Submissions of the Friends of the Northumberland Strait in Response to the call for Public Comments on the Environmental Assessment of the Replacement Effluent Treatment Facility Project

James Gunvaldsen Klaassen
and Sarah McDonald
Ecojustice
1801 Hollis Street, Suite 520
Halifax, NS B3J 3N4

March 8, 2019

ecojustice

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1. Overview

1. This submission is filed on behalf of our client, the Friends of the Northumberland Strait, a society registered under Nova Scotia's Society Act with a membership of Pictou residents and the Pictou County area community.

2. Northern Pulp Nova Scotia (NPNS) proposes a project to build a new Effluent Treatment Facility (ETF or the "project"). Please consider these submissions, and the accompanying appendices, as the comments of the Friends of the Northumberland Strait in relation to the Environmental Assessment (EA) process for the ETF.

3. The NPNS ETF is ill-conceived and is designed to externalize to the environment the costs of NPNS's enterprise. NPNS rejects the significant and viable alternative of installing a closed-loop system on the basis that it cannot make the same level of profits as it does with its current process.

4. The risks to the environment are too great to permit this project to proceed. Further, the application is a paper exercise rather than an in-depth investigation of an important ecosystem, and is missing crucial information. The application is based on inadequate and second-hand and often out-dated research and investigation, and relies on inappropriate methodology to make defective predictions. No significant effort was expended to measure and determine the actual conditions in the affected ecosystems. NPNS does not understand the environment in which it seeks to operate, it understates the risks of the project, and overstates the effectiveness of its proposed mitigation measures.

5. The risks are significant and NPNS has failed to discharge its burden to show that the project will not cause significant environmental effects or adverse effects, or that any such effects can be mitigated. The project proposes to discharge a daily average of 62,000,000 litres, and up to a maximum of 85,000,000 litres, of pulp mill effluent every day into the middle of the only herring spawning area in the southern Gulf of St. Lawrence. It will discharge effluent directly into lobster fishing grounds for as many as 82 local fishers, and could affect the lobster fishery for as many as 1800 lobster fishers from Nova Scotia and Prince Edward Island in the Strait.

It could have significant effects on the marine ecosystem and foundational species of the ecosystem, such as planktonic species, invertebrate and fish larvae, subtidal and intertidal invertebrates and plants, forage species and other marine organisms. There could also be significant human health impacts from air emissions, from contamination of freshwater, drinking water and soils, and from contamination and bioaccumulation of toxic substances in marine species and marine foods.

6. Further, the Minister has selected an EA process under the *Environment Act*[1] that does not permit sufficient time for the public and other affected groups and individuals to assess the voluminous materials filed by NPNS. NPNS has had four years to prepare this set of materials, but the public is given 30 days to respond. Provincial officials have worked closely with NPNS to develop the reports appended to NPNS's submission, but NPNS has chosen not to release them to the public until the last minute. Significant taxpayer funding has been provided to NPNS to develop the submission, but no corresponding funding has been made available to the public to hire their own scientific experts to review this submission. The Minister has acknowledged the unfairness of this process to the public[2], but appears content to push the matter to a quick conclusion.

7. As discussed in detail below, ample evidence is before the Minister to allow her to conclude that the project should be rejected, as it is likely that it will cause adverse effects or significant environmental effects that cannot be mitigated. Consequently the project should be rejected pursuant to section 34(1)(f) of the *Environment Act.*

8. In the alternative, the NPNS EA fails to provide information on many crucial aspects of the project. The Minister therefore does not have sufficient information and analysis before her to permit her to allow the project to proceed. Evidence of potential adverse effects or significant environmental effects that cannot be mitigated have been presented to the Minister from many sources within this EA process. Due to the multiple information gaps, lack of examination of significant issues, and lack of scientific support for the premises put forward by NPNS, as well as failure to provide evidence of mitigation measures and their effectiveness,

[1] *Environment Act*, S.N.S. 1994-1995, c. 1, and Part IV.
[2] Jean Laroche, "Northern Pulp's plans for pipeline, effluent treatment plant now public," CBC, February 7, 2019 [Appendix H-9].

the Minister must order a full environmental assessment report pursuant to section 34(1)(c)[3]. This is the only means by which the Minister or a Panel can comprehensively and objectively assess project impacts and permit full and informed public participation in this process, given the potential for harm posed by this project.

2. Introduction

a) Friends of the Northumberland Strait

9. The Friends of the Northumberland Strait (FONS) are community members from Pictou and surrounding area with a deep connection to Pictou County and the Northumberland Strait. Some have lived in Pictou County for their whole lives, and their families have lived here for generations. Others are drawn here to live or summer in the Strait area. They are business people, professionals and fishing families united by their love for this area and for the beautiful and sensitive ecosystem of the Northumberland Strait.

10. FONS began in 2017, and was formally incorporated as a society in 2018. Its members came together as it became clear that NPNS planned to solve its need for a new effluent treatment facility by discharging its treated effluent directly into the Northumberland Strait. Since the *Boat Harbour Act* prohibits NPNS from using Boat Harbour past January 31, 2020, it was easiest, and cheapest, for NPNS to get rid of its pulp mill effluent by treating it on-site, then piping it off its property and discharging it into the Northumberland Strait. FONS members were appalled by the prospect of up to 85,000,000 litres of hot treated effluent containing harmful chemicals, being pumped directly and continuously into the Strait every day. They are very concerned about the potential for serious and irreversible damage to Pictou County's air, soil, freshwater, wetlands and wildlife, and to the Strait ecosystem and the local economy it supports, including fisheries and tourism.

11. Since its formation, FONS has made substantial efforts to promote public awareness of these issues and provide opportunities to debate them, and to empower the public to communicate their concerns. FONS has hosted and supported public meetings, public rallies, media releases and briefings, and has operated a website and a dedicated Facebook page, in an attempt to

[3] *Environment Act*, S.N.S. 1994-1995, c. 1, s. 34(1)(c).

increase understanding of the project, and to better understand the community's concerns. FONS has also made presentations to local municipal councils, various political parties, community groups, and the Prince Edward Island Legislature's Standing Committee on Agriculture and Fisheries.

12. FONS' concerns will be set out in detail below. In summary, FONS submits that:

(i) The registration materials filed by NPNS are incomplete and do not comply with the requirements of section 9(1A) of the *Environmental Assessment Regulations*. The Project is therefore improperly registered and the current EA process is a nullity.

(ii) The ongoing EA process is inadequate and unfair, as it does not allow the public to assess the large amount of scientific documentation and conduct a comprehensive review of the information contained in NPNS's EA submission. NPNS failed to hold promised public information sessions, and held back from the public the majority of the scientific studies until registration;

(iii) The EA submission, although lengthy, lacks critical information, or sufficient detail, in crucial areas such as:

(a) The composition of the effluent to be discharged into the Northumberland Strait;
(b) Studies showing actual composition of raw effluent produced at the NPNS facility;
(c) Studies showing the nature and frequency of process interruptions and disruptions, leaks and spills at the NPNS facility and the impacts of same on effluent composition;
(d) Studies showing that the proposed ETF, which is not yet constructed, can and will in fact reliably and consistently discharge effluent which will meet any particular parameter, or whether it will meet the parameters which form the basis of the discussion in the EA submission;
(e) Studies and analyses regarding mercury issues associated with the project, including methylmercury, mercury and other metals in effluent, and mercury contamination of the NPNS/Canso site;
(f) Baseline data specific to either Caribou Harbour or Caribou Channel;
(g) Professional ecosystem studies in relation to the marine and terrestrial environments;
(h) Thorough and accurate modelling to determine mixing capabilities in Caribou Channel and how the effluent will fare as it circulates in the Strait;
(i) Analysis or engineering study of the impacts of ice scour on buried HDPE pipe or diffusers;
(j) Drawings or mapping/chart coordinates showing the precise pipeline route on the shore, in Caribou Harbour, and in Caribou Channel;
(k) Air emissions data from current operations from all stacks and vents; and
(l) Clear, effective and comprehensive mitigation plans, with substance and that take into account actual conditions in the local environment.

The above defects, individually and collectively, show that the NPNS EA is incomplete, based on inaccurate information and unproven assumptions, and is not supported by credible scientific studies in relevant disciplines.

(iv) Once the above defects are noted, the self-serving summary table in NPNS's Executive Summary, which provides a uniform assessment of the project as having no "significant residual environmental effects" clearly strains credibility. The conclusion fails to take proper account of the nature of pulp mill effluent, the gaps in the information presented by NPNS, and the sensitive environments into which it may be discharged. It is demonstrable proof of a failure to provide a balanced and objective submission of environmental impacts for the Minister's review.

b) Reasonable Apprehension of Bias

13. On February 12, 2019, on behalf of FONS, we submitted a package both to the Minister and to the Environmental Assessment Branch, at the address given for submission of public comments on this EA. That submission asked the Minister to recuse herself from the NPNS ETF EA process due to a significant conflict of interest. On March 6, 2019 we received a letter dated March 5, 2019 from the Minister, advising that the Minister would not be recusing herself from this EA process. We maintain the position, set out in our letter of February 12, 2019, that the Minister's involvement in the EA process gives rise to a reasonable apprehension of bias. As a result, the Minister must recuse herself in order to maintain public confidence and to ensure the integrity of the process.

c) Context of NPNS ETF EA

(i) Boat Harbour and past effluent discharges

14. A central premise of the NPNS' submission, and its public statements about the impacts of its operations on the Northumberland Strait, is the following:

> Since effluent has been discharging into the Strait for the past 50 years, it will cause no change to the ecosystem by discharging effluent in a new location.[4]

[4] Email string Nov. 15-17, 2017, NP response to media questions, (Appendix H-8).

15. The environmental cost of discharging effluent into a living ecosystem has, to date, been borne largely by the Boat Harbour Basin and by the residents of Pictou Landing First Nation. The environmental damage to Boat Harbour from continuous effluent discharge is an environmental disaster, which cannot be truly quantified in monetary terms.[5] However, using Boat Harbour as the effluent dump for 50 years has resulted in an estimated clean-up cost of over $200 Million. Fortunately, the abuse of Boat Harbour is scheduled to end on January 31, 2020.

16. Now, the proposed plan will dump effluent into the marine environment at outfall location CH-B in the Caribou Channel, just outside Caribou Harbour.

17. KSH Solutions Inc., pulp and paper engineering consultants advising NPNS, have touted the benefits of Boat Harbour Basin in reducing the harmfulness of NPNS effluent currently discharged at Point C.[6] In a KSH power point obtained via the *Freedom of Information and Protection of Privacy Act*, KSH describes Boat Harbour Basin as "[a] large, natural final polishing/stabilization basin [which] follows prior to release to the Northumberland Straight [sic]."[7] This "final polishing/stabilization basin" provides a "settling effect…prior to Point D, so the impact on marine environments is even less pronounced."[8] The slow flushing time in Boat Harbour Basin allows for settling of solids, cooling of effluent, and performs other useful filtering functions. By the time the effluent enters the Strait, it has already dumped a lot of its toxic cargo in Boat Harbour Basin. Nova Scotia taxpayers will be paying over $200 Million to clean that up.

18. The contribution of Boat Harbour Basin in the effluent discharge process is also acknowledged by NPNS officials. The Mill's Technical Manager said in an email dated November 29, 2017:

[5] Boat Harbour Remediation Project Handout, (Appendix H-11).
[6] Point C is the point at which effluent leaves the current treatment facility and enters Boat Harbour Basin. After it cools and polishes, it flows out of the Basin at Point D. The locations of Points C and D are shown on the aerial photo at p. 10, Figure 2.1-1 NPNS EA Submission.
[7] KSH Power point, excerpt (Appendix H-4).
[8] KSH Power point, excerpt (Appendix H-4).

> Some say effluent quality [with new ETF] will be worse than today because of all the polishing that is happening across the [Boat Harbour] basin – and they are correct to some extent.[9]

19. In the same email, the Technical Manager goes on to say:

> Effluent temperature [of new ETF] – hotter than now [Boat Harbour] big basin provides a lot of natural cooling today[10]

20. As acknowledged by engineering consultants and by NPNS, Boat Harbour has taken the brunt of the effluent discharge to date and there will be no comparable "buffer zone" effect on the effluent when discharged at CH-B. The only thing standing between the raw effluent from the mill and the fish spawning grounds, active fishing grounds, and marine ecosystem, is the proposed ETF, which remains largely a mystery, both in terms of what it is capable of doing, and what it will actually be used for by NPNS.

21. The NPNS EA does not answer the question of what will happen to the substances currently settling out in Boat Harbour. The EA does not provide objective scientific evidence as to the likelihood that the proposed ETF will, or can, actually change the effluent into a harmless and benign substance, or that it will meet any standard or will in fact be "better quality" than what is currently discharged into in Boat Harbour. The Minister is given only assumptions as to water quality characteristics, with no proof that these are realistically achievable or that NPNS actually intends to achieve them. The NPNS proposal is based on the premise that the effluent discharge into the Strait should simply be allowed to proceed, and that monitoring will be conducted to verify its safety at some vague point in the future. In perhaps two years after effluent discharge begins, someone will assess whether it has caused any problems. This approach is a recipe for environmental harm, and runs contrary to an underlying principle of the *Environment Act*, which requires that:

> the precautionary principle will be used in decision making so that where there are threats of serious or irreversible damage, the lack of full scientific certainty shall not be used as a reason for postponing measures to prevent environmental degradation…[11]

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[9] Email dated Nov. 29, 2017 from NPNS Technical Manager to NS TIR, p. 4 of 5 (Appendix H-10).
[10] Email dated Nov. 29, 2017 from NPNS Technical Manager to NS TIR, p. 4 of 5 (Appendix H-10).
[11] *Environment Act, supra*, ss2(a), b(ii); *Sorflaten v Nova Scotia (Minister of Environment),* 2018 NSSC 55 at para 38.

22. As discussed below, no testing or test results have been provided to show the effluent's composition. Most of the substances contained in raw effluent are not discussed, and their impacts on the marine, freshwater, terrestrial and atmospheric environments are not analysed. Likewise, as will be discussed further below, the Stantec modelling used to predict the effluent mixing and transport in the marine environment has fundamental flaws, and must be disregarded.

(ii) EA enforcement and compliance issues

23. In 2017, Nova Scotia's Auditor General issued a report in relation to environmental assessments conducted under the *Environment Act*.[12] From 2013 to 2016, of the 54 environmental assessments conducted, 53 were approved, amounting to an approval rate of over 98%.[13] This figure is of great concern, as it suggests that the environmental assessment process in Nova Scotia is a process by which projects receive a rubber stamp rather than a thorough and objective environmental review.

24. The Auditor General went on to conclude that, where environmental assessments are approved on conditions, it is likely that Nova Scotia Environment (NSE) does not monitor compliance or verify that the required conditions have been satisfied. Within a sample of 53 EA conditions examined by the Auditor General, 23 were not verified or monitored for compliance. As the Auditor General says:

> "Without monitoring, Nova Scotia Environment does not know if the terms and conditions of approved projects are effective in reducing impacts on the environment."[14]

25. Where there is a failure to monitor compliance with EA approval conditions, a failure to enforce conditions, or a failure to monitor whether an approved project is actually causing environmental harm, the risks of harm from such projects increases dramatically.

[12] Report of the Auditor General, November 2017, Chapter 4 Environmental Assessments, pp. 43-53 (Appendix H-12)
[13] Report of the Auditor General, para. 4.2, p. 45 (Appendix H-12)
[14] Report of the Auditor General, para. 4.5, p. 46 (Appendix H-12)

(iii) The Province's agreements with NPNS cannot fetter the Minister's discretion in making a decision on the EA

26. As set out in our February 12, 2019 submission, NPNS has stated publicly that its various contracts with the Province should be determinative of the results of administrative approval processes. In its 2015 appeal of the Industrial Approval issued by the Province, NPNS stated that "[g]overnment cannot arbitrarily revoke Northern Pulp's contractual rights under the Agreements with the Province by way of an administrative approval process."[15] Acceding to such an argument would constitute an unlawful fettering of the Minister's discretion in this matter. The Minister must consider contractual arrangements with NPNS to be an irrelevant consideration in this process, and make her decision under section 34(1) of the *Environment Act* without regard to such agreements.

(iv) The Minister must consider all possible outcomes under section 34(1) of the *Environment Act*, including rejection of the project

27. The Minister must consider all possible outcomes under section 34(1) of the *Environment Act*, including whether to reject the project outright.[16] Correspondence between NPNS and the NSE demonstrates a predisposition to approve the project, and shows that rejection of the proposal is not being considered by provincial officials.

28. For instance, by email dated November 14, 2017, NPNS wrote to the Deputy Minister of NSE requesting it be granted "regulatory certainty" by engaging in negotiations on a future IA [Industrial Approval] prior to the EA.[17] By letter of November 30, 2017, 14 months before the EA application was even submitted, NSE's Eastern Regional Director agreed to begin negotiations as to the terms of the Industrial Approval that would follow the EA.[18] The correspondence further shows that both parties appear to assume that the purpose of the EA is

[15] Letter from Ms. Terri Fraser, Technical Manager Northern Pulp Nova Scotia Corporation, to the Honourable Randy Delorey, Minister of Environment, 9 April 2015 (Appendix H-21).
[16] *Environment Act*, S.N.S. 1994-1995, c. 1, s. 34(1)(c).
[17] Email from Bruce Chapman to Deputy Minister Frances Martin, November 14, 2017 (redacted as provided by FOIPOP) (Appendix H-23)
[18] Letter to Bruce Chapman, Northern Pulp, from Paul Keats, Eastern Regional Director NSE, dated 30 November 2017 (Appendix H-20)

to establish effluent discharge levels[19], and not to determine whether the project will cause adverse effects or significant environmental effects that cannot be mitigated. The NSE letter agrees to negotiate those Industrial Approval items that "are not impacted by the future environmental assessment process".[20] The NSE official fails to recognize that all items in an Industrial Approval would potentially be impacted by the EA, since all terms are contingent upon EA approval, and all terms would be unnecessary should the ETF project be rejected.

29. If rejection of the project were actually under consideration by NSE, this would have been reflected in the correspondence. Taking a closed-minded approach is contrary to the Minister's duty in coming to a decision in this matter.

3. The environmental assessment scheme: the *Environment Act* and the *Environmental Assessment Regulations*

30. The current review of NPNS's proposed ETF is proceeding as a Class 1 environmental assessment. The review and decision-making process is governed by the *Environment Act* and the *Environmental Assessment Regulations* ["*EA Regs*"].

31. NPNS's proposed ETF was registered for EA on February 7, 2019. As per s 34(1) of the *Environment Act* and s 13(1) of the *EA Regs,* the Minister has 50 days from the registration date to determine whether:

(1) additional information is required;
(2) a focus report is required;
(3) an environmental-assessment report is required;
(4) all or part of the undertaking will be referred to alternate dispute resolution;
(5) a focus report or an environmental-assessment report is not required, and the undertaking may proceed; or
(6) the undertaking is rejected because of the likelihood that it will cause adverse effects or significant environmental effects that cannot be mitigated.[21]

[19] NSE letter to Bruce Chapman of 30 Nov 2017, *supra*, at page 1: "The upcoming environmental assessment will also be used to establish those [effluent discharge concentration] limits." (Appendix H-20)
[20] NSE letter to Bruce Chapman of 30 Nov 2017, *supra*, at page 2. (Appendix H-20)
[21] *Environment Act,* SNS 1994-95, c 1 at s 34(1).

32. The *EA Regs* provide additional details on the parameters of the Minister's decision. Section 13(1) of the *Regs* specifies the circumstances in which the Minister may select each of the options listed in s 34(1) of the *Environment Act* as follows:

> 13(1) No later than 50 days following the date of registration, the Minister shall advise the proponent in writing of the decision under subsection 34(2) of the Act
>
> (a) that the registration is insufficient to allow the Minister to make a decision and additional information is required;
> (b) that a review of the information indicates that there are no adverse effects or significant environmental effects which may be caused by the undertaking or that such effects are mitigable and the undertaking is approved subject to specified terms and conditions and any other approvals required by statute or regulation;
> (c) that a review of the information indicates that the adverse effects or significant environmental effects which may be caused by the undertaking are limited and that a focus report is required;
> (d) that a review of the information indicates that there may be adverse effects or significant environmental effects caused by the undertaking and an environmental-assessment report is required; or
> (e) that a review of the information indicates that there is a likelihood that the undertaking will cause adverse effects or significant environmental effects which are unacceptable and the undertaking is rejected.[22]

33. As per s 13(1)(b), the Minister can only approve an undertaking under s 34(1) of the *Environment Act* if she concludes that it *would not cause any* adverse effects or significant environmental effects, or that any such effects would be mitigable.

34. In order for an adverse effect or a significant environmental effect to be adequately mitigated for the purposes of s 13(1)(b) of the *EA Regs,* the effect in question must be mitigable to the point that its impact is less than "limited". This is based on the combined effect of subsections 13(1)(b) and (c) – as per subsection 13(1)(c), if the undertaking may cause even "limited" adverse effects or significant environmental effects, the Minister must order a focus report.

35. As a result, the Minister is only authorized to approve NPNS's proposed ETF under s 34(1) of the *Environment Act* if she is certain that there will be no adverse effects or significant environmental effects, or that such effects can be mitigated to the extent that they all but

[22] *Environmental Assessment Regulations,* NS Reg 26/95 ["*EA Regs*"].

disappear. This is consistent with the purposes of the *Environment Act,* including upholding the precautionary principle and maintaining environmental protection.[23]

36. NPNS employs various definitions of what it terms a "significant adverse residual environmental effect" when evaluating the proposed ETF's potential impact on "Valued Environmental Components" ("VECs"). These definitions do not appear anywhere in the *Environment Act* or the *EA Regs,* and the Minister should exercise due caution in relying on them when determining whether the proposed ETF will cause significant environmental effects. NPNS does not propose a definition of "adverse effect" or any similar term when evaluating the project's potential impacts on human health.

37. As will be outlined in detail in the following sections, NPNS's EA registration materials are far from sufficient to allow the Minister to approve the proposed ETF. The Minister cannot, and should not, rely on NPNS's "vague assurances" of mitigation and further studies to approve a project that could have widespread and devastating impacts on the Province's environment, economy, and rural communities.[24]

4. Procedural Issues

38. It is trite to state that, as a general rule, there is "[…] a duty of procedural fairness lying on every public authority making an administrative decision which is not of a legislative nature and which affects the rights, privileges or interests of an individual."[25]

39. The current EA process has been marred by numerous procedural defects, which have resulted in a violation of the duty of procedural fairness. These procedural defects have undermined the public's ability to fully engage in the EA, contrary to the *Environment Act*'s explicit goal of "providing access to information and facilitating effective public participation in the formulation of decisions affecting the environment […]".[26]

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[23] *Environment Act, supra*, ss2(a), b(ii); *Sorflaten v Nova Scotia (Minister of Environment),* 2018 NSSC 55 at para 38.
[24] *Taseko Mines Ltd v Canada (Minister of the Environment),* 2017 FC 1099 at paras 123-124.
[25] *Cardinal v Kent Institution,* [1985] 2 SCR 643 at 653.
[26] *Environment Act, supra*, ss2(h).

40. The procedural flaws impacting the ongoing EA are examined in detail in the following sections.

a) Barriers to public participation

41. NPNS has submitted a 614-page Registration Document to the Province, along with 18 Appendices. In total, there are almost 1,700 pages of materials for the public to review. Many of the documents included with NPNS's materials contain dense scientific and technical information which can be time consuming for a layperson to digest.

42. The ongoing Class 1 EA process provides only 30 days for the public to review and comment on NPNS's materials. This is far from an adequate comment period. Minister Miller herself has acknowledged that this process is defective, stating "I don't know that the public is really going to be able to fully digest everything that's been submitted."[27]

43. The impacts of this inadequate comment period on the public's ability to review and understand the EA materials are further aggravated by NPNS's failure to engage with the public in a thorough and transparent manner prior to registering its project for EA.

44. Both the *EA Regs* and NSE policy documents explicitly contemplate a proponent's responsibility to engage with members of the public who may be impacted by a proposed project, and to attempt to understand and address their concerns. For instance, when formulating a decision under s 34(1) of the *Environment Act,* the Minister must consider "[…] concerns expressed by the public and aboriginal people about the adverse effects or the environmental effects of the proposed undertaking."[28] Furthermore, in its "Citizen's Guide to Environmental Assessment," NSE declares that "[p]ublic participation is vital to the success of environmental assessment."[29]

45. NPNS and/or its representatives made numerous promises with respect to public engagement prior to registering its EA materials, many (if not most) of which went unfulfilled. NPNS held

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[27] Jean Laroche, "Northern Pulp's plans for pipeline, effluent treatment plant now public," CBC, February 7, 2019 [Appendix H-9].
[28] *EA Regs, supra,* at s 12(c).
[29] Nova Scotia Environment, *A Citizen's Guide to Environmental Assessment* (Halifax, NS: Nova Scotia Environment, 2017) at p 4. Link to: https://novascotia.ca/nse/ea/docs/EA.Guide-Citizens.pdf

two public "Open House" sessions, one in December 2017 and the other in January 2018.[30] Following the January 2018 Open House, Dillon Consulting (a consulting firm retained by NPNS to conduct the EA) committed in writing to conducting another series of Open House sessions in the spring of 2018.[31] Similarly, in July 2018 Dillon Consulting committed to holding another Open House session in the fall of 2018.[32] To the best of FONS' knowledge, neither of these commitments were upheld. As a result, the most recent public engagement session conducted by NPNS was over a full year before its EA materials were registered with the Province.

46. This failure to uphold explicit commitments made to members of the public is all the more egregious in light of the significant changes made to the ETF project beginning in October 2018. In July, 2018, NPNS announced that the pipeline route it had originally contemplated was not feasible. The planned route and outfall were therefore altered dramatically. We have been informed that Bruce Chapman, General Manager of the NPNS mill, made a verbal commitment to Krista Fulton of FONS on August 31, 2018 in a phone call at 11:34 a.m. that additional Open House sessions would be held regarding the new pipe route and outfall location. Mr. Chapman advised Ms. Fulton that: "Yes, we will have another Open House because that is what we promised."[33] However, despite this promise and NPNS's previous commitments, there were no public meetings held between the time the new route was selected and the date on which the EA materials were submitted to the Province.[34] A public information session has therefore never been held with respect to the new Caribou route and the CH-B outfall.

47. In addition, NPNS and/or its representatives committed on numerous occasions to releasing specialist studies completed as part of the EA to the public upon their completion.[35] These

[30] NPNS website Project Materials page, accessed January 31, 2019 (Appendix H-25).
[31] Letter from Annamarie Burgess to Jill Scanlan, dated January 22, 2018 (Appendix H-24)
[32] Letter from Annamarie Burgess to Jill Scanlan, dated July 9, 2018 (Appendix H-25).
[33] Personal conversation between Bruce Chapman and Krista Fulton, August 31, 2018.
[34] Brendan Ahern, "Lack of public consultation ahead of Northern Pulp's submission of Environmental assessment sparks backlash," The News, January 16, 2019 [Appendix H-6].
[35] Letters from Annamarie Burgess to Jill Scanlan, appendices H-24 and H-25; Northern Pulp, "Replacement Effluent Treatment Facility" webpage, accessed January 10, 2019 [Appendix H-28].

studies were to be made available on the ETF project website.[36] However, as of the date of its EA registration NPNS had only made a small portion of its specialist studies available to the public. Specifically, of the 18 Appendices included with its EA materials, NPNS only made two full appendices and three partial appendices available on its website prior to registration.[37]

48. NPNS's failure to make the vast majority of its specialist studies available to the public prior to the EA registration would be understandable if the studies in question had not been completed until the registration date (February 7, 2019). However, this is far from the case. The chart below lists all of the studies included in NPNS's EA materials and the dates on which they were completed.

Appendix	Title of study	Date	Posted on NPNS project website?
A	Joint Stock Record	November 8, 2018	**No**
B	NPNS Market Profile	January 26, 2018	Yes
C	Technology Selection Report	July 1, 2017	Yes
D	Veolia AnoxKaldnes Reference List	January 1, 2018	**No**
E	E1 – Stantec Final Caribou Discharge Receiving Water Study	December 19, 2018	Yes – this addendum was finalized on December 19, 2018, but not posted until mid-January 2019
	E2 – Stantec Response to Questions	January 5, 2018	Yes
	E3 – Stantec Preliminary Receiving Water Study Effluent Treatment Plant Replacement	August 11, 2018	Yes
F	Description of Marine Pipeline Construction	January 25, 2019	**No**

[36] The website address, at the time, was www.northernpulpeffluenttreatmentfacility.ca – This website still exists but you are redirected to another address.
[37] Northern Pulp, "Project Materials" webpage, accessed January 31, 2019 [Appendix H-26].

Appendix	Title of study	Date	Posted on NPNS project website?
G	Proposed EEM Program	January 2019 (date not specified)	**No**
H	Proposed Follow Up and Monitoring Program	January 2019 (date not specified)	**No**
I	I1 – Public Engagement Materials	December 2017; January 2018 (dates not specified)	Yes
	I2 – What We've Heard Summary Report	March 2018 (date not specified)	Yes
	I3 – Record of Project Website	January 16, 2019	**No**
	I4 – Stakeholder Meeting Minutes	December 21, 2017; February 8, 2018; February 20, 2018; October 22, 2018	**No**
J	J1 – 2016 EEM with Appendices	March 2016 (date not specified)	Yes
	J2 – What is Environmental Effects Monitoring	Undated	**No**
K	K1 – Stantec Air Dispersion Modeling Study of Replacement Effluent Treatment Facility	January 21, 2019	**No**
	K2 – Stantec Memo re Hoffman Report	June 15, 2018	**No**
L	L1 – Summary of Baseline Noise Monitoring	Undated	**No**
	L2 – Baseline Noise Monitoring Results for R1 – Maritime Oddfellows Home	December 18, 2017	**No**
	L3 – Baseline Noise Monitoring Results for R2 – 12 Birch Lane	December 17, 2017	**No**
	L4 – Baseline Noise Monitoring Results for R3 – 1220 Loch Broom Loop	December 18, 2017	**No**

Appendix	Title of study	Date	Posted on NPNS project website?
	L5 – Baseline Noise Monitoring Results for R4 – 108 Grant Abercrombie Branch Road	December 17, 2017	**No**
	L6 – Temperatures During Noise Monitoring Event	December 17, 2017	**No**
M	M1 – Watercourse Fish and Habitat Field Data Sheets	June 12, 2018	**No**
	M2 – Watercourses in the Vicinity of the Project Footprint Area Photo Plate	December 3, 2018	**No**
	M3 – Summary of General Physical Characteristics of Predicted Watercourse Crossings	December 3, 2018	**No**
	M4 – Maxxam Laboratory Certificates	December 17, 2018	**No**
	M5 – Middle River of Pictou Water Availability – Final Report	December 17, 2015	Yes
N	N1 – Potential Priority Animal Species	November 15, 2018	**No**
	N2 – Potential Priority Plant Species	November 15, 2018	**No**
O	O1 – Wetland Delineation Data Forms	June 12, 2018	**No**
	O2 – WESP_AC Functional Assessment Result Scores	Undated	**No**
P	Plant Data	Undated	**No**
Q	Q1 – Avian Survey Locations	June 30, 2018	**No**

Appendix	Title of study	Date	Posted on NPNS project website?
	Q2 – Map of MBBA Square 20NR25	April 13, 2006	**No**
	Q3 – MBBA Data Summary for Square 20NR25	November 22, 2018	**No**
	Q4 – Map of MBBA Square 20NR26	April 13, 2006	**No**
	Q5 – MBBA Data Summary for Square 20NR26	November 22, 2018	**No**
	Q6 – Results of all Avian Survey Efforts	June 20, 2018	**No**
R	Scientific Literature BKME Effects on Lobster	August 27, 2018 (amended January 25, 2019)	**No**

49. Most, if not all, of the listed studies could easily have been posted on the ETF project website prior to the project's registration for EA on February 7, 2019. Indeed, most of the studies were completed months before the registration date. It is unclear why NPNS chose not to post these studies on its website for public review upon their completion, as per its previous commitment. NPNS's failure to do so has unquestionably undermined the public's ability to review, understand, and provide thoughtful and fulsome comments on the EA materials.

50. In FONS's respectful submission, these clear procedural defects have resulted in violations of the duty of procedural fairness. Furthermore, as per subsection 12(d) of the *EA Regs,* when formulating a decision under subsection 34(1) of the *Environment Act,* the Minister is required to take into account "steps taken by the proponent to address environmental concerns expressed by the public and aboriginal people." NPNS's failure to uphold even its most basic commitments to engage the public illustrates that it has not listened to the public's concerns, let alone taken steps to address them. In light of this fundamental procedural flaw, the Minister cannot approve the proposed ETF.

b) Incomplete Registration Document

51. Subsection 9(1A)(b) of the *EA Regs* require that an EA registration document must include certain basic information. As detailed below, NPNS's Registration Document does not fulfill the requirements of subsections 9(1A)(b)(ix), (x) or (xii).

(i) Section 9(1A)(b)(ix): A description of the proposed undertaking

> NPNS describes the proposed ETF at Section 5.0 of its Registration Document. However, its project description fails entirely to address at least one significant component.
>
> KSH Consulting's *Technology Selection Summary,* at Appendix C to NPNS's Registration Document, speaks to the inclusion of an oxygen delignification system as part of the new ETF. However, there is no mention of oxygen delignification anywhere in the Registration Document. Furthermore, NPNS has previously stated that oxygen delignification would not be installed as part of the new ETF, but would be an "anticipated future upgrade" that would occur sometime after the new ETF became operational.[38]
>
> If an oxygen delignification system will be included as part of the new ETF, then NPNS must address this component as part of its project description as per subsection 9(1A)(b)(ix) of the *EA Regs.* If not, then NPNS must clarify that the KSH *Technology Selection Summary* does not accurately reflect the components of the proposed ETF.

(ii) Section 9(1A)(b)(x) Environmental Baseline Information

> NPNS's materials contain no environmental baseline information specific to the receiving environment, i.e.: Caribou Harbour and Caribou Channel.[39] And, as listed below in section 8, a large number of other baseline studies are noted as necessary but have not been completed. This baseline information is fundamental for an understanding of the receiving environments and for meaningful environmental effects monitoring.[40] NPNS has failed to satisfy this requirement.

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[38] Dillon Consulting, *Northern Pulp Nova Scotia – Replacement Effluent Treatment Facility – Information Submission to CEAA*, April 2018, excerpt [Appendix H-22].
[39] This is acknowledged in the NPNS EA submission at **Section 8.11.2, p 337**
[40] MacKay, A.A., *Northern Pulp's Effluent Disposal Plans – Issues and Answers*, February 2019 (MacKay commentary)(Appendix C-1), regarding the necessity of conducting species and chemical composition baseline surveys.

(iii) Section 9(1A)(b)(xii): All sources of any public funding for the proposed undertaking

> NPNS purports to fulfill this requirement at page 1 of its Registration Document, where it states as follows: "[a]t the date of Registration, the Province of Nova Scotia has made contributions to the cost planning and design of the project."
>
> This meagre description clearly does not identify all sources of any public funding for NPNS's proposed ETF. NPNS fails to even specify which provincial Departments provided the funds to which it refers. This cannot, and does not, fulfill the requirements of subsection 9(1A)(b)(xii).

52. In light of the above, the ETF project should not have been registered for EA. In the alternative, these omissions demonstrate that the Minister does not have sufficient information to approve the proposed ETF.

5. Closed Loop is a Viable Alternative to the ETF

53. At section 4.1 of its EA materials, NPNS briefly discusses alternatives to the project. FONS is of the view that a closed-loop system remains a viable choice compared to the proposed ETF, from an economic and environmental perspective.

54. A closed-loop effluent system is the only environmentally viable solution in this situation. A closed loop system would not discharge effluent into the environment and would allow the Mill to continue to produce pulp for the market.

55. NPNS retained Brian McClay and Associates to prepare a Global Market Profile[41] to look at whether NPNS could change from its current Northern Bleached Softwood Kraft production ("NBSK"), which produces effluent discharges into the environment, to a closed loop system. NPNS says that the Market Profile concludes that changing its production process to a closed loop system would mean that the mill would not "remain competitive", and that "NPNS must continue to operate by producing NBSK to be economically viable."[42]

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[41] Brian McClay and Associates, Global Market Profiles: NBSK, UKP & BCTMP, NPNS EA Submission, at Appendix B (the "Market Profile"). The terms of the retainer are not disclosed, and there is no indication of what information came directly from NPNS and the degree of independent analysis performed by Brian McClay and Associates.

[42] NPNS EA Submission, Registration Document, section 4.1, p. 26.

56. In fact, the Market Profile does not reach such conclusions, and states only that the current process is "the most competitively viable option by far".[43] Presumably, this means that the current process yields the highest profits.

57. The Market Profile does not say that changing production to a closed loop system would be unprofitable for NPNS. Rather, the Market Profile says simply that NPNS would have to compete in new markets and, in the case of Bleached Chemi-Thermo-Mechanical Pulp production, would require new equipment and would need to address electricity demand issues.[44] Whether this would be a real obstacle remains to be seen, but the Market Profile does not mention that, at present, NPNS produces its own power to satisfy 90% of its current electricity requirements[45] and that it is "almost self-sufficient in energy."[46] NPNS does not wish to make such an investment to modernize its operations and eliminate its effluent discharges. NPNS wants to characterize the solution as a stark choice between NPNS continuing to make its current profits and offloading the environmental problems to the Northumberland Strait, or closing the Mill entirely. This is a false choice and an oversimplification of the market and the choices facing NPNS. The Market Profile demonstrates that other options exist which are more environmentally acceptable and may also be economically viable.

58. It is also noted that the Market Profile provides no assessment of the economic costs to the taxpayers in relation to construction of the proposed ETF, or the economic cost to the community, or the environment, of current operations and the ETF. The Market Profile limits its scope solely and unquestioningly to NPNS profit margins and ignores the question of the economic/environmental burden externalized by NPNS operations.

59. Other potential alternatives or treatments, including evaporation, were never examined in any meaningful way. Discharge of effluent into the Strait was the only alternative given any serious consideration.

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[43] Market Profile, at page 2 "summary".
[44] Market Profile, at page 9.
[45] NPNS webpage, NPNS Operations Today, "Facts", at http://www.paperexcellence.com/npns-operations-today , accessed 28 Feb. 2019.
[46] NPNS webpage, NPNS Environment, at http://www.paperexcellence.com/npns-environment , accessed 28 Feb. 2019.

60. The arguments against a closed-loop system are not compelling, and can be answered via a change in product line. Given the environmental risks posed by this project, a closed-loop system is the best choice for the environment, the mill and the region.

6. Effluent composition

61. Possibly the most significant gap in the materials filed by NPNS and its consultants, is the complete lack of objective scientific reporting and test results regarding the composition of the effluent that is to be discharged from the proposed ETF into the herring spawning grounds and Caribou Channel. The Minister must have reliable and precise information about the actual effluent that will be entering the environment, in order to assess the impacts it will have on the environment. Without this information, an assessment of environmental impacts cannot proceed as it is impossible and absurd to assess the impacts of an unknown substance.

62. The only information about the characteristics and composition of the effluent that will flow out of the proposed ETF is described as "expected water quality characteristics". It appears in tables set out in the Receiving Water Studies.[47] As well, no explanation is provided as to why the data in these tables differs from one table to another: the expected water quality value for Total Nitrogen (TN) is listed as 3.0 mg/L in the August 2017 Preliminary Study, but 6.0 mg/L in the December 2018 Addendum.

63. In a letter dated October 5, 2017, an NSE official wrote to the NPNS General Manager, agreeing that NPNS could use the water quality characteristic numbers (as reproduced in Table 3-2 of the August 2017 Receiving Water Study) for "the design of the project" but that this agreement did "not encumber the Minister's decision following the EA process". The official went on to say:

> NSE is aware that current data from the facility indicates possible exceedances at point C for many of the parameters. **As part of the EA, Northern Pulp must demonstrate that the new treatment facility can achieve the numbers highlighted… above**. If any of the parameters, including maximum flow, require modifications to the mill itself to achieve the volumes and concentrations modelled in the study, Northern Pulp must also

[47] Stantec, Preliminary Receiving Water Study, August 17, 2017, p. 3.54 Table 3-2, NPNS EA Submission, Appendix E3; and Stantec, Addendum Receiving Water Study, December 19, 2019, p. 17, Table 3.2, NPNS EA Submission, Appendix E1.

> submit a plan to the Department indicating what changes are required to the Mill to achieve the maximum concentrations. [emphasis added][48]

64. The NPNS materials contain no assessment or studies done to demonstrate that the new treatment facility can achieve the assumed water quality characteristics. The Minister is asked to approve a project to construct a facility without being shown that it will work.

65. The Receiving Water Studies say that the expected water quality characteristics of the treated effluent were provided by KSH.[49] There are references to a KSH "brief" and report, and other KSH communications throughout the submission.[50] However, no report from KSH on the predicted effluent is provided. The only KSH-authored document is found at Appendix C, which is a Technology Selection Report. That report contains no information about the parameters of the effluent that will flow out of the diffusers into the marine environment, or that could leak out of breaks or ruptures in the pipe or at the ETF facility itself. There is an oblique reference to testing conducted in Sweden, but no results or report is provided.[51] NPNS has chosen not to provide any hard evidence that the effluent will achieve the parameters set out in its submission to the Minister, relying instead on hypothetical assumed parameters. This is a fundamental problem with the EA and is grounds for rejection of the entire submission.

66. NPNS's registration document expressly concedes that the information on which they rely is speculative and the assumptions untested.

> Due to uncertainty regarding effluent composition and approximate concentrations of substances present in the future treated effluent (which will not be verified until the project is operational), the identified candidate COPCs [chemicals of particular concern] in effluent are considered preliminary at this time.[52]

67. The EA registration demonstrates just how vague and speculative the information about the composition/characteristics of the effluent is, in the following statement:

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[48] Letter to General Manager, NPNS, from Supervisor of Environmental Assessment, NSE, dated October 5, 2017, p. 2 (Appendix H-15).
[49] Stantec, Preliminary Receiving Water Study, August 17, 2017, p. 3.54 Table 3-2, NPNS EA Submission, Appendix E3; and Stantec, Addendum Receiving Water Study, December 19, 2019, p. 17, Table 3.2, NPNS EA Submission, Appendix E1.
[50] NPNS EA Submission, Appendix E3, Stantec, Preliminary Receiving Water Study, August 17, 2017, section 2.1.3, p. 2.22 and "References" at p. 6.92; NPNS EA Submission, Section 9.2.4.2, p. 510.
[51] NPNS EA Submission, Section 4.2.1, p. 29. If testing was done, and was successful, one would assume that the results would be provided.
[52] NPNS EA Registration Document, Section 9.2.4.2, p. 506.

> While there are some uncertainties associated with the representativeness of the effluent chemistry characterization presented in Toxikos (2006) to the proposed future NPNS project effluent (as noted above), it is believed that there are sufficient similarities to state that the Toxikos (2006) information can serve as an indication of what may be expected in relation to NPNS project effluent composition/characteristics (KSH Consulting, personal communication).[53]

68. It is unusual to rely on a report from a mill which process different wood products and which discharges effluent into an entirely different ocean on the other side of the world, with different dynamics, temperatures etc., but not to provide a report summarizing and analyzing data from the actual mill that will be producing the effluent. As well, as has been noted elsewhere,[54] the mill being analysed by the Toxikos report was never built[55], so there is no way to compare those predictions with later actual results to determine the degree of accuracy of the predicted outcomes.

69. No attempt is made to explain the lack of data from NPNS or KSH regarding the precise effect of the ETF on the mill's effluent, despite the onus on NPNS to provide a complete set of information so the Minister can make a decision on the environmental impacts of the proposal.

70. The water quality characteristics assume that the components of the mill's effluent output will be more or less constant and stable. However, no evidence is provided for this. No information is provided about how the effluent composition may vary due to system disruptions, black liquor spills, equipment failures or a failure of the proposed ETF itself. Due to the age of this mill, it is possible that it will not be able to maintain a constant and predictable effluent flow and composition, and the chemistry of the effluent may vary considerably from time to time. As per the letter from Nova Scotia Environment of October 5, 2017, exceedances have been recorded at Point C where the effluent discharges into Boat Harbour Basin.[56] The fact that exceedances can occur demonstrates that the effluent flow is not constant or necessarily stable. The Minister should obtain a report regarding the nature and frequency of process

[53] NPNS EA Registration Document, Section 9.2.4.2, p. 507. While this statement is made in relation to a human health analysis, it demonstrates the lack of any certainty as to the actual effluent composition.
[54] Sweeney, E. *Comments on File No 1003, Environmental Assessment of NP's Proposed ETF*, Report, p. 2 Executive Summary (Appendix G-1).
[55] Timberbiz: Gunn's pulp mill permit lapses so land now for sale (Appendix H-14)
[56] Letter to General Manager, NPNS, from Supervisor of Environmental Assessment, NSE, dated October 5, 2017, p. 2 (Appendix H-15).

interruptions, disruptions, leaks and spills at the NPNS facility, and the impacts of such events on effluent composition.

71. The lack of any hard, provable data on the effluent that will come out of the end of the pipe ensures that the NPNS EA remains a hypothetical exercise. It is impossible to assess the impacts of an unknown substance. All the discussions regarding modelling and impacts are theoretical, as the assessment is not based in fact. The absence of scientific studies leads to the conclusion that NPNS is unable to prove the most fundamental component of their EA proposal, which is: "what is the composition of the effluent that NPNS proposes to discharge?" It would be an error for the Minister to accept an EA based on assumed effluent composition, rather than on proof of actual composition. This lack of basic information, despite its obvious centrality to the EA, must invalidate the submission.

7. Other effluent characteristics

72. Pulp mill effluent can contain many other components beyond those listed by NPNS as "expected water quality characteristics". Many of these are described in the context of human health impacts, but there is no discussion as to how they will fare in the receiving environment, whether that be the diffuser into the Caribou Channel, or via a leak or spill.[57]

73. Pulp mill effluent contains, or can contain, many toxic, bio-accumulative and carcinogenic components. Testing of raw effluent[58] from the Mill by the Boat Harbour Remediation Project reveals the presence of many compounds, including cadmium and mercury, which are problematic and bio-accumulative.[59] Mercury is often associated with pulp and paper operations.[60] The impacts of mercury and cadmium are not assessed in any meaningful way in the EA submission, yet they are clearly present in the effluent from the Mill and in the sediments in Boat Harbour Basin.[61] The long-term effects of discharging such substances into

[57] NPNS EA Submission, Section 9.2.4.2, p. 516 refers to a long list of substances, including mercury
[58] This relates to raw untreated effluent, which is different from the effluent which will be discharged after treatment in the proposed new ETF. The test results were provided by Ken Swain of the Boat Harbour Remediation Project in relation to raw effluent testing done in 2017 (Appendix H-1).
[59] Dr. Margaret Sears, *Comments regarding the Northern Pulp Nova Scotia Environmental Assessment Registration Document, Replacement Treatment Facility*, March 8, 2019, pp. 3 and 5, (Appendix F-1);
[60] Dr. Sears Report, at pp. 3 and 5 (Appendix F-1).
[61] Boat Harbour Remediation Project Handout, Appendix H-11.

the marine environment are not addressed in the NPNS submission, despite the potential impacts on the marine ecosystem and marine species and human health, as well on air quality via burning sludge. The impacts of these substances, being bio-accumulative, must be analyzed.

74. In an email from 2017 from NPNS to a provincial official, NPNS admitted it could not meet the CCME standard for certain metals, such as mercury, lead, aluminum, cadmium, iron, selenium and zinc.[62]

75. As mercury has been detected in raw effluent from the mill as recently as 2017[63], it requires assessment against mercury specific guidelines. The main route of exposure for wildlife in aquatic ecosystems is the consumption of contaminated aquatic prey species such as fish. To address this route of exposure there is a methylmercury CCME tissue residue guideline for protection of wildlife consumers of aquatic biota.[64] As the effluent will contain mercury, an assessment against the guideline should be conducted. Existing mercury levels in aquatic biota near the outfall should be measured, and the bio-accumulation that may occur from the exposure to the mercury in the effluent should be compared to the guideline. There is no indication that this guideline was reviewed and taken into account within the NPNS studies.

76. In a letter to NPNS dated June 14, 2017, NSE advised NPNS that "a receiving water study should address all potential substances of concern, not limited to those outlined in the Pulp and Paper Effluent Regulations."[65] No such list of all potential substances of concern appear in the receiving water study despite the express requirement that a list be provided and addressed.

77. The June 14, 2017 letter went on to say that "[t]he information provided to the Department should include one year's worth of effluent characterization data." Partial test results are referred to but not provided from several years, including 2002, 2003 and 1999, although it is not explained why it is necessary to go so far back in time to obtain test results. In any event,

[62] Email dated April 7, 2017, NP to Gary Porter, TIR with attached table (Appendix H-5)
[63] Test results were provided by Ken Swain of the Boat Harbour Remediation Project in relation to raw effluent testing done in 2017 (Appendix H-1).
[64] Canadian Tissue Residue Guidelines for the Protection of Wildlife Consumers of Biota – Methylmercury, CCME 2000. http://ceqg-rcqe.ccme.ca/download/en/294?redir=1551877575
[65] Letter to the NPNS General Manager, from Nova Scotia Environment, Engineering Specialist, dated 14 June 2017, p. 1 (Appendix H-6).

it does not appear that effluent characterization data for one full year appears in the materials filed by NPNS within this EA. These are glaring omissions, and without such information, the environmental impacts of harmful substances on receiving waters cannot be addressed.

8. Canso chemical site and mercury contamination

78. Dr. Meg Sears has prepared comments on the NPNS EA. The report from Dr. Sears speaks for itself and we present this report to the Minister for her consideration on this EA.[66]

79. As stated in Dr. Sears' report, serious mercury contamination issues are associated with the former Canso chemical chloro-alkali plant at the NPNS site.[67] This site is very close to, or immediately adjacent to, the site proposed for the new ETF.

80. The dangers presented by mercury and methylmercury are discussed above. It is a serious omission in this NPNS EA that there be no discussion of any environmental effects, or any discussion at all, in the NPNS materials in relation to the Canso site, and the mercury contamination. Likewise, there is no discussion about how construction of the ETF would affect the mercury contamination present in the bedrock and on the site. As Dr. Sears says, such information and analysis should be an essential component of any EA process.[68]

9. Failure to conduct primary studies and obtain baseline data

81. Section 8 of NPNS's EA materials, which is titled "Environmental Effects Assessment," focuses on 17 identified "Valued Environmental Components" (VECs). For over 50% (9/17) of the VECs examined in this section, NPNS failed to conduct its own primary research to determine baseline conditions. The following list identifies the VECs for which NPNS did not complete primary studies:

a) VEC: Freshwater Fish and Fish Habitat

EA Registration Document, Section 8.6.2.1, p 205: "It is noted that fall 2017 to summer 2018 field investigations were undertaken at the replacement ETF site, but an

[66] Dr. Margaret Sears, *Comments regarding the Northern Pulp Nova Scotia Environmental Assessment Registration Document, Replacement Treatment Facility*, March 8, 2019 (Appendix F-1).
[67] Dr. Sears' report, at p. 4 (Appendix F-1) . Partial decommissioning report for Canso site (Appendix H-2).
[68] Dr. Sears' report, at p. 4 (Appendix F-1).

alternate pipeline route was selected in the fall of 2018 […] and due to the timing of route selection, only a preliminary reconnaissance site visit was undertaken."

b) VEC: Wetlands

EA Registration Document, Section 8.7.2.3, p 224-225: "It should be noted that fall 2017 to summer 2018 field investigations were undertaken at the replacement ETF footprint area and surrounding area, but as an alternate pipeline route was selected in the fall of 2018 […] and due to the fall/winter timing of route selection, only a preliminary reconnaissance visit of the pipeline footprint area was undertaken."

c) VEC: Flora/Floral Priority Species

EA Registration Document, Section 8.8.2, p 245: "It is noted that fall 2017 to summer 2018 field investigations were undertaken at the replacement ETF footprint site, but an alternate pipeline route was only selected in the fall of 2018 […] and due to the fall/winter timing of route selection, only a preliminary reconnaissance visit was undertaken."

d) VEC: Terrestrial Wildlife/Priority Species

EA Registration Document, Section 8.9.2, p 269: "It is noted that fall 2017 to summer 2018 field investigations were undertaken at the replacement ETF site, but an alternate pipeline route was selected in the fall of 2018 […] and due to the timing of route selection, only a preliminary reconnaissance visit was undertaken."

e) VEC: Migratory Birds and Priority Bird Species/Habitat

EA Registration Document, Section 8.10.2.2, p 290: "[…] the proposed location of the pipeline changed following the completion of the avian program. As such, a significant portion of the [Project Footprint Area] (in the pipeline corridor) has not been surveyed for avian [Species of Conservation Concern] and/or [Species At Risk]."

f) VEC: Harbour Physical Environment, Water Quality, and Sediment Quality

EA Registration Document, Section 8.11.2, p 337: "The description of the existing conditions for the harbour physical environment, water quality, and sediment quality in the Northumberland Strait, Caribou Harbour, and Pictou Harbour is based on the results of previous research and existing scientific literature and environmental assessments; no field work was conducted as part of this EA Registration."

g) VEC: Marine Fish and Fish Habitat

EA Registration Document, Section 8.12.2, p 358: "The description of existing conditions is based on the results of previous research and existing scientific literature and environmental assessments; no field work was conducted as part of this EA Registration."

h) VEC: Marine Mammals, Sea Turtles and Marine Birds

EA Registration Document, Section 8.13.2, p 387: "The description of existing conditions for marine mammals, sea turtles, and marine birds in the Northumberland Strait is based on the results of previous research and existing scientific literature and environmental assessments; no field work was conducted as part of this EA Registration."

i) VEC: Marine Archaeological Resources

EA Registration Document, Section 8.16.2, p 458-459: "The assessment of effects on marine archaeological resources is based on background research and analysis of relevant geophysical and remote sensing data. […] An [Archaeological Resource Impact Assessment] of the marine environment has not been completed for this project but will be completed prior to construction."

82. The absence of this basic baseline research means that NPNS cannot accurately identify or describe the environment into which it proposes to introduce unknown toxic substances. In other words, NPNS cannot name the mammals, birds, fish, or plants, or describe the wetlands or harbour environment that will be impacted by its ETF with any certainty because it has not done the research.[69]

83. Instead of conducting its own primary research, NPNS purports to rely on previous research and existing scientific literature to support its assessment and its conclusion that there will be "no significant adverse residual environmental effects" on any of its identified VECs. However, this is highly problematic because the primary research cited by NPNS (or cited in the literature upon which NPNS relies) in many cases dates back decades.

84. For example, the Atlantic Canada Conservation Data Centre (AC CDC) report relied on by NPNS in support of its evaluation of the potential impacts on various species (including birds,

[69] MacKay, A.A., *Northern Pulp's Effluent Disposal Plans – Issues and Answers*, February 2019 (MacKay report)(Appendix C-1), regarding the necessity of conducting baseline surveys

terrestrial wildlife, marine mammals, fish, sea turtles, and others) purports to identify the species "known to occur" in the vicinity of the ETF project.[70] However, the majority of the data relied upon by AC CDC is over a decade old – and in some cases dates back over 50 years.[71] It is trite to state that the species residing in any particular area change over time. In the absence of current research, NPNS cannot purport to identify the species that may be affected by its project, much less evaluate the potential impacts on those species.

85. Similar conclusions can be drawn with respect to the non-species related VECs listed above (with the exception of the "wetlands" VEC, for which NPNS cites no research whatsoever in relation to the wetlands impacted by the new pipe route[72]).

86. It is particularly important to note once more that NPNS has not conducted baseline studies for over half of the environmental components that it purports to evaluate. In the absence of this critical information, it is impossible to understand how NPNS can conclude that its project will have "no significant adverse residual environmental effects" on any of the identified VECs. In our respectful submission, as a result of this glaring gap in NPNS's EA materials, the Minister cannot conclude with any certainty that the proposed ETF will have no significant environmental effects that cannot be mitigated. As a result, she cannot legally approve the proposed project.

87. Arthur MacKay has authored a commentary on aspects of the NPNS EA, and on behalf of FONS we hereby submit it to the Minister for consideration.[73] Mr. MacKay is an experienced fisheries biologist and consultant.[74] He co-authored an extensive study on the long-term effects of a pulp and paper mill, along with other industrial activity, on the St. Croix estuary in New Brunswick.[75]

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[70] NPNS Registration Document, Appendix N, p 1.
[71] NPNS Registration Document, Appendix N, p 18-21.
[72] NPNS Registration Document, Appendix O3.
[73] MacKay, A.A., *Northern Pulp's Effluent Disposal Plans – Issues and Answers*, February 2019 (MacKay report)(Appendix C-1)
[74] Art MacKay cv (Appendix C-1).
[75] Arthur MacKay, et al., 2010, "The St. Croix Estuary 1604 – 2004". It can be found at: https://issuu.com/artmackay/docs/healthofstcroixestuary

88. Mr. MacKay notes that NPNS has done few if any primary surveys to determine the vital ecosystem components of the target areas. He notes that the NP submission discusses mainly commercial fish species. While such species are important, it is not the full picture. He writes:

> …the foundational species of the ecosystem such as planktonic species, invertebrate and fish larvae, subtidal and intertidal invertebrates and plants, forage species, etc are not considered. Seasonality is an important issue and to truly understand ecosystem dynamics, at least 12 monthly surveys must be undertaken that include records for plankton, fish and invertebrate larvae, forage species, fish, bird, and mammals.[76]

89. His report provides some parameters that ought to have been followed in conducting baseline surveys for local species, as well as to obtain baseline chemical analyses.

90. Mr. MacKay warns that, in the absence of this basic information, the impact of the effluent from the proposed outfall pipe at Caribou Harbour or the proposed cleanup in Boat Harbour cannot be measured in the short term or long term.[77]

91. He concludes as follows:

> Frankly, in relation to the proposed pipeline, no work should begin until professional ecosystem surveys are undertaken at Caribou Harbour, Northumberland Strait at Caribou Harbour and Northumberland Strait at the Boat Harbour outfall (vital for comparison purposes). **In the absence of these necessary surveys, the Minister must be made aware that there can be no confidence in the purported lack of impacts stated and implied in the Northern Pulp environmental submission**. …[78]

j) Other missing studies

92. In addition to the missing studies identified above, the following are also absent:

(i) Baseline studies on Caribou Harbour and Caribou Channel. NPNS instead uses Pictou Harbour as a proxy (although no baseline study was conducted for Pictou Harbour either);[79]
(ii) Baseline data for the larger Strait area, regarding water quality and other municipal, industrial and agricultural discharges into the waters of the Strait;

[76] MacKay report, p. 3 (Appendix C-1).
[77] MacKay report, at p. 3 (Appendix C-1).
[78] MacKay report, p. 5 (Appendix C-1).
[79] NPNS EA Submission, Section 8.11.1, p. 336.

(iii) Studies regarding impacts of effluent from kraft pulp mills (without delignification) on species present in the Strait, including lobster, crab, herring and foundational ecosystem species;[80]
(iv) Engineering reports or drawings regarding the construction of the shoreline and marine portions of the pipeline, the route it will follow and how deeply it can be buried;
(v) Analysis or engineering study of the impacts of ice scour on buried HDPE pipe;
(vi) Modelling of effluent transport and dispersion from pipeline breaks, ruptures and leaks in marine, shoreline and terrestrial environments;
(vii) Air emissions data from current operations from all stacks and vents;
(viii) Studies showing the nature and frequency of process interruptions and disruptions, leaks and spills at the NPNS facility and the impacts of same on effluent composition;
(ix) Report and analysis on the Canso chemical site and mercury contamination and how it may be impacted by the construction and operation proposed ETF, and/or how it may impact effluent composition and risks of mercury contamination to the environment and human health, and
(x) Baseline data and cumulative effects of the project on the larger Northumberland Strait, taking into account other discharges and activities already affecting the Strait as a whole.

10. Long-term effects

93. A discussion of potential long term effects of the ETF project is noticeably absent from the NPNS EA submission as all impacts are deemed not to be residual. However, as identified throughout this submission, there are many potential and likely long-term effects that have not been meaningfully assessed. As per the Fringer Report, discussed below, had Stantec correctly used the models that were available, they would have discovered that it is likely that effluent will accumulate in Pictou and Caribou Harbours[81], and solids will settle out of the discharged effluent and onto the seabed.[82] Likewise, the long-term impacts of bio-accumulation of metals requires assessment for long term impacts on human and ecosystem health, and on the economics of the fishery.[83] Without it, the Minister cannot make a decision on the EA.

94. Arthur MacKay, in his report discussed above, notes the longer term impacts that should be expected due to exposure to effluent on an ongoing basis. This would include biological

[80] MacKay report, p. 3, discussion of "foundational species of the ecosystem" (Appendix C-1).
[81] Fringer report, p. 1 (Appendix A-1)
[82] Fringer report, pp. 4-5. (Appendix A-1)
[83] Discussed below. See also report by Dr. Sears, Appendix F-1.

magnification of toxins in the Harbour and Strait, and impacts on a broad range of marine organisms, including plankton, fish larvae, fish, birds, marine mammals and humans. He also notes creation of anoxic "dead zones," declines in marine invertebrates, fish, and some birds and mammals, and fishery closures due to the presence of toxic chemicals in fish caught for human consumption. All these effects, and many others, were observed and documented in his St. Croix study.[84]

11. Cumulative effects

95. The NPNS EA materials contain almost no discussion of the larger environment of the Strait and the southern Gulf, and the role of Caribou Channel and Caribou Harbour within that context. The discussion of cumulative effects in section 12 of the NPNS EA materials sets an artificially small area within which cumulative effects are examined. Even within that boundary, effects of agricultural activity are not discussed, and the impacts of existing municipal wastewater discharges are not taken into account. Further, due to the boundary in the EA submission, there is no discussion of the macro conditions in the Strait. No effort was made to take baseline measurements or to assess the carrying capacity of the Strait's Ecosystem overall and how it may be able to handle the proposed effluent discharge, or how that discharge may affect more distant parts of the Strait due to overall flows, currents and dynamics. The entire EA package and the discussion regarding cumulative effects are based on the findings of the Stantec modelling exercise, which is fundamentally flawed[85] and which fails to take into account what will happen to the effluent trail once it passes out of the immediate vicinity of Caribou Channel.

96. The cumulative impacts of current discharges of from agricultural activities, and from industrial and municipal wastewaters, emanating from Nova Scotia, New Brunswick and Prince Edward Island, are not examined. The role of climate change, and how it might interact with the project and impact consultant predictions, is likewise absent from the discussion.

[84] MacKay report, at pp 4-5. (Appendix C-1)
[85] See Dr. Fringer's report (Appendix A-1) and the discussion below regarding the Stantec modelling exercise.

Despite the presence of section 12 of the submission, the NPNS EA materials provide no comprehensive analysis of cumulative environmental effects.

12. Pulp and Paper Effluent Regulations

97. The *Fisheries Act*, coupled with the *Pulp and Paper Effluent Regulations* (*PPER*) permit discharge of pulp and paper effluent, up to certain measurable limits for certain characteristics.[86] However, mere compliance with the *PPER* does not prevent adverse effects or significant environmental effects that cannot be mitigated.

98. It is noted that the *PPER* are currently under review, with the goal of tightening them up, as up to 70% of pulp and paper mills still are considered to be harming the environment despite alleged compliance with the *PPER*. On February 1, 2019, Environment and Climate Change Canada officials appeared before the Standing Committee on Agriculture and Fisheries of the Prince Edward Island Legislature. In that appearance, an ECCC official stated, in part:

> Despite this high level of compliance with the existing effluent standard, the environmental effect studies have shown that the effluents from 70% of the pulp and paper mills across the country are having an effect on fish and/or, depending, fish habitat.[87]

99. The official also confirmed that the NPNS mill was included in the 70% of mills whose effluents are having an "impact on fish habitat".[88]

100. It is noted that the current conditions within Boat Harbour Basin have occurred, and continue to occur, despite ostensible regulatory compliance with the *PPER* over several decades.

13. Source of Mixing Zone Concept

101. The NPNS Submission and the receiving water studies on which it relies are based, in large part, on the misapplication of the concept of a 100 metre "standard mixing zone", within which

[86] *Pulp and Paper Effluent Regulations,* SOR/92-269 (*PPER*). The *PPER* are made under the *Fisheries* Act, R.S.C. 1985, c. F-14. See also the *Pulp and Paper Mill Effluent Chlorinated Dioxins and Furans Regulations*, SOR/92-267 made under the *Canadian Environmental Protection Act, 1999*, S.C. 1999, c. 33.
[87] Standing Committee Minutes, 1 Feb. 2019, p. 3 (Appendix H-13).
[88] Standing Committee Minutes, 1 Feb. 2019, p. 5 (Appendix H-13).

effluent components are projected to dilute to "background levels." In reality, the mixing zone that is proposed completely fails to comply with the basic requirements of a mixing zone, no matter what standard is applied. A mixing zone is entirely inappropriate given the realities of the receiving environment of Caribou Channel and Caribou Harbour.

102. The NPNS EA Submission states:

> Additionally, the project is designed with key established water quality guidelines and/or will meet ambient water quality (current background) at the edge of a standard mixing zone (CCME 2009 - Canada-wide Strategy for the Management of Municipal Wastewater Effluent)..[89]
> …
> The mixing zone for the discharged effluent was defined as the 100-m distance from the outfall pipe as per the Canadian Council of Ministers of the Environment (CCME) guidelines.[90]

103. NPNS cites CCME 2009 (Canada-wide Strategy for the Management of Municipal Wastewater Effluent) and the Atlantic Canada Wastewater Guidelines Manual as authority for its use of a mixing zone.[91] However, CCME 2009 is a municipal waste water guideline, which applies to government or public owners[92], not to private industrial pulp and paper mills like NPNS. Similarly, the Atlantic Canada Wastewater Guidelines Manual addresses municipal sewage, and not pulp and paper effluent.[93]

104. The significant differences between municipal waste water and pulp and paper effluent are underscored by the reality that they are regulated by two mutually exclusive sets of regulations made under the Fisheries Act. Municipal waste water is regulated via the *Wastewater Systems Effluent Regulations*[94], whereas pulp and paper effluent is governed by the PPER, as discussed

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[89] NPNS EA Submission, Registration Document, Section 5.6.1, p. 84.
[90] Stantec, Addendum Receiving Water Study, December 19, 2019, p. i, Executive Summary, NPNS EA Submission, Appendix E1.
[91] Stantec, Addendum Receiving Water Study, December 19, 2019, Section 3.1.2, p. 3.52, NPNS EA Submission, Appendix E1.
[92] *Canada-wide Strategy for the Management of Municipal Wastewater Effluent*, Canadian Council of Ministers of the Environment (CCME 2009). See definitions of "Municipal Wastewater Effluent" and "Owner" which do not include a private industrial operator like NPNS. Accessible at the following link: https://www.ccme.ca/files/Resources/municipal_wastewater_efflent/cda_wide_strategy_mwwe_final_e.pdf
[93] Atlantic Canada Wastewater Guidelines Manual, Environment Canada, 2006. The manual is an update of the former *Atlantic Canada Standards and Guidelines Manual for the Collection, Treatment and Disposal of Sanitary Sewage,* 2000 edition. https://novascotia.ca/nse/water/docs/AtlCanStdGuideSewage.pdf
[94] *Wastewater Systems Effluent Regulations* SOR/2012-139 (*WSER*). Subsection 2(5) of the *WSER* provides that the waste water regulations do not apply in respect of pulp and paper mills.

above. It is therefore in doubt whether CCME 2009 has any application to pulp and paper effluent and this EA.

105. It is questionable whether the CCME 2009 guidelines even continue to apply in relation to municipal wastewater, as they may have been superseded by the *Wastewater Systems Effluent Regulations*[95]. Under those regulations, made in 2012, the only 100 m mixing zone contemplated relates to discharge of municipal wastewater containing un-ionized ammonia. No comparable mixing zone is employed in the Fisheries Act or PPER relation to any pulp and paper effluent constituents.

14. Mixing zone does not apply in the context of outfall CH-B

106. Further, and more importantly, a mixing zone may not be used <u>at all</u> unless it satisfies important preconditions or requirements. These requirements are not discussed in NPNS's EA Submission. When they are considered, it becomes apparent that the proposed, or any, mixing zone is not appropriate at the outfall location proposed by NPNS and does not comply with CCME or NSE direction.

107. Nova Scotia Environment discussed the requirements for a mixing zone in correspondence to NPNS dated June 14, 2017.[96] The letter says, in part:

> A mixing zone is defined as an area of water contiguous to a point source discharge. A mixing zone is, under no circumstances, to be used as an alternative to reasonable and practical treatment….it is only one factor to be considered in establishing effluent requirements.
>
> …As a general principle, the use of mixing zones should be minimized and limited to conventional pollutants. The mixing zone principle does not apply to hazardous wastes…. Mixing zones also do not apply to bio-accumulative or persistence [sic] substances and despite the allowance of a mixing zone, effluent shall not be acutely toxic.
>
> …Mixing zones cannot interfere with other water uses such as…active fisheries… .[97]

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[95] *Wastewater Systems Effluent Regulations* SOR/2012-139 (*WSER*). Subsection 2(5) of the *WSER* provides that the waste water regulations do not apply in respect of pulp and paper mills.

[96] Letter to the NPNS General Manager, from Nova Scotia Environment, Engineering Specialist, dated 14 June 2017 (Appendix H-6).

[97] Letter to the NPNS General Manager, from Nova Scotia Environment, Engineering Specialist, dated 14 June 2017, p. 1 (Appendix H-6). The requirements for a mixing zone set out in this letter are similar to those found in the

108. Contrary to the directions in the June 14, 2017 letter, the mixing zone proposed by NPNS in this EA does not consider meaningfully, or in some cases even note the existence of, biotic communities and spawning areas, and the information provided about spawning areas is not accurate.[98] As well, given the presence of mercury and other bio-accumulative metals and compounds, the proposal does not comply with the requirement that no such substances be discharged within a mixing zone. Further, as CH-B is positioned within one of the last remaining herring spawning areas in the Strait, and within an important lobster fishing area,[99] it violates the express requirement that "mixing zones should not impinge upon…important fish spawning and/or fishing areas". [100] The Caribou Channel is in the middle of an extremely active fishery, yet this is not mentioned by the consultants who purport to apply the "CCME guidelines" that require such factors to be considered.

109. The NPNS submission fails to conduct any analysis of whether a mixing zone can actually be used at CH-B. There is no actual application of the NSE or CCME guidance. When the criteria are reviewed, NPNS fails most of them. The mixing zone concept cannot be applied to CH-B, and consequently, it is irrelevant how soon the substances within the effluent meet background conditions. The diffuser would be discharging harmful substances, including metals and solids, directly into a living ecosystem and spawning grounds, which supports an active fishery.

110. Caribou Channel is not an artificial 100 m dead zone which can be continuously loaded with effluent without consequence. The NP submission is based on an incorrect standard. In reality there is no water quality guideline which permits discharge of effluent into a spawning and active fishing area.

Guidelines on the Site-Specific Application of Water Quality Guidelines in Canada: Procedures for Deriving Numerical Water Quality Objectives, CCME 2003. http://cegg-rcqe.ccme.ca/download/en/221

[98] For more accurate information about herring spawning zones, see Egilsson, G., and MacCarthy, A., Caribou Harbour and Caribou Channel - dynamics, tides, ice, marine species and fisheries, February 21, 2019 (Appendix B-1).

[99] Egilsson, G and MacCarthy, A. (Appendix B-1).

[100] Letter to the NPNS General Manager, from Nova Scotia Environment, Engineering Specialist, dated 14 June 2017, p. 1 (Appendix H-6)..

15. Receiving environment – receiving water studies and near and far field modelling

111. The Stantec Receiving Water Studies, on which much of the NPNS EA is founded, are unreliable and the modelling exercise undertaken was not appropriate for the receiving environment. FONS submits that the Receiving Water Studies, and other materials based on the conclusions of those studies, must be disregarded and new, properly conducted studies must be included in an EA report.

112. A critique of the Stantec Receiving Water Studies has been prepared by Dr. Oliver Fringer of Stanford University, Stanford California USA and is appended to this submission.[101]

113. Dr. Fringer is an Associate Professor (with tenure), Department of Civil and Environmental Engineering, Stanford University. He is an oceanographer with expertise in numerical modelling of coastal dynamics.[102]

114. Dr. Fringer's report speaks for itself and we hereby submit it to the Minister for a detailed and thorough review. In summary, Dr. Fringer concludes that Stantec did not implement the MIKE 21 far-field model and the CORMIX near-field model appropriately. In this case, Stantec's implementation problems are significant. Dr. Fringer concludes that they lead:

> … to the incorrect conclusion that the environmental impacts will be negligible because the effluent concentrations are predicted to be unphysically low. **Instead, correct implementation of the models with more conservative and physically realistic scenarios would show that effluent concentrations in the region could be much larger and that effluent accumulation in Pictou and Caribou Harbours is likely.**[103] [emphasis added]

115. In this regard, Dr. Fringer states that Stantec's use of the two-dimensional MIKE 21 model is inappropriate as it fails to take into account local dynamics caused by wind, river inflows, offshore currents, ice, waves and storm surge. Due to the highly three-dimensional circulation in the region, a three-dimensional model (MIKE 3) should have been used to model the

[101] Fringer, O.B., *Review of near- and far-field modeling studies by Stantec Consulting for the Northern Pulp effluent treatment facility replacement project*, 7 March 2019 (Appendix A-1) (Fringer report)
[102] Oliver Fringer, CV, (Appendix A-2)
[103] Fringer Report, p. 1. (Appendix A-1)

behaviour of the effluent in the receiving water environment in relation to the outfall at CH-B, and the surrounding area.[104]

116. In this regard, we note that in May 2017 KSH recommended 3-D modelling be done in relation to alternative outfall locations D and D2.[105] Whether or not this recommendation was implemented, no 3-D far field modelling results have been provided within any reports filed within this EA despite the necessity of using 3-D far field modelling in generating accurate and reliable results.

117. Likewise, Dr. Fringer concludes that significant implementation issues in using the CORMIX near-field model have created unreliable results in the Receiving Water Studies. The ambient tidal current used to drive the CORMIX model is modelled by Stantec as much stronger than it would actually be during a neap tidal period. Tidal currents are even weaker during winter when ice cover decreases the strength of the tides. The CORMIX model also overestimates salinity as it does not take into account potential river inflow, which in turn leads to an overestimation of buoyancy and dilution.[106]

118. Dr. Fringer further notes that the Receiving Water Studies do not take into account settling of suspended solids during slack tides within 100m of the outfall, despite the potential for settling of such solids.[107]

119. Dr. Fringer notes:

> During each one-hour slack tide period, 173 kg of suspended solids would be discharged into the ocean from outfall CH-B. The solids that were discharged 30 minutes before slack tide would find themselves just 45 meters from the outfall, only to be transported back over the outfall again at the end of the next 30 minutes to be re-entrained into the outfall plume.
>
> … Furthermore, owing to the reduction in vertical turbulent mixing because of the weak currents during slack tides, there is a strong potential for the suspended solids in the effluent to settle out of the water column and onto the bed in the vicinity of the outfall.

[104] Fringer Report, p. 7 (Appendix A-1)
[105] Email May 29, 2017, KSH to NPNS and TIR, Alt D 2D modelling results (Appendix H-3).
[106] Fringer report at pp. 2 and 18-20 (Appendix A-1)
[107] Fringer Report, pp 4-5 and 21 (Appendix A-1)

> The effects of slack tides and the potential for settling of suspended solids is not discussed in the Stantec studies.[108]

120. Based on this clear and expert critique, FONS submits that the Receiving Water Studies do not provide sound information and data to the Minister that would permit the Minister to accept the conclusions of those Studies, or to conclude that discharge of effluent at the outfall will not cause adverse effects or significant environmental effects that cannot be mitigated. Rather, the critique requires the conclusion that the Receiving Water Studies cannot reliably determine the likelihood that adverse impacts or significant environmental effects will occur that cannot be mitigated in the receiving environment. As these studies form the backbone of the NPNS submission, NPNS has failed to discharge its onus to demonstrate that its proposal to discharge effluent into the Strait will not cause harm.

121. In addition, as discussed above regarding the mixing zone concept, NPNS has failed to provide and analyze certain types of information, within the Receiving Water Studies.

122. For all these reasons, the Receiving Water Studies must be rejected and their conclusions disregarded.

16. Local knowledge

123. We submit for the Minister's consideration three local knowledge summaries from individuals who have particular and detailed knowledge about local conditions.

1. Egilsson, G., and MacCarthy, A., Caribou Harbour and Caribou Channel - Dynamics, tides, ice, marine species and fisheries, February 21, 2019 (Appendix B-1);
2. Letter from Rob MacKay, Master Diver, dated March 5, 2019 (Appendix B-2); and
3. Letter from Barry Sutherland, dated March 4, 2019 (Appendix B-3).

124. Allan MacCarthy and Greg Egilsson are experienced fisherman who have fished in the immediate vicinity of the proposed outfall CH-B in Caribou Channel. Rob MacKay is a Master Diver with experience over three decades of diving in the Pictou area. Barry Sutherland has

[108] Fringer Report, pp 4-5 (Appendix A-1)

been fishing the Caribou area for 27 years. Between them they have lifetimes of observations about local conditions, including winds, currents, tides, ice and marine species.

125. Their summaries and letters speak for themselves.

126. Mr. Sutherland, Mr. MacCarthy and Mr. Egilsson are three of about eighty-two lobster fishers who fish in that area, including fishers from the Pictou Landing First Nation.

127. The three submissions listed above contain a wealth of information that was never gathered by any of NPNS's consultants. The actual observations described in these submissions provide real information which often contradicts the assumptions made within the NPNS materials, including the Stantec Receiving Water Studies.

128. Notably, the Receiving Water Studies fail to take into account crucial local conditions when they assess how the effluent would behave after discharge at CH-B. Mr. MacCarthy and Mr. Egilsson describe local currents, such as the Pictou Island counter clockwise gyre current. These submissions demonstrate that the Studies, and the NPNS submission generally, vastly underestimate the effects of ice, wind, tide and other dynamics, and demonstrate the vulnerability of a plastic pipe placed on, or buried in, the floor of Caribou Harbour and the Caribou Channel.

129. Among other things, Mr. Egilsson and Mr. MacCarthy note that the proposed outfall CH-B would be positioned within Mr. Egilsson's current lobster fishing area, very near to where he places his first traps of the day in lobster season. Mr. MacCarthy's lobster fishing area is immediately adjacent to CH-B and the entire area is a very active fishing zone. Many species are fished there, over the course of each year. Mr. Egilsson and Mr. MacCarthy also note that:

> **The proposed outfall CH-B is located in the middle of the last major active spawning area for Area 16F herring. Herring spawning grounds have compressed in the past few years as the stock has declined. Very little herring spawning occurs anywhere else in the Eastern Gulf.**[109] [emphasis added]

[109] Egilsson, G., and MacCarthy, A., Caribou Harbour and Caribou Channel - dynamics, tides, ice, marine species and fisheries, February 21, 2019 (Appendix B-1), at page 3 (Appendix B-1).

130. Likewise, Mr. MacKay has had a unique opportunity to observe the sea bottom in the Caribou area over the past several decades. He provides detailed information regarding the power and reach of ice and ice scour and how it can move extremely heavy items, as well as the soft shifting sea bottom in the area, and how these conditions could affect the effluent pipe that NPNS proposes to run through that area:

> The channel shifts from time to time mostly due to storms. Ice and tide also move sand around as it is very shallow in this area. Storms can pile ice up to 30 feet high which can dig deep into the soft bottom. This could damage the buried pipe.
>
> ...If the pipe is covered in armour stone, the sand on either side will be undermined by wind and wave action exposing the pipe to the full force of the ice in winter. If no armour stone is used, those same fall storms could easily expose the pipe, as anyone living near a beach knows how easily sand is shifted by storm winds and waves. Either way the pipe is unlikely to survive extreme conditions in this area.
>
> …The sea bottom in the area of the proposed pipe is very fragile. It's mostly sand and in the inner harbour, mud and eel grass. The eel grass is very fine and important to juveniles and larvae of lobster and crab.

131. Mr. Sutherland has shown that Caribou Harbour is a rock crab nursery. Rock crab are plentiful in that area, and are a food species which support lobster stocks. He writes, in part:

> Caribou Harbour is home to the largest commercial fishing fleet in the Northumberland Strait. The strong lobster catches in this area are the result of the continuous food supply from the rock crab nursery. The potential destruction of this crab habitat will have devastating consequences on the lobster industry in this area.[110]

132. He also expresses his concerns regarding the impact of noise and disruption from the installation and operation of the effluent pipeline and diffuser in this area.

133. These studies must inform any assessment of actual conditions in the area. Unfortunately, NPNS has failed to consider these issues in any significant way in its EA materials.

17. Monitoring and Accident Prevention

134. Throughout the lengthy period leading up to the current EA, members of the public expressed numerous concerns with respect to NPNS's ability to adequately monitor the proposed ETF

[110] Sutherland, at page 2 (Appendix B-3)

and to respond to accidents that could result in the unplanned release of treated or untreated effluent or other hazardous substances into the environment. NPNS has utterly failed to respond to these concerns in its EA materials. As a result, the Minister does not have enough information to make an informed decision as to whether spills from the proposed ETF may result in significant environmental effects and/or adverse effects.

135. In its EA materials, NPNS refers to an Environmental Protection Plan (EPP) and an Emergency Response and Contingency Plan (ERCP) that will be developed to address various aspects of its monitoring and accident response requirements. These plans will form part of an umbrella document known as an Environmental Management Plan (EMP).[111]

136. NPNS states that both the EPP and the ERCP will be prepared after it receives its EA approval.[112] In other words, neither the public nor the Minister will be given the information required to fully understand how NPNS intends to respond to spills, or monitor its ETF and mitigate the potential for accidents, until the project is well on its way to operation. Until that time, we are left only with vague statements indicating what the ERCP is "anticipated" to include[113] - and are told only that the EPP will address management and prevention of "accidents, malfunctions, or unplanned events".[114]

137. This lack of information is all the more problematic in light of the significant risks posed by ice coverage in Caribou Harbour and the Northumberland Strait. As per the MacCarthy and Egilsson submission, "[i]ce is typically present in the Caribou area from the end of December through April, but can set in earlier and remain later if temperatures are cooler than normal."[115] At a minimum, then, ice will be present in and around the NPNS pipe route for over 1/3 of the year. This ice includes "fast ice," which freezes to the bottom of the Harbour in shallower inshore areas.[116]

138. Common sense dictates that the ice, storms and other unpredictable marine conditions will hinder NPNS's ability to monitor its pipe and diffuser for damage and leaks, and to investigate

[111] NPNS Registration Document, section 5.3.1, p 49.
[112] *Ibid,* section 5.7.3, p 97 & section 10.5, p 533.
[113] *Ibid,* section 5.7.3, p 98.
[114] *Ibid,* s 10.5, p 533.
[115] MacCarthy & Egilsson, Appendix B-1, p 9.
[116] *Ibid.*

and repair spills in the marine environment. NPNS does not explain how it will conduct its monitoring and spill response activities in the presence of ice – in fact, its EA materials do not even acknowledge that ice may be an issue when it comes to monitoring and responding to spills. Likewise, the EA materials do not contain an examination of the particular effects of a prolonged and inaccessible effluent spill, at any point along the pipeline, or within the marine area under ice cover. Despite the lengthy ice-bound periods during the winter, and the significant possibility of damage by ice or other forces during the winter, NPNS provides no explanation of what could be done to protect the marine environment of Caribou Harbour or the Caribou Channel, before an opportunity arises to access and repair the damaged infrastructure. This is an obvious issue and a serious oversight that must be addressed prior to any EA approval.

18. Receiving environment – air quality

139. The ETF proposal includes the burning of sludge generated from effluent treatment. Like the effluent discussed above, the chemical composition of the sludge is largely unknown, and no studies have been provided analyzing the sludge composition and the impacts to air quality and human and environmental health from emissions arising from burning sludge.

140. Significant concerns exist in respect of burning sludge in the mill's power boiler, especially in combination with existing emissions at the mill. As noted by Dr. Sears, too little is known and provided about the composition of the sludge to provide any certainty as to whether air emissions will be problematic.[117] There is likewise a lack of information regarding toxic metals and polyaromatic hydrocarbons (PAHs) in air emissions.

141. Dr. Sears notes concerns regarding dioxins and furans associated with pulp mills.[118] She also notes an inaccuracy in the information provided by NPNS on this EA:

> It is stated in the EA Registration document (e.g., Table 6.7-1), "In fact, dioxins and furans testing for the last 5 years has consistently shown that all of the compounds required to be tested under the regulations have not been detected in NPNS' effluent (non-detect)." The dioxin-free message is not consistent with reports from Northern Pulp that are posted on the Nova Scotia government website,

[117] Dr. Sears' report, at p. 3 (Appendix F-1)
[118] Dr. Sears' report at pp. 10-12 (Appendix F-1)

> nor the data reported to the National Pollutant Release Inventory (NPRI).[17] NPRI data indicates that on average 3.6 tonnes of PAHs have been emitted to the air annually since 2006, and 8 mg TEQ dioxins/furans have been emitted annually since 2011.[119]

142. The NPRI data cited by Dr. Sears is appended to this submission.[120]

143. Dr. Sears also notes exceedances in air emissions of hydrogen sulphide associated with the mill.[121]

144. With respect to air quality, again actual testing of co-combustion of hog fuel and sludge in the power boiler has not occurred, but a "pilot study" is contemplated.[122] No explanation was provided as to why such testing could not have been done prior to the EA.

145. Air emission studies and information remain at best incomplete, and therefore an insufficient basis for any conclusion as to project environmental or health impacts. At worst, they show issues with emissions of dioxins and furans, and PAHs being emitted by the mill.

a) Hoffman report and rebuttal to Stantec critique

146. In a report in 2017, Emma Hoffman and co-researchers conducted a pilot study of air quality issues in the Pictou area.[123] The study investigated prioritized air toxic ambient VOC concentrations to determine whether these correlated with wind directions and whether there was an indication that toxic ambient VOCs were linked to the NPNS mill. The study acknowledged its limitations, but concluded that elevated levels of certain toxins were apparent when prevailing winds came from the direction of the mill.

147. At appendix K2, Stantec challenged these findings, and argued they should be disregarded. Ms. Hoffman answered the Stantec criticisms with an effective rebuttal, dated February 23, 2019 and attached to this package.[124] Ms. Hoffman described the Stantec article as containing

[119] Dr. Sears' report, at p. 11 (Appendix F-1)
[120] National Pollutant Release Inventory spreadsheet for NPNS, Appendix H-27.
[121] Dr. Sears' report, at p. 8 (Appendix F-1)
[122] NP EA Registration Document, Section 9.3, p. 519.
[123] Hoffman, E, et. al., *Pilot Study investigating ambient air toxics emissions near a Canadian kraft pulp and paper facility in Pictou County, Nova Scotia*, June 2017, Environ Sci Pollut Res 24(25):20685–20698 (Appendix E-1).
[124] Memo Hoffman, E., to Gunning, D. (Hoffman rebuttal), (Appendix E-1)

misrepresentations put forth by NPNS's EA of the scientific contributions her 2017 study provides. Ms. Hoffman's 2017 report, and her rebuttal speak for themselves, and we submit them to the Minister for consideration in this EA process.

148. Ms. Hoffman's rebuttal confirms the potential that toxic ambient VOCs are emanating from the mill:

> Compared to all other wind directions, prevailing winds from the northeast and the mill typically resulted in higher VOC concentrations for all compounds, except carbon tetrachloride, suggesting that the mill is likely a contributor to increased concentrations; however (as stated in the study), the origin(s) of VOCs are "*inconclusive*", and "*other local sources likely contribute to air toxics emissions*".[125]

149. Ms. Hoffman concludes as follows:

> In summary, the intent of this pilot study was to address local air quality conditions in a Nova Scotia rural community, which clearly indicates the need for further investigation. Moreover, this pilot study serves as a precursor to gaining awareness, so that government agencies adopt more stringent air quality regulations and monitoring programs to ensure health of all citizens is safeguarded and prioritized.[126]

150. We ask that the Minister likewise examine closely the data provided by NPNS in respect of air emissions, and the other aspects of this EA, and employ the precautionary approach when determining whether adverse effects or non-mitigable significant environmental effects will occur.

19. Human Health effects

a) Expert – Ellen Sweeney report

151. At Section 9.0 of NPNS's EA materials, we are provided with a "Human Health Evaluation". In theory, this section is intended to provide the Minister with the information she needs to evaluate whether the proposed ETF will cause "adverse effects" – which are defined in the

[125] Hoffman rebuttal, p. 2 (Appendix E-1)
[126] Hoffman rebuttal, p. 4. (Appendix E-1)

Environment Act as effects that impair or damage the environment, or change the environment in a manner that negatively affects "aspects of human health."[127]

152. Dr. Ellen Sweeney, Director of Strategic Research Initiatives at the Atlantic Partnership for Tomorrow's Health,[128] has reviewed and critiqued NPNS's Human Health Evaluation.[129] Dr. Sweeney's comments are appended to this submission for the Minister's review.

153. Overall, Dr. Sweeney concludes that the information provided by NPNS is far from sufficient to accurately assess the true impacts of the proposed ETF on the health of the surrounding communities.

154. Dr. Sweeney identifies numerous critical gaps in NPNS's Human Health Evaluation. For instance, NPNS states that specific effluent chemistry characteristics "will not be known with certainty until the project is operational."[130] As Dr. Sweeney notes, without detailed information identifying precisely what will be coming out of NPNS's proposed outfall, the Minister cannot possibly evaluate the risks and potential hazards with any degree of certainty.[131]

155. Additional flaws identified by Dr. Sweeney include the following: (1) a failure to provide supporting evidence relating to pulp and paper mill projects NPNS claims to be similar to its proposed ETF;[132] (2) a heavy reliance on a single study (the Toxikos report) pertaining to a project that was never built;[133] (3) a failure to examine potential fetal exposure to carcinogenic and endocrine disrupting chemicals;[134](4) a failure to evaluate the health risks associated with potential spills on land or in watersheds;[135] and (5) a failure to evaluate the potential health

[127] *Environment Act, supra* at s. 3(c).
[128] Dr. Sweeney, cv (Appendix G-2).
[129] Sweeney, E., *Comments on File No: 1003 – Environmental Assessment of Northern Pulp's Proposed Effluent Treatment Facility,* February 2019 (Appendix G-1) (Sweeney report).
[130] NPNS EA Submission, Registration Document, section 9.1, p 489.
[131] Sweeney report, Appendix G-1, p 4.
[132] *Ibid,* p 4.
[133] *Ibid,* p 4-5.
[134] *Ibid,* p 5.
[135] *Ibid,* p 10.

impacts of low dose cumulative exposures to toxic substances associated with the proposed ETF.[136]

156. Dr. Sweeney's report raises significant concerns with the quality and sufficiency of the Human Health Evaluation provided by NPNS. Given these critical flaws, the Minister cannot conclude with any certainty that the proposed ETF will not cause "adverse effects" that cannot be mitigated.

b) Expert - Daniel Rainham comments

157. Dr. Daniel Rainham of Dalhousie University has also critiqued various parts of NPNS's Human Health Evaluation.[137] Dr. Rainham is an Associate Professor and Director of Dalhousie's Environmental Science Department. He is also the Elizabeth May Chair in Sustainability and Environmental Health.[138] Dr. Rainham's report is appended to this submission for the Minister's review.

158. Dr. Rainham identifies similar concerns to those raised by Dr. Sweeney. For instance, he notes that NP did not provide detailed information with respect to the chemical composition of its effluent – although it was well within NPNS's capacity to do so.[139]

159. Additional information gaps identified by Dr. Rainham include the risks of exposure to emissions through methods such as the consumption of fish exposed to toxic substances,[140] and the chemical composition of the fine particulate pollution associated with the ETF project.[141] As a result of these and other flaws in NP's Human Health Evaluation, the Minister cannot accept NPNS's conclusion that there will be no significant impact on the health of the affected communities.

[136] *Ibid,* p 8-9.
[137] Rainham, D., *Comments on the document "Replacement Effluent Treatment Facility Project,* 5 March 2019 (Appendix D-1) (Rainham report).
[138] Dr. Rainham's CV (Appendix D-1).
[139] *Ibid*, p 2.
[140] *Ibid,* p 4.
[141] *Ibid,* p 5.

20. Conclusion

160. As stated in the first paragraphs of this submission, NPNS's Executive Summary advises that, on all aspects of the project, there will be no "significant residual environmental effects". As per the material submitted above, and the expert reports from qualified experts, this conclusion cannot stand. FONS submits that the information and analysis provided in this submission show that there is a very real possibility that adverse effects and non-mitigable significant environmental effects will occur in respect of the ETF project.

21. Decision Requested –ss 34(1) and 34(2) of the *Environmental Assessment Act* and ss. 13(1) of the *Environmental Assessment Regulations*

161. FONS submits that this submission and the accompanying Appendices have established that it is likely that the ETF project will cause adverse effects or significant environmental effects that cannot be mitigated. FONS therefore requests that the Minister reject the proposed undertaking pursuant to subsection 34(1)(f) of the *Environment Act* and subsection 13(1)(e) of the *Environmental Assessment Regulations.*

    In the alternative, FONS submits that the evidence before the Minister establishes that there may be adverse effects or significant environmental effects caused by the undertaking that cannot be mitigated, and that an environmental-assessment report is therefore required, pursuant to subsection 34(1)(c) of the *Environment Act*, and subsection 13(1)(d) of the *Environmental Assessment Regulations.*

162. Further and in any event of the above, FONS requests that it be provided with a written statement of the decision rendered by the Minister in relation to the environmental assessment of the undertaking, setting out the findings of fact upon which it is based and the reasons for the decision, pursuant to subsection 10(4) of the *Environment Act.*

Dated March 8, 2019, at Halifax Nova Scotia.

James Gunvaldsen Klaassen

Sarah McDonald

APPENDICES

APPENDIX A-1

Review of near- and far-field modeling studies by Stantec Consulting for the Northern Pulp effluent treatment facility replacement project

Prepared by:
Oliver Fringer, Ph.D.
San Francisco, CA, USA

March 7, 2019

Table of Contents

1. Executive Summary

This report provides a review of computer modeling of the fate and transport of effluent from proposed discharge locations in and around Pictou Harbour and offshore of Caribou Harbour near Pictou, Nova Scotia. The modeling work was carried out by Stantec Consulting for assessment of the Replacement Effluent Treatment Facility Project registered by Northern Pulp Nova Scotia Corporation. Simulations were conducted with accepted industry-standard models including the near-field CORMIX model and the far-field MIKE 21 model.

Owing to several problems related to the implementation of the CORMIX and MIKE 21 models, they overestimate the near- and far-field mixing and dilution of the effluent from the proposed outfalls, including the final outfall at site CH-B offshore of Caribou Harbour. This leads to the incorrect conclusion that the environmental impacts will be negligible because the effluent concentrations are predicted to be unphysically low. Instead, correct implementation of the models with more conservative and physically realistic scenarios would show that effluent concentrations in the region could be much larger and that effluent accumulation in Pictou and Caribou Harbours is likely.

The principle problems related to the far-field MIKE 21 modeling include:

1) Agreement between the model simulated currents and water levels and observed currents and water levels in Pictou Harbour is poor. Therefore, we can have no confidence that the model accurately predicts the far-field fate and transport of the effluent at any of the proposed outfall locations.

2) Use of the two-dimensional MIKE 21 model is inappropriate given the potentially strong vertical variability of currents driven by winds and river inflows in the region. These three-dimensional effects can significantly impact the far-field transport by exaggerating accumulation in Pictou and Caribou Harbours.

3) The far-field model scenarios using MIKE 21 omit or incorrectly simulate the impacts of winds, river inflows, offshore currents in the Northumberland Strait, ice, waves, and storm surge. These processes may significantly impact far-field mixing and dilution of effluent and lead to higher effluent concentrations throughout the region.

4) The figures showing maps of low effluent concentrations offshore of Caribou Harbour are misleading because the far-field model artificially dilutes the effluent. Nevertheless, the dilution factors are reported to be over 100 in most of the region surrounding the CH-B outfall, which is an overly optimistic result.

The principle problems related to the near-field CORMIX modeling include:

1) The ambient tidal current used to drive the CORMIX model offshore of Caribou Harbour is much stronger than the expected current during a neap tidal period. Tidal currents are even weaker during winter when there is ice cover which decreases the strength of the tides. Overestimation of the tidal currents gives an unrealistic overprediction of the near-field mixing and dilution of effluent, particularly during slack tides.

2) The ambient density employed in the CORMIX model is too saline because it does not take into account potential effects of river inflows. This makes the receiving waters too dense and leads to too much buoyancy-driven mixing of the effluent plume, thus leading to an overestimate of the near-field mixing and dilution. The CORMIX modeling also ignores the effect of vertical variability in salinity, which could be strong during periods of high river inflows and reduce the near-field mixing and dilution because fresh water layers near the surface may trap the effluent beneath them.

It should be noted that these problems are related to the implementation and choice of models, not to the models themselves. When implemented correctly, CORMIX and far-field models like MIKE 21 or its three-dimensional counterpart, MIKE 3, yield very reliable near- and far-field predictions of effluent transport.

2. Introduction

2.1. Overview

In this report I review the near- and far-field modeling studies conducted by Stantec Consulting to understand the fate of effluent from proposed outfalls located in and around Pictou and Caribou Harbours which are connected to the Northumberland Strait in Pictou County, Nova Scotia, Canada. These studies are part of the Environmental Assessment of the Replacement Effluent Treatment Facility Project registered by Northern Pulp Nova Scotia Corporation (Northern Pulp). Specifically, in this report I analyze the modeling studies contained in the following appendices included in the Environmental Assessment:

1) Appendix E1 – Stantec final Caribou discharge receiving water study (The final study)
2) Appendix E2 – Stantec response to questions
3) Appendix E3 – Stantec receiving water study effluent treatment plant replacement (The preliminary study)

In the preliminary study (Appendix E3), scenarios were conducted to study the effluent transport from two outfalls in (sites Alt-A and Alt-B) and offshore of (sites Alt-C and Alt-D) Pictou Harbour. It was deemed that the suggested outfall location Alt-D was not appropriate because of the potential for ice scour of the outfall in the relatively shallow water (11 m). The final study (Appendix E1) was then undertaken to assess the effluent transport from outfalls located offshore of Caribou Harbour in 20 m of water at sites CH-A and CH-B. Site CH-B was recommended as the location with the least environmental impact. In what follows, I will refer to these appendices as the "final study", the "response to questions", and the "preliminary study". Collectively, they will be referred to as "the studies" or "the Stantec studies".

Simulating the transport and fate of effluent from a coastal wastewater outfall requires two kinds of models. Roughly within 100 m of the outfall, effluent is diluted relatively rapidly by mixing with ambient ocean waters. This mixing is due to strong turbulence related to jet-like flow from the outfall ports and buoyancy arising from the difference in density between relatively warm and fresh effluent and colder and saltier receiving waters. In the studies reviewed here, this dilution process is simulated with CORMIX (Jirka et al. 1996), an industry standard near-field model that takes into account diffuser geometry and properties of the effluent and receiving waters. After the near-field turbulence and buoyant mechanisms have decayed, the fate and transport of the effluent is dictated by the larger-scale circulation in the coastal region surrounding the outfall. The far-field currents, salinity, and temperature are obtained with a hydrodynamic model that computes circulation in response to winds, tides, river inflows, and other relevant coastal processes. These currents are then used to compute the far-field transport and fate of the effluent. In the studies reviewed here, the MIKE 21 model (DHI 2017) was used to compute the far-field circulation and transport. This model is also an industry standard that has been applied extensively to study circulation and transport in coastal regions. While the CORMIX model is an appropriate choice for the near-field modeling, the MIKE 21 model is not appropriate for this study because it is a two-dimensional model, as discussed in Section 3.1 below.

It is common practice to use far-field models to supply ambient currents and environmental parameters like temperature and salinity to the near-field model. The near-field dilution results including the near-field concentration and vertical distribution of the effluent plume can be supplied to the far-field model. In the Stantec studies, the ambient currents needed

for the CORMIX model are taken from the MIKE 21 model, while the ambient density field for CORMIX is taken from measurements of temperature and salinity. The far-field MIKE 21 model does not use results from CORMIX. This is common given that only relative concentrations are needed to assess the far-field dilution when using a two-dimensional model like MIKE 21. As will be discussed in this report, however, a three-dimensional far-field model is needed, and this model requires information about the vertical distribution of the effluent plume from the near-field model.

2.2. Currents and dispersion in the coastal ocean

In coastal areas like the regions in and around Pictou and Caribou Harbours, the currents arise from a multitude of processes, although a simple categorization is to distinguish between the tides and all other non-tidal processes, such as wind-driven, river-driven, and large-scale ocean currents in the Northumberland Strait. A prevailing and misleading theme in the Stantec studies is the suggestion that, although some non-tidal processes are included in the modeling (albeit incorrectly), these non-tidal processes are not important because the tidal currents dominate the near- and far-field effluent transport. However, as discussed throughout this review, the non-tidal processes are extremely important for predicting the fate of the effluent in both the near-field and far-field.

Because of their oscillatory motion in time, tides transport effluent back and forth over an outfall, and with each oscillation the effluent is dispersed, leading to horizontal spreading of the effluent plume. This so-called tidal dispersion is strongest in regions where the tidal currents are both large and vary strongly in space, such as at the mouths of Caribou and Pictou Harbours. Although an outfall plume will spread due to tidal dispersion, there will not be much dilution of the effluent after many tidal cycles unless there are non-tidal currents that can transport the effluent away from the outfall. Without non-tidal currents, effluent would simply accumulate around outfall location CH-B and in nearby Caribou Harbour.

Accumulation of effluent in the vicinity of an outfall is strongest during slack tides, periods of low or negligible currents that occur twice during every tidal period, which is approximately 12 hours (the tidal period due to the moon is 12.42 hours and that due to the sun is 12 hours). The effects of slack tides are most pronounced during neap tides when tidal currents are weakest. For example, the maximum neap tidal current is approximately 10 cm/s at outfall location CH-B (based on the discussion presented in Section 4.2 below). With this tide, the tidal currents will be weaker than 2.5 cm/s for the one-hour period surrounding slack, or for approximately two hours (17%) of the entire tidal cycle. During each one-hour slack tide period, 173 kg[1] of suspended solids would be discharged into the ocean from outfall CH-B. The solids that were discharged 30 minutes before slack tide would find themselves just 45 meters from the outfall, only to be transported back over the outfall again at the end of the next 30 minutes to be re-entrained into the outfall plume. This demonstrates the importance of slack tide in the accumulation of effluent over an outfall diffuser due to the prolonged periods of relatively weak currents, particularly during the neap period of the spring-neap tidal cycle. Furthermore, owing to the reduction in vertical turbulent mixing because of the weak currents during slack tides, there is a strong potential for the suspended solids in the effluent to settle out of the water

[1] Based on a concentration of 48 mg/L and effluent flow rate of 1 m^3/s, from Table 3.2 of the final study.

column and onto the bed in the vicinity of the outfall. The effects of slack tides and the potential for settling of suspended solids is not discussed in the Stantec studies.

Fortunately for the health of coastal ecosystems, non-tidal currents exist to varying degrees in all coastal regions. In fact, the tides themselves produce non-tidal currents, much like ocean swell waves produce rip currents that have no wave-like signature. Non-tidal currents that are produced by the tides are generally smaller than other non-tidal currents in the region, such as wind-driven, river-driven, and large-scale ocean currents. While river flows and winds are included in the far-field modeling, these effects are not accurately simulated, as discussed in Section 3.1 below. There are large-scale ocean currents that are predominantly from the west to east in the Northumberland Strait at speeds ranging from 6-9 cm/s (Lauzier 1965). Another non-tidal current in the region is the counterclockwise circulation around Pictou Island that has been observed by local fisherman (MacCarthy and Egilsson 2019). This non-tidal current is likely driven by a combination of winds and tides. Although they are important in dictating the far-field transport of effluent, these non-tidal currents are regarded as not important and not included in the Stantec studies.

3. Review of the far-field modeling

3.1. Two- vs. three-dimensional modeling

The MIKE 21 model employed in the far-field simulations is not appropriate because it is two-dimensional and does not represent important three-dimensional processes in the region, such as wind-driven circulation and density effects arising from freshwater flows from rivers. A more appropriate model like MIKE 3 would need to be used to account for these effects.

The MIKE 21 model employed by Stantec is a two-dimensional model in that it computes the depth-averaged currents at each grid cell in the computational domain. Therefore, it assumes that the currents are constant with height above the bed in each grid cell. The three-dimensional equivalent of MIKE 21 is the MIKE 3 model (also by DHI), which computes the variability in currents as a function of height above the bed. The principal advantage of two-dimensional, depth-averaged models is that they are computationally efficient because three-dimensional models require addition of grid cells in the vertical direction. In the case of the Stantec simulations, a three-dimensional model would require at least 20 layers in the vertical which would increase the model runtime by at least a factor of 20.

Despite its computational efficiency, a two-dimensional model is not appropriate to simulate the far-field effluent transport because of the importance of three-dimensional processes in the coastal region around Pictou and Caribou Harbours arising from variations in salinity and temperature, which affects the density stratification. Density stratification due to salinity arises along coastlines where river inflows bring fresh water into the ocean. Because the river water is fresh, it is less dense than the salty ocean, thus inducing vertical variations in the salinity field in which the denser, salty water lies beneath the lighter, fresher water above. Temperature stratification also exists throughout the oceans since the upper layers tend to be heated by the sun, leaving warmer and lighter waters above colder and denser waters. Temperature stratification is weakest in winter months when incoming heat is weakest.

Salinity stratification is more important than temperature stratification in coastal waters where river effects can be important. For example, the top and bottom salinities in the Pictou

Road region in July 1995 were 23.7 and 31.2 ppt (parts per thousand by mass), respectively, while the top and bottom temperatures were 13.5°C and 14°C, respectively (Preliminary study, p. 2.21). This translates to a top-bottom difference in density of 5.8 kg/m^3 due to the salinity and 0.1 kg/m^3 due to temperature, using the UNESCO equation of state calculator (UNESCO 1981). In December 1998, the salinity stratification at the same location was weaker (top-bottom salinity difference of 2 ppt) although the temperature stratification was slightly stronger (top-bottom temperature difference of 2°C). The salinity stratification generally increases with increasing river flow and decreases with tidal flow strength, since tidal currents generate turbulence that tends to mix the salinity and temperature field and weaken the vertical density stratification. Measurements indicate that the surface salinity near the East River in the Pictou Harbour region varied from 20 ppt during low-flow periods to just 5 ppt during high-flow periods (Preliminary study, p. 2.21).

Ocean water is generally stratified in the vertical because density increases with depth, with lighter, less dense waters overlying heavier, denser waters. However, in the coastal ocean there is also horizontal variability in the salinity-induced density. At a river mouth, the water is fresh and there is no vertical salinity stratification, while in the ocean far from the river mouth the salinity is high, yet there is also weak vertical salinity stratification. The most important effect of this horizontal variability in density is to induce a three-dimensional circulation in which fresh, river waters flow seaward over denser ocean waters which flow landward. In addition to the implications for the near-field transport (See Section 4.2 below), the implication for far-field transport is that effluent may be transported into the harbours with the landward-flowing denser currents. This effect is accentuated in deeper waters, implying that it will be stronger in Pictou Harbour (which also has higher freshwater flows), although the shipping channel in Caribou Harbour can act as a conduit to transport effluent-rich ocean waters into the harbour.

A second three-dimensional effect that cannot be captured by a two-dimensional model is related to the winds. When aligned with the main axes of Pictou or Caribou Harbours, winds will drive currents downwind along the shallow edges while the flow in the central, deeper portions will be driven upwind. Since the dominant westerly winds (August-April[2]) in the region are generally aligned with the main axes of the harbours, they have the potential to drive surface effluent seaward and that at depth into the harbours. Wind-driven circulation is typically not as strong as that driven by the rivers or tides, although it can be important during periods with neap tides and low river inflows.

A two-dimensional model also cannot capture the variability of the effluent with depth. The assumption of two-dimensionality in the effluent field is reasonable when the three-dimensional effects in the flow field are relatively weak. In fact it is possible to approximate some three-dimensional processes quite well with a two-dimensional model, such as a process known as shear-flow dispersion. Because of bottom friction, currents are slower near the bed, and if there is wind-driven circulation, the currents may be stronger near the surface. Therefore, tracers[3] that are in regions of the water column with slower-moving currents will be transported more slowly in the horizontal than those in the faster-moving regions of the water column. This process can be thought of as horizontal dispersion of the tracer field because it is spreading horizontally, and can be approximated reasonably well in a two-dimensional model with a shear-

[2] https://weatherspark.com/y/28559/Average-Weather-in-Pictou-Canada-Year-Round

[3] A tracer is a substance that is transported passively with the flow without buoyancy effects.

flow dispersion coefficient. The MIKE 21 model includes many approximations like this to account for three-dimensional effects in the two-dimensional transport module, although these were not employed in the Stantec studies (Preliminary study Table 2-1; Final study Table 2-11: "No decay and no dispersion in the particle tracking module"). Indeed, these approximations are not suitable for estuarine environments given that they work best in riverine environments that are weakly stratified, weakly wind-driven, and lack tidal influence.

Regardless of the influence of dispersion on the two-dimensional transport, the lack of vertical variability in the modeled tracer prevents simulation of an effluent that in reality can vary quite strongly in the vertical. The proposed effluent will typically be less dense than the receiving waters (it is both fresher with a total dissolved solids concentration, or salinity, of 1-4 kg/m^3, and warmer, with a winter temperature of 25^{o}C and summer temperature of 37^{o}C; Preliminary report p. 3.54). Therefore, if the receiving waters are sufficiently salty and cold (See Section 4.2 below) the effluent is expected to rise to the surface and propagate as a surface plume that is just 1-2 m thick based on the CORMIX near-field results in the Stantec studies. Furthermore, the depth at which the plume propagates is not necessarily at the surface, particularly under high flow conditions in which the effluent may be more dense than the receiving waters (See Section 4.2 below). Therefore, it is possible that the effluent could be driven in a direction that is opposite to that in a two-dimensional model if a three-dimensional model were used.

In summary, while three-dimensional effects may not be important during some periods of the year, such as during periods of low river flows and weak winds, in general a three-dimensional model is needed to accurately simulate the far-field fate and transport of effluent from the proposed discharge locations. Indeed, the MIKE 21 manual (Page 2 of DHI 2017) states, "In water bodies with stratification, either by density or by species (ecology), a 3D model should be used. This is also the case for enclosed or semi-enclosed waters where wind-driven circulation occurs." One might argue that three-dimensional models take too much time to run because of the need to include many grid points in the vertical. However, the Stantec final study employed a computational mesh with 24,645 grid cells (15,872 were employed in the preliminary study). Three-dimensional effects would be resolved with reasonable confidence using 20 or more grid cells in the vertical, which would result in 492,900 grid cells in three dimensions. This problem size is well within the reaches of a model like MIKE 3 using modern desktop computers and is relatively low compared to the problem size in other modeling studies in which three-dimensionality is important, both for consulting and academic projects (see, e.g. MacWilliams et al. 2008). Therefore, Stantec should have used a three-dimensional model like MIKE 3 because the circulation in the region is highly three-dimensional and the computational overhead is not restrictive.

3.2. Model setup and forcing

Although rivers and winds are included in the MIKE 21 model, these have no bearing on the far-field results because the effects of winds and rivers are not correctly reproduced with a two-dimensional model. Other processes like waves, storm surges, and large-scale currents were also not included in the MIKE 21 model even though they are important. Finally, the MIKE 21 simulations were conducted over a one-month period which is not long enough to assess the potential for effluent to accumulate in the harbours over much longer periods.

Data from tidal, wind, and river inflow measurements were supplied to the MIKE 21 model using standard practices in coastal ocean modeling. However, owing to the two-dimensional nature of the model, the winds and river inflows have little to no bearing on the far-field results in the studies. Wind and river inflow data could be supplied to a three-dimensional model in a similar manner as it was supplied to the MIKE 21 model in the studies, although estimates for flows in all rivers and streams would need to be included (only the East River was included). As suggested in the Stantec studies, river inflows should be based on stream gauges when available, and based on approximations using the relative catchment area when unavailable (the East River inflow was inferred from measured flows in the Middle River at the Rocklin hydrometric station). With regard to tidal forcing, the standard practice was performed in which the observed tides at Wood Islands were reconstructed based on superposition of the most important components of the tides (using software such as T_TIDE; Pawlowicz 2002). However, the reduction in tidal amplitudes due to large-scale ice cover was not included in the tidal forcing (See Section 3.4 below).

The influence of wind-generated waves and swells were not included in the MIKE 21 model which is a reasonable assumption, although waves should be included during storms, as should the effect of storm surges (See Section 3.4 below). Finally, the west-to-east currents in the Northumberland Strait at speeds ranging from 6-9 cm/s (Lauzier 1965) should be included. These large-scale currents can have an important impact on transport by flushing a region that might otherwise accumulate with effluent without river flows or winds. While this will contribute to flushing of the proposed outfall at location CH-B near Caribou Harbour, it will drive the effluent southward with the potential to be entrained into Pictou and Boat Harbours. This effect is likely to be pronounced with three-dimensional modeling.

To evaluate the far-field dilution characteristics of effluent discharged from the proposed outfall locations, the MIKE 21 model was run over a total simulation time of one month during July 2016 for each outfall. This length of time is not sufficient to evaluate the effects of the effluent plumes given that the flow of effluent is not yet in equilibrium over such a short time period. The appropriate time period is dictated by the flushing time of the estuaries which can take days to months depending on the tides, river flows, winds, and large-scale circulation in Northumberland Strait. It is impossible to determine equilibrium from the spatial distributions of the effluent dilution factors (such as Figure 2.13 in the final study, showing the spatial distribution of the effluent dilution factor from the CH-B discharge location in the vicinity of Caribou Harbour after one month), since the effluent may still be accumulating in one of the harbours at the end of the month. A quantitative measure would need to be computed to demonstrate that the model is in equilibrium. For example, the total effluent mass in each harbour would need to be relatively constant in time, at least when averaged over a tidal cycle. Variations in forcing from processes that act over intervals that are longer than the tides (e.g. the spring-neap cycle, rainfall and associated river flow events, seasonal variations in winds), lead to associated slow variations in the effluent transport, and so these would need to be accounted for when assessing whether the total mass in the harbours is in equilibrium (see, e.g. Rayson et al. 2016).

In summary, the tides are the only component of the forcing in the far-field simulations that have any significant impact on the far-field dilution results. The other components of the forcing, including wind, river inflows, waves, storm surges, and large-scale currents are either not included or have little to no impact. Accurate representation of all of these effects would

need a three-dimensional model that is run for much longer than one month to account for possible accumulation in the harbours.

3.3. Model validation

Model validation is an important step in coastal ocean modeling because it demonstrates that the far-field model accurately predicts realistic currents, water levels, and other parameters. Not only is there no quantitative model validation in the studies, but the comparisons of water levels and currents to observations in Pictou Harbour demonstrate that the MIKE 21 model performs poorly. Therefore, the MIKE 21 model cannot be used to assess, with any level of confidence, the far-field behavior of the effluent discharged from the proposed outfall locations.

Validation is the most important step in coastal ocean modeling because it proves that the model is a faithful representation of what is happening in the real world. This gives the user confidence to use the model to analyze results obtained during the validation period, but more importantly during periods when there is no data so that predictions under a wide variety of scenarios can be made. An important component of validation is the availability of appropriate observational datasets. For two-dimensional modeling, these datasets should include time series of observations of sea-surface height and the east and west components of depth-averaged currents. Depending on the instrument, depth-averaged currents can be computed if the instrument measures currents throughout the water column (such as an acoustic Doppler current profiler, or ADCP), since these measurements can be averaged to produce an accurate representation of the depth-averaged currents. However, it is more common to measure currents at a point above the bed. If three-dimensional effects are weak, then the depth-averaged model result can be validated with the point measurement. Strong three-dimensionality makes it difficult to compare a point measurement to the result from a two-dimensional model, which should not be expected to produce the correct currents when three-dimensional effects are important. Three-dimensional models should be validated with velocity data at different heights above the bed in the water column and with time series of salinity and temperature near the bed and free-surface (to assess model ability to reproduce the stratification). Since three-dimensional models compute the vertical distribution of turbulent mixing, then it is desirable to obtain measurements of turbulence to validate the turbulence models. Ideally, models could validate the results of effluent transport, although such observational datasets are rare and so this is not common.

A common step that is often performed in coastal ocean model validation is what is referred to as calibration, in which model parameters that cannot be measured are varied to improve the results. Despite the availability of accurate bathymetry datasets, the bed roughness is rarely measured although it plays an important role in dictating the resistance by the bed on the flow. For example, beds covered with sands or gravels are rougher than beds that are covered with silts or muds, and so the resistance over sands and gravels should be higher. Sometimes, the roughness may be very large if there are bedforms like sand ripples or dunes. Even the drag by vegetation, corals, and kelp is modeled with an effective roughness (Fringer et al. 2019). In some cases, the roughness is approximated with knowledge of the distribution of sediments (this was accounted for in the near-field CORMIX modeling). However, the bottom roughness is more commonly used as a calibration or tuning parameter and varied to give the best match between observations and simulations. In the MIKE 21 model, the roughness is represented specifically

by the Manning's roughness parameter, which is used to compute the drag in flows with a free surface with given bed roughness properties.

After performing the appropriate calibration, it is standard practice to compare observations to simulations with quantitative metrics. There are many metrics available in the literature, although the most common are the mean error (also known as the bias), root-mean-square error, the coefficient of determination ("r-squared") and the lag, which is a measure of the time error between the observations and predictions. Another common metric is the skill score, which is a measure of the simulation error normalized by a measure of the spread in the observations. It is generally agreed upon in the coastal modeling community that a skill score greater than 0.65 characterizes excellent agreement between the model and observations (Allen et al. 2007). For simulations with tides, it is common to compare the amplitudes and phases of observed and modeled tidal constituents of both currents and water levels. These are particularly important to show that the model correctly captures the directions and magnitudes of the tidal currents. Examples of comprehensive validation of three-dimensional estuarine modeling studies can be found in MacWilliams et al. (2008) and Wang et al. (2011).

The MIKE 21 validation presented in the preliminary study by Stantec indicates that the model performs poorly because there is weak agreement between the simulations and observations. The validation is performed by running the model over a period in April 1990 when observations of water levels and currents in Pictou Harbour are available. Some statistics are computed, such as minimum, maximum, mean, and standard deviation, yet these statistics are computed separately for the observations and simulations and provide no objective measures for comparison like those found in the literature and discussed above. Despite a lack of quantitative comparisons, the qualitative comparisons represented by the figures in the preliminary study clearly indicate that the agreement between simulations and observations is poor. For example, Figure 1 below shows a comparison between simulated and measured water levels in Pictou Harbour (Figure 2-8 from the preliminary study). While the agreement in timing of the water level is good, most of the high- or low-water levels (indicated by the horizontal blue lines) are visibly incorrect. This lack of agreement could be due to wind and river forcing that was omitted from the model because of a "…lack of the simultaneous records of wind and river discharge during the period of model calibration in April 1990" (Preliminary study, p. 2.27). However, wind or flow events would produce disagreement in the tides over the duration of these events (over a few days each, such as during April 17-21), not throughout the entire record. Furthermore, attributing errors to incorrect forcing implies that the validation period is inappropriate because it does not allow for a demonstration of model fidelity through proper validation. Comparison of observed and simulated currents in Pictou Harbour in Figure 2-9 of the preliminary study shows that the model underpredicts the current speeds by roughly 20% at Location #1 and roughly 50% at Location #2, and in some cases by 80%. This level of disagreement is unjustifiable. Furthermore, there is no indication that the model correctly simulates the direction or timing of the currents since only current speeds are compared.

The differences between observations and simulations is attributed to "the nature of stratified currents through the water column from surface to the seabed, as well as the difference in bathymetry between the existing condition and that in 1990" (Preliminary study, p 2.28). If the difference is indeed due to stratification effects, then this justifies the need for a three-dimensional model. Differences in bathymetry would indicate that the choice of the validation period is not suitable because the circulation in the region was fundamentally different in 1990 than it was when the bathymetry datasets were collected over the past decade. Of course, it is

always desirable to use more recent observations to ensure that the results are not contaminated by differences between the dates in which the bathymetry and flow measurements were made. However, a more careful validation procedure and use of an appropriate model should be able to indicate whether this is the case and if more recent data is needed. Regardless, the bottom line is that simply more observations are needed to prove that the model simulations are accurate. Even if the validation indicated that the simulations of currents and water levels in Pictou Harbour were excellent, it would be difficult to argue that the model also correctly reproduced currents in and around Caribou Harbour unless there were observations of water levels and currents from at least one station in that region.

In summary, the validation suggests that the model does not correctly predict the magnitude, direction, or timing of the currents. Therefore, in addition to a lack of validation in or near Caribou Harbour, the results provide no confidence that the model can accurately compute the currents and simulate the subsequent far-field fate and transport of the effluent from any of the proposed outfall locations. Furthermore, the validation provides no measure of confidence that can be ascribed to the predictions of ambient currents or directions at any of the six sites for use in the near-field modeling studies (See Section 4.2 below).

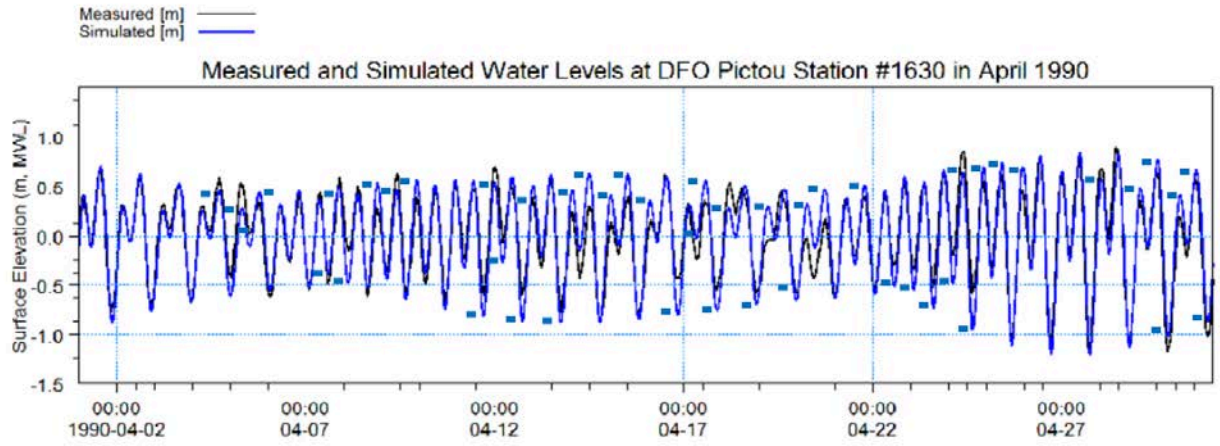

Figure 1: (Figure 2-8 from the preliminary study): Comparison of simulated to measured water levels in Pictou Harbour during April 1990. The blue horizontal lines were added to indicate incorrectly predicted low or high water levels.

3.4. Model scenarios

The scenarios that were conducted in the studies could only evaluate (unsuccessfully) the effect of the tides in a two-dimensional model. Many more scenarios are needed using a three-dimensional model to assess the potential impacts of winds, river inflows, large-scale currents in the Northumberland Strait, waves, storm surges, and ice during winter.

The far-field model scenarios in the studies were carried out with environmental conditions that are stated to minimize mixing of the effluent plume, thus producing conservative results. The conditions include use of "smaller tidal ranges, warmer ambient waters, less wind-driven surface currents, and lower freshwater flows from rivers" (Final report, p. 3). Warmer ambient waters during summer are conservative because, "in winter, mixing is effectively enhanced due to the larger difference in temperature and salinity (density) conditions" (Final report, p. 3). Wave and

storm surge conditions are not included in the model given that "surge tides generate turbulence and ultimately provide better and faster mixing conditions" (Answer #2, Response to questions).

While some of these conditions are indeed conservative, not all are relevant or necessarily conservative, particularly in a two-dimensional model. Because the far-field model is two-dimensional and there is no vertical density stratification, the far-field plume dynamics are insensitive to the density of the effluent plume. Therefore, two-dimensional results should be the same for ambient summer or winter temperature conditions. A difference between two-dimensional effluent transport results in summer and winter could, in principle, be based on different initial effluent concentrations derived from the near-field model while taking into account the different ambient conditions from observations. However, the discharged effluent concentration in the far-field model is arbitrary because the dilution factor is a ratio of the far-field to discharged effluent concentration, and thus the actual concentration discharged from the outfall is irrelevant. A reduction in tidal and wind-driven currents reduces the vertical mixing of the plume, although again this has no bearing on the far-field results because the plume is vertically well-mixed in the two-dimensional model. However, different tidal conditions affect the tidal dispersion in the two-dimensional model and thus the tides have a significant impact on the far-field results. Wind-driven currents also affect the far-field results, but these effects are weak in a two-dimensional model since it does not account for wind-driven recirculating currents. Smaller river inflows may also be more conservative because they would be less likely to flush effluent out of the harbours. However, wind and river inflow effects can only be correctly simulated with a three-dimensional model, since both winds and river inflows can transport effluent into the harbours (See Section 3.1 above). Finally, while waves and storm surges indeed provide more mixing and dilution in the near-field, the surge has the potential to transport offshore effluent into the harbours, thus it may potentially be less conservative in terms of far-field transport.

Ice plays a significant role in the circulation and far-field effluent transport in coastal areas like Pictou and Caribou Harbours, yet its effects were not incorporated into the MIKE 21 model in the Stantec studies. While there are frameworks that can couple a model for ice formation and melting to a model like MIKE 21 (e.g. Kusahara and Hasumi 2013), it is possible to approximate the effects of ice sheets by imposing friction at the ice-water interface in the circulation model that impedes the flow of water due to the friction from the ice (Georgas 2012). In smaller domains like those in the Stantec studies, in addition to friction from the ice, the tidal boundary conditions must be altered to account for the significant reduction in tidal amplitude due to ice cover over the Gulf of St. Lawrence (Smith et al. 2006). Alternatively, these boundary conditions must be obtained from data measured during winter when there is large-scale ice cover. In shallow areas, the flow may be completely blocked when ice freezes over the entire water column, in what is referred to as "fast ice" by fishermen in the Pictou area (MacCarthy and Egilsson 2019). In the final Stantec study (p. 3), it is indicated that a winter scenario and the associated effects of ice are not considered because "the presence of ice cover would increase turbulence at the ice/water interface by providing resistance to the ambient water currents, resulting in higher mixing and dilution". Indeed, higher mixing and dilution may take place and can be modeled in the near field with CORMIX, but turbulent mixing at the ice/water interface is not accounted for in the far-field model because it is two-dimensional. Instead, the effect of ice in the far-field model is to reduce the magnitude of the currents and reduce the potential for far-field dilution. Therefore, a winter model run with extensive ice cover and appropriate boundary

conditions is needed to represent a worst-case scenario for the far-field dispersion despite the substantial initial dilution of the strongly buoyant effluent during this period.

Overall, the scenarios in the Stantec reports do not reproduce the impact of different physical processes over the course of the year on the effluent transport in the region. In its current form, the far-field model can only be used to simulate the influence of tides on the far-field dispersion of the effluent plumes during low flow and low wind conditions in the absence of ice and large-scale currents. To obtain a good understanding of all of the possible scenarios that might impact the far-field transport, a three-dimensional model would need to be run under scenarios that demonstrated the effects of (1) strong/weak winds, (2) strong/weak river flows, (3) with/without ice cover (including the associated weaker tidal forcing and possibly fast ice), and (4) with/without large-scale currents through the Northumberland Strait. In each of these scenarios, the model would need to be run for at least as long as the flushing time to ensure that the far-field effluent field reaches equilibrium. If the flushing time is not much longer than a spring-neap tidal cycle, then additional scenarios would need to be run to understand the impact of strong (spring) vs. weak (neap) tides. The freshwater inflows would need to include all possible rivers and effluent from municipal wastewater treatment plants, given that the worst-case scenario may include freshening of the receiving waters to a point that significantly impacts the near-field dilution (See Section 4.2 below). Finally, storm surge scenarios would need to be studied given the possibility of strong waves and surges in the region, which could lead to significant accumulation in the harbours.

3.5. Results

The particle tracking module in MIKE 21 over-approximates the far-field mixing and dilution because of the assumption of uniformly distributed effluent mass throughout the volume of each grid cell. This gives the best-case scenario because it mixes the effluent from a point discharge completely over the water column, thus eliminating the possibility of higher concentrations confined to near-surface or mid-water layers of effluent. As a result, the assessment by Stantec that the far-field dilution factors for most of the region surrounding site CH-B are above 100 at the end of the one-month simulation period is overly optimistic. Accounting for vertical variability in the plume could lead to much smaller dilution factors but this would require a three-dimensional model. Dilution factors are also over-approximated in Caribou Harbour because the simulations are not run for long enough time to allow for accumulation of effluent in the harbour due to tidal dispersion.

As they are presented in the reports, the far-field modeling results provide only qualitative, and in some cases misleading, information about the far-field fate and transport of effluent from the proposed outfalls. The focus of this section is on Figures 2.5-2.13 in the final study, which depict extremely low concentrations of the effluent field around site CH-B. For example, in Figure 2.5 there is a small patch of effluent located over the outfall which appears to have a concentration of 2-3 mg/L. It is hard to imagine how the concentration of the effluent from the outfall could have diluted by nearly a factor of 50 (from 100 mg/L) even though this figure depicts the concentration field at slack tide during a neap tidal cycle. As discussed in Section 2.2 above, during slack tide we expect higher concentrations due to buildup of effluent because currents are too weak to induce any significant transport away from the outfall. Higher effluent concentrations are also expected because turbulent dispersion is ignored in the particle tracking

module of MIKE 21 to promote conservative dilution factors. It is possible that a diluted concentration from the outfall is imposed in the far-field model based on the near-field modeling results, although an arbitrary concentration of 100 mg/L is assumed given that the relative concentration is of interest.

The low concentrations in the figures can be explained by the particle tracking module that is used to transport effluent in MIKE 21. In the particle tracking module, the outfall is modeled as a point source from which particles with a given amount of mass are released at specified time intervals. After being released, the particles are transported by currents computed with the MIKE 21 hydrodynamic module. In the Stantec final study, the mass flow rate from the outfall is given by 0.1 kg/s, based on the assigned concentration of 100 mg/L and flow rate of 1 m^3/s. Therefore, if we assume that one particle is released from the outfall every hydrodynamic time step of 60 s (the details of how often particles are released are not provided, although this is a safe assumption), then it must be assigned a mass of 6 kg. It is possible to release particles at shorter intervals or multiple particles at each time step, with mass divided equally among the particles to ensure the same prescribed mass flow rate of 0.1 kg/s. However, there would be no difference between transport of a single particle and a group of particles because particles in a group do not spread over time due to a lack of turbulent dispersion, which is ignored by Stantec in the particle tracking simulations. In addition to a lack of dispersion, there is no decay assigned to the particles in the Stantec studies, and hence the mass of each particle remains fixed during the simulations.

To convert the distribution of particles to a concentration field on the hydrodynamic grid, the total mass in each grid cell (which is the sum of the masses of all of the particles in each cell) is divided by the volume of the grid cell. Assuming the grid resolution around site CH-B is approximately 25 m (based on the mesh shown in Figure 2.3 in the final study), then the volume of the prismatic grid cell containing the point release at the location of outfall CH-B is approximately 6000 m^3, based on a depth of 20 m and cross-sectional area of approximately 300 m^2. The minimum concentration in this cell can be estimated by assuming it is empty and then filled with 6 kg of effluent after one 60-s time step. Since it is assumed that this mass is uniformly distributed over the cell volume, the resulting effluent concentration will be 1 mg/L, implying a dilution factor of 100 relative to the assumed inflow concentration of 100 mg/L. This shows that conversion of the particle mass to a concentration field results in artificial mixing of the effluent, giving rise to effective mixing and dilution that depend to great extent on the mesh resolution, depth, and details of the particle release at the outfall (i.e. particle release time interval, mass per particle, number of particles per interval). Although these details are not provided in the Stantec studies, it is clear that much of the far-field dilution is an artifact of the way in which the concentration fields are calculated.

The artificial dilution arising from two-dimensional particle tracking simulations like that in the MIKE 21 model is a common feature of coastal ocean modeling. It is possible to reduce the dilution by increasing the particle release rate or by decreasing the grid size. However, decreasing the grid size is often difficult given computational constraints associated with far-field studies on grids that are finer than those in the Stantec studies. Regardless of grid resolution or the details of the particle tracking module, conclusions about far-field mixing and dilution derived from particle tracking results in a two-dimensional model should take the inherent overestimation of mixing and dilution factors into account. In this regard, Figures 2.5-2.13 in the final study cannot be used to conclude that the environmental impacts of the effluent from outfall CH-B are negligible simply because the dilution factor is at least 100 in most of the domain at

the end of the 1-month period. Instead, these dilution factors represent the best-case scenario in which the effluent is mixed over the water column instantaneously upon being released from the outfall. Owing to the buoyant nature of the near-field plume and other three-dimensional effects, the effluent could be confined to a layer much smaller than the depth (as discussed in Section 3.1). As indicated by the near-field modeling results in the final study, this layer can be as small as 1-2 m, which would lead to a reduction in the dilution factor in the region surrounding the CH-B outfall by a factor of 10 or more because the effluent is not completely mixed over the water column. A three-dimensional model would be able to account for the vertical variability of the effluent plume through use of the near-field model to inform the vertical variability in the vicinity of the outfall. This would reduce the artificial dilution associated with the assumption of complete mixing over the water column in a two-dimensional model.

An additional perplexing aspect of Figures 2.5-2.13 in the final study is that they appear to depict transport of patches created by pulses of effluent discharges rather than trails of effluent emanating from the continuous-in-time discharge at outfall CH-B. Examples of such an effluent field showing trails emanating from the outfall locations are depicted in Figures 2-20 and 2-21 from the preliminary study, which show the effluent concentration field surrounding sites Alt-C and Alt-D near Pictou Harbour. Effluent trails are not visible around site CH-B in Figures 2.5-2.13 from the final study because the overestimated dilution due to the particle tracking module produces concentrations in the trails that are too low to be visible with the given color scale. Instead, higher-concentration patches (that also have artificially low concentrations) oscillate with the tides while slowly propagating away from the outfall with the weak non-tidal flow produced by the tides (see Section 2.2 for a discussion of tidal vs. non-tidal flows). While these simulations indicate that there is some dilution of the effluent patches since their concentrations decay in time, the dilution is representative of the best-case scenario when compared to the effluent concentration at the outfall of 100 mg/L.

Another process that is likely reducing dilution factors but is not represented in the simulations is accumulation in Caribou Harbour. Figure 2.11 in the final study clearly shows a patch of effluent in the harbour at slack high tide, indicating that it was transported into the harbour during the previous flood tide. Although the patch appears to be leaving the harbour during the subsequent ebb tide (Figure 2.12 in the final study), tidal dispersion is expected to transport effluent into the harbour over many tidal cycles. Furthermore, although inclusion of turbulent dispersion in the particle tracking module would act to dilute the patches, it would accentuate the tidal dispersion and promote transport into the harbour, thereby reducing the dilution in the harbour after many tidal cycles. As discussed in Section 3.4, accumulation in Caribou Harbour would need to be quantified with simulations that were run for sufficient time to demonstrate that the effluent mass in the harbour was not changing in time.

In summary, when computing concentration fields from the particle tracking results, uniform and instantaneous mixing over the grid cell volumes leads to artificially low concentrations and high dilution factors associated with far-field effluent transport from site CH-B. While it is impossible to eliminate this effect, it can be thought of as the best-case scenario in which the outfall plume is uniformly mixed over the water column. As demonstrated by the near-field modeling results in the Stantec studies, this is clearly not the case. Instead, the plume is typically confined to a smaller region in the water column, which implies a much smaller dilution factor when compared to that arising from assuming a uniform effluent concentration over the depth. The artificially low concentrations and high dilution factors produce far-field effluent concentrations in the region surrounding the CH-B outfall after a month-long simulation

that are greater than 100, which is an overly optimistic result. The artificial dilution eliminates most of the visible effluent in the figures except for a few small patches that oscillate with the tides. Some of these are transported into Caribou Harbour, indicating the potential for accumulation in the harbour due to tidal dispersion, an effect that should be assessed with simulations over much longer time periods than the 31-day simulations conducted in the final study.

4. Review of the near-field modeling

4.1. Overview of CORMIX

The CORMIX model was used to compute the three-dimensional effluent concentration field in the near-field mixing zone, which is generally defined as the region within 100 m of the outfall. Near-field mixing involves detailed flow and turbulence processes over length scales that are much smaller than the grid in the far-field model. Therefore, they cannot be simulated with MIKE 21 and must be modeled with a near-field model like CORMIX. According to the CORMIX model, the "near-field" is defined as the region between the outfall and the point at which the buoyant plume interacts with a boundary, which can be the bed, the free surface or some intermediate layer in the water column. In this near-field region, the plume dynamics are initially dictated by the high velocity flow and turbulence emanating from the outfall ports which rapidly mix the effluent with ambient waters. Once the high momentum fluid has decelerated (typically within 5-10 meters of the outfall ports), buoyancy-driven turbulence and mixing take over as the plume rises to the surface or at some point in the water column where the plume density matches the density in the water. This could be the thermocline (a point below the surface that separates the warmer, surface waters from the colder, bottom waters) or the halocline (a point at which fresher river waters are separated from the denser, saltier ocean waters below). After reaching the surface or intermediate layer, subsequent dynamics are referred to as the "far-field" zone in CORMIX. In this zone, the plume is transported by the ambient currents while spreading laterally due to weaker buoyancy effects. Once the density of the plume mixes with that of its surroundings, it propagates as a passive plume (i.e. no longer spreading due to buoyancy) with the ambient currents while spreading laterally and horizontally due to the ambient turbulence. This stage of plume development is modeled in CORMIX in a way that is similar to how it would be modeled under similar ambient conditions in a three-dimensional circulation model like MIKE 3.

The CORMIX model predicts the shape of the near-field plume in three dimensions based on the relatively complex geometry of an outfall diffuser, including the ability to specify different numbers of ports and the specific geometry of how they are attached to the diffuser pipe resting on the bed. Because CORMIX solves for the plume characteristics in a much smaller area and over much shorter time periods when compared to those in the far-field model, the characteristics of the flow needed to drive CORMIX are much simpler than the boundary conditions needed to drive the MIKE 21 model. As a result, parameters in CORMIX are generally not tuned, unlike the far-field modeling which requires tuning of, for example, the bottom roughness to improve agreement between observed and simulated currents (See Section 3.3 above). Furthermore, validation of CORMIX results is generally not required given that, at least under the scenarios that can be simulated with the CORMIX package, we expect the model to produce a good approximation of the near-field dynamics. The downside to this simplicity is

that the results depend critically on choosing the effluent and ambient parameters that are representative of realistic worst-case conditions that would give the least amount of near-field dispersion and thus representative of the most conservative design scenario. As discussed in the next section, the receiving water conditions do not represent worst-case scenarios.

4.2. Near-field results at location CH-B

The receiving water current and ambient density field supplied to the CORMIX model to predict the near-field mixing and dilution at site CH-B are not representative of worst-case scenarios because the current is too strong and the ambient density is too high. This gives an over-prediction of the mixing and near-field dilution within the 100-m mixing zone surrounding site CH-B. The near-field effluent concentrations are expected to be higher, particularly during periods of high river inflows and when the tidal currents are weaker, such as during neap tides or when there is winter ice cover.

In the final study, two scenarios for the near-field mixing at site CH-B were conducted. The only difference between the two scenarios is the use of one port in the diffuser in the first scenario and three ports in the second. The dilution factor for the three-port design was roughly twice as large as that for the one-port design 100 m from the outfall (Table 3.4 in the final study). The three-port design at site CH-B had a dilution factor that was roughly 30% larger than the six-port design at site Alt-D (Table 4.1 in the final study shows results from site CH-B obtained in the final study and results from site Alt-D, which are repeated from the preliminary study). Despite the likely increase in the dilution factor at CH-B with six ports, it was concluded that the three-port design had a favorable seabed footprint with a lower potential to interact with the seabed than the six-port design, and hence the six-port design was not evaluated at site CH-B. Given the incorrect estimates of the worst-case currents and receiving water density discussed below, studies need to be conducted with three- and six-port designs to understand their characteristics under worst-case scenarios, particularly in the presence of vertical density stratification of the water column.

The inputs to the CORMIX model that have the most significant impact on the near-field mixing in the final study are the effluent flow rate and density and the ambient tidal currents and density. The effluent flow rate was fixed at the annual average rate of 0.98 m^3/s, while the effluent salinity was assumed to be 4 g/L = 4 kg/m^3, the densest value in the reported range of 1-4 g/L. The effluent temperature was reported to be 25°C in winter and 37°C in summer. The summer effluent temperature was chosen under the assumption that the plume would be least buoyant in summer when the receiving waters were at their warmest. The values chosen for the effluent salinity and temperature are stated to give an upper bound for its density, thus giving a conservative estimate for the dilution because more buoyancy-driven mixing is expected to take place if the effluent is less dense than the receiving waters. Using the UNESCO equation of state (UNESCO 1981), a salinity of 4 kg/m^3 and temperature of 37°C give an effluent density of 996 kg/m^3, the value used in the final study.

A key assumption in the CORMIX model is that the ambient currents are steady. Therefore, approximations are needed when applying CORMIX to tidal flows that are unsteady in that the ambient currents flowing past the outfall vary in magnitude and direction over the tidal cycle. When currents are weak, the effluent accumulates above the outfall and dilution is poor. However, the worst-case scenario occurs roughly one hour before or after slack tide when

currents are weak yet sufficient to re-entrain the effluent that was recently transported away from the discharge location in the opposite direction before slack tide. CORMIX requires information about the tidal period and peak currents and the magnitude of the ambient currents one hour before or after slack tide in order to provide an estimate of the worst-case scenario. The CORMIX manual (Page 33 of Jirka et al. 1996) also recommends that additional scenarios be conducted with tidal currents at intervals of one or two hours at different stages of the tidal cycle to ensure that all possible scenarios are analyzed.

Based on the information provided in the preliminary and final studies, the ambient current supplied to the CORMIX model does not represent the worst-case mixing scenario. The preliminary report mentions the use of tidal information in the CORMIX simulations, stating that, (p. 3.54) "The results are presented for a time step corresponding to 1 hour before slack tide conditions." However, in the final report only average (10 cm/s) and maximum (27 cm/s) tidal currents are supplied based on MIKE 21 simulations in July 2016 at site CH-B. There is no mention of the tidal current speed expected within one hour of slack tide, as needed for the worst-case calculation in CORMIX. Furthermore, simulations are not conducted during different phases of the tidal cycle as suggested in the CORMIX manual. These would demonstrate the impact of current speed and direction on the dilution factor. The direction, in particular, could impact the effect of the diffuser and port alignment relative to the oscillatory flow. An important implication of the worst-case slack tide is that suspended solids may settle onto the bed within 100 m of the outfall because of the weak currents, as discussed in Section 2.2 above. This possibility is not mentioned or modeled in the Stantec studies.

Regardless of whether the details of the tide are incorporated into CORMIX, the ambient currents applied to CORMIX in the final study are too large to represent a worst-case scenario. Based on Figure 2-14 in the preliminary report, which shows the Northumberland Strait water levels over the 31-day MIKE 21 simulation period, the weakest neap tide on July 14 has a tidal range of 0.6 m, which is more than three times smaller than the strongest spring tidal range of 2 m on July 5. Therefore, the average and maximum tidal currents used in the CORMIX scenarios are much larger than they would be in the worst-case scenario because they are impacted by the large spring tides. A more conservative, worst-case tide would be given by the weakest neap tide during the period, since the weaker currents would have significantly less near-field dilution than the average tide over the 31-day period. It is important to note that, given the insufficient far-field model validation presented in Section 3.3 above, the simulations of the currents at CH-B may not be representative of the actual currents. This implies that if the currents are underpredicted in Pictou Harbour, they will not necessarily be underpredicted at site CH-B, and therefore it is not valid to justify use of inaccurate far-field model results based on the notion that the errors would lead to a more conservative worst-case scenario.

The ambient density field supplied to the CORMIX model is equally as important as the ambient currents. Estimates of the ambient density of the receiving waters were based on observations because the far-field model is two-dimensional (See Section 3.1 above). However, because observations of temperature and salinity at site CH-B were not available, the ambient density was based on observations in the Pictou Road region in August 2014 and September 2006 (Appendix B, Preliminary study). In principle, this would provide a conservative receiving water density given the likelihood that the receiving water salinity, and hence its density, was lower in this region due to more inflows into Pictou Harbour than Caribou Harbour. However, as discussed below, this is not the case. Using data from Pictou Road region, the receiving water density was calculated as 1020 kg/m^3 based on a temperature of 17.6^{o}C and salinity of 28 ppt,

which are averages of the observations. With these salinities and temperatures, the effluent is (1020 kg/m^3 – 996 kg/m^3) = 24 kg/m^3 less dense than the receiving waters. According to Stantec, this provides sufficient buoyant mixing to produce far-field dilution factors computed by CORMIX that are within established water quality guidelines for the 100-m mixing zone. Owing to the strong near-field mixing by the three-port diffuser, the plume interacts with the bed up to 25 m away from the outfall. However, the dilution factor of 71 at 10 m indicates this should not be a source of concern for this value of the ambient density.

