violence, in addition to those offered and accepted by mainstream politics, such as public education campaigns. Finally, social movements involve a diverse range of organizations and individuals working towards the same general goals, though it is noted that the boundaries of a movement are fluid.

Traditional social movement scholars in Europe and North America have utilized a number of theoretical approaches which were developed independently of one another, with *collective behaviour*, *resource mobilization* and *political process theory* emerging in the United States and *new social movement theory* originating in Europe (Staggenborg, 2007). The social movement theory emerging from the United States was founded upon the idea that instances of collective behaviour were influenced by particular psychological and societal factors, and that rational actors were able to propel social movements by strategically mobilizing resources or political forms. In comparison, the traditional European social movement theory has more focus on the impact of socioeconomic structures, as well as with ideology and identity in terms of collective action (Dobrowolsky, 2008: 164). It has been noted that the efficacy of social movements only becomes apparent over time, through the challenging of cultural codes and conventions. Social movements suggest to the broader society that "alternative frameworks of meaning are possible and that the operational logic of power apparatuses is not the only possible 'rationality'" (Epstein, 1998: 346).

Collective Behaviour Theory

Collective behaviour theory emerged in the United States and theorists claim that collective behaviour occurs during a period of social disruption as opposed to being part

of a standard political process (Crossley, 2002). While there are different approaches to collective behaviour theory, Staggenborg (2007) notes several commonalities. It is believed that instances of collective behaviour occur as a result of cultural or structural breakdown or strain, such as instances of rapid social change or a dramatic event. Instances of collective behaviour exist outside of institutionalized structures and there is an emphasis placed on the role of social psychology and shared beliefs and among participants (Staggenborg, 2007: 12).

The theory of mass society is based on Durkheimian theory and proposes that collective behaviour emerges as an extreme response to social isolation. The mass society exists within conditions in which there are few groups which link individuals to mainstream society such as religious or community organizations. It is suggested that individuals experience feelings of alienation and anxiety as a result of isolation from social and political institutions creating susceptibility for recruitment by social movements, such as the German Nazis. However, it has since been proven that it is not isolated individuals who are most likely to participate in social movements, but rather those who are already involved in social networks and organizations (Staggenborg, 2007: 14).

The Chicago School approach to collective behaviour was developed in the 1920s by American sociologists who studied symbolic interactionism which focused on how actors create meanings through social interaction. Proponents of the Chicago School approach contend that collective behaviour emerges when established systems of meaning and sources of information have broken down creating situations in which

participants create new meanings to guide behaviours. There is an emphasis on how participants act collectively and create new goals, culture and organizational structures in the form of social change (Staggenborg, 2007: 12-13). Another approach to collective behaviour includes Smelser's theory from 1962 which offers a model with six interrelated determinants, including conditions of structural conduciveness to encourage specific types of behaviour; structural strain which creates a sense of deprivation; the growth and spread of generalized belief which creates meaning for participants; precipitating factors related to the generalized belief which create a specific target for action; mobilization for action; and finally, acts of social control which may attempt to prevent the collective behaviour. Both the Chicago School approach and Smelser's theory have been critiqued for placing too much emphasis on structural strains on society, when "strains may be a fairly constant feature of societies and the rise of movements may be better explained by factors such as political opportunities, resources and organization" (Staggenborg, 2007: 13-14; Klawiter, 2008a).

Resource Mobilization and Political Process Theories

North American social movement theory began to move away from concerns of collective behaviour theory in the 1970s. The resource mobilization and political process theories found that the collective behaviour theories did not adequately account for the new wave of protests that emerged in the 1960s (Klawiter, 2008a). Whereas the collective behaviour theory focused on the motivations of individuals as a psychological phenomenon, the newer perspectives framed social movements as political phenomena where individual participants are viewed as rational actors with clearly defined goals and

motivation. According to this perspective, social movements "arise out of pre-existing organization, engaging in both institutionalized and non-institutionalized forms of action" (Staggenborg, 2007: 16). In addition to political movement organizations, it is argued that other types of mobilizing institutions are involved in the recruitment of participants, including formal and informal networks, groups and organizations (Staggenborg, 2007).

Resource mobilization theory emphasizes the importance of resources, organization and opportunities for collective action in the mobilization of social movements. The availability of resources is believed to be of great importance to the success of social movements in this approach. Resources include both tangible assets such as funding, as well as intangible resources such as the availability and level of commitment among participants. Resources used and created by social movements may include moral resources such as legitimacy; cultural resources including strategic knowledge; social-organizational resources including infrastructures, networks and organizational structures; human resources which includes both the labour and experience of activists; and material resources such as capital and office space (Staggenborg, 2007: 16). It is noted that these resources may not necessarily come from aggrieved groups who benefit from the social movement, but rather from conscience constituents who contribute to movements but do not personally benefit from the results of the movement (McCarthy and Zald, 1987). However, Melucci (1985: 197-98) suggests that the resource mobilization approach avoids a macro-level analysis and does not allow for the consideration of the "cultural orientation of the emerging social conflicts."

The political process approach to social movement theory was advanced by Tilly, Zald, McAdam, McCarthy, and Tarrow. It was developed from resource mobilization theory and also influenced by new social movement theory from Europe (McCarthy and Zald, 1987; Smith, 2008; Tarrow, 1994). Political process theory identifies both opportunities and constraints related to the mobilization of social movements and the potential influence on the emergence and activities of social movements (Brown et al., 2004). This approach emphasizes the interactions of participants with the state and the role of political opportunities as occasions for collective action; social movements are most likely to occur when activists feel that conditions are favourable (Klawiter, 2008a; Smith, 2008; Staggenborg, 2007).

In political process theory, the nation-state is framed as the "primary enabler, suppressor, and target of social movements" (Klawiter, 2008a: 11). Social movements are not only influenced by political processes but can create opportunities for the movement itself and for other social movements (Staggenborg, 2007). The strong program of political process theory posited that political opportunities did not directly cause social movements, but that social movements develop as a result of and would not succeed without political opportunities (Klawiter, 2008a). The weak program utilizes Snow's (2007) concept of "framing processes" which frames and assigns meaning in the process of interpreting relevant events in the mobilization of participants. It is described as a conscious and strategic effort of participants to develop a shared understanding in the legitimation and mobilization of collective action (Klawiter, 2008a: 12; Snow, 2007:

384).[13] The use of frames was to respond to critics who note that resource mobilization theory and weak political process theory did not account for the importance of cultural factors in the development of social movements, including ideas and perceptions. Political process theory has been critiqued for overestimating the role of the nation-state as a primary target for social movements and contentious politics (Klawiter, 2008a).

New Social Movement Theory

New social movement theory was developed from a history of European tradition, Marxist analysis and critical theory. Key theorists involved in new social movement theory include Melucci, Habermas and Touraine and it emphasizes social movements in a post-industrial, advanced capitalist society including the environmental, gay and lesbian, student, and women's social movements which emerged in the 1960s and 1970s (Brown et al., 2004; Klawiter, 2008a; Staggenborg, 2007). As post-industrial societies produce an integration of economic, political, and cultural structures (Melucci, 1985), it is argued that new social movements differ from movements in the industrial society, such as the labour movement in terms of structure, types of constituents and overall ideology. New social movement theorists emphasize collective identity, and the shared experiences and values which lend themselves to collective agency (Staggenborg, 2007: 20-21).

Whereas previous social movement research in Europe reflected Marxist theory, an important factor in new social movement theory is the intentional dismissal of class as a central concern (Orsini, 2008). Unlike political process theory which focused its

[13] Snow's (2007) "framing processes" should be distinguished from the policy frames in chapter one's discussion of interpretive policy analysis. While framing processes assign meaning and shared understanding in mobilization processes, policy frames create a framework in which to interpret policy-related documents and the meanings used by different policy actors and communities (Yanow, 2000).

attention on formal politics and the nation-state, new social movement theory proposed a need for a more thorough understanding of the large-scale transformations that occurred in advanced, post-industrial capitalist societies and argued that the expansion of the state into the private sphere produced new kinds of social movements (Klawiter, 2008a: 16-17). New social movement theory is "postmodern, postmaterial, or uninterested in the economy of the state," drawing on framing processes to understand the conflict which reflects participants' engagement with issues of collective identity rather than specific economic interests (Orsini, 2008: 342).

Key outcomes of new social movement theory include new types of values, identities and organizations (Staggenborg, 2007: 23). Williams et al. (1995: 115) note that many new social movements include issues related to inequality that were not considered in earlier class-related movements, including gender, sexuality, ethnicity, age, and disability. However, the authors note that class is still an important factor when studying social movements, such as the environmental movement. For instance, the environmental movement has largely consisted of a white, middle-class membership, whereas often those most at risk for related health problems are "working class" (Williams et al., 1995).

Critiques of Traditional Social Movement Theory

Dobrowolsky (2008) suggests that while each of the traditional approaches to social movement theory has strengths and weaknesses, these approaches do not consider that social movements negotiate both issues of strategy and identity. It is argued that politics must be discussed as both a way in which traditional political theory is

understood and also as a location for larger political discourse; while social movements are affected by politics, they may also affect politics (Dobrowolsky, 2008: 164). It is suggested that social movements can be understood in terms of macro- and micro-processes (Orsini, 2008; Staggenborg, 2007). Macro-processes may involve three related concerns including systematic explanations of the rise of social actors and participants, the clarification of the relationship between the state and civil society, and the processes related to the formation of collective identities. Micro-processes include the dynamics involved in the mobilization processes, the organization of the social movement, and the role of individual participants (Orsini, 2008: 342). While new social movement theory may address the macro-level processes, resource mobilization theory may be more able to address the micro-level processes. Civil society should be viewed as both the target and terrain of collective action (Orsini, 2008).

Melucci (1985: 795) contends that the field of social movement theory must transition from empirical generalizations towards analytical definitions and defines a social movement as a "form of collective action (a) based on solidarity, (b) carrying on a conflict, [and] (c) breaking the limits of the system in which the action occurs." He argues that analysis of social movements should focus on the systemic relationships more so than the logic of participants, but also recognizing the structural conditions and the importance of organization as a critical site of observation as social movements operate within systems of opportunities and constraints (Melucci, 1985). To address the question of why movements originate when they do and how they attract and maintain support, Staggenborg (2007) contends that social movements do not develop quickly and are often

linked in some way to earlier movements. The development of a social movement is directly influenced by processes of mobilization in which a "group that shares grievances or interests gain collective control over resources" and recruitment of individuals which is "part of a broader process of mobilization involving the commitment of individual resources, such as time, money, and skills, to a cause" (Staggenborg, 2007: 26). It is important to note that mobilization and recruitment are not static, but ongoing processes that are influenced by large-scale socioeconomic and political changes, opportunities and threats, critical events, pre-existing or emergent organizations, leadership, resources, and frames (Staggenborg, 2007: 28).

In the 1990s, many theorists drew from studies of the sociology of culture, gender, emotions, and identity as they placed a greater emphasis on the importance of agency in social movement theory. The social constructionist approach allows for a greater understanding of the "the expressive, emotive, discursive, interpretive, identity- and solidarity-building activities in which social movement actors engage" (Klawiter, 2008a: 10). However, continuing the recognition of the importance of structural factors on participants themselves and social movements more broadly, it is noted that structural factors shape both the external world and the internal world, including us as participants and subjects, as "selves" (Klawiter, 2008a: 11).

Health Social Movements

Narrowing the focus from social movement research more broadly to health research specifically, health social movements offer an important opportunity to challenge political power, professional authority and develop personal collective identity

(Brown and Zavestoski, 2005). Health social movements may be defined as "collective challenges to medical policy, public health policy, belief systems, research[,] and practice which include an array of formal and informal organizations, supporters, networks of cooperation and media" (Brown and Zavestoski, 2005: 1). While not considered by most political process theorists, health social movements have become central to research conducted by medical sociologists and anthropologists in order to understand the ongoing transformation of bodies, biomedicine and health care, and subsequently our experiences of health, risk, disability, illness, and disease (Klawiter, 2008a: 289). Health social movements have very different goals than those found in traditional social movement theory. While the focus of traditional social movements has been at the level of state policy, health social movements often focus on targets at other levels, such as the health care system, biomedicine and traditional approaches to health, public health policy, and the recognition of experiential knowledge and the illness experience. Health social movements challenge power and authority, as well as our understanding about individual and collective identities (Orsini and Smith, 2010: 40).

Brown and Zavestoski (2005: 9) note three ways in particular in which health social movements are able to affect contemporary society. First, health social movements have the potential to produce changes in the public health care system in terms of health care delivery, social policy and regulation. Secondly, health social movements can affect contemporary society through changes produced in the field of medical science, including promoting new and innovative hypotheses and methodological approaches to research, as well as advocating for changes in funding priorities. Finally, health social movements can

influence society by calling for processes of democracy within institutions that influence medical research and policy-making. Health social movements act as a "critical counter-authority aimed at democratizing and reshaping social policy and regulation in a way that transforms the socioeconomic and political conditions that underlie distributions of health and disease among populations" (Brown and Zavestoski, 2005: 14).

Concepts from resource mobilization, political process and framing theories are utilized in the conception of health social movements. The resource mobilization theory acknowledges knowledge, experience and networks as important resources to be utilized by health social movements; the political process theory may explain the processes utilized in health movements related to political opportunities; and framing processes include the importance of the use of emotions, grievance and experiential knowledge (Brown et al., 2004). However, the traditional approaches to social movement theory do not account for the role of class which is an important consideration in terms of access to health care, as well as health outcomes (Brown et al., 2004).

Health social movements most often address i) access to, or provision of, health-care services; ii) health inequality and inequity based on race, ethnicity, gender, class and/or sexuality; and iii) disease, illness experience, disability and contested illness (Brown, 2007: 26). The purpose of health access social movements is to seek equitable access to healthcare, as well as improved delivery of services. Examples of health access social movements include the US mobilizations for national healthcare reform and extension of health insurance to the uninsured (Brown, 2007). Groups associated with constituency-based health social movements address disproportionate outcomes and

oversight by the scientific community while addressing health inequality and inequity related to issues of race, ethnicity, gender, class, and/or sexuality differences. Constituency-based movements include the women's health movement, the gay and lesbian movement, and the environmental justice movement (Brown, 2007).

Until recently, the majority of health social movements focused more on expanding access to and improving the quality of health care which is reflected in the health access and constituency-based movements. The third category, embodied health movements, does address some issues of health care access but focuses more on the personal understanding and experience of illness (Brown and Zavestoski, 2005: 3). Embodied health movements include the tobacco control, HIV/AIDS and breast cancer movements (Brown, 2007). Embodied health movements address the experience of disease, disability or illness by challenging science on etiology, diagnosis, treatment, and prevention. These movements also focus on contested illness that may be unexplained by current medical knowledge or illnesses that have environmental explanations which are often disputed. Contested illness is defined as that which is "dismissed as illegitimate, - framed as 'difficult,' psychosomatic, or even non-existent - by researchers, health practitioners, and policy-makers operating within conventional paradigms of knowledge" (Moss and Teghtsoonian, 2008: 7). Research on contestation addresses illness not only through diagnosis and treatment, but also examines the mechanisms though which social practices, discourses and institutional processes shape conventional understandings of illness (Moss and Teghtsoonian, 2008). It is argued that virtually all diseases that can be attributed to environmental causes are highly contested because of the scientific

limitations related to the burden of proof and potential liability issues (Brown, Kroll-Smith and Gunter, 2000; Shriver and Kennedy, 2005). The status of illnesses as contested since the Second World War has arisen from several sources including i) the illnesses themselves stemming from the production use and disposal practices of the past half century; ii) a reflection of the growing uncertainty over the specific causes and expression symptoms; and iii) the popular participation in science and politics making the identification of illness and its causes much more public (Brown, 2007: 230).

Participants in embodied health movements organize to achieve medical recognition of contested illnesses, research and treatment.[14] Interestingly, members of these groups may also include people who are not ill themselves but see themselves at risk for the disease, as well as those who experience the disease through family connections. Brown et al. (2004: 54-55, 2012a: 18-21) provide an overview of the ideal characteristics and tactics that embodied health movements are unique in possessing. They include: i) introducing the biological body to social movements; ii) challenging existing medical and/or scientific knowledge and practice; and iii) activists' involvement

[14] While breast cancer and other environment-related illnesses such as multiple chemical sensitivity are contested illnesses, there are important differences surrounding the concepts of visibility and acceptability. When considering multiple chemical sensitivity, the contested nature lies not only with environmental links to disease but in the challenges of acceptance and in fact, to the very existence of the disease itself. Sufferers with multiple chemical sensitivity experience struggles with legitimacy and lack an accepted sick role due to insufficient scientific credibility. This is not the case when considering the diagnosis and treatment of breast cancer which is well established within biomedicine and the dominant epidemiological paradigm. The current treatment options for breast cancer, including surgery, radiation and chemotherapy are the same regardless of the etiology of disease; no one is disputing its existence or challenging its associated illness experience. There are clear parallels of invisibility between multiple chemical sensitivity and breast cancer in the invisibility of the environmental risks themselves which are often impossible to detect through human senses (Shriver and Kennedy, 2005), and subsequently impossible to avoid. For further discussion of contested illness and multiple chemical sensitivity, refer to Alaimo (2010), Ashford and Miller (1998), Dumit (2006), Kroll-Smith and Floyd (1997), Lipson (2004), Moss and Teghtsoonian (2008), Nash (2006), Shriver and Kennedy (2005), and Shriver, White and Kebede (1998).

and collaboration with scientists and health professionals in pursuing treatment, prevention, research, and expanded funding.

The first characteristic involves the experience of the disease within the body producing a particular "disease identity" which may or may not be stigmatized. The disease identity represents the intersection of the social construction of illness with the lived personal experience of a biological disease process. It is important to note that those with the disease experience are in a unique position of living with the disease process, the personal experiences, interpersonal effects, and the social ramifications of the illness (Brown et al., 2004: 55-56). Brown et al. (2004: 56) argue that the significance of the experience in the embodiment of a disease is reflected in the options available to an embodied health social movement when it is mobilized. Those who experience the disease identity can either work within or against the system which produces the scientific and medical knowledge (Brown et al., 2004, 2012a). That system plays a direct role in determining whether an illness is contested or not. The ability to work within or against this system may be impacted by a number of factors including whether or not the disease is contested. The personal experiences possessed by those with a disease identity within an embodied health movement prove valuable in terms of a lived experience and perspective that is not available to others, as well as instilling a moral credibility to the social movement within both the public and scientific realms (Brown et al., 2004: 56). A collective illness identity emerges when individuals develop a "cognitive, moral, and emotional connection" with other illness sufferers (Brown et al., 2004: 60).

The second tactic used by embodied health movements involves challenging the existing medical and/or scientific knowledge and practice. Embodied health movements are ultimately tied to the production of scientific knowledge and to changes in practice as social movement participants seek support for their illness claims from these institutions. What differentiates embodied health movements from other social movements in the challenge to medical and scientific knowledge is the involvement and utilization of experiential knowledge related to environments, bodies and illnesses (Brown et al., 2004: 56). The third tactic specific to embodied health movements involves the collaboration of activists with scientists and health professionals. Participants in this social movement must simultaneously challenge and collaborate with the fields of science, medicine and public health (Brown et al., 2012a: 19). This collaboration occurs as activists attempt to pursue treatment, prevention, research, and expanded funding for their illness (Brown et al., 2004: 54-55).

While embodied health movements may be unique in possessing each of the three traits, they are also similar to other social movements as mobilization is dependent on the emergence of a collective identity. In the case of illnesses, the initial approach involves working within established social institutions. However, if science and biomedicine fail to recognize the illness experience and offer accounts of the disease that activists do not accept, they may adopt an identity of an aggrieved illness sufferer and proceed with collective action (Brown, 2007: 27-28). The concepts of collective identity and disease identity are combined to provide a discussion about the politicized collective illness identity in which the collective identity is "linked to a broader social critique that views

structural inequalities and the uneven distribution of social power as responsible for the causes and/or triggers of the disease" (Brown et al., 2004: 60, 2012a: 22). One of the factors involved in the development of a politicized collective illness identity is a common experience with government, medical and scientific institutions which create the dominant epidemiological paradigm. The critique situated within the politicized collective illness identity removes the onus of responsibility for both the treatment and prevention of disease from the individual and places it on social institutions. Activists criticize the biomedical model which they argue treats disease as a discrete entity occupying the body and in turn, the body as a discrete entity which is separate from the person occupying it (Brown et al., 2004: 61, 67, 2012a). The characteristics of embodied health movements are reflected in breast cancer social movements which will be examined in depth in the following section after addressing the historical context.

The History of Breast Cancer and Disease Regimes

It is necessary to consider the history of breast cancer and its disease regimes in order to fully understand the contemporary context of the disease. Historically, ideas about women's risk for developing cancer were entangled with ideas about women's "essential nature" (Jasen, 2002: 20). During the Enlightenment period (1750s-early 1800s), the association between menopause and cancer was supported by humoral theory which promoted the idea that the breasts become engorged and developed tumours after menstruation ceased and the body became "uncleansed." Disease theory during this time observed that health status was negotiated by the body and mind, with a level of responsibility placed on the individual themselves (Jasen, 2002). The belief in

psychosomatic causes of disease was especially strong at this time, as "[w]omen, made of frail fibers, were seen to have easily impressionable souls and unquiet hearts readily carried away by lively imagination" (Bronfen, 1998: 114).

During the Victorian era (mid 1800s-1900), there was a shift towards research at the cellular level, although the association between breast cancer and hysteria was still common in medical literature in the late 19th century. Interestingly, it was during this era that public silence surrounding breast cancer became deeply entrenched. This silence was perpetuated by the notion that deaths caused by cancer were a social taboo in middle- and upper-class society, as well as being compounded by the breast's association with sexuality and as a violation of the mother's nourishing breast (Jasen, 2002: 28-29; Ehrenreich and English, 2011).

Klawiter (2008a) provides an important contribution to the literature surrounding breast cancer social movements with an alternative approach which focuses on the disease regimes in which breast cancer was medically managed in individuals and publicly administered in populations. Disease regimes are defined as consisting of the "institutionalized practices, authoritative discourses, emotional vocabularies, visual images, and social scripts through which diseases are socially constructed, medically managed, publicly administered, and subjectively experienced" (Klawiter, 2008a: 33). A disease regime includes interlinked practices through which a disease is medically managed in individual bodies and publicly administered across populations.

When the concept is applied to examine the regimes of medicalization and biomedicalization related to breast cancer (Klawiter, 2008a, 2008b), disease regimes of

breast cancer are mapped along the two axes of biopower. The first axis, the biopolitics of populations involves the public administration of disease and includes the discourses and practices of public health such as health promotion, education, population surveillance, epidemiology, and environmental health sciences. The second axis of biopower, the anatomo-politics of individual bodies involves the medical management of disease through the discourses and practices of clinical medicine including screening, diagnosis, treatment, and clinical research (Klawiter, 2008a: 33). Rather than the voluntary subjects of disease regimes such as scientists, physicians, healthcare professionals, Klawiter (2008a) focuses on the involuntary subjects who are recruited and incorporated into the regime through its discourses and practices. It is important to note that involuntary does not mean unwilling, rather disease regimes are most effective when the subjects are willing and able to participate in the processes.

The first regime of breast cancer, the regime of medicalization, occurred during the early 1900s after a shift in impressions of the human body. Humoral medicine was gradually replaced by scientific medicine which was founded upon new "technologies of seeing" including the microscope and medical dissection (Klawiter, 2008a). The research on breast cancer during the first half of the 20th century placed a significant emphasis on the natural pathology of the breast and away from causal factors outside the body, including dangers of "civilization" or trauma to the breast (Jasen, 2002). Though this process was initially resisted by both women and their physicians, it was during this time period that breast cancer became distinguished from other cancers, with its own origins

which could be treated through surgery. By framing breast cancer as a curable disease, this regime re-framed breast cancer patients as potentially curable (Klawiter, 2008a: 75).

As scientific medicine was institutionalized, it became an elite profession with largely white, upper-class, Christian men acting as its practitioners. The regime of medicalization transformed the power dynamic between physicians and patients in the clinical relationship, creating a new social script of the "sick role" which located the power and authority with male physicians and placed female patients in a position of compliance. The patient's narrative about her illness experience no longer held significant value, but rather the diagnosis came from the physician who now focused on the body's interior while subsequently creating new meanings of illness and reinforcing and reproducing the dominant gender order (Klawiter, 2008a: 62-63).

It was during the interwar period that a war on cancer was declared by government and the medical profession in which "only neglected cancer is incurable" (Jasen, 2002: 36; King, 2008). Breast cancer patients who were subjects of the medicalization regime were shaped and influenced in particular ways including i) experiences within their diagnoses and treatments; ii) the norms of non-disclosure rooted within interactions between physicians, surgeons and patients; and iii) the normalization processes which encouraged "cured" women to return to their daily activities and pass as "normal" women (Klawiter, 2008a: 75). The regime of medicalization was deeply entrenched in the gender roles and norms of this time period. Women were blamed for failing to be vigilant in detecting breast lumps with surgeons and pathologists promoting the "notion that women's greatest risk lay in the failure to be vigilant in detecting and

reporting suspicious lumps" (Jasen, 2002: 36). Male surgeons and occasionally the patient's husbands made the decisions regarding treatments, often without consulting the patient and, at the same time, requiring her compliance and obedience (Jasen, 2002; King, 2008; Klawiter, 2008a).

Breast cancer was diagnosed through surgical biopsy during the regime of medicalization, and because breast cancer was viewed as a localized disease, the Halsted radical mastectomy became the dominant treatment among North American surgeons. Until the early 1970s, rather than performing two separate procedures, a radical mastectomy was performed if the biopsy results were malignant, while the patient remained unconscious (Klawiter, 2008a: 76). The radical mastectomy involved removing the entire breast in addition to the chest wall muscles, lymph glands and fat located under the skin (Ley, 2009). When the breast cancer patient awoke from the biopsy and radical mastectomy, she awoke "not as a cancer patient but as a mastectomee who had been successfully treated for a condition that was not called by name, at least not in front of the patient" (Klawiter, 2008a: 77). The sick role which emerged in this regime segregated and isolated those who were ill from those who were not and did not allow for the forming of a collectivity. The sick role "channels deviance so that the two most dangerous potentialities [to the medical establishment], namely, group formation and successful establishment of the claim to legitimacy, are avoided" (Parsons, 1951: 477; Klawiter, 2008b). For instance, in the case of new mastectomees, patients were required to leave the temporary sick role and return to their regular lives and responsibilities immediately. The "formation of disease-related identities, solidarities, social networks,

and other forms of biosociality[15] was thus heavily constrained by and within the regime of medicalization" (Klawiter, 2008a: 37).

A second regime of breast cancer, the regime of biomedicalization emerged during the 1970s and 1980s with new developments in biomedical research and cancer epidemiology. This regime moved discourses and practices of risk to the forefront and included changes in the practices of education, and measures and promotion of early detection, diagnosis, disclosure, treatment, and rehabilitation. In considering the public administration of disease, this included the development of new screening practices and the construction of all women, regardless of whether they are symptomatic or not, into risky subjects who are responsible for the status of their own health and must participate in the screening practices (Klawiter, 2008a: 86).