Rather than using average salinity and temperature values of observations for the ambient, a more conservative scenario for the near-field modeling would have been to use the freshest and warmest observations in the region, which should be 23 ppt instead of 28 ppt and 19.4^{o}C instead of 17.6^{o}C (Appendix B, Preliminary study). This would give a receiving water density that is 4 kg/m^3 less dense than the value used in the final study, yielding a less buoyant effluent plume and less near-field dilution. While it is unlikely that the water temperature would be much warmer than 20^{o}C in the region, waters warmer than 20^{o}C would contribute much less to potential reductions in ambient density than lower salinity values. This is because the density can vary by as much as 25 kg/m^3 due to the 0-31 ppt salinity range in the region (based on data from Galbraith et al. 2014), while it can only vary by 3 kg/m^3 due to the 0-20^{o}C temperature range. In fact, the salinity value of 28 ppt that was used for the scenario is close to the maximum observed salinity in the region of 31 ppt, thus reflecting close to the best- rather than worst-case salinity for buoyancy-driven near-field dilution at site CH-B. A worst-case salinity is likely much smaller given that salinity observations in the East River range from 20 ppt during low-flow periods to as low as 5 ppt during high-flow periods (Preliminary study, p. 2.21). Lower salinity values are also likely near Caribou Harbour, although perhaps not as low given that flows into Caribou Harbour are weaker than those into Pictou Harbour. Nevertheless, all inflows in the region are expected to lower the salinity of the receiving waters surrounding the proposed outfalls in the studies.

The effect of salinity on the near-field dilution is weakest in winter when inflows are at their lowest. Combined with the colder receiving waters, winter ambient density scenarios are not needed given their potential to drive more buoyancy-driven turbulence and near-field dilution. However, given the weaker tidal currents due to ice cover in winter, scenarios would need to be conducted with worst-case winter density values for the ambient and effluent combined with model-derived worst-case weak winter tides during the period of peak ice cover.

In addition to the potential for low salinities to impact the near-field dilution by reducing the effluent buoyancy at site CH-B, low salinities indicate the existence of vertical stratification in which fresher, river water overlies saltier, denser ocean water. For example, observations in the Pictou Road region indicate a top-bottom salinity difference in July 1995 of 7.5 ppt (Preliminary study, p. 2.21), which is the dominant driver of the top-bottom density difference of 5.8 kg/m^3 (See Section 3.1 above). The stratification can reduce near-field dilution by trapping the effluent in a layer beneath the ocean surface where the density of the effluent matches that of the water column. Additionally, the trapping leads to far-field transport at depth rather than at the surface, thus having the potential to propagate toward the fresh water source. In the case of site CH-B, this would mean transport of the effluent into Caribou Harbour (See Section 3.1 above for a more thorough discussion of three-dimensional far-field effects). The CORMIX model has the ability to simulate near-field dilution in the presence of vertically-stratified waters, and the manual suggests including these effects when the vertical variation in density is greater than 0.1 kg/m^3 (Page 33 of Jirka et al. 1996), significantly smaller than the observed top-bottom density

difference of 5.8 kg/m^3 mentioned above. Therefore, worst-case dilution scenarios at CH-B should be devised that take into account the potential for low salinity and stratification arising from high freshwater inflows in the region. These scenarios would need to be devised using results from three-dimensional, far-field modeling.

5. Summary

The MIKE 21 and CORMIX models were used to simulate the distribution of near- and far-field effluent discharged from proposed outfall locations in and near Pictou and Caribou Harbours. Although there are numerous metrics that are commonly used to validate far-field model results like those in the MIKE 21 simulations, these are not calculated in the study. Instead, only qualitative comparisons to observations are made, and these indicate that the far-field model is poorly reproducing the currents and water levels throughout the domain. Therefore, as it is implemented, the far-field model is inaccurate and cannot be trusted to faithfully represent actual circulation and transport dynamics in the region. Given the strong three-dimensional nature of the circulation and transport dynamics due to the winds and fresh water flows in the region, three-dimensional processes are expected to significantly impact the far-field transport. Therefore, the two-dimensional MIKE 21 model is not appropriate for use in this study.

In addition to the inaccurate nature of the far-field model, the scenarios that are presented are not representative of the multitude of processes that can impact the far-field circulation and effluent transport. While there is some qualitative evaluation of the impacts of tidal currents on the far-field fate of the effluent, the two-dimensional nature of the MIKE 21 model makes it impossible to predict the effects of strong winds or strong river inflows, effects that can significantly impact the far-field dynamics. For example, freshwater flows and wind-driven circulation can drive effluent into Caribou Harbour from site CH-B, leading to more accumulation than what might be predicted by the two-dimensional model. Furthermore, although near-field dilution may be accentuated in winter owing to the stronger temperature difference between the effluent and receiving waters, there is no assessment of the potential worst-case winter scenario in which reduced tidal currents due to ice cover may significantly reduce both near- and far-field dispersion. Similarly, while the turbulence and mixing due to storm surges and waves would likely increase near-field dilution, there are no simulations conducted to assess their impact on far-field transport, including the potential for accumulation of effluent in the harbours. Finally, the simulations are not conducted over sufficiently long time periods that are needed to ensure that the simulated far-field dilution factors are in equilibrium, making it impossible to assess the potential for accumulation of effluent in regions of the domain with weaker dispersion and flushing, such as the harbours.

Qualitative representation of the far-field dilution dynamics around site CH-B in the figures indicates fundamental inconsistencies with how the effluent concentrations are being computed and interpreted. The concentrations are unphysically low because the model assumes uniform effluent concentrations within each grid cell. This leads to an over-approximation of the far-field mixing and dilution and overly optimistic conclusions about the far-field dilution factors in the vicinity of the outfall at site CH-B, which are reported to be above 100 in most of the region after a one-month simulation. In reality, the effluent concentrations can vary significantly in the vertical, since effluent plumes can be confined to layers near the surface or mid-water, leading to higher concentrations and smaller, more realistic dilution factors. Due to the artificial dilution, trails of effluent emanating from the outfall are not visible in the figures because their

concentrations are too small to appear with the given color scale. Instead, small patches of effluent oscillate with the tides, with some propagating into Caribou Harbour. These indicate the potential for accumulation of effluent in Caribou Harbour by tidal dispersion, an effect that can only be captured with simulations that are run over much longer time periods.

Based on the near-field results obtained with the CORMIX model in the final study, Stantec concluded that the dilution factors near the outfall located at site CH-B are within established water quality guidelines for the 100-m mixing zone. However, the ambient currents and densities supplied to CORMIX are not representative of worst-case near-field dilution scenarios. The currents are based on the average and peak tidal currents at site CH-B over the 31-day simulation period, which are too high because the data include two spring tides. A worst-case tidal current would be better represented by a neap tide during this period, which has smaller currents and is therefore expected to induce less near-field dilution, particularly when accounting for accumulation during slack tide. Weaker tidal currents due to winter ice cover further reduce the potential for near-field dilution, although this scenario is also not investigated. Finally, despite the potential for settling of suspended solids during slack tides within 100 m of the outfall, this is not mentioned in the Stantec studies.

In addition to the overestimated tidal currents, the ambient density supplied to CORMIX is also not representative of a potential worst-case scenario. The salinity used to compute the receiving water density is more representative of the maximum salinity in the region, which gives an effluent that is far too buoyant and thus an overprediction of the near-field buoyancy-driven mixing and dilution. The worst-case salinity, and hence receiving water density, should be much lower given the potential for high river flows to reduce the salinity in the region. Furthermore, high river flows would produce vertical salinity stratification or layering in which fresh water overlies salt water, an effect that can be included in the CORMIX model and further acts to reduce near-field dilution.

6. References

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Smith, G.C., F.J. Saucier, and D. Straub, 2006, Response of the lower St. Lawrence Estuary to external forcing in winter, Journal of Physical Oceanography, 36, 1485–1501, (Freely available online).

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APPENDIX A-2

Oliver B. Fringer

Curriculum Vitae

Academic History

2003 Ph.D. in Civil and Environmental Engineering
Stanford University, Department of Civil and Environmental Engineering
Dissertation: Numerical simulations of breaking interfacial waves

1996 Master of Science in Aeronautics and Astronautics
Stanford University, Department of Aeronautics and Astronautics

1995 Bachelor of Science in Aerospace Engineering, *cum laude*
Princeton University, Department of Mechanical and Aerospace Engineering

Employment Record

2011-present Associate Professor (with tenure), Dept. of Civil and Environmental Engineering, Stanford University

2003-2011 Assistant Professor, Dept. of Civil and Environmental Engineering, Stanford University

2006 Engineering Consultant, Chevron Energy Technology Company

2002-2003 Acting Assistant Professor, Dept. of Civil and Environmental Engineering, Stanford University

2002-2003 Lecturer, Depts. of Mathematics and Computer Science, University of the Western Cape, Cape Town, South Africa

2001-2002 Postdoctoral Researcher, Environmental Fluid Mechanics Laboratory, Stanford University

1996-2001 Research Assistant, Dept. of Civil and Environmental Engineering, Stanford University.

1994-1995 Summer Research Assistant, Dept. of Mechanical and Aerospace Engineering, Princeton University.

1993 Summer Intern, U. S. Dept of State, Foreign Building Operations, La Paz, Bolivia.

1992 Summer Intern, U. S. Consulate, La Paz, Bolivia.

Professional Activities

Scientific committees and conference sessions organized

Co-organizer for session, “Internal Waves/Tides and Sediment Processes on Continental Margins”, 2018 Ocean Sciences Meeting.

Co-organizer and chair for session, “Multiscale topographic effects on large-scale flow: From wakes and lee waves to small-scale turbulence and mixing”, 2018 Ocean Sciences Meeting.

Conference Chair, 16th International Workshop on Multi-scale (Un)-structured mesh numerical Modelling for coastal, shelf and global ocean dynamics, Stanford, CA, August 29-September 1, 2017.

Scientific Committee, 15th International Workshop on Multi-scale (Un)-structured mesh numerical Modelling for coastal, shelf and global ocean dynamics, Toulouse, France, September 27-29, 2016.

Scientific Committee, 14th International Workshop on Multi-scale (Un)-structured mesh numerical Modelling for coastal, shelf and global ocean dynamics, Portland, OR, September 28-30, 2015.

Co-organizer for Session, “Measuring and modeling internal waves and the turbulence cascade: a tribute to David Tang”, 2014 Ocean Sciences Meeting.

Organizing Committee, 63rd Annual Meeting of the APS Division of Fluid Dynamics, 2014.

International Scientific Committee, 7th International Symposium on Environmental Hydraulics, Singapore, 2014.

Scientific Committee, 11th International Workshop on Multi-scale (Un)-structured mesh numerical Modelling for coastal, shelf and global ocean dynamics, Delft, Netherlands, 28-30 August 2012.

Co-organizer for Session, “Transport and mixing due to nonlinear internal gravity waves”, 2012 Ocean Sciences Meeting.

Co-organizer for Session, "Mini-Symposium on Computational Strategies for the Simulation of Nonlinear Waves and Turbulence in Environmental Flows", 63rd Annual Meeting of the APS Division of Fluid Dynamics, 2010.

Scientific Committee, Geophysical and Astrophysical Waves, Les Houches, Chamonix, Feb 6-11, 2011.

Scientific Committee, 9th International workshop on Multiscale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, Cambridge, MA, 17-20 August 2010.

Scientific Committee, Third International Symposium on Shallow Flows, June 2012, U. Iowa.

Scientific Committee, Ninth International Workshop on Unstructured Grid Modeling of Coastal and Ocean flows (2009).

Session organizer and chair (jointly with T. Peacock and D. Farmer) for AGU Fall Meeting Session OS15: "Internal Waves" (2008)

Session organizer and chair (jointly with S. Ramp and J. Lynch) for AGU Ocean Sciences Session O86: " Nonlinear Internal Wave Observations, Dynamics, and Acoustic Impacts" (2008)

International Scientific Committee, 15th Congress of Asia and Pacific Division of International Association of Hydraulic Engineering and Research, Chennai (2006)

Session organizer and chair (jointly with J. Nash) for AGU Ocean Sciences Session OS11J: "Dynamics of highly nonlinear internal waves" (2006)

External thesis evaluator

1. Subasha Wickramarachchi, “The hydrodynamics of two-dimensional oscillating flows over ripples: The effects of asymmetries in ripple shape and currents”, The University of Waterloo, 2017.

2. Cintia Luz Ramón Casañas, “Hydrodynamics and mixing at river confluences: On the influence of buoyancy and the tides”, The University of Granada, 2016.

3. Mario César Acosta Cobos, “Computational improvement of 3D hydrodynamic semi-implicit models for oceans and continental water simulations”, The University of Granada, 2016.

4. Olga Kleptsova, “On techniques for modelling coastal and ocean flows with unstructured meshes”, Technical University of Delft, 2013.

Reviewer/advisory service

NERRS Science Collaborative Research & Integrated Assessment Reviewer (2016)

San Francisco Estuary Institute Bay Modeling Advisory Team (2013)

Link Foundation Selection Committee (2013, 2014)

National Science Foundation (NSF) Reviewer, Physical Oceanography Program (2003-)

National Science Foundation (NSF) Panelist, Collaboration in Mathematical Geosciences, Jun 2-4, 2010.

Dept. of Energy (DOE) Computational Science Graduate Fellowship (CSGF) application screening committee, (2009-)

Journal referee

Advances in Water Resources (2009, 2010)
Boundary-Layer Meteorology (2009)
Coastal Engineering (2009)
Communications in Nonlinear Science and Numerical Simulation (2011)
Computers and Fluids (2010, 2011)
Computers and Geosciences (2009)
Continental Shelf Research (2004, 2013)
Deep-Sea Research (2011)
Dynamics of Atmospheres and Oceans (2007)
Ecological Applications (2006)
Environmental Fluid Mechanics (2013, 2015, 2017)
Environmental Practice (2015)
Estuaries and Coasts (2007, 2015, 2016)
European Journal of Mechanics - B/Fluids (2008, 2011)
Flow, Turbulence and Combustion (2013)
Geophysical Research Letters (2008, 2009, 2011, 2012, 2016)
International Journal for Numerical Methods in Fluids (2007, 2008, 2012, 2013, 2015)
International Journal of Computational Methods (2014)
Journal of Computational Physics (2006, 2010)
Journal of Fluid Mechanics (2003, 2006, 2007, 2008, 2010, 2011, 2012, 2013, 2014, 2015, 2016)
Journal of Geophysical Research: Earth Surface (2015)
Journal of Geophysical Research: Oceans (2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2016, 2017)
Journal of Hydraulic Engineering (2006, 2007, 2009, 2011, 2013, 2014, 2015)
Journal of Hydraulic Research (2013, 2016)
Journal of Hydrodynamics (2012)
Journal of Hydrology (2014)
Journal of Physical Oceanography (2006, 2010, 2011, 2013, 2014, 2015, 2016)
Limnology and Oceanography Letters (2017)
Limnology and Oceanography Methods (2008)
Marine Geology (2012)
Monthly Weather Review (2007)

Nonlinear Processes in Geophysics (2017)
Ocean Dynamics (2010)
Ocean Modelling (2007, 2008, 2010, 2011, 2012, 2013, 2016, 2017)
Oceanography (2017)
Physics of Fluids (2006, 2007, 2008, 2009, 2010, 2011, 2014, 2015)
San Francisco Estuary and Watershed Science (2014, 2016)
The Sea (2017)
Water Resources Research (2013)

Editorial boards

Ocean Modelling – Editor (2018-)

Journal of Water Waves – Associate Editor (2017-)

Environmental Fluid Mechanics (2017-)

University and Departmental Service

Stanford Interdisciplinary Graduate Fellowship selection committee (2017)

Environmental Fluid Mechanics and Hydrology Program Coordinator (2014-)

Chair, Promotion Committee (associate professor) (2014-2015)

CEE Vision Committee (2014-2016)

SOE Undergraduate Council (2013)

Woods Institute EVP Selection Committee (2013-2016)

CEE Undergraduate Curriculum Committee (2012-2015)

CEE ABET Representative for Environmental Engineering Degree (2011-2012)

CEE Faculty search committee (2012)

Premajor advisor (2012-2014)

Faculty Member, Institute for Computational and Mathematical Engineering (ICME) (2003-)

DOE Computational Science Graduate Fellowship (CSGF) coordinator for Stanford University (2004-)

Admissions chair, Environmental Fluid Mechanics and Hydrology Program, Dept. of Civil and Environmental Engineering (2009-2011, 2013, 2016, 2017)

Sophomore academic advisor (2005-2006)

Mechanical Engineering Flow Physics and Computation faculty search committee member (2005)

Freshman academic advisor (2004-2005)

Institute for Computational and Mathematical Engineering (ICME) curriculum committee member (2003-2004)

Center for African Studies (CAS) search committee member for South Africa Teaching Fellowship (2003)

Dissertation Reading Committee Member:

M. Barkdull, (Principal advisor: S. Monismith) Ph.D. 2016.
K. Cheng, (Principal advisor: L. Hildemann) Ph.D. 2010.
M. Chui, (Principal advisor: D. Freyberg) Ph.D. 2009.
K. Davis (Principal advisor: S. Monismith) Ph.D. 2008.
J. Dunckley (Principal advisor: J. Koseff) Ph.D. 2012.
S. Giddings, (Principal advisor: S. Monismith) Ph.D. 2010.
K. Gleichauf (Principal advisor: S. Monismith) Ph.D. 2015
R. Holmes (Principal advisor: L. Thomas) Ph.D. 2016.
J. Krall (Principal advisor: D. Freyberg) Ph.D. 2014
R. Moniz (Principal advisor: S. Monismith) Ph.D. 2014
N. Nidzieko (Principal advisor: S. Monismith) Ph.D. 2009.
T. Reddy, (Principal advisor: K. Arrigo) Ph.D. 2009.
J. Rogers, (Principal advisor: S. Monismith) Ph.D. 2015
L. Samuel, (Principal advisor: S. Monismith) Ph.D. 2014
M. Squibb, (Principal advisor: S. Monismith) Ph.D. 2014
E. Sta. Maria, (Principal advisor: M. Jacobson) Ph.D. 2013
J. Steinbuck (Principal advisor: S. Monismith) Ph.D. 2009.
L. Walter (Principal advisor: S. Monismith) Ph.D. 2011
R. Walter (Principle advisor: S. Monismith) Ph.D. 2014
J. Weitzman (Principal advisor: J. Koseff) Ph.D. 2013
D. Whitt (Principal advisor: L. Thomas) Ph.D. 2014
V. Sridharan (Principal advisor: S. Monismith) Ph.D. 2015
G. Zhao, (Principal advisor: R. Street) Ph.D. 2009.
R. Zeller, (Principal advisor: J. Koseff) Ph.D. 2014.
D. Zheng, (Principal advisor: L. Hildemann) Ph.D. 2016.

University Oral Examination (Chair)

J. Bae, Computational and Mathematical Engineering (2018)
S. Bose, Aeronautics and Astronautics (2012)
P. Constantine, Computational and Mathematical Engineering (2009)
S. Davis, Geological and Environmental Sciences (2008)
H. Hamilton, Aeronautics and Astronautics (2004)
C. Hamman, Mechanical Engineering (2015)
K. Hosseini, Aeronautics and Astronautics (2005)
S. Infeld, Aeronautics and Astronautics (2005)
M. Ji, Mechanical Engineering (2006)
S. Kang, Mechanical Engineering (2008)
S. Kumar, Aeronautics and Astronautics (2012)
M. Lande, Mechanical Engineering (2011)
G. Lotto, Geophysics (2018)
D. Macklin, Bioengineering (2017)
M. McDowell, Materials Science and Engineering (2013)
K. Moffett, Environmental Earth System Science (2010)
M. Mortazavi, Mechanical Engineering (2015)
L. Katrina ole-MoiYoi, E-IPER (2016)
B. Olson, Aeronautics and Astronautics (2013)
D. Phillips, Mechanical Engineering (2012)
B. Saenz, Environmental Earth System Science (2011)
N. Santhanam, Aeronautics and Astronautics (2004)
J. Seo, Mechanical Engineering (2016)
M. Shoeybi, Mechanical Engineering (2010)
V. Somandepalli, Mechanical Engineering (2006)
D. You, Mechanical Engineering (2003)
C. Yu, Aeronautics and Astronautics (2014)

University Oral Examination (Examiner)

L. Samuel, Civil and Environmental Engineering (2014)
I. Benekos, Civil and Environmental Engineering (2005)
N. Grumet, Geological and Environmental Sciences (2004)
P. Ray, Mechanical Engineering (2006)
A. Santoro, Civil and Environmental Engineering (2008)
R. Simons, Civil and Environmental Engineering (2004)
J. Thompson, Civil and Environmental Engineering (2015)

Awards and Honors

Outstanding Reviewer, Ocean Modelling, 2016

Lorenz G. Straub Award for best dissertation by former Ph.D. student Bing Wang, 2011.

Lorenz G. Straub Award for best dissertation by former Ph.D. student Subhas Karan Venayagamoorthy, 2009.

Presidential Early Career Award for Scientists and Engineers (PECASE), Office of Science and Technology Policy, 2009.

Young Investigator Award, Office of Naval Research, 2008.

Frederick A. Howes Scholar in Computational Science, Department of Energy, 2003.

South Africa Teaching Fellow, Department of African and African-American Studies, Stanford University, 2002-2003.

Bibliographical Information

Publications

Author order is based on percentage of work performed or contributed, except for the PI on the project or paper who is typically listed as last author. Ph.D. student names are in bold, supervised postdoctoral researcher names are in italics.

Refereed Journal Publications

1. **K. S. Nelson** and O. B. Fringer, 2018, "Sediment dynamics in wind-wave dominated shallow water environments", J. Geophys. Res.-Oceans, 123, 6996-7015, doi:10.1029/2018JC013894.
2. M. Traer, A. Fildani, O. Fringer, T. McHargue, and G. Hilley, 2018, "Turbidity current dynamics: Part 1. Model formulation and identification of flow equilibrium conditions resulting from flow stripping and overspill", J. Geophysical Research - Earth Surface, 123, 501–519, doi:10.1002/2017JF004200
3. M. Traer, A. Fildani, O. Fringer, T. McHargue, and G. Hilley, 2018, "Turbidity current dynamics: Part 2. Simulating flow evolution toward equilbrium in idealized channels", Journal of Geophysical Research – Earth Surface, 123, 520–534, doi: 10.1002/2017JF004202
4. B. Wang, L. Cao, F. Micheli, R. L. Naylor, and O. B. Fringer, 2018, "The effects of intensive aquaculture on nutrient residence time and transport in a coastal embayment", Environmental Fluid Mechanics, 18 (6), 1321–1349 doi:10.1007/s10652-018-9595-7
5. **K. R. Scheu**, D. A. Fong, S. G. Monismith, and O. B. Fringer, 2018, "Modeling sedimentation dynamics of sediment-laden river intrusions in a rotationally-influenced, stratified lake", Water Resources Research, 54, 4084–4107, doi:10.1029/2017WR021533

6. Y.-J. Chou, K. S. Nelson, R. C. Holleman, O. B. Fringer, M. T. Stacey, J. R. Lacy, S. G. Monismith, and J. R. Koseff, 2018, "Three-dimensional modeling of fine sediment transport by waves and currents in a shallow estuary", J. Geophys. Res.-Oceans., 123, doi:10.1029/2017JC013064
7. M. D. Rayson, G. N. Ivey, N. L. Jones, and O. B. Fringer, 2018, "Resolving high-frequency internal waves generated at an isolated coral atoll using an unstructured grid ocean model", Ocean Model., 122, 67-84, doi:10.1016/j.ocemod.2017.12.007
8. M. D. Rayson, E. S. Gross, R. D. Hetland, and O. B. Fringer, 2017, "Using an isohaline flux analysis to predict the salt content in an unsteady estuary", J. Phys. Oceanogr., 47, 2811-2828, doi:10.1175/JPO-D-16-0134.1
9. **E. T. Mayer** and O. B. Fringer, 2017, "An unambiguous definition of the Froude number for lee waves in the deep ocean", Journal of Fluid Mechanics, 831, doi:10.1017/jfm.2017.701
10. E. Masunaga, O. B. Fringer, Y. Kitade, H. Yamazaki, and S. Gallager, 2017, "Dynamics and energetics of trapped diurnal internal Kelvin waves around a mid-latitude island", Journal of Physical Oceanography, 47, 2479-2498, doi:10.1175/JPO-D-16-0167.1
11. **R. S. Arthur**, S. K. Venayagamoorthy, J. R. Koseff, and O. B. Fringer, 2017, "How we compute N matters to estimates of mixing in stratified flows", Journal of Fluid Mechanics, 831, doi:10.1017/jfm.2017.679
12. **K. S. Nelson** and O. B. Fringer, 2017, "Reducing spin-up time for simulations of turbulent channel flow", Physics of Fluids, 29, 105101, doi:10.1063/1.4993489
13. L. M. M. Herdman, J. L. Hench, O. Fringer, and S. G. Monismith, 2017, "Behavior of a wave-driven buoyant surface jet on a coral reef", Journal of Geophysical Research-Oceans, 122 (5), 4088-4109, doi:10.1002/2016JC011729
14. M. M. Flint, O. Fringer, S. L. Billington, D. Freyberg, and N. S. Diffenbaugh, 2017, "Historical analysis of hydraulic bridge collapses in the continental United States", Journal of Infrastructure Systems, 23 (3), 04017005, doi:10.1061/(ASCE)IS.1943-555X.0000354
15. E. Masunaga, **R. S. Arthur**, O. B. Fringer, and H. Yamazaki, 2017, "Sediment resuspension and the generation of intermediate nepheloid layers by shoaling internal bores", Journal of Marine Systems, 170, 31-41, doi:10.1016/j.jmarsys.2017.01.017
16. **R. S. Arthur**, J. R. Koseff, and O. B. Fringer, 2017, "Local vs. volume-integrated turbulence and mixing in breaking internal waves on slopes", Journal of Fluid Mechanics, 815, 169-198, doi:10.1017/jfm.2017.36
17. J. S. Rogers, S. G. Monismith, O. B. Fringer, D. A. Koweek, and R. B. Dunbar, 2017 ,"A coupled wave-hydrodynamic model of an atoll with high friction: Mechanisms for flow, connectivity, and ecological implications", Ocean Modelling, 110, 66–82, doi:10.1016/j.ocemod.2016.12.012
18. *M. D. Rayson*, E. S. Gross, R. D. Hetland, and O. B. Fringer, 2016, "Time scales in Galveston Bay: An unsteady estuary", Journal of Geophysical Research-Oceans, 121, 2268-2285, doi: 10.1002/2015JC011181
19. E. Masunaga, O. B. Fringer, H. Yamazaki, and K. Amakasu, 2016, "Strong turbulent mixing induced by internal bores interacting with internal tide-driven vertically sheared flow", Geophysical Research Letters, 43, 2094-2101, doi:10.1002/2016GL067812

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Refereed Conference/Symposia Proceedings

1. **B. Wang**, O. B. Fringer, and M. T. Stacey, 2012, “Interpreting the mixing efficiency from two-equation turbulence closure models”, Proceedings of the 3rd International Symposium on Shallow Flows, Iowa, USA.
2. **V. P. Chua**, and O. B. Fringer, 2012, "Impact of tidal dispersion and time scales on numerical diffusion in unstructured-grid estuarine modeling", Proceedings of the 3rd International Symposium on Shallow Flows, Iowa, USA.
3. *M. D. Rayson*, N. L. Jones, G. N. Ivey, and O. B. Fringer, 2011, “Internal hydraulic jump formation in a deep water, continuously-stratified, unsteady channel flow”, 7th International Symposium on Stratified Flows, Rome.
4. O. B. Fringer and **B. Wang**, 2010, "Analysis of stratified flow and separation over complex bathymetry in a field-scale estuarine model ", Proceedings of the 2010 DoD HPCMP Users Group Conference, IEEE Computer Society, 171-176, (invited), Schaumburg, IL, USA, doi:10.1109/HPCMP-UGC.2010.14
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6. R. Q. Wang, A. W. K. Law, E. E. Adams, and O. B. Fringer, 2009, “The determination of formation number for starting buoyant jet”, Proceedings of the 2nd International Symposium on Computational Mechanics (ISCM II) and 12th International Conference on Enhancement and Promotion of Computational Methods in Engineering and Science, AIP Conference Proceedings, v. 1233, 1636-1641. doi: 10.1063/1.3452156, Hong Kong.
7. R. Q. Wang, A. W. K. Law, E. E. Adams and O. B. Fringer, 2009, "Large-Eddy Simulation of Starting Buoyant Jets", Proceedings of the 33rd International Association of Hydraulic Engineering and Research (IAHR) Biennial Congress, Vancouver, Canada.
8. O. B. Fringer and **Z. Zhang**, 2008, "High-Resolution Simulations of Nonlinear Internal Gravity Waves in the South China Sea", Proceedings of the DoD HPCMP Users Group Conference, 2008, DOD HPCMP, 43-46. doi: 10.1109/DoD.HPCMP.UGC.2008.46, Seattle, WA, USA.
9. **Y.-J. Chou** and O. B. Fringer, 2007, "Modeling Sediment Suspension in High Reynolds Number Flow Using Large Eddy Simulation", Proceedings of the 5th International Symposium on Environmental Hydraulics, Tempe, AZ, USA.
10. *M. F. Barad* and O. B. Fringer, 2007, "Numerical simulations of shear instabilities in open-ocean internal gravity waves", Proceedings of the 5th International Symposium on Environmental Hydraulics, Tempe, AZ, USA.
11. *S. K. Venayagamoorthy*, O. B. Fringer, J. R. Koseff, and R. L. Naylor, 2007, "Simulations of mixing and transport of dissolved wasted discharged from an aquaculture pen", Proceedings of the 5th International Symposium on Environmental Hydraulics, Tempe, AZ, USA.

12. **B. Wang** and O. B. Fringer, 2007, "Modeling the dynamics of the Snohomish River Estuary with a finite volume, unstructured-grid parallel coastal ocean simulator", Proceedings of the 5th International Symposium on Environmental Hydraulics, Tempe, AZ, USA.
13. **Z. Zhang** and O. B. Fringer, 2006, "A Numerical Study of Nonlinear Internal Wave Generation in the Luzon Strait", Proceedings of the 6th International Symposium on Stratified Flows, pp 300-305, Perth, Australia.
14. *M. F. Barad*, O. B. Fringer, and P. Colella, 2006, "Multiscale simulations of internal gravity waves", Proceedings of the 6th International Symposium on Stratified Flows, pp 722-727, Perth, Australia.
15. **S. M. Jachec**, O. B. Fringer, M. Gerritsen, and R. L. Street, 2006, "The Three-Dimensional, Time-Dependent Nature of Internal Waves Entering Monterey Submarine Canyon", Proceedings of the 6th International Symposium on Stratified Flows, pp 294-299, Perth, Australia.
16. **S. K. Venayagamoorthy** and O. B. Fringer, 2006, "The dynamics of breaking internal gravity waves over a shelf break", Proceedings of the 6th International Symposium on Stratified Flows, pp 384-389, Perth, Australia.
17. O. B. Fringer, E. S. Gross, M. Meuleners, and G. N. Ivey, 2006. "Coupled ROMS-SUNTANS simulations of highly nonlinear internal gravity waves on the Australian northwest shelf", Proceedings of the 6th International Symposium on Stratified Flows, pp 533-538, Perth, Australia.
18. **S. M. Jachec**, O. B. Fringer, M. Gerritsen, and R. L. Street, 2006. "Effects of Grid Resolution on the Simulation of Internal Tides", Proceedings of the 16th International Offshore and Polar Engineering Conference, v. III, pp 432-438, San Francisco, CA, USA.
19. **D. Kang** and O. B. Fringer, 2006. "Efficient Computation of the Nonhydrostatic Pressure", Proceedings of the 16th International Offshore and Polar Engineering Conference, v. III, pp 414-419, San Francisco, CA, USA.
20. **S. K. Venayagamoorthy** and O. B. Fringer, 2006. "Internal wave energetics on a shelf break", Proceedings of the 16th International Offshore and Polar Engineering Conference, v. III, pp 473-480, San Francisco, CA, USA.
21. **Y. Chou** and O. B. Fringer, 2005, "An unstructured immersed boundary method for simulation of flow over complex topography", Proceedings of the 9th International Conference on Estuarine and Coastal Modeling, pp. 568-584. doi: 10.1061/40876(209)33, Charleston, SC, USA.
22. **D. Kang** and O. B. Fringer, 2005, "Time accuracy for pressure methods for nonhydrostatic free-surface flows", Proceedings of the 9th International Conference on Estuarine and Coastal Modeling, pp. 419-433. doi: 10.1061/40876(209)24, Charlston, SC, USA.
23. **S. K. Venayagamoorthy** and O. B. Fringer, 2004, "Energy partitioning in breaking internal waves on slopes", In: Environmental Hydraulics and Sustainable Water Management, Proceedings of the 4th International Symposium on Environmental Hydraulics and 14th Congress of Asia and Pacific Division, International Association of Hydraulic Engineering

and Research, 15-18 December 2004, Hong Kong, v. I, Edited by J.H.W. Lee, K.M. Lam, pp. 1051-1056.

24. O. B. Fringer, M. Gerritsen, and R. L. Street, 2004, "Internal waves in Monterey Bay: An application of SUNTANS", In: Environmental Hydraulics and Sustainable Water Management, Proceedings of the 4th International Symposium on Environmental Hydraulics and 14th Congress of Asia and Pacific Division, International Association of Hydraulic Engineering and Research, 15-18 December 2004, Hong Kong, v. I, Edited by J.H.W. Lee, K.M. Lam, pp. 67-75 (invited).
25. O. B. Fringer, S. W. Armfield, and R. L. Street, 2003, "A nonstaggered curvilinear grid pressure correction method applied to interfacial waves", Proceedings of the 2nd International Conference on Heat transfer, Fluid Mechanics, and Thermodynamics (HEFAT), Victoria Falls, Zambia.
26. O. B. Fringer, S. W. Armfield, and R. L. Street, 2000, "Direct numerical simulation of unstable finite amplitude progressive interfacial waves", Proceedings of the 5th International Symposium on Stratified Flows, pp. 749-754, Vancouver, Canada.
27. O. B. Fringer and R. L. Street, 2001, "The dynamics of breaking progressive interfacial waves", Proceedings of the 3rd International Symposium on Environmental Hydraulics, Tempe, AZ, USA.

Non-refereed Conference/Symposia Proceedings

1. **G. T. C. Gil** and O. B. Fringer, 2016, “Particle transport due to trapped cores”, 8th International Symposium on Stratified Flows, San Diego, CA.
2. **R. S. Arthur,** S. K. Venayagamoorthy, J. R. Koseff, and O. B. Fringer, 2016, **“**Quantification of highly unsteady and inhomogeneous stratified turbulence in breaking internal waves on slopes”, 8th International Symposium on Stratified Flows, San Diego, CA.
3. O. B. Fringer, 2009, “Towards nonhydrostatic ocean modeling with large-eddy simulation”, Oceanography in 2025: Proceedings of a Workshop, pp 81-83, The National Academies Press.

Edited Works in Print or in Press

1. A. Desbonnet, Ed., 2008, Ecosystem-based Estuary Management: A Case Study of Narragansett Bay, Chapter 14, "Circulation and pollutant transport dynamics in Narragansett Bay", by J. Craig Swanson & Malcolm L. Spaulding, Springer Series on Environmental Management, New York: Springer.

Presentations

Invited Plenary Talks and Distinguished Lectures

1. O. B. Fringer and **Y. Zhang**, 2016, “Subgrid hydrodynamics and sediment transport modeling on unstructured grids”, 15th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, September 27-29, Toulouse, France (keynote).
2. O. B. Fringer, 2016, “Numerical simulations to understand the dynamics, energetics, and mixing of breaking internal gravity waves”, B’Waves 2016, June 13-17, Bergen, Norway (keynote).
3. O. B. Fringer and **R. S. Arthur**, 2016, “Transport and mixing due to breaking internal gravity waves on slopes”, European Congress on Computational Methods in Applied Sciences and Engineering, June 5-10, Crete, Greece (keynote).
4. **B. Wang**, O. B. Fringer and M. Gerritsen, 2007, "Numerical techniques in a parallel, unstructured-grid, finite-volume coastal ocean simulation tool", Ninth U.S. National Congress on Computational Mechanics, San Francisco, CA (keynote).
5. O. B. Fringer, 2004, "Fluids, Math, Computers, and the Environment", Southern California Applied Mathematics Symposium (SOCAMS), Claremont, CA (keynote).

Other Invited Presentations

1. O. B. Fringer, **K.R. Scheu**, D. A. Fong, and S. G. Monismith, 2017, “Modeling intrusive, sediment-laden gravity currents in a rotationally-influenced lake”, IUTAM/AMERIMECH SYMPOSIUM on the Dynamics of gravity currents, September 25-27, Santa Barbara, CA.
2. O. B. Fringer and **Y. Zhang**, 2016, “Subgrid bathymetry for seamless 1d, 2d, and 3d hydrodynamics and sediment transport modeling in SUNTANS”, California Water and Environmental Modeling Forum, April 11-13, Folsom, CA.
3. **Y. Zhang**, O. Fringer, I. Huang, D. Fong, and S. Monismith, 2015, “Sediment transport modeling in a San Francisco Bay salt marsh”, California Water and Environmental Modeling Forum, March 11, Folsom, CA.
4. O. B. Fringer, 2015, “Three-dimensional coupled wind-wave and cohesive sediment transport modeling in South San Francisco Bay”, 2015 SIAM Conference on Computational Science and Engineering, March 13-18, Salt Lake City, UT.
5. **Y. Zhang**, O. B. Fringer, I. Huang, D. A. Fong, and S. G. Monismith, 2015, “The Impact of Vegetation and Culverts on Sediment Transport in a San Francisco Bay Salt Marsh”, SIAM Conference on Mathematical and Computational Issues in the Geosciences, June 29-July 2, Stanford, CA.
6. *M. Rayson*, E. Gross, and O. B. Fringer, 2015, “Challenges in three-dimensional hydrodynamic modelling of the shallow bays and estuaries along the Gulf of Mexico coast”, SIAM Conference on Mathematical and Computational Issues in the Geosciences, June 29-July 2, Stanford, CA.

7. O. B. Fringer and **R. S. Arthur**, 2015, “Direct numerical simulation of transport and mixing in breaking internal waves on slopes”, 13th U.S. National Congress on Computational Mechanics, July 27-30, San Diego, CA.
8. **Y. Zhang** and O. B. Fringer, 2015, “1D, 2D, and 3D Unstructured-grid modeling of sediment transport in a salt-marsh estuary”, 13th U.S. National Congress on Computational Mechanics, July 27-30, San Diego, CA.
9. O. B. Fringer, S. Vitousek, and **Y. Zhang**, 2015, “A model to simulate nonhydrostatic internal gravity waves in the ocean”, AGU Fall Meeting Abstract NG13B-07, December 14, San Francisco, CA.
10. O. B. Fringer, 2013, “Modeling internal wave-induced transport in the coastal ocean”, Workshop on Modeling in Support of Coastal Hypoxia, Acidification and Nutrient Management in the California Current, December 10-11, Costa Mesa, California.
11. O. B. Fringer, 2013, “Towards large-eddy simulation of internal waves in the coastal ocean”, Gordon Research Conference on Coastal Ocean Circulation, Biddeford, Maine.
12. O. B. Fringer and **P. J. Wolfram**, 2013, "Dealing with divergence errors and noise in C-grid finite-volume hydrodynamic models", Advances on Computational Mechanics: A Conference Celebrating the 70th Birthday of Thomas J. R. Hughes, San Diego.
13. O. B. Fringer, **S. Vitousek**, and **P. J. Wolfram**, 2012, “Finite-volume, nonhydrostatic ocean modeling on unstructured grids”, 1st International Conference on Frontiers in Computational Physics: Modeling the Earth System, Boulder.
14. R.C. Holleman, E.S. Gross, L.J. MacVean, M.T. Stacey, and O.B. Fringer, 2012, “Modelling Hydrodynamics, Sediment Transport and Provenance in the South San Francisco Bay Salt Ponds”, AGU Fall Meeting, San Francisco, CA, Abstract OS23D-04.
15. O. B. Fringer, 2011, "Grid resolution requirements and computational overhead in nonhydrostatic coastal ocean modeling", Minisymposium "Recent advances in coastal ocean modeling", SIAM Conference on Mathematical & Computational Issues in the Geosciences, Long Beach, CA.
16. O. B. Fringer and **B. Wang**, 2010, "High-resolution numerical simulation of surface salinity variability over an abrupt sill in a salt-wedge estuary", American Geophysical Union (AGU) Fall Meeting, San Francisco, CA.
17. O. B. Fringer, 2010, "Three-Dimensional Modeling of Sediment Dynamics in San Francisco Bay Using the SUNTANS Model", The 6th Biennial Bay-Delta Science Conference, Sacramento, CA..
18. O. B. Fringer and **B. Wang**, 2010, "Analysis of Stratified Flow and Separation Over Complex Bathymetry in a Field-Scale Estuarine Model", DOD HPCMP Users Group Conference, Shaumburg, IL
19. O. B. Fringer and **B. Wang**, 2010, "Challenges in high-resolution simulations of macrotidal estuaries", American Geophysical Union (AGU) Ocean Sciences Meeting, Eos Trans. AGU, 91(26), Ocean Sci. Meet. Suppl., Abstract IT25H-04, Portland, OR.

20. **D. Kang** and O. B. Fringer, 2010, "The energetics of barotropic and baroclinic tides in the Monterey Bay area", American Geophysical Union (AGU) Ocean Sciences Meeting, Eos Trans. AGU, 91(26), Ocean Sci. Meet. Suppl., Abstract PO31C-03, Portland, OR.
21. **Z. Zhang**, O. B. Fringer, and S. R. Ramp, 2010, "Determining the phase in the tide at which internal waves are generated over ridges", American Geophysical Union (AGU) Ocean Sciences Meeting, Eos Trans. AGU, 91(26), Ocean Sci. Meet. Suppl., Abstract PO43C-02, Portland, OR.
22. O. B. Fringer, 2009, "Multi-scale numerical simulation of internal waves in the ocean", 4th Warnemunde Turbulence Days Workshop, Warnemunde, Germany.
23. O. B. Fringer, *S. K. Venayagamoorthy*, and J. R. Koseff, 2009, "Characteristics of waste plumes from aquaculture pens in the marine environment", AAAS Annual Meeting, Chicago, IL.
24. O. B. Fringer, 2009, "High-resolution 3D hydrodynamics and sediment transport modeling of San Francisco Bay", Interagency Ecological Program (IEP) "Physical Modeling and Fish Management" workshop, Sacramento, CA.
25. **B. Wang** and O. B. Fringer, 2008, "High-resolution simulations of a salinity front interacting with complex geometry and intertidal mudflats", American Geophysical Union (AGU) Ocean Sciences Meeting, Orlando, FL.
26. O. B. Fringer, 2007, "Multiscale simulations of internal waves and other coastal processes", Gordon Research Conference on Coastal Ocean Modeling, New London, NH.
27. **Z. Zhang** and O. B. Fringer, 2007, "Nonhydrostatic effects of nonlinear internal wave propagation in the South China Sea", American Geophysical Union (AGU) Joint Assembly Meeting, Eos Trans. AGU, 88 (23), Jt. Assem. Suppl., Abstract OS41A-06, Acapulco, Mexico.
28. O. B. Fringer, 2006, "Parallel performance of a nonhydrostatic, unstructured-grid coastal ocean model", National Science Foundation Petascale Computing and the Geosciences Workshop, La Jolla, CA.

Contributed Conference Presentations

1. **K. Nelson** and O. B. Fringer, 2018, “Unexpected fluid and sediment transport dynamics in shallow-water wave and current driven environments”, AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
2. **Y. Zhang**, S. Vitousek, and O. B. Fringer, 2018, “An adaptive vertical coordinate for unstructured-grid, nonhydrostatic ocean modeling”, AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
3. O. B. Fringer and **R. S. Arthur**, 2018, “Transport and dispersion due to breaking internal gravity waves on slopes”, AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
4. **E. Mayer** and O. B. Fringer, 2018, “The lee-wave Froude number and its intuition”, AGU Ocean Sciences Meeting, February 11-16, Portland, OR.

5. **J. Adelson**, R. Holleman, and O. B. Fringer, 2018, "Observations of Suspended Sediment Dynamics in San Francisco Bay using Landsat 7 Imagery", AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
6. *J. Rogers*, D. Ko, and O. B. Fringer, 2018, "A framework for seamless one-way nesting of internal wave-resolving ocean models", AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
7. **K. Scheu**, O. B. Fringer, D. Fong, and S. G. Monismith, 2018, "The role of lateral boundaries in sediment transport due to river plumes in rotational, stratified environments", AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
8. **S. White, J. Adelson**, D. Freyberg, and O. B. Fringer, 2018, "Estimating Sediment Budget in South San Francisco Bay from Limited Streamflow Data", AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
9. E. Masunaga, O. B. Fringer, H. Yamazaki, R. S. Arthur, and K. Wada, 2018, "Numerical simulations and observations of nonlinear internal tides in shallow coastal regions", AGU Ocean Sciences Meeting, February 11-16, Portland, OR.
10. O. B. Fringer, **R. S. Arthur**, S. K. Venayagamoorthy, and J. R. Koseff, 2017, "The effect of different methods to compute N on estimates of mixing in stratified flows", 70th Annual Meeting of the APS Division of Fluid Dynamics, November 19-21, Denver, CO.
11. **Y. Zhang**, S. Vitousek, and O.B. Fringer, 2017, "A hybrid vertical coordinate for unstructured-grid, nonhydrostatic ocean modeling", The 16th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, August 29-September 1, Stanford, CA.
12. B. Wang, L. Cao, O.B. Fringer, F. Micheli, and R. Naylor, 2017, "Model study of the effects of intensive aquaculture on residence time and nutrient transport in a coastal embayment", The 16th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, August 29-September 1, Stanford, CA.
13. **W. Chen**, S. L. Billington, and O. B. Fringer, 2017, "An unstructured-grid, cut-cell model for scour simulation", The 16th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, August 29-September 1, Stanford, CA.
14. E. Masunaga, O.B. Fringer, H. Yamazaki, 2017, "Nonlinear internal wave dynamics and sediment transport processes investigated with the SUNTANS model", The 16th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, August 29-September 1, Stanford, CA.
15. **J. H. Adelson** and O. B. Fringer, 2017, "Remote Sensing of Sediment Dynamics and Critical Shear Stress in San Francisco Bay", Gordon Research Conference on Coastal Ocean Dynamics, June 11-16, Biddeford, ME.
16. **E. T. Mayer** and O. B. Fringer, 2017, "The dynamics of unmapped bathymetry: Lee waves", Gordon Research Conference on Coastal Ocean Dynamics, June 11-16, Biddeford, ME.

17. E. Masunaga, G. Auger, M. Rayson, O. Fringer, Y. Uchiyama, and H. Yamazaki, 2017, "Numerical simulations of the interaction between internal waves and the Kuroshio Current over the Izu-Ogasawara Ridge", AOGS 14th Annual Meeting, August 6-11, Singapore.
18. **K. Nelson** and O. B. Fringer, 2016, "Understanding the effects of sediment stratification in shallow wave and current driven environments", AGU Fall Meeting, December 12-16, San Francisco, CA.
19. **J. Adelson**, N. Kau, and O. B. Fringer, 2016, "Remote sensing to infer surface SPM in San Francisco Bay", 9th Biennial Bay-Delta Science Conference, November 15-17, Sacramento, CA.
20. **J. Adelson**, R. James, V. Chirayath, and O.B. Fringer, 2016, "Calibration and Testing of an Active Multispectral Instrument for Remote Sensing Suspended Particulate Matter", Ocean Optics Conference, November 8-12, Victoria, BC, Canada.
21. **K. Nelson** and O. B. Fringer, 2016, "Reducing spin-up time for DNS and LES of turbulent channel flow", 69th Annual Meeting of the APS Division of Fluid Dynamics, 61 (20), Abstract KP1.00134, November 20-22, Portland, OR.
22. E. Masunaga, O. B. Fringer, and H. Yamazaki, 2016, "Generation mechanisms and energetics of internal waves around an island", PO33B-05, AGU Ocean Sciences Meeting Abstract MG14A-1901, February 21-26, Portland, Oregon
23. **R. S. Arthur**, J. R. Koseff, and O. B. Fringer, 2016, "Local vs. bulk measures of the mixing efficiency in breaking internal waves on slopes", AGU Ocean Sciences Meeting Abstract PO24E-2998, February 21-26, Portland, Oregon.
24. **K. R. Scheu**, D. A. Fong, S. G. Monismith, and O. B. Fringer, 2016, "Sedimentation dynamics of a sediment-laden river intrusions in a large alpine lake", AGU Ocean Sciences Meeting Abstract MG14A-1901, February 21-26, Portland, Oregon.
25. S. Y. Litvin, J. M. Beers, C. B. Woodson, P. Leary, O. B. Fringer, J. A. Goldbogen, F. Micheli, S. G. Monismith, G. N. Somero, 2016, "Quantifying physiological, behavioral and ecological consequences of hypoxic events in kelp forest", AGU Ocean Sciences Meeting Abstract ME24E-0759, February 21-26, Portland, Oregon.
26. *M. D. Rayson*, E. S. Gross, and O. B. Fringer, 2015, "Physical processes controlling tracer exchange at the mouth of Galveston Bay", Gulf of Mexico Oil Spill and Ecosystem Science Conference, February 16-19, Houston, TX.
27. **R. S. Arthur** and O. B. Fringer, 2015, "Transport by breaking internal waves on slopes", Gordon Research Conference on Coastal Ocean Modeling, June 7-12, Biddeford, ME.
28. **K. Nelson**, O. B. Fringer, and Y.J. Chou, 2015, "A three-dimensional sediment transport model and its application for studying shoal and channel sediment dynamics", Gordon Research Conference on Coastal Ocean Modeling, June 7-12, Biddeford, ME.
29. *M. Rayson*, E. Gross, R. Hetland, and O. Fringer, 2015, "Characterizing and modelling salinity variability in an estuary with transient river forcing", Gordon Research Conference on Coastal Ocean Modeling, June 7-12, Biddeford, ME.

30. **K. Scheu**, D. Fong, S. Monismith, and O. Fringer, 2015, "Modeling sedimentation dynamics of a sediment-laden river plume in a large alpine lake", Gordon Research Conference on Coastal Ocean Modeling, June 7-12, Biddeford, ME.
31. **Y. Zhang** and O. B. Fringer, 2015, "New developments and applications of the parallel finite-volume unstructured-grid SUNTANS model for sediment transport within estuarine marshes", Gordon Research Conference on Coastal Ocean Modeling, June 7-12, Biddeford, ME.
32. O. B. Fringer, *M. D. Rayson*, and P. J. Wolfram, 2015, "Are unstructured grids needed? Comparison of the accuracy of finite-volume unstructured to curvilinear and Cartesian grid ocean models", 14th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, September 28-30, Portland, OR.
33. O. B. Fringer and **Y. Zhang**, 2015, "Subgrid bathymetry for hydrodynamics and sediment transport in SUNTANS", 14th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, September 28-30, Portland, OR.
34. L. Zaninetta and O. B. Fringer, 2014, "New Ways to Assess Natural Recovery of Sediments in Lake Maggiore, Italy", 30th Annual International Conference on Soils, Sediments, Water, and Energy, October 20-23, Amherst, MA.
35. **R.S. Arthur** and O. B. Fringer, 2014, "Turbulent dynamics of breaking internal gravity waves on slopes", 67th Annual Meeting of the APS Division of Fluid Dynamics, 59 (20), Abstract BAPS.2014.DFD.R11.1, November 23-25, San Francisco, CA.
36. O. B. Fringer, **P. Wolfram**, N. Monsen, K. Gleichauf, D. Fong, and S. G. Monismith, 2014, "Comparison of mixing at a junction computed with two- and three-dimensional models to the simple flow-weighting scheme used in one-dimensional models", 8th Biennial Bay-Delta Science Conference, October 28-30, Sacramento, CA.
37. M. van der Wegen, L. Lucas, N. Knowles, D. Senn, B. Jaffe, E. Elias, P. Barnard, M. Stacey, O. Fringer, E. Gross, T. Fregoso, R.C. Martyr, F. Achete, E. Melger, F. Baart, H. Los, T. Troost, J. Smits, D. Roelvink, 2014, "Building a Public Community around the D3D-FM San Francisco Bay-Delta Model", 8th Biennial Bay-Delta Science Conference, October 28-30, Sacramento, CA.
38. *M. D. Rayson*, E. S. Gross, R. D. Hetland, O. B. Fringer, 2014, "Tracer age as a diagnostic for understanding the relationship between surface and boundary forcing and estuarine circulation", Poster 3-63, Gulf of Mexico Oil Spill & Ecosystem Science Conference, January 26-28, Mobile, AL.
39. **R. S. Arthur** and O. B. Fringer, 2014, "Transport and mixing by breaking internal waves on slopes", 61st Annual Eastern Pacific Ocean Conference (EPOC). September 17-20, Mt. Hood, OR.
40. **R. S. Arthur** and O. B. Fringer, 2014, "Cross-stream variability in breaking internal waves on slopes", Nonlinear effects in internal waves conference, June 9-12, Cornell, NY.

41. K. G. Gleichauf, **P. Wolfram**, N. Monsen, O. Fringer, and S. Monismith, 2014, "Dispersion mechanisms in a tidal river junction in the Sacremento-San Joaquin Delta, CA", AGU Ocean Sciences Meeting Abstract 14592, February 23-28, Honolulu, HI.
42. **R. S. Arthur** and O. B. Fringer, 2014, "The three-dimensional structure and energetics of breaking internal waves on slopes", AGU Ocean Sciences Meeting Abstract 13316, February 23-28, Honolulu, HI.
43. N. L. Jones, C. E. Bluteau, *M. D. Rayson*, O. B. Fringer, and G. N. Ivey, 2014, "Internal tide mixing on the Australian Northwest continental shelf and slope", AGU Ocean Sciences Meeting Abstract 15994, February 23-28, Honolulu, HI.
44. O. B. Fringer, *B. Wang*, N L. Jones, and G. N. Ivey, 2014, "Numerical modeling of nonlinear and nonhydrostatic internal waves on the Australian North West shelf", AGU Ocean Sciences Meeting Abstract 16541, February 23-28, Honolulu, HI.
45. *M. D. Rayson*, O. B. Fringer, E. S. Gross, and R. D. Hetland, 2014, "Application of a nested, unstructured mesh hydrodynamic model to a bay in the Gulf of Mexico", AGU Ocean Sciences Meeting Abstract 16922, February 23-28, Honolulu, HI.
46. **K. Scheu**, D. Fong, S. Monismith, and O. Fringer, 2014, "Seasonal variability of sediment deposition into a large alpine lake", AGU Ocean Sciences Meeting Abstract 17645, February 23-28, Honolulu, HI.
47. N. Tahvildari, T. Peacock, and O. B. Fringer, 2014, "A parametric study of nonlinear and nonhydrostatic effects on internal tide generation over a submerged ridge", AGU Ocean Sciences Meeting Abstract 16837, February 23-28, Honolulu, HI.
48. **S. Vitousek** and O. B. Fringer, 2014, "A nonhydrostatic isopycnal-coordinate ocean model", AGU Ocean Sciences Meeting Abstract 15863, February 23-28, Honolulu, HI.
49. **R. S. Arthur** and O. B. Fringer, 2013, "Dissipation and mixing in breaking internal gravity waves on slopes", Gordon Research Conference on Coastal Ocean Circulation, Biddeford, Maine.
50. N. E. Monsen, **P. Wolfram**, K. Gleichauf, O. Fringer, and S. G. Monismith, 2013, "Development of a SUNTANS model for the Sacramento-San Joaquin Delta", 2013 California Water and Environmental Modeling Forum (CWEMF) Annual Meeting, April 22-24, Folsom, California.
51. *M. Rayson*, E. Gross, and O. B. Fringer, 2013, "Residual circulation in a shallow, micro-tidal estuary: Galveston Bay, TX", Gordon Research Conference on Coastal Ocean Circulation, June 9-14, Biddeford, Maine.
52. *M. Rayson*, E. S. Gross, and O. B. Fringer, 2013, "Age as a diagnostic for understanding the link between surface and boundary forcing and estuarine circulation", The 12th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, September 16-19, Austin, Texas.
53. *M. Rayson*, E. S. Gross, and O. B. Fringer, 2013, "Development of a high-resolution, three-dimensional hydrodynamic model of Galveston Bay", Gulf of Mexico Oil Spill and Ecosystem Science Conference, January 21-23, New Orleans, Louisiana.

54. **K. R. Scheu,** O. B. Fringer, S. G. Monismith, and D. A. Fong, 2013, "Sediment transport due to river inflows into large alpine lakes", KITP Conference: Particle-Laden Flows in Nature, December 16-19, U. C. Santa Barbara.
55. **K. R. Scheu,** S. G. Monismith, D. A. Fong, and O. B. Fringer, 2013, "Seasonal variability of sediment and contaminant transport in Lake Maggiore, Italy", 16th Workshop on Physical Processes in Natural Waters, July 8-11, Queensland, Australia (awarded best presentation).
56. **S. Vitousek** and O. B. Fringer, 2013, "A Nonhydrostatic Isopycnal Model for the Simulation of Internal Gravity Waves", Gordon Research Conference on Coastal Ocean Circulation, June 9-14, Biddeford, Maine.
57. **P. Wolfram,** N. Monsen, K. Gleichauf, O. Fringer, and S. Monismith, 2013, **"**Tidal dispersion and the impact of small-scale flow features in channel junctions", Gordon Research Conference on Coastal Ocean Circulation, Biddeford, Maine.
58. **P. Wolfram,** N. E. Monsen, K. Gleichauf, O. Fringer, and S. G. Monismith, 2013, "Computing Secondary Flows in the Delta: The Problem of Noise on Unstructured C-grids", California Water and Environmental Modeling Forum (CWEMF) Annual Meeting, April 22-24, Folsom, California.
59. **Y. Zhang** and O. B. Fringer, 2013, "New developments for the parallel finite-volume unstructured-grid SUNTANS model", The 12th International workshop on Multi-scale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, September 16-19, Austin.
60. *Y. Chou*, R. C. Holleman, S. Lee, C. Chang, O. B. Fringer, M. T. Stacey, S. G. Monismith, and J. R. Koseff, 2012, "Three-Dimensional Numerical Modeling of Sediment Suspension in San Francisco Bay", AGU Fall Meeting, San Francisco, CA, Abstract OS21C-1767.
61. **R. S. Arthur**, R.K. Walter, C.B. Woodson, O.B. Fringer, and S.G. Monismith,2012, "Field and Numerical Investigation of Nearshore Internal Bores", 59th Annual Eastern Pacific Ocean Conference (EPOC), September 22, 2012. Mt. Hood, OR.
62. **P. Wolfram** and O. Fringer, 2012, "Mitigating divergence-error oscillations in triangular C grids for nonlinear and nonhydrostatic flows", Workshop on Multiscale Modelling of Coastal, Shelf and Ocean Dynamics (IMUM), Delft, Netherlands.
63. **P. Wolfram**, N. Monsen, K. Gleichauf, O. Fringer, and S. Monismith, 2012, "Characterizing the effect of small scale flow features on dispersion within channel junctions", 7th Biennial Bay-Delta Science Conference, Sacramento, CA.
64. N. E. Monsen, **P. Wolfram**, K. Gleichauf, O. Fringer, and S. Monismith, 2012, "The Devil is in the details: why the representation of the flow field, especially at junctions, matters in order to simulate dispersion in the Delta", 7th Biennial Bay-Delta Science Conference, Sacramento, CA.
65. **S. Koltakov,** G. Iaccarrino, and O. B. Fringer, 2012, "Inferring bottom bathymetry from free-surface flow features", SIAM Conference on Uncertainty Quantification, Raleigh, North Carolina.

66. **V. P. Chua**, O. B. Fringer, D. A. Fong, S. G. Monismith, and J. R. Koseff, 2012, "Modeling the impact of sea-level rise on salinity intrusion in San Francisco Bay", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 9460.
67. R. B. Zeller, J. S. Weitzman, M. E. Abbett, O. B. Fringer, and J. R. Koseff, 2012, "Seagrass blade dynamics in unidirectional, oscillatory, and combined flows", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 9801.
68. **G. Trigo Cabrita Gil** and O. B. Fringer, 2012, "Lagrangian- and Eulerian-mean effects in progressive internal gravity waves", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 10029.
69. **K. R. Scheu**, O. B. Fringer, S. G. Monismith, D. Lin, and R. G. Luthy, 2012, "Rotational effects on sediment and DDT transport within a large lake (Lake Maggiore, Italy)", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 10877.
70. K. T. Gleichauf, **P. J. Wolfram,** S. G. Monismith, O. B. Fringer, N. E. Monsen, and A. M. Bayen, 2012, "Small-scale hydrodynamics in tidal river junctions in the Sacramento-San Joaquin River Delta", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 12139.
71. **S. Vitousek** and O. B. Fringer, 2012, "Grid resolution requirements in modeling internal waves", 2012 Ocean Sciences Meeting, Salt Lake City, Abstract 12494.
72. **B. Wang, P. J. Wolfram,** G. Zhao, and O. B. Fringer, 2011, "Reconstruction of vector fields for semi-Lagrangian advection on unstructured, staggered grids", The 10th International Workshop on Multiscale (Un-)structured Mesh Numerical Modelling for coastal, shelf and global ocean dynamics, Bremerhaven, Germany, August 22-25.
73. R. Holleman, **V. Chua**, M. Stacey, and O. B. Fringer, 2011, "Numerical scalar diffusion on flow-aligned unstructured, finite-volume grids", The 10th International Workshop on Multiscale (Un-)structured Mesh Numerical Modelling for coastal, shelf and global ocean dynamics, Bremerhaven, Germany, August 22-25.
74. O. B. Fringer and **S. Vitousek**, 2011, "Physical vs numerical dispersion in nonhydrostatic internal wave modeling", Conference on Geophysical and Astrophysical Internal Waves, Les Houches, France.
75. S. Sankaranarayanan and O. B. Fringer, 2010, "Dynamics of Low-frequency fluctuations in San Francisco Bay due to upwelling", AGU Fall Meeting, San Francisco, CA, USA.
76. *Y. Chou* and O. B. Fringer, 2010, "Coupled Wave-Current Numerical Simulation of Cohesive Sediment Transport in San Francisco Bay using SUNTANS", AGU Fall Meeting, San Francisco, CA, USA.
77. **Gil, G. T. C.**, and O. B. Fringer, 2010, "On the potential for transport via internal tides in the coastal ocean", 63rd Annual Meeting of the APS Division of Fluid Dynamics, 55 (16), Long Beach, CA, USA.
78. O. B. Fringer and **Z. Zhang**, 2010, "Understanding internal wave generation in the South China Sea using three-dimensional numerical simulations", BIRS Coordinated Mathematical Modeling of Internal Waves, Banff, Canada.

79. **B. Wang** and O. B. Fringer, 2010, "High-resolution simulations of stratified flow and separation over an abrupt sill in a shallow estuary", 9th International workshop on Multiscale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, MIT, Cambridge, MA, USA.
80. **V. Chua** and O. B. Fringer, 2010, "Assessing the effects of numerical diffusion in a three-dimensional unstructured-grid model of a periodically-stratified estuary", 9th International workshop on Multiscale (Un)-structured mesh numerical Modeling for coastal, shelf, and global ocean dynamics, MIT, Cambridge, MA, USA.
81. **Y. Chou** and O. B. Fringer, 2010, "Numerical simulation of combined wave-current flows over dynamic sand ripples", Eos Trans. AGU, 91(26), Ocean Sci. Meet. Suppl., Abstract PO15E-30, Portland, OR, USA.
82. **B. Wang**, O. B. Fringer, and E. S. Gross, 2010, "Understanding turbulent mixing in a partially stratified estuary using SUNTANS", Eos Trans. AGU, 91(26), Ocean Sci. Meet. Suppl., Abstract PO45I-10, Portland, OR, USA.
83. O. B. Fringer and *M. F. Barad*, 2009, "Numerical simulation of shear instabilities in interfacial gravity waves", 4th Warnemunde Turbulence Days Workshop, Warnemunde, Germany.
84. *M. Barad* and O. B. Fringer, 2009, "Large-eddy simulation of coherent flow structures in a river", DOD HPCMP Users Group Conference, San Diego, CA, USA.
85. **Y. Chou** and O. B. Fringer, 2009, "Numerical simulation of turbulence-induced sediment transport and the resulting bedform dynamics", Workshop on modeling of turbidity currents and related gravity currents, Santa Barbara, CA, USA.
86. **Y. Chou** and O. B. Fringer, 2009, "Numerical Simulation of Turbulence-Induced Bedform Initiation", 62nd Annual Meeting of the APS Division of Fluid Dynamics, 54 (19), Abstract BAPS.2009.DFD.AC.6, http://meetings.aps.org/link/BAPS.2009.DFD.AC.6, Chicago, IL, USA.
87. O. Fringer and *M. Barad*, 2009, "Numerical simulation of shear instabilities in interfacial gravity waves", 62nd Annual Meeting of the APS Division of Fluid Dynamics, 54 (19), Abstract BAPS.2009.DFD.HS.4, http://meetings.aps.org/link/BAPS.2009.DFD.HS.4, Chicago, IL, USA.
88. **Chou, Y.**, Fringer, O. B., and J. R. Lacy, 2008, "Numerical Study of Sediment Suspension over Bedforms in Combined Flows", Eos Trans. AGU, 89(53), Fall Meet. Suppl., Abstract OS21E-1204, San Francisco, CA, USA.
89. *S. K. Venayagamoorthy*, O. B. Fringer, R. Naylor, and J. R. Koseff, 2008, " Numerical simulations of aquaculture dissolved waste transport in a coastal embayment", 61st Annual Meeting of the APS Division of Fluid Dynamics, 53 (15), Abstract BAPS.2008.DFD.HV.9, http://meetings.aps.org/link/BAPS.2008.DFD.HV.9, San Antonio, TX, USA.
90. O. B. Fringer, 2008, "Nonhydrostatic effects of internal solitary-like waves and possible parameterizations for hydrostatic models", Eos Trans. AGU, 89(53), Fall Meet. Suppl., Abstract OS51F-08, San Francisco, CA, USA.

91. **Wang, B.**, and O. B. Fringer, 2008, "Nonhydrostatic effects in high-resolution estuarine simulations", Seventh International Workshop on Unstructured Grid Numerical Modelling of Coastal, Shelf and Ocean Flows, Halifax, Canada.
92. *M. F. Barad*, O. B. Fringer, and P. MacCready, 2008, "Surface Signatures Generated by one- and two-dimensional sinusoidal bathymetry", AGU Ocean Sciences Mtng., Orland, FL, USA.
93. **D. Kang** and O. B. Fringer, 2008, "Simulation of the interaction of mesoscale currents with internal tides", AGU Ocean Sciences Mtng., Orlando, FL, USA.
94. *S. K. Venayagamoorthy*, O. B. Fringer, J. R. Koseff, and R. L. Naylor, 2008, "Simulations of mixing and transport of dissolved wasted discharged from near-coastal aquaculture pens", AGU Ocean Sciences Mtng., Orlando, FL, USA.
95. **Z. Zhang** and O. B. Fringer, 2008, "Numerical simulation of the generation of nonlinear internal gravity waves in the South China Sea"", AGU Ocean Sciences Mtng., Orlando, FL, USA.
96. *S. K. Venayagamoorthy*, O. B. Fringer, J. R. Koseff, and R. L. Naylor, 2007, "Simulations of Mixing and Transport of Dissolved Waste Discharged From a Submerged Aquaculture pen", Eos Trans. AGU, 88 (23), Jt. Assem. Suppl., Abstract OS23G-02, Acapulco, Mexico.
97. O. B. Fringer, S. J. Macumber, and A. B. Boehm, 2007, "Transport due to Internal Waves in the San Pedro Bay Region", Eos Trans. AGU, 88 (23), Jt. Assem. Suppl., Abstract OS23G-04, Acapulco, Mexico.
98. **S. M. Jachec**, O. B. Fringer, M. Gerritsen, and R. L. Street, 2006, "Understanding Internal Tides within Monterey Bay: from Beams to Modes", Eos Trans. AGU, 87(52), Fall Meet. Suppl., Abstract OS13A-1542, San Francisco, CA.
99. **Z. Zhang** and O. B. Fringer, 2006, "Simulation of Nonlinear Internal Wave Generation in the Northeastern South China Sea", Eos Trans. AGU, 87(52), Fall Meet. Suppl., Abstract OS13A-1533, San Francisco, CA.
100. **B. Wang**, O. B. Fringer, and R. L. Street, 2006, "Application of an unstructured-grid, finite-volume parallel coastal ocean simulator to the Snohomish River Estuary", Proceedings of the fifth international workshop on unstructured mesh numerical modelling of coastal, shelf and ocean flows, Miami, FL, USA.
101. **S. K. Venayagamoorthy** and O. B. Fringer, 2006. " Dynamics of internal boluses across a shelf break", 59th Annual Meeting of the Division of Fluid Dynamics, American Physical Society, Abstract BAPS.2006.DFD.FG.3, http://meetings.aps.org/link/BAPS.2006.DFD.FG.3, Tampa Bay, FL, USA.
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Former Ph.D. Students (in order of graduation year)

1. Kurt Nelson, 2018, Thesis: "Simulating sediment dynamics in shallow-water wave- and current-driven environments"
2. Yun Zhang, 2017, Thesis: "Numerical modeling for hydrodynamics and suspended sediment transport in estuarine marshes"
3. Gonçalo Gil, 2017, Thesis: "Mass transport and shear-flow dispersion due to nonlinear internal gravity waves"
4. Kara Scheu, 2016, Thesis: "Sediment transport due to river plumes in stratified, rotationally-influenced lakes"
5. Robert Arthur, 2015, Thesis: "Numerical investigation of breaking internal waves on slopes: Dynamics, energetics, and transport"

6. Sean Vitousek, 2014, Thesis: "Towards internal wave resolving simulations of the ocean"
7. Phillip Wolfram, 2014, Thesis: "Secondary flows and dispersion in channel junctions"
8. Sergey Koltakov, 2013, Thesis: "Bathymetry inference from free-surface flow features using large-eddy simulation"
9. Bing Wang, 2012, Thesis: "Multiscale numerical simulations of a complex macrotidal tidal-river estuary"
10. Vivien Chua, 2012, Thesis: "Three-dimensional, unstructured-grid numerical simulations of hydrodynamics and scalar transport in San Francisco Bay"
11. Dujuan Kang, 2011, Thesis: "Energetics and dynamics of internal tides in Monterey Bay using numerical simulations"
12. Zhonghua Zhang, 2010, Thesis: "Numerical simulations of nonlinear internal waves in the South China Sea"
13. Yi-Ju Chou, 2009, Thesis: "Numerical study of sand ripple dynamics in turbulent flows"
14. Sheng Chen, 2009, Thesis: "Adaptive error estimators for electromagnetic field solvers"
15. Steven Jachec, 2007, Thesis: "Understanding the evolution and energetics of internal tides within Monterey Bay via numerical simulations"
16. Karan Venayagamoorthy, 2006, Thesis: "Energetics and dynamics of internal waves on a shelf break using numerical simulations"

APPENDIX A-3

The **tidal period (PERIOD)** must be supplied; in most cases it is 12.4 hours, but in some locations it may vary slightly. The **maximum tidal velocity (UAmax)** for the location must be specified; this can usually be taken as the average of the absolute values of the two actual maxima, independent of their direction. A CORMIX design case consists then of an instantaneous ambient condition, before, at or after one of the two slack tides. Hence, the analyst must specify the **time** (in hours) **before, at, or after slack** that defines the design condition, followed by the actual **tidal ambient velocity (UA)** at that time. The ambient depth conditions are then those corresponding to that time.

In general, tidal simulations should be repeated for several time intervals (usually hourly or two-hourly intervals will suffice) before and after slack time to determine plume characteristics in unsteady ambient conditions.

Strongly unsteady conditions can also occur in other environments, such as in wind-induced current reversals in shallow lakes or coastal areas. In this case, any typical reversal period can be analyzed following an approach similar to the above.

4.3.4 Ambient Density Specification

Information about the density distribution in the ambient water body is very important for the correct prediction of effluent discharge plume behavior. CORMIX first inquires whether the ambient water is **fresh water** or **non-fresh** (i.e. brackish or saline). If the ambient water is fresh and above 4 ^{o}C, the system provides the option of entering ambient temperature data so that the ambient density values can be internally computed from an equation of state. This is the recommended option for specifying the density of fresh water, even though ambient temperature per se is not needed for the analysis of mixing conditions. In the case of salt water conditions, Figure 4.3 is included as a practical guide for specifying the density if "salinity values" in parts-per-thousand (ppt) are available for the water body. Typical open ocean salinities are in the range 33 - 35 ppt.

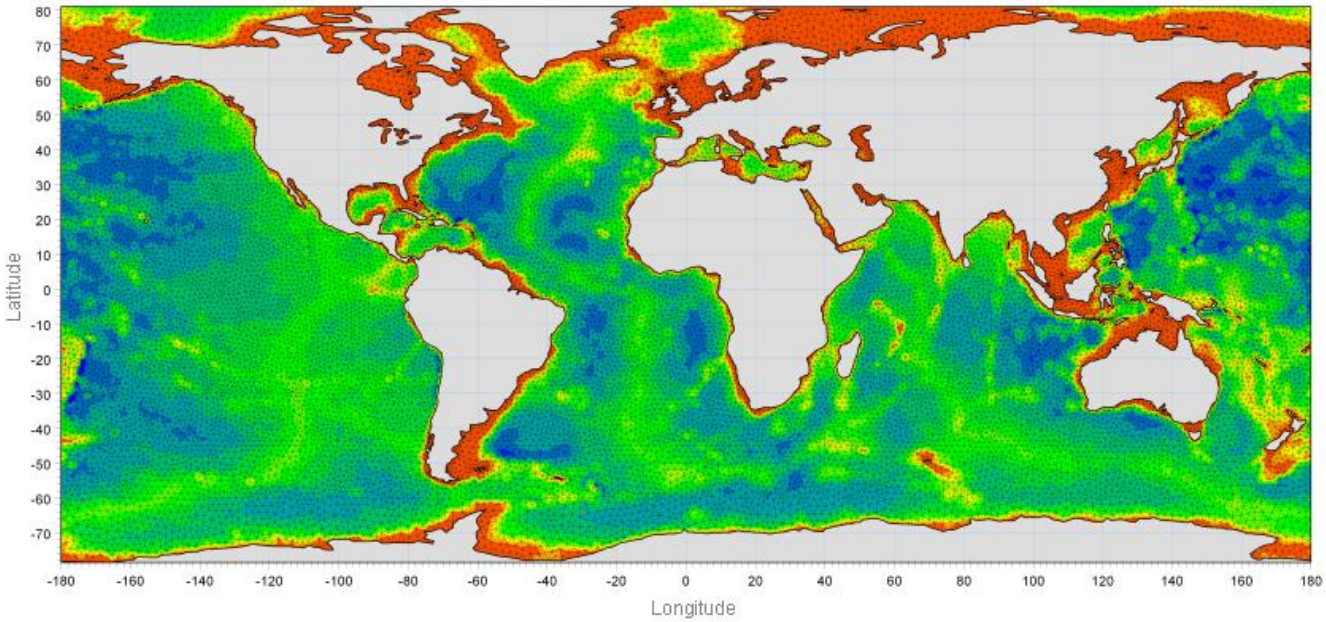
The user then specifies whether the ambient density (or temperature) can be considered as **uniform** or as **non-uniform** within the water body, and in particular within the expected plume regions. As a practical guide, vertical variation in density of less than 0.1 kg/m^3 or in temperature of less than 1 ^{o}C can be neglected. For uniform conditions, the **average ambient density** or **average temperature** must be specified.

When conditions are non-uniform, CORMIX requires that the actual measured vertical density distribution be approximated by one of three schematic stratification profile types illustrated in Figure 4.4. These are: Type A, linear density profile; Type B, two-layer system with constant densities and density jump; Type C, constant density surface layer with linear density profile in bottom layer separated by a density jump. Corresponding profile types exist for approximating a temperature distribution when it is used for specifying the density distribution.

Note: When in doubt about the specification of the ambient density values it is reasonable to first simplify as much as possible. The sensitivity of a given assumption can be explored in subsequent CORMIX simulations. Furthermore, if CORMIX indicates indeed a flow configuration (flow class) with near-field stability, additional studies with the post-processor option CORJET (see Section 6.1) can be performed to investigate *any arbitrary density distribution*.

After selecting the stratification approximation to be used, the user then enters all appropriate density (or temperature) values and **pycnocline heights (HINT)** to fully specify the profiles. The pycnocline is defined as zone or level of strong density change that separates the upper and lower layers of the water column. The program checks the density specification to insure that stable ambient stratification exists (i.e. the density at higher elevations must not exceed that at lower elevations).

Note that a dynamically correct approximation of the actua ldensity distribution should keep a balance between over-and under-estimationof the actual data similar to a best-fit in regression analysis. If simulation results indicate internal plume trapping, then it is



MIKE 21 & MIKE 3 FLOW MODEL FM supports both Cartesian and spherical coordinates. Spherical coordinates are usually applied for regional and global sea circulation applications. The chart shows the computational mesh and bathymetry for the planet Earth generated by the MIKE Zero Mesh Generator

MIKE 21 & MIKE 3 Flow Model FM - Hydrodynamic Module

The Hydrodynamic Module provides the basis for computations performed in many other modules, but can also be used alone. It simulates the water level variations and flows in response to a variety of forcing functions on flood plains, in lakes, estuaries and coastal areas.

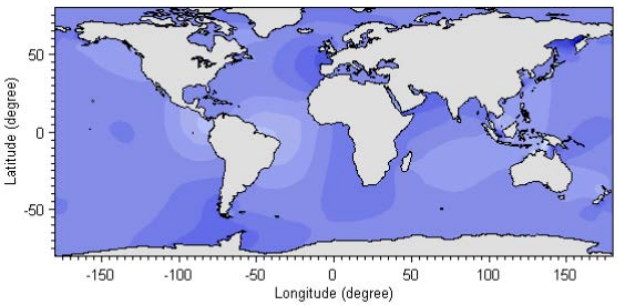
Application Areas

The Hydrodynamic Module included in MIKE 21 & MIKE 3 Flow Model FM simulates unsteady flow taking into account density variations, bathymetry and external forcings.

The choice between 2D and 3D model depends on a number of factors. For example, in shallow waters, wind and tidal current are often sufficient to keep the water column well-mixed, i.e. homogeneous in salinity and temperature. In such cases a 2D model can be used. In water bodies with stratification, either by density or by species (ecology), a 3D model should be used. This is also the case for enclosed or semi-enclosed waters where wind-driven circulation occurs.

Typical application areas are

- Assessment of hydrographic conditions for design, construction and operation of structures and plants in stratified and non-stratified waters
- Environmental impact assessment studies
- Coastal and oceanographic circulation studies
- Optimization of port and coastal protection infrastructures
- Lake and reservoir hydrodynamics
- Cooling water, recirculation and desalination
- Coastal flooding and storm surge
- Inland flooding and overland flow modelling
- Forecast and warning systems



Example of a global tide application of MIKE 21 Flow Model FM. Results from such a model can be used as boundary conditions for regional scale forecast or hindcast models

APPENDIX A-4

Drift Bottle Observations in Northumberland Strait, Gulf of St. Lawrence[1]

By L. M. Lauzier

Fisheries Research Board of Canada
Biological Station, St. Andrews, N.B.

ABSTRACT

Drift bottles have been released at fixed stations and during cruises in Northumberland Strait from 1960 to 1963. From the 2741 releases, there have been 1213 recoveries before the winter seasons. The overall high percentage recovery (44%) and the large number of returns not far from the point of release and within a short time after releases are featured.

The drift bottle recoveries suggest a surface non-tidal drift through Northumberland Strait from the northwest and west to the southeast and east. An outward drift into the Gulf of St. Lawrence, along the northwest coast of Prince Edward Island is inferred.

The speeds of the drift were generally greater than 3 miles a day on the average. They reached 5 miles a day (0.2 knot).

RÉSUMÉ

Plusieurs lancers de bouteilles dérivantes ont été faits dans le détroit de Northumberland, de 1960 à 1963, à de nombreux points échelonnés lors des croisières ainsi que quotidiennement à des points fixes. On a retrouvé, avant l'hiver, 1213 des 2741 bouteilles lancées. La grande moyenne du pourcentage des bouteilles retrouvées était élevée, 44%. Les nombreuses découvertes à une faible distance du lancement et la courte durée des dérives sont caractéristiques.

On peut déduire des trajets des bouteilles une dérive des eaux superficielles le long du détroit de Northumberland, du nord-ouest et de l'ouest, vers le sud-est et l'est. Un mouvement de sortie des eaux de surface du détroit dans le golfe Saint-Laurent semble aussi être une déduction logique.

Les vitesses de dérives sont habituellement plus grandes que 3 milles par jour, en moyenne. Elles peuvent atteindre 5 milles par jour (0.2 noeud).

INTRODUCTION

Northumberland strait, lying between the Prince Edward Island and New Brunswick–Nova Scotia coasts (Fig. 1), is an important commercial fisheries area for lobster and herring. Over the past 15 years a study of abundance and distribution of lobster larvae in the northern sector has been the main biological effort (Scarratt, 1964). The program included concurrent monitoring of oceanographic conditions and drift bottle experiments during 4 years, 1960–63. Earlier circulation studies in the Strait have been reported by Dawson (1913) and Farquharson (1959, 1962).

The purpose of the present study is to show seasonal and year-to-year changes of the non-tidal drift in the northern sector of Northumberland Strait and its relation to the drift in the central and eastern sectors of the Strait,

[1]Received for publication September 17, 1964.

as well as to that of the outside waters. The study was initiated to assist biologists interested in the circulation as it affects the biological content of the waters and the production of certain commercial species in the area. The results described here are pertinent to a very thin layer of surface waters. Work is being carried out to extend observations to a deeper layer and also along the bottom.

DRIFT BOTTLE RELEASES

Figure 1 shows the segmentation of Northumberland Strait referred to in this paper. The releases by segments were:

1. In northern Northumberland Strait (Northern and Egmont Bay segments).

   For 4 years, starting in 1960, regular releases of drift bottles were made repeatedly at one station from the end of May to October, and at two additional stations from June to September. The total number of releases every year at these stations in the northern segment varied from 116 to 206. This is called the *Pandalus* series. In June 1963, a large-scale release of 204 bottles was made at 32 positions covering both segments.

2. In central Northumberland Strait (Central segment).

   During 1961 and 1962, daily releases of drift bottles were made between Cape Tormentine, N.B., and Port Borden, P.E.I., from CNR

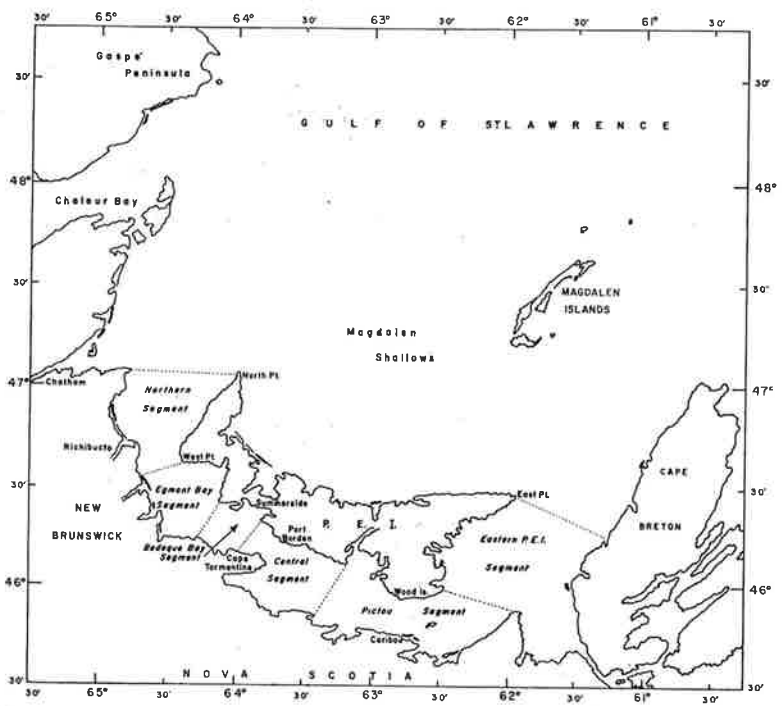


FIG. 1. Map of area showing the segmentation of Northumberland Strait used in this paper.

Abegweit (*Abegweit* series). From July to December 1961 and June to December 1962, 710 and 360 bottles were released, respectively.

3. In eastern Northumberland Strait (Pictou and Eastern P.E.I. segments).

From May to December 1962, daily releases of drift bottles in the Pictou segment between Caribou, N.S., and Wood Islands, P.E.I., totalled 780 (*Selkirk* series). In June 1963, 46 bottles were released at 7 stations covering the Eastern P.E.I. segment.

4. Outside Northumberland Strait.

During seven cruises, between 1959 and 1963, 974 bottles were released north of Northumberland Strait, along the New Brunswick coast, and at the entrance of Bay of Chaleur up to the Gaspé Peninsula in a strip 40–45 miles wide. A total of 99 and 90 bottles were released, respectively, in 1960 and 1963 between Prince Edward Island and the Magdalen Islands.

ANALYSIS OF RECOVERIES

Spatial Distribution and Inferred Drift

The overall percentage of the recoveries made in the same year as the releases was maximum for releases made in northern Northumberland Strait, at 54%. From releases made in central and eastern sectors of the Strait, recoveries were 46% and 27%, respectively. A certain number of recoveries was made after a winter season, approximating 3%, 7%, and 3% from the northern, central, and eastern sectors, respectively. Most of these bottles which were recovered in Northumberland Strait had been released in the last quarter of the year. Table I gives a summary of releases and recoveries from the fixed stations.

Table I. Summary of releases and recoveries of drift bottles from fixed stations in Northumberland Strait.

	Released	Recovered	
	No.	No.	%
Pandalus series			
1960 May–October	172	100	58
1961 May–November	206	108	52
1962 May–November	160	96	60
1963 April–October	116	51	44
Abegweit series			
1961 July–December	710	336	48
1962 June–December	360	154	43
Selkirk series			
1962 May–December	780	213	27

NORTHERN NORTHUMBERLAND STRAIT

The recoveries from the three fixed stations of the *Pandalus* series are illustrated in Fig. 2 and 3. These are seasonal recoveries totalled independently of the month of release. The distribution of recoveries by season of release would be very slightly different from that of Fig. 2 and 3, because of the short time between release and recovery.

More recoveries were made on the Prince Edward Island side than on the New Brunswick side of the Strait, indicating an easterly component of the drift of surface waters. Generally 50% or more of the recoveries — 61% on the average — are recorded in the area of release. Within the northern segment, the recoveries infer either a cyclonic eddy or a southerly drift along the New Brunswick coast and a northeasterly drift along the Prince Edward Island coast. This northeasterly movement of the surface waters is no doubt the main avenue for bottles drifting out of the northern Strait towards the Magdalen Islands, Newfoundland, Cape Breton, and the north coast of Prince Edward Island.

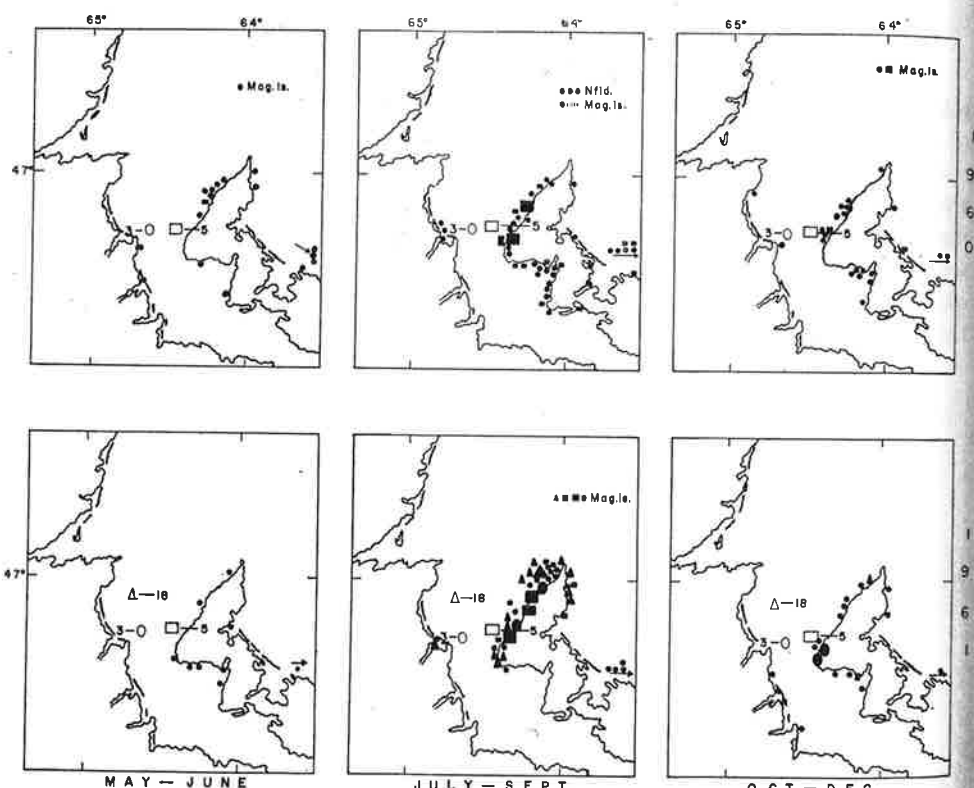


FIG. 2. Distribution of drift bottle recoveries, *Pandalus* series, from May to December 1960 and 1961. Open symbols: release stations 3, 5, and 18; closed symbols: recoveries. A small symbol represents one recovery, and a large symbol represents five recoveries.

From recoveries to the south, it is inferred that very few of the bottles that drifted into the Egmont Bay segment drifted further into the Bedeque Bay segment. The circulation in the Egmont Bay segment seems to be that of an eddy with inflow from the north along the New Brunswick coast and main outflow to the north along the Prince Edward Island coast. A predominant easterly drift is inferred from the large number of recoveries along the Prince Edward Island coast.

Even though the releases at fixed stations are few, the rates of recovery, 40–70%, are sufficient to show seasonal and year-to-year variations. The relative number of recoveries on the New Brunswick side of Northumberland Strait has a tendency to increase from summer to autumn. As shown in Table II, the percentage recovery from fixed station releases has a tendency to be minimum for July and maximum for September. The low percentage in July seems to be associated with a small proportion of recoveries within the area of releases, the northern segment, and a large proportion of recoveries away from it, either outside the Strait or in the Egmont Bay and Bedeque segments. However, a seasonal high percentage recovery is not necessarily associated

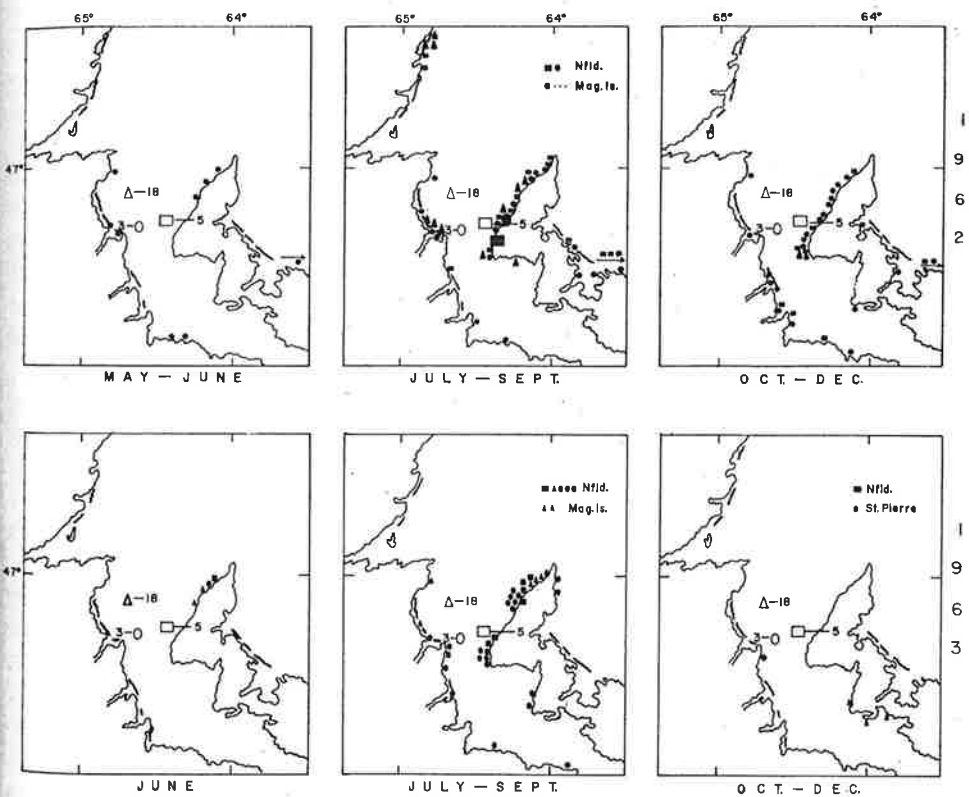


FIG. 3. Distribution of drift bottle recoveries, *Pandalus* series, from May to December 1962 and 1963. Open symbols: release stations 3, 5, and 18; closed symbols: recoveries. A small symbol represents one recovery, and a large symbol represents five recoveries.

TABLE II. Drift bottle recoveries from releases made during July and September in the northern segment, 1960–64.

	July	September
Number of releases:	128	86
Recoveries:		
in northern segment (area of release)	20	33
in Egmont Bay and Bedeque segments	13	18
outside Northumberland Strait	18	10
Total number	51	61
Percentage recoveries	40	71

with a large proportion of recoveries within the area of release. The conditions which seem to be prevalent in July are those of either divergence or dispersion from the area or rapid flushing of the area.

As shown in Fig. 2 and 3, during 1961 and 1963, a relatively high proportion of recoveries occurred in the northern segment, the area of release, associated with a relatively low proportion in the Egmont Bay segment and outside the Strait. Opposite conditions were predominant in 1960 and 1962. In 1963 the overall percentage recovery was the smallest in 4 years, and the July conditions described earlier still existed. Other contrasting conditions are also shown in the Magdalen Islands and Newfoundland recoveries; they amount to 16% of all the outside recoveries in 1962 and 80% in 1963. This could be an indication of stronger circulation over the Magdalen Shallows in 1963 than in 1962.

The recoveries from the releases made during June 1963 at several stations covering both northern and Egmont Bay segments are grouped in Table III. They infer a circulation pattern similar to that deduced from the fixed stations releases and recoveries; namely, a strong movement from west to east in the northern and Egmont Bay segments, and as an eddy in the northern segment confirming a northeasterly current along the Prince Edward Island coast. However, the inferred drift of surface waters towards the central segment of the Strait, and even east of it, is more definite than previously observed. For the bottles released in the northern segment, 31% of all the recoveries were made south of the Egmont Bay segment, as compared with the previous average of 2%. For the bottles released in the Egmont Bay segment, 86% followed a southeasterly direction towards the central and eastern sectors of the Strait. As a corollary, the inferred drift of surface waters to the east past North Point, P.E.I., was rather restricted, since only 10% of all the recoveries were recorded outside the Strait. The conditions in the second half of June 1963 were those

TABLE III. Drift bottle recoveries from releases made in June 1963 in northern and Egmont Bay segments.

	Northern segment	Egmont Bay segment
Number of releases	156	48
Number of recoveries in:		
Northern segment		
P.E.I. side	37	–
Mainland	–	–
Egmont Bay segment		
P.E.I. side	20	3
Mainland	3	1
Bedeque Bay segment		
P.E.I. side	3	–
Mainland	12	5
Central segment		
P.E.I. side	5	7
Mainland	8	5
Pictou and eastern P.E.I. segments		
P.E.I. side	4	2
Mainland	1	5
Outside	12	1

of relatively strong circulation through Northumberland Strait. This had not been observed before. It is not surprising that such differences should occur between recoveries from cruise-type releases involving a large number within a short time, and recoveries from fixed station-type releases involving repeated releases of a small number of bottles at regular intervals. The former gives inferences of the circulation over a short period of time, the latter of the average circulation over a wide range of conditions.

CENTRAL NORTHUMBERLAND STRAIT

A large number of drift bottles was released in the central part of Northumberland Strait between Cape Tormentine, N.B., and Port Borden, P.E.I. This was termed the *Abegweit* series. For the summer releases, 1961 and 1962, the percentage recovery (67%) was greater, on the average, than for the *Pandalus* series (52%). The percentage recovery decreased for those released during the autumn.

The seasonal recoveries in 1961 and 1962 are shown in Fig. 4 and 5, respectively. The salient features of the recoveries are: (1) their large proportions on the Prince Edward Island side in comparison with the New Brunswick side; (2) their concentrations at a short distance from the point of release; (3) their relative spread to the east and their absence in the Egmont Bay

segment. The inferred predominant drift from the mainland to Prince Edward Island was relatively stronger in 1961 than in 1962. This west–east drift seems to weaken during the autumn (October–December) as compared with the summer (July–September). The same variations were inferred from recoveries of the *Pandalus* series.

The *Abegweit* series, similar to the *Pandalus* series, inferred a somewhat restricted drift. However, there is an inference of a definite southeasterly drift from the central segment into the Pictou segment but only occasionally further to the east. The much less frequent westerly drift towards the Bedeque Bay segment seems to increase from summer to autumn.

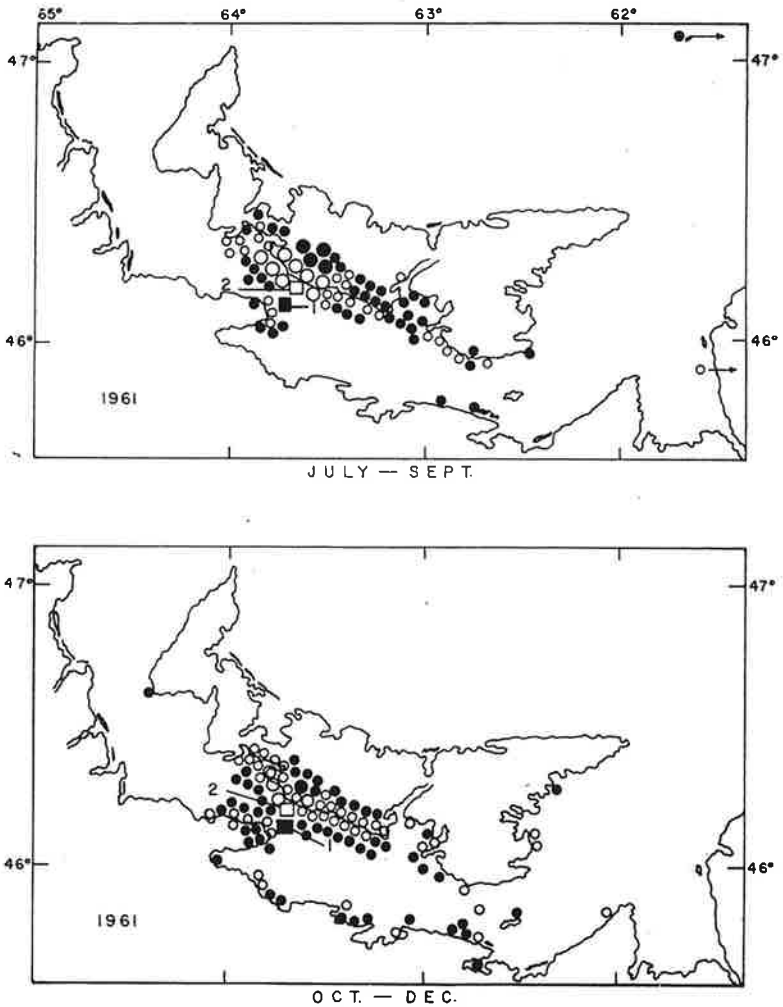


FIG. 4. Distribution of drift bottle recoveries, *Abegweit* series, from July to December 1961. Squares: release stations 1 and 2; circles: recoveries. A small circle represents one recovery, and a large circle represents ten recoveries.

EASTERN NORTHUMBERLAND STRAIT

In 1962 only, the eastern sector of Northumberland Strait was studied at the same time as the central and northern sectors. The overall percentage recovery from the *Selkirk* series released between Wood Islands, P.E.I., and Caribou, N.S., was 27%, the lowest of the three series in Northumberland Strait. It was 35% for summer month releases, still the lowest of the three series.

As inferred from the recoveries (Fig. 6), a predominant drift from the mainland to Prince Edward Island is one of the features of spring and summer seasons. There is almost a reverse of this drift during autumn. There is also

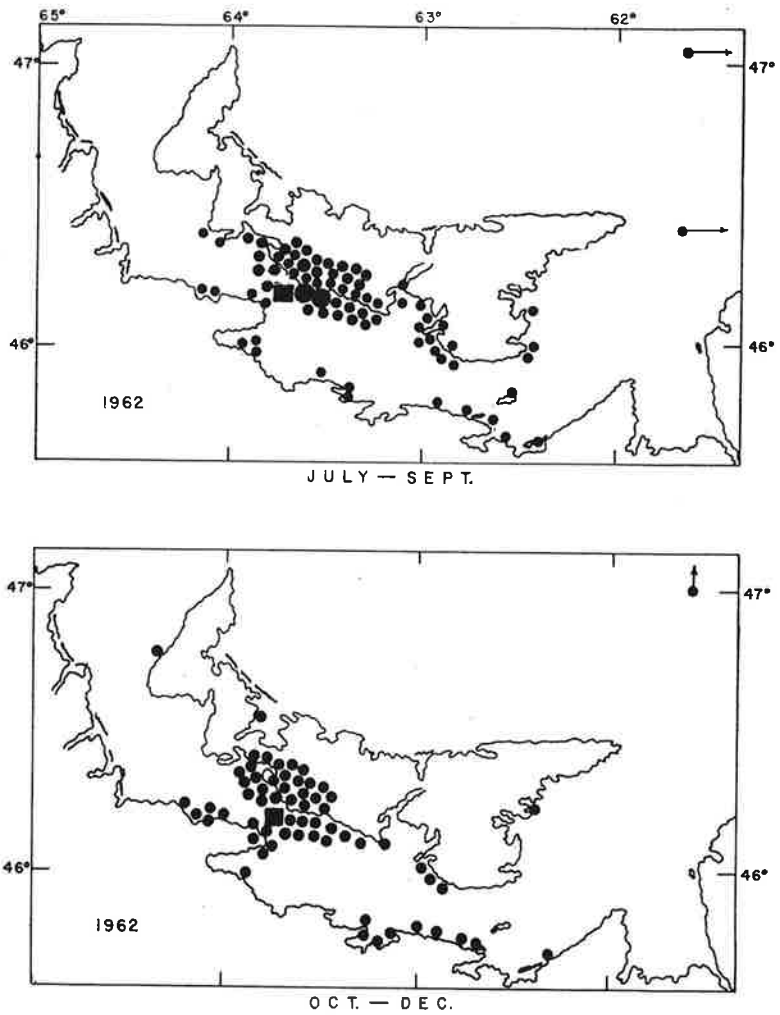


FIG. 5. Distribution of drift bottle recoveries, *Abegweit* series, from July to December 1962. Squares: release station; circles: recoveries. A small circle represents one recovery, and a large circle represents ten recoveries.

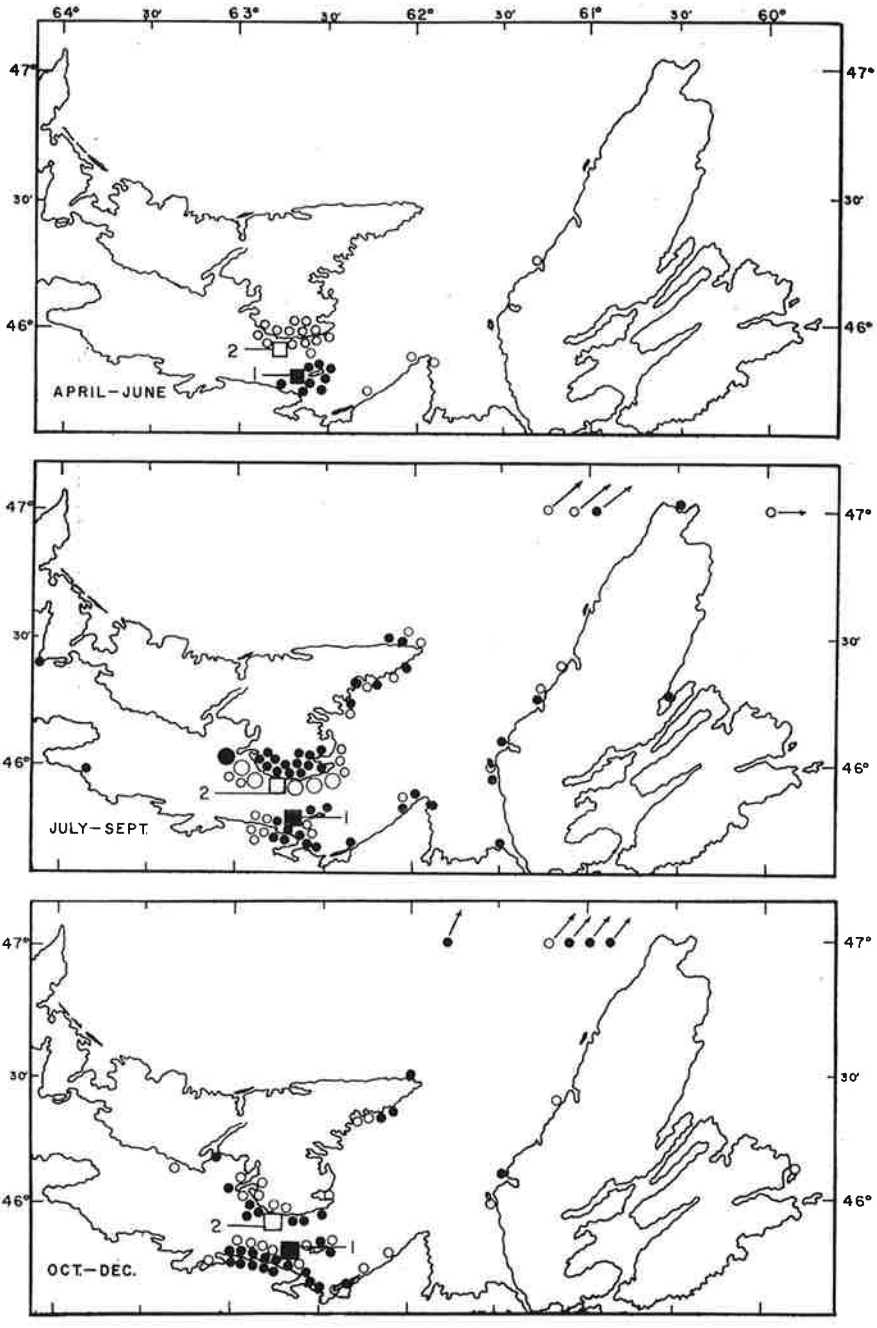


FIG. 6. Distribution of drift bottle recoveries, *Selkirk* series, from April to December 1962. Squares: release stations 1 and 2; circles: recoveries. A small circle represents one recovery, and a large circle represents ten recoveries.

a certain amount of westward drift but it hardly extends into the central segment. A definite easterly drift from the Pictou segment to the eastern P.E.I. segment and beyond is inferred. From the summer releases, 22% of all the recoveries are recorded to the east more than 20 miles from the point of release, and 15% more than 40 miles away. At the same time 75% of all recoveries were observed within 15 miles of the point of release. There is no strong evidence of drift on the south side of the Strait opposite to that on the north side.

In June 1963, 46 drift bottles were released at seven stations in the eastern P.E.I. segment at approximately the same time other releases were conducted in the northern end of the Strait. A percentage recovery of 50% was recorded for the releases in the eastern P.E.I. segment. More than half of these recoveries infer an easterly drift towards outside the area of release, while the others infer a westerly drift, up to 25 miles west of the point of release. There are similarities between the northern segment and the eastern P.E.I. segment. To someone in Northumberland Strait, looking towards the open Gulf, there seems to be a southward drift on the left hand side of the segments and a northeasterly drift on the right hand side. The presence of cyclonic eddies is inferred in both cases.

OUTSIDE RELEASES

On several occasions, drift bottles were released during cruises in the vicinity of Northumberland Strait. The surface non-tidal drift over the southwestern Gulf of St. Lawrence as inferred from drift bottle recoveries seems to be generally to the south and to the east (Bumpus and Lauzier, 1964). It is important to assess the contribution of outside waters to Northumberland Strait. From 974 bottles released north of the Strait, 247 bottles were recovered (before winter) in various parts of the Gulf. Less than 9% (21) of all recoveries or 2.2% of all releases were reported from Northumberland Strait, most of them in the northern segment, and none as far as the central segment. These originated only from some of the cruises.

The inferred circulation in the area north of Northumberland Strait seems to be in most cases to the southeast or east and in at least 10% of the cases to the south towards the Strait. However, it is impossible to estimate the extent of a southerly drift which makes a "u-turn" in northern Northumberland Strait: into the northern segment along the New Brunswick coast and out into the Gulf again along the Prince Edward Island coast. It seems that Northumberland Strait does receive on the average a rather small "share" of the surface waters just north of it.

Time Distribution and Inferred Speed

The overall percentage recoveries for the summer month releases in 1962 were 63%, 61%, and 35% for the *Pandalus*, *Abegweit*, and *Selkirk* series, respectively. The time taken to recover a definite proportion of released bottles

varies from one series to the other, depending on their location in the Strait. From the *Abegweit* series, 25% of the releases in the central segment were recovered within 10 days; from the *Pandalus* series, 25% of the releases in the northern segment were recovered in 17 days; and from the releases in Pictou segment, the *Selkirk* series, 25% were recovered in 23 days.

Such a short lifetime afloat of an *Abegweit* bottle is not surprising if one considers the geographical location of the points of release, the predominant drift from the mainland to Prince Edward Island, and the rate of recovery within 10 miles of the point of release — 67% of all recoveries. It took a longer time to recover 25% of the bottles from *Pandalus* and *Selkirk* series because these were released in wider parts of the Strait which are adjacent to the open Gulf. The time given for "25% recovery" is not constant for a given segment but the shorter period always seems to be characteristic of the *Abegweit* series, released in the central segment.

The inferred speed of surface drift has been calculated in two different ways, after eliminating the recoveries recorded within 10 miles from the point of release. First, an average speed was computed from the fastest bottles (the upper third of the speed frequency) irrespective of the distance travelled outside the 10-mile radius; this was the technique used by Bumpus and Lauzier (1964). Second, another average speed was computed from most of the bottles which had travelled a certain distance, always greater than 10 miles; some very slow ones were excluded.

In using the fastest bottles an attempt was made to show seasonal variations of speed and direction; however, no such attempt was made with the other technique. The average speeds are listed in Table IV. As expected, the speeds computed for trips longer than 20 miles are approaching the values calculated for the fastest bottles. In general, the longer the "trip", the faster

TABLE IV. Average surface drift in miles per day from June to October.

Series segment	*Pandalus* northern	*Abegweit* central	*Selkirk* Pictou
Fastest bottles[a]	4.5 (NE)	3.5	3.5
	3 (SE)		
Most bottles			
10–15-mile trips	1.6	1.3	1.8
15–20-mile trips	2.9	1.3	1.5
20–50-mile trips	3.7	3.3	2.3
>50-mile trips	3.5	–	2.7

[a]Upper third of the speed frequency and drifts of 10 miles or more

is the drift. Possibly a short trip may be a series of drifts back and forth or drifts around an eddy and ending not too far from the point of release. A longer trip may be a more straightforward drift free from aleatory motion. It should be realized that the speed values listed here are minima and in some cases may be much smaller than the actual speed of the drift. However, they represent the speed of a thin surface layer 18–20 cm deep. The residual currents recorded by the Canadian Hydrographic Service (Farquharson, 1959) are of somewhat lower strength. They vary generally between 1 and 2 miles a day and represent the speed of a residual current at a depth of 7 m.

In the northern segment, the northeast drift always seemed to be faster than the southwest drift by a factor that varies between 1.3 and 2.5. The maximum speeds occurred in July for the northern segment and from June to August for the Pictou segment. They may reach 5 miles a day. There were no seasonal tendencies in the inferred speeds for the central segment.

DISCUSSION

Because of the high proportion of local recoveries, the three series *Pandalus*, *Abegweit*, and *Selkirk* seem to indicate that they are three "stocks" that do not "intermingle". Most recoveries infer a drift to a certain point along the Strait but very seldom further. There seems to be a drift from northwest to southeast at various points along the Strait. Bottles released in a relatively small body of water have a short average lifetime and are subjected to a high rate of recovery. From the rate of recovery, the average speed, and the distance to be travelled to go from one area of release to the other, it is possible to estimate the chances for bottles released in the central segment of the Strait to drift into another area of release. With an average speed of 2 miles a day, it would take 25 days to reach another area of release; by that time, 40–45% of the bottles released would have already been recovered. In the case of the bottles released in the northern segment, the assumed speed is higher and the rate of recovery within the first month, lower. Then 25% of the bottles released would have been recovered before being able to reach the area of release in the central segment, if all the bottles had drifted into the segments to the southeast. In fact only 15% did so. Consequently the experiment cannot demonstrate continuity of flow.

The recovery charts (Fig. 2–6) do not imply a continuous drift from one end of the Strait to the other. However, the similarities in the speeds within various segments, the consistency and some predominance of southeasterly and easterly drift, as well as the lack of long drifts westward in the central and Pictou segments infer a resultant drift of surface water in Northumberland Strait from the northwest to the southeast. Figure 7 shows the average surface circulation in Northumberland Strait as inferred from drift bottle releases and recoveries. The inference of a resultant drift from the northwest to the southeast is strengthened by consideration of the seasonal variations of salinity conditions. During the summer, a large body of relatively low salinity

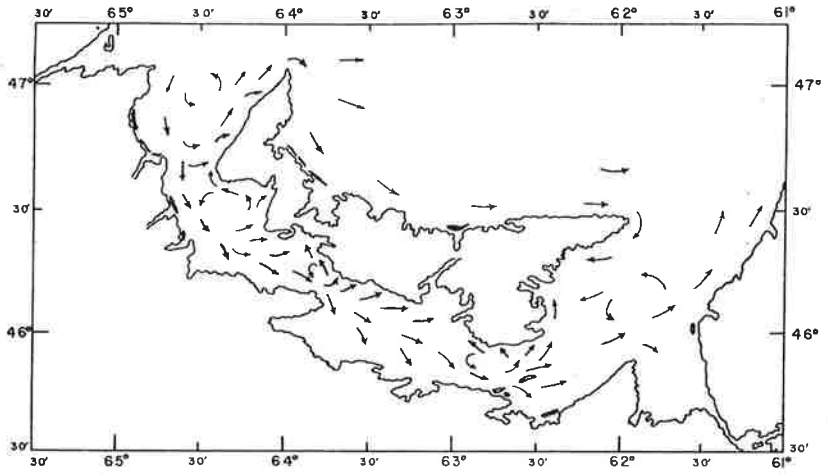


FIG. 7. The inferred non-tidal drift in Northumberland Strait.

water drifts southward over the Magdalen Shallows. A summer minimum of salinity is also observed in Northumberland Strait (Lauzier, 1957).

It was mentioned previously that, in general, the relative distribution of recoveries from the July *Pandalus* series is different from average conditions. It was presumed then that either dispersion or rapid flushing occurred in the area. Approximately at the time there is a summer minimum of salinity and a definite gradient of salinity between the western and eastern ends of Northumberland Strait which might be partly responsible for a stronger circulation through the Strait, or at least in both the northern and Egmont Bay segments.

The predominant drift in Northumberland Strait from the mainland to Prince Edward Island during the summer months was observed to diminish during the autumn all over the Strait and almost to the point of a reversal in the Pictou segment. This might be associated with the weakening of the southwest winds and the strengthening of the easterly winds in the autumn. Although the outside contributions to Northumberland Strait from the north are generally small, they were greater in June and August 1959 and May 1960 than at any other time of observation. From one cruise as many as 23% of the recoveries were reported in Northumberland Strait. In most cases, when bottles were recovered in the Strait, the resultant winds (as observed at Summerside, P.E.I.) had either a stronger easterly or a weaker southerly component than usual. When bottles released outside were not recovered in the Strait, the resultant winds seemed to have a stronger component than usual from either the south or the west. These are the only relationships found so far, between the direction of the surface drift and the resultant winds on a monthly basis.

It is assumed that the bottles drift with the water and its biological content such as lobster and herring larvae. Drift bottle observations give some of

the history of particles of water (Lagrangian observations) in contrast with current-meter observations which give the history of what goes on at one point (Eulerian observations) without knowledge of origin or past history of the particles in motion. Most biological and oceanographic observations are of the Eulerian type. The circulation of the surface waters in Northumberland Strait illustrated in Fig. 7 should be taken as the most probable circulation pattern under various conditions. From the data on hand it is impossible to say how constant and how fast is the renewal of waters and the biological content in a given segment, or how long the water stays in a segment. However, eddies seem to exist at both ends of the Strait. There is a northwest–southeast drift through the Strait but it is not known if this inferred drift is counterbalanced by a reverse drift which is impossible to observe because of the prevalent onshore drift along Prince Edward Island. It is hoped that the answers to this and many other unknowns concerning water circulation in Northumberland Strait will be revealed through further research.

SUMMARY

The surface circulation in Northumberland Strait has been studied from drift bottle experiments at fixed stations and during cruises. Emphasis was given to the northern sector of the Strait, starting in 1960, but the studies covered the whole Strait in 1962. From releases of 2741 bottles in 1960 to 1963 the overall percentage recovery was 44%. The percentage recovery for a year or for the summer months was generally higher (above 60%) in the northern part of the Strait, and lower (less than 40%) in the eastern end of the Strait.

The main features of the inferred surface non-tidal drift are:

1. A general movement of the surface waters through the Strait from the northwest and west to the southeast and east.
2. A predominant drift from the mainland to Prince Edward Island, stronger in the summer than in the autumn.
3. A cyclonic eddy in the northern entrance of the Strait with a southerly drift along the New Brunswick coast and northeasterly drift along the Prince Edward Island coast, the latter being the main avenue towards the open Gulf.
4. Other eddies are suggested mainly south of West Point, P.E.I., and at the eastern entrance of the Strait.
5. The speeds seem to vary seasonally in certain parts of the Strait. They are generally greater than 3 miles a day on the average, and may reach 5 miles a day.

In general the rate of drift bottle recoveries is high for the releases made in the central part of the Strait, where 25% of the bottles released are recovered within 10 days. It takes 17 and 23 days to recover the same proportion of those released in the northern and southern ends, respectively.

ACKNOWLEDGMENTS

The author is indebted to Dr D.G. Wilder of the lobster investigations at the Biological Station, St. Andrews, N.B., for his assistance in organizing the drift bottle releases from M.V. *Pandalus II* starting in 1960; to the masters of *Pandalus II* (Fisheries Research Board), of *Abegweit* (Canadian National Railways), and of *Lord Selkirk* (Northumberland Ferries) for their co-operation in the task of drift bottle releases. The assistance of Messrs J.G. Clark and A.W. Brown in the organization and co-ordination of releases and in the compilation of drift bottle data is gratefully acknowledged. Drift bottle data related to outside releases in 1959 were supplied to the author by Dr Y.M.L. Jean.

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APPENDIX B-1

Caribou Harbour and Caribou Channel - dynamics, tides, ice, marine species and fisheries

21 February 2019

Submission from Local Fishermen Allan MacCarthy and Greg Egilsson

Introduction

Allan MacCarthy and Greg Egilsson are two of approximately 82 (lobster) and 100 (herring) local fishers who fish in the immediate area encompassing and surrounding the effluent outfall discharge location (CH-B) proposed by Northern Pulp. Both have a lifetime of experience fishing in the productive waters of the Northumberland Strait, in the very area where Northern Pulp now seeks to discharge its effluent.

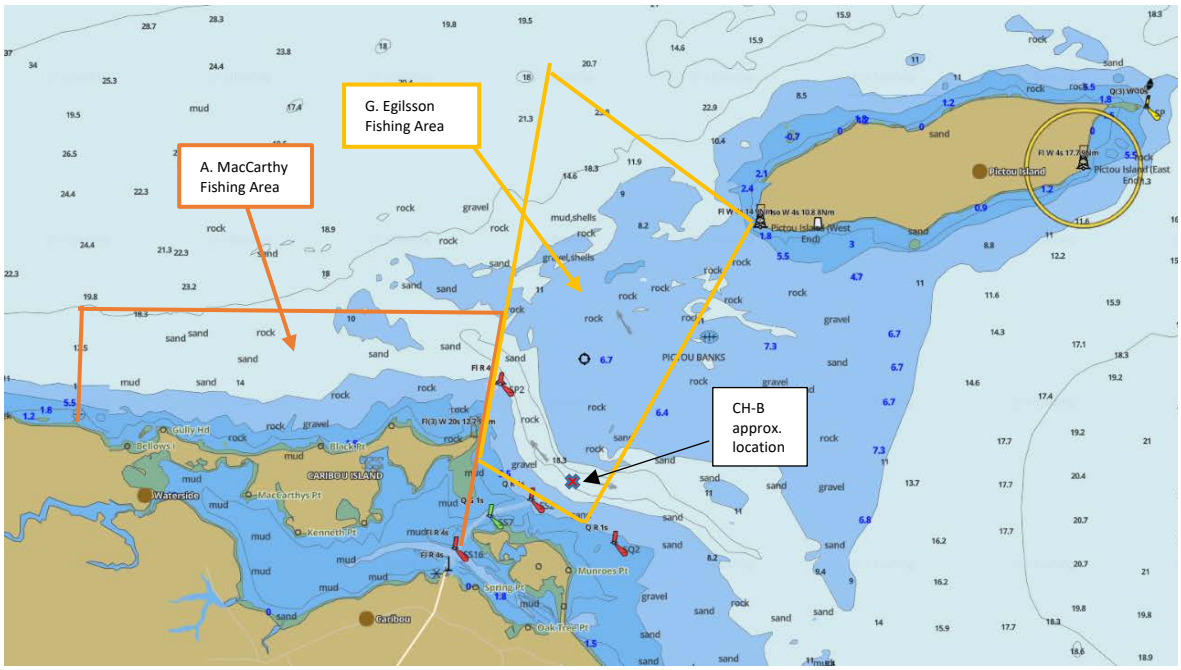
Allan MacCarthy is a life-time resident and fisherman in the Caribou-Pictou area. He started out fishing with his dad and his uncle, and has since fished on his own for 40 years. For the last 17 years he has fished in his boat called the "Red Trapper". Mr. MacCarthy holds a Fishing Master Class II and Watch Keeping Mate Certifications. He has both trained in and taught navigation and meteorology at the School of Fisheries in Pictou (now part of the Nova Scotia Community College). Mr. MacCarthy is a past member of the DFO/Industry Gulf Herring Advisory Committee and Scallop Advisory Committee. He fishes lobster, herring, rock crab and scallops in the Northumberland Strait. He has also fished mackerel and, prior to 1998, groundfish there.

Mr. MacCarthy's lobster fishing grounds are located in the Caribou area, and cover an area of approximately 20 nautical miles^2 (37 km^2) (5 nautical miles (9.2km) east-west x 4 nautical miles (7.4km) north-south), positioned to the north of Caribou Island (see the chart below). The eastern edge of his fishing area is approximately ½ nautical mile from the outfall location (CH-B) proposed by Northern Pulp. Mr. MacCarthy's fishing grounds overlap with about 13 or 14 other fishers who use some of the same territory.

Greg Egilsson is likewise a life-long fisherman in the Caribou area. He has fished on his own for 30 years, and for 10 years before that with other fishermen. In his vessel, the "Jenny CE", he fishes lobster, herring and scallops, and has also fished mackerel and groundfish in earlier years. Like Mr. MacCarthy, Mr. Egilsson holds Fishing Master and Watch Keeping Mate certifications and lectured on navigation and meteorology at the Pictou School of Fisheries. He is Chairman of the Gulf NS Herring Federation and represents Area 16F herring fishers (PEI & NS) at local and Gulf Small Pelagics Advisory Committee meetings. He is also a member of the working group for that Committee.

Mr. Egilsson's lobster fishing grounds are adjacent to Mr. MacCarthy's to the east (see chart below), but angle northeast toward Pictou Island, and include an area northwest from that line. The proposed outfall point CH-B is positioned inside his territory at the southeast corner. Mr. Egilsson often hauls his first trawl of the day during lobster season in roughly the same location as Northern Pulp's proposed outfall. His fishing grounds overlap with those of about 20 other fishers.

The lobster fishing grounds of Mr. MacCarthy and Mr. Egilsson are marked approximately on the following chart. However, it is important to note that they are only 2 of up to 82 lobster fishers who fish in the Caribou area.



Lobster Fishing Grounds – Allan MacCarthy and Greg Egilsson

Fishing in the Caribou Area

Fishing seasons in the Caribou area follow approximately this pattern annually:

Lobster– May and June

Herring – September to Mid-October

Rock Crab – early August to end of November

Scallops – November to mid-December

Oysters – are harvested in the fall but grow year round in Caribou Harbour

Local fishermen use several wharves near to the Caribou-Prince Edward Island Ferry Wharf, including Logans, Little Entrance Wharf, Sinclair's Island, Pictou Island, and Pictou Landing First Nation. The public fish mackerel, bass and smelts off the Caribou wharf.

There are approximately 25 commercial tuna fishing vessels and 8 tuna charter vessels that fish for tuna in the middle grounds (between Caribou and Pictou Island), within a commercial fishery, or for sport and tourism.

Moving out from the immediate proposed outfall location, many more fishers work in the Strait, which supports approximately 1800 fishers from Nova Scotia and Prince Edward Island. PEI is approximately 9 nautical miles (16.7 km) across the Strait from the outfall site.

Fish species

Lobster

The Caribou Channel and the Strait support a large lobster population. The area falls within Lobster Fishing Area (LFA) 26A-1. As indicated above, 82 lobster fishers fish in the area, including the approximately 12 fishers from Pictou Landing First Nation whose fishing grounds are located to the southeast of the proposed outfall.

Lobster larvae float at or near the water surface for about 12 weeks, starting mid-July each year. The larvae are swept along by the currents and may circle Pictou Island in the gyre. Once the lobster begin to mature they drop to the sea floor. Mature lobsters feed on the floor of the Strait, and will be affected by any alteration to the seabed, and the accumulation of pulp mill solids or contaminants in their feeding area.

Lobster are very sensitive to temperature changes. If water temperature rises or drops quickly, lobster typically "disappear" – meaning that they become inactive and do not enter lobster traps.

The Caribou Channel hosts a productive lobster fishery, as do most other parts of the Strait. In the Caribou area, approximately 82 fishers set about 280 traps each, for an estimated total of 22,960 traps (5 traps per trawl). Lobster season falls within May and June. Lobsters are retained if they are of sufficient size[1]; but undersized lobsters, berried females (females carrying eggs) and windows (large females above 110 mm) are returned to the water to ensure a sustainable population.

Herring

As mentioned above, approximately 100 Nova Scotia herring fishers, and fishers from Prince Edward Island fish in the grounds in the immediate area of the proposed outfall at CH-B. Herring fishers go where the fish are. In the 2018 herring fishery in the Caribou area, 90 percent of the herring were caught within a 2 mile radius of the proposed outfall location (CH-B).

Herring is fished in September and October. It is used as a food fish and also for lobster bait and is important in the food chain for numerous species. Herring stocks have been declining and DFO has designated herring as a "low caution" stock, the step before critical – DFO is watching the fishery closely to determine if it will remain viable. The proposed outfall CH-B is located in the middle of the last major active spawning area for Area 16F herring. Herring spawning grounds have compressed in the past few years as the stock has declined. Very little herring spawning occurs anywhere else in the Eastern Gulf.

During the herring fishery, it is not unusual for the Caribou-Prince Edward Island Ferry to navigate around the herring vessels, as they sometimes fish right in the ferry channel.

Rock Crab

Mr. MacCarthy also fishes for rock crab, from August to the end of November each year. He and others fish out in a wide area, including near the proposed outfall location CH-B. There are 22 rock crab fishers in the local area. Mr. MacCarthy and Mr. Egilsson have observed large numbers of juvenile rock crab in

[1] In 2018, must have a carapace size of at least 73 mm in LFA 26A-1

Caribou Harbour, which is viewed as a crab nursery. Mature crabs tend to seek out cooler deeper water in summer and shallower water in winter.

Scallops

Mr. Egilsson and Mr. MacCarthy fish scallops in the Caribou area from November to mid-December, outside the DFO-imposed Scallop Buffer Zone. DFO prohibits scallop fishing within one mile of shore or of Pictou Island, in order to protect juvenile lobster and lobster nursery habitat. DFO's website[2] designates this habitat as important to biodiversity conservation, due to its importance for the life-cycle of the species.

Scallops are filter feeders, making them susceptible to contaminant build-up in their local environment.

Detail of Scallop Buffer Zone – SFA 24

Oysters

While neither Mr. Egilsson nor Mr. MacCarthy harvest oysters themselves, they advise that there is an oyster fishery in Caribou Harbour. Caribou Harbour is open to the Strait only by way of the ferry channel, and Harbour water moves in and out only via that channel, due to blockage of "the Little Entrance" channel into the Strait. As filter feeders, oysters fished in the Harbour are particularly vulnerable to any build-up of contaminants that could occur. As stated below, tides and currents can push water from the Caribou Channel into Caribou Harbour, where it may accumulate until conditions change. The proposed effluent outfall at CH-B is located close to the ferry route by which the water enters the Harbour.

[2] DFO - Scallop Buffer Zones (SFA 21, 22, 24) - http://www.dfo-mpo.gc.ca/oceans/oeabcm-amcepz/refuges/sfa-zpp-eng.html

Bathymetry and Depths

Northern Pulp's proposed outfall at point CH-B in the Caribou Channel is to be placed in a relatively deep and narrow part of the channel, 20 m deep and about 0.1 nautical miles (0.2 km) wide. From there the channel slopes upward to the much shallower waters found on either side of the channel (at depths of approximately 7 to 9 m).

Depths are shown in metres in the attached chart. A pipeline leading to the outfall will have to travel across areas with depths in a range of 0 to 8 m. As discussed further below, this makes the pipeline leading to the outfall very vulnerable to ice damage and increases the likelihood of rupture and leakage.

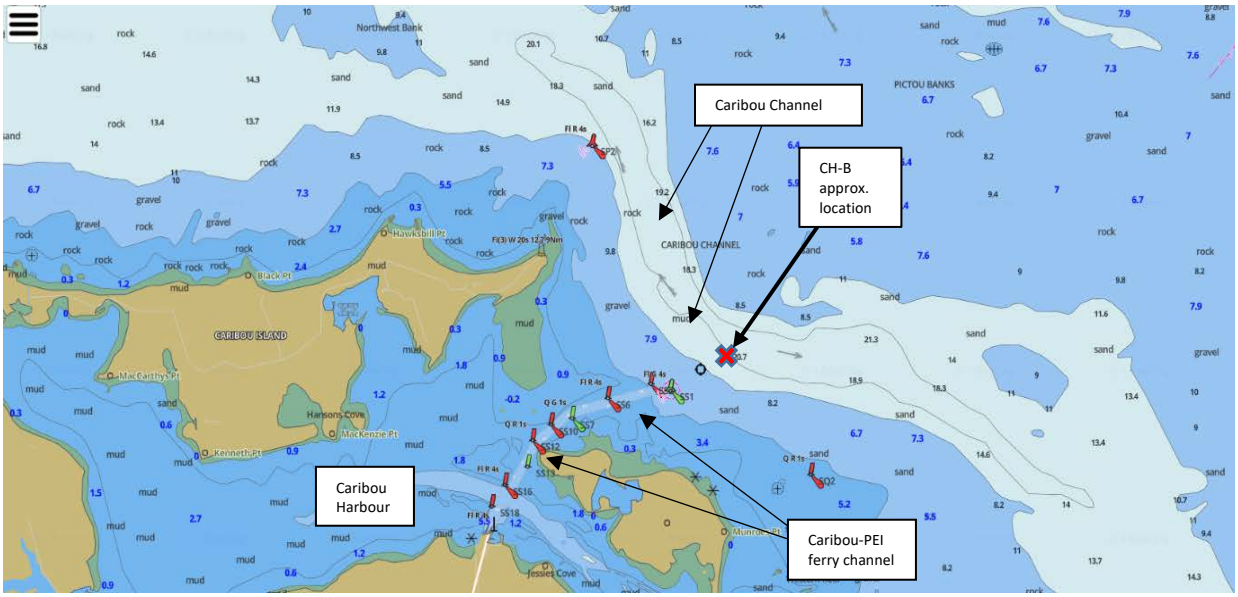


Chart of Caribou Channel and surrounding area

The Caribou-Prince Edward Island Ferry departs Caribou Harbour via the ferry channel. The ferry channel has to be dredged to maintain sufficient depth for the ferry. Prior to the creation of the dredged ferry channel, people would walk their cattle at low tide through the shallow water between Munroe's Island and Caribou Island.

Until recent decades, there was a passage between Munroe's Island and the mainland called the "little entrance". Winds and tides have since filled the little entrance and blocked that passage, effectively joining Munroe's Island to the mainland and limiting the flushing of Caribou Harbour. This is indicative of the shifting sea bottom, which is common in this area.

Currents and Tides

Currents are complex in the Caribou Channel area and are very localized. The water moves back and forth with the tides, but on the south side of the Channel will also flow into Caribou Harbour. The channel area between Caribou Island and Pictou Island is narrower than the Strait on either end of the channel, and the water must speed up as the tides push it through in both directions. As the water moves through the channel, it causes an upwelling effect up the slopes on either side of the channel.

Northern Pulp's materials say that water movement due to tides is southeast and northwest.[3] While the falling tide does move southeast, the rising tide generally moves more north than northwest. Northern Pulp has made very general assumptions about its proposed outfall location, but has taken no steps to actually measure currents in the outfall area, or along the pipeline route, despite its proposed placement in the midst of an area used by local fishers.

The tide moves southeastwards through the channel, and then roughly northwards, and repeats this pattern, on approximately 6 hour intervals. Tides vary in height, but there is a roughly 4 to 5 foot differential between low and high tide, with 6 feet on a full moon. There is an approximately 1 hour period of slack tide between each high and low tide during which the water does not flow in any significant way and no meaningful dispersal would occur in Caribou Channel. Slack tides can last up to 2 hours during the summer months.

Slack tide periods in Caribou Harbour

In Caribou Harbour, there are four slack tide periods per day. Fishermen estimate that in the vicinity of the proposed outfall, slack tides last on average 45 minutes, four times a day.

Local fishermen describe a slack tide as "when everything stops, even in a dynamic area." They also describe water movement as much slower than normal for a period of time before and after a slack tide period. Slack tide periods are a preferred time for herring fishing.

Periods of slack tide vary in different bodies of water and under different conditions. Currents, wind direction and stress, and tidal heights affect the duration of slack tide periods.

Duration of slack water at a given location is inversely related to the height of the tide at that location. The smaller the tide, the longer the period of slack tide will last. Tidal flow in the Caribou Harbour area can be quite small. See the image on the next page.

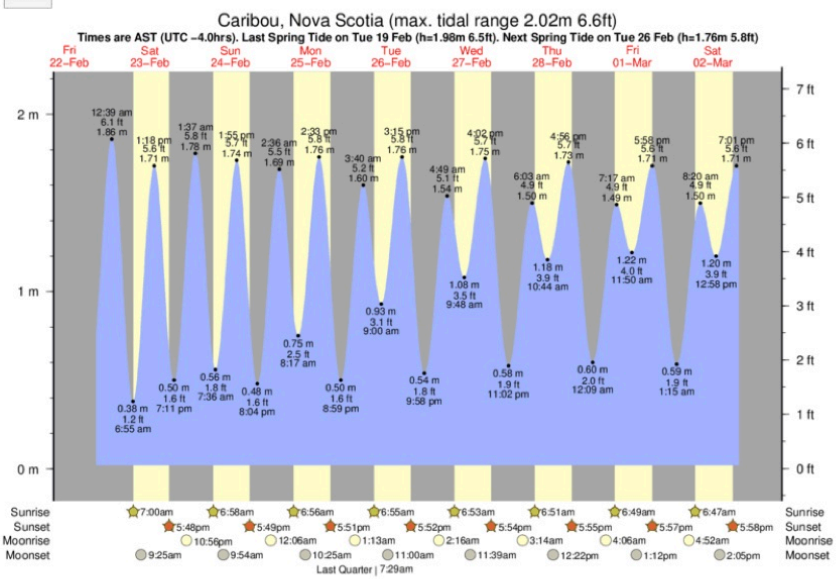
[3] Addendum to Receiving Water Study, p. 18.

tide-forecast
free tidal predictions for the whole World

TIDE CHART FOR CARIBOU SHOWING LOW AND HIGH TIDE TIMES FOR THE NEXT 7 DAYS

Tide Times are AST (UTC -4.0hrs). Last Spring High Tide at Caribou was on Tue 19 Feb (height:1.98m 6.5ft). Next high Spring Tide at Caribou will be on Tue 26 Feb (height:1.76m 5.8ft).

Tide datum: Mean Lower Low Water.



Caribou Tide Chart. The largest known tidal range at Caribou is 2.02m 6.6 feet.

CARIBOU TIDE CHART KEY:

The tide chart above shows the height and times of high tide and low tide for **Caribou, Nova Scotia**. The red flashing dot shows the tide time right now. The yellow shading corresponds to daylight hours between sunrise and sunset at Caribou.

NOTE: Use of this site for Caribou, Nova Scotia tide times is subject to our terms.

Variations in the strength of the current will also vary the time when the stream reverses, altering the time and duration of slack water. Northern Pulp's Receiving Water Study does not account for slack tides or their effect on predicted diffusion of effluent released from the proposed pipe at CH-B.

Northern Pulp's submissions do not take into account storms and storm surges and the heavier waves that can occur, especially in the fall. During rough weather and storm surges, very high water levels occur within the Harbour, and can be 5 or 6 feet higher than typical as shown in the photos on the next page.

These extreme tide events can last for several days, when combined with winds and waves from the northeast, holding the high water in Caribou Harbour. At such times, if effluent were to be discharged in the channel at CH-B, it would not disperse to the east or north – rather it would be pushed into Caribou Harbour and might accumulate there until the dynamic changed.

Regular High tide in Caribou Harbour

High tide in Caribou Harbour during a storm in the fall of 2018

Water can also enter the Harbour via movement of vessel traffic. When the PEI ferry enters Caribou Harbour from the Caribou Channel, it pushes in a wall of water with it from the channel. The wave is high enough to "surf".

As well, the water in the Caribou Channel is part of a gyre/current that circles Pictou Island, in a counter-clockwise rotation, rather than strictly east-west. The Pictou Island gyre is well-known to fishers. In one winter during the 1930s, a ferry vessel became stuck in the ice south of Pictou Island. The gyre moved the vessel with the ice around Pictou Island three times over the winter, until the thaw released the ice's grip on the ferry in the spring. Gyres in the Strait and localized currents in the Caribou Channel and around Pictou Island were also noted in a series of drift bottle studies conducted by DFO scientists in the 1960s[4]. Mr. MacCarthy and Mr. Egilsson believe that the significant effects of the Pictou Island gyre, and how it might affect effluent dispersal or build-up, are not analysed or considered in Northern Pulp's materials.

Northern Pulp's materials fail to account for the significant localized water flow dynamics in the Caribou area, and in the Strait overall. Northern Pulp's failure to take basic measurements of currents and other dynamics results in incorrect and oversimplified modelling of crucial water flows.

Swells and Wave action

The largest waves in the Caribou area occur in the fall. They come generally from the Northwest with the prevailing winds, although the largest storms with the highest tides come out of the Northeast every fall and winter. Allan and Greg observe that storms have become more frequent and more violent over the past 40 years.

[4] Lauzier, L.M., 1965. Drift bottle observations in Northumberland Strait, Gulf of St. Lawrence. Journal Fisheries Research Board of Canada 22(2), 353-368.

Ice

Ice is typically present in the Caribou area from the end of December through April, but can set in earlier and remain later if temperatures are cooler than normal. Due to ice conditions, the Caribou-Prince Edward Island Ferry does not run from late December until the beginning of May. See in Appendix A, the Ice Chart for 3 February 2019 for the Gulf of St. Lawrence, including the Northumberland Strait. This chart is updated daily and Mr. MacCarthy reviews it on a regular basis.

In the shallows of the inshore area the ice freezes to the bottom. The fishermen call this “fast ice”. Further out into the deeper water drift ice forms on the surface. As water splashes on to the ice, it builds up into thicker and thicker layers.

These photos were taken by Mr. MacCarthy in 2014 off Caribou Island, and show the fast ice in the foreground and the ridge of sea ice farther out.

In the photo below, the sea ice is resting on the bottom which makes it stand far out of the water.

When, due to ice thickness or low tide, ice sheets rest on the sea floor, they will freeze to boulders and sediment. When high tide returns it lifts the ice and frozen material from the sea floor and moves it elsewhere. The ice in the Strait has picked up concrete vessel anchors weighing 2200 pounds and moved them far from their original location, such that they could not be located by divers. Ice also moved a 5000 lb piece of armour stone a considerable distance within the Strait.If wind or currents move the ice sheets, ice can also scour across the sea floor. Scour can be enhanced if boulders or other material have frozen on to the bottom of the ice. Ice scour can occur quite deep, even as deep as the proposed outfall position. As indicated in Stantec's Preliminary Receiving Water Study, in 1991 an undersea fiber optic cable to Prince Edward Island was broken by ice at a depth of over 18 metres.[5] Divers have reported ice scour occurring as deep as 20 m.

[5] Stantec, Preliminary Receiving Water Study, August 2017, at page 4.81.

Ice can be pushed up on to shore by the wind. In spring the ice begins breaking up into large icebergs and ice pans. At this time, when the wind blows from the northeast, it pushes the ice into harbours where it can pile up 25 (7.6m) to 30 feet (9m) high. See the photo below, taken near Caribou Lighthouse.

Given the dynamics observed by Mr. MacCarthy and Mr. Egilsson, any pipeline or diffuser installed on or under the sea floor, whether in shallow water or at a depth of 20 m, would be vulnerable to ice damage. Likewise, any piping infrastructure on or near the shore could be damaged from ice moving on to the shore. Attempts to protect the pipeline by trenching or covering it with armour stone would be insufficient, given the ice's ability to scour and move heavy objects and sediment, and the shifting sea bottom in that area.

Wind

Mr. MacCarthy and Mr. Egilsson reviewed the wind directions as recorded in a recent air emissions study of the Pictou Area.[6] They agree that Figure 2 in the study report accurately captured the typical wind patterns[7] but note that the winds are much stronger offshore than at the Granton station where the data was tracked for the Pictou study.

Figure 2 is reproduced below:

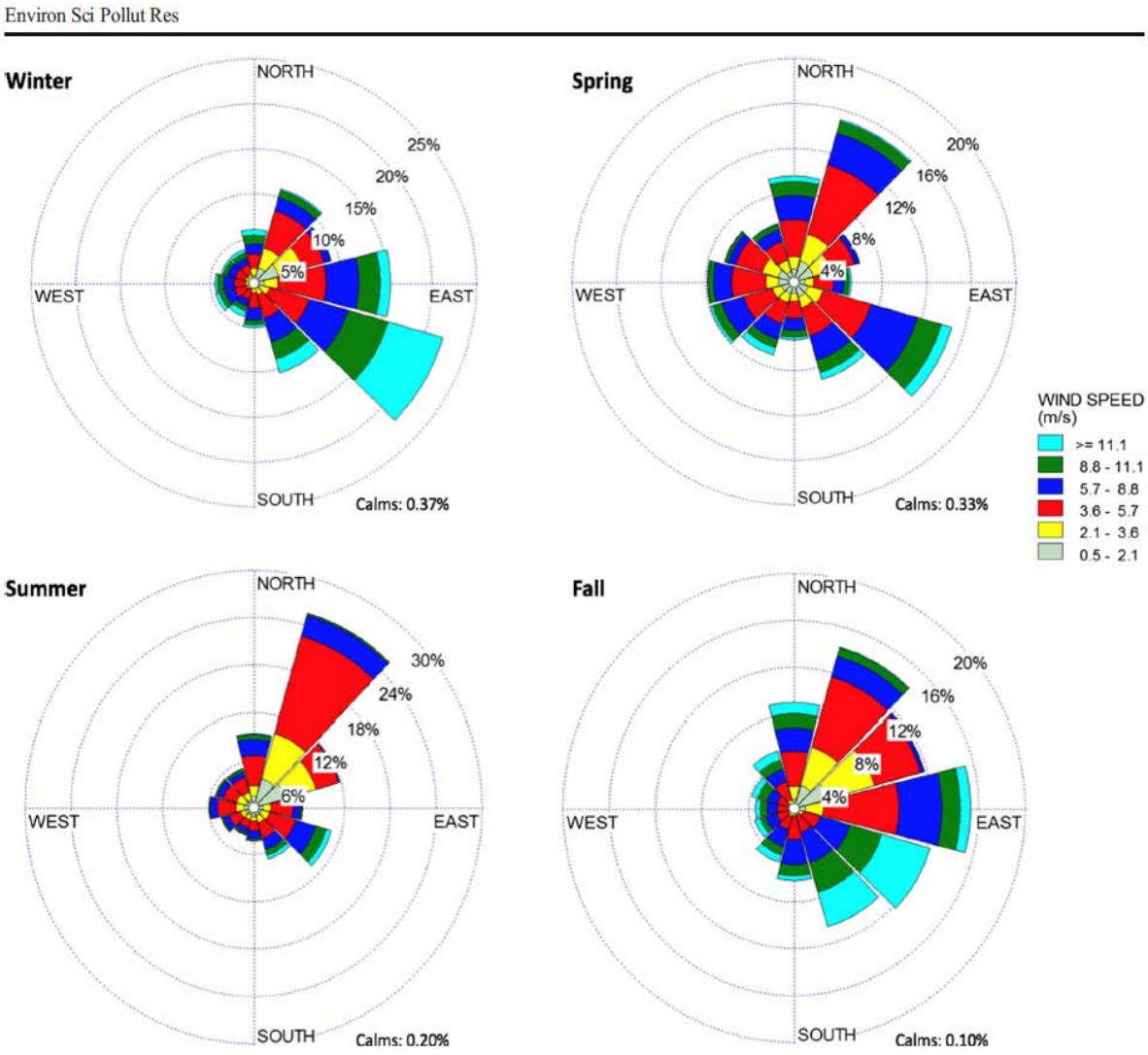


Fig. 2 Seasonal (2006–2013) wind rose simulations using WRPlot View™ (blowing to direction). Percentages represent frequency of wind direction

[6] Hoffman, E., Guernsey, J.R., Walker, T.R. et al., Pilot study investigating ambient air toxics emissions near a Canadian kraft pulp and paper facility in Pictou County, Nova Scotia, Environmental Science and Pollution Research (2017) 24: 20685. https://doi.org/10.1007/s11356-017-9719-5.