During the late 1970s, feminist health activists began to agitate against the one-step biopsy and Halsted radical mastectomy, calling it paternalistic and patriarchal. They argued that the procedures denied women the right to be informed of their diagnoses and to participate in the decision making process (Boehmer, 2000; Ley, 2009). The processes involved in the medical management of disease in the regime of biomedicalization include the emergence of informed consent, the refinement of surgical procedures, increased use of adjuvant therapies, redefining the roles and responsibilities of patients and physicians, and the development of rehabilitation programs which addressed the experiences of isolation among breast cancer patients (Klawiter, 2008a).

[15] Biosociality signifies the ways in which the practices of science, public health and medicine enable the formation of new subjects and social groups (Klawiter, 2008a: 27).

Historically breast cancer was a private, even secretive disease associated with feelings of shame. Breast cancer emerged into the public domain in the 1970s through the influence of feminism and the women's movement,[16] as well as the role of the media and the public breast cancer cases of prominent women such as Shirley Temple Black, Betty Ford and Happy Rockefeller who encouraged early detection and intervention (King, 2008; Ley, 2009; Sherwin, 2006). While these prominent women were willing to speak publicly about their experiences with breast cancer, it is important to note that their positions of privilege and status influenced their ability to do so.

Breast cancer was now framed as a disease for which every woman is at risk and required continual vigilance by individual women. Measures of surveillance and detection were heavily promoted as the "moral duty" of women, including engaging in breast self-exams, clinical examinations and mammographic screening. The temporary sick role for symptomatic women from the regime of medicalization was replaced by a permanent risk role for all women (Klawiter, 2008a: 38). While the processes involved in the regime of biomedicalization did not improve breast cancer incidence or mortality rates during the 1970s or 1980s, the subjects and social relations of the disease regime were transformed. This created the conditions for biosociality and collective action among the "risky subjects" within this regime, including asymptomatic women, women

[16] In addition to the women's health movement and feminism, the breast cancer social movement has been significantly influenced by the HIV/AIDS movement. Despite differences in the history, biological and social epidemiology of breast cancer and HIV/AIDS, it was the politicization of HIV/AIDS that paved the way for the politicization of breast cancer and the participation of women who had not previously been active in social movements (Boehmer, 2000; Epstein, 1998). For a complete discussion of the relationship of activism between breast cancer and HIV/AIDS, refer to Ulrike Boehmer's (2000) book *The Personal and the Political: Women's Activism in Response to the Breast Cancer and AIDS Epidemics*.

in treatment for breast cancer, women at risk of recurrence, and women in remission (Klawiter, 2008a: 39-40).

The women's health movement was grounded during the third wave of the feminist movement.[17] Specifically, the cancer movement in Canada and the United States became organized around a feminist analysis, taking the position that cancer is a political issue. Boehmer (2000: 99) points to a collective feminist identity which is negotiated between politically experienced feminists and women with no prior political engagement. Since the early 1990s there has been an ongoing cultural transformation in which understandings of breast cancer have shifted from that of a historically stigmatizing disease of individuals suffering in isolation, to that of a neglected epidemic at the center of public debate and political organizing. It has become common for many women with breast cancer to dismiss the label of "patient" and embrace an identity associated with being a "survivor" (King, 2008: x). This cultural transformation led to the development of three distinct cultures of action in the San Francisco Bay Area (Klawiter, 2008a).

Breast Cancer and Cultures of Action

Klawiter (2008a: 44) uses cultures of action as a "heuristic device for conceptualizing and mapping patterns of similarity and difference within social movements." Cultures of action are produced by individuals, groups, agencies, organizations, councils, corporations, and coalitions and involve shared goals, assumptions and discourses among interactions involving allies and opponents. They

[17] During the third wave of feminism, the women's health movement focused specifically on the politics of reproduction, including issues surrounding sexuality, birth control, pregnancy, childbirth, breast-feeding, forced sterilizations, unnecessary hysterectomies, and the safety of pharmaceutical technologies including the birth control pill, hormone therapy and the DES (diethylstilbestrol) controversy (Klawiter, 2008a: 167).

change over time as a result of influence from the actions of members and relationships with other cultures of action, as well as the shifting dynamics in the discourses and practices the culture of action is attempting to influence (Klawiter, 2008a; Zavestoski et al., 2004). Cultures of action "are not simple constellations of ideas, frames, cognitions, or identities. Rather, they enact, embody, and articulate (visually and verbally) particular visions of what is and what ought to be" (Klawiter, 2008a: 44).

During the same time period in the 1990s, three different cultures of action emerged within the Bay Area of San Francisco. Moffett (2003: 290) contends that breast cancer advocacy groups have three goals in particular, including: i) raising awareness about breast cancer and promoting the use of biomedical processes, such as mammographic technologies; ii) providing emotional support for women in varying stages of the disease and their treatment; and iii) raising funds or promoting that funds be allocated towards scientific research for breast cancer. These goals are reflected in Klawiter's (2008a) first culture of action; however it is not the case in the second or third. Each specific culture of action employs different discourses related to breast cancer, promotes different identities and body politics, and supports different agendas and priorities. Each culture of action also draws upon distinct understandings of gender, race, class, and sexuality, and has diverse perceptions of and relationships to science and biomedicine, capitalism, corporate philanthropy and cause-related marketing, and the pharmaceutical industry (Klawiter, 2008a: 45).

1) Culture of Early Detection and Screening Activism

The discourse surrounding breast health began to emerge in the early 1990s; these discussions were linked exclusively to breast cancer screening as part of awareness and early detection campaigns. This is evidenced by the focus of the *culture of early detection and screening activism* which emerged in the San Francisco Bay Area and drew upon the strong evidence and science related to the detection and treatment of breast cancer (Brown et al., 2002; Klawiter, 2008a). Similar to the breast cancer awareness campaigns of the 1970s and 1980s, this culture of action involves the promotion of breast self-examination, clinical breast exams and mammographic screening as life-saving technologies while simultaneously placing the onus of responsibility to comply with screening for early detection on individual women. The unique aspects which emerged during the 1990s and distinguished this culture of action from previous campaigns include three developments in particular: i) the interpenetration of the state, private industry and breast cancer screening advocacy; ii) the rise of mass-participation fund-raising events; and iii) growing pressure to expand mammographic screening to medically marginalized communities (Klawiter, 2008a: 131-32).

The early detection and screening activism culture of action challenged the assumption in social movement theory that social movements have clear boundaries which can be distinguished from the state, private industry and philanthropic organizations, and those social movements must engage in contentious forms of protest (Tarrow, 1994). In addition to individuals, this particular culture of action involved public agencies, professional organizations, health care organizations, and private

industry. Rather than engaging in the contentious forms of protest embraced by the other cultures of action in this area, a culture of consensus emerged which "privileged the identity of 'breast cancer survivor' and tied this identity to the physical display of heteronormative femininity" (Klawiter, 2008a: 134).

The discourse utilized in this culture of action focused exclusively on a lack of awareness about breast cancer and the financial, cultural and physical barriers to screening. Concerns about access to mammographic screening for medically marginalized women, particularly low-income, uninsured women of colour became a priority at this time (Klawiter, 2008a). It is important to note that this was not unique to the San Francisco Bay Area, but was also occurring nationally and internationally. Parallels can be drawn between these issues in the United States and similar concerns about access to mammographic screening in rural and geographically isolated areas of Canada.

The promotion of mammography within this culture of action increased women's concern about their individual risk of developing breast cancer. The discovery of the "breast cancer genes," $BRCA_1$ and $BRCA_2$ during the 1990s altered the discussion surrounding women's risk of breast cancer and the options available in determining this risk. An estimated 5-10 percent of breast cancer diagnoses involve hereditary forms of cancer and women may seek to engage with the health care system to obtain this information through genetic testing regardless of their risk profile (Bottorff et al., 2002; Bouchard et al., 2004; Rees et al., 2001). It is suggested that testing for $BRCA_1$ and $BRCA_2$ genetic mutations may be the first widespread utilization of pre-symptomatic

genetic testing transforming general medicine into predictive medicine (Bouchard et al., 2004). Although the majority of breast cancer diagnoses do not involve genetic mutation, its presence does increase the risk of an invasive breast cancer diagnosis[18] (Klawiter, 2008a). Women with $BRCA_1$ and $BRCA_2$ genetic mutations are faced with uncertainty about if and when they will develop breast cancer and how to manage this risk (Lippman, 1998; Rees et al., 2001). The preventive measures offered by geneticists and physicians to patients with $BRCA_1$ and $BRCA_2$ genes include increased surveillance, breast self-examination, mammography screening, chemoprevention, and prophylactic surgery in the form of a preventive mastectomy (Bouchard et al., 2004).

Genetic screening contributes to the biomedicalization regime of breast cancer and the framing of all women as "risky subjects" (Klawiter, 2008a: 262). Due to the widespread prevalence of cancer in western society, Jain (2007a) contends that everyone lives with some degree of prognosis. The effects of genetic screening for breast cancer are not limited to the individual being tested, but also have an impact on close relatives who are living and those who have not yet been conceived. The knowledge of genetic mutations linked to breast cancer increases the experience of risk and anxiety among both the carrier and his or her extended family (Klawiter, 2008a: 262).

While prophylactic surgery has a strong history in the regime of medicalization, the option of chemoprevention emerged in the late 1990s. The Breast Cancer Prevention Trial included 13,338 Canadian and American women, and tested the breast cancer treatment drug tamoxifen against a placebo in "high risk" women who had a 1.7 percent

[18] The increase in risk of developing invasive breast cancer with the presence of $BRCA_1$ and $BRCA_2$ genes is between 36-85 percent depending to which study one refers (Klawiter, 2008a: 262).

or higher risk of being diagnosed with breast cancer in the next five years. This percentage was the average risk a 60-year old woman had of developing breast cancer in the United States. The results of this clinical trial indicated that the group of women receiving tamoxifen were approximately half as likely to develop invasive breast cancer as the control group (Batt, 2002; Klawiter, 2008a). There was extensive media coverage around this issue at the time suggesting that "[w]e know for the first time in history that we can prevent cancer through pharmaceuticals" (Batt, 2001). The subsequent decision by the United States Food and Drug Administration to approve tamoxifen for supplemental and preventive use in healthy women considered to be at "high risk" was considered controversial by women's health organizations. The decision was critiqued in terms of the promotion of the drug by pharmaceutical company, Astra Zeneca, to physicians and direct-to-consumer advertisements targeting women. By focusing on medication for the "prevention" of breast cancer, the advertisements removed focus from the causes of the disease. The decision was also critiqued as data indicated that women taking tamoxifen were twice as likely to develop endometrial cancer, three times more likely to develop pulmonary embolisms, and fifty percent more likely to suffer a stroke (Batt and Lippman, 2010; Klawiter, 2008a: 263-66). In fact, the use of tamoxifen on healthy women was described as "disease substitution" due to the number of other life-threatening illnesses associated with taking the drug (Batt and Lippman, 2010: 49; Fosket, 2004: 293). These policies assume that risks associated with health should be "managed rather than reduced or eliminated" (CWHN, 2003).

The representative symbol associated with the culture of early detection and screening activism is the now well-known pink ribbon -- the gold-standard in cause-related marketing. Cause-related marketing emerged in the mid-1980s as a strategic marketing tool which allowed companies to simultaneously associate themselves with a particular cause while concurrently increasing profits and developing reputations as good corporate citizens (King, 2008: 9). During this time, cause-related marketing transformed from short-term promotions of one to two months with charitable organizations towards long-term commitments which directly link the company to a particular "cause" in the minds of consumers. Marketing professionals are clear that while the long-term strategy may be viewed by the public as less opportunistic than short-term campaigns, cause-related marketing campaigns themselves are and should be seen as "first and foremost a strategy for selling products, rather than an altruistic or philanthropic activity" (King, 2008: 10).

The now widely recognizable and corporate-influenced pink ribbon has its origins within a grassroots movement. Inspired by the red ribbon associated with the HIV/AIDS movement, Charlotte Haley began distributing peach ribbons to raise awareness about breast cancer and funds for the prevention of the disease (Harvey and Strahilevitz, 2009; Moffett, 2003). She distributed postcards with the peach ribbons that stated: "The National Cancer Institute's annual budget is $1.8 billion, only 5 percent goes for cancer prevention. Help us wake up our legislators and America by wearing this ribbon" (BCA, 2011a). However, Haley was not interested in commercializing her efforts and refused to partner with cosmetics company, Estée Lauder. Based on focus group research, Estée

Lauder created, produced and marketed the *pink* ribbon, with the colour choice representing heterosexual femininity and hope (Estée Lauder, 2010; Jain, 2007b).

Demonstrating the principles of cause-related marketing, breast cancer awareness became linked to corporations during the 1990s. Industries such as fitness, fashion and cosmetics used breast cancer as a way to differentiate their products from others, while increasing their visibility in relation to female consumers, elevating their corporate image, and increasing profit-margins (Harvey and Strahilevitz, 2009; King, 2008, 2010). The combination of cause-related marketing and breast cancer has resulted in a clear case of cause-related consumption with the successful pink ribbon and National Breast Cancer Awareness Month campaigns that encourage the public to make purchases in order to "support breast cancer." Fundraising events such as Run/Race for the Cure involve hundreds of thousands of participants across Canada and the United States each year. These events exclusively promote positive messages combining images of feminine triumph, strength, positivity, hope, and beauty (Batt, 1994; Klawiter, 2008a).

There was an interesting dynamic during this time in which women had developed a personal relationship to breast cancer, either as a patient themselves or knowing someone else with breast cancer. The pervasiveness of awareness campaigns resulted in the commercialization of breast cancer presenting the disease through a very specific and narrow lens. The decades of early detection promotion had created a category of white, middle-class women as both "risky subjects" and consumers (Klawiter, 2008a: 132). This culture of action benefits from the cause-related marketing, pink ribbon and philanthropic activities related to breast cancer while blurring the

boundaries between social movements, the state and private industry. The tangible successes of this movement lie in addressing the unequal access to mammographic screening in medically marginalized communities. This culture of action operates on a discourse of hope which advertises a sense of control for women through participation in early detection and screening activism, despite being risky subjects (Klawiter, 2008a).

2) Culture of Patient Empowerment and Feminist Treatment Activism

The second culture of action that emerged in the Bay Area, *patient empowerment and feminist treatment activism*, occurred during a time when cancer was viewed as an "acceptable epidemic" and was in conflict with the culture of early detection and screening activism (Klawiter, 2008a: 164). Participants in this movement were influenced by the women's health movement and the lesbian community and worked towards creating a discourse that was feminist, anti-racist, not exclusively heterosexual, accommodating towards people with (dis)abilities, and recognized non-Western alternative therapies (Klawiter, 2008a: 170). By addressing and combining concerns of racism, classism and sexism, participants in this culture of action believe that breast cancer is influenced as much by factors of economic, social and cultural factors as genetics. Thus, for them, addressing issues of breast health requires engaging with these plural environments (Davis and Webster, 2002; Eisenstein, 2001; Potts, 2004a).

This culture of action constructed a feminist discourse to emphasize the importance of empowerment for breast cancer patients. The feminist cancer organizations in the Bay Area, such as Breast Cancer Action and the Breast Cancer Fund, promoted the empowerment of women with breast cancer, and challenged the positive discourse of

'survival.' They scorned the unscarred feminine bodies that were utilized and promoted in the mainstream media and within the culture of screening and activism underlying a heteronormative framework (Klawiter, 2008a). Feminist activists challenge the "cheery deary" positive discourse promoted by pink ribbon activists with

> narratives that drew attention to the false promises and misrepresentation of the cancer establishment, to the ineffectiveness of mammographic screening, the unreliability and toxicity of treatments, the chronic nature of the disease for many women, the inadequacy of research, the lack of scientific understanding and medical progress on the disease, the emphasis on individual risk factors, and the low priority given to cancer prevention (Klawiter, 2008a: 175).

They created social spaces which promoted alternative images, discourses and ways of embodying breast cancer (Ehrenreich, 2001; King, 2010; Klawiter, 2008a).

The culture of patient empowerment and feminist breast cancer activism was founded upon a culture of caring and compassion for women diagnosed with breast cancer, and thus involved advocating for direct services and support. While the feminist cancer activists supported the promotion of universal access to mammographic screening for all women, this culture of action also challenged the idea that unpleasant emotions such as sorrow, grief and aggression should be suppressed. Rather than buying into the symbolic pink ribbon, feminist cancer activists wore "Cancer Sucks" buttons. They showed photographs of bald, one-breasted women while arguing that the survival discourse and "pretty pink ribbons distorted the ugly realities of the disease" (Klawiter, 2008a: 169; Matuschka, 2012; Sulik, 2011).

3) Culture of Cancer Prevention and Environmental Risk

The third culture of action that emerged in the Bay Area in the 1990s, the *culture of cancer prevention and environmental risk*, frames breast cancer as a 21st century

phenomenon by engaging with issues of environmental health (Klawiter, 2008a). Breast cancer is framed as representing the hazards associated with industrialization emblazoned onto women's bodies (Sherwin, 2006: 18). Historically, social movements have relied not on science but on ethical and moral appeals to promote change. Couch and Kroll-Smith (2000: 384) find that contemporary environmental social movements are organized around more than a populist appeal to ethical or moral rights. Rather, activists believe we are endangered by the production, use and disposal of environmental contaminants and utilize scientific, technical and medical expert knowledge with moral and ethical arguments about the right to a safe environment. These movements combine resources from civic rights and environmental justice movements with toxicology, risk assessment and biomedicine in order to frame claims which aim to change the actions or policies of institutions.

The culture of cancer prevention and environmental risk utilized the appeal of the discourse of early detection to challenge the personal lifestyle and responsibility surrounding the dominant epidemiological paradigm of breast cancer in order to promote a message of cancer prevention (Klawiter, 2008a). This culture of action recognizes that the lifestyle choices and behaviours women are encouraged to engage with in order to prevent breast cancer, such as diet, exercise and age at which she has her first child, are significantly influenced by her socioeconomic status and her cultural environment, and do not account for factors that are beyond her personal control (Leopold, 1999). King (2010: 107) argues that this discourse which locates risk factors in individual behaviours "operates more to detract attention away from external variables that might be implicated

in high incidence rates (industrial pollution, for instance), rather than to demonize women with breast cancer." The idea of bodies existing separately from their environments distorts the complexity involved and there is a call for a recognition of "the interpenetration of bodies and their overlapping environments" (Eisenstein, 2001: 84). The environmental breast cancer movement has worked towards four goals in particular: i) to broaden public awareness of potential environmental causes of breast cancer; ii) to increase research into environmental causes of breast cancer; iii) to create policy which could prevent environmental causes of breast cancer; and iv) to increase activist participation in research (Brown, 2007: 44; Brown et al., 2004: 66-67; McCormick et al., 2003: 546).

In this perspective, "the gendered experience of breast cancer leads...[activists] to experience their disease not as a personal trouble to be dealt with through lifestyle changes, but as a condition caused by social and environmental factors that are shaped by powerful social institutions" (Zavestoski et al., 2004: 569). As Sulik (2011: 372) argues, "[t]he cultural equation of breasts, and having breasts, with women's heterosexual identity enables pink ribbon products to trivialize and ignore the realities of breast cancer while simultaneously degrading women and putting them in their place." Similar to the feminist breast cancer activists, the environmental breast cancer movement problematizes the heterosexual norms of femininity that are utilized in the media's portrayal of breast cancer. This portrayal is furthered by the involvement of the beauty and fashion industries in the events and campaigns associated with National Breast Cancer

Awareness Month, and the mainstream breast cancer movement's promotion of heteronormative femininities (Brown, 2007; Jain, 2007b).

Zavestoski et al. (2004: 565) note three specific considerations related to gender which create difficulties in the attempts by activists' to transform popular and medical notions of breast cancer and situate them into a broader social and environmental context. The constraints include depictions of activists as "hysterical women" which has its roots in the 19th century medical literature linking breast cancer and hysteria (Jasen, 2002). The second element includes a marginalized illness experience of breast cancer where women are socialized and encouraged to present themselves as having "normal" bodies. The "struggle for normalcy often begins as soon as the disease is detected, intensifies as treatment becomes more aggressive, and continues long after the disease is cured" (Schulzke, 2011: 37). Finally, the third element involves the sexualization of breast cancer through the media. There are varying degrees of sexualization used in breast cancer cause-related marketing campaigns, including those that are overtly sexualized with images objectifying women's breasts and slogans that include "great breasts are worth fighting for," "save the ta-tas," and "don't let cancer steal second base" (Sulik, 2011; Save the Ta-Tas, 2012; Total Pro Sports, 2010; Zazzle, 2009). While this sexualization results in greater media coverage, it also parallels the experience of breast cancer with the loss of one's sexual identity which shifts attention away from important structural critiques (Zavestoski et al., 2004: 576). In this case, important questions which should be asked include "what is being bought and sold in advertisements, and in the name of 'the cause'?"

Couch and Kroll-Smith (2000: 388) find that in this movement, there are "people who find the authoritative voices of science and medicine unable to make sense of their bodies and environments. Importantly, they are doing more than questioning the use of expert knowledge. Indeed, they often become experts themselves." It is in this respect that, despite constraints, there are also a number of ways that gender can enable the efforts of activists including: i) a unique perspective on health and illness as a result of women's marginalization; ii) a holistic view of social change involving knowledge, personal experience and action; and iii) solidarity and social networks which result from a shared sense of subordination (Zavestoski et al., 2004: 564). Perhaps most importantly, activists utilize their embodied knowledge and lay expertise which creates a unique perspective while they work to "transform personal experience into scientific knowledge and then into political action" (Zavestoski et al., 2004: 572).

It is argued that scientific challenges and policy implications are far more complex with contemporary contested illnesses. Past examples of contested illnesses include black lung disease attributed to coal mining and asbestosis or mesothelioma attributed to asbestos exposure. These diseases became established through lay discovery and unions, occupational health and safety organizations and sympathetic scientists who challenged the dominant epidemiological paradigm to demonstrate a path of causation (Brown, 2007; Markowitz and Rosner, 2002). For instance, an active trade union health and safety movement worked towards exposing workplace hazards in Ontario (Brophy et al., 2007: 238). This movement resulted in the provincial government establishing a Royal Commission in the early 1980s in order to examine the health and safety issues

arising from the use of asbestos in Ontario (Brophy et al., 2007; Dupré et al., 1984). Between 1980-2002, approximately 1,487 cases of mesothelioma were diagnosed among men in Ontario (Brophy et al., 2007). However, it is Sarnia, Ontario that is the "epicentre of asbestos disease" (Wordsworth, 2012: 32). Hospital data for Sarnia from the 1990s demonstrates that the overall cancer rate was approximately thirty-four percent higher than the provincial average, the lung cancer rate was fifty percent higher, the mesothelioma rate was five times higher, and the asbestosis rate was nine times higher (Mittelstaedt, 2004). It is suggested that the statistics around asbestos-related disease incidence are likely to be underestimated based on an under-diagnosis and poor record keeping related to occupational health issues (Brophy et al., 2007; Mittelstaedt, 2004).[19] While the economic cost of protecting coal miners and people working with asbestos fell primarily on industry, members of the culture of cancer prevention and environmental risk suggest that the environmental causes implicated in breast cancer are linked to "the heart of the entire economic system and require massive policy shifts" (Brown 2007: 229).

Activists utilized confrontational politics and public protests in their attempts to challenge private industry, local and state government, the other cultures of action, and public attitudes and perceptions. While the culture of early detection and screening activism uses the pink ribbon as its representative symbol, the culture of cancer prevention and environmental risk utilizes a poison skull to demonstrate the health

[19] For additional discussion of asbestos exposure in Ontario, refer to Brophy et al. (2007), Dupré et al. (1984), Landsberg (2012), Mittelstaedt (2004), and Wordsworth (2012).

hazards associated with environmental contaminants. They posit that the economic interest in maximizing profits often conflict with efforts of disease prevention (Leopold, 1999; Potts, 2004b). Wilkinson (2007: 424) speaks to the explicit links with the commercialization of breast cancer. She specifically addresses the breast cancer "industry," and the profits associated with mammographic screening services, radiotherapy and chemotherapy, and drug treatments.

The National Breast Cancer Awareness Month and the pink ribbon campaign is a clear example of successful cause-related marketing and associated corporations such as Avon, Revlon, General Motors, and Nike maintain a safe distance from feminist and environmental breast cancer activism (Moffett, 2003). The primary sponsor of this campaign, AstraZeneca, is critiqued by environmental breast cancer movement activists because in addition to manufacturing tamoxifen, it also produces pesticides, including the carcinogen acetochlor and one of its manufacturing plants is reportedly the third largest source of airborne carcinogenic pollution in the United States (Sulik, 2011; Wilkinson, 2007: 424). AstraZeneca also has the authority to approve or disapprove all printed materials used in campaigns during Breast Cancer Awareness Month and, not surprisingly, this literature does not include mention of the potential role of environmental contaminants in causing breast cancer (Sherwin, 2006; Wilkinson, 2007).

There is very little transparency when examining the percentage of revenues corporations donate from purchases of pink ribbon products during Breast Cancer Awareness Month to breast cancer research, treatment, screening, prevention, or education (Harvey and Strahilevitz, 2009; Moffett, 2003). Questions that may be asked

when purchasing pink ribbon products include: is there a cap on the amount of money the company will donate and has the maximum amount already been met; is the company contributing to the increasing incidence rates of breast cancer through everyday exposures to their products; and what organization will receive the funds and how will they be used (BCA, 2011a). Indeed, King (2010: 108) argues that there is "nothing inherently uncontroversial about breast cancer….[T]he disease has been manufactured as such over two decades of organizing that has gradually been incorporated into conservative political agendas, the programs of large nonprofits in partnerships with the cancer industries, and corporate marketing strategies." Activists in the culture of cancer prevention and environmental risk problematize, critique and question three aspects of the National Breast Cancer Awareness Month in particular. The first is that it legitimizes and promotes early detection programs as the only public health approach to breast cancer and does not recognize a causal link between environmental contaminants and breast cancer. The second is that the very multinational corporations that participate are also contributing to the development of cancer through the production of toxic products including pesticides, plastics and their industrial by-products, such as dioxin. The third is that certain corporations, such as pharmaceutical companies, profit from both the diagnosis *and* the treatment of breast cancer, and this information is concealed from the public (Klawiter, 2008a: 201).

Jain (2007b: 519) contends that the use of cause-related marketing in pink ribbon campaigns to increase profits and build name recognition among consumers, while "cover[ing] up their production of carcinogens bears the name 'pinkwashing'...which

obscures the links among the production, suffering and obfuscation of disease." The term "pinkwashing" is used to describe a company or organization that claims to care about breast cancer by promoting a pink ribbon product, but at the same time produces, manufactures and/or sells products that are linked to disease (BCA, 2011a).[20] Pink ribbon culture has become more than a successful cause-related marketing campaign:

> [I]t has become a distinct cultural system that is integrated into the fabric of [North] American life. Grounded in advocacy, deeply held beliefs about gender and femininity, mass-mediated consumption, and the cancer industry, pink ribbon culture has transformed breast cancer from an important social problem that requires complicated social and medical solutions to a popular item for public consumption (Sulik, 2011: 9).

Each of the three cultures of action, the culture of early detection and screening activism, the culture of patient empowerment and feminist treatment activism, and the culture of cancer prevention and environmental risk provided important contributions to the breast cancer social movement which can be seen throughout the United States and Canada. The breast cancer movement provides a unique example of activists' efforts that utilize ideologies from health, environment and women's movements (McCormick et al., 2003). The cultures of action which emerged in the 1990s helped to shape the breast cancer social movement into one of the most popular and influential movements of the last twenty-five years (Klawiter, 2008a). As the breast cancer social movement is

[20] It should be noted that the concept of "pinkwashing" is also being used to describe the practice of a state, corporation or organization using "gay rights rhetoric" in order to present a particular image and to detract from other practices (Dhoot, 2012). Sarah Schulman published a widely cited op-ed in the *New York Times* grounding this global practice in Israel with a deliberate juxtaposition of Israeli LBGT citizens and Palestinian citizens (Schulman, 2011). In this context, pinkwashing can draw upon the "emotional legacy of homophobia" in its framing of LGBT citizens in order to distract from other, more controversial aspects of state behaviour (Schulman, 2011). For additional information on pinkwashing and the LGBT community, refer to Dhoot (2012), Fung (2013), Ng (2013), and Schulman (2011).

ongoing and diverse, it is important to consider the varying constructions of risk in relation to the development of the disease, everyday exposures to toxic substances and outcomes for women's health.

Risk and the Risk Society

Risk is a pervasive concept related to human existence in contemporary western societies (Lupton, 1999). Sociocultural perspectives on risk emphasize the social and cultural contexts in which risk is understood, factors that approaches rooted in the natural sciences and biomedicine are criticized for neglecting. In an interdisciplinary perspective, risk is viewed as a cultural and political concept associated with ideas about choice, responsibility and blame. Lupton (1999) points to categories of risk[21] which concern individuals and institutions in contemporary western societies that are indicative of the broader sociocultural, political and economic context in which they exist including environmental risks such as pollution, radiation and chemical contaminants. This specific category of risk should be considered along with its relationship to health outcomes.

When considering the ontology of risk, Rigakos and Law (2009) contend that risk by its own definition does not exist, rather it is an unrealized potentiality which is fulfilled when it is measured by researchers or observed by lay populations. Risk embodies the "potentiality for a negative occurrence which must be understood for the

[21] The six categories of risk include environmental risks such as pollution, radiation and chemical contaminants; lifestyle risks such as those linked to the consumption of food and drugs; medical risks related to medical care and treatment; interpersonal risks related to personal relationships, sexuality and gender roles; economic risks including under- and unemployment; and criminal risks as a result of being a participant in or potential victim of illegal activities (Lupton, 1999: 13-4). For the purposes of this research, I am primarily concerned with the category of environmental risks and their relationship with health outcomes.

purposes of avoidance or control" (Rigakos and Law, 2009: 80). While realists tend to agree about the epistemology of risk as rooted in science and real in existence, there is discord when considering the specific nature of this reality, the ontology of risk and potential involvement of social and cultural dynamics (Rigakos and Law, 2009). Lupton (1999) offers a continuum which demonstrates the epistemology of approaches to risk. The perspectives based in the natural sciences and biomedicine have a realist epistemology in which risk is an objective hazard that can be measured independently of social and cultural processes. In contrast, sociocultural perspectives often frame the discussions of risk "by identifying underlying cultural structures, hierarchies and categories that serve to define risk knowledges and practices" (Lupton, 1999: 25-26). Thus, in several of these perspectives, risk is considered to be more of a subjective phenomenon than an objectively measurable one. Finally, the risk society perspective views risk as an objective and real hazard that is mediated, perceived and responded to through social, cultural and political processes (Lupton, 1999: 35).

Beck (1992) combines objectivism and cultural relativism in his approach to risk. He views risks as real in existence but points to the weakness of an objectivist, realist approach founded in the natural sciences because in a quest for objectivity, it fails to recognize the ways in which 'scientific facts,' like other perspectives on risk, are "situated and interpreted in cultural and political contexts" (Lupton, 1999: 60). A cultural relativist approach emphasizes the contextual aspects of risk responses and recognizes that what concerns a particular social group in a specific historical context may not concern another. However, Beck (1992) argues that such an approach fails to recognize

the unique nature of contemporary environmental risks in western society. Thus he seeks to integrate both perspectives into a sociological approach to risk which incorporates a scientific objectivist perspective recognizing that risks do exist, and a cultural relativist perspective which recognizes that nature and causes of risk are conceptualized differently in contemporary western societies than in previous eras (Lupton, 1999: 61).

According to the risk society perspective as theorized by Beck, risk is viewed as the probability of physical harm due to technological processes and as a systematic way of dealing with hazards and insecurities induced and introduced by modernization itself (Beck, 1992: 4, 21). The risk society perspective describes a phase of development in society in which the social, political, ecological, and individual risk created by the momentum of innovation increasingly elude the control and protective institutions of industrial society (Beck, 1992, 1995). Unlike the risks of early industrialism, contemporary nuclear, chemical, ecological, and biological threats are unlimited across both space and time, as they cross international borders and have the potential to affect future generations (Beck, 1992). Therefore, risks are more difficult to calculate, manage and avoid than in past eras (Lupton, 1999).

Beck (1992) and Giddens (1990) argue that contemporary society is characterized by a critique of the processes of modernity, and thus industrial society itself. This society is no longer unproblematically viewed as producing "goods," such as wealth and employment, but is now seen to produce many of the dangers from which we feel threatened, including environmental pollution and contaminants. The production and management of risk is framed as a human responsibility and the central institutions of

contemporary society, including government, industry and science, are singled out as the main producers of risk (Beck, 1992, 1995, 1996; Giddens, 1990). Beck (1995) reserves the term "risk society" for the contemporary era and notes distinct features of risk in late industrialism compared with pre- and early industrialism. One significant difference is that the type of risk, including environmental contamination and radiation, differs in contemporary societies than in previous eras. Since the Second World War contemporary western societies have been confronted with threats to human life on an unprecedented and previously unknown scale. Beck (1995) contrasts the calculability of risk with those from pre-industrial eras which included plague and famine, but also magic, gods and demons which were incalculable as they were believed to be caused by external and supernatural causes. During early industrialism, risks became calculable through the instatement of insurance and compensation schemes (Beck, 1995). However, the modernist rules of causation and the processes of risk calculation fail in the risk society as contemporary risks may be minimized through technology but it is not possible to eliminate the risk entirely (Beck, 1995: 76-77; Lupton, 1999).

The transition into a period of threats to social, economic and political order is presented as a challenge to the present and future, and as a justification of the risk society itself. The entry into the risk society occurs at the moment when hazards which are now decided and produced by society undermine the established safety systems of the state's existing risk calculations (Beck, 1996). In the past risks were traced to a lack of hygienic technology, such as in the case of noxious fumes in 19th century London sewers. Interestingly, today many hazards are both imperceptible to the senses and are a result of

industrial overproduction. There are risks associated with modernization itself and because of the continually evolving forms of technology, the calculability of the consequences of risk becomes impossible (Beck, 1992). Unlike the risks of early industrialism, contemporary nuclear, chemical, ecological, and biological threats found in the risk society are i) not limitable, either spatially or temporally; ii) not accountable according to the prevailing rules of causality, guilt and liability; and iii) neither compensable nor insurable (Beck, 1995: 2; 1996: 31). The known and unintended consequences in the risk society have emerged as previously unknown entities in history and western society (Beck, 1992).

The risk society describes a period of time in which the hazards produced in the growth of industrial society become predominant (Beck, 1996: 28-29). The risk society constitutes "the end of the antithesis between nature and society" so that nature can no longer be understood separately from society and contemporary cultural activity or society from nature (Beck, 1992: 80; Adam, 1996). These risks include radioactivity, which completely evades human perceptive abilities, as well as toxic substances and pollutants in the air, water and food sources, and their short-and long-term effects on plants, animals and people. The risks produce

> systematic and often irreversible harm, generally remain invisible, are based on causal interpretations, and thus initially only exist in terms of the (scientific or anti-scientific) knowledge about them. They can thus be changed, magnified, dramatized or minimized within knowledge, and to that extent they are particularly open to social definition and construction. Hence the mass media and the scientific and legal professions in charge of defining risk become key social and political positions (Beck, 1992: 23).

Beck (1996) identifies two phases when considering industrial society and the risk society. The first phase involves the systematic production of self-endangerment and its consequences but which are not a topic of public debate or political conflict. This scenario is altered when the hazards of industrial society dominate public, political and private debates. At this time, the institutions of industrial society, including government, science and industry, produce and legitimize hazards which they cannot control. Industrial society sees and criticizes itself as a risk society. Society still makes decisions and acts on the pattern of the old industrial society; however, at this time debates and conflicts which originate in the dynamic of the risk society are now being applied to interest organizations, the legal system and politics (Beck, 1996: 27-28). It is important to note that the risk society is still at the same time an industrial society because it is mainly industry, in conjunction with science, that is involved in the creation of the risks involved in the risk society (Beck, 1992: 3).

Three observations have been made about the risk society in particular. The physical risks are always created within social systems, such as organizations and institutions which are supposed to manage and control the risky activity. Therefore, the magnitude of the physical risks is a direct function of the quality of social relations and processes. The primary risk is social dependency upon institutions; these institutional actors may be inaccessible to the people affected by the risks in question (Beck, 1992: 4). Giddens (1990) also sees modern institutions as playing a key role in the risk and uncertainty associated with contemporary western societies. He points to both the pace and scope of change as unique to this time period. The rapidity of change in conditions is

extreme and demonstrated in the case of technology which is pervasive and reflected in the global nature of risks (Giddens, 1990: 6). These risks negate the standard separation between past, present and future and create an uncertainty of the implications for future generations (Adam, 1996).

The aspects of risk related to value-laden social constructs create an appropriate venue for reflexive inquiry (Rigakos and Law, 2009). The concept of reflexive modernization may be introduced when considering the stages of industrial society, the risk society and their consequences. The concept of risk is "directly bound to the concept of reflexive modernization" (Beck, 1992: 21). The shift towards reflexivity is an unintended side effect of the contemporary industrialized society and the risks produced. It is the "process of modernity coming to examine and critique itself" (Lupton, 1999: 66). Reflexive modernization does not signify reflection, but rather self-confrontation with the consequences of the risk society that cannot adequately be addressed and overcome in the system of industrial society. The risks cannot be measured by industrial society's own institutionalized standards (Beck, 1996: 28). The concept of risk is linked to reflexivity because "anxieties about contemporary risks pose questions about current practices" (Lupton, 1999: 66). The risk society becomes reflexive through processes including the awareness of the global nature of risk triggering new impulses towards the development of co-operative international institutions and the boundaries of the political being removed, leading to worldwide alliances (Lupton, 1999: 66).

Through these processes, the risk society becomes a "world risk society" where the public sphere of debate and action is globalized (Lupton, 1999). Processes of

globalization, including the pervasiveness of technology are unique to the late 20th and early 21st centuries and connect diverse populations creating a world risk society. The historical dualistic discussions of nature and culture and people's relation to environments indicate that nature is separate from cultural activity. Traditional social science understandings of nature and culture are impacted by the dissolution of the boundaries between people and their physical environments, as well as geographical boundaries in the risk society (Adam, 1996: 89-90). The risks produced through industrial processes are not just environmental problems but represent an institutional crisis as the institutions in which the public places its trust, including government, industry and science, fail to protect our health (Beck, 1995). Beck (1995: 2) argues that "[t]hreats are produced industrially, externalized economically, individualized juridically, legitimized scientifically, and minimized politically." In this view of the world risk society, there is a global citizenship in which traditional means of defining identity linked to locality are exchanged for a focus on the world-wide perspective, as environmental risks are an invisible reality and create a global future and common experience regardless of geographical location (Adam, 1996; Lupton, 1999).

The globalization of risk creates far-reaching consequences. Both Beck (1992) and Giddens (1990) contend that the nature of globalized risks does not respect the class divide or geographical boundaries of the world. In an elimination of the "other," the nature of globalized risk transcends social and economic considerations. It is possible to frame risks such as radioactivity, nuclear technology and toxic substances as not respecting geographical boundaries and affecting the global population regardless of

location or socioeconomic status. The pervasiveness of environmental risks and the geographical span demonstrates that locally-produced risks can result in globally-produced consequences (Mythen, 2004: 32). Unlike Beck, Giddens (1990: 125-26) does acknowledge that risks are "differentially distributed between the privileged and the underprivileged," but neither proponent of the risk society provides an in-depth analysis of how issues of class may still be prevalent in the risk society.

The increased social awareness of the detrimental impact of human practices on the environment leads to the social cognition and environmental impact of risk becoming a global issue. This leads to Beck's claim that risks within the risk society dissolve hierarchies of class and geography (Mythen, 2004: 32). However, this does not allow for the recognition that these global risks often affect already marginalized or historically-oppressed populations disproportionately and the risks are experienced in profoundly different ways. Marshall (1999: 269) observes that historically, corporations have located hazardous industries in communities of low socioeconomic status, choosing "the path of least resistance." In fact, the environmental justice and environmental racism literature situated in the social sciences demonstrates that communities of low socioeconomic status are systematically and disproportionately affected by technological hazards such as toxic contamination, oil spills and radioactive waste storage (Marshall, 1999; Scholsberg, 2007). The unequal distribution of environmental risks cannot be adequately understood through a framework which emphasizes one factor to the exclusion of other relevant factors (Brulle and Pellow, 2006). Environmental justice is the first framework to explicitly link environment, race, class, gender, and social justice concerns and the

disproportionate burden faced by at-risk populations[22] (Taylor, 2000: 42). A critique grounded in environmental justice is able to consider the lived experience and experiential knowledge of those most affected by the risks which is clearly missing from the risk society. Proponents of environmental justice in social movements call for i) equity in the distribution of environmental risk; ii) acknowledgement of the diversity of participants and experiences of affected communities; and iii) participation in the political processes which create and manage environmental policy and thus, assess and manage the associated risks (Scholsberg, 2007: 517). Environmental breast cancer activists' efforts to

> integrate the needs of socially and economically marginalized women into the environmental breast cancer movement have not only broadened the movement's demographic base, however, but also highlight the ways in which gender, race, and class shape understandings of the environmental breast cancer problem, the strategies for addressing it, and disease prevention efforts more generally (Ley, 2009: 138).

The intersection of sex and gender with other determinants of women's health is of particular relevance when considering potential outcomes related to environmental health, contaminants and breast cancer (Hankivsky et al., 2010).

The risk society perspective offers an overarching theoretical framework for this research that acknowledges risks and a causal relationship with environmental health. However, there are important gaps in this perspective and the framework is augmented with environmental justice literature which allows for a broader consideration of the

[22] For additional discussion of environmental justice and health concerns, refer to Brown et al. (2012), Buzzelli (2008), Dhillon and Young (2010), Fletcher (2003), Hoover et al. (2012), MacDonald and Rang (2007), and Scott (2009a).

globalization of risks, the politics of risks and hazards, and the pitfalls associated with the individualization of risk and illness (Hess, 2004).

Beck (1995) distinguishes two stages in the ecological conflict in the risk society. The first stage is a struggle to uncover the risks and their environment and health implications which must be exposed despite industrial expansion and progress. The second stage occurs when knowledge about the risk is accepted in principle, but there is no remediation and thus a conflict surrounding issues of accountability arises. Beck (1995: 8) contends that

> the ecological issue, considered politically and sociologically, focuses at heart on a *systematic, legalized violation of fundamental civil rights*—the citizen's right to life and freedom from bodily harm. This violation is not going on incidentally, accidentally, or individually, but in broad daylight, as part of the development of industry, prosperity, and technical rationality in the glare of the mass media and in an alert democracy of citizen's groups (emphasis in original text).

Beck (1992: 71) suggests that if risks are not recognized scientifically then they do not exist legally, medically, technologically, or socially and subsequently are not prevented, treated or compensated for.

Conclusions

The theoretical framework for this research draws from seemingly disparate bodies of literature and concepts including sex- and gender-based analysis, social movement theory, and the risk society perspective. In my view, each is necessary for conducting an effective analysis of Canada's body of law, policy and practice related to toxic substances, and for prioritizing a primary prevention approach to breast cancer. First, breast cancer is a disease that primarily affects women, and there is a growing body of evidence that suggests at least some of its incidence is related to endocrine disrupting

chemicals understood to affect bodies in ways profoundly influenced by sex and gender considerations. The fact that sex- and gender-based analysis has not been a central feature of the law, policy or practice governing the regulation of toxics justifies its inclusion into the analytical approach taken here. Second, the culture of cancer prevention and environmental risk which emerged in San Francisco and the related and widespread environmental breast cancer movement has shaped the way that the law, policy and practices related to toxic substances have been understood. By acknowledging the risks associated with everyday exposures to toxic substances and the associated detrimental health outcomes, including the development of breast cancer, and by making these political issues, those social movements have generated a widespread call for a shift away from the dominant epidemiological paradigm of breast cancer. And third, the influence of the biomedicalization regime has resulted in all women being framed as permanently at risk for developing the disease and as responsible for their own health outcomes. This "risk role" can be understood through the risk society perspective which acknowledges the unique nature of contemporary risks which are produced and managed through social, cultural and political factors.

There are three axes along which struggles for a paradigm shift against the dominant epidemiological paradigm occur and which reflect the varying levels of prevention, risk factors and public participation in research.[23] The first axis is concerned

[23] Brown et al. (2006) argue that challenges to the breast cancer dominant epidemiological paradigm are located primarily within the United States as a result of the strength of the breast cancer movement and particularly the environmental breast cancer movement. However, I would suggest that the challenges to the dominant epidemiological paradigm are also occurring in other western countries, including Canada.

with the level of prevention and whether the focus of research involves treatment, intervention or prevention (Brown et al., 2006). Degrees of prevention include primary, secondary and tertiary prevention and are well-utilized in the field of health promotion. Primary prevention promotes the prevention of disease among specific populations and is most relevant for this research in its potential to truly prevent disease from a public health context. In an environmental health framework, these strategies would include the objective of reducing human exposure to environmental contaminants and are consistent with the efforts of the culture of action of cancer prevention and environmental risk (Brown et al., 2006: 511-12; Klawiter, 2008a). Secondary prevention efforts promote access to screening measures, early detection of disease and timely intervention. For breast cancer, measures of secondary prevention include breast self-examination, biopsy and mammography (Brown et al., 2006: 512). Aspects of secondary prevention efforts reflect the efforts of both the culture of early detection and screening activism and the culture of patient empowerment and feminist treatment activism (Klawiter, 2008a). Finally, tertiary prevention efforts attempt to minimize the health effects of disease. Efforts of tertiary prevention in breast cancer involve the traditional interventions including surgery, radiation, chemotherapy, and medication (Brown et al., 2006: 512).

While the first axis focuses on health interventions and levels of prevention, the second axis focuses on research itself, both at the level of the individual and the community. The traditional biomedical approach to disease focuses on individual risk factors, including biological, genetic and lifestyle factors. In contrast to an approach which focuses on individual characteristics, models from health promotion, political

economy of health, and social production of disease question how economic, political and environmental factors may influence health outcomes. In this framework, disease prevention occurs through changes in industrial production practices rather than individual behaviour or forms of medical treatment. A more broadly-based population approach focuses on the relationship between bodies and macro-level structures and asks why some groups of women have higher incidence rates of breast cancer than other groups. These considerations are not accounted for in an individual approach to breast cancer (Brown et al., 2006: 517-18). Brown et al. (2006: 518) contend that "[i]n terms of intervention, the population based approach is more radical because it implies the need for mass environmental control measures or the alteration of socioeconomic norms that give rise to widespread hazardous exposures and collective behaviors that enhance the vulnerability of certain communities to disease."

The third axis within the struggle for a paradigm shift in the breast cancer dominant epidemiological paradigm pertains to the involvement of lay activists in the research process. Brown et al. (2006) use a continuum to illustrate this process. On one end of the continuum, research is conducted independently, and laypersons may participate as subjects in a study but without the possibility of contributing to the research questions, methodology, data analysis, or the dissemination process. On the opposite end of this continuum, laypersons are actively involved and collaborate in the research process by providing important and substantive contributions. The "magnitude of lay involvement in breast cancer research signifies the broad societal importance of the

disease itself and is representative of campaigns for public representation in other illnesses as well" (Brown et al., 2006: 525).

Breast cancer is an important area of research for developing critical theory, policy and practice (Wilkinson, 2007). Breast cancer in contemporary society has distinct similarities to the disease a hundred years ago. In both time periods, the medically accepted forms of treatment carry significant risks, and cannot offer a guaranteed cure for the disease. Similarly, in both time periods, there are concerns surrounding the effectiveness of disease prevention (Leopold, 1999). Considering the history of breast cancer clearly demonstrates how something that appears to be an objective concept is influenced by cultural factors, and the influence of contemporary beliefs about gender, the mind, bodies, and personal responsibility has implications for discussions of illness (Jasen, 2002: 42).

It has become clear from sociocultural perspectives on risk that understandings of disease and health cannot be separated from the social and political contexts in which they arise (Nash, 2006). Risk is a pervasive concept related to human existence in contemporary western societies and is associated with notions of choice, responsibility and blame (Lupton, 1999). In the risk society,

> [r]isks lie across the distinction between theory and practice, across the borders of specialities and disciplines, across specialized competences and institutional responsibilities, across the distinction between value and fact (and thus between ethics and science), and across the realms of politics, the public sphere, science and the economy, which are seemingly undivided institutions (Beck, 1992: 70).

The incidence of harm related to toxic substances in the risk society is "not only significant, intentional, and expected, but [is] also...inherent to our practices of

production and consumption" (Scott, 2008: 296). Therefore, environmental health issues are so strongly contested because they are so intricately linked to the production and consumption processes in contemporary western society (Beck, 1992; Brown, 2007).

Definitions of illness are continually shifting and evolving with social forces playing an integral role in the social construction of illness (Shriver, White and Kebede, 1998). The acceptance of environmental causation of disease is further complicated by issues of uncertainty and the problems of knowing which are consistent themes in discussions of contested illnesses, including breast cancer. Ley (2009: 36-37) contends that the issue of uncertainty creates difficulty in calling for more protective environmental policies within a regulatory system that "demands proof of harm before taking action." Contemporary environments are filled with manufactured risks created by corporations and government which are difficult to measure, predict and control (Brown, Kroll-Smith and Gunter, 2000; Giddens, 1990). The inherent uncertainty associated with these risks is grounded in the interests of those responsible for their production. As Adam (1996: 97) argues, "insistence on certainty and 'proof' for situations characterized by indeterminacy, unpredictability and multiple time-lags is central to much of the political complacency about environmental problems." There are problems associated with the i) rapid changes occurring in the contemporary environment, and ii) the limited capacity of experts and their systems for fully assessing and evaluating these changes (Brown, Kroll-Smith and Gunter, 2000). The latency period between exposure and identifiable symptoms could be months, years or even decades which complicates the question of proof with respect to the causal connection between toxic substances and illnesses such

as breast cancer. This temporal gap, combined with the mobility of individuals in contemporary society, makes it difficult to connect symptoms with particular locations and exposures and further complicates attempts to challenge contemporary assumptions about the separation of bodies and contaminated environments (Nash, 2006: 181).

Brown, Kroll-Smith and Gunter (2000) provide some important considerations surrounding the uncertainty related to environmental health controversies and contested illnesses. There is uncertainty surrounding the body's past exposures to potentially hazardous environments, the potentially synergistic effects, and the lack of a history of exposure during interactions with the medical profession. There has traditionally been a great deal of uncertainty around the low-dose response relationship in toxicology and the difficulty producing data about the effects of chronic, low-level toxic exposures on human health (Brown, Kroll-Smith and Gunter, 2000). However, a number of recent publications focus on the health effects of low-dose exposures to toxic substances, and endocrine disrupting chemicals in particular. When the original *State of the Science of Endocrine Disrupting Chemicals* report was published in 2002 (WHO, ILO and UNEP, 2002), the evidence linking endocrine disrupting chemicals to human health outcomes was described as "weak" (Bienkowski, 2013a). The state of the science has evolved considerably in the past ten years and the newly published report concludes that endocrine disrupting chemicals "have the capacity to interfere with tissue and organ development and function, and therefore may alter susceptibility to different types of diseases throughout life. This is a global threat that needs to be resolved" (UNEP and WHO, 2012: xv).

Similarly, the European Environment Agency has developed a working definition of the precautionary principle since the publication of the first *Late Lessons from Early Warnings* report in 2001 (EEA, 2001) to reflect advances in science and research over the past decade and recognizing the implications of toxic substances for the environment and human health.

> The precautionary principle provides justification for public policy and other actions in situations of scientific complexity, uncertainty and ignorance, where there may be a need to act in order to avoid, or reduce, potentially serious or irreversible threats to health and/or the environment, using an appropriate strength of scientific evidence, and taking into account the pros and cons of action and inaction and their distribution (EEA, 2013: 681).

Finally, regarding the toxicology concept that "the dose makes the poison" in the traditional dose-response relationship, recent research has demonstrated that low-dose exposures of endocrine disrupting chemicals can have effects that are not predicted at higher doses (Vandenberg et al., 2012).

The final uncertainty described by Brown, Kroll-Smith and Gunter (2000: 11) involves problems associated with diagnosis as the authors suggest that physicians often do not possess the technology or knowledge to determine a causal link between exposure to environmental contaminants and a specific disease. While this uncertainty still exists and environmental links to health remain contested, there is a clear need for precaution and prevention of disease, especially in the context of women's health. The increasing number of chronic diseases in contemporary society, including breast cancer, cannot be adequately addressed within the individualist paradigm for the management of infectious diseases (Davis and Webster, 2002). Breast cancer is clearly influenced by sociocultural, political, economic, and environmental factors and advocating for increased research

without changing the environmental regulatory system is not enough to protect women's health (Ley, 2009: 82).

Chapter 3

The History of Environmental Health Policy in Canada

Introduction

This chapter will provide a descriptive history of Canadian policy related to environmental health drawing from federal legislation including the *Environmental Contaminants Act* and the *Canadian Environmental Protection Act*; government publications from the 1970s to the present, including Environment Canada and Health Canada; and grey literature from environmental and health organizations. This chapter considers the evolution of legislation and public health policy designed to protect Canadian citizens from exposure to toxic substances and the associated adverse health outcomes.

The chapter begins its overview of the history of health policy in Canada with the influential Lalonde Report written in 1974, one of the first policy documents to recognize the interacting influences on health outcomes, including the environment. It then moves to the *Environmental Contaminants Act*, which was the first piece of federal environmental legislation, followed by the the *Canadian Environmental Protection Act, 1988*. Here, an extensive review of the Act was paralleled by the implementation of the *Toxic Substances Management Policy*. This is followed by a discussion of the revised *Canadian Environmental Protection Act* which received Royal Assent in 1999, its review process, and the 2006 *Chemicals Management Plan*. The *Chemicals Management Plan* is the most recent tool for the assessment and management of toxic substances and the risks to the environment and human health. The detailed policy history that follows provides

the foundation necessary for the more in-depth and critical analysis in chapters four and five which examines the relationship between theory and practice in Canada's regulatory regime for toxics and the potential for protecting Canadian women's health, their risk for developing breast cancer and the potential for preventing the disease.

An Overview of the History of Health Policy in Canada

As discussed in chapter two, issues of health, especially those related to breast cancer and disease regimes, have historically been viewed as private matters rather than of public concern. Health issues were regarded primarily as the responsibility of the family and possibly charitable or religious institutions, whereas government intervention was limited. From 1867 to 1919, the Department of Agriculture was responsible for any health-related concerns in Canada (Ham, 2001). The first federal health department was established in 1919 and reconstituted in 1993 during which time its responsibilities included conducting public health studies, the regulation of food and drugs, the inspection of medical devices, the administration of health care insurance, and the dissemination of general information services related to health conditions and practices (Maioni, 2004; Miller Chenier, 2002).