[7] Pilot study investigating ambient air toxics emissions, *supra*, at p. 20689, Figure 2.

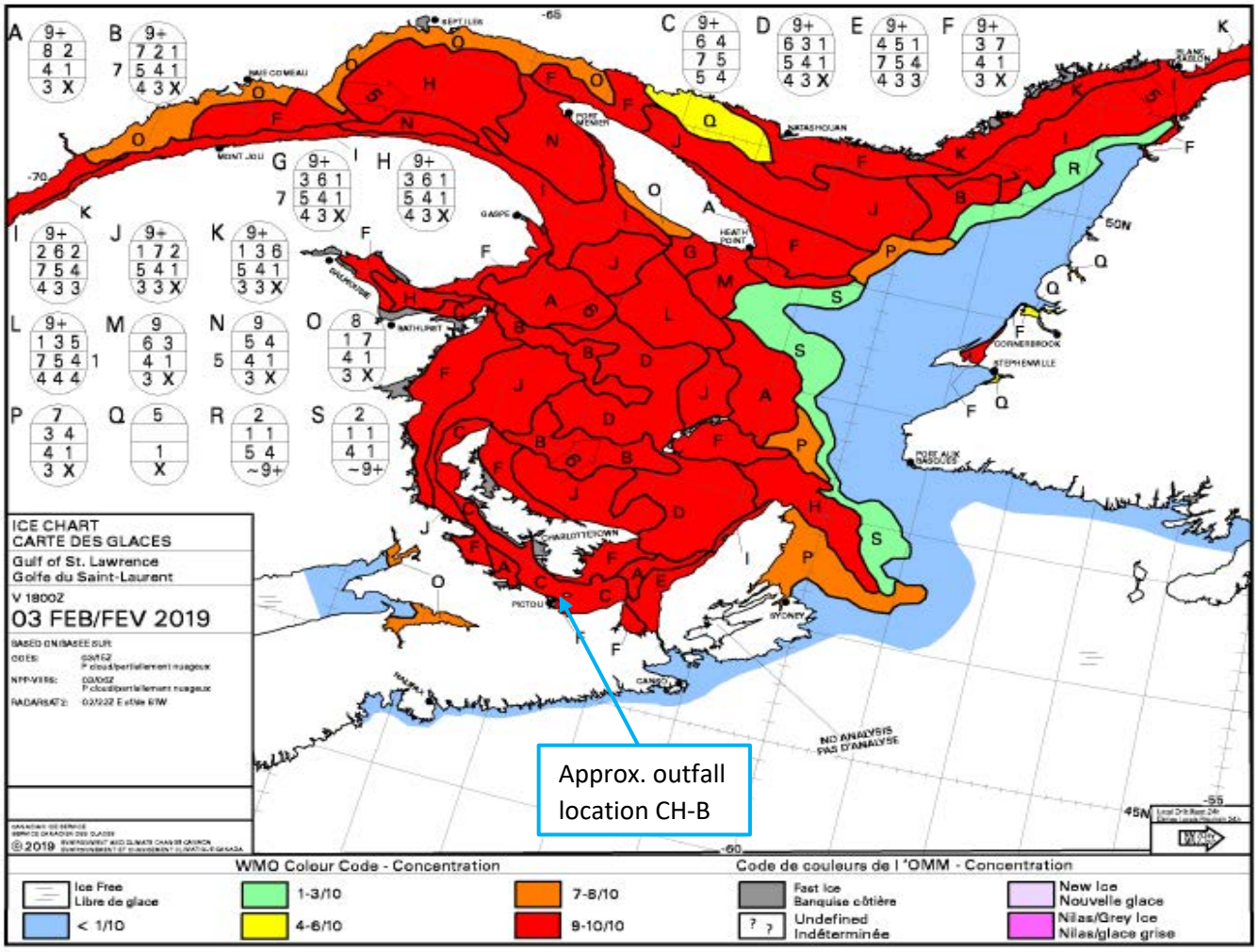
Conclusion

Mr. MacCarthy and Mr. Egilsson believe that much of the above information was not taken into account by Northern Pulp and its consultants in the process of developing its materials and providing information on the ability of the Caribou Channel and the Northumberland Strait to neutralize the impacts of a continuous effluent flow into that environment.

Contrary to the conclusions of Northern Pulp's consultants, the carrying capacity of the Strait, combined with the currents, overall and local, does not permit quick and complete dispersal of the constant flow of effluent and its constituents. The effluent will not simply vanish, but will circulate and build up within the system of local tides, currents and dynamics in the Strait, creating an unreasonable and serious risk of harm to fish and fish habitat.

Further, the pipeline and the diffusers are extremely vulnerable to damage from ice, storms and other forces, likely resulting in effluent discharge into Caribou Harbour, or other parts of the Caribou area. Such discharges could go undetected for weeks or months due to ice cover, and because there is no effective monitoring equipment installed on the pipeline and diffuser. Likewise, in the event of a rupture or leak, pipeline infrastructure repair would be extremely difficult or impossible prior to ice breakup in spring. All of these factors present serious individual and cumulative risks to the fishery and to the marine environment.

APPENDIX A


ICE CHART
CARTE DES GLACES
Gulf of St. Lawrence
Golfe du Saint-Laurent
V 1800Z
03 FEB/FEV 2019
© 2019
Approx. outfall location CH-B
NO ANALYSIS
PAS D'ANALYSE
WMO Colour Code - Concentration
Code de couleurs de l 'OMM - Concentration
Ice Free
Libre de glace
< 1/10
1-3/10
4-6/10
7-8/10
9-10/10
Fast Ice
Banquise côtière
Undefined
Indéterminée
New Ice
Nouvelle glace
Nilas/Grey Ice
Nilas/glace grise

APPENDIX B-2

March 5, 2019
Re: Northern Pulp Replacement Effluent Treatment Project

My name is Rob MacKay. I live in Central Caribou, NS. I am a master diver and have been for almost 3 decades. I have over 4000 dives in and around the Pictou County area. I have spent many years on the bottom in the Caribou area and I have seen a lot of changes made by ice each year.

Here are a few examples.

1. Gary, a fisher from Toney River, asked me to go and look for a couple of lobster traps he lost in 32 feet of water. I came across an ice scour on the bottom that was probably 4 feet deep and wide enough that you could fit a small car in. This was located just west of Toney River, in lobster bottom about ½ km from land. This would be about 10 miles from the proposed outfall location.

2. Another fisher from Toney River named Barry had a trawl hung up on bottom several years ago and he asked me to go look for it. It was tangled up in a buoy and chain from Prince Edward Island. The buoy had dragged from northern PEI. It weighed approximately 5000 pounds and had a 4000-pound stone mooring attached to it. We figured out where the buoy was from by prying a tag with serial number off the solar panel on the side of the buoy. It was a fairway buoy, one of the larger ones. This was found just west of the Ballast Island, Caribou Island, about 5 miles from the proposed outfall location, about ½ km from land.

3. In Caribou Harbour I used to help every spring installing the navigation buoys in the channel with Ed George and Paul Logan. Sometimes the ice would drag them for miles and it was all we could do to drag them back into place. Some of the large navigation buoys in Caribou are about 30" around and about 23 feet long. Buoys weigh from 150 to 5000 lbs. Buoy anchors are 2000-pound blocks with 500 pounds of chain on them depending on the depth. Larger buoys would have double the weight on them. DFO tend to use larger ones, such as the Gull Rock buoy and the last green Caribou buoy which is also a RACON buoy that shows up on radar as a Morse code letter. The navigation buoys are located in the channel in Caribou Harbour, parallel to the proposed pipe route.

The channel shifts from time to time mostly due to storms. Ice and tide also move sand around as it is very shallow in this area. Storms can pile ice up to 30 feet high which can dig deep into the soft bottom. This could damage the buried pipe.

If the pipe is covered in armour stone, the sand on either side will be undermined by wind and wave action exposing the pipe to the full force of the ice in winter. If no armour stone is used, those same fall storms could easily expose the pipe, as anyone living near a beach knows how easily sand is shifted by storm winds and waves. Either way the pipe is unlikely to survive extreme conditions in this area.

The armour stone proposed for this pipe would likely be moved around by the ice and could end up in the channel used by Northumberland Ferries which is barely deep enough to begin with.

The sea bottom in the area of the proposed pipe is very fragile. It's mostly sand and in the inner harbour, mud and eel grass. The eel grass is very fine and important to juveniles and larvae of lobster and crab.

I have personally witnessed the things that I have described above.

Robert L MacKay
711 Central Caribou Rd, NS
B0K1H0

APPENDIX B-3

March 4, 2019

Re: Northern Pulp Replacement Effluent Treatment Facility Environmental Assessment

My name is Barry Sutherland. I have been a fisherman for 27 years. I fish in and around the area of Caribou Harbour and have great concern over the possibility of an effluent pipe in this area.

Caribou Harbour is one of the largest rock crab nurseries in the Eastern Gulf. It contains millions of female and juvenile crab. I have fished rock crab for several years. I fish Hillsborough, PEI and from Wallace Harbour to Caribou Harbour in Nova Scotia. Female crab can be found in all the bays and inlets, but for some reason Caribou Harbour holds a far greater number of female and juvenile crab than all the other areas.

The survival of the lobster industry in this end of the Strait depends on the health of this rock crab nursery. Crab is the main food supply for lobster.

I am concerned about the potential impact of a pipe carrying and **continuously discharging up to 3 million litres an hour** of effluent through three diffusers, and the potential that this will produce noise and/or vibration that would disturb marine life.

I would like to draw the Minister's attention to the potential impact of noise and/or vibration from the diffusers on the marine species in Caribou Harbour and surrounding area. This issue has not been addressed in Northern Pulp's submission. I believe this issue must be fully addressed before any approval is given. Noise and vibration can carry long distances under water and change conditions which make a habitat favourable for many species.

I also believe that Northern Pulp's plan to excavate a trench through the centre of this very special eco-system may disrupt the fishery in this area.

The DFO description of Scallop Buffer Zone 21, 22 and 24, which runs 1 nautical mile from the nearest point of land and includes the entire proposed pipeline and outfall, states that the ecological components of interest protected by the buffer zone are" juvenile American lobster and habitat that is important to biodiversity" and states "specifically American lobster nursery habitat is important for the life-cycle of the species."

The description also states, "No other human activities that take place in this area are incompatible with the conservation of the ecological components of interest" and that "These closures offer protection to other important species and habitats in the southern Gulf of St. Lawrence." As an example, DFO refers to SFA 22 containing "rock crab (an important prey for several species and a commercial species." http://www.dfo-mpo.gc.ca/oceans/oeabcm-amcepz/refuges/sfa-zpp-eng.html

Caribou Harbour is home to the largest commercial fishing fleet in the Northumberland Strait. The strong lobster catches in this area are the result of the continuous food supply from the rock crab nursery. The potential destruction of this crab habitat will have devastating consequences on the lobster industry in this area. No studies have been done on the possible effect noise and/or vibration from this effluent pipe could have on local marine life. When foreign material is dumped into the environment, it will have an effect. Sometimes it takes years to realize how big that effect will be and then it is too late to fix it. Northern Pulp needs do something environmentally friendly, not something that is economically beneficial to themselves.

Sincerely,

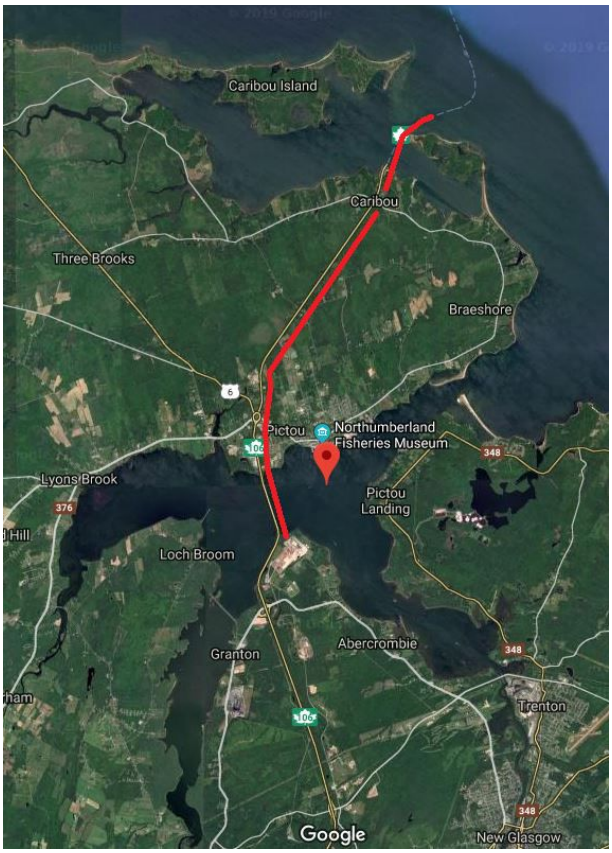
Barry Sutherland
40 Sutherlands Lane
RR2 Scotsburn, Pictou County
Nova Scotia B0K1R0
debbie_and_barry@hotmail.com

APPENDIX C-1

Northern Pulp's Effluent Disposal Plans - Issues and Answers

by Arthur A. MacKay
February 2019

BACKGROUND



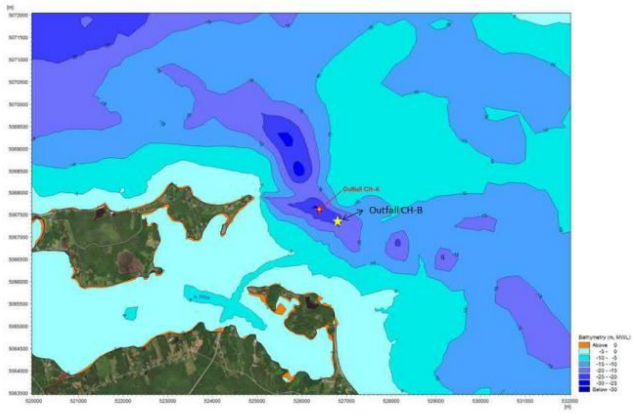


Figure 2.4 **Bathymetry and Outfall Locations CH-A and CH-B in Caribou Harbour Offshore**

Northern Pulp Nova Scotia Corporation (Northern Pulp) started operations in 1967 and is located in Pictou County, NS, on the shore of Pictou Harbour. The company states that it "*manufactures 280,000 tonnes of Kraft pulp annually, primarily for export*". It claims to have 300 employees and to generate $200 million annually into the Nova Scotia Economy.(1)

On February 7, 2019, Northern Pulp Nova Scotia Corporation (Northern Pulp) registered a project for environmental assessment. The purpose of the Project is to replace the existing effluent treatment facility (ETF) with a new one to treat wastewater received from the Northern Pulp pulp mill at Abercrombie Point, Pictou County.

Northern Pulp intends to use a biological activated sludge treatment process which combines moving bed biofilm reactor technology with conventional activated sludge. Once treated, effluent would be sent via an approximately 15.5 kilometers-long pipeline. The effluent pipeline would follow the Highway 106 for approximately 11.4 kilometers, then enter the marine environment near the Northumberland Ferries marine terminal, and continue for approximately 4.1 kilometers through Caribou Harbour to the Northumberland Strait where the treated effluent would be discharged via an engineered diffuser. (Modified from 3)

There are also plans to eliminate the existing treatment facility at Boat Harbour.

ADDENDUM RECEIVING WATER STUDY FOR NORTHERN PULP EFFLUENT TREATMENT FACILITY REPLACEMENT PROJECT – ADDITIONAL OUTFALL LOCATION CH-B, CARIBOU POINT, NOVA SCOTIA

Table 3.5 Water Quality at the End of the Mixing Zone for a Three-port Diffuser

Parameter	Unit	Effluent Daily Maximum Limit	CCME, Marine Guideline	Ambient Conditions	End of Mixing Zone at 100 m from Diffuser	Distance (m) from Diffuser Ambient Condition is Reached
Adsorbable Organic Halides (AOX)	mg/L	7.8	n/a	Trace amount	0.05	n/a
Total Nitrogen (TN)	mg/L	6.0	45[1]	0.24	0.24	< 2
Total Phosphorus (TP)	mg/L	1.5	n/a	0.35	0.35	< 2
Colour	TCU	750	n/a	10.8	10.8	< 5
Chemical Oxygen Demand (COD)	mg/L	725	n/a	n/a	5.0	n/a
Biochemical Oxygen Demand (BOD_5)	mg/L	48	n/a	n/a	0.33	n/a
Total Suspended Solids (TSS)	mg/L	48	Narrative [2]	8.5	8.5	< 2
Dissolved Oxygen	mg/L	> 1.5	>8	7.2	7.2	< 2
pH	-	7.0 - 8.5	7.0 - 8.7	8.0	8.0	< 2
Temperature (summer)	°C	37	Narrative [3]	17.6	17.7	< 2
Temperature (winter)	°C	25	Narrative [3]	0	<0.1	< 2
Salinity	g/L	4	Narrative [4]	28	28	< 2

n/a – not available
[1] - CCME marine limit for NO_3^- as N
[2] - Maximum average increase of 5 mg/L from background levels for longer-term exposures (e.g., inputs lasting between 24 h and 30 d)
[3] - Human activities should not cause changes in ambient temperature of marine and estuarine water to exceed ±1°C at any time, location, or depth
[4] - Human activities should not cause the salinity (parts per thousand [‰], expressed here in g/L) of marine and estuarine waters to fluctuate by more than 10% of the natural level expected at that time and depth.

This figure shows the estimated water quality for some common parameters such as halides, nitrogen, phosphates, oxygen demand, dissolved oxygen, pH, temperature and salinity at the outfall and at varying distances away.

The purpose is to show that dilution occurs and as a result, all is well. However, except for Board Harbour data, I can find no definitive statement about the toxic chemicals that are of real concern in the Strait and in Caribou Harbour. This includes, among other things, cadmium, dioxins, furans, mercury, polycyclic aromatic hydrocarbons (PAHs), petroleum hydrocarbons, zinc, etc. (8)

How toxic are these chemicals? You can check each of these at:

http://scorecard.goodguide.com/chemical-profiles/ which, for each chemical, provides information on Human Health Hazards, Hazard Rankings, Chemical Use Profile, Chemicals Rank in the United States, Regulatory Coverage, Basic Testing to Identify Chemical Hazards, Information Needed for Safety Assessment, and Links to associated information.

ASSESSMENT:

The “Replacement Effluent Treatment Facility Project” (11) is a massive document with 15 large sections and numerous appendices. “Section 8 - Environmental Effects Assessment” alone contains 356 pages that cover the following topics: Atmospheric Environment, Acoustic environment, Soils and Geology, Surface Water, Groundwater, Freshwater and Fish Habitat, Wetlands, Flora/Floral Priority Species, Terrestrial Wildlife/Priority Species, Migratory Birds and Priority Bird Species/Habitat, Harbour Physical Environment, Water Quality, and Sediment Quality, Marine Fish and Fish Habitat, Marine Mammals, Sea Turtles and Marine Birds, Socio-Economic Environment, Indigenous Peoples Use of Land and Resources, Marine Archaeological Resources, Terrestrial Heritage Resources, Effects of the Environment on the Project.

While the work itself is impressive and one would expect that it does impress in most circles, it has serious flaws which limit its short-term and long-term value in ensuring the ecosystem health of Caribou Harbour and Northumberland Strait. Unfortunately, it seems that few if any primary surveys have been done to determine the vital ecosystem components of the target areas apart from the engineering, construction surveys. As far as the biological components of the area goes, much of the material quoted is obtained from other studies of various purpose and extent, from opinions expressed and from limited surveys undertaken many years ago (plankton results from 1992 are used, for example).

In addition, only “Valued Environmental Components” and “Priority Species” are considered. Thus, only species that are deemed to be important such as commercial fish, are considered in the report. As a consequence, the foundational species of the ecosystem such as planktonic species, invertebrate and fish larvae, subtidal and intertidal invertebrates and plants, forage species, etc are not considered. Seasonality is an important issue and to truly understand ecosystem dynamics, at least 12 monthly surveys must be undertaken that include records for plankton, fish and invertebrate larvae, forage species, fish, bird, and mammals.

Also, baseline chemical analyses are required for water and bottom samples at predetermined sites which can be revisited for ongoing monitoring purposes in future years. The following figure is an example of how these sites should be selected to adequately cover the area under question. The white lines are transects used to survey marine organisms and bottom type. The red dots are sediment samples. For details on how sampling may be carried out see Chapter 2 at https://issuu.com/artmackay/docs/healthofstcroixestuary

In the absence of this information, the impact of the effluent from the proposed outfall pipe at Caribou Harbour or the proposed cleanup at Boat Harbour cannot be measured in the short term or long term. More particularly, the “out-of-sight-out-of-mind” pipeline will be unlikely to foster any response to future

deleterious impacts in Caribou Harbour and the Northumberland Strait … or for that matter any improvements that might be made in Pictou Harbour.

While there seems to be an abundance of engineering studies, fishermen and other knowledgeable individuals have provided some descriptive information on the abundant life in Caribou Harbour and the Northumberland Strait, I have found no reference to professional biological surveys in Caribou Harbour, Pictou Harbour or the adjacent section of the Northumberland Strait.(12)

Many of the issues associated with the outfall proposed for Caribou Harbour are outlined in a letter to NS Advocate by John Collins (6) and others which can be summarized as follows:

1. The one ton of solids that will discharge daily with the millions of liters of effluent are still scientifically considered toxic waste.
2. The composition of toxic wastes is, at least in part, known.
3. Enormous amounts of water are required to dilute the effluent to permissible concentrations allowable in the receiving waters.
4. The average effluent temperature released in the summer will be 37 degrees C when the Straits ambient temperatures are already nearing 20 C and lobster larvae are present and may be impacted by this temperature difference.Other plankton and larvae will also be impacted by temperature and pressure changes.
5. Lobster larvae survival diminishes rapidly when the water temperature surpasses 20º C and is already a major concern without the addition of the millions of liters of hot effluent.
6. According to Northern Pulp, the effluent temperature and salinity will meet that of the ambient surrounding water within 100 meters of the discharge point. However, some calculations suggest the proposed system will impact 65,000 square meters of area (See John Collins 5).
7. The proposed discharge location is a narrow channel, just outside the mouth of Caribou Harbour and just alongside the ferry channel which is dredged regularly.

It appears that no long term and adequate surveys have been done for Pictou Harbour. However, such surveys have been done for the St. Croix River and Estuary on the border between Maine and New Brunswick (See: https://issuu.com/artmackay/docs/healthofstcroixestuary), Saint John Harbour, and elsewhere and these surveys suggest that similar conditions can be found in Pictou Harbour and in the adjacent Strait area. Since effluent will be introduced continuously into the Strait at levels substantially elevated from those coming out of Boat Harbour into the Strait, the impacts can be expected to be substantially higher on a day-by-day basis. While Caribou Harbour will not be subjected to the same direct pollution, the incursion of seawater containing effluent from the outfall should be expected to create conditions similar to those that occur in Pictou Harbour, albeit over a longer period of time. Impacts that can be expected include but are not limited to:

1. Caribou Harbour - Tidal incursions of effluent can be expected from the outfall in the strait as well as from potential leaks and accidents. As a consequence effluent impacts will occur in Caribou Harbour estuary which has been identified as a small, shallow harbour “rich with fish of all sorts, including lobster, crab, mussels, clams, mackerel, etc.”(5)
2. Toxic Chemicals can be expected to accumulate in the sediments of Caribou Harbour and the Northumberland Strait at, as yet, unknown locations.

3. As in the St. Croix, biological magnification of toxins will occur in the Harbour and in Northumberland Strait. A broad range of marine organisms including plankton, fish larvae, fish, birds, marine mammals and humans will be impacted directly or indirectly at lethal and sublethal levels. While these impacts will be observable in Caribou Harbour in a relatively short time, they will only be observable in the strait after more time has passed. This is partly why annual long term monitoring is required.
4. As in the St. Croix, toxic chemicals and physical alterations (pH, temperature, residual solids, etc.) will impose negative impacts on life forms, particularly at the planktonic level and will foster impacts on organisms up the food chain.
5. As in the St. Croix, over time anoxic “Dead Zones” should be anticipated.
6. As in the St. Croix, decline in marine invertebrates and fish as well as some birds and mammals can be anticipated. Those that remain are likely to harbour toxic chemicals.
7. As in the St. Croix, negative impacts on the local fisheries should be anticipated with consequence loss of income.
8. As in the St. Croix, some fisheries may be closed due to the presence of toxic chemicals in fish caught for human consumption.
9. As in the St. Croix, the impacts in the marine system are shared by multiple jurisdictions - Nova Scotia, Prince Edward Island, New Brunswick and the Federal government of Canada. All of these jurisdictions must be included in the crafting of studies and the decisions made to proceed with this proposal for an outfall off Caribou Harbour.
10. As in the St. Croix, the proposed outfall may impact several coastal parks and tourist destinations as well as human properties along all of the shores.

RECOMMENDATIONS:

The problems associated with the outfall proposed for Caribou Harbour are outlined and well defined by local knowledge. However, the knowledge base seems to be largely related to economically valuable and obvious species only and a literature search has not found any extensive science-based, ecosystem surveys and reports. Unless appropriate studies are undertaken, likely impacts are hard if not impossible to assess and cannot be tracked over the long term.

Effluent must be tested in the laboratory for lethal and sub-lethal impacts by an independent laboratory using appropriate local freshwater and marine species from all target areas including Caribou Harbour and the rivers flowing into it.

Frankly, in relation to the proposed pipeline, no work should begin until professional ecosystem surveys are undertaken at Caribou Harbour, Northumberland Strait at Caribou Harbour and Northumberland Strait at the Boat Harbour outfall (vital for comparison purposes). In the absence of these necessary surveys, the Minister must be made aware that there can be no confidence in the purported lack of impacts stated and implied in the Northern Pulp environmental submission. Additionally, the Minister will be remiss in supplying necessary data that will be required by subsequent Ministers to make essential decisions related to future environmental impacts in Caribou Harbour and Northumberland Strait.

Make no mistake, adequate surveys take time and requires highly qualified and experienced field staff. While running sufficient transects and collecting sufficient samples is time consuming, seasonal variations in abundance and distribution of marine organisms dictates designing a sampling regime that takes this into account. As a consequence, a full year at least should be designated for the work. Proposals must include an appropriate ongoing annual monitoring regime.

THE AUTHOR

As CEO and owner of research and consulting company Marine Research Associates Ltd, Art MacKay has had over 50 years of professional experience studying the marine environment of the east coast. In relation to this presentation he has several decades of field surveys and research relating to the pulp mill pollution in the St. Croix River and Estuary in New Brunswick. Other applicable experience includes studies on effluent and ecosystem impacts of the Point Lepreau Nuclear Generating Station, The Coleson Cove Generating Station, Saint John Harbour, coastal quarries in New Brunswick and Nova Scotia as well as mill impacts on the Humber River in Corner Brook Newfoundland. Additional information can be found at: https://1drv.ms/b/s!AlJhIkiSGh1knL5BB8Fyl05bnPdYJQ

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[9] "The St. Croix Estuary 1604 - 2004 by Art MacKay - issuu." https://issuu.com/artmackay/docs/healthofstcroixestuary. Accessed 23 Feb. 2019.
[10] "How a pulp mill killed commercial fisheries in New Brunswick's St ...." 9 Jan. 2019, https://nsadvocate.org/2019/01/09/how-a-pulp-mill-killed-commercial-fisheries-in-new-brunswicks-st-croix-estuary-and-its-lessons-for-the-northumberland-strait/. Accessed 24 Feb. 2019.
[11] "Replacement Effluent Treatment Facility Project | Environmental ...." 7 Feb. 2019, https://novascotia.ca/nse/ea/Replacement_Effluent_Treatment_Facility_Project/. Accessed 24 Feb. 2019.
[12] "Northumberland Strait Ecosystem Overview Report Moncton, New ...." http://docplayer.net/33332787-Northumberland-strait-ecosystem-overview-report-moncton-new-brunswick-final-report.html. Accessed 28 Feb. 2019.

APPENDIX C-2

Art MacKay

SUMMARY

Art received his BSc in Biology at the UNB in 1961 and carried out graduate studies at McGill University in Montreal, Quebec for 3 years. He taught at McGill, the U of Victoria, and UNB before establishing a biological consulting business in N.B. in 1964 and aquaculture companies at Deer Island, N.B. and Eastport, Maine in 1979 and 1980, respectively. From 1964 to 1987, principal work included collection and supply of living marine organisms for schools, universities and research establishments around the world, including Case Western Reserve University, the University of Maryland, Woods Hole Oceanographic Institute, National Institutes of Health, Technion in Israel, New Brunswick Research and Productivity Council and many others. Consulting activities included the identification of aquatic and marine inventories, environmental monitoring and mitigation for nuclear power plants, oil refineries, gas pipelines and other industrial developments. Art is credited with establishing the first successful commercial Atlantic salmon sea farms in both NB and ME. He has worked in the United States, Canada, Norway, Thailand, and India.

He has had continuous involvement with education on various levels; has taught junior high, high school and university levels and has presented lectures and field courses to many private and public organizations, including the UNB, U of Toronto, U of Waterloo, U of Western Ontario, U of Guelph, Huntsman Marine Science Centre, New Brunswick Community College, and others. He has lectured on leadership in the Canadian Navy and has made many presentations on behalf of the aquaculture industry and conservation efforts. He was Education Director at the Atlantic Salmon Federation for 3 years.

Art has a background in publishing, printing, graphic design and display construction. He is an artist and commercial illustrator and has sold numerous paintings and illustrations.

Specialties: Fisheries, aquaculture, environment, marine biology, business development.

APPENDIX D-1

DALHOUSIE UNIVERSITY
FACULTY OF SCIENCE

March 5, 2019

James Gunvaldsen Klaassen
Barrister & Solicitor | Ecojustice
520-1801 Hollis Street, Halifax, NS B3J 3N4

Dear Mr. Gunvaldsen Klaassen,

Please find below comments on the document "Replacement Effluent Treatment Facility Project," prepared by Dillon Consulting for North Pulp Nova Scotia Corporation, and submitted for environmental assessment, in accordance with Part IV of the Environment Act.

My comments are based on my training, research and experience as an Associate Professor, Endowed Chair in Sustainability and Environmental Health, Director of the Environmental Science program, and as a Senior Research Scholar with the Healthy Populations Institute at Dalhousie University in the fields of environmental epidemiology and health geography. I hold a Ph.D. in Population Health Science from the University of Ottawa and have conducted research in the area of air pollution epidemiology for more than a decade. I have also participated as a Co-investigator on a Canadian Institutes of Health Research-funded project to evaluate the health and environmental conditions of residents in Pictou Landing First Nation (PLFN) which employed a two-eyed seeing approach to evaluate environmental exposure and community health status in the community.

Given my background the following comments explore the potential for health-related impacts arising from the project and ongoing operation of the pulp mill, and form a response to the following questions:

1. Please identify the portions of the materials filed by Northern Pulp as part of its registration package on which you are qualified to comment based on your expertise.

I have reviewed specific sections of the Registration Document as they relate to my expertise, including Sections 1-7, Section 8.1 on air emissions, Section 8.2 on noise, and Sections 9.1, 9.2.1, and 9.2.4.1 on health effects.

2. Within the portions of the materials identified in your response to question 1, what is your opinion with respect to the major findings and conclusions expressed in the materials?

Section 6.7, Page 117: Concerns by community members living in Pictou Landing First Nation (PLFN) and others were raised about perceived high incidence of cancers. The use of the 2018 NSCCP report (Saint-Jacques et al., 2018) is not an adequate response to this concern for several reasons. The report estimates cancer incidence for the county (Pictou) and not for sub-regions within the county or for populations surrounding the mill. In addition, the report does not provide detailed reporting of specific cancers that could be linked to ingestion of weakly-chlorinated compounds, such as bladder, pancreatic and other cancer sites, via consumption of marine or terrestrial food sources, or the inhalation of fine particles from stack emissions.

Saint-Jacques N, Dewar R, Nauta L, MacIntyre M. 2018. Nova Scotia Cancer Incidence and Survival. Statistics Update: Focusing on 2011-15. Registry & Analytics, Nova Scotia Cancer Care Program, Nova Scotia Health Authority.

Section 9.1: The Registration Document identifies two main sources of emissions or discharges that may result in potential human exposure to toxins, including air emissions from the replacement ETF and existing mill and the marine treated effluent diffuser. The evaluation does not include the estimation of human health impacts, even if low risk, arising from contamination of ground and well water supplies due to leakage from the effluent pipe from mechanical or connection failures at drain valve locations.

Regarding effluent characteristics the report claims that the actual chemical composition, including specific substances in the treated effluent and their associated concentration, are not known with certainty until operations begin. However, it is well within the capacity of the proponent to develop emission/effluent models with varying degrees of uncertainty associated with chemical composition. What is known is that the effluent will contain low but detectable levels of chlorinated by-products, some of which are high toxic, mutagenic and that can bioaccumulate even in small amounts.

The registration document refers to a toxicological report authored by scientists with Toxikos (2006) that reported negligible risks to human health from consumption of marine-based food sources exposed to effluent discharge near the diffuser. However, the report does not include assessment of chlorinated aliphatic compounds which may still be present at low concentrations even under modern effluent treatment conditions. The reason provided for this omission is the notion that modern kraft pulp production uses chlorine dioxide as a bleaching agent which results in significant reductions in chlorinated residual compounds in effluent.

However, although concentrations of chlorinated compounds have decreased significantly from levels generated by mills employing elemental chlorine as a bleaching agent, they have not been eliminated by the use of chlorine dioxide (Bajpai, 2015).

Bajpai P. Green Chemistry and Sustainability in Pulp and Paper Industry. Switzerland: Springer International Publishing, 2015.

Toxikos. 2006. Human Health Risk Assessment – Bell Bay Pulp Mill Effluent. Prepared for: Gunns Ltd. Prepared by: Toxikos Pty Ltd., Toxicology Consultants. TR081205-RJF. July, 2006.

Section 9.2.1: The registration document identifies three exposure pathways, including inhalation, ingestion and dermal absorption. However, it is now well recognized, supported by a substantive evidence base, that exposure to harmful or beneficial characteristics of the environment may also occur through visual and aural routes leading to physiological impacts, eg. via damage to hearing, or the development of psychological impacts leading to increased stress response and mental health challenges. The development of (perceived) community illness (e.g. cancer incidence) in PLFN offers an interesting case example of how contamination of the environment, particularly environments with long-standing valued ecosystem components (VECs), e.g. hunting, fishing, recreational and spiritual values, leads to a severing of community connections to the surrounding environment and a real or perceived impact of contamination on health in the community. Additional impacts may also arise in the broader community, particularly among the fishing sector which may experience challenges with the sale of products sourced from locations near the effluent diffuser.

Section 9.2.4.1 : The registration document outlines the identification of Chemicals of Potential Concern (COPC) in air emissions associated with different aspects of the project. Specifically, the report evaluates emissions regulated in Nova Scotia under current air quality regulations and existing approvals for the emissions of fine particles. These pollutants include, and are limited to, carbon monoxide (CO), hydrogen sulphide (H_2S), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), total suspended particulate matter (TSP), and fine particulate matter ($PM_{2.5}$). According to the report, ambient air monitoring data for the last three years show no exceedances when assessed against current Nova Scotia Air Quality Regulations, other than the occasional instances of excess H_2S releases. However, the report provides no data or discussion with reference to speciation of particulates, specifically fine particles ($PM_{2.5}$) which may have significant health impacts at any level above background.

Much of the epidemiological literature on the association between fine particles and human health impacts employs measurements of particle mass of a specific aerodiameter as a fraction of air volume, and as a function of exposure. However, not all particles and their associated chemical composition are created equally; those arising from emissions associated with the creation of kraft pulp will have chemical signatures and composition that differs from other sources. Typical *speciation methods* will enable detection of particle- and gas-phase organic and carbonyl compounds, metals, as well as determination of air toxics typical to emissions from pulp and paper operations using recovery boilers. Although there are several sources of air emissions, including fine particle emissions, within the study area, speciation of fine particles would provide data on the fraction of $PM_{2.5}$ associated with mill operations and the potential for human exposure to air toxics characteristic to kraft pulp operations.

Section 9.2.4.2: In addition to air emissions, human exposures to mill emissions are possible via ingestion of marine organisms exposed to substances released at the location of the effluent diffuser (Page 495). Of greatest concern would be chlorinated COPCs such as chlorinated aliphatic hydrocarbons, chlorinated organics and metals such as cadmium and mercury. In addition to chlorinated compounds, the effluent is likely to contain endocrine disrupting compounds such as androstenediones and other substances with androgenic or estrogenic properties. These substances may interfere with endocrine balance critical to fish or reproduction of other marine organisms, thus affecting associated populations relevant to commercial and recreational fishing operations. The proposed Environmental Assessment follow-up and performance monitoring program should include monitoring programs of marine species relevant to human exposure pathways.

3. What is your view as to the reliability of the underlying research conducted and relied upon in reaching the findings and conclusions? What if any information gaps or data gaps exist within the materials? If so, what impacts, if any, do those gaps have on the data and on the reliability of the conclusions that can be drawn, or are drawn, from that data?

I am confident in the reliability of the underlying research conducted and relied upon in supporting the findings and conclusion presented in sections of the Registration Document reviewed for the purposes of developing the comments in this letter. However, there are instances of statements and conclusions made in the document that are not well supported by the evidence referenced.

For example, reference to provincial cancer statistics released at the county level do not provide evidence of no negative effect on human health associated with exposures to mill effluent among residents of PLFN (Page 117).

In another example, the document relies heavily on analysis (Toxikos, 2006) of effluent from a proposed mill. In other words, the true chemical composition of the effluent is unknown and toxicological analysis was based on sample effluent taken from operating mills of similar design at dilutions equal to or less than 10% which does not account for potential diffusion problems.

In terms of routes of human exposure, the registration document omits research on the psychological impacts of the project, limiting exposures to traditional routes such as inhalation, ingestion and dermal absorption. The notion of releasing pollution (formally identified as effluent) into the local environment will invariably increase stress levels in the communities affected, regardless of efforts made to dilute the pollution in the receiving media. The result of the omission is an inability to recognize and assess health effects of stress associated with perceived contamination of the local environment and potential impacts to local livelihoods.

The registration document also omits reference to the practice of speciation as a legitimate approach to the identification of fine particulate pollution origin. Although measured concentrations of fine respirable particulate pollution are within guideline values it should also be recognized that: 1) there is no safe level of exposure to fine particulate pollution, and 2) the chemical composition of particles varies with source so that the toxicity of particulate pollution will also vary with the source. The omission of any reference to speciation limits the reliability of the conclusion of no significant impact arising from the proposed project or continuing mill operations.

4. What significant assumptions are relied upon in the materials filed by Northern Pulp, and what is your view as to the reliability of each such assumption? What is the impact of each assumption on the findings contained in the Northern Pulp materials?

There are several significant assumptions contained with the materials filed by Northern Pulp.

Assumption 1: There is no evidence of increased cancer risk associated with mill operations, either historic or proposed modifications to these operations. The materials do not provide any evidence in support of this assumption other than a provincial report of cancer prevalence for the county as a whole. The use of this report would support the assumption that there is no variation in cancer prevalence within the county.

Assumption 2: The analysis and conclusions reached as part of a toxicological analysis of potential effluent (not actual effluent) from a proposed mill in another location are applicable to the proposed project. While the science underlying the analysis is likely to be sound, the conclusions may not be as reliable as portrayed in the registration document.

Assumption 3: The identification of human exposures assumes no impact on health associated with perceived risk of pollution discharge, regardless of dilution, in areas of concern including local wetlands, groundwater recharge zones, and in active fishing grounds. The conclusion of no significant impact on health is potentially erroneous in this context.

Assumption 4: Current air emissions from the mill are well within provincial and federal air quality guidelines and thus there are no potential health effects from air pollution exposures. This assumption does not acknowledge the fact that there are no "safe" concentrations of air pollution, particularly for particulate air pollution which has been shown to have health effects even at low concentrations (Cohen et al., 2017; ECCC, 2017). Moreover, the materials assume that particulate air pollution, regardless of source, will have similar health impacts. However, without conducting speciation analysis on particles collected in receptor communities, the impact of mill emissions cannot be discounted by concluding that there are a variety of air pollution sources within the vicinity of local populations (e.g., power plant, tire plant, vehicular emissions, fugitive dusts, etc.). The conclusion of no significant impact on health may not be accurate.

Cohen et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. Lancet 2017;389:1907-18.

http://ec.gc.ca/indicateurs-indicators/default.asp?lang=en&n=CB7B92BA-1&pedisable=true. Accessed: February 22, 2019.

5. Within the portions of the materials identified in your response to question 1, are there any [findings/issues/etc] on which you are unable to express an opinion? If so, why are you unable to express an opinion? What, if anything, would enable you to express such an opinion?

There are no portions in the sections identified in my response on which I was unable to express an opinion.

Thank you for the opportunity to provide comments on the environmental assessment materials submitted by Northern Pulp.

Sincerely,

Daniel Rainham, Ph.D.
Director, Environmental Science
Senior Research Scholar, Institute of Population Health

APPENDIX D-2

DANIEL G. C. RAINHAM, PH.D.

CURRICULUM VITAE

TABLE OF CONTENTS

Daniel Rainham, Ph.D.

PERSONAL:

Citizenship:	Canadian, British
D.O.B.:	January 24, 1971
Professional Address:	Faculty of Science, Dalhousie University Life Sciences Centre, Rm. 820 P.O. Box 15000 1355 Oxford Street, Halifax, Nova Scotia, B3H 4R2, CANADA Office: +1 (902) 494-1286 Fax: +1 (902) 494-1123 E-mail: daniel.rainham@dal.ca
Internet Addresses:	Elizabeth May Chair in Sustainability and Environmental Health *emaychair.dal.ca* Research Site *danielrainham.ca*
Professional Affiliations:	Canadian Public Health Association International Society for Children's Health and the Environment International Society for Environmental Epidemiology American/Canadian Association of Geographers Children and Nature Network Geomatics Association of Nova Scotia

EDUCATION:

2003-2009 (Ph.D.)	University of Ottawa, Ottawa, Ontario, Canada Major: Population Health Science; Specialization: Health & Place Supervisors: Daniel Krewski, Ph.D. and Ian McDowell, Ph.D.
1998-2000 (M.Sc.)	University of Alberta, Edmonton, Alberta, Canada Major: Atmospheric Sciences; Specialization: Medical Geography Supervisor: Karen Tomic, Ph.D.
1994-1998 (B.E.S. hon.)	University of Waterloo, Waterloo, Ontario, Canada Major: Environment & Resource Studies; Minor: Music Supervisor: Paul Kay, Ph.D.

OVERVIEW OF RESEARCH PROGRAM

I am an Associate Professor in Environmental Science and Senior Research Scholar with the Healthy Populations Institute at Dalhousie University. My research is focused at the nexus of population health science, environmental epidemiology and health geography. Specifically, I investigate people-environment interactions and how these interactions affect health and well being. These efforts are supported by innovations in wearable sensors and spatial analytics, and have most recently been applied to patient management strategies, physical activity interventions and empirical research on the role of nature contact in supporting healthy behaviours.

PROFESSIONAL EXPERIENCE

Position	Organization	Department	Start Date (MM/YR)	End Date (MM/YR)
I. Current Appointments				
Director	Dalhousie University	Environmental Science	07/2015	-
Associate Professor (Chair in Sustainability and Environmental Health)	Dalhousie University	Environmental Science	07/2014	-
Assistant Professor (Chair in Sustainability and Environmental Health)	Dalhousie University	Environmental Science	01/2009	06/2014
Assistant Professor – Jointly Appointed	Dalhousie University	College of Sustainability	09/2009	07/2015
Assistant Professor – Cross Appointed	Dalhousie University	School of Planning	09/2010	-
Assistant Professor – Cross Appointed	Dalhousie University	Earth Sciences	09/2010	-
Assistant Professor – Cross Appointed	Dalhousie University	Community Health and Epidemiology	09/2012	-
Assistant Professor – Cross Appointed	Dalhousie University	Health and Human Performance	09/2012	-
Assistant Professor – Cross Appointed	Dalhousie University	School for Resource and Environmental Studies	09/2012	-

II. *Previous Appointments*

Research Associate	University of Ottawa	McLaughlin Centre for Population Health Risk Assessment	09/2004	12/2008
Lecturer	Dalhousie University	Health and Human Performance	09/2001	12/2002
Instructor	MacEwan University	Faculty of Arts and Science	09/1998	12/1999

AWARDS AND HONOURS

Award	***Institution***	***Amount***	***Start Date (MM/YR)***	***End Date (MM/YR)***
Elizabeth May Chair in Sustainability and Environmental Health	Dalhousie University	Endowment (Approx. $2.3 million)	01/2009	-
CIHR Doctoral Research Award	University of Ottawa	$19,900/annum	04/2004	04/2007
Doctoral Admission Scholarship	University of Ottawa	$11,000/annum	09/2004	09/2008
Research Development Award	Environment Canada	$14,000	09/1999	05/2000
Alberta Graduate Scholarship	University of Alberta	$9,000	09/1999	09/2000
J. Gordin Kaplan Graduate Award	University of Alberta	$1,000	09/1998	09/1999
Senate Scholarship	University of Waterloo	$500	01/1996	04/1996

RESEARCH SUPPORT

Research Grants and Contracts (funded)

Date	Agency (Role)	Title	Amount
2019-2022	Canadian Institutes of Health Research Villeneuve P, Crouse D, Dales R, Griffith L, Hystad P, Raina P, **Rainham DG**, Ross N.	Does residential proximity to natural environments impact the mental health and well-being of participants of the Canadian Longitudinal Study on Aging?	$191,251
2019-2020	Canadian Institutes of Health Research **Rainham DG**, Crouse D, Gilliland J, Villeneuve P, van den Bosch M, Collyer C, Duinker P.	Optimizing Health and Sustainability Interventions on Schoolgrounds (OHASIS): Increasing Green Infrastructure for Healthy Cities	$100,000
2019-2020	Nova Scotia Health Research Foundation Stone M, Joshi N, Miller L, **Rainham DG**, Cawley J, Rehmen L, Turner J, Kolen A, Kirk S, McIsaac J-L.	#PLEYRocks: A knowledge sharing strategy to improve children's physical literacy through quality outdoor play in Nova Scotia early years settings	$10,000
2018-2021	Nova Scotia Health Research Foundation St. Jacques N, Terashima M, Dummer T, **Rainham DG**, Brown P, Purcell J.	Nova Scotia Community Cancer Matrix	$107,661
2018	Canadian Institutes of Health Research **Rainham DG**, Crouse D (Co-PI), Collyer C, Gilliland J, Katapally T, Villeneuve P, van den Bosch M.	A Nature-based Solutions Research Incubator (NBSRI) for Healthy Cities	$10,000
2018	Canadian Institutes of Health Research Katapally T, Osgood N, **Rainham DG**	A System Sciences Approach to Building Healthy, Equitable, and Smart Cities: a citizen science-driven population health intervention	$10,000
2018	Canadian Institutes of Health Research Kestens Y (PI), Fuller D, **Rainham DG (Co-Inv)**, Widener M, Winters M.	INTervention Research Across Cities Team	$10,000

Date	Agency (Role)	Title	Amount
2017-2018	Canadian Institutes of Health Research **Rainham DG (PI)**, Fuller D, Gilliland J, Kestens Y, Shooshtari M.	Location Sensor Analysis Platform (LSAP)	$53,750
2017	Canadian Institutes of Health Research, Public Health Agency of Canada, MaRS Discovery **Rainham DG (PI)**, Buckeridge D, Doherty S, Stewart B.	National Dashboard of PASS Health Behaviours	$10,000
2017-2020	Nova Scotia Health Research Foundation Terashima M, **Rainham DG (Co-Inv)**, Kirk S, Kephart G, Habib M.	Spatial Accessibility to Four Services and their Influence on Health Inequality	$147,792
2016 – 2021	Canadian Institutes of Health Research Brook J, Stieb D, Awadalla P, Brauer M, Hu H, Mcgrail K, Subbarao P (PIs), Pantelimon O, Atkinson S, Azad M, Carlsten C, Chaumont D, Copes R, Davies H, Demers P, Dunn J, Evans G, Feddema J, Habib M, Hakami A, Hatzopoulou M, Henry D, Johnson M, Lou W-Y, Mclaughlin J, **Rainham D (Co-Inv)**, Ross N, Smargiassi A, Takaro T, van den Bosch M, Villeneuve P.	CANadian Urban Environmental (CANUE) Health Research Consortium	$4,165,000
2016 – 2018	Saskatchewan Health Research Foundation Katapally T, Tremblay M, Larouche R, Osgood N, Longo J, **Rainham DG**, Leatherdale S, Ferguson L.	Smart Active Living Policy	$118,500
2016 - 2017	Canadian Institutes of Health Research Villeneuve P (PI), Brauer M, Hakami A, Weichenthal S, Davies H, Kennedy J, Moola F, Peters C, Pinault L, **Rainham D (Co-Inv)**, Tjepkema M.	Mortality Impacts of the Urban Built Environment in Canada: Evaluating the Role of Environmental and Socio-Demographic Factors	$48,823
2015 - 2020	Social Sciences and Humanities Research Council of Canada Ramos H (PI), Grant J, Haan M, Kaida L, McDonald T, Radice M, **Rainham (Co-Inv)**, Stoddart M, Thériault L, Wilkes R.	Perceptions of Change in Atlantic Canadian Cities	$418,924
2014 - 2016	Dalhousie Research Development Fund (SSHRC) **Rainham D (PI)**, Johnson S	Evaluating the Role of Nature Contact on Cognitive, Social and Physical Outcomes in Older Adults	$4,460

Date	Agency (Role)	Title	Amount
2014 – 2016	Heart and Stroke Foundation of Nova Scotia/CIHR (Bridge Funding) Blanchard C (PI), Warburton D, Rhodes R, King-Shier K, Grace S, Reid R, Sweet S, Giacomantonio N, McGowan E, Saunders T, **Rainham D (Co-Inv)**, Witcher C.	Cardiac rehAbilitation sedentaRy bEahviour correlateS (CARES)	$59,820
2013 – 2015	Canadian Institutes of Health Research Muhajarine N, Fuller D, Katapally T, Osgood N, **Rainham D (Co-Inv)**, Stanley K.	A Step towards Creating Active Urban Communities: Informing Policy by Identifying and Mapping Locations of Seasonal Activity Accumulation	$388,756
2012 - 2015	Canadian Institutes of Health Research Blanchard C (PI), Fowles J, Giacomantonio N, Plotnikoff R, **Rainham D (Co-Inv)**, Rhodes R, Shields C, Vallis M.	Lifestyle Activity Correlates for Diabetics (LIFE)	$384,679
2012 - 2014	Nova Scotia Health Research Foundation Dummer T (PI), Otley A, Parker L, **Rainham D (Co-Inv)**	Why does Nova Scotia have the highest incidence rate of inflammatory bowel disease (IBD) in the world? Exploring the role of environmental factors	$99,750
2012 - 2015	Canadian Institutes of Health Research Castleden H (Co-PI), Martin D (Co-PI), Adams M, Campbell D, Clow B, Gibson M, Jamieson R, **Rainham D (Co-Inv)**, Russell R, Steenbeek A, and Collaborators.	A' two-eyed seeing' approach to researching environmental health concerns with Pictou Landing First Nation	$444,639
2012 - 2013	Social Sciences and Humanities Research Council of Canada Johnson S (PI), **Rainham D (Co-PI)**, McGonnell M.	Effects of Exposure to Nature in Children: An Interdisciplinary Exploration	$34,110
2012 - 2013	Canadian Breast Cancer Foundation Blanchard C (Co-PI), Keats M (Co-PI), **Rainham D (Co-Inv)**, Younis T.	Breast Cancer Environmental Activity Correlates Trial (BREAST)	$143,568
2011 - 2012	Health Canada **Rainham D (PI)**	School-based Air Quality Study	$22,015

Date	Agency (Role)	Title	Amount
2011 - 2012	Dalhousie Research Development Fund Johnson S (PI), **Rainham (Co-PI)**	The Effects of Nature Exposure on Attention and Well-being in Children	$4,490
2011 - 2012	Capital Health Research Fund Blanchard C (PI), Keats M, **Rainham D (Co-Inv)**	Environmental influences on physical activity behaviours in breast cancer patients: A pilot study	$14,784
2011 - 2014	Canadian Institutes of Health Research **Kirk S (Co-PI), Rainham D (Co-PI)**, Rehman L, Shearer C, Blanchard C, Chircop A.	TIME (Tools, Information, Motivation, Environment) for health: A multi-level intervention to promote healthy eating in children and their families	$598,553
2011 - 2013	Cancer Research Society Dummer T, **Rainham D (Co-Inv)**, Parker L.	Cancer risk and outdoor air pollution in Halifax Regional Municipality, Nova Scotia	$117,520
2011 - 2013	Health Canada Gibson M (Co-PI), **Rainham D (Co-PI),** Duck T, Pierce J, Pegg M, Satish M, Jamieson R.	Halifax Marine Emissions Study	$265,920
2011	Nova Scotia Health Research Foundation Kirk S (PI), Shearer C, Barro K, **Rainham D (Co-Inv)**	Obesity and Health Inequities	$10,000
2011 - 2012	Nova Scotia Lung Association **Rainham D (PI).**	Map of Radon Potential for Health Risk Exposure Assessment	$8,320
2011 - 2014	Canadian Foundation for Innovation **Rainham D (PI).**	UrbanSense: Infrastructure for an Urban-Scale Environmental Sensor Network	$300,809
2010 - 2011	Health Canada **Rainham D (PI)**, Dummer T.	Measurement and Analysis of Air Quality and Noise in Halifax Regional Municipality	$55,609
2010 - 2011	Canadian Institutes of Health Research **Rainham D (PI)**, Shookner M, Blanchard C, Lyons R.	Knowledge Synthesis on Measures of Environmental Deprivation in Health Inequalities (MEDHI) Research: A Knowledge Synthesis on the Environment	$72,864

Date	Agency (Role)	Title	Amount
2010 – 2012	Nova Scotia Health Research Foundation **Rainham D (PI)**, Blanchard C.	Development of a Health Geo-Informatics Service (HEALGIS)	$48,792
2010 – 2013	Canadian Institutes of Health Research Blanchard C (PI), Lyons R, **Rainham D (Co-Inv)**, Murnaghan D, Rhodes R, Giacomantonio N, Young W, Reid R, Kirkland S, & Spence J.	Environmental Physical Activity Correlates after Cardiac Hospitalization (EPOCH)	$484,988
2010 – 2013	Nova Scotia Health Research Foundation Blanchard C (PI), **Rainham D (Co-Inv)**, Giacomantonio N, Lyons R.	Environmental Correlates Of Cardiac Rehabilitation (ENCORE)	$145,269
2009 – 2010	Capital Health Innovation Fund Blanchard C (PI), **Rainham D (Co-Inv)**, Giacomantonio N.	Community and Environmental Factors Influencing Drop-out and Adherence to Cardiac Rehabilitation	$25,000
2009 – 2010	Heart and Stroke Foundation Canada Blanchard C (PI), **Rainham D (Co-Inv)**, Giacomantonio N, Cyr C, Dufour-Doiron M.	Community and Environmental Factors Influencing Drop-out and Adherence to Cardiac Rehabilitation	$14,985
2009 – 2010	Public Health Agency of Canada **Rainham D (PI)**, Willison M.	Healthy Housing, Health Community Project	$35,000
2009 – 2010	QEII Research Foundation Parker K (PI), **Rainham D (Co-PI).**	Development of an Objective Evaluation Device to Measure the Impact of Learning Wheelchair Skills	$5,000
2009	Canadian Institutes of Health Research **Rainham D (Co-PI)**, Blanchard C (Co-PI).	Environmental Correlates Of Cardiac Rehabilitation (ENCORE) - Pilot	$9,853
2007 – 2012	Canadian Institutes of Health Research / Heart and Stroke Foundation Lyons R (PI), Grant J (Co-PI), Arthur M, Blanchard C, Chircop A, Dummer T, Kirk S, Parker L, Pitter R, Rehman L, **Rainham D (Co-Inv)**, Shields C.	Optimizing Investments in the Built Environment to Reduce Youth Obesity	$597,437

Research Grants and Contracts in Development

Date	Agency (Role)	Title	Amount
2019-2022	Canadian Institutes of Health Research **Rainham, D (PI),** James P, Villeneuve P, Crouse D, Lanphear B.	The Impact of Green Space on Mental Health of Canadian Children	$ 330,000

Total Funding to Date (since 2009): **$ 9,788,691**

Total Funding As Principal Investigator (*incl. Co-Pi)* : **$ 1,639,545**

PUBLICATIONS

Theses:

1. Rainham DG. (2008) *Human healthscapes as an approach to measuring context in research on place and health*. Ph.D. Thesis, Institute of Population Health, University of Ottawa, ON. pp. 231.

2. Rainham DG. (2000) Atmospheric risk factors of human mortality. M.Sc. Thesis, Earth and Atmospheric Sciences, University of Alberta, AB. pp. 146.

Refereed Journal Articles: Citations=1842; h-index=19; i10-index=26; RG Score=31.55
(students/trainees are underlined)

51 Houser N, Cawley J, Kolen A, **Rainham DG**, Rehman L, Turner J, Kirk S, Stone M. A loose-parts randomized controlled trial to promote active outdoor play in preschool-aged children: Physical Literacy in the Early Years (PLEY) project. Methods and Protocols 2019; (Submitted).

50 Stone M, Houser N, Cawley J, Kolen A, **Rainham DG**, Rehman L, Turner J; Kirk S. Accelerometry-measured physical activity and sedentary behaviour of preschoolers in Nova Scotia, Canada. Applied Physiology, Nutrition and Metabolism 2019. http://dx.doi.org/10.1139/apnm-2018-0683.

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Book Chapters:

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Non-Refereed Contributions and Reports:

1. Pictou Landing Native Women's Group, Castelden H, Lewis D, Jamieson R, Gibson M, **Rainham DG**, Rusself R, Martin D, Hart C. Our Ancestors Are in Our Land,

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2. Gibson M, **Rainham DG**. Halifax Marine Emissions Study: Final Report. Contract report for Dr. Amanda Wheeler, Exposure Assessment Section, Health Canada, Ottawa, Ontario. March 2013.

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Papers/Abstracts Published in Conference Proceedings:

1. Villeneuve P, Root A, Kumar N, Ambrose S, DiMuzio J, **Rainham DG**, Ysseldyk R. Are Neighbourhood Walkability and Greenness Associated with Increased Physical Activity and Better Self-Rated Health? Findings from the Beyond Health Survey of Adults in Ottawa, Canada. Journal of Transport and Health 2017;5:S88.

2. Awan T, **Rainham DG**, Johnson S, McSweeney J. Children want to spend time in outdoor nature but rarely do: A study exploring how children in Nova Scotia engage with nature. 29th International Congress of Applied Psychology (ICAP 2018), Montreal, QC, June 26-30, 2018.

3. Forbes CC, Keats M, **Rainham DG**, Younis T, Blanchard CM. (2017, June). Environments associated with moderate-to-vigorous physical activity in breast cancer survivors in Nova Scotia, Canada. Annual meeting of the International Society for Behavioural Nutrition and Physical Activity, Victoria, British Columbia.

4. Daley K, Jamieson R, **Rainham DG**, Castleden H, Truelstrup Hansen L, Harper S, Furgal C, Ell E. The social context of drinking water, sanitation and public health in the Arctic territory of Nunavut, Canada. In ARTEK Event 2016 – International Conference Sanitation in Cold Climate Regions. (pp. 21-22). Arctic Technology Centre, DTU Technical University of Denmark.

5. Balish S, **Rainham DG**, Blanchard CM. Digit-ratio (2D:4D) predicts youth sport motivation through feelings of self-assurance. Proceedings of the SCAPPS (Canadian Society for Psychomotor Learning and Sport Psychology) 2016 Annual Conference. Journal of Exercise, Movement and Sport 2016;48(1).

6. Lewis D, Castleden H, Martin D, Jamieson R, **Rainham DG**, Gibson M, & Pictou Landing Native Women's Group. Understanding the impacts of industrial development on health within a relational worldview. Royal Geographical Society Annual International Conference 2016, London, UK.

7. **Rainham DG**. A wireless sensor network for urban environmental health monitoring: Urbansense. Proceedings of the 9th International Symposium on Digital Earth (ISDE9), Halifax, Nova Scotia. 2016. http://iopscience.iop.org/article/10.1088/1755-1315/34/1/011006

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12. Poirier A, Dodds L, Johnson M, Dummer T, **Rainham DG**. Associations between exposure to air pollution and gestational hypertension in urban Halifax. Journal of Epidemiology and Community Health 2015;69:A2.

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14. Ferrier S, Blanchard CM, Giacomantonio N, **Rainham DG**, Murnaghan D, Rhodes R, Reid R, Warburton D, Spence J, King-Shier K, McGowan E, Lyons J, Kirkland S. Demographic and clinical correlates of sedentary behaviour in heart disease patients. Proceedings of the SCAPPS (Canadian Society for Psychomotor Learning and Sport Psychology) 2013 Annual Conference. Journal of Exercise, Movement, and Sport 2013;44(1).

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16. Blanchard, C.M., **Rainham, D**., McSweeney, J., Spence, J.C., McDonnell, L., Reid, B., Rhodes, R., McGannon, K., Edwards, N. Community socioeconomic status, urban sprawl, and the perceived environment's relationship to physical activity during home-based cardiac rehabilitation. Journal of Cardiopulmonary Prevention and Rehabilitation 2013;1, S5.

17. Keats M, Blanchard CM, Tyrrell A, **Rainham DG**, Younis T. Environmental influences on physical activity behaviours in breast cancer patients: a pilot study. Proceedings of the SCAPPS (Canadian Society for Psychomotor Learning and

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21. Krewski D, **Rainham DG**. Ambient air pollution and population health: Overview of health effects posters presented at the 2003 AIRNET/NERAM Conference. In: Craig L, Krewski D, Shortreed J, Samet J. (Eds), Strategies for Clean Air and Health: Proceedings of AIRNET Annual Conference/NERAM International Colloquium. Rome, Italy. November 5-7, 2003.

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Publications in Submitted

1. Cleveland T, Dec P, **RainhamDG**. Shorter Roads go a Long Way: The relationship between density and road length per resident within and between cities. Submitted to Land Use and Transport (Nov 2018).

Book Reviews and Popular Writing:

1. Rainham DG. There's More to Health Than Health Care. Health: An Ecosystem Approach, Jean Lebel, Ottawa: International Development Research Centre, 2003. Alternatives Journal 2004; 30(4): 36-7 [on-line at: www.alternativesjournal.ca].

2. Rainham DG. Global climate change: is global warming a health warning? Encompass 1999; 4(2): [on-line at: www.encompass.org/4-2/global.htm].

PRESENTATIONS (ALONE UNLESS OTHERWISE NOTED)

Invited Presentations:

2018 Location, Location, Location: Adding Value to Physical Activity Research. Annual Larry Maloney Lecture. Dalhousie University. March 24, 2018

2017 A Public Health Perspective on Urban Trees. Atlantic Urban Forest Conference. Fredericton, New Brunswick. November 7-8, 2017.

2017 Impact of Contact with Nature on Attention in Children. Children's Health and the Environment Workshop & Symposium: Moving Research to Action for Healthy Kids. Ivey Spencer Leadership Centre, Western University; London, Canada, June 26-28, 2017.

2017 Advancing Intervention to Reduce Risk of Obesity in Children. Children's Health and the Environment Workshop & Symposium: Moving Research to Action for Healthy Kids. Ivey Spencer Leadership Centre, Western University; London, Canada, June 26-28, 2017.

2013 Cities as if Ecology Mattered. PK13: Dense in the City, Halifax, NS.

2012 Exploring Physical Activity Behaviour Using Life Patterns Data. IWK Interdisciplinary Research Conference. Halifax, NS.

2012 Environmental Exposure to Electromagnetic Radiation. CH&E Seminar Series. Department of Community Health and Epidemiology, Dalhousie University. Halifax, NS.

2012 Location Tracking and Applications in Health Research. Spatial Intelligence for Health Meeting, ESRI Canada. Halifax, NS.

2012 Radon Soil Gas Exposure in HRM: Linking Geology and Probability. Atlantic Respirology and Critical Care Conference. Halifax, NS.

2012 Health Mapping. Atlantic Association on Planning Technicians Workshop. Centre of Geographic Sciences, NSCC. Lawrencetown, NS.

2012 Extreme Heat and Climate Change Vulnerability. Vulnerability to Global Changing Environments and Impacts on Health in Maritime Canada: Latest Research Findings and Implications for Policy. Halifax, NS.

2012 Using Life Patterns Data to Explore How Built Environments Shape Human Behaviours and Health. Place and Health: Shaping the Built Environment of New Brunswick and PEI. Canadian Public Health Association. Fredericton, NB.

2012 Free Range Kids: Classifying Youth Physical Activity Domains in the Built Environment. Dalla Lana School of Public Health & Bridgepoint Health. Toronto, ON.

2011 Nurturing Nature. Physical Activity Practitioners Exchange. Halifax, NS.

2011 Halifax Spatial and Personal Exposure Assessment Study. Air Quality and Health Research in Atlantic Canada: New Directions and Opportunities. Dalhousie University.

2011 The Contexts of Rehabilitation. Rehabilitation Research, Faculty of Health Professions, Dalhousie University. Halifax, NS.

2011 Examining the Issue of Overweight and Obesity in Nova Scotia from a Health Geomatics Perspective. Community Health and Epidemiology Seminar Series. Faculty of Medicine, Dalhousie University.

2010 Children's Health in an Age of Nature. Evergreen. Toronto, ON.

2010 Children's Health in the Age of Nature. Great Lakes Public Health Network. McMaster University.

2010 How Built Environments Influence Health. Ecology Action Centre Conversation Series on Healthy Urban Development, Halifax, NS.

2010 Biodiversity and Children's Health. Children's Health and Environment Workshop. University of Western Ontario.

2009 The Value of a Sustainability Perspective in Global Health Diplomacy. 16th Annual Canadian Conference on International Health, Canadian Society for International Health. Ottawa, Ontario.

2009 Human Health in the Age of Nature. Invited Keynote. Crossroads in Health Conference. Dalhousie University

2008 Context in Research on Place and Health. Invited Lecture. Occupational and Environmental Health (CHE 6001). Community Health and Epidemiology Department, Faculty of Medicine, Dalhousie University, Halifax, NS.

2008 The Place of Occupations. Invited Lecture. Occupational Therapy (Occu 2000): Occupation and Daily Life. Faculty of Health Professions, , Dalhousie University, Halifax, NS.

2008 A Sense of Possibility: What Does Governance for Health and Sustainability Look Like? International Health Speaker Series, International Health Office, Faculty of Medicine, Dalhousie University, Halifax, NS.

2007 Are Cities a Health Hazard. Café Scientifique. Canadian Institutes of Health Research. Halifax, Nova Scotia.

2007 When an Apple a Day Keeps Sustainability Away. 2nd Annual Atlantic Sustainable Campuses Conference. Sierra Youth Coalition. University of King's College. Halifax, Nova Scotia.

2006 GIS for Health and Epidemiology. GIS Day. Dalhousie University. Halifax, Nova Scotia.

2006 Geomatics for Population Health and the Healthscapes Study. Geomatics Atlantic 2006 Conference, Acadia University, Wolfville, Nova Scotia.

2006 Climate Change and Human Health. Our Forests, Our Health. Joint Meeting of: Nova Scotia Forest Technicians Association Canadian Institute of Forestry – NS Section Registered Professional Foresters Association of Nova Scotia. Truro, Nova Scotia.

2004 Air Quality and Human Health. International Youth Summit on Sustainable Urban Transportation, Ottawa, ON.

2003 Krewski D, **Rainham DG**. Overview of Health Effects Posters. AIRNET/NERAM Joint Colloquium: Strategies for Clean Air and Health. Rome, Italy. November 6, 2003.

2002 Human Health Response to Atmospheric Change – Review of Research Evidence Linking Climate Change, Air Pollution, and Health. Climate Change and Health Issues Research Network. The Impact of Climate Change on Air Pollution and Health. Summary Report of the First Workshop. Best Western Crystal Palace Hotel, Moncton, NB.

Scientific:

2019 Alwan T, Johnson S, **Rainham DG**. (2019). Children perceive time in nature as restorative despite minimal exposure to nature. Canadian Psychological Association 80th Annual National Convention, Halifax, N.S. May 31- June 2, 2019.

2019 Liu Y, Goudreau S, Oiamo T, **Rainham DG**, Hatzopoulou M, Chen H, Davis H, Tremblay M, Johnson J, Bockstael A, Leroux T, Smargiassi A.(2019). Comparison of land use regression and random forests models on estimating noise levels in five Canadian cities. Canadian Society for Epidemiology and Biostatistics 2019 Biennial National Conference, Ottawa, ON. May 13-15, 2019.

2019 Liu Y, Goudreau S, Oiamo T, **Rainham DG**, Hatzopoulou M, Chen H, Davis H, Tremblay M, Johnson J, Smargiassi A.(2019). Estimation of double exposures to outdoor nitrogen dioxide and environmental noise in five Canadian cities. 2019 HEI Annual Conference, Seattle, Washington, May 5-7, 2019.

2018 Trecarten N, Rhodes R, Warburton D, Murnaghan D, King-Shier K, Spece J, Reid R, Giacomantonio N, **Rainham D**, Kirkland S, McGowan E, Blanchard CM. (2018). Examination of sedentary time in patients with coronary heart disease. Presented at the Canadian Association Cardiovascular Prevention and Rehabilitation, Toronto, Ontario.

2016 **Rainham** DG, Crouse DL. Nature-health interconnections: Evidence, mechanisms and current research. Canadian Association of Geographers. Halifax, Nova Scotia, May 30-June 3, 2016.

2016 Bennett M, **Rainham DG**, Drage J. Radon potential mapping: Sensitivity to input weighting selection. Canadian Association of Geographers. Halifax, Nova Scotia, May 30-June 3, 2016.

2016 Bennett M, **Rainham DG**. The relationship between greenness and physical activity in Canadian children and adults. Canadian Association of Geographers. Halifax, Nova Scotia, May 30-June 3, 2016.

2016 Castleden H, Francis S, Lewis D, Strickland D, Denny C, Martin D, Jamieson R, **Rainham D**, Russell R, Gibson M. (May 11-14, 2016). "Our ancestors are in our land, water, and air: A Two-Eyed Seeing approach to researching environmental health concerns with Pictou Landing First Nation. Community-Campus Partnerships for Health's 14th International Conference Journey to Justice: Creating Change Through Partnerships. New Orleans, Louisiana, USA.

2015 Blanchard CM, Campbell N, **Rainham DG**, Giacomantonio N. (2015). Translating physical activity recommendations into steps per day for cardiac rehabilitation patients. Canadian Society for Psychomotor Learning and Sport Psychology, Edmonton, Alberta.

2015 Balish S, Deaner R, **Rainham DG**, Blanchard CM. Sex differences in sport across countries: Does gender inequality and digit ratio (2D:4D) matter? Northeastern Evolutionary Psychology Society (NEEPS), 9th Annual Meeting, Suffolk University in Boston, Massachusetts.

2015 Poirier A, Dodds L, Dummer T, **Rainham DG**, Maguire B, Johnson M. Maternal exposure to air pollution and adverse birth outcomes in Halifax, Nova Scotia. Canadian Society for Epidemiology and Biostatistics 2015 Conference, Toronto, Ontario, Canada.

2014 Daley K, Jamieson R, Rainham DG, Truelstrup Hansen L. (December). Assessing exposure pathways and human health risks related to wastewater treatment in Inuit communities. Arctic Change 2014, Ottawa, Ontario, Canada.

2014 Balish SM, Rainham DG, Blanchard CM. (2014, November). Community Size and Sport and Exercise Participation Across 28 Countries. Verbal presentation at SCAPPS (Société Canadienne d'Apprentissage Psychomoteur et de Psychologie Sportive) annual meetings, London, Ontario, Canada.

2013 Rehman L, Shearer C, **Rainham DG,** Kirk S. Recreation facility food environments: Exploring opportunities to improve family food choices. Qualitative Health Research Conference, Halifax, NS.

2013 **Rainham DG**, Blanchard C, Dummer T, Kirk S, Lyons R, Rehman L, Shearer C. Activity Space Measures of Access to Residential and School Neighbourhood Food Environments, Diet and BMI among Youth. International Medical Geography Symposium, Michigan State University, East Lansing, MI.

2013 Balish, S.M., Evans, B., Blanchard, C., & **Rainham, D.G** (June 2013). Female Participation Profiles and Reasons for Physical Activity Stratified by Gender-Inequality of Host Country. Verbal Presentation of the 2013 Annual Meeting International Society for Behavioral Nutrition and Physical Activity, Ghent, Belgium.

2013 **Rainham DG**, Cui Y, Nauta L, Parker L, Dummer T. Lung Cancer and Exposure to Air Pollution in Halifax, Nova Scotia. Canadian Association of Geographers Conference, St. John's, NF.

2013 Balish, S., McLaren, C., **Rainham, DG**., & Blanchard, C.M. Correlates of Youth Sport Attrition: Review, Synthesis, and Future Directions. Presentation at the North American Society for the Psychology of Sport and Physical Activity, New Orleans, Louisiana, US

2012 Balish S, Blanchard C, **Rainham D**. The Social Ecology of Team-Sport Participation in Youth: A Multilevel, Gender-Specific Approach. Sports Canada Research Initiative Conference, Ottawa, ON.

2012 Jason, T., Blanchard, C.M., **Rainham, D**., & Giacomantonio, N. (2012). The effect of one's living environment on physical activity during cardiac rehabilitation. Canadian Society for Psychomotor Learning and Sport Psychology, Halifax, NS.

2012 Keats, M., Blanchard, C.M., Tyrrell, A., **Rainham, D**., & Younis, T. (2012). Environmental influences on physical activity behaviours in breast cancer patients: A pilot study. Canadian Society for Psychomotor Learning and Sport Psychology, Halifax, NS.

2012 **Rainham DG**, Shearer C, Kirk S, Blanchard C, Dummer T, Shields C, Bates C, Lyons R. Navigating Nutrition in the Neighbourhood. American Associations of Geographers Annual Conference. New York, NY.

2012 Blanchard, C.M., Shearer, C., **Rainham, D**., Kirk, S., Shields, C., Pitter, R., Dummer, T., & Lyons, R. (2012). Physical activity in adolescents: The role of the built environment from a GPS perspective. Presented at the North American Society for the Psychology of Sport and Physical Activity, Hawaii, USA.

2011 **Rainham DG**, Blanchard C, Dummer T, Kirk S, Shearer C, Bates C, and the ENACT Team. (July 2011). Spatial classification of youth activity patterns. International Medical Geography Symposium. Durham, UK.

2011 McSweeney J, **Rainham DG**, Bates C, Gibson M, Guernsey J. (July 2011). Using wearable global positioning technology to assess personal exposure to fine airborne particles. . International Medical Geography Symposium. Durham, UK.

2011 Neimanis A, Castleden H, **Rainham DG**. (May 2011). (Health) Justice for All: Creating a Tool for Environmental Justice with an Ecological Integrity Lens to Improve Well Being. Canadian Association of Geographers, Calgary, AB.

2010 Pitter R, Shields C, Chircop A, **Rainham D**G, Shearer C, Rehman L, Blanchard C, Flannery M. (November, 2010). I Think my Neighbourhood is Safe, So Why is Safety a Barrier to Physical Activity? North American Society for the Sociology of Sport (NASSS) 2010 Conference: Producing Knowledge, Producing Bodies: Cross-Currents in Sociologies of Sport and Physical Culture. San Diego, CA.

2010 Blanchard, C.M., **Rainham, D**., McSweeney, J., Spence, J.C., McDonnell, L., Reid, B., Rhodes, R., McGannon, K., Edwards, N. (2010). Community socioeconomic status, urban sprawl, and the perceived environment's relationship to physical activity during home-based cardiac rehabilitation. Canadian Association for Cardiac Rehabilitation, Montreal, QC.

2010 Patricia Manuel, Andrea Chircop, **Daniel Rainham**, Jill L. Grant, Laurene Rehman, Gillian McGinnis, Meredith Flannery, Renee Lyons, Trevor Dummer, Chris Blanchard. (October 2010) You want us to do what, where? Investing in youth health through the built environment. 48th International Making Cities Livable Conference True Urbanism: Planning Healthy and Child-Friendly Communities. Charleston, SC.

2010 Neimanis A, Castleden H, **Rainham DG**. (July 2010). A Systematic Review of the Literature on Environmental Justice: What about Ecological Integrity. Ecohealth 2010. London, UK.

2010 Shearer, C., Grant, J., Lyons, R., Arthur, M., Blanchard, C., Chircop, A., Dummer, T. Kirk, S., Manuel, P., McHugh, T., Parker, L., Pitter, R., **Rainham, D**., Rehman, L., & Shields, C. (July 2010). Environmentally-linked disparities in youth physical activity and nutrition. 20th IUHPE World Conferences on Health Promotion. Geneva, Switzerland.

2010 **Rainham DG**, Blanchard C, Chircop A, Dummer T, Kirk S, Rehman L, the ENACT Team. How much and where? Using actimetry and global positioning systems (GPS) to assess the relationship between physical activity and the built environment. Paper presented at the Canadian Public Health Association Centennial Conference, June 13-16, 2010, Toronto, ON.

2009 **Rainham DG**, Wilson J. Socioeconomic Metabolism, Health and Ecological Thresholds. International Medical Geography Symposium. Hamilton, ON.

2009 Grant, J., Lyons, R., Arthur, M., Blanchard, C., Chircop, A., Dummer, T. Kirk, S., Manuel, P., McHugh, T., Parker, L., Pitter, R., **Rainham, D**., Rehman, L., Shearer, C., & Shields, C. (2009, June). Optimizing investments in the built environment to reduce youth obesity: Methodological approaches and dilemmas. Poster session presented at the 7th International Conference on Diet and Activity Methods, Washington, DC.

2008 McHugh, T., Shearer, C., Grant, J., Lyons, R. , Arthur, M., Blanchard, C., Chircop, A., Dummer, T., Kirk, S., Manuel, P., Parker, L., Pitter, R., **Rainham, D**., Rehman, L., & Shields, C. Optimizing investments in the built environment to reduce youth obesity. Poster session presented at the Childhood & Adolescent Obesity 2008 Conference, Vancouver, BC.

2008 Is the Path to Human Well-being Inevitably Unsustainable? Public Health in Canada: Reducing Health Inequalities through Evidence and Action. Canadian Public Health Association 2008 Annual Conference. Halifax, NS.

2008 Gibson, M.D., Guernsey, J.R., **Rainham, D**., Gould, R., Hore, P., Garand, L., Beauchamp, S., Waugh, D., Brook, J.R., McPherson, J., Bryden, B., Maher, R., King., G. Pilot Comparative Personal/Indoor/Outdoor/Urban/Rural Exposure Studies for Ground-Level Ozone (O3) and Fine Respirable Particles. Canadian Public Health Association 2008 Annual Conference. Halifax, NS.

2007 The Spatial Existence of People and Their Health. Public Health in Canada: From Politics to People. Canadian Public Health Association 2007 Annual Conference. Ottawa, ON.

2007 Governance for Population Health and Sustainability. Ecological Integrity and a Sustainable Society Conference. Dalhousie University. Halifax, NS.

2006 Geovisualization of Health and Social Capital Data Derived from Statistics Canada Surveys. Statistics Canada 23rd International Methodology Symposium: Methodological Issues in Measuring Population Health. Gatineau, QC.

2004 **Rainham DG**, Tomic KE, Sheridan SC, Burnett RT. Synoptic weather patterns and modification of the association between air pollution and human mortality. 16th Conference on Biometeorology and Aerobiology and the Fifth Symposium on the Urban Environment, American Meteorological Society, Vancouver, British Columbia

2004 Population Health and the Health of the Biosphere. Global Ecological Governance for Eco-Justice and Public Health. Global Ecological Integrity Group. Montreal, QC.

2003 Examining Sustainability and Population Health. 10th Annual Conference of the Environmental Studies Association of Canada, Halifax, NS.

2002 Atmospheric Risks to Public Health – What is the Evidence? Urban Heat Island Summit. The Clean Air Partnership. Toronto, ON.

2001 Smoyer-Tomic KE, Kuhn R, **Rainham DG**. Natural Hazards Assessment: Heat Waves. Canadian Natural Hazards Assessment Second Workshop. Mississauga, ON.

2001 Smoyer KE, **Rainham DG**, Pereira D. In Search of a Heat Stress Index for Southern Ontario. Association of American Geographers Annual Meeting. New York, NY.

2000 Smoyer KE, **Rainham DG**. When Weather Kills: The Search for a Heat Stress Index Continues. 9th International Symposium in Medical Geography. Montreal, QC.

2000 Contaminated Communities and Continuums of Causation: Perspectives From Environmental Epidemiology. ESAC 2000: Exploring the Shades of Environmentalism. Congress of the Social Sciences and Humanities, University of Alberta. Edmonton, AB.

2000 Modelling Atmospheric Risk Factors of Human Mortality. EcoSummit 2000. Westin Nova Scotia, Halifax, NS.

2000 Smoyer KE, **Rainham DG**, Hewko JN. Hot in the City: Heat Waves and Health in Southern Ontario. Centre for Health Promotion Studies Research Symposium Series. Edmonton, AB.

2000 Nonparametric Smoothing Techniques: Applications in Environmental Epidemiology. Area-Wide Epidemiology and Biostatistics Conference, University of Alberta. Edmonton, AB.

1999 Smoyer KE, **Rainham DG**. Humidex and Mortality Response in Toronto, 1980-1996. Public Health Impact of Extreme Heat Workshop. Toronto Public Health. Toronto, ON.

1999 Healthy Cities and Shires in Rural Australia: The Cambooya Experience. Canadian Association of Geographers Conference. Lethbridge, AB.

1999 **Rainham DG**, Smoyer KE. Weather-Related Mortality in Southern Ontario. Western Division, Canadian Association of Geographers, Annual Meeting. Kelowna, BC.

SERVICE

Workshops:

2018 Spatial Analytics for Health Research using ArcGIS. Maritime SPOR (Strategy for Patient-Oriented Research) Support Unit. Dalhousie University. February 22-23.

2017 Spatial Analytics for Health Research using ArcGIS. Maritime SPOR (Strategy for Patient-Oriented Research) Support Unit. University of New Brunswick. November 9-10.

2016 Spatial Analytics for Health Research using ArcGIS. Maritime SPOR (Strategy for Patient-Oriented Research) Support Unit. Dalhousie University. June 20-21.

2016 Spatial Analytics for Health Research using ArcGIS. Maritime SPOR (Strategy for Patient-Oriented Research) Support Unit. Atlantic Veterinary College, University of Prince Edward Island. June 20-21.

Guest Lectures:

2018 Noise!. Guest Lecture to ENVS 2500, November 2, 2018.

2018 Environmental Determinants of Physical Activity and Obesity. Guest Lecture to KINE 4709, October 18, 2018.

2018 Urban Nature as Health Promotion. Guest Lecture to ENVS 1200, April 3, 2018.

2017 An Introduction to Systems Thinking. Guest Lecture to ENVS 3501, September 27, 2018.

2017	Environmental Determinants of Physical Activity and Obesity. Guest Lecture to KINE 4709, September 18, 2017.
2016	How to Conduct a GIS-Based Research Project. Guest Lecture to the GIS Certificate Project Course, January 16, 2016.
2015	Kids Need Nature. Guest Lecture to the SHAD Valley Program, July 9, 2015
2015	Thinking Spatially: GIS in Epidemiology. Guest Lecture to HESA 5320: Managerial Epidemiology. February 2, 2015.
2013	Space, Place and Health. Guest lecture to CH&E 6042: Determinants of Health in Human Populations. March 27, 2013.
2013	Spatial Thinking: Applications to Resource Decisions. Guest Lecture to ENVI 5500: Socio-political Dimensions of Resource and Environmental Management. March 11, 2013.
2013	Environment and Health: An Introduction. Guest lecture to ENVS 1000, Introduction to Environmental Science. January 10, 2013.
2012	Health Geomatics. Guest lecture to ENVS 3400, Environment and Human Health. March 27, 2012.
2011	How Our Environment Affects our Health. Guest lecture to ENVS 1000, Introduction to Environmental Science. March 10, 2011.
2011	Governance for Sustainability. Guest Lecture to ENVI 5500. January 26, 2011.
2010	Place and Health. Guest lecture to CH&E 6042, Determinants of Health in Human Populations. March 18, 2010.
2010	What is Environmental Health? Guest lecture to ENVS 1000, Introduction to Environmental Science. January 21, 2010.

Grant Review:

Date	Organization/Role	Opportunity/Committee
2012 - Present	CIHR Operating Grants (PH)	Institute of Population and Public Health
2016	Health Canada - Clean Air Regulatory Agenda (CARA)	Environmental and Radiation Health Sciences Directorate

2012 - 2015	CIHR Training Grants (PROPEL)	Population Interventions for Chronic Disease Prevention
2012 - 2013	Nova Scotia Health Research Foundation Scientific Officer (Training)	Establishment Grants Health Policy, Health Services and Outcomes
2012	Health Effects Institute Proposal Review	Rosenblith New Investigator Award
2011 - Present	Heart and Stroke Foundation of Nova Scotia Proposal Review	Bright Red Awards Scientific Advisory Committee
2010	National Institutes of Health/Centers for Disease Control Proposal Review	FOA EH09-001 Climate Change: Environmental Impact on Human Health
2009	Northern Ireland Chest Heart & Stroke Proposal Review	Reference Number: 200925 Climate Change and Cardiovascular Health
2007 - Present	Canadian Public Health Association Abstract Review	Canadian Public Health Association Annual Conference / Abstract Review

Institutional Service:

Date	Committee	Location
2017 – Present	Director, ESRI Centre of Excellence	Dalhousie University
2017 - 2018	Chair, Senate Review Committee for Faculty of Arts and Social Sciences	Dalhousie Senate
2016 - Present	Selection Committee for Director of the Interdisciplinary PhD Program	Faculty of Graduate Studies
2015 - Present	Faculty Council	Faculty of Science

2015 - Present	Director, Environmental Science	Faculty of Science
2015	Faculty Search	School for Resource and Environmental Studies, Faculty of Management
2014 - 2016	Member, Executive Committee	Healthy Populations Institute
2014	Tenure and Promotion Committee	School of Planning, Faculty of Architecture and Planning
2014 -	GIS Centre Advisory Committee	Killam Library
2011 -	Scientific Advisory Committee	Heart and Stroke Foundation of Nova Scotia
2009 -	Environment Committee Representative	Science Atlantic
2009 - 2015	Undergraduate Student Research Awards Committee	Faculty of Science
2009 - 2014	Curriculum Development and Awards Committees	College of Sustainability
2007 - 2009	Executive Committee, Institute of Population Health	University of Ottawa
2007	GIS Workshop Steering Committee	Nova Scotia Health Research Foundation

Journal Review (last 5 years):

American Journal of Preventive Medicine
BMC Public Health
Canadian Journal of Public Health
Diabetes Technology and Therapeutics
Environmental Research
Health and Place
International Journal of Health Geographics
Journal of Epidemiology and Community Health
Journal of the Air and Waste Management Association
Social Science & Medicine
Spatial and Spatiotemporal Epidemiology

SUPERVISION OF POST-DOCTORAL FELLOWS

1. Terashima, M. (2012-14). Mapping the social-ecological determinants of youth sport participation: A community level perspective. Interdisciplinary PhD program, Faculty of Graduate Studies, Dalhousie University.
   2012 – 2014: CIHR Fellowship / $80,000

   Peer-Reviewed Publications:
   1. A small-area analysis of inequalities in chronic disease prevalence across urban and non-urban communities in the Province of Nova Scotia, Canada, 2007–2011. (BMJ Open)

   2. Should we enhance the commonly used deprivation index for a regional context? (Can J Pub Health)

GRADUATE SUPERVISION

DOCTORAL STUDENTS

1. Barber, B. (2017-). An Investigation of Super-Users of Emergency Health Services in Halifax. Ph.D. in Health program, Faculty of Health, Dalhousie University.
   2017 – 2019: Nova Scotia Graduate Scholarship / $15,000/annum

2. Balish, S. (2011-2016). Mapping the social-ecological determinants of youth sport participation: A community level perspective. Interdisciplinary PhD program, Faculty of Graduate Studies, Dalhousie University.
   2012 – 2015: SSHRC Doctoral Research Award / $105,000
   2012 – 2014: President's Award, Dalhousie University / $17,226
   2012 – 2103: CIHR / HSFC Fellow in Population Intervention for Chronic Disease Prevention Top-up Award / $2000
   2011 – 2012: Dalhousie FGS Award / $6,000
   2011 – 2012: Doctoral Stipend / CIHR / $17,850

3. Daley, K. (2013-present). Human Health Risk and Wastewater Treatment in Arctic Canada. Interdisciplinary PhD program, Faculty of Graduate Studies, Dalhousie University.
   2013 - 2017: Northern Municipal Wastewater Management' Project / $64,500

4. McSweeney, J. (2010-2016). Nurturing nature and the human psyche: Understanding the impact of indoor nature exposure on depression. Interdisciplinary PhD program, Faculty of Graduate Studies, Dalhousie University.
   2013 - 2014: SSHRC Doctoral Research Award / $35,000 (1 yr)
   2011 - 2013: NSHRF Scotia Support Scholarship / $35,000
   2010 – 2013: CIHR / HSFC Fellow in Population Intervention for Chronic Disease Prevention / $17,500

MASTER'S STUDENTS

1. O'Brien, K. (2011-2013). Radon Soil Gas Mapping in Halifax Regional Municipality. Masters in Earth Sciences, Faculty of Science, Dalhousie University.
   2011-2012: Nova Scotia Lung Association: Legacy Research Grant / $5,000

2. McCurdy, J. (2012-2014). Community Mapping of Elder Oral Histories in Pictou Landing-Boat Harbour. Masters in Resource and Environmental Management, Faculty of Management, Dalhousie University.

3. Neimanis, A. (2010-2012). Environmental Justice: Making the Case for Ecological Integrity. Masters of Environmental Studies, Faculty of Management, Dalhousie University.
   2010 - 2012: SSHRC Masters Scholarship / $35,000

UNDERGRADUATE SUPERVISION

HONOURS STUDENTS

Year	Name (Degree)	Title
2017-18	Chantelle-Lyn Fynn	The relationship between urban tree canopy cover and socioeconomic status in urban Halifax
2015-16	Rachel Shin (Environmental Science)	Exploring the influence of nature exposure on risk-based decision-making
2014-15	Allison Welk (Environmental Science)	Association Between Access to Neighbourhood Greenness and Physical Activity in Nova Scotia Youth
2012-13	Kit Moran (Environmental Science)	An Investigation into the Ability of Urban Green Space to Provide Stress Restoration
2012-13	Caroline McNamee (Environmental Science)	Evaluation of Public Recreational Greenspace in Halifax for Physical Activity Promotion Using the Quality of Public Open Space Tool
2010-11	Emily Stewart (Environmental Science)	Spatial and Temporal Mapping of Radiofrequency Fields
2009-10	Alexandre Girard (Community Design)	Elderly Walkability in Spryfield, Nova Scotia
2008-9	Gavin King (Environmental Science)	Community Environmental Noise and the Built Environment in Two Halifax Neighbourhoods
2005-6	Rory Cantwell (Environmental Science)	The Relationship Between Ecological Footprint and Population Health in Twenty-Two Canadian Municipalities
2005-6	Erin Balser (Environmental Science)	Assessing the Sustainability of the University of King's College

2005-6	Ashley Greenspoon (International Development)	The Tale of Two Medicines: Examining the Influence of Western Medicine on Traditional Medicine in Developing Countries
2005-6	Nicholas Ahlers (International Development)	Ecotourism in Botswana: The Okavango Polers Trust (OPT) and a Sustainable Approach to Ecosystem Health

Courses Taught

1. Environmental Science (ENVS2000): Urban Field School
   Urban Field School introduces students to environmental science issues within an urban setting. By the end of the course a student will be able to: discuss how humans are components of ecosystems; differentiate urban an non-urban areas and systems; understand how plants and animals exist and interact with humans; understand human-dominated landscapes; engage in scientifically informed discussions related to urban areas and urbanization; and demonstrate proficiency in a variety of environmental science techniques including mapping, sampling, and measurement.

2. Environmental Science (ENVS2100): Environmental Informatics
   Environmental Informatics is the knowledge skills and tools which enable information to be collected, managed and disseminated to support research in environmental science. Students develop skills for the analysis, evaluation and synthesis of knowledge in environmental science. Information systems, tools, and techniques are introduced and applied to current environmental challenges.

3. Environmental Science (ENVS3400): Environment and Human Health
   You and everything around you is part of the environment. Every aspect of the environment, from the air you breathe to the water you drink, from the roads you travel to the waste you produce, may affect how you feel and ultimately your well being. Environmental health is a discipline that focuses on the interrelationships between people and their environment, promotes human health and well being, and fosters a safe healthful environment. In this course we will learn about the tools and perspectives needed to investigate the relationships between human health and the environment. We will also examine a variety of current issues from the global to the local and critically evaluate the systems required to support human health in the context of environmental sustainability. Weekly laboratory exercises will teach students how theories and methods from ecology, epidemiology and geomatics (GIS, GPS, and remote sensing technologies) can be employed in environment and health research.

4. Sustainability 3000 (SUST3000): Environmental Decision Making
   Making decisions regarding our individual and collective choices for a sustainable future requires decisions to account for the uncertainty and complexity inherent to human development that is sensitive to ecological constraints along with competing human values. Given this context, this course sets out to explore a number of key challenges that confound decision-making generally along with a variety of decision support tools that help us integrate diverse knowledge and values for theoretically better outcomes. While many of the issues and approaches covered transcend human decision contexts,

throughout the course we will draw on resource and environment related examples and contexts and consider the implications of disconnecting development decision-making from sustainability.

5. Environmental Science (ENVS4901/2): Honours Thesis
   An honours thesis comprised of an independent investigation to develop mastery of the basic skills of problem definition, proposal preparation and project implementation is the key to dealing with a wide range of "real-life" situations, both on and off the career path. The journey includes lectures and tutorials on proposal writing, research design and methodology, and an independent environmental science research project carried out under the supervision of an approved faculty member. An honours thesis gives you the opportunity to conduct an independent research project over the course of the full academic year. The experience will help you to decide if you would be interested in a career in research as well as the aptitude to be a successful researcher in environmental science.

I also contribute to the Environmental Science Field School classes (ENVS3001) teaching geographic information systems and sampling methods.

TEACHING SUBJECT MATTER

- Teaching in epidemiologic and geomatics research methods, environmental science, measurement and methods, and health geography
- Medical school teaching on population and environmental health
- Graduate and undergraduate supervision in epidemiology, environmental science and sustainability research

RESEARCH AND IDEAS IN THE MEDIA:

November 2017: What neighbourhood noise drives you nuts? CBC Maritime Noon, November 3, 2017.

November 2017: Noise and health effects. CBC Radio 1: Mainstreet. November 2017.

November 2014: Dal study looks at physical effects of exposure to nature. By Ben Cousins. UNews, November 18, 2014.

May 2013: Chronic illness high near pulp mill. By Daniel Campbell. The News, May 3, 2013.

July 2012: The future of downtown: re-ripening Barrington Street. By Chris Benjamin. Openfile, July 6, 2012.

June 2012: Getting kids moving. Maritime Noon, CBC, Halifax, Nova Scotia (.mp3).

June 2012: A television interview about the Environment, Nutrition and Physical Activity Project and the results of the paper published in the American Journal of Preventive Medicine. CTV Morning Live, Halifax, Nova Scotia.

May 2012: City kids get more exercise than rural ones. The Chronicle Herald, Halifax, Nova Scotia (online).

April 2012: Obesity linked to neighbourhood features: Do you live in a fat neighborhood? ABC News (online).

April 2012: New technology maps the surprising subtleties of childhood and teen obesity. Scientific American (Observations Blog),

August 2011: A television interview about the Legacy Research Grant and Kelsey O'Brien's work on radon mapping. CTV Morning Live. Halifax, Nova Scotia.

July 2011: Feds to probe air pollution from ships; Study will analyze data from 55 air quality monitors in Halifax. The Chronicle Herald. Halifax, Nova Scotia.

August 2010: Researcher wants to probe air quality. The Chronicle Herald. Halifax, Nova Scotia.

November 2009: Sustainable Offices 101. Green Living Magazine.

February 2009: Tracking our eco-footprints. The Coast. Halifax, Nova Scotia.

October 2008: The Joy of Parking: Hot Meters Map and Map of Parking Ticket Intensity. CBC News Feature Report. Halifax, Nova Scotia.

February 2007: A night on the town: Interactive Map of Reported Crimes. CBC News Feature Report. Halifax, Nova Scotia.

June 2009: Is your neighbourhood healthy? Harrowsmith Country Life (Vol. 30, pp. 108-9).

March 2006: He knows where you've been. Dalhousie Alumni Magazine. Halifax, Nova Scotia.

APPENDIX E-1

February 23, 2019

IN RESPONSE TO: Dillon Consulting Limited (2019). Northern Pulp Nova Scotia environmental assessment registration document: replacement effluent treatment facility. https://www.novascotia.ca/nse/

Dear Dave Gunning,

Thank you for your interest in our study, "*Pilot study investigating ambient air toxics emissions near a Canadian kraft pulp and paper facility in Pictou County, Nova Scotia*" (Hoffman et al. 2017). The following is a response to the misrepresentation put forth by Northern Pulp's Environmental Assessment (EA) of the scientific contributions our study provides.

Explanation of key topics (Hoffman et al. 2017):

- There is growing concern about the toxicity of volatile organic compounds (VOCs) (Cicolella 2008), their presence in air, and the consequences of long-term, low-dose exposure to these agents. Airborne VOCs are varied and widespread pollutants (e.g., hydrocarbons, aromatics, and some chlorinated compounds) and are increasingly recognized as important precursors to $PM_{2.5}$ and ground-level O_3 formation through photochemical reactions (Ryerson et al. 2001). Many VOCs are included in the US Environmental Protection Agency (EPA) air toxics list.
- "Air toxics" are defined as "those pollutants that cause or may cause cancer or other serious health effects […] or adverse environmental and ecological effects" (EPA 2015a).
- Ambient air monitoring in the US is conducted in accordance with the Clean Air Act (CAA) (Clean Air Act 1970). CAA amendments identify 187 air toxics, which form the basis for EPA's approach to regulating emissions (EPA 2015a). Of these, EPA identified 30 air toxics that pose the greatest potential health threat in urban areas (EPA 2015b). Using the risk-based principles outlined in CAA, EPA developed the National Air Toxics Assessment (NATA), a comprehensive evaluation tool that prioritizes efforts to regulate emissions of air toxics (EPA 2015c). Such a rigorous initiative has not been implemented in Canada, where no federal guidelines exist for ambient air toxics. Yet, some of these air toxics, as noted in our publication, have been identified as associated with increased risks for chronic disease. See, for example, Paul Villeneuve et al. (2013) (https://www.ncbi.nlm.nih.gov/pubmed/23369806) which showed an increase risk of cancer mortality associated with elevated ambient air benzene concentrations in urban Toronto.
- The National Air Pollution Surveillance (NAPS) program was established to monitor and assess ambient (outdoor) air quality at various urban and rural areas across Canada. This program focuses primarily on the criterion air pollutants (nitrogen dioxide, ozone, $PM_{2.5}$ and sulphur dioxide). Several EPA-designated air toxics are routinely quantified in Canada; however, at a subset of NAPS monitoring sites.
- VOCs selected for analysis in this investigation were based on EPA's list of 30 urban air toxics (EPA 2015b) and *National Air Toxics Trends Station Work Plan Template* (EPA

2015d). Therefore, this investigation represents one of the few peer-reviewed published studies on record about airborne VOCs in rural Canada.

This was a "*pilot*" study (as indicated by the title and subsequent sections of the study); therefore, it was not meant to provide causal evidence to implicate the presence of airborne VOCs as solely emanating from Northern Pulp ("the mill"), as the EA suggests. Nevertheless, we were interested in documenting VOC-related air quality in the vicinity of the mill, given that pulp mills are present across rural Canada. Publicly accessible Environment and Climate Change Canada (ECCC) data (VOC concentrations [Granton NAPS ID: 31201, located southwest of the mill], and local meteorological conditions [Caribou Point]) (http://climate.weather.gc.ca/climateData/; http://maps-cartes.ec.gc.ca/rnspa-naps/) were examined using temporal (2006–2013) and spatial analytic methods to investigate prioritized air toxic ambient VOC concentrations near a pulp plant to determine whether these emissions concentrations were in the range of US EPA air toxic levels (EPA 2015d) (https://www3.epa.gov/ttnamti1/files/ambient/airtox/nattsworkplantemplate.pdf).

Results highlight associations with wind direction and the Granton NAPS site's ambient VOC concentrations in relation to the location of the mill. Compared to all other wind directions, prevailing winds from the northeast and the mill typically resulted in higher VOC concentrations for all compounds, except carbon tetrachloride, suggesting that the mill is likely a contributor to increased concentrations; however (as stated in the study), the origin(s) of VOCs are "*inconclusive*", and "*other local sources likely contribute to air toxics emissions*". The mill's EA states that "[this study] did not attempt to rule out contributions from other potential sources of VOCs in the area", which is clearly not a true statement - other potential local emission sources were discussed in detail in the publication. Figure 1, for example, is a map displaying other local point source emitters in the community (e.g., tire manufacturing facility, coal-fired thermal electrical generating station).

VOCs (1,3-butadiene, benzene, and carbon tetrachloride) routinely exceeded EPA air toxics-associated cancer risk thresholds, regardless of whether the mill contributed to these VOC levels, and is a significant finding that warrants further investigation. The EA's statement: *"When other study uncertainties are considered […] there is no current air quality issue with the seven targeted VOCs in the Pictou County area"* is misleading. Due to the limited number of sampling sites, the problem with the location of the sampling site in relation to the location of the mill, and the short duration of our study, we explicitly identified the need for further investigation on this question. As commonly identified by environmental researchers, absence of evidence is not necessarily evidence of absence. Therefore, the limitations caused by sparse data does not necessarily mean there is no problem with air emissions in this community and there is no justification for this erroneous conclusion, as stated in the EA.

The EA statement, *"The seven VOCs are not known (based on literature review) to be associated with pulp and paper mill activities and air emissions to any significant extent",* is both unclear and undefined. Furthermore, the EA does not specify how the literature was reviewed/cited to support this statement. According to the mill's own self-reported NPRI report in 2012, 143.18

tonnes of VOCs were atmospherically emitted on-site (ECCC 2012). An estimated 3.195 tonnes of benzene were released to the air from a stack higher than 50 m and 0.022 tonnes were released within 50 m of the ground. Benzene can combine with chlorinated hydrocarbons associated with the Kraft bleaching process to form a range of toxic compounds which can be volatilized. Although trichloroethylene, tetrachloroethylene, and carbon tetrachloride were not officially reported to have been released by Northern Pulp, these VOCs may become airborne through evaporation from pulp and paper wastewater (Soskolne and Sieswerda 2010). Boat Harbour (the mill's effluent treatment facility) may therefore contribute to ambient concentrations of VOCs. The major chlorinated hydrocarbon emitted into the air from bleached kraft pulp mills of concern is chloroform, which is produced by heating a mixture of chlorine and either chloromethane or methane (EPA 1985). However, we address other local and area sources which likely contribute to the observed VOC concentrations, which warrants further investigation. We note that direct links between 1,3-butdiene and vinyl chloride with pulp and paper industries have not been reported in the scientific literature to date.

With regards to the EA statement, "For the carcinogenic inhalation TRVs that were applied, the authors did not adjust these values from the default USEPA target cancer risk level of 1 in 1 million to the target cancer risk level that is current public health policy in Nova Scotia and most other provinces (i.e., 1 in 100,000). Thus, the TRVs for carcinogens cited in the paper should have been ten times higher than indicated. This correction would alter the conclusions of the study substantially in that for the seven VOCs considered, there would be no to negligible exceedances of the TRVs that were applied", their proposal is not relevant given that Health Canada has no guidelines nor process in place that is required for such comparisons. In contrast, the US NATA process is based upon a 2005 scientific risk assessment process well laid out by EPA which established the cancer risk levels to which the ECCC data were compared (Hoffman et al. 2017). Furthermore, it should be stated that our study was published in an excellent, internationally-recognized environmental science peer-reviewed journal (*Environmental Science and Pollution Research*) and met the journal's quality control standards.

To reiterate the value of this study: "*Despite study limitations, this is one of few investigations documenting elevated concentrations of certain VOCs air toxics to be associated with pulp and paper emissions in a community. Findings support the need for more research on the extent to which air toxics emissions exist in pulp and paper towns and contribute to poor health in nearby communities.*" Various recommendations were put forth to improve the rigor and validity of the present study (e.g., a field component consisting of real-time measurements of ambient air toxics; a comprehensive risk assessment to investigate uncertainties that have implications for risk estimates in the present study).

Furthermore, we addressed various limitations and gaps in air quality monitoring, not only locally but nationally, and provided recommendations how air quality management could be improved to support informed public health decisions (e.g., epidemiological research of human exposures to air toxics emissions in the ambient Pictou environment with appropriate considerations, as outlined; more strategically placed air monitoring stations; evaluation of a wide-suite of air toxics) - topics ECCC and applicable stakeholders should consider. To the

contrary and to my surprise, the Granton NAPS station has now been decommissioned. This information vacuum only emphasizes the need for more research on these questions.

In summary, the intent of this pilot study was to address local air quality conditions in a Nova Scotia rural community, which clearly indicates the need for further investigation. Moreover, this pilot study serves as a precursor to gaining awareness, so that government agencies adopt more stringent air quality regulations and monitoring programs to ensure health of all citizens is safeguarded and prioritized.

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APPENDIX E-2

Pilot study investigating ambient air toxics emissions near a Canadian kraft pulp and paper facility in Pictou County, Nova Scotia

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RESEARCH ARTICLE

Pilot study investigating ambient air toxics emissions near a Canadian kraft pulp and paper facility in Pictou County, Nova Scotia

Emma Hoffman[1] · **Judith R. Guernsey**[2] · **Tony R. Walker**[1] · **Jong Sung Kim**[2] · **Kate Sherren**[1] · **Pantelis Andreou**[2]

Received: 15 December 2016 / Accepted: 21 June 2017

Abstract Air toxics are airborne pollutants known or suspected to cause cancer or other serious health effects, including certain volatile organic compounds (VOCs), prioritized by the US Environmental Protection Agency (EPA). While several EPA-designated air toxics are monitored at a subset of Canadian National Air Pollution Surveillance (NAPS) sites, Canada has no specific "air toxics" control priorities. Although pulp and paper (P&P) mills are major industrial emitters of air pollutants, few studies quantified the spectrum of air quality exposures. Moreover, most NAPS monitoring sites are in urban centers; in contrast, rural NAPS sites are sparse with few exposure risk records. The objective of this pilot study was to investigate prioritized air toxic ambient VOC concentrations using NAPS hourly emissions data from a rural Pictou, Nova Scotia Kraft P&P town to document concentration levels, and to determine whether these concentrations correlated with wind direction at the NAPS site (located southwest of the mill). Publicly accessible Environment and Climate Change Canada data (VOC concentrations [Granton NAPS ID: 31201] and local meteorological conditions [Caribou Point]) were examined using temporal (2006–2013) and spatial analytic methods. Results revealed several VOCs (1,3-butadiene, benzene, and carbon tetrachloride) routinely exceeded EPA air toxics-associated cancer risk thresholds. 1,3-Butadiene and tetrachloroethylene were significantly higher ($p < 0.05$) when prevailing wind direction blew from the northeast and the mill towards the NAPS site. Conversely, when prevailing winds originated from the southwest towards the mill, higher median VOC air toxics concentrations at the NAPS site, except carbon tetrachloride, were not observed. Despite study limitations, this is one of few investigations documenting elevated concentrations of certain VOCs air toxics to be associated with P&P emissions in a community. Findings support the need for more research on the extent to which air toxics emissions exist in P&P towns and contribute to poor health in nearby communities.

Keywords Air toxics · Air quality · Volatile organic compounds (VOCs) · Community health · Pulp and paper · Cancer risk

Responsible editor: Constantini Samara

Electronic supplementary material The online version of this article (doi:10.1007/s11356-017-9719-5) contains supplementary material, which is available to authorized users.

✉ Emma Hoffman
emma.hoffman@dal.ca

[1] School for Resource and Environmental Studies, Dalhousie University, Halifax, NS, Canada

[2] Department of Community Health and Epidemiology, Dalhousie University, Halifax, NS, Canada

Introduction

Poor ambient air quality is an increasing global concern with recent revelations that 92% of the world's population is exposed to air pollution levels above the World Health Organization (WHO) air quality guidelines (WHO 2006, 2016; Kelly and Fussell 2015). Ambient air pollution is widely recognized and increasingly associated with a wide range of acute and chronic adverse health effects, including cancer, cardiovascular, respiratory, and mortality outcomes (IOM 2011; Villeneuve et al. 2013; ECCC 2015a). The pathological mechanisms by which these toxic exposures exert their effects are not well understood. WHO highlights the need for research in order to better inform exposure-response relationships (WHO 2016).

Published online: 15 July 2017

Most air pollution surveillance activities are limited to measurement of respirable fine particulate matter ≤2.5 μm ($PM_{2.5}$), without regard to their specific chemical composition and criterion air contaminant (CAC) gases (i.e., nitrogen oxides [NO_x] and ground-level ozone [O_3]). There is growing concern about the toxicity of volatile organic compounds (VOCs) (Cicolella 2008) and the consequences of long-term, low-dose exposure to these agents. VOCs are varied and widespread air pollutants (e.g., hydrocarbons, aromatics, and some chlorinated compounds) that are increasingly recognized as important precursors to $PM_{2.5}$ and ground-level O_3 formation through photochemical reactions (Ryerson et al. 2001). Atmospheric deposition of VOCs may contaminate other environmental media (e.g., soils, sediments, and biota) (ATSDR 2014a; MacAskill et al. 2016). Many VOCs are included in the US Environmental Protection Agency (EPA) "air toxics" list. "Air toxics" are defined as "those pollutants that cause or may cause cancer or other serious health effects […] or adverse environmental and ecological effects" (EPA 2015a).

According to the Canadian Environmental Protection Act 1999 (CEPA), VOC releases are acknowledged as a health concern, but, due to their highly volatile properties, are challenging to monitor and manage (CCME 2011). Although no specific Canadian legislative or regulatory tools address ambient VOC levels, emissions are indirectly controlled through regulatory mitigation of $PM_{2.5}$ and ground-level O_3 under the Canadian Ambient Air Quality Standards (CAAQS). Under CEPA, it is mandatory for owners or facility operators, who meet reporting requirements, to self-report pollutant releases to air, water, and land to Environment and Climate Change Canada (ECCC)'s National Pollutant Release Inventory (NPRI) (ECCC 2014). While this provides a disincentive to those industries releasing these agents, there is less regulatory control or routine monitoring of these agents in Canada which, in turn, limits scientific understanding of sources, exposures, and the effectiveness of current control measures across the country.

Ambient air monitoring in the US is conducted in accordance with the *Clean Air Act* (CAA) (Clean Air Act 1970). CAA amendments identify 187 air toxics, which form the basis for EPA's approach to regulating emissions (EPA 2015a). Of these, EPA identified 30 air toxics that pose the greatest potential health threat in urban areas (EPA 2015b). Although many CEPA-toxic or equivalent agents are monitored by the National Air Pollution Surveillance (NAPS) network, it includes a selection (not all) of EPA's list of prioritized air toxics, and the main criteria for air toxics monitoring in Canada has been their potential contribution to ambient PM and ground-level O_3 (Galarneau et al. 2016). Consequently, there are gaps in understanding of air toxics concentrations across the NAPS network. Using the risk-based principles outlined in CAA, EPA developed the National Air Toxics Assessment (NATA), a comprehensive evaluation tool that prioritizes efforts to regulate emissions of air toxics (EPA 2015c). Such a rigorous initiative has yet to be implemented in Canada, where no federal guidelines exist for ambient air toxics.

Despite economic benefits of the P&P industry, it generates large quantities of atmospheric and effluent emissions, resulting in environmental degradation (Hewitt et al. 2006; Hoffman et al. 2015; Hoffman et al. 2017). P&P mill emissions vary depending on the pulping method, wood species, and by the age and technology used (Soskolne and Sieswerda 2010). P&P mills are industrial emitters of air toxics, although few investigations (e.g., the Nez Perce National Air Toxics Program, funded by EPA [STI 2009]) have characterized ambient concentrations in nearby communities.

Potential adverse health effects associated with exposure to air pollutants in the vicinity and downwind from P&P facilities include respiratory disease, neurophysical symptoms, and higher risks of contracting lung cancer (Soto et al. 1991; Toren et al. 1996; Mirabelli and Wing 2006). Yet, few investigations reported adverse health effects from chronic community-level ambient exposures to P&P mills emissions in Canada (Mirabelli and Wing 2006; Soskolne and Sieswerda 2010). While there have been a number of occupational epidemiological studies of P&P workers, these investigations have not been extended to examine community exposures, due to research design challenges including ecological fallacy (i.e., inferences made about individuals deduced from the population) in community studies (Soskolne and Sieswerda 2010). Additionally, most of these studies focused on respiratory disease outcomes; there is a dearth of epidemiological studies of cardiovascular effects or cancer effects in these communities.

Decades-long concerns for perceived higher incidence and mortality rates for all-cause cancer, cardiovascular disease, chronic respiratory disease, and diabetes (Reid 1989; PCHA 2008; Statistics Canada 2013) in PC, Nova Scotia, have generated considerable community antipathy among residents towards a local P&P mill (Hoffman et al. 2015). This bleached kraft P&P mill ("the mill") is located approximately 3 km south of the town of Pictou (population 3500) and produces approximately 280,000 t of bleached kraft pulp annually from softwood and hardwood chips (NP 2016b) (Fig. 1) and has been in production since 1967 (Ogden 1972). Public backlash gained momentum during 2014 due to the failure of the recovery boiler electrostatic precipitator (i.e., particulate filtration device).

Environmental reporting by the mill, when compared against provincial and federal regulatory compliance standards, contrasted to local perceptions of impacts. Most environmental monitoring reports indicated some

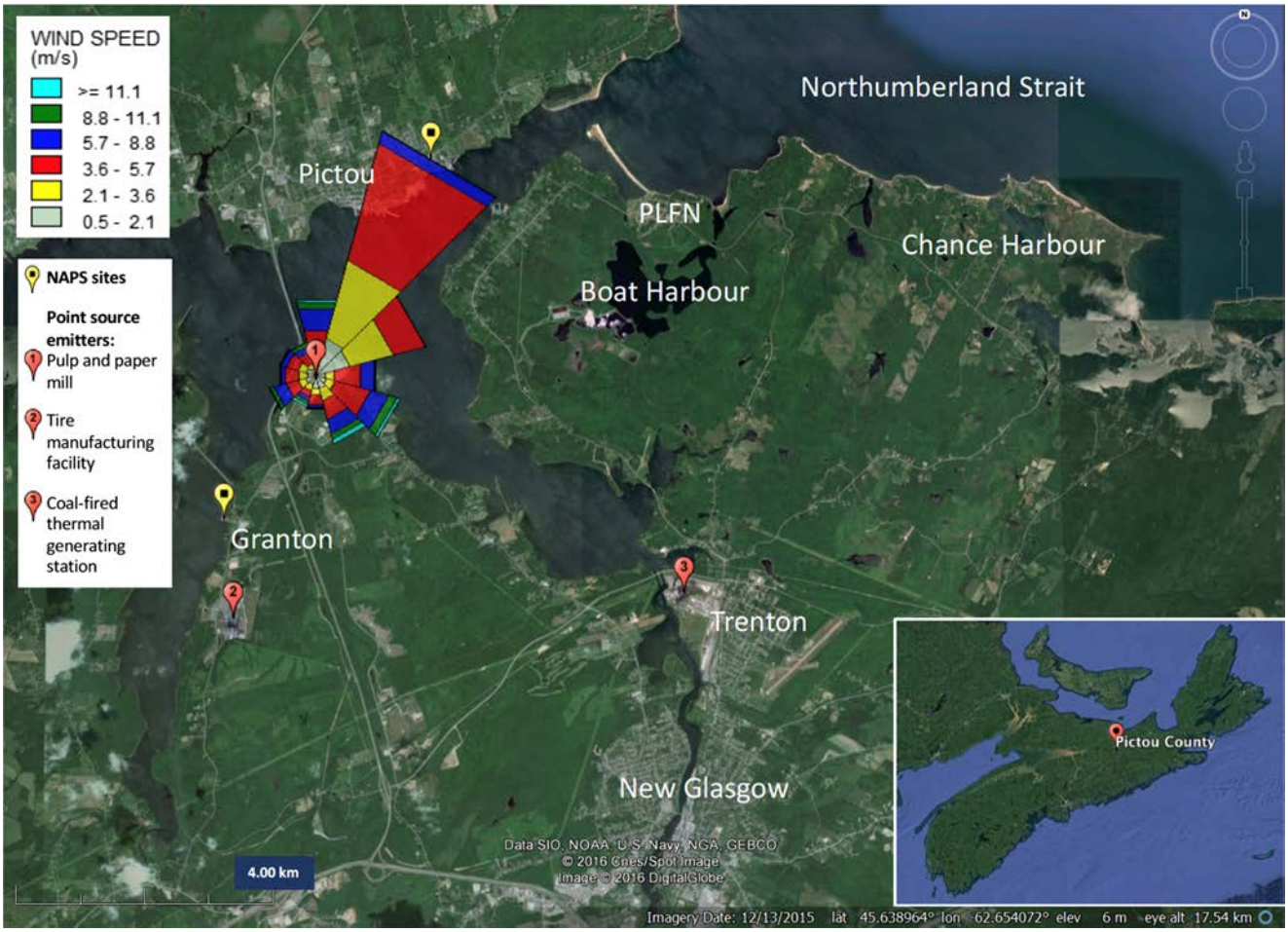

Fig. 1 Summer (2006–2013) wind rose simulation using WRPlot View™ (blowing to direction) with the mill as the focal point (*1*), relative to communities (e.g., Pictou and Pictou Landing First Nation [PLFN]), NAPS discrete receptor sites, and other local point source emitters (e.g., tire manufacturing facility (*2*), coal-fired thermal electrical generating station (*3*)). The length of each radial spoke represents the relative frequency of wind direction (©Google Earth)

level of compliance in atmospheric emissions, but when compliance targets were exceeded, there were inconsistent regulatory enforcement (Hoffman et al. 2015). The mill is required to report emissions to NPRI: conduct third-party stack testing, continuous emission monitoring of total reduced sulfur (TRS), and ambient air monitoring for pollutants found in the Nova Scotia Air Quality Regulations pursuant to the *Environment Act* (NSE 2015). The mill's air emission monitoring data are reviewed by provincial and federal regulators to ensure compliance with applicable environmental permits and air quality objectives (ECCC 2014). Hoffman et al. (2015) provided detailed information on new and existing environmental policies that impose pollution abatement in the P&P industry in Canada, particularly the PC mill (e.g., 2015 Industrial Approval).

These are critical research gaps both in relation to community exposures to VOCs in P&P communities and in regard to adverse health effects resulting from chronic exposure to P&P emissions which are a concern given the potential adverse health outcomes that VOCs and other P&P air emissions pose. This further justifies the need for more research to characterize air quality in this particular subset of industrial communities, which have often been neglected because of their remote locations.

An intensive study of specific ambient air toxic emissions in PC has not been undertaken. The aim of this pilot study was to assess levels of PC community exposures to VOC air toxics emissions from 2006 to 2013, and to evaluate these data in relation to potential risks suggested by EPA air toxic guidelines. The main objective of this study was to determine whether wind direction correlated with prioritized air toxic ambient VOC concentrations at a nearby NAPS site (Granton). As the Granton NAPS site is positioned southwest of the mill, it was hypothesized that prevailing winds (PW) from northerly and northeasterly directions would

positively correlate with an increase in ambient VOC concentrations, as capturing potential VOCs from the mill's plume would be optimized (Fig. 1).

Materials and methods

Spatial and temporal sampling

Historical meteorological and NAPS data from the Granton NAPS site discrete receptor (ID: 31201) were collected from publically assessable ECCC databases (http://climate.weather.gc.ca/climateData/; http://maps-cartes.ec.gc.ca/rnspa-naps/). Hourly surface wind observations (i.e., speed and direction to the nearest 10°) were obtained from the closest EC meteorological station, Caribou Point (45.767° N; 62.683° W), located ~10 km north of the mill (45.652° N; 62.718° W). Temporal data for ambient VOCs monitored at the Granton NAPS station were limited to 2006 to 2013.

Nova Scotia Environment (NSE) operates both NAPS monitoring stations in PC: (i) downtown Pictou (ID: 30901) located 3.5 km northeast of the mill and (ii) Granton (ID: 31201) located 2.5 km southwest of the mill (Fig. 1). The Pictou NAPS site routinely monitors NO, NO_2, NO_x, O_3, $PM_{2.5}$, TRS (not VOCs), and wind characteristics, whereas the Granton site monitors 36 VOC species. Multi-component VOC monitoring at NAPS sites are conducted using canister sampling and gas chromatography/mass spectrometry (GC/MS) (CCME 2011). Sampling of 24 h (midnight to midnight) cumulative ambient air samples ($\mu g/m^3$) are taken on a 1-in-6-day schedule by pumping ambient air into pressurized stainless steel SUMMA® canisters and analyzed by an EC accredited Laboratory (CCME 2011; Galarneau et al. 2016).

Statistical analyses

Variation of meteorological conditions and VOC concentrations were assessed by conducting a spatiotemporal analysis to characterize ambient air toxics emissions in PC from 2006 to 2013. Various analytical methods can be applied to concentration data to estimate source apportionments of air pollutants to provide additional insights into the source/receptor relationships to guide development of more effective air quality management strategies (Hopke 2016). However, given the limitations of having complete VOC data from only one NAPS monitoring station in the region, a full chemical mass balance analysis to identify and apportion sources of atmospheric contaminants were not conducted in this study.

Wind rose plots were generated with WRPlot View™ (©Lakes Environmental Software) to simulate seasonal and spatial variation of wind direction (°) frequency and wind speed (m/s) with the mill as the focal point. Although simplified, wind rose models have proven utility for estimating spatial gradients for fate and transport of pollutants from emission sources (Gibson et al. 2013). Summer, when local people spend more time outdoors (and more vulnerable to outdoor pollution exposure), was a focus of this study (Figs. 1 and 2).

This pilot study was conducted to determine whether ambient concentrations of VOCs exceeded their EPA-associated cancer and/or noncancer risk thresholds, to help identify potential human health concerns in PC. VOCs selected for analysis were based on EPA's list of 30 urban air toxics (EPA 2015b) and *National Air Toxics Trends Station Work Plan Template* (EPA 2015d). Health Canada and the province of Nova Scotia currently do not have specific guidelines for air toxics exposures. Therefore, EPA thresholds were considered a more acceptable standard for carcinogenic exposures in this study. Cancer risk threshold refers to the probability of contracting cancer if exposed to a substance every day over the course of a lifetime (assumed to be 70 years for the purposes of NATA risk characterization). Lower threshold values correspond with higher toxicity. Noncancer risk threshold is associated with effects other than cancer, based on reference concentrations via the "hazard quotient" ratio (HQ; exposure divided by appropriate chronic or acute value) (EPA 2015c). The HQ should not be interpreted as a probability of adverse effects. Noncancer risk thresholds are typically higher compared to cancer risk thresholds, as lower concentrations can elicit a carcinogenic response, whereas other diseases are not triggered until higher exposure thresholds are reached. US and Canadian method detection limits (MDL) are provided (Health Canada 2010; EPA 2015d) (Table 1).

The mill is located approximately 40° northeast of the Granton NAPS site. The selected PW range expected to result in increased VOC concentrations at the NAPS site (±40° either side of the mill [80° total]). A narrower range may be more accurate; however, due to the sample size of VOC samples, the selected range captured more data. All other wind directions (AOWD) represent ranges outside PW (i.e., >80°, <360°). AOWD represent sampling days when no time PW blew from the selected range (i.e., 0 h). VOC concentrations for AOWD were compared to when PW were present for at least 1 h.

Hourly meteorological data were compiled to correspond with ambient VOC sampling. Hourly wind direction within defined PW range (i.e., 360°–80°) was assigned a value of 1; AOWD were assigned a value of 0. Daily totals represented the proportion of time with PW (i.e., 1–24 h) compared to AOWD. Daily

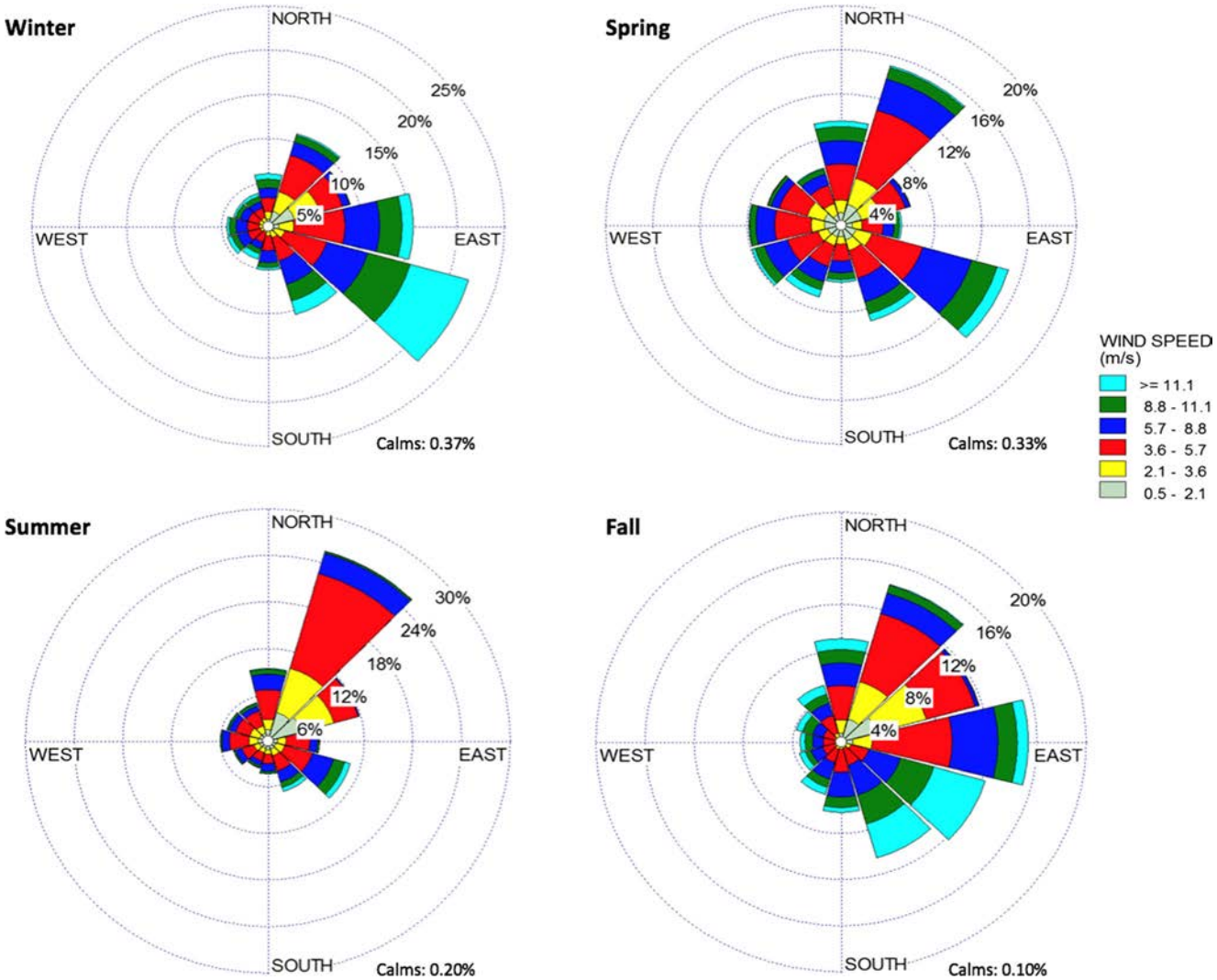

Fig. 2 Seasonal (2006–2013) wind rose simulations using WRPlot View™ (blowing to direction). Percentages represent frequency of wind direction

totals corresponding to VOC sampling were paired. Increasing proportions of PW (AOWD [0 h], ≥1 h, ≥4 h, ≥8 h, ≥12 h) categorized VOC concentrations, which were predicted to correlate with higher VOC concentrations. To test the effect of wind direction and season on ambient VOC concentrations, multivariate analysis of variance (MANOVA) and univariate analysis of variance (ANOVA) were applied in ©R. Due to right-skewed distributions for all VOCs, except for carbon tetrachloride, statistical procedures were performed on both raw and log-transformation of VOC concentrations (Supplementary material Table S1).

Box plots and histograms were used to compare VOC concentrations with PW ≥ 1 h to AOWD on an annual and seasonal basis in relation to their respective cancer and noncancer risk thresholds. See Supplementary material for histograms, and additional box-and-whisker plots illustrating VOC concentrations with increasing time categories with PW (AOWD [0 h], ≥1 h, ≥4 h, ≥8 h, ≥12 h) (Figs. S2 and S4). Box plots display the distribution of data based on a five-number summary: minimum, first quartile, median, third quartile, and maximum. The central rectangle ("box") spans the first to the third quartile (i.e., interquartile range [IQR]). The horizontal line segment within the box represents the median, and "whiskers" above and below the box represent the minimum and maximum. Radar plots consist of a sequence of angular spokes, whose length extending from the center along a separate axis is proportional to the magnitude of the variable relative to the magnitude of the variable across all data points. Lines connect the data values for each spoke. Radar plots were used to display seasonal variation of median VOC concentrations for PW ≥ 1 h and AOWD. One-tailed *t* tests, assuming unequal variance, were performed to determine whether seasonal variation associated with increasing proportions of time

Table 1 List of priority air toxics (i.e., VOCs), associated cancer/noncancer risk thresholds (μg/m^3), and method detection limits (MDL) (μg/m^3) (Health Canada 2010; EPA 2015d)

VOC	Cancer risk[a] (μg/m^3)	Noncancer risk at HQ = 0.1[b] (μg/m^3)	MDL[c] (NATS) (μg/m^3)	MDL (Health Canada) (μg/m^3)
Chloroform	–	9.8	0.50	0.089
1,3-Butadiene	0.0300	0.2	0.10	0.055
Vinyl chloride	0.1100	10.0	0.11	0.046
Benzene	0.1300	3.0	0.13	0.038
Carbon tetrachloride	0.1700	19.0	0.17	0.123
Trichloroethylene	0.2083	0.2	0.20	0.190
Tetrachloroethylene	3.8462	4.0	0.17	0.120

[a] Cancer risk threshold: the probability of contracting cancer over the course of a lifetime (assumed to be 70 years for the purposes of NATA risk characterization). Lower threshold values correspond with higher toxicity (EPA 2015c)

[b] Noncancer risk threshold: the risk associated with effects other than cancer, based on the reference concentration via a ratio known as the "hazard quotient" (HQ; the exposure divided by the appropriate chronic or acute value)

[c] MDL: the lowest concentration that can be detected with confidence. NATA and Health Canada's MDLs are listed for comparison (Health Canada 2010; EPA 2015d)

with PW (i.e., ≥1 h, ≥4 h, ≥8 h, ≥12 h) resulted in a significant increase ($p < 0.05$) in VOC concentrations compared to AOWD (see Supplementary material, Table S3).

Quality control

Standard procedures of the Meteorological Service of Canada have been developed in accordance with internationally recommended procedures established by the World Meteorological Organization (ECCC 2013a). As part of the quality assurance and quality control (QA/QC) program, observational meteorological data are subjected to a computer analysis or review to reveal possible errors. EC and the operating agency are jointly responsible for the NAPS network QA/QC program. Elements of the program include site selection, sampling system requirements, instrument calibration and reference standard requirements, and inter-laboratory testing and performance audits. With few exceptions, analyzers are accorded with EPA designation as either a reference or equivalent method for ambient air monitoring (ECCC 2004). ECCC's air quality laboratories use International Organization for Standardization requirements (i.e., ISO 9001:2008 or ISO/IEC 17025:2005) (ECCC 2013b).

Results and discussion

Meteorological observations

Wind rose simulations illustrate seasonal variability with respect to wind direction, with the mill as the focal point (Fig. 2). During summer, wind blew NNE (25.47%) and ENE (11.80%) directions (aggregate range 15°–75°) towards Pictou and Pictou Landing First Nation (PLFN) (Figs. 1 and 2). Wind blew less frequently towards S-NNW (aggregate range 165°–345°) towards the Graton NAPS site. During winter, wind typically prevailed from the north; the highest frequency (21.99%) blowing ESE, followed by E (15.37%) directions (aggregate range 75°–135°) (Fig. 1). Spring and fall have meteorological characteristics that are similar to summer and winter and were considered transitional periods. Pictou, PLFN, Chance Harbour, Trenton and New Glasgow are communities close to the mill that are downwind of annual PW (range 15°–165°). The Granton NAPS site correlates poorly with seasonal or annual wind directions (Fig. 2).

VOC concentrations

Carbon tetrachloride had the highest median concentration (0.5452 μg/m^3) and vinyl chloride had the lowest (0.0014 μg/m^3) (Fig. 3). Carbon tetrachloride concentrations exceeded its EPA cancer risk threshold (0.1700 μg/m^3) for all samples, with maximum and minimum concentrations of 0.7047 and 0.2892 μg/m^3, respectively. Benzene concentrations exceeded its cancer risk threshold (0.1300 μg/m^3) for most samples, with maximum and minimum concentrations of 1.889 and 0.0266 μg/m^3, respectively. Concentrations of 1,3-butadiene concentrations occasionally exceeded its cancer risk threshold (0.0300 μg/m^3), with maximum and minimum concentrations of 0.1062 and 0 μg/m^3, respectively (Fig. 3). Consequently, 1,3-butadiene, benzene, and carbon tetrachloride were air toxics of primary concern in terms of local

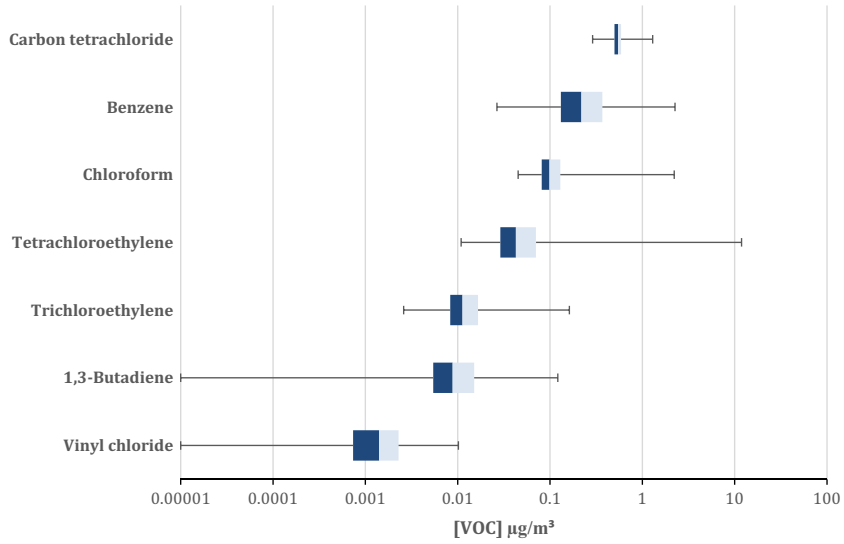

Fig. 3 Relative VOC concentrations (µg/m^3) (2006–2013). Should not be interpreted as orders of magnitude of toxicity. Minimum concentration for 1,3-butadiene and vinyl chloride is 0 µg/m^3 or undetectable

population risk. Other VOCs are presented in Supplementary material (Fig. S5).

ANOVA and MANOVA results revealed that 1,3-butadiene was significantly higher with the presence of PW ≥ 1 h (p = 0.001and p = 0.01 for raw and log-transformed data, respectively). Tetrachloroethylene was also statistically higher with the presence of PW ≥ 1 h (p < 0.01) for log-transformed data. Benzene approached significance with the presence of PW ≥ 1 h (p = 0.07) for log-transformed data. Although not statistically significant, median concentrations of other VOCs, except carbon tetrachloride, were equal or marginally higher with presence of PW ≥ 1 h compared to AOWD. Season had a consistent significant effect on VOC concentrations, except chloroform and tetrachloroethylene (Supplementary material Fig. S2 and Table S1).

Box plots combined with radar graphs illustrate seasonal variation of VOC concentrations of primary concern (i.e., 1,3-butadiene, benzene, carbon tetrachloride) under PW ≥ 1 h and AOWD conditions (Figs. 4, 5, and 6). Median VOC concentrations associated with PW ≥ 1 h and AOWD display parallel

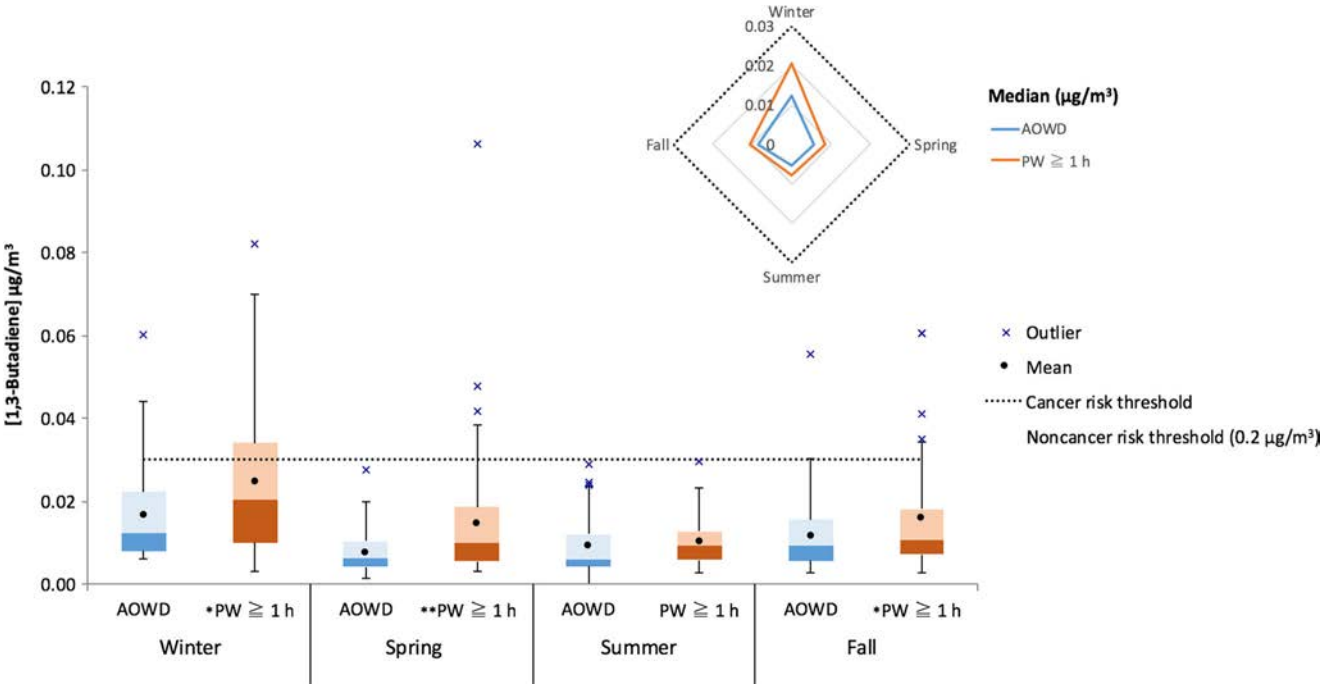

Fig. 4 Seasonal variation (2006–2013) of [1,3-butadiene] (µg/m^3) comparing AOWD to PW for at least 1 h on sampling days (i.e., 360° − 80°), relative to associated cancer and noncancer risk thresholds. Minimum concentration is 0 µg/m^3 or undetectable. Significant differences indicated as *<0.05; **<0.01

seasonal trends. Concentrations of 1,3-butadiene (Fig. 4) and benzene (Fig. 5) exhibit seasonal variation. Both have evidently higher concentrations during winter, with summer having overall lowest concentrations. In addition to exceeding cancer risk thresholds, *t* test results revealed that 1,3-butadiene and benzene concentrations were significantly higher with the presence of PW ≥ 1 h compared to AOWD during at least two seasons, including spring and fall. Conversely, median carbon tetrachloride concentrations showed little variation (Fig. 6). Regardless of season or wind direction, all carbon tetrachloride samples exceeded its associated cancer risk.

This pilot study presents findings of a secondary analysis of 8 years of air toxic VOC exposure data associated with ambient air quality in a Canadian P&P town. Concentrations of three ambient outdoor air toxics routinely exceeded EPA air toxics-associated cancer risk thresholds and are consequently of primary health concern in relation to population health risk in PC: 1,3-butadiene, benzene, and carbon tetrachloride. Exceedance in cancer risk thresholds for these air toxics is consistent in the literature (e.g., Morello-Frosch et al. 2000). The extent to which threshold exceedances of 1,3-butadiene adversely affect human health is poorly understood, with little toxicity information available to compare with cancer risk estimates (Morello-Frosch et al. 2000). With respect to benzene exposure, most monitoring data are associated with occupational studies (ATSDR 2007a), where long-term exposure can cause leukemia (ATSDR 2007b). High exposure to carbon tetrachloride can cause liver, kidney, and central nervous system damage (ATSDR 2005). Combinations of air toxics may have additive or synergistic adverse health effects (Morello-Frosch et al. 2000). Therefore, exposure to mixed VOCs might pose health risks to facility employees and neighboring residents (An et al. 2014; He et al. 2015).