A working document was published in 1974 which is frequently cited as revolutionizing understandings about health, identifying the need for intersectoral collaboration, and acknowledging the importance of multiple interventions in order to properly address the determinants of health (Canadian Population Health Initiative, 2002; Glouberman and Millar, 2003). Marc Lalonde, Minister of National Health and Welfare under the Liberal government, wrote *A New Perspective on the Health of Canadians*

which focused on the state of Canadians' health and proposed a new approach for addressing health outcomes (Lalonde, 1974). The health status of the Canadian population was framed as one of the significant problem areas with health outcomes including life expectancy, rates of mortality and morbidity, and causes of death (Lalonde, 1974: 19). Due to the lack of consensus regarding an established conceptual framework in the analysis of health, Lalonde (1974: 31) proposed the utilization of a "health field" concept that was developed by considering the underlying factors associated with the health status of Canadian citizens. The health field includes four broad elements and is proposed as a tool for the analysis of health problems, as well as determining the health needs of Canadians and how those needs might be properly addressed.

The first element proposed by Lalonde (1974: 31) is *human biology*, "all those aspects of health, both physical and mental, which are developed within the human body as a consequence of the basic biology of man and the organic make-up of the individual" (Lalonde, 1974: 31). Human biology is linked to a variety of health issues including genetic disorders and chronic diseases such as diabetes, arthritis and cancer. The second element utilized in the health field concept is the *environment* which involves "matters related to health which are external to the human body and over which the individual has little or no control" (Lalonde, 1974: 32). It was recognized at this time that health status can be impacted by both social and physical environments and that individuals cannot prevent health hazards associated with the pollution and contamination of air, water and food supplies (Lalonde, 1974).

The third category of the health field involves the *lifestyle* of individuals and the decisions and behaviours which impact their health status. The language used in the discussion of lifestyle factors is contradictory: placing blame on the individual for creating self-imposed risks which may contribute to illness or death such as smoking cigarettes and consuming alcohol, while simultaneously labelling the individual as a "victim" (Lalonde, 1974: 32). The final category within the health field involves the *health care organization* which consists of the "quantity, quality, arrangement, nature and relationships of people and resources in the provision of health care" (Lalonde, 1974: 32). The health care organization is more commonly referred to as the health care system and includes related institutions, professionals, practices, and treatments (Lalonde, 1974).

Prior to the 1970s most efforts to improve health status in Canadian society and the majority of direct health expenditures focused on the health care organization. However, as Lalonde (1974: 32) notes, the main causes of sickness and death in Canada are rooted in human biology, environment and lifestyle. A significant challenge encountered when attempting to improve the health status of the Canadian population is that the power to do so is dispersed among individual citizens, governments, health professions, and institutions. The Lalonde Report suggests that this creates fragmented responsibility and imbalanced approaches. The comprehensive nature of the health field concept allows for health problems to be traced to one or a combination of the four elements and to examine their significance and interaction (Lalonde, 1974).[24]

[24] The influential Lalonde Report and the emphasis on interacting influences on health outcomes was a precursor to the rise of research focused on the social determinants of health that emerged in the 1980s and continues to evolve in 2013 (O'Neill et al, 2007; Mikkonen and Raphael, 2010; Raphael, 2003).

At the time of the Lalonde Report, the Government of Canada was committed to pursuing two broad objectives related to health outcomes: reducing mental and physical health hazards for citizens considered to be at increased risk, and improving accessibility of good mental and physical health care for individuals that encounter barriers to accessing such care (Lalonde, 1974). In order to achieve these objectives, five specific strategies were proposed:

> 1) a *health promotion strategy* to inform, influence and assist individuals and organizations to accept additional responsibility and become active participants in matters related to mental and physical health;
>
> 2) a *regulatory strategy* to use federal regulatory powers to reduce hazards related to mental and physical health, as well as promoting similar practices at the provincial level;
>
> 3) a *research strategy* to discover and apply information related to mental and physical health problems;
>
> 4) a *health care efficiency strategy* to assist the provinces in reorganizing the delivery of mental and physical health care to address issues of cost and accessibility; and
>
> 5) a *goal-setting strategy* designed to develop goals for improving the mental and physical health status of Canadians and the efficiency of the health care system overall (Lalonde, 1974: 66).

Health promotion in Canada is rooted in the Lalonde Report's proposal that human biology, lifestyle, environment, and the health care organization have a direct influence on the health status of Canadian citizens (Health Canada, 2002a; Lalonde, 1974). The five objectives were designed to create a participatory framework where health promotion is distinguished from both health protection and disease prevention. While *health protection* efforts are concerned with maintaining health status by addressing

intermediate health threats, and *disease prevention* attempts to anticipate and avoid imminent health threats, *health promotion* moves beyond maintaining health to improving health status and focuses on long-term health gains (Health Canada, 2002a). There was a rapid growth of interventions including health education in public schools, and social marketing public awareness campaigns focused on tobacco use, exercise and healthy diets in an attempt to influence individual knowledge, attitudes and behaviours (Health Canada, 2002a; PHAC, 1997). These early health promotion initiatives focused directly on the lifestyle component and the related links between health status and personal risk behaviours (Boyce, 2002) creating a precedent for individual responsibility for health outcomes.

However, by the early 1980s there was increasing concern about the limitations of health promotion campaigns that focused solely on lifestyle, individual choices and personal behaviours. In *Achieving Health for All*, Jake Epp, Minister of National Health and Welfare under the Progressive Conservative government, utilized a population-based approach to health as a complement to the healthcare system, and to identify aspects in health policy and practice that resulted in disparities and negative health outcomes among Canadian citizens (Epp, 1986; Parliament of Canada, 2008a). Epp (1986) called for an integration of concepts from public health, health education and public policy towards health promotion in order to reduce inequities, increase prevention efforts and enhance Canadian's capacity to cope. It was argued that framing the causal relationship between lifestyle and behaviour with health outcomes does not adequately account for interacting factors that also play a significant role (Epp, 1986: 5). Instead, Epp (1986: 7)

recommended three mechanisms for effective health promotion: i) *self-care*, the decisions and actions individuals can take in the interest of their own health and well-being; ii) *mutual aid*, the actions one can take to assist others; and iii) *healthy environments*, the creation of conditions and surroundings that are conducive to good health (Epp, 1986: 7; Health Canada, 2004b). Environmental change is framed as the most complex and difficult of the three mechanisms necessary for effective health promotion. Epp (1986: 9) concluded that it is "time to clearly articulate a direction which is designed expressly to promote the health of Canadians."

The first international conference on health promotion was held in Ottawa in November 1986 and was co-sponsored by the World Health Organization, the Canadian Public Health Association, and Health and Welfare Canada. The five key strategies involved in the framework of health promotion included building healthy public policy, creating supportive environments, strengthening community action, developing personal skills, and reorienting health services (World Health Organization, 1986). A significant outcome of the conference was the publication of the *Ottawa Charter for Health Promotion* which has since become influential in the practice of health promotion both across Canada and internationally (PHAC, 1997; WHO, 1986). A perspective grounded in population health research emerged which recognized the impact of structural conditions such as poverty and discrimination on health status. Structural factors were now being considered along with environmental factors including the physical, social, cultural, and economic environments that impact the health of the Canadian population (Health Canada, 2002a). While Canada emerged as a public leader in health promotion at

this time, there was a disconnect between public health policy and emerging environmental legislation.

Environmental Contaminants Act

Unlike health protection statutes such as the *Food and Drugs Act*[25] which dates back to 1920, legislation designed to protect the Canadian environment has been developed more recently. The Department of the Environment was established in 1972 and the first piece of legislation that focused on environmental protection, the *Environmental Contaminants Act*, was promulgated in 1975 under the Liberal government.[26] The *Environmental Contaminants Act* was administered to address the environmental and health risks posed by toxic chemicals, under the rubric of "toxic substances management." It also developed a domestic response to international initiatives at the level of the Organisation for Economic Co-operation and Development (OECD) to manage the risks associated with polychlorinated biphenyls (PCBs) (Leiss, 2001; Meek and Armstrong, 2007: 592).

There was an increase in concern about the causal relationship between the environment and human health at this time. Public awareness surrounding this relationship was influenced by widespread media coverage of events such as the industrial dumpsite at Love Canal, New York in 1978; a nuclear power plant accident at

[25] The *Food and Drugs Act* was established in 1920 and focused on preventing adulteration, unsanitary production, fraudulent labelling, and subsequently licensing requirements for drugs. By 1951, pharmaceutical manufacturers were legally required to obtain regulatory approval before marketing their drugs. However, the thalidomide tragedy of the early 1960s resulted in a strengthening of Health Canada's regulatory abilities (Health Canada, 2010: 17).

[26] Environmental Contaminants Act, R.S.C. 1975.

Three Mile Island, Pennsylvania in 1979; a gas leak in Bhopal, India in 1984; and the world's worst nuclear power accident at Chernobyl, in the former USSR in 1986. A discussion of new "environmental risks," featuring aspects of collective risk, long latency periods, and irreversible impacts was distinguished from traditional environmental problems such as floods, earthquakes and tornadoes. The new risks included suspected carcinogens, mutagens and heavy metals which pose "long-term, serious threats of uncertain likelihood to health and life" (Page, 1978: 218). Researchers at the time emphasized that research findings demonstrated "that the release of certain chemicals into [hu]man's environment…[could] lead to the production of cancer, birth defects, genetic damage and a range of acute and chronic diseases" (Nemetz et al., 1981: 3).

Concern surrounding the visibility of environmental risks is consistent with Beck's (1992) later work on the risk society. It may be argued that the nature of environmental risks are visible because of the potential negative outcomes associated with a particular risk. However, Page (1978) proposes that the visibility of environmental risk is also impacted by a number of different factors and in particular ways. An important consideration is that environmental risks lack visibility when considering the potential for lengthy latency periods between exposure and health outcomes. It is suggested that the low dose concentrations of environmental pollutants result in a lack of visibility. The acknowledgement of the risk associated with the environment may also be affected by one risk receiving more attention than another, such as a recognized carcinogen over a suspected carcinogen or a contested contaminant (Page, 1978: 222-23). The identification of new environmental problems include the production of synthetic

chemicals which may be toxic, carcinogenic, mutagenic, or teratogenic. These new risks are described as being "less susceptible to management through existing regulatory, legal and economic institutions" (Page, 1978: 207-8).

The *Environmental Contaminants Act* did not require either assessment or testing of environmental contaminants for potential impact on human health or the environment prior to their release into the Canadian environment. Under the Act, if the Minister of the Environment and the Minister of National Health and Welfare believed that a substance may enter the environment in quantities or concentrations that may constitute a danger to human health or the environment, they possessed the authority to i) require commercial producers of that substance or class of substances to provide the government with notification of activities and information about the substances; and ii) require producers and importers to conduct tests which the Ministers may reasonably require (Nemetz et al., 1981: 123). Thus, industry was only required to submit testing information about environmental contaminants if the Ministers had reason to believe that a substance may enter the environment in amounts that are a danger to human health or the environment based on existing information (Meek and Armstrong, 2007).

Toner (2002: 76-77) contends that the Liberal government's lack of enforcement was a result of their "lack of political will" to challenge: i) claims from the provinces that federal regulatory efforts were a jurisdictional infringement; and ii) claims from industry stakeholders that environmental regulation would result in an undue burden and in job loss. Like other federal statutes, the *Environmental Contaminants Act* could only be meaningful and effective when specific regulations were made under it (Nemetz et al.,

1981: 124). In this context, Environment Canada lacked the resources to effectively administer the *Environmental Contaminants Act*. For instance, Environment Canada only assessed five chemicals over a period of ten years (Leiss, 2001: 202-03). A Consultative Committee was established in 1985 to review proposals to strengthen the *Environmental Contaminants Act* (Environment Canada, 2002; Environmental Contaminants Act Consultative Committee, 1986). Consistent with Page's (1978) assessment of new environmental risk problems, the legislative review determined that the *Environmental Contaminants Act* was unable to adequately address the scope of problems associated with environmental contaminants.

> Not only has the number of chemicals increased dramatically over the past 20 years or so, but so have the quantities of them that are produced. Global production of organic chemicals, for example, increased from about 1 million tonnes a year in the 1930s to 7 million in 1950, 63 million in 1970 and about 250 million in 1985. Annual production now tends to double every seven or eight years (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 22).

The Canadian Environmental Protection Act, 1988 (CEPA)

After a process involving extensive public consultation and task force recommendations, including the legislative review conducted by the Environmental Contaminants Act Consultative Committee, a bill was drafted to provide a revised approach and include multiple aspects of environmental protection under one statute. A more comprehensive approach to chemicals management was recommended in order to manage the complete life cycle of toxic substances from the "cradle to grave" (Douglas and Hébert, 1998; House of Commons Standing Committee on Environment and Sustainable Development, 1995; Toner, 2002). Bill C-74, the *Canadian Environmental*

Protection Act[27] was introduced to the House of Commons in June 1987 under the Progressive Conservative government. Extensive amendments were made to Bill C-74 in Committee over the following year and the Bill was passed and became active legislation on June 30, 1988 (House of Commons Standing Committee on Environment and Sustainable Development, 1995; McRobert and Cooper, 2000).

Lucien Bouchard, Minister of the Environment in 1988 promoted the need for a "strong federal role" in environmental protection:

> If there is a special role for the federal government, it is the development of national environmental protection standards and practices. The very nature of environmental problems demands this. Too often, the solutions adopted to control polluting emissions or hazardous waste, for example, differ from province to province…Ottawa must play a key role in the harmonization of standards and methods (Harrison, 1996: 121).

CEPA became Canada's primary legislation aimed at protecting the environment. In addition to the *Environmental Contaminants Act*, CEPA also replaced or combined environmental protection statutes including the *Clean Air Act*, the *Ocean Dumping Control Act*, and parts of both the *Canada Water Act* and the *Department of the Environment Act* into one single piece of larger legislation (Meek and Armstrong, 2007: 592). Part II of CEPA created a regulatory regime that allowed the Government of Canada to control toxic substances, including processes of manufacturing, importation and disposal. One of CEPA's guiding principles is the management of pollution, and Environment Canada and Health Canada[28] became jointly responsible for the risk

[27] Canadian Environmental Protection Act, R.S.C. 1988; herein after described as CEPA.

[28] Health Canada promotes its commitment to improving the lives of Canadian citizens and to making Canada's population among the healthiest in the world in terms of longevity, lifestyle and effective use of the public health care system (Health Canada, 2008a: 1). The objectives of the federal department include

assessment and management of toxic substances (Health Canada, 1995: 19). A substance is defined in section 3 as any distinguishable kind of organic or inorganic matter, whether animate or inanimate. Under section 11, a substance will be considered toxic if it enters or may enter the environment in a quantity or concentration under conditions that i) have or may have an immediate or long-term effect on the environment; ii) constitute or may constitute a danger to the environment on which life depends; or iii) constitute or may constitute a danger in Canada to human life or health. The inclusion of the word "may" when considering the danger to human life or health reflects a change in the language which allows for the potential of harm from that used in the *Environmental Contaminants Act*.

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preventing and reducing risks to the individual health of Canadians and the overall environment; promoting healthier lifestyles; ensuring high quality health services that are both efficient and accessible; integrating renewal of the health care system with longer term plans in the areas of health prevention, promotion and protection; and reducing health inequalities to help Canadian citizens make informed decisions about their health (Health Canada, 2007b). Health Canada (2008a: 31-3) offers four strategic outcomes that guide their attempt to provide long-term benefits to Canadians:

i) *An accessible and sustainable health system responsive to the health needs of Canadians* in order to promote the national coordination and development of a knowledge base to address health and health care priorities. Health Canada seeks to facilitate health system adaptation towards change in technology, society, industry, and the environment in order to protect Canadian citizens from health risks and provide access to quality health care;

ii) *Access to safe and effective health products, food and information for healthy choices* to protect the health and safety of Canadian citizens. Scientific and technical expertise is emphasized in research conducted to contribute to evidence-based decision-making and regulation. Evidence-based decision-making has gained increased attention within the health policy environment and Health Canada seeks to advance evidence-based policy and regulatory decision-making within the department (Dobrow et al, 2004; Health Canada, 2008a);

iii) *Reduced health and environmental risks from products and substances, and sustainable living and working environments*. Health Canada aims to advance scientific research and utilize evidence-based research to develop health promotion and harm prevention programs, policies and regulations; and

iv) *Improve health outcomes and the reduction of health inequalities between First Nations and Inuit and other Canadians*. Health Canada will use science and research to accurately define health risks, trends and emerging issues related to the health status of First Nations and Inuit Canadians in order to support the effective design and delivery of health programs.

While toxicity is understood to involve the "inherent capability of a substance to cause harm" and does not include considerations of exposure, section 11 of CEPA equates toxicity with risk and the understanding that "harm to the environment or human health is a function of both the intrinsic toxicity…and the extent of exposure" (Health Canada, 1994: 2). The inclusion of the exposure component in determining if a substance is classified as toxic under CEPA means that a substance "cannot be regulated merely for having the inherent potential to cause harm; it must also be shown to be entering or likely to enter the environment at levels sufficient to cause harm" (Cooper et al., 2000: 202). Health Canada (1994: 2) finds that the definition of toxic under section 11 allows for principles of health risk assessment, but that the three risk assessment endpoints do not address any aspects of risk management including: i) a finding of "toxic" under CEPA; ii) a finding of "not considered to be toxic" under CEPA; or iii) a finding of "insufficient information to conclude whether or not the compound is toxic."

CEPA includes two broad categories of substances. Under section 25, the Minister of the Environment was required to compile a list of substances for the first category, the Domestic Substances List. The Domestic Substances List includes existing substances that were manufactured or imported into Canada in a quantity of not less than 100 kilograms in any one calendar year, or were in commerce or used for commercial manufacturing purposes in Canada between January 1, 1984 and December 31, 1986. Due to limitations in the notification and information gathering provisions of the prior legislation, the *Environmental Contaminants Act*, the majority of the 23,000 existing substances, also known as "legacy chemicals," were put into the marketplace without any

risk assessment that evaluated them for potential detrimental effects on human health and the environment (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 22).

CEPA also required the Minister of the Environment to compile a list of substances which were new to Canadian society and commerce after 1986 and were not part of the Domestic Substances List. The Non-Domestic Substances List is based on the United States Environmental Protection Agency's *Toxic Substances Control Act* (TSCA) Chemical Substances Inventory for 1985 and includes more than 58,000 substances (Environment Canada, 2010b). It required that an assessment be conducted on all new substances for their potential impact on human health and the environment before their introduction into the Canadian market (House of Commons Standing Committee on Environment and Sustainable Development, 1995). Substances on the Non-Domestic Substances List are subject to less onerous notification requirements than the Domestic Substances List (Lucas, 1999; Environment Canada, 2010b).

Both the Domestic Substances List and Non-Domestic Substances List were published in a Supplement of the *Canada Gazette* on January 25, 1991 (Lucas, 1998: 155), and the Domestic Substances List was published in Part II, the Official Regulations of the *Canada Gazette* on May 4, 1994 (Health Canada, 2003a: 22). Under section 33, if a substance is determined to be toxic by meeting the requirements outlined in section 11, the Minister of the Environment and Minister of Health can recommend to the Governor in Council that the substance be placed on the Toxic Substances List in Schedule 1 of the Act. Twenty-six substances were originally placed on the Toxic Substances List under

CEPA, including asbestos, benzene, mercury, lead, and chlorofluorocarbons (CFCs) (Environment Canada, 2010c; Health Canada, 1994).[29]

Section 12 of CEPA required that the Minister of Health and Minister of the Environment establish a Priority Substances List which "identifies substances to be assessed on a priority basis to determine whether they are toxic" under CEPA and where the substances pose a risk to the environment or the health of Canadians (Environment Canada, 2011a). A priority substance may involve a chemical, a group or class of chemicals, effluents, or wastes (Environment Canada, 2011a). The requirements of the Priority Substances Assessment Program include an in-depth assessment of the substance to determine the risks to the environment and human health. The assessment reports must include the characteristics of the substance, how it enters the environment, and the effects of and risks to human health and the environment as a result of exposure to the substance (Environment Canada, n/da). A report and summary must be published in the *Canada Gazette* with the decision of the Minister of Health and the Minister of the Environment regarding a whether a substance on the Priority Substances List will be listed as toxic under Schedule 1 of CEPA. The assessment must be completed within five years of publication in the *Canada Gazette* and if it has not been completed, a Canadian citizen can file a notice of objection to the Minister of the Environment requesting a review

[29] The twenty-six substances placed on the Schedule 1 List of Toxic Substances of CEPA included asbestos, 1, 1, 1-trichloroethane, benzene, bis(chloromethyl) ether, bromochlorodifluoromethane, bromofluorocarbons, bromotrifluoromethane, chlorobiphenyls, chlorofluorocarbon, chloromethyl methyl ether, dibenzo-para-dioxin, dibenzofuran, dibromotetrafluoroethane, dodecachloropentacyclo [5.3.0.02,6.03,9.04,8] decane (Mirex), fuel containing toxic substances that are dangerous goods within the meaning of section 2 of the *Transportation of Dangerous Goods Act*, 1992, hydrobromofluorocarbons, hydrochlorofluorocarbons, lead, mercury, methyl bromide, polybrominated biphenyls, polychlorinated dibenzo-para-dioxins, polychlorinated dibenzofurans, polychlorinated terphenyls, tetrachloromethane (carbon tetrachloride), and vinyl chloride (Environment Canada, 2010c).

board inquire whether the substance under consideration is toxic or capable of becoming toxic (sections 14 and 89(5)).

Environmental assessments and human health assessments were completed under the Priority Substances Assessments Program (Environment Canada, 2008). The first Priority Substances List (PSL1) was published in the *Canada Gazette* on February 11, 1989 and included 44 substances or groups of substances (Health Canada, 1994).[30] If a substance on the Priority Substances List was found to be "not toxic" under CEPA, the substance was deleted from the list as was the case for methyl tertiarybutyl ether and toluene (Lucas, 1998: 155). Twenty-five substances of the original 44 that were assessed under the first Priority Substances List met the criteria for being classified in CEPA. Based on the recommendations of a multi-stakeholder Expert Advisory Panel, those 25 substances were added to the second Priority Substances List (PSL2) of CEPA (Environment Canada, n/da; Environment Canada, 2008).

The second Priority Substances List was published in the *Canada Gazette* on December 16, 1995 and contained 25 substances or classes of substances including single

[30] The substances on the first Priority Substances List include 1,1,1-trichloroethane, 1,1,2,2-tetrachloroethane, 1,2-dichlorobenzene, 1,2-dichloroethane, 1,4-dichlorobenzene, 3,3'-dichlorobenzidine, 3,5-dimethylaniline, benzene, benzidine, bis (2-chloroethyl) ether, bis (2-ethylhexyl) phthalate, bis (chloromethyl) ether, chlorinated paraffins, chlorinated wastewater effluents, chlorobenzene, chloromethyl methyl ether, creosote-contaminated sites, dibutyl phthalate, dichloromethane, di-n-octyl phthalate, effluents from pulp mills using bleaching, hexachlorobenzene, hexavalent chromium compounds, inorganic arsenic compounds, inorganic cadmium compounds, inorganic fluorides, methyl methacrylate, methyl tertiary-butyl ether, organotin compounds, oxidic, sulphidic and soluble, inorganic nickel compounds, pentachlorobenzene, polychlorinated dibenzodioxins, polychlorinated dibenzofurans, polycyclic aromatic hydrocarbons, refractory ceramic fibre, styrene, tetrachlorobenzenes, tetrachloroethylene, trichlorobenzenes, trichloroethylene, toluene, used crankcase oils, and xylenes (Environment Canada, 2008).

chemicals, mixtures and effluents.[31] Environment Canada and Health Canada have completed risk assessments that consider the impact on both the environment and human health for the second Priority Substances List. The draft assessment reports are available for a 60-day comment period to the public and then revised and a final copy is published with the determination of whether a substance is considered to be toxic under CEPA (Environment Canada, 2006). If a substance is determined to be toxic under CEPA, the Minister of the Environment and Minister of Health can choose from risk management control options including environmental quality or releases, guidelines, codes of conduct, or specific regulations controlling the release, handling, storage, transportation, or disposal of a toxic substance. The proposed regulation must be published in Part I of the *Canada Gazette* (Lucas, 1998: 156).

There is a legislative requirement that the Government of Canada must review CEPA every five years under a process that involves public consultation (section 139 of the Act). The House of Commons Standing Committee of Environment and Sustainable Development was tasked with conducting the first review of CEPA on June 10, 1994. The five years of the review period are described as being "characterized by highly charged tension among champions of health, environment, labour and other public

[31] The substances on the second Priority Substances List include acetaldehyde, acrolein, acrylonitrile, aluminum chloride, aluminum nitrate and aluminum sulphate, ammonia in the aquatic environment, 1,3-butadiene, butylbenzylphthalate (BBP), carbon disulfide, chloroform, N,N-dimethylformamide (DMF), ethylene glycol, ethylene oxide, formaldehyde, hexachlorobutadiene (HCBD), inorganic chloramines, 2-methoxy ethanol, 2-ethoxy ethanol, 2-butoxy ethanol, N-nitrosodimethylamine (NDMA), nonylphenol and its ethoxylates (NPE), phenol, releases from primary and secondary copper smelters and copper refineries, releases from primary and secondary zinc smelters and zinc refineries, releases of radionuclides from nuclear facilities (effects on non-human species), respirable particulate matter less than or equal to 10 microns, road salts, and textile mill effluents (Environment Canada, 2006).

interests, the government, and the regulated chemical and other affected industries" (Kwasniak, 1999).

Toxic Substances Management Policy

During the CEPA review period, the *Toxic Substances Management Policy* was developed by the Liberal government after consultations with stakeholders held between September 1994 and April 1995 (Environment Canada, 1995). The *Toxic Substances Management Policy* was released in June 1995 and is still operational today. According to Environment Canada (1995: 1), it is designed around a "preventive and precautionary approach to deal with all substances that enter the environment" which may negatively impact the environment or human health. This policy is intended to guide regulatory and non-regulatory programs within federal jurisdiction, and is designed to help determine the risk assessment and management processes for toxic substances in Canada. The risk assessment process under the *Toxic Substances Management Policy* estimates the degree and likelihood of adverse effects as a result of exposure to a toxic substance in the environment. Risk management under this policy involves selecting and implementing management options around a particular risk associated with toxic substances while considering a range of legal, economic and social factors (Environment Canada, 1995: 7).

A toxic substance will be considered for systematic assessment if a federal, provincial, or international program or a Canadian citizen identifies a substance as potentially harmful to the environment and/or human health (Environment Canada, 1995). The key objectives of the *Toxic Substances Management Policy* include the assessment and management of two specific groups of substances. In order to be

classified as a Track 1 substance, the substance must meet four criteria including being toxic under CEPA, persistent, bioaccumulative, and anthropogenic. The precise details of the criteria include:

- *CEPA-toxic*: A substance is considered toxic if it meets the criteria as defined in section 11 of CEPA where a substance is entering or may enter the environment in a concentration or under conditions that i) have or may have an immediate or long-term harmful effect on the environment or its biological diversity; ii) constitute or may constitute a danger to the environment on which life depends; or iii) constitute or may constitute a danger in Canada to human life or health;

- *Persistence*: A substance can be defined as persistent in air, water, sediment, or soil where environmental persistence refers to the "length of time in environmental media and is usually defined in terms of half-life – the time required for the concentration of a substance to diminish to half its original value" (Environment Canada, 1995: 8);

- *Bioaccumulation:* A substance can be considered bioaccumulative through a process in which the "substance accumulates in a living organism either from the surrounding medium or through food containing the substance" (Environment Canada, 1995: 8); and

- *Predominantly Anthropogenic:* A substance must be primarily produced as a result of human activity as opposed to contributions to the environmental medium from natural sources (Environment Canada, 1995: 8).

It is noted that persistence and bioaccumulation ranges may vary as they are influenced by factors such as the intrinsic properties of a substance, conditions in the environment, and the ecosystem under consideration. Thus, expert judgment and weight of scientific evidence are used to determine if the four criteria are fulfilled (Environment Canada, 1995: 9).

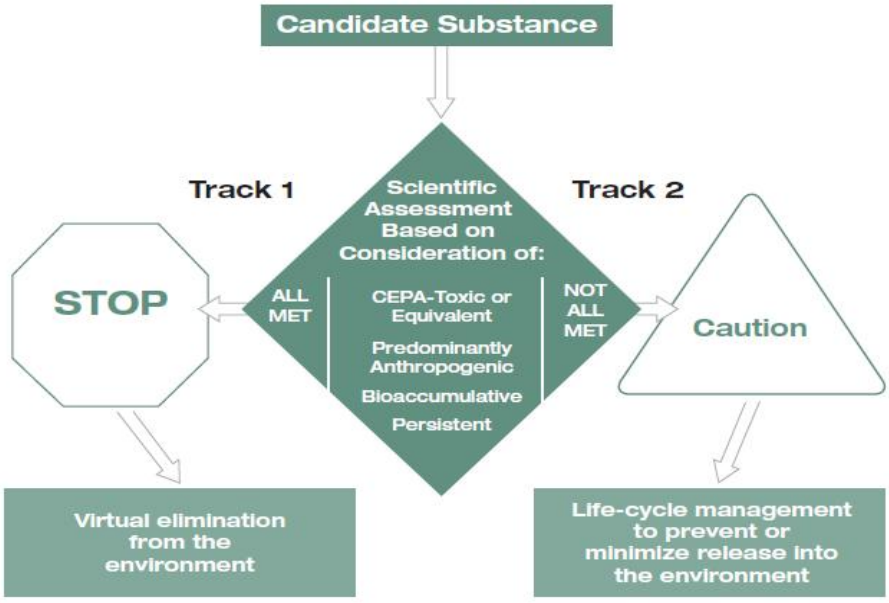
The *Toxic Substances Management Policy* proposes pollution prevention strategies to avoid the measurable release of Track 1 substances in order to minimize exposure to the environment and human health. If a Track 1 substance meets the required

criteria and cannot be adequately managed throughout its lifecycle, it may qualify for "virtual elimination." The persistence and bioaccumulation criteria for individual chemical substances cannot be used for complex mixtures or groups of substances. However, a Track 1 substance that is present in a complex mixture can be a candidate for virtual elimination if the assessment and management process accounts for this (Environment Canada, 1995: 9). The *Toxic Substances Management Policy* proposes to achieve virtual elimination by "addressing sources of release to the environment or by removing or managing the substance if it is already in the environment" (Environment Canada, 1995: 5). This policy places the onus of responsibility on the producers or users of a Track 1 substance to prove that it will not be released into the Canadian environment in measurable concentrations during its life cycle. While the policy claims the objective of the virtual elimination of a substance from the environment is established regardless of socioeconomic factors, it also clearly states that "management plans such as targets and schedules to achieve that long-term objective will be based on analyses of environmental and human health risks as well as social, economic and technical considerations" (Environment Canada, 1995: 5). Track 1 substances are to be monitored in the environment to ensure the compliance with and effectiveness of the risk management process (Environment Canada, 1995).

The second category of chemicals addressed by the *Toxic Substances Management Policy* involves Track 2 substances which do not meet the four criteria including being anthropogenic, bioaccumulative, persistent, and CEPA-toxic. In this case, rather than a virtual elimination approach, risk management for these substances includes

a “life-cycle management” approach that focuses on pollution prevention, pollution control, and remediation in order to prevent or minimize the release of Track 2 substances into the environment. Legal, economic and social factors are included in determining the risk management process. While pollution control or remediation strategies may be utilized, the federal government considers pollution prevention to be the most cost-effective risk management strategy (Environment Canada, 1995: 7).

An overview of the overall risk assessment and management process in the *Toxic Substances Management Policy* can be seen in figure below:



(Environment Canada, 1995: 4).

Ultimately, the *Toxic Substances Management Policy* is promoted as a precautionary approach in the identification of toxic substances and the implementation of cost-effective measures to prevent negative impacts on the environment and human health. This policy is publicized as serving as “the centrepiece for the country’s position on

managing toxic substances in discussions and negotiations with the world community" (Environment Canada, 1995: 4). The *Toxic Substances Management Policy* was released just two weeks before the House of Commons Standing Committee on Environment and Sustainable Development was scheduled to release its federally mandated review of CEPA, which focused on the effectiveness of the legislation and recommended changes to strengthen the Act in order to protect the Canadian environment and human health (House of Commons Standing Committee on Environment and Sustainable Development, 1995). The Canadian Environmental Law Association and the Canadian Institute for Environmental Law and Policy[32] (1996) critiqued the *Toxic Substances Management Policy* by pointing to concerns raised during the public consultation process which were not incorporated into the final policy. Further, they argued that the House of Commons Standing Committee on Environment and Sustainable Development was responsible for reviewing the same issues as part of their legislative review. It is suggested that there is an "inescapable conclusion that the TSMP [*Toxic Substances Management Policy*] was released to pre-empt a more full and comprehensive debate and to thwart the kinds of reforms that were to be forthcoming by the Standing Committee" (CELA and CIELAP, 1996: 101).

[32] The Canadian Environmental Law Association (CELA) was established in 1970 as a non-profit, public interest organization to use existing laws to protect the environment and to advocate for environmental law reforms. One of CELA's primary objectives is to prevent harm to human and ecosystem health through the use of precautionary measures (CELA, 2012a). The Canadian Institute for Environmental Law and Policy (CIELAP) was founded in 1970 as not-for-profit research and education organization and one of Canada's top environmental think tanks (CIELAP, n/d). CIELAP is no longer actively performing research and analysis after a decision by the Board in 2011 based on changes in funding and an overlap with CELA (CIELAP, 2011).

CEPA Parliamentary Review

Under the Liberal government, the House of Commons Standing Committee on Environment and Sustainable Development[33] conducted extensive hearings as part of its review process including nation-wide consultations with stakeholders comprised of members of the public, as well as representatives from environment, health and labour organizations, government, academia, and industry. The Standing Committee released *It's About Our Health! Towards Pollution Prevention: CEPA Revisited* in June 1995. The in-depth and detailed report contains 382 pages reviewing CEPA and provides 141 recommendations to the Government of Canada with the potential to strengthen and improve the legislation (House of Commons Standing Committee on Environment and Sustainable Development, 1995).

The Standing Committee cites Environment Canada's 1991 report, *The State of Canada's Environment* to illustrate the challenges involved with the use of chemicals in industrialized society.

> In seeking to reap the abundant benefits they offer, people may also inadvertently run the risk of doing serious harm to the environment and human health. The problem that Canada faces, as a society that is highly dependent on chemicals, is

[33] Members of the Standing Committee on Environment and Sustainable Development included: *Chair*: Charles Caccia, M.P., for Davenport; *Vice-Chairs*: Karen Kraft Sloan, M.P., for York-Simcoe and Monica Guay, M.P., for Laurentides; *Members*: Peter Adams, M.P., for Peterborough, Paul DeVillers, M.P., for Simcoe North, John Finlay, M.P., for Oxford, Paul Forseth, M.P., for New Westminster-Burnaby, Bill Gilmour, M.P., for Comox-Alberni, Clifford Lincoln, M.P., for Lachine-Lac-Saint-Louis, Pat O'Brien, M.P., for London-Middlesex, and Roger Pomerleau, M.P., for Anou-Rivière-des-Prairies; *Associate Members*: Jim Abott, M.P., for Kootenay East, Rex Crawford, M.P., for Kent, Stan Dromisky, M.P., for Thunder Bay-Atikokan, Bob Mills, M.P., for Red Deer, Len Taylor, M.P., for The Battlefords-Meadow Lake, and Andrew Telegdi, M.P., for Waterloo; and *Other Member Who Participated*: Benoît Sauvageau, M.P., for Terrebonne. The *Clerk of the Committee* was Normand Radford. *Research Staff of the Committee* included Pascale Collas, Thomas Curran, Monique Hébert, Margaret Smith, and Ruth Wherry (seconded to the Committee by Environment Canada) from the Research Branch, Library of Parliament, and François Bregha and John Moffett from Resource Futures International. *Other Staff* included Susan Waters (House of Commons Standing Committee on Environment and Sustainable Development, 1995: iv).

> how to realize the benefits of these substances while avoiding the damage they may cause or, at least, reducing the risk of such damage to acceptable levels (Environment Canada cited in House of Commons Standing Committee on Environment and Sustainable Development, 1995: 30).

This demonstrates an early recognition of the risks associated with toxic substances and understanding about the level of acceptability surrounding those environment and health risks.

The House of Commons Standing Committee on Environment and Sustainable Development (1995) specifically notes the capacity of toxic substances to persist and bioaccumulate in the environment to the point where they pose a danger to both ecosystem and human health. The Committee acknowledges the

> …mounting evidence [which] continue[s] to reinforce concerns about the effects of persistent toxic substances. Long-term exposure of fish, wildlife and humans to these substances has been linked to reproductive, metabolical, neurological and behavioural abnormalities; to immunity suppression leading to susceptibility to infections and other life-threatening problems; and to increasing levels of breast and other cancers. Available evidence also points to the long-term reproductive and intergenerational effects (International Joint Commission cited in House of Commons Standing Committee on Environment and Sustainable Development, 1995: 30).[34]

In its brief submitted to the Standing Committee, the Canadian Environmental Law Association speaks to concerns around toxic substances which have the potential to act as endocrine disrupters. The Standing Committee recognizes the increasing body of evidence around toxic substances, particularly those with persistent and bioaccumulative properties, including the potential for detrimental health outcomes such as reproductive,

[34] The International Joint Commission between Canada and the United States recognizes that each country is impacted by the other's actions related to lake and river systems located on the border with the purpose of managing and preventing pollution. The International Joint Commission publishes biennial reports on the water quality of the Great Lakes which can be found at http://www.ijc.org/en_/Biennial_Reports (International Joint Commission, 2013).

developmental and behavioural abnormalities. The report states that the "possible effects of such chemicals on the reproductive integrity of humans, particularly the suggested estrogenic properties of some pollutants, have now developed into a priority issue" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 33).

Consistent with Beck's (1992) theory of the risk society and argument that contemporary chemical threats are unlimited across both space and time as they cross territorial borders and have the potential to affect future generations, the Committee contends that pollution can no longer be viewed only as a local problem. For instance, pesticides and PCBs produced in industrial and agricultural regions of North America are evident in wildlife in Northern Canada and high levels of PCBs have been found in the breast milk of aboriginal women in northern communities (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 30-31).

The House of Commons Standing Committee on Environment and Sustainable Development expressed concern with the definition of toxic under section 11 of CEPA which determines a substance to be toxic if it enters or may enter the environment in a quantity or concentration under conditions that i) have or may have an immediate or long-term effect on the environment; ii) constitute or may constitute a danger to the environment on which life depends; or iii) constitute or may constitute a danger in Canada to human life or health. In this definition of toxic, "there must be a possibility that the substance will *enter* the environment, that living organisms will be *exposed* to the substance, and that there will be an actual or probable *effect* resulting from that exposure"

(House of Commons Standing Committee on Environment and Sustainable Development, 1995: 61). Accordingly, an *entry assessment*, *exposure assessment* and *effects assessment* must be conducted as part of the environmental and human health risk assessment processes under CEPA. The entry assessment determines whether a substance is entering or may enter the environment, and thus it requires that the major sources and releases of the substance be quantified. The exposure assessment must establish and quantify the relationship of exposure to the substance and the living organisms and human population by measuring the concentrations in air, soil, water, and sediment, and in the case of human health extrapolating those findings into probable exposures to humans. Finally, the effects assessment must determine acceptable concentrations for natural populations, communities and ecosystems exposed to the substance, and establish whether acceptable concentrations are exceeded in the environment (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 61-62). An acceptable concentration of a substance is defined in Environment Canada's risk assessment guidelines as the "maximum substance concentration that causes no immediate or long-term harmful effect to the (natural) population, community or ecosystem under consideration" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 62).

The House of Commons Standing Committee on Environment and Sustainable Development (1995: 59) found that Canadian citizens have an expectation of protective legislation and rigorous enforcement of standards around toxic substances which may contain carcinogenic and endocrine disrupting properties. A key component in CEPA's

potential to protect the environment and human health from the effects of toxic substances lies in the risk assessment process. This is the "pivotal point around which turn the functions of risk management – including scheduling, regulations, compliance, and enforcement" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 59). Unlike the assessment and management of Track 1 and Track 2 substances which were proposed (and subsequently adopted and implemented) as part of the *Toxic Substances Management Policy*, the Standing Committee recommended three tracks for the assessment and management of toxic substances. The three tracks included:

- *Track 1 substances* which would establish a presumption of sunsetting for any substance that is sunsetted or banned in a Canadian province or a member nation of the OECD, as well as for any substance that is persistent, bioaccumulative and inherently toxic;

- *Track 2 substances* which would involve a designation of toxic for any substance that is regulated in any Canadian province or in a member nation of the OECD, unless the proponent can demonstrate extraordinary reasons why the substance should not be regulated; and

- *Track 3 substances* which would involve the continued assessment of existing substances through the Priority Substances List process. The Priority Substances List program should be revised to include more classes of substances, effluents and waste streams, as well as applying a "stop-clock" provision for substances for which there is insufficient information needed to complete an assessment. The Minister of the Environment should have the authority to declare the substance toxic under CEPA even if the needed information is not available or forthcoming (Douglas et al., 1997; House of Commons Standing Committee on Environment and Sustainable Development, 1995: xxii).

The Standing Committee expressed concern with both the timing of the release of the *Toxic Substances Management Policy* and its content (Douglas et al., 1997).[35] The *Toxic Substances Management Policy* continues to use the section 11 definition of toxic, which equates toxicity with risk, and includes a risk assessment approach in which the exposure component plays a pivotal role. The risk assessment process under section 11 of CEPA considers the toxicity of a substance and the extent of exposure of a population to that substance (Health Canada, 1994). During the legislative review process, the Standing Committee received feedback suggesting that a hazard assessment process may be more appropriate than risk assessment where hazard is the "intrinsic capability of a substance to do harm, while risk is the probability of harm associated with exposure to various levels of a substance" (Health Canada, 1995: 13; House of Commons Standing Committee on Environment and Sustainable Development, 1995). A hazard assessment approach considers the intrinsic or inherent toxicity of a substance as the primary component in determining regulation and risk management strategies, rather than exposure. "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).

[35] The Standing Committee was critical of the *Toxic Substances Management Policy* because substances that were toxic, persistent and bioaccumulative would be allowed to be used in commerce if the proponent could demonstrate that the substance would not be released into the environment. The Standing Committee's proposal for assessing and managing toxic substances would have "cast a wider net, thereby leading to the eventual elimination of a greater number of substances of concern" (Douglas et al., 1997).

The human health risk assessment conducted under CEPA focuses on both risk and exposure, but not hazard (House of Commons Standing Committee on Environment and Sustainable Development, 1995). Health Canada (1995: 13) contends that the approach to protecting human health from toxic substances must "be aimed at controlling those substances that will have the greatest potential impact on the public's health…[which is] a function of both intrinsic toxicity and exposure."[36] Health Canada (1995: 40) 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." It is argued that the level of risk increases with an increase in exposure (Health Canada, 1995).

In contrast, in the Canadian Institute for Environmental Law and Policy's submission to the Standing Committee, it was suggested that the definition of toxic under CEPA be revised to emphasize the intrinsic characteristics of a substance and the potential to cause harm to the environment or human health, rather than the exposure component which considers the quantity or concentration of a substance that will cause a negative effect (CIELAP, 1994: 9). Recognizing the merits associated with the hazard assessment process, the House of Commons Standing Committee on Environment and Sustainable Development (1995: xxii) recommended revising CEPA's definition of toxic to include both risk assessment and hazard assessment.

[36] Health Canada (1995: 13-14) provides an illustrative example using knives to contrast hazard- and risk-based approaches. The intrinsic property of knives being sharp demonstrates the hazard associated with the object. Rather than just considering the hazard associated with children and knives which would require that knives be removed from the home entirely, an exposure-based approach is recommended in which the knives should be placed in a locked drawer which reduces the risk by preventing exposure.

While the precautionary principle has been associated with the protection of the environment, it had not yet been traditionally or explicitly linked to preventing the health outcomes associated with exposure to toxic substances (Health Canada, 1995: 10). The House of Commons Standing Committee on Environment and Sustainable Development (1995: 54) concluded that precautionary measures should be used under circumstances where an activity or substance poses a serious threat of harm to the environment or human health, even if the outcome is uncertain. The precautionary principle was said to promote sustainable development and was supported by many of the stakeholders involved in the consultation process as part of the legislative review of CEPA. For instance, the Canadian Bar Association indicated that CEPA would benefit from the precautionary principle.

> The determination of a toxic chemical is arrived at through a classification process and the restriction of the release of that toxic chemical appears to be based on nothing short of scientific certainty. The same process is subsequently applied (substance by substance) for each and every other toxic chemical. Because this approach is proving too lengthy to attain its objectives within a reasonable time period, it does not fit with the notion of sustainable development. Instead, amendments should be made to Part II of CEPA that reflect a precautionary approach to managing toxic chemicals (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 55).

Health Canada (1995: 11) suggested that the precautionary principle is an inherent part of the human health risk assessments conducted under CEPA, but allows that it may be appropriate to explicitly highlight the precautionary principle in the Act and refer to it in the Preamble. The Standing Committee formally recommended that the precautionary principle be incorporated as a guiding principle of CEPA, included in the Preamble of the Act, and that all provisions of CEPA should be interpreted within the framework of the

precautionary principle. "CEPA should define the precautionary principle to mean that, in respect of all substances suspected of posing a threat to the environment or to human health on the basis of weight of evidence, lack of full scientific certainty shall not be sufficient reasons for postponing preventive or remedial measures" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 56).

The two Standing Committee members from the Official Opposition, the Environment and Sustainable Development critic and the Deputy critic, both members of the Bloc Québécois, wrote a dissenting opinion which was included in the submission (House of Commons Standing Committee on Environment and Sustainable Development, 1995). Despite allowing that CEPA did not succeed in its intended impact, they "disagreed profoundly with the solutions proposed by the Committee for improving the effectiveness of CEPA and of environmental issues generally" (Douglas et al., 1997). While federal environmental legislation was intended to resolve jurisdictional issues and overlap between and among the provinces, territories and the country, there were still those who favoured a secular approach and localized regulatory regimes (Girard et al., 2010). The Opposition members rejected the report in its entirety stating that it was unfairly biased against the provinces by advocating a federal, centralized approach to environmental management in Canada (Douglas et al., 1997).[37]

[37] It should be noted that the Quebec government was the strongest provincial opponent to CEPA. However, "provincial efforts to resist federal involvement in the environmental field may have resulted in a more intrusive state" (Harrison, 1996: 131). CEPA acknowledges provincial authority, but it contains strict equivalency standards with the intent of promoting national standards (Harrison, 1996).

Under Standing Order 109, the House of Commons Standing Committee on Environment and Sustainable Development requested that the federal government deliver a response to the recommendations within 150 days (Douglas et al., 1997). The official response from the Government of Canada was tabled on December 14, 1995 in a document entitled *Environmental Protection Legislation Designed for the Future – A Renewed CEPA*. It stated that the domestic and international agenda had changed dramatically since CEPA's proclamation in 1988:

> New concepts and approaches, such as sustainable development, the precautionary principle and pollution prevention, have evolved since CEPA first came into effect. Consequently, the renewed *Act* would be based on guiding principles. They would include statements on pollution prevention, the ecosystem approach, biodiversity, intergovernmental cooperation, science and the precautionary principle, economic responsibility and user/producer responsibility (Minister of Supply and Services Canada, 1995: 7).

There is agreement with the Standing Committee's interpretation of section 11 of CEPA in that the Government must consider whether the substance is entering or may enter the environment, the degree of exposure as a result, and the levels of exposure that can cause adverse effects to occur. The Government agreed that it "must consider the risk posed by substances before rendering a conclusion. Understanding the nature (including sources) and extent of the risk enables the Government to prioritize dangers to human health and the environment and to focus controls where they will have the greatest benefit" (Minister of Supply and Services Canada, 1995: 70). While the Government agreed that inherent toxicity plays a role in determining the risk associated with a substance, it resisted considering an approach that would include hazard assessment and concluded that the ultimate role of inherent toxicity is to be used in conjunction with data on

exposure in order to form the basis for assessing risk (Minister of Supply and Services Canada, 1995: 70).

Overall, the Government's response is very much focused on the *Toxic Substances Management Policy*, both in its content and as a precursor to the upcoming revisions to CEPA. The *Toxic Substances Management Policy* established the direction for all federal government departments around the assessment and management of risk associated with toxic substances (Minister of Supply and Services Canada, 1995: 71). At the time, the Government of Canada established that the key policy direction of the *Toxic Substances Management Policy* was strongly supported by industry, and would be incorporated into a revised CEPA, consistent with the Government's regulatory reform agenda (Minister of Supply and Services Canada, 1995: 5). The House of Commons Standing Committee on Environment and Sustainable Development (1996) expressed significant concerns and criticisms around the Government's response its report.[38]

[38] The House of Commons Standing Committee on Environment and Sustainable Development's (1996) response to the Government proposal to reform CEPA included an extensive list of signatories who formally endorsed the document. The organizations included Action! Environment, NFLD; Alberta Federation of Labour, AB; Allergy Foundation of Canada, SK; Amis de l'environment de Brandon, PQ; Animal Alliance, ON; APT Environment, ON; Banff Recycling Society, AB; Biomedical Waste Incineration Ban Incineration, ON; Bruce Peninsula Environment Group, ON; Canadian Auto Workers, ON; Canadian Auto Workers Lower Mainland-Environment Committee, BC; Canadian Environmental Law Association, ON; Canadian Institute for Environmental Law and Policy, ON; Canadian Labour Congress, ON; Canadian Organic Growers Inc., ON; Canadian Union of Public Employees, ON; CAW Windsor Regional Environment Council, ON; Centre for Long Term Environmental Action, NFLD; CHOICES!, MB; Citizens' Clearinghouse on Waste Management, ON; Citizens Environment Alliance of Southwestern Ontario, ON; Citizens for Renewable Energy, ON; Citizens' Network on Waste Management, ON; Clean North, ON; Clean Nova Scotia, NS; Coalition of Ontario Doctors for the Environment, ON; Common Frontiers, ON; Concerned Citizens of Ashfield and Area, ON; Concerned Citizens of Manitoba, MB; Conservation Council of New Brunswick, NB; Cosy Covers Corporation, ON; Earth Wise, ON; East Coast Environmental Law Association, NS; Ecology Awareness Group Landscape and Environment, ON; Environmental Coalition of Prince Edward Island, PEI; Environmental Component Public Service Alliance of Canada, ON; Environmental Law Centre, AB; Environmental Mining Council of British Columbia, BC; Environmental Resource Centre, AB; Friends of Lily Lake, AB; Friends of the Earth, ON; Furiously Opposed to All Dumping, ON; Georgia Strait Alliance, BC; Great Lakes United, PQ;

> Despite the Committee's recommendations, industry and pro-industry departments – like Natural Resources Canada, Industry Canada and Agriculture Canada – attempted to discredit the Standing Committee's report. Their efforts contributed to the tabling of a weak, dilute government response to the report. The government response […] does not reflect the breadth and scope of the Committee recommendations (House of Commons Standing Committee on Environment and Sustainable Development, 1996: 2).

In particular, the House of Commons Standing Committee on Environment and Sustainable Development suggested that the government's response inadequately responded to concerns around pollution prevention and toxic substances. The Standing Committee took issue with the lack of a clear commitment to phase out production and use of substances which are inherently dangerous, persistent, bioaccumulative, or disruptive to the endocrine system. For instance, substances with toxic properties such as toluene may still be declared non-toxic under CEPA (House of Commons Standing Committee on Environment and Sustainable Development, 1996: 5; CELA and CIELAP, 1996).

Green Alternatives Institute of Alberta, AB; Greenpeace, ON; Greensville Against Serious Pollution, ON; Guideposts for a Sustainable Future, ON; Healthy Sustainable Communities Association (National Capital Region), ON; Hickory Falls Rate Payers Association, ON; Housing Fairness Association, ON; Human Ecology Liaison People, BC; Incineration Counteracts the Environment, ON; Learning Disabilities Association of Canada, ON; Manitoba Federation of Labour, MB; Manitoba Future Forest Alliance, MB; National Farmers Union, SK; National Union of Public and General Employees, ON; Northwatch, ON; Nova Scotia Public Interest Research Group, NS; Ocean Voice International, ON; Ontario Federation of Labour, ON; Ontario Health Advocacy Association, ON; Ontario Health Care, ON; Ontario Public Health Association Environment Work Group, ON; Ontario Streams, ON; Ontario Toxic Waste Research Coalition, ON; Pembina Institute for Appropriate Development, AB; Pictou Harbour Environmental Protection Project, NS; Poetical Asylum, PEI; Pollution Probe, ON; Prairie Acid Rain Coalition, AB; Prince Edward Island Stranding Network, PEI; Protect Our Water and Environmental Resources, ON; Research for Unbleached, BC; Sierra Club of Canada, ON; Sierra Club of Eastern Canada, ON; Sierra Club Prairie Chapter, MB; St. Clair River International Citizens' Network, ON; Stop and Tell Our Politicians Society, AB; Stop Environmental Deregulation in Canada, York University, ON; Stop Incineration United in Yards Anywhere, ON; Time to Respect Earth's Ecosystem, MB; Toronto Environmental Alliance, ON; Town of Pickering Waste Reduction Committee, ON; Toxics Watch Society, AB; Tusket River Environmental Protection Association, NS; Voice of the Earth Society, NS; Waste Not; ON; Wastewise, ON; Western Canada Wilderness Committee, AB; Windsor and Area Coalition for Social Justice, ON; Windsor and District Labour Council Environment Committee, ON; Women's Network on Health and the Environment, ON; and the World Wildlife Fund Canada, ON.

In a detailed reaction to the Government's official response, referenced in the Standing Committee's response, the Canadian Environmental Law Association and Canadian Institute for Environmental Law and Policy (1996) expressed concerns that under the *Toxic Substances Management Policy*, the "environment" does not include the occupational environment, and the definitions of virtual elimination, persistence and bioaccumulation which are inconsistent with those set by agencies such as the International Joint Commission and the government's pollution prevention policy statement. The required criteria for substances to be classified as Track 1 are suggested to be too limited and that a combination of toxicity and persistence, or toxicity and bioaccumulation should be sufficient rather than toxicity, persistence *and* bioaccumulation. The deliberate use and management of Track 1 substances are also allowed to continue within the "no measurable release" provisions of the virtual elimination requirement of the *Toxic Substances Management Policy* (CELA and CIELAP, 1996: 100-01). In a response to the government proposal to reform CEPA, the House of Commons Standing Committee on Environment and Sustainable Development (1996: 2) contends that Canadians expected a strong federal regulatory leadership to be reflected the Government's response and expressed disappointment in the Government's proposal which fails to "implement aggressive [pollution] prevention and regulation of toxic chemicals."