Emission sources within the defined PW range, N to ENE of the Granton NAPS site, may be a causal factor for the increase in VOC concentrations, except carbon tetrachloride. The largest point source emitter within this range is likely the mill; however, the origin(s) of VOCs are inconclusive. According to the mill's most recent substance report submitted to NPRI in 2012, 143.18 t of VOCs were atmospherically emitted on-site (ECCC 2012). An estimated 3.195 t of benzene were released to the air from a stack higher than 50 m and 0.022 t were released within 50 m of the ground. Additionally, benzo(a)anthracene and of benzo(a)phenanthrene were emitted to the air (9.7 and 6.7 kg, respectively) and deposited on-site (0.753 and 0.142 kg, respectively) (ECCC 2012). Although trichloroethylene, tetrachloroethylene, and carbon tetrachloride were not reported to have been released, they may become airborne through evaporation from P&P wastewater (Soskolne and Sieswerda 2010). Boat Harbour (the mill's effluent treatment facility) may therefore contribute to ambient concentrations of VOCs. Collectively, these emissions may have contributed to the ambient atmospheric levels of VOCs measured at the Granton NAPS site. While

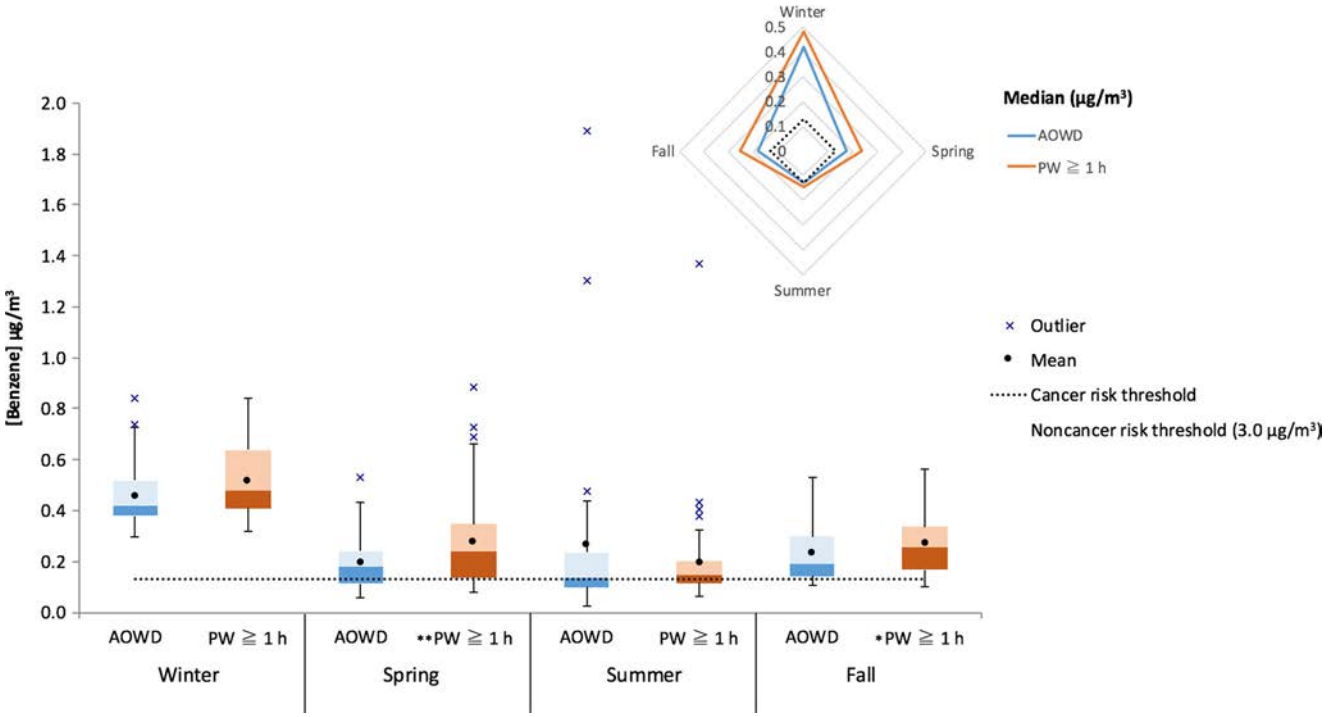

Fig. 5 Seasonal variation (2006–2013) of [benzene] (µg/m^3) comparing AOWD to PW for at least 1 h on sampling days (i.e., 360° − 80°), relative to associated cancer and noncancer risk thresholds. Significant differences indicated as *<0.05; **<0.01

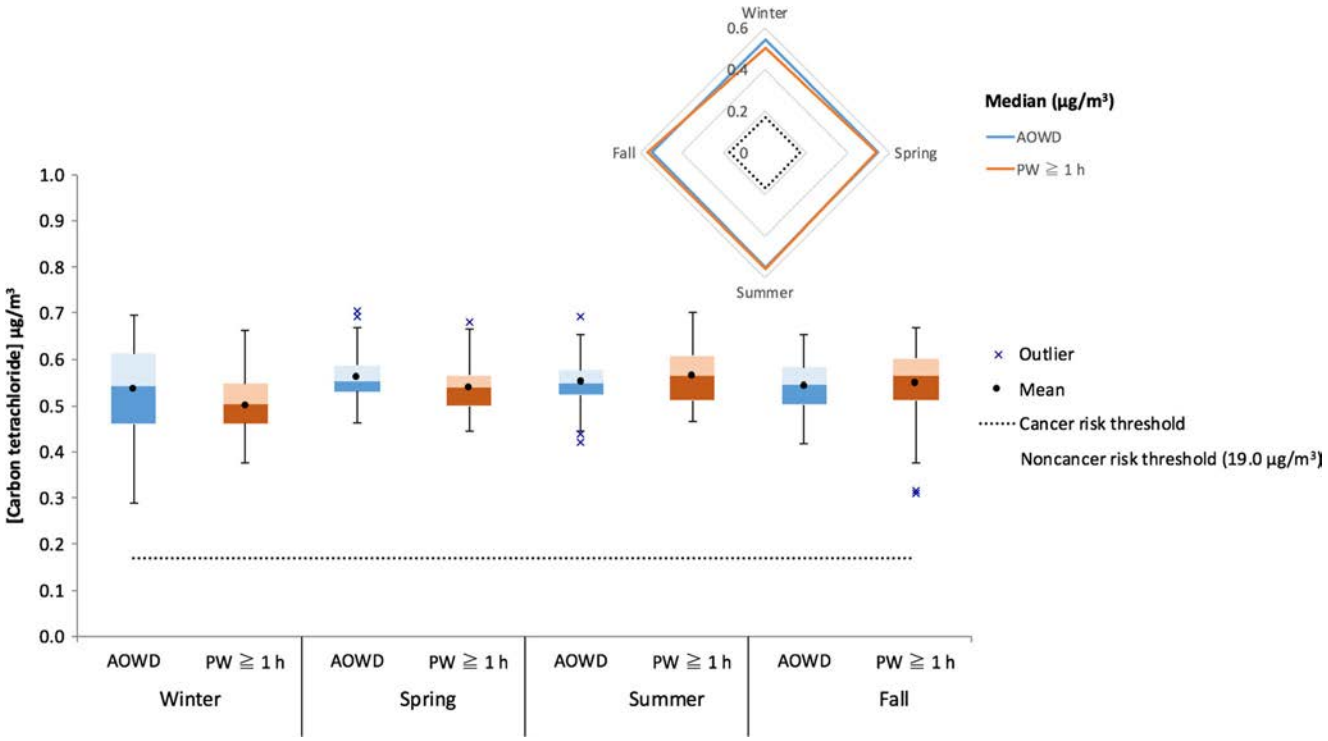

Fig. 6 Seasonal variation (2006–2013) of [carbon tetrachloride] (µg/m^3) comparing AOWD to PW for at least 1 h on sampling days (i.e., 360° − 80°), relative to associated cancer and noncancer risk thresholds

NPRI provides detailed information on pollutant releases, data are self-reported by facilities, with no audits to ensure accuracy. Consequently, data quality may be compromised.

The major chlorinated hydrocarbon emitted into the air from bleached kraft pulp mills of concern is chloroform, which is produced by heating a mixture of chlorine and either chloromethane or methane (EPA 1985). Although chloroform is a recognized by-product of the chlorination process in the P&P industry, it has been suggested that up to 90% of total emission sources may be natural in origin and is widely dispersed in marine environments (McCulloch 2003). As PC is located along the coast of the Northumberland Strait, marine environments may have contributed to the observed ambient chloroform concentrations (see Supplementary material).

Results implicate the mill as a source of air toxics (particularly 1,3-butatdiene and tetrachloroethylene); however, other local sources likely contribute to air toxics emissions. Area and mobile sources have been reported to largely contribute to concentrations of 1,3-butadiene (ATSDR 2014b) and benzene (ATSDR 2007a). Because the Granton NAPS site is located near a highway and access roads, vehicle emissions may have contributed to the observed concentrations of these compounds. A coal-fired thermal generating station and a tire manufacturing facility (located 7 km E and 1.5 km S from the Granton NAPS site, respectively) may be other local point source emitters of VOCs (e.g., 1,3-butadiene is used to make synthetic rubber [ATSDR 2014b]) (Fig. 1). According to the latest NPRI substance reports: the tire manufacturing facility released 220 t of atmospheric VOCs, whereas no VOC releases were reported by the thermal generating station (ECCC 2015b), despite that coal combustion is a significant contributor (Chagger et al. 1999). Direct links between 1,3-butdiene and vinyl chloride with P&P industries were not found in the literature.

Major monitored pollutants at the mill include NO_x, sulfur dioxide (SO_2), and total PM ([TPM] upper size limit of 100 µm diameter) (NP 2016a). A 2013 study concluded that $PM_{2.5}$ concentrations were highest (0.88 µg/m^3) downwind from the mill from using an AERMOD atmospheric dispersion model (Gibson et al. 2013), though this investigation used Halifax wind speed and direction meteorological data (130 km to the south). Hoffman et al. (2015) reported an analysis of 2013 data showing that when Pictou is downwind of the mill, average 1 h ambient $PM_{2.5}$ concentrations result in a twofold increase (12.96 µg/m^3), compared to all other wind directions (5.73 µg/m^3), suggesting the mill is likely the primary contributor of ambient $PM_{2.5}$ in the community. Additionally, TRS, TPM, $PM_{2.5}$, and coarse particulate matter ≤10 µm (PM_{10}) emission exceedances at the mill during 2012 were two to three

orders of magnitude higher than five similar Canadian kraft P&P mills; however, VOC emissions were comparable (Hoffman et al. 2015).

A comparable ambient air toxics monitoring study of a P&P community was conducted in the metropolitan Lewiston, Idaho area and the Nez Perce Reservation (STI 2009). Findings revealed that concentrations of formaldehyde and acetaldehyde were much higher than expected relative to Lewiston's size. Chloroform, tetrachloroethylene, and trichloroethylene were highest at monitoring sites nearest the mill, which presumably contributed at least 50% to pollutant concentrations (STI 2009). However, due to insufficient information on local concentrations of anthropogenic and biogenic VOCs, it was inconclusive whether the mill was a causal factor.

Recent (2009–2013) measurements of ambient CEPA-toxic or equivalent agents monitored at NAPS sites revealed that 11 air toxics, including benzene, chloroform, trichloroethylene, and tetrachloroethylene exceeded ambient air quality guidelines set by respective Canadian jurisdictions (Galarneau et al. 2016). An additional 16 air toxics approached guidelines. Although these guidelines are not necessarily enforceable, CEPA outlines provisions for toxic compounds and are thus subject to risk management actions. Air toxics' contribution to poor health on a regional and national scale has not been thoroughly investigated; therefore, calls into question the effectiveness of current toxic substance management in Canada.

Nova Scotia is known as the "tail pipe of North America," due to being within the trajectory of long-range transport of emissions from transboundary sources along the Eastern Seaboard, plus central and eastern Canada (NSE 2014). Background levels of air pollution that originate from resuspension and natural sources has been found to be major contributors to concentrations of carbon tetrachloride and benzene (Morello-Frosch et al. 2000). Background levels, in combination to carbon tetrachloride's capacity to persist in the atmosphere for a least a year, may explain why observed concentrations are consistently above its associated cancer risk threshold at the Granton NAPS site, regardless of wind direction.

Atmosphere circulation plays a complex role in dispersion, transformation, and removal of pollutants. The dispersion of pollutants from source emitters (e.g., smokestacks) is affected by crosswind mixing in both horizontal and vertical directions. Meteorological variables, including wind speed, wind direction, temperature, humidity, precipitation (process of removal), and atmospheric pressure are the main drivers of variation in pollutant concentrations and dispersion (Bates and Caton 2002). Furthermore, gravitational settling is important for pollutants with larger molecular weights (Oliver 2008); heavier particles settle or deposit closer to emission sources (Walker et al. 2003a, 2003b). Gravitational settling may also explain the high concentrations of carbon tetrachloride.

Topography and coastal conditions can affect wind characteristics (e.g., direction, speed) and the behavior of pollutant transport. A sea breeze that is trapped under descending warmer air from land can exaggerate conditions at coastal zones, a phenomenon known as coastal inversion (Bates and Caton 2002). In addition, turbulent winds along the coast may influence wind characteristics at the Caribou Point meteorological station, and the fate and transport of pollutants. Such coastal conditions, in combination with transboundary air pollution, may be occurrences that coastal areas experience in Nova Scotia, including PC.

Seasonal variability

Seasonal variability exists for both meteorological conditions and VOC concentrations. Variations in meteorological conditions, the nature and intensity of emissions from nearby sources, and photochemical activity are factors that could have led to the observed seasonal variability of outdoor VOC levels (Al-Khulaifi et al. 2014). Of the three VOCs considered particular concern in this study, 1,3-butdaiene and benzene exhibited the highest concentrations during winter. Photochemical reactions involved with ground-level O_3 formation are catalyzed by ultraviolet radiation and temperature. Therefore, peak ground-level O_3 levels typically occur during warm days with sufficient sunlight exposure; thus, people are more vulnerable to exposure during summer. The opposite is true during winter, when available light is diminished, and temperatures are colder (ATSDR 2014b).

Demographic behavior and technological improvements that aim to mitigate emissions (e.g., smokestack precipitator installation in 2015) also need to be considered when evaluating pollutant concentrations. For instance, households in the Atlantic provinces are heated primarily with oil, electricity, and wood or wood pellets (Statistics Canada 2011); therefore, as residential heating increases during winter, biogenic VOCs (e.g., benzene [ATSDR 2007a], 1,3-butadiene [ATSDR 2014b]) from wood burning may have contributed to higher concentrations of these compounds observed in this study. As the mill operates on a 24/7 schedule (ECCC 2012), atmospheric VOC emissions were assumed consistent throughout the year.

Implications

Location of ambient air quality monitoring stations has a direct impact on the observed concentrations of pollutants

(Craig et al. 2008). Based on the time series and spatial analyses, wind direction appears to play a key role in the Granton NAPS site's ability to monitor ambient VOCs from the mill. PW ≥ 1 h from the selected range (360°–80°) typically resulted in equal or higher VOC concentrations for all compounds, except carbon tetrachloride, compared to AOWD (Figs. 4, 5, and 6; Supplementary material), suggesting that the mill is likely a causal factor. Furthermore, as there is a higher frequency of northerly winds blowing towards the south during winter (Fig. 2), the Granton NAPS site is more likely to capture ambient pollutants from the mill's atmospheric emissions. Southwest PW blowing towards Pictou dominate during the summer months when people are more vulnerable to ambient air pollution exposure. Due to Pictou's geography, air toxics from the Eastern Seaboard in combination with local emission sources, including the mill, converge there; hence, higher concentrations of VOCs are expected in Pictou during summer. Subsequently, southwest PW are expected to result in lower VOC concentrations at the Granton NAPS site, as capturing the mill's atmospheric emissions would not be optimized. Therefore, VOC concentrations at the Granton NAPS site during winter would likely be representative of ambient VOC concentrations in Pictou during summer. Moreover, Pictou's considerably larger population base compared to the rural area of Granton further confirms that the NAPS site is not strategically positioned to accurately represent ambient levels of air toxics where there is higher residential exposure.

Study limitations

This study only evaluated exposure to ambient VOC air pollutants. Human exposure to air pollution is a combination of both outdoor and indoor environments and varies according to daily activity patterns and the conditions of specific settings. Secondary data analysis was used in this study; therefore, the ecological nature of these findings limit the explicit attribution of ambient air toxic exposures to the risk potential for cancer for community residents. Personal exposure monitoring, more detailed spatial analysis of ambient conditions, and source apportionment studies would be required to establish more explicitly the health risk associated with these exposures. The analysis was limited by the inability to examine the interaction of local meteorological conditions. Meteorological data were retrieved from Caribou Point, located approximately 10 km from the mill; consequently, coastal conditions may cause differences in meteorological measurements between sites.

Future research and monitoring

A *field component* consisting of real-time measurements of ambient air toxics would improve the rigor and validly of the present study. Although labor intensive, air toxics samples can be analyzed with a high degree of accuracy (Craig et al. 2008). Because monitoring stations are typically fixed, government-approved *atmospheric dispersion modeling* that considers landscape dynamics and seasonal meteorological variability (e.g., ©CALPUFF, ©AERMOD) would more accurately estimate spatial patterns of air toxics dispersion, and human exposure at the population or individual level (EPA 2013). This would require numerous stations within the community so would likely only be feasible for a specific research investigation. Further, installation of a new precipitator in 2015 has likely changed in ambient conditions. A follow-up assessment would provide a comparison to these findings to determine if VOC levels have improved. Additional research includes applying a *Conditional Probability Function* to calculate the probability that an air pollution source is located within a particular wind direction sector to help determine direction of a source from a NAPS discrete receptor site, and conducting an analysis of the effect of mixing height on measured VOC concentrations to further investigate seasonal patterns.

Investigation of health outcomes might involve longitudinal epidemiological research of human exposures to air toxics emissions in the ambient Pictou environment with appropriate consideration for latency of health outcomes, while controlling for indoor and occupational sources and other contextual factors. Several recent Canadian nationwide cohort studies that may provide a foundation for such investigations have been described (e.g., Crouse et al. 2012).

A *comprehensive risk assessment* investigates uncertainties that have implications for risk estimates in the present study, including those surrounding toxicity information (Morello-Frosch et al. 2000). More research is required to determine what cancer and noncancer risks are from ambient air toxics exposure. Further, it is important to consider synergistic effects of a full suite of ambient pollutants, and physical and chemical processes involved in fate and transport of these compounds. Comprehensive emission inventories are necessary to thoroughly address (i.e., characterize, model, and manage) air quality issues (CEC 2009). Collectively, these research efforts aim to better inform air quality management, composed of federal (e.g., ECCC, Health Canada) and provincial (e.g., NSE, Nova Scotia Department of Health and Wellness) government and public health agencies, how best to proceed to ensure the health of residents in industrial communities is prioritized. Implications of the current findings warrant further investigation.

Given the contribution emissions from local sources have to regional, national, and global airsheds, local mitigation initiatives should be an integral part of air quality strategies. There is no common approach to assess health effects of a mixture of pollutants, as they tend to be site specific; hence, an assortment of effective measures may be required. Case studies that provide evidence of effective of air quality

management interventions and guidance documents for risk managers may help inform air quality management for stakeholders (Craig et al. 2008).

To address potential adverse health effects associated with degraded air quality, Health Canada, the Public Health Agency of Canada (PHAC) and provincial partners might work collaboratively with local stakeholders to mitigate health risks and improve efficient industrial technology, while balancing economic, political, and social factors in development and implementation of air quality management. Mitigating industrial emissions has beneficial outcomes for wellbeing (Clougherty 2010); environmental stewardship and governance fosters a more proactive and cleaner environment, while building trusting relationships between industrial stakeholders (Pascal et al. 2013). "A comprehensive enforcement program with mandatory reporting of emissions, […] and meaningful penalties for noncompliance ensures that emission standards are being met" (Craig et al. 2008), and facility operators are held accountable. Data collected internally by the mill is not readily available. To improve transparency, siting rationale for air quality monitoring stations and accompanying data should be provided as part of a commitment to corporate responsibility of the mill (Hoffman et al. 2015).

To improve air quality conditions, stakeholders could increase the capacity for surveillance, assessment, and response to air quality. Furthermore, evaluation of a wide-suite of air toxics, including NATA compounds not measured by the NAPS network (particularly prioritized air toxics) would contribute to ensuring that air quality in Canada is adequately studied. Therefore, ECCC and NSE should consider implementation of a long-term monitoring program for priority air toxics that is comparable to the NATA network monitored by EPA to characterize air toxics exposure on local, regional, and national scales. Data will be useful to help mitigate emissions and achieve acceptable air quality standards that do not exceed cancer or noncancer risk thresholds.

ECCC should also consider the feasibility of installing and maintaining additional strategically placed NAPS sites to improve air pollution evaluation in both rural and urban areas, as well as in microenvironments (e.g., near point source emitters, high-traffic areas) (Craig et al. 2008). More effective communication of the results is required to increase transparency among stakeholders, including the public (Hoffman et al. 2015). Based on the population's risk of exposure, it is strongly recommended that ambient air toxics monitoring to be incorporated at the established NAPS station in Pictou to optimize capturing of said air toxics, and to best correlate pertinent results. Additionally, atmospheric dispersion modeling should use local meteorological data; therefore, meteorological data should also be collected concurrently at NAPS sites to help identify source emitters.

Measurement of individual VOC compounds is necessary to provide insight into their contribution to $PM_{2.5}$ and ground-level O_3 formation. Data would be useful to help target large source emitters and aid regulatory enforcement. Establishment of stringent and/or adapted air quality standards that encompass more air toxics (e.g., VOCs) fosters strong public support and political engagement to address air quality issues. Moreover, health impacts associated with background air pollution should be estimated. Air quality management programs are human resource intensive; therefore, they must have clear and feasible short- and long-term objectives. These initiatives gain predictive insights on atmospheric chemistry, and engage and support relevant sectors in the development and implementation of policies to reduce health risks associated with air pollution exposure (Craig et al. 2008).

Conclusions

Findings reveal that 1,3-butadiene, benzene, and carbon tetrachloride exceeded their respective cancer risk thresholds and are of primary health concern in terms of population risk. Results highlight associations with wind direction and the Granton NAPS site's ambient VOC concentrations in relation to location of the pulp mill. Compared to AOWD, PW from the selected range (360°–80°) typically resulted in higher VOC concentrations for all compounds, except carbon tetrachloride, suggesting that the mill is likely a contributor to increased concentrations. In addition, there are clear seasonal variations of meteorological conditions and VOC concentrations. Southwest PW blowing towards Pictou dominate during summer months, when people spend more time outdoors, and consequently are exposed to higher concentrations. Due to Pictou's geography, air toxics from transboundary and local sources may converge in summer, resulting in higher VOC concentrations. Findings suggest the Granton NAPS site is not positioned to accurately represent ambient levels of toxicity in PC. Therefore, ECCC and NSE should consider incorporating ambient air toxics (e.g., VOCs) monitoring at the established Pictou NAPS site where there is higher residential exposure.

Future research will provide air quality management with a comprehensive characterization of air toxics to support informed public health decisions. Moreover, this pilot study may serve as a precursor to gaining awareness, so that government agencies adopt more stringent air quality regulations and monitoring programs to ensure health of citizens is safeguarded and prioritized.

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APPENDIX F-1

Comments regarding the Northern Pulp, Nova Scotia *Environmental Assessment Registration Document. Replacement Effluent Treatment Facility*

Margaret (Meg) Sears, M.Eng., PhD
RR 1, 107 Mast Ln
Dunrobin, ON
K0A 1T0
Email: MegSears@ncf.ca
Telephone: 613 297-6042

March 8, 2019

The following are comments with regard to the Environmental Assessment (EA) Registration documents submitted in support of the Northern Pulp effluent treatment proposal.

By way of introduction, please find my curriculum vitae attached to this report. I am trained in chemical engineering, applied chemistry and biochemical engineering (doctoral level). I have subsequent experience examining toxicants in environmental and human health, and regulatory issues; clinical epidemiology / medical research; research specifically into arsenic, cadmium, lead and mercury; and have published in the peer-reviewed literature on biosorption of toxic metals, as well as health effects of toxicants and medical approaches to environmentally-linked disease in clinical practice. I have examined environmental toxicants and human health in a number of situations and locations, ranging from chemicals used at CFB Gagetown, to air quality in the Peace River district of Alberta. I have also worked in modelling enhanced oil recovery, which is similar to but more complex than hydrology, and is relevant in consideration of groundwater flows.

My comments regarding the current Northern Pulp EA are limited due to the short timeframe within the selected Nova Scotia process. I would have provided a more fulsome review of a more comprehensive selection of topics, if more time had been available. I am focusing on what I consider to be some important gaps and shortcomings in the information provided, and the issues considered. In the present case, omission by NPNS and its consultants of topics for consideration, and presentation of incomplete or unreliable information can lead to under-estimation and lack of consideration of potential harms. Decision-making on this EA, in the absence of a more complete submission from NPNS, could result in missed opportunities to make wiser choices and to avoid future harms and liabilities, and could result in further (potentially preventable) future harms.

In response to the EA Registration documents, I herein:

1. comment briefly on section 9, pertaining to scoping of a potential future human health review;

2. address implications and reports of specific toxicants (mercury, cadmium, reduced sulphur compounds [RSCs] and polyaromatic hydrocarbon [PAH] chemicals, including chlorinated dioxins and furans); and in this context
3. broaden consideration to include sensitive and directly applicable human and environmental health indicators and effects.

In summary, the current Environmental Assessment lacks:

- Data and analysis regarding some unmentioned and unaddressed hazards, particularly mercury, associated with the Canso Chemicals Ltd. legacy landfill as well as mercury spilled into bedrock beyond the landfills. This includes an underground mercury plume, and related risks to drinking water and the surrounding environment, biota and all who may consume affected foods. Precise locations are unclear and the Canso legacy is not mentioned in EA Registration documentation, but the landfill abuts or overlaps the proposed effluent treatment facility;
- Sufficient information to establish baseline environmental conditions in terms of mill operations and emissions (in part because the oxygen delignification equipment, a key component promised to the public, and important for pollution reduction, is not yet in place), as well as contamination of the environment, ecosystems and foods;
- Sufficient information to establish baseline health and contamination of the surrounding lands, water, biota and populations.

In the absence of necessary data and analyses, the current EA is incomplete. In the absence of information, the Minister cannot make a decision with confidence that the proposed Effluent Treatment Plan will not result in serious harms to the environment, or to people living and working in the area. The Minister cannot ensure that necessary baseline data and surveillance are in place for early alerts to increasing toxicants or ecological changes. Any approvals would appear to be ill-informed and premature at best.

Section 1. Comments on EA registration document section 9, regarding scoping of a potential human health risk assessment (HHRA), or a human health evaluation (HRA)

Section 9 of the EA registration document, "Human Health Evaluation" is 31 pages long, describing what a human health risk assessment (HHRA) might consider, and arguing for exclusion of considerations on the basis of supposed irrelevance. Should a HHRA be required in the future, this section argues for a narrow scope in terms of:

- chemicals of potential concern;
- populations;
- potential effects related to the proposed waste water treatment project; and
- issues that arose historically.

NPNS is seeking an overly narrow scope for the assessment. This approach is not acceptable, as it would fail to address significant issues, including but not limited to:

- scoping is argued in the context of a proposed mill (Toxikos, 2006), that did not have the history of and location on a site with pre-existing toxic chemicals (this mill was never built);
- the fact that air emissions and effluent composition will not be known with certainty until after the NPNS mill equipment is updated, new equipment is installed, and burning of sludge is implemented;
- lack of analyses of anticipated sludge composition, and effects on air emissions of toxic metals and polyaromatic hydrocarbons (PAHs);
- omission of the foetus and pregnant mother as a population of concern;
- the fact that historical issues such as mercury, and PAH (including dioxin and furan) contamination will continue for extended periods of time; and
- contaminant analyses of foods available on the land and in the waterways and ocean.

Section 2. Comments on EA registration document sections reporting environmental contaminants mercury, cadmium, reduced sulphur compounds and polyaromatic hydrocarbons (including dioxins/furans)

Mercury

Mercury, including methylmercury, is an established developmental and neurological toxicant, and is recognized as a global pollutant for action under the Minimata Convention. Canada has ratified the Minimata Convention. Methylmercury, the common form of organic mercury, is formed via microbial action, under oxygen-poor or anaerobic conditions such as in wetlands and sediments. Methylmercury is lipophilic ("fat loving"), so it accumulates in fatty tissues. Methylmercury in fish and other aquatic organisms poses risks to fishers, and individuals and communities relying upon these foods. Other forms of mercury (vapour and salts) are also highly toxic.

Mercury was historically essential to the process to generate the chlorine and sodium hydroxide that were used to bleach pulp, in "chor-alkali" plants. Mercury pollution is well known for being associated with chor-alkali plants at Canadian pulp mills. Mercury in waterways such as the English-Wabigoon river system in Ontario has debilitated generations of Aboriginal peoples.[1] Most chlor-alkali plants were closed during the 1970s,[2] but Canso Chemical Ltd. operated through the 1980s.[3] Appendix I1-B in the EA Registration package indicates that with the closure of Canso in 1992, "Generation of mercury and dioxins & furans from chlor-alkali process ceases." Unfortunately, concerns regarding these persistent toxicants linger to this day.

Mercury contamination exists at the Northern Pulp Site
A Statutory Declaration by John Daniel Currie, dated July 8th, 1997, reveals that Canso Chemicals Ltd. operated a chlor-alkali plant and associated landfills at Abercrombie Point, on a property approximately 23.29 acres in size, abutting the Northern Pulp Mill site and immediately adjacent to the site of the proposed Effluent Treatment Facility. The Canso Chemicals Ltd. Decommissioning Final Report (Attention Mr. Dan Currie, January 26, 2000) indicates that mercury infiltrated the bedrock more than eight metres below the room where the mercury cells produced chlorine gas and alkaline water. Thus, substantial quantities of mercury apparently remain on the site, in landfills created to hold wastes during operation and subsequent demolition, on the site of the demolished Canso buildings, and in the bedrock.

The location, condition and contents of the mercury-containing Canso site would have to be confirmed, to avoid further releasing mercury into the local environment during construction of the new effluent treatment facility, via disturbance of landfills, or as a result of alteration of groundwater flow and drainage. Groundwater in the area should be monitored, with a view to intercepting mercury contamination as it spreads.

The Decommissioning Report speculated (perhaps optimistically) that the mercury plume would eventually (in perhaps, approximately 200 years) emerge underwater in Pictou Harbour. That said, it is acknowledged that the mercury might surface sooner, closer to its source, potentially affecting the shoreline and wetlands, and presumably groundwater quality en route. **Vagaries of fractured bedrock make it virtually impossible to predict mercury migration, so to protect neighbours' drinking water and to anticipate impacts on the environment and marine food sources, it is important to investigate migration of the mercury plume, and biological receptors within the landscape that may accumulate mercury (e.g., wetlands species and shellfish).** This should be an essential component of any EA process.

Importantly, albeit without having examined existing detailed geological investigations should they exist, I wish to flag the possibility that the mercury plume may intercept wells in the neighbourhood. If the plume intercepts one well, there is potential for contamination of several wells, via contamination of any aquifer that is common among wells. With fractured bedrock, it is not necessarily the case that the closest wells would be affected first. Hydrogeological testing and well water testing would have to be carried out.

Wherever the mercury plume encounters pulp mill effluents or organic materials, methylmercury might be formed.

Among dozens of metals and other chemicals, mercury and methylmercury were *not* measured in the assessment (Appendix M4 – Surface Water Data, Dillon). It is surprising that mercury was omitted from the report, as mercury (and

methylmercury) would typically be a standard element in a large panel of environmental analyses such as is reported in Appendix M4.

Lack of analyses of mercury species is a serious omission, given that:

- **mercury is an important toxicant in any environmental assessment of an old pulp mill that previously hosted a chlor-alkali plant on site. Contamination may spread into ground- and surface water, and may enter effluent during treatment;**
- **methylmercury is a well known contaminant of aquatic species, posing risks throughout the food chain. Populations along the coast may be high consumers of these foods, posing risks to all ages and particularly to unborn children;**
- **there is a history on this site of mercury contamination remaining in landfills and the bedrock, from Canso Chemicals Ltd. Mercury contamination is documented in the immediate area of the proposed effluent treatment facility. The current status of this mercury plume is undisclosed. Disturbance of mercury containing landfill, soils and rock during construction and operation may accelerate toxicant mobilization and amplify harms to environmental and human health;**
- **there was a well publicized, third party report of mercury in the immediate area (e.g., https://www.capebretonpost.com/news/local/report-shows-heavy-metals-in-pulp-mill-effluent-7486/);**
- **as the regulator, and recipient and custodian of all documentation, Nova Scotia Environment should be well aware of this history; and**
- **Dillon Consulting prepared both the final decommissioning report for Canso Chemicals Ltd., as well as the EA documents for Northern Pulp, so clearly should have been aware of the issue.**

Cadmium

Cadmium is a highly toxic heavy metal that occurs naturally, and can be accumulated from the soil by plants. Cadmium is at elevated concentrations in pulp mill effluent, because the naturally occurring element is concentrated from high volumes of biomass. Cadmium also bioaccumulates in liver, kidney and bone, building up with age. Toxicologically, cadmium is somewhat similar to, but much more potent than lead (environmental quality standards for water and air are typically 10 to 100 fold lower for cadmium than for lead). According to the International Agency for Research on Cancer (IARC) cadmium is a probable human carcinogen.[4] Among numerous other health effects, cadmium is toxic to the developing child, affects multiple organ systems including the kidneys, liver, and cardiovascular system, disrupts the endocrine (hormone) system, and weakens bones.[5,6,7,8]

Cadmium may accumulate in wildlife, including fish and crustaceans, and is the reason for consumption restrictions for wild foods such as shellfish, and organ meats. [9,10]

It is claimed in section 9 that cadmium has not been detected in NPNS emissions. The spreadsheet of 2017 analyses of water from the Boat Harbour Raw Effluent Ditch, (author Wayne Williams), indicates that the detection limit for cadmium was 0.01 µg/L, which is identical to the provincial ecological criterion for surface water. Thus, the cadmium analyses would not detect levels below the ecological criterion. In fact, cadmium may be of limited solubility in these waters, and should be monitored in particulates and sediments. Of note, this is consistent with shellfish being particularly susceptible to contamination.

Table 1 summarises cadmium levels in "watercourse" samples, as reported in Appendix M. Cadmium at WC05 was at levels almost an order of magnitude higher than elsewhere (cadmium was also elevated at WC03). Levels at WC03 and WC05 exceeded Canadian Water Quality Guidelines (Table 2) for the protection of aquatic life, both for fresh and marine waters. I did not identify a map of these locations, but apparently these lowest numbered samples are closer to and may be more likely to be impacted by NPNS.

I bring up the topic of cadmium in the absence of definitive evidence in the data presented that it is of high impact in this particular instance. Rather, cadmium is a highly toxic element, is expected to be a significant issue in this instance, has not been sufficiently investigated with regard to NPNS, and bears investigation as part of any EA.

Table 1. Cadmium levels (µg/L) in watercourse samples, December 3, 2018. (ref. Appendix M4)

Location	Cadmium concentration (µg/L)
WC16	0.037
WC15	0.056
WC14	0.024
WC13A	0.018
WC13B	0.017
WC12	<0.01
WC11	0.033
WC10	0.019
WC09	0.05
WC08	0.032
WC06	0.041
WC05	0.43
WC03	0.23

Table 2. The Canadian Water Quality Guidelines (CWQG) for the protection of aquatic life for cadmium

	Long-term Exposure (µg/L)	Short-term Exposure (µg/L)
Freshwater	0.09[a]	1.0[b]
Marine	0.12	NRG

NRG = no recommended guideline

a The long-term CWQG of 0.09 $\mu g \cdot L^{-1}$ is for waters of 50 mg $CaCO_3$/L hardness. At other hardness values, the CWQG can be calculated with the equation $CWQG = 10^{\{0.83(\log[hardness]) - 2.46\}}$, valid for hardness between 17 and 280 mg $CaCO_3$/L

b The short-term benchmark concentration of 1.0 $\mu g \cdot L^{-1}$ is for waters of 50 mg $CaCO_3 \cdot L^{-1}$ hardness. At other hardness values, the benchmark can be calculated with the equation $Benchmark = 10^{\{1.016(\log[hardness]) - 1.71\}}$, valid for hardness between 5.3 and 360 mg $CaCO_3$/L

Hydrogen Sulphide

Hydrogen sulphide, with its characteristic "rotten eggs" smell, is the simplest of a group of chemicals called "reduced sulphur compounds" (RSCs), also reported as "total reduced sulphur" (TRS). Other RSCs include carbonyl sulphide and carbon disulphide that do not have strong smells, as well as mercaptans and others that do have strong odours (a trace quantity is used as an alert chemical in natural gas). RSCs are the compounds largely responsible for the odour of pulp mills.

RSCs are sometimes characterized inaccurately as mere nuisance odours, but they are in fact toxic to the nervous system and to multiple organs.[11] Acute, episodic exposure to malodorous sulphur air pollutants released by a pulp mill causes breathing difficulties, and other respiratory, ocular (eye) and neuropsychological symptoms.[12]

Larger RSC molecules may be longer acting than simple hydrogen sulphide. Numerous reduced sulphur compounds exert substance-specific toxicities, but then are ultimately metabolized to create hydrogen sulphide, so they can all also have similarly toxic effects in the body. These include multi-system adverse effects, with life-long implications for the foetus and developing child (e.g., irritation of eyes, nose and throat, cough, headache, fatigue and malaise, nausea and vomiting, and headache, confusion and depression, and effects on the central and peripheral nervous system with chronic exposure). [11,13]

Pulp mill emissions of RSCs contain many more chemicals, and greater quantities of reduced sulphur, than may be measured or estimated as hydrogen sulphide *per se*. If this is true in the present case, the analyses reported in the EA Registration package (reproduced below) may under-estimate the load of reduced sulphur, and thereby its biological effects.

A 2017 study by Hoffman et al. [14], [1] was extensively discussed in the Environmental Assessment document (section 8.1.2.2). The Hoffman report clearly indicates, using straightforward treatment of routinely gathered data, that some toxic volatile chemicals measured in Pictou probably originate at the Northern Pulp facility. The data is from routine Canadian pollutant monitoring as well as weather data.

Northern Pulp monitoring reports posted on the Nova Scotia website, and modelling in the EA Registration package, all indicate exceedances of hydrogen sulphide or RSCs. These exceedances are predicted to continue with the new effluent treatment facility. The following tables, reproduced from the EA Registration package, indicate that hydrogen sulphide has been and is expected to continue to exceed permissible concentrations.

AIR DISPERSION MODELLING STUDY – REPLACEMENT EFFLUENT TREATMENT FACILITY

January 21, 2019

Hydrogen Sulphide

The maximum predicted ground level concentrations for hydrogen sulphide (H_2S) at each discrete receptor are presented in Table 6.5.

Table 6.5 Maximum Predicted Ground Level Concentrations (GLC) for H_2S – Existing

Receptor ID/Regulatory Limit	UTM Coordinates		1-hour (µg/m³)	24-hour (µg/m³)
	Easting (m)	Northing (m)		
Regulatory Limit	**-**	**-**	**42**	**8**
1	519768	5055219	20.4	0.86
2	520907	5053346	18.1	1.16
3	522480	5053951	7.69	0.77
4	522963	5054415	40.5	2.69
5	522899	5054854	37.0	2.55
6	524552	5055699	**96.6**	4.90
7	524337	5056312	34.8	2.21
8	526942	5057565	15.4	1.46
9	528826	5055486	2.52	0.24
10	522169	5058110	2.21	0.58

Bold – indicates an exceedance

Exceedances of the Nova Scotia maximum permissible ground level concentration limit for H_2S for the 1-hour averaging period were predicted at discrete receptor 6. The source contributing to the exceedance at discrete receptor 6 is the existing ETF. There were no exceedances of the 24-hour maximum permissible ground level concentration limit.

Further analysis of the above modelling results, including a exceedance frequency analysis at receptor 6, are discussed in Section 7 below.

[1] This study is found at Appendix E-2 of the Friends of the Northumberland Strait/Ecojustice submission, along with a response at Appendix E-1, from Ms. Hoffman to Stantec's critique in the NPNS EA submission.

Hydrogen Sulphide

The maximum predicted ground level concentrations for hydrogen sulphide (H_2S) at each discrete receptor are presented in Table 6.11. No exceedances of the Nova Scotia maximum 1-hour permissible ground level concentration limits were predicted for H_2S. For the 24-hour time averaging period, the maximum predicted concentration of H_2S at Receptor 5 was slightly above the maximum permissible ground level concentration limit.

Table 6.11 Maximum Predicted Ground Level Concentrations (GLC) for H_2S - Future

Receptor ID/Regulatory Limit	UTM Coordinates		1-hour ($\mu g/m^3$)	24-hour ($\mu g/m^3$)
	Easting (m)	Northing (m)		
Regulatory Limit	-	-	**42**	**8**
1	519768	5055219	9.46	1.89
2	520907	5053346	10.4	2.01
3	522480	5053951	12.2	2.66
4	522963	5054415	18.0	4.40
5	522899	5054854	21.3	**8.52**
6	524552	5055699	2.15	0.75
7	524337	5056312	10.6	2.50
8	526942	5057565	7.21	1.14
9	528826	5055486	1.08	0.23
10	522169	5058110	1.59	0.61
Bold – indicates an exceedance				

Further analysis of the above modelling results is presented in Section 7 below.

Table 7.1 Emission Impact Summary Table - Existing Operation

Contaminant	Maximum Predicted Concentration at Nearby Specific Receptors (ug/m^3)	Averaging Period	Reference Criteria* (ug/m^3)	Percentage of Criteria (%)
Carbon monoxide (CO)	340	1-hour	34,600	1%
	200	8-hour	12,700	2%
Hydrogen sulphide (H_2S)	**96.6**	1-hour	42	**230%**
	4.9	24-hour	8	61%
Nitrogen dioxide (NO_2)	29	1 hour	400	7%
	0.78	Annual	100	1%
Sulphur dioxide (SO_2)	57.3	1 hour	900	6%
	11.1	24 hour	300	4%
	0.68	Annual	60	1%
Total suspended particulate (TSP)	51.1	24-hour	120	43%
	3.35	Annual	70	5%
$PM_{2.5}$	7.92	24-hour	28	28%
	0.68	Annual	10	7%
*The criteria for the listed contaminants are Maximum Permissible Ground Level Concentration Limits specified in the Nova Scotia *Air Quality Regulations* except for $PM_{2.5}$, which criteria are Canadian Ambient Air Quality Standards.				

These results, copied from the EA Registration package, indicate both past and projected exceedances of hydrogen sulphide exposures. One might think that prediction of reaching a high proportion, let alone exceedances of air pollution guidelines, would send proponents back to the drawing board. This was not the case. Following these reports and predictions of excessive levels of RSCs, it is explained not that these are the result of the pulp mill, but that these are the result of abnormal weather conditions or "meteorological anomalies." It is then recommended that these may be ignored. The nature of anomalies is not detailed, but could include situations such as temperature inversions that impede atmospheric mixing. The consultants then explain that by using the 9th highest hourly concentration rather than the highest concentration in modelled years (i.e., previously observed "extremes" are removed), the hydrogen sulphide levels are predicted to be within compliance at a residential area close to the existing effluent treatment facility. The report concludes that although hydrogen sulphide exceedances are modelled, that with elimination of meteorological anomalies it is anticipated that exceedances will not be experienced.

It is unreasonable and certainly not precautionary, particularly in this era of climate change, to base environmental assessments on assumptions that "meteorological anomalies" will not repeat. Indeed, with weather anomalies of all kinds becoming more frequent and extreme, the opposite is expected, and should be assumed and factored into assessments.

Furthermore, approving and thereby facilitating periodic exceedances of health-based exposure criteria, for communities that include the most vulnerable of individuals and who are more susceptible to adverse effects, is contrary to the purpose of limits, and the mandate to protect public health. Process changes for pollution prevention and capture, and validation of emissions to land, water, air and deposition on soil, would be necessary to improve confidence that these communities will no longer be exposed to excessive levels of hazardous RSCs.

Dioxins and furans

Dioxins and furans ("dioxins") are among the most toxic chemicals that may be emitted from pulp mills. Their creation and release has been reduced as pulp mills have switched from elemental chlorine, to chlorine dioxide for bleaching. Dioxin levels are further reduced if oxygen is used in delignification, and peroxide is used later in the process. Sweden leads the world in advanced technologies for pulp manufacture.[15]

2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD), made famous as a contaminant of the defoliant Agent Orange used in the Vietnam War, is one of the most toxic, persistent, bioaccumulative man-made chemicals. This chemical is considered to be the most toxic, in the family of related dioxins and furans. With three chemical rings in a row, these are examples of poly-aromatic hydrocarbons (PAHs). Chlorinated PAHs have

chlorine atoms attached to the carbon structure. As a result of comparative toxicity based on historical experiences and scientific methods, dioxins and furans with four or more chlorine atoms (maximum eight chlorine atoms), including at the positions numbered 2, 3, 7 and 8, are considered extraordinarily toxic. Other dioxins are pro-rated in terms of toxicity, to calculate total toxic equivalents (TEQs).

An advance in pulp manufacture was made with introduction of chlorine dioxide bleaching that results in lower levels of chlorinated PAHs. Oxygen de-lignification, to which Northern Pulp committed during public consultation (see EA Registration document, Appendix 6), also assists to reduce use of chlorine based bleach and releases of chlorinated PAHs.

PAHs form, and may be chlorinated, during pulping and bleaching. PAHs can also form during combustion, and chlorination during combustion may be enhanced when the fuel contains higher levels of chlorine.[16] This is expected to be the case with addition of sludge to feed the boiler. Temperatures also affect formation of PAHs.[16] Thus, rigorous analyses of emissions and environmental contaminants are necessary to provide a baseline, as well as to monitor emissions and effects before and following operational changes in the pulp mill.

It is stated in the EA Registration document (e.g., Table 6.7-1), "In fact, dioxins and furans testing for the last 5 years has consistently shown that all of the compounds required to be tested under the regulations have not been detected in NPNS' effluent (non-detect)." The dioxin-free message is not consistent with reports from Northern Pulp that are posted on the Nova Scotia government website, nor the data reported to the National Pollutant Release Inventory (NPRI).[17] NPRI data indicates that on average 3.6 tonnes of PAHs have been emitted to the air annually since 2006, and 8 mg TEQ dioxins/furans have been emitted annually since 2011.

Environment Australia published a helpful review of chlorinated dioxin and furan emissions.[18] Of particular interest is a series of diagrams of typical dioxin "fingerprints" or patterns of emissions from various processes, as discussed below.

Biomass entering the pulp mill should contain little material with chlorine atoms bound to carbon atoms. In this case, typically there would be significant proportions of dioxins with four to six chlorine atoms (that have the highest TEQs), and lower proportions of dioxins with seven or eight chlorine atoms.

The opposite is reported for Northern Pulp in analyses reported on the Nova Scotia website, with all dioxin findings restricted to the hepta- and octo- (seven and eight) chlorinated forms. This is highly unusual. It is not consistent with the Australian experience,[18] and is also not consistent with reports of changes in dioxin fingerprints in fish affected by emissions from a pulp mill on Lake Superior, after bleaching was switched from chlorine to chlorine dioxide.[19]

Although I know of no evidence of either of the following actually happening, I offer two possible explanations from my experience and research, for this unusual situation. There may be laboratory deficiencies and/or irregularities during analyses, or alternatively, wood chips treated with polychlorinated phenols (wood preservatives) may have been processed at the facility.[18,20] These bear investigation.

In summary, regarding PAHs, including dioxins and furans:

- **Total chlorine free pulp production, with innovations to reduce water, waste and pollution, should be reassessed independently, because advanced technologies are established internationally. At a bare minimum, oxygen delignification must be installed as promised, to help to reduce toxicity of effluents, including both chlorinated PAHs (in particular dioxins and furans) and non-chlorinated PAHs;**
- **independent verification of laboratory analyses should be conducted, with blinded field blanks and, importantly, spiked samples included with field samples;**
- **possible origins of the unusual dioxin finger-print observed in samples from recent years should be investigated, including boiler fuels;**
- **the statement by Dillon, on behalf of Northern Pulp, that dioxins and furans have not been detected for the past five years is not true. This should be withdrawn. Releases of PAHs and other NPRI data should be reported.**

Looking for contaminants where they will be found, and pose hazards

While monitoring emissions directly is important for any industry, these samples do not adequately predict what is important – the levels of toxicants in the environment, and in foods and people. PAHs and metals emitted by NPNS are bioaccumulative. For example, lipophilic ("fat loving") PAHs tend to bioaccumulate in fatty tissues, [19] so analysis of aquatic organisms may reveal substantial levels despite "no detection" results in water or air. Similarly, metals attached to particulates may not be detected in finely filtered water or air, but may nevertheless build up in plants and animals. It is of course easier to measure pollutants when they are at higher concentrations, and is more relevant to the environment as well as to populations, to measure toxicants where they accrue and pose greatest hazards and risks.

According to a 2017 study of foods consumed by First Nations in the Atlantic Provinces, oysters and organ meat are highest in cadmium.[21] Mussels [22] and lobster hepatopancreas have been monitored for toxic chemicals in other situations (Appendix R of the Environmental Assessment). There are many options for relevant, sensitive longitudinal monitoring. It is beyond the scope of this work to offer recommendations, but it may also be time to expand toxicant and toxicity testing beyond simple acute fish mortality.

Summary

In summary, in my view, it is essential that the environmental assessment information for this project must include, at a minimum:

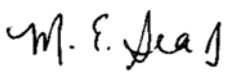
- independent scoping of a human health assessment, to ensure that the details and particulars of the diverse populations, vulnerabilities, changes in exposure within the particular contexts of these populations and their diets, and current industrial legacy pollution are adequately addressed;
- assessment of progressive measures to modernize equipment and processes in the mill, to eliminate pollutant exceedances, and to advance technologies and practices, to place NPNS as a leader in environmentally preferable pulp production, as well as promotion of public and environmental health;
- baseline and regular surveillance monitoring that is accurate, verified, reliable and relevant to the environment and human populations;
- investigation and assessment of any proposed developments, in recognition of legacy mercury contamination in proximity of the proposed effluent treatment facility;
- plans to minimize contamination and to monitor for contaminants and related effects, using both conventional measures as well as with assays of biological samples (e.g. shellfish).

Unfortunately, I could identify none of these in the current EA Registration package. What is more, inaccurate statements denying pollution that occurred cast a shadow over the reliability of the EA.

The above comments address a few, but given the time frame certainly not all, shortcomings and misperceptions within the Northern Pulp Environmental Assessment Registration documents. It is my opinion that the current EA Registration package is insufficient to permit adequate assessment, and that any permissions or approvals on this basis are not justified.

I hope that the above is of assistance, and am available for further discussions in this important matter.

Respectfully Submitted,



Margaret (Meg) Sears, M.Eng., PhD
RR 1, 107 Mast Ln
Dunrobin, ON
K0A 1T0
Email: MegSears@ncf.ca
Telephone: 613 297-6042

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APPENDIX F-2

Margaret (Meg) E. Sears (M.Eng., Ph.D.)

RR 1, 107 Mast Ln.

Dunrobin, Ont. Canada. K0A 1T0

+1 (613) 832-2806 (landline) / +1 (613) 297-6042 (cell phone)
email: megsears@ncf.ca

Capabilities and Experience:

Broad interests include health and medicine, epidemiology and toxicology, chemistry, ecology, biology and chemical engineering. Specific interests in ongoing work include environmental health, and scientific evidence synthesis for hazard and risk assessment, and public policy. A current focus is on multi-factorial contributors to chronic disease, and filling data gaps in environmental health epidemiology.

Activities include researching, assessing, reviewing and reporting in the scientific literature, for government bodies, peer-reviewed journals, and civil society organizations. My work includes liaising with a broad network of scientific experts, physicians and others on topics related to environment and health. I also frequently observe and participate in stakeholder meetings regarding the Chemicals Management Plan, and participate in federal government consultations and other meetings on chemicals management and pesticides. In 2015, two reports from the Parliamentary Standing Committee on Health included my recommendations regarding pesticides, and health effects of radiofrequency radiation. The *Canadian Environmental Protection Act* Parliamentary Committee and the *Canadian Environmental Assessment Act* Panel recommendations include some of my recommendations, and I participated with others and Canadian Environmental Law Association staff in drafting proposed amendments to the *Canadian Environmental Protection Act (1999)*. I have written key documents regarding environmental sensitivities and worked with groups of affected as well as health care professionals.

Experience includes working with groups of researchers on large scientific reports, including research question identification, literature searches, data extraction, analysis and review, writing, editing, managing references and maintaining version control. I chair the civil society organization *Prevent Cancer Now* and work with numerous other civil society organizations in Canada and internationally regarding toxic exposures. I have also twice been a medical journal guest editor. I have also conducted consultations among professionals, and citizens' groups, and prepared and presented scientific committee and tribunal submissions.

Public speaking includes lecturing at the Universities of Ottawa and Toronto, and Lakehead University, and numerous public presentations regarding topics in environmental health.

Topics recently addressed include hazard and risk assessment per federal legislation, and more specifically epidemiology, toxicology (including pesticides), endocrine disruptors, toxic elements, systematic review in environmental health, and electromagnetic radiation in public and personal health.

Diverse laboratory and field experience in chemical engineering, applied chemistry, and microbiology; including occupational health and safety, and microbiological and petrochemical industry research.

Academic Background

1986 Doctor of Philosophy, McGill University. Effects of growth conditions on biosorption by *Rhizopus* biosorbents.

1981 Masters of Chemical Engineering, McGill University. Measurement and mathematical modelling of biosorption of uranyl ion by biomass of the mould *Rhizopus arrhizus*.

1979 Bachelors of Applied Chemistry and Chemical Engineering, with Honours, University of Toronto.

Appointments

Senior Clinical Research Associate, presently working with Dr. Richard van der Jagt at the Environmental Health Information Infrastructure; previously under Dr. David Moher at the Centre for Practice-Changing Research, Epidemiology, at the Ottawa Hospital Research Institute

Associate with University of Sherbrooke, working with Dr. Isabelle Gaboury and colleagues

Previously Adjunct Investigator at the Children's Hospital of Eastern Ontario (this type of appointment was discontinued)

Professional Membership

Canadian Paediatric Society, including Environmental Health Section.
Canadian Public Health Association
Ontario Public Health Association
International Network for Epidemiology and Policy

Awards and Grants

2013 Carleton Lee Award, American Academy of Environmental Medicine

2007-2009 Canadian Institutes of Health Research / Social Sciences and Humanities Research Council grant for a scoping review on the toxic elements arsenic, cadmium, lead and mercury.

1980-1985 Natural Sciences and Engineering Research Council scholarship for post-graduate studies

Work Experience – University instruction

2012, 2014, 2017 University of Ottawa EVS 3131 (undergraduate) and Capstone Masters programs - Supervised and participated in student projects. Lectured on Environmental Health.

2010 – 2012 Lakehead University and the Northern School of Medicine - PUBL5213 Environmental and Occupational Public Health (Masters of Public Health). Lectured on toxicology and epidemiology, land use planning, evidence synthesis, pesticides, toxic metals and endocrine disruption, in a distance-learning course.

2011, 2012 Lectured on toxicology epidemiology and evidence synthesis, as well as pesticides, in ENV 341, at University of Toronto.

2009, 2010 Lectured on toxicology, epidemiology, evidence synthesis, pesticides and toxic metals in HSS3303 at the University of Ottawa.

Work Experience – selected

2018 Preparation of an Indoor Air Quality Module regarding Chemical Sensitivities, for the Canadian National Research Council.

2013-2014 – Health expert in the Proceeding of the Alberta Energy Regulator re. health effects of bitumen emissions in the Peace River area.

2012 Health expert in Fortis BC hearing re. Smart Meters

2004-on Systematic Reviews in the Centre for Practice Changing Research at the Ottawa Hospital Research Institute, under Dr. David Moher. Many aspects of evidence review and synthesis, and editorial responsibility for large medical scientific research reports.

2003-on Work with diverse medical researchers, on data analysis, presentation and writing, in Ottawa, Toronto and at the University of Sherbrooke.

2011 In conjunction with physicians associated with the Ontario College of Family Physicians, I conducted literature searches, synthesis of information and co-writing of a report regarding updating the Greig Record for child and adolescent primary care visits with Family Physicians.

I organized and spoke at a meeting to present Toxic Metals in Canadians scoping review findings and to gain insights from physicians, clinical and toxicological researchers, and public health officials.

2009 Co-authored "Air Travel and Chemical Sensitivities" for the Canadian Transportation Authority.

2008 Canadian Institutes of Health Research Primary Investigator - "Toxic Metals in Canadians and their Environments: Exposures, health effects, and physician and public health management strategies - A Scoping Review"

2008-on Occasionally assist the David Suzuki Foundation with scientific review of documents regarding environmental health topics, including pesticides and cosmetics.

Prepared affidavits with regard to health effects of herbicides (and contaminants) used at CFB Gagetown, NB.

Lectured on epidemiology, toxicology and synthesis of scientific evidence in environmental health, pesticides, toxic elements (particularly arsenic, cadmium, lead and mercury) as well as scientific writing, in undergraduate courses at the University of Ottawa and University of Toronto, and at the graduate level (Masters of Public Health) at Lakehead University.

2006 Prepared "A Medical Perspective on Environmental Sensitivities" for the Canadian Human Rights Commission, including research review, and consultation with physicians, architects and civil society organizations.

2002-on Writing, and assisting medical researchers and others with drafting of research documents.

- This work includes data extraction, review of data and statistics, review of the medical background information, literature review updates, and planning, drafting and version control.
- Articles have included systematic reviews, randomized controlled trials, other interventional and observational studies, and commentaries.
- Topics include pesticide assessment and 2,4-D, environmental sensitivities, medical ethics, medical education, diabetes in children, probiotics, sexuality and fertility

following spinal cord injury, breast cancer care, child car-seats and booster-seats, nocturnal enuresis, omega-3 fatty acids and infant health, computerized physician order-entry systems in the context of bronchiolitis, childhood arthritis, models of medical practice and collaboration, lipid modifying agents, drug delivery, TPMT assessment in thiopurine therapy, morphine monitoring nursing practice, nutritional supplements and drugs for cardiovascular health, and online medical education.

2002 Drafted "Frequently Asked Questions" responses regarding breast milk contamination, flame retardants, West Nile virus and insect repellents, for the Canadian Institute of Child Health.

1979-80 Research engineer at Gulf Canada's research facility in Sheridan Pk., Mississauga. Constructed and operated small-scale laboratory simulation of heavy oil cracking, as well as mathematical modelling of enhanced oil recovery (akin to 4-phase hydrogeology).

1975-79 During summers prior to and during undergraduate studies, worked in UofT Chemical Engineering laboratories (including tar sands oil extraction), and at Imperial Oil Research laboratories.

Volunteer Activities

2017 Member of the Science Committee for the Canadian Public Health Association 2018 Conference Planning Committee

2017 Core leadership group and 2018 Conference planning for the Canadian Alliance for Regional Risk Factor Surveillance

2015-on member of Waste Watch Ottawa, that brings strong evidence regarding waste management to Ottawa City Council

2011- on Board member (currently Chair) of *Prevent Cancer Now* (www.preventcancernow.ca). Responsible for writing and editing publicly available documents, media, and numerous public presentations regarding cancer prevention.

With Dr. van der Jagt, initiating a national environmental health information infrastructure for investigation of links between environmental quality and health.

2011- on Member of the Sustainability Committee ("Green Team") at the Children's Hospital of Eastern Ontario, Ottawa.

2001- on Established, along with other mothers, the Ottawa Neuroblastoma Research Fund (CHEO)

2001- on Work with physicians and various organizations regarding pesticides and health.

2002- on (currently quiescent) Founding member of the Coalition for a Healthy Ottawa and the Canadian Coalition for Health and the Environment. We synthesized and promoted research on pesticides and health, as well as synthesis of scientific evidence, in efforts to reduce use of pesticides in urban areas, and for vector control, with these and other groups across Canada.

1995-8 Member of the Board of Directors of *Les Petits Ballets*, in charge of publicity.

1989-on (currently quiescent) Founding member and Secretary for the Wetlands Preservation Group of West Carleton, working for environmental protection before the Ontario Municipal Board, Environmental Assessment Advisory Committee, in court, and before the Sewell Commission on land use planning.

Peer-reviewed publications

Fernández, C., A. A. de Salles, M. E. Sears, R. D. Morris, and D. L. Davis. "Absorption of Wireless Radiation in the Child versus Adult Brain and Eye from Cell Phone Conversation or Virtual Reality." Environmental Research, June 5, 2018. https://doi.org/10.1016/j.envres.2018.05.013.

Sears, Margaret E. Chelation: Harnessing and Enhancing Heavy Metal Detoxification:A Review. The Scientific World Journal 2013 (April 18, 2013). doi:10.1155/2013/219840.

Kanji, Salmaan, Dugald Seely, Fatemeh Yazdi, Jennifer Tetzlaff, Kavita Singh, Alexander Tsertsvadze, Andrea C. Tricco, et al. "Interactions of Commonly Used Dietary Supplements with Cardiovascular Drugs: a Systematic Review." Systematic Reviews 1, no. 1 (May 31, 2012): 26.

Seely D, Kanji S, Yazdi F, Tetzlaff J, Singh K, Tsertsvadze A, Sears ME, Tricco A, Ooi TC, Turek M, Tsouros S, Skidmore B, Daniel R, Ansari MT. Dietary Supplements in Adults Taking Cardiovascular Drugs. Comparative Effectiveness Review No. 51. (Prepared by the University of Ottawa Evidence-based Practice Center under Contract No. HHSA 290-2007- 10059-I.) AHRQ Publication No. 12-EHC021-EF. Rockville, MD: Agency for Healthcare Research and Quality. April 2012.

Sears, Margaret E., Kathleen J. Kerr, and Riina I. Bray. Arsenic, Cadmium, Lead, and Mercury in Sweat: A Systematic Review. J Environ Public Health. Article ID 184745 (2012): 1–10.

Sears, Margaret E., and Stephen J. Genuis. Environmental Determinants of Chronic Disease and Medical Approaches: Recognition, Avoidance, Supportive Therapy, and Detoxification. J Environ Public Health. Article ID 356798 (2012): 1–15.

Loit E, Tricco AC, Tsouros S, Sears M, Ansari MT, Booth RA. Pre-analytic and analytic sources of variations in thiopurine methyltransferase activity measurement in patients prescribed thiopurine-based drugs: A systematic review. Clin Biochem (March 18, 2011). 44(10-11):751-757.

Sears M. Toxic Metals Injuries. Paed Child Health. 2011;16(3):152.

Sharma M, Ansari MT, Soares-Weiser K, Abou-setta AM, Ooi TC, Sears M, Yazdi F, Tsertsvadze A, Moher D. Systematic review: comparative effectiveness and harms of combination therapy and monotherapy for dyslipidemia. Annals of Internal Medicine 151, no. 9 (November 3, 2009): 622-630.

Sharma M, Ansari MT, Soares-Weiser K, Abou-setta AM, Ooi TC, Sears M, Yazdi F, Tsertsvadze A, Moher D. Comparative Effectiveness of Lipid-Modifying Agents [Internet]. Rockville (MD): Agency for Healthcare Research and Quality (US); 2009 Sep.

Sears M and Walker CR. Dioxins in Children. Paediatric and Perinatal Drug Therapy, 2007; 8 (3):134.

Sears M, Walker CR, van der Jagt RHC, Claman P. Pesticide assessment: Protecting public health on the home turf. Paediatr Child Health 2006;11(4):229-234.

Irwin D, Vaillancourt R, Dalgleish D, Thomas M, Grenier S, Wong E, Wright M, Sears M, Doherty D, Gaboury I. Standard concentrations of high alert drug infusions across paediatric acute care. Paed. Child Health 2008;13(5):371-376.

Treen-Sears ME, Volesky B, Neufeld RJ. Ion exchange/complexation of the uranyl ion by Rhizopus biosorbent. Biotechnol. Bioeng. 1984;26(11):1323–1329.

Treen-Sears ME, Martin SM, Volesky B. Propagation of Rhizopus javanicus Biosorbent. Appl Environ Microbiol. 1984; 48(1):137–141.

Treen-Sears ME, Martin SM, Volesky B. Control of Rhizopus Biosorbents' Quality During Propagation.
Fundamentals of Applied Biohydrometallurgy; Vancouver, BC; Canada; 21-24 Aug. 1985. pp. 305-308.

Volesky B, Sears M, Neufeld RJ, Tsezos M. Recovery of strategic elements by biosorption. Annal NY Acad Sci 1983.

Nadeau JS, Treen ME, Boocock DGB. Mass transfer effects in a nitric oxide dosimeter. Anal. Chem. 1978;50(13):1871-1873.

Guest Editor: *Journal of Environmental and Public Health.* Special issue on Incorporating Environmental Health into Clinical Medicine, published spring of 2012.

Guest Editor: *The Scientific World Journal*, Special issue on Environmental Health, published spring 2013

Peer Reviewer: Canadian Medical Association Journal; European Journal of Internal Medicine; Reproductive Toxicology; Journal of Forensic Sciences; Paediatrics and Child Health; Human and Experimental Toxicology; Public Health; Science of the Total Environment; International Journal of Environmental Research and Public Health; Current Oncology.

Book Chapter

Davis, D, M Sears, A Miller, R Bray. Microwave/Radiofrequency Radiation and Human Health. In *Integrative Environmental Medicine.* A Cohen, FS vom Saal Eds. Oxford University Press. March 2017.

Recent Conference Participation

2018 Core organizing committee for CARRFS pre-conference session, and speaker (**Escalating Chronic Disease in Young Canadians – surveillance for environmental links**) at the collaboration session during the Canadian Public Health Association conference, Montreal.

2017 Presentations and participation in an experts forum at The Hebrew University, hosted by the Israeli Institute for Advanced Studies, and the Environmental Health Trust. **"Wireless Radiation and Human Health."** Jerusalem, January 2017.

2015 **"Scientific Review to Support Public Policy Regarding Exposure to Radiation from Wireless Communications Devices."** Poster. International Bioelectromagnetics Conference. Alisomar, California. June 2015

2014 "**Search and ye shall find environmental health concerns: e.g. Peace River Proceeding1769924."** Invited Presentation. Under Western Skies Conference. Calgary. September 2014.

2013 **"Harvesting the Best from the Wilderness: Moving from Scouts' Common Sense, to Evidence-Based Practice for Environmental Health."** Invited presentation. American Academy of Environmental Medicine. Phoenix, Arizona

Selected non-peer reviewed medical / scientific articles

Ongoing, submissions regarding policy and laws improvements, and scientific comments regarding substances and exposures to the Government of Canada and others, on behalf of *Prevent Cancer Now*. http://www.preventcancernow.ca/main/resources/cancer-prevention-submissions

Healthy Children/Healthy Environment: Improving the Odds: Part 2

(authors in alphabetical order) Riina I. Bray, M. Janet Kasperski, Lynn M. Marshall, Margaret E. Sears.
[March 31, 2011. Respectfully submitted on behalf of the Ontario College of Family Physicians to the Environmental Health Program, Health Canada.]

Air Travel and Chemical Sensitivities

John Molot, Lynn Marshall and Meg Sears
[March 2009 – prepared for the Canadian Transportation Authority]

The Medical Perspective on Environmental Sensitivities

Margaret E. Sears [February 2007 – prepared for the Canadian Human Rights Commission, in collaboration with the Ontario College of Family Physicians Environmental Health Committee, and other academics, physicians and architects]
Available at: http://www.chrc-ccdp.gc.ca/eng/content/medical-perspective-environmental-sensitivities

Comments on the Pest Management Regulatory Agency's Use of Uncertainty and Safety Factors in the Human Health Risk Assessment of Pesticides

M.E. Sears, C.S. Findlay, N. Arya, L. Marshall, M. Sanborn, K.J. Kerr, J. Kasperski
[2007 – prepared on behalf of, and submitted to the Pest Management Regulatory Agency (PMRA) of Health Canada, by the Environmental Health Committee, Ontario College of Family Physicians]

APPENDIX F-3

Chapter 4
The Greening of the Pulp and Paper Industry: Sweden in Comparative Perspective

Ann-Kristin Bergquist and Kristina Söderholm

4.1 Introduction

Since the 1960s, concern about environmental degradation has greatly altered the commercial conditions for most industries in the Western world, but the global pulp and paper industry (PPI) has encountered more serious challenges than most other industries. The reason is that pulp and paper production, especially bleached pulp manufacturing, is extremely damaging to the environment. Thus, pulp mills cause, among other things, emissions of odorous gases and sulphur compounds into the air and of organic and inorganic chemicals including chlorinated compounds and other substances into waterways. Thanks to technological developments, however, the environmental situation has been greatly improved during the last five decades with reduced emissions; in many cases the reduction has been more than 90%. This chapter concerns the environmentally driven transformation of the PPI and examines it in a country which has pioneered parts of the greening process in this industry, namely Sweden. This nation is one of the world's leading pulp and paper countries, as it ranks as the ninth largest producer of paper and the fourth largest producer of pulp (Lamberg et al. 2012; Järvinen et al. 2012; Bergquist and Keskitalo 2016).

For any polluting industry, technology is at the very core of the challenge to reduce its environmental impact (Jaffe et al. 2005) and indeed this has been the case for the PPI (Bajpai 2011). Technological strategies and timing for investments have differed between countries and regions depending on the different national jurisdictions, organizational solutions, demand characteristics and geographical circumstances. The

A.-K. Bergquist (✉)
Department of Geography and Economic History, Umeå University, Umeå, Sweden
e-mail: ann-kristin.bergquist@umu.se

K. Söderholm
Department of Business Administration, Technology and Social Sciences, Luleå University of Technology, Luleå, Sweden
e-mail: Kristina.Soderholm@ltu.se

T. Särkkä et al. (eds.), *Technological Transformation in the Global Pulp and Paper Industry 1800–2018*, World Forests 23, https://doi.org/10.1007/978-3-319-94962-8_4

literature on corporate environmentalism, which has stressed companies' modes of responding to environmental issues, has generally identified a movement along an evolutionary, adaptive learning process that formed specific attitudes or means of response during certain periods (Hoffman and Bansal 2012). An important conclusion from this research when it comes to technological strategies is that before the 1990s, firms typically employed end-of-pipe approaches to achieve regulatory compliance, which practically means external treatment of effluents after they have left the plant (Lee and Rhee 2005; Frondel et al. 2007; Hoffman 1997). The alternative technological approach, internal process changes (e.g., in-plant measures that prevent or reduce effluents before they leave the plant, sometimes also referred to as 'cleaner production technologies'), has subsequently been acknowledged and adopted as a more advantageous and effective strategy (Sinclair 1990).

As will be discussed in this chapter, Swedish PPI has not followed the textbook example of the learning process from end-of-pipe technology to "cleaner" production processes. The Swedish PPI, in contrast to both its North American and Finnish counterparts, embarked already in the 1970s on developing internal process alterations rather than end-of-pipe solutions, as the main technological strategy for pollution control (Bergquist and Söderholm 2015; Harrison 2002). This early strategy became formative for the Swedish PPI's green technological development for decades to come. Kemp and Soete (1990) have argued that, compared to 'normal' technological efforts in industry, pollution control efforts are generally more focused on incremental improvements in 'cleaning' technology, following relatively well-established technological strategies of 'progress'. This argument suggests that pollution control technologies are particularly 'path dependent' and breaking the 'path' demands a lot of investments and even a new way of thinking. The notion of path dependence can be seen from several perspectives; from the simple assertion that 'history matters' to the problem of imperfect information and the appreciation that institutional arrangements may have limits, and the phenomenon of increasing returns (e.g. Liebowitz and Margolis 1995; Pierson 2000). When it comes to technology, path dependence can provide very powerful incentives for limiting actions to incremental technological solutions, which in turn strengthen established lines of action (Geels 2004; Kemp and Soete 1990; David 1994). Technological approaches in one country, as well as divergent technological strategies between different pulp and paper producing countries, might therefore be considered in the logic of technological path dependence.

For several reasons the greening of the PPI has been a core challenge for the sector. Environmental regulations have greatly altered the commercial conditions for the PPI over the past five decades (as for most industries), and since the 1980s, market pressure has also interacted with environmental regulation as a driver towards greener production processes and products. In addition, since the 1970s, the drive to use energy more efficiently has been increasingly linked with environmental issues, not least through the issue of climate change. Overall, the greening of the PPI has been a complex process. It has evolved along several different paths in combination with other business challenges, with the latter stemming from changed input

prices on, for instance, energy, wood and labour and also changes in market competition, governmental policies, and new technologies outside the immediate realm of pollution control.

4.2 Environmental Impacts of Pulp and Paper Production: A Short Background

The environmental impact of pulp and paper mills has been, and still is, serious from many aspects (Table 4.1). It causes or produces human toxicity, ecotoxicity, photochemical oxidations, acidification, nutrification, solid wastes and climate change (Bajpai 2011). Still, the situation is much better today than it was in the past; wastewater from a modern pulp mill contains only a small fraction of the contaminants that it did in the 1960s. One of the most serious problems is the very large quantity of wastewater resulting from pulp manufacturing, and it is contaminated with a number of organic and inorganic chemicals including lignin, cellulosic compounds, phenols, mercaptans, sulphides and chlorinated compounds (Thompson et al. 2001). The PPI is also a large user of both energy and water, and toxic chemicals. For example, in the United States (US) the paper industry is the third largest energy user among manufacturing industries, accounting for 11% of domestic energy consumption in 2010 (World Watch Institute 2015). The most significant environmental problems occur in the stage of the pulping process (in relation to paper production), which is why the most severe problems in the past were found in pulp producing countries, such as Sweden, Finland, Canada and the US although pulp importing countries, such as the United Kingdom and Germany also have struggled with severe problems (Bajpai 2015; Bergquist and Söderholm 2015; Mutz 2009).

Pulp can be roughly divided into the processes (and products) of mechanical and chemical pulp. Each process varies in terms of immediate environmental impact depending upon the amount and type of energy and chemicals used. Pulp mills are, however, traditionally associated specifically with the discharge of high levels of wastewater contaminated with organic matter. In the chemical pulp process, wood chips are first cooked in chemicals and then the solution is washed to produce "clean pulp", which consists of only cellulose fibres. Dirty "wash water", containing cooking chemicals and dissolved lignin, are thereafter sent to a recovery boiler[1] from which the cooking chemicals are recovered for reuse and the lignin is burned for power generation. Wastewater containing the remaining organic matter and chemicals is then discharged (Gunningham et al. 2003, pp. 10–11).

The most serious environmental problem related to pulp manufacturing in the past was caused by the bleaching process. It takes place in stages, generally alternating between acid and alkaline stages. The use of elemental chlorine (Cl_2) as the bleaching agent became dominant in the 1950s (Norberg-Boom and Rossi 1998) and was

[1] The invention of the recovery boiler by G. H. Tomlinson in the early 1930s was a milestone in the advancement of the Kraft pulp process.

Table 4.1 Major pollution problems caused by pulp and paper production

Suspended solids (SS)	Have its origins in bark, pieces of fibres and filling, and coating agents. Consumes oxygen when decaying and can be carriers of poisonous substances
Organic matter in general (BOD and COD)	Uses oxygen from water. May cause oxygen deficiency in waterways, which leads to the death of, i.e., fish and cause severe damage to the ecosystem
Chlorinated organic compounds (AOX)	From mills using elemental chlorine in their bleaching sequence. Wastewater containing organic matter formed by elemental chlorine reacting with wood products to form absorbable organic halide (AOX). AOX has become an accepted measure of chlorinated organic material, and is used to monitor and regulate bleached Kraft pulp mill effluents. AOX is used as a surrogate parameter of dioxins in wastewater (and stack gas) from pulp mills
Water consumption	Pulp mills are major water users. Consumption of fresh water can seriously harm habitats near mills from reduced water levels (necessary for fish) and changed water temperature (also a critical environmental factor for fish)
Sulphur dioxide (SO_2) and reduced sulfur compounds	Leads to acid rain and causes soil degeneration
Nitrogen oxides (NOx)	Gases composed of nitrogen and oxygen formed during combustion. In moist air, the substances are converted into nitrogen oxides and then nitric acid, which creates acid rain. Originates from recovery boilers in the Kraft pulp process
Sulphur compounds (TRS gases)	Smell

Source Ince et al. (2011), Skogsindustrierna (1995), Nilsson (2007)

subsequently replaced after the 1980s with new bleaching agents and methods such as the elemental chlorine free (ECF) and total chlorine free (TCF) bleaching.

4.3 Environmental Regulation as a Driver for Technology Development

The Swedish PPI has been central to the Swedish economy ever since the introduction of mechanical pulp in the 1850s and chemical pulp in the 1870s. Already at the time of the First World War Sweden was the world's second largest pulp producer after the US and the world's largest pulp exporter (Fahlström 1948; Rydberg 1990; Järvinen et al. 2012). As the PPI represented both one of the largest industrial sectors in Sweden and one of the biggest polluters, it has received much attention in Swedish policy to control industrial pollution, including joint state-industry efforts in R&D to control it (Bergquist and Söderholm 2011).

Over time environmental policies have grown more complex with a long-term shift in focus from local conflicts over water pollution and odour problems in the late nineteenth century to transnational and global problems such as climate change in the late twentieth century. This development has taken place in stages (McNeill 2000). Hence, in the 1960s, Sweden along with many other western societies saw the initiation of serious governmental action to control industrial pollution in parallel to a growing environmental awareness in society (Lönnroth 2010). In contrast to the US, environmental awareness in Sweden grew mainly amongst experts and not the public in the 1960s and 1970s. It is often assumed that heavy polluting industries did not focus seriously on mitigating their environmental impacts before this environmental awakening. There is plenty of historical research, however, which illustrates how companies and even industrial sectors undertook action to control pollution due to local conflicts and governmental intervention before the 1960s and sometimes they did so even proactively to avoid a bad outcome for business (for an updated literature overview, see Bergquist 2017). In fact, the Swedish PPI undertook action to control pollution long before the 1960s due to increasing local criticism and the industry's own awareness of the negative environmental effects from production. This in turn spurred joint efforts to develop new and cleaner technologies already in the early 1900s (Söderholm and Bergquist 2012). For example, by 1908 all Swedish sulphate pulp producers had united to form the so-called *Sulphate Pulp Committee* to develop technology to lessen the odour problem stemming from the sulphate pulp process.[2] Central reasons behind these initiatives were rising concerns about industrial pollution that had been expressed in the Swedish Parliament in the early 1900s, along with court cases concerning the contaminating activities of single pulp mills. These court cases built on early health- and water protective regulation, which to some degree included regulation of industrial pollution. For instance, the Public Health Act of 1874 constituted the first governmental "all-embracing" attempt to address sanitary issues. In the 1910s, the Swedish government presented a proposal to implement stricter legislation against air and water pollution. However, the proposal was rejected in the early 1920s due to an economic recession (Söderholm 2009; Lundgren 1974).

Pollution problems, however, increased in tandem with a growing Swedish economy in the 1930s, whereupon the Swedish government initiated a process of tightening the regulation of water pollution. Hence the Water Act was reformed in 1942, whereby it introduced a concession system according to which enterprises such as chemical pulp and sugar mills and textile factories had to apply to the Water Court for permission to operate. Still, air pollution was not addressed properly until 1963, and then through the formation of the State Air Pollution Control Board (Lundgren 1999). Finally, in 1969, the Environmental Protection Act (EPAct), the first uniform Swedish framework for regulating air and water pollution, noise and other disturbing activities from industrial plants, was passed by the Swedish Parliament. Years before the final bill was enacted, the construction of a modern system for environmental

[2]For a more detailed overview of regulation and organization of the Swedish PPI during this period, see Söderholm and Bergquist (2012).

protection had begun in Sweden. Thus, in 1967 the government created the National Environmental Protection Agency (EPA), a unified body for almost the entire area covered by the Environmental Protection Act (Lundqvist 1971). Soon other countries followed this lead, such as the US, which established its own EPA in 1970. Still, in the neighbouring pulp producing country of Finland, environmental policy developed at a slower pace (Söderholm et al. 2017). The National Board of Waters was created in 1970, but it was not until 1983 that a unified body to handle a major part of the environmental issues founded there (i.e., the Ministry of the Environment) (Joas 1997).

From the 1970s until the 1990s, the Swedish EPAct was the main tool to control pollution and other environmental problems related to Swedish industry, and it fundamentally changed the conditions for the operations of the Swedish PPI. Thus, in the 1970s and the 1980s it forced the sector to increase considerably its green R&D activities and to undertake deep emission cuts (Bergquist and Söderholm 2011). The regulatory approach was based on case-by-case assessments whereby permits had to be reassessed and renewed every 10 years or after production increased. It relied on so-called performance standards rather than technology standards and these were negotiated with each plant owner, sometimes over extended periods of time. The standards were typically implemented in combination with extended compliance periods, meaning that the companies were giving necessary time to develop and test technology. In these ways, the regulatory approach provided scope for environmental innovation and permitted the affected companies to coordinate pollution abatement measures with productive investments (Bergquist et al. 2013). In 1999, the EPAct along with 15 other acts were amalgamated into the Swedish Environmental Code and the responsibility for issuing permits was thereby transferred from the Franchise Board of Environmental Protection (the organ responsible for issuing permits since 1969) to the Environmental Courts (Michanek and Zetterberg 2007).

The Swedish PPI has also been governed by European Union (EU) environmental legislation ever since Sweden became an EU-member in 1995. One example is the Integrated Pollution Prevention Control Directive (IPPC Directive) of 1996 and 2008. The IPPC Directive is a key instrument in the EU's environmental legislation, and its purpose is to achieve 'integrated pollution prevention' and control of the pollution occurring at large industrial installations (Schoenberger 2009).[3] Further, in 2011, the Industrial Emission Directive (IED)[4] came into force, meaning all Member States before 7 January 2013, had to incorporate the IED into national leg-

[3]The conditions of required permits have to be based on Best Available Techniques (BAT), and in 1997, the Sevilla Process was established to develop BAT. Since then, 33 BAT Reference Documents (BREFs) have been drafted, adopted and published containing ambitious consumption and emission levels which cannot be found anywhere else (Schoenberger 2009).

[4]The IED is the successor of the IPPC Directive and, in essence, is about minimizing pollution from various industrial sources throughout the EU. Operators of industrial installations are covered by Annex I of the IED and are required to obtain an integrated permit from the relevant EU country authorities. Permit conditions including emission limit values (ELVs) must be based on the Best Available Techniques (BAT) as defined in the IPPC Directive.

islation. In Sweden the directive was included in the Industrial Emission Regulation (*Industriutsläppsförordning* 2013, p. 250) (SEPA 2016).

Since the 1990s in Sweden, policies targeting the PPI sector have embraced a blend of mandatory governmental and voluntary stipulations concerning social and environmental requirements. Thus, in line with international trends, environmental management systems (EMAS) have been implemented in virtually all Swedish pulp and paper mills. And most mills have chosen to certify their environmental management systems under either the global standard ISO 14001 and/or the European EMAS scheme. Thus, in 2013, 97% of the pulp and 98% of the paper produced in Sweden was manufactured under certified environmental management systems (Swedish Forest Industry Federation 2015). Essentially the EMAS scheme is an opportunity for producers to formally demonstrate from year to year their improved environmental performance to customers and partners, and is thus a form of 'eco-labelling'. The opportunity for the Swedish PPI to engage in 'eco-labeling' first appeared in the 1980s (see below). Regulatory measures to stimulate improvements in energy efficiency and lower carbon dioxide emissions have developed as an additional control box parallel to other emissions (see e.g. Thollander and Ottosson 2008; Henriksson et al. 2012). Also, in 2005 the voluntary Swedish Program for Energy Saving (PFE) came into force (Henriksson et al. 2012).

4.4 The "Spring-Cleaning" of the Swedish PPI

4.4.1 Internal Process Changes and Structural Rationalizations

The magnitude of pollution control implied by the EPAct in 1969 made the Swedish pulp and paper producers conclude that it was only through reconstructed and new plants embodying the most novel techniques that the discharges could be substantially reduced and requirements met (Wohlfart 1971b, p. 320). In this context, the Swedish pulp producers strategically aimed for production expansion based on the adoption of the sulphate process in the late 1960s, since the calcium-based sulphite mills had many environmental disadvantages in terms of their discharges of BOD, lignin, gases and dust, and chemical recovery. The discharges of the sulphite mills required radical external purification works while the sulphate process had the potential to recover chemicals and at the same time generate electricity (Wohlfart 1971b). Thus, the enforcement of the EPAct made it economically impossible for some mills to continue operating, especially small, inefficient mills, many of which were forced to shut down. And it was in this context that Swedish sulphite pulp mills were almost totally phased out. The Domsjö sulphite mill owned by the Modo Group is, however, one of few sulphite mills that survived in Sweden after the 1970s (Söderholm and Bergquist 2013).

Contributing to the reduction of discharges from the PPI was a process of structural rationalization, and Sweden was not the only country to experience this phenomenon. The OECD stated in 1973 that replacing small, uneconomical and environmentally obsolete units with larger more energy- and resource-efficient mills would create cost-effective means of reducing discharges per production ratio opportunity without deviating from the business inclination to seek productivity gains through capacity expansion. The approaches, however, differed between countries. In most cases, oxygen depletion of waterways required the rapid adoption of stringent Biochemical Oxygen Demand (BOD) limits, which typically locked industry into end-of-pipe measures (Rajotte 2003; OECD 1973). In Sweden, however, the pulp and paper producers at the time of the enforcement of the EPAct had a basic approach developed, one that already defined decades of environmental development activities and which they aimed at maintaining (i.e., to cope with the pollutants inside the mills and not when they "leave the plant") (Wohlfart 1971a, pp. 432–434).

The Swedish PPI's approach to dealing with pollution through changing internal processes can be traced back to the 1940s and 1950s and the pioneering R&D activities then taking place within collaborative platforms established jointly by the industry sector (see Sect. 4.4.3 below). While the first collaborative initiatives of the sector to deal with pollution problems had taken place already at the turn of the twentieth century, the R&D activities thereafter increased in tandem with the development of environmental policy up to the 1960s. During this novel period, attention was foremost directed towards improved efficiency and reduced fiber emissions. The practice of discharging fibers was highly inefficient as it resulted in 15% waste. Increased efficiency was therefore closely linked to the ability to reduce discharges of organic materials (Söderholm and Bergquist 2012). Also Swedish authorities and other industrial sectors, such as the metal smelting industry, early on chose to focus on in-plant measures and primary effluents instead of external measures (Lindmark and Bergquist 2008). For example, in describing this strategy, the environmental manager of Stora Enso in the 1990s explained that 'internal process changes have a completely different potential for the business. An end-of-pipe is a cost. An integrated solution can provide so much more. It can provide opportunities to increase production, reduce operating costs, and simply increase productivity. The key to all the improvements we have seen over the years is that the industry has been able to expand. The basic rule is that environmental improvements are a consequence of the corporate will to invest and its propensity to do so in the future. You take care of environmental costs as an integrated part.'[5]

4.4.2 Emission Reductions and Costs

During the 1970s and 1980s, costs for environmental investments were considerable for the Swedish PPI and accounted for 9–14% of the total investments (see Table 4.2).

[5]Interview with Per G Broman, Falun, 9 May 2006.

Table 4.2 Environmental investments in the Swedish pulp and paper industry, 1968–1988. Real prices (2000) million SEK (deflated using investment price index for the pulp and paper industry)

	68–70	71–75	76–79	80–84	85–88	Total
Environmental investments	930.2	4254.7	2372.9	2235.1	3388.9	13101.5
Environmental investments (share of total investments)	6	12.15	13.67	9.9	13.5	na

Source Swedish Forest Industries' Water and Air Pollution Research Foundation (SSVL) 'SSVL 74–85' (1989), see *Miljöskyddskostnader inom Svensk Skogsindustri 1985 t o m 1991. Appendix 2 and 20.* The estimates are based on questionnaire data collected by SSVL during the periods 1968–1970, 1971–1975, 1976–1979, 1980–1984, and 1985–1988. For a detailed accounting, see Söderholm and Bergquist (2012)

Over 60% of these investment costs involved altering internal processes that aimed to decrease water use and improve chemical and fibre recycling while about 14% of the costs concerned external wastewater purification measures. The cost share for air-purification measures amounted to only 15% (SSVL 1991, p. 93).

Costs were the highest among pulp producers (in comparison to paper producers), a sector in which the share of environmental investments in 1985–1988 accounted for as much as 17.5% of total investments (SSVL 1989, Appendix 10). This share was even higher among producers of bleached pulp at the beginning of the 1990s due to the discovery in the mid-1980s of the formation of dioxin in the process of pulp bleaching. Reported expenditures of US mills during the 1970s were on average 24% (i.e., higher than for the Swedish mills). However, during the 1980s capital expenditures on pollution control in the US PPI declined to 8.1% (Smith 1997, pp. 109ff).

The environmental investments undertaken by Swedish pulp and paper producers generated significant emission cuts. One example occurred in terms of Chemical Oxygen Demand (COD), which over the period 1970–1995 decreased from approximately 2.3 to 0.4 million tons annually. Moreover, from the mid-1980s to 2010 emissions of absorbable organic halides (AOX) declined by 97%.[6] AOX is associated with dioxin and is a measure of total halogens (chlorine, bromine, iodine). Emissions of sulphur, AOX and COD are shown in Figs. 4.1, 4.2 and 4.3, while pulp production is shown in Fig. 4.4.

[6] http://www.skogsindustrierna.se/skogsindustrin/branschstatistik/hallbarhet/. Accessed 19 January 2018.

4.4.3 Firm Collaboration

A central part of the technologies that generated the significant emission cuts illustrated in Figs. 4.1, 4.2, 4.3 and 4.4 were developed within collaborative R&D arenas of the Swedish PPI. And at the time of the enforcement of the EPAct (1969) these measures were supported by environmental authorities and involved various actors of importance to the innovation process. Two new collaborative environmental R&D

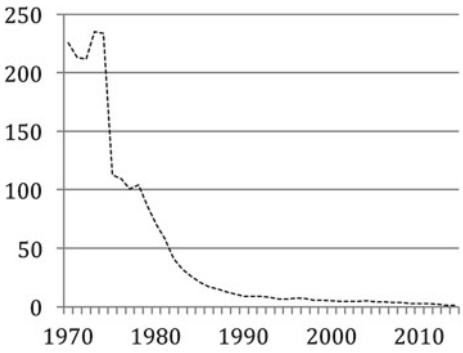

Fig. 4.1 Emissions of sulphur 1970–2014 (tons). *Source* Skogsindustriernas miljödatabas

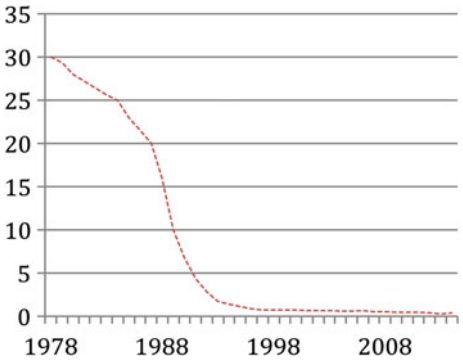

Fig. 4.2 Emissions of AOX 1978–2014 (tons). *Source* Skogsindustriernas miljödatabas

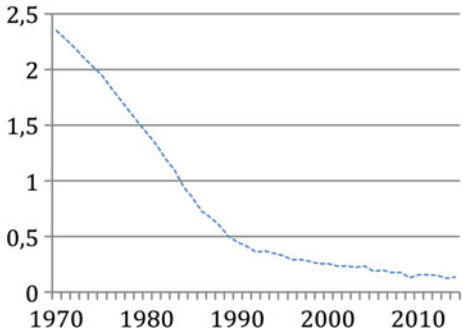

Fig. 4.3 Emissions of COD 1970–2014 (million tons). *Source* Skogsindustriernas miljödatabas

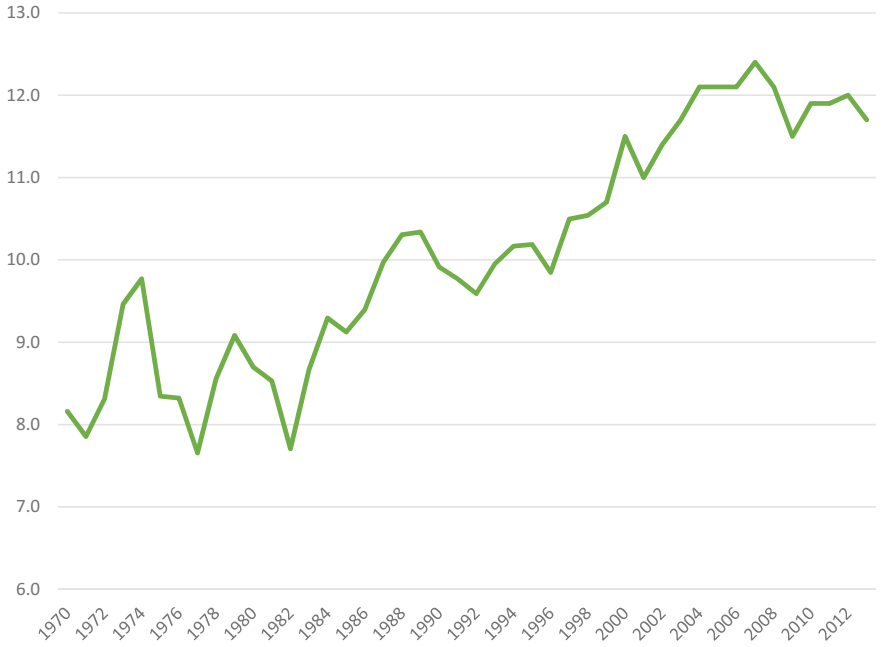

Fig. 4.4 Swedish pulp production 1970–2013 (million tons). *Source* Skogsindustriernas miljödatabas

platforms were established by the Swedish PPI in the 1960s. These were the state- and industry-funded Institute for Water and Air Protection (IVL), created in 1966, and the Forest Industries' Water and Air Pollution Research Foundation (Stiftelsen Skogsindustriernas Vatten- och Luftvårdsforskning, hereafter SSVL), established in 1969. The motive for creating the two new platforms was the recognized need for effective collaborative efforts in environmental R&D to manage costs and risks related to emerging stricter environmental requirements. SSVL consisted not only of a broad set of representatives from private companies, research institutions, and industry interest groups, but also of consultants, equipment suppliers, and research institutions outside the immediate sector (Bergquist and Söderholm 2011).

The furthering of green knowledge was accomplished in collaboration also with state agencies, particularly within IVL. The Institute was jointly founded in 1966 by the Swedish government and the Swedish pulp and paper industry in collaboration with other industrial sectors. Its primary assignment was to conduct research on the relationship between industrial production and environmental problems and to identify effective solutions. Closely affiliated with the Institute was the Industry Water and Air Protection Agency (Industrins Vatten och Luftvård AB), a service company that was also established in 1966. Together with SSVL, IVL supported knowledge diffusion and technology development of decisive importance to the green reconstruction of the Swedish PPI from the 1960s through the 1970s and 1980s

Table 4.3 Industry-wide collaborative platforms of the Swedish pulp and paper industry for environmental R&D until 1973

Organisation/Institute	Time period
Sulphate Pulp Committee	1908–1909 (?)
Swedish Pulp and Paper Research Institute (STFI)	1945—Changed name to Innventia AB in 2009
Central Laboratory of the Pulp Industry	1936 (ascended into STFI in 1968/69)
Water Pollution Committee	(1937) 1945–1953/54
Water Laboratory of the Forest Industry (SIV)	1953/55–1964 (was first governed by the Water Protection Committee of the Forest Industries, but in a few years turned into the Forest Industries' Water Protection Council respectively the Forest Industries' Water Protection Research Foundation, which in turn later became SSVL)
Swedish Forest Industries' Water and Air Pollution Research Foundation (SSVL)	1963—still active
Institute for Water and Air Protection (IVL)	1966 (today IVL Swedish Environmental Research Institute)
Service company of IVL	1966 (acquired by the Swedish Steam Boiler Association in 1982)
Energy Committee	1973—still active

Sources Söderholm and Bergquist (2012, 2016)

(Bergquist and Söderholm 2011). Table 4.3 provides an overview of the collaborative platforms.

It should be mentioned that the knowledge generated within IVL and SSVL was applied by the Franchise Board of Environmental Protection (FBEP) while enforcing the EPAct through the issuing of individual permits. The network connected to the environmental R&D activities, including both industry and environmental authorities, hosted technical competence and contributed to a shared understanding of reasonable courses of action. There were, however, conflicting opinions on how far industry could and should go in terms of environmental compliance.

4.5 Towards ECF and TCF Bleached Pulp

The replacement of elemental chlorine as a bleaching agent with the alternative ECF and TCF methods is an internationally well-known, environment-related technological shift of the PPI. Technically the ECF-process means a complete replacement of chlorine (Cl_2) with chlorine dioxide gas (ClO_2). By replacing Cl_2 with ClO_2 the levels of absorbable organic halogens (AOX) are greatly reduced. The TCF-process in turn completely eliminates the use of both Cl_2 and ClO_2 and instead typically

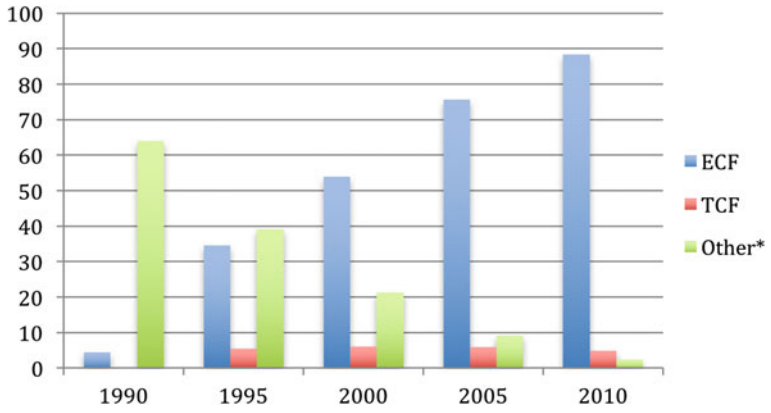

Fig. 4.5 Bleached chemical pulp production in the world 1990–2010 (million tons). *Source* Alliance for Environmental Technology (2012). *Pulp bleached with some molecular chlorine

uses hydrogen peroxide (H_2O_2) and/or ozone (O_3) as substitute bleaching agents. The development of ECF and TCF was further aided by improvements in the oxygen delignification technology, which is used in a pre-bleaching stage.

The shift towards alternative bleaching methods was dramatic. Soon after the US EPA in 1985 detected low concentrations of the extremely toxic and highly chlorinated hydrocarbon group dioxins in fish caught downstream from a few pulp mills, Swedish environmental authorities immediately followed up with their own investigations. In 1986, they concluded that crabs collected near the effluent site of a Swedish pulp mill contained dioxins, too. In the years to come the issue of bleached pulp was raised on both public and governmental agendas throughout the world and regulatory processes were initiated in pulp producing countries. Hence, the risks of dioxin were intensively discussed during the European environmental debate and Sweden was, already in 1988, the first country in the world to set up a regulatory standard for controlling dioxins discharged from pulp mills (Bergquist and Söderholm 2015). Until recently the dioxin alarm has caused a major shift in bleaching technologies worldwide. In 1990, 93% of the bleached pulp produced in the world was bleached by elemental chlorine and in 2010, this figure had dropped to only 2.7%. Figure 4.5 illustrates this development.

It is widely recognized that the technologies diffused more rapidly in the Nordic countries compared to North America, and it is mainly explained by the rise of green consumerism in key markets for Nordic producers while the demand for chlorine free paper was absent in North America (Marcus 1999; Smith and Rajotte 2001; Harrison 2002; Norberg-Bohm and Rossi 1998; Reinstaller 2005; Smith 1997; Popp et al. 2011). Figure 4.6 illustrates the diffusion of ECF and TCF in the Nordic countries since 1990.

Yet, it gives a highly simplified picture to bundle the Nordic countries in this respect. It is rarely highlighted that the great majority of the pulp producers a few

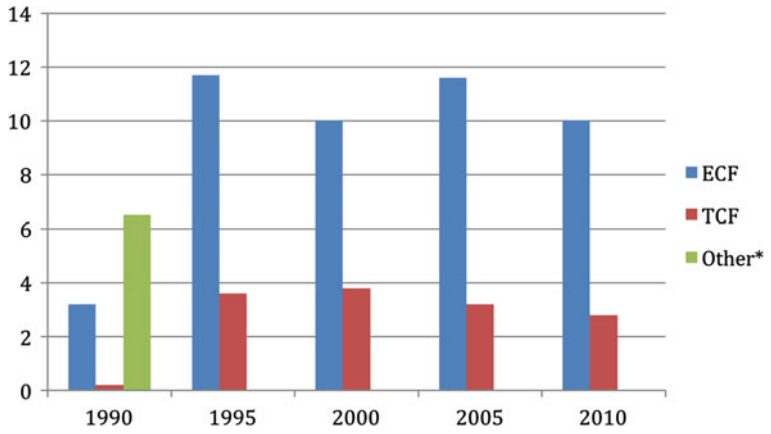

Fig. 4.6 Bleached chemical pulp production in the Nordic countries 1990–2010 (million tons). *Source* Alliance for Environmental Technology (AET) (2012). *Pulp bleached with some molecular chlorine

years into the 1990s that had switched to TCF were of Swedish origin and only two were Finnish (Reinstaller 2005, p. 1373). Moreover, Swedish producers, unlike Finnish ones, pioneered in the development of several core technologies for the ECF and TCF processes in the 1970s and 1980s, such as oxygen delignification, advanced batch cooking and ozone bleaching (Kramer 2000). The pioneering development of the Swedish producers in this matter should be viewed in the collaborative context of the long-term environmental R&D activities discussed above, and where important technological steps were achieved through networks of actors rather than by single firms. Thus, already from the mid-1970s, projects were initiated within both SSVL and the Swedish Pulp and Paper Research Institute (STFI) that focused largely on improving the pulp bleaching process.

The projects typically aimed at decreasing the use of chemicals while increasing the level of delignification in the boiler and in the subsequent oxygen delignification. The main motives in these instances were both to decrease costs and cut emissions from the bleaching process. Thus, already in the early 1970s a pilot plant for oxygen delignification was constructed at a Swedish mill in relation to a SSVL-project (Jerkeman 2007), and two decades later the technology was installed at all Swedish pulp mills (SSVL 1991). At the time, the Nordic Council of Ministers considered the oxygen delignification technology the single most effective process-oriented device to reduce chlorinated organic compounds (Nordic Council of Ministers 1989, pp. 21–23). Also during the 1980s, methods of boiling the pulp to very low levels of lignin, which in turn reduced the need for bleaching chemicals even further (Modified Continuous Cooking/Super Batch Cooking), were developed jointly (and later widely diffused) in the Swedish PPI (Jirvall and Noodapera 1995).

Industrial pollution was regulated only in a limited way in Finland before the late 1980s, and the preferred method for controlling it was by end-of-pipe technology and

particularly the activated sludge method. In 1989, it was applied by a large number of Finnish sulphate/kraft pulp mills, whereas Swedish mills instead were better outfitted with pollution prevention methods such as oxygen delignification, modified cooking and high chlorine dioxide substitution (Auer 1996). Hence, before green consumerism grew in key markets for Nordic producers in the 1990s, regulation was the main trigger for the technological shift towards ECF and TCF among Swedish pulp mills. The Swedish FBEP took an initial and clear standpoint on the dioxin issue already in 1986, when it delivered final conditions for a sulphate pulp mill that declared that the wastewater from the bleaching process caused significant local damage to water-based organisms. In 1988, the Swedish Parliament in turn adopted a proposal which stipulated that industrial emissions of chlorinated organic compounds must be reduced to 1.5 kg AOX/ton of pulp (the normal emission level was about 4 kg per ton of pulp at this time). At this stage the Swedish Forest Industries Federation identified necessary investments to meet the requirements that were now being enforced to be 4 billion SEK, and for some mills it represented up to 50% of the annual investments until 1992 (Bergquist and Söderholm 2015).

The final important technological steps towards TCF pulp were taken by the Swedish PPI in the very last years of the 1980s due to rather forceful regulatory action. In 1988 the Swedish government imposed on an individual mill the most stringent standards ever, namely a maximum of 0.5 kg AOX per ton. The mill, Aspa, was a small producer of market pulp, however, with a sensitive location on a lake that supplied drinking water for several communities. Already in the spring of 1989 the mill had installed a method which produced these low emissions, however, after further development work and only a year later the so called Lignox process[7] allowed for the production of TCF pulp. Hence, the small Swedish firm ASPA was the first mill in the world to produce TCF pulp. Still, it is important to note that while the development work physically took place at ASPA, the knowledge underpinning the transition stemmed from the long-term, industry-wide R&D collaboration described above (Bergquist and Söderholm 2015). In 1991, ASPA would, in collaboration with Greenpeace, produce an exact copy of "Das Plagiat", the weekly *Der Spiegel* printed on paper made of such pulp. It was the first time a magazine was published on chlorine-free paper and this accomplishment has been recognized as having had a great impact on the German public and, by extension, also the greater northern European market (Waluszewski and Håkansson 2004). For the first time it was possible to market paper products with eco-labelling, an opportunity which the Swedish producers quickly embraced. At this time the Swedish Nature Conservation Association had launched two environmental classes ("low chlorine" and "chlorine-free") for bleached paper qualities which got major impact and helped the Swedish pulp producers in their first-time marketing of green products by this new 'eco-labelling' standard (i.e., chlorine-free pulp and paper products) (Bergquist and Söderholm 2015).

[7]The Lignox process: oxygen-bleached pulp is treated with hydrogen peroxide at high temperatures after the removal of heavy metals with a complexing agent. Subsequent final bleaching takes place with peroxide and chlorine dioxide.

In 1994 the majority of the Nordic mills that had switched to TCF were of Swedish origin (remaining Swedish mills had all switched to ECF). In contrast, only two mills in Finland had opted for TCF by this time. Also in the US, the companies showed little interest in shifting to TCF. Only two mills had adopted TCF in 1994 (Reinstaller 2005, p. 1373). Thus, also in the US the policy process moved more slowly and when the Swedish PPI aimed to produce ECF and TCF pulp in the early 1990s the US EPA still worked on defining the standard settings for controlling dioxin emissions. For the US PPI, the dioxin issue meant something completely new as the mills could not compel only by installing end-of-pipe treatments, but had to invest significant funds in adopting new bleaching technology. The US industry had never had a strategy to deal with pollution with internal process changes (Gunningham et al. 2003, p. 16; see also Smith 1997). Thus, resistance to making the change was strong within the sector because it would come both at a very high cost and without any advantages on the American market (Reinstaller 2005, 1380). This helps explain why Greenpeace failed in its attempt in 1992 to convince the American *Time* magazine to switch to publishing on TCF paper. Further, Georgia Pacific, one of the leading pulp and paper producers in the US, announced in 1992 to consumers that it would not market TCF pulp, having decided to invest in chlorine dioxide substitution instead. Customers who wanted TCF pulps would have to seek other suppliers. This was possible for the company to do and say as as the demand for TCF pulp was very low on the US market (Smith 1997, p. 131). And together with a slower regulatory process, US firms could wait to alter the technology. In this context, in 1989 the US Office of Technology Assessment (OTA) reported on the compliance of Swedish pulp mills to more stringent standards of chlorinated organics, whereas it was unreasonable to hold US firms to such standards due to both economic risks and scientific uncertainties (Powell 1997, p. 12).

In sum, although the shift towards ECF and TCF apparently happened swiftly in the Swedish case, the development-path towards environmentally friendlier bleaching methods was the outcome of decades-long industrial efforts to increase efficiency and reduce the use of chemicals by changing internal processes, in combination with long-term and continuously strengthened environmental legislation.

4.6 Energy Transition

In parallel to the "greening" of industry with respect to increased pollution control, two other issues have had a major impact on the greening of the PPI since the early 1970s. The first is the increased energy prices in the wake of the oil crises of the 1970s and the second is the fear of a shortage of wood. Neither issue was initially related to environmental concerns, but became increasingly so as they drove serious concerns about energy use and triggered energy savings as well as recycling.

Indeed, pulp and paper production is energy-intensive. The PPI is the largest energy user in Sweden and accounts for 52% of the total industrial energy use (SEA 2017, p. 21). In the EU the PPI accounts for 14% of the total industrial energy

use (Jönsson 2011). Although energy usage was always important to the PPI due to its cost, the 1970s caused the need for energy savings and finding substitutes for oil to become preeminent concerns, and both the Swedish PPI and the Swedish government turned their attention to phasing out oil from pulp and paper production. As a consequence, the energy mix of the Swedish PPI underwent radical changes over the 1970s and 1980s, and a large-scale substitution of oil took place. In 1973, for instance, oil accounted for 43% of the total external energy use and in 1984 the share of oil had decreased to 16%. Moreover, in 2011 the corresponding number was 5% (Skogsindustrierna 2012).

Oil reduction was mainly achieved through increased use of internal biofuels (external biofuels remained fairly constant); between 1973 and 1984 the share of energy generated from internal biofuels increased from 55 to 72%, and in 2011 the share of biofuels was 79% (Fig. 4.7). The biofuels mainly consisted of by-products from the pulp manufacturing process, where the biggest share is generated from black liquor[8] and the rest from bark and wood residues (Federation of Swedish Forest Industry 2012). The overall reduction in the use of oil was also made possible through other energy efficiency improvements and increased internal production of electricity through back-pressure turbine power generation (Lindmark et al. 2011; Bergquist and Söderholm 2016). Of central importance for this development, which included a great deal of incremental technology development, was the already established collaborative strategy of firms within the industry and between the PPI and state authorities. Thus, a large number of energy projects were subsequently conducted through inter-firm and state-industry collaborative R&D platforms established after the 1940s, such as STFI and SSVL (Table 4.3). Still, to manage the new energy challenge the Swedish PPI had already appointed in 1973 a standing Energy committee consisting of 12 members from among management and technical personnel within the sector.

Between 1973 and 1977, no fewer than 51 new energy projects had been initiated or at least proposed within the sector. These involved 37 energy conservation and 14 energy generation projects, most conducted/proposed in collaboration with organisations closely associated with the sector, such as STFI, the Swedish Cellulose and Paper Mill Association (SCPF), and the Steam Generator Association (in Swedish Ångpanneföreningen), but also universities and research institutes outside the sector (e.g., the Thermal Engineering Research Institute (in Swedish Värmeforsk) (Marklund 1994, p. 143). Underpinning the energy transition of the Swedish PPI was also a proactive governmental strategy to emphasize knowledge management and collaboration with industry along with the substitution of oil with internal biofuels. The Swedish government assigned significant subsidies for prototypes and demonstration plants by its energy policy decision in 1975, and such activities increased greatly over time. In the 1970s, new processes and technologies on a factory-wide scale could be subsidised by as much as 50% (Bergquist and Söderholm 2016).

[8] In the pulping process, cooking chemicals, known as white liquor are used to break out and dissolve the lignin whereas the white liquor becomes black liquor. There are basically two main process streams coming out of a pulp mill: cellulose fibres and black liquor.

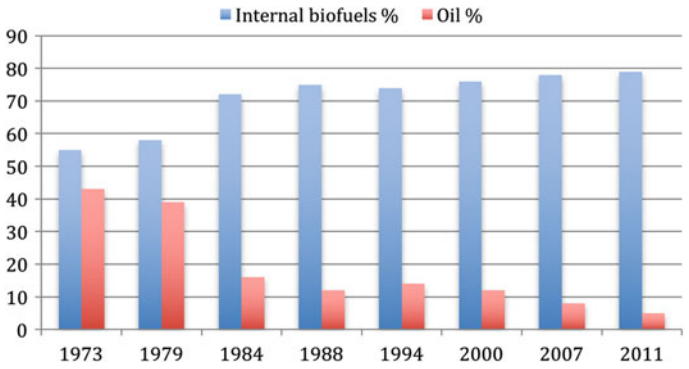

Fig. 4.7 Consumption of internal biofuels and oil in the Swedish pulp and paper industry 1973–2011 (% of total energy use). *Source* Skogsindustrierna (2012)

The energy transition of the Swedish PPI was further driven by the fact that focus was directed towards unutilized potential energy sources, whereby a previous waste problem with bark and chips now could be transformed into energy savings and improved energy efficiency. Moreover, the PPI feared shortages of wood and sought means of increasing its energy efficiency, and these forces led it to increase its use of recycled paper in pulp production; producing pulp from recycled paper requires only one-fifth of the energy needed produce it from virgin wood. Thus, from not have being utilised at all in Swedish pulp production prior to 1975, recycled paper came to be an important raw material. Of central importance for this development were improved methods for removing ink and other contaminants, such as plastic, from the recycled paper, and the introduction in 1975 of a compulsory collection system for old newsprint from households (Bergquist and Söderholm 2016).

The improved energy efficiency over the 1970s and 1980s coincided with an on-going structural change in industry, namely a trend towards fewer and larger production units and an improved ability to take advantage of economies of scale (Järvinen et al. 2012). The concentration of production units allowed, among other things, a higher degree of integrated pulp and paper production whereby the energy intensive step of drying the pulp (at the pulp mill, only to dissolve it again at the paper mill) was avoided (Bergquist and Söderholm 2016).

Today the energy efficiency of the Swedish (and Finnish) PPI is higher than that of other major pulp producing countries such as Brazil, the US and Canada (Fracoro et al. 2012). However, the sector's total energy use has stayed relatively constant due to considerable production expansion, not the least in mechanical pulp, which has contributed to increased use of electricity beginning in the early 1980s (Bergquist and Söderholm 2016). As a result, between 1973 and 2011 the total energy consumption of the Swedish PPI increased from 55 to 57 TWh (Skogsindustrierna 2012).

4.7 Environmentally Driven Transformation—The Past and the Future

Over the second half of the twentieth century, environmentally related issues have played a central role in the overall transformation of the Swedish PPI, essentially as new or altered technologies were required to comply with tightening environmental regulations. In the Swedish context, the greening of polluting manufacturing industries occurred within a collaborative regulatory framework. Knowledge was flowing quite openly between Swedish PPI companies and between environmental authorities and the regulated industry (Bergquist and Söderholm 2011). Thus, the significant environmental improvements accomplished within the Swedish PPI over the period can only be fully comprehended by acknowledging the role of collaborative environmental R&D activities among firms within the sector and between the sector and the state. Furthermore, the development after the 1980s illustrates how the emergence of green consumerism came to play a role as a driver towards environmental improvements.

The PPI has developed from being considered one of the greatest polluters of the twentieth century to an industry that is essential for the transition towards a more sustainable (bio-based) economy. In the EU, the PPI today already constitutes the biggest single industrial producer and user of renewable energy; 56% of the industry's primary annual energy consumption is bio-based (CEPI 2013, p. 50). And there are big hopes, especially within the Nordic countries, that the PPI will take the lead in clean-tech innovation and green growth in terms of new products and the second generation of "green" fuels. The trend is global and most pulp and paper companies are working in this direction. The sector, particularly in Sweden, is at the same time undergoing substantial change due to stricter environmental regulations, unstable oil price, energy policies, global competitiveness and structural changes. In addition, the increasing use of electronic rather than paper-based communications are pushing the PPI towards self-renewal to improve its profitability (Backlund and Nordström 2014), whereby it appears as if parts of the pulp industry have experienced a shift towards becoming more "biorefineries" than pulp producers (Hamaguchi et al. 2012). One promising technology in this context is black liquor gasification (BLG[9]) for the production of fossil free transportation fuels. Here the Swedish company Chemrec has been a key player. Thus, in 2009 and after 20 years of R&D on BLG, Chemrec constructed a pilot plant with financial support from the European Community's Framework Programmes and the Swedish Energy Agency. However, after the BLG demonstration technology did not develop as expected and there was a lack of investment interest in the project, the pilot plant was shut down and remains so. One suggested reason for the lack of interest was the uncertain market conditions for biodiesel and methanol (SEA 2016). Still estimates suggest that BLG could supply as much as 7% of Sweden's total—not merely its industrial—demand for electricity, or as much as 30% of the nation's demand for transportation fuels. Outside the

[9]BLG is in its essence a process in which a clean synthesis gas is produced from black liquor by converting its biomass content into gaseous energy carriers (Bajpai 2014, p. 3).

Nordic countries, BLG technology is also developing in the US and Asian pulp producing countries (Bajpai 2014, pp. 4–5). Another example of on-gong renewals is the Finnish Metsä Group's investment in the 'bioproduct' mill in Äänekoski, Central Finland, which represents the largest forestry investment ever made in Europe, with a value of EUR 1.2 billion. It is the first next-generation product mill in the world, were the production of energy, along with the pulp production, play a crucial role (Metsä Group 2017).

Another trend in the PPI's development towards environmental improvements, partly from new products within the area of bioenergy, is a continued expansion of the traditional pulp and paper production driven by an increased global demand for cardboard and sanitary papers. For example, the Swedish pulp and paper company SCA is currently rebuilding its Östrand mill (due to start up in 2018) in Sweden and has publicized that a large-scale investment programme will lead to 'the largest production line for bleached softwood sulphate pulp in the world'. Above all the firm has stressed that the mill will become 'world class in terms of product quality, environment and competitiveness' by becoming 'the leader in terms of resource management' and 'generate surplus energy' to be sold in the form of 'green electricity and district heating'. Also, 'both TCF and ECF pulp' will be produced with raw materials from 'sustainable forestry'.[10] Overall the Swedish example illustrates, however, that the transition towards cleaner and more energy efficient pulp and paper production is the result of long-term and incremental processes, whereby the technology development has been shaped by both evolving markets, long-term collaborative R&D and shifting concerns in society about the natural environment and human well-being.

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IChemE

0957–5820/00/$10.00+0.00

Trans IChemE, Vol 78, Part B, January 2000

THE ROLE OF CHLORINE IN DIOXIN FORMATION

B. K. GULLETT, A. F. SAROFIM*, K. A. SMITH* and C. PROCACCINI*

US Environmental Protection Agency, National Risk Management Research Laboratory, North Carolina, USA
**Department of Chemical Engineering, Massachusetts Institute of Technology, Cambridge, Massachusetts, USA*

There is poor correlation between total chlorine in waste streams and formation of polychlorinated dibenzodioxin and polychlorinated dibenzofuran (PCDD/F) during waste combustion. This is because the active chlorine (Cl) species are strongly dependent upon combustion conditions. For homogeneous conditions, trace amounts of a hydrocarbon species (benzene) injected into the effluent from complete combustion of a mixed chlorocarbon fuel (ethylene and chloromethane) results in formation of stable, oxygenated and chlorinated compounds. This occurs over a broad range of temperatures (400–900°C), provided that a fraction of the system chlorine is in the Cl radical form. Cl is the only form in which chlorine can react, in gas-phase, with stable hydrocarbon species, and these reactions are very fast, even in the low temperature regime. Molecular chlorine (Cl_2) may subsequently participate in reactions with carbon-based radicals, and these reactions are the primary source of chlorinated products. Hydrogen chloride (HCl)—the major chlorine species in the products—can react with oxidizing radicals (eg, OH) and promptly form significant amounts of Cl. Gas temperature has a great influence on the final distribution of products. At 800–900°C, practically all of the benzene which was attacked by Cl is converted to carbon monoxide and small unsaturated hydrocarbons by subsequent reactions with oxygen. At about 750°C, measurable concentrations of chlorobenzenes are formed. At lower temperatures (400–600°C), chlorophenols become a large fraction (up to 15%) of the total reacted benzene. Heterogeneous reactions result in the formation of Cl_2 through catalyzed reactions (most actively by copper salts) and promotion of carbon-Cl bond formation. The latter is dependent on the ash surface species and type, ash adsorptive characteristics, temperature, and presence of regenerative Cl in the waste combustion products.

Keywords: polychlorinated dibenzodioxin; dibenzofuran; air pollution; combustion; chlorination.

INTRODUCTION

The public concern with the emissions of 'dioxins' or polychlorinated dibenzodioxin and polychlorinated dibenzofuran (PCDD/F) from incinerators has resulted in periodic calls for the banning of chlorine from waste streams. This paper reviews the mechanisms for the formation of dioxins to try to establish a better understanding of the role of chlorine in the dioxin formation process in order to guide both control measures and regulations on the composition of the waste stream.

The formation of PCDD/F as trace by-product pollutants from combustion processes has been extensively studied over the last 15 years. These chemicals are highly persistent, bioaccumulating in the environment and ending up in the food chain. The more toxic form has been classified as a 'known' human carcinogen[1], and they have been linked with tumour formation, learning deficits, endometriosis, immunosuppression, and developmental/reproductive effects[2,3] in experimental animals.

PCDD and PCDF are each composed of eight homologue groups (mono through octa), distinguished by the number of chlorine (Cl) atoms attached to their ring structures. The eight homologue groups have varying numbers of structural isomers, totalling 210 congeners (75 of PCDD, 135 of PCDF). Of these 210, 17 are considered toxic to varying degrees due to their biological activity, and each is Cl-substituted in at least the 2,3,7,8 positions. Each of these 2,3,7,8 isomers has been assigned a toxicity equivalency factor (TEF) which, when combined with the concentration, produces a weighted toxic equivalency (TEQ) of the mixture.

The occurrence of PCDD/F in the environment, as deduced from the depth profiling in sediments, has grown dramatically since the 1930s in parallel with the growth with the production of chlorinated organic compounds[4]. This has led to the attribution of the dioxins to primarily anthropogenic sources with incineration implicated as a major source[5]. The dioxins are widely distributed globally[6] with the concentrations being generally highest in industrialized countries.

Despite the predominance of PCDD/F from combustion sources, our understanding of the formation mechanisms and reaction rates in combustion systems has not yet reached the point where we can predict the potential of a system to form PCDD/F or take adequate measures to substantively prevent formation. This is due to the extremely difficult nature of the reaction mechanism: only trace concentrations of products are formed; multiple gas- and solid-phase reactions are involved; the combustor reaction environment is difficult to simulate; the reactants themselves are present at only trace concentrations; simultaneous chlorination, dechlorination, formation, and decomposition reactions occur; and monitoring PCDD/F

formation is possible only through application of long and difficult manual sampling methods and costly chemical analyses.

THEORIES AND FACTORS

PCDD/F are formed via both homogeneous and heterogeneous processes with the heterogeneous pathway generally considered to be the more important. There are two main mechanistic theories to account for observed PCDD/F heterogeneous formation of which the distinction is likely to be blurred by further research. The first theory is the so-called '*de novo* synthesis' mechanism which has been postulated and demonstrated by Stieglitz and coworkers[7]. In this mechanism, the presence of fly ash containing unburnt aromatic moieties and metal catalysts is essential. Gas-phase Cl, likely as hydrogen chloride (HCl), is said to form metal - chloride ligands on the fly ash surface, after which Cl is transferred to carbon (C). The subsequent metal-catalysed oxidation/gasification of the fly ash surface releases these C-Cl structures, some of which comprise the subject PCDD/F pollutants, and others which form related compounds such as chlorophenols, chlorobenzenes, and non-aromatic chlorinated hydrocarbons. The second theory is the so-called 'precursor' theory which has been demonstrated by Dickson and Karasek[8] and Karasek and Dickson[9]. In this mechanism, combustion byproducts including compounds such as chlorophenol or chlorobenzenes react on the catalyst-laden fly ash surfaces to form PCDD/F through condensation reactions. More recently, it has been demonstrated[10] that polycyclic aromatic hydrocarbons (PAHs) also chlorinate and decouple to form isomer-specific PCDD/F compounds. Both of these theories have been adequately validated in laboratory experiments. Arguments based on relative reaction rates, product congener profile comparisons, and temperature dependencies have been used to support both theories. The relevance of each of the theories, however, is difficult to ascertain: laboratory simulations of assumed microscale phenomena leave considerable doubt regarding their ability to replicate or represent conditions in a practical combustion system. The actual situation may be such that the carbon source is derived from both condensing, gas-phase organics and volatilizing, fly-ash-derived organics, the relative magnitude of which is due to system- and operational-specific variables but both of which subsequently participate in the same, or a similar, reaction mechanism.

At any rate, the generally requisite conditions for formation of PCDD/F are: (1) incomplete combustion of an organic fuel; (2) the presence of trace metals and surfaces to act as catalysts; (3) a temperature/time history providing at least 1 s below 600°C; and (4) a source of Cl. The focus in this paper will be Cl: evolution of various Cl species, Cl species concentrations and temperature/time dependency, mechanisms in carbon chlorination, and combustion factors that affect Cl species.

CORRELATION OF PCDD/F WITH CHLORINE IN WASTE

Data from some 169 municipal waste combustors, hazardous waste incinerators, medical waste incinerators, cement kilns, hazardous-waste-fired boilers, biomass combustors, and laboratory- and pilot-scale combustors were examined to determine if there was a correlation between the chlorine content in the feed and the PCDD/F concentrations in the stack[11]. The chlorine content in the feed varied from less than 0.1% for some biomass combustors to over 80% for some hazardous waste incinerators. The chlorine in the waste stream was in the form of inorganic salts as well as synthetic organic products including polyvinyl chloride (PVC). The results of the statistical analysis of the data showed that there was no significant effect of chlorine in the feed and the PCCD/F concentrations in the stack for field data although well controlled laboratory experiments showed an increasing concentration of PCDD/F with inlet chlorine content. The conclusion of the study was that in field studies the effects of confounding variables such as the temperature of the particulate control device, the excess air, and other combustor operating conditions masked any effect of the chlorine content in the feed. In order to understand such data, it is therefore important to understand what the active form of chlorine is and how it is influenced by changes in design and operating conditions of combustors and air pollution control devices.

Chlorine is undeniably required for formation of PCDD/F but only trace amounts in the fuel feed are needed to provide the minimum amount. For example, while emissions of PCDD/F from coal combustion are generally considered to be low, if measurable, even coal with less than 1 ppt (part per trillion, 1×10^{-12}) of Cl has more than sufficient Cl to produce PCDD/F that would exceed common regulatory limits. So the relevant question is not what amount of chlorine is limiting but what are the pathways for PCDD/F versus other chlorinated pollutants?

Homogeneous Formation

Which form of chlorine favours the formation of chlorinated hydrocarbons and PCDD/F?

The three forms of chlorine present in the post-combustion products of organic wastes are HCl, Cl, and chlorine (Cl_2). The hypothesis, for which support will be provided, is that the active form of chlorine is atomic chlorine and not total chlorine and that the concentrations of chlorinated hydrocarbons can be changed significantly by manipulating post-combustion conditions to modify the concentrations of either the atomic chlorine or hydrocarbon intermediates.

While the majority of the system chlorine in the products is in the HCl form, a non-negligible fraction may be present as Cl radicals and Cl_2. These two species (as opposed to HCl, which has very little reactivity in the combustion environment) can react with organic compounds. Cl radicals can readily abstract H atoms from stable hydrocarbons, forming carbon-based radicals and HCl. At high temperatures, 1400–1500°C, equilibrium indicates that a minor, but not negligible, fraction of the total system chlorine is in the Cl radical form. Furthermore, kinetic constraints may cause the persistence of high concentrations of Cl radicals, which exit the primary zone of chlorocarbon flames. The high concentrations of Cl radicals that survive during the cool-down of combustion products can promote the fast, gas-phase formation of chlorinated and oxychlorinated pollutants, under conditions possibly arising during practical combustion processes.

The concentrations of HCl, Cl, and Cl_2 can be calculated for different conditions by taking the following reactions into account.

Interconversion reactions of Cl and HCl:

$$HCl + OH = H_2O + Cl$$

$$HCl + O = OH + Cl$$

$$HCl + M = H + Cl + M$$

$$HCl + O_2 = HO_2 + Cl$$

Interconversion reactions of Cl and Cl_2:

$$Cl_2 + M = Cl + Cl + M$$

$$Cl_2 + O = ClO + Cl$$

$$Cl_2 + H = HCl + Cl$$

The kinetics of the reactions are relatively well understood, and the concentration of Cl and Cl_2 can be calculated with fair confidence[12,13]. The factors that favour high chlorine concentrations are: (1) high temperatures; (2) rapid cooling from high temperatures because of the slow rate of recombination of the chlorine atoms; and (3) high oxygen or OH concentrations because of their abstraction of H from HCl.

As the temperatures are reduced, the atomic chlorine recombines to form Cl_2. High concentrations of molecular chlorine in combustion products are therefore an indirect measure of high concentrations of atomic chlorine.

What conditions favour the formation of chlorinated hydrocarbons?

The reaction of chlorine atoms with hydrocarbons in the presence of oxygen will depend upon the temperature of reaction. This will be demonstrated here by summarizing results by the authors of injecting benzene into the lean post-flame combustion products of a chlorinated hydrocarbon after the combustion products had been cooled to different temperatures[13].

High-temperature oxidation: At 1100°C, benzene was promptly oxidized and converted to CO_2 (Figure 1-a). At the same time, a significant increase in the concentration of Cl_2 in the quenched samples was observed (Figure 1-b) indicating that new Cl radicals were generated by the reaction of HCl with oxygen-containing radicals (primarily OH). The OH radicals needed to drive this process were those generated during the full oxidation of the carbon species. These results indicate that benzene was oxidized primarily by the initial attack of oxygen species. H abstraction by Cl radicals certainly occurred, at least to some extent, and the resulting phenyl radicals were completely oxidized to carbon dioxide (CO_2). It can be concluded that, due to the high temperature, only the products of the complete oxidation of benzene were formed.

Intermediate temperature reactions: A similar experiment conducted at 900°C (Figure 1-a) showed that about 100 ppm of benzene is initially consumed and converted to smaller secondary products (primarily carbon monoxide (CO), acetylene, and vinyl acetylene). After the initial phase (0–2 ms), benzene oxidation continues at a much slower

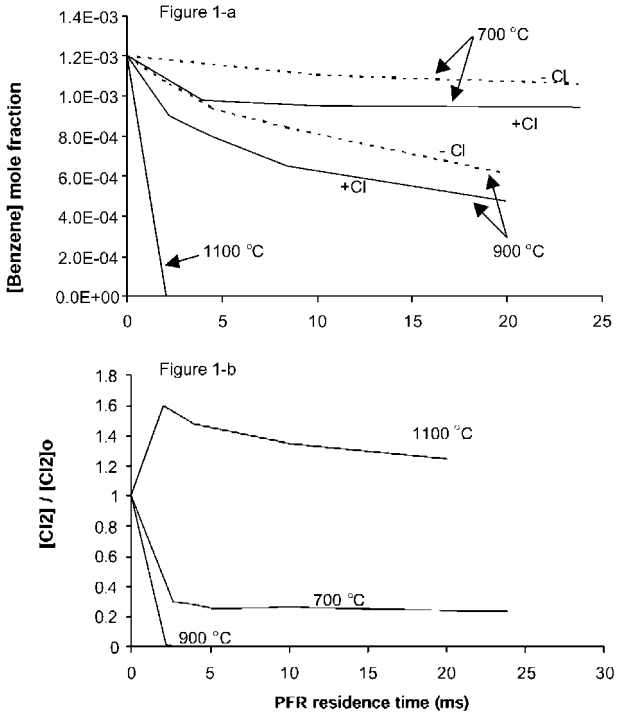

Figure 1. Concentration profiles of benzene (Figure 1-a), and Cl_2 (Figure 1-b) in quenched combustion products. Measurements relative to three different plug flow reactor (PFR) temperatures (700, 900, and 1100°C) are reported. For benzene measurements, data relative to control experiments (dashed lines labelled '-Cl') are also reported. During the control experiments, no chloromethane (ie, the only chlorine source) was fed to the primary reactor stage and the benzene concentration was measured. The comparison of the benzene profiles with and without chlorine in the system allows one to estimate the fraction of benzene which was consumed through the direct interaction with chlorine species. In Figure 1-b, data on Cl_2 concentration refer to the measurements in quenched combustion gas and are normalized with the Cl_2 concentrations measured just upstream of the secondary benzene injection. The initial values of the Cl_2 mole fractions are 1.6×10^{-4}, 1.4×10^{-4}, and 1.1×10^{-4}, respectively, for the 1100, 900, and 700°C measurements.

rate, yielding an increasing amount of the same secondary products. Given the relatively low temperature, the oxidation process was not complete and, after a residence time of 25 ms, no measurable CO_2 production was observed. A parallel decrease in the concentration of Cl_2 in the quenched products (Figure 1-b) indicates that the initial benzene consumption occurred through reactions with Cl radicals. The likely products of the reaction of benzene and Cl were HCl and phenyl radicals. The latter were subsequently attacked by oxygen species and converted to secondary products. Chlorobenzene, the only chlorinated product of incomplete combustion (PIC) observed, accounted for just a minor, although non-negligible, fraction of the conversion products (about 1.5 ppm mole fraction, which was equivalent to 1.5% of the total benzene consumed). Chlorobenzene may have formed, at least in part, by the reaction:

$$\text{phenyl} + Cl_2 \rightarrow \text{chlorobenzene} + Cl$$

Lower reaction temperatures: A third experiment was conducted at 700°C and, this time, benzene consumption was observed practically only in the initial phase of the process (Figure 1-a). The product distribution included the same secondary reaction products observed at 900°C

and a much larger fraction of chlorinated or oxychlorinated species (the sum of the concentrations of chlorobenzene, 2-chlorophenol, and 4-chlorophenol, measured after a residence time of 25 ms, accounted for about 12% of the total benzene consumed). The concentration profile of Cl_2 (measured in the quenched samples) showed a different pattern than in the two previous cases. After a prompt decrease in mole fraction to about one fourth of the initial concentration, the Cl_2 level remained practically constant. This indicates that, in fact, not all the Cl_2 measured in the quenched products sampled before the benzene injection was, at 700°C, in the form of Cl, but only about three-fourths.

The above results are consistent with the following mechanism:

(1) At a low enough temperature (eg, 700°C), benzene reacts with chlorine species and oxygen in a process initiated either by H abstraction by Cl radicals or via Cl attack on the benzene ring.
(2) The resulting radical further reacts with oxygen, yielding secondary products and chlorophenols, or can react with Cl_2, yielding chlorobenzene. Chlorobenzene might also be formed by the recombination of phenyl and Cl radicals.

Can chlorinated hydrocarbon formation be controlled by controlling the chlorine atom concentration?

Support for the above role of chlorine atoms has been obtained by injecting ethylene upstream of the benzene addition. If ethylene is injected at 1250°C, it burns and generates OH which augments the Cl atom concentration through the reaction $OH + HCl = Cl + H_2O$. In this case the production of chlorobenzene and chlorophenols was augmented by a factor equal to the increase in Cl atom concentration. If the ethylene is injected in the lower temperature regime, again upstream of the benzene injection, it will react with and consume the chlorine radicals. In this case, the chlorobenzene and chlorophenol consumption is suppressed.

What determines polychlorination?

Chlorine atoms in the lower temperature regime are the source of chlorination. As long as the concentration of the hydrocarbon injected is greater than that of the chlorine atom, monochlorinated hydrocarbons will result. Polychlorinated products are produced once the chlorine atom concentration exceeds that of the hydrocarbon.

Heterogeneous Formation

The mechanism of carbon chlorination is a significant question relating to an understanding of the mechanism of chlorinated organic formation.

Is a fuel or waste C-Cl bond required for subsequent formation?

With an understanding that PCDD/F emissions are due to formation processes in the combustor and not simply the result of incomplete combustion of a PCDD/F-containing feed stock, the next likely question concerns whether organic Cl is necessary for formation. This is a contentious issue, particularly regarding the role of specific forms of chlorinated organics, mainly PVC.

Numerous studies (eg, Wikström *et al.*[14]) have shown that inorganic chloride sources are comparatively equivalent to organic chloride sources for formation of PCDD/F. This suggests that pre-existing C-Cl bonds are not necessary for formation; chlorination readily occurs in the combustion process. This apparent equivalency may be partly understood by studying the role of inorganic chlorides in producing HCl in combustors. Experiments by Uchida *et al.*[15] showed that high temperature (>700°C) production of HCl from alkaline and alkaline earth chlorides occurs and is promoted by the presence of alumina (Al_2O_3) and silicon dioxide (SiO_2), common compounds in fly ash. If the presence of HCl then (at least) partially determines the extent of formation of PCDD/F, then even inorganic chlorides can lead to PCDD/F emissions.

Can gas-phase chlorination occur at lower temperatures?

Direct, quantitative chlorination of phenol by gas-phase Cl_2 has been demonstrated[16,17,18] with a maximum at 400°C; significantly less chlorination was observed using HCl as the Cl source (at a maximum of 600°C). As temperatures cool, equilibrium predicts the dominant Cl species to be Cl_2. However, the rate of Cl_2 formation is extremely slow such that most Cl is in the form of HCl as it leaves the stack. It has been hypothesized[19] that some Cl_2 forms as the result of a surface-catalysed reaction (the 'Deacon' reaction) between HCl and O_2:

$$2HCl + \tfrac{1}{2}\,O_2 \leftrightarrow H_2O + Cl_2$$

This first-order reaction occurs[16] with high yields, and the Cu catalyst can be reactivated by addition of HCl to the system. More rapid cooling minimizes this reaction[20] by slowing the recombination of Cl to Cl_2. This reaction is catalysed by metals, such as CuCl, of various oxidation states. Gullett *et al.*[17] analysed Cu_2O, CuO, and Cu and found nearly equivalent Deacon activity at a peak temperature equivalent to that of maximum reactivity of Cl_2 with phenol (400°C). No Deacon activity (Cl_2 production) was noted for Fe, Fe_2O_3, and $FeCl_2.4H_2O$. Fly ash testing indicated that 1.3% of HCl was converted to Cl_2.

Are surfaces necessary for C-Cl bond formation? Does chlorination occur as a result of gas-solid or solid-solid reactions?

Taylor *et al.*[21] propose that indirect chlorination of carbon via a transfer from surface Cu occurs as opposed to direct chlorination by gaseous precursors of Cl· or Cl_2. This work, and that of Froese and Hutzinger[22], showed that chlorination of acetylene (C_2H_2), a known precursor of aromatics and soot, occurs in the presence of surfaces and a chlorine source. Taylor *et al.*[21] proposed a surface transfer of Cl from $CuCl_2$ to C_2H_2 to form a chlorovinylic intermediate, with the presence of these surfaces reducing the formation temperature from 700 to 150°C. Formation of this intermediate is the rate limiting step (30.5 kcal mole^{-1}), and it is more energetically favourable than the Deacon process (48.7 kcal mole^{-1}). The authors report this reaction at as low as 150°C with $CuCl_2$ (although they show significant yields only at temperatures $\approx$ 300°C), whereas the Deacon process with CuO occurs at temperatures in excess of 300°C. Subsequent Cl transfers result in dichloroethylene or chloroacetylene (Taylor *et al.*[21]). Repetition

of these steps results in production of multiple chlorinated ethylenes or acetylenes. A lack of unchlorinated molecular growth products indicates that chlorination occurred prior to C growth reactions. Regeneration of the $CuCl_2$ is possible with HCl supply; hence, the metal serves as a stoichiometric oxidant and not a catalyst. The role of CuCl species in PCDD/F formation via Cl transfer is evidenced by variation in the colour of the Cu, reflecting changes in the Cu oxidation states with temperature. This is a plausible pathway, although it does not explain evidence for direct gas-phase chlorination of phenolic precursors (via electrophilic aromatic substitution reactions) nor did the tests account for possible decomposition of the copper chloride into Cl and/or Cl_2 gas (as shown in Gullett *et al.*[16,17]).

Considerable work (see Chaltykyan[23]), mostly in the field of aqueous catalyst reactions, supports the catalytic role of Cu-Cl species in formation of aromatic aldehydes; aryl halides; hydrolysis of chlorobenzene to form phenol and allyl chlorides to form alcohols; transformation of allenes to form halogenated dienes; dimerization, polymerization, and hydrochlorination of acetylene; and dechlorination reactions. Cu readily forms complexes with electron-donating ligands. When multiple ligands are present, equilibrium is determined by the polarizability of the competing ligands. Thus, copper-catalysed reactions are made up of complicated successive reactions consisting of acts of complex formation and break-down into the original components, activation of ligand molecules in the complex, and conversion of ligand molecule in molecules of the final product. This supports the proposed role of copper catalysts as 'transfer' agents for Cl (as indicated by Taylor *et al.*[21]).

Solution chemistry has shown that acetylene in concentrated aqueous solutions of cuprous chloride is extremely reactive. Acetylene polymerization is also possible using an acidified solution of cuprous chloride and ammonium chloride with acetylene[24]. A number of non-chlorinated products result, including butenyne (C_4H_4)$CH_2=CH\text{-}C\equiv H$ and traces of acteylene hexamer ($C_{12}H_{12}$) and chloroderivates. Nieuwland[24] hypothesized that one or two acetylene molecules displace the ammonium chloride and enter into the cuprous complex. This complex decomposes to form, possibly, $CH_2=C=$ which reacts with an unreacted acetylene to form butenyne.

Gas-phase work has also shown the role of copper catalysts. Freidlin *et al.*[25] showed the gas-phase hydrolysis reaction of chlorobenzene over silica gel to be greatly enhanced in the presence of $CuCl_2$, CuCl, and Cu powder, resulting in the formation of phenol and HCl. Others[26,27] have measured continuous hydrochlorination of acteylene over dry metal chlorides (Pt, Hg, Cu, Fe, and Ba) supported on activated carbon (180–195°C). Processes to improve vinyl chloride preparation from acteylene and HCl also make use of cuprous chloride.

Similarly, hydrochlorination of butenyne is aided by KCl and NH_4Cl, but mostly by $CaCl_2$ to form products such as 4-chloro-1,2-butadiene[28] and chloropropene[29]. In the presence of copper salts, a second HCl will be added[28]. Likewise, dechlorination reactions also occur. The Cu-catalysed dechlorination of tetrachlorophenol was observed when passing H_2 and tetrachlorophenol over cuprous chloride[30].

Does PCDD/F suppression by SO_2 act through an effect on the Cl reaction?

The studied effect of sulphur dioxide (SO_2) on preventing formation of PCDD/F may also offer some indirect clues regarding the mechanism and role of Cl in the formation of chlorinated organics. Much research has shown, in bench[31,32], pilot[33,34] and field tests[35], that the presence of S, likely as SO_2, effects suppression of formation of chlorinated organics. This effect may explain why coal-fired boilers, despite sufficient levels of Cl, Cu, organics and particle surfaces, are not significant PCDD/F emitters[36].

There are several possible hypotheses for mechanistic interference by the S species. It has been proposed[19] and demonstrated at 400°C[32] that an effect of S is to deplete the Cl_2 levels through the gas-phase reaction

$$Cl_2 + SO_2 + H_2O \leftrightarrow 2\,HCl + SO_3$$

perhaps through a sulphuryl chloride (SO_2Cl_2) intermediate. This reaction occurs and may be carbon-catalysed. This reaction would presumably inhibit reaction of Cl_2 into aromatic structures via electrophilic aromatic substitution reactions due to reductions in the amount of available Cl. A preliminary concentration effect has been observed such that S/Cl (not Cl_2) ratios around or above, approximately, unity are necessary to produced decreases in PCDD/F formation[34].

Another hypothesis suggests that the presence of higher concentrations of sulphur during coal combustion shifts the phase distribution of metallic deposits away from the chlorides necessary for PCDD/F formation. Experimental and equilibrium analyses by Chen *et al.*[37] found that the presence of sulphide (added as sodium sulphide, Na_2S) inhibited the formation of the more volatile metallic chlorides, instead resulting in more metallic oxides than chlorides. This may limit the activity of the metal catalysts for PCDD/F formation through reductions in the available metal chloride concentrations. Likewise, equilibrium calculations by Verhulst *et al.*[38] similarly show that the presence of S stabilizes many metals in sulphate phases below 800°C. These metal sulphates can displace chlorides; for Cu, the formation of copper sulphate ($CuSO_4$) completely dominates the formation of copper chloride (CuCl) at temperatures below 500°C. In the absence of S, gaseous Cu_xCl_x species still exist between 300 and 500°C. Increasing Cl levels result in increased Cu volatilization.

Miller and Krause[39] also found that chloride salts on wall deposits react with SO_2 and oxygen near the boiler tubes to evolve high concentrations of HCl directly adjacent to the metal. These observations are consistent with work by Uchida *et al.*[15] who found that the presence of SO_2 resulted in the overall reaction ('Hargreave reaction'):

$$2NaCl + SO_2 + \tfrac{1}{2}\,O_2 + H_2O \Leftrightarrow Na_2SO_4 + 2\,HCl$$

producing HCl(g). The equilibrium constants for these types of reactions increase with decreasing temperatures. An analogous reaction with CuCl or $CuCl_2$ may be responsible, at least partly, for the reduced formation of PCDD/F observed in combustion systems.

CONCLUSIONS

Major progress has been made in our understanding of the homogeneous and heterogeneous pathways to the polychlorinated hydrocarbons, including PCDD/F. This understanding shows that total chlorine is an imperfect measure of PCDD/F formation potential. For the homogeneous pathway, the concentration of chlorine atoms is the major factor, and its concentration can be augmented by reactions contributing to OH formation or suppressed by hydrocarbon injection of reactants such as hydrogen. The heterogeneous reactions depend on ash surface species and type, ash adsorptive characteristics, temperature, and the presence of regenerative Cl. Knowledge of the chemistry not only explains the imperfect correlation of total chlorine content and PCDD/F formation but can also lead to the development of control strategies which are targeted at suppressing the active chlorine species, either atomic chlorine for the homogeneous pathway or active surface ligands for the heterogeneous pathway.

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ADDRESS

Correspondence concerning this paper should be addressed to Dr B. K. Gullett, US Environmental Protection Agency, National Risk Management Research Laboratory, Air Pollution Technology Branch (MD-65), Research Triangle Park, NC 27711, USA. E-mail: gullett.brian@epa.gov

This paper was presented at the 2nd International Symposium on Incineration and Flue Gas Treatment Technologies, organized by IChemE and held at the University of Sheffield, UK, 4–6 July 1999.

The South Karelia Air Pollution Study: Acute Health Effects of Malodorous Sulfur Air Pollutants Released by a Pulp Mill

Tari Haahtela, MD, Olli Marttila, MSc, Vesa Vilkka, MD, Paavo Jäppinen, MD, and Jouni J. K. Jaakkola, MD

ABSTRACT

We evaluated the acute health effects of a strong emission of malodorous sulfur compounds released from a pulp mill in South Karelia, Finland. The 24-hour ambient air concentrations of hydrogen sulfide for the two emission days were 35 and 43 $\mu g/m^3$ (maximum 4-hour 135 $\mu g/m^3$). A questionnaire was distributed after the high exposure and later after a low exposure period to 29 households with 75 subjects living in the nearby community. During the high exposure, 63% of the respondents reported experience of at least one symptom compared to 26% during the reference period. Every third participant reported difficulties in breathing. In the 45 subjects responding to both questionnaires more eye, respiratory and neuropsychological symptoms occurred during the exposure compared to the reference period. The strong malodorous emission from a pulp mill caused an alarming amount of adverse effects in the exposed population. (*Am J Public Health.* 1992;82:603–605)

Introduction

Large populations in northern Europe and North America are often or constantly exposed to malodorous sulfur air pollutants such as hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), and methyl sulfides produced by the paper and pulp industry. The knowledge of the adverse effects of these pollutants is sparse, although people living in the neighborhood affected commonly complain of discomfort and express concern about the health effects.

A cluster of paper mills producing wood pulp is located in South Karelia, in southeastern Finland. The two sulfate pulp mills, located in the small city of Imatra (33 000 inhabitants), were, in 1989, estimated to emit annually 2800 tons of H_2S into the air. The annual mean (2 $\mu g/m^3$) concentration of sulfur dioxide (SO_2) is low because of the use of natural gas as the source of industrial energy.

During 2 days in September 1987, a strong emission, with H_2S as the major component and mesityloxide as a minor component, was released from one of the two pulp mills, exposing the local population to intensive catty odor for the 2 days. We evaluated the acute health effects of the emission by comparing the occurrence of symptoms during the high-exposure period and 4 months later during a low-exposure period.

Material and Methods

During the introduction of a new processing technique, a strong malodorous emission was released from the pulp mill using the sulfate method for production, and increased concentrations of H_2S were measured for 2 days at our monitoring station located in a nearby community about 1 km southeast of the source of exposure. The H_2S exposure was measured by absorbing the air sample to cadmium hydroxide.[1] The 4-hour concentrations of H_2S during the high exposure were, on the average, 4 to 5 times higher, and a maximum of 20 times higher, than the levels before and after the peak emission, during which there was inversion of the air and no wind (Figure 1). The highest 4-hour concentration of H_2S, 135 $\mu g/m^3$, was measured at 2 AM. The 24-hour averages for the 2 days were 35 and 43 $\mu g/m^3$.

During the peak emission of malodorous sulfur compounds, the mean 1-hour concentration of SO_2 was only 3 $\mu g/m^3$. It was not possible to measure mesityloxide in the ambient air. Mesityloxide is produced inside pulp mills under the same kinds of conditions that occurred during the peak emission. The presence of mesityloxide was recognized in the air by its characteristic catty odor.

A reference period of 2 days was chosen about 4 months later, in January 1988. During the 2 reference days no odor was sensed, and the concentration of H_2S was low (0.1 to 3.5 $\mu g/m^3$ for 4 hours). The SO_2 concentration was low, 3 $\mu g/m^3$, as during the high-exposure period.

An open-ended questionnaire was distributed 10 days after the high exposure to all 29 households of the nearby community; 66 (88%) of a possible 75 subjects responded. Six subjects were excluded because of missing information, leaving 60 subjects: 44 adults (15 men and 29 women; mean age = 45 years and range = 17 to

Tari Haahtela is with the Department of Allergic Diseases, Helsinki University Central Hospital, Helsinki, Finland. Olli Marttila is with the South Karelia Allergy and Environment Institute, Joutseno, Finland. Vesa Vilkka is with the Department of Pulmonary Diseases, Tiuru Hospital, Joutseno, Finland. Paavo Jäppinen is with the Vuoksenlaakson Occupational Health Centre, Imatra, Finland. Jouni J. K. Jaakola is with the Department of Public Health, University of Helsinki, Helsinki, Finland.

Requests for reprints should be sent to Tari Haahtela, MD, Department of Allergic Diseases, Helsinki University Central Hospital, Meilahdentie 2, 00250 Helsinki, Finland.

This paper was submitted to the Journal November 26, 1990, and accepted with revisions July 9, 1991.

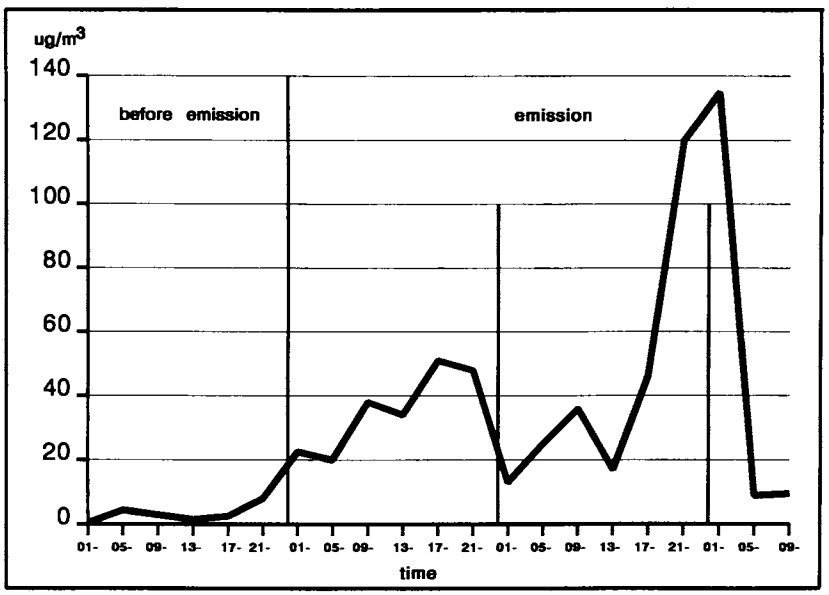

FIGURE 1—Four-hour concentrations of hydrogen sulfide before and during the emission from the first day at 1 AM to the fourth day at 9 AM at the monitoring station located about 1 km from the pulp mill.

TABLE 1—Prevalence of Symptoms in Subjects Who Responded after the High-Exposure Period (n = 60) and after the Reference Period (n = 66)

	High-Exposure Period		Reference Period	
	n	%	n	%
Eye symptoms	13	22	1	2***
Males	5	21	0	0
Females	8	22	1	3
Nasal symptoms	5	8	13	20
Males	2	8	5	16
Females	3	8	8	24
Cough or pharyngeal irritation	9	15	3	5
Males	5	21	1	3
Females	4	11	2	6
Breathlessness	21	35	1	2***
Males	7	29	0	0
Females	14	39	1	3
Nausea	14	23	3	5**
Males	8	33	1	3
Females	6	17	2	6
Headache	14	23	5	8*
Males	5	21	1	3
Females	9	25	4	12
Mental symptoms[a]	6	10	0	0*
Males	1	4	0	0
Females	5	14	0	0

Note. Differences between prevalences were obtained using a χ^2-test.
[a]Depression, anxiety.
*$p < .05$.
**$p < .01$.
***$p < .001$.

85) and 16 children (9 boys and 7 girls; mean age = 7 years and range = 1 to 15). The respondents were asked to describe the symptoms they had experienced during the days of peak exposure, without mention of the exceptionally high emission of pollutants or what kinds of symptoms the emission may generate. Parents responded on behalf of their children.

An identical questionnaire was distributed to the same households 4 months later. The link of this reference questionnaire to the first inquiry was not revealed. The response rate was 79%, no data had to be excluded, and 66 persons were included in the analyses. The effect of confounding was controlled by studying the intraindividual difference in the symptoms in the subpopulation of 45 subjects who responded to both questionnaires. The statistical significance of the difference in the occurrence of the symptoms during the high-exposure and reference periods was assessed by a χ^2-test and McNemar's test.[2]

Results

The unadjusted prevalences of the different symptoms in the two population samples are given in Table 1. Large proportions of the participants reported breathlessness (35%) and mental symptoms (10%) during the high-exposure period, while no such symptoms were indicated during the reference period. The prevalence of eye symptoms (22% vs 2%), cough or pharyngeal irritation (15% vs 5%), and nausea (14% vs 3%) were also significantly larger during the high exposure. However, nasal symptoms (8% vs 20%) were more common during the reference period than the high-exposure period, although the difference was not significant. Altogether, 63% of the subjects reported experience of at least one symptom during the high-exposure period compared with 26% during the reference period.

The presence of symptoms during the exposure and reference periods in the 45 subjects who responded to both questionnaires is shown in Table 2. Eye symptoms, cough or pharyngeal irritation, breathlessness, nausea, and headache were experienced more often during the exposure period than during the reference period. The difference was significant for breathlessness.

Discussion

The occurrence of the symptoms during the high-exposure period was large: Every third participant reported difficulties in breathing, and every fourth participant indicated irritation of eyes, headache, and nausea. Mental symptoms were also common. The prevalence of these symptoms was significantly larger than those during the reference period in the comparison of the two samples. The direction of the differences was similar in the intraindividual comparison of those who responded to both questionnaires, al-

though significance was not reached in all of the symptoms because of the smaller number of subjects than in the two-sample comparison. Interestingly, more subjects reported nasal symptoms during the reference period than during the high-exposure period. A possible explanation is that breathing difficulties due to exposure were so prominent that attention was not paid to mild nasal symptoms. The highest 4-hour concentration of H_2S was 135 $\mu g/m^3$, which is nine times higher than the guideline in Canada (15 $\mu g/m^3$). There are no guidelines for ambient air H_2S in Finland.

The observed symptoms correspond to physiological effects of acute exposure of H_2S suggesting direct irritative effect on mucous membranes and eye conjunctivae, although in far smaller concentrations than described earlier.[3] Previously, we have observed an increased amount of eye and nasal symptoms and cough among subjects living in the most polluted area of Imatra.[4]

According to the World Health Organization (WHO) guideline values for H_2S, the highest acceptable half-hour concentration is 7 $\mu g/m^3$ for odor nuisance, along with a daily mean of 150 $\mu g/m^3$ for health hazards.[5] During the period of high exposure, the highest measured 4-hour concentration (135 $\mu g/m^3$) and 24-hour averages (35 and 42 $\mu g/m^3$) were well below the recommendation. These kind of emissions are rather commonplace and are always possible wherever wood pulp production takes place. It seems likely that the WHO guideline value of 150 $\mu g/m^3$ for H_2S is too high and does not provide prevention from adverse health effects. Furthermore, the presence of other pollutants should be taken into account, because H_2S is rarely the only hazardous component of air pollution. □

TABLE 2—Occurrence of Symptoms during the High-Exposure and Reference Periods in the 45 Subjects Who Responded to Both Questionnaires

	Exposure Period Only, No.	Reference Period Only, No.	Both Periods, No.	No Symptoms, No.	Significance[a]	
					t	P
Eye symptoms	5	1	0	39	1.63	NS
Nasal symptoms	3	11	1	30	2.14	<.05
Cough or pharyngeal irritation	5	1	0	39	1.63	NS
Breathlessness	16	0	1	28	4.00	<.001
Nausea	6	2	1	36	1.41	NS
Headache	8	2	2	33	1.90	NS
Mental symptoms[b]	2	0	0	43	1.41	NS

Note. NS = not significant.
[a]McNemar's test of significance.
[b]Depression, anxiety.

Acknowledgments

This study was supported by the Academy of Finland and the Ministry of the Environment.

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Science of the Total Environment 468–469 (2014) 950–957

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Signs and symptoms of methylmercury contamination in a First Nations community in Northwestern Ontario, Canada

Shigeru Takaoka [a,*], Tadashi Fujino [a], Nobuyuki Hotta [b], Keishi Ueda [c], Masanobu Hanada [d], Masami Tajiri [d], Yukari Inoue [d]

[a] Minamata Kyoritsu Hospital, Sakurai-cho 2-2-12, Minamata 867-0045, Japan
[b] Sakura-ga-oka Hospital, Ikeda 3-44-1, Kumamoto 860-0082, Japan
[c] Kumamoto Seimei Hospital, Toroku 5-1-37, Kumamoto 862-0970, Japan
[d] The Open Research Center for Minamata Studies, Kumamoto Gakuen University, Ohe 2-5-1, Chuoh-ku, Kumamoto 862-8680, Japan

HIGHLIGHTS

- Similarities between the prevalence of complaints in Minamata and Grassy Narrows
- Similarities in neurological findings were also found.
- Quantitative sensory measurements gave similar results for impairments.
- Younger Canadians were less severely affected than older ones.
- Results suggest that subjects from Grassy Narrows had methylmercury poisoning.

ARTICLE INFO

Article history:
Received 26 June 2013
Received in revised form 5 September 2013
Accepted 5 September 2013
Available online 1 October 2013

Editor: Frank Riget

Keywords:
Methylmercury
Complaints
Neurological findings
Sensory disturbance
Quantitative somatosensory measurements

ABSTRACT

In 1970, fish caught in the English–Wabigoon River system in northwestern Ontario, Canada, were found to be contaminated with mercury coming from a chlor-alkali plant in the province. In the 1970s, patients exhibiting some of the symptoms of the Hunter–Russell syndrome (e.g. paresthesias, visual field constriction, ataxia, impaired hearing, and speech impairment) were reported by some researchers. However attempts to diagnose the patients as suffering from methylmercury poisoning proved to be controversial. In order to research the presence of methylmercury contamination, and show that the patients, through eating contaminated fish, were suffering from methylmercury poisoning, we studied the results of subjective complaints, neurological findings, and quantitative somatosensory measurements gathered in Grassy Narrows Indian Reservation, Ontario, in March, 2010. At that time, the population of the Grassy Narrows settlement was around 900. Ninety-one residents volunteered to be examined. From them, we selected 80 people who were older than 15 years old, and divided them into two groups. Canadian Younger (CY): 36 residents who were from 16 to 45 years old. Canadian Older (CO): 44 residents who were from 46 to 76 years old. We compared them to Japanese Exposed (JE): 88 methylmercury exposed residents from the Minamata district in Japan, and Japanese Control (JC): 164 control residents from non-polluted areas in Japan. Complaints and abnormal neurological findings were more prevalent and quantitative sensory measurements were worse in the two Canadian groups and the Japanese Exposed group than in the Japanese Control group. Complaints, neurological findings and quantitative sensory measurements were similar in Canadian Older and Japanese Exposed. The results for Canadian Younger fell between those of Canadian Older and Japanese Control. These findings indicate that the clinical signs and symptoms of the residents of Grassy Narrows are almost the same as those recorded for Minamata disease in Japan.

1. Introduction

Since the 1960s, a chlor-alkali plant had been releasing waste products contaminated with mercury into the waters of the English–Wabigoon River system in northwestern Ontario, Canada. In the first examples of fish, from the river system, that were contaminated with methylmercury (Bligh, 1970) levels of up to 16 μg/g were reported. Fimreite and Reynolds (1973) found highly contaminated fish with a maximum

* Corresponding author. Tel.: +81 966 63 1704; fax: +81 966 62 2044.
E-mail addresses: stakaoka@x.email.ne.jp (S. Takaoka), tds-fujino@se.kcn-tv.ne.jp (T. Fujino), hanada@kumagaku.ac.jp (M. Hanada), mtajiri@kumagaku.ac.jp (M. Tajiri), iyukari@kumagaku.ac.jp (Y. Inoue).

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http://dx.doi.org/10.1016/j.scitotenv.2013.09.015

methylmercury concentration of 27.8 μg/g in 1970. The contamination in fish there was almost equal to that measured in fish from Minamata Bay, Japan, where a barracuda was recorded as having 23 μg/g in May 1961 (Fujiki and Irukayama, 1979). The average mercury concentrations, measured in 1971, in walleye and northern pike from Ball Lake, English–Wabigoon River, were 1.99 μg/g and 5.05 μg/g respectively (Armstrong and Scott, 1979).

In 2003, results of similar measurements were 0.40 μg/g in walleye and 0.85 μg/g in northern pike (Kinghorn et al., 2007). The concentrations of mercury in fish are declining, but they are still higher than those found in fish in Minamata Bay, Japan, where the average concentration of mercury in fish has been from 0.17 to 0.42 μg/g during the period of 1998-2004. (Kindaichi and Matsuyama, 2005). Residents living in that area of Japan, still continue to be exposed to low levels of mercury from the eating of locally caught fish.

The two First Nations' communities living in Grassy Narrows and Whitedog Indian Reservations became victims of methylmercury poisoning through the consumption of locally caught fish. Patients with some of the Hunter–Russell syndrome symptoms have been reported in these reserves (Harada et al., 1976). Canadian researchers also examined the indigenous residents of the areas. Wheatley et al. (1979) reported that effects associated with methylmercury poisoning were observed, but they concluded that direct impacts on human health were difficult to prove.

Pathological studies of a cat from Whitedog, which exhibited acute neurological symptoms, revealed high mercury levels in the brain similar to those of Minamata disease in Japan (Takeuchi et al., 1977). Another cat from neighboring Grassy Narrows showed no symptoms, but on closer examination it was shown to have high mercury levels in the brain and latent defects caused by methylmercury poisoning (Takeuchi et al., 1977).

Since 2004, more than 60,000 people from the Minamata district have been diagnosed with abnormal neurological symptoms. In many cases, the abnormalities were being reported for the first time since the release of methylmercury contaminated wastewater was stopped in 1968 (Takaoka et al., 2009). In chronic methylmercury poisoning the somatosensory disturbance from cortical damage is specific and sensory disturbance is present either in all four limbs or throughout the body, sometimes with the exception of the face and head. To detect such abnormalities, quantitative sensory measurements are useful (Takaoka et al., 2008). So we carried out a health survey that included the examination of subjective complaints, neurological symptoms, and quantitative sensory measurements in Grassy Narrows, Ontario, Canada, in 2010 and tried to reassess the effect of methylmercury in this area.

2. Materials and methods

2.1. Subjects

The study was carried out in March 2010 in Grassy Narrows, Ontario, Canada. We contacted a Grassy Narrows support group and asked for their help. They informed the indigenous population of our coming survey to detect any health effects from methylmercury poisoning and asked for volunteers to take part in the study. The Chief of Grassy Narrows First Nation went on the local radio to ask for volunteers for our research. Posters, informing of our coming research project and our wish for volunteers, were displayed in the band office and a local store. Of the approximately 900 residents, 91 volunteered to be examined. We selected 80 residents, who were older than 15, for the study. Subjects were informed verbally and in writing about the examination method, how the data would be used and that their confidentiality would be protected. Each participant gave their written consent.

We divided them into two groups. (1) Group Canadian Younger (CY): consisted of 36 residents who were from 16 to 45 years old (M/F = 18/18, 34.4 ± 9.5 years old). (2) Group Canadian Older (CO): consisted of 44 residents who were from 46 to 76 years old (M/F = 19/25, 57.5 ± 8.1 years old).

We studied subjective symptoms, neurological findings and carried out four quantitative sensory measurements (including minimal tactile sensation, vibration, position sense, and two-point discrimination) among the 80 residents. In order to assess the state of health of the residents of Grassy Narrows, we compared them to a group of Japanese residents who had also been exposed to methylmercury as well as a group of Japanese control residents. These residents were the same people we had used as subjects in our previous study (Takaoka et al., 2008).

(3) Group Japanese Exposed (JE): was comprised of 88 randomly selected Japanese subjects. They were chosen from residents of the Minamata area who had been exposed to methylmercury and who had been included in our former study at the Minamata Kyoritsu Hospital and the Kyoritsu Neurology and Rehabilitation Clinic between November 2004 and April 2005. Their ages matched those of the corresponding Canadian group but the genders could not be matched exactly (M/F = 38/50, 59.0 ± 7.5). They answered a detailed questionnaire and were given a neurological examination including the same four quantitative sensory measurements as we performed in Canada. All of them were born before 1969. In our former study, we separated the subjects into those with and without neurological or neurologically related diseases, but we found that there was little difference between those two groups. So in this study, Group JE included subjects both with and without such related diseases.

(4) Group Japanese Control (JC): consisted of 164 residents who lived in other districts, e.g. around Fukuoka City, Kumamoto City, and Kagoshima City, aged between 40 and 79. In the control group, people who had lived around Minamata City or who suffered from a neurological disease or a neurologically related illness were excluded. The control subjects were examined between February and May 2006 (M/F = 67/97, 58.4 ± 11.6).

2.2. Epidemiological conditions and questionnaire on complications

The questionnaire, both in Canada and Japan, included questions to determine the subject's exposure to methylmercury and asked for information regarding place of residence, dietary habits, occupational history, medical complications and the health and medical histories of family members.

The questionnaire on complaints for the Canadian groups (CO and CY) consisted of 47 questions related to sensory impairment (7 items), somatic pain (4), visual impairment (3), hearing impairment (3), tasting and smelling problems (3), in-coordination of the extremities (4), other movement impairment (11), vertigo and dizziness (4), general complaints (2), and mental and intellectual problems (6).

The questionnaire on complaints for the Japanese groups (JE and JC) consisted of about 50 questions. We selected 35 relevant questions from the Japanese questionnaire and used them in the Canadian one. They were as follows: sensory impairment (4 items), somatic pain (3), visual impairment (3), hearing impairment (3), tasting and smelling problems (3), in-coordination of the extremities (5), other movement impairment (4), vertigo and dizziness (4), general complaints (2), and mental and intellectual problems (4).

In answer to questions on health complaints, subjects were asked to select one of 4 possible choices. 1) Yes, always, 2) Yes, sometimes, 3) Yes, in the past but not at present, and 4) No, never. The prevalence of each complaint was calculated for each group and then compared between the four groups. All subjects completed the questionnaire before their medical examination. Subjects who could not complete the questionnaire by themselves were asked the questions verbally. All questionnaires were reviewed prior to the examinations.

2.3. Standard neurological examination

A standard neurological examination was carried out on 32 of the 36 subjects in Group CY and on all 44 subjects in Group CO. Dysarthria, auditory disturbance, visual constriction, finger–nose test with the eyes open and closed, diadochokinesis, heel–shin test, gait disturbances, tandem gait test, Mann's test, balancing on one foot with eyes open and closed, and superficial sensory disturbance (touch and pain) were used and tested for. The examination methods and criteria were almost the same as the former study (Takaoka et al., 2008).

Dysarthria, auditory disturbance, visual constriction, postural hand tremors, gait disturbances and Romberg's sign were judged to be either present or absent. Dysarthria, auditory disturbance, and visual field constriction were judged by the examining physician without using special instruments. Tunnel vision was considered to be present when the confrontation test showed a lateral field of vision of 80° or less.

Limb and truncal ataxia were judged as being either absent, mildly abnormal or moderately to severely abnormal. The results of the finger–nose test and the heel–shin test were judged to be positive not only when there was constant dysmetria or decomposition but also when there was uncertain dysmetria, decomposition, or very slow movement involved. Dysdiadochokinesis was judged to be present from when there was a constant abnormality to when there was an uncertain abnormality or slow movement. Tandem gait disturbance was judged as present not only when the subject could not walk more than five steps but also when they could walk five steps but were unstable. In the balancing on one foot test, disequilibrium was judged as present when it was impossible to balance more than 3 s or when the subject was unstable, but could keep their balance for more than 3 s.

The examinations were carried out in Canada by one neurologist and four psychiatrists. The neurologist and two of the psychiatrists had previous experience in carrying out these tests from earlier examinations of Minamata disease patients. The other two psychiatrists had been briefed and trained to carry out the tests. In Japan, Group JE was examined by six physicians and one neurologist and Group JC was examined by forty-five physicians, several of whom were neurologists.

Because there was not enough data collected from Group JE for the finger–nose test with eyes closed, Mann's test, Romberg's sign or balancing on one foot with eyes closed test, we excluded that information from our calculations.

2.4. Quantitative sensory measurements

Four categories of tests were used in the quantitative measurement of the somatosensory system. All four categories were the same as in the former study (Takaoka et al., 2008).

2.4.1. Assessment of minimal tactile sense

The minimal tactile sense was measured by the Semmes–Weinstein monofilament test. Twenty kinds of filaments from 0.008 to 300 g were used. Subjects were tested with eyes closed after receiving clear instructions on which locations would be tested. Each filament was pushed until it bent about 90° for about a second. The threshold was the smallest size filament which a subject could feel as touch. The trial was performed starting from a smaller size filament and then gradually increasing the size.

Each filament was tested only once except when the subject was unsure. In that case the examiner, using the same filament, repeated the test an odd number of times and selected the dominant response. If a subject could not detect the maximum filament (300 g), we defined the threshold as 400 g for calculation. Testing was carried out on the lower lip, upper chest and ventral sides of both index fingers and big toes.

2.4.2. Vibration sense

Vibration sense was measured by using a 128 Hz tuning fork. The vibration test was carried out on the middle or upper sternum, the radial side of both wrists and the fibular side of both ankles. Subjects were told to indicate when they no longer could feel the vibrations. The examiner simultaneously struck a tuning fork and started a stopwatch, then placed the base of the fork against the area to be tested. The time interval until the subject reacted was recorded.

2.4.3. Position sense

The testing of position sense was carried out on subjects with their eyes closed. A ruler with a millimeter scale was used. Tests were carried out on the subject's index fingers and big toes. A ruler was placed vertically alongside the digit. With the digit held horizontally, the zero point was the position of the digit's nail on the ruler's scale. The examiner held the digit by its sides and moved it either up or down, holding the position for about 1 s. The subject was asked to indicate whether they felt the movement and if it was up or down. The upward or downward movement started from 5 mm from the zero point and was considered to be the minimum value. The movement was increased in 5 mm increments and the subject's threshold distance was recorded.

Each trial was carried out once for each distance except when the subject was uncertain. In that case the examiner would repeat the test again for an odd number of times and choose the dominant response. If the subject could not feel the maximum movement, the threshold was defined as the maximum distance plus 5 mm.

2.4.4. Two-point discrimination

The two-point discrimination test was carried out on subjects while their eyes were closed. A drafting divider, with the legs set at different distances, was pressed against the subject's skin at an angle of 30 to 45° to a depth of between 1 and 2 mm for about 1 s. The distances between the points used in the test were 1, 2, 3, 4, 5, 6, 8, 10, 12, 15, 20, 25, 30 and 36 mm. The threshold recorded was the shortest distance at which a subject answered correctly. This method was performed on the lower lip and the ventral side of each index finger. If the subject was unable to detect the maximum distance of 36 mm, the threshold was defined as 40 mm for calculation purposes.

In testing Groups CO and CY, the “Yes/No” method was used. However, when a subject felt that 1-point stimulation was actually 2-point stimulation, they were re-tested using the “2-alternative, forced-choice method”. In Groups JE and JC the “2-alternative, forced-choice method” was used exclusively.

2.5. Statistical methods

All the calculations were performed using MS Excel and SPSS. Chi square analysis was used in MS Excel when the prevalence was compared, and t-test was used in MS Excel when the average was compared. The correlations were calculated by SPSS.

When we calculated the minimal tactile sense, we converted the grams to the evaluator size using the equation: Evaluator size $=$ $\log([\text{gram}]) + 4$.

3. Results

3.1. The subjects' backgrounds

The age and sex of the subjects are shown in Table 1. The age and sex in Groups CO, JC and JE were not statistically different. The age of Group CY was less than the other groups.

From Table 2, we can see that 86% of Group CO and 83% of Group CY answered the question on how they obtained fish and over 60% of Groups CO and CY answered that they are catching fish by themselves. Also from Table 2, we can see that 41% of Group JE and 1% of Group JC

Table 1
Age and sex of each group.

	Age	n (M/F)
Group CO	57.5 ± 8.1	44 (19/25)
Group CY	34.4 ± 9.5	36 (18/18)
Group JE	59.0 ± 7.5	88 (38/50)
Group JC	58.4 ± 11.6	164 (67/97)

belonged to a fisher family. Ninety-three percent of Group JE and 25% of Group JC ate fish every day.

Table 3 shows the relevant medical history of Group CO and Group CY. The information was collected through interviews. On the question of hypertension, although some subjects had no recorded history of hypertension, high blood pressure was detected during the tests. If we detected a systolic pressure of 170 or more or and diastolic pressure of 110 or more in a subject, we recorded that hypertension was present. Some neurological or neurologically related diseases were found in 47 subjects (53%) of Group JE. No neurological or neurologically related diseases were found in Group JC.

3.2. Questionnaire on medical complaints

Medical complaints reported from each group are shown in Table 4, and the p-values by chi square among the four groups are shown in Table 5. In general, the prevalence in both "Yes, always" and "Yes, always" + "Yes, sometimes" in Groups JE, CO, and CY was statistically higher than Group JC. In general, the prevalence in Group JE and Group CO was similar. For most of the complaints Group CY showed a lower prevalence than Group CO.

The questionnaire consisted of questions related to specific and non-specific symptoms in the methylmercury polluted area. Specific symptoms included numbness, difficulty in speaking, fine-finger tasks, buttoning, losing slippers while walking, difficulty in differentiating tastes, detecting smells etc. Non-specific symptoms included headaches, forgetfulness, general fatigue, and so on. In specific symptoms, the prevalence of the answer "Yes, always" + "Yes, sometimes" was lower in Table 4. A tendency to a similar prevalence was observed both in specific and non-specific symptoms.

The correlation efficient among four groups was shown in Table 7. In "Yes, always" answers, the correlation between Groups JE and CO was highest, and was followed by the one between Groups CO and CY. In "Yes, always" + "Yes, sometimes" answers the correlation between Groups CO and CY was highest, and was followed by the one between Groups JE and CO and between Groups JE and CY.

Table 2
Fish sources and frequency of ingestion.

In Canada	Group CO	Group CY
Self-caught fish	28/44 (64%)	24/36 (67%)
Fish caught by relatives and friends, purchased etc.	30/44 (68%)	22/36 (61%)
A combination of both the above	38/44 (86%)	30/36 (83%)
In Japan	**Group JE**	**Group JC**
Fishermen	10/88 (11%)	0/164 (0%)
Fishermen's families	36/88 (41%)	1/164 (1%)
Sport fishing/recreational fishing	62/88 (70%)	17/161 (11%)[b]
Daily ingestion	81/87 (93%)[a]	40/157 (25%)[c]
Ingestion more than once a week	85/87 (98%)[a]	119/157 (76%)[c]

[a] Datum for 1 person was missing.
[b] Data for 3 persons were missing.
[c] Data for 7 persons were missing.

Table 3
Complications in Canadian subjects.

	Group CO	Group CY
Hypertension	15 (34%)	16 (44%)
Diabetes mellitus	8 (18%)	3 (8%)
Stroke	3 (7%)	1 (3%)
Lung diseases	0 (0%)	1 (3%)
Stomach diseases	6 (14%)	1 (3%)
Heart diseases	1 (2%)	0 (0%)
Orthopedic diseases	7 (16%)	4 (11%)
Psychiatric diseases	3 (7%)	1 (3%)

3.3. Neurological examination

The neurological findings for each group and p-values by chi-square among the four groups are shown in Table 6. The prevalence was generally highest in Group JE, and the prevalence of all the findings of Groups JE and CO was statistically higher than Group JC. All the findings of Group CY were less prevalent than Group CO, and seven of twelve findings of Group CO showed statistically higher prevalence than Group CY.

The most prevalent findings in Groups CO are tandem gait abnormality and somatosensory disturbances of pain and touch, followed by hearing impairment, truncal ataxia other than tandem gait abnormality, upper extremity ataxia (finger–nose test and diadochokinesis), and lower extremity ataxia (heel–shin test). The prevalence of visual constriction or dysarthria was lower. Although the orders of prevalence for the positive findings were different, the tendencies of the abnormalities were similar between Groups CO and JE.

Correlation among four groups was shown in Table 7. Correlations between Groups JE and CY, Groups CO and CY, and Groups JE and CO were higher than those between Group JC and other three groups, and p-values were lower.

3.4. Quantitative sensory measurements

3.4.1. Minimal tactile sensitivity using the Semmes–Weinstein monofilament test

Thirty-three subjects (75%) of Group CO and 24 subjects (67%) of Group CY were examined. One set of data for the lower lip in Group CO and one set of data for the left and right big toes in Group CY were absent. 84 of 88 subjects (95%) in Group JE (in which three sets of data were missing for the lower lip) and all 164 subjects of Group JC were examined and calculated.

Fig. 1 shows the results of the minimal tactile sensitivity using the Semmes–Weinstein monofilament test. The results were worst in Group CO, and there were distinct statistical differences between Groups CO, CY, and JC. Group JE was, in general, positioned between Groups CO and CY.

3.4.2. Vibration sense

Thirty-five subjects from Group CO and 25 subjects from Group CY were examined. Two subjects from Group CO and one subject from Group CY were unable to carry out the test correctly and were excluded from the results. Consequently, the results of 33 subjects (75%) from Group CO and 24 subjects (67%) from Group CY were used in the calculation. Three sets of data for the right ankle and 4 sets of data for the left ankle were absent for Group CO. All 88 subjects of Group JE and all 164 subjects of Group JC (in which one set of data for the chest and one set of data for the left and right ankles were absent) were examined and calculated.

Fig. 2 shows that the vibration sense was worst in Group JE, followed by Group CO, Group CY and Group JC. Most of them had statistical differences between each other.

Table 4
Percentage of complaints.

		Yes, always				Yes, always + Yes, sometimes			
		Group CO	Group CY	Group JE	Group JC	Group CO	Group CY	Group JE	Group JC
1	Numbness in hands	40%	11%	49%	2%	86%	75%	92%	6%
2	Numbness in legs	39%	8%	42%	1%	91%	61%	86%	6%
3	Cannot judge bath water temperature	26%	8%	15%	0%	47%	31%	35%	0%
4	No pain when burned or wounded	23%	17%	16%	0%	65%	49%	44%	0%
5	Hand weakness	34%	22%	56%	3%	78%	69%	81%	5%
6	Leg weakness	35%	11%	49%	2%	79%	54%	77%	4%
7	Tremor in hand	23%	19%	24%	2%	74%	67%	68%	4%
8	Difficulty in speaking	2%	6%	8%	0%	51%	53%	62%	2%
9	Difficulty in fine finger tasks	47%	14%	57%	0%	77%	72%	86%	7%
10	Dropping things	9%	8%	16%	0%	77%	47%	73%	6%
11	Difficulty in buttoning clothes	21%	6%	25%	0%	49%	28%	57%	0%
12	Stumbling	29%	17%	5%	0%	79%	58%	65%	1%
13	Losing slippers while walking	8%	0%	23%	0%	50%	27%	70%	1%
14	Disturbed vision	33%	19%	49%	3%	93%	75%	87%	15%
15	Difficulty in finding things in shops	13%	9%	29%	0%	63%	46%	72%	6%
16	Limited peripheral vision	32%	14%	25%	1%	59%	31%	64%	4%
17	Difficulty in hearing	42%	25%	35%	7%	72%	61%	71%	15%
18	Tinnitus	30%	19%	34%	6%	86%	81%	80%	17%
19	Can hear, but cannot understand	5%	3%	7%	1%	65%	64%	45%	6%
20	Difficulty in differentiating tastes	28%	6%	18%	0%	53%	28%	46%	1%
21	Difficulty in sampling food while cooking	26%	0%	15%	1%	48%	23%	47%	1%
22	Difficulty in detecting smells	28%	11%	22%	0%	49%	28%	48%	4%
23	Muscle cramps	33%	17%	31%	3%	98%	91%	91%	29%
24	Headaches	37%	25%	37%	1%	88%	81%	86%	30%
25	Shoulder stiffness	24%	6%	64%	10%	95%	53%	95%	51%
26	Forgetfulness	21%	17%	34%	1%	81%	72%	97%	52%
27	Inability to hold concentration at work	26%	3%	28%	0%	74%	58%	67%	11%
28	Lack of motivation to do things	16%	0%	26%	1%	58%	56%	86%	21%
29	Irritation	21%	22%	31%	0%	72%	78%	85%	33%
30	Difficulty in sleeping	37%	39%	39%	3%	74%	69%	84%	19%
31	Dizziness when standing up	23%	14%	15%	0%	86%	83%	85%	17%
32	Feeling of head spinning	0%	3%	3%	1%	49%	58%	63%	5%
33	Swaying dizziness	5%	3%	5%	1%	72%	72%	57%	6%
34	Dizziness, bordering on fainting	7%	0%	5%	1%	47%	22%	46%	2%
35	General fatigue	40%	31%	45%	2%	95%	78%	87%	25%

3.4.3. Position sense

Twenty-eight subjects from Group CO and 24 subjects from Group CY were examined. One subject from Group CO and one subject from Group CY were unable to carry out the test correctly and were excluded from the results. Consequently, the results of 27 subjects (61%) from Group CO and 23 subjects (64%) from Group CY were used in the calculation. One set of data for the left and the right big toe, both upward and downward movements, was absent for Group CO. Eighty-seven subjects from Group JE (in which one set of data for both big toes in both directions was absent) and all 164 subjects of Group JC were examined and calculated.

Fig. 3 shows that the position sense was worst in Group JE, followed by Group CO, Group CY, and Group JC. Although there were some exceptions, most of the groups had statistical differences.

3.4.4. Two-point discrimination

Thirty-four subjects from Group CO and 25 subjects from Group CY were examined. Two subjects from Group CO and one subject from Group CY were unable to carry out the test correctly and were excluded from the results. Consequently, the results of 32 subjects (73%) from Group CO and 24 subjects (67%) from Group CY were used in the calculation. 87 of 88 subjects (99%) from Group JE and all 164 subjects of Group JC (in which one set of data for the right index finger was absent) were examined and calculated.

Fig. 4 shows that the position sense was worst in Group JE or Group CO, followed by Group CY and finally Group JC. Most of them had statistical differences, except for the results for index fingers when comparing Group JE with Group CO.

Table 5
p-Value for complaints — comparing groups.

		$p < 0.01$	$p < 0.05$	n.s.
"Yes, always"	CO, JC	All others	Nos. 25, 34	Nos. 8, 19, 32, 33
	CY, JC	All others	Nos. 1, 2, 6, 8, 11, 18, 20	Nos. 13, 19, 21, 25, 27, 28, 32, 33, 34
	JE, JC	All others	Nos. 12, 19	Nos. 32, 33, 34
	CO, CY	Nos. 1, 2, 9, 21	Nos. 6, 20, 27, 28	All others
	JE, CO	Nos. 12, 25	No. 5	All others
	JE, CY	Nos. 1, 2, 5, 6, 9, 13, 14, 25, 27, 28	Nos. 11, 15, 21	All others
"Yes, always" + "Yes, sometimes"	CO, JC	All	–	–
	CY, JC	All others	No. 26	No.25
	JE, JC	All	–	–
	CO, CY	Nos. 2, 25	Nos. 6, 10, 16, 20, 21, 34, 35	All others
	JE, CO	No. 28	Nos. 4, 19, 26	All others
	JE, CY	Nos. 2, 11, 13, 16, 25, 26, 28	Nos. 1, 6, 10, 15, 21, 34	All others

Table 6
Neurological findings and group comparisons.

Examination	Prevalence of abnormality				p-Value					
	Group CO	Group CY	Group JE	Group JC	CO, JC	CY, JC	JE, JC	CO, CY	JE, CO	JE, CY
Hearing impairment	49%	17%	32%	6%	<0.01	n.s.	<0.01	<0.05	n.s.	n.s.
Visual constriction	14%	0%	28%	0%	<0.01	n.s.	<0.01	n.s.	n.s.	<0.01
Dysarthria	17%	3%	18%	1%	<0.01	n.s.	<0.01	n.s.	n.s.	n.s.
Postural tremor	20%	19%	28%	3%	<0.01	<0.01	<0.01	n.s.	n.s.	n.s.
Normal gait—unstable	28%	6%	30%	5%	<0.01	n.s.	<0.01	<0.05	n.s.	<0.05
Tandem gait—unstable	87%	34%	68%	7%	<0.01	<0.01	<0.01	<0.01	<0.05	<0.01
Finger–nose test (eyes open)	21%	0%	47%	0%	<0.01	n.s.	<0.01	<0.05	<0.01	<0.01
Adiadokokinesis	23%	3%	43%	1%	<0.01	n.s.	<0.01	<0.05	n.s.	<0.01
Balancing on one foot (eyes open)	53%	22%	63%	8%	<0.01	<0.05	<0.01	<0.05	n.s.	<0.01
Heel–knee test	24%	0%	50%	1%	<0.01	n.s.	<0.01	<0.05	<0.05	<0.01
Touch disturbance (four limbs)	50%	44%	83%	1%	<0.01	<0.01	<0.01	n.s.	<0.01	<0.01
Pain disturbance (four limbs)	59%	50%	97%	1%	<0.01	<0.01	<0.01	n.s.	<0.01	<0.01

4. Discussion

Our results show that subjective complaints were most prevalent in Group JE, followed by Groups CO, CY and JC. Similar and high prevalence of more specific complaints from methylmercury poisoning between Groups JE and CO suggest that these similarities are mainly related to methylmercury poisoning. The increase of non-specific complaints, which was found in Group JE, was also observed in Group CO.

The high prevalence of somatosensory disturbance and following ataxic movement in Groups JE and CO suggests that those two groups are similarly affected by methylmercury and Group CY is affected to a smaller extent by methylmercury. Harada et al. (1976) conducted health surveys on 89 indigenous inhabitants of Grassy Narrows and Whitedog and found sensory disturbance (47.6%), abnormality in visual field (18.0%), impaired hearing (44.9%), ataxia (9.0%), and dysarthria (5.6%). These findings together with the Hunter–Russell syndrome suggest methylmercury pollution in these communities.

Except for the high prevalence of tandem gait abnormality and hearing loss, the pattern of prevalence is almost the same as our study from the Minamata area in the 1970s (Fujino, 1994). Harada and his colleague visited Grassy Narrows in 2002 (Harada et al., 2005a) and Grassy Narrows and Whitedog in 2004 (Harada et al., 2005b). In 2002, glove and stocking type of sensory disturbance (54.4%), tunnel vision (10.5%), ataxia (12.3%) and impaired speech (12.3%) were observed in 57 subjects (Harada et al., 2005a). Combining the results of both studies, 2002 and 2004, glove and stocking type sensory disturbance was found in 65.1% of the cases, tunnel vision in 10.8%, ataxia in 25.1% and impaired speech in 6.9% of the 175 subjects (Harada et al., 2005b). As the criteria used for determining ataxia was not described in those papers, it is possible that the actual percentage could be greater than the papers suggested. We show the raw data for ataxic movement in this study.

Our present study differs from the former studies of 1975, 2002 and 2004, in that quantitative measurements of somatosensory disturbance were added to the study. The results of two-point discrimination sensitivity of the lower lip, vibration sense and position sense in big toes were also worse in Group JE, followed by Groups CO, CY and JC. Somatosensory acuity was most impaired in Group JE. Whereas minimal tactile sensitivity of the lower lip and left big toe were worst in Group CO, followed by Groups JE, CY and JC. The reason for these differences is, as yet, unascertained. As to two-point discrimination, the "Yes/No" method was used in testing Groups CO and CY, and the "2-alternative, forced-choice method" was used in testing of Groups JE and JC. But, in our experience, there were no great differences between results of these two methods.

Quantitative measurements showed that somatosensory disturbance, showing a similar pattern, was present in all tested body areas. These patterns are assumed to be the characteristics of somatosensory disturbance due to a uniformly injured central nervous system, especially the sensory cortex.

The similarity in the symptoms of abnormalities, neurological findings and quantitative measurements in Groups JE and CO were assumed to be produced by methylmercury poisoning. The intermediate abnormalities in Group CY suggest a milder and continuing effect of methylmercury poisoning in the younger generations of this area.

There are some limitations in this study. The first one is that the subjects in the study consisted of applicants who volunteered for the examination. The subjects in Groups JE, CO and CY ate a lot of fish, but the subjects in Group JC ate less fish. Also the lifestyle and occupations of the Japanese and the Canadian subjects are likely to be quite different. Also, the lifestyle and occupations were not the same in the two Japanese groups JE and JC.

Secondly we had no control area in Canada. Instead, we compared the Canadian results with the results from a control area in Japan, as well as from an exposed area in Japan.

Thirdly, there can be other factors involved which can affect the findings. In Groups CO and CY, some neurological diseases were found which confused the analysis of the test results. In order to overcome this limitation, we tried to compare a wide range of symptoms and complaints, neurological findings and quantitative measurements.

In the earlier study in Japan, we found that some complaints, symptoms, neurological findings, and quantitative measurements recorded could be affected by the presence of certain other neurological diseases. However, the similar patterns of symptoms, neurological findings, and quantitative sensory measurements cannot be explained without taking

Table 7
Correlation of prevalence of complaints and neurological examinations — group comparisons.

		CO, JC	CY, JC	JE, JC	CO, CY	JE, CO	JE, CY
"Yes, always"	Correlation coefficient	0.315	0.301	0.549	0.630	0.712	0.450
	p-Value	n.s.	n.s.	<0.01	<0.01	<0.01	<0.01
"Yes, always" + "Yes, sometimes"	Correlation coefficient	0.578	0.521	0.693	0.813	0.787	0.712
	p-Value	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Neurological examination	Correlation coefficient	0.628	0.242	0.048	0.784	0.708	0.825
	p-Value	<0.05	n.s.	n.s.	<0.01	<0.01	<0.01

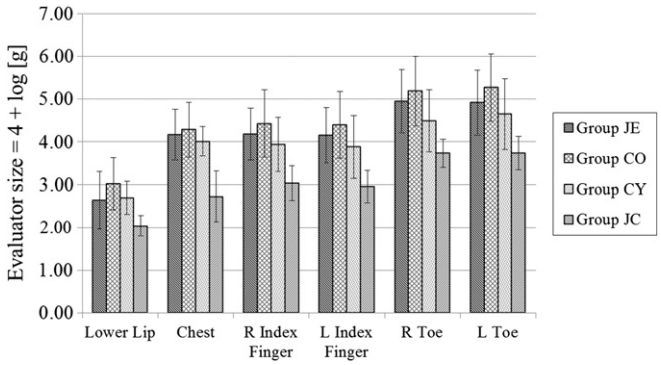

Fig. 1. Threshold for minimal tactile sense — group comparisons. JC, JE: $p < 0.01$ (all locations), JC, CO: $p < 0.01$ (all locations), JC, CY: $p < 0.01$ (all locations), JE, CO: $p < 0.01$ (lower lip), $p < 0.05$ (L toe), n.s. (other locations), JE, CY: $p < 0.01$ (R toe), $p < 0.05$ (chest and R index finger), n.s. (other locations), and CO, CY: $p < 0.05$ (chest), $p < 0.01$ (other locations).

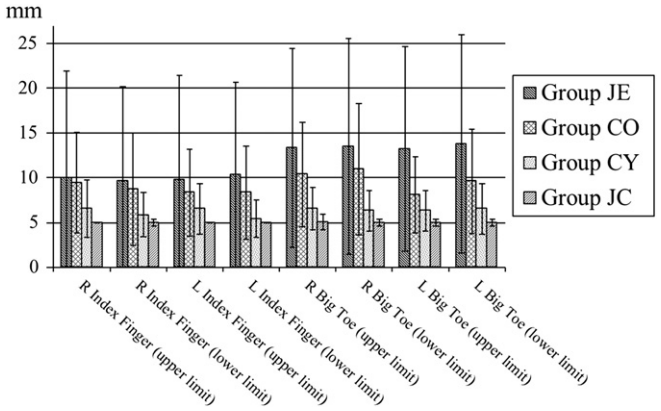

Fig. 3. Threshold for position sense — group comparisons. JC, JE: $p < 0.01$ (all locations), JC, CO: $p < 0.01$ (all locations), JC, CY: $p < 0.05$ (R index finger—lower), n.s. (L index finger—lower), $p < 0.01$ (all other locations), JE, CO: $p < 0.01$ (L big toe—upper & lower), $p < 0.05$ (R big toe—upper), n.s. (all other locations), JE, CY: $p < 0.05$ (L index finger—upper), $p < 0.01$ (all other locations), and CO, CY: $p < 0.05$ (R index finger—lower, L index finger—upper, L big toe—upper), $p < 0.01$ (all other locations).

into account the possibility of the presence of methylmercury poisoning (Takaoka et al., 2008).

After Harada's paper in 1976, the health effects of methylmercury on indigenous Canadians (First Nations) were reported by Canadian researchers. Barbeau et al. (1976) reported that he discovered symptoms like Minamata disease patients in Quebec, but he soon modified and toned down his conclusions. Shephard (1979) admitted the higher exposure in Canadian Indians and insisted that there was no agreement on the clinical diagnosis of mercury poisoning. But no conclusive human health-related data of Canadian Indians were presented by them. Wheatley et al. (1979) reported that they failed to prove abnormalities attributable to methylmercury. But his study in 1979 was based on clinical and pathological test results taken from only one male Cree Indian.

In north-western Quebec, McKeown-Eyssen and Ruedy (1983) studied individuals from the Cree First Nation and reported incoordination (15.4% in males/6.5% in females in Mistassini, 44.2% in males/26.5% in females in Great Whale), abnormal perception of sensations (5.7% in males/4.3% in females in Mistassini, 9.3% in males/2.1% in females in Great Whale). The results for subjects who had visual field scores of over 55° were 16.0% in males and 19.4% in females in Mistassini, and 0.7% in males and 15.5% in females in Great Whale. Although the patterns of abnormalities are very different from our results, the presence of these abnormalities could suggest milder methylmercury poisoning. The Canadian researchers did not, however, come to this conclusion. It would appear that they did not follow up and observe the individuals for a longer period of time, which is unfortunate. In our new Minamata disease cases, observed from November 2004 to April 2005, about half of them were considered to have had their first abnormalities after 1968, the year when the Chisso Company ceased to release methylmercury contaminated industrial wastewater (Takaoka et al., 2009).

Spitzer et al. (1988) found no abnormalities in north-western Quebec when they compared people from the Cree First Nation with a control group. In this study, the exposed group (SDD: The Self Designated Disease Group) and other three control groups were compared. Visual constriction was observed in 4.9% of SDD and in 0.0–1.7% of other groups. Peripheral sensory disturbance (written as "peripheral neuropathy" in the paper) was observed in 9.9% of SDD and in 1.5–3.1% of other groups. There was a higher tendency in the prevalence in SDD, but there was no statistical difference between SDD and other groups.

In examining the Cree people, McKeown-Eyssen et al. (1990) emphasized inter-observer variation. It is more important, however, to do the same examination in the control area and to set criteria for judging the results of the examination, rather than to repeat an examination in the same group. Even though there were some variations and differences between the results obtained by the screener and the results of the neurological examination by the neurologist, e.g. prevalence of tremor (screener 40.2%, neurologist 29.9%) and incoordination (screener 22.8%, neurologist 15.4%) in men in Mistassini, they were still high. They also had differences in criteria when identifying neurological abnormalities, but it should be possible to set standard criteria for the assessment of the neurological findings similar to our methods.

Changeability of neurological functions is quite common in cases of Minamata disease. In Minamata disease the range of four limb

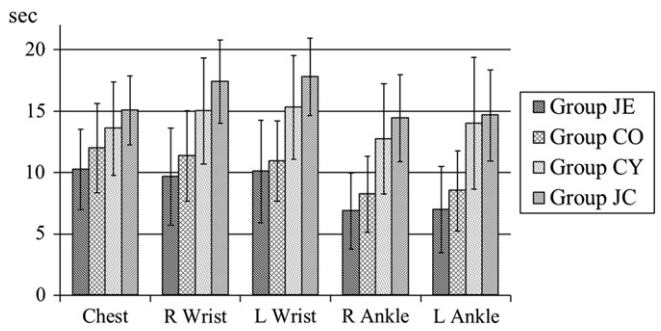

Fig. 2. Threshold for vibration sense — group comparisons. JC, JE: $p < 0.01$ (all locations), JC, CO: $p < 0.01$ (all locations), JC, CY: $p < 0.01$ (bilateral wrist), $p < 0.05$ (chest and R. ankle), n.s. (L. ankle), JE, CO: $p < 0.01$ (chest), n.s. (L wrist), $p < 0.05$ (other locations), JE, CY: $p < 0.01$ (all locations), and CO, CY: n.s. (chest), $p < 0.01$ (all other locations).

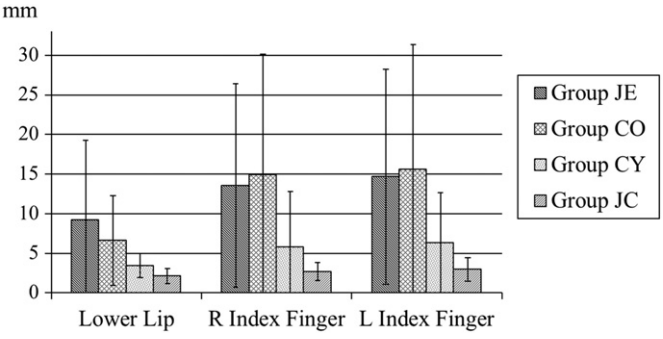

Fig. 4. Threshold for two-point discrimination — group comparisons. JC, JE: $p < 0.01$ (all locations), JC, CO: $p < 0.01$ (all locations), JC, CY: $p < 0.05$ (R index finger), $p < 0.01$ (other locations), JE, CO: $p < 0.05$ (lower lip), n.s. (other locations), JE, CY: $p < 0.01$ (all locations), and CO, CY: $p < 0.01$ (all locations).

somatosensory disturbance is changeable. Uchino and Araki (1984) reported on 100 cases of Minamata disease. In 77 of the 100 cases the examination for superficial sensory disturbance was carried out from 2 to 5 times, and 63 of the 77 cases (82%) were recorded as "unstable type" in which the area of the body showing somatosensory disturbance changed. The most important factor in showing the effect of environmental pollution on the inhabitants of an area is the ability to gather data and information from both the people exposed to the pollution and from a control group which has not been exposed.

There are some other Canadian studies about the exposure to methylmercury (Wheatley and Paradis, 1996; Wheatley et al., 1997; Wheatley and Paradis, 1998), but there are few that take into account epidemiological human health data. Wheatley (1996) stressed that the people of the First Nations understanding of mercury contamination is influenced by their holistic concepts of health and environment. But there was too little physical data available to corroborate this concept. Without the presence of human physical data, we cannot evaluate psychological, social and spiritual values of health.

5. Conclusions

The data collected from methylmercury-exposed indigenous residents of Grassy Narrows suggest that they were poisoned by methylmercury. The health abnormalities also appear to exist in a milder form in the younger generations from Grassy Narrows.

Information on funding sources

The study was conducted partly with funding from Grants-in-Aid for Scientific Research of Japan Society for the Promotion of Science (JSPS) (No. 20330118). The title of the funding is "Studies on the reappraisal of the real situation of Minamata disease damages and their social impact in the past half-century."

Conflict of interest

There is no conflict of interest in this study.

Acknowledgment

We, the authors, would like to express our thanks to the late Masazumi Harada of the Open Research Center for Minamata Studies, Kumamoto Gakuen University, for his planning and involvement in carrying out this research, by dedicating this paper to his memory.

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APPENDIX G-1

Comments on *File No: 1003*
Environmental Assessment of Northern Pulp's Proposed Effluent Treatment Facility

Ellen Sweeney, PhD
Halifax, Nova Scotia
February 2019

Executive Summary

Overall, the material provided in *Section 9.0: Human Health Evaluation* of the environmental assessment is lacking, and additional content is required in order to provide a fully informed assessment. The information provided falls short and does not include an exposure assessment, toxicity assessment or thoughtful risk characterization. The scoping or screening level approach to the human health evaluation is superficial. The content is missing supporting evidence for the claims that are made, its reference materials are dated and rely heavily on the Toxikos (2006) report, and there is misinterpretation of key concepts throughout.

Unfortunately, it is not possible to fully assess the potential health impacts of the proposed effluent as Northern Pulp states that "various project details [are] under development and/or in the process of being refined" and specific effluent chemistry characteristics "will not be known with certainty until the project is operational" (p. 489, 491). This lack of detailed information about the proposed effluent is problematic, it provides an inaccurate and ultimately incomplete environmental assessment and makes it difficult to truly assess its content and to comment on risks and potential hazards without additional information.
It is very concerning that the environmental assessment did not include existing reference material available on Pictou Landing (e.g., Pictou Landing Native Women's Group et al., 2016; Castleden et al., 2016). There was no formal consultation by Northern Pulp and/or its consultants with Pictou Landing First Nation, and no information has been provided regarding consultation that may have occurred or is in progress with the Province.

There is only a single study identified as having a similar method/approach to the Northern Pulp project, a proposed elemental chlorine free kraft pulp mill in Bell Bay, Tasmania. The human health evaluation is largely dependent upon the Toxikos (2006) report, *Marine Impact Assessment – Bell Bay Pulp Mill Effluent* from Bell Bay, Tasmania. Perhaps most concerning about the reliance on the Tasmania project is that it is not explicitly clear that this data comes from a proposal rather than an operational pulp mill. It is not reassuring that Northern Pulp has relied so heavily on a singular proposed pulp mill and cannot point to another example of the proposed work that would provide evidence of successes and/or failures, as well as lessons learned.

The information provided in Northern Pulp's environmental assessment is insufficient in addressing the potential health risks associated with the proposed work. Ultimately, by not providing information on the treated effluent itself, the environmental assessment is incomplete which makes it difficult to truly assess its content and to comment on risks and potential hazards without additional information. The information provided in the human health evaluation does not provide sufficient evidence to support its claims. This project would benefit from a complete risk assessment process, including complete information and evidence about the treated effluent, and a consultation with Pictou Landing First Nations and other community members. The assessment should apply sex- and gender-based analysis and incorporate an in-depth understanding about the limitations of threshold values in accounting for low dose cumulative exposures, as well as a hazard assessment which accounts for the intrinsic hazard or intrinsic toxicity of the substance(s) and the potential to cause harm.

Introduction

I am writing in regard to the Environmental Assessment of Northern Pulp's proposed effluent treatment facility in Pictou County, Nova Scotia. I have been asked to comment on this environmental assessment which will be reviewed by the Minister of Environment, the Honourable Margaret Miller. I hope the Minister will carefully consider the feedback received as part of the public comment period of the environmental assessment in making her decision.

My research background has resulted in experience and expertise across broad disciplines, such as environmental health, population health and sex- and gender-based analysis, and in specific areas such as breast cancer, occupational health and cancer prevention. I completed a PhD at York University in the only interdisciplinary Environmental Studies doctoral program Canada. My dissertation research examines the body of Canadian law, policy and practice which encompasses Canada's regulatory regime for toxic substances. It demonstrates that these approaches are not inherently precautionary and do not enact a primary prevention approach to women's health, and breast cancer in particular. I have translated this research into a book chapter, four peer-reviewed articles, 13 local, national and international presentations, as well as contributions for non-academic audiences.

I play an active role in an advisory and research capacity for organizations focused on health and environmental health issues. I was the Acting Director for the National Network on Environments and Women's Health in 2012-2013. I have served in an advisory capacity for this national organization, as well as for Breast Cancer Action Quebec. I am an affiliate scientist for the Beatrice Hunter Cancer Research Institute and a researcher on the ENRICH project which focuses on environmental racism in Mi'kmaq and African Nova Scotian communities. I am also a member of the Board of Directors for Prevent Cancer Now and the Scientific Review Panel of Breast Cancer UK, both organizations that focus on preventing cancer by reducing exposure to carcinogens and hazardous chemicals.

I am currently the Director of Strategic Research Initiatives at the Atlantic Partnership for Tomorrow's Health (Atlantic PATH) study. Atlantic PATH is part of the Canadian Partnership for Tomorrow Project, a national multi-cohort prospective research study with more than 300,000 participants. The data collected includes questionnaire data, physical measures and biological samples (blood, urine, saliva, toenails). My research at Atlantic PATH and active participation in our multi-disciplinary research team has resulted in seven published articles to date, with one currently accepted, two under review and one pending submission. In my role as the Director of Strategic Research Initiatives at Atlantic PATH, I am also responsible for overseeing the data access process which involves working directly with researchers and the research platform that holds data and biological samples for more than 35,000 participants.

Human health evaluation

As my expertise lies in health research, I focused on the related material in Northern Pulp's environmental assessment. Overall, I find the material provided in *Section 9.0: Human*

Health Evaluation to be lacking, and that additional content is required in order to provide a fully informed assessment. The information provided falls short and does not include an exposure assessment, toxicity assessment or thoughtful risk characterization. The scoping or screening level approach to the human health evaluation is superficial. The content is missing supporting evidence for the claims that are made, its reference materials are dated and rely heavily on the Toxikos (2006) report, and there is misinterpretation of key concepts throughout.

Vague language is used throughout the discussion (e.g., "the marine study boundary is considered to be a radius of *a couple to a few hundred metres*…" (p. 494, emphasis added) which does not aid in the evaluation of the environmental assessment. There are also problematic language choices such as "implausible" which negates any level of risk, rather than framing something as unlikely to occur.

Unfortunately, it is not possible to fully assess the potential health impacts of the proposed effluent as Northern Pulp states that "various project details [are] under development and/or in the process of being refined" and specific effluent chemistry characteristics "will not be known with certainty until the project is operational" (p. 489, 491). This lack of detailed information about the proposed effluent is problematic, it provides an inaccurate and ultimately incomplete environmental assessment and makes it difficult to truly assess its content and to comment on risks and potential hazards without additional information.

While Northern Pulp does acknowledge that there are other pulp and paper mill projects that may share similarities to the proposed project in Pictou County, the language is both vague and at times contradictory. For example, it is stated that key aspects of these studies, such as exposure pathways and receptors "cannot be assumed to be directly applicable to the current project," but that they "may facilitate to some degree the identification of key exposure pathways [and] receptors…and may also inform on certain assessment approaches" (p. 492). There is no supporting evidence offered related to these projects.

There is only a single study identified as having a similar method/approach to the Northern Pulp project, a proposed elemental chlorine free kraft pulp mill in Bell Bay, Tasmania.[1] The human health evaluation is largely dependent upon the Toxikos (2006) report, *Marine Impact Assessment – Bell Bay Pulp Mill Effluent* from Bell Bay, Tasmania. Northern Pulp appears to rely heavily on this report, while also critiquing its findings. It is framed as a "highly conservative assessment that substantially overestimated exposure and risk to potential human consumers of fish and shellfish that may be influenced by the effluent diffuser discharge in Bell Bay" (p. 491) without providing any substantive context or evidence for this claim.

Perhaps most concerning about the reliance on the Tasmania project is that it is not explicitly clear that this data comes from a proposal rather than an operational pulp mill. From the media coverage available, it becomes apparent that the Gunns Ltd. timber

[1] It should be noted that the Tasmanian mill process would focus on hardwood eucalyptus, as opposed to softwood coniferous wood species in Nova Scotia.

company collapsed and that the permits for the mill have lapsed. It is not reassuring that Northern Pulp has relied so heavily on a singular proposed pulp mill and cannot point to another example of the proposed work that would provide evidence of successes and/or failures, as well as lessons learned.

It is well established that Atlantic Canada has among the highest rates of cancer and other chronic disease in Canada (Marrett et al., 2008; Canadian Cancer Society's Steering Committee on Cancer Statistics, 2012; Xie et al., 2015; Sweeney et al., 2017a). There are numerous factors that must be considered regarding cancer outcomes, including lifestyle and behavioural factors, genetics, and environmental exposures. As demonstrated in the research report published on Pictou Landing (which is not referenced in the environmental assessment), there is significant concern about the impact that the mill has had in the past and will have in the future on the health of local residents and the broader environment (Pictou Landing Native Women's Group et al., 2016). Northern Pulp states that the proposed project is "expected to have no negative effects on human health" (p. 117). It also references a recent report from the Nova Scotia Health Authority (NSHA) in response to concerns that there is a high incidence of cancer [and other chronic health issues] experienced by Pictou Landing First Nation and surrounding communities. To support this claim, Northern Pulp refers to the NSHA report by stating that the incident rate of all cancers for men in Pictou County is below the provincial average. However, this interpretation could be skewed by the low rates of prostate cancer in Pictou County, as well as by considering the county as a whole rather than at the community-level (Saint-Jacques et al., 2018). It should also be noted that Statistics Canada data indicates that the cancer incidence among men, women and the total cancer cases in Pictou County-Guysborough Antigonish Strait are above the Canadian average (Statistics Canada, 2013).

Northern Pulp suggests that there are limited data on traditional marine food item harvesting and consumption patterns within the Pictou Landing First Nation, as well as the broader experiences of this community. The amount of space dedicated to this topic seems wholly inadequate (p. 485). It is very concerning that the environmental assessment did not include existing reference material available on Pictou Landing (e.g., Pictou Landing Native Women's Group et al., 2016; Castleden et al., 2016), and that the process did not include a formal consultation with Pictou Landing First Nation, as well as with those who could speak to the other community marine recreational patterns. It is stated that a Pictou Landing First Nation dietary survey would be preferable but may not be feasible (p. 499), however, this should be a requirement. A fulsome consultation process with Pictou Landing First Nation should be undertaken, as well as with key experts, such as Dr. Diana (Dee) Lewis who spent seven years researching the impacts of the pulp and paper mill on the health of Pictou Landing First Nation.

Gaps in understanding of environmental exposures and potential health outcomes

I have numerous concerns about the information presented in section 9.2.2: *Identification of Potential Human Receptors and Their Characterization*. This section starts by describing a human receptor as a hypothetical person who may experience exposure and encompasses infants, toddlers, children, adolescents, and adults (p. 496). This definition, as well as the

proposed human receptor age classes (p. 497) exclude fetal exposure which is an important component of exposure to carcinogenic and endocrine disrupting chemicals and intergenerational outcomes. Biomonitoring research demonstrates that virtually all pregnant women in the United States experience body burdens with reproductive and developmental toxic substances such as lead, mercury, toluene, perchlorate, phthalates, bisphenol A (BPA), pesticides, polychlorinated biphenyls (PCBs), perfluorochemicals (PFCs), and polybrominated diphenyl ethers (PBDEs) (Sutton et al., 2010, 2012; Woodruff et al., 2011; Parry et al., 2018). Similar findings have emerged from biomonitoring studies in other western countries, including Canada (Environmental Defence, 2006, 2013; Basu et al., 2013; Sweeney, 2014, 2017; Health Canada, 2018). The body burden associated with exposure to toxic substances that is revealed through biomonitoring processes can be viewed as representing the personalization of pollution -- pollution is no longer something external that occurs in isolation outside of the body (Altman et al., 2008; Sweeney, 2017). These toxic substances can cross the placental barrier which results in infants who are born ''pre-polluted'' and can result in intergenerational health outcomes (Leffall and Kripke, 2010: 98; Mallozzi et al., 2016; Eisen et al., 2018; Lee and Mykitiuk; 2018).

Northern Pulp also suggests that females may be more sensitive than males as a "function of differences in physiological, endocrine and biochemical parameters" without providing any evidence of this statement (p. 498). In fact, carcinogenic and endocrine disrupting chemicals have been detected in human seminal fluid (Chapin et al., 2004; Marques-Pinto and Carvalho, 2013; UNEP and WHO, 2013; Jeng, 2014; Sweeney, 2017; Rehman et al., 2018; Adoamneia et al., 2018; Smarr et al., 2018). The impairment of testicular development and reproductive function in males have also been associated with fetal exposure to toxic substances (Royal College of Obstetricians and Gynaecologists, 2013; Sweeney, 2017).

It is noted that those with the greatest potential for exposure to toxic substances and/or those who have the greatest sensitivity or potential to develop adverse effects should be considered in a formal human health risk assessment. However, without conducting a human health risk assessment, the next sentence seems to predict that there will be no potential health risks found among these populations and thus, there would be no risk (p. 497). Furthermore, it is suggested that infants and children may be more sensitive to exposure to some toxic substances than adults. This does not allow for fully understanding vulnerable or susceptible populations, including women who may be more susceptible to exposures to toxic substances and subsequent health outcomes based on the timing of exposure and windows of susceptibility. These windows of susceptibility involve periods of development or hormonal activity in which women's bodies may be more susceptible. They occur throughout the lifecourse and include the prenatal period, childhood, puberty, menstruation, pregnancy, and menopause (Birnbaum, 2009; Diamanti-Kandarakis et al., 2009; Schwarzman and Janssen, 2010; Schug et al., 2011; Brophy et al., 2012; Mallozzi et al., 2016; Gray et al., 2017; Herceg et al., 2018; Cohn et al., 2019).

There is also a great need for a sex- and gender-based analysis to be applied to any evaluation of health, risk and exposure to toxic substances. The analysis of sex and gender in health research has emerged as an increasingly important methodology which

necessitates the consideration of impacts on both men and women, as well as identifying the shortcomings which emerge as a result. The foundation of sex- and gender-based analysis is the understanding that both biology and social experiences, and thus sex and gender, impact the health status of Canadian citizens (Sweeney, 2014; Canadian Institutes of Health Research – Institute of Gender and Health, 2019). In this instance, Northern Pulp is conflating the two concepts when they speak about gender (p. 498).

In brief, sex refers to biological and genetic characteristics which are manifested in one's anatomy, physiology and hormones. Sex includes the "specific capacities of our bodies, and affects the propensity and trajectory of diseases and health conditions" (Greaves, 2009: 3). Gender should not be confused or conflated with sex as it is a social construct that "extends beyond the boundaries of biologically defined categories of sex" (Benoit and Shumka, 2009: 7). Gender includes the social, cultural and economic factors that influence the socially constructed roles and relationships, personality traits, attitudes, behaviours, values, and influence that a particular society assigns to women, men and other gender groups (Clow et al., 2009; Greaves, 2009; Sweeney, 2014). The consideration of gender in health research is especially critical as it can "determine different exposures to certain risks, different treatment-seeking patterns, or differential impacts of social and economic determinants of health" (Hankivsky, 2007: 155).

The discussion related to sex, stated incorrectly in the environmental assessment as gender, does not provide evidence to support the statements about exposure and risk among male and female receptors. There also seems to be some misunderstanding about "body weight" which is stated as both having little difference between men and women, and as a relevant factor potentially affecting female receptors due to a "slightly higher intake rate to body weight ratios" (p. 498). However, there is a difference between body weight and body fat distribution. For instance, women may be at higher risk for health outcomes related to exposure to toxic substances which tend to concentrate in body fat and are often related to estrogen receptors, and women tend to have a higher ratio of body fat and estrogen levels than men (Nickerson, 2006; Assembly of First Nations Environmental Stewardship Unit, 2009; Clow et al., 2009; Sweeney, 2014). It is important to ask questions about levels of susceptibility and sex-linked differences when considering environmental exposures which raises additional issues related to sex-specific variations in disease, health and illness (Sweeney, 2014).

The discussion of "receptors" in this section is lacking. In this case, a "human receptor" is described as someone who lives, visits or works an area where exposure may occur, and the "receptor type," influenced by physical and behavioural characteristics, is used to determine the amount of chemical exposure received by the human receptor (p. 496). The discussion would benefit from evidence to support the claims, likewise the definitions would benefit from being fleshed out. For instance, the use of the term "Indigenous receptors" is unclear. Is this describing "human receptors" with First Nations ancestry and is thus focused on relevant social determinants of health which are not adequately explored, or are there specific biological receptors relevant to members of the Pictou Landing First Nation? Perhaps the discussion of receptors would also benefit from

consideration of biological receptors, such as estrogen receptors which are impacted by endocrine disrupting chemicals (Shanle and Xu, 2011; Li et al., 2013; Barrett, 2014).

Key concepts in assessing exposure to toxic substances

Should a risk assessment process be required moving forward, there are also gaps in this process that should be highlighted. Throughout the human health evaluation, there is a reliance on the concept of risk posed to human health *if there is significant exposure* to toxic substances. This approach is grounded in the risk assessment process that determines toxicological effects employing the traditional dose-response relationship whereby "the dose makes the poison" and utilizing threshold values. The use of threshold values in the risk assessment process suggests that threshold effects occur only at a specific level of exposure (Sweeney, 2014). Health Canada contends that a toxicological threshold exists below which adverse effects do not occur.

> Below a certain minimum dose,…compensatory mechanisms can mitigate the adverse effects of a substance, even on a continuing basis. At higher dose levels, however, the ability of the organisms to compensate or adapt becomes overwhelmed, leading to an impairment in organ function or development of disease state (Health Canada, 2007: 6; Sweeney, 2014).

Endocrinologists, environmental health researchers and advocates are raising competing paradigms to contest the reliance on toxicology and the dose-response relationship in risk assessment processes (Pesch et al., 2004; Vandenberg et al., 2009, 2012; Ritter, 2011; Grossman, 2012, 2013; Darbre and Fernandez, 2013; Sweeney, 2014; Zoeller and Vandenberg, 2015; Mallozzi et al., 2016). The traditional dose-response relationship posits that toxicological effects increase with increased exposure and dose of a toxic substance (Health Canada, 2007). The high dose animal testing and linear extrapolation utilized in toxicology does not allow for the potential of health effects occurring below the "safe" levels utilized in evaluating threshold values (Birnbaum, 2012; Brophy et al., 2013). Vandenberg et al. (2012) analyzed hundreds of epidemiological studies in order to demonstrate the impact of low-dose effects of endocrine disrupting chemicals on human health in comparing the role of non-monotonic responses and the traditional dose-response relationship.

> Whether low doses of EDCs influence certain human disorders is no longer conjecture, because epidemiological studies show that environmental exposures to EDCs are associated with human diseases and disabilities.…[W]hen nonmonontonic dose response curves occur, the effects of low doses cannot be predicted by the effects at high doses. Thus, fundamental changes in chemical testing and safety determination are needed to protect human health (Vandenberg et al., 2012).

Thus, the argument that the amount of exposure, whether it is considered "significant," does not allow for the acknowledgement of low-dose cumulative exposures. Exposure data and threshold effects, the premise of risk assessment, do not adequately account for the possibility of substances such as endocrine disrupting chemicals that have low dose,

cumulative and synergistic effects as a result of exposure to complex mixtures of toxic substances (Rochon Ford and Sweeney, 2015). While mixtures of chemicals are briefly mentioned in the environmental assessment (p. 512), there is no discussion of synergistic effects and related health outcomes.

Another gap in human health risk assessments conducted in Canada is that they focus on both risk and exposure, but not *hazard*. Health Canada does not recommend the use of hazard assessment over risk assessment because the potential for harmful effects is "wholly dependent upon the extent of exposure," and that the level of risk increases with an increase in exposure (Health Canada, 1995). As noted above, this does not account for low-dose cumulative exposures and the potential health outcomes. Conversely, environmental health experts recommend the use and implementation of a *hazard assessment*, which does not contain an exposure requirement but rather includes an assessment of the intrinsic hazard or intrinsic toxicity of the substance and its potential to cause harm (Sweeney, 2014; Kienzler et al., 2016; Syberg and Hansen, 2016; Solecki et al., 2017). "The issue of how much of the substance enters the environment is not taken into account. The possibility that an inherently toxic substance *might* enter the environment is accepted as reason enough to trigger the regulatory process" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 60).[2]

Potential examples of exposure to toxic substances

Northern Pulp references toluene as having a measurable exposure above recommended detection limits in recent chemical analyses of current treated mill effluent (p. 516). Toluene is an example of a toxic substance that possesses inherently toxic properties but was found to be not toxic under the *Canadian Environmental Protection Act* because of the exposure requirement and thus is not subject to federal risk management provisions. This substance is linked to numerous health concerns including developmental and reproductive toxicity, neurotoxicity, and organ system toxicity (Sweeney, 2014). In California, toluene falls under the risk management provisions of the *Safe Drinking Water and Toxic Enforcement Act*, also known as Proposition 65. Proposition 65 requires the state to publish a list of toxic substances that are known to cause cancer, birth defects or other reproductive harm and which must be updated at least once a year. Industry and businesses must notify citizens about "significant amounts of chemicals in the products they purchase, in their homes or workplaces, or that are released into the environment" (Office of Environmental Health Hazard Assessment, 2013). To some extent, the onus of responsibility is placed on the individual to use the information provided though Proposition 65 to reduce exposures that may not be adequately controlled under other state or federal regulation. However, this law has also created incentives for manufacturers to remove toxic substances that are listed as part of this initiative. For example, following their inclusion on the list, toluene was removed from many nail care products (Office of Environmental Health Hazard Assessment, 2013; Sweeney, 2014). The European Union placed restrictions on toluene in 2004 so that the substance "shall not be placed on the

[2] For a more detailed discussion of risk vs. hazard assessment and threshold vs. non-threshold values, please refer to Sweeney, 2014.

market, or used, as a substance or in mixtures in a concentration equal or greater than 0.1% by weight where the substance or mixture is used in adhesives or spray paints intended for supply to the general public" (Armstrong and Dupont, 2012: 52).

Northern Pulp also reports a significant list of metals tested in 2002 that were above recommended detection limits, including lead and arsenic. Exposure to lead through drinking water is an issue of increasing concern, particularly with recent high-profile cases of lead-contaminated water (Bellinger, 2016; Hanna-Attisha et al., 2016). Low-level effects of lead are associated with neurodevelopmental, neurodegenerative, cardiovascular, renal, reproductive, and developmental impacts (Sweeney et al., 2017b). The maximum acceptable concentration level for drinking water in Canada is 10 µg/L, whereas the current blood intervention level is 10 µg/dL. The health effects related to lead exposure are well established and there is evidence that blood lead levels as low as 5 µg/dL are associated with adverse health effects in both children and adults. Water and toenail samples from Nova Scotia residents have been analyzed for lead concentrations. Approximately 46% of Nova Scotia residents use well water as their primary source of drinking water. Water from dug wells had higher lead concentrations compared to drilled wells, and the lowest lead levels were found in water from municipal supplies. Although the majority of the lead levels in the drinking water provided by individuals in Nova Scotia were below the Canadian maximum acceptable concentration level, there were outliers, particularly among unregulated private well water sources. Given the health effects that are linked to low-level exposures, any exposure to lead in water sources remains a concern (Sweeney et al., 2017b).

Long-term exposure to environmental arsenic has been associated with many chronic diseases, including cancer and diabetes (Hughes et al., 2011). There are a number of local ongoing projects exploring arsenic and arsenic speciation exposure and associated health conditions, including cancer and diabetes. An article currently under review found that the percentage of monomethylarsonic acid (%MMA) was found to be significantly higher in the toenails of individuals in Atlantic Canada with arsenic-related cancers, compared to healthy individuals with similar total arsenic exposure (Smith et al., Under Review). Other toxic substances highlighted in Northern Pulp's discussion of substances above recommended detection limits in 2018 include hydrocarbons, cyanide, mercury, other metals and metalloids, and trace polyaromatic hydrocarbons (PAHs); there are also various health concerns with these substances.

It is particularly concerning that the risks associated with a potential accident where the pipeline travels across land and through watersheds in Pictou County are not considered in the human health evaluation. It is suggested that exposure pathways (e.g., air, ground water) "do not merit consideration in relation to the project" (p. 496), but exposures to toxic substances via air pollution or in drinking water do have potential for detrimental health outcomes and should be fully considered (e.g., Hoffman et al., 2017).

Finally, it is concerning that Northern Pulp uses non-committal language in discussing environmental monitoring in the marine area where treated effluent will be discharged. For example, stating that there "will likely be environmental monitoring" (p. 519), rather

than committing to monitoring practices as part of comprehensive risk management practices.

Conclusion

In sum, I do not believe that the information provided in Northern Pulp's environmental assessment is sufficient in addressing the potential health risks associated with the proposed work. Ultimately, by not providing information on the treated effluent itself, the environmental assessment is incomplete which makes it difficult to truly assess its content and to comment on risks and potential hazards without additional information. The information provided in the human health evaluation does not provide sufficient evidence to support its claims. This project would benefit from a complete risk assessment process, including complete information and evidence about the treated effluent, and a consultation with Pictou Landing First Nations and other community members. The assessment should apply sex- and gender-based analysis and incorporate an in-depth understanding about the limitations of threshold values in accounting for low dose cumulative exposures, as well as a hazard assessment which accounts for the intrinsic hazard or intrinsic toxicity of the substance(s) and the potential to cause harm.

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APPENDIX G-2

Ellen Sweeney, PhD

c/o Atlantic PATH, Dalhousie University
1494 Carlton Street, PO Box 15000, Halifax, Nova Scotia B3H 4R2
Email: Ellen.Sweeney@dal.ca Phone: (902) 494-7818

EDUCATION

PhD, Faculty of Environmental Studies, York University, 2013
Specialization: Environmental Health
Dissertation: *Preventing Breast Cancer: An Analysis of Canada's Regulatory Regime for Chemicals*

Canadian Institutes of Health Research Institute of Gender and Health Summer Institute
Intensive training on gender, sex and health research under the guidance of expert mentors
July 5-9, 2010
Vancouver, British Columbia

Master of Arts, Social Anthropology, Dalhousie University, 2006
Specialization: Health and Illness (Environmental Health)
Thesis: *Biographical Disruption and the Environmental Health Controversy at the New Waterford Consolidated Hospital*

Bachelor of Arts, Honours, Social Anthropology, Dalhousie University, 2003
Dean's List (2002-2003)

RESEARCH CONTRIBUTIONS

Refereed Publications:

Sweeney, E., Yu, Z.M., Dummer. T.J.B., Cui, Y., DeClercq, V., Forbes, C., Grandy, S.A., Keats, M., Parker, L., Adisesh, A. (Submission Pending). "The Effect of Shift Work on Cardiometabolic Health and Depression: Findings from the Atlantic PATH Study." *International Archives of Occupational and Environmental Health*.

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Sweeney, E. (2011). Book Review: *Dodging the Toxic Bullet: How to Protect Yourself from Everyday Environmental Hazards* by David Richard Boyd. Green Book Reviews.

Gahagan, J., **Sweeney, E.**, Worthington, C., Perry, D., Satzinger, F., and Rogers, E. (2008). "Research Ethics Issues for Biomedical HIV/AIDS Researchers in International Settings – Perspectives from Canadian Experience." *International Journal of Infectious Diseases*, 12(6).

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Media Releases:

"Re-Think Pink During Breast Cancer Awareness Month: A Prevent Cancer Now Challenge to Canadians." Media Release for Prevent Cancer Now. October 14, 2016.

"Chemical exposures contributing to elevated breast cancer risk in some occupations: Research findings." Media Release for the National Network on Environments and Women's Health. Distributed internationally. November 19, 2012.

Select Non-Refereed Publications:

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Sweeney, E. (2012). *Breast cancer risk in relation to occupations with exposure to carcinogens and endocrine disruptors: a Canadian case-control study. Summary of Research Findings.* Canadian Women's Health Network and National Network on Environments and Women's Health.

Sweeney, E. (2011). "Breast Cancer as a Contested Illness." *Proceedings from the 16th Annual Graduate Student Symposium.* York Institute for Health Research (YIHR). York University. Toronto, Ontario.

Sweeney, E., Gahagan, J., and Langille, D. (2007). *Framework for Action: Youth Sexual Health in Nova Scotia Literature Review - "Attitudes Towards Youth Sexual Health.* Nova Scotia Department of Health Promotion and Public Protection.

Sweeney, E. and Gahagan, J. (2007). *Nova Scotia – Sierra Leone Programme (NSSLP) Peer Health Education Programme. Mid-Term Evaluation Report.* Nova Scotia-Gambia Association.

Published Abstracts:

Sweeney, E. (2015). "Contesting Everyday Exposures: Case Studies in Toxic Exposure, Public Health and Canada's Regulatory Regime." *Discard Studies: Studies of Waste, Pollution, and Externalities.* http://discardstudies.com/.

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Gahagan, J., **Sweeney, E.**, Jackson, R., Mill, J., Dykeman, M., Ricci, C., Patrick, C., and Benton, A. (2009). "HIV Risk, Historical Trauma and Systemic Inequities among Aboriginal Women in Canada." *The Canadian Journal of Infectious Diseases & Medical Microbiology, Vol. 19, Supplement B: 104B.*

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Jackson, R., Gahagan, J., **Sweeney, E.**, Mill, J., Dykeman, M., Benton, A., Patrick, C., and Ricci, C. (2009). "A Review of Methods and Models of Culturally Sensitive HIV Prevention Programs for Canada's Aboriginal Population." *The Canadian Journal of Infectious Diseases & Medical Microbiology, Vol. 19, Supplement B: 85B.*

Gahagan J., **Sweeney, E.,** and Baxter, J. (2007). "Peer Health Education: Lessons Learned from Sierra Leone." *The Canadian Journal of Infectious Diseases and Medical Microbiology, Vol. 18, Supplement B: 90B.*

Presentations as a Guest Speaker:

Invited Speaker. "Environmental Exposures and Breast Cancer: Considerations for Primary Prevention." Beatrice Hunter Cancer Research Institute Seminar Series. April 6, 2018.

Invited Speaker. "Exclusion, Extraction, and the Politics of Breast Cancer: The US-Middle East Partnership for Breast Cancer Awareness" by Samantha King. Faculty of Environmental Studies, York University. Co-Sponsored by the Canadian Women's Health Network and the National Network on Environments and Women's Health. March 20, 2012.

Invited Panel Discussant. Screening of Sandra Steingraber's story, *Living Downstream,* a film about cancer and the environment by Chanda Chevannes. Presented by the National Network on Environments and Women's Health. November 10, 2010.

Refereed Conference Presentations:

Sweeney, E., Cui, Y., DeClercq, V., Forbes, C., Grandy, S., Hicks, J., Keats, M., Parker, L., Yu, Z.M., Dummer, T.J.B. (2018). "Atlantic Partnership for Tomorrow's Health: Opportunities for Collaborative Health Research." Poster presentation at the Healthy Living, Healthy Life Conference. Halifax, Nova Scotia.

Sweeney, E., Cui, Y., DeClercq. (2018). "A Profile of Breast Cancer in Atlantic Canada: The Atlantic Partnership for Tomorrow's Health (Atlantic PATH) Study." Poster Presentation at Improving Breast Cancer Outcomes Through Fundamental Research. Halifax, Nova Scotia.

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Vena, J., Dummer, T.J.B., Le, N., Chu, J., Lai, C., Hicks, J., **Sweeney, E.**, Keats, M., Awadalla, P., McDonald, K., Jacquemont, S., Obadia, A., Noisel, N., Fortier, I., Davison, A., Chappell, H. (2018). "Use of mixed methods for follow-up survey implementation in five large geographically dispersed cohorts in Canada: successes and challenges in the Canadian Partnership for Tomorrow Project." Oral presentation at the International Conference on the Methodology of Longitudinal Surveys. Essex, England.

Sweeney, E., Cui, Y., DeClercq, V., Forbes, C., Grandy, S., Keats, M., Parker, L., Thompson, D., Yu, Z.M., and Dummer, T. (2018). "Cohort Profile: The Atlantic Partnership for Tomorrow's Health (Atlantic PATH) Study." Poster Presentation at the Canadian Nutrition Society Conference. Halifax, Nova Scotia.

Forbes, C., Yu, Z.M., Cui, Y., DeClercq, V., Grandy, S., Keats, M., Parker, L., **Sweeney, E.**, Dummer, T.J.B. (2018). "Prevalence and comparison of obesity and health behaviours between urban and rural residents: an Atlantic PATH study." Poster presentation at the Society of Behavioral Medicine Conference. New Orleans, Louisiana.

Sweeney, E., Yu, Z.M., Dummer, T.J.B., Cui, Y., DeClercq, V., Forbes, C., Grandy, S.A., Keats, M., Parker, L., Adisesh, A. (2018). "The Effect of Shift Work on Cardiometabolic Health: Findings from the Atlantic PATH Cohort Study." Oral presentation at the International Congress on Occupational Health. Dublin, Ireland.

Sweeney, E., Yu, Z.M., Dummer, T.J.B., Cui, Y., DeClercq, V., Forbes, C., Grandy, S.A., Keats, M., Parker, L., Adisesh, A. (2017). "The Effect of Shift Work on Cardiometabolic Health: Findings from the Atlantic PATH Cohort Study." Poster presentation at the 9th Annual New Brunswick Health Research Conference. Moncton, New Brunswick.

Atlantic PATH Team. (2017). Pre-Conference Session: An Overview of the Canadian Partnership for Tomorrow Project and the Atlantic Partnership for Tomorrow's Health." Canadian Public Health Association Conference. Halifax, Nova Scotia.

Sweeney, E., Cui, Y., DeClercq, V., Forbes, C., Grandy, S., Keats, M., Parker, L., Thompson, D., Yu, Z.M., and Dummer, T. (2017). "Cohort Profile: The Atlantic Partnership for Tomorrow's Health (Atlantic PATH) Study." Poster Presentation at the Canadian Public Health Association Conference. Halifax, Nova Scotia.

Yu, Z.M., Cui, Y., DeClercq, V., Forbes, C., Grandy, S., Keats, M., Parker, L., **Sweeney, E.**, and Dummer, T. (2017). "Fruit and Vegetable Intake and Obesity among Populations in Eastern Canada: the Atlantic Partnership for Tomorrow's Health Study." Poster Presentation at the Canadian Public Health Association Conference. Halifax, Nova Scotia.

DeClercq, V., Cui, Y., Forbes, C., Grandy, S., Keats, M., Parker, L., **Sweeney, E.**, Yu, Z.M., and Dummer, T. (2017). "Sleep and obesity in the Atlantic PATH cohort." Poster Presentation at the Canadian Public Health Association Conference. Halifax, Nova Scotia.

Forbes, C., Cui, Y., DeClercq, V., Grandy, S., Keats, M., Parker, L., **Sweeney, E.**, Yu, Z.M., and Dummer, T. (2017). "A comparison of the physical activity and sitting time correlates among Atlantic Canadians." Poster Presentation at the Canadian Public Health Association Conference. Halifax, Nova Scotia.

Cui, Y., DeClercq, V., Forbes, C., Grandy, S., Keats, M., Parker, L., **Sweeney, E.**, Yu, Z.M., and Dummer, T. (2017). "Association between Physical Activity and Self-Rated Health in Atlantic Canadians." Poster Presentation at the Canadian Public Health Association Conference. Halifax, Nova Scotia.

Dummer, T.J.B, and Atlantic PATH team. (2017). "Multimorbidity and physical activity in Atlantic Canadians." Oral presentation at the Society of Behavioral Medicine Conference. San Diego, California.

Traynor, R., Curran, J., Bishop, A., Cassidy, C., Hayden, J., Lawrence, L., McIssac, J., McNeil, K., Snelgrove-Clarke, E., **Sweeney, E.**, and Urquhart, R. (2016). "Designing policy and practice change interventions: Findings from a learning collaborative initiative to build capacity in KT practice." Poster presentation at KT Canada Annual Scientific Meeting. Toronto, Ontario.

Sweeney, E. (2013). "Canada's Toxic Regulatory Regime: A Discussion of Gender, Risk and Breast Cancer." Oral Presentation at the Environmental Health Conference: Science and Policy to Protect Future Generations. Boston, Massachusetts.

Sweeney, E. (2013). "Canada's Regulatory Regime for Chemicals: Implications for Breast Cancer and Risk." Oral Presentation at the York Institute for Health Research (YIHR) Graduate Student Research Symposium. York University. Toronto, Ontario.

Sweeney, E. (2012). "Women's Environmental Health: The Importance of Primary Prevention in Canadian Health Policy." Oral Presentation at the Canadian Institutes of Health Research (CIHR) Institute of Gender and Health, Advancing Excellence in Gender, Sex and Health Research Conference. Montreal, Quebec.

Sweeney, E. (2012). "Pink Ribbon Fatigue: A Critique of Cause-Related Marketing." Oral Presentation at the Environmental Studies Association of Canada (ESAC) Conference. Waterloo, Ontario.

Sweeney, E. (2012). "Environmental Health: A Call for Primary Prevention in Public Health Policy and Education." Oral Presentation at the Environmental Studies Association of Canada (ESAC) Conference. Waterloo, Ontario.

Sweeney. E. (2012). "Moving Beyond Breast Cancer Awareness Campaigns." Oral Presentation at the York Institute for Health Research (YIHR) Graduate Student Research Symposium. York University. Toronto, Ontario.

Sweeney, E. (2011). "The Gendered Implications of Breast Cancer, Risk and the Environment." Oral Presentation at the Environmental Studies Association of Canada (ESAC) Conference. Fredericton, New Brunswick.

Sweeney, E. (2011). "Breast Cancer as a Contested Illness." Oral Presentation at the York Institute for Health Research (YIHR) Graduate Student Research Symposium. York University. Toronto, Ontario.

Gahagan, J., **Sweeney, E.**, Jackson, R., Mill, J., Dykeman, M., Ricci, C., Patrick, C., and Benton, A. (2009). "HIV Risk, Historical Trauma and Systemic Inequities among Aboriginal Women in Canada." Poster Presentation at the 18th Annual Canadian Conference on HIV/AIDS Research (CAHR). Vancouver, British Columbia.

Gahagan, J., **Sweeney, E.**, Jackson, R., Mill, J., Dykeman, M., Patrick, C., Ricci, C., and Benton, A. (2009). "Challenges and Barriers to Health Care HIV Service Delivery: The Experience of Aboriginal Women in Canada." Poster Presentation at the 18th Annual Canadian Conference on HIV/AIDS Research (CAHR). Vancouver, British Columbia.

Jackson, R., Gahagan, J., **Sweeney, E.**, Mill, J., Dykeman, M., Benton, A., Patrick, C., and Ricci, C. (2009). "A Review of Methods and Models of Culturally Sensitive HIV Prevention Programs

for Canada's Aboriginal Population." Poster Presentation at the 18th Annual Canadian Conference on HIV/AIDS Research (CAHR). Vancouver, British Columbia.

Sweeney, E. (2008). "Contested Illness and the Environmental Health Controversy at the New Waterford Consolidated Hospital." Oral Presentation at the International EcoHealth Forum. Merida, Mexico.

Gahagan, J., Worthington, C., **Sweeney, E.**, Satzinger, F., and Rogers, E. (2008). "Ethics Issues for Canadian HIV/AIDS Researchers in International Settings." Poster Presentation at the 15th Canadian Conference on International Health. Ottawa, Ontario.

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Gahagan J., **Sweeney, E.** and Baxter, J. (2007). "Peer Health Education: Lessons Learned from Sierra Leone." Poster Presentation at the 16th Annual Canadian Conference on HIV/AIDS Research (CAHR). Toronto, Ontario.

Sweeney, E. (2007). "The Environmental Health Controversy at the New Waterford Consolidated Hospital." Oral Presentation at the International Ecological Integrity and a Sustainable Society Conference. Halifax, Nova Scotia.

Sweeney, E. (2006). "When Environment-Related Illness Strikes the Health Care Profession: An Exploratory Case Study of the New Waterford Consolidated Hospital." Oral Presentation at From the Cradle to the Grave: Future Perspectives on the Social History of Health and Healthcare. Glasgow, Scotland.

PROFESSIONAL EXPERIENCE

Adjunct Professor. Graduate Studies, Dalhousie University. June 2018-Present.

Director, Strategic Research Initiatives. Atlantic Partnership for Tomorrow's Health (Atlantic PATH). (Research investigating the environmental, lifestyle and genetic factors related to the development of cancer in Atlantic Canada). April 2017-Present.

Health Research Scientist. Atlantic Partnership for Tomorrow's Health (Atlantic PATH). Halifax, Nova Scotia. February 2016-Present.

Research Associate. REAL Knowledge Program and REAL Evaluation Services, Nova Scotia Health Research Foundation (NSHRF). Halifax, Nova Scotia. January 2014-February 2016.

Advisor to CIHR Team Grants. "Effects of Brominated Flame Retardants on Reproductive Health: Animal, Human, Ethical, Legal and Social Studies" and "'Green' Plasticizers: Impact of Exposure to Phthalates, their metabolites and 'green' plasticizers on male reproductive health." June 2013-2015. University of Western Ontario.

Acting Executive Director. National Network on Environments and Women's Health (NNEWH). York University. September 2012-July 2013.

Research and Writing Contract. Summary of research findings on a Canadian project related to breast cancer risk and occupational exposure to carcinogens and endocrine disruptors. Canadian Women's Health Network (CWHN) and National Network on Environments and Women's Health (NNEWH). August-November 2012.

Project Coordinator. "HIV Prevention in Canada: A Meta-Ethnographic Synthesis of Current Knowledge." School of Health and Human Performance, Dalhousie University. April 2008-April 2009.

Research Associate. "The Canadian Sexual Health Assessment Model: Establishing Indicators to Evaluate the Sexual Health of Canadians." School of Health and Human Performance, Dalhousie University (PI: University of Alberta). June 2007-March 2008.

Research Associate. Various Projects. School of Health and Human Performance, Dalhousie University. November 2007-March 2008.

Research Assistant. "The Problematization of OxyContin and the Treatment of Pain." Department of Sociology and Social Anthropology, Dalhousie University. November 2005-November 2007.

Project Coordinator. "Determinants of Adolescent Pregnancy: Factors Influencing Youth Behaviours in a Rural Nova Scotia Community." Department of Community Health and Epidemiology, Dalhousie University. November 2006-October 2007.

Research Associate. Various Projects. Atlantic Interdisciplinary Research Network (AIRN) for Social and Behavioural Issues in Hepatitis C and HIV/AIDS. November 2006-June 2007.

Research Associate. Evaluation of "Peer Health Education in Sierra Leone" for the Nova Scotia Gambia Association. School of Health and Human Performance, Dalhousie University. November 2006-April 2007.

Research Associate. "Framework for Action: Youth Sexual Health in Nova Scotia." Nova Scotia Department of Health Promotion and Public Protection and Dalhousie University. January 2007-March 2007.

Executive Member, Communications Officer. Canadian Union of Public Employees, Local 3912. 2004-2005.

Research Assistant. "Fluoride Mouthrinse Program." School of Dental Hygiene, Dalhousie University. October 2004-December 2004.

Referrals Clerk (Habitat Referrals Tracking System). Department of Fisheries and Oceans, Habitat Management Division. Dartmouth, Nova Scotia. Term Positions: 2001-2003.

TEACHING EXPERIENCE

Teaching Assistant (Tutorial Leader). "ENVS 1000: Introduction to Environmental Studies: Earth in our Hands." Faculty of Environmental Studies, York University. 2011-2012.

Teaching Assistant. "ENVS 2150: Environment, Technology and a Sustainable Society." Faculty of Environmental Studies, York University. 2010.

Teaching Assistant (Tutorial Leader). "ENVS 1000: Introduction to Environmental Studies: Earth in our Hands." Faculty of Environmental Studies, York University. 2009-2010.

Teaching Assistant. "SOSA 1100: Introduction to Anthropology." Department of Sociology and Social Anthropology, Dalhousie University. 2005-2006.

Teaching Assistant. "SOSA 2100: Environment and Culture." Department of Sociology and Social Anthropology, Dalhousie University. 2003-2006.

SELECT FUNDING AND AWARDS

Murphy, R., Dummer, T., Le, N., Vena, J., DeClercq, V., Cui, Y., Keats, M., Grandy, S., Sweeney, E. Awadalla, P., Brenner, D., Chu, Q., and Di Sebastiano, K. (2019). "Beyond Smoking: Investigating Risk Factors for Lung Cancer in the Canadian Partnership for Tomorrow Project." CIHR Operating Grant: Data Analysis Using Existing Databases and Cohorts. Value: $100,000.

Adisesh, A., Dummer, T., Kim, J.S., Keats, M., Sweeney, E. (2018). "Cadmium, Arsenic and other metal exposures as determinants of prostate cancer in the Canadian Atlantic provinces." Prostate Cancer Canada and New Brunswick Health Research Foundation. Value: $180,000.

Kim, J.S., Sweeney, E., Adisesh, A. (2018). "Arsenic speciation profiling for evaluating the association between cancer and arsenic exposure using toenail biomarkers in the Atlantic PATH Cohort Study: Feasibility and preliminary health outcomes." Nova Scotia Health Authority Research Fund Application. Value: $25,000.

Adisesh, A., Fleming, D., Kim, J.S., Sweeney, E., Dummer, E. (2018). Measurement methods for Lead in toenails: towards a novel biomonitoring of occupational exposure. WorkSafeNB Chief Medical Officer Occupational Medicine Research Fund. Value: $14,900.

Kim, J.S., Sweeney, E., and Sweeney, C. (2018). Metabolic Profiling of Human Biospecimens for Identification of Novel Biomarkers Associated with Pesticide Exposure Using a Non-Targeted Approach. Seed Funding. Beatrice Hunter Cancer Research Institute. Value: $10,000.

Waldron, I., Beaton, S., Haluza-Day, R., Mitchell, L., Teelucksingh, C., Thomas, R., Rutland, T., Sweeney, E. (Awarded). "Over the Line: A Bilateral Conversation on Race, Place, and the Environment." Connection Grant. Social Sciences and Humanities Research Council of Canada (SSHRC). 2017-2018. Value: $50,000.

Waldron, I. and Sweeney, E. (2016). "Over the Line: A bilateral conversation about the health effects of race, place and the environment." Nova Scotia Health Research Foundation Knowledge Sharing Support Award. Value: $10,000.

Kim, J.S., Sweeney, E., and Smith, N. (2016). "Toenail Biomarkers of Environmental Arsenic Exposure and Metabolism, and their Relation to Skin cancer, Lung cancer, and Diabetes Mellitus." Seed Funding. Beatrice Hunter Cancer Research Institute. Value: $10,000.

PSY-CA Consortium (Netherlands). (Awarded). "Psychosocial factors and cancer incidence: a pre-planned meta-analysis of the PSYchosocial." Dutch Cancer Society. 2017-2022. Value: 1,327,072 Euros ($1,967,211 CAD).

Provost Dissertation Scholarship (Awarded to ten graduate students at York University). Faculty of Graduate Studies, York University. 2012-2013.Value: $22,000.

National Network on Environments and Women's Health (NNEWH) Conference Costs. Environmental Health Conference: Science and Policy to Protect Future Generations. Boston, Massachusetts. March 2013. Value: $1500.

Canadian Institutes of Health Research (CIHR) Institute of Gender and Health Conference Scholarship. "Advancing Excellence in Gender, Sex and Health Research." Montreal, Quebec. October 2012. Value: $750

Ontario Graduate Scholarship (*Declined*). 2012-2013. Value: $15,000

Ontario Graduate Scholarship. 2011-2012. Value: $15,000

Ontario Graduate Scholarship. 2010-2011. Value: $15,000

National Network on Environments and Women's Health (NNEWH) Registration Costs. Canadian Institutes for Health Research, Institute of Gender and Health Conference - "Innovations in Gender, Sex and Health Research." Toronto, Ontario. November 2010. Value: $200

Canadian Institutes of Health Research, Institute of Gender and Health Summer Institute. July 5-9, 2010. Vancouver, British Columbia. Costs of travel, accommodations and meals.

PhD Funding. Faculty of Environmental Studies, York University. 2009-2010. Value: $22,400.

EcoHealth Forum and International Development Research Centre (IDRC) Travel Award. International EcoHealth Forum. Merida, Mexico. December 2008. Value: $1500

Nova Scotia Health Research Foundation Travel Award. From the Cradle to the Grave: Future Perspectives on the Social History of Health and Healthcare Conference. Glasgow, Scotland. January 2006. Value: $2500

PROFESSIONAL SERVICE

2017-Present	Scientific Review Panel Member, Breast Cancer UK
2017-Present	Affiliate Member, Beatrice Hunter Cancer Research Institute
2016-Present	Board of Directors, Prevent Cancer Now
2016-Present	Editorial Board Member, *Palgrave Communications*
2016-Present	Senior Editorial Board, *Journal for Undergraduate Ethnography*
2016-Present	Working Group Member, Nova Scotia Environmental Rights Working Group
2015-Present	Working Group Member, Environmental Noxiousness, Racial Inequities and Community Health Project (ENRICH)
2015-Present	Advisory Board Member, *Health Tomorrow: Interdisciplinarity and Internationality*
2015-Present	Reviewer, *Health Tomorrow: Interdisciplinarity and Internationality*
2015-2016	Knowledge Translation Mentor, REAL Change Intervention Workshop Curriculum
2015-2016	Guest Editor, *Women & Environments International Magazine* Sustainable Consumption Issue
2013-Present	Scientific Advisory Board Member, Breast Cancer Action Quebec (formerly Breast Cancer Action Montreal)
2013-Present	Advisor, National Network on Environments and Women's Health
2013	National Organizing Committee Member, "Preventing Environmental and Occupational Endocrine Disrupting Chemical Exposures" and Public

	Event: "Hormone Disrupters Unmasked: How Chemical Exposures are Harming Human Health." Workshop. Toronto, Ontario.
2012-2015	Editor, *Health Tomorrow: Interdisciplinarity and Internationality*
2012	Reviewer, Canadian Institutes of Health Research (CIHR) Institute of Gender and Health, Advancing Excellence in Gender, Sex and Health Research Conference
2011	Guest Editor, *Women & Environments International Magazine* Women, Gender and Labour Issue (February-August 2011)
2010-Present	Copy Editor, *Anthropology Matters Journal*, United Kingdom.
2009-2013	Volunteer, Women's Healthy Environments Network (WHEN). Toronto, Ontario.
2008-2009	Advisory Board Member, National Network on Environments and Women's Health (NNEWH).
2008-2009	Steering Committee Member, "The Gendered Effects of Chronic Low Dose Exposures to Chemicals in Canadian Drinking Water." National Network on Environments and Women's Health (NNEWH).
2008-2009	Project Lead and National Organizing Committee Member, "Women and HIV in Canada: The Past, the Present, and the Future – Implications for Research, Policy and Practice." Satellite Session at the 18th Annual Canadian Conference on HIV/AIDS Research (CAHR). Vancouver, British Columbia.
2007	Volunteer, 16th Annual Canadian Conference on HIV/AIDS Research (CAHR). Toronto, Ontario.
2003	Volunteer, Canadian Anthropology Society (CASCA) Conference. Halifax, Nova Scotia.

APPENDIX G-3

"Put It Near the Indians": Indigenous Perspectives on Pulp Mill Contaminants in Their Traditional Territories (Pictou Landing First Nation, Canada)

Heather Castleden, Ella Bennett, Pictou Landing Native Women Group, Diana Lewis, Debbie Martin

Progress in Community Health Partnerships: Research, Education, and Action, Volume 11, Issue 1, Spring 2017, pp. 25-33 (Article)

Published by Johns Hopkins University Press
DOI: https://doi.org/10.1353/cpr.2017.0004

"Put It Near the Indians": Indigenous Perspectives on Pulp Mill Contaminants in Their Traditional Territories (Pictou Landing First Nation, Canada)

Heather Castleden, PhD[1], Ella Bennett, MES[2] , Pictou Landing Native Women's Group[3] , Diana Lewis, PhD (ABD)[2], Debbie Martin, PhD[2]

(1) Queen's University; (2) Dalhousie University; (3) Pictou Landing First Nation

Submitted 20 January 2016, revised 26 July 2016, accepted 27 August 2016.

ABSTRACT

Background: Pictou Landing First Nation (PLFN), a small Mi'kmaw community on the Canadian east coast, has had a relationship with a tidal estuary known as A'se'k for millennia. In the 1960s, it became the site of effluen disposal from a nearby pulp mill. Almost immediately, health concerns regularly and consistently reverberated throughout the community.

Objectives: The Pictou Landing Native Women's Group (PLNWG) formed a community-based participatory research (CBPR) partnership with an academic team to conceptualize community well-being in the context of environment and human health connections. This paper documents Mi'kmaw Elders' stories of A'se'k before it became contaminated.

Methods: Using narrative inquiry vis-à-vis oral histories, we carried out conversational interviews with 10 Elders from PLFN. These interviews were thematically analyzed and 're-storied' through a process of (w)holistic content analysis.

Results: Our findings present four broad story layers, recounting the themes that emerged through analysis and presenting a broad Mi'kmaw narrative of A'se'k. These story layers share: what A'se'k originally provided, the historical/cultural context of PLFN, changes to land and health after the mill was put in, and reflections on the past and future of A'se'k.

Conclusions: Our research offers a novel contribution to the literature by showing how Mi'kmaw perspectives on the pollution at A'se'k reveal the close connection between Mi'kmaw livelihood, local ecologies, and health and well-being. Our research also provides insights into the way the research relationship developed between the PLNWG and the academic team, providing a pathway for others seeking to decolonize the research landscape.

Keywords

Indigenous health, environmental justice, social justice, oral histories, narrative inquiry, environmental health equity, community-based participatory research, Canada, decolonizing research, Two-Eyed Seeing

FROM A'SE'K TO BOAT HARBOUR: SITING POLLUTING INDUSTRIES ON INDIGENOUS LANDS

Mi'kmaw peoples have inhabited the Canadian Maritimes for millennia. It is where Mi'kmaw origin stories begin and where Mi'kmaw peoples continue to raise families, share knowledge of the land, and live in relationship to it through oral traditions.[1] Indigenous relationships to the territory from time immemorial have led to close connections to the environment; healthy connections manifest in the vitality of Indigenous well-being.[2–6] Indigenous cultures, languages, and knowledges flow from the land; health and well-being flo from cultural strength.[7–10] Healthy lands and healthy peoples include spiritual, emotional, mental, and sociocultural connectedness in many Indigenous contexts; these functions have a profound impact on overall community health, well-being, and resilience.[1–12]

For the Mi'kmaw peoples of Pictou Landing First Nation (PLFN), one place—A'se'k—is a culturally significant tidal estuary adjacent to the community (with an on-reserve population of 476[13]). It was known for its highly productive sub-

sistence fisheries, and recreational and medicinal functions. In the late 1700s, soon after the arrival of British settlers, A'se'k became known as "Boat Harbour," a site for ship building. Fift years ago, an effluen (wastewater) treatment facility associated with a nearby bleached Kraft pulp mill was established, releasing daily approximately 85 million liters of effluent into A'se'k.

Like many other Indigenous locales across the colonized Canadian landscape, Boat Harbour is a highly contested and politically charged site. Although Mi'kmaw lands and waters have never been the subject of a land cession treaty or a Crown grant (only Peace and Friendship Treaties were signed in the 1700s), when the mill was proposed, the then-Chief of the PLFN signed an agreement for the treatment facility with the province on the promise that A'se'k would remain clean.[14] Soon after the mill became operational (1967), it became clear that the promise was an empty one. Untreated effluen was dumped directly into Boat Harbour without treatment until later that year, resulting in immediate fish kills and significant social, psychological, and cultural impacts on the community. Since then, a legacy of broken government promises has continued, through unfulfilled commitments to relocate the waste treatment facility and remediate A'se'k. As Mascarenhas observes, "whether by conscious design or institutional neglect, [First Nations] communities face some of the worst environmental devastation in the nation."[15p570]

Over the next 20 and more years, the company's emissions standards fell through regulatory gaps (i.e., 'grandfather' clauses), thus not needing to meet contemporary standards until the 1990s. Although the pulp and paper industry has improved its environmental performance improvements in its lifetime, the enduring impacts on the natural environment are vast and continue to pose risks to human health and well-being.[16] Air emissions from such mills include a number of malodourous sulfur compounds, as well as particulate matter, sulfur oxides, nitrogen oxides, and volatile organic compounds.[17–19] A complex mixture of roughly 300 known chemicals can be found in this type of effluen [20] known to be highly toxic and a major source of pollution.[21–27] The impacts on the local ecology have led to fundamental difference in how the PLFN interacts with the land so much so that Mi'kmaw members no longer gather foods and medicines there. A'se'k/Boat Harbour quickly transformed into a place of fear, anxiety, and unrest.

A NEW PARTNERSHIP FOR COMMUNITY HEALTH AND WELL-BEING

The effluen from the mill has compromised the ecological integrity of the once-healthy tidal estuary[28,29] and, as a result, the PLFN lost the use of A'se'k and surrounding landscape. Community concerns around contaminated land, water, and air extend to unease about the potential negative effects on residents' health. Frustrated with government inaction and wary of government and industry reports that they were out of harm's way, in 2010 the PLNWG, a volunteer group of women from across the community that supports community-based activities, fundraising, charitable work, and other emergent projects, began mobilizing around these concerns. First, the PLNWG invited Lewis, a Mi'kmaw woman from Sipekne'katik First Nation, with graduate-level training in environmental studies to discuss the women's concerns and what could be done about it. With the PLNWG's permission, Lewis then invited Castleden, a community-based participatory settler (non-Indigenous) researcher with more than 15 years of experience working with Indigenous peoples, and Bennett, her settler graduate student, to meet with the PLNWG in the community (a 2-hour drive from the university).

A CBPR partnership began to form over the next 16 months, centered on listening to and learning from each other (and shedding tears too—for the land, the Elders, and future generations). This period of relationship building involved monthly meetings in the community "drinking tea,"[30] and a 3-day retreat of social, cultural, recreational, and intellectual interactions. Having determined the women's priorities, we invited a number of scientists with expertise in GIS, air quality, water quality, soil analysis, and ecotoxicology to join our team, asking them to commit to a CBPR partnership through a signed agreement to honor the PLNWG's protocols for engagement and dissemination. From there, we collaboratively wrote a grant proposal, securing funds to conduct the research and continue our relational process of maintaining good hearts and minds across the cross-cultural research team.[31]

Hinging on the important role Mi'kmaw women have played in protecting and preserving the land, water, and air while acting as guardians for future generations, the initial partnership between the PLNWG and the research team acknowledged the need to conceptualize community health

and well-being in the context of environment and human health connections from Indigenous perspectives. Indigenous knowledge holders represent thousands of years of contact and experience with the local environment[32] and so the PLNWG made it clear early on in our research relationship that they were increasingly concerned with documenting community Elders' stories about what A'se'k was like before the effluen treatment facility began operations. The women were concerned that the decades of colonial disruption to Mi'kmaw ways of life put Mi'kmaw oral traditions at risk and as community Elders were passing, so too, were their stories. Thus, the women prioritized this aspect of our CBPR plan. The research we describe herein is one arm of a multiyear research program using Indigenous and Western science to answer the PLNWG's research question: "Are we getting sick from Boat Harbour?" In addressing this question, our scope is not limited to physical health, but extends to exploring the impacts of Boat Harbour on the mental, emotional, and spiritual well-being of the community as a whole. Gathering Elders' stories about A'se'k helped to build a (w)holistic understanding of how Boat Harbour has changed the community's health and sense of well-being.

METHODS

Our CBPR approach "provide[d] a launch pad for the recognition and inclusion of Indigenous epistemologies and community participation."[33p11] This was of critical importance for moving forward, given the track record of unethical research in many Indigenous contexts.[34] CBPR principles framed our theoretical approach to inquiry as well as the methodological choices made in our research partnership.

Like many who endeavor to carryout CBPR research from the academy,[30,34] we encountered our share of institutional hurdles, but with frequent and ongoing dialogue, our research was approved by the University Research Ethics Board and the Mi'kmaw Ethics Watch,[35] an entity specific to the Mi'kmaw Nation that was established to ensure that research involving Mi'kmaw peoples is done in a culturally appropriate way that safeguards Indigenous knowledge.[36] In keeping with the revised *Tri-Council's Policy Statement: Ethical Conduct for Research Involving Humans,*[37] participants opted to use their real names.

Three advisors from within the PLNWG elected to give Bennett (who collected the data) guidance over the course of the study. Two advisors were Elders, fluent Mi'kmaw speakers, and were included as participants, and—equally important—Bennett had established rapport with all of them. Regular community-based meetings helped to refine the path of inquiry and provide updates to the PLNWG throughout the research process. In particular, the advisors provided suggestions on participant recruitment, which resulted in 10 Elders agreeing to participate (eight of whom were women). The also provided guidance on how to appropriately collect data, they shared community and cultural protocols, and they translated interview data as needed. Beyond the integral research guidance, they welcomed Bennett into their homes during the 5-month period of data collection, had many conversations over meals and tea, and offered friendship and familiarity in an environment to which she was an "outsider."[30,38–40]

Story as Method/Narrative Inquiry

Storytelling is an important means by which much Indigenous knowledge is transmitted through generations. The use of story as a research method honors the process of conversation, dialogue, or narrative exchange to value participants' voices in the research process.[41–46] Narrative inquiry, conversational interviewing, and oral histories have thus been considered appropriate methods in many action research contexts,[47] and they align well with honoring the oral traditions of many Indigenous cultures.[42,48] Stories allow us to understand and explore phenomenon through lived experience[45,49] and illuminate voices that are often excluded from dominant narratives. Doing so can act to challenge established "orthodoxies" and "conventional ways of thinking."[43] The suggestion by Blodgett et al. that "conversational interviews provide the researcher and the participants with flexibility to co-create both *what* is said and *how* things are said during the interviews"[50p524] aligned well with our goals for doing CBPR in a good way.[51]

Similar to general theme areas developed by other researchers,[50,52] the PLNWG–academic research team crafte a conversational oral history guide. Members of the PLNWG suggested that it would be most appropriate to have younger community members, who had formed a relationship with Bennett, introduce her to potential participants. Thus, participants were recruited using purposeful and snowball sampling methods guided by the inclusion criteria set out by members of the PLNWG.

The interviews were unfolding dialogues with participants.

Beyond recollections of A'se'k and changes to the environment and human health, conversations included details of participants' lives, their families, and significant moments in their community's history. Four interviews were one-on-one, five involved another member of the PLNWG and/or a participants' family member for all or part of the interview, and in one instance two participants were interviewed at the same time. All interviews were conducted at participants' homes. Many interview participants used Mi'kmaw words and phrases. The interviewer intentionally avoided disrupting the natural flow and later sought translation (if appropriate) from one of the team's advisors, fluent in the Mi'kmaw language.

The PLNWG wanted the interviews to be video-recorded to create a digital archive according to the OCAP® principles[53] of First Nations' ownership, control, access, and possession of research data (*OCAP® is a registered trademark of the First Nations Information Governance Centre [FNIGC]; see www.FNIGC.ca/OCAP)*. Six participants agreed to video, two opted for audio recorded interviews only, and one chose to interview without recording devices (hand-written notes were taken). Although the interviews ranged from 35 to 75 minutes, the time spent together often exceeded multiple hours. For example, Bennett stayed afterward to visit and in some instances went for walks, shared food, or ceremonially smudged with the smoke of sacred medicines, which is practiced by (some, not all) Mi'kmaw peoples and other Indigenous Nations. Interviews were transcribed and copies were hand-delivered to participants, giving them the opportunity to review and make any corrections to the content.

Interview data were analyzed thematically using a constant comparative method (a process of moving back and forth between the data and themes from existing literature),[1–14] through which a number of emergent themes were identifi d and coded. From there, a holistic content analysis was undertaken, where the function of analysis moves toward retelling the story in an attempt to provide an account of participants' lived experiences.[54–56] Specifically, we threaded together temporal dimensions of each story so that these could be understood in relation to each other, which is particularly well-suited for this type of analysis.[57] Over several months, preliminary findings were vetted with both PLNWG and individual participants during in-person meetings, thus allowing for analytic refinement, an important function of CBPR[30]; moreover, the feedback helped to ground our interpretations in a Mi'kmaw ontology and epistemology. Bennett's thesis, upon which this manuscript is based, was defended openly in front of a community audience in PLFN, not the university—a first for that institution.

RESULTS

The initial process of coding the data served as an important level of analysis, but to present the findings along the lines of discrete themes would risk taking elements of the participants' stories out of context from their place in the broader collective narrative.[58] Instead, vignettes of the following four broad story layers emerged as a basis for a holistic narrative of A'se'k. Pieces of narrative from across the participants were then woven together to reconstruct each story layer. By presenting the findings as stories, our goal is to honor the oral tradition of Mi'kmaw culture by enacting interpretive responsibilities on the audience.[45,46,50]

Story Layer 1—"All Seasons, All Purpose": What A'se'k Provided

It was always known as *A'se'k* before it was called Boat Harbour. It was a recreational place for us, but also our livelihood, a playground, and a work area. There was something to do with every season, like an all-purpose place.

"It was thought of as the other room, where food is stored. Like—nature was storing the food there, 'cause it was there all year round." (Mary Irene Nicholas)

There was a time when most of our food was from there. Every family was hunting, fishing, trapping, and gathering. We ate healthier then. The salmon ran in the streams, and so many smelts we would take home buckets and buckets of them. We would go down with our shovels and buckets and dig up clams, cooking them right there on the shore.

"That was safe haven for all of us. Everything that we needed was there. " (Sadie Francis)

We did lots of berry picking there, and gathered other plants and medicines too. Women would collect mayflo - ers and blueberries—sell them in town for a little extra pin money. Older folks knew about the Indian medicines that came from the woods. Going to A'se'k was like a family outing for us. Sometimes there would be a bunch of families gathered, cooking and eating together right there on the shore.

"We would swim and skate, sometimes make a great big bonfire and we'd skate around. Oh my god, it was beautiful—sometimes it's just the moonlight." (Martha Denny)

Story Layer 2—"A'se'k Was a Refuge": Historical/Cultural Context

We have a connection to these places; our ancestors have occupied this space for thousands of years. The spirit of our people is here. We feel connected to our ancestors in this way. Every time our people ate, it came from the land around us. It is what kept you alive, and it is what kept the people around you alive. When you hunted a deer or a moose, it did not belong to you, it belonged to the community.

"The meat was divided accordingly, nobody was left behind. The men would be up all night carving the meat, and people would come by to pick up their share . . . The people. . . . were looking after the community." (Sadie Francis)

After European contact and after the reserve system and Indian Act were in place, a lot of our men were going down to the States for work, or maybe looking for a better life. Some men could find odd jobs around the area, as labourers mostly. It would help get us by.

"Back then they were mostly trying to survive. I watched my dad working so hard to get so little." (Don Francis)

And our kids were being taken away to the Indian Residential School. The Indian Agents would come down to our community, and just take them. But A'se'k, that was like a refuge, a safe place for all of us that they would not venture out to.

When they decided to dump that effluen into A'se'k, everything was supposed to stay 'ok'. We had no reason to assume otherwise, until we learned of the White man's way—*aklasie'wey*. Some people had come down and talked to Chief and Council, duped them into signing that agreement. Some crooked people. Dishonest people. But that is how the Indian Affairs and the non-Native society has been. Their main goal was to get rid of the Indians. It has always been about the almighty dollar for them.

"Well, I guess they didn't want to put it anywhere else in town. Let's put it near the Indians—Native people close by, we'll dump it on them! . . . Let them deal with it. But it's always us that got dumped on. That's how they treated us I guess." (Mary Ellen Denny)

Story Layer 3—"After the Mill Went in . . . ": Changes to Land and Health

At first, there was nothing to it really, just a mill. But then we saw all the fish dying. The rabbits and the deer—they seemed to disappear. And if we did hunt one, they had strange lumps. All those swampy areas that we used to get our cranberries, all that is under water now, and we do not even know if our medicines are good anymore.

"Our air is polluted, our water is polluted, our land is polluted . . . And they're all connected." (Diane Denny)

The pollution is not just in the water, it is in the air too. Sometimes that stink can be so bad we cannot even sit outside. In the beginning it turned our houses black. We found out it was the sulphur drawing the lead out of the paint, so they gave us money to repaint our houses. What is it doing to us? It is everywhere, there is no getting away from it.

"We had to change our diet. The things that we were accustomed to for thousands and thousands of years, those were all of a sudden not available to us anymore. We had to resort to another way of life. And now we have people that have diabetes, heart disease." (Sadie Francis)

When the land went, so did our health. It is not just the rabbits getting those lumps, it is our people now too. Skin cysts and cancer. A lot of kids have breathing problems, asthma, nosebleeds, sinus headaches, and it is like that stink does something to your nose. There seems to be a lot more cancer. Growing up, we never knew what cancer was but all of a sudden there are so many different kinds of cancer down here. Looking at all these health problems, we cannot help but wonder if that pollution is the reason.

Story Layer 4—"Lost, Gone, and Changed": Looking Back, Looking Ahead

It is too bad what happened there; it was such a beautiful place. So now nobody goes down there to hunt or trap, get eels or smelts, snare rabbits or fish. There is no place for kids to walk along the shore, or swim in the summertime. Nothing grows there or lives there anymore, and if it did—we would not trust it. Our community has lost their trust in that food, and our connection seems to have suffered too.

Food from the land was the way it was before Europeans arrived, but in the last couple hundred years it was also a safety net. A'se'k was something our people could fall back on. When the groceries were running low, we always knew

we could get food from there. But when the pollution came, we did not even have that anymore. And now the young people, our youth, they are not out there learning in the woods from their Elders.

Everything is gone for us there, and it is like we are getting poorer while that mill is getting richer. We wonder what could have been . . . just think of the beautiful things we could have done down there. And it is not just us that lost out. Everybody all around here lost out, the non-Native community too. And there has been anger. Th re has been blame. It has divided families, divided our community.

"Everything we used to do, we can't do. What we were brought up on, it's all been taken away." (Don Francis)

We had something good and sacred here. But our stories are slowly being lost in the older generations. We need to tell the younger generations, share our stories and share our knowledge, so that the memory of a clean A'se'k can be preserved. History is not meant to be kept in a closet; it's not doing any good there. It is meant to be shared.

"I had a dream once. I dreamt it was clean, and our community became rich from it. And everybody worked together, in my dream." (Louise Sapier)

DISCUSSION

Substantively, our research offers Mi'kmaw perspectives on the highly contentious "Boat Harbour issue" from Elders who have lived through the loss of A'se'k. For Indigenous peoples, the ability to practice subsistence livelihoods facilitates a connection to the local environment. Many of the Elders discussed community sharing practices, which were an important cultural function in PLFN before the pollution at A'se'k. Such practices are foundational to Mi'kmaw values, upholding kinship ties and connections with Mi'kmaw knowledge systems.[14,59–61] PLNWG members consequently spoke of the links between the pollution at A'se'k and the dramatic decrease of individual and community engagement with the land and waterways. Although sharing continues to function in a contemporary context in PLFN and some members continue to engage in traditional harvesting activities (albeit usually far from their contaminated homeland), it is overwhelmingly clear from the Elders in this study that trust in food and medicines from the land has been compromised radically. The Elders suggested that a lack of engagement with the land is especially true for the younger generation, many of whom do not hunt or fish, or know what foods and medicines are available on Mi'kmaw lands because they do not have a place to engage in these activities. Most important, our data indicate that Boat Harbour is indeed making the community "sick," by highlighting the ways PLFN's loss of A'se'k and surrounding land use has compromised the community's physical, sociocultural, emotional, and spiritual health and well-being.

Methodologically, the relationship building between the PLNWG and the academic members of the team offers an example of working toward CBPR's goals of balancing power and fostering trust between research partners, and also aligns with the calls made by Indigenous scholars, leaders, and community members to further decolonize the research landscape. Our unstructured oral history interview style facilitated a co-creation of knowledge; participants were able to guide the research conversations, and "re-storying" the data together maintained a holistic representation by drawing entirely from the narratives of participants for collective story layers. Th oral history interviewing process also enabled intergenerational knowledge sharing in that younger family members of participants were often present. Finally, although researchers typically destroy data after 5 to 7 years, the recorded interviews that were conducted with these Elders will remain in the hands of the PLNWG, allowing future generations to have the opportunity to engage with their Elders' memories and perspectives. Already, the stories from the Elders and other aspects of the larger research project are providing important teaching resources for PLFN, grounded in (and helping to revive) Mi'kmaw concepts and connections to their lands.

Although our findings reflect the voices of only some PLFN Elders, it is important to recognize that there are other Elders and Indigenous knowledge holders who hold important cultural relationships with A'se'k. This research is, therefore, "one move in a continuing dialogue"[62] and with community researchers continuing to document Elders' oral histories, the story of A'se'k for PLFN will continue to evolve and be shared. As for our partnership, it is now going on 6 years, and we hold annual PLNWG retreats to reflect on where we have come from and establish where we are going next (see www.heclab.com for updates).

Concerning issues of voice and representation in research, the nature of CBPR, and whether it is an inherently emancipatory research tool,[63–65] we note research is not about *giving* voice to participants; they have always, and will always, have a voice. Moreover, as scholars Vannini and Gladue note, "as

researchers, we are not writing to give voice to theoretical ideas hidden in the cracks of "reality" or even to give voice to the marginalized. Rather, we are writing to begin to share Mi'kmaw voices. With this in mind, cross cultural research must begin with the people and end with the people and everything in between."[66p157] Our research partnership addresses the penchant for dismissing Indigenous voices by honoring and illuminating the voices of Elders from PLFN by reconstructing stories about A'se'k from those who remember it as a healthy, thriving, culturally significant place. Since the efflue began flowing, the area has become a source of environmental contamination and serious health concerns that are difficul to measure using Western methodologies alone, but as this study concludes, not only has it negatively impacted the environment, but Boat Harbour has also compromised Mi'kmaw sociocultural, emotional, and spiritual health and well-being.

CONCLUDING COMMENTS

The story has not ended. In 2015, after significant pressure from PLFN and constituents in the county (and perhaps awareness that the results of this study were forthcoming), the provincial government legislated to stop the flow of effluen and remediate the site by 2020. Moreover, in referring to the manner in which the mill was sited 50 years ago as an "injustice," the Provincial Court of Nova Scotia sentenced the Northern Pulp Nova Scotia Corporation (current owner of the mill) with a $225,000 CAD fine in May 2016 for a 2014 offenc concerning 47 million liters of effluen escape; $75,000 CAD of this award went to PLFN for purposes related to conservation and restoration. Precisely how the site will be remediated, and whether it will be remediated according to Mi'kmaw values and PLFN's vision or simply to current Western standards, remains to be seen.

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Endocrine-Disrupting Chemicals: An Endocrine Society Scientific Statement

Evanthia Diamanti-Kandarakis, Jean-Pierre Bourguignon, Linda C. Giudice, Russ Hauser, Gail S. Prins, Ana M. Soto, R. Thomas Zoeller, and Andrea C. Gore

Endocrine Section of First Department of Medicine (E.D.-K.), Laiko Hospital, Medical School University of Athens, 11527 Athens, Greece; Department of Pediatrics (J.-P.B.), Centre Hospitalier Universitaire de Liege, 4000 Liege, Belgium; Department of Obstetrics, Gynecology, and Reproductive Sciences (L.C.G.), University of California San Francisco, San Francisco, California 94131; Department of Environmental Health (R.H.), Harvard School of Public Health, Boston, Massachusetts 02115; Department of Urology (G.S.P.), University of Illinois at Chicago, Chicago, Illinois 60612; Department of Anatomy and Cell Biology (A.M.S.), Tufts University School of Medicine, Boston, Massachusetts 02111; Biology Department (R.T.Z.), University of Massachusetts, Amherst, Massachusetts 01003; and Division of Pharmacology and Toxicology (A.C.G.), The University of Texas at Austin, Austin, Texas 78712

There is growing interest in the possible health threat posed by endocrine-disrupting chemicals (EDCs), which are substances in our environment, food, and consumer products that interfere with hormone biosynthesis, metabolism, or action resulting in a deviation from normal homeostatic control or reproduction. In this first Scientific Statement of The Endocrine Society, we present the evidence that endocrine disruptors have effects on male and female reproduction, breast development and cancer, prostate cancer, neuroendocrinology, thyroid, metabolism and obesity, and cardiovascular endocrinology. Results from animal models, human clinical observations, and epidemiological studies converge to implicate EDCs as a significant concern to public health. The mechanisms of EDCs involve divergent pathways including (but not limited to) estrogenic, antiandrogenic, thyroid, peroxisome proliferator-activated receptor γ, retinoid, and actions through other nuclear receptors; steroidogenic enzymes; neurotransmitter receptors and systems; and many other pathways that are highly conserved in wildlife and humans, and which can be modeled in laboratory *in vitro* and *in vivo* models. Furthermore, EDCs represent a broad class of molecules such as organochlorinated pesticides and industrial chemicals, plastics and plasticizers, fuels, and many other chemicals that are present in the environment or are in widespread use. We make a number of recommendations to increase understanding of effects of EDCs, including enhancing increased basic and clinical research, invoking the precautionary principle, and advocating involvement of individual and scientific society stakeholders in communicating and implementing changes in public policy and awareness. **(*Endocrine Reviews* 30: 293–342, 2009)**

ISSN Print 0021-972X ISSN Online 1945-7197
Printed in U.S.A.

doi: 10.1210/er.2009-0002 Received February 2, 2009. Accepted April 17, 2009.

Abbreviations: AGE, Advanced glycation end-product; AhR, aryl hydrocarbon receptor; AR, androgen receptor; BPA, bisphenol A; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; DES, diethylstilbestrol; DMBA, dimethylbenzanthracene; EDC, endocrine-disrupting compound; ER, estrogen receptor; HPA, hypothalamic-pituitary-adrenal axis; HPG, hypothalamic-pituitary-gonadal axis; HPT, hypothalamic-pituitary-thyroid axis; IUGR, intrauterine growth retardation; IVF, *in vitro* fertilization; MBP, monobutyl phthalate; MBzP, monobenzyl phthalate; NIS, sodium/iodide symporter; PBB, polybrominated biphenyl; PBDE, polybrominated diphenyl ether; PCB, polychlorinated biphenyl; PCOS, polycystic ovarian syndrome; POF, premature ovarian failure; PPARγ, peroxisome proliferator-activated receptor γ; PTU, 6-propyl-2-thiouracil; RXR, retinoic X receptor; TBBPA, tetrabromobisphenol-A; TBG, T_4-binding globulin; TBT, tributyltin; TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin; TDS, testicular dysgenesis syndrome; TGCC, testicular germ cell cancer; TPO, thyroperoxidase; TR, thyroid receptor; TRE, thyroid response element; TTR, transthyretin.

I. General Introduction to Endocrine Disruption

An endocrine-disrupting compound was defined by the U.S. Environmental Protection Agency (EPA) as "an exogenous agent that interferes with synthesis, secretion, transport, metabolism, binding action, or elimination of natural blood-borne hormones that are present in the body and are responsible for homeostasis, reproduction, and developmental process." Our understanding of the mechanisms by which endocrine disruptors exert their effect has grown. Endocrine-disrupting chemicals (EDCs) were originally thought to exert actions primarily through nuclear hormone receptors, including estrogen receptors (ERs), androgen receptors (ARs), progesterone receptors, thyroid receptors (TRs), and retinoid receptors, among others. Today, basic scientific research shows that the mechanisms are much broader than originally recognized. Thus, endocrine disruptors act via nuclear receptors, nonnuclear steroid hormone receptors (*e.g.*, membrane ERs), nonsteroid receptors (*e.g.*, neurotransmitter receptors such as the serotonin receptor, dopamine receptor, norepinephrine receptor), orphan receptors [*e.g.*, aryl hydrocarbon receptor (AhR)—an orphan receptor], enzymatic pathways involved in steroid biosynthesis and/or metabolism, and numerous other mechanisms that converge upon endocrine and reproductive systems. Thus, from a physiological perspective, an endocrine-disrupting substance is a compound, either natural or synthetic, which, through environmental or inappropriate developmental exposures, alters the hormonal and homeostatic systems that enable the organism to communicate with and respond to its environment.

The group of molecules identified as endocrine disruptors is highly heterogeneous and includes synthetic chemicals used as industrial solvents/lubricants and their byproducts [polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), dioxins], plastics [bisphenol A (BPA)], plasticizers (phthalates), pesticides [methoxychlor, chlorpyrifos, dichlorodiphenyltrichloroethane (DDT)], fungicides (vinclozolin), and pharmaceutical agents [diethylstilbestrol (DES)].

Natural chemicals found in human and animal food (*e.g.*, phytoestrogens, including genistein and coumestrol) can also act as endocrine disruptors. These substances, whereas generally thought to have relatively low binding affinity to ERs, are widely consumed and are components of infant formula (1, 2). A recent study reported that urinary concentrations of the phytoestrogens genistein and daidzein were about 500-fold higher in infants fed soy formula compared with those fed cow's milk formula (3). Therefore, the potential for endocrine disruption by phytoestrogens needs to be considered.

A challenge to the field of endocrine disruption is that these substances are diverse and may not appear to share any structural similarity other than usually being small molecular mass (<1000 Daltons) compounds. Thus, it is difficult to predict whether a compound may or may not exert endocrine-disrupting actions. Nevertheless, in very broad terms, EDCs such as dioxins, PCBs, PBBs, and pesticides often contain halogen group substitutions by chlorine and bromine. They often have a phenolic moiety that is thought to mimic natural steroid hormones and enable EDCs to interact with steroid hormone receptors as analogs or antagonists. Even heavy metals and metalloids may have estrogenic activity, suggesting that these compounds are EDCs as well as more generalized toxicants. Several classes of EDCs act as antiandrogens and as thyroid hormone receptor agonists or antagonists, and more recently, androgenic EDCs have been identified.

The sources of exposure to EDCs are diverse and vary widely around the world. The situation is constantly evolving because some EDCs were banned decades ago and others more recently, with significant differences between countries. In this respect, migrating people provide a model to study cessation and/or onset of exposure depending on contamination of the original and new milieus. There are also several historical examples of toxic spills or contamination from PCBs and dioxins that show a direct causal relationship between a chemical and the manifestation of an endocrine or reproductive dysfunction in humans and wildlife. However, these types of single exposures are not representative of more common widespread persistent exposure to a broad mix of indoor and outdoor chemicals and contaminants. Industrialized areas are typically characterized by contamination from a wide range of industrial chemicals that may leach into soil and groundwater. These complex mixtures enter the food chain and accumulate in animals higher up the food chain such as humans, American bald eagles, polar bears, and other predatory animals. Exposure occurs through drinking contaminated water, breathing contaminated air, ingesting food, or contacting contaminated soil. People who work with pesticides, fungicides, and industrial chemicals are at particularly high risk for exposure and thus for developing a reproductive or endocrine abnormality.

Some EDCs were designed to have long half-lives; this was beneficial for their industrial use, but it has turned out to be quite detrimental to wildlife and humans. Because these substances do not decay easily, they may not be metabolized, or they may be metabolized or broken down into more toxic compounds than the parent molecule; even substances that were banned decades ago remain in high levels in the environment, and they can be detected as part of the body burden of virtually every tested individual animal or human (4, 5). In fact, some endocrine disruptors are detectable in so-called "pristine" environments at remote distances from the site they were produced, used, or released due to water and air currents and via migratory animals that spend part of their life in a contaminated area, to become incorporated into the food chain in an otherwise uncontaminated region. Others, such as BPA, may not be as persistent [although recent evidence (*e.g.*, Ref. 6) suggests longer half-lives) but are so widespread in their use that there is prevalent human exposure.

A. Important issues in endocrine disruption

A number of issues have proven to be key to a full understanding of mechanisms of action and consequences of exposure to EDCs. These have been reviewed previously in detail (7), and several of them are listed here in brief.

1. Age at exposure

Exposure of an adult to an EDC may have very different consequences from exposure to a developing fetus or infant. In fact, the field of endocrine disruption has embraced the terminology "the fetal basis of adult disease" (8) to describe observations that the environment of a developing organism, which includes the maternal environment (eutherian mammals), the egg (other vertebrates), and the external environment, interacts with the individual's genes to determine the propensity of that individual to develop a disease or dysfunction later in life. In this Scientific Statement, we extend this concept beyond the fetal period to the early postnatal developmental period when organs continue to undergo substantial development. Thus, we will henceforward use the terminology "the developmental basis of adult disease."

2. Latency from exposure

The developmental basis of adult disease also has implicit in its name the concept that there is a lag between the time of exposure and the manifestation of a disorder. In other words, consequences of developmental exposure may not be immediately apparent early in life but may be manifested in adulthood or during aging.

3. Importance of mixtures

If individuals and populations are exposed to an EDC, it is likely that other environmental pollutants are involved because contamination of environments is rarely due to a single compound. Furthermore, effects of different classes of EDCs may be additive or even synergistic (9).

4. Nontraditional dose-response dynamics

There are several properties of EDCs that have caused controversy. First, even infinitesimally low levels of exposure—indeed, any level of exposure at all—may cause endocrine or reproductive abnormalities, particularly if exposure occurs during a critical developmental window (10). Surprisingly, low doses may even exert more potent effects than higher doses. Second, EDCs may exert nontraditional dose-response curves, such as inverted-U or U-shaped curves (11). Both of these concepts have been known for hormone and neurotransmitter actions, but only in the past decade have they begun to be appreciated for EDCs.

5. Transgenerational, epigenetic effects

EDCs may affect not only the exposed individual but also the children and subsequent generations. Recent evidence suggests that the mechanism of transmission may in some cases involve the germline (12) and may be nongenomic. That is, effects may be transmitted not due to mutation of the DNA sequence, but rather through modifications to factors that regulate gene expression such as DNA methylation and histone acetylation.

B. The role of endocrinologists in discerning effects of EDCs

The field of endocrine disruption has particular pertinence to endocrinologists. In general, persistent endocrine disruptors have low water solubility and extremely high lipid solubility, leading to their bioaccumulation in adipose tissue. The properties of these substances are particularly well suited for study by endocrinologists because they so often activate or antagonize hormone receptors. There is no endocrine system that is immune to these substances, because of the shared properties of the chemicals and the similarities of the receptors (13) and enzymes involved in the synthesis, release, and degradation of hormones (Fig 1). Therefore, the role of this Scientific Statement is to provide perspectives on representative outcomes of exposures to endocrine disruptors and evidence for their effects in wildlife, laboratory animals, and humans.

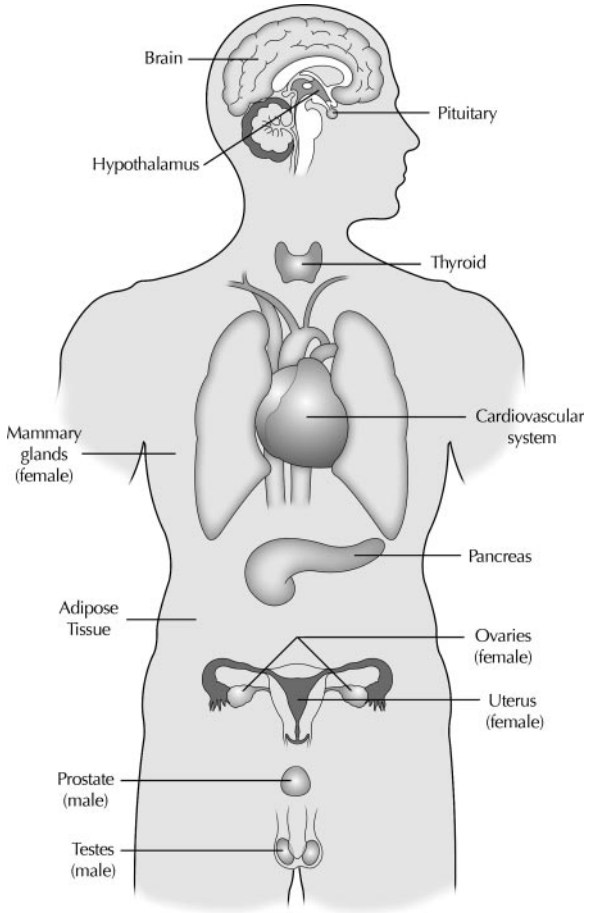

FIG. 1. Model of the endocrine systems targeted by endocrine-disrupting chemicals as discussed in this article. This figure demonstrates that all hormone-sensitive physiological systems are vulnerable to EDCs, including brain and hypothalamic neuroendocrine systems; pituitary; thyroid; cardiovascular system; mammary gland; adipose tissue; pancreas; ovary and uterus in females; and testes and prostate in males.

II. Overview of Endocrine Disruption and Reproductive Health from a Clinical Perspective

A. Clinical aspects of endocrine disruption in humans

For a clinician taking care of an individual patient, there are numerous challenges in ascertaining EDC involvement in a particular disorder. Each person has unique exposure to a variety of both known and unknown EDCs. Individual differences in metabolism and body composition will create considerable variability in the half-life and persistence of EDCs, as well as their degradation in body fluids and tissues. Susceptibility to EDCs may vary according to genetic polymorphisms. In addition, human disorders are more likely the result of chronic exposure to low amounts of mixtures of EDCs. The latency between exposure to EDCs and occurrence of clinical disorders creates further challenges when one attempts to establish a relationship at the level of a given individual.

Epidemiological studies at the level of populations in a country or a region are crucial to alert researchers about geographical or secular trends in prevalence of disorders pointing to possible environmental factors. Registries with data on particular diseases or cell/organ donors may provide valuable contributions. For instance, the observation of adverse trends in male reproductive health together with declining sperm count in Denmark and other countries has led to the hypothesis of environmental con-

taminants being harmful to reproduction (14). Unfortunately, it is virtually impossible to make direct links between such epidemiological observations and exposure to given chemicals. Regional differences in certain reproductive disorders (infertility, cancer) that may be tied to contamination by compounds used locally such as in agriculture, industrial accident, or product misuse/abuse in subpopulations can also be informative (14, 15). Finally, a comparison of disorders before and after migration to a new environment may reveal exposure and/or susceptibility to exposure to EDCs (16).

As already mentioned, a critical concern is the potential lag between exposure to EDCs and the manifestation of a clinical disorder. In humans, this period may be years or decades. In the case of reproduction, infertility cannot be assessed until the exposed individual has attained a certain age, again resulting in a lag between early exposure and manifestation of a dysfunction. Delayed or early puberty cannot be assessed until this event actually takes place, although timing of puberty could involve programming many years earlier during fetal life. Interestingly, an increased likelihood of early puberty was observed in subjects born with intrauterine growth retardation (IUGR) (17, 18), suggesting a link between developmental programming and reproductive maturation. As discussed below, development of vaginal adenocarcinoma in women exposed fetally to DES (19) and the association of carcinoma *in situ* in the fetal testis with the development of testicular cancer in adulthood (14, 20) are examples of links between the fetal environment and the occurrence of adult disease.

The timing of exposure is key to human disease because there are critical developmental periods during which there may be increased susceptibility to environmental endocrine disruptors. In those cases in which disruption is directed toward programming of a function, *e.g.*, reproductive health, this may interfere with early life organization, followed by a latent period, after which the function becomes activated and the dysfunction can become obvious. For reproductive function in both humans and animals, fetal life is most vulnerable because there are rapid structural and functional events. The roles of sex steroids in sexual differentiation and thyroid hormones in brain development are of paramount importance at that time. Early postnatal life is also a time when maturation is still rapid (*e.g.*, the central nervous system undergoes significant development at this time, including the hypothalamus which controls reproduction; see *Section VII*). The organization of the neuroendocrine control of reproduction is not completed at birth and remains sensitive to the interaction of steroids or EDCs neonatally such as has been shown for the control of ovulation in rodents. Breast or formula feeding could be of particular significance due to the capacity of human milk to concentrate EDCs in the former and the potential high intake of phytoestrogens in soy milk and/or plasticizers in formula-containing cans in the latter. It is apparent that the developmental basis of adult disease is an important concept for understanding endocrine disruption of reproductive function in humans.

B. Clinical dimorphism of EDCs on male and female reproduction

A spectrum of disorders throughout life, some of which are sexually dimorphic, can be related to endocrine disruption (Table 1). Male sexual differentiation is androgen-dependent (and potentially estrogen-dependent), whereas female differentiation occurs largely independently of estrogens and androgens. Therefore, it is expected that different dis-

TABLE 1. Disorders of the human reproductive system possibly involving EDCs in their pathogenesis: A sexually dimorphic life cycle perspective

	Fetal/neonatal	Prepubertal	Pubertal	Adult
Processes	Intrauterine growth Sexual differentiation	Adrenarche	Gonadarche	Spermatogenesis Ovulation Hormonal control of prostate, breast, uterus, and lactation
Male disorders	IUGR (15) Cryptorchidism (14, 20)[a] Hypospadias (14, 20)[a]	Premature pubarche	Small testes and high FSH (18) Early puberty (25) Delayed puberty (25)	Oligospermia (14, 20)[a] Testicular cancer (14, 20)[a] Prostate hyperplasia (24)
Female disorders	IUGR	Premature thelarche (25) Peripheral precocious puberty (17) Premature pubarche (18)	Secondary central precocious puberty (17, 27) PCOS (18, 25) Delayed ovulatory cycles (17, 18)	Vaginal adenocarcinoma (19, 28) Disorders of ovulation (29) Benign breast disease (29, 31) Breast cancer (30, 31) Uterine fibroids (29) Disturbed lactation (29)

[a] Cryptorchidism, hypospadias, oligospermia and testicular cancer are four components of the "testicular dysgenesis syndrome" as a common entity.

orders are seen in males and females as a result of EDC effects that overall mimic estrogens and/or antagonize androgens.