The Canadian Environmental Protection Act, 1999 (CEPA 1999)

A year after submitting its response to the House of Commons Standing Committee on Environment and Sustainable Development's recommendations, the

Liberal government introduced Bill C-74 which was tabled in Parliament in December 1996, but did not receive a second reading and died when the general federal election was called in April 1997 (Douglas et al., 1997). Subsequently Bill C-32 was introduced to the House of Commons in March 1998, received a second reading in April 1998, was studied for a year by the House of Commons Standing Committee on Environment and Sustainable Development, and then received a third reading in June 1999. Bill C-32 replaced CEPA at this time and the *Canadian Environmental Protection Act, 1999*[39] was implemented (Douglas and Hébert, 1999a, 1999b). The lengthy review of CEPA resulted in a statute that was five times longer than the original Act and included new concepts such as "sustainable development" and the "precautionary principle" which had not yet been applied to the management of toxic substances (Government of Canada, 2005a; House of Commons Standing Committee on Environment and Sustainable Development, 2007: 4). The operative parts of CEPA 1999 are divided into administration; public participation; information gathering, objectives, guidelines and codes of practice; pollution prevention; controlling toxic substances; animate products of biotechnology; controlling pollution and managing wastes; environmental matters related to emergencies; government operations and federal and aboriginal land; enforcement; miscellaneous matters; and consequential amendments, repeal, transitional provision and coming into force. For the purposes of this dissertation and its focus on the regulatory

[39] Canadian Environmental Protection Act, R.S.C. 1999, c. 33; herein after described as CEPA 1999.

regime for chemicals, the most relevant sections include pollution prevention and controlling toxic substances.

CEPA 1999 is designated as an "Act respecting pollution prevention and the protection of the environment and human health in order to contribute to sustainable development" where the environment involves the components of the Earth including air, land and water; all layers of the atmosphere; all organic and inorganic matter and living organisms; and the interacting natural systems that include the former components. Sustainable development in CEPA 1999 refers to development that meets the needs of the present without compromising the ability of future generations to meet their own needs. The Government of Canada (2005a) promotes the revised Act as contributing to sustainable development by preventing pollution; promoting coordinated action with provinces, territories, Aboriginal governments, and federal departments in order to achieve the highest level of environmental quality for the health of Canadian citizens; managing risks from harmful substances[40]; and virtually eliminating the releases of the substances determined to be the most dangerous.

The new principles outlined in the preamble of CEPA 1999 are grounded in concepts including sustainable development, pollution prevention, an ecosystem approach, and the precautionary principle. The role of the Government of Canada is to demonstrate national leadership and fulfil international obligations in establishing

[40] A substance in CEPA was defined as any distinguishable kind of organic or inorganic matter, whether animate or inanimate. This definition has been expanded in CEPA 1999 to include "any distinguishable kind of organic or inorganic matter, whether animate or inanimate, and includes…any mixture that is a combination of substances…or any complex mixtures of different molecules that are contained in effluents, emissions or wastes that result from any work, undertaking or activity." Health Canada (2003: 24) notes that the substances "encompass…discrete chemical compounds, classes of chemicals, emissions and effluents and products of biotechnology, including microorganisms."

environmental standards, ecosystem objectives and environmental quality guidelines and codes of practice. The responsibilities of Environment Canada and Health Canada include:

- a commitment to implementing pollution prevention as a national goal and a priority for environmental protection;
- acknowledging the need to virtually eliminate the most persistent and bioaccumulative toxic substances;
- the need to control and manage pollutants and wastes if their release into the environment cannot be prevented;
- recognizing the risks associated with toxic substances in the environment and that substances cannot be contained within geographic boundaries once released; and
- removing threats to biological diversity through pollution prevention, controlling and managing the risk of adverse effects associated with the use and release of toxic substances, and the virtual elimination of persistent bioaccumulative toxic substances.

A number of the inclusions in CEPA 1999 such as the ecosystem approach, the role of pollution prevention and the precautionary principle were recommended by the House of Commons Standing Committee on Environment and Sustainable Development's (1995) review of the original Act. Pollution prevention as the cornerstone of CEPA 1999 reflected a shift in focus from the management of pollution which was one of the guiding principles of the original CEPA (Environment Canada, 2010a). This shift was consistent with the Liberal Party's 1993 platform which made toxic substances a significant focus, and emphasized that pollution would be reduced at the source (Swimmer, 1997; Toner, 2002; Juillet and Toner, 1997).

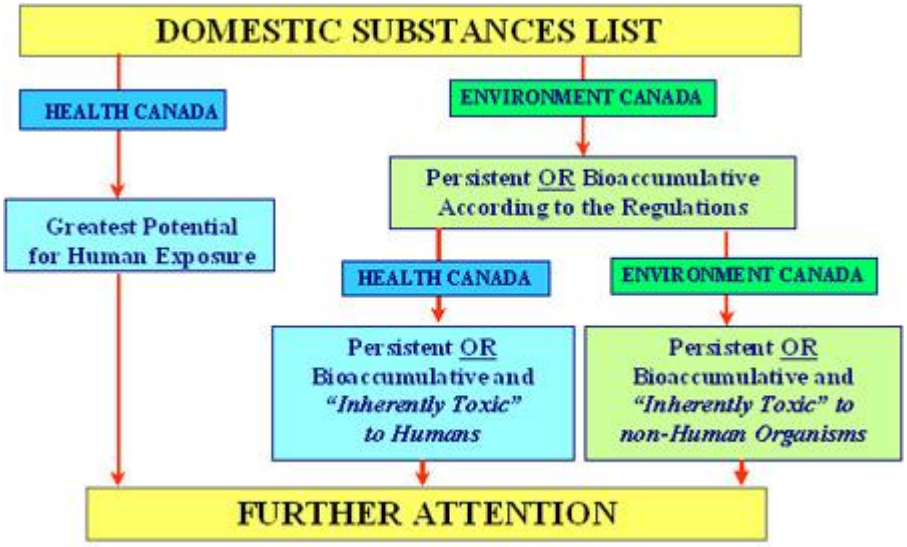
A substance is considered to be toxic under section 64 of CEPA 1999, with the exception of "inherently toxic," if it is entering or may enter the environment in a

quantity or concentration or under conditions that i) have or may have an immediate or long term harmful effect on the environment or its biological diversity; ii) constitute or may constitute a danger to the environment on which life depends; or iii) constitute or may constitute a danger in Canada to human life or health. The assessment and management of risks from toxic substances are the principal objectives of CEPA 1999, and are promoted by Environment Canada as being proactive, preventive and precautionary (Environment Canada, n/da). The risk management tools under CEPA 1999 "range from guidelines or codes of practice through to requiring the preparation and implementation of pollution prevention plans, environmental emergency plans and regulations, including economic instruments" (Government of Canada, 2005b: 2).

The revised Act continues work with the Domestic Substances List established in the original CEPA. This includes the 23,000 existing substances as outlined in section 25 of CEPA and section 66 of CEPA 1999, which were manufactured or imported into Canada in a quantity of not less than 100 kilograms in any one calendar year, or were in commerce or used for commercial manufacturing purposes in Canada between January 1, 1984 and December 31, 1986. CEPA 1999 provides a "framework for the identification/ prioritization of [e]xisting [s]ubstances for risk assessment and the control or management of those considered to pose a risk to human health and/or the environment. This framework is broad, evidence-based, open and transparent and builds upon work done in other jurisdictions" (Health Canada, 2003a: 16). Section 73(1) of CEPA 1999 required the Minister of the Environment and the Minister of Health to *categorize* the 23,000 existing substances on the Domestic Substances List which

(a) may present, to individuals in Canada, the greatest potential for exposure; or
(b) are persistent or bioaccumulative in accordance with the regulations, and inherently toxic to human beings or to nonhuman organisms, as determined by laboratory or other studies.

It should be noted that the concept of inherent toxicity is not defined in CEPA 1999. The toxic substances on the Domestic Substances List that are used in the highest quantities and that come into direct contact with the general public are considered to have the greatest potential for exposure (Health Canada, 2003a: 21). A basic overview of the categorization process can be seen below:



(Environment Canada, 2011b).

The Act included a provision requiring the categorization of existing substances to be completed within seven years of its Royal Assent. The mandate of the categorization process is to identify the substances to be considered in subsequent phases of assessment including screening assessments under section 74 and in-depth assessments for Priority Assessment under section 76 of CEPA 1999 (Health Canada, 2005c: 5). Section 74 requires the Minister of the Environment and Minister of Health to conduct

screening assessments of substances in order to determine whether a substance is toxic or capable of becoming toxic. A screening assessment is conducted if the Ministers determine that a substance on the Domestic Substances List has the greatest potential for exposure, or is persistent or bioaccumulative, and inherently toxic. Section 76 requires the Minister of the Environment and the Minister of Health to compile the Priority Substances List which includes the substances that the Ministers are satisfied should be given priority in assessing whether they are toxic or capable of becoming toxic. After the screening assessment has been conducted, the Ministers can propose one of the measures outlined in section 77(2) which may include: taking no further action in respect of the substance; adding the substance to the Priority Substances List if it is not already included; or recommending that the substance be added to the List of Toxic Substances in Schedule 1, and the implementation of virtual elimination under subsection 65(3) where applicable.[41]

Virtual elimination of toxic substances released into the environment as a result of human activity was a new addition to CEPA 1999. Under section 65(1) the virtual elimination of a toxic substance is defined as the ultimate reduction of the quantity or concentration of the substance in the release below the level of quantification specified by the Ministers in the Virtual Elimination List. The level of quantification is defined in section 65.1 as the lowest concentration that can be accurately measured using sensitive but routine sampling and analytical methods. When the level of quantification for a

[41] The Ministers are required to publish their preliminary findings including a summary of the science for feedback from the public over 60 days. After considering the public comments, the Ministers must publish a final proposal (Environment Canada and Health Canada, 2006).

substance on the Virtual Elimination List has been specified, the Ministers are required to prescribe the quantity or concentration of the substance that may be released into the environment either alone or in combination with any other substance from any source or type of source, and must account for environmental and health risks, as well as any relevant social, economic or technical matters.

The List of Toxic Substances in Schedule 1 of the original CEPA was "rolled over" and incorporated into Schedule 1 of CEPA 1999. It was determined that no assessment under section 64 of CEPA 1999 would be required for the twenty-six substances on Schedule 1 as they all met the criteria for toxic (Environment Canada, 2010c). Environment Canada and Health Canada (2006: 14) explain that CEPA 1999 does not provide specific information about the type of assessment to be conducted under the main risk assessment pathways including the screening assessment, Priority Substances List, and addition to Schedule 1. Policy must be used to determine the difference between a screening assessment and a Priority Substances List assessment, the latter of which may be used under situations that require in-depth input from the public (Environment Canada and Health Canada, 2006: 14).

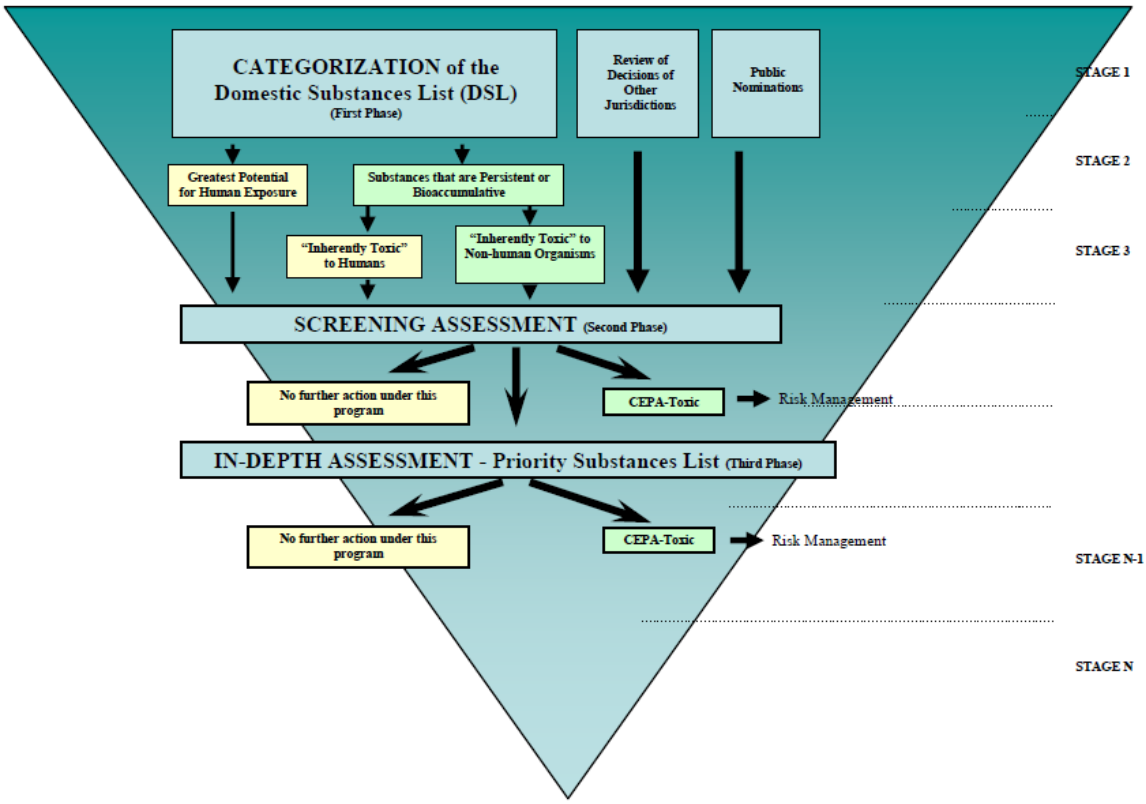
Health Canada's responsibilities as part of this process included categorizing all 23,000 substances on the Domestic Substances List in order to determine which substances are potentially harmful to human health and thus require further consideration. The substances were studied and categorized as to whether they possess the greatest potential for human exposure, and are persistent, bioaccumulative and inherently toxic to humans under section 73 of CEPA 1999 (Health Canada, 2005c; Health Canada,

2009b).[42] This process falls under section 68 of CEPA 1999 in which a Minister may collect or generate data and conduct investigations respecting any matter in relation to a toxic substance; correlate and evaluate any data collected or generated and publish results of any investigations; and provide information and make recommendations respecting any matter in relation to a toxic substance, including measures to control the presence of the substance in the environment. If a substance is declared to be CEPA-toxic, a health risk assessment involves:

- the identification of the critical adverse health effect associated with exposure to the substance;
- analysis of the dose-response relationship; and
- the determination of the extent to which the population or subset of the population are exposed to the substance; and relating the exposure to a measure of the dose-response relationship for the critical effect (Health Canada, 2004c: 10).

A more detailed diagram of the assessment process of the Domestic Substance List under CEPA 1999 which can be seen below:

[42] Under section 73 of CEPA 1999, Environment Canada is responsible for the categorization of substances on the Domestic Substances List that are persistent and/or bioaccumulative and inherently toxic to non-human organisms.



(Health Canada, 2009b: 1).

When it began in 2000, it was anticipated that approximately 4700 of the 23,000 substances assessed would meet the categorization criteria and those substances would undergo a screening assessment (Environment Canada and Health Canada, 2006). When it was completed in 2006, Canada claimed to be the "first country in the world to have examined the hazardous properties of all its 'existing substances' providing an information baseline on all of those substances" (Environment Canada and Health Canada, 2006: 16).[43]

[43] Environment Canada and Health Canada (2006: 12) do acknowledge other similar initiatives including the High Production Volume initiative by the Organisation for Economic Cooperation and Development (OECD) and the European Union's Registration, Evaluation and Authorization of Chemicals (REACH) program. The High Production Volume Chemicals Initiative was launched in 1998 with the OECD and the International Council of Chemicals Associations (IICA). This program collected screening-level data to be

The results determined that 4300 of the 23,000 substances examined were classified as priorities for further action under the newly implemented *Chemicals Management Plan* (Health Canada, 2010a). The remaining 19,000 substances did not meet the criteria for categorization (Environment Canada, 2010e). However, the House of Commons Standing Committee on Environment and Sustainable Development (2007: 12) suggested that these 19,000 substances should not be identified as "safe" based on the results of the categorization process. These substances may possess persistent, bioaccumulative or inherently toxic properties. It is also noted that persistence may be an issue of concern not only "because they break down slowly in the environment, but because there is a continuous supply of [the substance]" (House of Commons Standing Committee on Environment and Sustainable Development, 2007: 13). Five hundred chemicals were classified as the highest priorities for immediate action, 2600 were classified as medium priorities, and 1200 chemicals were classified as low priorities. Environment Canada and Health Canada must determine the priorities for the risk assessment and management of the substances that meet the categorization criteria on the

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used in hazard assessments which consider acute toxicity, repeat dose toxicity, reproductive and developmental toxicity, genetic toxicity, ecotoxicity, and the environmental fate of the chemical (IICA, 2013; OECD, 2013a). The OECD's Cooperative Chemicals Assessment Programme was established based on the High Production Volume Chemicals Programme in order to assess more chemicals in a shorter time period, address all chemicals on the market; and avoid duplication of work occurring in other member countries. The focus of the Cooperative Chemicals Assessment Programme includes the dissemination of the hazards associated with chemicals; development and application of integrated approaches to testing and assessment; avoiding duplication among member countries; and providing a forum to exchange experience (OECD, 2013b). REACH became active legislation in the European Union in 2007 and seeks to "improve the protection of human health and the environment through the better and earlier identification of the intrinsic properties of chemical substances" by placing the burden of proof on industry (Europa, 2013). Manufacturers and importers of chemicals are required to manage the risks associated with the toxic substances and to provide safety information. REACH also calls for substitution when suitable alternatives can be utilized. The provisions of REACH are being phased-in over an 11 year period (Europa, 2013).

Domestic Substances List, as well as disseminate the categorization results to the public (Environment Canada and Health Canada, 2006: 16).

CEPA 1999 Parliamentary Review

CEPA 1999 requires a mandatory Parliamentary review every five years under section 343 and a review was scheduled for March 31, 2005. In preparation for the parliamentary review and under the Liberal government, Environment Canada and Health Canada (2004) published a scoping review to provide background information for a public engagement process; created a website to provide Canadian citizens with information on the CEPA 1999 review process and accept comments submitted online; and held six regional workshops across Canada in 2005 for feedback from the public about CEPA 1999. Environment Canada and Health Canada consulted with municipal governments, Aboriginal organizations, industry and business interests, and civil society, and solicited advice from provincial and territorial governments in advance of producing an *Issues Paper* on CEPA 1999 under the new Conservative federal government in 2006 (Environment Canada and Health Canada, 2006; Environment Canada, 2012).[44] The new federal government presents itself as being

[44] The Canadian Environmental Network Toxics Caucus was involved in the consultation process and submitted an agenda representing environmental non-governmental organizations across Canada. It concluded that despite efforts to control pollution under CEPA 1999, "the volume of dangerous chemicals released into Canada's environment continues to increase" (Canadian Environmental Network Toxics Caucus, 2005: 3). This report was supported and endorsed by the Allergy and Environmental Health Association of Quebec, Beyond Factory Farming Coalition, Canadian Environmental Law Association, Canadian Network for Environmental Education and Communication, Citizens Environment Alliance of Southwestern Ontario, Citizens Network on Waste Management, Coalition for Alternatives to Pesticides, Environmental Defence, Environmental Health Association of Nova Scotia, Great Lakes United, Inter-Church Uranium Committee Educational Cooperative, New Brunswick Lung Association, Ontario Toxic Waste Research Coalition, Sierra Legal Defence Fund, Sierra Youth Coalition, South Peace Environment Association, STORM Coalition, Under the Sleeping Buffalo Research, and World Wildlife Fund Canada (Canadian Environmental Network Toxics Caucus, 2005).

> committed to ensuring the environmental laws and policies promote the over-arching national goal of attaining the highest levels of environmental quality so as to enhance the well-being of Canadians, protect human health, preserve the quality of the environment and advance the country's long-term economic competitiveness (Environment Canada and Health Canada, 2006: 3).

Environment Canada and Health Canada (2006) conclude that CEPA 1999 provides a solid basis for continuing to protect the environment and human health, but concede that there are opportunities for improving the implementation of Act.

Two Parliamentary Committees were appointed in April 2006 to formally review CEPA 1999 including the House of Commons Standing Committee on Environment and Sustainable Development[45] and the House of Commons Standing Committee on Energy, the Environment and Natural Resources[46] (Environment Canada, 2012).[47] Under the

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[45] Members of the Standing Committee on Environment and Sustainable Development included: *Chair:* Bob Mills, M.P.; *Vice-Chairs:* Bernard Bigras and Hon. Geoff Regan; *Members:* Mike Allen, Nathan Cullen, Luc Harvey, Marcel Lussier, David McGuinty, Anthona Rota, Francis Scarpaleggia, Maurice Vellacott, and Mark Warawa; and *Other Members Who Participated:* Catherine Bell, Don H. Bell, Dennis Bevington, Steven Blaney, Hon. Scott Brison, Paule Brunelle, Blaine Calkins, Rodger Cuzner, Jean-Claude D'Amours, Patricia Davidson, Dean Del Mastro, Paul Dewar, Ken Epp, Mark Eyking, Hon. John Godfrey, Laurie Hawn, Mark Holland, Michael Ignatieff, Brian Jean, Hon. Marlene Jennings, Hon. Lawrence MacAulay, Luc Malo, Pat Martin, Christian Ouellet, Daniel Petit, Pierre Poilievre, Pablo Rodriguez, Denise Savoie, Mario Silva, Scott Simms, Lloyd St. Amand, Paul Steckle Hon. Gilbert Thibault, Chris Warkentin, Jeff Watson, Blair Wilson, and Borys Wrzesnewskyj. The *Clerk of the Committee* was Justin Vaive. *Research Staff of the Committee* included Tim Williams, Sam Banks and Kriten Douglas from the Library of Parliament, Parliamentary Information and Research Service (House of Commons Standing Committee on Environment and Sustainable Development, 2007: iii).

[46] Members of the Standing Senate Committee on Energy, the Environment and Natural Resources included: *Chair:* the Honorable Tommy Banks, *Deputy Chair:* the Honorable Pierre Claude Nolin, and the Honorable Willie Adams, the Honorable Bert Brown, the Honorable Ethel Cochrane, the Honorable Colin Kenny, the Honorable Elaine McCoy, the Honorable Lorna Milne, the Honorable Grant Mitchell, the Honorable Nick Sibbeston, the Honorable Mira Spivack, and the Honorable Marilyn Trenholme Counsell. *Ex-officio members of the committee* included the the Honorable Senators Hervieux-Payette P.C., (or Tardif) and LeBreton, P.C., (or Comeau), and the Honourable Senators Angus, Campbell, Carney, Chaput, Cordy, Cowan, Dawson, Fox, Fraser, Grafstein, Hubley, Lavigne, Mercer, Nolin, Nancy Ruth, Peterson, Robichaud, Segal and Tkachuk. *Research Staff of the Committee* included Kristen Douglas (principal), Lynne Myers, Sam Banks, analysts and Amelia Bellamy-Royds from the Library of Parliament, Parliamentary Information and Research Services. The *Clerk of the Committee* was Eric Jacques, Committees Directorate, and the *Administrative Assistant* was Nicole Bédard, Administrative Assistant,

legislation, the Standing Committees have up to one year to complete their review, though an extension may be granted if necessary. CEPA 1999 requires the final reports to be submitted to Parliament, and the Government of Canada has 150 days to provide a response as to whether or how the Act will be revised (s. 343(2), CEPA 1999; Environment Canada and Health Canada, 2004).

The House of Commons Standing Committee on Environment and Sustainable Development presented its report to the House of Commons in May 2007 with a seventy-five page report which included thirty-one recommendations. *The Canadian Environmental Protection Act, 1999 – Five Year Review: Closing the Gaps* focused primarily on examining the content and implementation of Part 5 of the Act, Controlling Toxic Substances. The objectives of CEPA 1999 include contributing to sustainable development through pollution prevention; promoting coordinated access across the country to achieve the highest environmental quality for the health of all Canadian citizens; and managing risks from harmful substances, while virtually eliminating releases of the most dangerous toxic substances. However, questions are raised about the

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Committees Directorate (House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008: i).

[47] The Parliamentary Review Committees received formal submissions from environmental organizations critiquing the implementation of CEPA 1999. A submission by Pollution Watch (representing the Canadian Environmental Law Association and Environmental Defence) included 34 specific recommendations to revise and reform CEPA 1999 in order to "address key gaps in federal law that enable ongoing exposure to toxic substances" (Pollution Watch, 2006: 3). The recommendations included shorter assessment periods, stronger mandatory deadlines and including the role of sensitive stages of human development and vulnerable populations in risk assessment processes (Lafrenière, n/d; Pollution Watch, 2006). Submissions by both Pollution Watch (2006) and the Ontario Public Health Association (2006) note the increasing incidence of detrimental health outcomes linked to exposure to toxic substances and the subsequent healthcare costs. It is suggested that the review of CEPA 1999 offered an opportunity to address these issues and to "close gaps in our regulatory system thus ensuring that Canadian's health and the environment are adequately protected" (Ontario Public Health Association, 2006: 3).

efficacy of CEPA 1999 and whether the objectives of the Act were being met; the report specifically notes the increasing emissions of toxic substances; and the very limited use of virtual elimination provisions to date (House of Commons Standing Committee on Environment and Sustainable Development, 2007: 6). As of 2007, the Government of Canada had not implemented many of the provisions under CEPA 1999 including:

- the authority to create regulations that control products containing toxic substances (never been used);
- the authority to create interim orders regarding potentially dangerous substances (never been used);
- the authority to request information on substances that the Minister of Health or Environment suspects are or could become toxic (limited use); and
- the authority to require virtually elimination of persistent, bioaccumulative and inherently toxic substances (occurred for only one substance which was not in commerce) (House of Commons Standing Committee on Environment and Sustainable Development, 2007: 5).

The Standing Committee considered how the Government of Canada might improve the implementation of CEPA 1999 so the objectives of the Act might be met (House of Commons Standing Committee on Environment and Sustainable Development, 2007).

The House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources extended its timeline three times during the review period, from October 31, 2007 to February 29, 2008, and ultimately submitted its final report on March 4, 2008 (Environment Canada, 2012). *The Canadian Environmental Protection Act (1999, c. 33) Rx: Strengthen and Apply Diligently* is a fifty-five page report which utilizes two comprehensive case studies, on mercury and perfluorinated compounds, in order to consider "whether, how and to what extent…[toxic substances] are currently

being managed under the Act, and how successful the management has been in protecting the health and well being of Canadians and the environment" (House of Commons Standing Committee on Energy, the Environment and Natural Resources, 2008: 6). The Senate Standing Committee provides twenty-four recommendations and suggests that the ineffectiveness of CEPA 1999 is related to a lack of will to implement and enforce the Act, as well as failure to devote the necessary resources for the implementation and enforcement (House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008: 3).