In the male (Table 2), cryptorchidism, hypospadias, oligospermia, and testicular cancer have been proposed to be linked as the testicular dysgenesis syndrome (TDS) arising from disturbed prenatal testicular development (14, 21). Such links are important because they could mean that several disorders occur at different periods throughout life in a single individual as a result of exposure to a given EDC (or mixture) at a particular period. The epidemiological data relating TDS with environmental disruptors are indirect, and we still lack direct evidence of EDC involvement in the pathogenesis of TDS in humans (see *Section V*). In the rodent, however, a TDS-like condition can be observed after fetal exposure to phthalates (20), and the reduced anogenital distance observed in the rat (22) was observed in a recent epidemiological study on human male newborns (23). Several studies have shown a strong association of low birth weight with hypospadias and cryptorchidism, suggesting that they have a common determinant (15).

Other pathologies in males are linked to EDC exposure. Prostate hyperplasia has been described after exposure to BPA (24). In adolescence, boys born with IUGR have small testes and elevated serum FSH, together with low inhibin B levels (18) that could be related to some of the TDS disorders. Divergent data have been reported on effects of EDCs on pubertal timing in the male (25).

In the female (Table 3), premature thelarche has been reported in girls exposed to phthalates (26), although these data need to be replicated. Sexual precocity presumably of peripheral origin initially and secondarily central could be related to exposure to the insecticide DDT in girls migrating for international adoption (17). A neuroendocrine mechanism is suggested by experiments in a rodent model (27) (see *Section VII*). An association of premature pubarche and ovulatory disorders with EDCs is suggested indirectly by links with IUGR at birth and metabolic syndrome in adulthood (18).

In the adult female, the first evidence of endocrine disruption was provided almost 40 yr ago through observations of uncommon vaginal adenocarcinoma in daughters born 15–22 yr earlier to women treated with the potent synthetic estrogen DES during pregnancy (19). Subsequently, DES effects and mechanisms have been substantiated in animal models (28). Thus, robust clinical observations together with experimental data support the causal role of DES in female reproductive disorders. However, the link between disorders such as premature pubarche and EDCs is so far indirect and weak, based on epidemiological association with both IUGR and ovulatory disorders. The implications of EDCs have been proposed in other disorders of the female reproductive system, including disorders of ovulation and lactation, benign breast disease, breast cancer, endometriosis, and uterine fibroids (29–32).

C. Experimental and clinical evidence of EDCs and potential mechanisms

In Tables 2 and 3, some experimental and clinical observations of disturbed reproductive systems are listed for

TABLE 2. Effects of some specific EDCs on the male reproductive system

EDC	Exposed animal and effects	Possible translation to the clinical condition	Potential mechanisms
Vinclozolin	Fetal rat: hypospadias (36); undescended testes, prepubertal (37); delayed puberty (38), prostate disease among subsequent generations (34)		Epigenetic: altered DNA methylation in germ cell lines (12, 34)
DES	Fetal rats: hypospadias, cryptorchidism, micropenis, increased transmitted susceptibility to malignancies (28)	Hypospadias, cryptorchidism, micropenis, epididymal cysts (28)	Increased ERα expression in epididymis (43) Reduced insulin-like factor 3 (465)
DDT	Adult rats: decreased fertility (466)	Cryptorchidism	
DDE		Cryptorchidism	
Phthalates	Reduced anogenital distance (22)	Reduced anogenital distance (23) and Leydig cell function, hypospadias	Decreased testosterone synthesis (468)
	Cryptorchidism (467)	Cryptorchidism (14, 20)	
	Oligospermia	Reduced fertility (14, 20)	
PCBs	Fetal rat: decreased spermatogenesis, delayed puberty	Reduced penile length, delayed sexual maturation, reduced fertility Fetal: testis cancer	
BPA	Increased prostate size (469) Aberrant development of prostate and urethra (470) Prostate cancer (122) Increased anogenital distanceAltered periductal stroma in the prostate (471)		Increased ERα expression in hypothalamus (42) Increased AR expression in prostate (469)

TABLE 3. Effects of some specific EDCs on the female reproductive system

EDC	Exposed animal and effects	Possible translation to the clinical condition	Potential mechanisms
Vinclozolin	Fetal rat: multisystem disorders including tumors (12)		Epigenetic: altered DNA methylation in germ cell line (12); reduced ERα expression in uterus (44)
DES	Fetal mouse: transmitted susceptibility to malignancies (39)	Vaginal adenocarcinoma in daughters of women treated with DES during pregnancy (19)	
DDT/DDE	Immature female rat: sexual precocity (27)	Precocious and early puberty (17) Reduced fertility in daughters of exposed women (472) <15 yr: increased breast cancer risk	Neuroendocrine effect through estrogen receptors, kainate receptors, and AhRs (27)
BPA	Inhibited mammary duct development and increased branching (145) Increased mammary gland density, increased number of terminal ends (146) Reduced weight of vagina (473) Endometrial stimulation (473) Early puberty (474, 475)	Miscarriages	Inhibition of apoptotic activity in breast (145) Increased number of progesterone receptor-positive epithelial cells Reduced sulfotransferase inactivation of estradiol (45, 46) Nongenomic activation of ERK1/2 (476)
PCBs	Fetal and early postnatal rat: neuroendocrine effects in two generations, and behavioral changes (296, 477)		Actions on estrogen receptors, neurotransmitter receptors
Dioxins	Fetal rat: altered breast development and increased susceptibility for mammary cancer (478) Early pubertal rat: blocked ovulation		Inhibition of cyclooxygenase2 via AhR (479)
Phthalates		Premature thelarche (25)	

selected EDCs. The evidence from human epidemiological studies is partial and indirect (see *Section V*). Mechanistic studies are ethically and practically very limited in humans and have to rely on data obtained using animal experiments (*in vivo* and *in vitro* models), although these models can have limitations. Clinical and experimental studies correlate DES effects quite convincingly in both sexes. In the male, rodent studies using phthalates and, to a lesser extent, PCBs model TDS entirely or partly. In the female, some rodent studies are consistent with DDT/dichlorodiphenyldichloroethylene (DDE) involvement in sexual precocity.

The following considerations emphasize some of the concepts emerging from the available data.

1. Heritability

There may be transgenerational effects of EDCs due to overt mutation or to more subtle modifications of gene expression independent of mutation (*i.e.*, epigenetic effects). Epigenetic effects of EDCs include context-dependent transmission (*e.g.*, the causal factor persists across generations; Ref. 33) or germline-dependent mechanisms (*i.e.*, the germline itself is affected; Refs. 12, 34, and 35). An example of germline transmission of an epigenetically modified trait is shown in a rat model for the fungicide vinclozolin and is manifested by a higher likelihood of metabolic disorders, tumors, and reproductive dysfunctions in the next four generations (12, 34–38). In the case of DES, there are both human and experimental observations indicating heritability (19, 28, 39).

2. Diversity and complexity of mechanisms

EDCs often act via more than one mechanism. Some EDCs have mixed steroidal properties: for example, a single EDC may be both estrogenic and antiandrogenic. EDCs may be broken down or metabolized to generate subproducts with different properties. For instance, the estrogen agonist DDT is metabolized into the androgen antagonist DDE (27). The balance between estrogenic and androgenic properties of EDCs can be biologically significant because reproduction of both sexes involves an interplay of androgens and estrogens. In humans, early breast development occurs in girls with a highly active variant of CYP3A4, a cytochrome p450 enzyme involved in inactivating testosterone (40), and premature thelarche occurs with antiandrogenic phthalates (25). Similar androgen-estrogen interactions have been reported in DES-treated rats in which reduced androgen secretion or action sensitized the animals to the estrogenic effects of DES (41). Moreover, many organs are targeted by sex steroids and are thereby vulnerable to endocrine disruption, including the hypothalamic-pituitary-gonadal system, breast, uterus, cervix, vagina, brain, and nonreproductive tissues

such as bone, muscle, and skin (Fig. 1). In the case of humans, a peripheral effect in the reproductive system (*e.g.*, breast development) can result from direct EDC effects (peripheral puberty) and/or endogenous estrogen increase through premature neuroendocrine maturation (central puberty) (17, 27), but these may be difficult to distinguish. For instance, EDC effects can involve altered ERα expression in hypothalamus (42) and epididymis (43) or uterus (44). Along with the direct influence of EDCs on estrogen or androgen actions, they can affect endogenous steroid production through negative and positive feedback, effects that may differ depending on developmental stage. Also, there are multiple levels of interactions with steroid action (receptor or promoters), synthesis (*e.g.*, aromatase stimulation by atrazine), and metabolism [*e.g.*, sulfotransferase (45)]. Finally, there are coexisting mechanisms not directly mediated at the hypothalamic-pituitary-gonadal (HPG) system. For instance, reproductive dysfunction can result from thyroid disruption (46) or nonspecific interference of reduced energy intake (47).

3. Limits of translational models

The *in vivo* animal models may be difficult to extrapolate to humans for several reasons, including species differences in ontogeny of reproductive system and functions, differences in metabolism of sex steroids, difficulty in estimating exposure to mixtures, and variable body burdens. As already mentioned, exposure to EDCs is complex. For example, mixtures are likely to be the usual form of exposure to EDCs, but they are difficult to approximate in experimental models. Moreover, the effects may not be additive; nevertheless, a combination of low doses of substances that individually are inactive may result in a biological perturbation (48). Despite these limitations, considering the substantial conservation of endocrine and reproductive processes across species, it is certainly reasonable to use animal models for understanding human processes, as long as these potential differences are taken into account.

III. Clinical and Translational Impacts of EDCs on Female Reproduction

A. Introduction to female reproductive development and function

Development and function of the female reproductive tract depends on coordinated biological processes that, if altered by endogenous or exogenous factors during critical periods of development or during different life stage, could have significantly adverse effects on women's health and reproductive function and outcomes. For example, the full complement of cell types in the human ovary depends on successful germ cell migration from the yolk sac during the first trimester and differentiation into oocytes with associated somatic cells to form the functional unit of the primordial follicle by the second to third trimesters of gestation. Factors that interfere with germ cell migration or follicle formation can result in abnormal functioning of this tissue with significant reproductive consequences. Also, the oocyte is arrested in the diplotene stage of late prophase until meitic divisions occur beginning at puberty (meiosis I) and after fertilization (meiosis II), and abnormalities in these processes can have a profound impact on reproductive outcomes, such as aneuploidy, premature ovarian failure (POF), and miscarriage. In addition, whereas Mullerian tract formation begins at 8 wk gestation with fusion of the Mullerian ducts and subsequent differentiation into the uterus (endometrium, myometrium), cervix, and upper vagina, uterine differentiation with regard to formation of luminal epithelium, glandular epithelium, and stromal components is mostly a postnatal event, with functionality of response to steroid hormones beginning at puberty. Interference with these processes can predispose women to infertility, ectopic gestation, poor pregnancy outcomes, and other reproductive disorders that may be programmed during development (*e.g.*, endometriosis, uterine fibroids). Thus, abnormal development or alterations at other times in the life cycle can alter anatomy and functionality of the female reproductive tract and thus can alter the reproductive potential of affected individuals and their offspring.

Most female reproductive disorders are well described with regard to clinical presentation, histological evaluation of involved tissues where applicable, and diagnostic classification. However, whereas few are polygenic inherited traits and some are due to infections, the pathogenesis of the vast majority of female reproductive disorders is not well understood. This has hindered a preventive strategy to their development and/or exacerbation, and in some cases limited the development of effective therapies for symptoms and associated morbidities.

A key question arises as to whether EDCs contribute to the development of female reproductive disorders, particularly those occurring during a critical window of susceptibility: *in utero*, neonatally, in childhood, during puberty, and during adulthood. There are increasing data from wildlife studies and laboratory studies with rodents, ungulates, and nonhuman primates that support a role of EDCs in the pathogenesis of several female reproductive disorders, including polycystic ovarian syndrome, aneuploidy, POF, reproductive tract anomalies, uterine fibroids, endometriosis, and ectopic gestation (for reviews, see Refs. 29 and 49–54; also see Table 4). Many of the mechanisms are understood and, moreover, are conserved

TABLE 4. Female reproductive disorders and their possible relationships to EDCs: Some experimental and human data

Female reproductive disorder	Experimental data	Human epidemiological data
Reproductive tract abnormalities/ malignancies	Mice prenatally exposed to DES have structural abnormalities of the oviduct, uterus, cervix, and vagina, leiomyoma, infertility-subfertility, immune dysfunction, ovarian cysts, ovarian tumors, vaginal adenocarcinoma (480)	*In utero* exposure to DES: abnormal cervical, uterine, and oviduct anatomy (481), vaginal adenocarcinoma (19), subfertility and infertility, ectopic pregnancy (480)
Endometriosis	Adult monkey exposed to TCDD (dioxin): promotion of growth and survival of endometriosis impants (110)	↑ plasma concentrations of DEHP in women with endometriosis *vs.* controls (113); ↑ levels of phthalates (DnBP, BBP, DnOP, DEHP) in Indian women with endometriosis *vs.* controls (114)
Precocious puberty	Immature female rat exposed to DDT: sexual precocity (27)	High levels of the DDT metabolite p,p′-DDE, in plasma from foreign immigrant girls with precocious puberty in Belgium (482)
	Female mouse fetuses exposed to BPA: early puberty (474)	Breastfed girls exposed to high levels of PBB *in utero* (≥7 ppm): earlier age at menarche (483)
Premature thelarche		Higher levels of phthalates and its major metabolite mono-(2-ethylhexyl) phthalate in serum of girls from Puerto Rico with premature breast development (26)
Disturbed lactation	Rodents exposed to atrazine: impaired lactation through prolactin inhibition (484)	Negative correlation between DDE (metabolic product of DDT) and duration of lactation (484)
Breast abnormalities/ cancer	Fetal rats exposed to dioxins (TCDD): altered breast development and ↑ susceptibility for mammary cancer (478)	Limited and conflicting evidence
	Mice exposed to BPA: altered organization of the mammary anlagen, accelerated ductal development, and inhibition of lumen formation in the fetus (128)	M2 polymorphism in the cytochrome P450 1A1 gene modify the association between PCB exposure and risk of breast cancer (51)
	Mice exposed to BPA: increased number of epithelial structures (145, 146)	
	Rats exposed perinatally to BPA: development of preneoplastic lesions (intraductal hyperplasias) and carcinomas *in situ* (148)	
	Rats exposed perinatally to BPA; increased susceptibility to neoplastic development (149)	
	Rats: lactational exposure to BPA: shortening of the latency period and increased tumor multiplicity after carcinogen challenge (150)	
	Mice exposed to BPA: development of preneoplastic lesions (intraductal hyperplasias) (147)	
PCOS	Prenatal exposure to high levels of testosterone results in fetal programming of PCOS traits (60, 61)	Increased levels of serum AGEs in women with PCOS and positive correlation between AGE proteins and testosterone levels (64)
	Rats fed with high *vs.* low AGE diet: ↑ androgens– ↑ ovarian volume and AGE ovarian deposition (461)	In polycystic ovaries, increased immunostaining of colocalized AGEs, RAGEs, and activated nuclear factor-κB (211, 485)
Fertility and fecundity	Mice prenatally exposed to DES (480)	Isolation of persistent organochlorine chemicals from ovarian follicular fluid of women undergoing IVF (51)
		Indications that exposure to pesticides may contribute to female infertility in some occupationally exposed groups (484)

↑, Increased; DEHP, di-(2-ethylhexyl) phthalate; DnBP, di-n-butyl phthalate; BBP, butyl benzyl phthalate; DnOP, di-n-octyl phthalate.

between animals and humans. Herein, we describe some of the clinical implications of these associations.

B. Polycystic ovarian syndrome (PCOS)

PCOS is a heterogeneous syndrome characterized by persistent anovulation, oligo- or amenorrhea, and hyperandrogenism in the absence of thyroid, pituitary, and/or adrenal disease (55–57). At the level of the ovary, there is recruitment and growth of follicles to the small antral stage, without selection of a dominant, preovulatory follicle, leading to accumulation of multiple, small, antral follicles (58). Hyperfunctioning of the theca and relative hypofunctioning of the granulosa cells accompany the acyclicity of the syndrome. Many, but not all women with PCOS have

relatively high circulating levels of LH, compared with FSH, believed to be due to insensitivity to steroid hormone feedback. However, this does not fully account for the observed increase in thecal androgen production or the relative quiescence and sometimes frank FSH resistance of the granulosa cells. This complex disorder likely has its origins both within and outside the hypothalamic-pituitary-ovarian axis, and metabolic, neuroendocrine, and other endocrine regulators likely contribute to its manifestation. Obesity and insulin resistance occur in about 50% of women with PCOS, and obese women have a 12% risk of having PCOS (59). PCOS has multiple physiological processes (*e.g.*, neuroendocrine functioning and feedback mechanisms, ovarian steroidogenesis, insulin resistance, and obesity) that are regulated by hormonal and metabolic parameters. Hence, endocrine disruption by environmental chemicals may indeed contribute to the pathogenesis of PCOS.

In sheep and rhesus monkeys, prenatal exposure to high levels of testosterone results in fetal programming of PCOS traits (60). Specifically, high levels of testosterone exposure at gestational d 40–60 and 100–115 result in rhesus monkey females who, in adulthood, have anovulatory infertility, hypersecretion of LH, elevated circulating levels of testosterone, neuroendocrine feedback defects, central adiposity and compensatory insulin resistance, and polycystic ovaries with ovarian hyperandrogenism and follicular arrest in adulthood (60, 61). In the sheep model, a similar PCOS phenotype, along with IUGR and compensatory catch-up growth after birth, derives from prenatal exposure to exogenous testosterone (60, 62). In rhesus monkey and sheep, unlike rodents, follicular differentiation is completed during fetal life. Thus, it is plausible that *in utero* exposure of human female fetuses to androgen-like EDCs could result in PCOS in adulthood, along with associated metabolic disorders. Very recent evidence for androgenic properties of personal-care products such as triclocarban (63) add to the possibility of environmental androgens, although a connection to PCOS has not yet been drawn.

There are numerous candidate genes associated with predisposition to developing PCOS in women (57, 64), and how and if these interact with prenatal androgen-like factors to promote the PCOS phenotype in women has not been determined. Nonetheless, PCOS is a debilitating disorder in women, occurring in 6.6% of the reproductive-age population (65–67); it is a leading cause of subfertility and is associated with increased lifetime risks for cardiovascular disease and type II diabetes (55). In addition to these clinical impacts on patients, the cost to the health care system for PCOS diagnosis and treatment is substantial, totaling in 2004 about $4.4 billion in the United States alone (68). These facts underscore the need to understand potential EDC contributions to the development of PCOS in an effort to minimize such exposures and maximize prevention.

Other pathways may be involved in endocrine disruption of PCOS. Women with PCOS have higher levels of the EDC BPA (69), and increased testosterone in these women is consistent with decreased clearance of BPA (70). Although adult exposures do not necessarily imply earlier exposures in life, especially with EDCs of relatively short half-lives, there are data demonstrating nearly 5-fold higher levels of BPA in amniotic fluid compared with other body fluids, suggesting significant prenatal exposure (71). Although a cause and effect of BPA and PCOS have not been demonstrated definitively, the biological plausibility is interesting and worthy of further consideration.

C. Premature ovarian failure, decreased ovarian reserve, aneuploidy, granulosa steroidogenesis

POF (cessation of proper ovarian function before the age of 40) occurs in about 1% of reproductive-age women (72). Although in some cases the causation is known, for the vast majority of women with POF this is not the case, and there are stages of susceptibility during organogenesis and adult exposures that could contribute to POF.

Because the total ovarian follicle complement is established before birth in humans (73), anything that interferes with this, resulting in a decreased ovarian follicle resting pool, can result in POF. For example, disruption of germ cell migration from the genital ridge into the developing gonad results in ovarian dysgenesis. The resting pool undergoes a baseline level of apoptosis, and TNF-α, Fas ligand, and androgens stimulate this in the resting pool, as well as in the growing pool (74). Also, once a cohort of follicles is recruited during a given cycle in women, survival factors (FSH, estradiol, and growth factors, *e.g.*, IGFs) are important for escape from apoptosis of the dominant follicle. Recent data in the mouse show that selective activation of the K-ras pathway in the oocyte results in rapid follicular development and depletion (75). Interestingly, adult and *in utero* exposures of mice to BPA have resulted in damage to oocytes (76, 77). Specifically, adult exposures result in abnormalities in alignment of chromosomes on the meiotic spindle and aneuploidy, which, while not leading to ovarian senescence, does lead to aneuploid gametes and offspring (76). However, BPA given to pregnant dams during midgestation affects the developing ovary with resulting abnormalities in meiotic prophase, including synaptic defects, and mature animals exposed *in utero* have an increase in aneuploid oocytes and embryos (77). Such alterations also lead to cell cycle arrest and oocyte death, thus depleting the complement of normal oocytes (77). Currently, there are no data on *in utero* or

adult exposure to BPA and aneuploidy in humans, but the possibility that there are parallels is compelling.

Interestingly, mice exposed *in utero* to DES, between d 9–16 gestation, have a dose-dependent decrease in reproductive capacity, including decreased numbers of litters and litter size and decreased numbers of oocytes (30%) ovulated in response to gonadotropin stimulation with all oocytes degenerating in the DES-exposed group, as well as numerous reproductive tract anatomic abnormalities (78). In women with *in utero* exposure to DES, Hatch *et al.* (79) reported an earlier age of menopause between the 43–55 yr olds, and the average age of menopause was 52.2 yr in unexposed women and 51.5 yr in exposed women. The effect of DES increased with cumulative doses and was highest in a cohort of highest *in utero* exposure during the 1950s (79). These observations are consistent with a smaller follicle pool and fewer oocytes ovulated, as in DES-exposed mice after ovulation induction (78).

Of interest are human data that demonstrate unequivocally that adult exposure in women to cigarette smoke results in decreased fecundity, decreased success rates in *in vitro* fertilization (IVF), decreased ovarian reserve (higher basal cycle d 3 FSH and stimulated parameters), earlier menopause by 1–4 yr, and an increased miscarriage rate (80, 81). The mechanism appears to be mediated by the AhR-mediated apoptosis of oocytes, with accelerated loss of ovarian follicles. Interestingly, exposure of rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) *in utero* and through the end of reproductive life results in a dose-dependent onset of premature reproductive senescence, likely due to direct effects on ovarian function (82).

Thus, whereas POF may occur in a relatively small percentage of the population, there are several alarming signals that should not be ignored. For example, the age group with the fastest growing rate of involuntary subfertility is 15- to 24-yr-old women (83). Also, the known effects of environmental contaminants on oocyte survival, aneuploidy, decreased ovarian reserve, and infertility described above underscore how much at risk the population may be for reproductive compromise.

With regard to ovarian granulosa steroidogenesis, several EDCs have effects on this process (84). For example, TCDD (10 ppm) decreases FSH-stimulated LH receptor mRNA expression and half-life in cultured granulosa (85). DDE increases vascular endothelial growth factor and IGF-I expression in luteinized granulosa from IVF patients, suggesting a contribution to impaired steroidogenesis and perhaps infertility (86). Recently, Kwintkiewicz and Giudice (87, 88) have shown, in preliminary studies, that BPA decreases proliferation and FSH-induced aromatase expression via activation of peroxisome proliferator-activated receptor γ (PPAR-γ) and increases IGF-I and IGF receptor type I in human granulosa-like tumor cells and luteinized human granulosa from IVF subjects. These data suggest that EDCs may have local effects on ovarian function in adult women.

D. Reproductive tract anomalies

Disruption of female reproductive tract development by the EDC DES is well documented (89). A characteristic T-shaped uterus, abnormal oviductal anatomy and function, and abnormal cervical anatomy are characteristic of this *in utero* exposure, observed in adulthood (90), as well as in female fetuses and neonates exposed *in utero* to DES (91). Some of these effects are believed to occur through ERα (92) and abnormal regulation of Hox genes (93, 94). Clinically, an increased risk of ectopic pregnancy, preterm delivery, miscarriage, and infertility all point to the devastating effect an endocrine disruptor may have on female fertility and reproductive health (89). It is certainly plausible that other EDCs with similar actions as DES could result in some cases of unexplained infertility, ectopic pregnancies, miscarriages, and premature deliveries. Although another major health consequence of DES exposure *in utero* was development of rare vaginal cancer in DES daughters, this may be an extreme response to the dosage of DES or specific to pathways activated by DES itself. Other EDCs may not result in these effects, although they may contribute to the fertility and pregnancy compromises cited above. Of utmost importance clinically is the awareness of DES exposure (and perhaps other EDC exposures) and appropriate physical exam, possible colposcopy of the vagina/cervix, cervical and vaginal cytology annually, and careful monitoring for fertility potential and during pregnancy for ectopic gestation and preterm delivery (89, 95).

E. Uterine leiomyomas

Uterine leiomyomas (fibroids) are benign smooth muscle tumors of the myometrium that can cause morbidity for women, including menorrhagia, abdominal pain, pelvic prolapse, and infertility and miscarriage (96). They are the most common tumor of the reproductive tract in women and comprise the leading cause for hysterectomy and the second leading cause of inpatient surgery in the United States, with health care costs exceeding $2 billion in 2004 (97). The prevalence rate of uterine leiomyomas is approximately 25–50%, with a preponderance occurring in African-American women (97). The greatest risk factor in adult women is prolonged exposure to unopposed estrogen. Whether *in utero* exposure to DES increases a woman's lifetime risk of developing uterine fibroids is controversial, as the method to detect fibroids in two different studies influenced the outcome (98, 99). Specifically, in a

study of 1731 women exposed to DES and 848 matched unexposed controls, no association was found ($P = 0.68$) when histological confirmation after myomectomy or hysterectomy was used to document uterine fibroids (98). In contrast, when ultrasound was used to determine the presence of fibroids in DES-exposed *vs.* DES-unexposed women, a significant relationship was found (odds ratio, 2.4; 95% confidence interval, 1.1–5.4) in DES-exposed women and uterine fibroids (99). However, there are strong animal data to support development of uterine fibroids in adulthood after *in utero* exposure to EDCs, especially DES (for reviews, see Refs. 49, 50, and 52). Newbold *et al.* (100) reported that CD-1 mice develop uterine leiomyomas if exposed *in utero* or neonatally to DES, whereas unexposed mice do not. Furthermore, the Eker rat, which has a germ-line mutation in the rat homolog of the tuberus sclerosis complex 2 tumor suppressor gene, spontaneously develops uterine leiomyomas (101). The number, size, and growth rate of the fibroids increase significantly when the rat is exposed to DES on postnatal d 3–5 and 10–12, but not 17–19 (102), an effect that can be diminished with prior oophorectomy (102). These data overall strongly suggest developmental programming and gene-environment interactions for the increased risk of uterine lyomyomas in this rat model (103). In addition to mice, the Eker rat, and some dogs, the Baltic gray seal that has high organochlorine body burden also develops uterine leiomyomas (104). As with most environmental causes of abnormalities in the reproductive tract (and other tissues and organs), direct cause and effect relationships are difficult to establish. However, as in many of the other abnormalities in this Scientific Statement, the likelihood of such a relationship is plausible.

F. Endometriosis

Endometriosis is an estrogen-dependent gynecological disorder associated with pelvic pain and infertility. It occurs in 6–10% of women and up to 50% of women with pelvic pain and infertility. In 2002, the total health care costs estimated in the United States for diagnosis and treatment of endometriosis totaled approximately $22 billion (105). There are suggestive animal data of adult exposure to EDCs and development of or exacerbation of existing disease, and there is evidence that *in utero* exposure in humans to DES results in an increased relative risk = 1.9 (95% confidence interval, 1.2–2.8) (106). Most striking are the observations of rhesus monkeys administered different doses of TCDD and their subsequent development of endometriosis (107, 108). Although this study had low sample size and confounding variables that brought into question the relationship between endometriosis and TCDD (49, 52, 109), another study revealed that adult exposure of cynomolgus monkey to TCDD promotes growth and survival of endometriosis implants (110), indicating that this EDC is involved in the progression, if not pathogenesis, of this disorder. Similar data were obtained in rodent models of endometriosis in which human endometrium is transplanted into mouse and rat peritoneum, and the established lesions grew larger when animals were exposed to TCDD *in utero* and as adults (111, 112), underscoring the estrogen (and EDC) dependence of this disorder.

There are also correlative findings of phthalate levels in plasma and endometriosis. For example, Cobellis *et al.* (113) found high plasma concentrations of di-(2-ethylhexyl)-phthalate in women with endometriosis, and an association of phthalate esters with endometriosis was found among Indian women (114). Thus, the evidence is accumulating of correlations between EDCs in the circulation of women with endometriosis, although a cause-and-effect relationship has yet to be established, which is not uncommon in reproductive environmental toxicity.

Endometriosis is believed to be due to retrograde menstruation and transplantation of endometrial fragments and cells into the peritoneal cavity. Because nearly all women have retrograde menstruation but relatively few have endometriosis, the disorder is also believed to involve a dysfunctional immune response, *i.e.*, activated macrophages in the peritoneal cavity with robust secretion of inflammatory cytokines but without clearance of disease. An interesting model of early-life immune insult and developmental immunotoxicity suggests that *in utero* exposures to specific insults may reprogram the immune system, resulting in disorders such as chronic fatigue syndrome, cancer, and autoimmune disorders. Whether this has any relevance to the development or progression of endometriosis in adult women has not been explored but warrants further evaluation. Interestingly, TCDD and a therapy for endometriosis, danazol, both have effects on the adult immune system, although effects on the developing immune system are not known.

Although the infertility associated with endometriosis for the most part can be treated with advanced reproductive technologies, less success has been achieved with treatment of endometriosis-related pain. Because the pathogenesis of the associated pain is not known with certainty, therapies are empiric and include agents directed to minimize inflammation (nonsteroidal antiinflammatory drugs, danazol), progestins and androgens (to oppose estrogen actions), GnRH analogs (to inhibit gonadotropin secretion and thus ovarian estradiol production), and aromatase inhibitors (to inhibit estradiol synthesis by the ovary and endometriotic lesions), as well as surgical ablation or excision of the disease, when possible. Most of these therapies are effective in

up to 50–60% of affected women, with either intolerable side effects (*e.g.*, profound hypoestrogenism) or recurrence of pain (*e.g.*, after surgery) (115). Thus, prevention is key to this disorder, as is understanding the pathogenesis so that therapies for pain can be devised appropriately and administered.

IV. Endocrine Disruptors, Mammary Gland Development, and Breast Cancer

It has been hypothesized that the significant increase of the incidence of breast cancer in the industrialized world observed during the last 50 yr may be due to exposure to hormonally active chemicals, particularly xenoestrogens (116). A similar increase in the incidence of testicular cancer and malformations of the male genital tract and decreased quantity and quality of human sperm have been observed during the same half century, again suggesting a link to the introduction of these chemicals into the environment (117) (see *Sections II and V*).

A. Windows of vulnerability to carcinogenic agents and "natural" risk factors

The standard risk factors for developing breast cancer include age at menarche, first pregnancy, menopause, lactation, and parity. All of these factors are related to lifetime exposures to ovarian hormones. It is also known that there are developmental periods of enhanced vulnerability (see *Section I*). For example, sensitivity to radiation is highest during puberty. Additionally, pregnancy increases the risk of breast cancer in the short term (118) and decreases it in the long term (119). More recently, epidemiological studies have revealed that the intrauterine environment may also influence the risk to develop breast cancer later in life. Studies comparing human dizygotic twins and single births revealed that the propensity to breast cancer is enhanced in female twins, and this outcome was attributed to excess estrogen exposure in dizygotic twins during gestation (120).

B. Theories of carcinogenesis

A majority of researchers support the idea that cancer is due to the accumulation of mutations in a cell [the somatic mutation theory (121)]. In contrast, supporters of the theory of developmental origins of adult disease are proposing that changes in the epigenome play a central role in carcinogenesis (see *Section VI*).

Both the genetic and epigenetic theories of carcinogenesis imply that cancer originates in a cell that has undergone genetic and/or epigenetic changes, which ultimately results in dysregulated cell proliferation (122). Alternatively, the tissue organization field theory postulates that carcinogenesis represents a problem of tissue organization, comparable to organogenesis gone awry, and that proliferation is the default state of all cells (123–125). According to this theory, carcinogens, as well as teratogens, would disrupt the normal dynamic interaction of neighboring cells and tissues during early development and throughout adulthood (126).

During postnatal life, the mammary gland undergoes massive architectural changes, comparable to those usually associated with organogenesis. These changes occur in response to alterations in endogenous hormone levels such as those associated with puberty and pregnancy and can be induced experimentally by endocrine manipulation. Many studies of endocrine disruptors have illustrated that developmental exposure to these exogenous hormone mimics can alter normal patterns of tissue organization and hence disrupt stromal-epithelial interactions (127, 128). These changes may disturb important regulatory mechanisms and enhance the potential for neoplastic lesions.

C. Susceptibility of the breast during puberty and adulthood

Several epidemiological studies explored the link between exposure to endocrine disruptors and breast cancer incidence. In general, these are case-control studies that usually measure exposure to a single chemical at the time of breast cancer diagnosis. This type of study has produced inconsistent results. Prospective studies that measured exposure several years before cancer diagnosis revealed a positive link between breast cancer and chemical exposure to toxaphene (129) and DDT (130). In particular, a study linked DDT with an increased risk of breast cancer when the exposure was measured before 14 yr of age. This study used samples taken before the banning of DDT for agricultural use and hence represents higher exposures than those measured today. Humans, however, are exposed to a plethora of hormonally active chemicals with different metabolic profiles. Moreover, individuals living in the same area may be exposed to a different mixture of chemicals due to different diets and to migration history. These facts imply that a single chemical cannot be construed as a marker of total exposure. Not surprisingly, one case-control study reported a significant correlation between total xenoestrogen exposure and breast cancer (131).

How xenoestrogen exposure during the period of sexual maturity may result in mammary gland carcinogenesis remains unsolved; this is not surprising because the mechanisms underlying hormonal carcinogenesis are still unknown. One possibility, compatible with all the cancer hypotheses briefly discussed above, is that xenoestrogens may extend the length of the period of ductal growth and alveologenesis during the menstrual cycle. This period is also characterized by proliferative activity in the glandular

epithelium. For example, ductal cell proliferation in the breast is maximal from the late follicular phase and throughout the luteal phase, *i.e.*, when endogenous estrogen levels are high (132). The ubiquitous presence of xenoestrogens in foods, their persistence, and their lack of binding to the plasma carrier protein SHBG (127) may result in relatively constant levels in blood. These xenoestrogens would act additively with ovarian estrogens and thus advance by a few days the period of ductal growth. Hence, a small and maintained increase of estrogenic activity during the period of low ovarian output could be sufficient to "promote" carcinogenesis by increasing the number of cells that undergo proliferation menstrual cycle after menstrual cycle, an explanation consistent with the somatic mutation theory. An alternative explanation, consistent with the tissue organization field theory, is that estrogens acting as morphogens would enhance tissue remodeling through stroma epithelium interactions and increase the likelihood of producing alterations of tissue architecture. This notion is supported by data showing that recombination of normal mammary epithelial cells with stroma exposed to carcinogenic agents results in the development of epithelial neoplasias (133) and that conversely, recombination of mammary carcinoma cells with stroma from multiparous animals (which are refractory to carcinogens) results in the normalization of the neoplastic phenotype (126).

D. Susceptibility of the mammary gland during the perinatal period

Direct evidence of prenatal estrogen exposure and breast cancer risk is being gathered from the cohort of women born to mothers treated with DES during pregnancy and is discussed above (see *Sections II and III*). These women are now reaching the age at which breast cancer becomes more prevalent. In the cohort of these women who are aged 40 yr and older, there is a 2.5-fold increase in the incidence of breast cancer compared with unexposed women of the same age (134, 135), suggesting that indeed, prenatal exposure to synthetic estrogens may play an important role in the development of breast neoplasms. Consistent with this, experiments in rats showed that prenatal exposure to DES resulted in increased mammary cancer incidence during adulthood (136, 137). These experiments illustrated that rats exposed prenatally to DES and challenged with the chemical carcinogen dimethylbenzanthracene (DMBA) at puberty had a significantly greater incidence of palpable mammary tumors at 10 months of age than animals exposed prenatally to vehicle. In addition, the tumor latency period was shorter in the DES-exposed compared with the vehicle-exposed group (130). Both the epidemiological and experimental data are consistent with the hypothesis that excessive estrogen exposure during development may increase the risk of developing breast cancer.

In utero exposure to tamoxifen, an estrogen antagonist and partial agonist, has also been shown to increase the incidence of mammary tumors when the exposed offspring are challenged with DMBA at puberty. Eighteen weeks after the challenge, 95% of the tamoxifen-exposed animals developed tumors, compared with 50% of the vehicle-treated rats (138). However, in the above-mentioned studies, both DES and tamoxifen were administered at high pharmacological doses to reflect the medical use of these agents, whereas the effects of twinning mentioned above represent a physiological range of endogenous hormone levels to which developing fetuses are exposed.

E. Perinatal exposure to environmentally relevant levels of endocrine disruptors

There is a third type of exposure that needs to be addressed: the inadvertent and continuous exposure of fetuses to environmentally active chemicals, such as dioxins and BPA (Table 4).

1. Dioxins

Depending on the context (time of exposure, organ, presence or absence of estrogens) dioxins have either estrogenic or antiestrogenic effects. Despite cross-talk between the aryl hydrocarbon and ERs (139), the mechanisms underlying these opposite effects have yet to be elucidated. Rats exposed prenatally (gestational d 15) to TCDD and challenged with the chemical carcinogen DMBA at 50 d of age showed increased tumor incidence, increased number of tumors per animal, and shorter latency period than rats exposed prenatally to vehicle and to DMBA at 50 d of age. These TCDD-exposed animals had increased numbers of terminal end buds at puberty (140). Because these structures are believed to be the site where mammary cancer arises, these results were interpreted as evidence that TCDD increased the propensity to cancer by altering mammary gland morphogenesis. Interestingly, Fenton (31) showed that prenatal exposure to TCDD results in impaired development of terminal end buds that remain in the gland for prolonged periods, whereas in the normal animals terminal end buds are transient structures that regress when ductal development is completed.

2. BPA, a ubiquitous xenoestrogen

The ubiquitous use of BPA provides great potential for exposure of both the developing fetus, indirectly through maternal exposure, and the neonate, directly through ingestion of tinned food, infant formula, or maternal milk (11). Indeed, BPA has been measured in maternal and fetal

plasma and placental tissue at birth in humans (141). A recently published study conducted by the Centers for Disease Control, the first using a reference human population, showed that 92.6% of over 2500 Americans had BPA in their urine (142). Measured urine concentrations were significantly higher in children and adolescents compared with adults. BPA has also been measured in the milk of lactating mothers. These data indicate that the developing human fetus and neonate are readily exposed to this chemical.

In rodents, BPA has been shown to readily cross the placenta (143, 144) and bind α-fetoprotein (the estrogen-binding protein that prevents maternal estrogen from entering the circulation of the fetus) with negligible affinity relative to estradiol; this results in enhanced bioavailability during neonatal development. BPA is present in the mouse fetus and amniotic fluid during maternal exposure in higher concentrations than that of maternal blood.

The U.S. EPA has established the safe daily intake of BPA to be 50 μg/kg body weight/d based on the assumption that the main source of exposure is oral through food ingestion. However, recent publications suggest that food is not the only relevant source of exposure and that the half-life of BPA in humans is longer than expected (6). Numerous publications addressing fetal exposures to BPA have used parenteral administration. This practice was based on one hand on the fact that the fetus is exposed to BPA through the internal milieu of the mother, and on the other hand that parenteral administration via an osmotic minipump allows for a precise and constant level of exposure. Using this route of administration, exposure of a pregnant mouse dam to 25 and 250 ng BPA/kg body weight/d (namely, 2000 and 200 times lower than the safe dose) for 14 d beginning on d 8 gestation has been shown to impact certain aspects of development in their female offspring. When examined on gestational d 18, fetuses of mothers exposed to the higher dose of BPA exhibited altered growth parameters of the mammary gland anlagen. Changes in the appearance of the mammary epithelium were observed, such as decreased cell size and delayed lumen formation, as well as increased ductal area. In the stroma, BPA exposure promoted advanced maturation of the fat pad and altered localization of fibrous collagen (128). Because maturation of the fat pad is the driving event for ductal growth and branching, it is likely that the increased ductal area in BPA-exposed animals is due to the accelerated formation of their fat pads. By postnatal d 10, in the offspring born to mothers exposed to either dose of BPA, the percentage of proliferating epithelial cells was significantly decreased relative to those not exposed. At 30 d of age, the area and number of terminal end buds relative to the gland ductal area increased, whereas cell death in these structures decreased in BPA-exposed offspring compared with controls. It is likely that the reduced cell death in the terminal end buds of BPA-exposed females may be the cause of the observed ductal growth delay because cell death is essential for both the hollowing and the outward growth of the subtending duct. Collectively, these effects observed at puberty may be attributed to an increased sensitivity to estradiol that has been observed in the BPA-exposed animals (145). Because of the new epidemiological data cited above and the effects found in the low-dose animal studies using parenteral exposure, the EPA recommendations need to be reevaluated.

In animals exposed perinatally to BPA, there was also a significant increase of ductal epithelial cells that were positive for progesterone receptor at puberty. These positive cells were localized in clusters, suggesting future branching points. Indeed, lateral branching was significantly enhanced at 4 months of age in offspring born to mothers exposed to 25 ng BPA/kg body weight/d (145). These results are compatible with the notion that increased sensitivity to estrogens drives the induction of progesterone receptors in epithelial cells, leading to an increase in lateral branching. By 6 months of age, perinatally exposed virgin mice exhibit mammary glands that resemble those of a pregnant mouse, as reflected by a significant increase in the percentage of ducts, terminal ends, terminal ducts, and alveolar buds (146). Additionally, intraductal hyperplasias, which are considered preneoplastic lesions, were observed starting at 3 months of age (147).

To explore the links between prenatal BPA exposure and mammary gland neoplasia, a rat model was chosen because it closely resembles the human disease regarding estrogen dependency and histopathology. BPA was administered to pregnant dams at doses of 2.5, 25, 250, and 1000 μg/kg body weight/d. Fetal exposure to BPA, from gestational d 9 to postnatal d 1, resulted in the development of carcinomas *in situ* in the mammary glands of 33% of the rats exposed to 250 μg/kg body weight/d, whereas none of the unexposed animals developed neoplasias (148). These cancers were only observed after the animals had reached young adult age. Fetal exposure to BPA significantly increased the number of precancerous lesions (intraductal proliferation) by three to four times, an effect also observed in puberty and during adult life. The lesions observed in the BPA-exposed animals were highly proliferative and contained abundant ER-positive cells, suggesting that the proliferative activity in these lesions may be estrogen mediated. Comparable preneoplastic lesions were found in a study using a different rat strain (149). Additionally, this study found stromal alterations such as desmoplasia and mast cell invasion; these features are often observed during neoplastic development. Moreover,

when challenged with a subcarcinogenic dose of nitrosomethylurea, only the BPA-exposed animals developed palpable tumors (carcinomas). The period of vulnerability of the mammary gland to BPA does not cease at the neonatal stage. BPA exposure during lactation followed to exposure to the carcinogen DMBA resulted in mammary tumor multiplicity and reduced tumor latency compared with control animals (exposed solely to DMBA) (150). These results indicate that perinatal exposure to environmentally relevant doses of BPA results in persistent alterations in mammary gland morphogenesis, development of precancerous lesions, and carcinoma *in situ*. Moreover, the altered growth parameters noted in the developing mammary gland on embryonic d 18 suggest that the fetal gland is a direct target of BPA, and that these alterations cause the mammary gland phenotypes observed in perinatally exposed mice at puberty and adulthood.

In summary, exposure to estrogens throughout a woman's life, including the period of intrauterine development, is a risk factor for the development of breast cancer. The increased incidence of breast cancer noted during the last 50 yr may have been caused, in part, by exposure of women to estrogen-mimicking chemicals that have been released into the environment from industrial and commercial sources. Epidemiological studies suggest that exposure to xenoestrogens such as DES during fetal development, to DDT around puberty, and to a mixture of xenoestrogens around menopause increases this risk. Animal studies show that exposure *in utero* to the xenoestrogen BPA increases this risk. Moreover, these animal studies suggest that estrogens act as morphogens and that excessive perinatal exposure results in structural and functional alterations that are further exacerbated by exposure to ovarian steroids at puberty and beyond. These altered structures include preneoplastic lesions, such as intraductal hyperplasias, and carcinomas *in situ*. Additionally, these mammary glands are more vulnerable than their normal counterparts to carcinogenic stimuli. Exposures to other endocrine disruptors that are not estrogenic, such as dioxins, were reported to increase breast cancer incidence in humans and to alter mammary gland development in animal models. Collectively, these data support the notion that endocrine disruptors alter mammary gland morphogenesis and that the resulting dysgenic gland becomes more prone to neoplastic development.

V. Male Reproductive and Developmental Health: The Human Evidence

A. Introduction to male reproductive health

The mechanisms through which environmental chemicals alter the endocrine system are elucidated through experimental animal studies and *in vitro* systems. In epidemiological studies it is generally not possible to explore potential mechanisms. Nevertheless, epidemiological studies are essential to our understanding of the potential risks, or lack thereof, of EDCs on human reproductive function and development.

Human evidence of altered male reproductive and developmental health in relation to EDCs is limited (Table 2). As has been shown in the recent Third National Report by the Center for Disease Control (151), humans are exposed, at a minimum, to hundreds of environmental chemicals, of which dozens are known EDCs. A major limitation of epidemiological studies is that they generally only measure human exposure to a single EDC, or at best to a set of isomers or congeners within a family of EDCs. A fuller understanding of potential human health risks requires studying the complex mixtures to which we are exposed. This limitation, already raised in other sections, should be kept at the forefront as the current epidemiological evidence on health risks from EDCs is presented.

For the purposes of this report, the male reproductive health endpoints under consideration include, among others: 1) disrupted reproductive function, manifest as reduced semen quality and infertility; 2) altered fetal development, manifest as urogenital tract abnormalities, including hypospadias and cryptorchidism; and 3) testicular germ cell cancer (TGCC).

B. Male reproductive function and development

1. TDS: A unifying hypothesis

Skakkebaek *et al.* (21) hypothesized that diminished semen quality, TGCC, and male urogenital tract anomalies may share a common causal pathway. They defined this triad as the TDS. The hypothesis invokes a common pathway by which EDCs, and other environmental chemicals and genetic factors, may lead to abnormal development of the fetal testis, producing testicular dysgenesis that can manifest as an increased risk of urogenital abnormalities in newborn males, as well as altered semen quality and TGCC in young men. As a cautionary note, the manifestations (or symptoms) of TDS have other causes apart from testicular dysgenesis.

It is hypothesized that TDS is due to prenatal Leydig and Sertoli cell dysfunction with secondary androgen insufficiency and impaired germ cell development. This should not be confused with the clinical diagnosis of dysgenetic testes, which is associated with genital ambiguity and a high risk of testicular malignancy (152). The existence of TDS as a distinct clinical entity and of possible associations with EDCs is an area of active research.

C. Semen quality: Temporal trends and EDC exposure

The epidemiological evidence on temporal trends in semen quality remains inconsistent. Some studies suggest that human semen quality has declined during the previous 50 yr (153–155), whereas other studies have not reported a decline (156–158). Despite the potential importance and relevance of early life exposure to EDCs, the epidemiological evidence on the relationship between semen quality and exposure to EDCs is limited to the assessment of adult exposure to EDCs. In the cases of PCBs, pesticides (persistent and nonpersistent), and phthalates, limited epidemiological evidence supports a relationship between adult exposure and reduced semen quality. However, most studies are cross-sectional in design; thus exposure and semen parameters were assessed at the same point in time. Although there are few studies in humans on the effects of developmental exposures to chemicals and semen quality in adulthood, this has been shown in animal models. Anway and Skinner (12) showed direct as well as transgenerational effects of EDCs on semen quality after intrauterine exposure.

1. Phthalates and semen quality

The diesters of 1,2-benzenedicarboxylic acid (phthalic acid), commonly known as phthalates, are a group of manmade chemicals widely used in industrial applications. They are primarily used as plasticizers in the manufacture of flexible vinyl plastic which, in turn, is used in consumer products, flooring, and wall coverings, food contact applications, and medical devices (159–161). They are also used in personal-care products (*e.g.*, perfumes, lotions, cosmetics), as solvents and plasticizers for cellulose acetate, and in making lacquers, varnishes, and coatings, including those used to provide timed releases in some pharmaceuticals (159, 162, 163).

Human exposure to phthalates is widespread and occurs through ingestion, inhalation, and dermal contact (160–165). Parenteral exposure from medical devices and products containing phthalates are important sources of high exposure to phthalates, primarily di-(2-ethylhexyl) phthalate (DEHP) (161, 166). Phthalates have biological half-lives measured in hours, are rapidly metabolized, and are excreted in urine and feces (160–163). The most common biomonitoring approach for investigating human exposure to phthalates is the measurement of urinary concentrations of phthalate metabolites.

There are few epidemiological studies on phthalates and semen quality. A large study on male partners of subfertile couples from an infertility clinic in Massachusetts (167, 168) found associations between monobutyl phthalate (MBP; the hydrolytic metabolite of dibutyl phthalate) and below World Health Organization (WHO) reference value sperm motility and sperm concentration. There was also a dose-response relationship between monobenzyl phthalate (MBzP, the primary hydrolytic metabolite of butylbenzylphthalate) and below WHO reference value sperm concentration. In contrast to the U.S. study, in a Swedish study there were no relationships of MBP or MBzP with any of the semen parameters (169). Potential reasons explaining why the two studies found differing results include differences in age and fertility of the study populations. The Swedish study population consisted of young men (median age, 18 yr; range, 18–21 yr) from the general population, whereas in the U.S. study the median age of the men from an infertility clinic was 35.5 yr and ranged from 22 to 54 yr. None of the men from the infertility clinic were 21 yr of age or younger. Men presenting to an infertility clinic may be more "susceptible" to reproductive toxicants, including phthalates, than men from the general population. Furthermore, it is also unclear whether middle-aged men, compared with young men, are more susceptible to reproductive toxicants because of an age-related response to the toxicant.

2. PCBs and semen quality

PCBs are a class of synthetic, persistent, lipophilic, halogenated aromatic compounds that were widely used in industrial and consumer products for decades before their production was banned in the late 1970s. PCBs were used in cutting oils, lubricants, and as electrical insulators. As a result of their extensive use and persistence, PCBs remain ubiquitous environmental contaminants. They are biologically concentrated and stored in human adipose tissue. The general population is exposed primarily through ingestion of contaminated foods (*e.g.*, fish, meat, and dairy products), because PCBs can bioaccumulate up the food chain. As a result of their persistence and ubiquity, measurable levels of serum PCBs are found in the majority of the U.S. general population (170). Serum levels of PCBs are an integrated measure of internal dose, reflecting exposure from all sources over the previous years; depending on the congener, the half-life of PCBs in the blood ranges from 1 to 10 or more years (171, 172). Notably, there are 209 different possible chlorine substitutions on the biphenyl backbone of PCBs, with the resulting PCB molecules having different structural, functional, and toxicological properties (173, 174).

The epidemiological evidence on the relationship between PCBs and semen quality support an inverse association of PCBs with reduced semen quality, specifically reduced sperm motility. Such relationships have been consistently reported across studies performed in different countries (India, The Netherlands, Taiwan, Sweden, and the United States). The associations were found across a range of PCB levels, suggesting that there was not a threshold. The PCB levels in these studies ranged from low back-

ground levels (175–177), to high background levels due to consumption of contaminated fish (178), to even higher exposure levels due to ingestion of contaminated rice oil (179, 180).

3. Dioxins and semen quality

A recently published study of dioxin exposure and semen quality suggested that timing of exposure may have an impact upon the response (181). A chemical plant explosion in 1976 in Seveso, Italy, led to environmental contamination with high levels of TCDD. Exposed men in three age groups (1–9, 10–17, and 18–26 yr of age in 1976) were studied in 1998. Interestingly, the men exposed prepubertally (1 to 9 yr) had an inverse association between serum TCDD concentrations and semen quality, specifically sperm count and motility, whereas the men exposed at ages 10–17 yr had a positive association with semen quality, referred to as stimulatory by the authors. The men exposed at 18–26 yr of age had no associations of TCDD with semen quality. Men exposed at both 1–9 and 10–17 yr of age had lower estradiol and higher FSH concentrations compared with unexposed men. These results suggest that the timing of exposure, *i.e.*, life stage, may have importance in determining the impact of environmental exposures.

4. Nonpersistent pesticides and semen quality

Nonpersistent pesticides (also referred to as "contemporary-use pesticides") are chemical mixtures that are currently available for application to control insects (insecticides), weeds (herbicides), fungi (fungicides) or other pests (*e.g.*, rodenticides), as opposed to pesticides that have been banned from use in most countries (*e.g.*, many of the formerly popular organochlorine pesticides such as DDT). Three common classes of nonpersistent pesticides in use today include organophosphates, carbamates, and pyrethroids. Although environmentally nonpersistent, the extensive use of pest control in these various settings results in a majority of the general population being exposed to some of the more widely used pesticides at low levels. Exposure among the general population occurs primarily through the ingestion of foods that contain low levels of pesticide residue or through inhalation and/or dermal exposure in or around the home and in other indoor environments.

Several epidemiological studies suggest an association between nonpersistent pesticide exposure and altered semen quality. Most of the data are from occupational studies involving simultaneous exposure to several pesticides (182–191). Two recent studies found associations between pesticide exposures representative of the general population and reduced semen quality (192, 193).

In a small study on male partners of pregnant women, Swan *et al.* (192) compared urinary concentrations of pesticide biomarkers in 34 men with sperm concentration, motility, and morphology below the median (defined as cases) to 52 men with above-median semen parameters (defined as controls). They found elevated odds ratios for poorer semen quality in relation to urinary concentrations of alachlor mercapturate, 2-isopropoxy-4-methyl-pyrimidinol (diazinon metabolite), atrazine mercapturate, 1-naphthol (carbaryl and naphthalene metabolite), and 3,5,6-trichloro-2-pyridinol (chlorpyrifos metabolite).

In a study among 272 men from an infertility clinic, Meeker *et al.* (193) found inverse associations between urinary levels of 1-naphthol, a metabolite of both carbaryl and naphthalene, with sperm concentration and motility. They also found a suggestive inverse relationship between the urinary metabolite of chlorpyrifos (3,5,6-trichloro-2-pyridinol) and sperm motility.

In summary, in addition to evidence from occupational studies, there are limited human studies suggesting reduced semen quality in relation to nonoccupational exposure to nonpersistent pesticides, specifically some herbicides and insecticides.

D. Male urogenital tract malformations

Epidemiological studies provide inconclusive evidence on temporal trends in cryptorchidism and hypospadias. Studies show that the prevalence of cryptorchidism is variable and geographically specific (194), with temporal upward trends noted in some studies but not others (15, 195, 196). The prevalence data for cryptorchidism are difficult to interpret because of the limitations of registry-based data and how they are obtained, changes in clinical practice that emphasize earlier diagnosis and treatment, confounding factors such as birth weight and prematurity, and inaccurate diagnosis related to changes in testicular position (spontaneous descent or secondary "ascent") over time (197). Similarly, data for hypospadias prevalence are difficult to interpret. Although prevalence temporally increased in some locations, other reports showed no trends over time (195, 198–200). Ascertainment bias may also easily exist for this anomaly, particularly for milder forms, because both false-negative and false-positive diagnoses may be made in newborns based on circumcision status.

Epidemiological evidence for EDC exposure and cryptorchidism or hypospadias is limited. Maternal serum concentrations of PCBs, DDT, or DDE (primary metabolite of DDT) were weakly associated or not associated with cryptorchidism or hypospadias in offspring (201–204).

The relationship of parental or general community pesticide exposure with hypospadias or cryptorchidism is suggestive (205–210), but there is the need for further

research that explores maternal and/or paternal exposure to specific pesticides with urogenital anomalies.

In one of the only human studies on phthalates and male genital development, Swan *et al.* (23) determined "anogenital index" (anogenital distance/body weight) and testicular position in young boys (mean age, 16 months) and corresponding maternal levels of urinary phthalate metabolites at three separate clinical sites. In this study, the authors found significant inverse relationships between the highest maternal levels of MBP, MBzP, monoethyl phthalate, and monoisobutyl phthalate and anogenital index (odds ratio for MBP, 10.2; 95% confidence interval, 2.5–42.2), although MEP has not been linked to reproductive anomalies in rodent studies based on oral administration rather than transdermal, which is the route for human exposure via its use in personal-care products (197). The implication of a reduced anogenital index in rats is well defined, but the clinical implications of reduced anogenital index in human male infants is unknown.

In summary, the strongest epidemiological data that link EDC exposure to cryptorchidism and/or hypospadias are those suggesting an association between residency in agricultural areas and/or measures of direct parental exposure to nonorganochlorine pesticides, without providing insight into specific potentially causative agents. However, these data are not necessarily consistent for both anomalies or congruent with observations made in animal experiments. Further studies will be needed to provide a clearer understanding of the role(s) of specific EDCs in the etiology of genital anomalies in man.

E. Testicular germ cell cancer

Epidemiological studies show both geographical variability and dramatic recent upward trends in the incidence rate of TGCC (212–216). The steep temporal rise over a relatively short period of several decades suggests that genetic factors alone cannot explain it. Therefore, environmental and lifestyle factors have been hypothesized to play a role. Evidence for environmental and lifestyle factors is supported by migration studies in which the first generation of immigrants have incidence rates similar to their country of origin (birth), but their offspring had rates similar to men in the country in which they were born and raised (217).

The earliest suggestion of epidemiological evidence related to prenatal estrogen exposure and increased risk of TGCC came from a study in 1979 (218). However, other studies have not consistently confirmed these earlier results (219). At present, the evidence on EDCs and risk of TGCC is very limited. Interestingly, in a novel case-control study on EDCs and TGCC, Hardell *et al.* (220, 221) did not find associations between serum concentrations of organochlorines among cases and controls and risk of TGCC, but instead found that blood organochlorine levels measured in their mothers, decades after their sons' birth, were predictive of increased risk. The organochlorines measured included PCBs, p,p′-DDE (primary long-lived metabolites of DDT), and hexachlorobenzene, a fungicide. The study was small (44 case mothers and 45 control mothers), and the median time from the fetal period until blood sampling for the cases and controls was approximately 30 yr. It is important to keep in mind that despite the long period between the etiological relevant exposure window and measurement of organochlorines, their long half-lives, on the order of years to a decade, makes it possible to estimate historic exposure using the mothers' blood samples. Therefore, the limited studies suggest that *in utero* exposure to environment EDCs represents the relevant etiological window of exposure. If this is borne out to be true, it will mean that epidemiologists need to consider innovative study designs to better assess prenatal exposure windows for endpoints that may not manifest for decades. Prospective pregnancy cohort follow-up studies for TGCC would be difficult and costly to implement because TGCC is a rare cancer and prospective study would require unrealistically large cohorts.

F. Conclusions

This section has tried to provide highlights and insights into the current state of the epidemiological evidence on the relationship between EDCs and male reproductive and developmental health. The overview was not meant to be an exhaustive review of the evidence, but rather a synthesis of the current knowledge in an ever-changing field of inquiry and discovery. Although there is current scientific, public, and governmental interest in the potential health risks of exposure to EDCs, the human evidence on associations of EDCs with altered male reproductive health endpoints remains limited and, in certain instances, inconsistent across studies. This highlights the need for further epidemiological research on these classes of EDCs.

VI. Prostate Cancer

A. Introduction to prostate cancer

Prostate cancer is the most common solid cancer in males and the second leading cause of cancer deaths in American men (222). In addition, benign prostatic hyperplasia is the most common benign neoplasm, occurring in approximately 50% of all men by the age of 60. The basis for these high rates of abnormal prostatic growth is not well understood despite decades of extensive research on the topic. Nonetheless, it is accepted that steroids play a fundamental role in the initiation and progression of prostate cancer, which forms the basis for hormonal treatment strategies. Men who have undergone early castration do

not develop prostatic carcinoma (223). Charles Huggins received the Nobel Prize for his work revealing that regression of prostate cancer can be initially achieved by castration and androgen blockade (224). In addition to androgens, it has been proposed that estrogens are involved in the etiology of benign prostatic hyperplasia and prostatic cancer (225–227), and the use of antiestrogens has been recently recognized to have a therapeutic role in prostate cancer management (228, 229). The prostate gland contains both ERα and ERβ during development and into adulthood, with ERα primarily found in stromal cells and ERβ in differentiated epithelium (230). It is also believed that prostatic developmental events under the regulation by steroids early in life may be linked to the predisposition of this structure to high rates of disease in adult men (231, 232). Moreover, the prostate gland is particularly sensitive to estrogen exposures during the critical developmental period relative to adult estrogenic responses (233).

The established risk factors for prostate cancer are age and race. African-American men have the highest incidence of prostate cancer worldwide, at rates 2-fold those for Caucasian-American counterparts. Family history (genetics), diet, and environmental factors are also recognized to impact prostate cancer risk. However, in the human population, direct connections between EDCs and prostate cancer risk have not been established. Due to the hormonal basis of this disease and the evidence that dietary compounds high in phytoestrogens (*e.g.*, genistein) can control prostate cancer growth in humans, there is reasonable cause to evaluate and understand any potential relationship between environmental EDCs and prostate cancer risk. Because there are difficulties in directly associating prostate cancer risk in humans with EDC exposures, potential risk(s) will have to be ascertained from research with animal models, particularly those that are responsive to environmentally relevant exposures. The sections below summarize the evidence obtained from epidemiological studies, *in vitro* studies with human prostate cells, and *in vivo* studies in animal models that indicate associations between EDCs and prostate cancer, carcinogenesis, and/or susceptibility (Fig. 1).

B. Evidence and mechanisms for EDC effects on the prostate

1. Farming and pesticides

The most compelling data for a link between prostate cancer and environmental factors outside of diet in humans comes from the established occupational hazard of farming and increased prostate cancer rates (234–236). Although several variables may contribute to this association, chronic or intermittent exposures to pesticides are the most likely explanation (236, 237). A large epidemiology study (Agricultural Health Study) conducted collaboratively between the National Cancer Institute, the National Institute of Environmental Health Sciences, and the EPA examined agricultural lifestyles and health in approximately 90,000 participants in North Carolina and Iowa since 1993 (www.aghealth.org). Evaluation of more than 55,000 pesticide applicators revealed a direct link between increased prostate cancer rates and exposure to methyl bromide, a fungicide with unknown mechanism of action (236). In addition, six pesticides (of 45 common agricultural pesticides) showed significant correlation with exposure and increased prostate cancer rates in men with a familial history of the disease, suggesting gene-environment interactions. These six agents were chlorpyrifos, fonofos, coumaphos, phorate, permethrin, and butylate (236, 238). The first four compounds are thiophosphates that share a common chemical structure. These agents are acetylcholine esterase inhibitors and have not been shown to have direct estrogenic or antiandrogenic activities. However, a literature search found that these compounds have marked capacity to inhibit p450 enzymes. Chlorpyrifos, fonofos, and phorate strongly inhibit CYP1A2 and CYP3A4, which are the major p450s that metabolize estradiol, estrone, and testosterone in the liver (239, 240). Thus it is possible that exposure to these compounds can interfere with metabolism of steroid hormones and, in so doing, disturb the normal hormonal balance that might contribute to increased prostate cancer risk. A similar mechanism of endocrine disruption *in vivo* has been identified for PCBs and polyhalogenated aromatic hydrocarbons (including dioxins, BPA, and dibenzofurans) through marked inhibition of estrogen sulfotransferase, which in turn elevates bioavailable estrogens in target organs (45, 241).

2. Environmental estrogens

In men, chronically elevated estrogens have been associated with increased risk of prostate cancer (227). In rodents, natural estrogens combined with androgens induce prostate cancer (225, 242). For simplicity, we herein refer to environmental estrogens as molecules with identified estrogenic activity (estrogen mimics), primarily through ER activation.

a. DES. *In utero* DES exposure is an important model of endocrine disruption and provides proof-of-principle for exogenous estrogenic agents altering the function and pathology of various end-organs. Maternal usage of DES during pregnancy resulted in more extensive prostatic squamous metaplasia in human male offspring than observed with maternal estradiol alone (243). Although this prostatic metaplasia eventually resolved during postnatal life, ectasia and persistent distortion of ductal ar-

chitecture remained (244). These findings have led to the postulation that men exposed *in utero* to DES may be at increased risk for prostatic disease later in life (245), although the limited population studies conducted to date have not identified an association (245). Nonetheless, several studies with DES in mouse and rat models have demonstrated significant abnormalities in the adult prostate, including increased susceptibility to adult-onset carcinogenesis after early DES exposures (246–249). It is important to note that developmental exposure to DES, as with other environmental estrogens, has been shown to exhibit a biphasic dose-response curve with regard to several end-organ responses, and this has been shown to be true for prostatic responses as well (250). Low-dose fetal exposure to DES or BPA (see below) resulted in larger prostate size in adulthood compared with controls, an effect associated with increased levels of prostatic ARs. This contrasts with smaller prostate sizes, dysplasia, and aging-associated increases in carcinogenesis found after perinatal high-dose DES exposures as noted above. This differential prostatic response to low *vs.* high doses of DES and other EDCs must be kept in mind when evaluating human exposures to EDCs because the lack of a response at high doses may not translate into a lack of negative effects at low, environmentally relevant doses of EDCs.

b. BPA. BPA is a synthetic monomer used in the production of polycarbonate plastics and epoxy resins and is one of the highest production synthetic compounds worldwide. Importantly, conjugated BPA was detected in the urine of 93% of the U.S. population in a recent screen conducted by the Center for Disease Control. Although the relative binding affinity of BPA for ERα and ERβ and its capacity to activate ER-dependent transcription is approximately 1,000 to 10,000 lower than estradiol or DES (1, 251), BPA was capable of activating an estrogen-responsive luciferase reporter at levels that were 50% of 17β-estradiol activation (252). Thus, whereas BPA may have a significantly lower potency than endogenous estrogens *in vitro*, it is a full agonist for both ERα and ERβ. Furthermore, BPA induces ER through nongenomic pathways with an EC_{50} equivalent to 17β-estradiol, suggesting that *in vivo* estrogenic activity of BPA may be due to nongenomic activation of ER (253, 254).

The carcinogenic potential of BPA was recently evaluated by an expert panel convened by the EPA and the National Institute of Environmental Health Sciences, and the written report, which includes prostate cancer findings, has been published (255). In summary, there is evidence using *in vitro* prostate cell cultures and rodent models showing that BPA can modulate prostate cell proliferation and increase susceptibility of the prostate gland to hormonal carcinogenesis. Using transcriptional assays, BPA (1 nM) was found to activate a mutated AR (AR-T877A) that is frequently found in advanced prostate cancers of patients who relapsed after androgen deprivation therapy (256). Furthermore, BPA exposure led to unscheduled cell cycle progression and cellular proliferation in the absence of androgen in LNCaP cells that expressed this mutant AR. Because BPA had no impact on wild-type AR, these findings demonstrate that the common gain-of-function AR mutant had attained the ability to utilize BPA as an agonist. Importantly, the BPA effects were greatest at lower doses of BPA compared with high-dose exposures. *In vivo* analyses of the impact of BPA on human prostate tumor growth and recurrence was performed utilizing a xenograft model (257). At low doses equivalent to human exposures, prostate tumor size increased after BPA exposure when compared with placebo control mice. Additionally, mice in the BPA cohort demonstrated an earlier rise in prostate-specific antigen (biochemical failure), which indicates that BPA significantly shortened the time to therapeutic relapse. These outcomes underscore the need for further study of the effects of BPA on tumor progression and therapeutic efficacy.

Recent studies using a rat model have shown that early-life exposure to environmentally relevant levels of BPA can increase susceptibility to prostate carcinogenesis, possibly by developmentally reprogramming carcinogenic risk (122, 258). Rats were exposed to low doses of BPA (10 μg/kg body weight) during the early postnatal period when the prostate undergoes morphogenesis. In adulthood, estradiol levels were elevated 3-fold through the use of implants for 16 wk. Rats exposed neonatally showed a significant increase in the incidence (100 *vs.* 40%) and grade of prostatic intraepithelial neoplasia lesions compared with rats neonatally exposed to oil alone. These lesions in BPA-exposed rats exhibited high levels of proliferation and apoptosis suggestive of perturbed homeostasis leading to pathological lesions. Furthermore, prostates from BPA-exposed animals were shown to have permanent epigenetic changes with altered DNA methylation patterns in multiple genes that resulted in altered gene transcription. Together, these findings indicate that BPA may "imprint" the prostate through epigenetic modifications, resulting in predisposition to carcinogenesis.

c. PCBs. PCBs are persistent organic pollutants that are fat-soluble and bioaccumulate in human body fat deposits. Many PCBs have estrogenic or antiandrogenic activity and as such, may perturb the prostate gland. A recent analysis in Swedish men with and without prostate cancer of adipose tissue PCB concentrations revealed a significant association between PCB levels in the higher quadrants and prostate cancer odds ratio, with the most marked associations for PCB 153 and transchlordane (259). An extensive epidemiological study of capacitor manufacturing plant workers exposed to high levels of PCBs revealed a

strong exposure-response relationship for prostate cancer mortality (260). These results support previous findings of correlations between PCB 153 and 180 and prostate cancer risk in electric utility workers (261, 262). Although estrogenic activity of these compounds is a suspected mode of action, there is also evidence that PCBs inhibit estrogen sulfotransferase activity in the liver and effectively increase bioavailable estrogen in the body (45). Further investigation using animal models is warranted for PCBs and prostate cancer risk.

d. UV filters. Recent reports have shown that UV light filters used to protect against the sun have estrogenic activity (263). In particular, 4-methylbenzylidene camphor and 3-benzidene camphor are ERβ ligands (264). Although there are no studies on these UV filters and human prostate cancer, two reports indicate that early life exposure to these compounds can alter prostate gland development, growth, and gene expression in the rat prostate (263, 265). Thus, it is possible that the fetal prostate in humans may be affected after maternal use of these compounds, although this remains to be examined.

e. Cadmium. Cadmium has been shown to act as a ligand for the ER and function as an estrogenic mimic. Although some large epidemiological studies indicated a relationship between cadmium exposure and rates of prostate cancer, these findings have been challenged in other reports (266). Cadmium has been shown to have proliferative action on human prostate cells *in vitro* through an ER-dependent mechanism, and this exposure was associated with progression to androgen independence (267). In addition, prostatic tumors have been experimentally induced by oral exposure to cadmium (268). Because cadmium bioaccumulates in the body, further epidemiological analysis of cadmium and prostate cancer risk is warranted, particularly in men with occupational exposures.

f. Arsenic. Exposure to arsenic has long been associated with a number of diseases, including cancers (269). More recently, it has been documented that arsenic may mediate some of these effects through endocrine disruption, specifically through interaction with ERs and activation of estrogen-regulated genes (270). A recent report has found that arsenic induced malignant transformation of prostate epithelial cells *in vitro*, driving them toward an androgen-independent state (271). Progression to androgen-independent growth was shown to be mediated through Ras-MAPK pathways, and thus, it is possible that membrane ERs may mediate this effect. Epidemiological studies have shown an association between arsenic exposure and prostate cancer mortality in Taiwan (272), a finding that was substantiated in a more recent study in the United States (273). Thus it remains a possibility that endocrine disruption by arsenic can contribute to prostate cancer risk, and further research on this topic is essential.

3. Antiandrogens

Endocrine disruption that might affect the prostate gland can also be derived through antiandrogenic pathways. Because prostate cancer is an androgen-dependent disease, a brief review of known effects of some of these agents on the prostate gland is presented.

a. Vinclozolin. Vinclozolin is a fungicide that is used as a pesticide on crops. It possesses known antiandrogenic properties through interference with AR activity (274). Rats exposed to vinclozolin during early development were reported to have reduced prostate gland growth and size (275). Recently, maternal exposure to vinclozolin was shown to produce transgenerational effects with adverse consequences on the prostate gland, including atrophy and prostatitis for four generations (34, 276). However, because vinclozolin functions through AR antagonism, it is unexpected that vinclozolin will lead to prostate cancer.

b. DDT/DDE. DDT and its metabolic derivative p,p′-DDE were widely used as pesticides in the United States, and their use is still in effect in other countries. Although many reproductive abnormalities have been found with DDT/DDE, there is no known association between its exposure and prostate cancer risk (277). Again, due to its antiandrogenic actions, it is not expected to drive prostate cancer. A number of key questions remain unresolved but merit future investigation, not just in prostate cancer but in other fields (Boxes 1 and 2). Spanning from the molecular to the clinical, they highlight the need for a better understanding of the pathogenesis of prostate cancer and the potential role of EDCs in this process.

BOX 1. Recommendations for research on prostate cancer

- It remains unclear whether EDC exposures directly induce or promote prostate cancer. If either occurs, it will be necessary to determine the mode of action.
- It will be important to determine whether estrogenic or antiandrogenic EDCs modulate disease risk or progression in the adult male. One possibility may be that EDC exposure may influence prostate cancer susceptibility in subpopulations of men. If so, it would be important to determine the other risk factors that EDCs might synergize with to influence prostate cancer incidence and/or progression.
- It is unknown whether there is an additive or synergistic effect from EDC mixtures and prostate cancer risk or growth.
- It is necessary to determine whether the *in utero* developing human prostate is sensitive to EDCs and whether this may influence the prostate cancer risk in the aging male.
- Epidemiology studies need to be undertaken to evaluate the long-term outcome for prostate cancer incidence, grade, stage, and progression in DES-exposed sons.
- The most appropriate life stages for examining EDC and prostate cancer risk need to be assessed.
- An unexplored and important issue is whether there may be a transgenerational risk for prostate cancer as a function of EDC exposures.
- Are there epigenetic pathways that mediate developmental exposures to EDCs and prostate disease with aging?
- It will be important to establish molecular markers for EDC exposures as they relate to prostate disease risk.

BOX 2. Recommendations for research and practice regarding EDCs

- Clinical research
  - In newborns with IUGR and/or anomalies of sexual differentiation including cryptorchidism and hypospadias, screen for EDCs in maternal serum and in breast milk, and archive biological samples for further screening.
  - Prioritize a search for early (*i.e.*, neonatal period and infancy) biomarkers of EDC effects and early indicators of exposure to EDCs during fetal life.
  - Identify groups at high or low risk of exposure to EDCs for prospective studies correlating indicators of early exposure with subsequent clinical characteristics throughout infancy, childhood, and adolescence.
  - Search for and study polymorphisms in enzymes (*e.g.*, CYP enzymes) that predispose groups/individuals to greater/lesser vulnerability to EDCs.
  - Develop intervention strategies to decrease or reverse the influence of EDCs on prostate health.
  - Identify the chemical or chemicals in pesticides that negatively impact risk for prostate cancer, breast cancer, endometriosis, and others in humans.
  - Develop markers for total xenoestrogens or antiandrogen exposure in humans.
- Basic science
  - Molecular studies *in vitro* and with *in vivo* animal models are needed to identify pathways for EDC influence on endocrine tissues.
  - The mechanisms by which EDCs affect neuroendocrine systems need to be ascertained. In addition, studies on EDCs on several of these systems are very underrepresented, and these fields need to be expanded.
  - Roles of steroid and nonsteroid pathways need to be better differentiated and ascertained.
  - More information on low-dose effects of EDCs and their mechanisms is needed.
  - The transgenerational, epigenetic effects of EDCs need to be much more broadly studied across different endocrine and reproductive systems.
  - The interaction of EDCs with central nervous system developmental processes dependent on thyroid hormones (*e.g.*, cochlear development) or sex steroids (*e.g.*, hippocampal development) warrant early *in vitro* and *in vivo* studies.
  - The effects in animal stem cells or progenitor cells in different tissues could decipher EDC-sensitive genes possibly used as reporters in early biomarking.
  - Basic science research on effects of EDCs on diabetes and glucose intolerance is merited.
  - There is a gap in knowledge about the mechanisms by which EDCs act as "obesogens," particularly in how these processes develop.
- Epidemiology
  - Large prospective epidemiological studies need to be undertaken to examine the relationships between EDC exposures, particularly agents with estrogenic and antiandrogenic activity, and relevant endpoints as identified in this report. The National Children's Study will be especially critical to this undertaking.
  - Identify populations or subgroups with high exposures to EDCs and conduct exposure-response studies among these populations.
  - Perform epidemiological studies that incorporate measurement of exposure to multiple EDCs, allowing for the study of human health effects from chemical mixtures.
  - Develop and incorporate validated biomarkers of EDC exposures and relevant outcomes into new and ongoing epidemiological studies.
  - Observations from occupational and environmental exposures in humans and their corresponding disease states should inform what animal studies should be performed and which EDCs should be studied, and should be used to inform policy decisions regarding human exposures to EDCs.
- Clinical practice
  - Set up early detection programs for testis cancer in the follow-up management of infertile men with poor semen quality.
  - Take a careful history of onset of reproductive disorders along with an occupational and environmental exposure history.
  - Think "epidemiologically" about the patients: that is, consider possible exposure to EDCs in geographical or community subgroups showing unexpectedly high prevalence of any of the disorders possibly related to EDCs.
  - Clinicians can advise patients about exposures, minimizing risks, and abiding by the "precautionary principle" to preserve their reproductive health and that of generations thereafter.
  - Health care professionals need to be educated in sources and effects of environmental contaminant exposures *in utero* and across the life span.
  - Health care professionals need to have access to straightforward and accurate health information tools to share with patients.
  - Clinicians should be made aware of the potential risks posed by EDCs. This would, for instance, help them to seek evidence for exposure when treating patients presenting with early thelarche or puberty.

VII. Neuroendocrine Targets of EDCs

The central neuroendocrine systems of the body serve as an interface between the brain and the endocrine systems in the rest of the body. These neuroendocrine systems control diverse functions such as reproduction, stress, growth, lactation, metabolism and energy balance (including thyroid), osmoregulation, and other processes involved in homeostasis. Considering that these neuroendocrine systems mediate the ability of the organism to respond to its environment through rapid (neuronal) and more sustained (endocrine) responses, it is not surprising that they are targeted by environmental EDCs (reviewed in Refs. 7, 278, and 279). Furthermore, neuroendocrine cells in the brain have both neuronal and endocrine properties, which is important in the context of endocrine disruption because EDCs can have neurobiological and neurotoxic effects (279), along with the endocrine effects discussed in this Scientific Statement.

The physiological processes controlled by central neuroendocrine systems are highly complex, making an understanding of neuroendocrine disruption a particular challenge. Each of these neuroendocrine systems comprises several interdependent levels of organization: the brain (specifically the hypothalamus), the pituitary gland, and often a target organ. These levels of organization may each produce a unique hormone(s) or a complex protein (*e.g.*, breast milk), and each level also responds to the hormones produced by the other levels via feedback mechanisms (280). Here, we will discuss the evidence for central neuroendocrine systems as targets for

EDCs (Fig. 1). The bulk of the literature to date has studied primarily the reproductive (HPG) system and secondarily the thyroid neuroendocrine system. The latter will be considered in detail in *Section VIII*, so the former (reproductive neuroendocrinology) will be the focus of the current discussion. Other neuroendocrine systems remain understudied and are only briefly mentioned. Nevertheless, they merit much more investigation in the future.

A. Endocrine disruption of reproductive neuroendocrine systems

1. GnRH neurons

Of the neuroendocrine systems, the reproductive HPG axis is best studied in the arena of endocrine disruption. The control of reproductive neuroendocrine function involves a group of neurons in the basal hypothalamus that synthesize and release the decapeptide GnRH (281). GnRH release drives reproduction throughout the life cycle, and this is the primary stimulus to the rest of the reproductive axis (the pituitary and gonads). GnRH release stimulates gonadotropin release from the anterior pituitary gland, which in turn activates steroidogenesis and gametogenesis in the ovary and testis. Steroid hormones produced by the gonad act on other target tissues that express estrogen, progestin, and/or ARs, a concept that is fundamental to endocrine disruption because so many EDCs act to interfere with steroid hormone actions. A second important concept is that sex steroids also control the hypothalamic GnRH neurons, but this involves indirect effects because GnRH neurons do not express most of the receptors for steroid hormones (282). This introduces the important point that other cells in the brain that express steroid hormone receptors and that regulate GnRH cells through afferent neural inputs are targets for EDCs. These points also relate to evidence that EDCs can act upon neurotransmitter systems that, at first glance, may not seem to have relevance to neuroendocrine control. For example, EDCs have been shown to cause neurotoxicity of noradrenergic, serotonergic, dopaminergic and other neurotransmitter-containing neurons (reviewed in Refs. 2 and 279). Considering that all of these neuronal types have been shown to express steroid hormone receptors and all of these cell types can project to and regulate GnRH neurons (281), this is a mechanism for convergence of effects of EDCs on the link between neural and endocrine systems.

One of the biggest challenges with the neuroendocrine system is gaining access to it. Hypothalamic neuroendocrine cells such as GnRH neurons are located in the hypothalamus at the base of the brain, making them difficult to access in animal models and impossible in humans. The hypothalamic-releasing hormones are not released in sufficiently high quantities to be detectable in peripheral circulation. Therefore, assays of hypothalamic function rely on hormone measurements of their corresponding pituitary hormones. If the pituitary sensitivity to hypothalamic output is compromised, then it is impossible to distinguish a primary hypothalamic or pituitary effect of an EDC. This has necessitated the use of animal models or *in vitro* assays to directly ascertain effects of EDCs on neuroendocrine peptide gene expression or release.

A reliable model for the GnRH system is the hypothalamic GT1 cell lines that have been used for nearly two decades as a proxy for the GnRH neuron *in vivo* (283). For example, PCBs (284) and organochlorine pesticides (methoxychlor, chlorpyrifos) (285) have been tested in this context. Application of these EDCs to GT1 cells caused significant changes in GnRH gene expression, GnRH peptide release, and the morphology of the GT1–7 cells. Interestingly, these substances often acted by nonlinear dose-response curves, with intermediate dosages exerting the greatest effects, typical of hormonally-active substances (286, 287). Moreover, unlike traditional toxicological studies, effects of these environmental contaminants were in many cases stimulatory to the GnRH response. When comparisons were made to estradiol, at least some of the effects of these EDCs mimicked effects of estrogens on GT1 cell morphology, proliferation, and gene expression. In addition, blockade of ERs with ICI 182,780 diminished some of the actions of EDCs. Together, these data suggest that EDCs may directly target GnRH cell lines. Nevertheless, caution must be taken in interpreting these data, because the GT1 cells express some molecules not detectable in the animal's GnRH cell, including some nuclear steroid hormone receptors. Other cell line models for neuroendocrine cells are available and may be useful in screening substances for neuroendocrine disrupting activities.

Studies using explanted hypothalamic dissections in a perifusion model from 15-d-old female rats tested effects of several EDCs on glutamate-evoked GnRH release, the latter model used as a reliable way of stimulating GnRH secretion (288). Of the EDCs tested, o,p′-DDT had the greatest stimulation of glutamate-evoked GnRH release, and BPA had a lesser effect. Not all EDCs were stimulatory; methoxychlor and p,p′-DDE had no effect in this *in vitro* model. Collectively, these data suggest EDC effects on GnRH release in a hypothalamic explant model (288). Finally, antagonists to the ER or AhR blocked effects of DDT, suggesting mediation of these endocrine-disrupting properties by these nuclear receptors and invoking a potential mechanism of action. In another study, this same group showed that DDT, but not DDE, decreased the interpulse interval of GnRH pulses (*i.e.*, increased pulse fre-

quency), again consistent with stimulatory effects of these EDCs on GnRH release (27).

Mammalian *in vivo* studies also implicate GnRH neurons as targets for EDCs. O'Byrne's laboratory (289) has shown that coumestrol suppresses LH release (a proxy for GnRH) and the GnRH pulse generator. Bourguignon's laboratory (27) reported that DDT accelerated the timing of puberty in female rats and altered the LH response to GnRH, although surprisingly, this was decreased in the DDT animals. Not all aspects of GnRH function are affected by all EDCs: Patisaul *et al.* (290) showed that expression of the immediate early gene fos in GnRH neurons was not altered by neonatal genistein or BPA. By contrast, data from Gore's laboratory (291) suggest that EDCs can stimulate GnRH mRNA levels in laboratory rats. In the rabbit, prenatal vinclozolin (an endocrine-disrupting fungicide) decreased numbers of GnRH neurons in selected brain regions (292). Together, these results suggest actions of EDCs on GnRH neurons, although much more research is necessary to reconcile these data and better understand the mechanisms. These findings are not really surprising, considering that GnRH neurons act as the interface between endocrine and neural systems, but they are important because they show this level of regulation with the HPG axis.

Studies in fish also demonstrate effects of EDCs on the GnRH system. The strongest work has been published by the collaboration of Khan and Thomas (293). Using the Atlantic croaker as an experimental model, these labs showed that PCBs decreased preoptic-hypothalamic GnRH content, pituitary GnRH receptors, and the LH response to GnRH challenge (293). This effect was mimicked by an inhibitor of serotonin synthesis suggesting the possible mediation of effects of PCBs by the serotonergic pathway.

2. EDC effects on sexually dimorphic brain regions and behavior

The regions of the hypothalamus that control reproductive neuroendocrine systems undergo development during specific time periods, in large part due to exposures to endogenous steroid hormones such as estrogens and androgens. Although this is a simplification, it is speculated that the brains of male mammals become masculinized and defeminized due to actions of estradiol and testosterone produced by the developing (embryonic and early postnatal) testis. In female rodents, the best-studied model for endocrine disruption, the ovary is relatively quiescent during these developmental periods, and their brains are thought to be feminized and demasculinized due to the relative absence of exposure to these steroid hormones (reviewed in Ref. 294). However, it is important to note that the human ovary does produce estradiol (295), so there are species differences. Nevertheless, the developmental basis of adult disease applies to the development of the reproductive neuroendocrine system through actions during critical periods of sexual differentiation.

It should be apparent that exogenous hormones that may perturb steroidal actions through actions such as binding to steroid receptors, changing steroid metabolism, and others would have effects on the developing neuroendocrine system in a sexually dimorphic manner. There has been considerable and consistent research that shows that PCBs, phytoestrogens, fungicides, pesticides, and other xenobiotics can disrupt brain sexual differentiation (294). This type of disruption has a high likelihood of affecting both reproductive physiology and behavior later in life, and indeed, there is strong evidence in rodent models that reproductive success is diminished as a consequence (reviewed in Ref. 7). Early life exposure (late embryonic and/or early postnatal) to low doses of PCBs (296–298) or soy (299) significantly and adversely affected mating behaviors in female rats. Early postnatal treatment with coumestrol (a phytoestrogen) diminished masculine and feminine sexual behaviors (300, 301). These results are consistent with a functional outcome for effects of EDCs in the neuroendocrine hypothalamus.

A recently published collaborative study demonstrated significant effects of prenatal vinclozolin on mate preference behavior in F3 descendents. In brief, pregnant rats were treated with vinclozolin or vehicle. The F1 vinclozolin male offspring developed latent disease in adulthood, consistent with the developmental basis of adult disease (35). Moreover, this phenotype was passed on to subsequent generations (through F3) via paternal germline transmission, due to epigenetic modification of specific genes. We evaluated the attractiveness of the male F3 vinclozolin descendents in comparison to F3 vehicle descendants in a mate preference test in which females were given the opportunity to spend time with a descendent of both treatments. The results showed a profound difference, with females spending significantly more time with an F3-vehicle compared with an F3-vinclozolin descendant male (302). These results show differences in behavior, as well as evolutionary impact on mating success, caused by endocrine disruption. Notably, these F3 descendants had no personal body burden or exposure to vinclozolin, and it has been postulated that the basis of the discrimination in the mate choice test was due to a transgenerational, epigenetically transmitted trait (302).

B. Hypothalamic-pituitary-adrenal (HPA) effects of EDCs

As articulated by Harvey *et al.* (303), "The adrenal is arguably *the* neglected organ in endocrine toxicology, and the lack of recognition of the importance of adrenal function in a regulatory endocrine disruption context and the need for an adrenal toxicology assessment strategy has

been pointed out." Numerous pharmaceuticals can affect the HPA axis, but this phenomenon has not, to our knowledge, been systematically studied for EDCs. Findings that the HPA axis is sensitive to HPG hormones suggest a potential mechanism by which EDCs may disrupt the HPA axis as well. Alternatively, EDCs may act directly upon the glucocorticoid or mineralocorticoid receptors or on steroidogenic pathways. EDCs including PCBs, dioxin, lindane, and others can affect synthesis of adrenal steroids, but specific effects on the neuroendocrine control of HPA function are lacking (303). This is an important area for future research.

C. Thyroid, metabolism, and growth

The hypothalamic-pituitary-thyroid (HPT) axis provides a critical test of the developmental basis of adult disease hypothesis because normal development and the acquisition of adult functions are dependent upon a euthyroid environment in the developing organism (304). Just a few examples are provided in this section because *Section VIII* provides a comprehensive review of endocrine disruption of thyroid systems. In rats, PCB congeners can affect the HPT axis at several levels, including a reduction in the T_4 or TSH response to TRH (305). Low-dose exposure of pregnant rats to polybrominated diphenyl ether (PBDE) on d 6 of gestation reduced T_4 levels in both dams and offspring (the latter measured on postnatal d 22), although it is unknown whether this is due to direct thyroid or neuroendocrine actions (306). Gray seals with higher blubber concentrations of industrial organochlorine compounds have lower total and free T_3 concentrations (307). Considerably more information on the subject of thyroid disruption is provided in *Section VIII*.

The control of metabolism and energy balance extends well beyond the HPT axis. In the context of endocrine disruption, there are reports on effects of fetal DES, the prototypical estrogenic endocrine disruptor, on obesity in adulthood and even on a successive generation of mice (308). Although the exact mechanisms for such effects are not understood, the fact that the hypothalamus contains a complex neural circuitry that regulates energy and metabolic homeostasis suggests the possibility for this being a neuroendocrine action. Further discussion of this topic is in *Sections VIII and IX* of this Scientific Statement.

To our knowledge, there is little published work on neuroendocrine disruption of somatic growth. Although studies in fish show reductions in the gonadosomatic index, animals exposed to refuse or water waste (309), the mechanism for these effects and the respective roles of the growth, as opposed to the reproductive, axes are not known.

D. Hormonal targets of neuroendocrine disruption

There are both hormonally dependent and independent pathways by which EDCs exert neuroendocrine actions. EDCs may act upon nuclear hormone receptors that are expressed in hypothalamic or pituitary cells, thereby exerting feedback effects. Steroid hormone receptors are expressed abundantly in hypothalamus and other brain areas that control neuroendocrine functions (310–312). Along with "classical" nuclear steroid hormone-mediated actions, EDCs may exert actions via membrane steroid receptors (313, 314) (reviewed in Ref. 315). These and other steroid-sensitive pathways are obvious targets by which EDCs act upon neuroendocrine systems.

The neuroendocrine actions of EDCs may occur via nonhormonally mediated mechanisms. Numerous neurotransmitter systems such as dopamine, norepinephrine, serotonin, glutamate, and others are sensitive to endocrine disruption (reviewed in Ref. 2). This point is important because it explains neurological effects of EDCs on cognition, learning, memory, and other nonreproductive behaviors, but it may also relate to reproductive neuroendocrine systems. As already mentioned, these neurotransmitters may coexpress steroid hormone receptors, so this steroid-sensitive circuitry may be an important target of EDC actions on neurotransmission.

Neuroendocrine systems are critically involved in the control of vertebrate homeostasis and physiology. Although we tend to think of them as independent systems, in fact there is considerable cross-talk among them. This is an important consideration in determining effects of EDCs; whereas no discernible effect may be determined in one system, it is important to evaluate the other systems for subtle but physiologically relevant effects. Therefore, there is a great need for additional interdisciplinary research on effects of EDCs in neuroendocrine systems. However, high-throughput assays for neuroendocrine effects of EDCs are difficult to develop due to the nature of these complex physiological systems. For example, it is impossible to test the "developmental basis of adult disease" hypothesis in a cell line. Animal studies are by nature labor intensive, particularly when they necessitate exposures during critical periods and when performed in species that give birth to litters as opposed to individuals, an intrauterine organization that is very different from the situation in humans. Thus, carefully designed neuroendocrine studies on EDCs in rodents need to take the litter composition and intra- and interlitter variability into consideration.

VIII. Thyroid Disruption

A. Introduction to thyroid systems

Thyroid hormone is essential for normal brain development, for the control of metabolism, and for many aspects of normal adult physiology. Therefore, changes in the function of the thyroid gland or interference with the

ability of thyroid hormone to exert its action may produce effects on development, metabolism, or adult physiology. The goal of this section is to provide a brief overview of the literature regarding the mechanisms by which environmental chemicals may interfere with thyroid hormone action, which will require a brief background of thyroid endocrinology. In addition, we will describe some of the information in humans that indicate the extent to which environmental chemicals may be acting on thyroid hormone signaling in humans.

B. Environmental chemicals impacting thyroid function

A large number of industrial chemicals have been shown to reduce circulating levels of thyroid hormone. Brucker-Davis (316) and Howdeshell (317) have extensively reviewed this topic. Howdeshell categorized these chemicals (more than 150 in all) according to the mechanism by which the chemical was known to cause a reduction in serum thyroid hormone (see Table 1 in Ref. 317 for a full list). This point serves to illustrate clearly that there are many industrial chemicals that can interfere with thyroid function by acting on different points of regulation of thyroid hormone synthesis, release, transport through the blood, metabolism of thyroid hormone, and thyroid hormone clearance. In addition, many natural substances are known to affect thyroid function, including low iodine as well as goitrogens in various foods (318, 319). The current section on thyroid disruption will emphasize the mechanisms by which chemicals are known to interfere with thyroid hormone action and highlight some recent information on the effects of chemicals on thyroid hormone receptors.

The first step in thyroid hormone synthesis is the uptake of iodide into the thyrocyte by the sodium/iodide symporter (NIS) (320). Iodine is essential for thyroid hormone synthesis, and iodine deficiency is an important public health problem worldwide (321). Thus, chemicals that interfere with the NIS may interfere with thyroid hormone synthesis or may exacerbate problems of iodine deficiency. A good example of this is that of perchlorate. This chemical is used as an oxidant in solid rocket propellants, in ordnance, fireworks, airbag deployment systems, and others (322). Because of the environmental stability of perchlorate, it has become a widespread contaminant in drinking and irrigation waters and in food (323), such that perchlorate contamination is nearly ubiquitous in the U.S. population (324). Experimental studies in humans indicate that the serum half-life of perchlorate is about 8 h and that a dose of about 5.2 μg/kg·d is sufficient to begin to reduce iodide uptake into the thyroid gland (325). Thus, it was surprising that Blount *et al.* (326) found that urinary perchlorate levels were associated with serum TSH in the general population of women (not in men). It is perhaps not surprising that this association was greater in women with urinary iodine below 100 μg/liter and stronger still among these women who smoke (327) because cigarettes contain thiocyanates that also inhibit iodine uptake. Because infants are particularly vulnerable to thyroid hormone insufficiency (328) and because perchlorate levels are particularly high in breast milk (329), it is of concern that perchlorate may be affecting thyroid hormone signaling in early infant development in some proportion of the U.S. population (330). However, several studies have failed to identify such a relationship. For example, Amitai *et al.* (331) recently reported that newborn T_4 levels, taken as part of the newborn screening program, were not different on average in babies born in neighborhoods known to be highly contaminated with perchlorate in drinking water compared with babies born in neighborhoods with lower-level perchlorate contamination. These findings are more consistent with a number of studies employing newborn T_4 screening data and location of residence as a proxy measure of perchlorate contamination (for review, see Ref. 322).

There are a number of chemicals that can interfere with iodide uptake by the NIS (332), including chlorate, thiocyanate, and nitrates that are particularly prevalent. It is likely that the effect of one of these chemicals (*e.g.*, perchlorate) on iodide uptake will depend on the presence and concentration of the others and with iodine itself (333).

Iodide, the form of iodine that enters the cell, must be oxidized to a higher oxidation state before it is transferred to the precursor of thyroid hormone, thyroglobulin (334). Of the known biological oxidizing agents, only H_2O_2 and O_2 are capable of oxidizing iodide (335). Organification of iodine is controlled by the enzyme thyroperoxidase (TPO), a heme-containing enzyme. A number of compounds are known to block TPO. A prototypical one is 6-propyl-2-thiouracil (PTU), a methylmercaptoimidazole that has been intensively studied in animals and in humans and is used therapeutically to treat patients with Graves' disease (336). As a class (the 2-mercapto-4-hydroxy-6-propyl-pyrimidines), PTU is representative of compounds found in the environment that can affect thyroid function. PTU is well known to reduce circulating levels of T_4 and T_3 and to increase circulating levels of TSH (405) and has been extensively used in mechanistic research focused on identifying the role of thyroid hormone in brain development. The ability of PTU to reduce circulating thyroid hormone levels has been exploited in the treatment of hyperthyroidism in humans, including in pregnant and lactating women (337). PTU is generally believed to produce deleterious effects in animals by causing a dose-dependent reduction in circulating levels of thyroid hormone. This reduction is caused by the ability of PTU to inhibit directly the function of the TPO enzyme (338).

Other TPO inhibitors include the isoflavones, especially those found in soy protein (*e.g.*, genistein, coumesterol; reviewed in Ref. 339). In humans, goiter has been reported in infants fed soy formula (340–342). In addition, teenage children diagnosed with autoimmune thyroid disease were found to have twice the rate of occurrence if they had consumed soy formula as infants (343). Boker *et al.* (344) recently reviewed the dietary sources of a variety of isoflavones, showing that these are common dietary components. These isoflavones are also so-called "phytoestrogens," which are highly enriched in some commercial preparations.

C. Environmental chemicals impacting thyroid hormone transport, metabolism, and clearance

Once secreted into the blood, thyroid hormones are carried by specific proteins. In humans, about 75% of T_4 is bound to T_4-binding globulin (TBG), 15–20% is bound to transthyretin (TTR; also called T_4 binding prealbumin or TBPA), and the remaining 5–10% is bound to albumin or is free (0.02%) (345, 346).

The role of serum binding proteins for thyroid hormone in thyroid homeostasis is not well understood. No single serum T_4 binding protein is essential for good health or for the maintenance of a euthyroid state in humans (347). There are a number of clinical situations in which serum binding proteins are elevated or reduced (even completely absent) and the thyroid state is normal. Therefore, despite large increases or decreases in serum total T_4 and T_3 concentrations in some of these patients, serum free hormone and TSH are normal (348). In contrast, there is evidence that the role of serum binding proteins such as TBG is to allow the equal distribution of hormone delivery to a tissue. Mendel *et al.* (349) found that ^{125}I-T_4 was evenly distributed in the rodent liver after a single pass through the tissue only if serum binding proteins were present in the perfusate. However, the identity of the serum binding protein (*e.g.*, TTR *vs.* TBG) did not alter the pattern or intensity of T_4 uptake.

There is some evidence that TTR is important in transport of thyroid hormone across the blood-brain barrier. In large part, this concept is derived from the observation that TTR is produced in the choroid plexus (350–352). However, this concept is not supported by the observation that mice carrying a targeted deletion of the TTR gene have normal concentrations of T_4 in the brain (353, 354). A number of chemicals have been shown to displace T_4 from TTR *in vitro*. In fact, some chemicals bind to TTR with higher affinity than does T_4 itself (355–357); however, the consequences of this binding are not completely clear. One hypothesis is that chemicals can reduce serum total T_4 levels by inhibiting T_4 binding to TTR (358). Perhaps this displacement may also increase T_4 clearance by the liver. However, TTR in the choroid plexus appears to be important for thyroid hormone action in the brain (359), and TTR may mediate transport of environmental chemicals into various compartments such as placenta (360). Thus, chemical binding to the TTR may not only decrease the availability of thyroid hormone to various tissues, it may also selectively target these chemicals for transport and uptake.

Many chemicals are known to decrease the serum half-life of T_4 by inducing liver enzymes that glucuronidate T_4 (361–363). These enzymes uridine diphosphate glucuronyl transferase can be induced by dioxin-like compounds acting on the AhR or through the pregnane X-receptor or constitutive androstane receptor nuclear receptors (364). These chemicals fall into many industrial categories including pesticides of many types (365). The classes of industrial chemicals known to interact with the thyroid system have been reviewed previously and will not be emphasized here (see Refs. 316, 317, 365, and 366).

Once in the serum, thyroid hormones can be taken up into tissues by selective transporters (367) to enter cells. This issue has been particularly investigated because the finding that children with a genetic defect in the MCT8 gene exhibit severe neurological and behavioral disorders (Allan-Herndon-Dudley syndrome; Ref. 368). There are a number of transporters that are likely to be important in the control of thyroid hormone uptake into various tissues and cells. However, little is known— or has been tested— about the ability of specific environmental or industrial chemicals to interfere with T_4 or T_3 transporter function.

Inside the cell, T_4 can be converted to T_3 by the type 1 or type 2 deiodinase. These outer-ring deiodinases are essential for thyroid hormone action (369). For example, the type 2 deiodinase knockout mouse exhibits a form of pituitary resistance to thyroid hormone negative feedback in which both serum T_4 and TSH are elevated (370), indicating that the conversion of T_4 to T_3 in pituitary cells is an important step in thyroid hormone action. A number of environmental chemicals affect deiodinase activity including PCBs (360, 371, 372) and others (317). Environmental chemicals that affect deiodinase activity may have effects that are not entirely consistent with the appearance of "hypothyroidism" and, therefore, may be difficult to recognize in the absence of mechanistic studies.

D. Environmental chemicals impacting the thyroid hormone receptor

1. PCBs

Despite early speculations that environmental chemicals may act as imperfect thyroid hormone analogs (373), few studies had tested this hypothesis until recently. Now,

several recent reports show that a broad range of chemicals to which humans are routinely, and inadvertently, exposed can bind to TRs and may produce complex effects on thyroid hormone signaling. Perhaps the best example is that of PCBs—industrial chemicals consisting of paired phenyl rings with various degrees of chlorination (374). Although the production of PCBs was banned in the mid 1970s, these contaminants are routinely detected in the environment (375) and in human tissues (376). PCB body burden is associated with lower full-scale IQ, reduced visual recognition memory, attention deficits, and motor deficits (377–381).

PCBs can reduce circulating levels of T_4 in animals (382–384), and some authors propose that PCBs exert neurotoxic effects on the developing brain by causing a state of relative hypothyroidism (385, 386). In addition, PCB body burden has been found to be associated with thyroid hormone in some, but not all, human studies (366, 387). Interestingly, measures of thyroid function at birth are associated with maternal, infant, and delivery factors, and this may explain why some studies fail to identify an association between PCB exposures and measures of thyroid function at birth (388, 389).

The concept that PCBs can exert a neurotoxic effect on the developing brain by causing a state of relative hypothyroidism is supported by the observations that the ototoxic effect of PCB exposure in rats can be partially ameliorated by T_4 replacement (390), and that the cerebellum, a tissue highly sensitive to thyroid hormone insufficiency (391), is targeted by PCB exposure. PCBs alter motor behavior associated with cerebellar function, as well as cerebellar anatomy (392). Interestingly, PCB exposure is associated with an increase in expression of glial fibrillary acidic protein (392), which is also increased by thyroid hormone insufficiency (393). Finally, in young children, the association between PCB body burden and behavioral measures of response inhibition is stronger in those children that have a smaller corpus callosum (394), an area of the brain affected by thyroid hormone (395). Thus, it is possible that PCBs exert at least some neurotoxic effects on the developing cerebellum by causing a state of relative hypothyroidism.

However, PCB exposure does not produce consistent effects on animals that are indicative of thyroid hormone insufficiency, such as body weight gain during development (382) or the timing of eye opening (390). In addition, despite the reduction in serum T_4, PCB exposure increases the expression of several thyroid hormone-responsive genes in the fetal (396, 397) and neonatal (382) brain. These observations are consistent with the hypothesis that at least some individual PCB congeners, or their metabolites, can act as TR agonists *in vivo*. Recently, Kitamura *et al.* (398) reported that nine separate hydroxylated PCB congeners can bind to the rat TR with an IC_{50} as low as 5 μM. In addition, using a human neuroprogenitor cell line, Fritsche *et al.* (399) found that a specific PCB congener could mimic the ability of T_3 in increasing oligodendrocyte differentiation and that this effect was blocked by the selective TR antagonist NH3. Finally, Arulmozhiraja and Morita (400) have identified several PCB congeners that exhibit weak thyroid hormone activity in a yeast two-hybrid assay optimized to identify such activity.

Not all recent reports indicate that PCBs act as agonists on the TR. Kimura-Kuroda *et al.* (401) have found that two separate hydroxylated PCBs interfere with T_3-dependent neurite outgrowth in mouse cerebellar granule cell primary cultures. In addition, Bogazzi *et al.* (402) found that a commercial mixture of PCBs (Aroclor 1254) exhibited specific binding to the rat TRβ at approximately 10 μM. This concentration inhibited TR action on the malic enzyme promoter in a chloramphenicol acetyltransferase assay, and this effect required an intact thyroid response element (TRE). However, the PCB mixture did not alter the ability of TR to bind to the malic enzyme TRE in a gel shift assay. In contrast, Iwasaki *et al.* (403) found that a specific hydroxylated PCB congener inhibits TR-mediated transcriptional activation in a luciferase assay at concentrations as low as 10^{-10} M. This effect was observed in several cell lines, but was not observed using a glucocorticoid response element. Miyazaki *et al.* (404) followed this report by showing that PCBs can dissociate TR:retinoic X receptor (RXR) heterodimers from a TRE.

It is clear that PCBs are neurotoxic in humans and animals and that they can interact directly with the TR. However, the consequences of PCB exposure on TR action appear to be quite complex. This complexity includes acting as an agonist or antagonist and may include TR isoform selectivity inasmuch as most studies have been performed using the TRβ, leaving the TRα relatively unstudied in this context. In addition, considering that there are 209 different chlorine substitution patterns on the biphenyl backbone and that these can be metabolized [hydroxyl and methylsulfonyl metabolites (173, 174)], it is possible that different chemical species exert different effects. Finally, PCBs may exert different actions on TRs depending on associated heterodimer partners, promoter structure, or different cofactors. This complexity will be important to pursue because the effect of PCB exposure in humans is far better studied than for structurally related compounds such as PBBs and PBDEs. Thus, mechanistic studies on PCBs can be more easily and effectively coupled to specific human health outcomes.

2. BPA

BPA (4,4′ isopropylidenediphenol) is produced at a rate of more than 800 million kilograms annually in the United

States alone (418) and is used primarily in the manufacture of plastics including polycarbonate plastics, epoxy resins that coat food cans, and in dental sealants (406, 407). Howe *et al.* (406) estimated human consumption of BPA from epoxy-lined food cans alone to be about 6.6 μg per person per day. BPA has been reported in concentrations of 1–10 ng/ml in serum of pregnant women, in the amniotic fluid of their fetus, and in cord serum taken at birth (71, 408). Moreover, BPA concentrations of up to 100 ng/g were reported in placenta (408). BPA is also halogenated (brominated or chlorinated) to produce flame retardants. Tetrabromobisphenol A (TBBPA) is the most commonly used, with more than 60,000 tons produced annually (409, 410). Thomsen *et al.* (411) recently reported that brominated flame retardants, including TBBPA, have increased in human serum from 1977–1999 with concentrations in adults ranging from 0.4 to 3.3 ng/g serum lipids. However, infants (0–4 yr) exhibited serum concentrations that ranged from 1.6 to 3.5 times higher (411).

Considering this pattern of human exposure, it is potentially important that BPA has been shown to bind to the TR (412). Although best studied for its actions on the nuclear ER (413), binding with a K_i of approximately 10^{-5} M (414, 415), and more recently for the membrane ER (416), BPA also binds to and antagonizes T_3 activation of the TR (412, 417) with a K_i of approximately 10^{-4} M. As little as 10^{-6} M BPA significantly inhibits TR-mediated gene activation (412). Moreover, Moriyama *et al.* (412) found that BPA reduced T_3-mediated gene expression in culture by enhancing the interaction with nuclear receptor corepressors. Interestingly, Zoeller *et al.* (418) found that developmental exposure to BPA in rats produces an endocrine profile similar to that observed in thyroid resistance syndrome (419). Specifically, T_4 levels were elevated during development in the pups of BPA-treated animals, but TSH levels were not different from controls (418). This profile is consistent with BPA inhibition of TRβ-mediated negative feedback. However, the thyroid hormone-response gene RC3 was elevated in the dentate gyrus of these BPA-treated animals (418). Because the TRα isoform is expressed in the dentate gyrus, the authors concluded that BPA could be a selective TRβ antagonist *in vivo*.

If BPA acts as a TR antagonist *in vivo*, it is predictable that specific developmental events and behaviors would be affected by developmental exposure to BPA. In this regard, Seiwa *et al.* (420) have shown that BPA blocks T_3-induced oligodendrocyte development from precursor cells. In addition, there may be an association between the thyroid resistance syndrome and attention deficit-hyperactivity disorder in humans (421) and in rats (422); therefore, it is potentially important that BPA-exposed rats exhibit attention deficit-hyperactivity disorder-like symptoms (423).

Despite the antagonistic effects of BPA on the TRβ, halogenated BPAs appear to act as TR agonists (417). Both TBBPA and tetrachlorobisphenol A can bind to the thyroid hormone receptor and induce GH3 cell proliferation and GH production (417). Thus, these compounds may exert agonistic effects on the TR, and this could be important during early brain development. For example, thyroid hormone of maternal origin can regulate gene expression in the fetal brain (424–426); one of these genes codes for Hes1 (397). Considering the role of HES proteins in fate specification in the early cortex (427, 428), the observation that industrial chemicals can activate the TR and increase HES expression (397) may indicate that these chemicals can exert subtle effects on early differentiative events.

3. PBDEs

PBDEs may also bind to the thyroid hormone receptor (reviewed in Ref. 429). Marsh *et al.* (430) demonstrated that two hydroxylated PBDEs can bind to both TRα and TRβ, but with a significant preference for TRβ.

IX. Environmental Chemicals, Obesity, and Metabolism

A. Introduction to EDCs and the obesity epidemic

Obesity, defined as body fat greater than 25% in men or greater than 30% in women, is fast becoming a significant human health crisis (431). More than 30% of adults in the United States are defined as clinically obese (431, 432), and an analogous rise is observed in pediatric populations, with a tripled increase in the obesity rate from ages 6–19 yr during the last five decades (433). The prevalence of obesity has risen dramatically in wealthy developed countries, and it is also on the rise in poor nations. The WHO has declared excessive weight as one of the top 10 health risks in the world and has estimated that the number of overweight people in the world is now greater than the number of undernourished. The rise in the incidence in obesity matches the rise in the use and distribution of industrial chemicals that may be playing a role in generation of obesity (434), suggesting that EDCs may be linked to this epidemic.

Obesity has deleterious effects on human health by increasing the risk of associated metabolic abnormalities such as insulin resistance, hyperinsulinemia, hypertension, and dislipidemia—all components of the metabolic syndrome—which constitute, in turn, major risk factors for the development of diabetes mellitus type 2 and coronary heart disease. The etiology of the obesity epidemic has been partly attributed to alterations in food intake, with the prevalence of a Westernized-style diet characterized by high caloric uptake as well as a lack of physical

activity representative of a sedentary lifestyle. However, the mechanisms still remain unclear, and except for a genetic predisposition and lifestyle modifications, scientific research implies the impact of environmental substances in the generative roots of obesity. Grün and Blumberg (435, 436) have coined the terminology "obesogens" in reference to molecules that inappropriately regulate lipid metabolism and adipogenesis to promote obesity.

Obesity also relates to the fetal (developmental) origins of adult disease. Children of women who experienced famine during pregnancy exhibit symptoms of the metabolic syndrome as adults (437). Moreover, it is becoming evident that an important risk factor for development of this metabolic syndrome is low birthweight (438, 439). These studies indicate that developmental events occurring *in utero* and perhaps in the immediate perinatal period can affect metabolic functions that can lead to the metabolic syndrome in adulthood (431).

B. Environmental estrogens and obesity

White adipose tissue metabolism is under the control of the sympathetic nervous system and is modulated by hormones including sex steroids. The impact of environmental estrogens on adipose tissue may be through direct modulation of lipogenesis, lipolysis, and adipogenesis, or indirect by affecting food consumption and leptin secretion targeting the central nervous system or lipid homeostasis in liver (440).

The estrogenic pharmaceutical chemical DES illuminates the relationship between perinatal exposures and latent development of high body weight and obesity. Moreover, there is a complex relationship between the concentration of estrogen to which pregnant animals are exposed and the weight of the offspring in adulthood (432). Specifically, according to a recent experiment by Newbold *et al.* (432), mice neonatally exposed to DES experience increased body weight in adulthood associated with excess abdominal body fat. Interestingly, the dose of DES determines the chronic manifestation of the observed alterations, with high doses leading to initially decreased body weight and a peripubertal "catch-up" and low doses causing an increase in weight detectable only in adulthood. Moreover, the timing is important because gestational administration in rodents results in the offspring's low birth weight, an unchanged metabolic characteristic throughout life (432). Along with an increase in body fat stores, the adipokines leptin and adiponectin, IL-6 (an inflammatory marker), and triglycerides were all elevated in DES-exposed mice (432).

An *in vitro* study using a culture system of 3T3-L1 preadipocytes showed that 4-nonylphenol and BPA stimulated lipid accumulation, accelerating their differentiation to mature adipocytes in a time- and concentration-dependent way (441). The underlying mechanism appeared to involve up-regulation of gene expression involved in lipid metabolism and adipocyte differentiation. In the second part of the experiment, fat accumulation was observed in human hepatocellular carcinoma cell lines exposed to those endocrine disruptors (441). These findings are consistent with previous *in vitro* studies using mouse fibroblast cell lines in which a link between environmental chemicals including nonylphenol, BPA, and genistein in the development of body weight imbalance was suggested (431, 432).

C. Peroxisome proliferator-activated receptor (PPAR) γ and organotins

PPARγ is a member of the nuclear receptor superfamily and constitutes a major regulator of adipogenesis. It is primarily expressed in adipose tissue, and its activation promotes adipocyte differentiation as well as the induction of lipogenic enzymes. Additionally, it contributes to maintenance of metabolic homeostasis through transcriptional activation of genes implicated in energy balance (442). During its activation, PPARγ forms a heterodimer with RXR-α, and the complex binds to PPAR response elements in the regulatory regions (promoters) of target genes ultimately involved in the regulation of fatty acid storage and the repression of lipolysis.

Experimental evidence highlights that nuclear receptor superfamily and specifically PPARγ are molecular targets for endocrine disruptors, in particular organotin compounds such as tributyltin (TBT) and triphenyltin, which have been widely used in agriculture and industry. Kanayama *et al.* (443) showed that TBT and triphenyltin functioned as agonists of PPARγ and RXR, acting as high-affinity ligands at levels comparable to known endogenous ligands. Moreover, administration of those xenobiotics in preadipocyte cell lines resulted in adipocyte differentiation through PPARγ (443). In mice, TBT induced the differentiation of adipocytes *in vitro* and increased adipose mass *in vivo* by RXR and PPARγ activation (444).

It is possible that PPARγ signaling can interact with that of estrogen to influence adipogenesis. These findings have been reviewed recently (435, 436, 444) and represent an important example of the mechanism by which environmental chemicals can interfere with body weight regulation. In addition, at high doses, TBT can inhibit aromatase enzyme activity in adipose tissue directly, leading to decreased estradiol levels and down-regulation of ER target genes. TBT at moderate to high doses inhibits the activity of 11β-hydroxysteroid dehydrogenase 2, resulting in decreased inactivation of cortisol. It has been hypothesized that the increased local glucocorticoid levels could influence late stages in adipocyte differentiation and thus, metabolic regulation (435, 436).

D. Phytoestrogens

In recent years, efforts to implement healthier eating habits have resulted in an increased consumption of soy products and supplements and hence, increased exposure to phytoestrogens. Genistein is the principal phytoestrogen in soy and has a wide range of biological actions. It binds to ERα and ERβ but also displays antiestrogenic action (445). At low concentrations, genistein was found to act as estrogen and exert an inhibitory effect on lipogenesis. There are also sex differences in the effect of genistein on adipose deposition and insulin resistance, an effect that involves the ERβ (446). At higher concentrations, genistein promotes lipogenesis through the molecular pathway of PPARγ, an ER-independent pathway (445).

E. Endocrine disruptors, diabetes, and glucose homeostasis

The incidence of diabetes mellitus has tripled over recent decades, with an estimated 177 million people affected worldwide (447). It is speculated that by the year 2030 the prevalence of diabetes will increase to 4.4% worldwide (from 2.8% in 2000) with more than 300 million diabetic adults (448). Regarding the young population, epidemiological studies show an alarming increase in the incidence of diabetes mellitus type 2 (449).

Based on the links between endocrine disruptors and disturbances of reproduction, metabolism, and links to adult dysfunctions and cancer, it is reasonable to propose a connection between EDCs and diabetes as well as prediabetic disturbances. Indeed, epidemiological studies have linked high dioxin levels with increased risk for diabetes or altered glucose metabolism (450). Animal models also support this hypothesis. Alonso-Magdalena *et al.* (447) undertook an *in vivo* experiment to evaluate the impact of BPA on pancreatic β-cell function. Its biological action was compared with 17β-estradiol. The results showed that acute treatments with either estradiol or BPA caused a temporary hyperinsulinemia, whereas longer-term exposure provoked insulin resistance with chronic increased insulin levels, an aggravating factor for the development of diabetes mellitus (447). Recently, in conditioned media from human breast, sc and visceral adipose explants, it was demonstrated that BPA at environmentally relevant doses (0.1 and 1 nM) inhibits the release of adiponectin, an adipocyte-specific hormone that increases insulin sensitivity. Therefore, factors that suppress adiponectin release could aggravate insulin resistance and susceptibility to obesity-related syndromes like metabolic syndrome and type 2 diabetes mellitus. However, the mechanisms by which BPA suppresses adiponectin and the receptors involved remain to be determined (451).

Pancreatic α-cells have also been suggested as potential targets for endocrine disruption. Low doses of BPA and DES were shown to impair the molecular signaling that leads to secretion of glucagon by suppressing intracellular calcium ion oscillations in α-cells in response to low blood glucose levels through a nongenomic mechanism (452).

The above experiments suggest that low doses of endocrine disruptors can disrupt pancreatic physiology, affecting both insulin- and glucagon-secretory cells, leading to changes in the regulation of glucose and lipid metabolism. The underlying mechanisms involve at the very least classical ER-mediated but also nongenomic actions. Further investigations are required to elucidate the potential associations with human health. Importantly, whereas current evidence represents experimental data from laboratory animals or *in vitro* studies, no direct association with humans has yet been established, with the exception of the epidemiological studies discussed above.

F. Endocrine disruptors and cardiovascular systems

The obesity phenotype may lead to a dysmetabolic state with atherogenic, inflammatory, prothrombotic abnormalities that not only accelerate the progression of cardiovascular disease but also create favorable "subsoil" for an acute myocardial infarct (453). Therefore, the cardiovascular system is also a target of environmental chemicals that interfere with intracellular signaling of hormonal and inflammatory pathways.

G. Estrogenic EDCs and cardioprotection

Phytoestrogens have been shown to exert cardioprotective effects. Female rats fed a high phytoestrogen diet exhibited cardioprotection against adverse left ventricular remodeling (454) and reduction of myocardial necrosis, increased myocardial contractility, and decreased occurrence of ventricular arrhythmias. Genistein was also associated with reduced levels of TNF-α and blunted myocardial intercellular adhesion molecule-1 expression (455). Moreover, it was shown that in people at high risk of cardiovascular events, a greater isoflavone intake is associated with better vascular endothelial function and lower carotid atherosclerotic burden (456).

Regarding human populations, there are some epidemiological studies that suggest that high phytoestrogen intake is inversely associated with cardiovascular risk factors and development of cardiovascular disease (457). Moreover, it was shown that in people at high risk of cardiovascular events, a greater isoflavone intake is associated with better vascular endothelial function and lower carotid atherosclerotic burden (456). However, these epidemiological observations need clinical confirmation.

H. Advanced glycation end-products (AGEs)

Recent data clearly suggest that a heterogeneous group of exogenous advanced glycation end-products (AGEs)

have a negative impact on cardiometabolic tissues. Tobacco use (458) and food cooked at high temperatures, precooked meals, and some beverages contain large amounts of AGEs that are absorbed from the human gastrointestinal tract (459). AGEs cause tissue injury through intracellular generation of free radicals and triggering oxidative stress, through the interaction of AGEs with a multiligand, cell surface receptor called RAGE, and endocrine signaling pathway. There is evidence in experimental animals and humans for a link between exogenous AGEs and an increase in cardiometabolic risk markers. It is notable that mice chronically fed a high-AGE diet, compared with those fed a low-AGE, high-fat diet exhibited relative insulin resistance accompanied by modifications in pancreatic cellular architecture compatible with hyperplasia and hypertrophy and loss of islet of Langerhans structure (460). Another *in vivo* chronic experiment involved feeding intact female rats a high-AGE diet for 6 months. This resulted in increased fasting glucose and insulin levels independent of the degree of obesity as well as hormonal alterations (461). Uribarri *et al.* (462) showed that a single oral administration of an AGE-rich beverage acutely (within 90 min) resulted in temporarily impaired endothelial function assessed by flow-mediated arterial vasodilation, increased serum C-reactive protein, and plasminogen activator inhibitor-1 levels in both diabetic and healthy subjects.

I. Conclusions

The literature demonstrates a role of EDCs in the etiology of complex diseases such as obesity, diabetes mellitus, and cardiovascular disease, yet these processes are still poorly understood. Although the evidence is limited, accumulating data are pointing to the potential role of endocrine disruptors either directly or indirectly in the pathogenesis of adipogenesis and diabetes, the major epidemics of the modern world. Taking into consideration the wide spectrum of industrial chemicals to which an average consumer might be exposed, a rational hypothesis is that the scientific community may inadvertently ignore the effect of several other compounds that might in turn constitute potential "obesogens" or promoters of glycemic disturbances. Further research is required to elucidate all potential interactions between environmental substances and metabolic dysregulation.

X. Recommendations for the Future

A. Linking basic research to clinical practice

It should be clear from this Scientific Statement that there is considerable work to be done. A reconciliation of the basic experimental data with observations in humans needs to be achieved through translation in both directions, from bench to bedside and from bedside (and populations) to bench. An example of how human observation and basic research have successfully converged was provided by DES exposure in humans, which revealed that the human syndrome is faithfully replicated in rodent models. Furthermore, we now know that DES exposure in key developmental life stages can have a spectrum of effects spanning female reproduction, male reproduction, obesity, and breast cancer. It is interesting that in the case of breast cancer, an increased incidence is being reported now that the DES human cohort is reaching the age of breast cancer prevalence. The mouse model predicted this outcome 25 yr before the human data became available. In the case of reproductive cancers, the human and mouse data have since been confirmed in rats, hamsters, and monkeys (463). This is a compelling story from the perspective of both animal models and human exposures on the developmental basis of adult endocrine disease.

Another estrogenic compound, BPA, is also linked to a wide variety of endocrine dysfunction. BPA exposure, particularly in development, increases the risk of mammary cancer, obesity, diabetes, and reproductive and neuroendocrine disorders. The human evidence for BPA is mounting; recently, Lang *et al.* (464) published a cross-sectional analysis on the relationship between concentrations of urinary BPA and chronic disease states in over 1400 adults in the United States. They found a significant correlation between BPA concentrations in urine with cardiovascular disease and abnormal concentrations of liver enzymes. It would be really interesting to be able to relate the relationship of these outcomes with developmental/fetal exposure to BPA and other xenobiotics. However, epidemiological research on fetal exposure would be logistically difficult and costly because exposures must be measured at several different time points, including gestation, whereas the outcome may not be manifest in some cases until 50 or more years after the initial fetal exposure. Given the reproducibility of the human DES syndrome in rodents and recent evidence for commonalities in a relationship between BPA and cardiovascular endocrine disease, it is obvious that more research in animal models is necessary to enrich our knowledge of the mechanisms by which endocrine disruptors increase the risk of disease.

A challenge to understanding the relationship between EDCs and health abnormalities is that EDCs are a "moving target." Individuals and populations are exposed to ever-changing patterns of production and use of these compounds. They also tend to be released into the environment as mixtures, rather than individual chemicals. Therefore, it is important to understand the effects of simultaneous coexposures to these chemicals, which may interact additively, multiplicatively (synergistically), or

antagonistically (48). There are limited data on the interactions between chemicals within a class or across classes of chemicals. Presently, there are good analytical methods for measuring exposures to a variety of endocrine disruptors in humans. An increased understanding of the potential human health risks of exposure to mixtures of EDC is important but remains very understudied. Hence, measurement of body burden of the most prevalent xenobiotics would probably be the best strategy for finding a link between exposure and effect. Once known, this could be related to mechanistic studies in laboratory models, and future experiments could be designed to evaluate the effects of combinations of common EDCs in the laboratory, with the obvious caveat that it will not be possible to mimic every possible combination and dose. Despite these challenges, evolving and innovative technologies designed to improve the assessment of human exposure and reproductive and endocrine health endpoints should provide enhanced opportunities for improving our understanding of these relationships.

B. Endocrine disruption and the public

At the recent Summit on Environmental Challenges to Reproductive Health and Fertility at the University of California, San Francisco, recommendations were made regarding future research, health care, policy, community action, and occupational health (49). Included in these recommendations were enhancing collaborations among and between researchers and granting agencies and promoting critical research directions, including prenatal exposures in the National Children's Health Study, leveraging specific laboratory data into the National Health and Nutrition Examination Survey study, developing biomarkers of exposure and disease, and increasing the funding for effects of chemicals on the epigenome, developmental programming, transgenerational effects, and cross-talk among endocrine systems and metabolic and immune systems (49). In addition, for health care professionals, being educated in sources and effects of environmental contaminant exposures *in utero* and across the life span, as well as having straightforward health information tools to share this information with patients and for public education in general are recommended.

C. Prevention and the "precautionary principle"

Although more experiments are being performed to find the hows and whys, what should be done to protect humans? The key to minimizing morbidity is preventing the disorders in the first place. However, recommendations for prevention are difficult to make because exposure to one chemical at a given time rarely reflects the current exposure history or ongoing risks of humans during development or at other life stages, and we usually do not know what exposures an individual has had *in utero* or in other life stages.

In the absence of direct information regarding cause and effect, the precautionary principle is critical to enhancing reproductive and endocrine health (49). As endocrinologists, we suggest that The Endocrine Society actively engages in lobbying for regulation seeking to decrease human exposure to the many endocrine-disrupting agents. Scientific societies should also partner to pool their intellectual resources and to increase the ranks of experts with knowledge about EDCs who can communicate to other researchers, clinicians, community advocates, and politicians.

D. Specific recommendations for future research

Although direct causal links between exposures to EDCs and disease states in humans are difficult to draw, results from basic research and epidemiological studies make it clear that more screening for exposures and targeting at-risk groups is a high priority. In addition to enhancing research in these areas, an important and effective approach is prevention of disease. Our chemical policies at local, state, and national levels, as well as globally, need to be formulated, financed, and implemented to ensure the best public health. Additional specific recommendations of this group are shown in Box 2. By communicating these priorities to basic and clinical researchers, physicians, community advocates, and the public at large, we are hopeful that early identification and intervention will be facilitated.

Acknowledgments

We thank Dr. Loretta Doan for the critical role that she played in facilitating all communications of this taskforce; Dr. Alan Schneyer for support and guidance; Ms. Maggie Haworth for expert assistance with publication-related activities; the Research Affairs Core Committee of The Endocrine Society; and The Scientific Statements Taskforce of The Endocrine Society.

Address all correspondence and requests for reprints to: Andrea C. Gore, Ph.D., The University of Texas at Austin, College of Pharmacy, 1 University Station, A1915, Austin, Texas 78712. E-mail: andrea.gore@mail.utexas.edu.

This work was supported by Grants NIH ES07784 (to A.C.G.), NIH ES015584 (to G.S.P.), NIH ES09718 (to R.H.), NIH ES01230, ES015182, ES08314 (to A.M.S.), European Commission (EDEN project, QLRT-2001-00269), Belgian Study Group for Pediatric Endocrinology, Belgian Fonds de la Recherche Scientific Medicale, grants 3.4567.09 and 3.4573.05 (to J.P.B.).

Disclosure Summary: E.D.-K., J.-P.B., L.C.G., R.H., G.S.P., and A.C.G. have nothing to declare. A.M.S. has served as a consultant to Emerson Poynter LLP. R.T.Z. has served on the EPA Scientific Advisory Board, has received honoraria for university lectures, and has served as an expert witness in federal court.

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Final Report
2010–2016

Pictou Landing Native Women's Group
and
H. Castleden, D. Lewis, R. Jamieson, M. Gibson,
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Prepared for:
Pictou Landing First Nation

How to cite this report: Pictou Landing Native Women's Group (c/o Sheila Francis, Past President), Castleden, H., Lewis, D., Jamieson, R., Gibson, M., Rainham, D., Russell, R., Martin, D., & Hart, C. (2016). "Our Ancestors Are in Our Land, Water, and Air": A Two-Eyed Seeing Approach to Researching Environmental Health Concerns with Pictou Landing First Nation – Final Report.

Executive Summary

A'se'k, "the other room," is the tidal estuary commonly known today as Boat Harbour, adjacent to Pictou Landing First Nation on the Northumberland Strait of Nova Scotia, Canada. Historically, A'se'k was a gathering place that exemplified the Mi'kmaw ethic of sharing: food, knowledge, and skills were exchanged there between generations and amongst family groups. These uses of the land and its recreational, physical, mental, spiritual, cultural, and emotional purposes have been compromised since a pulp and paper mill was built in 1965 and began operating in 1967.

Almost 50 years later, the effects of the pollution on the surrounding land, air, soils, and water are not fully known and understood. Out of concern for their children, families, and community, the women of the Pictou Landing Native Women's Group (PLNWG) mobilized around the issue of Boat Harbour in 2010, inviting academic members to join them in exploring their question, "Is Boat Harbour making us sick?" It is due to their initial collective and continual organizing that the research this report describes was carried out.

This multi-year community-based participatory project, co-led by Past PLNWG President Sheila Francis and Dr. Heather Castleden (formerly with Dalhousie University, now at Queen's University), employed a Two-Eyed Seeing approach. Dr. Debbie Martin, with her expertise in this approach of bringing Indigenous and Western knowledge systems (ways of seeing the world and ways of doing research) together, was consulted in the development of our research design and continually throughout the project.

Four Research Retreats and five Community Dinners provided opportunities to share and discuss community-relevant concerns of environment and health in a multi-generational and female-centred forum. This, in turn, helped establish, revisit, reconfirm, and revise goals and priorities throughout the research partnership between and within the academic members of the team and the PLNWG. Monthly one-page updates were sent to Pictou Landing First Nation, c/o Chief Andrea Paul.

The Two-Eyed Seeing approach resulted in the use of diverse methods, including documenting oral histories; conducting a comprehensive literature review; establishing a web-based community map; carrying out a Youth Camp; creating digital stories; conducting a community-wide Environmental Health Survey, deploying an array of environmental monitoring techniques including water, air, soil, and sediment sampling and analysis as well as mammal analysis and dendrochronology; and sharing our new knowledge across multiple platforms. The varied methods allowed for a broad assessment of health concerns; they intersect and overlap to inform a wholistic understanding of individual and collective health in Pictou Landing First Nation.

Oral histories, collected in large part by Ms. Ella Bennett, illustrated intergenerational impacts as Elders expressed their concerns over the decline of their community's engagement with the land and waterways that they have witnessed since the start of the pulp and paper mill's operations and use of the Boat Harbour Effluent Treatment Facility. Our literature review, conducted in large part by Ms. Ziyun Wang, found that, unsurprisingly, the limited research to date has focused on physical health impacts and concerns. The review also identified three gaps in the literature: (1) legislation in Nova Scotia has never (and does not) require human health risk assessments; (2) regulations left a gap of more than 20 years when the mill operated without effluent control; and (3) little research has been conducted on the topic of tidal flush.

The Environmental Health Survey, led by Ms. Diana Lewis, assessed community-wide environmental and human health concerns, perceptions, and experiences. The face-to-face survey, conducted by members of the PLNWG, reflected questions that the PLNWG wanted information on and received an exceptional 60% response rate. Findings are highlighted here; a detailed Community Report of the survey results is forthcoming. While it is clear that Pictou Landing First Nation members have been experiencing poorer health outcomes compared to other First Nations people living on reserve both provincially and nationally, they have not been the beneficiaries of the socioeconomic benefits that typically come with, and are often touted as the impetus for, regional industrial development.

Dr. Mark Gibson led air quality sampling in summer 2013 and spring and summer 2014 using passive and real-time samplers across 13 sites. Ammonia, nitrogen dioxide, sulfur dioxide, volatile organic compounds, particulate matter, and dioxins and furans were monitored and all of the air quality data were found to be below the Canadian Council of Ministers of the Environment's (CCME) limits. That said, Dr. Gibson noted that there were certainly challenges to monitoring air quality, including equipment malfunction and the unknown impact on the data of the mill's closure during summer 2014 after the effluent spill.

Dr. Rob Jamieson led water sampling, testing for total suspended solids, conductivity, *E. coli*, nitrogen, phosphorus, a range of metals, biological oxygen demand, dissolved oxygen, pH levels, and dioxins and furans across eight sites. In general, samples from within Boat Harbour were above the limits specified by the Canadian Water Quality Guidelines for the Protection of Aquatic Life. Samples from surrounding sites mostly met guidelines, with the exception of some heavy metals: silver, zinc, iron, and lead.

Dr. Jamieson also led the soil quality analysis. Soil was first sampled at 14 sites and was analyzed for various metals as well as dioxins and furans. Follow-up sampling occurred to confirm the first round's findings and drew from six sites. The soil results indicated

that contaminant concentrations in these samples generally met Canadian guidelines for protection of human and environmental health, and that the samples between Pictou Landing First Nation and the town of Pictou were of similar quality.

Dr. Ron Russell led the ecotoxicology and sediment analysis to determine aquatic toxicity and what chemicals were present in the samples. The sediment analysis revealed the presence of dioxins and furans well above the CCME limits for environmental quality (human health guidelines don't exist). The chemicals, however, are mainly present in the sediment, and contact with Boat Harbour sediments should be avoided. Dr. Russell's determination of aquatic toxicity found that Boat Harbour is unable to support aquatic life due to hyper-eutrophication (causing low oxygen), high water temperature, decreased sunlight penetration, and toxic chemical inputs.

Dr. Russell's mammal analysis found that detectable but low concentrations of dioxins and dioxin-like compounds were found in beaver and muskrat tissues. Liver exhibited consistently higher concentrations than muscle since metabolic detoxification pathways are predominantly found in the liver. Toxicity of dioxins in mammal tissue was approximately the same as plankton and significantly lower than that calculated for sediment. Beaver and muskrat are not part of the commercial food industry; however, the low concentrations of dioxins detected in both muscle and liver of these mammals still exceeded European Union guidelines for commercial meat, indicating local wild meats should not be consumed.

Dr. Russell also conducted an analysis of the June 2014 effluent spill near Boat Harbour. He found that most metals in samples from the effluent exceeded guidelines for the protection of aquatic life, with copper and lead as the worst cases (exceeding the guidelines by greater than 10 times).

Mr. Geoff Kershaw undertook basic dendrochronology, the analysis of tree rings, to determine whether there were impacts to tree growth in the area. Twenty trees were sampled from an old-age white spruce stand near Boat Harbour and, for comparison, 18 trees were sampled from a control site. While the analysis showed statistical differences suggesting unique growth-influencing factors at each site, it is unclear whether these differences are associated with pulp mill activity.

Three key legacies of the project are detailed in this report. First, Dr. Daniel Rainham carried out an interactive community mapping activity for the project, bringing together sites of data collection and stories associated with the data. In doing so, he has created a legacy map that can be explored online to visualize land use in terms of traditional and recreational use over time as well as places relevant to the project (e.g., air sampling sites, soil sampling sites, tree sampling sites, places where community members used to go for traditional medicines and traditional foods).

Second, a capacity-building legacy of the project has been the training and certification of water monitors in Pictou Landing. Ms. Kim Strickland, Ms. Colleen Denny, and Ms. Lucie Francis are trained to use the equipment associated with Wet-Pro, a community-based water monitoring kit received from CURA H2O.

Third, and also related to capacity-building, a Youth Camp took place over two weeks in 2015. Ms. Cecilia Jenning and Ms. Kim Strickland brought five Mi'kmaq youth from Pictou Landing together to learn about the PLNWG's research project and to share their perspectives concerning Boat Harbour in the form of digital stories.

This report is one knowledge-sharing product of many from our work together; more are described herein. The Research Team is proud of this community-owned research.

Foreword by Sheila Francis, Past President
Pictou Landing Native Women's Group

Here it is: Our final report after 6+ years of research, meetings, presentations and laughs!

I must say, this has been a long and emotional journey, not just for me but especially for the women of the community. At the same time, it has been one of empowerment and voice. Many women in our community have shown themselves to be leaders through this project. They have given themselves that opportunity to express their concerns, their fears, and their hopes.

I would like to thank the University members of our Research Team who helped us with the scientific part of this project. Your expertise enabled us to understand the technical aspects and you put it in a language we could understand. But most of all, you brought your humanness. You cried with us. You gave us a safe and compassionate space. You heard us.

I would like to thank Heather Castleden, our Lead Academic Researcher. Right from the start, you were our partner. You did not come in and assert your credentials or your experience. You did not minimize our lack of expertise as scientists. What you brought was what we had never received before – compassion, safety, someone who listened to our concerns and who really cared. I think that was the most important thing we needed to move this project forward so successfully. From the bottom of my heart Heather, I thank you.

To the ladies who played a role in this project:

Whatever conclusions you have taken from this research study, I hope one of them is the fact that you were a part of this study. You led this study. You controlled this study. You are the authors of this study. I hope you will continue to demand and express your concern for your and your family's health, and the health of our community. I hope you will continue to use your voice. I want to thank you for allowing me to represent you. I had to step out of my own comfort zone many times to tell your story, our story, but I would do it again for you. I really love that we worked together, we spent time together, we began to create an understanding for and about each other. I hope we will continue to work as a team. I really appreciate you giving me the chance to move this forward.

All my best!

Sheila Francis

Acknowledgements

Our team wishes to offer its sincerest thanks to everyone from Pictou Landing First Nation for supporting this community-based research project that was led by the Pictou Landing Native Women's Group with Sheila Francis (Past President) serving as the Community Research Lead and Heather Castleden serving as the University Research Lead, joined by Diana Lewis (Dalhousie University), Daniel Rainham (Dalhousie University), Debbie Martin (Dalhousie University), Mark Gibson (Dalhousie University), Rob Jamieson (Dalhousie University), and Ron Russell (Saint Mary's University).

Our deepest and warmest heartfelt thanks to the Elders of the Pictou Landing Native Women's Group for their guidance throughout the project, and to the Community Research Associates from Pictou Landing without whom this work would not have been possible: Kim Strickland and Colleen Denny, and the surveyors who conducted the Environmental Health Survey: Colleen Denny, Kim Strickland, Pam Denny, Haley Bernard, Jordan Francis, Sheila Francis, Fran Nicholas, April Nicholas, Darlene Bachiri, Holly Francis, Heather Mills, Sylvia Francis, and Loretta Sylliboy. Special thanks to Haley Bernard and Lucie Francis for their internship work on the project, Durney Nicholas for trapping mammals for analysis, and Dakota Francis for his work with the Boat Harbour Youth Camp.

Thank you to each student whose graduate work has fulfilled pieces of this project: Diana "Dee" Lewis (PhD, Environmental Health Survey), Ella Bennett (MES, Oral Histories), Ziyun Wang (MREM, Health Canada Literature Review), and Jane McCurdy (MREM, Community Map). Thanks also to the many university-based Research Assistants involved in this project, including Chris Garda, Cecilia Jennings, Codey Bennett, Geoff Kershaw, James Kuchta, Jenny Hayward, Justine Lywood, and Rick Scott, for their integral roles in data collection and beyond. We are also grateful for Emily Skinner and Catherine Hart, who served as consecutive Project Coordinators. Thanks also to the CURA H_2O team, namely, Oliver Wood and Sarah Weston, for supporting the WetPro component of this project. And finally, enormous thanks to Martha Stiegman, Catherine Martin, and Frank Clifford for documenting moments of this project on film throughout its duration.

Funding for this research was provided by a Canadian Institutes of Health Research Operating Grant (# 119395), a Nova Scotia Health Research Foundation Development Initiative Grant and the in-kind contributions of the Health, Environments, and Communities Research Lab (Castleden), the Centre for Water Resources Studies (Jamieson), the Atmospheric Forensics Research Group, (Gibson), the Spatial Intelligence for Health Knowledge Lab (Rainham) and the Biology Laboratory (Russell). In-kind contributions were also provided by the Pictou Landing Native Women's Group.

Project Team

Sheila Francis, Community Research Lead

Heather Castleden, Academic Research Lead

Diana "Dee" Lewis, PhD Candidate; Environmental Health Survey Lead

Kim Strickland, Community Research Associate

Colleen Denny (bottom), Community Research Associate

Ella Bennett, Master's Student, Oral Histories

Rob Jamieson, Soil and Water Quality

Ron Russell, Ecotoxicology

Mark Gibson, Air Quality

Daniel Rainham, Community Mapping

Debbie Martin, Academic Co-Investigator

Catherine Hart, Project Coordinator (2014-2016)

James Kuchta, Research Assistant, Air Quality

Codey Bennett, Research Assistant, Air Quality

Chris Garda, Research Assistant, WetPro Monitoring and Pamphlet Creation

Emily Skinner, Project Coordinator (2012-2014)

Cecilia Jennings, Research Assistant, Boat Harbour Youth Camp

Dakota Francis, Community Research Assistant, Boat Harbour Youth Camp

Geoff Kershaw, Research Assistant, Land, Water, and Air Quality

Ziyun Wang, Master's Student, Literature Review and Gap Analysis

Rick Scott, Research Assistant, Water Quality

Jenny Hayward, Research Assistant, Soil Quality

Jane McCurdy, Master's Student, GIS Mapping

Lucie Francis, Atlantic Aboriginal Health Research Program Award Recipient, Ecotoxicology

Haley Bernard, Atlantic Aboriginal Health Research Program Award Recipient, Oral Histories & Environmental Health Survey

Fran Nicholas, Research Assistant, Environmental Health Survey

Sylvia Francis, Research Assistant, Environmental Health Survey

Loretta Sylliboy (right), Research Assistant, Environmental Health Survey

Heather Mills, Research Assistant, Environmental Health Survey

April Nicholas, Research Assistant, Environmental Health Survey

Darlene Bachiri (right), Research Assistant, Environmental Health Survey

Pam Denny, Research Assistant, Environmental Health Survey

Jordan Francis, Research Assistant, Environmental Health Survey

Justine Lywood, Research Assistant, Water Quality

Durney Nicholas, Trapper, Ecotoxicology Analysis

Picture Unavailable

Holly Francis, Research Assistant, Environmental Health Survey

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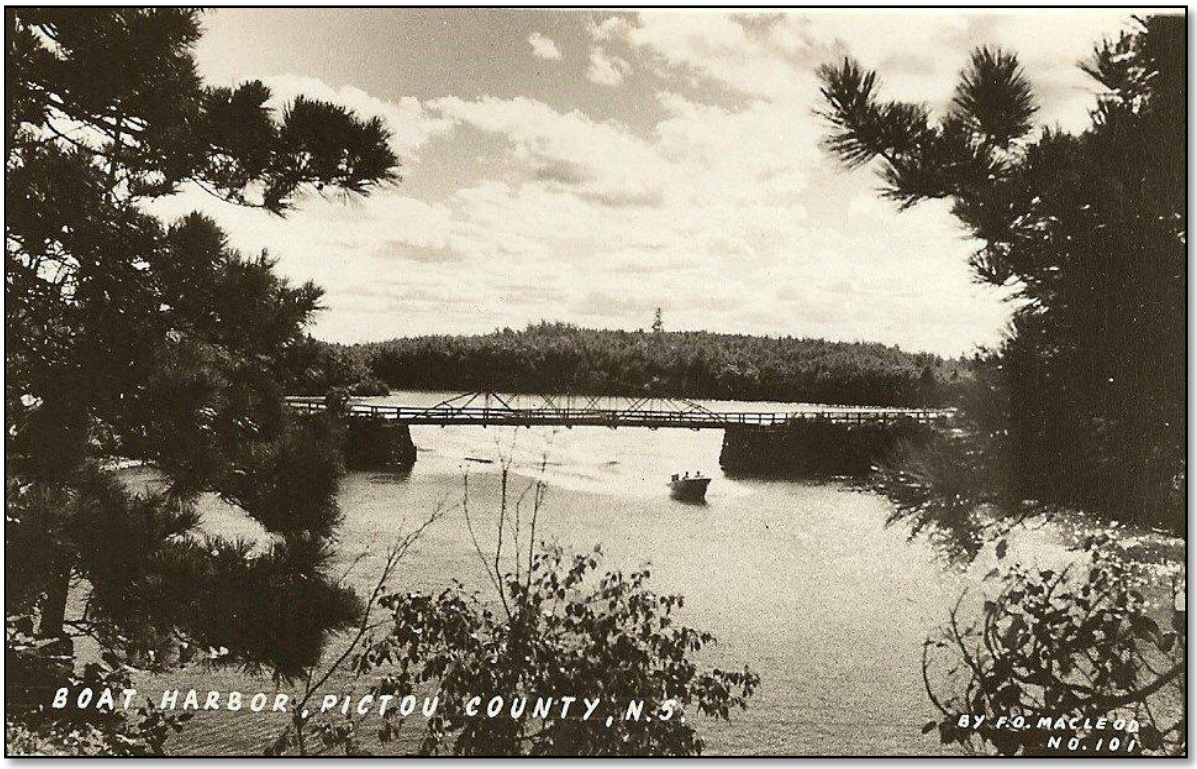
1. Introduction

Mi'kmaw peoples have lived in Mi'kma'ki (now frequently referred to as the Canadian Maritimes) for hundreds of generations. It is where our Mi'kmaw origin stories begin and where we, the Mi'kmaw people, continue to raise our families and live on the land. For those of us from Pictou Landing, A'se'k is "the other room." This tidal estuary is commonly known today as Boat Harbour, Nova Scotia. But historically, A'se'k was our gathering place that exemplified our Mi'kmaw ethic of sharing: food, knowledge, and skills were exchanged there between generations and amongst family groups (see www.plfn.ca).

A'se'k and its recreational, physical, mental, spiritual, and emotional purposes have been compromised since a pulp and paper mill was built nearby and began dumping its effluent into this cherished body of water. For nearly 50 years, we – our Elders, our leaders, all of us – have been trying to redress this environmental and social injustice.



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In 2010, the women of the Pictou Landing Native Women's Group (PLNWG) mobilized as a result of our health and environmental concerns related to Boat Harbour, and it is

due to this initial collective and continual effort to organize that the research described in this report was carried out.

To date, more than 200 studies have been completed to learn about what contaminants are in Boat Harbour, of which about 80 studies are relevant today (Nova Scotia Government, 2015). A review of the research that has taken place found that "very few reports" contained information about air, sediment, soil, game animals, vegetation, human activity patterns, fish and shellfish eating habits, and non-human living organisms on land and in water (DCL [2012] as cited in Hoffman et al., 2015).

Scholar Michael Mascarenhas (2007) observes, "whether by conscious design or institutional neglect, [First Nations] communities face some of the worst environmental devastation in the nation" (p. 570), and Boat Harbour is evidently an example of the environmental injustice and health inequity that he describes. The effects of the pollution on the surrounding land, air, soils, and water are not fully known and understood; "while consultants' reports exist, they are patchwork at best, limited by time and resources, lacking in longitudinal quality, and difficult to access, much less interpret" (Castleden & Bennett, 2011).

A Brief History of the Pulp and Paper Mill at Abercrombie Point

In 1957, Scott Paper Company Ltd. acquired lands at Abercrombie Point and established a kraft pulp and paper mill there. In 1967, the mill began operating and dumping its effluent (wastewater) into Boat Harbour; it continues to do so today at a rate of approximately 85 million litres per day (Jacques Whitford Environment & Beak Consultants, 1992). The effluent from the mill was dumped directly into Boat Harbour without being treated until a treatment plant was built on Simpsons Road adjacent to Pictou Landing First Nation in the early 1970s (Nova Scotia Government, 2015). Even so, Scott Paper admitted in a brochure mailed to all Pictou County households in the fall of 1989 that the mill equipment was largely of 1967 technology and, in terms of air emissions, was not designed to meet modern mill standards (Reid, 1989).

Industrial operations like those at the mill at Abercrombie Point involve two stages – kraft pulping and bleaching. Kraft pulping and bleaching typically consume huge amounts of fresh water and considerable quantities of chemicals, meaning the wastewater from the kraft bleaching processes contains various toxic chemicals such as dioxins and furans, polycyclic aromatic hydrocarbons (PAHs), hydrogen sulfite, and mercury (Pokhrel & Viraraghavan, 2004). The Nova Scotia Government acknowledges that there are dioxins and furans in Boat Harbour (2015).

Wastewater treatment at Boat Harbour happens in two stages (previously three; see Stantec, 2004). Once effluent is delivered to the treatment facility by pipeline, primary treatment takes place in the settling basin followed by secondary treatment in the aerated stabilization basin before the water is released into Northumberland

Photo Credits: University of King's College Journalism (2009)

Strait (Stantec, 2011). In 2015, an industrial approval included three main conditions for Northern Pulp (the company that eventually bought Scott Paper): (1) lower annual emission limits for particulate matter and sulfur dioxide beginning January 2016; (2) stack testing of recovery and power boilers doubling to four times a

year; and (3) a restriction of water use, which would lead to a 35% reduction of effluent being dumped into Boat Harbour on a daily basis, and a subsequent 25% reduction by 2020 (Hoffman et al., 2015). After appeals by Northern Pulp about these conditions, a final decision was released in February 2016, which included revisions to the third condition (see Miller, 2016).

Pulp and Paper Mill and Government Interactions with Pictou Landing First Nation

Concerns about how the mill's operations impact the land, air, soil, and water have been expressed since its establishment. Yet Nova Scotia has repeatedly granted approval to the mill to continue its operations. A timeline of interactions between the mill, the province, and Pictou Landing First Nation is presented in Table 1.

Table 1
Pulp and Paper Mill and Government Interactions with Pictou Landing First Nation (Adapted from Hoffman et al., 2015 and New Glasgow News, 2010)

1964	Scott Maritimes decides to build a pulp and paper mill at Abercrombie Point.
1965	Pictou Landing First Nation Chief and Council state concerns about odour. The province and Scott Maritimes take them to a similar mill site in Saint John, New Brunswick, to show another mill with no odour (it was not operating at the time). An agreement-in-principal is signed.
1965	Soon after, a resolution is signed for a lump sum payment of $60,000 to Pictou Landing First Nation for permanent loss of fishing and hunting revenue and other benefits derived from land/estuary use, with a final settlement subject to further negotiations between the province and Indian Affairs.
1967	Mill begins operating.
1986	Pictou Landing First Nation begins action against federal government for breach of fiduciary duty in Boat Harbour.
1991	Federal government negotiates settlement with Pictou Landing First Nation. Province promises to abate adverse effects of effluent when agreement expires in 1995.
1993	The federal government and Pictou Landing First Nation agree to settle out of court for $35 million.
1995	No alternative effluent treatment site identified before agreement expired. Province promises closure of Boat Harbour by December 2005.
2004	Mill ownership transferred to Neenah Paper.
2005	Province and mill request extension to December 2008.
2008-2009	Band Council Chief meets with negotiator and advises province that they will not agree to further licence extensions. Meetings halt following provincial election. When talks resume, transportation minister requests more time to study issues.

Table 1, cont.

2008	Province asks Pictou Landing First Nation not to protest extension of licence to December 2008 and promises not to extend it without consultation, but province extends licence after December 2008 on a month-to-month basis.
2010	Pictou Landing First Nation asks province to terminate licence, effective June 30. After no change, a lawsuit is filed against province and mill.
2014	Effluent leak sparks protest by residents seeking a commitment from province for firm deadlines to find an alternative location for mill's effluent and remediation of Boat Harbour. Pictou Landing First Nation Chief and Nova Scotia's Minister of Environment sign an agreement. Government commits to legally implementing a timeline to stop flow of effluent into Boat Harbour and site's remediation by June 30, 2015. Clean the Air concert raises awareness about mill's emissions. Public consultation on behalf of mill for public to voice opinions related to mill's industrial approval renewal application. Smoke stack precipitator shutdown triggers ministerial order requiring the mill to install new air pollution equipment by May 2015.
2015	New industrial approvals issued by Nova Scotia Environment for improvements in air emissions, water use, and effluent. Mill immediately appeals new industrial approvals claiming that they are tougher than rest of the pulp and paper industry emission standards. US company hired to supply and install new air pollution equipment pulls out of $22 million project before completion. *Boat Harbour Act* is passed, stating that the Boat Harbour Effluent Treatment Facility will close by 2020.
2016	Northern Pulp appeals the industrial approval issued by the province. Nova Scotia subsequently amends its conditions, and Northern Pulp drops the appeal. Nova Scotia court issues a fine to Northern Pulp in the amount of $225,000 for the company's effluent spill in 2014. Of this amount, $75,000 will go to the Mi'kmaw Conservation Group, $75,000 to the Pictou County Rivers Association, and $75,000 to eligible recipients in the area, including residents of Pictou Landing First Nation.

Developing a Community-Academic Research Partnership

After discussions with the Nova Scotia Native Women's Association president, **Cheryl Maloney**, the PLNWG invited **Diana "Dee" Lewis**, a Mi'kmaw woman from Sipekne'katik First Nation with graduate-level training in environmental studies, to

discuss the women's concerns and what could be done about them. Early on, there was discussion about traditional knowledge and the traditional uses of Boat Harbour, and the potential of oral histories and community mapping as ways of exploring this further. With the PLNWG's permission, Lewis then invited **Dr. Heather Castleden**, a community-based participatory Settler (non-Indigenous) researcher from (then) Dalhousie University with 15+ years experience working with Indigenous peoples, and **Ella Bennett**, her Settler graduate student, to meet with the PLNWG in the community in November 2010 to discuss the future of PLNWG's interests in research and action about Boat Harbour. Discussion continued about how some of the questions the PLNWG had about Boat Harbour could be answered through Dee's doctoral research and Ella's Master's research (see Figure 1).

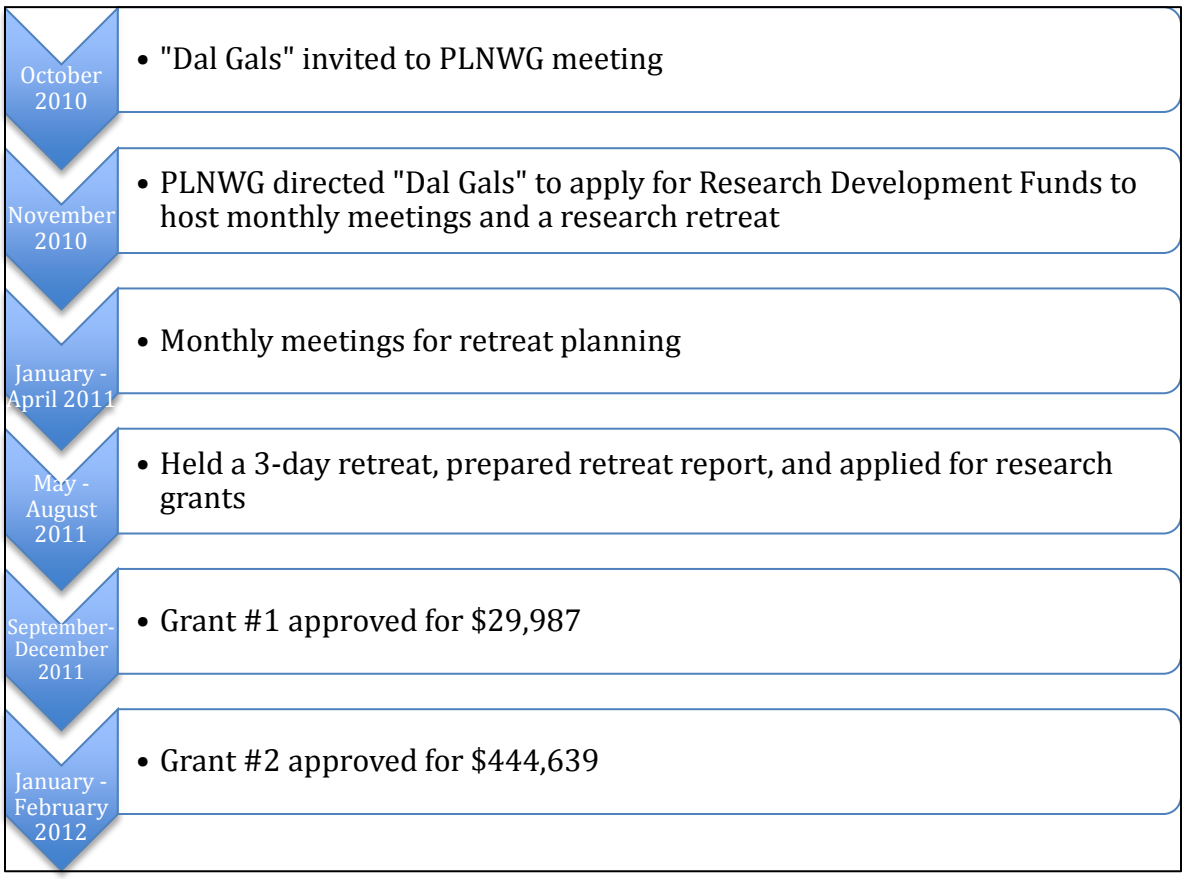

Figure 1. Timeline of our early research partnership.

At this November meeting, three key components of a research relationship between the PLNWG and the trio of researchers, who became known as "the Dal Gals," emerged:

1. The community's health must be the number one priority of the research;

2. The research partnership should result in transparent and impartial research with direction coming from the women, but with the consideration of all community members' input; and
3. The research must have a focus on capacity-building, including training community researchers and involving students from the community.

The research relationship began with meetings that had an atmosphere where the women could share their concerns and goals for community health and wellbeing, as well as discuss ways to move forward on positive environment and health reform; in other words, we "spent the first year drinking tea!" (Castleden, Sloan Morgan, & Lamb, 2012, p. 16).

From the beginning, the research partnership sought to embody what are known as the "4 Rs" of research (Kirkness & Barnhardt, 1991): to be relevant, respectful, responsible, and carry out reciprocal research, with shared decision-making about and ownership of the research plan, the information we collect, how we analyze and make sense of what we collect, and how/when results are shared with others.

Following direction from the PLNWG, Heather applied for and received funding to hold a research retreat (Grant #1). Then, after the first research retreat, and with guidance and support from the PLNWG, Heather led a second grant submission to the Canadian Institutes of Health Research (Grant #2). Included on the team were other researchers: **Dr. Rob Jamieson** (Dalhousie University), **Dr. Mark Gibson** (Dalhousie University), **Dr. Daniel Rainham** (Dalhousie University), **Dr. Debbie Martin** (Dalhousie University), and **Dr. Ron Russell** (Saint Mary's University). The PLNWG and university researchers were then awarded Grant #2 to explore the question: "Is Boat Harbour making us sick?" using Indigenous and Western approaches to the research design.

The approach to health within this community-based project was wholistic; it did not solely focus on physical health like previous research, but equally looked at mental, spiritual, and emotional wellbeing. Central to the project and research partnership was the guiding principle of Two-Eyed Seeing (Figure 2, below).

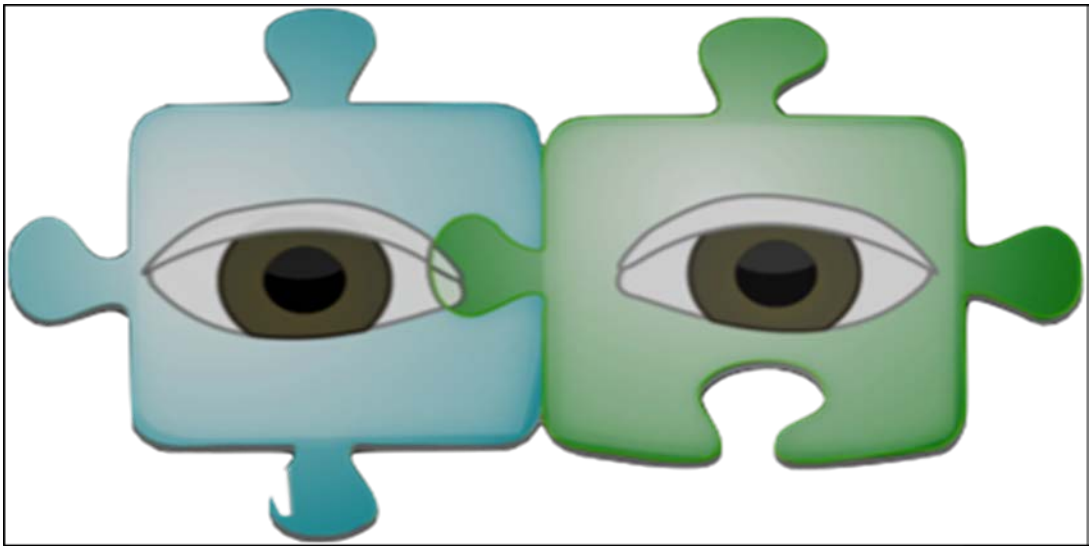

Figure 2. Two-Eyed Seeing illustration (reprinted with permission of Dr. Cheryl Bartlett from http://www.integrativescience.ca/Principles/TwoEyedSeeing/).

The concept of Two-Eyed Seeing is attributed to Mi'kmaw Elders Albert and Murdena Marshall, who live in Eskasoni First Nation, and their colleague Dr. Cheryl Bartlett, a professor emerita of biology and former Tier 1 Canada Research Chair in Integrative Science. Two-Eyed Seeing is "learning to see from one eye with the strengths of Indigenous knowledges and ways of knowing, and from the other eye with the strengths of Western knowledges and ways of knowing… and learning to use both these eyes together, for the benefit of all" (Bartlett, Marshall, Marshall, & Iwama, 2015, p. 280). Two-Eyed Seeing has been central to the project as the PLNWG and their university partners have sought to conduct relevant, respectful, responsible, and reciprocal research.

2. Research Retreats

Research retreats were held, approximately annually, over the course of the community-university partnership as a means to strengthen and maintain good relationships between all team members and as spaces where important decision-making about the project occurred. Each retreat served different purposes, balancing relationship-building with research design, knowledge sharing, and reporting of findings. They were held in multiple places in Nova Scotia (Halifax, Tatamagouche, Truro) across the life course of the project. Each retreat is briefly described below. Retreat Reports for the first, third, and fourth retreats can be downloaded from www.heclab.com.

First Research Retreat (2011)

"We have the voice, we need the steps to move forward… that's called empowerment."

The first PLNWG Research Retreat was funded through a grant awarded by the Nova Scotia Health Research Foundation. The retreat was titled *Epitik Mawi-ta'jik* and took place from April 29 to May 1, 2011, in Kjipjutuk (Halifax), and it was an important outcome of the evolving research partnership. Six academics and professionals from Atlantic Canada were invited to share their knowledge and expertise with the PLNWG. The retreat gave women from the community the opportunity to gain knowledge, share their concerns, and discuss relevant and accessible research priorities for the future.

Specifically, the goals as stated in the Retreat Report (Castleden & Bennett, 2011) of the *Epitik Mawi-ta'jik* were to:

1. Provide an opportunity to share and discuss community-relevant concerns of environment and health in an inclusive, multi-generational, and female-centred forum;

2. Increase the environmental and health literacy of retreat participants, especially as pertaining to Boat Harbour;
3. Allow participants to acquire and discuss information provided by invited panelists, who have particular backgrounds in environment and health fields; and
4. Establish goals and priorities for a continuing community-based participatory research partnership.

"We can't just respond to research, we need to be the drivers of it!"

"Our women are strong and tough and we're able to do it!"

After a night of social relationship-building, the women gathered for two days of learning and sharing, each day opening and closing with a prayer. The following panelists were invited to give presentations based on their relevant expertise:

1. **Deborah Carver** (*Executive Director of the East Coast Environmental Law Association*) discussed the legal issues surrounding Boat Harbour.
2. **Barbara Clow** (*Executive Director of the Atlantic Centre of Excellence for Women's Health and Associate Professor in the Faculty of Health Professions at Dalhousie University*) discussed the relationship between gender and health in the context of Boat Harbour.
3. **Debbie Martin** (*member of NunatuKavut and Assistant Professor in the School of Health and Human Performance at Dalhousie University*) presented on Indigenous rights and the importance of moving forward acknowledging that health should be examined from all four directions: physical, mental, emotional, and spiritual.

4. **Inka Milewski** (*expert marine biologist*) discussed health and environmental (in)justice.
5. **Daniel Rainham** (*Assistant Professor in the Environmental Science Program at Dalhousie University*) described how his approach could capture meaning and story in a map.
6. **Ron Russell** (*Associate Professor in the Biology Department at St. Mary's University*) presented the language needed to articulate possible impacts of the mill's effluent from a physical science perspective.

In addition to panelist presentations, there was a morning spent developing health and environmental literacy with retreat participants. With a comprehensive list of over 50 concepts, tools, and terms related to health and environment, this session allowed the women to acquire a strong foundation on which to build throughout the retreat and for their collaborative work in the future. Booklets of the terms were also provided for everyone to take home. The women who attended the retreat described it as being extremely important, and the momentum it generated carried forward next steps of the research relationship as established by the PLNWG (for example, the Environmental Health Survey and the submission of Grant #2 to the Canadian Institutes of Health Research).

Second Research Retreat (2012)

"I suffered a lot of injustice, but now in this room, I feel empowered."

The second PLNWG Research Retreat was held at the Tatamagouche Centre (April 28-29, 2012) to establish a good mind and heart across the team. It included prayers, ceremony, and sharing circles; shared food and meals; a traditional medicine walk and an archaeological walk; a screening of the CBC documentary about Boat Harbour; shared laughter and tears, bowling and bonfires; a review of OCAP,[1] TCPS2,[2] and the 4 Rs of research; and the development and confirmation of a Research Agreement. The research team members were taught by invited Elders **Albert** and **Murdena Marshall** from Eskasoni First Nation and **Dr. Cheryl Bartlett** (Professor Emeritus at Cape Breton University) about the Guiding Principle of Two-Eyed Seeing (see end of Introduction for a definition of Two-Eyed Seeing).

[1] Standing for ownership, control, access, and possession, OCAP asserts that First Nations control research in their communities and that they own the information and determine how it will be used. OCAP® is a registered trademark of the First Nations Information Governance Centre (www.FNIGC.ca).

[2] The TCPS2 is the second edition of the *Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans.* A joint policy of Canada's three main federal granting agencies in health, social sciences, and natural sciences, it is informed by international ethics practices and provides guidance to researchers working with people.

Indigenous scholar David Newhouse (2008) speaks of the Haudenosaunee concept of Ganigonhi:oh ("a good mind") in regard to the balance between passion and reason; researchers need to balance these as they navigate institutional, professional, personal, and community interests. Participants in the second retreat discussed approaching research with "a good mind" in terms of researchers' roles in water and air sampling, the progress that had been made, and the challenges they encountered. Ron Russell and **Rob Jamieson** also delivered updates on ecotoxicology and water respectively, and Daniel spoke about the status of the community map.

"We don't know how healthy our medicines are – we're afraid to go and collect them."

"This is all I ever wanted: the chance for people to speak up… to be able to sit together and get out feelings out… it's so powerful."

Third Research Retreat (2014)

The third PLNWG Research Retreat was titled *Sharing What We Were Learning: Where We've Come From, Where We Are Going* and took place March 1 and 2, 2014, in Kjipjutuk (Halifax) with 28 women in attendance. The first morning began with a welcome, introductions, and an overview of the research partnership provided by **Sheila Francis** (President, PLNWG) and **Heather Castleden**. **Dee Lewis** gave an update on the Environmental Health Survey, which had concluded on December 31, 2013. There was discussion about the potential for a documentary film about the overall project.

Three things emerged from the discussion about what stories the film could tell: (1) the history of Pictou Landing First Nation and the important role A'se'k played for the community; (2) the unjust placement of the Treatment Facility in the 1960s and broken government promises since then; and (3) the impact of the pollution and loss of use of the land, especially the reverberating impacts on younger generations.[3]

Jordan Francis, a student from Pictou Landing First Nation, and her supervisor, **Irena Knezevic**, presented on a project Jordan had been working on since the previous summer. Jordan's project, part of a larger one conducted through the Food Action and Research Centre (FoodARC) at Mount Saint Vincent University, involved Storysharing and Photovoice sessions with 18 participants. The results of the project showed that the community of Pictou Landing as a whole struggles to obtain affordable, nutritious food and that Boat Harbour has contributed to this struggle because people have felt they don't have access to food from the land since the estuary began receiving wastewater. Jordan also reported that people are hesitant to grow their own food because they don't trust the soil. This research led to the addition of soil sampling to the overall Boat Harbour project.

[3] In 2014-2015, the PLNWG decided to carry out soil analysis in backyards out of interest in having individual and community gardens. The funds allocated to the film were redistributed to do the (expensive) soil analysis. In 2016, Heather and the PLNWG applied for additional funds to complete the film but the application was not successful in the funding competition.

Kim Strickland and **Colleen Denny** delivered a presentation titled *A Day in the Life of a Community Researcher,* in which they talked about their critical involvement in the project to keep things running smoothly. They described their roles in water sampling and air quality monitoring as well as the challenges they had experienced. They also explained how their families motivated them to do this work.

At the end of the first day a photobooth activity was organized where everyone was invited to complete the sentences "A'se'k is…" and "The Boat Harbour project is important to me because..." on a chalkboard and have their photo taken. After a full day, everyone headed downtown to enjoy dinner and a performance at the Grafton Street Dinner Theatre.

On the second day, Rob Jamieson presented on water quality testing he had completed and Ron Russell presented on the aquatic toxicity tests he'd conducted. Ron's results indicate that the water is acutely toxic to creatures, meaning the effects happen in the short term. Because Daniel Rainham and **Mark Gibson** regrettably could

not attend, **Emily Skinner** shared updates about the community mapping and air quality monitoring components of the project. The day concluded with a sharing circle to reflect on the retreat. **Trina Roache** from APTN (Aboriginal Peoples Television Network) also came by the retreat and conducted interviews with some of the women in attendance as part of a three-part television series about Boat Harbour.[4]

Fourth Research Retreat (2015)

The fourth PLNWG Research Retreat took place December 12 and 13 in Wékopekwitk (Truro), Nova Scotia. The PLNWG arrived in the morning on December 12, and the day began with the opportunity to review a draft of this Final Project report as well as a draft Environmental Health Survey Community Report. The meeting opened with a prayer led by **Jackie Alex** as well as a song and drumming by Mi'kmaw filmmaker **Catherine Martin**. With two full days ahead of the group, it was important for the meeting to start, as all other meetings and retreats had, with allowing those present to enter discussions with open hearts and open minds. **Mary Irene Nicholas** also welcomed the group and noted that while this retreat would be the last one held specifically for this project, it signified a new beginning, not an ending.

Heather and Dee began the presentations with an oral history update – Dee had conducted three more in the fall of 2015. They also screened a short film that Ella had made, which was a collage of oral histories about Boat Harbour. The PLNWG decided to make the film available online through the HEC Lab website as well as Pictou Landing First Nation's website. Dee then presented findings from the Environmental Health Survey. The PLNWG offered comments and discussed the benefits and compromises of using microdata from provincial and regional databases, comments which Dee indicated she would continue to consider as she finalized the Community Report.
The afternoon consisted of a Science Panel, comprising Daniel, Rob, Mark, and Ron. Daniel began the panel by illustrating what the community map is capable of showing by turning layers "on" and "off," and in doing so communicating different information around land use and the project. The decision to make the map a legacy of the project was reconfirmed, meaning it will only be editable by way of request to Daniel. The second panellist, Rob Jamieson, reiterated findings from the water quality analysis and reported, for the first time, findings from the soil quality analysis. Mark Gibson delivered an update on the air quality analysis, which included more recent data sets from spring and summer 2014 and the modelling of pollutants. Ron concluded the panel by presenting the ecotoxicology aspect of the project, reiterating his aquatic toxicity report and reporting on the chemical analysis he also conducted. At the time of the retreat, the mammal analysis had not been completed. Throughout discussions

[4] The APTN series can be viewed here: http://aptn.ca/news/2014/03/26/pictou-landing-researches-health-effects-polluted-harbor/.

during the Science Panel, the PLNWG emphasized their concern about the potential danger that the removal of sediment posed as well as perceived negative health impacts of the smell in the area: considerations in the context of future remediation and restoration processes.

At the end of Day 1, we held a sharing circle where participants reflected on the transformational path of this research and emphasized gratefulness for the time, resources, and spirit that each person contributed to this critical work. Retreat attendees then enjoyed dinner at Frank & Gino's who hosted us in their private room, making for a festive night of friendship and food.

On Day 2, Heather provided a budget overview of spending to date and what the remaining funds were committed to. The PLNWG reconfirmed the decision to pursue a documentary film; filmmaker Catherine Martin led the discussion to get a sense of possibilities for the film – as before, the women expressed it was a critical tool to document the story of the PLNWG mobilization and communicate Elders' stories about A'se'k. After the retreat, a grant application was submitted to the Canadian Institutes of Health Research. Unfortunately in July 2016 Heather received notice that the funding application had been rejected; however, there is the possibility of continued efforts to secure film funding.

Other opportunities for future collaborations were discussed, such as a way to connect Elders and youth in the community and the potential formation of a community-based Boat Harbour group, which would include men and youth as well as women. Heather and the PLNWG also took the time to discuss the Canadian Alliance for Healthy Hearts and Minds cohort project that Pictou Landing First Nation was invited to be a part of. It was decided that this would be an initiative taken on by Chief and Council, instead of the PLNWG.

Cecilia Jennings, Kim, and **Lexy Strickland** also presented on the Youth Camp's activities from summer 2015. They screened the youth's powerful and moving digital stories made by **Madison Nicholas**, **Hunter Francis**, and **Alexandria Francis**, as well as one by Cecilia. **Carter Hatfield** and **Laela Denny** also attended the camp. The stories will be made available on the HEC Lab website after the project's final Community Dinner.

In an emotional final roundtable discussion, there was an appreciation for the trust and sharing that this partnership enabled as well as pride in the work that had taken place with an eye forward on what would be next to come.

3. Oral Histories

Oral histories addressed topics of family heritage and future, health and wellbeing, childhood/youth of the storyteller, community, Boat Harbour, reasons for leaving or staying in Pictou Landing First Nation, and memories. The oral history interview guide was adapted from the Community-University Research Montreal Life Stories Project (2007-2012) at the Centre for Oral History and Digital Storytelling, Concordia University. In November 2011, **Ella Bennett** began to document oral histories to reconstruct what Boat Harbour was like before the mill opened. **Hayley Bernard**, while majoring in Mi'kmaq Studies at Cape Breton University, was awarded an undergraduate summer internship by the Atlantic Aboriginal Health Research Program and also collected several oral histories. **Dee Lewis** continued to record oral histories in the fall of 2015 and completed three as of this report's publication (September 2016).

Ella's graduate work, and Haley and Dee's continuing work, documenting oral histories offers Mi'kmaw perspectives on Boat Harbour from Elders who have lived through the loss of A'se'k and surrounding land. Many of the Elders discussed sharing practices in great length. Residents of Pictou Landing First Nation also spoke about the relationship between the pollution at A'se'k and the dramatic decrease of individual and community engagement with their lands and waterways. While sharing practices continue to function in Pictou Landing today and some people continue to engage in traditional harvesting activities (although usually away from Pictou Landing), it was clear from the Elders Ella spoke with that trust in food and medicines from the land has been significantly compromised (Castleden et al., in press). The Elders also suggested that the lack of engagement with the land is especially true for youth. Many youth do not hunt or fish or know what foods and medicines are available on the land where they're from and where they live because they have lost a place to engage in these activities. In this sense, the pulp mill and treatment facility have compromised the community's physical, sociocultural, emotional, and spiritual health and wellbeing.

Ella's thesis, as well as the transcribed oral histories, are available to the community by contacting Sheila Francis.[5]

A'se'k: All Seasons, All Purpose

"That was safe haven for all of us. Everything that we needed was there." ~Sadie Francis

A'se'k: After the Mill Went In

"Well, I guess they didn't want to put it anywhere else in town. Let's put it near the Indians – Native people close by, we'll dump it on them! … Let them deal with it. But it's always us that got dumped on. That's how they treated us I guess…" ~Mary Ellen Denny

A'se'k: No More

"Everything we used to do, we can't do. What we were brought up on, it's all been taken away." ~Don Francis

A'se'k: The Future

"I had a dream once. I dreamt it was clean, and our community became rich from it. And everybody worked together, in my dream…" ~Louise Sapier

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[5] Ella defended her thesis, titled *"We Had Something Good and Sacred Here": Restorying A'Se'k with Pictou Landing First Nation*, in June 2013 in Pictou Landing. It was the first time a Dalhousie University student defended a thesis in a Mi'kmaw community, and residents of Pictou Landing attended. At the defence, she also screened the short film she made that was a compilation of the oral histories she had documented; the film can be viewed online at the HEC Lab website as well as Pictou Landing First Nation's website.

4. Literature Review

Pulling the Plug on Boat Harbour: A Synthesis Review and Gap Analysis of Existing Environmental and Human Health Assessment Including Pictou Landing First Nation (1968-2007) by Ziyun Wang, Master of Resource and Environmental Management

Ziyun Wang, an international graduate student working with **Heather Castleden**, conducted a literature review in 2012 to determine what information about Boat Harbour existed and what was missing. Ziyun systematically reviewed and synthesized 70 government-funded and industry-produced environmental health studies published between 1968 and 2007. Her synthesis focused on 29 reports about health impact assessment and risk assessment in the Boat Harbour ecosystem as they most closely related to the Pictou Landing Native Women's Group's concerns, and resulted in three areas of findings: (1) water quality, (2) air quality, and (3) remediation options.

1. The water quality synthesis includes findings from Health Canada as well as the Canada and Pictou Landing First Nation Joint Environmental and Health Monitoring Committee (JEHMC). JEHMC has been directly sampling from the community drinking water system with three production wells to fulfill Canada's mandatory Environmental Health Program for all Aboriginal communities. Community drinking water has been sampled twice a year and then compared to the Guidelines for Canadian Drinking Water Quality (Health Canada, 2014). The JEHMC also sampled weekly for bacteria between 1998 and 2004. The most recent report (2005) from the JEHMC (provided by the PLNWG) reported there were no negative impacts to the community drinking water quality. Earlier negative observations included a drop in groundwater level, which has led to elevated levels of lead and barium. In addition, there were four instances of bacterial contamination in community drinking water, most likely resulting from activities near the water supply wells or the distribution pipes.

2. In terms of air quality, very few site-specific air quality monitoring events had been conducted at the time of this review. There is therefore very little information available on air pollutants in and around Boat Harbour. Starting in 1991, Nova Scotia Environment began monitoring total reduced sulfur in Pictou County, and the mill implemented additional emission controls in that same year. In February and July of 1995, Environment Canada assessed the levels of dioxins and furans in the ambient air in Pictou Landing First Nation to address community concerns about smog. Environment Canada's (1996) study findings did not find elevated levels of dioxins and furans, so no changes in operations by the mill were required.

3. Remediation options have included the installation of a rock berm in 1991 to control fish kills in the estuary. The rock berm was partially removed in 1993. Two other options were suggested in the 1990s: (1) Diffusing the effluent from the Aerated Stabilization

Basin to the Mackenzie Head and a plan for shoreline remediation, and (2) Opening Boat Harbour to tidal flush (opening it up to the Northumberland Strait again). To date, none of the proposed remediation promises have been enacted.

Three key gaps were found through this review:

1. Legislation in Nova Scotia does not (and has never) required human health risk assessments. There was one conducted in 2004 regarding the decommissioning of the Boat Harbour Effluent Treatment Facility, but as with other assessment processes the focus was only on physical health.

2. Federal pulp and paper effluent regulations left a gap of more than 20 years for the mill to operate without wastewater quality control.

3. There has been little research on the effects of tidal flush on deep water ecosystems. The impact of sediment from Boat Harbour travelling out to the ocean is unknown, and it is likely to be a major one. It is thought that this would speed up the eutrophication process because diluted effluent would increase light availability, meaning algae would be able to grow. Eutrophication is the result of increased algae (and other plant growth) in a water body, which reduces the amount of dissolved oxygen that other life forms (e.g., fish) in the water need to live. To address the concern of sediment release, dredging could be performed; however, our review found literature stating it is likely that only 50% of contaminated sediment could be removed, and that if the wastewater continued to flow into Boat Harbour, then it would not be possible to complete the dredging.

5. Environmental Health Survey

Alaptmeg aqq mawte'meg mst gogwe'l klamon ula utan jajigkaktow/Looking and Gathering Everything so This Community Will Be Healthy – Identifying, Documenting, Mapping, and Mobilizing Environment and Health Knowledge in Pictou Landing: An Environmental Health Survey by Diana Lewis, PhD Candidate

The Environmental Health Survey (EHS) began development in 2010 soon after the Pictou Landing Native Women's Group (PLNWG) had been meeting regularly to fundraise for and support community and cultural activities and to discuss community priorities. They asked to meet with representatives of Dalhousie University to explore the possibility of working together on a health research project to determine whether the community was getting sick from Boat Harbour. The PLNWG envisioned developing a survey instrument that would move beyond anecdotal health stories to reliable data about the community's health.

The overarching goal of the door-to-door EHS was to assess community-wide concerns and perceptions of environmental impacts, health problems, and access to healthcare, as well as the impacts on traditional, cultural, and spiritual activities affecting residents of the community. Prior to the survey, no independent scholarly study had specifically examined the relationship between the environmental contamination at Boat Harbour and its influence on the health of the people who live in Pictou Landing First Nation (PLFN). The EHS serves as the focus of **Diana** (**Dee) Lewis**'s PhD research, and she led the survey development.

The gap in knowledge about Mi'kmaw health is, in part, due to the fact that while Statistics Canada regularly undertakes major national surveys on the health of Canadians, Aboriginal people living on reserve have often been excluded (Mi'kmaq Health Research Group, 2007). In 1997, the Assembly of First Nations began collecting data, developing the First Nations Regional Health Survey (RHS) for longitudinal study of First Nations and Inuit health, and it continues to do so today. The 13 Mi'kmaw communities in Nova Scotia participate in the survey, but the sample size for Pictou Landing is too small to disaggregate the data from the regional data set, and would not show statistical significance even if this were possible. Most importantly, the focus of the previous RHS survey was not on environmental health.

The definition of health used in the survey reflects the National Aboriginal Health Organization's definition that health is a balance between the "physical, mental, emotional and spiritual realms as well as the environment, culture, family, and community" (First Nations Centre, 2007, p. 1). The EHS was revised and adapted from the RHS and "Our Environment, Our Health: A Community-based Participatory

Environmental Health Survey in Richmond, California" (Cohen, Lopez, Malloy, & Morello-Frosch, 2012).

The PLNWG agreed to retain 140 questions from the two surveys which covered a wide range of health questions, including physical, mental, emotional, and spiritual health; as well, 157 new questions were added that were central to the cultural, intergenerational, and spiritual concerns of the community. Dee worked with the women over several months to ensure all of the questions they wanted included in the survey were there, and the questions, in relation to the needs of the community, would be answered. She also consulted with the "Science Guys," **Daniel Rainham, Mark Gibson, Rob Jamieson**, and **Ron Russell**, to include questions that would be helpful to them with their work. The final EHS was a 297-question, 70-page survey instrument that took 90-120 minutes to complete.

The survey collected demographic information (age, education, employment, income – Tables 2 and 3); residence (air quality in home, water quality); health (perceptions of health, chronic conditions, access to health care, skin conditions, allergies, cancers, family history); traditional, cultural, spiritual information (ability to practice, access); residential school attendance; experiences of racism; and measures of quality of life, perceptions, and beliefs. These questions, unlike the RHS, allowed us to get to household-level data, which was the goal of the PLNWG.

Dee trained PLNWG members to administer the household-level Environmental Health Survey, and data collection began in November 2012. The first round of community research assistants to conduct the survey with Pictou Landing residents were **Haley Bernard, Colleen Denny, Pam Denny, Jordan Francis, Sheila Francis,** and **Kim Strickland**. Seven additional surveyors were also trained in the fall of 2013: **Darlene Bachiri, Holly Francis, Sylvia Francis, Heather Mills, April Nicholas, Fran Nicholas,** and

Loretta Sylliboy. The survey concluded on December 31, 2013, with a total of 279 out of a potential 470 survey respondents – an exceptional 60% response rate.

Dee has been able to compare EHS responses with data reported in 2013 in *The Health of the Nova Scotia Mi'kmaq Population* (NS RHS), using Nova Scotia First Nations Regional Health Survey data from 2008/10. She is also using publicly available data from the 2012 *National Report on Adults, Youth and Children Living in First Nation Communities* (N RHS), using national First Nations Regional Health Survey data from 2008/10 (FNIGC, 2012). Dee requested and was granted access to Statistics Canada data at the Atlantic Regional Data Centre at Dalhousie University, including the Canadian Cancer Registry, the Canadian Community Health Survey, the Survey on Living with Chronic Disease in Canada, and the Maternity Experiences Survey. As she continues with her analysis of the data in her PhD thesis, she will compare these data to the EHS and identify trends.

Dee's full analysis will be reported in a complete *Community Report – Environmental Health Survey*. Here, we provide a brief overview of key findings.

Table 2
Environmental Health Survey – Demographics

Variable	Category	Frequency (Total)	%
Gender	Female	150	56
	Male	117	44
		(267)	(100)
Marital status (Adults only 18 years +)	Single, never married	89	51
	Married, common law	62	36
	Separated, divorced, widowed	22	13
		(173)	(100)
Ages	0-11	46	18.1
	12-19 years	47	18.5
	20-29 years	45	17.7
	30-39 years	42	16.6
	40-49 years	28	11.0
	50-59 years	26	10.2
	60 years and older	20	7.9
		(254)	(100)
Highest level of education completed (Adults only 18 years +)	Less than high school	66	39
	High school graduate	54	32
	College/trade school	40	24
	Bachelor	7	4
	Graduate	2	1
		(169)	(100)

Table 3
Environmental Health Survey – Employment and Income

Variable	Category	Frequency (Total)	%
Employment status (Adults only 18 years +)	Employed F/T	37	22
	Employed P/T or seasonal	41	24
	Unemployed	73	43
	Retired	6	4
	Never worked	12	7
		(169)	(100)
Income ($) (if working)	Less than 10,000	16	20
	10,000 to 19,999	23	28
	20,000 to 29,999	19	24
	30,000 to 39,999	13	16
	40,000 to 49,999	2	2
	Over 50,000	8	10
		(81)	(100)
Job category	Management/professional	13	13
	Administrative/sales	16	16
	Trade, transport, equipment operator	9	9
	Fishing, hunting, forestry, mining	24	24
	Care worker	5	5
	Student/Other (research, gardening, security, etc.)	32	32
		(99)	(100)
Source of income (if not working*)	Unemployment insurance	27	21
	Social assistance	59	47
	Pension/disability	8	6
	Child Tax Benefit/CTB and other	18	14
	Social assistance and other	7	6
	Education allowance/other	7	6
		(126)	(100)
If not working, why? (Adults only 18 years +)	Illness/disability	13	12
	Caring for family	19	17
	Seasonal layoff	13	12
	Retired	6	5
	No work available	26	24
	Student/other	33	30
		(110)	(100)

In surveys, self-reported health (Figure 3) is deemed a meaningful and reliable measure of current health that has been tested against mortality rates and results of clinical examinations. Beyond that, perceived self-rated health is accepted as a robust broad indicator of health status and wellbeing, incorporating the physical, emotional, and

personal aspects of health, and over the years it has maintained its reliability as a measure of current health. Moreover, self-rated health has been shown to be valid for use in different cultural groups including Indigenous populations, who have different perceptions of health based on a relational worldview (Chandola & Jenkinson, 2000; Sibthorpe, Anderson, & Cunningham, 2001; Strawbridge & Wallhagen, 1999; Wilson, Rosenberg, & Abonyi, 2011).

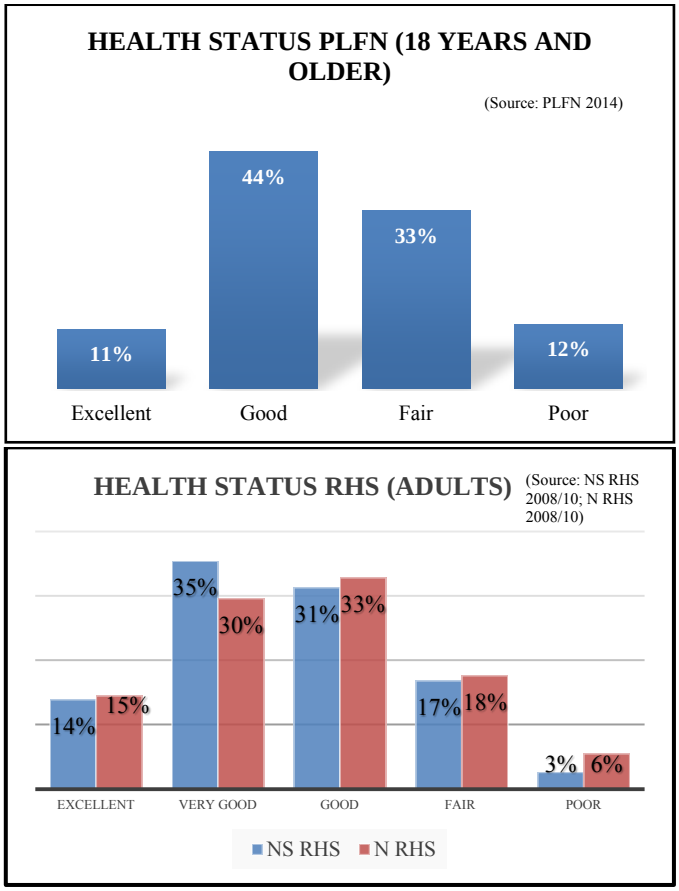


Figure 3. Environmental Health Survey – Self-reported health status (PLFN – Pictou Landing First Nation; NS RHS – Nova Scotia Regional Health Survey; N RHS – National Regional Health Survey).

For all adults aged 18 years and older in PLFN, 55% report their health as good to excellent compared to 81% of Nova Scotia First Nation adults on reserve and 77% of all First Nation adults on reserve nationally. Conversely, 45% of PLFN adult respondents report that their health is fair to poor, while 19% Nova Scotia First Nation adults on reserve and 23% First Nation adults on reserve nationally do.

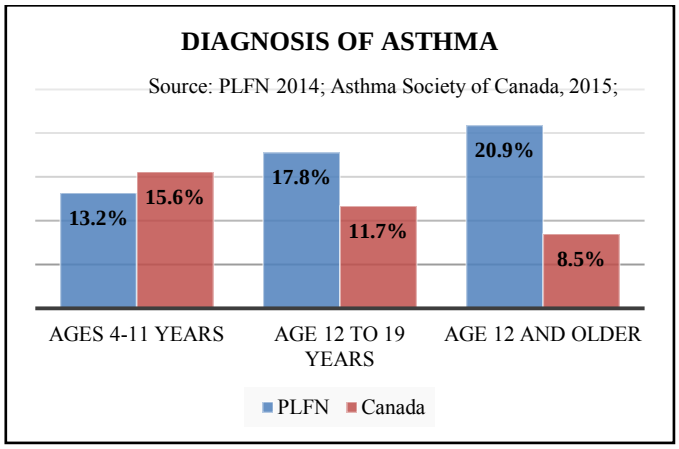


Figure 4. Environmental Health Survey – Asthma (PLFN – Pictou Landing First Nation).

According to the Asthma Society of Canada (2015), asthma is a disease of the lungs in which the airways become blocked or narrowed causing breathing difficulty. It is divided into two types: allergic (extrinsic) asthma and non-allergic (intrinsic) asthma. There are a number of potential causes, including heredity, the environment, and an impaired immune system. Risk factors (triggers) include a family history of asthma/allergies (including eczema and allergic rhinitis), exposure to tobacco smoke, mould, or pollen, and exposure to chemicals, odours, or pollution.

Figure 4 shows that younger Pictou Landing residents reflect similar percentages as other Canadians, but of PLFN youth aged 12 to 19 years, 17.8% have been diagnosed with asthma, compared to 11.7% of Canadian youth ages 12 to 19 years. Almost 2.5 times more PLFN community members age 12 and over have been diagnosed than in the general population. No one under the age of 4 has been diagnosed with asthma.

One trigger for asthma, as noted above, is mould or mildew in the home. Figure 5 shows that PLFN homes have more problems with mould and mildew than other First Nations communities.

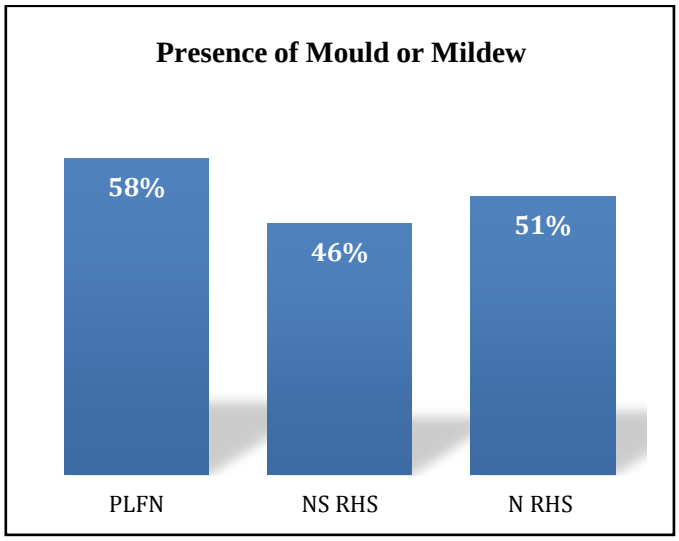


Figure 5. Environmental Health Survey – Mould or mildew in the home (PLFN – Pictou Landing First Nation; NS RHS – Nova Scotia Regional Health Survey; N RHS – National Regional Health Survey).

More than twice as many people from Pictou Landing do not think their water is safe to drink as compared to a national survey of Canadians (Figure 6, top chart). Consequently, more than 80% of PLFN respondents worry that the community water supply will impact their health (Figure 6, middle chart). As a result, 76% of respondents in PLFN drink bottled water compared to 71% of respondents on reserve nationally, and compared to 29% of Canadians in general (Figure 6, bottom chart).

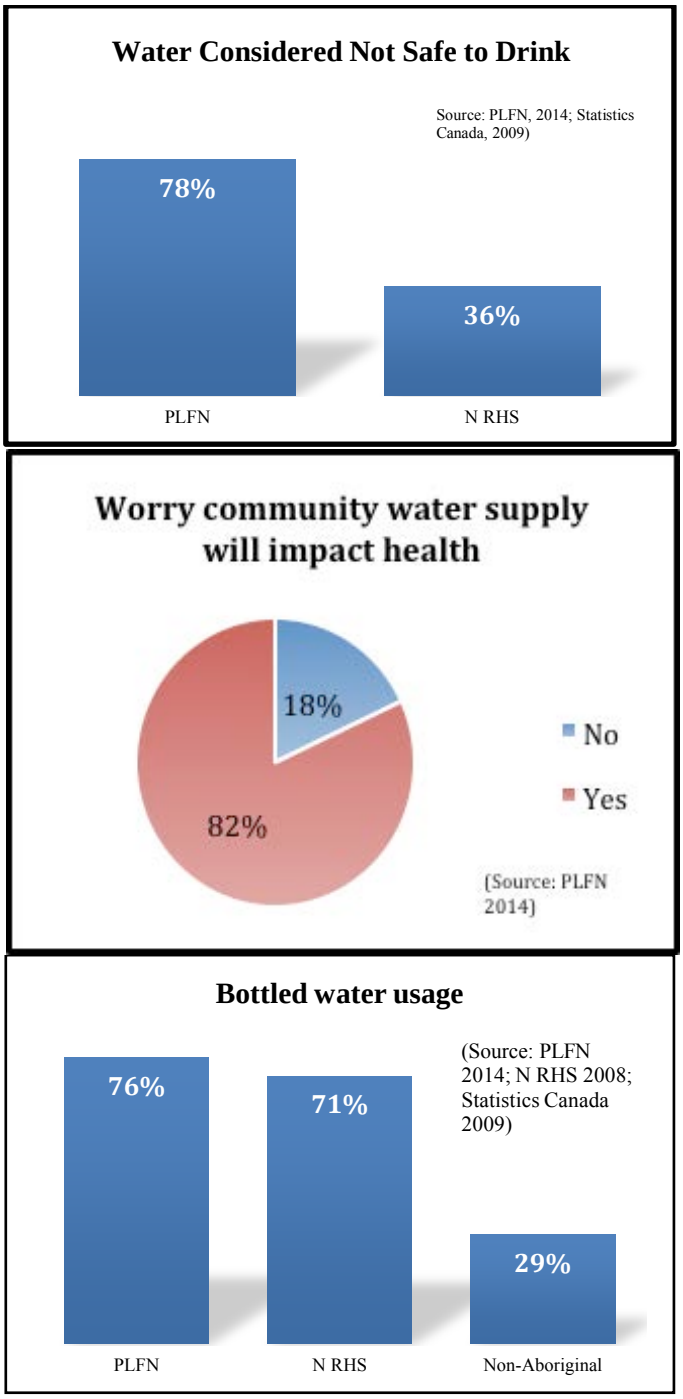


Figure 6. Environmental Health Survey – Safety of drinking water (PLFN – Pictou Landing First Nation; N RHS – National Regional Health Survey).

The Canadian Community Health Survey does not ask about mental health in the same way we asked about it in the EHS survey of PLFN members. However, when asking Canadians 12 years of age or older about their perceived mental health in 2013, 8%

report their mental health as fair to poor (Statistics Canada, 2013).[6] In comparison, 68% of PLFN residents of the same age report that they sometimes or often feel down or depressed.

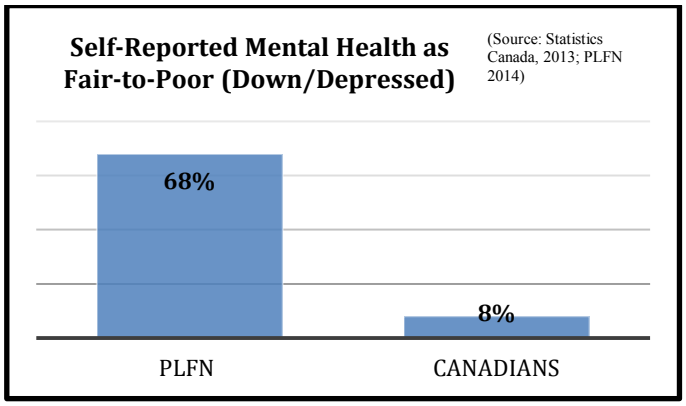


Figure 7. Environmental Health Survey – Mental health (PLFN – Pictou Landing First Nation).

Respondents were asked about whether they practise traditional activities now and whether they had practised these activities in the past (Figure 8) . All activities – use of traditional medicines, eating game, gathering shellfish and berries – showed a significant drop.

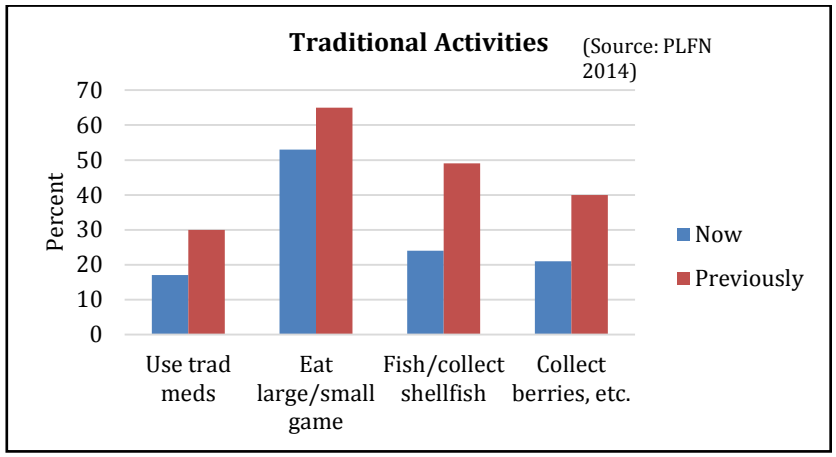


Figure 8. Environmental Health Survey – Traditional practices.

Figure 9 shows that 40% of PLFN members experienced racism in the past year. Racism, and the inevitable stress of being socially excluded, is damaging to health and wellbeing (Reading & Wien, 2009; Ziersch, Gallaher, Baum, & Bentley, 2011). Krieger

[6] Source: Statistics Canada, Canadian Community Health Survey. CANSIM table no(s).: 105-0501 (rates), 105-0503 (age-standardized rates).

(2001) defines racial discrimination as the process by which members of a socially defined racial group are treated unfairly because of membership of that group. Racism can occur at three levels – institutional, interpersonal, and internalized. It is important to understand how racism is constructed and practised, and rather than focusing on the disadvantaged position of the individual, it is also important to consider the impact of "whiteness and its associated privileges" and the inequity that produces (Durey, 2015, p. 197). Being subjected to discrimination and racism, it is now recognized (Castleden, Martin, & Lewis, 2016), has significant impacts on health.

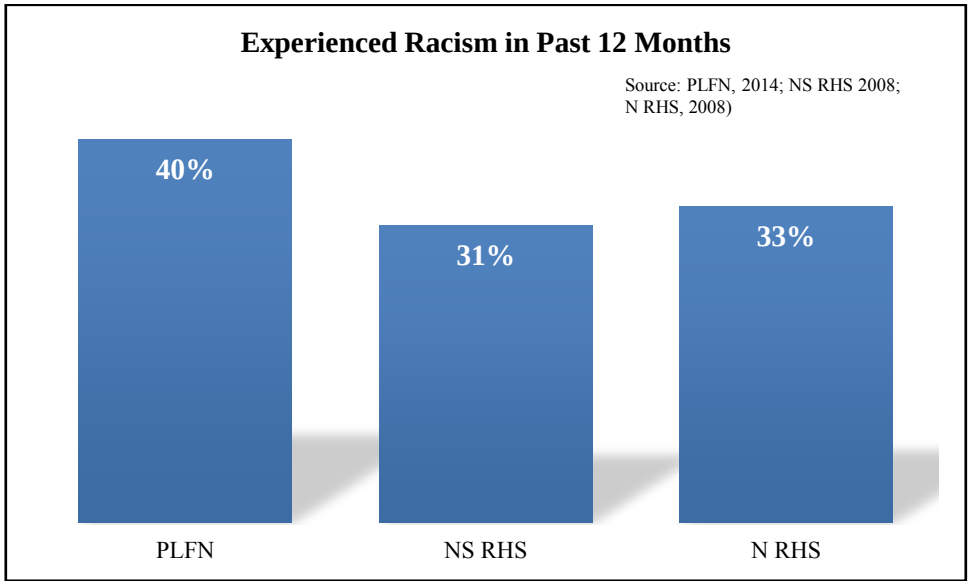
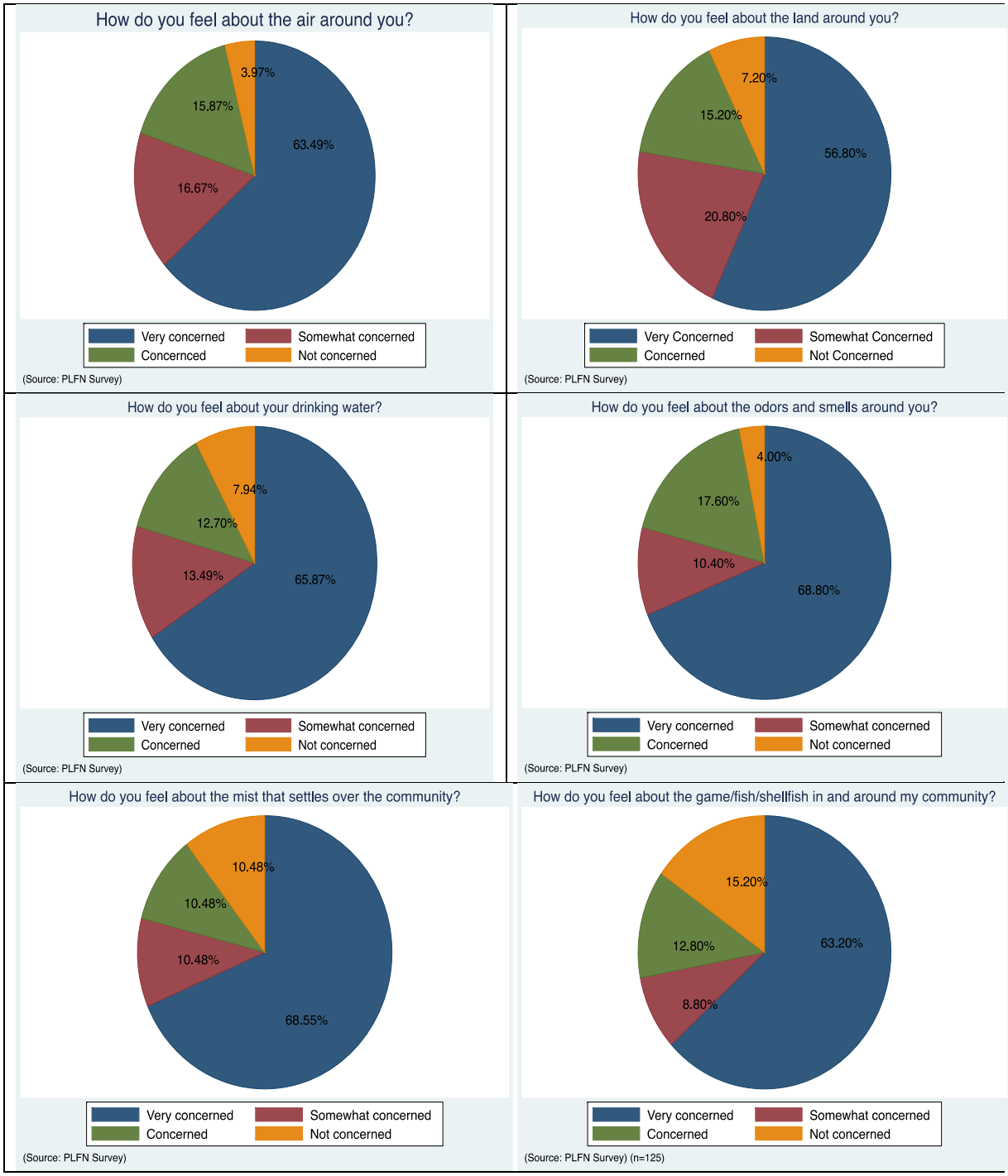
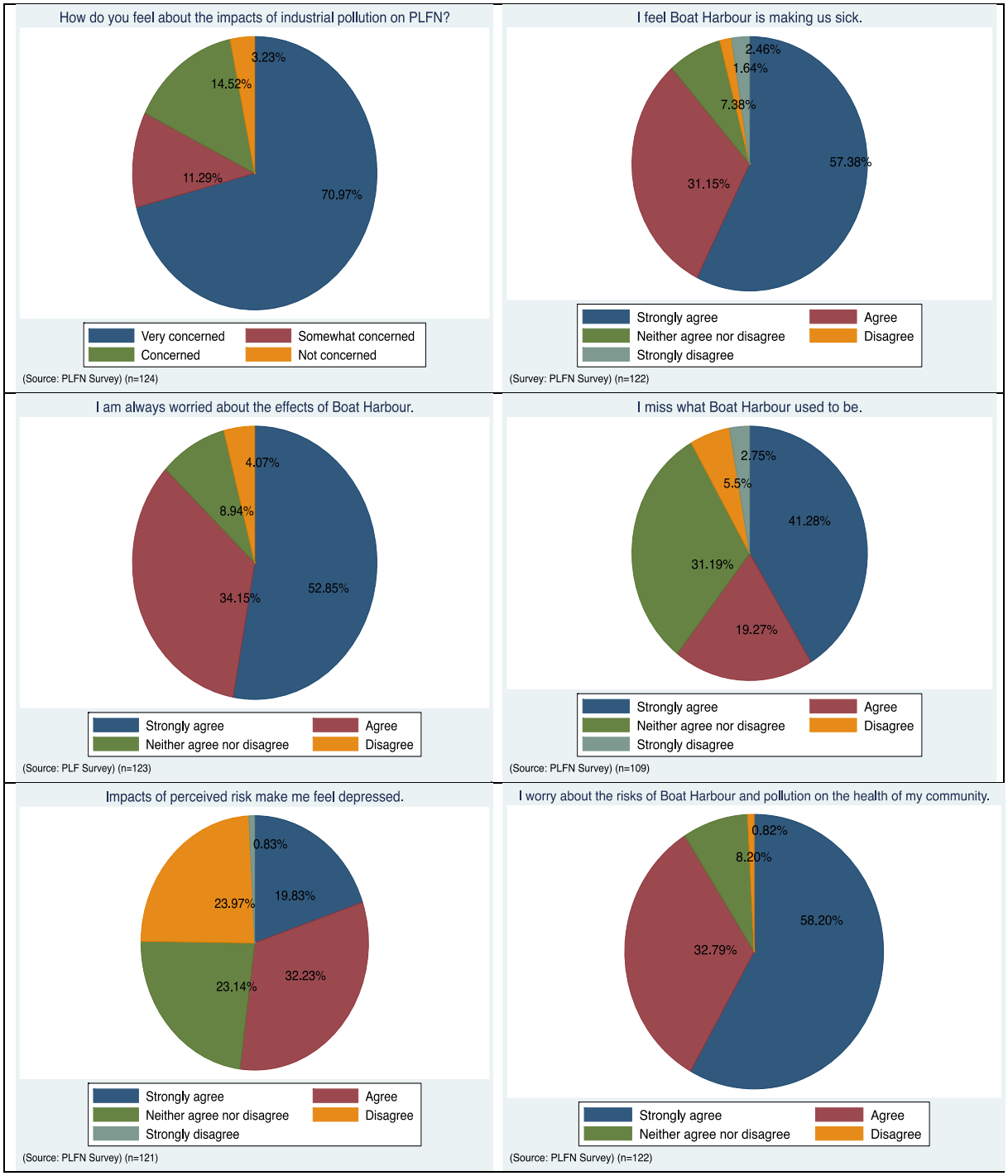


Figure 9. Environmental Health Survey – Racism (PLFN – Pictou Landing First Nation; NS RHS – Nova Scotia Regional Health Survey; N RHS – National Regional Health Survey).

During the collaborative development of the EHS survey questions, the PLNWG expressed concern about the air and water that surrounds their community, the odours that the members are exposed to, and the mist that settles on their land. We therefore developed the survey to determine whether the community as a whole was feeling the same types and levels of concern. As evidenced in the charts (Figures 10-21), it is clear that self-reported perspectives on their local environment convey a high level of concern and worry about the impacts that Boat Harbour is having on the health of the community through many different vectors, with most residents reporting that they agree or strongly agree that Boat Harbour is making them sick.



Figures 10-15. How do you feel about …?



Figures 16-21. How do you feel about …?

Summary

When we compare PLFN adult respondents' self-reports about poor health outcomes with those of other First Nations, both provincially and nationally, PLFN adults are reporting higher rates of poor health. Particularly worth noting is that more than half (approximately 60%) of adults in PLFN miss what Boat Harbour used to be when it was known as A'se'k, even though they may have not experienced A'se'k within their lifetime, having only heard stories that have been passed down to them over the years from their Elders.

Not only are PLFN residents experiencing poorer health outcomes compared to other First Nations, both provincially and nationally, they are at a disadvantage socioeconomically. The majority of households are headed by single parents, with a level of education at high school or less. Half the adult respondents 18 years or older are either unemployed or have never worked, and even if working, almost half exist on incomes below $20,000 a year. Almost half of the households in PLFN exist on social assistance alone. If, indeed, the pulp mill was brought to Nova Scotia to benefit the residents of the region, these benefits have clearly not materialized in the community that may be paying the highest price.

6. Air Quality

The Atmospheric Forensics Research Group's part of the project was led by **Mark Gibson** (Associate Professor, Department of Civil and Resource Engineering, Dalhousie University). PLNWG members **Kim Strickland** and **Colleen Denny** were hired as Community Research Associates to work with Research Assistants **Geoff Kershaw**, **James Kuchta**, and **Codey Bennett** to manage the monitoring equipment and to sample. Air quality data were collected during the summer of 2013, spring of 2014, and summer of 2014. Harsh winters and a loss of power to the equipment that needed electricity to run real-time measurements meant the winters of 2013 and 2014 were excluded, reducing data completeness. Once the air quality monitoring was completed, the results were compared to existing data and regulatory air standards.

A total of 13 samplers (11 passive samplers and 2 real-time samplers) were set up throughout the community to sample indoor and outdoor air quality (see Figure 22).

Figure 22. Map of passive and active air monitoring sites used during the air quality monitoring.

Passive Sampling

Eleven passive samplers were set up around Pictou Landing, Boat Harbour, and downwind between the mill and the community. Wind patterns, the terrain, and distance were all taken into account with site selection. Passive sampling requires the sample to be transported to a lab to be analyzed. Two passive air samplers were used: Ogawas and thermal desorption tubes (TDTs; Figure 23). Ogawas and TDTs were paired at all 11 passive sampling sites, where the Ogawas measured **ammonia, sulfur dioxide**, and **nitrogen dioxide**, and the TDTs measured **volatile organic compounds (VOCs)**.

Figure 23. Deployment of passive samplers: Ogawa (left) and thermal desorption tube (right).

Real-time Sampling

The real-time monitors were set up at two sites (Mary Ellen's and Geoff Hatin' Us) and included monitors called VRae, Dylos, and DustTrak, monitoring the air at the two sites for one week. Real-time sampling allows the sample to be analyzed at the sample location.

· VRae measured **ammonium, nitrogen dioxide**, **sulfur dioxide,** and **hydrogen sulfide** (rotten egg smell). A VRae real-time monitor was set up to measure carbon monoxide, hydrogen sulfide, oxygen, and ammonia but was unsuccessful due to a terminal malfunction of the instrument.
· The Dylos measured the **number of particles** smaller than 2.5 µm (called $PM_{2.5}$) and 10 µm (PM_{10}).

· The DustTrak measured the **concentration** of the tiny dust particles of 2.5 µm, which you breathe in but are too small to see (e.g., nitrogen dioxide, sulfur dioxide, and ammonia). The particles come from a mixture of natural and human-made sources (e.g., sea salt spray, wind-blown dust, wood smoke, smokestacks, vehicle exhaust, aerosolized droplets from the Boat Harbour lagoon).

One 48-hour real-time sample of **dioxins and furans** was taken at two sites. No dioxins or furans were determined in the air at Pictou Landing First Nation. According to the National Pollution Release Inventory (publicly available and self-reported data), Northern Pulp releases 0.008 g-TEQ (toxic equivalency) of dioxins and furans per year, which can be considered a very small amount similar to other industries that also emit dioxins and furans (Environment Canada, 2013). An attempt was made to model the emission of dioxins and furans from the Northern Pulp mill using the publicly available emissions rates. However, because the emission rate is extremely small (2.535×10^{-10} g/sec) the model could not run because the surface concentrations were so low and completely uniform across the model, including Pictou Landing First Nation. This further highlights the low air emissions of dioxins and furans that are reported as being released from Northern Pulp.

Results

Ammonia

The highest ammonia concentration was found at the Settling Pond Outfall, with the second highest ammonia concentration found at Ground Zero (see Figure 24). The lowest ammonia concentrations were upwind of Boat Harbour. This is reasonable evidence to suggest that the elevated ammonia found at the Settling Pond Outfall and at Ground Zero are due to emissions from the mill (Ground Zero) and Boat

Harbour treatment lagoon. However, the ammonia concentrations observed in Pictou Landing First Nation are well below any ambient and occupational exposure limits and do not pose any health concern (Alberta Environment, 2005) – see Table 4. Ambient standards were not found; therefore occupational standards were used here.

Table 4
Ammonia (NH_3) *Concentration Results*

Seasonal mean (average)		Total mean	Maximum NH_3 observed	Human health occupational exposure limit (8 hours)
Summer 2013	4.90 ppb			
Spring 2014	1.09 ppb	4.39 ppb	15.25 ppb	25 ppb
Summer 2014	3.18 ppb			

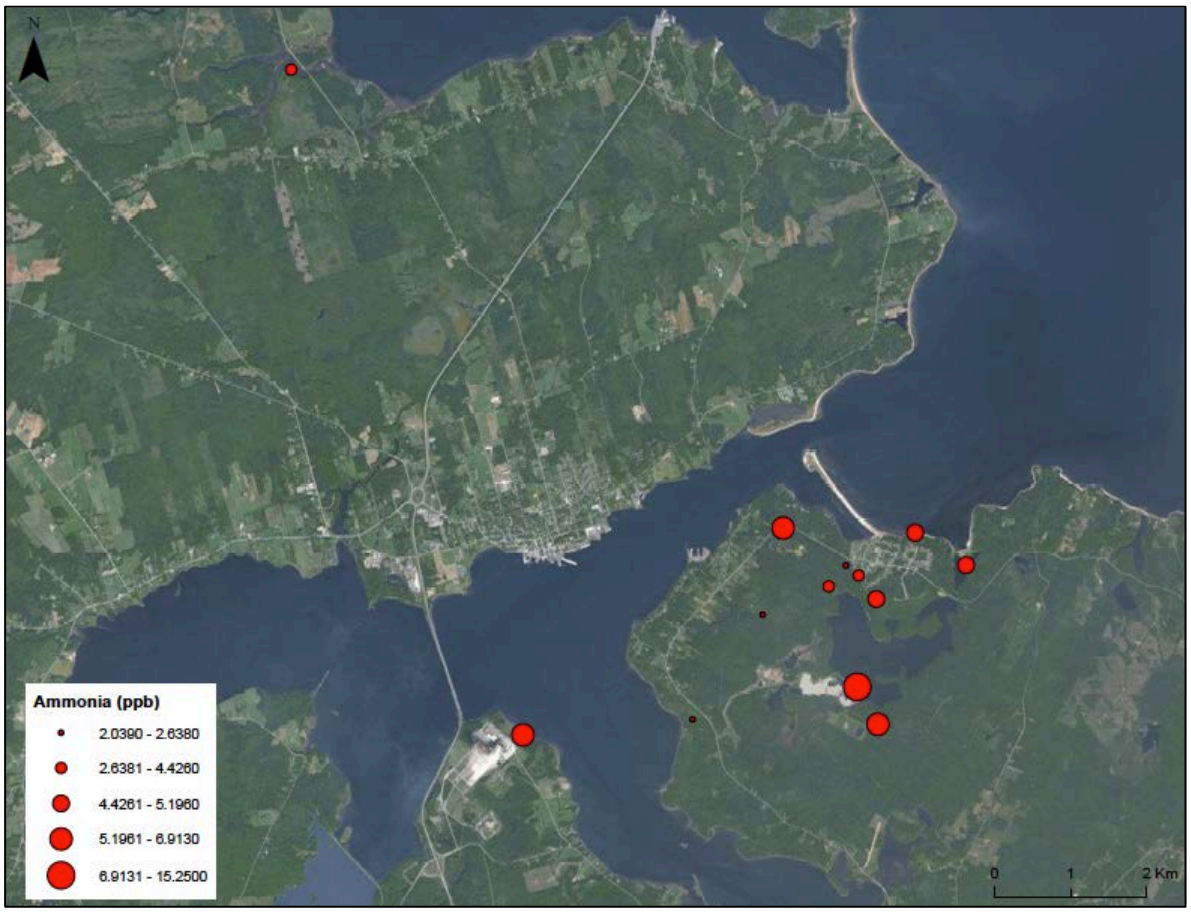


Figure 24. Spatial map of the average ammonia concentrations observed June 27, 2013 to July 31, 2014.

Sulfur Dioxide

The annual average **sulfur dioxide** concentration from Pictou Landing First Nation was 0.62 ppb, roughly double that found in Sydney and Cape Breton Highlands National Park, but still well below Canada's annual maximum air quality standard (22.9 ppb) – see Table 5.

Table 5
Sulfur Dioxide (SO_2) *Concentration Results*

Seasonal mean (average)		**Total mean**	**Maximum SO_2 observed**	**Canada's annual maximum air quality standard**
Summer 2013	0.70 ppb			
Spring 2014	0.41 ppb	0.62 ppb	5.56 ppb	22.9 ppb
Summer 2014	3.18 ppb			

The sulfur dioxide in Pictou Landing is likely not related to the Boat Harbour lagoon, but can be considered more of a background air pollutant carried from upwind sources that likely include some emissions from the mill and other local sources such as Michelin Tires and residential heating oil combustion.

A possible, but not definitive, reason for the increase in sulfur dioxide observed downwind of the mill is that stack plumes are impacting the ground as one moves away from the mill. This hypothesis is further supported because the prevailing wind is from the direction of the mill.

The sulfur dioxide concentrations are higher at the Boat Harbour Outfall (see Figure 25). This is an unexpected result as there are no strong sources of sulfur dioxide found at this site. However, it could be due to sea breezes carrying sulfur dioxide back onshore. Still, the sulfur dioxide concentrations observed across all sampling sites and sampling seasons are well below the US Environmental Protection Agency's National Air Quality Standards, even the 1-hour maximum level (75 ppb), and the Canadian Council of Ministers of the Environment Canada Wide Standard maximum annual acceptable amount (22.9 ppb).

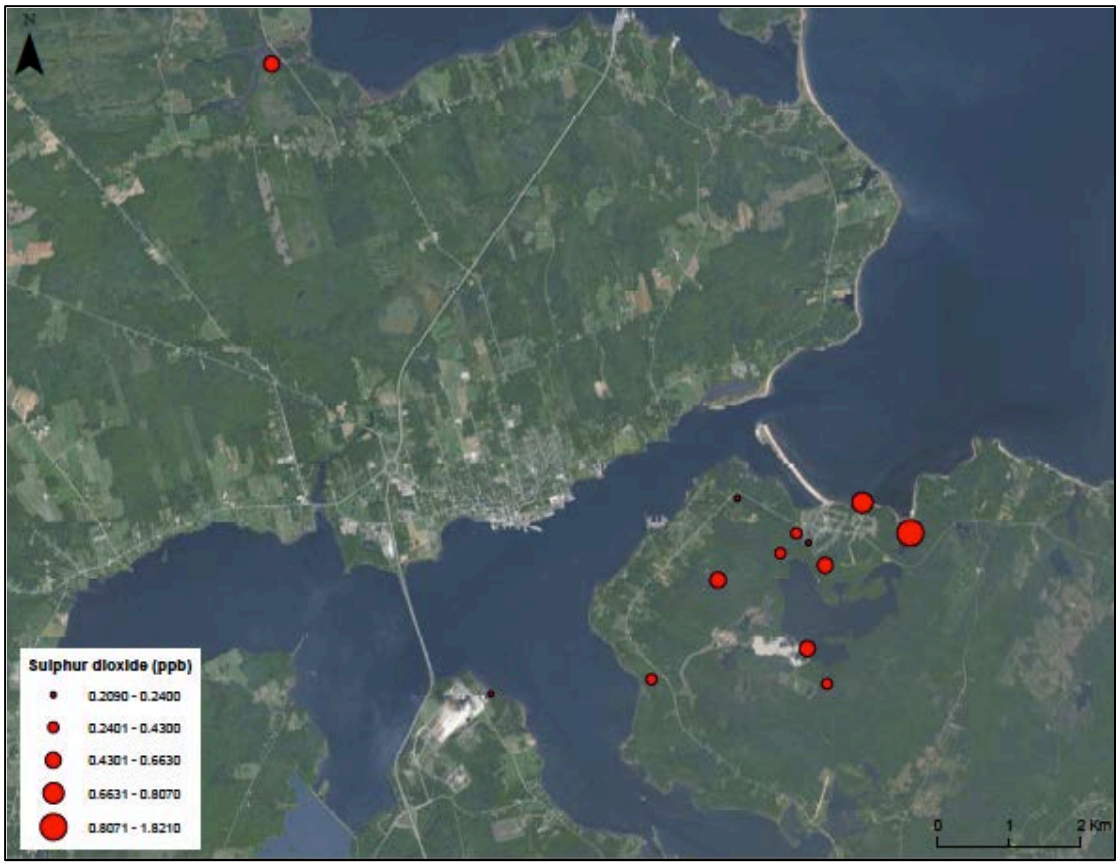


Figure 25. Spatial map of the average surface sulfur dioxide concentrations observed June 27, 2013 to July 31, 2014.

Nitrogen Dioxide

The nitrogen dioxide observed in Pictou Landing First Nation (see Table 6) can be considered to be similar to a city the size of Sydney, but still very low compared to larger cities in Canada that can experience nitrogen dioxide of approximately 10 ppb (metropolitan city roadways range between 22 ppb and 32 ppb; Brook, Dann, Galarneau, Herod, & Charland, 2014). As nitrogen dioxide is strongly linked to high-energy chemical reactions, for example, combustion of fuel for power, space heating, and transport, the nitrogen dioxide observed would be a mixture of background long-range transport into Pictou Landing First Nation likely combined with the mill, Michelin, New Glasgow, Trenton Power Station, residential heating and cooking, and local vehicle traffic. The highest average nitrogen dioxide (2.5-3.5 ppb) is found at the Boat Harbour aeration lagoon outflow (Settling Pond Outfall site; see Figure 26).

Table 6
Nitrogen Dioxide (NO_2) *Concentration Results*

Seasonal mean (average)		Total mean	Maximum NO_2 observed	Canada's maximum desirable annual average
Summer 2013	1.38 ppb			
Spring 2014	0.75 ppb	1.25 ppb	6.02 ppb	32 ppb
Summer 2014	3.18 ppb			

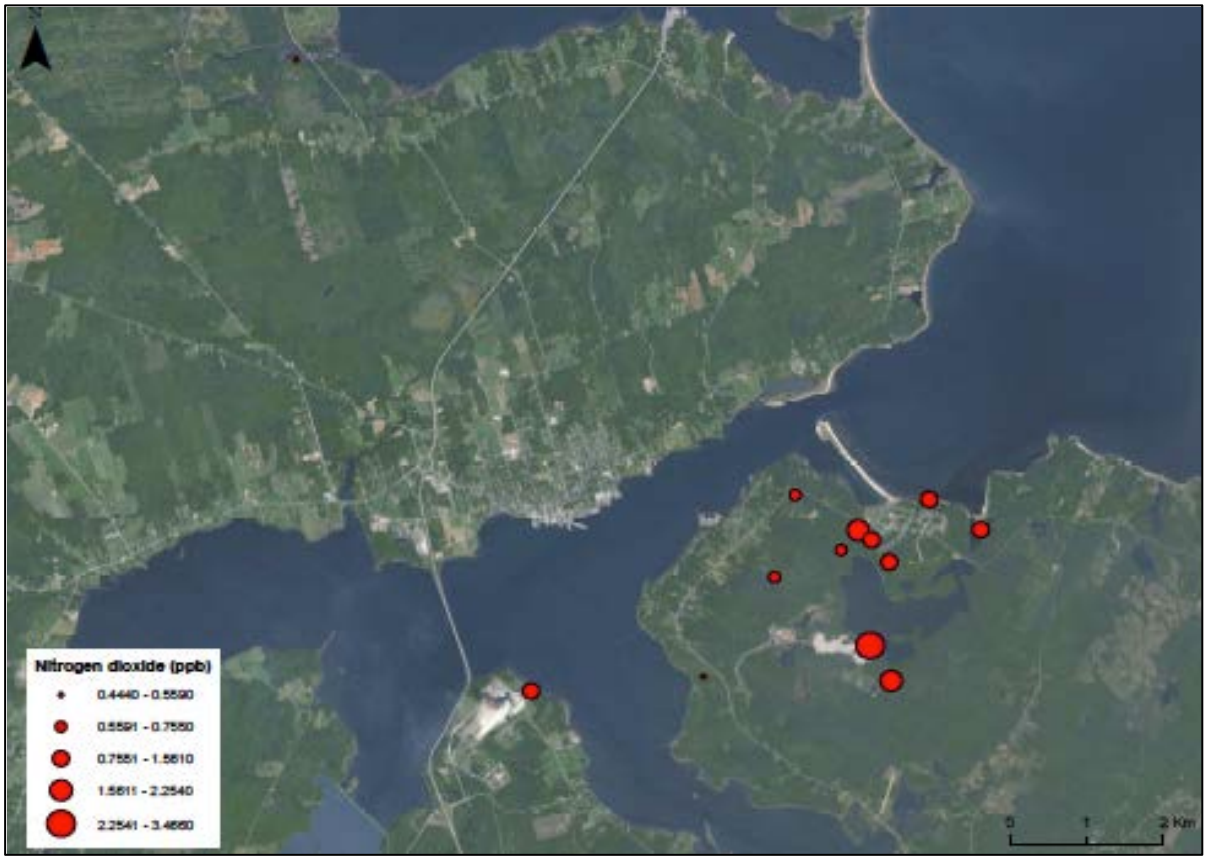


Figure 26. Spatial map of the average surface nitrogen dioxide concentration observed from June 27, 2013 to July 31, 2014.

As nitrogen dioxide is related to combustion, it is highly likely this is either diesel power equipment related to the aeration lagoon or lagoon service vehicles. The mean nitrogen dioxide concentrations are well below Canada's maximum desirable annual average regulations (32 ppb).

Volatile Organic Compounds

To help place the VOCs measured at Pictou Landing into context, two samples of VOCs were collected in Halifax August 17-24, 2013, and August 24-31, 2013, and compared with VOC samples collected between June 28 and July 4, 2013, at Boat Harbour Outfall and Caribou Control. The comparison is shown in Figure 27.

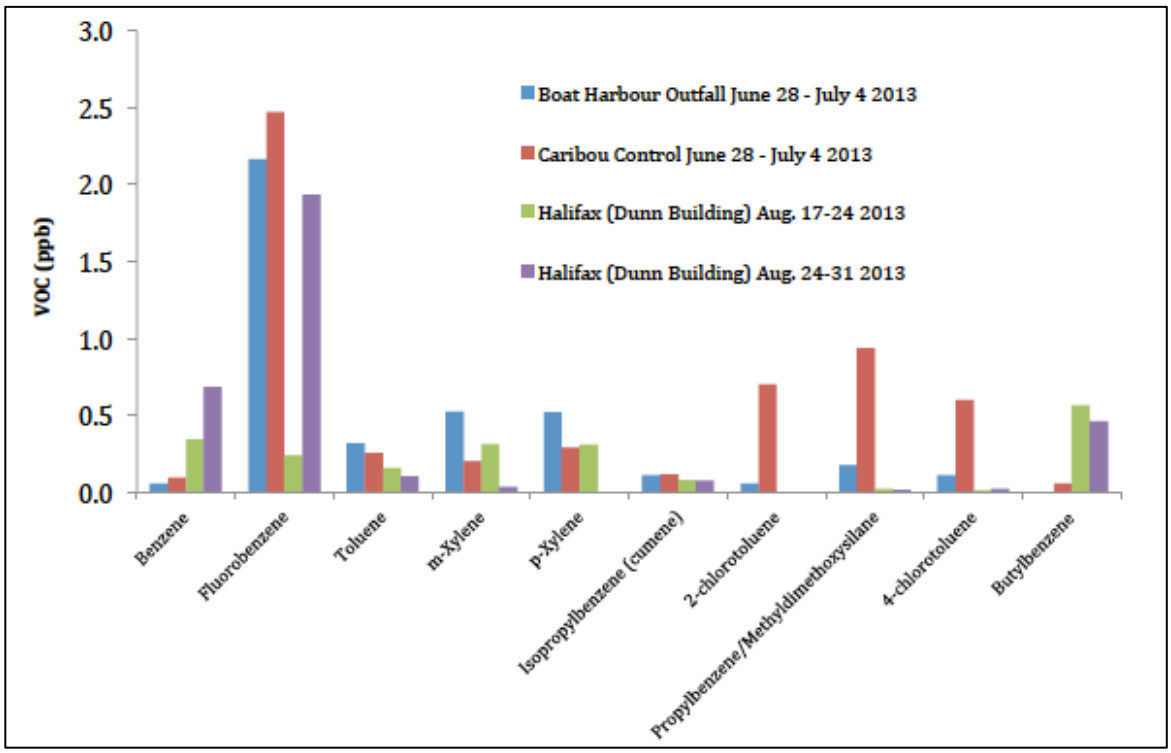


Figure 27. Comparison of volatile organic compounds (VOCs) measured at Pictou Landing First Nation with samples collected in Halifax.

In this regional comparison, five VOCs (fluorobenzene, 2-chlorotoluene, propylbenzene, methyldimethoxysilane, and 4-chlorotoluene) were observed in greater amounts at Caribou Control and Boat Harbour Outfall compared with Halifax. The use of chlorine and fluorine in the kraft pulp and paper process and the large amounts of aromatic substances from the tree pulp and other chemicals are well known. The higher concentrations of these VOCs found at Caribou Control and Boat Harbour are likely related to emissions from the mill and being carried by the prevailing wind to Caribou Control and entering Boat Harbour by the mill's effluent stream. The fact that these chemicals tend to be lower in the Halifax also adds weight to this argument.

The vehicle-related VOCs (benzene, toluene, and xylenes) were strongly associated with proximity to roads within Pictou Landing First Nation. The majority of VOCs that had higher concentrations within Pictou Landing appeared not to be associated with

Boat Harbour. Their source is likely related to vehicle emissions and wood and fossil fuel combustion for space heating and cooking. It is difficult to determine if any of these VOCs are related to the mill at this time. All of the VOCs measured were below ambient/indoor/occupational air quality guidelines.

Particulate Matter

$PM_{2.5}$, which is associated with gas-to-particle conversion or from combustion, accounts for 64% of the PM_{10} mass concentration. Approximately 70% of the $PM_{2.5}$ mass observed in Nova Scotia is from long-range transport from the northeastern United States and southern Ontario, carried to the region by the prevailing wind (Gibson et al., 2015).

Table 7 illustrates through fine particle number counts (associated with combustion and gas-to-particle conversions) and coarse particle number counts (related to wind-blown crystal material and ocean spray) that there are more fine particles per cubic centimetre that can be inhaled into our lungs than coarse particles. However, based upon these data, the concentration of $PM_{2.5}$ and fine number counts are very low when compared to Beijing, where $PM_{2.5}$ regularly reaches 500 $\mu g/m^3$. It can be observed in Table 7 that the maximum observed $PM_{2.5}$ concentration (35.45 $\mu g/m^3$) is greater than the Canada Wide Standard (28 $\mu g/m^3$). However, the maximum observed in Table 7 is for a five-minute average measurement and therefore not directly comparable with the Canada Wide Standard, which is an average of the 12 worst days measured over three years. It is common to see short-term "spikes" in monitoring over short periods of time such as five minutes. Once these are averaged over a day (e.g., 4.35 $\mu g/m^3$ in Table 7), they become much reduced and more comparable to the Canada Wide Standards. To conclude, airborne particulate matter mass and number observed in Pictou Landing First Nation are well below air quality standards where they exist.

Table 7
Particulate Matter (PM) Concentration Results

Particle size	Average observed PM concentration	Minimum observed PM concentration	Maximum observed PM concentration	Air quality guideline
$PM_{2.5}$	4.35 $\mu g/m^3$	0.91 $\mu g/m^3$	35.45 $\mu g/m^3$	28 $\mu g/m^{3*}$
PM_{10}	6.77 $\mu g/m^3$	2.73 $\mu g/m^3$	39.55 $\mu g/m^3$	40 $\mu g/m^{3**}$

***** Guideline is taken from the Canadian ambient air quality guidelines (*Canadian Environmental Protection Act*, 1999)

****** Guideline is European Commission Standard for annual average (Directive 2008/50/EC of the European Parliament); there is no PM_{10} standard in Canada

An analysis of wind direction for PM mass and number concentrations was also performed. The main wind directional dependence for all three PM size fractions is from the NW and SW. The SW wind direction is in line with Boat Harbour but also the town of New Glasgow and the major highway. It is therefore difficult to determine if Boat Harbour is the source of $PM_{10}/_{2.5}/_{1}$ or other upwind local and long-range sources, which warrants further investigation. The $PM_{10}/_{2.5}/_{1}$ associated with NW airflow are in line with Pictou Landing First Nation and the roads therein, so are likely associated with gas combustion.

Since air pollution cannot be monitored everywhere at the same time, American Meteorological Society and United States Environmental Protection Agency Regulatory Model (AERMOD) air dispersion modelling offers a solution by estimating the impact of emissions from point sources on surface air quality within any given modelling area (Gibson et al., 2009, 2013a, 2013b). AERMOD was used to model the air dispersion of $PM_{2.5}$ from the Northern Pulp Mill stacks. The run time of the stacks was assumed to be 24 hours a day for a whole year. The AERMOD results illustrate how Pictou Landing First Nation is impacted by the mill's stack emissions (see Table 8 below).

Table 8
Summary of AERMOD $PM_{2.5}$ Surface Concentrations Attributable to Emissions from the Northern Pulp Mill

AERMOD modelling period	**$PM_{2.5}$ concentration range (µg/m^3)**
1-hr, Annual maximum	2.1 – 96.7
Annual average	0.013 – 0.451
1-hr, Winter maximum	1.0 – 96.7
Winter average	0.009 – 0.440
1-hr, Spring maximum	0.8 – 84.0
Spring average	0.014 – 0.646
1-hr, Summer maximum	0.5 – 54.3
Summer average	0.008 – 0.837
1-hr, Fall maximum	0.9 – 98.2
Fall average	0.014 – 0.440

The AERMOD surface concentrations maps showed that the highest concentrations of $PM_{2.5}$ are seen closer to the stacks; however, some locations farther downwind also show increased $PM_{2.5}$. These locations farther downwind are located on hills that likely intercept the mill stack plume centerline that has the highest $PM_{2.5}$ concentration. The maximum surface $PM_{2.5}$ concentration estimated to be attributed to the mill is 0.837 µg/m^3, and was found during the summer. The maximum annual average was

found to be 0.451 µg/m^3. These concentrations are well below the $PM_{2.5}$ Canada Wide Standard for a 24-hour period of 28 µg/m^3. Therefore, the estimated $PM_{2.5}$ mass concentrations emissions from the mill, based upon reported emissions rates and stack characteristics, are of little impact to Pictou Landing First Nation. However, $PM_{2.5}$ health effects are not just related to the mass concentration but also the chemical, biological, and physical composition of the $PM_{2.5}$. Therefore, detailed information on the composition of the $PM_{2.5}$ modelled from the mill would also be needed to determine the potential health effects of the mill's $PM_{2.5}$ emissions on Pictou Landing residents. The AERMOD air dispersion modelling of $PM_{2.5}$ from the mill showed that there was some ground impact in Pictou Landing First Nation but at very low concentrations.

Despite not being able to obtain air quality data for the winters and falls of 2013 and 2014, a substantial data set was collected. All of the air quality data collected were below US National Ambient Air Quality Standards, the Canada Wide Standard, international standards, and/or occupational exposure standards where they existed. Some of the challenges encountered were inconsistent sampling times which made comparisons based on time problematic. There were also occasional Ogawa and TDT samples that were removed from the sites, and power losses at the active sites were not discovered until much later, leading to missed periods of time for data collection. Indoor air quality could also be a significant source of personal exposure, but the time and resource constraints within this project meant that these data were not collected.

7. Water Quality

Rob Jamieson (Associate Professor and Canada Research Chair, Department of Civil and Resource Engineering, Dalhousie University) led the water quality analysis component of the project. Staff from the Centre for Water Resources Studies, **Rick Scott** and **Justine Lywood**, also supported water sampling that was conducted with **Colleen Denny** and **Kim Strickland**. The findings reflect the water quality data that were collected in various locations around the community and from within Boat Harbour in the summer of 2013 (see Figure 28).

Water quality was sampled at eight locations. For each sample a range of tests were performed back in the lab. Water was tested for total **suspended solids**, **conductivity**, ***E. coli***, **nitrogen**, **phosphorus**, and a range of **metals**. The water was also tested for its **biological oxygen demand**, **dissolved oxygen**, and **pH**. **Dioxins and furans** were tested in two Boat Harbour water samples that were taken on July 24, 2013. Of the six sites, two are located in Boat Harbour (BH1 and BH2), one is located at the mouth of Boat Harbour (BHB), one is located in a brackish zone downstream of the mouth (BHOF), and two are reference sites: one on a spring that discharges into Boat Harbour (FF), and one on a surface water system adjacent to Boat Harbour (FP).

Figure 28. Water sampling sites. *Note:* The pink sites were chosen as reference sites, not affected by Boat Harbour effluent.

What's being sampled?

Suspended solids are all particles in the water column that will not pass through a filter. As levels of total suspended solids increase, a water body begins to lose its ability to support a diversity of aquatic life.

Conductivity was also tested; it is an indirect way to measure the total dissolved solids of the water. A high conductivity indicates that the water is most likely impacted by industrial discharge.

E. coli is a type of fecal coliform bacteria found in the intestines of humans and animals. If present, it points to recent sewage or animal waste contamination that is not necessarily harmful, but which indicates the possibility of other harmful pathogens in the water.

Nitrogen and **phosphorus** are often found to be the limiting factors of plant growth in aquatic systems. The more of these nutrients that are present, the more plant growth can arise which means less oxygen in the water and a more stressful environment for aquatic life.

Metals that were tested for in the water samples included aluminum, silver, arsenic, cadmium, copper, iron, nickel, lead, selenium, uranium, and zinc, with different levels of toxicity. Some, like arsenic, are naturally occurring in Nova Scotia.

Biological oxygen demand is the amount of oxygen consumed by microorganisms in decomposing organic matter in a period of five days. If more oxygen is consumed than is produced, the amount of **dissolved oxygen** declines, which can negatively affect aquatic life.

The **pH** shows if the water is basic or acidic. Changes in pH can affect how chemicals dissolve in the water and whether organisms will be affected by them at all.

Dioxins (polychlorinated dibenzodioxins) and **furans** (polychlorinated dibenzofurans) are highly persistent compounds with a strong affinity for sediments and a high potential for accumulating in biological tissues. Dioxins and furans enter the environment mainly through waste incineration and **pulp and paper processing**, and have been found in very small amounts in all parts of the environment including air, water, soil, sediments, animals, and foods. All animals and humans in Canada are exposed to some level of these substances. Large exposures can lead to a variety of serious health problems (Health Canada, 2006).

Results

In general, the Canadian Water Quality Guidelines for the Protection of Aquatic Life (CCME, 2007b) **were exceeded by water samples within Boat Harbour** (BH1, BH2, and BHB – marked green in Figure 28) and directly leaving Boat Harbour (BHOF). For the surrounding sample sites, FF and FP, water quality guidelines were met, except in the case of some heavy metals (silver – both sites; zinc – FF only; iron and lead – FP only).

- The amount of total **suspended solids** in Boat Harbour and directly leaving Boat Harbour were above available guidelines for aquatic health, but still within typical guidelines for wastewater effluent.
- **Conductivity** levels in samples from within Boat Harbour and the BHOF sites indicate waters are most likely impacted by industrial discharge.
- ***E. coli*** levels at all sites were within guidelines considered acceptable for recreational use. On two occasions, *E. coli* levels were beyond what is considered safe for irrigation onto food crops (BHB on July 12, FP on July 26).
- The amount of **nitrogen** in water samples was below the water quality guideline for the protection of aquatic life.
- **Phosphorus** national guidelines for the protection of aquatic life do not exist, but there are trigger ranges delineated by the CCME that characterize ecosystems with different phosphorus concentrations. Within Boat Harbour, the concentrations exceed the hyper-eutrophic trigger range. Hyper-eutrophic aquatic systems are characterized by excessive plant and algae growth, poor water clarity, and low dissolved oxygen levels, making it difficult for aquatic life to survive.
- Of the metals tested, **aluminum, iron, lead, silver,** and **zinc** were all above CCME recommended levels for protection of aquatic life.
- **Biological oxygen demand** from within Boat Harbour and BHOF (Boat Harbour Outfall) were generally 5-12 mg/L, which is a typical concentration for effluent leaving a sewage treatment plant, but indicates that Boat Harbour is influenced by organic wastewater discharges.
- **pH** levels were within the accepted range for aquatic life.
- Both water samples tested for **dioxins and furans** were below available guidelines for drinking water quality (10 TEQ). There are no guidelines for the protection of aquatic life.

In summary, the water quality analysis demonstrated that the discharge of pulp and paper mill effluent to Boat Harbour has degraded the quality of water within Boat Harbour, with concentrations of several water quality parameters exceeding available guidelines for the protection of aquatic life. The water quality measured in the reference sites was much better, but a small number of samples still had concentrations of metals that exceeded water quality guidelines for the protection of aquatic life. A number of different industrial sources in the region could be contributing to these background levels of metals found within the reference sites.

8. Soil Quality

In addition to water quality, **Rob Jamieson** led the soil sampling component of the project. Representatives from the Centre for Water Resources Studies, Dalhousie University, collected soil samples from current or potential future sites of vegetable gardens at 13 residential properties around Pictou Landing First Nation (Samples #1-12 and #14) and one reference location near the shore of Boat Harbour (Sample #13) on October 3, 2014. The soil was analyzed for various **metals** as well as **dioxins and furans**, and the results were compared to national guidelines for acceptable concentrations of dioxins, furans, and metals in soil. Metals that were tested for included aluminum, antimony, arsenic, barium, beryllium, bismuth, boron, cadmium, chromium, cobalt, copper, iron, lead, lithium, manganese, mercury, molybdenum, nickel, rubidium, selenium, silver, strontium, thallium, tin, uranium, vanadium, and zinc.

On June 25, 2015, additional soil sampling was completed to confirm the 2014 sampling results. Soil samples were collected from two residential properties (Samples #15 and #16), one recreational property in Pictou Landing First Nation (Sample #17), and three reference locations in the town of Pictou (Samples #18-20). The soil samples were again tested for dioxins, furans, and metals.

Three soil subsamples were taken at each property and were then combined to obtain a composite sample. The sampling included the upper layer of the soil (to a depth of 0.2 m) so the results represent only these depths. The sample locations are shown in Figure 29.

Results

The federal guidelines from the Canadian Council of Ministers of the Environment (CCME, 2014) were used to compare the soil sample concentration results to the recommended Canadian guidelines for the protection of human and environmental health.

Metals

For three samples, metal concentrations in the soil exceeded the federal guidelines. Two instances of **arsenic** concentrations above the guideline of 12 mg/kg were found, in Sample #1 (41 mg/kg) and Sample #16 (13 mg/kg). Arsenic is common in the natural geology of many regions in Nova Scotia and therefore the observed concentration may be of natural origin. However, as the levels do exceed the CCME guideline, it would still be advised to avoid using the soil on these two properties for the establishment of vegetable gardens, and to minimize disturbance and contact with the soil.

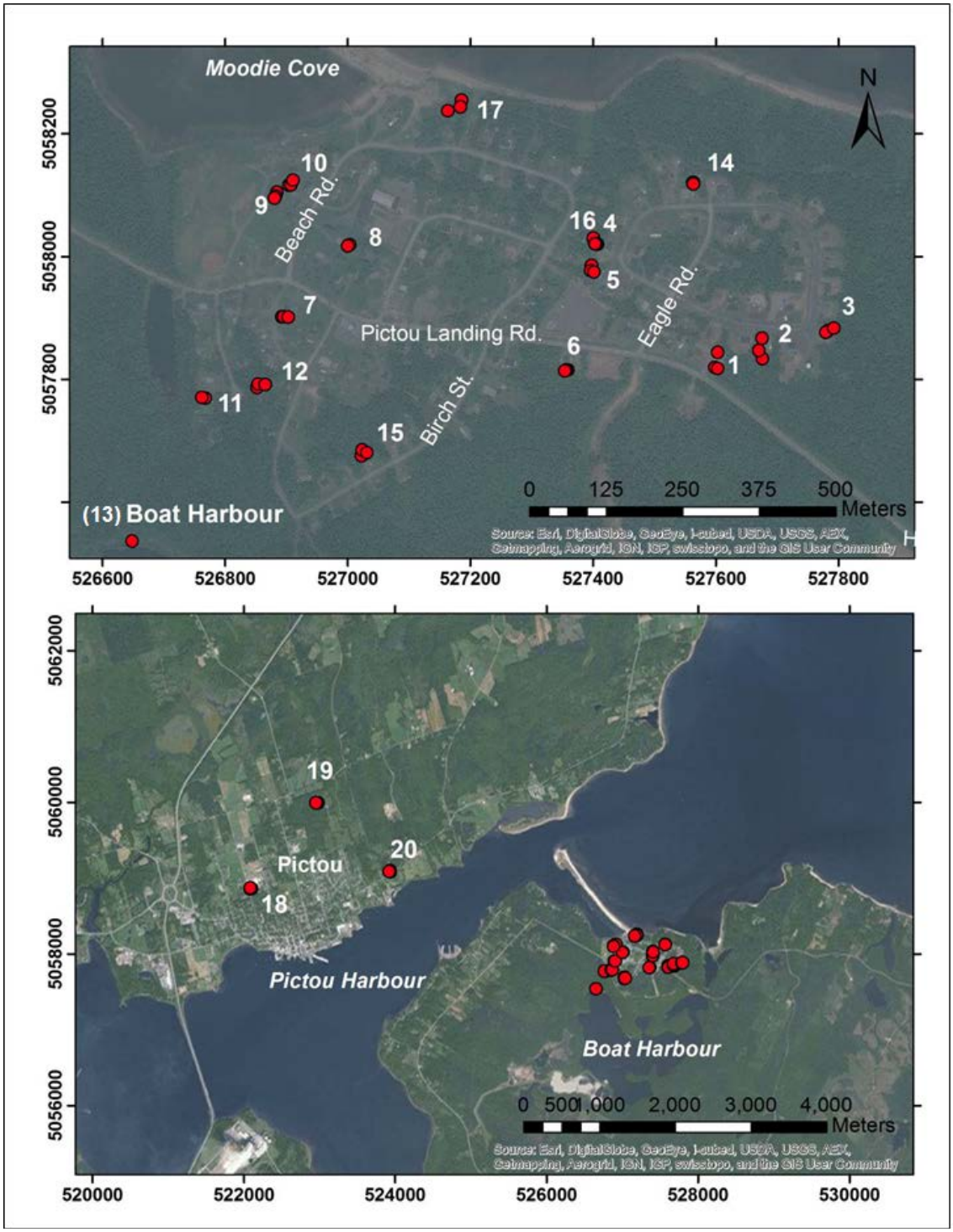


Figure 29. Plan view map of the 2014 and 2015 soil sampling locations in (upper map) Pictou Landing First Nation and (lower map) the town of Pictou.

In addition, **copper** exceeded the guideline level at one site, Sample #4. The copper concentration at that site was 330 mg/kg, well above the CCME environmental quality guideline of 63 mg/kg, but not above the CCME level for human health effects (1100 mg/kg). The source of the copper in Sample #4 is unknown; however, that sample was taken directly from an existing vegetable garden. If fertilizer was added to this garden, there might have been introduced copper from copper sulfate, which is present in some common soil amendments. Site #4 was resampled during the second round of sampling, and the copper concentration was then below the CCME guidelines.

Dioxins and Furans

The results for **dioxins and furans** were compared to the Canadian Soil Quality Guidelines (CCME, 2002). The guideline value is based on background concentrations observed in the environment and not on observed or predicted adverse human health effects. Therefore, any concentrations observed above the dioxin and furan guideline level may not necessarily cause negative human health effects.

In the first round of sampling, two soil samples had slight exceedances of the CCME guideline value for dioxins and furans. The exceedances were observed in Samples #4 and #6, where concentrations were 8.77 and 5.28 TEQ (ng kg^{-1}), respectively. The CCME guideline is 4 ng kg^{-1}. The source of dioxins and furans are unknown in these two samples. The property where Sample #6 was collected had imported fill in the backyard at the sample collection points. It is possible that the slightly elevated concentrations of dioxins and furans may be due to a previous use of the imported fill. Sample #4 (which also had elevated levels of copper in the first sampling round) showed elevated dioxins and furans as well. According to Rob, while the dioxin and furan concentration was above the CCME guidelines, it is typical of the range of soil concentrations found throughout Canada. In personal communication Rob stated: "There are small amounts of these chemicals in soils everywhere due to atmospheric deposition." Site #4 was resampled during the second round of sampling and concentrations of all contaminants, including dioxins and furans, were below the CCME guidelines.

In summary, the soil results indicate **the soil is generally safe** for vegetable harvest intended for human consumption and direct contact in terms of metals and dioxin and furan concentrations. Contaminant concentrations in samples obtained from Pictou Landing First Nation and the background samples from the town of Pictou were similar.

9. Ecotoxicology and Sediment Analysis

Ron Russell (Associate Professor, Department of Biology, Saint Mary's University) led the ecotoxicology analysis, which sought to investigate how living things are affected by Boat Harbour. His objective was to collect and analyze Boat Harbour sediments, plankton, and mammals for a variety of potentially toxic chemicals commonly observed in environmental samples. He also attempted to estimate the toxicity of Boat Harbour water to a variety of common vertebrates and describe the structure of the aquatic ecosystem.

In the summer of 2013, Ron, Community Research Associates **Kim Strickland** and **Colleen Denny,** and Research Assistant **Geoff Kershaw** collected sediment and plankton samples from three locations: (1) near the aeration pond outfall, (2) near the dam, and (3) midway between the previous two locations. They collected three sediment samples at each location; thus, in total they collected nine soil/sediment samples, which underwent chemical analysis to test for **polychlorinated biphenyls (PCBs), historic use pesticides, chlorophenols, brominated diphenyl ethers,** and **polychlorinated dibenzodioxins and furans**. Samples were prepared and analyzed by Research Productivity Consultants (RPC) of Fredericton, New Brunswick. Plankton sampling was intended to provide qualitative data only, and four samples were taken from within Boat Harbour.

Ron also collected about 200 L of water to use in toxicity tests. Ron exposed wood frog tadpoles, green frog tadpoles, recent metamorphs, and adult and young-of-year mummichogs (small killifish) to Boat Harbour water of varying dilutions for 7 days (acute toxicity tests) in Ron's lab. Adult frogs were exposed to 100% and 75% (diluted) Boat Harbour water for over 96 hours.

Additionally, a resident of Pictou Landing, **Durney Nicholas**, trapped a total of 12 muskrats and four beavers around Boat Harbour for tissue sample analysis between November 2014 and January 2015. Tissue samples were taken from these animals and compared to sediments from the bottom of Boat Harbour. Samples were once again prepared and analyzed by RPC.

Guidelines

Federal guidelines from the CCME and Environment Canada were used to compare the pollutant concentrations in sediments with the recommended Canadian guidelines for the protection of aquatic life. Canadian soil guidelines for the protection of human and environmental health and tissue guidelines for the protection of consumers of aquatic wildlife were also used where appropriate (CCME, 2014). Results for total PCBs were compared to sediment guidelines for the protection of aquatic life and soil guidelines for the protection of environmental and human health. Sediment guidelines for chlorophenols are not available. CCME sediment quality guidelines for dioxins and furans for the protection of aquatic life were compared against sediment sample results. There are no consumption guidelines for dioxins in human food in Canada. The European Union has established maximum levels of dioxin and related compounds in the European human food chain. The European Union does not state maximums for wild-caught terrestrial food, but has developed guidelines for a number of domesticated livestock species (EU, 2011). This report distinguishes between muscle and liver dioxin concentrations and considers the toxic contribution of dioxin-like PCBs to the overall toxicity (TEQ) of the meat.

Results

Plankton Community Structure

Boat Harbour exhibits a distinct truncated community profile for plankton where the rotifers are the largest planktonic organisms present in all samples. Common groups such as copepods, cladocerans, ostracods, and insects were absent from all samples. Equally alarming was the low biodiversity observed in these samples. High densities and biomasses of known pollution-tolerant species were observed in the Boat Harbour samples.

Toxicity Tests

Mummichogs are known to be highly tolerant of fluctuating salinities, temperatures, hypoxia, and pollution (Eisler, 1986). For these reasons, they are considered to be the ideal candidate for acute and chronic toxicity experiments. Both adult and young-of-year mummichogs exhibited 100% mortality in the full concentration Boat Harbour water by 168 hr (Figure 30). The young fish reached 100% mortality at 120 hr while the adults endured somewhat longer to 168 hr with the undiluted Boat Harbour water. The adult mummichogs reached about 60% mortality in the 75% dilution water, and about 35% mortality in the 50% dilution water, after 168 hr. Adult mummichog mortality in the 25% dilution water did not differ from the control which contained no Boat Harbour water.

Young mummichogs showed greater mortality earlier in the experiment than adults (Figure 30). Young mummichogs reached 100% mortality in the 75% Boat Harbour water dilution and about 30% mortality in the 25% dilution after 168 hr. It is clear that young fish are less able to persist in Boat Harbour water than adults.

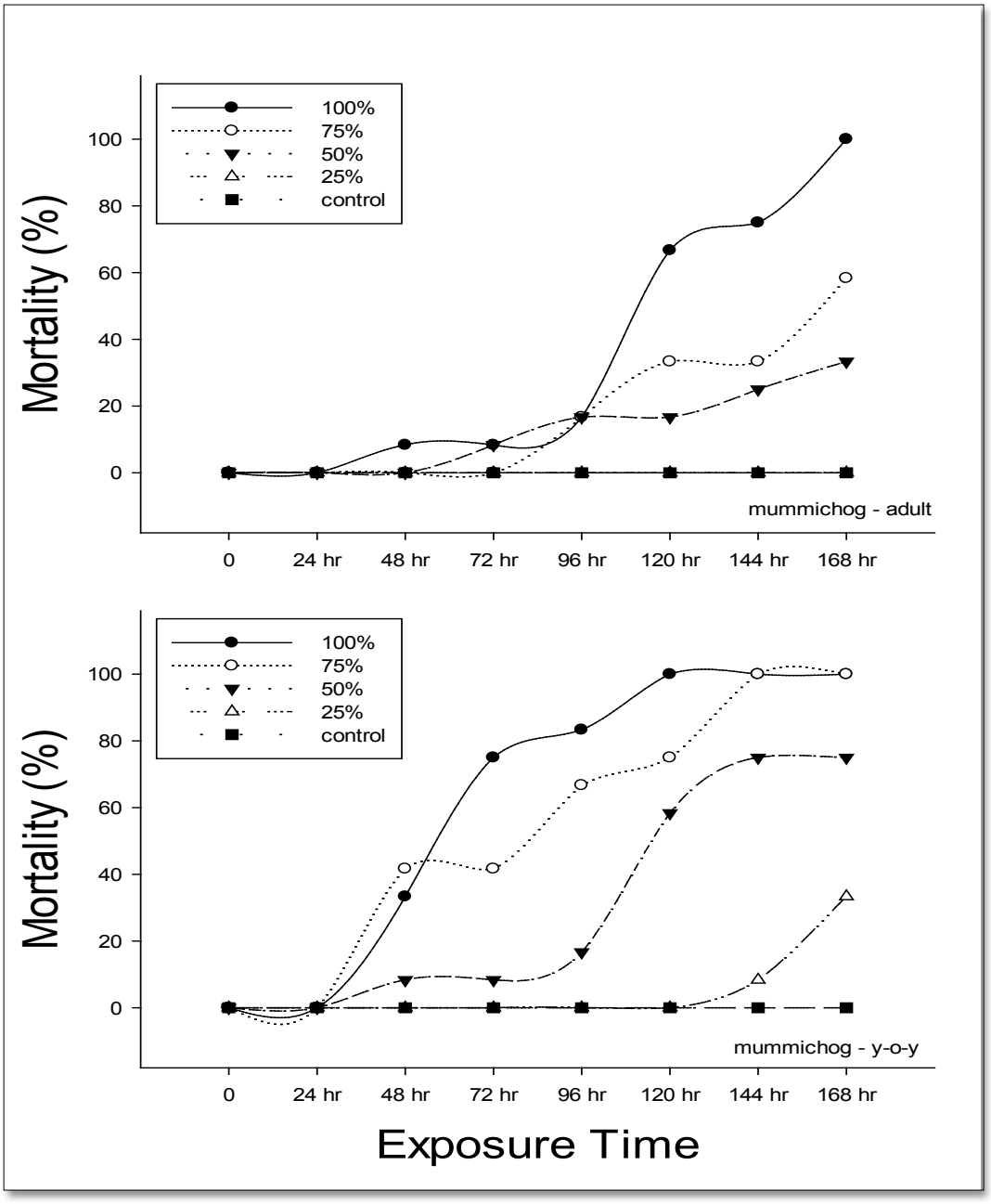


Figure 30. Acute toxicity of Boat Harbour water (undiluted and diluted to 75%, 50%, and 25%) to adult and young-of-year mummichogs.

Wood frogs have a near cosmopolitan distribution in Canada. They are a forest-dwelling species and reproduce in fishless ponds, so they are expected to be common around Boat Harbour; however, none were observed during the course of this study. Wood frog tadpoles reached 100% mortality in all dilutions of Boat Harbour water and reached 100% mortality in the full-concentration Boat Harbour water in less than 48 hr (Figure 31).

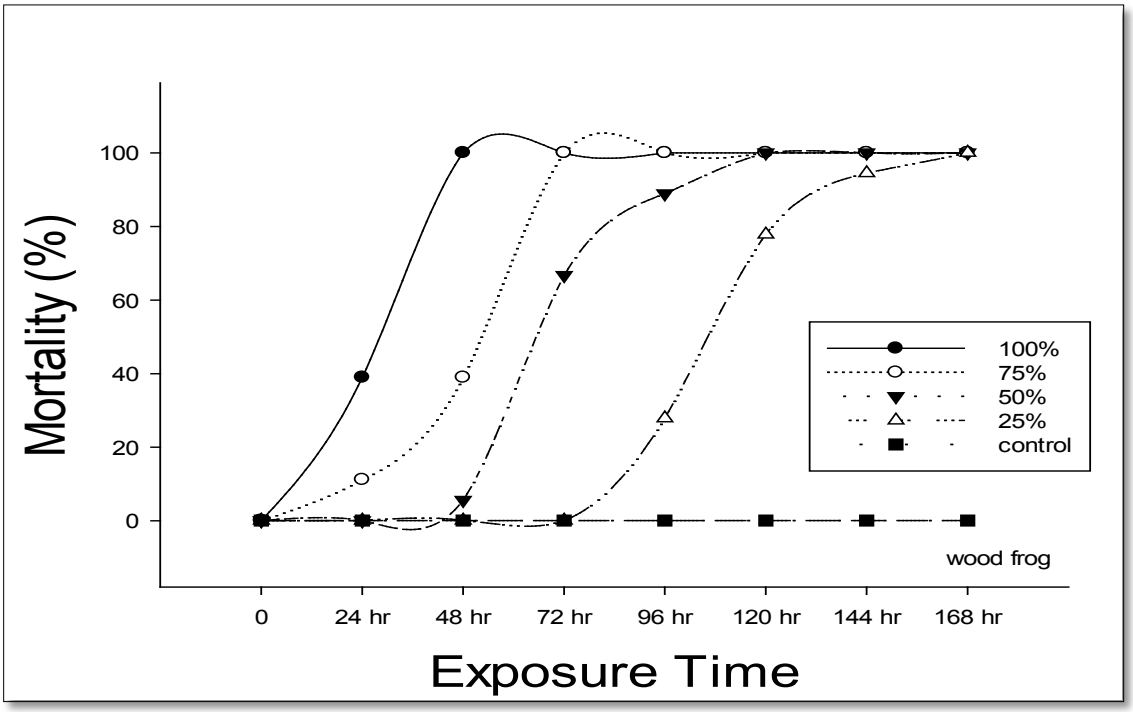


Figure 31. Acute toxicity of Boat Harbour water (undiluted and diluted to 75%, 50%, and 25%) to wood frog tadpoles.

Green frogs are a common and widely distributed species in Nova Scotia. They are tolerant of pollution and a pioneer species so are expected to be present in and around Boat Harbour. Adult green frogs were observed around the lower periphery of Boat Harbour. Stage 25 green frog tadpoles are the first feeding stage in these organisms. Tadpoles at this stage reached 100% mortality in the 100%, 75%, and 50% dilutions in less than 168 hr (Figure 32). The 25% dilution caused about 50% mortality in the same time frame. Stage 30 tadpoles are significantly larger and more fully developed than stage 25 green frog tadpoles. The stage 30 green frog tadpoles were clearly better able to survive, with 100% mortality observed in only the full concentration of Boat Harbour water (Figure 32).

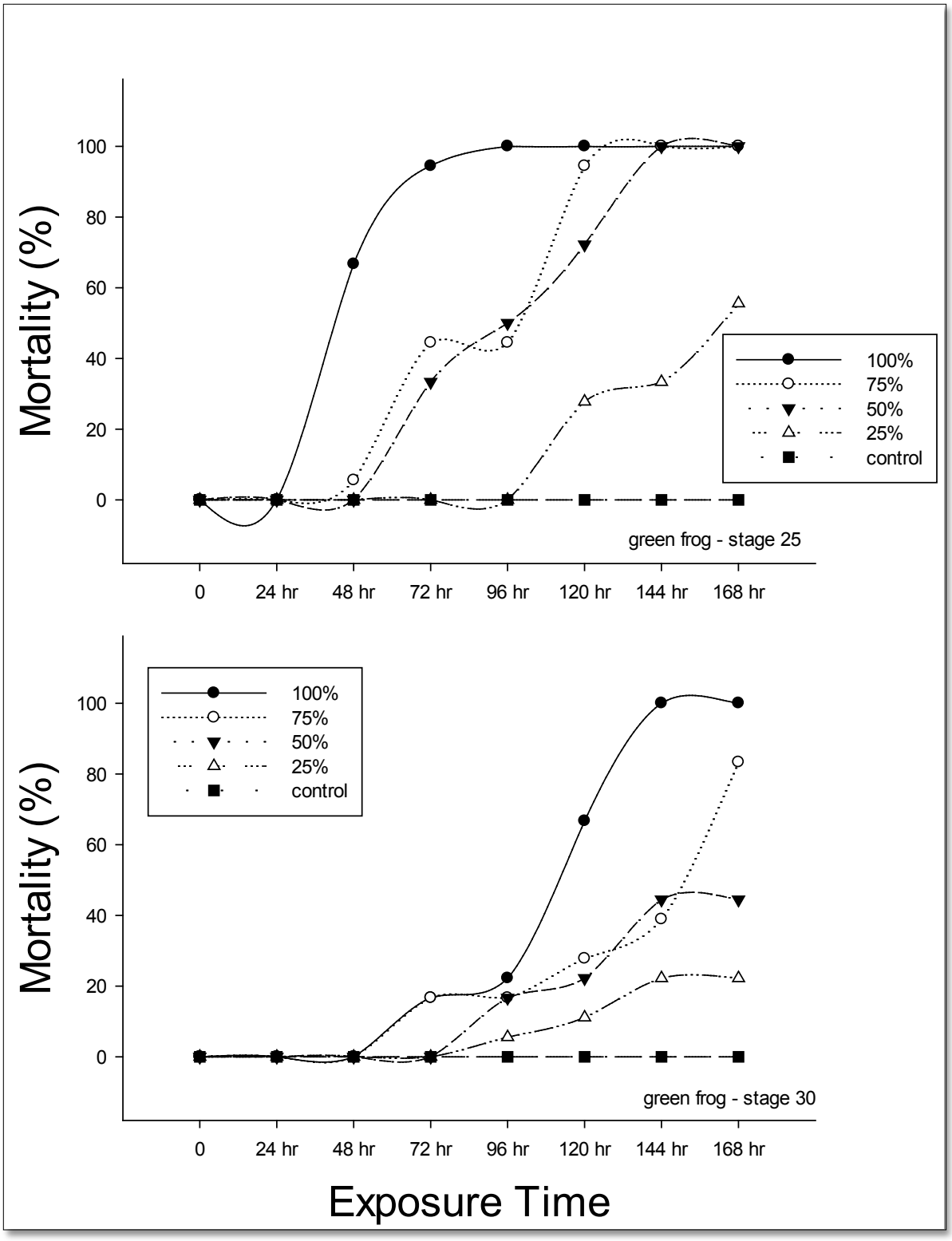


Figure 32. Acute toxicity of Boat Harbour water (undiluted and diluted to 75%, 50%, and 25%) to stage 25 and stage 30 green frog tadpoles.

Bullfrogs are the largest and potentially most tolerant of the amphibians to pollution (Weis, 1975). They are commonly found in rivers and large lakes are thus expected to be present in Boat Harbour. Bullfrogs were never observed in Boat Harbour over the course of this study. Bullfrog tadpoles showed a similar pattern of mortality as green frog tadpoles (Figure 33), with 100% mortality occurring in both the full concentration and 75% concentration treatments.

These data indicate that the three amphibian species probably cannot reproduce in Boat Harbour due to acute toxicity to early life stages.

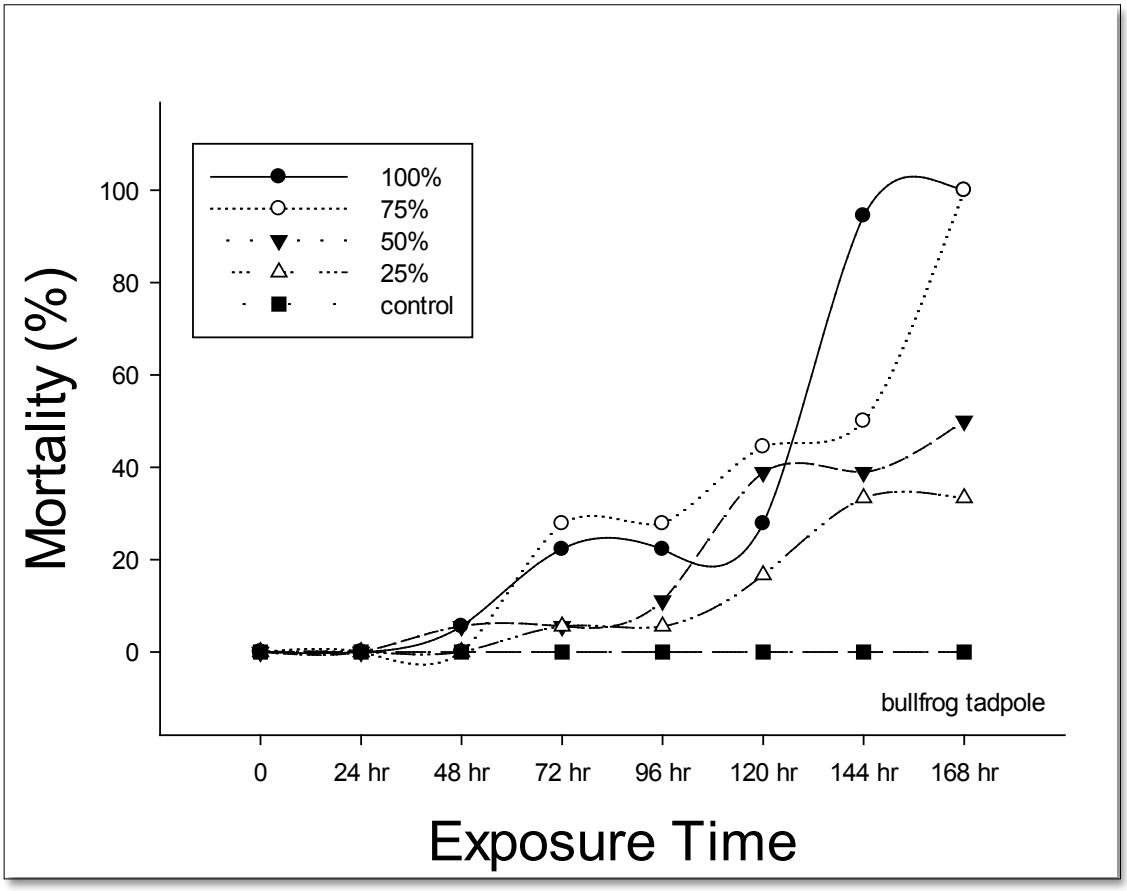


Figure 33. Acute toxicity of Boat Harbour water (undiluted and diluted to 75%, 50%, and 25%) to bullfrog tadpoles.

PCBs in Sediments and Plankton

All PCB concentrations measured in plankton and sediments were below CCME guidelines and are thus of little concern. PCB concentrations in Boat Harbour sediments are best described as background and do not indicate a point source in the area. Dioxin-like PCBs were routinely detected in both sediment and plankton; however, their

contribution to the TEQ was very low, typically less than 1 ng TEQ/kg. This indicates that observed toxic effects were most likely not due to PCB accumulation.

Pesticides in Sediments

All pesticide concentrations in Boat Harbour sediment were below CCME guidelines and below the detection limit of the analytical method of 0.05 mg/kg. This most likely indicates low historic use of pesticides in the Boat Harbour area. Toxicity arising from the past application of pesticides is of low concern.

Chlorophenols in Sediments

Chlorophenol concentrations were low. There are no sediment guidelines for phenols; however, all concentrations measured for phenols in sediments were well below the soil quality guideline of 3.8 mg/kg for phenol and 7.6 mg/kg for pentachlorophenol. Phenols are highly toxic, bleached kraft mill waste products, but there is little accumulation of these compounds in Boat Harbour sediments.

Polybrominated Diphenyl Ethers (PBDEs)

PBDEs were observed in all plankton and sediments samples. All concentrations were below federal sediment quality guidelines (Environment Canada, 2013). Relatively low concentrations of PBDEs were observed in sediments with no apparent pattern among sediment sampling locations. PBDE concentrations were lower yet in plankton and lower again in Boat Harbour mammals. There is no indication of magnification of PBDEs in the Boat Harbour ecosystem.

Polychlorinated Dibenzodioxins and Furans (PCDDs and PCDFs)

The distribution of dioxins and furans in sediments show that concentrations were greatest at the midway location and least near the aeration pond outfall. There is no simple explanation for this distribution. It could be due to historic changes in PCDD and PCDF concentrations in effluent, spatially variable sediment deposition, or the bottom profile of the sampled locations.

The most toxic sediments were found at the midway location, and the least toxic sediments were at the aeration pond outfall. The only conclusion possible is that PCDDs and PCDFs are found in Boat Harbour sediments, but they are not evenly distributed. PCDD and PCDF TEQs exceed both CCME interim sediment quality guidelines and probable effect level guidelines (CCME, 2001). Toxic effects on aquatic life are expected based on the calculated TEQs, and it is clear that the aquatic community is severely degraded.

Mean plankton TEQ for dioxins, dibenzofurans, and dioxin-like PCBs was 0.20 pg TEQ/g wet weight (SD = 0.03 ng TEQ/kg; Table 9), which is less than the CCME guideline. Clearly, PCDDs and PCDFs are accumulating in the Boat Harbour planktonic food web;

however, the severely degraded aquatic community with truncated structure may present few opportunities for these highly bioaccumulative compounds to be magnified with passage through the food web.

Table 9
Dioxins (Including Furans) and Dioxin-like PCB TEQs (ng WHO-TEQ/kg wet weight) in Boat Harbour Plankton

Sample	Dioxins	Dioxin-like PCBs	Dioxins + Dioxin-like PCBs
Plankton #1	0.18	0.02	0.20
Plankton #2	0.24	0.01	0.25
Plankton #3	0.18	0.02	0.20
Mean	0.20	0.02	0.22
Std. Dev.	0.03	0.01	0.03

Table 10 outlines wet weight TEQs for dioxins (including furans) and dioxin-like PCBs for Boat Harbour beaver and muskrat muscle and liver tissues. Dioxin TEQs in beaver muscle were slightly less than dioxin TEQs in plankton. Dioxin TEQs for beaver liver and muskrat muscle and liver were greater than TEQs in plankton; however, standard deviations were large in beaver liver and muskrat tissues, obscuring any real differences between these tissues and plankton. There is no clear evidence for magnification of dioxins and furans in beavers and muskrats.

Table 10
Dioxins (Including Furans) and Dioxin-like PCB TEQs (ng WHO-TEQ/kg wet weight) in Beaver and Muskrat Muscle and Liver

	Dioxins		Dioxin-like PCBs		Dioxins + Dioxin-like PCBs	
Sample	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Beaver muscle	0.11	0.05	0.02	0.01	0.13	0.05
Beaver liver	0.60	0.22	0.07	0.05	0.68	0.27
Muskrat muscle	0.47	0.27	0.01	0.01	0.48	0.27
Muskrat liver	0.63	0.27	0.02	0.01	0.66	0.28

The European Union has specified maximum levels of dioxin and dioxin-like chemicals allowable in human food, but currently no similar guideline exists in Canada. TEQs for dioxins and dioxin-like PCBs are expressed on a per unit lipid basis since these are highly lipid-soluble compounds and are expected to be found predominantly in fatty tissues. Lipid-based TEQs for beaver and muskrat tissues exceed European Union guidelines for all meat except the guideline for beef and mutton (Table 11). Considering variability in the data set, all beaver and muskrat tissue TEQs are within one standard

deviation of the EU guideline. One must use this guideline with caution, since wild-caught foods such as beaver and muskrat do not appear on any guideline, presumably since they are traditionally not found in commercial food preparation and retail marketing.

Table 11
Dioxins (Including Furans) and Dioxin-like PCB TEQs (ng WHO-TEQ/kg lipid weight) in Beaver and Muskrat Muscle and Liver

	Dioxins		Dioxin-like PCBs		Dioxins + Dioxin-like PCBs	
Sample	**Mean**	**Std. Dev.**	**Mean**	**Std. Dev.**	**Mean**	**Std. Dev.**
Beaver muscle	2.43	0.83	0.47	0.13	2.90	0.94
Beaver liver	13.92	4.31	1.58	0.60	15.50	4.66
Muskrat muscle	8.21	0.79	0.31	0.19	8.53	0.78
Muskrat liver	15.41	6.83	0.59	0.10	16.00	6.92

It is clear that the Boat Harbour aquatic ecosystem is severely degraded, most likely due to a combination of hyper-eutrophication (resulting in low oxygen), high water temperature, decreased sunlight penetration due to heavy coloration, and toxic chemical input. This is not a new observation. Peer (1972) documented significant changes in the marine benthic community downstream of Boat Harbour a mere two years following the opening of the kraft pulp mill at Abercrombie Point. Proposed causes included changes in tidal flow caused by damming the previously tidal estuary and the Middle and West Rivers (Peer, 1972) and promotion of toxicity of pulp mill effluents due to flocculation of material upon entry to the marine environment.

Boat Harbour water is toxic to both fish and amphibians in the lab. None of the experimental groups survived 168 hr (7 days) in undiluted Boat Harbour water; however, adult mummichogs, stage 30 green frog tadpoles, and bullfrog tadpoles all survived over 7 days exposure in dilute Boat Harbour water. While Boat Harbour water may not exhibit acute toxicity over the traditional 96 hr exposure period to adults and more highly developed stages of organisms, it is acutely toxic at early developmental stages as demonstrated by calculated median lethal concentrations. This would prohibit any of the organisms tested from establishing viable populations in Boat Harbour under current environmental conditions. It must be considered that these toxicity tests conducted under laboratory conditions minimized the negative effects of high water temperature and hypoxia that were present in Boat Harbour. The toxicity tests therefore slightly *underestimate* the real conditions in Boat Harbour at the water collection period.

PCBs, persistent pesticides, and chlorophenols were detected at low concentrations or below detection limits in sediment and plankton. All of these compounds are capable of eliciting a wide range of toxic responses in aquatic organisms and humans and thus could contribute to the overall toxicity observed in Boat Harbour. The small concentrations in sediments indicate that these compounds do not pose a significant problem in Boat Harbour, presently or in the future. Sediments can act as a repository for many persistent organic compounds, releasing them into the aquatic environment for many generations (Russell, Hecnar, & Haffner, 1995). Since PCBs and most of the pesticides present in the analysis have been banned in Canada for many years, it is highly unlikely that they will increase in concentration in sediments in the future. Chlorophenols are mostly water-soluble chemicals and do not accumulate to a great degree in sediments.

Polybrominated diphenyl ethers (PBDEs) are structurally similar to PCBs, and like PCBs, they exhibit nervous system toxicity, reproductive and developmental disruption, endocrine disruption, and cancer at high doses (Birnbaum & Staskal, 2004). They are highly persistent in the environment, subject to long-range transport in the atmosphere, and highly bioaccumulative. These compounds were detected in all sediments, plankton, and mammal tissues. Sediment concentrations were below published guidelines and PBDE concentrations in plankton and mammal tissues were low. There was no indication of magnification through food web processes, possibly since the Boat Harbour food web is degraded and there are few opportunities for biomagnification. PBDEs in the environment is a global issue, but these compounds probably do not make a significant contribution to the toxicology concerns at Boat Harbour due to their low concentrations. Due to the persistent nature of highly brominated PBDEs, they will be retained in the sediments for many generations.

Dioxins and dioxin-like compounds were detected in all samples. The TEQ for dioxins was above the interim sediment quality guideline and probable effects level for all sampling locations. Dioxins and dioxin-like compounds are persistent chemicals and are expected to accumulate in sediments and in lipids (fat) of biota. The concentrations of PCDDs and PCDFs measured in Boat Harbour sediments raise concerns.

Dioxin concentrations in plankton were considerably less than concentrations measured in sediments. Boat Harbour plankton are most likely contaminated by the freely dissolved portion of dioxins in the water column and by contaminated suspended particles. Food web effects are not apparent since the Boat Harbour food web is very short. Tertiary (third-level) consumers and successive levels of primary and secondary consumers and producers are absent. There are few opportunities for biological magnification of dioxins by passage through the food web structure, unlike less disturbed ecosystems (Braune & Simon, 2003; Ross et al., 2004), where tertiary predators can accumulate high concentrations of dioxins.

Concentrations of dioxins and dioxin-like compounds in beaver and muskrat tissues were low, but detectable. Liver exhibited consistently higher concentrations than muscle, since metabolic detoxification pathways are predominantly found in the liver. Dioxin TEQs in mammal tissues were approximately the same as plankton and significantly lower than TEQs calculated for sediment.

There are no guidelines for consumption of dioxins and furans in Canadian foods. The CCME soil quality guideline for soil ingestion is 4 ng TEQ/kg (4 pg TEQ/g; CCME, 2002, 2007a) for the protection of environmental and human health. It is recognized that dioxins and dioxin-like compounds are accumulated primarily through the diet (Fries, 1995). Greater than 90% of human exposure to dioxins is through diet, mainly meat, dairy products, fish, and shellfish (WHO, 2014). Health Canada recommends a "tolerable" level of dioxin consumption from all sources of 2.3 pg/kg of body weight/day and 70 pg/kg of body weight/month (Health Canada, 2005). Beaver and muskrat are not part of the commercial food industry; however, dioxins in both muscle and liver of these mammals exceed European Union guidelines for most commercial meats.

Recommendations

There are multiple serious problems with the ecotoxicology of Boat Harbour. Remediation of any single issue could exacerbate other problems, so a remediation plan should consider all problems. The primary organic pollutants of concern in sediments are dioxins and furans. Contact with Boat Harbour sediments should be avoided. The movement of these pollutants off site should be evaluated, particularly potential contamination of downstream marine habitats. Additionally, biological vectors of dioxins and furans moving these pollutants from the aquatic environment to the terrestrial environment should be assessed. Although concentrations of dioxins and furans in tested mammals were low, consumption of beaver and muskrat tissues, particularly liver, could still result in exceeding recommended consumption limits.

10. Tree Core Sampling

In the summer of 2013, **Geoff Kershaw, Colleen Denny,** and **Kim Strickland** cored tree rings for testing and analysis at the Mount Allison Dendrochronology Laboratory (MAD Lab, now the Mistik Askiwin Dendrochronology Laboratory at the University of Saskatchewan).

Dendrochronology is the analysis of tree rings formed in wood as a means of measuring changes in environmental factors that influence a tree's growth (Speer, 2010). Tree core sampling was done because tree rings have been used successfully to document the onset and intensity of chlorinated hydrocarbon pollution elsewhere (Yanosky, Hansen, & Schening, 2001). As well, sulfur compounds are released to the atmosphere in the kraft bleaching process (Ali & Sreekrishnan, 2001), which may cause acid rain and soil acidification issues that negatively affect forest wellbeing (Fox, Kincaid, Nash, Young, & Fritts, 1986; Savva & Berninger, 2010).

Twenty trees were sampled from an old-age (ca. 95 years) white spruce stand by Boat Harbour, with two cores taken at breast height from each tree (total of 40 cores). For comparison, 36 cores from 18 trees were sampled from a comparably aged (ca. 97 years) white spruce stand at the Caribou Control site (see Figure 34). Caribou Control was selected because it had characteristics considered

similar to what Boat Harbour would have been like before the dam and aeration ponds were installed. It was hypothesized that the sites would have similar but distinct histories, as proven with different statistical tests on the changes of yearly growth, and it was also hypothesized that stronger growth suppression would be obvious at the Boat Harbour site.

Figure 34. Tree sampling sites (Boat Harbour and Caribou Control).

Results

While the analysis showed statistical differences suggesting unique growth-influencing factor(s) at each site, it is unclear if these differences are associated with pulp mill activity. For example, causes could include insect outbreaks (Hogg, Brandt, & Kochtubajda, 2005), flooding (Speer, 2010),

coastal ice storms (Lafon & Speer, 2002), or other factors. Also, a growth suppression trend in Boat Harbour tree growth was not observed; instead, the Boat Harbour trees have a tendency to exhibit growth enhancement trends for both raw and standardized chronologies, while Caribou Control does not.

This is contrary to the hypothesis that pollution impacts will result in negative growth effects. As such, there is not sufficient evidence in this analysis to support the hypothesis that pulp mill activity has impacted growth of trees surrounding Boat Harbour. A more thorough assessment would require long-term emissions data from the mill and chemical analysis of tree tissues.

It could be that Caribou Control was not an effective reference and that 10 km was not a large enough buffer between sites (this meshes with **Mark Gibson's** interpretation of the air quality data once his analysis was completed as well). The best potential for establishing greater certainty in tree-ring associations with Northern Pulp pollution would be to expand the number of sites involved in the analysis. Data tracking pulp mill emissions via the main mill stacks and Boat Harbour settling ponds would also enhance our ability to identify pollution's association with tree growth. Dendrochemistry is another potential route for future research because trees internalize pollutants and retain them within their cells as they grow. Using additional sites with a focus on trying to determine any effects of wind direction from the mill site is also recommended.

11. Analysis of the June 2014 Spill

On June 10, 2014, a member of Pictou Landing First Nation discovered a leak at a pipe that was carrying effluent from the Northern Pulp mill to Boat Harbour. The following day, **Ron Russell,** a lab assistant of Ron's, and **Emily Skinner** travelled to Pictou Landing to collect water and plankton samples from the effluent spill site and Boat Harbour dam/bridge. They collected effluent water from the environment and not directly from the pipe. **Kim Strickland** and Emily also collected samples of the effluent from the spill to send to the Centre of Water Resources Studies for testing of biological oxygen demand (BOD), chemical oxygen demand (COD), ammonia-nitrogen, and bacteria (*E. coli*).

The water quality analysis did not detect *E. coli*. Values for BOD, COD, and ammonia-nitrogen were typical for pulp mill effluent. It is suspected that the effluent had probably become diluted before the samples were collected, which took place 24 hours after the discovery of the spill.

The metal concentrations in the effluent were compared to available CCME guidelines for the protection of aquatic life (freshwater). Most metals (aluminum, cadmium, copper, nickel, lead, selenium, silver, zinc) with associated guidelines exceeded said guidelines, with **copper and lead** as the worst cases – exceeding the guidelines by greater than 10 times. The water samples that Ron collected from the pipeline burst had octochloride dioxide (OCDD) in it, but this was likely from atmospheric fallout and was also present in the soil sample. OCDD concentrations detected were high (1350 pg/L). OCDD is the most persistent but least toxic of the influential dioxins. Furthermore, elevated concentrations, as in this sample, indicate an "old" source or environmental sink. The elevated OCDD could also be attributed to atmospheric deposition from a combustion source over time. As noted in the water quality section, there are no CCME water quality guidelines for dioxins at this time.

Samples were collected on June 17, 2014, for **Mark Gibson**'s lab, at least one week after the effluent leak occurred. At the time of sampling, the leak site had been excavated and the effluent had been removed and pumped to a natural pond adjacent to Boat Harbour. It is unknown how much time had passed between the effluent being removed and the sampling. Water was collected at three different locations that were exposed to the effluent. The first was from the small pool at the bottom of the excavated hole where the leak had originated (see Figure 35). The second was collected from the exposed effluent-carrying pipe (see right on Figure 35), and the third was collected from the pond where the effluent had been transferred. The water samples were tested for volatile organic compounds (VOCs). Twenty-four VOCs were detected between the three samples. When the results were compared to the Canadian Council of Ministers of the Environment regulations for freshwater (CCME,

2014), none of the VOCs detected exceeded the regulatory limits, though not all VOCs have associated guidelines.

Figure 35. Collection site for two of the effluent samples.

The VOC results were also compared to reference studies conducted in Halifax. The VOCs that are related to vehicles and combustion were present in both Pictou Landing and Halifax. However, 4-chlorotoluene and 2-chlorotoluene were present only in Pictou Landing from the ruptured effluent pipe. Chlorine and aromatics are present during the kraft and pulp and paper process and these are the likely source of the chlorinated aromatics. Further research could possibly confirm this.

Since the effluent spill, in March 2015, the *Boat Harbour Act* was passed, which legislated the closure of the Boat Harbour treatment facility by January 30, 2020. And on May 11, 2016, Judge Del W. Atwood released his Sentencing Decision for *R. v. Northern Pulp Nova Scotia Corporation,* 2016 NSPC 29. On January 20, Northern Pulp had pleaded guilty to breaching subsection 36(3) and subsection 40(2) of the *Fisheries Act* for the

illegal spill of pulp and paper effluent in water frequented by fish and/or in any other place where the effluent would then enter into water frequented by fish. The judge's opening of his Sentencing Decision is worth noting:

> *The first element of truth and reconciliation is truth. The undeniable truth is that the experience of the Pictou Landing First Nation has been one of subjugation and suppression under the Canadian federation. It shares this history with other First Nations of Canada, as described succinctly in the Report of the Truth and Reconciliation Commission of Canada. An instance of that injustice was the manner in which a pulp mill came to [sic] located at Abercrombie Point in Pictou County fifty years ago; along with the mill was built an effluent-treatment plant in Boat Harbour.*

The spill in June 2014 released an estimated 47,000,000 litres of effluent into the East River/Pictou Harbour from the deteriorating pipe. Afterwards, Northern Pulp responded to and implemented remedial measures put forth by Environment Canada. Additionally, the prosecution's obligation to consult with Pictou Landing First Nation about the spill's impact led to a presentation by Chief Andrea Paul about the impact to the community. Chief Paul discussed the environmental impacts in the context of the area's historical environmental degradation as a result of the pulp and paper mill. She noted that the spill re-victimized Pictou Landing residents and relayed their feelings that they had disappointed the environment; that the burial grounds at Indian Cross Point would be destroyed; and that the commercial, food, and ceremonial fishery would be negatively impacted as well.