Both the House of Commons Standing Committee on Environment and Sustainability (2007) and the House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources (2008) criticize the Government of Canada for a lack of reporting on information about pollution and environmental and human health. Section 44(1)(f) of CEPA 1999 requires the Minister of the Environment to publish a periodic report on the state of the Canadian environment. Specifically, the Minister will publish, arrange for the publication of or distribute through an information clearinghouse, information respecting pollution prevention; pertinent information with respect to all aspects of environmental quality; and a periodic report on the state of the Canadian environment. The goals of these reports are to provide "timely, accurate, and accessible environmental information, integrated with socioeconomic factors, to improve decision-making and support progress towards sustainability" (House of Commons Standing Committee on Environment and Sustainability, 2007: 8). It is argued that this practice of monitoring, reporting and communicating was "virtually abandoned" since the in-depth

publications on the state of Canada's environment in 1991 and 1996 (House of Commons Standing Committee on Environment and Sustainability, 2007). While CEPA 1999 does not include specific information as to how often the reports on the state of the environment should be completed, the Standing Committee on Environment and Sustainable Development (2007) recommends the reinstatement of timely reports and the Standing Senate Committee on Energy, the Environment and Natural Resources (2008) suggests that reports should be published no less frequently than every ten years.

Both the House of Commons Standing Committee on Environment and Sustainable Development (2007) and the Standing Senate Committee on Energy, the Environment and Natural Resources (2008) note the necessity of meeting reasonable and mandated timelines. Mandatory timelines have tended be effective where they exist in CEPA 1999, such as the seven years to complete the categorization process as part of the Domestic Substances List. However, concerns were expressed around the required screening level assessments which determine if the substances are CEPA-toxic, because no timeline was specified in the legislation for them to be completed. The Standing Committee recommended that if the screening assessment determines that a substance is toxic, there should be a maximum of two years from the assessment to the implementation of a risk management plan, and five years if the screening concludes the need for a full Priority Substances List assessment (House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008: 24-27).[48]

[48] The House of Commons Standing Committee on Environment and Sustainable Development (2007: 26) provides this discussion around the importance of "reasonable timelines" based on examples where the assessment of toxic substances did not occur in a timely fashion. For instance, the listing of

The Government tabled an interim response to the House of Commons Standing Committee on Environment and Sustainable Development report in October 2007 (Parliament of Canada, 2007). The interim response was vague and non-specific in addressing the Standing Committee's recommendations; it did not specify how or if they would be addressed. The interim response indicated while CEPA 1999 is fundamentally sound and does not require significant changes, refinements to the Act would strengthen its implementation (Parliament of Canada, 2007). After the House of Commons Standing Committee on Energy, the Environment and Natural Resources submitted its report in 2008, the Government of Canada was to table a final consolidated response to both committee reports addressing the recommendations and potentially proposing various improvements to the Act (Environment Canada, 2012; Parliament of Canada, 2007). However, a final response from the government was not released. There have been no official revisions to the legislation as it is not mandatory to incorporate the Standing Committees' recommendations from the review period. The next five year review of CEPA 1999 should have been triggered on March 31, 2010 but the review was suspended (Environment Canada, 2011a). Despite comprehensive reviews and being officially scheduled for review twice, CEPA 1999 has not changed since the Act was implemented in 1999. It should be scheduled for another review as of March 31, 2015.

Chemicals Management Plan

Toner (2008) notes that when the Conservative government came into power in 2006, it had shown little commitment to prioritizing the environment in its electoral

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trichloroethylene on the Priority Substances List to the publication of its management plan took over thirteen years.

campaign or the first Speech from the Throne. However, there was a shift in public opinion in 2006-2007 with Canadian citizens prioritizing the environment as a policy issue of concern (Toner, 2008: 3). The launch of Canada's *Chemicals Management Plan* was announced by Prime Minister Stephen Harper, Minister of Health Rona Ambrose and Minister of the Environment Tony Clement in December 2006. The government promoted an approach that was "tough on toxics" as part a "comprehensive environmental agenda" (Bueckert, 2006; Campion-Smith, 2006; Conservative, 2013; Prime Minister of Canada, 2006a; Scott, 2009b; Weeks, 2006). CEPA 1999 is the primary statute under which the *Chemicals Management Plan* is implemented. The Government indicated that the *Chemicals Management Plan* was designed to assess and manage the risk of all chemical substances categorized as part of the Domestic Substances List as potentially harmful to human health or the environment by 2020. Prime Minister Harper promoted the *Chemicals Management Plan* as including

> realistic and enforceable measures that will substantially increase protection of Canadians from dangerous chemicals. In fact, it will make Canada a world leader in the testing and regulation of chemicals that are used in thousands of industrial and consumer products….Over the next four years, we will tighten regulations and accelerate risk assessment for thousands of chemicals. Our plan will require substantial investment of public funds, but in the long run it will save money by reducing expenditures on public health and the clean-up of contaminated land and water.
>
> While Canada has always been responsible when it comes to chemical management, I'm proud to say that we will become a world leader because of today's announcement. Although since 1994, new chemicals substances produced or imported into our country have been subject to rigorous assessment by federal government scientists, some 23,000 "legacy" chemicals have not undergone the assessment required of new substances. All developed countries face the same challenge, and all have committed to safely manage chemicals by 2020.

> Canada has now become the first country in the world to achieve full categorization of our legacy chemicals. We are ahead of America and Europe, and Canada's New Government is committed to keeping our nation at the forefront of health and environmental protection. Our chemicals management plan is the next step in the process (Prime Minister of Canada, 2006b).

The *Chemicals Management Plan* is managed by Environment Canada and Health Canada and incorporates all the existing federal chemical programs into one single strategic policy in order to address routes of exposure to chronic and acute hazardous substances (Treasury Board of Canada, 2012). Health Canada (2010: 35) promotes the *Chemicals Management Plan* and its

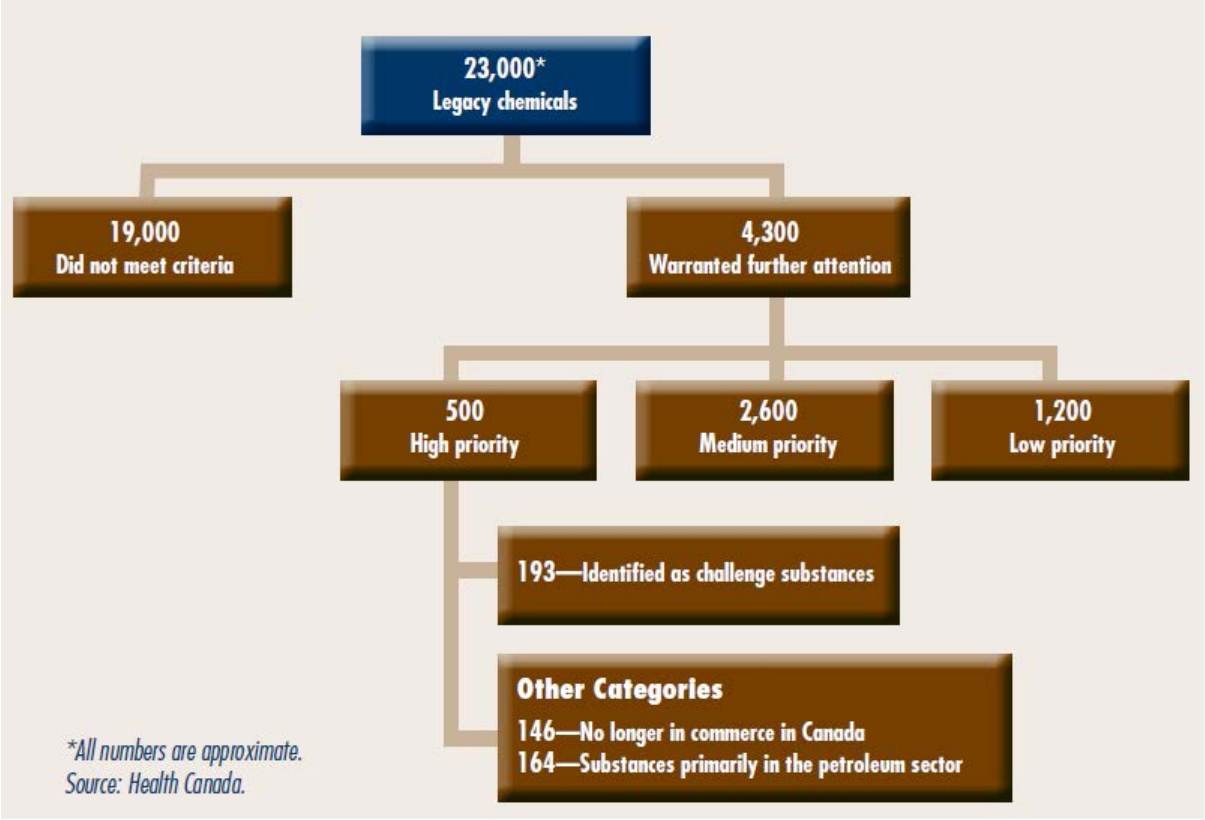
> innovative approach to regulation [which] supports the use of the best-placed and most effective Act to address the potential risks of a chemical substance…[The] government's regulatory actions should be proportional to the identified risks, as well as be the most cost effective and efficient in achieving the risk-management objective.

The *Chemicals Management Plan* is designed to protect human health and the environment by taking immediate action on chemical substances of high concern; undertaking regulatory activities to address sectors such as consumer products, food, pharmaceuticals, personal care products, and pesticides by using the most-appropriate Act; investing in research including biomonitoring to examine the health impacts associated with chemical exposures; and evaluating the success of risk management and control measures (Health Canada, 2010a: 34). The three key elements of the *Chemicals Management Plan* include a challenge to industry and stakeholders for immediate action on toxic substances of high concern; regulatory activities around food, cosmetics, drugs, and pesticides; and investment in research and monitoring about both the effects of toxic

substances on human health and the environment, and as a way to measure the success of risk management processes (Government of Canada, 2011a: 1-2).

The 500 high priority substances identified during the categorization process will be addressed through three mechanisms including the Ministerial Challenge Program (also known as "the Challenge), the Petroleum Sector Stream Approach[49] and through the Significant New Activities (SNAc) provisions. Of the chemicals that were classified as the highest priorities for immediate action, 193 substances were identified as part of the Challenge, with assessments to be completed between 2007-2010, 146 were no longer in commerce and classified under the significant new activity provisions, and 164 were substances used primarily in the petroleum sector (Health Canada, 2010a). An overview of the "Chemicals Categorization Process: A Large-Scale Priority-Setting Exercise" can be seen in the diagram below:

[49] The Petroleum Sector Stream approach includes 160 substances identified as high priorities through the categorization process. The majority of high priority petroleum substances are used or manufactured during petroleum refining or bitumen/heavy crude oil upgrading activities. Environment Canada and Health Canada are responsible for the assessment and management of risk associated with these substances (Government of Canada, 2013a).



(Health Canada, 2010a: 33)

The Challenge involves collecting data from industry under the information gathering provisions of section 71 of CEPA 1999. It includes a notice published in the *Canada Gazette* and a Challenge Questionnaire. Information required as part of the questionnaire includes details about the total quantity of a substance that was manufactured, imported, released, used, or sold for use in Canada; the concentration of the substance in a mixture, product or manufactured item; and use pattern codes and North American Industry Classification system codes that apply to the use of a substance (Government of Canada, 2009a). There is also a non-mandatory request that industry and stakeholders submit additional information that may be used as part of the risk

assessment process, and to develop best practices around risk management and product stewardship (Government of Canada, 2011b). For instance, information may be provided about the import, manufacture and use quantities; substance and product use details; releases to the environment and protocols for spill management; current and potential risk management and product stewardship actions; existing legislative and regulatory programs which control or manage the substance; and information to support the development of a regulatory impact assessment. The information is intended to assist the government in designing approaches and tools for the risk management of the Challenge substances (Government of Canada, 2009a, 2010a).

The 193 substances as part of the Challenge were divided into twelve smaller groups called "batches" to be addressed sequentially and launched within a three-year timeframe. Beginning in February 2007, a new group of 15-30 substances were released every three months for a six-month comment period from industry and stakeholder groups (Government of Canada, 2009a; 2010a). Screening risk assessments were conducted for each batch of substances by Health Canada and Environment Canada (Tilman and Rochon Ford, 2010).

The *Chemicals Management Plan* utilizes a risk management approach which includes scientific assessment and monitoring, combined with a variety of tools for the protection of human and environmental health (Health Canada, 2010a). The non-governmental organization, Environmental Defence[50], reported in 2011 that of the 193

[50] Environmental Defense is a Canadian environmental action organization whose research and campaigns focus on banning harmful chemicals, protecting green space, greening power sources, cleaning beaches, greening the economy, and detoxing Canadians (Environment Defense, 2013a).

high priority substances assessed as part of the Challenge between 2007 and 2010, twenty-five substances were determined to be toxic under CEPA 1999 and added to the Toxic Substances List, fourteen were determined to be toxic and proposed for addition to the Toxic Substances List, and six will likely be concluded as toxic in final assessments and proposed for addition to the Toxic Substances List (Environmental Defence, 2011: 14). Batches one and two each found nine substances to be toxic. However, the number of substances found to be toxic decreased in subsequent batches with three substances in batch three, four substances in batch four, two substances in batch five, one substance in batch six, and three substances in batch seven. There has been frequent use of the "future use notification" measure[51] and the proposed cosmetic ingredient hotlist[52] which are both

[51] The future use notification tool was identified early in the Challenge, but it was determined that the SNAc provisions of CEPA 1999 would fulfill this risk management measure. The future use notification tool wording was replaced with Significant New Activity provisions wording, eliminating the need to develop another regulatory initiative (Environment Canada, 2013a).

[52] The Cosmetic Ingredient Hotlist contains a list of prohibited and restricted cosmetic ingredients in Canada (Government of Canada, 2011c; Health Canada, 2011a, 2011b). The Hotlist is based in legislation including section 2 of the *Food and Drugs Act* which addresses the definitions of regulated products; section 16 of the *Food and Drugs Act* which states that no person shall sell a cosmetic product that contains a substance that may injure the health of the user; and section 24 of the *Cosmetic Regulations* which requires that the label of a cosmetic product presenting an avoidable hazard include directions for safe use. If a restricted ingredient is in a product, a "cautionary statement of direction for use associated with an ingredient mitigates the hazard of the product" (Health Canada, 2011b). Health Canada scientists use evidence-based decision making and weight of evidence in their risk assessments (CCTFA, 2007). Evidence-based decision-making is the "systematic application of the best available evidence in the evaluation of options for decision-making in clinical, management and policy settings" (Ham, 2001: 99). While evidence-based medicine focuses on the individual-clinical level, evidence-based decision-making and health policy focuses on the population-policy level (Dobrow et al, 2004: 208). The fundamental concepts of an evidence-based decision are evidence and context. It is the interaction between evidence and context in evidence-based decision making that is most critical to the development of evidence-based health policy (Dobrow et al, 2004). Health Canada may implement risk management measures including banning ingredients or restricting use through the Hotlist, requiring labelling, or requiring the product be removed from stores (CCTFA, 2007). Manufacturers may have to remove the ingredient from the formulation; reduce the concentration of the ingredient to an acceptable level; provide evidence that the product is safe for its intended use; confirm that the product is labelled as required; and confirm that the product is sold in child-resistant packaging (Health Canada, 2011b). However, the Hotlist has been criticized as it has no legal authority and cannot be enforced (David Suzuki Foundation, n/d). de Leon and

non-regulatory risk management measures. At the same time, the number of significant new activity (SNAc) provisions have steadily increased from batch one through batch seven and were applied to thirty-three substances while only three substances have been scheduled for virtual elimination (de Leon, 2010).

Under section 80 of CEPA 1999, significant new activity (SNAc) provisions apply when the Minister of the Environment or Health conclude that a substance is entering the environment in a quantity which is significantly greater than the previous release. These provisions may also apply if the substance is entering the environment in a manner that is significantly different than the previous release into the environment. The significant new activity provisions were applied to 146 substances classified as high priority under the *Chemicals Management Plan*. These substances were categorized as persistent, bioaccumulative and inherently toxic, but were not currently in commerce in Canada. If the Minister of the Environment or the Minister of Health is concerned that a significant new activity of a substance on the Domestic Substances List is being reintroduced into Canadian commerce which may result in the substance being classified

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Madray (2009) note important gaps in the Hotlist such as a lack of clarity around whether manufacturers or importers abide by the provided limits, and the Hotlist does not require exporters of cosmetic products to comply with the regulations. "This is a significant flaw, not only of the Hotlist but of the management regime for toxic chemicals in Canada. The use of CEPA toxic chemicals should not be permitted for products intended for the export market" (de Leon and Madray, 2009: 2). The Hotlist does not provide consideration for the impact on vulnerable populations who are exposed to the substances (de Leon and Madray, 2009). For a full discussion of vulnerable populations and exposure to toxic substances, refer to chapter 5. It should also be noted that personal care products which may be classified as drugs are not regulated under the *Food and Drugs Act* because they possess a therapeutic function, such as antiperspirants, face cream with a UV rating, anti-aging lotion, toothpaste, and hand sanitizers. Products which may be regulated as natural health products if they contain natural ingredients with a therapeutic function are also not regulated under the Act. Despite substances which are found to be toxic under section 64 of CEPA 1999, the Hotlist and labelling requirements in the *Cosmetic Regulations* do not apply to personal care products which are classified as drugs or natural health products (David Suzuki Foundation, n/d).

as CEPA-toxic, the Minister of the Environment may amend the Domestic Substances List under section 87(3) so that the new use of the new substance is evaluated (de Leon, 2010; Government of Canada, 2012a, 2012b). The outcomes of an assessment of a significant new activity may result in the substance being suspected of being toxic or capable of becoming toxic for the proposed activities, or not suspected of being toxic or capable of becoming toxic for the proposed activities. If the substance is suspected of being toxic or capable of becoming toxic, the Ministers may

> i) permit any person to manufacture or import the substance in relation to the Significant New Activity, subject to any conditions that the Ministers may specify;
>
> ii) prohibit any person from importing or manufacturing the substance in relation to the Significant New Activity; or
>
> iii) request any person to provide any additional information or submit the results of any testing that the Ministers consider necessary for assessing whether the substance is toxic or capable of becoming toxic, as a result of the Significant New Activity (Government of Canada, 2012b).

In a letter to the Director Generals of Environment Canada and Health Canada, de Leon et al. (2010) suggest that there has been an over-reliance on Significant New Activity provisions under the *Chemicals Management Plan* and express concern around the high priority substances that were identified for Significant New Activity Notices. "Prior to the release of the CMP [(*Chemicals Management Plan*)], the original intention was to apply SNAcs to substances considered "new" to Canada and subject to the New Substances Notification Regulation. Under the CMP, we have noticed a continuing trend toward issuing SNAcs to high hazard-low volume 'existing' substances without designating them as CEPA toxic" (de Leon et al., 2010: 7). A number of specific

concerns around the Significant New Activity provisions under the *Chemicals Management Plan* are raised including:

- The threshold for reporting use is 100kg which means that there may be uses of the substance below the reporting threshold and that does not apply to a Significant New Activity issuance;

- The Significant New Activity approach requires further assessment of toxic substances under the New Substances Program and these results are not required to apply elimination or reduction strategies as part of risk management, regardless of the original data gathered in the categorization process;

- The substances to be assessed and managed under the Significant New Activity approach of the *Chemicals Management Plan* include 146 from the high priority substances and thirty-three from batches one through seven. A more protective and precautionary approach would involve listing all these substances as toxic under CEPA 1999 and proposing to add them to CEPA's Prohibition of Specific Toxic Chemicals Regulations; and

- Despite the efforts by environmental non-governmental organizations to raise the issue of applying Significant New Activity notices under the *Chemicals Management Plan*, there has been very limited public policy debate around this issue (de Leon et al., 2010: 7-8).

The Significant New Activity approach does not fully protect human health and the environment. de Leon et al. (2010) conclude in urging the government to designate substances that meet the hazard criteria and are not in use, manufactured or imported into Canada as toxic under CEPA 1999, and add the CEPA-toxic chemicals to Schedule 1.

Other provisions under the *Chemicals Management Plan* include assessing the medium priority substances, monitoring and research including the Canadian Health Measures Survey and the Maternal Infant Research on Environmental Chemicals; mandatory ingredient labelling of cosmetics[53], regulations to address environmental risks

[53] Importantly, this labelling does not include "fragrance" or "parfum" ingredients which are protected by proprietary conditions.

from pharmaceuticals and personal care products under the *Food and Drugs Act*; rapid screening of lower risk chemicals; and accelerated re-evaluation of pesticides under the *Pest Control Products Act* (Government of Canada, 2010b; 2011a).

In October 2011, the federal government announced the second phase of the *Chemicals Management Plan* focused on consumer product safety. Minister of the Environment, Peter Kent stated that this phase "is both an investment in the health of the Canadian economy and our environment. Canadians want to have confidence in the products they use everyday, and reassurance that they are not harmful to the environment" (Health Canada, 2011d). Specifically, the Government stated that this phase will focus on continuing to improve product safety in Canada; completing assessments of 500 substances across nine categories; and investing in additional research for substances such as bisphenol A (BPA), flame retardants, and substances that affect hormone function and the environment. There are approximately 1,000 additional substances to be assessed over a five year period through other initiatives including the rapid screening of substances which pose "little or no risk" (Health Canada, 2011d).

As part of the second phase of the *Chemicals Management* Plan, the Substance Groupings Initiative involves assessment of priority substances between 2011 and 2016 in order to assess and manage the potential health and environmental risks associated with nine groups of substances including aromatic azo- and benzidine-based substances, boron-containing substances, internationally classified substances, certain organic flame retardants, cobalt-containing substances, methyldiphenyl diisocyanates and diamines, phthlates, selenium-containing substances, and substituted diphenylamines (Government

of Canada, 2012c, 2013b, 2013c; Laemy, 2012). This initiative emerged as a result of longstanding critiques, including from other jurisdictions, of the substance-by-substance approach rather than focusing on classes of substances (Denmark Ministry of the Environment Environmental Protection Agency, 2013; House of Commons Standing Committee on Environment and Sustainable Development, 1995; McClenaghan et al., 2003).

The substance groupings were chosen based on structural or functional similarities and assembled based on considerations of the ability to support informed substitution decisions, timing of international actions, stakeholder implications, assessment efficiencies, potential exposure to children and human health, and risk management efficiencies (Laemy, 2012: 6). The priorities for the assessment of the groupings initiative include potential hazard and exposure of the substances; efficiencies and effectiveness of risk assessment and risk management; transparency in the assessment and engagement with stakeholders throughout the process; and adaptability with the process regarding sub-groupings that may be revised as information becomes available (Government of Canada, 2011d).

The second phase of the *Chemicals Management Plan* also involves an inventory update for the Domestic Substances List as the original data for these substances may be out of date, some substances may no longer be in commerce, or their use and volume may have changed. There were originally 23,000 substances published on the Domestic Substances List in 1994, but there are currently 28,000 listed substances. Information from the updated Domestic Substances List will be used for risk assessment and

management activities, monitoring trends, and priority setting. Phase one of the update was launched in 2009 and included approximately 500 toxic substances. Phase two was launched in December 2012 and will involve approximately 2700 substances requiring reporting on chemicals manufactured or imported over 100kg alone or in a mixture in the 2011 calendar year, as well as polymers manufactured or imported over 1000kg alone or in a mixture in the 2011 calendar year (Government of Canada, 2013c; Télasco, 2012).

Conclusions

The proliferation of chemical contaminants used in industry, agriculture and consumer products in industrialized societies has led to increasing concern among Canadian citizens. Specific issues of concern are the health effects of exposure to environmental contaminants and particularly the long-term exposure to low-doses of contaminants, as well as the effects on developing foetuses, infants and young children (Health Canada, 2002b: 6). The measurement of the impact of environmental contaminants on human health is an important public policy challenge (Health Canada, 2002b). Public health and environmental policies have the potential to reduce environment-related diseases and contribute to significant improvements in public health.

Health Canada (2010a) has a wide range of instruments available that may be used in an attempt to achieve its public policy objectives including regulatory instruments such as legislation and regulations which are legally binding, and less formal non-regulatory instruments that encourage particular behaviours or actions such as economic incentives or disincentives and public education campaigns (Health Canada, 2010a: 7). The department works from the position that a completely risk-free environment is

neither realistic nor achievable, but rather that governments need a systematic decision-making process to determine the level of risk that is acceptable to both the environment and public health. Health Canada's approach to hazard identification, risk assessment and risk management is to develop a course of action that is evidence-based and cost effective. Risk should be reduced while also accounting for social, cultural, ethical, political, economic, and legal considerations (Health Canada, 2002b: 7). The importance of national environmental standards are emphasized as the only way to "ensure the right of all Canadians to the same minimum levels of health and environmental protection" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 15). The following chapters will investigate whether this claim is implemented in practice.

Chapter 4

Tracing Toxics: Considering Toxicity, Exposure, Precaution and Accountability

Introduction

In order to understand how the concept has evolved, this chapter examines the history of the concept of "toxicity" in Canadian legislation and the tensions between stakeholder groups during the regulatory review process. It explores how toxicity is grounded in risk assessment processes which are designed to assess the impact of toxic substances on the environment and human health. It also considers the role of toxicity in exposure assessments, threshold and non-threshold effects of exposure to toxic substances, and the requirements for virtual elimination under CEPA 1999. The chapter provides a brief overview of the case of siloxane D5, which was declared toxic under CEPA 1999 as part of the Challenge. This declaration was reversed after an objection was filed by the Silicones Environmental, Health and Safety Council of North America. The case study raises central questions about the regulation of toxic substances, risk, precaution, the contested nature of toxicity, and the influence of socioeconomic interests. This chapter also explores the precautionary principle which was included in Canadian legislation for the first time in CEPA 1999. However, the legislation is critiqued for not operationalizing the principle in the assessment of toxic substances. It provides an overview of the growing concern of the effects of endocrine disrupting chemicals, their inclusion in CEPA 1999 and whether the precautionary principle has been adequately implemented in risk management practices. Finally, it examines the debate between exposure-based and hazard-based assessments of risk and considers the efficacy of

Canadian law, policy and practice in preventing detrimental health outcomes and enacting primary prevention.

Tracing Toxicity, Risk and Exposure

In light of continued contestation around environmental health outcomes, it is interesting to reflect upon the history and context of environmental health and risk. Issues of (in)visibility of environmental contaminants and a tendency to emphasize some risks over others were recognized when the *Environmental Contaminants Act* was implemented (Page, 1978). A paper written by the Economic Council of Canada on the regulation of toxic chemicals in the environment in 1981 acknowledged the latent, long-term health effects of environmental contaminants. It emphasized that exposure to environmental contaminants can result in the development of cancer, birth defects, genetic damage, and other acute and chronic diseases (Nemetz et al., 1981). During the parliamentary review process of CEPA 1988, ninety percent of people surveyed were concerned about the effect of pollution on human health and additional surveys conducted at this time revealed that the presence of toxic chemicals in the environment was a major concern for Canadian citizens (House of Commons Standing Committee on Environment and Sustainable Development, 1995).

The Lalonde Report, which highlighted the environment as a determinant of health status, was an early indicator of environments as causal factors related to health outcomes (Lalonde, 1974). There has been an evolution in the language and terminology around toxic substances, as well as the formal definition of a "toxic substance" in Canada. Concerns were raised about the language used to describe environmental

chemicals at each stage of the parliamentary review of environmental legislation. Industry stakeholders felt that the use of "contaminants" to describe substances of concern in the *Environmental Contaminants Act* of 1975 unfairly stigmatized the industry and the substances themselves. The introduction of the term "toxic substances" in CEPA 1988 was meant to be less value-laden (House of Commons Standing Committee on Environment and Sustainable Development, 1995).