The judge's decision included a reflection on her presentation, as he noted:

> *In my view, while the historical account in Chief Paul's statement might extend beyond what counsel assert is admissible, the truth of the damaging impact that the pulp mill at Abercrombie Point and its toxic effluent-treatment site at Boat Harbour has had on the well-being of the Pictou Landing First Nation – and continues to have – is so conspicuous and notorious as to be beyond dispute.* (R. v. Northern Pulp Nova Scotia Corporation, 2016)

Importantly, Judge Atwood noted his decision was a part of transitional justice and that reconciliation would move forward in a small way in light of it. The sentence of the court was that Northern Pulp be fined in the amount of $225,000. It recommended that the fine be distributed by awarding $75,000 to the Mi'kmaw Conservation Group, $75,000 to the Pictou County Rivers Association, and $75,000 to Pictou Landing First Nation to be used to conserve, protect, and restore fish and fish habitat in Pictou County and in other waters fished by Pictou Landing First Nation.

12. Community Mapping

Daniel Rainham (Associate Professor and Elizabeth May Chair in Sustainability and Environmental Health, Environmental Science Program, Dalhousie University) supervised the community mapping actities of his trainee, **Jane McCurdy**, a summer intern through Dalhousie University's Master of Resource and Environmental Management program. They created a "web map" configured with text, photos, video clips, and website links to display traditional, cultural, and spiritual practices and recreational activities connected to community-identified places over time – past, present, and future. The community map shows changes in the way residents of Pictou Landing have interacted with their land over time.

Jane worked with **Heather Castleden** and **Dee Lewis** to read the oral histories documented by **Ella Bennett** to discern places of importance for fishing; hunting; gathering berries, seaweed, and medicines; and other recreational, cultural, and spiritual practices. **Kim Strickland** reviewed the additional oral histories that Dee documented for other places of importance. Dee also reviewed responses to the Environmental Health Survey for important places. The Pictou Landing Native Women's Group (PLNWG) were then able to use printed maps to pinpoint the place names

referenced in the stories to include in the web map. The map has also been geocoded with air, water, sediment, tree, and soil sampling sites as well as trapping sites.

The community map is now embedded in Daniel's website (SILK-Lab: www.silk-lab.org/pictou-landing-first-nation-community-ma) and is to be linked into the Pictou Landing First Nation website as well as Heather Castleden's Research Lab website (www.heclab.com) as a legacy map. As well, Jane prepared a user-friendly guide for navigating the map: "Web Mapping Application: A User Guide for the Pictou Landing First Nation's Community Web Mapping," as well as an amendment called "Steps for Accessing a Web Mapping Application via ArcGIS Online." The User Guide explains that a Web Mapping Application is a "living map," meaning changes can be made to it. The map has background layers, which are called the basemaps and show the geographic area of Pictou Landing First Nation. Other layers can sit on top of the basemaps to display the map's many features. To access the map, you can visit the websites noted above and then explore basemaps and layers of information. Directions for navigating the map have been pulled from Jane McCurdy's User Guide and briefly noted below.

Figure 36 displays the basemap options that are available within the map – click the dropdown arrow next to the *Basemap* icon to view thumbnails of each basemap.

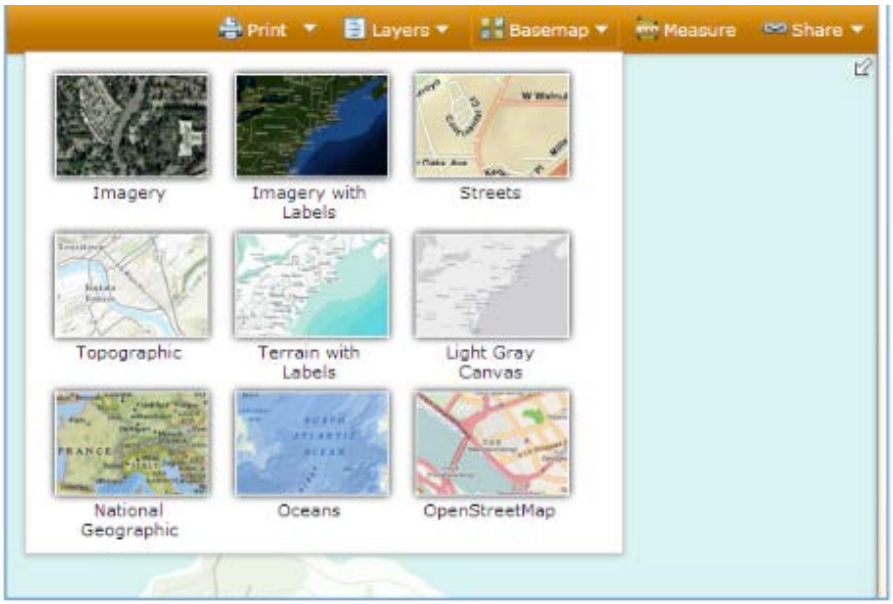


Figure 36. Assortment of basemaps to display as a background for the data.

Figure 37 shows two figures from the User Guide: Figure 2 from the User Guide illustrates that clicking the arrow in the top right-hand corner allows you to either show or hide the *Map Overview*. Figure 3 from the User Guide illustrates how to turn layers on and off – how to select what types of information you want to see displayed at any one

time by the map. In order to select which layers to display, click the dropdown arrow next to the *Layers* icon to check/uncheck the layers you would like to have visible on the map.

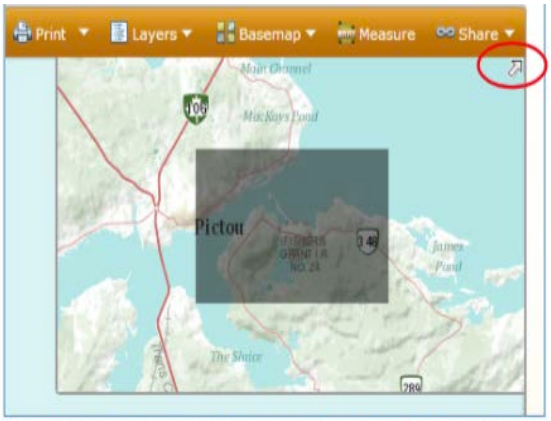


Figure 2: Clicking the arrow in the corner shows and hides Map Overview.

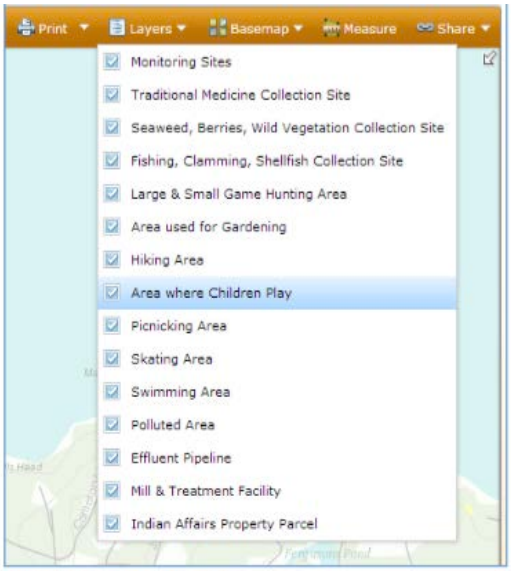


Figure 3: Display of layer list.

Figure 37. Two figures from the Web Mapping Application's User Guide.

Figure 38 shows what a layer of the map looks like and the informational pop-up that is visible when you click on a point of data on the map.

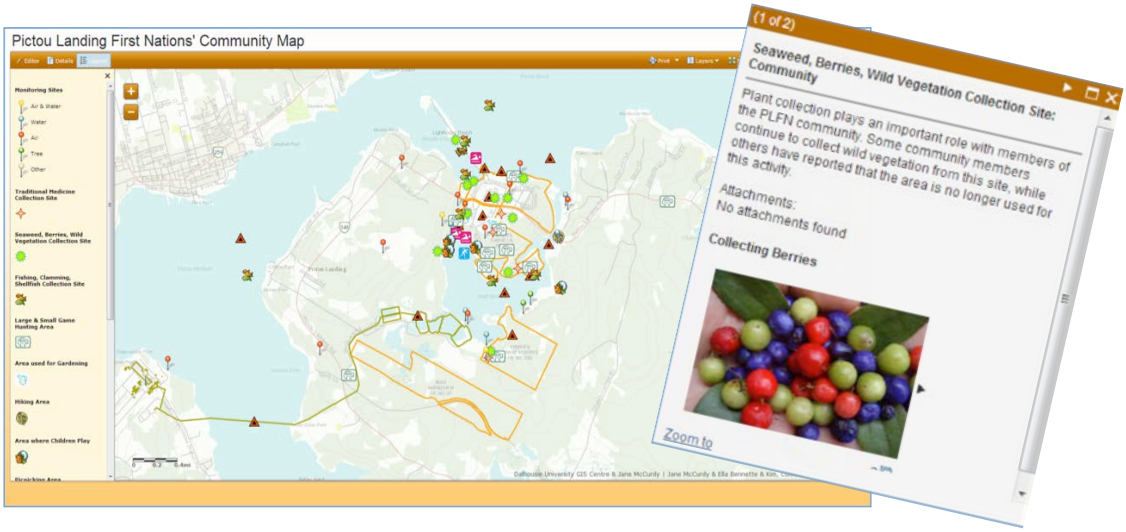


Figure 38. Map layer and informational pop-up.

You can also print and share the map via email, Facebook, or Twitter. And if revisions or additions to the map are needed, the PLNWG can contact Daniel Rainham to do so (Daniel.rainham@dal.ca).

13. Wet-Pro Water Monitoring

The CURA H2O project (Community-Based Water Monitoring and Management, housed at Saint Mary's University; http://curah2o.com) is a community-university research alliance to establish volunteer water monitoring in Nova Scotia; **Heather Castleden** was a member of the CURA H2O team. The project introduces communities to Wet-Pro, which is an online training course in water quality monitoring with an accompanying monitoring equipment toolkit for community-based water monitoring groups. In May 2013, **Sheila Francis, Kim Strickland, Colleen Denny**, **Lucie Francis, Emily Skinner** and **Heather** attended the CURA H2O Community-Based Water Monitoring Program Design and Database workshop at Saint Mary's University to design a water monitoring plan for Pictou Landing. As a result of their training, the PLNWG received a Wet-Pro kit to begin collecting water samples. The Wet-Pro kits have benefited this research as a legacy piece of the project because the community now has three certified water quality monitors: Kim Strickland, Colleen Denny, and Lucie Francis. The PLNWG can access their Wet-Pro kit any time via CURA H2O, which is the steward of this equipment.

Regular monitoring can characterize the health of the ecosystem, detect changes, and establish baseline data including conductivity, pH, and dissolved oxygen.

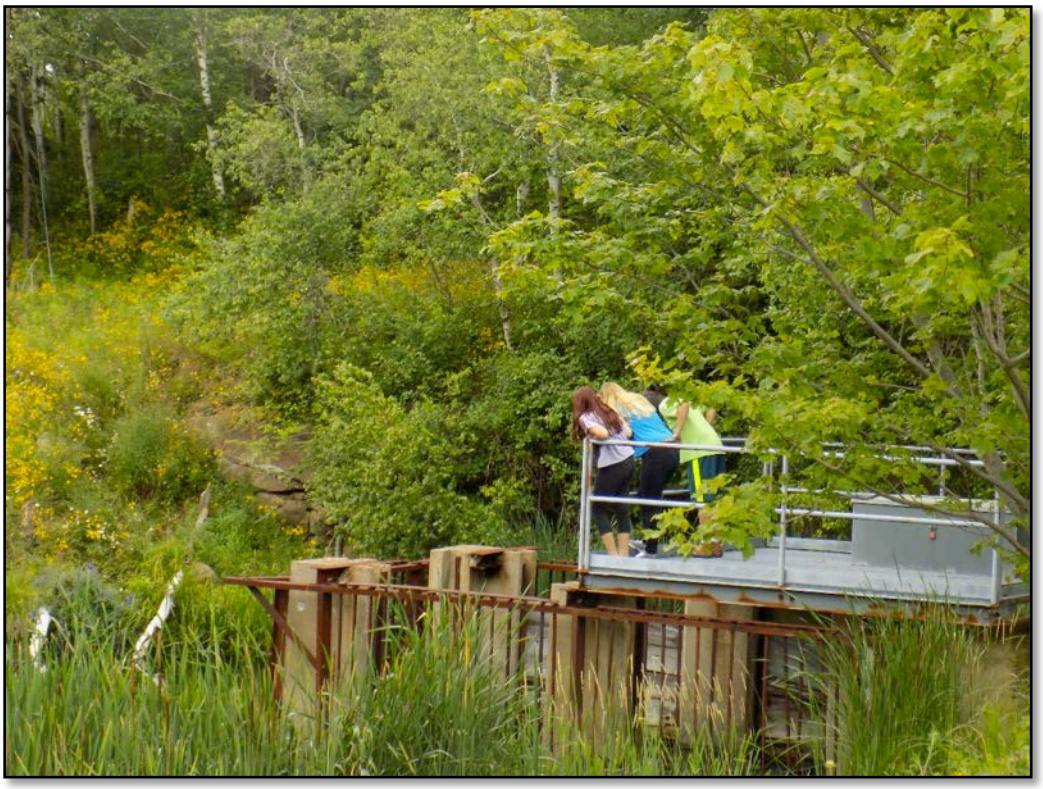
The Wet-Pro Liaisons, **Oliver Woods** and **Sarah Weston**, have supported the Wet-Pro initiative by attending meetings to discuss strategic water monitoring, supporting the individual water monitors, evaluating the functioning of the monitoring equipment from season to season, and facilitating workshops and sampling. For example, Oliver had to replace a probe in the equipment in the summer of 2014. Oliver, Sarah, and **Melissa Healey** also travelled to Pictou Landing in the summer of 2013 to support water testing and site selection with Kim, Colleen, **Chris Garda**, and **Geoff Kershaw**. In May 2014, Kim and Colleen, and subsequently Kim in 2015, attended the Atlantic Watershed Stewardship Showcase as part of the continuing training. The Wet-Pro team at Saint Mary's University are available to ensure water quality monitoring can continue in Pictou Landing and the equipment, while owned by the PLNWG, will continue to be stored there throughout the monitoring off-season. It will be up to the PLNWG to determine whether continued monitoring can and should occur.[7]

[7] "The CURA H2O project officially wrapped up in September 2016, and the Community-Based Environmental Monitoring Network continues to develop and maintain major CURA H2O legacy pieces such as the training course and database. Partnerships with government agencies and major universities continue to examine how this data can be integrated into governmental water management and provide a more comprehensive set of data than would otherwise be available through government resources alone." (http://curah2o.com)

14. Boat Harbour Youth Camp, July 2015

Throughout this research, the importance of multi-generational perspectives in understanding Indigenous health and environmental change was recognized. In the months approaching summer 2015, the Pictou Landing Native Women's Group (PLNWG) indicated it was time to engage and understand the perspectives of the youth, who represent the future leaders and decision-makers of the community and who have only ever experienced Boat Harbour as a toxic waste facility.

Over four days in July 2015, a group of five youth, **Laela Denny, Madison Nicholas, Hunter Francis, Alexandria Francis,** and **Carter Hatfield,** came together for the Boat Harbour Youth Camp, led by **Cecilia Jennings** (Heather Castleden's Master's student), **Kim Strickland**, and **Dakota Francis** (a summer student from Pictou Landing First Nation). The purposes of the camp were (1) to teach youth from Pictou Landing about the PLNWG's research project and (2) to gather perspectives from Pictou Landing First Nation youth about their experiences living with Boat Harbour. The group met for four days over two weeks, during which they learned about community mapping and water sampling, took a field trip to Halifax, and created digital stories.



Camp Structure

Day 1: Community Mapping and Learning About the PLNWG
The camp opened with introductions and watching the *A'se'k* documentary by **Christian Francis** and **Haley Bernard.** The documentary was the starting point for a conversation about the youths' impressions of Boat Harbour and what health means for them as individuals and for the whole community. Then, using GPS units borrowed from Dalhousie University, the group participated in a community mapping workshop. They went for a walk around the community, each taking GPS coordinates at places that represented good and negative effects on their health. Each participant was given a journal to take field notes throughout the camp and also used the journal to record notes about each GPS point.

Dee Lewis visited from Halifax and talked to the group about the PLNWG's participation in the project and screened the *Land & Sea* documentary about Boat Harbour as well as the digital stories made by herself, Kim, **Sheila Francis, Colleen Denny,** and **Darlene Bachiri**.

Day 2: Field Trip to Halifax
The camp group travelled to Dalhousie University, where they visited the Centre for Water Resources Studies. **Jenny Hayward** (Rob Jamieson's research associate) gave a tour of three different environmental engineering labs and talked about some of the sampling that has happened at Boat Harbour.

The group also visited the Health Geomatics Laboratory, where **Daniel Rainham** gave a lesson in map-making. Using the GPS points collected in the community walk during Day 1, each youth began a map to represent their own "health landscapes" in Pictou Landing.

Days 3 & 4: Wet-Pro Sampling and Digital Stories

On the third day, **Emma Wattie** and **Sarah Weston** from CURA H2O (Saint Mary's University) visited Pictou Landing First Nation to teach a Wet-Pro workshop. They taught the youth how to evaluate water quality and take measurements with the Wet-Pro kit. Everyone got a chance to try out the Wet-Pro kit, and the youth sampled and compared water from three sites around Pictou Landing First Nation.

In the afternoon and during the last day, the group worked on their digital stories, writing scripts in their journals, collecting photos from the Internet and from throughout their time at the camp, recording their voice-overs, and making final edits.

Sharing the Stories

For some of the youth participating in the camp, this was the first time they had been asked to share their feelings about Boat Harbour publicly. Three digital stories were made by youth during the camp, by Madison, Hunter, and Alexandria (Cecilia made one

as well). The narratives shared in the videos speak to the sense of loss experienced by youth in Pictou Landing, and the feelings of frustration at a legacy of broken promises. Even though these youth have never lived with a clean Boat Harbour, they carry the desire to be connected with A'se'k.

The youth camp sought to address and engage youth on questions of health and the environment. Overall, the camp opened up a space for learning and discussion among the group of participants. And it offered a chance for knowledge translation and for sharing the work of the PLNWG and research team over the past few years.

15. Knowledge-sharing Activities and Capacity-building

Throughout the project, reporting back to the community was important. Through discussions at Pictou Landing Native Women's Group (PLNWG) meetings, Research Retreats, and Community Dinners, changes were made to research protocols and goals. Examples of how information was shared include: monthly Chief and Council updates; PLNWG meetings; Community Dinners; annual retreats; documentary film and photography projects; Health, Environment, and Communities Research Lab website (heclab.com); media interviews; and a variety of publications including the Pictou Landing First Nation newsletter, academic journal articles, final reports, and booklets.

Event Participation and Conference Presentations

- **Heather Castleden, Dee Lewis,** and **Chief Andrea Paul** attended the Atlantic Policy Congress Atlantic First Nations Health Conference in Moncton, New Brunswick, to present on the process of developing the community-university research partnership in November 2012.

- **Ziyun Wang** presented her analysis of the Health Canada reports to the women at a PLNWG meeting before presenting "Pulling the Plug on Boat Harbor: A Synthesis Review and Gap Analysis of Existing Environmental and Health Studies" for her Master's degree in Environmental Studies in December 2012.

- **Ziyun** also presented at the Annual Atlantic Canadian Association of Geographers Conference in Halifax during the fall of 2012.

- **Dee** delivered a guest lecture titled "Gender and Culture: The Social and Health Impact Assessment in Environmental Assessment" to Heather's Socio-Political Dimensions of Resource and Environmental Management class in the winter of 2012.

- **Dee, Sheila Francis,** and **Heather** were invited to present at the Atlantic Aboriginal Economic Development Integrated Research Program (AAEDIRP) conference titled "Working Alongside Aboriginal Peoples in Research," hosted in Dartmouth February 5-7, 2013. Only Dee and Sheila could attend, and the subject of their presentation was the process of developing the community-university research partnership.

- In the summer of 2013, **Heather** brought her class to Pictou Landing as part of her course, Indigenous Perspectives on Resource and Environmental Management. **Colleen Denny** and **Kim Strickland** gave the group a tour of the community, introduced the project, and discussed their roles as Research Associates.

- **Sheila, Kim, Colleen, Dee**, and **Heather** presented their digital stories at the Community-Campus Partnerships for Health 13th International Conference, "From Rhetoric to Reality: Achieving Authentic, Equitable and Transformative Partnerships" (April 30–May 3, 2014) in Chicago, Illinois. The stories are about their personal journeys and project experiences and can be viewed online at www.heclab.com.

- In June of 2014, **Dee** and **Sheila** travelled to Happy Valley-Goose Bay; they were invited by FemNorthNet to participate in a process with other Indigenous women: those from Labrador who will be increasingly impacted by the Muskrat Falls project, and those from Nova Scotia who will be impacted by the Maritime Link.

- **Heather, Kim, Dee,** and **Darlene Bachiri** attended the International Network in Indigenous Health Knowledge and Development (INIHKD) in partnership with the Manitoba Network Environment for Aboriginal Health Research (NEAHR) Conference in Winnipeg, Manitoba, from October 5 to October 10, 2014.

- **Dee** facilitated a session for Environmental Justice Alliances on November 22, 2014, at St. Francis Xavier University called "Building Alliances and Seeking Reconciliation with Mi'kmaq Women: A Day of Action and Dialogues." **Sheila** served as a panelist. The session included a short overview of the project and a discussion about the issues and strategies to build alliances for positive actions to protect, uphold, and implement Indigenous rights.

- On January 23, 2015, **Dee** presented her PhD thesis proposal at Dalhousie University, titled "Tlilnuo'lti'k – Weji-sqalia'timk – How We Will Be Mi'kmaq on Our Land: Working Together with Pictou Landing First Nation to Redefine a Healthy Community."

- **Sheila, Heather, Kim, Colleen, Dee,** and **Darlene** presented their digital stories as a group at the Citizen Science 2015 Conference held February 11-12, 2015, in San Jose, California.

- **Dee** presented at Environmental Racism and First Nation Women – International Women's Week on March 4, 2015, at the Antigonish Women's Resource Centre.

- **Heather** presented a poster at the Community-Campus Partnerships for Health 15th International Conference, "Journey to Justice: Creating Change Through Partnerships," in New Orleans, Lousiana (May 11–14, 2016), where the poster presentation was awarded second place in the Viewers' Choice Award category.

- **Sheila, Kim, Dee,** and **Lexy Strickland** are scheduled to present at Dalhousie University's Indigenous Speaker Series in September 2016.

Peer-reviewed Academic Articles

- Given the remarkable response rate (approximately 60%) for the Environmental Health Survey, **Dee Lewis** led the development and submission of a manuscript on lessons learned from the process of conducting a survey using community-based participatory research methods. The article, authored by **Dee, Heather Castleden, Sheila Francis, Kim Strickland, Colleen Denny** and the **PLNWG**, is called "Increasing Response Rates on Face-to-face Surveys with Indigenous Communities in Canada: Lessons from Pictou Landing," and it was published by the journal *Progress in Community Health Partnerships* (2016).

- An article titled "'Put It Near the Indians': Indigenous Perspectives on Pulp Mill Contaminants in Their Traditional Territories (Pictou Landing, Canada)," authored by **Heather, Ella Bennett, PLNWG, Dee,** and **Debbie Martin,** has been accepted for publication by the journal *Progress in Community Health Partnerships*.

- A third peer-reviewed article is in preparation regarding the environmental monitoring components of the project: air, water, soil, and ecotoxicology.

Community Dinners

- The first Community Dinner was held on February 16, 2012. This dinner was a celebratory occasion as we had just received notice that our Canadian Institutes of Health Research Operating Grant had been approved.

- A second Community Dinner was held in Pictou Landing First Nation on January 15, 2013, to provide research activity updates. **Kim Strickland** and **Colleen Denny** organized and advertised the event and arranged for dinner to be served to about 40 people who attended.

- A third Community Dinner was held in Pictou Landing on June 18, 2013. More than 80 community members attended the BBQ organized by **Kim** and **Colleen**. Research activity updates were given by **Heather Castleden, Dee Lewis, Jane McCurdy** (on behalf of **Daniel Rainham**), **Mark Gibson, Rob Jamieson,** and **Ron Russell**.

- A fourth Community Dinner was held April 8, 2014. The agenda included a discussion about how the research project came to be and a summary of the first three years of working together, updates about the project components, and a presentation by **Kim** and **Colleen** about their roles as Community Research Associates.

- A fifth and final Community Dinner will be held to release this report to the community.

Awards

- **Haley Bernard** received an internship award from the Atlantic Aboriginal Health Research Program to work on the project in 2012.

- **Lucie Francis** received an internship award from the Atlantic Aboriginal Health Research Program to work on the project in 2012 with **Ron Russell**.

- **Diana Lewis** received a Canadian Institutes of Health Research Doctoral Research Award in May 2013 for three years to continue research with the PLNWG.

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- Information Morning Nova Scotia Interview with **Heather Castleden.** (2014, August 5).

- CBC/Global TV Interview with **Mark Gibson** (2014, August 7). *Northern Pulp newsmaker*. http://www.cbc.ca/player/play/2483990998

- **Martha Stiegman** and **Ella Bennett** filmed many aspects of the research before **Cathy Martin** and **Frank Clifford** joined the team to film research activities. We hope to obtain additional funding for Cathy Martin to produce a documentary using all the filmed material in the future.

Training

- **Dee Lewis** attended the Centre for Environmental Health Equity's National Training Program, "Knowledge Leaders in Children's Environmental Health," held in Vancouver, British Columbia, in 2012.

- **Kim Strickland** and **Colleen Denny** were hired as Community-Based Research Associates on the project on November 1, 2012. Colleen worked as a Research Associate until spring 2014 and Kim continued until June 2016.

- **Lucie Francis** and **Haley Bernard** were both awarded Atlantic Aboriginal Health Research Program summer internships to conduct research activities; respectively, they worked on ecotoxicology and oral histories.

- Thirteen women from the community administered Environmental Health Surveys: **Colleen, Kim, Pam Denny, Haley Bernard, Jordan Francis, Sheila Francis, Fran Nicholas, April Nicholas, Darlene Bachiri, Holly Francis, Heather Mills, Sylvia Francis,** and **Loretta Sylliboy** between 2012 and 2013.

- **Dee** gave two Environmental Impact Assessment (EIA) workshops in Pictou Landing in the fall of 2015 to inform the women about the language used in the Environmental Assessment process and case studies illustrating how they work. The first session involved learning the terminology specific to EIAs, and understanding the differences between types of EIAs and the basics of the process. The second session reviewed relevant case studies to put the previous learning into practice.

Theses, Reports, and Booklets

- "Final Report of Epitik Mawi-ta'jik: Pictou Landing Women's Retreat" (2011), prepared by **Dr. Heather Castleden** and **Ms. Ella Bennett** (School for Resource and Environmental Studies, Dalhousie University).

- "Pulling the Plug on Boat Harbour: A Synthesis and Gap Analysis of Existing Environmental and Human Health Assessments Including Pictou Landing First Nation, 1968-2007" (2012), prepared by **Ms. Ziyun Wang** (School for Resource and Environmental Studies, Dalhousie University).

- "Community Report: Boat Harbour Water Quality" (2013), prepared by **Dr. Rob Jamieson** (Centre for Water Resources Studies, Dalhousie University).

- "The Pictou Landing Native Women's Association: Boat Harbour Project" (2013), prepared by **Mr. Chris Garda** and **Ms. Kim Strickland** (available at www.heclab.com). It is an eight-page brochure with beautiful photographs, a description of the research team, an introduction to the pulp mill's history and the subsequent PLNWG mobilization, Two-Eyed Seeing, and the components of the project (ecotoxicology, air monitoring, oral histories, environmental health surveys, water monitoring, and community mapping).

- "Third Annual Pictou Landing Women's Research Retreat: Final Report" (2014), prepared by **Dr. Heather Castleden, Ms. Ella Bennett,** and **Ms. Emily Skinner** (School for Resource and Environmental Studies, Dalhousie University).

- "Soil Sampling Results for Dioxins, Furans and Metals in Pictou Landing First Nation, NS" (2014), prepared by **Dr. Rob Jamieson** (Centre for Water Resource Studies, Dalhousie University).

- "Our Ancestors Are in Our Land, Water, and Air: A Two-Eyed Seeing Approach to Researching Environmental Health Concerns with Pictou Landing First Nation: Air Quality Report" (2015), prepared by **Dr. Mark Gibson** (Atmospheric Forensics Research Group, Dalhousie University).

- "Sediment and Plankton Sampling for PCBs, Pesticides, Chlorophenols, Polybrominated Diphenyl Ethers, and Polychlorinated Dibenzodioxins and Furans" (2015), prepared by **Dr. Ron Russell** (Department of Biology, Saint Mary's University).

- "Fourth Annual Pictou Landing Women's Research Retreat Report" (2016), prepared by **Ms. Catherine Hart** (Health, Environments, and Communities Research Lab, Department of Geography and Planning, Queen's University).

- "Community Report: Environmental Health Survey" (forthcoming), prepared by **Ms. Diana Lewis** (Health, Environments, and Communities Research Lab, Department of Sociology and Social Anthropology, Dalhousie University).

"Not only are we going to do scientifically sound research… but it's going to come from us!"

(First Research Retreat, 2010)

16. Concluding Comments

Our six-year community-based participatory research project to explore the potential mental, physical, emotional, and spiritual health impacts of Boat Harbour for the members of Pictou Landing First Nation was, at times, arduous, rewarding, emotional, and certainly a transformative experience for those involved. Mobilizing the women of Pictou Landing, through the Pictou Landing Native Women's Group (PLNWG), around an issue that concerned all of our community, is an example of how Mi'kmaw women are revitalizing our roles as leaders and protectors in our community. For the academic members of this research team, it was a privilege and an honour for us to be invited to work with the PLNWG and support the women's research goals.

A'se'k was a place of highly productive subsistence fisheries, with recreational and medicinal functions for the Mi'kmaq of Pictou Landing. Although the land has never been subject to a treaty beyond "Peace and Friendship," 50 years ago the Boat Harbour Treatment Facility began releasing approximately 85 million litres of pulp mill effluent into A'se'k daily. We do not know, with absolute certainty, how much pollution the Mill has released and continues to release into the local environment, but we know that respiratory illness (e.g., asthma) is substantial in the community. When the mill started operating, fish kills were immediate, and significant social, psychological, and cultural impacts continue to affect the community. This is, in part, due to the mill's continued operation and consequent pollution. But it is also due to a legacy of broken government promises through unfulfilled commitments to relocate the waste treatment facility and remediate the estuary. A'se'k/Boat Harbour has, undoubtedly, transformed into a place of dis-ease, anxiety, and unrest (Castleden et al., in press).

While this report marks the conclusion of our research project, it does not mark the conclusion of our relationships and our commitments to each other, and the lands, waters, and air around us. Our story is not over…

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Journal of Steroid Biochemistry & Molecular Biology 127 (2011) 204–215

Contents lists available at SciVerse ScienceDirect

Journal of Steroid Biochemistry and Molecular Biology

journal homepage: www.elsevier.com/locate/jsbmb

Review

Endocrine disrupting chemicals and disease susceptibility

Thaddeus T. Schug[a,*], Amanda Janesick[b], Bruce Blumberg[b], Jerrold J. Heindel[a]

[a] National Institute of Environmental Health Sciences, Division of Extramural Research and Training, Cellular, Organ and Systems Pathobiology Branch, Research Triangle Park, NC 27709, USA
[b] Department of Developmental and Cell Biology, 2011 Biological Sciences 3, University of California, Irvine, CA 92697-2300, United States

ARTICLE INFO

Article history:
Received 4 August 2011
Accepted 6 August 2011

Keywords:
Endocrine disrupting chemicals
Hormone
Disease susceptibility
Bisphenol A
Nuclear receptor
Obesogen
Low dose effects
Developmental windows of susceptibility

ABSTRACT

Environmental chemicals have significant impacts on biological systems. Chemical exposures during early stages of development can disrupt normal patterns of development and thus dramatically alter disease susceptibility later in life. Endocrine disrupting chemicals (EDCs) interfere with the body's endocrine system and produce adverse developmental, reproductive, neurological, cardiovascular, metabolic and immune effects in humans. A wide range of substances, both natural and man-made, are thought to cause endocrine disruption, including pharmaceuticals, dioxin and dioxin-like compounds, polychlorinated biphenyls, DDT and other pesticides, and components of plastics such as bisphenol A (BPA) and phthalates. EDCs are found in many everyday products – including plastic bottles, metal food cans, detergents, flame retardants, food additives, toys, cosmetics, and pesticides. EDCs interfere with the synthesis, secretion, transport, activity, or elimination of natural hormones. This interference can block or mimic hormone action, causing a wide range of effects. This review focuses on the mechanisms and modes of action by which EDCs alter hormone signaling. It also includes brief overviews of select disease endpoints associated with endocrine disruption.

Published by Elsevier Ltd.

Contents

1. Introduction

EDs are synthetic chemicals that were originally designed for a specific action such as a pesticide, plasticizer, or solvent, but now have been found to have a side effect that when absorbed into the body causes them to either mimic or block hormones and disrupt the body's normal functions. This disruption can occur by altering normal hormone levels, inhibiting or stimulating the production and metabolism of hormones, or changing the way hormones travel through the body, thus affecting the functions that these hormones control. EDCs were originally thought to exert their actions solely through nuclear hormone receptors, including estrogen receptors (ERs), androgen receptors (ARs), progesterone receptors, thyroid receptors (TRs), and retinoid receptors, among others (Table 1) [1]. However, recent evidence shows that the mechanisms by which EDCs act are much broader than originally recognized. Indeed,

* Corresponding author. Tel.: +1 919 541 9469.
E-mail address: schugt@niehs.nih.gov (T.T. Schug).

0960-0760/$ – see front matter. Published by Elsevier Ltd.
doi:10.1016/j.jsbmb.2011.08.007

Table 1
Select human nuclear receptors and related functions.

Receptor	Abbreviation	Physiological function	Endogenous ligand	Examples of endocrine Disrupting chemicals
Androgen	AR	Male sexual development	Testosterone	Pesticides Phthalates Plasticisers Polyhalogenated compounds
Estrogen	ER α, β GPR30 (non-nuclear)	Female sexual development	Estradiol	Alklyphenols BPA Dioxins Furans Halogenated hydrocarbons Heavy metals
Thyroid hormone	TR α, β	Metabolism Heart rate	Thyroid hormone	BPA Dioxins Furans PBDEs PCBs Perchlorates Pesticides Phalates Phytoestrogens
Progesterone	PR	Female sexual development	Progesterone	BPA Fungicides Herbicides Insecticides
Arylhydrocarbon	AhR	Circadian rhythm Metabolism Neurogenesis Organ development Stress response	Unknown	Dioxins Flavonoids Herbicides Indoles PCBs Pesticides
Peroxisome proliferator-activated	PPAR α, β, λ	Lipid homeostasis	Lipids/fatty acids	BPA Organotins
Glucocordicoid	GR α, β	Development Metabolism Stress response	Cortisol	Arsenic BPA Phthalates

studies have shown that in addition to altering nuclear receptor signaling, EDCs are capable of acting through nonsteroid receptors, transcriptional coactivators, enzymatic pathways involved in steroid biosynthesis and/or metabolism, and numerous other mechanisms that converge upon endocrine and reproductive systems [1,2]. Other less well known mechanisms of action of EDCs include direct effects on genes [3] and their epigenetic impact [4]. These effects are particularly troubling since alterations in genetic programming during early stages of development may have profound effects years later and may also lead to transgenerational inheritance of disease (Fig. 1) [5].

There are several characteristics of the endocrine system that must be understood in order to develop a full understanding of the mechanisms of actions and the consequences of exposure to EDCs. For instance, similar to hormones, EDCs can function at very low doses in a tissue specific manner. EDCs may also exert non-traditional dose–responses due to the complicated dynamics of hormone receptor occupancy and saturation. Thus low doses may have more impact on a target tissue than higher doses, and the effects and dose–response curve may be entirely different. The age at which an individual is exposed to an EDC also has important implications on resulting health consequences. Indeed, it is now clear that exposure to EDCs during development results in different effects than exposures during adulthood. Adults require higher concentrations for EDCs to cause toxicity and their effects only last as long as the EDC is present. Low dose exposure during development can result in disruptions that lasts long after the EDC is gone from the body. For this reason, the field of endocrine disruption coined the term "the fetal basis of adult disease", or FeBAD, to describe the interactions between the developing organism and the environment that determine the propensity of that individual to develop disease later in life [1]. This concept has been extended beyond the fetal period to include the early postnatal developmental period when organs continue to undergo substantial development. DOHaD (developmental origins of health and disease) describes the interactions between the developing organism and the environment that determine the propensity of that individual to develop disease across its lifespan [1].

Evidence in animal models suggests that EDCs may affect not only the exposed individual but also the offspring and subsequent generations. The mechanism of transmission involves non-genomic modifications of the germ line such as changes in DNA methylation and histone acetylation. Altogether, EDCs pose a significant challenge to our industrialized society and to the health of humans and the environment. Indeed, due to their wide commercial use and direct link to adverse human health outcomes, the Endocrine Society published a scientific statement in 2009 indicating that endocrine disruptors pose a "significant concern for public health" [1].

2. Modes of action

2.1. Nuclear receptor signaling

EDCs are structurally similar to many hormones, function at extremely low concentrations, and many have lipophilic properties. EDCs are capable of mimicking natural hormones and maintain similar modes of action, transport, and storage within tissues. The properties of these chemicals, while unintended, make them particularly well suited for activating or antagonizing nuclear hormone receptors. Thus, there is virtually no endocrine system immune to these substances, because of the shared properties and similarities of receptors and enzymes involved in the synthesis, release, and degradation of hormones (Table 1) [1].

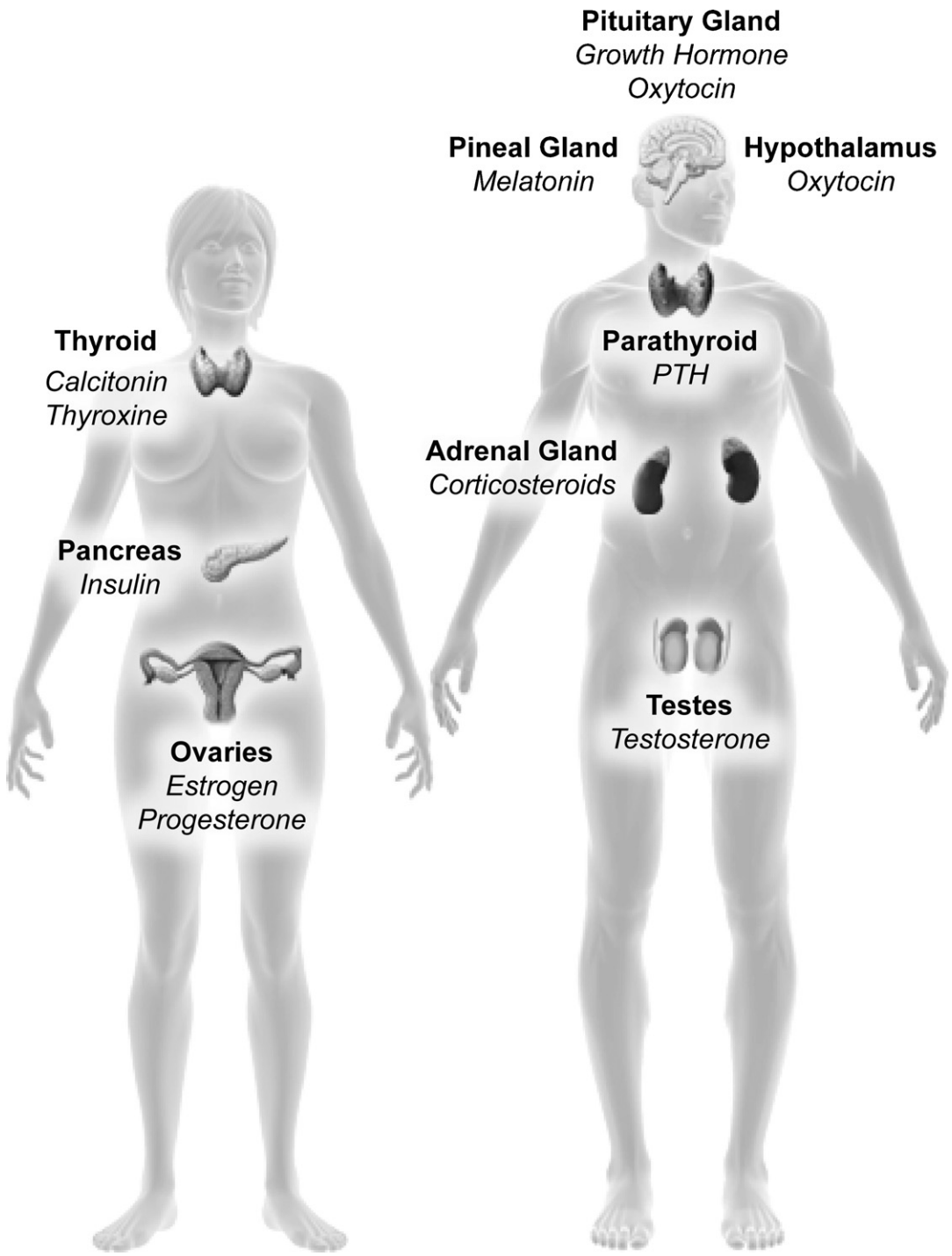

Fig. 1. Model of the endocrine systems targeted by EDCs. This figure illustrates that all major endocrine organs are vulnerable to endocrine disruption, including the HPA axis, reproductive organs, the pancreas, and the thyroid gland. EDCs are also known to impact hormone-dependent metabolic systems and brain function.

The nuclear hormone receptors are a super family of transcription factors that play important roles in both physiology and disease. In humans, there are some 48 nuclear receptors and many remain "orphans" as their endogenous ligands are yet to be determined. Research on the roles of nuclear receptors has been limited largely to the use of synthetic agonists, as well as genetic approaches to alter expression. This contrasts with the estrogen receptors (ERα and ERβ), which have been extensively studied [6]. These receptors remain at the center of endocrine disruption research, as outlined below, and results from these studies may provide a model for how other nuclear receptors interact with hormone mimics.

The estrogens are a group of steroid hormones produced by enzymatic modification of cholesterol. The primary estrogen of the reproductive years in females is 17β-estradiol (estradiol), which is derived from testosterone by aromatase activity. There are a wide range of natural and synthetic molecules that can activate ERα and ERβ. Natural estrogens include those produced by plants (phytoestrogens) and fungi (mycoestrogens). Synthetic ER activators include those intentionally produced for use in humans

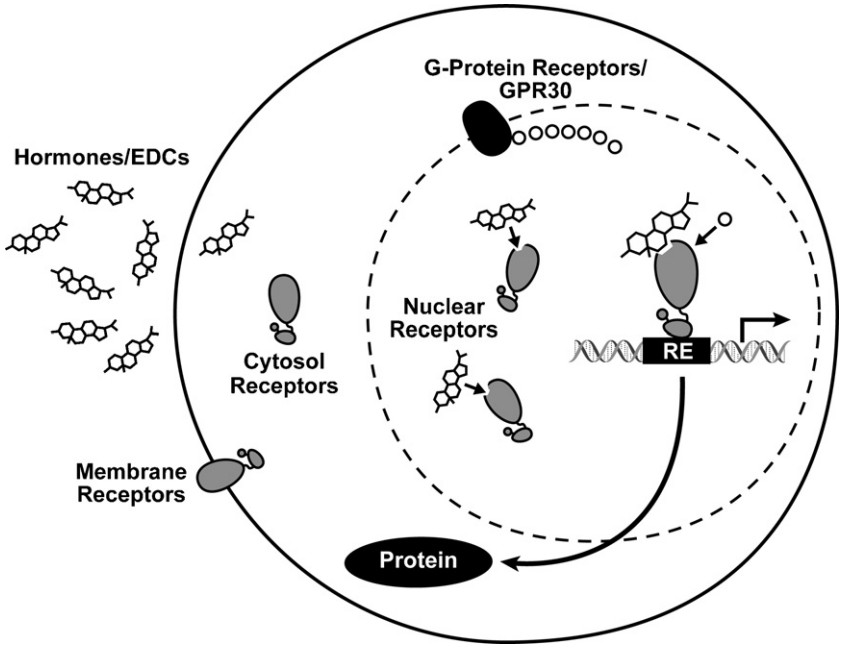

Fig. 2. Illustration of steroid hormone receptor signaling pathway. Hormones, or hormone mimics, bind to membrane or cytosol receptors, which in turn shuttle to the nucleus and attach themselves to response elements (REs), where they work to regulate gene transcription and ultimately protein production. Some receptors reside solely in the nucleus atop REs in inactive forms and become activated upon hormone binding. EDCs can alter this signaling process by binding to steroid receptors and either activating or inhibiting transcriptional response.

(e.g., diethylstilbestrol) as well as chemicals targeted for other uses that have unintended ER-modulating activities (e.g., DDT, methoxychlor). Identifying chemicals that display estrogenic activity is now a major focus of the research done on endocrine disruption.

Estrogenic compounds disrupt normal development via interaction with one of the estrogen receptors. There are three types of receptors for estrogens: the nuclear estrogen receptors (ERs), the membrane bound estrogen receptors (which are variants of the nuclear ERs), and the estrogen G protein-coupled receptor (GPR30), which is a membrane-bound protein with a high affinity toward estrogen. The main function of the ER is as a DNA-binding transcription factor that regulates gene expression and subsequent downstream responses (Fig. 2).

While some EDCs act as estrogen mimics, others have estrogenic activity but they are not true estrogens. For example, BPA was designed as a synthetic estrogen and has been shown to bind to the estrogen receptors (ERα, ERβ, and to the membrane ER), resulting in a cellular signal transduction cascade that is indicative of an estrogen response. [7,8] However, detailed examination of its effects on gene expression in a variety of tissues indicates that, while there is significant overlap, BPA does not stimulate the same suite of genes as estradiol. In addition there is mounting evidence that EDCs such as BPA interact with other nuclear receptors, albeit at higher concentrations. For example, one study found that BPA binds to the thyroid hormone receptor (TR) with a lower affinity than the estrogen receptor [9]. However, others believe BPA acts as an indirect antagonist of thyroid hormone (TH) and that its effects on TH action in vivo are likely dependent on the composition and relative abundance of cofactors available in the cell [10]. Studies have also shown that BPA binds to the ubiquitous aryl hydrocarbon receptor (AhR) [11]. This is not surprising because AhR is thought to be activated by many chemicals and likely mediates toxicity through several signaling pathways [11]. Thus, EDCs are not hormones, but they do display hormone-like properties that can have wide-ranging effects on cellular systems.

The focus of EDC research has been on estrogens, androgens and thyroid agonists and antagonists, but it is now clear that there are EDCs that affect other receptors and metabolic systems. Another nuclear hormone receptor targeted by EDCs is the peroxisome proliferator-activated receptor gamma (PPARγ) [reviewed in 12]. PPARγwas shown to be a key regulator of adipogenesis in vitro and in vivo and is important clinically as a target for drugs that ameliorate insulin resistance in type II diabetes. PPARγ functions as a heterodimer with the retinoid 'X' receptor, RXR; the RXR-PPARγ heterodimer is a ligand modulated transcription factor that directly regulates the expression of its target genes [13]. PPARγ is thought to be the master regulator of adipogenesis because it plays an important role in nearly all aspects of adipocyte biology [reviewed in 14, 15]. Activation of PPARγ2 in preadipocytes induces them to differentiate into adipocytes and PPARγ is required for this process in vitro and in vivo [16,17]. Moreover, expression of PPARγ is sufficient to transform susceptible stem cells into preadipocytes [18]. Activating the PPARγ pathway drives mutipotent stromal stem cells to enter the adipogenic pathway whereas inhibition of PPARγ expression promotes an osteogenic fate [reviewed in 19, 20]. It is known that humans whose diabetes is being treated with rosiglitazone (a drug that activates PPARγ) develop more adipocytes and gain weight [21]. Therefore it is reasonable to hypothesize that chemicals capable of activating PPARγ might have the same effect [reviewed in 22]. More research is needed to determine the extent to which EDCs interact will all nuclear receptors.

2.2. Low dose effects

For many years, toxicologists have relied on the presumption that "the dose makes the poison", first proposed by the Swiss physician and alchemist, Paracelsusin the 1500s. This view predicts that higher doses of a chemical will cause greater harm than low doses. This model is traditionally used by regulators to establish risk assessment profiles of chemicals. It relies on a monotonic dose–response curve generated from high and moderate dose

Fig. 3. Model illustrating early life exposures may cause functional changes at cellular levels that lead to changes in physiological status, and ultimately adult disease.

measurements that are linearly extrapolated downward to predict toxicity at very low doses. However, multiple studies on EDCs contradict this concept and question the adequacy of traditional toxicology testing paradigms for detecting low dose effects of EDCs. Indeed, these reports suggest that, similar to hormones, EDCs are capable of eliciting bi-phasic dose responses for many different endpoints at many levels of organization. These U-shaped and inverted U-shaped non-monotonic dose–response (NMDR) curves are used as evidence that very low concentrations of EDCs can effect endpoints such as cell proliferation and organ development.

NMDR curves have been described for numerous EDCs [23]. However, much controversy surrounds determining internal concentrations, the active metabolites, and the actual daily exposure levels of EDCs. The duration and route of exposure may also have a big influence on how the chemical is metabolized and whether or not the chemical remains biologically active. Additionally, the "low dose" levels at which these chemicals function are lower than those typically used in standard toxicology testing. This makes it difficult for toxicologists to use traditional rodent models to predict relevant endpoints for human exposures, when testing proceeds from a high dose and stops when a "no observed adverse effects level" (NOAEL) is reached.

Despite the controversy surrounding the "low dose" concept, there are several reasons why dose–response curves to toxicants may be non-monotonic. For example, the induction of metabolizing enzymes or conjugation of substrates my result in a U-shaped dose response for some endpoints. A recent study by Gualtieri et al., using Sertoli cells exposed to various doses of BPA (0.5 nM–100 μM), demonstrated that only intermediate doses (10 μM–50 μM), not high or low doses, induced an incremental increase in cell protecting glutathione levels [24]. Their findings show that detoxification through direct conjugation was enhanced at intermediate levels and cell viability was negatively affected at high and low doses where the cells were incapable of eliciting a response mechanism.

Several studies have suggested that non-monotonic responses can be explained by the down-regulation of receptors at higher hormone levels [25,26]. There is also evidence that NMDR curves are generated by the integration of two or more monotonic dose response curves that occur through different pathways affecting a common endpoint with opposing effects [27,28]. Furthermore, adaptive responses through complex cell signaling pathways and feed-back mechanisms could cause non-monotonic effects that are inconsistent with traditional dose–response curves. For example, Bouskine et al. reported that BPA stimulates JKT-1 cell proliferation in vitro in an inverse U-shape dose–response curve [29]. The authors propose that BPA activates two different signaling pathways that are distinct in both signaling mechanism and the time frame of response. In summary, making predictions about the safety of chemicals by testing at moderate or high doses is not appropriate when very low doses of endocrine disruptors can alter biochemical and morphological endpoints in a manner that is not necessarily predicted by exposures at much higher doses [30]. Lastly, it was proposed in a theoretical treatment that non-monotonic systems result from a loss of negative feedback and that such systems can be converted back into monotonic systems by adding back negative feedback [31]. This has important implications for EDCs since it well known that most hormonal signaling pathways are regulated by negative feedback and it has been demonstrated that EDCs differentially affect the stability of nuclear receptor proteins and ligands [reviewed in 32].

2.3. Developmental windows of susceptibility

Adult exposure to endocrine disrupting chemicals is certainly an important factor in adverse health outcomes, however focus on the fetus and/or neonate is of primary concern since developing organisms are extremely sensitive to perturbation by chemicals with hormone-like activity. Adverse effects may be most pronounced in the developing organism and occur at concentrations of the chemical that are far below levels that would be considered harmful in the adult [33,34]. Some of the reasons for this increased sensitivity include the fact that the protective mechanisms that are available to the adult such as DNA repair mechanisms, a competent immune system, detoxifying enzymes, liver metabolism, and the blood/brain barrier are not fully functional in the fetus or newborn. In addition, the developing organism has an increased metabolic rate as compared to an adult which, in some cases, may result in increased toxicity [34]. Finally, prenatal exposure to environmental factors can modify normal cellular and tissue development and function through developmental programming, such that individuals may have a higher risk of reproductive pathologies and metabolic and hormonal disorders later in life. Thus, exposures during critical windows of perinatal development may not manifest until much later in life. While fetal development is commonly known to be a period of increased sensitivity to chemical insult, childhood and adolescence are also marked by continued maturation of key endocrine systems, and are therefore susceptible to chemical exposure. Indeed, the DOHaD hypothesis, first proposed by David Barker in 1997, showed that poor in-utero nutrition resulted in high rates of disease manifested later in life [35]. This concept now includes non-nutritional early life exposures that have been shown to alter the body's physiology. Thus the DOHaD paradigm provides a framework to assess the effect of not only early nutrition but also EDCs on long-term health (Fig. 3) [36].

Of special concern are man-made hormone mimicking chemicals capable of evading defense mechanisms and misdirecting developmental decisions. Recent studies document detectable amounts of a variety of EDCs such as phthalates, polybrominated diphenyl ethers, polycyclic aromatic hydrocarbons, and BPA in pregnant women, fetuses, newborns, young children and adolescents [42–47]. Since each organ system has a different developmental trajectory, and the sensitive window for exposures to cause toxicity varies during tissue development, the effects of exposures are dependent not only on the type and dose of the chemical, but also when the exposure occurs [48]. These studies illustrate that the in utero developmental period is a critically sensitive window of vulnerability. Disruptions during this time-frame can lead to subtle functional changes that may not emerge until later in life [49]. Evidence now suggests that early life exposures to toxic chemicals can be directly associated with subsequent increases in the rates

Table 2
Developmentally induced diseases (**Human**[*]).

System	Disease	Chemicals
Reproductive/endocrine	Breast/prostate cancer	BPA
	Endometriosis	Dioxin, PCBs
	Infertility	Estrogens, pesticides, phthalates
	Diabetes/metabolic syndrome	BPA
	Early puberty[*]	Estrogens, BPA
	Obesity[*]	BPA, organochlorine pesticides, organotins
Immune/autoimmune	Susceptibility to infections	Dioxin
	Autoimmune disease	Dioxin
Pulmono-cardiovascular	Asthma[*]	Air pollution
	Heart disease/Hypertension	BPA
	Stroke	PCBs
Brain/nervous	Alzheimer's disease	Lead
	Parkinson's disease	Pesticides
	ADHD/learning disabilities[*]	PCBs, lead, ethanol, organochlorine pesticides

of many of the most common human diseases and the diseases that have increased the most in the last 20 years including asthma, learning and behavioral problems, early puberty, infertility, breast and prostate cancer, Parkinson's disease, obesity and other diseases (Table 2) [49–51].

Heindel and Newbold [52] described several important principles that demonstrate how early life environmental exposures contribute to increased risks of adult disease. First, chemical exposures can have both tissue-specific and time-specific consequences on growth and development. As long as tissue is developing, it is susceptible to disruptions from environmental exposures. These disruptions can result from changes in gene expression, protein activity, cell communication or other mechanisms. Secondly, the initiating in utero exposure may act alone or in concert with other environmental stressors. That is, the risk of developing disease in adulthood can be due to the combined insults over a lifetime. Thirdly, the pathophysiology may be manifested in a disease that otherwise might not have occurred and disease progression may have variable latent periods. Finally, the effects of environmental chemical exposures can be transgenerational, thus affecting future generations.

2.4. Epigenetic programming

One general mechanism by which prenatal and postnatal exposures could be linked to phenotypic changes later in life is through the alteration of epigenetic marks, which have a central role in determining the functional output of the information that is stored in the genome [37]. The term epigenetics refers to the factors around DNA that regulate its activity but are independent of the DNA sequence. While there are several factors that can modify DNA to alter gene expression, such as histone remodeling and regulation by small non-coding RNAs, we focus here on the ability of environmental chemicals to reprogram DNA through changes in methylation patterns. DNA methylation takes place at the carbon-5 position of cytosine in CpGdinucleotides due to DNA methyltransferases [38]. Methyl-binding proteins then attach to these sites and subsequently attract other chromatin modifying proteins, with the end result being a silencing of the methylated gene. On the other hand, hypomethylated genes tend to be more accessible to transcriptional machinery and can generate increased and inappropriate gene expression (Fig. 4) [39].

During development, the epigenome cycles through a series of precisely timed methylation changes designed to ensure proper development. The appropriate timing and extraordinary accuracy of methylation in the gametes and following fertilization makes this highly concerted system particularly vulnerable to interference from environmental exposures [40]. The highly orchestrated processes that occur during these critical developmental periods gives rise to concerns about vulnerability during early stages of life. For instance, epigenetic marks such as methylation patterns are laid down during development and are responsible for the programming necessary to transform stem cells into differentiated cells and tissues. The loss and subsequent reestablishment of the epigenetic profile in the developing embryo comprise a critically sensitive period during which the system is particularly vulnerable to environmental influences. Exposures to environmental chemicals, many with endocrine activity, can alter the epigenetic programming of both somatic and germ cells inducing subtle functional changes leading to disease later in life and in future generations.

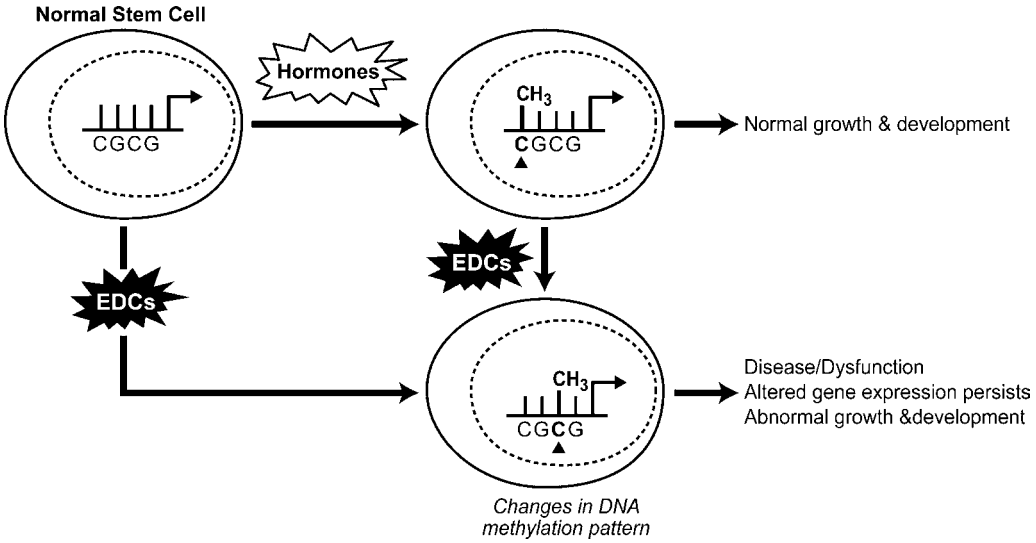

Fig. 4. Model depicting how EDCs can alter methylation patterns and normal epigenetic programming in cells. Alterations in the epigenetic status of somatic cells can lead to disease in developing tissues, whereas changes in the epigenetic programming in stem cells can lead to multi- and transgeneration effects in the offspring.

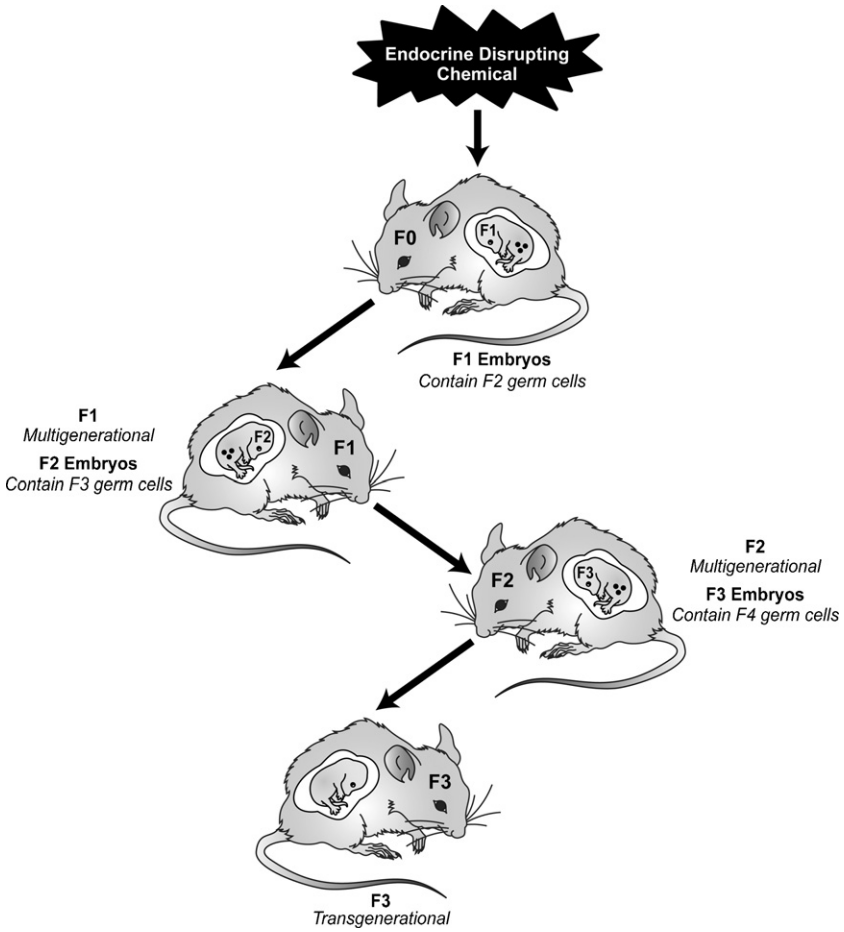

Fig. 5. EDCs many promote epigenetic alterations that influence somatic cells and so the disease status of the individual exposed (F0 generation). In pregnant females, EDC exposure could also cause epigenetic modifications in the next two generations (F1 and F2) through the fetus and its germ line. The effect of such multigenerational exposure in subsequent generations (F3 and beyond) would be considered a transgenerational phenotype.

The exact mechanism whereby environmental chemicals alter the epigenome has not been definitively established. It is known that following fertilization, the DNA methylation pattern in the sperm-derived pronucleus is actively removed (excluding imprinted genes). However, the enzymatic machinery responsible for demethylation is largely unknown. Recently, Activation-Induced cytidine Deaminase (AID) has been found to be highly expressed in primordial germ cells. AID was previously thought to act as a single-stranded DNA deaminase involved in recombination of the immunoglobulin genes during class switching [41]. However, AID activity may also play a role in DNA methylation in primordial germ cells and in the early embryo and in disrupting the action of DNA methytransferase activity during periods of programming. Emerging developments in technology necessary for accurate mapping of the epigenome during developmental periods will undoubtedly lead to a better understanding of the role of endocrine disruptors in disease susceptibility across the lifespan and across generations.

2.5. Transgenerational actions of endocrine disruptors

The majority of environmental factors act on somatic tissues and influence the physiology of the individual exposed [5]. However, in some cases these environmental factors promote a heritable transmission of the disease phenotype through successive generations [5,42,43]. The heritable transmission of this environmentally induced phenotype is referred to as epigenetic transgenerational inheritance. A classic example of a multigenerational phenotype resulting from an environmental chemical involves prenatal exposure to the potent synthetic estrogen diethylstilbestrol (DES), which was prescribed to reduce the risk of pregnancy complications and losses during the 1950s and 1960s [44,45]. Exposure of a gestating female to DES was found to promote an abnormal reproductive tract and gonadal dysfunction in the F1 generation males and females, as well as abnormal female reproductive tract function in the F2 generation [33]. It is interesting that the F1 and F2 generations display different disease phenotypes. Studies are currently underway to determine whether early life exposure to DES promotes multigenerational phenotypes [46,47]. Another example of multigenerational exposure was demonstrated using the anti-androgenic drug, flutamide. Many other chemicals have also been implicated in promoting toxicity for multiple generations, including BPA [48,49], polycyclic hydrocarbons [50,51], cocaine [51], pesticides [52], and phytoestrogens [53–55]. It is important to note that the multigenerational effects mentioned above involving direct exposures and phenotypes are not considered transgenerational because they are not transmitted solely through the germline. Only effects appearing in the F3 generation are considered to be truly transgenerational (Fig. 5) [5].

One of the first studies to demonstrate epigenetic, transgenerational effects of an endocrine disruptor involved the analysis of vinclozolin actions on the male germ line in rats [56]. Vinclozolin is a fungicide commonly used in agriculture that is known for its anti-androgenic endocrine action [57]. In this study Skinner et al. showed that exposing a pregnant rat to vinclozolin or methoxychlor (an estrogeneic pesticide) during embryonic days

8–14 caused defects in spermatogenic capacity, which were transmitted through at least four subsequent generations. Interestingly, the transgenerational phenotypes observed in the animals also included adult onset diseases, such as kidney disease, immune abnormalities, prostate lesions and cancer [56,58]. Subsequently, others have observed changes in behavior and learning capacity following vinclozolin exposure [59–63], including transgenerational changes in mate preferences and anxiety behavior [63]. These transgenerational effects were only seen when the exposure window overlapped with critical developmental processes such as germ cell methylation in the differentiating testis.

3. Selected disease endpoints

3.1. Male reproduction and development

Given the fact that both hormone production and action are regulated in large part by the reproductive tissue, it is not surprising that EDCs contribute to many adverse reproductive health outcomes in developing and adult humans. Epidemiological data has revealed an increase in male reproductive function disorders over the past 50 years, suggesting a correlative relationship with the increasing amounts of EDCs in the environment [64]. In the context of male reproductive health, EDCs have been linked to (1) disrupted reproductive function, displayed as reduced semen quality and infertility; (2) altered fetal development, displayed as urogenital tract abnormalities, including hypospadias and cryptorchidism, and (3) testicular germ cell cancer (TGCC) [1,65]. As previously mentioned, the potential lag between exposure to EDCs and the manifestation of a clinical reproductive disorder is of critical concern. In humans, this period may be years or decades post exposure because sexual maturity and fertility cannot be assessed until the exposed individual has attained a certain age [1].

Skakkebaek et al. [66] have suggested that the incidences of cryptorchidism, hypospadias and poor semen quality are risk factors for one another and that they are all predictive of developing testicular germ cell cancers. This quartet is defined as the testicular dysgenesis syndrome (TDS). They propose that the etiology of TDS lies in the diminished androgen action in fetal developmental periods and has a negative impact on the proper functioning of Sertoli cells (the cells supporting germ cells) and Lydig cells (where androgen synthesis occurs). This hypothesis proposes a strong association between environmental exposures and development of TDS.

Identifying environmental causes of TDS in humans is difficult because developing fetal tissues are inaccessible for examination. Thus, the majority of mechanistic evidence linking EDCs to TDS comes from animal experiments. It is possible to experimentally induce all the elements of TDS, except for germ cell cancer, by exposing pregnant rats to phthalates and other chemicals that block androgen action [67]. This model is referred to as the "phthalate syndrome" model, and it comprises non-descent of testis, malformations of the external genitalia, poor semen quality, and malformations of other sex organs [68]. The causes of phthalate syndrome center on suppression of fetal androgen action, which is the key driver of male reproductive organ development. Phthalates lower levels of testosterone and its derivatives by interfering with the uptake of steroid hormone precursors into fetal Leydig cells where steroid synthesis takes place. The net results are malformations of internal reproductive organs, hypospadias, retained nipples, and feminized anal–genital distance (AGD) [68]. Certain pesticides are able to block the androgen receptor, or interfere with the conversion of testosterone into dihydrotestosterone, thus producing effects similar to phthalate syndrome. Androgen action also is essential for proliferation and development of Sertoli cells, which are necessary for sperm production. Altogether, EDC-mediated disruption of androgen action during fetal development results in reduced fertility later in life [69].

Epidemiological studies have identified an association between chemical exposure (e.g., to phthalates, polychlorinated biphenyls (PCBs), dioxins, and nonpersistent pesticides) and reduced semen quality. In a U.S.-based study, Duty et al. [70] found links between monobutyl phthalate exposure and poor sperm motility and concentrations. A study of dioxin exposure conducted by Mocarelli et al. [71], suggests that timing of exposure has a significant impact on semen quality. This study was based on men exposed to high levels of TCCD as a result of a chemical plant explosion in 1976 in Seveso, Italy. Men exposed prepubertally (1–9 years of age) demonstrated poor semen quality as adults. Interestingly, men exposed between 10–17 and 18–27 years of age showed slightly positive or no differences in semen quality, respectively. Several occupational studies have found associations between pesticide exposure and reduced semen quality [72–78]. In a study on male partners of pregnant women, Swan et al. [79] found elevated odds ratios for poorer semen quality in relation to urinary concentrations of several common pesticides. Meeker et al. [80] also found an inverse relationship between urinary pesticide levels and sperm concentration and motilityin men. While there are clear associations between EDCs and diminished male reproductive health, there is a clear need for further epidemiological studies to identify the classes of chemicals, exposure levels, and the most critical windows of susceptibility important to male reproductive health.

3.2. Female reproduction and development

The ability of EDCs to alter reproductive function and health in females has been clearly demonstrated by the consequences of DES use in pregnant women. The daughters of women given DES while pregnant were shown to have rare cervicovaginal cancers [81,82], decreased fertility and increases in rates of ectopic pregnancy [83], and early menopause [84]. Many of these disorders have been replicated in laboratory animals treated with DES during gestation [44,85–89]. As Newbold points out [87], the lessons learned from 40 years of DES research in humans and animals are that the female fetus is susceptible to environmentally induced reproductive abnormalities, that gonadal organogenesis is sensitive to synthetic hormones and hormone mimics during critical exposure windows, and that reproductive disease may not appear until decades after exposures.

Proper development of ovarian follicles in the fetus is dependent on estrogen exposure during critical periods of development. For instance, mice treated with DES on postnatal day 1–5 develop multioocytic follicles as adults [90]. Therefore, maintaining a homeostatic balance of local and systemic hormones during follicle development is necessary for normal follicle development and germ cell quality [91]. Perturbations in hormone signaling resulting from chemical exposures during developmental periods could contribute to ovarian disorders and declining conception rates in human populations [92]. And while the mechanisms by which EDCs alter follicle development are not fully understood, there is evidence that these chemicals are contributing to increased rates of aneuploidy [93], polycystic ovary syndrome (PCOS) [94,95], premature ovarian failure (POF) [94,95], and altered cyclicity and fecundity [96–100]. For example, studies have shown that prenatal exposure to BPA causes irregular cycles in mice, which is likely due to hypothalamic alterations in the circuitry that controls luteinizing hormone (LH) secretion and ovulation [97,101]. In humans, altered cyclicity has been reported in individuals exposed to organochlorine pesticides. Indeed, cycle irregularities have been noted in women whose mothers were exposed in utero to DES [46].

Uterine fibroids (leiomyomas) are the most common tumor of the female reproductive system [102], occurring in 25–50% of all women. The risk of the development of uterine fibroids increases with age during premenopausal years, but tumors typically regress with the onset of menopause [103]. Obesity, age at menarche and unopposed estrogen signaling have been shown to increase the risks for fibroids [104]. The best characterized animal model for the study of uterine fibroids is the Eker rat. A mutation of the tuberous sclerosis complex 2 (Tsc2) tumor suppressor gene causes females to develop spontaneous uterine fibroids at a high frequency [105]. Studies using this model have shown that exposure to EDCs increases the incidence of fibroids in these animals [106]. Developmental exposure to DES causes rats that are genetically predisposed to uterine tumors to develop even more tumors of a larger size, but fails to induce tumors in wild-type rats. Importantly, DES exposure imparts a hormonal imprint on the developing uterus that causes an increase in estrogen-responsive gene expression [91]. The potential for DES to cause uterine fibroids in humans is less clear. Two studies on DES daughters came to different conclusions. In a study of 2570 women born during the period DES was prescribed, no association was found between prenatal exposure and uterine fibroids [107]. Another study of 1188 women found a significant relationship between DES exposure and uterine fibroids [108]. On analysis of these studies, Baird and Newbold concluded that there was a definitive increase in uterine fibroids in DES daughter and the discrepancies between the studies was due to the differences and sensitivities of the methods used to detect the tumors [108].

In summary, both animal and human studies suggest a role of EDCs in altering female reproductive development. Data from animal experiments show that EDC exposure during critical periods of development, both prenatal and neonatal, can induces functional changes that appear later in life. There are data gaps in understanding the mechanisms by which EDCs carry out their action, but it is clear that to reduce the risk of reproductive disorders we must take action to reduce exposure to these chemicals.

3.3. Obesity and metabolic disorders

There is now compelling evidence linking prenatal exposures to a variety of chemicals with altered developmental programming that may lead to weight gain [22] and metabolic disturbances such as diabetes later in life [109,110]. One well-studied example concerns the effects of maternal tobacco smoking. Babies born to mothers who smoke are typically born with a low birth weight but experience increased risk of obesity, cardiovascular disease and metabolic syndrome later in life, thus some component(s) of tobacco smoke that are transported to the fetus are “obesogens” [111].

Obesogens are functionally defined as chemicals that promote weight gain by acting directly on fat cells (to increase their number or the storage of fat) or indirectly by altering mechanism through which the body regulates appetite and satiety, by altering basal metabolic rate, or by altering energy balance to favor the storage of calories [reviewed in 22, 112]. Many known obesogens are EDCs that can act as direct ligands for nuclear hormone receptors, or affect components in metabolic signaling pathways under hormonal control [22]. Indeed, environmental chemicals such as tributyltin (TBT) and triphenyltin (TPT) are known to stimulate adipogenesis in vitro and in vivo. TBT and TPT are nanomolar affinity ligands for the RXR-PPARγ heterodimer [113,114] and stimulate 3T3-L1 preadipocytes to differentiate into adipocytes [113–115] in a PPARγ-dependent manner [116,117]. The ligand-binding pocket of PPARγ is large and considered to be promiscuous [118]; therefore, it is not surprising that an increasing number of other chemicals with dissimilar structures have been shown to be PPARγ ligands [reviewed in 12]. It is currently unknown how many environmental chemicals activate PPARγ and whether some or all of these will ultimately turn out to be obesogens but there is little doubt that activating PPARγ is an important pathway for adipogenesis and obesity (Fig. 6) [12,22].

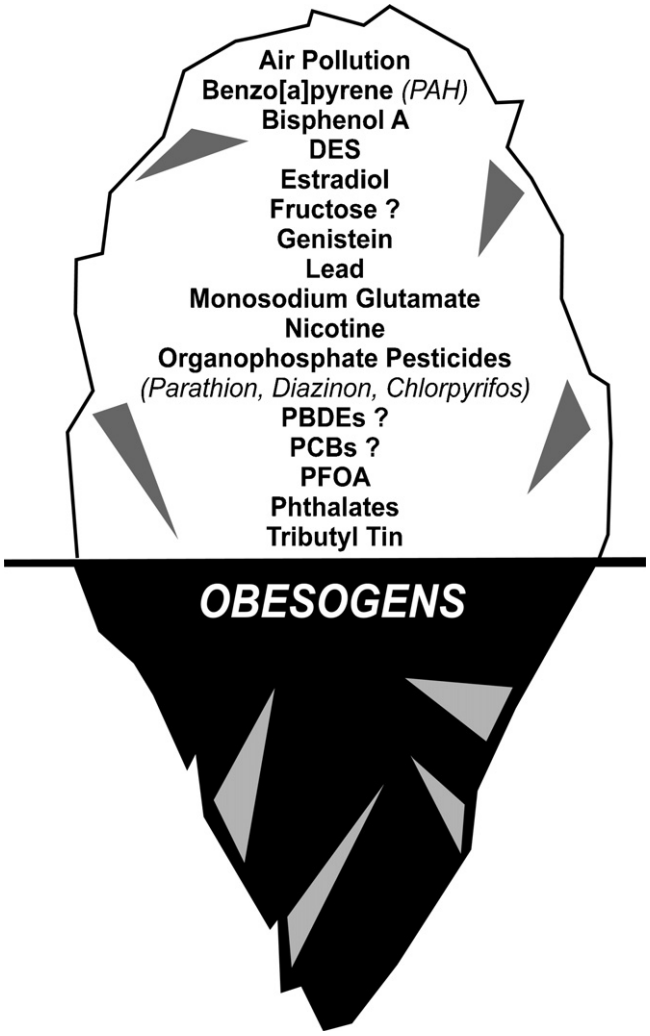

Fig. 6. The iceberg illustration indicates that there is evidence that exposure to certain EDCs during results in obesity in animal models. Only a few chemicals have been thoroughly studied in humans, thus possible that many more chemicals will be found below “the tip of the iceberg” that impact obesity.

Mature adipocytes are generated from multipotent stromal cells (MSCs) found in almost all fetal and adult tissues [119]. MSCs can differentiate into bone, adipose tissue, cartilage, muscle, in vitro and are thought to help maintain these tissues in the adult. Exposure of pregnant mice to TBT or the pharmaceutical obesogen rosiglitazone produced an MSC population that was predisposed to differentiate into adipocytes at the expense of bone [116]. Although the effects of TBT exposure on adults remains unexplored, it is known that rosiglitazone treatment increases weight and fat cell number in humans [120]; therefore, it is likely that TBT has the same effect. Intriguingly, MSCs derived from mice exposed to TBT in utero showed epigenetic alterations in the methylation status of the CpG islands of adipogenic genes such as AP2 and PPARγ which presumably led to the observed increase in the number of preadipocytes in the MSC compartment and in the frequency with which MSCs differentiated into adipocytes upon adipogenic stimulation [116]. Ultimately, it will be quite important to understand

how the setpoint for adipocyte number is programmed in humans and how this can be altered by EDC exposure.

There is growing concern in the scientific community that EDCs may be contributing to the rapid increased rates of diabetes and metabolic syndrome. It is of particular concern that the incidence of both obesity and diabetes are rising rapidly in the young. While there can be no argument that eating calorie-dense, nutrient-poor food in large portions combined with lack of exercise plays an important role, the rapid rise in obesity and diabetes in the young suggests the influence of early life exposures to chemicals may be playing an important role. Indeed, there is a growing body of literature linking exposure to EDCs such as BPA, dioxins, organochlorine and organophosphate pesticides with the incidence of metabolic syndrome and diabetes [109,110,121]. It is know that obesity and diabetes are linked and many of these same chemicals are associated with weight gain/obesity [22,112,122] and diabetes. While the precise metabolic pathways targeted by most of these chemicals are uncertain at present, the data linking EDCs with obesity, metabolic syndrome and diabetes are strong and the number of studies finding positive association is growing. Understanding the molecular mechanisms involved in the role of epigenetics and early life exposures will provide important insights into the etiology of these chronic disorders and should play an important role in designing effective prevention strategies.

4. Conclusion

Humans are exposed to thousands of chemicals during their lifetime, through the air, food, and water. A significant number of these chemicals can be toxic since they can disrupt the endocrine system. Over the past decade, the list of chemicals with endocrine disrupting activity has dramatically increased [123]. Evidence has shown that EDCs compromise the reproductive system, thyroid signaling mechanisms, as well as tissues and organs associated with energy metabolism, glucose control, fat cell development and satiety. Indeed, it is plausible that all endocrine systems are to some degree affected by environmental chemical exposures. Since EDCs activate the same receptors and signaling pathways as hormones and act at low concentrations, they are subject to the same biological regulatory systems as hormones. And since hormones control all aspects of physiology across the lifespan, the same can be expected from EDCs.

Hormones play a critical role in tissue development and the programming of stem cells and tissues during the developmental process. The same can be said for EDCs. The DOHaD paradigm illustrates that many, if not all, diseases have their origin during development. EDCs pose the most risk during the developmental period as they alter programming, which leads to increased susceptibility to disease later in life. Testing for chemicals with endocrine disrupting activity can be challenging as the effects are often subtle (functional changes such as alterations in epigenetic marks, and changes in gene expression), and they can manifest effects later in life, long after the EDC is eliminated from the body. Over the past 40 years, there has been a significant increase in a variety of endocrine-associated diseases including, infertility, premature puberty, ADHD, obesity and diabetes, and endocrine cancers such as prostate, ovarian and breast. It is biologically plausible that EDCs are playing a significant role in these and other diseases.

The notion that EDCs are significantly impacting human health is of great concern. More data is needed to expand the list of tissues affected by EDCs, and more effort is needed to identify and classify the diseases and dysfunctions they are causing in humans and animal models. Nevertheless, current data is sufficient to identify a public health problem that must be addressed. There must be concerted efforts to reduce exposures to EDCs across the lifespan, with particular emphasis in pregnant women and infants. In addition, it is important for scientists to develop biomarkers to measure exposure to EDCs during development periods. These biomarkers could be used to identify windows of susceptibility to EDCs and to develop early therapeutic interventions.

5. Statement

This article may be the work product of an employee or group of employees of the National Institute of Environmental Health Sciences (NIEHS), National Institutes of Health (NIH), however, the statements, opinions or conclusions contained therein do not necessarily represent the statements, opinions or conclusions of NIEHS, NIH or the United States government.

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PREVENTING BREAST CANCER:

AN ANALYSIS OF CANADA'S REGULATORY REGIME FOR CHEMICALS

ELLEN C. SWEENEY

A DISSERTATION SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

GRADUATE PROGRAM IN ENVIRONMENTAL STUDIES
YORK UNIVERSITY
TORONTO, CANADA

DECEMBER, 2013

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Examination Committee members:

1. Dr. Dayna Scott
2. Dr. Miriam Smith
3. Dr. Aryn Martin
4. Dr. Barbara Rahder
5. Dr. Abby Lippman

Abstract

Breast cancer is the most commonly diagnosed cancer in women worldwide. The incidence rates are such that one in nine Canadian women will be diagnosed in her lifetime. While social science research has demonstrated the influence of social, political, economic, and environmental factors on health outcomes, many still emphasize the role of traditional risk factors for breast cancer, such as family history or diet. However, these factors are unable to account for the increased incidence of the disease in industrialized countries. This leads to a call for more attention to the environmental links to breast cancer, including the 'everyday exposures' to toxic substances that we experience in our daily lives, which often include mammary carcinogens and endocrine disrupting chemicals. The *Canadian Environmental Protection Act, 1999* and the federal government's *Chemicals Management Plan* are designed to protect the environment and the entire Canadian population from risks associated with exposure to toxic substances. This dissertation research examines the body of Canadian law, policy and practice which encompasses Canada's regulatory regime for toxic substances. The regime is evaluated from a population health and primary prevention perspective. I asked: are the laws, policies and practices governing the everyday exposures to toxic substances in Canada inherently precautionary? And do they enact a primary prevention approach to women's health?

The primary prevention of environmental health outcomes has not been a strong feature of public health policy and legislation in Canada, despite the efforts of environmental breast cancer activists who advocate for a precautionary approach. This research is steeped in politicized debates as it engages with issues central to women's health, risk and the environment. I examine how the issues are communicated and understood, who the policies are designed to protect, and where the burden of risk is presumed to lie. I consider whether the policies capture the need for prevention and action related to women's health. This research seeks to identify gaps in the law, policy and practice and in doing so, concludes that women's health is not adequately protected from detrimental health outcomes as a result of everyday exposures to toxic substances, including breast cancer.

Dedication

I would like to dedicate this dissertation to my father, Reg Sweeney (1949-2007). Our parents instilled us with a strong work ethic and only ever asked that we try our best. Dad, I know you would be proud and I miss you every day.

Acknowledgements

The Faculty of Environmental Studies at York University is one of the only places where I could have conducted this research. This faculty encourages its students to conduct critical work drawing from and across multiple disciplines which was essential for a project linking women's health, toxic substances, risk, and regulation.

I was very fortunate to have the opportunity to work with my supervisor, Dr. Dayna Scott. She was an invaluable resource with her research and expertise in women's and environmental health. I'm grateful for the thoughtful discussions that helped to shape my work. I would also like to thank my committee members, Professor Liora Salter and Dr. Miriam Smith for their feedback throughout the various stages of the program, including comprehensive examinations, the dissertation proposal when I was wisely advised to narrow the scope of my project, and the draft dissertation.

I would like to thank Dr. Liette Gilbert for her support during my time in the program, particularly in my first year when she was the PhD Program Coordinator and teaching ENVS 8102. Her advice was priceless as I was navigating the first year of the program, and I continued to refer to materials from this course throughout my time at York.

I would like to acknowledge the financial support I received in the form of Ontario Graduate Scholarships (2010-2012) and York University's Provost Dissertation scholarship (2012-2013).

I'm very grateful for my time spent at the National Network on Environments and Women's Health. I would like to thank the NNEWH team for their continually inspiring commitment to advancing women's health research. It was a pleasure working closely with Dayna, Anne Rochon Ford, Jyoti Phartiyal, Jim Brophy, Margaret Keith, Michael Gilbertson, and Bob DeMatteo -- all of whom I admire both personally and professionally.

Many thanks to all of the members of my examining committee including Dayna and Miriam, as well as Dr. Aryn Martin (internal external), Dr. Barbara Rahder (chair and Dean's representative), and the external examiner, Dr. Abby Lippman for a critical and engaging discussion which has given me plenty to think about moving forward.

I'm very thankful for support of good friends, both new and old, near and far. In particular, I would like to my colleagues and members of a writing support group including Amanda Di Battista, Sonja Killoran-McKibbin, Andrew Mark, and Catie Ady-Bell. Thanks for the encouragement, as well as sharp eyes and always thoughtful feedback during our time together.

I would especially like to thank my Mom, Reeta Sweeney whose support and encouragement is invaluable and has never wavered. I would like to thank my brother Mark for his support. I want to wish him congratulations on completing his PhD in History from the University of Waterloo in November this year. I'm so proud! We would have never imagined that we'd both be completing PhDs, but I'm glad that we're also still the same people who grew up in Enfield, Nova Scotia. Finally, I would like to acknowledge the memory of my grandmothers who both taught me important life lessons and passed away during my time at York University, Erma Catherine Sweeney (1923-2009) and Mary Christena Sutherland (1923-2012).

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List of Abbreviations

ACEWH - Atlantic Centre of Excellence for Women's Health
BBP – n-butyl benzyl phthalates
BCCEWH - British Columbia Centre for Excellence in Women's Health
BHA - butylated hydroxyanisole
BHT - butylated hydroxytoluene
BPA - bisphenol A
BFR - brominated flame retardants
CEPA - *Canadian Environmental Protection Act*, R.S.C. 1988
CEPA 1999 - *Canadian Environmental Protection Act*, R.S.C. 1999, c. 33
CESAF - Centre of Excellence for Women's Health - Consortium Université de Montréal
CFCs - chlorofluorocarbons
CMP - *Chemicals Management Plan*
CWHN - Canadian Women's Health Network
DBP - di-n-butyl phthalates
DDT - dichlorodiphenyltrichloroethane
DEA - diethylalomine
DEHP - di(2-ethylhexyl) phthalates
DES - diethylstilbestrol
DIBP - diisobutyl phthalates
DMF - N,N-dimethylformamide
EDC - endocrine disrupting chemical
GBA - gender based-analysis
HCBD - hexachlorobutadiene
IARC - International Agency for Research on Cancer
IICA - International Council of Chemicals Associations
LGBT – Lesbian, Gay, Bisexual, and Transgender
LOAEL - Lowest-Observed-Adverse-Effect-Level
NDMA - N-nitrosodimethylamine
NO(A)EL - No-Observed-(Adverse)-Effect-Level
NNEWH - National Network on Environments and Women's Health
NPE - nonylphenol and its ethoxylates
NTP - National Toxicology Program
OECD - Organisation for Economic Co-operation and Development
PAH - polycyclic aromatic hydrocarbons
PBDE - polybrominated diphenyl ether flame retardants
PCB - polychlorinated biphenyls
PCEWH - Prairie Women's Health Centre of Excellence
PEG - polyethylene glycol compounds
PFC - perfluorinated chemicals
PFOS - perfluorooctane sulfonate
PSL1 - first Priority Substances List

PSL2 - second Priority Substances List
REACH - Registration, Evaluation and Authorization of Chemicals
RoHS - Restriction of the use of certain Hazardous Substances in electrical and electric equipment
RQASF - Réseau Québécois d'Action pour la Santé des Femmes (Quebec Womens' Health Action Network)
Siloxane D4 - octamethylcyclotetrasiloxane
Siloxane D5 - cyclopentasiloxane, decamethyl-
SNAc - Significant New Activity
TSCA - *Toxic Substances Control Act* (US)
TSMP - *Toxic Substances Management Policy*
USEPA - United States Environmental Protection Agency
WHCR - Women and Health Care Reform
WHP - Women and Health Protection

Chapter 1

Introduction

Introduction

Advances in medical and environmental science have created an awareness of the relationship between toxic agents and human health (Somers, 2001). Kroll-Smith and Kelly (2008) utilize the term of "ecological impairment" in their examination of the evolution of the relationship between bodies and environments. They consider the historical progression of bodies in environments, including industrial environments and industrial elements in bodies. The authors focus their attention on the 19th century in order to consider how changing environments were believed to have the capacity to alter or change the body. During this time, environment and disease were linked to landscape, topography and changes in climate (Kroll-Smith and Kelly, 2008: 306-07). However, between 1900 and the early 1960s, there was a shift in ideas about the relationship between the body and environment. The field of medicine became professionalized and the germ theory of disease emerged to surpass the role of landscapes, topographies and climates as explanations for health and sickness. Environmental links to disease were dismissed in this phase in favour of germ theory and explanations focused on microbes entering the body. There was a definite shift away from any attention that might have been paid to the increasingly chemical-laden industrial environments with medical, political and public emphasis placed on personal hygiene and self-regulation (Kroll-Smith and Kelly, 2008).

The third period, beginning in the mid-1960s, marks a historically significant transformation around the causality of disease and health outcomes (Chernomas and Donner, 2004). During this period, environments are viewed as "encroaching upon, invading, and indeed poisoning bodies" through human-made, synthetic environments (Kroll-Smith and Kelly, 2008: 309-10). The increase in disease incidence in western society corresponds with the increased mechanization and industrialization beginning in the late 20th century (Brophy et al., forthcoming). The global production of chemicals increased 400-fold from 1930-2000, and the increase in the production and use of toxic substances occurred largely without assessing the potential risks to the environment and human health (Environment Canada and Health Canada, 2004: 27). There is no agreement regarding pathophysiology for most contemporary toxic exposures in western society, and one cannot simply claim to have a disease when there is not a clear etiological explanation of exposures and symptoms. Under this new paradigm, it is common to acknowledge both the complex changes in the relationship between bodies and environments and the implications these relationships have for clinical medicine (Kroll-Smith and Kelly, 2008: 316). Environments are viewed as possible sources of impairment and it is recognized that some bodies that are exposed may experience health problems as a result. Proponents of this view argue that environments, like bodies, can be injured. However, unlike an impaired body that is unlikely to pose a risk to other bodies, *an impaired environment places bodies in danger* (Kroll-Smith and Kelly, 2008: 316-17, emphasis added).

The period in which industrial elements have permeated bodies has generated new scholarship which posits that "the human is always intermeshed with the more-than-human world" and the "substance of the human is ultimately inseparable from 'the environment'" (Alaimo, 2010: 2). The contemporary risks associated with exposure to toxic substances simultaneously embody Rob Nixon's (2011) concept of "slow violence" and Rachel Carson's (1962) "death-by-indirection." In the risk society, risks are defined as the probability of physical harm occurring as a result of technological processes. The dangers associated with an increase in chemical contaminants include risks to bodies that are both pervasive and cumulative. These risks are unlimited across both space and time, as they cross all territorial borders and have the potential to affect future generations (Beck, 1992). These "landscapes of risk" embody the hazards which become inscribed on women's bodies and may result in detrimental health outcomes (Alaimo, 2010). By viewing environments and bodies not as distinct entities, but rather as intricately linked, this discussion provides a place to situate critical research and analysis on environmental health.

"As the 21st century unfolds we are witnessing important changes in, and vigorous debates about, the ways in which people in the industrialized West understand and experience illness" (Moss and Teghtsoonian, 2008: 3). The fullest definition of environmental health problems includes all of the health hazards found in our living and working conditions, including bacteria and viruses in human waste; animal vectors for infectious diseases; surface-water and groundwater pollution; air pollution; chemical and petroleum product spills and explosions; and disasters such as floods, hurricanes,

landslides, and fires which may be natural or caused by humans (Brown, 2007). To narrow the definition to the understanding reflected in most research and policy on environmental health, environmental health problems may be defined as the "health effects caused by toxic substances in people's immediate or proximate surroundings" (Brown, 2007: 1). Brown (2007: 2) prefers to use the term "environmentally induced diseases" as the causation is linked to environmental factors, but the environmental factors may also interact with genetic predispositions or with some personal behaviours. Concerns around environmentally induced diseases focus on acute and chronic exposures to toxic substances which may be linked to cancer, respiratory diseases, and problems associated with the immune and endocrine systems. These health conditions affect the lives of Canadian residents and have implications for the health care system, social services and the economy (Health Canada, 2010a: 32).

Recognizing the carcinogenic, bioaccumulative and persistent nature of environmental contaminants, there is a call for a shift away from the biomedical model of disease which focuses on the diagnosis and treatment of breast cancer and towards research which focuses on environmental causes and primary prevention. My research will contribute to these debates with an explicit focus on preventive health policy in order to augment disease prevention efforts. The Government of Canada has a "duty to protect the environment, including its biological diversity, and human health, from any of the adverse effects of the use and release of toxic substances, pollutants and wastes" under the Administrative Duties of the *Canadian Environmental Protection Act, 1999*. In this research, I conduct an interpretive policy analysis of Canadian environmental law, policy

and practice which encompasses the regulatory regime for toxic substances. I focus specifically on the *Canadian Environmental Protection Act* and the *Chemicals Management Plan*, and frame the research with a cancer prevention lens.

Situating Breast Cancer: Issues of Risk, Responsibility and Prevention

Toxic exposure has provoked a substantial amount of debate and conflict, policymaking, legislation, public awareness, media attention, and social movement activity. It has also prompted vigorous debate between laypeople and professionals, citizens and government and among professionals themselves (Brown, 2007). My research investigates whether the environmental legislation and policies which are designed to protect the environment and human health capture the need for prevention and action related to protecting women's health in Canada. Breast cancer has been a notable touchstone as the "rising incidence of breast cancer in the decades following World War II paralleled the proliferation of synthetic chemicals" (Gray et al., 2009: 45). Because known attributable risk factors cannot account for the increased incidence of breast cancer, particularly in industrialized countries, it has become necessary to consider environmental links to breast cancer including mammary carcinogens and endocrine disrupting chemicals[1] through everyday exposures to industrial chemicals and toxic substances in consumer products (Schwarzman and Janssen, 2010).

[1] Endocrine disrupting chemicals are natural and human-made substances that can mimic or interfere with the function of hormones in the body (NIEHS, 2013b). Endocrine disrupting chemicals can disrupt the endocrine system in three specific ways: i) by mimicking a natural hormone in the body which may result in a signal stronger than the natural hormone or a signal that occurs at the "wrong" time; ii) by binding to a receptor within a cell and preventing the correct hormone from binding and resulting in abnormal reactions within the body; and iii) by blocking cell receptors which interferes with how normal hormones and receptors function in the body (CCOHS, 2013; Labelle, 2000). Endocrine disrupting chemicals are found in a wide range of substances including polychlorinated biphenyls (PCBs), dioxins, pharmaceuticals including

Breast cancer is a public health issue of concern: it is the most commonly diagnosed cancer in women worldwide (WHO, 2013). Breast cancer rates in Canada are among the highest in the world with similar incidence rates as the United States, northern Europe and Australia. Breast cancer is the most common cancer in Canadian women under age 50, ages 50-69 and over age 70, as well as being the most common cancer-related cause of death for women under 50 (CCS and NCIC, 2007). One in nine women will develop breast cancer in her lifetime and approximately sixty-five Canadian women are diagnosed with breast cancer every day. An estimated 23,800 women in Canada will be diagnosed this year and 5,000 will die as a result (BCSC, 2013a).[2]

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diethylstilbestrol (DES), dichlorodiphenyltrichloroethane (DDT) and other pesticides, and plasticizers including bisphenol A (BPA) (CCOHS, 2013; NIEHS, 2013b). These substances may interfere with the endocrine system in humans and wildlife resulting in adverse developmental, reproductive, neurological and immune effects (NIEHS, 2010, 2013b).

[2] It should be noted that breast cancer can occur in men. However, it is rare with men accounting for less than one percent of diagnosed cases (CCS and NCIC, 2007). An important example of environmental exposures and male cases of breast cancer involves Camp Lejeune which is a Marine Corps Base in North Carolina. Marines and Naval personnel, residents, family members and civilians who lived in military base housing at Camp Lejeune were exposed to contaminated water through the release of volatile organic compounds into the drinking water from 1957 to 1987 (TFTPTF, 2010). Camp Lejeune was officially listed as a Superfund site in 1989 under a federal program by the United States Environmental Protection Agency which addresses uncontrolled hazardous waste sites (USEPA, 2013a). Approximately 750,000 to 1,000,000 people were exposed to trichloroethylene, tetrachloroethylene, benzene, and vinyl chloride which are linked to miscarriage, birth defects, childhood leukemia, and other forms of cancer (Ordonez, 2012; Semper Fi: Always Faithful, 2013). Eighty-one cases of male breast cancer have been linked to exposures at Camp Lejeune as of November 2012 resulting in the single largest cluster of male breast cancer (Partain, 2012). A newly released study by the Agency for Toxic Substances and Disease Registry cites a report from 1985 where levels of trichloroethylene were 18,900 parts per billion in a drinking water well at Camp Lejeune, nearly 4,000 times today's maximum allowable limit of 5 parts per billion (Breed, Waggoner and Biesecker, 2013). Leaders of the House and Senate Veterans Affairs Committees stated that this is "possibly the worst example of water contamination" in the history of the United States (Ordonez, 2012). The House of Representatives approved the *Janey Ensminger Act* which provides health care to affected military personnel and their family members if they lived or worked at least thirty days at Camp Lejeune from 1957 to 1987 and their health condition is listed in the Bill as being associated with exposure to the chemical contaminants (Ordonez and Barrett, 2012). For additional information about this case, refer to the Agency for Toxic Substances Disease Registry (2013); The Few, The Proud, The Forgotten (2012); and Williams (2012).

Most illnesses in western society are viewed through a unique dominant epidemiological paradigm which involves a specific set of beliefs and practices about a disease, its causation and treatment that are embedded in science, government and public life (Brown, 2007: 18). The dominant epidemiological paradigm is directly influenced by the biomedical model of disease which focuses on anatomy and physiology, and causes of disease at the cellular, hormonal and genetic levels (Rosser, 2000). This paradigm is characterized by a "hegemonic outlook on disease that emphasizes individual behavioural factors rather than environmental and social factors as keys to disease prevention" (Brown, 2007: 21). The dominant epidemiological paradigm places the onus of responsibility strictly on the individual and does not acknowledge other determinants of health. The dominant epidemiological paradigm involved in studying breast cancer is

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Another case of groundwater contaminated by trichloroethylene occurred in Shannon, Quebec which is located close to and shares an aquifer with a military base. CFB Valcartier used trichloroethylene as an industrial solvent prior to 1980. The trichloroethylene was then stored in human-made open lagoons to allow the substance to evaporate. However, the trichloroethylene leached through the lagoons and contaminated the aquifer and private wells in Shannon. Base officials became aware of the contamination in 1997, but residents of Shannon were not formally informed at this time by the Department of National Defense. Water samples from a private well in 2000 found that trichloroethylene levels were 200 times higher than acceptable levels. Emergency measures were ordered at this time by Quebec Public Health Officials including drinking only bottled water and showering with open windows (CBC Fifth Estate, 2007; Stephen, 2009). It is suggested that the contamination of the water resulted in an increased incidence of cancer in Shannon with as many as 500 cases (CBC, 2009). A class action lawsuit was filed in 2003 against the Government of Canada and the Department of National Defense representing 3,500 residents of Shannon. The lawsuit claims that the government knew about the exposure of residents to trichloroethylene and that they drank the contaminated water for 22 years (CBC, 2009, 2011a, 2011b). *Spieser v. Canada* began in 2011 and took place over 115 days with 74 witnesses including 23 experts in the fields of hydrogeology, toxicology, epidemiology, and oncology. The Honourable Justice Bernard Godbout found that the plaintiff did not meet the burden of proof regarding causation of health outcomes and did not order punitive damages (Gagné and Mosian, 2012). However, compensatory damages were awarded in the amount of $12,000 per person for the "fears, worries, troubles and nuisances associated with the fact of having lost a source of drinking water in such circumstances" (Gagné and Mosian, 2012). The Quebec City Regional Public Health Agency has recently hired eight international experts to examine the 500 cases of cancer. "The expected rate for brain cancer is one in every 20,000. With a population of 5000[,] Shannon had 20 brain-cancer cases. That's 80 times the normal expected rate" (Séguin, 2013). Residents hope to use the findings of this study when the case goes to the Quebec Court of Appeal in 2014 (CBC, 2013a, Séguin, 2013).

based on a biomedical model of disease and attributes causation to individual-level factors, including diet, exercise, age at first parity, and genetics (Brown, 2007; Nash, 2006). This approach places an emphasis on individual-level approaches to prevention, detection and treatment including changes in lifestyle such as diet, utilization of mammographic technology to detect tumours, and treatment options which include surgery, radiation and chemotherapy. The dominant epidemiological paradigm is utilized by the biomedical community and the mainstream breast cancer movement and it frames breast cancer as a preventable disease by placing the onus of responsibility on the individual in terms of managing personal risk factors and behaviours, and downplaying social, structural, political, economic, and environmental factors that influence the disease (Zavestoski et al., 2004; Orsini, 2007).

There are multiple symbolic meanings associated with women's breasts in Western society, including representations of sexual pleasure and desire, nurturing and motherhood. Women's breasts are also now associated with ideas about danger and risk: the "risk of disease, risk of defeminisation, risk of deformity, [and] risk of death" (Klawiter, 2008a: xx). A special issue of the *Canadian Cancer Statistics* report provides a discussion of the risk factors associated with the development of breast cancer which can be categorized as modifiable or non-modifiable. Non-modifiable characteristics include reproductive and hormonal factors, and heredity associated with a family history of the disease and genetic mutations (CCS and NCIC, 2007; PHAC, 2009, 2012). A woman's lifetime exposure to estrogen is tied to her risk of developing breast cancer. For instance, early menstruation beginning at age eleven or younger and late menopause increases the

number of years the breast tissue is exposed to estrogen. As estrogen levels are lowered during pregnancy and breast feeding, full-term pregnancies before the age of twenty are thought to lower a woman's risk of developing breast cancer, whereas pregnancies after the age of thirty-five or never becoming pregnant are linked to an increased risk (National Cancer Institute, 2011). An estimated five to ten percent of breast cancer diagnoses involve a specific genetic and hereditary component such as the "breast cancer genes," $BRCA_1$ and $BRCA_2$ which were discovered during the 1990s (CCS and NCIC, 2007). Other non-modifiable biological risk factors include high breast density which is associated with a higher risk of breast cancer, and previous breast conditions with biopsies showing abnormal cells (PHAC, 2009, 2012).

Modifiable risk factors associated with the development of breast cancer include lifestyle and behavioural risk factors. The predominant view of cancer prevention focuses "almost exclusively on individual lifestyle changes" (Chernomas and Donner, 2004: 4). This view is promoted by Health Canada, the Public Health Agency of Canada, and mainstream cancer and breast cancer organizations including the Canadian Cancer Society, the Canadian Breast Cancer Foundation, the Breast Cancer Society of Canada, and the Canadian Breast Cancer Network (BCSC, 2013b, 2013c; Canadian Cancer Society, 2008, 2013a; CBCF, 2012a, 2012b; CBCN, 2013a; Health Canada, 2012a, PHAC, 2009, 2012). The "risky behaviours" include using tobacco, consuming alcohol, not engaging in physical activity, exposure to the sun, and a diet high in fat, red meat, sugar, and processed foods. The Canadian Cancer Society and the National Cancer

Institute of Canada (2007: 74) conclude that the best opportunities for primary prevention and reducing the risk of developing breast cancer are

> eating a healthy diet and being physically active throughout life (thereby maintaining a healthy body weight), minimizing alcohol consumption and avoiding nonessential hormones. Regular participation in high quality screening programs will further lower the breast cancer burden by reducing mortality and improving prognosis.

This official narrative rarely concedes that the modifiable "lifestyle" factors account for only a fraction of breast cancer incidence. Even the *Canadian Cancer Statistics* report, which acknowledges this, places the onus of responsibility solely on the individual with its emphasis on personal behaviours (CCS and NCIC, 2007).

There is a long history which focuses on the role of lifestyle and personal behaviours in health outcomes. The individualization of health and illness has resulted in a "responsibilization paradigm" (Orsini, 2007: 349). This ideology places the onus of responsibility on the individual and suggests that the risk factors for health are controllable if one makes the appropriate lifestyle choices. If one does not behave accordingly or if one does and still becomes ill, there are elements of blame placed on the individual. The responsibilization paradigm places the individual at the centre of disease prevention where "cancer prevention is depoliticised and reduced to behaviour modification" (Brophy, 2004: 60).

The mainstay of breast cancer prevention remains early detection and treatment (Shah, 2003: 221). However, measures of detection and prevention are often conflated in the discourse surrounding breast cancer. In the United Kingdom, United States and Canada, public health policy has a very clear policy promoting detection over primary

prevention (Potts, 2004a). McCormick et al. (2003: 550) note that the American Cancer Society and the National Cancer Institute have a long history arguing that "mammography is the best form of prevention." The Canadian Task Force on Preventive Health Care[3] provides recommendations around clinical breast exams and mammography for women aged forty to seventy-four who are at an average risk of developing breast cancer, but do not apply to women who are at an increased risk with a personal history of the disease, known $BRCA_1$ or $BRCA_2$ mutations, prior chest wall radiation, or a history of the disease in a first degree relative. Recommendations are not made for women over seventy-five due to a lack of data (Canadian Task Force on Preventive Health Care, 2013b). Prior to a 2011 update on screening guidelines, the Task Force recommended annual screening with a clinical breast exam and mammography for women aged fifty to sixty-nine. Women aged forty to forty-nine were encouraged to receive counselling in order to make their decision around the potential benefits and risks associated with mammographic screening and the age at which they wish to begin testing (Shah, 2003: 221).

> Recent evidence has shown that there is sufficient evidence to exclude the routine teaching of breast self examination (BSE) for the periodic health examination of women aged 40 to 69, and there is insufficient evidence to evaluate its effectiveness in women younger than 40 and older than 70 (Shah, 2003: 222).

[3] The Canadian Task Force on Preventive Health Care is an independent body which consists of fourteen primary care and prevention experts who promote the need for evidence-informed preventive activities in primary care in Canada. The Task Force develops and disseminates practice guidelines for primary and preventive care based on systematic analyses of scientific evidence (Canadian Task Force on Preventive Health Care, 2013a).

The most recent screening guidelines from 2011 recommend routine screening with mammography every two to three years for women aged fifty to sixty-nine and aged seventy to seventy-four. Women aged forty to forty-nine are no longer recommended to engage with routine mammographic screening (Canadian Task Force on Preventive Health Care, 2013b). This new recommendation[4] is based on evidence of false positive test results being higher in this age group which may have "undesirable consequences" and "lead to further investigation, including other unnecessary procedures such as breast removal" (Canadian Task Force on Preventive Health Care, 2013a, 2013b). Breast cancer organizations promote mammography as part of "preventive health care" (CBCF, 2012c). The Breast Cancer Society of Canada (2013a) promotes early detection as a means of prevention, including breast self-exams, clinical breast exams and mammography. However, as advocates note, once a tumour has been detected, prevention has ultimately failed.

The established risk factors such as a family history account for less than half of diagnosed cases (Gray, 2010; Parkin et al., 2011). Breast cancer is a multifactorial disease caused by a combination of hormonal, genetic, lifestyle, and environmental factors (Gray, 2010). It is argued that a truly primary prevention focused approach would involve attempting to prevent the disease before it develops. Primary prevention may be broadly defined as "the protection of health by personal and community-wide efforts…[which] consist of measures aimed at preventing the inception of a pathological

[4] The Canadian Task Force on Preventive Health Care will provide an updated report of recommendations around prevention and screening practices within five years of the 2011 guidelines (Canadian Task Force on Preventive Health Care, 2013b).

process or the occurrence of disease" (Tomatis and Huff, 2001: 458). A primary prevention approach to a multi-factorial disease such as breast cancer would "aim to reduce and eliminate as far as possible, human exposures to all substances or agents that are known to be, or suspected of being, implicated in the disease process" (UK Working Group on the Primary Prevention of Breast Cancer, 2005: 10). A primary prevention focus towards environmental health outcomes as a result of exposure to toxic substances has been historically underrepresented in public health policy and legislation.

A woman's risk of developing breast cancer may be increased by exposure to mammary carcinogens and exogenous estrogenic compounds. Brophy et al. (2012: 2) describe the endocrine disruptor theory which contends that "the timing of exposure is important to varying susceptibility, particularly during critical periods of breast development when breast tissue is less differentiated, but also predicts that effects may occur at low doses." Exogenous exposures may include use of hormone replacement therapy and oral contraceptives, but also includes exposure to environmental contaminants. Exposure to endocrine disrupting chemicals may play a particular role during key periods of development or "windows of susceptibility" including the prenatal period, childhood, puberty, menstruation, pregnancy, and menopause (Birnbaum, 2009; Brophy et al., 2012; Cooper et al., 2000; Diamanti et al., 2009; Gray, 2010; Schug et al., 2011; and Schwarzman and Janssen, 2010).[5]

[5] It should also be noted that the endocrine disrupter discourse contains aspects of heteronormativity. "It seems that the horror associated with the theory of 'feminisation' as a manifestation of underlying endocrine disruption must be tied to its potential for completely disrupting the 'heterosexual matrix'" (Scott, 2009a). Di Chiro (2010: 201) explores how heteronormativity is found within the anti-toxics discourse and rhetoric. Exposure to toxic substances results in undermining or distorting the "natural" including biologies, ecologies, bodies, and reproductive processes. She suggests that this has resulted in a

In the United States, the President's Cancer Panel produced a report in 2009 that calls for reducing the risk of developing cancer associated with the widespread and ubiquitous exposure to toxic substances. The Panel was "particularly concerned to find that the true burden of environmentally induced cancer has been grossly underestimated" (Daghofer, 2010; Reuben, 2010: 5). Director of the Science and Environmental Health Network, Dr. Ted Schettler described the report as an "integrated and comprehensive critique" and suggested that the Panel "underscored that regulatory agencies should reduce exposures even when absolute proof of harm was unavailable," drawing on the precautionary principle (Cone, 2010).

The Interagency Breast Cancer and Environmental Research Coordinating Committee[6] published a report in 2013 that calls for making prevention the key to reducing the burden of breast cancer. This report recognizes environmental contaminants, as well traditional risk factors for breast cancer including lifestyle and behavioural factors and other social determinants of health (IBCERCC, 2013). Jeanne Rizzo, co-chair of the Committee and President and CEO of the Breast Cancer Fund, states that the report demonstrates that research and programs "focused on preventing breast cancer need as much attention as treatment and a cure" (Goldman, 2013). Rizzo notes that

> [w]e're extending life with breast cancer, making it a chronic disease, but we're not preventing it. We have to take a look at early life exposures, in utero,

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"sex panic" in which heterosexist and queerphobic arguments reinforce "what and who are constructed as normal and natural" (Di Chiro, 2010: 202). For an in-depth exploration of queer ecologies, refer to *Queer Ecologies: Sex, Nature, Politics, Desire*, edited by Mortimer-Sandilands and Erickson (2010).

[6] The Interagency Breast Cancer and Environmental Research Coordinating Committee was established by the United States Secretary of Health and Human Services after Congress passed the *Breast Cancer and Environmental Research Act* in October 2008. The Committee consisted of federal and non-federal representatives (NIEHS, 2013a).

> childhood, puberty, pregnancy and lactation. Those are the periods when you get set up for breast cancer. How does a pregnant woman protect her child? How do we create policy so that she doesn't have to be a toxicologist when she goes shopping? (Grady, 2013).

The Committee found that identifying and mitigating the environmental causes of breast cancer is the key to reducing the number of new cases and recommends a breast cancer prevention strategy to prioritize and increase government funding in breast cancer prevention (Forman, 2013; IBCERCC, 2013; Rizzo, 2013). "Prevention requires we close the knowledge-to-action gap and translate science into preventive public health actions that can impact breast cancer incidence in the future" (Rizzo, 2013).

Canadian Environmental Law, Policy and Practice

Health Canada and Environment Canada are jointly responsible for the risk assessment and management associated with toxic substances. The first *Canadian Environmental Protection Act* in 1988 included two broad categories of substances. The first was the 28,000 "existing substances" that were manufactured, imported or in commercial use in Canada between January 1, 1984 and December 31, 1986. The majority of these substances had not been evaluated for potential detrimental effects on human health and the environment and were placed on the so-called Domestic Substances List. The second category included substances which were new to Canadian society and commerce and were not part of the Domestic Substances List. It was required that an assessment be conducted on all new substances for their potential impact on human health and the environment before their introduction to the Canadian market. One of the guiding principles of the original *Canadian Environmental Protection Act, 1988* was the "management of pollution" (House of Commons Standing Committee on Environment

and Sustainable Development, 1995). However, there was a shift in focus in the *Canadian Environmental Protection Act, 1999* in which "pollution prevention" became the cornerstone in order to achieve the highest level of environmental quality for the health of Canadian citizens (Environment Canada, 2010a). Section 73 of the revised Act required that all existing substances on the Domestic Substances List be categorized according to which substances presented the greatest potential for exposure for individuals in Canada, and which were considered persistent, bioaccumulative and 'inherently toxic' to human beings or nonhuman organisms. The categorization of the Domestic Substances List was completed between 2000 and 2006 and the results determined that 4,300 of the 23,000 substances examined were classified as priorities for further action. The newly implemented *Chemicals Management Plan* identified five hundred chemicals classified as the highest priorities for immediate action (Health Canada, 2010a: 33). The *Chemicals Management Plan* is designed to assess and manage the risk of all chemical substances categorized as potentially harmful to human health or the environment under the *Canadian Environmental Protection Act* by 2020.

Analyzing the *Canadian Environmental Protection Act* and the *Chemicals Management Plan* from a primary prevention perspective has allowed me to tie together my interests in women's health, risk and disease prevention. It is suggested that the traditional focus of Canadian health policy has been on health care policy with an emphasis on the treatment of diseases and injuries, rather than on disease prevention. As others have noted, "[t]here is clear recognition that we need to move from a system focused predominantly on health care to one more oriented to improved health status"

(Miller Chenier, 2002: 13). My research is aligned with the argument that comprehensive preventive health policy has the potential to contribute to efforts in disease control and population health outcomes.

Research Design and Methodology

This research utilizes a population health approach framing breast cancer with a primary prevention perspective and as a disease influenced by social conditions (Chernomas and Donner, 2004). The nature of critical analysis of social problems requires an interdisciplinary approach and I draw upon theory and methods utilized in sociology and social anthropology, health, gender studies, and environmental studies. Policy analysis may be considered to be an "applied social science discipline which uses multiple methods of inquiry and arguments to produce and transform policy-relevant information that may be utilized in political settings to resolve policy problems" (Dunn, 1981: 35; Fischer, 2003: 1).

The primary data sources for this research are documents related to Canada's regulatory regime for toxic substances. I draw upon government publications, grey literature and media coverage in analyzing environmental law, policy and practice with a particular focus on the *Canadian Environmental Protection Act* and the *Chemicals Management Plan.* My goal in this research is to determine not only what policies exist, but also to examine how the issues are communicated and understood, who the policies are designed to protect, and where the burden for assuming risk is presumed to lie. I question whether the policies capture the need for prevention and action related to protecting women's health, whether the precautionary principle is implemented, and if

they enact primary prevention in approaches to women's health. I explore whether issues of sex and gender are accounted for in the legislation and policies which is of particular relevance in issues related to women's health outcomes. I investigate issues which are contested in nature including environmental links to disease and the debate surrounding exposure-based approaches and hazard-based approaches to risk assessment. I also examine the shifting and contested concept of toxicity which I follow from its inception to its current form under the regulatory regime. During the research process, I seek to identify gaps in the law, policy and practice which do not adequately protect women's health.

The sources of qualitative data for this research include documents from the Canadian federal government; environmental, women's health, cancer, and breast cancer organizations including mainstream organizations and members of the environmental breast cancer social movement; and relevant media coverage. The data sources include:

Government Publications: Website searches include all documents related to breast cancer and all other relevant documents from Environment Canada, Health Canada, the Government of Canada and the *Canada Gazette* (1988-2012) related to the *Canadian Environmental Protection Act* and *Chemicals Management Plan.* Additional documents related to environmental health, cancer, breast cancer, human biomonitoring, public health policy, health impact assessments, environmental assessments and health, toxic substances, environmental contaminants, exposure and human health, as well as initiatives such as the Canadian Partnership for Tomorrow Project (a pan-Canadian project to learn more about causes of cancer and other chronic diseases) and Cancer 2020 (an action plan for cancer prevention and detection) are included. Any documents that could not be obtained directly from government websites, archives, university or public libraries were requested in electronic format or hard copy.

Grey Literature: Grey literature searches include websites of organizations including but not limited to the Atlantic Centre of Excellence for Women's Health, Breast Cancer Action, Breast Cancer Action Montreal, the Breast Cancer Society of Canada, the Breast Cancer Fund, the British Columbia Centre of Excellence for Women's Health, the Canadian Breast Cancer Foundation, the Canadian Breast Cancer Network, the Canadian

Cancer Society, the Canadian Environmental Network, the Canadian Environmental Law Association, the Canadian Network for Human Health and the Environment, the Canadian Women's Health Network, the Centre for Environmental Health Equity, the David Suzuki Foundation, Ecojustice, Environmental Defence, the Environmental Working Group, FemmeToxic, the National Network on Environments and Women's Health, the Prairie Women's Health Centre of Excellence, Prevent Cancer Now, the Quebec Women's Health Action Network, Women and Health Care Reform, Women and Health Protection, and the Women's Healthy Environments Network. Any documents that could not be obtained directly from websites of organizations were requested in electronic format or hard copy. Search terms included but were not limited to: [Canadian Environmental Protection Act], [CEPA], [Chemicals Management Plan], [CMP], [environment and health], [environment], [toxic], [toxic substances], [breast cancer], [breast cancer and environment], [disease prevention], and [prevention].

Media Coverage: Media coverage for this research includes coverage from 1988-2012 in Canadian national newspaper *The Globe and Mail* and the *CBC News Archives* using York University newspaper search engines (Canadian Newsstand and Factiva). Search terms included but were not limited to: [Canadian Environmental Protection Act], [CEPA], [Chemicals Management Plan], [CMP], [environment and health], [breast cancer], [environment and breast cancer], [toxic], and [toxic substances]. Other relevant news media coverage emerged from the *Toronto Star*, *CTV News*, the *Huffington Post*, the *New York Times*, as well as other news sources up to July 2013.

These policy documents were examined and analyzed through multiple readings of the texts and interpretive policy analysis. Interpretive approaches to policy analysis consider both *what* specific policies mean and *how* the policies work by exploring the processes through which meanings of policy are communicated and questioning who the intended audiences are. This process also questions what context-specific meanings are embedded in relevant policy documents which include symbolic language, objects and actions (Salter, forthcoming; Yanow, 1996, 2000: 8).

In conducting interpretive policy analysis, it is necessary to identify groups of policy documents and stakeholders in order to determine how a policy and policy process are understood. A question of central importance and consideration is how the policy issue is being framed by the various parties to the debate. Policy frames may be

expressed through language in order to shape perceptions and understandings. In this case, frames are used to create a framework in which to interpret policy-related documents. Frames may be used to direct attention towards some elements while also specifically diverting attention from other elements (Yanow, 2000: 11-12). It is necessary to "map the 'architecture' of debate relative to the policy issue under investigation, by identifying the language and its entailments (understandings, actions, meanings) used by different communities in their framing of the issue" (Yanow, 2000: 12-13). This process is of particular relevance in order to understand the varying perspectives involved in this research. Different groups of policy actors frame the issues in distinct ways related to issues of risk, exposure to toxic substances, and health outcomes including breast cancer.

My method involves four specific steps in conducting interpretive policy analysis. The first step involves identifying the policy documents that hold significant meaning for policy-relevant actors, stakeholders and interpretive communities for a given policy issue. These documents include but were not limited to the *Environmental Contaminants Act,* the Lalonde Report, the *Canadian Environmental Protection Act, 1988* and *1999* and its review processes, the *Chemicals Management Plan* and other documents related to public health, breast cancer, risk, and toxic substances. The second step involves identifying the communities relevant to the policy issue that create or interpret the policy documents and meanings. The relevant communities in this research include but are not limited to the Government of Canada, Environment Canada, Health Canada, stakeholders in the review process of the *Canadian Environmental Protection Act*, and women's health, cancer and breast cancer organizations. It is possible to have multiple interpretive communities and

multiple interpretations of policy documents and the first two steps may be conducted concurrently as the policy documents and interpretive communities are intricately linked and each step leads back to each other (Yanow, 2000: 20). The third step in this process in continuing to analyze the policy documents involves the identification of the discourses utilized by the various interpretive communities in order to determine the meanings that are considered to be important. Finally, the fourth step involves identifying the meanings that are in conflict between or among interpretive communities and their conceptual sources. This process has the potential to demonstrate the implications of different meanings and interpretations for policy formulation and outcomes (Fischer, 2003; Yanow, 2000: 20). Building on these steps, additional questions that I asked throughout the interpretive analysis process include:

- Who are the subjects and what are the objects? What are the relationships between them?
- What information is present in the documents? What information is absent in the documents?
- Who made the decisions about what information is included or excluded in the documents?
- Who produced the documents? What is the nature of their role and relationship in the context of the document and the broader policy context?
- How was the document produced? What are the processes and who are the people involved?
- How is the information related to the broader policy context? (Ginger, 2006: 346-47).

The process and questions involved in conducting interpretive policy analysis allows for engaging with the policy documents in order to determine whether Canadian law, policy

and practice is inhekrently precautionary and enacts a primary prevention approach to women's health.

Overview of the Dissertation

This research will provide important policy lessons in its examination of Canadian law, policy and practice, with an explicit focus on primary prevention which is supported by current research linking breast cancer and toxic substances. The research results will be of interest to those studying environmental health, as well as to the policy sector and non-governmental organizations. It contributes to the larger body of breast cancer research with analysis and discussion around the issues of gender, risk and precaution by examining the impact of policy on disease prevention and its implication for the health of women across Canada.

This chapter provided an introduction to the overall context within which the research is situated including issues of risk, responsibility and precaution related to breast cancer. Chapter two provides a literature review which draws upon concepts, methods and theories which are situated in interdisciplinary but related fields including environmental studies, sociology, medical anthropology, health, and gender studies. It provides the theoretical underpinning and framework for this dissertation research in three substantive areas including i) sex- and gender-based analysis, ii) social movement theory and health social movements, and iii) risk and the risk society. Sex- and gender-based analysis has emerged as an important methodology in conducting health research. This chapter provides an overview of sex- and gender-based analysis and this lens is applied throughout the dissertation research. The chapter then introduces traditional

social movement theory in order to position more recent health social movement theory and its relationship to the multi-faceted and diverse breast cancer social movement. The history of breast cancer includes two specific disease regimes -- the regime of medicalization which began in the 1900s and the regime of biomedicalization which emerged in the 1970s and 1980s -- in which the disease is medically managed in individual bodies and publicly administered across populations. The role of biosociality played a significant role in the formation of shared experiences among patients and the creation of a breast cancer social movement through engagement with the practices of science, public health and medicine which enabled the formation of shared experiences among patients and the creation of a breast cancer social movement (Klawiter, 2008).[7] The nature of breast cancer calls for examining questions and constructions of risk related to the development of the disease and everyday exposures to toxic substances. These questions of risk are engaged with by exploring Beck's (1992) theory of the risk society which views contemporary risks as unique hazards which are created and managed through social, cultural and political factors. The risk society perspective is augmented with considerations from environmental justice literature in order to provide the overarching framework for the dissertation as it connects issues of risk, toxic substances and environmental health.

Chapter three offers an overview of the evolution of legislation and public health policy which were designed to protect Canadian citizens from exposure to toxic

[7] While it would have been interesting to explore the way that the regulatory regime, perhaps even its failures, have enabled the formation of the breast social movement and the environmental breast cancer movement specifically, it is beyond the scope of this dissertation.

substances. It draws upon legislation, government publications and grey literature in order to provide a descriptive history of Canadian policy from the 1970s when environmental issues were emerging as widespread concerns to the present. The chapter begins with the influential Lalonde Report, *A New Perspective on the Health of Canadians* which provided a new approach for addressing health outcomes. It then presents the first environmental legislation, the *Environmental Contaminants Act* and the introduction of the *Canadian Environmental Protection Act, 1988.* The revised *Canadian Environmental Protection Act* was implemented in 1999. The Act was promoted as reflecting a shift in the regulatory approach from pollution management to pollution prevention and it is still the primary piece of legislation which governs environmental protection in Canada. Finally, the chapter presents the *Chemicals Management Plan* which was introduced in 2006 and is the most recent tool for the assessment and management of risks associated with toxic substances. The review of environmental health policy presented in this chapter is necessary in order for the more in-depth and critical analysis which follows in chapters four and five.

Chapters four and five examine the relationship between theory and practice in Canadian law, policy and practice and the potential for protecting women's health. Chapter four examines the history of the concept of "toxicity" in Canadian legislation, how it has evolved and its contested nature. The assessment of toxicity is grounded in the current risk assessment processes which are based in toxicology, and rely on exposure estimates and an inherent assumption of threshold effects. The chapter provides a brief overview of the siloxane D5 case which raises important questions about the regulation

of toxic substances, the contested nature of risk and toxicity, and the influence of socioeconomic interests. It then introduces the concept of the precautionary principle, along with a growing concern about the effects of endocrine disrupting chemicals, and questions whether the precautionary principle is implemented in risk management processes. The chapter concludes by examining the debate between exposure-based and hazard-based risk assessments which is central to the evaluation of whether Canadian law, policy and practice is enacting a primary prevention approach related to women's health.

Chapter five draws upon government publications, grey literature and media coverage in order to explore questions and tensions around risk, precaution and prevention. It begins with a discussion of the role and implementation of sex- and gender-based analysis in Canadian health policy. The chapter then poses a series of questions in order to explore issues of risk and responsibility. Where is the burden of risk in preventing health outcomes presumed to lie? The responsibilization trend and the practices of precautionary consumption both fit comfortably into the dominant epidemiological paradigm of breast cancer. Both place the onus of responsibility for risk and disease prevention on the individual. These dynamics are explored in-depth. Who is at risk and who are the policies designed to protect? The *Canadian Environmental Protection Act, 1999* does not specifically address any populations of concern. While children are identified as a population of concern by Health Canada, women are not viewed as an at-risk or susceptible population of concern under the legislation and policy. While bisphenol A (BPA) has been regulated in baby bottles in Canada, this regulation is

explored and critiqued. Occupational exposures to toxic substances including BPA are considered, as well as the challenges related to issues of accountability and compensation. It examines the messaging and campaigns around breast cancer by women's health and cancer organizations including the mainstream Canadian organizations and members of the environmental breast cancer movement. It concludes by discussing the impact of broad federal funding cuts on citizen participation in legislation and policy, and organizations which conducted critical, feminist research, policy and advocacy work.

Finally, chapter six calls for a paradigm shift from a reactionary to a preventative approach to health policy. It concludes that the Canadian regulatory regime is not truly precautionary and does not enact a primary prevention approach. The chapter addresses the specific gaps in the law, policy and practice that would need to be addressed in order to truly protect women's health from detrimental health outcomes such as breast cancer.

Chapter 2

Literature Review: Situating Interdisciplinary Research

Introduction[8]

The nature of environmental health research is necessarily interdisciplinary. As Moss and Teghtsoonian (2008: 3) suggest, "we are witnessing important changes in, and vigorous debates about, the ways in which people in the industrialized West understand and experience illness" in the twenty-first century. In order to understand the complex issues involved in this field, it is necessary to draw upon concepts, methods and theories situated in related literatures including environmental studies, sociology, medical anthropology, health, and gender studies. This chapter will provide the theoretical underpinning and framework for the research. The chapter begins with an introduction to sex- and gender-based analysis and the importance of applying this lens in health research and policy analysis which is reflected throughout the dissertation. The chapter then introduces traditional social movement theory in order to situate the emergence of contemporary health social movements with a specific focus on breast cancer. It investigates the history of breast cancer as a disease and the emergence of the distinct factions of the breast cancer social movement which have different understandings and engagements with issues related to gender, race, class, and sexuality, and varying relationships to science, biomedicine and cause-related marketing. Finally, the chapter

[8] Please note that sections of this chapter were previously published and are reproduced here in revised form with slight modifications. See Sweeney, E. (2012a). "Tracing the Role of Gender in the History of Breast Cancer Social Movements." *Women's Health and Urban Life,* 11(1); and Killoran-McKibbin, S. and E. Sweeney. "Selling Pink: Feminizing the Non-Profit Industrial Complex Through Ribbons and LemonAid" which is under review in a Women's Studies journal.

explores Beck's (1992) theory of the risk society which provides an overarching framework for the dissertation by engaging with issues of risk, exposure to toxic substances, environmental health, and the risk a woman has of developing breast cancer. In this chapter, there is a call for a paradigm shift from the dominant epidemiological paradigm of breast cancer to the promotion of primary prevention within public health, regulation and policy which is grounded in the environmental breast cancer movement.

Sex- and Gender-Based Analysis

Greaves (2009) notes that gender was first introduced into health research by social philosophers and social scientists. Since then it has become an important consideration in health research, policy, programming, and service development, particularly as the health determinants model gains more widespread acceptance and support, and gender has been identified as a key determinant.[9] The analysis of sex and gender in health research has emerged as an increasingly important methodology which

[9] The history of health determinants in Canada influenced both the direction of research about population health and the development of government policies designed to improve health. The Lalonde Report which is discussed in detail in Chapter 3 is cited as the first stage of health promotion in Canada and was founded upon the health field concept (Glouberman and Millar, 2003; Lalonde, 1974). Different health organizations and researchers identify various determinants which have a direct impact on the health status of a given population (Benoit and Shumka, 2009). Health Canada first identified nine determinants of health in 1994 as part of a focus on population health and in response to the critique of health promotion strategies. The original nine determinants included income and social status; social support networks; education; employment and working conditions; physical environments; biology and genetic endowment; personal health practices and coping skills; healthy child development; and health services (Health Canada, 2002). The Public Health Agency of Canada (2011) utilizes twelve determinants of health including income and social status; social support networks; education and literacy; employment/working conditions; social environments; physical environments; personal health practices and coping skills; healthy child development; biological and genetic endowment; health services; gender; and culture. The World Health Organization (2011) promotes ten health determinants including income and social status; social support networks; education; economic environment; social environment; physical environment; personal individual characteristics and behaviour; genetics; health services; and gender. In Dennis Raphael's most recent work, he offers fourteen determinants including income and income distribution; education; unemployment and job security; employment and working conditions; early childhood development; food insecurity; social exclusion; social safety networks; health services; Aboriginal status; gender; race; and disability (Raphael and Mikkohen, 2010).

necessitates the consideration of impacts on both men and women, as well as identifying the shortcomings which emerge as a result. The "integration of a sex- and gender-based analysis makes for better science and more inclusive policies" (Lewis, 2011: 5).

The foundation of sex- and gender-based analysis is the understanding that both biology *and* social experiences, and thus sex *and* gender, impact the health status of Canadian citizens. In order to conduct thoughtful and effective health research, it is necessary to clarify the concepts behind sex and gender. Sex typically refers to biological and genetic characteristics which are manifested in one's anatomy, physiology and hormones. Sex includes the "specific capacities of our bodies, and affects the propensity and trajectory of diseases and health conditions" (Greaves, 2009: 3). While sex plays an important role in reproductive health, there are also important considerations in terms of male and female bodies differing in their susceptibility to disease and differing in reactions to substances including alcohol, tobacco, over-the-counter, prescription or illegal drugs because of differences in metabolism, blood chemistry and body fat composition (Batt, 2007; Clow et al., 2009). For example, women may be at higher risk for health issues related to exposure to environmental contaminants which tend to concentrate in body fat and are often related to estrogen receptors, and women tend to have a higher ratio of body fat and estrogen levels than men (Assembly of First Nations Environmental Stewardship Unit, 2009; Clow et al., 2009; Nickerson, 2006; Women's College Hospital, 2013). It is important to ask questions about levels of susceptibility, body size or sex-linked differences which raises additional issues related to sex-specific variations in disease, health and illness (Clow et al., 2009: 11).

Gender should not be confused or conflated with sex as it is a social construct that "extends beyond the boundaries of biologically defined categories of sex" (Benoit and Shumka, 2009: 7). Gender includes the social, cultural and economic factors that influence the socially constructed roles and relationships, personality traits, attitudes, behaviours, values, and influence that a particular society assigns to women, men and other gender groups such as transgendered and two spirited persons (Clow et al., 2009: 11; Greaves, 2009: 3). The consideration of gender in health research is especially critical as it can "determine different exposures to certain risks, different treatment-seeking patterns, or differential impacts of social and economic determinants of health" (Hankivsky, 2007a: 155). Matters related to gender are relevant in every society and affect every population and individual. As sex has been treated as having two distinct categories of male or female, traditionally gender has also been treated in this manner in categories of masculinity or femininity. However, this binary is inadequate and does not account for a continuum of characteristics and behaviours. There are also people who do not identify as male or female or reject those categories entirely (Clow et al., 2009: 12).

Clow et al. (2009: 12-14) and Johnson et al. (2009) offer four specific dimensions of gender including gender identity, gender roles, gender relations, and institutionalized gender which may have an impact on the health outcomes of a given population.

- *Gender identity* involves one's sense of being a "woman" or a "man" and is developed within the prescriptions related to the "appropriate expression" of gender for the biological sex (Clow et al., 2009; Johnson et al., 2009). It should be noted that in some instances or in some cultures, gender identity does not fall into dichotomous categories (Benoit and Shumka, 2009);

- *Gender roles* include the ways in which gender identities are expressed and the behavioural norms within societies which influence individuals' actions,

expectations and experiences in their daily lives (Clow et al., 2009: 12; Johnson et al., 2009). Gender norms may shape the illness experience, as well as what health care issues are researched, what health care services are available, and the quality of patient care (Benoit and Shumka, 2009: 7);

- *Gender relations* involve interactions and how people are treated based on their ascribed gender. For instance, there is a stereotype that women need to be "protected" and this may affect the ways in which women experience illness and approaches to treatment[10] (Clow et al., 2009: 13; Johnson et al., 2009); and

- *Institutionalized gender* involves the experiences, roles and relationships which are framed by social institutions such as the media, education, legal, and health care systems, and religious and political establishments. These institutions influence the social norms that "define, reproduce and often justify different expectations and opportunities" for women, men, girls, and boys, such as "social and family roles, job segregation, job limitations, dress codes, health practices, and differential access to resources such as money, food or political power" (Clow et al., 2009: 13; Johnson et al., 2009).

Clow et al. (2009) add the concepts of equity and diversity to sex and gender as key considerations in sex- and gender-based analysis. Equity refers to the inequalities and gender oppression that may result in detrimental health outcomes. Diversity concerns recognize that experiences with gender identity, gender roles, gender relations, and institutionalized gender are specific,

> particular to a certain time and place, and social, cultural, economic and political situation...and because gender differences and inequalities in a particular place combine with the effects of other forms of social division such as class and ethnicity, not all women or all men experience gender-related health problems or issues in the same way (Clow et al., 2009: 14).

The four core concepts of sex, gender, equity, and diversity create a framework for exploring and understanding experiences of health and illness, and evaluating the extent

[10] For an example of gender relations and health care, refer to the section on *The History of Breast Cancer and Disease Regimes* in this chapter for a discussion linking breast cancer and hysteria, as well as issues of informed consent with male physicians and husbands making treatment decisions on behalf of breast cancer patients.

to which a society's responses are "equal, fair, effective and efficient" (Clow et al., 2009: 16; Greaves, 2009).

Sex- and gender-based analysis involves more than understanding the differences or similarities *between* women and men, but also examines the differences *among* groups of women and men (Greaves 2009). A sex- and gender-based analysis also considers other determinants of health and explores how diversity within and between subgroups of women and men may affect health outcomes. Thus, the intersection of sex and gender are considered alongside issues of age, race, ethnicity, culture, geographic location, sexual orientation, and socioeconomic status (Tudiver, 2009).[11]

A health determinants framework may be used from an empirical, theoretical or policy perspective and attempts to understand the myriad of "interrelated social, cultural, environmental and biological factors that affect the health of individuals and communities" (Benoit and Shumka, 2009: 1). A health determinants framework acknowledges the interaction between predisposing genetic and biological factors, and social and cultural influences that impact individual attitudes and behaviours to positively or negatively affect health. The purpose of this framework is not merely to understand how various factors individually affect the health of a population, but to also understand

[11] Importantly, sex- and gender-based analysis can allow for further analyses engaging questions of sexuality and sexual orientation. Sex, gender and sexuality intersect in numerous ways, impacting various populations differently. Research indicates higher incidence rates of breast cancer among certain groups who are differentially affected by the intersection of sex, gender and sexuality. For instance, research suggests that lesbian women have increased rates of breast cancer, possibly as a result of reproductive factors such as being less likely to have given birth, or more likely to have done so later in life; or other factors such as body mass index and alcohol consumption; and barriers to screening and poor patient-provider communication within the healthcare system. For additional information, refer to Boehmer (2002), Brandenburg et al. (2007), Brown and Tracy (2008), Dribble et al. (2004), Kavanaugh-Lynch et al. (2002), and O'Hanlan et al. (2002).

why there are differences in health status and health outcomes and the influence of an unequal distribution of resources (Benoit and Shumka, 2009: 2).

The health determinants literature often fails to include sex and gender as determinants of health or includes one but not the other, though it should be noted that sex may also be viewed as a biological determinant which may be why some lists of social determinants of health do not include it. Sex and gender have often been conflated, used interchangeably and applied as one variable in health research rather than as constructs which cross-cut other variables in influencing health status (Benoit and Shumka, 2009; Tudiver, 2009). In their discussion about gender and determinants of health, Benoit and Shumka (2009: 5-6) note a systemic bias as a result of the historical health research that was based solely on the experiences of men and an inability to disentangle biological and social conditions which influence health outcomes. Thus, gender is now acknowledged as an important variable in policy analysis. Gender-based analysis requires "a solid knowledge of gender trends in society and the collection of information that furthers the understanding of the ways that gender interacts with policy, how policy may reinforce existing power structures based on gender, or how policy may reproduce gender inequalities (Hankivsky, 2007b: 114-15). Benoit and Shumka (2009: 11) offer a gender-inspired health determinants model which assigns equal importance of sex and gender to other fundamental health determinants. This model demonstrates causal connections between *fundamental macro-level determinants* including sex, gender,

socioeconomic status,[12] race, ethnicity, immigrant status, age, and geographic location; *access to key meso-level resources* such as employment status, education, childcare, safe neighbourhoods, and health services; *proximal micro-level determinants* such as smoking, diet and exercise; and *morbidity and mortality* as health outcomes. It is argued that sex- and gender-based analysis is essential for improving the health of Canadians in conducting health research and in the development and implementation of health programs and policies. Applying this lens is particularly relevant in research on women's health and is utilized throughout the dissertation.

Social Movement Theory

The history of breast cancer as a disease and the associated social movements must be examined in order to understand the influence of sociocultural, political, economic, and environmental factors. Women are often key actors in the mobilization around public health issues, including breast cancer (Williams et al., 1995). The tradition of hiddenness and invisibility with breast cancer led women to seek support from each other and to "form associations that could serve as the basis for organizing and taking action to improve treatment and to increase public awareness" (Schulzke, 2011: 43). The

[12] In addition to including sex and gender as fundamental determinants of health, this model recognizes how inequalities in health are associated with social class or socioeconomic status as measured by education, occupation and income which is consistent with discussions of the importance of socioeconomic status in Health Canada's *Health Policy Research Bulletins* (Health Canada, 2004a; 2005a; 2005b; 2007a; 2009a). This perspective considers a community's physical, social and public policy environments in relation to health status. Research demonstrates that health risks are not evenly distributed across the population but rather are disproportionately affecting those living with low socioeconomic status. Place can be thought of as a "geographic area where men, women, boys and girls all live in their diversity" (Health Canada, 2007a: 7). However, in order to properly analyze the complexities involved in the relationships between people, place and health, place ought to be viewed as more than simply geographical locations. Place may be conceptualized as "environments consisting of physical, cultural, political, economic and social components, with each component contributing in complex ways to the differential health risks experienced by a population" (Health Canada, 2007a: 8).

public awareness related to breast cancer as a disease along with a perception of susceptibility and risk act to mobilize support for collective action (Brown et al., 2002). Despite being organized around one specific disease, breast cancer is also one of the broadest of health social movements drawing from multiple influences and crossing institutional domains, disease regimes, fields of contention, and cultures of action (Klawiter, 2008a: 248).

Social movement theory provides a useful lens for examining breast cancer as a health social movement. Social movements have the potential to increase public awareness, provide political challenges towards government, issue scientific challenges to medicine and science and for changes in organizations such as health-related charities, and influence the distribution of power and authority among organizations within a movement (McCormick et al., 2003: 573). The success of a social movement hinges on the ability to strike a balance between the need to have the platform and priorities institutionalized so that broad and enduring changes can be made, and the need to remain flexible so as to generate the pressures necessary for adaptation. The movement's goals must be embraced by the prevailing power structure, but the movement must also maintain its ability to pressure and successfully influence governing institutions when new actions are required or more ambitious policies need to be pursued (Bryner, 2001).

This section will first provide a brief overview of traditional social movement theory before focusing on health social movement theory more specifically. I will examine the history of breast cancer as a disease which will be discussed in terms of disease regimes related to medicalization and biomedicalization (Klawiter, 2008a). The

groundwork for the breast cancer social movement began in the 1970s with public education work and the women's health movement, followed by HIV/AIDS activism in the 1980s which provided a new model for public impact on health policy, and the formation of national coalitions and significant lobbying efforts for increased research in the 1990s (Finley, 1995). Feminist, postcolonial and queer theories have pushed the boundaries of concepts such as woman, sex, and gender and highlight the importance of analyses in health that "contextualize women in their diverse social and economic circumstances and understand gender as inseparable from other forms of social difference such as race, ethnicity, culture, class, sexual orientation, gender identity and ability" (Morrow et al., 2007: 9). The three distinct cultures of action in the breast cancer social movement which emerged in the 1990s in the Bay Area of San Francisco are examined in depth (Klawiter, 2008a). The cultures of action and specifically the environmental breast cancer movement remain influential and provide important context for a primary prevention approach to the disease.

Traditional Social Movement Theory

Staggenborg (2007: 2) offers key questions to be considered in the study of social movements including why movements originate when they do, how they attract and maintain support, how they present issues and formulate strategies and tactics, how they structure organizations, how they change cultures, why they generate opposition and sometimes decline, and how and why they succeed or fail in achieving their objectives. Social movements are key agents for change in society. The development of the modern social movement was made possible by the development of the nation-state which is

noted as the most important and often only actor with the capacity to act on claims (Meyer, 2000: 39). Instances of change at the level of legislation and policy as a result of social movements may be rare, but changes more commonly occur that are local and cultural in nature. Social movements problematize the way in which we live our lives and call for changes in thought and action. Social movements are natural experiments in power, legitimation and democracy and the dynamics of social movements allow for the examination of the broader political structures of society (Crossley, 2002).

Tarrow (1994: 3-4) defines social movements as "collective challenges by people with common purposes and solidarity in sustained interaction with elites, opponents and authorities." Social movement theorists make the distinction between social movement groups and other organized groups such as political parties and interest groups by regarding social movements as challengers that are outside of the established power structure, whereas political parties and interest groups may have some degree of access as insiders. A social movement community consists of networks of individuals, cultural groups, alternative institutions, institutional supporters, and political movement organizations. The interactions of social movements involve social movement participants, the target of the social movement, the public, and other actors relevant to a specific movement. Social movements are sustained through multiple campaigns or multiple episodes of collective action within a single campaign (Staggenborg, 2007: 5-6). While the most common denominator of social movements is interest, it is the participants' recognition of their common interests that provides the potential for mobilization and collective action. It is by sustaining the collective action against

opponents that a contentious episode can become a social movement and it is the common purpose, collective identities, and an identifiable challenge that allows this process to occur (Tarrow, 1994).

Issues of collective identity play a key role in the development of social movements and in the experiences of participants. Collective identity involves an "individual's cognitive, moral, and emotional connection with a broader community. It is a perception of a shared status or relation, which may be imagined rather than experienced directly, and it is distinct from personal identities, although it may form part of a personal identity" (Brown et al., 2004: 60). The collective identity may be embedded within a specific social movement organization, within the social movement itself, and within the solidarity group involved. It is expressed through styles of dress, language, demeanour, and discourse and is not static but rather the result of fluid processes (Boehmer, 2000). Social movements can demonstrate how experiential knowledge and expertise are integral to collective identities which are involved in social, legal and political claims made by a social movement (Orsini and Smith, 2010).

Meyer (2000: 39-41) notes four elements which distinguish social movements from other social and political phenomena. The first element involves social movements making claims on the state or another authority which is viewed as having the capacity to address the grievances of participants. The second element is the challenging of cultural codes and the transformation of the lives of participants which allows for the acknowledgement and utilization of experiential knowledge. Social movements use tactics including demonstrations, boycotts, pickets, civil disobedience, and political