But industry representatives also expressed concerns with the use of the term "toxic substances" and the name of Schedule 1, the List of Toxic Substances under CEPA 1999. Because the Minister of the Environment and Minister of Health consider both the hazard and exposure of a substance that is defined as CEPA-toxic before placing it on Schedule 1, it was noted that a substance may be placed on the List of Toxic Substances as a result of detrimental effects at a high exposure level, but may be commonly (and safely, in their view) used under other circumstances. Industry representatives argued that "because of this, their products were being given an unfair stigma" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 45). For instance, the examples of road salt and ammonia were repeatedly raised in stakeholder consultations. Road salt[54] met two of the criteria to be defined as toxic including the potential to have an immediate or long-term harmful effect on the environment or its biological diversity, and the potential to constitute a danger to the environment on which life depends, although it was not added to Schedule 1. Ammonia was added to Schedule 1

[54] Similar to Health Canada's (1995) illustrative example using knives to contrast hazard- and risk-based approaches in chapter three, it is suggested that the public may easily confuse "toxic" and "high hazard" and may think they are "sprinkling such a substance on their french fries" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 45-46).

after a risk assessment of ammonia in the aquatic environment determined that it has the potential to have immediate or long-term effects on the environment or biological diversity. The main objection to the use of the word "toxic" by industry is that "it gives all Schedule 1 substances the same connotation of being something to be avoided at all costs" (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 45). Industry stakeholders recommended removing "toxic" entirely and replacing it with "substances to be managed," while stakeholders from environmental and health organizations suggested that the removal of the toxic substances language could diminish the importance of the impact of the regulatory decisions. While there have not been any revisions to CEPA 1999 during legislative review, there were two attempts to remove "toxic" from all or parts of the Act in Bill C-43 from the 38th Parliament, first session in 2005, and Bill C-30 of the 39th Parliament, first session in 2007 (House of Commons Standing Committee on Environment and Sustainable Development, 2007: 45-46).

> Under section 64 of CEPA 1999, a substance is considered "toxic" if
>
> > it is entering or may enter the environment in a quantity or concentration or under conditions that i) have or may have an immediate or long-term harmful effect on the environment or its biological diversity; ii) constitute or may constitute a danger to the environment on which life depends; or iii) constitute or may constitute a danger in Canada to human life or health.

Health Canada (2007c) equates the concept of risk with the definition of toxic under CEPA 1999 as it encompasses both the exposure to a substance and the hazard or inherent toxicity of a substance. Simply put, toxicity is equated with risk and this is the understanding that is used in Health Canada's decision-making framework for risk

assessment and risk management. In order to evaluate the potential impact of a toxic substance on human health, Health Canada (2013a) assesses the risk by: i) reviewing relevant decisions of other jurisdictions; ii) conducting initial screening assessments which consider the hazardous properties of the substance, routes of exposure and the potential to harm human health; and iii) conducting in-depth assessments for substances which are placed on the Priority Substances List which includes a critical and comprehensive analysis of the risks to human health (Health Canada, 2008b).[55] The risk assessments consider the use, hazard, exposure, and environmental fate of the substance, as well as the risk to human health. They consider acute exposure at the individual-level and chronic exposure at the population-level (Environment Canada, 2013b; Saner, 2010).

A risk assessment is designed to determine a range of toxicological effects by utilizing the dose-response relationship. The traditional dose-response relationship posits that the "nature, number, severity, incidence[,] and/or prevalence of specific toxicological effects *increase with increasing exposure*, as determined by the dose, duration and frequency" of the toxic substance (Health Canada, 2007c, emphasis added). Toxicological effects may be classified as "threshold" or "non-threshold." Threshold effects only occur above a certain level of exposure (Health Canada, 2007c; Saner, 2010). Health Canada (2010: 6) defines a toxicological threshold as a "dose below which no adverse effects to the exposed organism will occur." Under this model small doses of a toxic substance are expected to be tolerated by the human body because of metabolic

[55] Refer to chapter three for additional detail on human health risk assessments and these processes in CEPA 1999.

detoxification, physiological homeostasis, and cellular adaptation and repairs. Risk assessments attempt to identify the highest dose of a substance that does not result in adverse health outcomes, also known as the "No-Observed-(Adverse)-Effect-Level" (NO(A)EL) (Health Canada, 2007c: 6).[56]

Non-threshold effects, in contrast, are considered to occur at any level of exposure to a substance. During screening level assessments of existing substances under CEPA 1999, a non-threshold risk for a cancer endpoint results in the substance being found CEPA-toxic (Health Canada, 2000; Saner, 2010: 11). After this designation, the exposure component is considered in subsequent decisions, such as determining whether the substance will be added to Schedule 1 of the Act, and which risk management measures will be taken (Health Canada, 2007c: 6-7).[57]

Ultimately, determinations of toxicity are dependent upon "whether or not the potential level of exposure is below that for which the health risk is considered significant, or for which the health risk is considered negligible" (Health Canada, 2007c: 3). Risk assessments utilize "margins of exposure" in order to determine the ratio between the NOE(A)L and estimated exposure level of the substance (Barnes and Dourson, 1988; Scott and Lewis, forthcoming; USEPA, 2000). The margin of exposure

[56] If the data does not allow for the determination of a NO(A)EL, the "Lowest-Observed-Adverse-Effect-Level" (LOAEL) would be used indicating the lowest dose at which an adverse effect occurs (Health Canada, 2007c: 6).

[57] For new substances, hazard and exposure are considered concurrently. A *de minimus* ("essentially negligible") risk level is determined for new substances which do not have a threshold effect. Substances will be classified as Group 1 ("Carcinogenic to Humans") or Group 2 ("Probably Carcinogenic to Humans") where the substance will be considered toxic if its risk is not negligible. Substances will be classified as Group 3 ("Possibly Carcinogenic to Humans," "Possible Human Germ Cell Mutagen" or if the weight of evidence indicates genotoxicity in somatic cells) where the substance will be suspected of being toxic if the risk is not negligible (Health Canada, 2007c: 8).

may be used in determining both non-cancer and cancer endpoints and are based on broad, population-level estimates (Environment Canada, n/db; Government of Canada, 2013d).

Human health exposure assessments consider exposure through a variety of sources including food, air, water, dust, soil, and consumer products, and a variety of pathways including ingestion, inhalation and dermal absorption. The exposure assessments consider scenarios including direct exposure and environmental (indirect) exposure. Direct exposure to toxic substances occurs most often through inhalation and dermal contact. Direct exposure of the general public results from "direct contact with, or close proximity to, the chemical during any part of its lifecycle, whether knowingly or not" (Health Canada, 2007c: 4). Environmental (indirect) exposure occurs when there are toxic substances present in food, drinking water, domestic and recreational water, air, dust, and soil. Environmental exposure occurs as a result of substances entering the "general environment through industrial waste streams, from releases from intended industrial uses, air emissions, household wastewater and landfill sites" (Health Canada, 2007c: 5). Direct human exposure is distinguished from indirect exposure, as there is no pathway in the environment that interferes between the point of release and the point of exposure. However, it may not always be possible to make a distinction between the two exposures. The toxicological effects which may occur are evaluated as part of the exposure assessment and include organ- or system-specific effects such as cardiovascular or neurological/behavioural; reproductive and developmental; immunological; carcinogenic; or mutagenic effects (Health Canada, 2007c: 4-5).

Section 65(1) of CEPA 1999 gives a legislative basis for the virtual elimination requirement of the Toxic Substances Management Policy for Track 1 substances. Here, virtual elimination is defined as the ultimate reduction of the quantity or concentration of the substance in the release below the level of quantification specified by the Ministers in the Virtual Elimination List. Section 77(4) of CEPA 1999 states that when

> i) the substance is persistent and bioaccumulative in accordance with the regulations; ii) the presence of the substance in the environment results primarily as a result of human activity; and iii) the substance is not a naturally occurring radionuclide or a naturally occurring inorganic substance, the Ministers shall propose the implementation of virtual elimination under subsection 65(3).

Both Williams (2006) and the House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources (2008) raised concerns about substances needing to be *both* persistent and bioaccumulative in order to qualify for virtual elimination. This requirement means that substances that are CEPA-toxic and persistent, but do not bioaccumulate, are not targeted for virtual elimination under CEPA 1999. It is suggested that the definition of virtual elimination be revised to be broader in scope, similar to the one used in the International Joint Commission and the Great Lakes Water Quality Agreement (Williams, 2006; House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008).

The Standing Senate Committee (2008) conducted a case study that focused on perfluorinated compounds and on perfluorooctane sulfonate (PFOS) in particular. PFOS is a synthetic chemical that was imported from the United States to Canada to be used in numerous processes and products including water, oil, soil, and grease repellents, firefighting foams, hydraulic fluids, mining and oil surfactants, and carpet spot removers.

A State of the Science report prepared by Health Canada as part of a screening health assessment for PFOS determined that the majority of Canadian citizens have low levels of perfluorinated compounds, including PFOS, in their blood. However, the report ultimately concluded that there are adequate margins of exposure to prevent detrimental health outcomes (Health Canada, 2006; Health Canada, 2007d). Health Canada concluded that PFOS and its salts meet the criteria for persistence under CEPA 1999 and that "while the weight of scientific evidence indicates that PFOS and its salts are also bioaccumulative[,]…the relevant data for these substances do not meet the numeric criteria for bioaccumulation as defined in the CEPA 1999 *Persistence and Bioaccumulation Regulations*" (House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008: 33). Based on this result, PFOS did not qualify for virtual elimination under CEPA 1999 and environmental non-governmental organizations suggested that the criteria for bioaccumulation be expanded. Consequently, a Member of Parliament introduced the Private Member's Bill C-298 to Parliament in October 2007 to add PFOS to the Virtual Elimination list and the Bill received Royal Assent in April 2008 (House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008; Parliament of Canada, 2008b).

In the next section, I illustrate the degree to which findings of toxicity are contested in the case of siloxane D5 which was determined to be a toxic substance under CEPA 1999. The Silicones Environmental, Health, and Safety Council of North America subsequently filed an objection which reflects the deeply vested interests around issues of production and risk.

Case Study: Siloxane D5 (CEPA-toxic?)

Siloxane D5 (cyclopentasiloxane, decamethyl-) is an industrial chemical used in a variety of products and processes. It is not manufactured in Canada, but it is imported into the country as a pure substance, in mixtures with other cyclic siloxanes, as a residual in silicone polymers, and in finished consumer products. Based on information received as part of a notice published under section 71 of CEPA 1999, between 1,000,000 and 10,000,000 kilograms of siloxane D5 were imported into Canada in 2006 (Environment Canada and Health Canada, 2008a). The most common use of siloxane D5 in Canada is in blending and formulating personal care products, such as hair and skin care products, antiperspirants and deodorants. It is also used in manufacturing silicone polymers, and in textiles, paints, sealants, lubricants, plastics, non-medical ingredients in pharmaceuticals, silicone polymers, food additives, surface treatments for wounds, and medical devices (Environment Canada, 2012f; Government of Canada, 2012d). Siloxane D5 may be released into the environment as a result of industrial processes and from the use and disposal of personal care products. Thus, air, wastewater and soil are the "principal receiving environmental media for [siloxane] D5 based on its physical-chemical properties and its use patterns" (Environment Canada and Health Canada, 2008: ii).

It was determined during the categorization process of the Domestic Substances List that siloxane D5 was in commerce in Canada, and met the ecological criteria for persistence, bioaccumulation potential, and inherent toxicity to non-human organisms. This substance was identified as a high priority for screening assessment and included in the Challenge of the *Chemicals Management Plan*. Siloxane D5 was originally assessed

as part of Batch 2 under the Challenge (Environment Canada and Health Canada, 2008a). A notice announcing the release of the final screening assessment report for siloxane D5 was published in the *Canada Gazette* in January 2009 (Government of Canada, 2009b, 2012e). The screening assessment conducted by Environment Canada and Health Canada (2008a: 51) concluded that siloxane D5 has the potential to cause ecological harm and meets the criteria for being defined as "toxic" under section 64 of CEPA 1999. It was determined by the Minister of the Environment that siloxane D5 "is entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity" (Environment Canada and Health Canada, 2008a: 51).

Siloxane D5 was not classified as a priority for in-depth assessment of the potential risks to human health during the categorization process. However, a human health assessment was conducted based on its structure and use pattern similarity to siloxane D4 (octamethylcyclotetrasiloxane) which was determined to be a high priority for assessment for risks to the environment and human health under CEPA 1999. The screening assessment of siloxane D5 considered exposure, health effects and the characterization of risk to human health. The estimates of exposure relied on the use of models and use pattern data which were not from Canadian studies. It is suggested that the extent of use of the substance in personal care products and in other products may be lower than the estimated dose and that exposure estimates from personal care products may be overestimated (Environment Canada and Health Canada, 2008a: 43-45).

The health effects of siloxanes are not covered extensively in the literature. It is noted in the health effects assessment that siloxane D5 has not been classified for carcinogenicity, genotoxicity or reproductive/developmental toxicity by an international agency. The assessment does reference reports by the Danish Environmental Protection Agency and the United States Environmental Protection Agency (Environment Canada and Health Canada, 2008a). While acute toxicity, irritant effects, sensitization and genotoxicity are not reported to be health effects of concern for siloxane D5, Lassen (2005) found that there are potential health effects related to repeated exposure to the lung and potential carcinogenic effects including uterine tumours. This is consistent with a study submitted by Dow Corning to the United States Environmental Protection Agency under the *Toxic Substances Control Act*. The study evaluated chronic toxicity and carcinogenicity of siloxane D5 on rats and concluded that the highest level of exposure resulted in a significant increase in uterine tumours (USEPA, 2009). Environment Canada and Health Canada (2008a) suggest that the high exposure levels related to these findings may be due to threshold effects.

The screening assessment identified considerable uncertainties in the risk of siloxane D5 to human health. For example, it noted that the assessment did not:

> take into consideration a full analysis of the mechanism of action of decamethylcyclopentasiloxane and it does not take into account possible differences between humans and experimental species in sensitivity to effects induced by this substance. There is uncertainty surrounding the mechanism of carcinogenicity following exposure via the inhalation route….There is uncertainty regarding the estimation of exposure and systemic dose because of the use of modelling and a lack of Canadian data (Environment Canada and Health Canada, 2008a: 50-51).

However, based on the available information and the overall findings of the screening assessment, it was determined that siloxane D5 is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to human life or health in Canada (Environment Canada and Health Canada, 2008a). Thus, the finding of toxicity made under the Challenge was based on meeting the criteria for ecological toxicity alone.

A proposed order to add siloxane D5 to Schedule 1, List of Toxic Substances under CEPA 1999 was published in the *Canada Gazette* in May 2009. The Government stated that the addition of siloxane D5 to Schedule 1 under section 90(1) would allow for the development of regulatory and non-regulatory measures to manage the human health and/or environmental risks (Government of Canada, 2009c). As risk management measures, the Government of Canada proposed the creation of concentration limits for siloxane D5 in order to minimize its release into municipal wastewater streams through the use of personal care products. It also proposed limiting the release of the substance into the environment from wastewater produced as part of manufacturing processes (Environment Canada, 2010f).

Within two months, in July 2009, a Notice of Objection was filed by the Silicones Environmental, Health, and Safety Council of North America which is a not-for-profit trade association representing silicone chemical producers and importers in North America (Thomas, 2009).[58] The trade association requested the establishment of a formal

[58] After a forty year history, the Silicones Environmental, Health, and Safety Council transitioned to become the Silicones Environmental, Health, and Safety Center in January 2013. The Silicones Environmental, Health, and Safety Center is a sector group of the American Chemistry Council with membership representing over ninety percent of the silicone chemical manufacturing capacity in North

Board of Review in response to the proposal to add siloxane D5 to Schedule 1. Under section 333(2) of CEPA 1999, the Minister of the Environment may establish a board of review to conduct an inquiry "into the nature and extent of danger" posed by siloxane D5. The Notice of Objection was filed by Karluss Thomas, Executive Director of the Silicones Environmental, Health, and Safety Council of North America and argued that a Board of Review

> is warranted as the Proposed Order to add…[siloxane] D5 to Schedule 1 is based on final screening assessments…that have been conducted in a manner that is not consistent with the best available science. Use of the best available science would not have resulted in the conclusion that…[siloxane] D5 "may cause adverse effects to aquatic organisms in certain Canadian environments" and "have the potential to cause ecological harm" (Thomas, 2009).

The Notice of Objection is primarily concerned with conflicting evidence around the bioaccumulative potential of siloxane D5. Further, the Silicones Environmental, Health, and Safety Council of North America contended that there would be potential socioeconomic consequences if siloxane D5 were listed on Schedule 1, including "severe global market impacts to Canadian companies importing, processing, and using, these substances." The trade association called for a Board of Review to "be convened to prevent a premature, inadequately supported Schedule 1 listing" (Thomas, 2009).

Upon receiving the Notice of Objection, the Government of Canada (2012e) considered new scientific information on siloxane D5 which became available from industry studies submitted to Environment Canada in January 2010, as well as scientific studies conducted by Environment Canada and in other jurisdictions. Based on the

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America including Bluestar Silicones, Dow Corning Corporation, Evonik Goldschmidt Corporation, Momentive Performance Materials, Shin-Etsu Silicones of America, Milliken (formerly SiVance), and Wacker Chemical Corporation (American Chemistry Council, Inc., 2013).

availability of new information, the Minister of the Environment established a Board of Review in August 2010. According to the terms of reference, the Board of Review would conduct an inquiry into the nature and extent of the danger posed by siloxane D5 and submit a final report to the Minister of the Environment before March 31, 2011 (Government of Canada, 2010c). The Chair of the Board of Review submitted a letter dated November 12, 2010 to the Minister of the Environment requesting an extension for the final report. The Board received information suggesting that new information would not be available until the end of 2010 or early 2011 and required an adequate amount of time in order to conduct a "thorough and comprehensive review of the nature and extent of the dangers posed by Siloxane D5." The Board committed to submitting its final report and recommendations by September 30, 2011. The Board of Review received a letter from the Minister of the Environment acknowledging this request on August 30, 2011. This letter required the report be translated into French and extended the final deadline for submission to October 31, 2011 (Siloxane D5 Board of Review, 2011: 76-77).

The Minister of the Environment appointed three toxicologists[59] to serve on the Board of Review. The Board consulted with each of the parties involved in the proceedings including Environment Canada; the applicant, Silicones Environmental, Health and Safety Council of North America; and the interveners, the Canadian

[59] The three members of the Board of Review included Chair, Dr. John Giesy who is the Canada Research Chair of Environmental Toxicology, Department of Veterinary Biomedical Sciences and Toxicology Centre at the University of Saskatchewan and a Distinguished Professor of Zoology Emeritus at Michigan State University; Dr. Sam Kacew who is the Associate Director of Toxicology at the McLauglin Centre for Population Health Risk Assessment and Professor in the Department of Cellular and Molecular Medicine at the University of Ottawa; and Dr. Keith Ross Solomon who is a Professor Emeritus in the Department of Environmental Science and Director of the Centre for Toxicology at the University of Guelph (Government of Canada, 2011e).

Cosmetic, Toiletry and Fragrance Association, and a coalition consisting of the Canadian Environmental Law Association, the International Institute of Concern for Public Health, Chemical Sensitivities Manitoba, and the Crooked Creek Conservancy Society of Athabasca. The Board of Review determined that it would focus its review on the nature and extent of the danger posed by siloxane D5 to the environment based on the initial screening assessment, additional information that became available, and direction from the Minister of the Environment (Government of Canada, 2010c; Siloxane D5 Board of Review, 2011).

The Board of Review investigated the nature and extent of the risk posed by siloxane D5 to the environment and whether detrimental effects may occur as a result of exposure to siloxane D5. The Board conducted a *de novo* risk assessment[60] that considered all available information surrounding the intrinsic physical and chemical properties of siloxane D5, as well as its toxicity, uses, exposures, and effects (Siloxane D5 Board of Review, 2011: 10). Specific findings of the Board of Review included:

- Siloxane D5 exceeds the regulatory threshold for persistence, but does not exceed the thresholds established in the *Persistence and Bioaccumulation Regulations*;
- While siloxane D5 can be accumulated into organisms from environmental matrices or food, it does not biomagnify through the foodchain; and
- Siloxane D5 will not accumulate to sufficient concentrations to cause detrimental effects in organisms in air, water, soils, or sediments (Siloxane D5 Board of Review, 2011: 9).

Thus, the Board of Review concluded that siloxane D5 does not pose a danger to the environment or its biological diversity (Siloxane D5 Board of Review, 2011: 9).

[60] A *de novo* risk assessment means that the Board of Review did not assess whether the conclusions of the Ministers of the Environment and Health were reasonable, but rather conducted its own assessment.

Based on the conclusions provided by the Board of Review, the federal government published a revised decision on siloxane D5 in February 2012. In reversing its decision, the Government concluded that siloxane D5 does not meet any of the criteria under section 64 of CEPA 1999 and that this substance is not entering the environment in a quantity or under conditions that constitute a danger to the environment. The Government formally annulled the original decision to add siloxane D5 to the List of Toxic Substances and all related risk management activities (Government of Canada, 2012e, 2012f).

The Board of Review for siloxane D5 was the first to be established under CEPA 1999. The reversal of the original decision by the Government of Canada, prompted by the Silicones Environmental, Health, and Safety Council of North America, raises questions about the application of the precautionary principle and may set a precedent for future regulation of toxic substances. In particular, the Board's conclusions "may make it more difficult for federal scientists to build a case for restricting problematic chemicals in future, particularly at a time when Environment Canada is already facing severe cuts to its overall budgets and still faces the task of completing assessments of approximately 1500 chemicals under Canada's Chemicals Management Plan over the next few years" (CELA, 2012b).

The Role of the Precautionary Principle

Decision-making under CEPA 1999 is said to be guided by the application of the precautionary principle. The precautionary principle is often promoted by environmental health advocates as an approach that encourages regulatory action when some evidence

of harm exists, despite uncertainty or contestation (Ley, 2009: 81). Vogel (2012: 75) suggests that it may be "the dangers that we do not yet adequately understand or know about [that] are likely to be more serious than those about which we already know." The precautionary principle is grounded in "anticipatory action in the absence of complete proof of harm, particularly where there is scientific uncertainty about causal links" and allows for decision-makers to act in a precautionary and protective manner in order to prevent harm to humans and the environment from exposure to toxic substances (Tickner, 1997). This principle guides a precautionary approach to decision-making in order to make risk assessment and management decisions around pollution prevention and the release of toxic substances into the environment (Ogilvie, 2001; Tickner, 1997).

The preamble of CEPA 1999 commits the Government of Canada to implementing the precautionary principle "where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation." This is consistent with the most widely used definition of the precautionary principle from the Rio Declaration on Environment and Development which was established in 1992 (Ogilvie, 2001). Principle 15 states that "[i]n order to protect the environment, the precautionary approach shall be widely applied.…Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation" (United Nations, 1992).

Vlek (2009) considers the Rio Declaration version of the precautionary principle used in CEPA 1999 to be weaker than the definition proposed at the Wingspread Conference in 1998 by treaty negotiators, activists, academics, and scientists from Canada, the United States and Europe. Participants in the conference

> believe existing environmental regulations and other decisions, particularly those based on risk assessment, have failed to protect adequately human health and the environment…[T]here is compelling evidence that damage to humans and the worldwide environment is of such magnitude and seriousness that new principles for conducting human activities are necessary (Science and Environmental Health Network, 2013).

The Wingspread Consensus Statement promotes the implementation of the precautionary principle when "an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof" (Science and Environmental Health Network, 2013; Wilson, 2005). Unlike the definition in CEPA 1999, the Wingspread definition does not include provisions around "serious or irreversible damage" or cost-effectiveness. Many health and environmental groups promote the Wingspread definition as an alternative to the Rio Declaration definition used in CEPA 1999 because it also implies a duty to act (McClenaghan et al. 2003; Ogilvie, 2001).

The precautionary principle is also included in the administrative duties of CEPA 1999 under section 2(1) where the government must

> (a) exercise its powers in a manner that protects the environment and human health, applies the precautionary principle that, where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as reason for postponing cost-effective measures to prevent environmental degradation, and promotes and reinforces enforceable pollution prevention approaches;

> (j) protect the environment, including its biological diversity, and human health, from the risk of any adverse effects of the use and release of toxic substances, pollutants and wastes; and
>
> (k) endeavour to act expeditiously and diligently to assess whether existing substances or those new to Canada are toxic or capable of becoming toxic and assess the risk that such substances pose to the environment and human life and health.

While the precautionary principle allows for the justification of taking action, Environment Canada and Health Canada (2004: 11) note that the complexity of environment and health issues mean that most decisions cannot reflect absolute certainty and the use of the precautionary principle will vary from case-to-case depending on the degree of scientific certainty and the irreversibility or potential damage.

The House of Commons Standing Committee on Environment and Sustainable Development (2007) contends that the inclusion of the precautionary principle in the administrative principles of the Act means that the government is obliged to apply it. However, CEPA 1999 is critiqued for not operationalizing the precautionary principle in the assessment of toxic substances (Scott and Lewis, forthcoming). Environment and health advocates argue that there is a duty to act in accordance with the precautionary principle, to protect human health and the environment from exposure to substances which are CEPA-toxic and substances which are inherently toxic and have the potential to cause harm.

The House of Commons Standing Committee on Energy, the Environment and Natural Resources (2008: 4) note concerns that the legislation is sound but inadequately implemented. The Committee "believes that the lack of will to implement and enforce the

Act, and a shortage of necessary resources for that implementation and enforcement, are the weak links in the effectiveness of the CEPA [1999] environmental protection regime." There has been "little to no action taken to limit or manage a chemical before a complicated legal and political process confirms its toxicity" (CELA, 2007: 49). Dr. Kapil Khatter of PollutionWatch also pointed to the mechanisms under CEPA 1999 that had not yet been utilized.

> CEPA [1999] gives the federal government the powers to regulate any substance that it deems to endanger our health or the environment. It offers the government a range of tools to reduce pollution and to prevent harm. CEPA [1999], though, has not been effective in reducing pollution in Canada or in getting the worst chemicals off the market (House of Commons Standing Committee on Energy, the Environment and Natural Resources, 2008: 4).

The regulatory system is based on the principle that a certain level of risk is unavoidable which raises questions about the "acceptability of risk." Early risk management was based around the idea that the public could be completely protected from all risks (Health Canada, 1998). This is simply not possible within the risk society where we are exposed to substances which are invisible, do not respect territorial boundaries and have lengthy latency periods. These risks and subsequent health outcomes are often not the result of one single high-dose exposure, but rather the result of synergistic exposure to long-term cumulative doses of complex mixtures of substances. There is a lack of consensus around levels of acceptability and the risks associated with exposure to toxic substances. For example, in 1999, a Member of Parliament, the Honourable Charles Caccia asked "[w]hat comfort is it to Canadians if toxic chemicals get catalogued and assessed, but not necessarily eliminated?" (Batt, 2002: 5; Parliament of Canada, 1999).

Industry stakeholders propose that risk is acceptable in the name of socioeconomic progress. By factoring "*financial* risks and benefits into the same equation as *health* risks and benefits, risk management frames illness as an acceptable trade-off for economic prosperity and/or jobs" (Batt, 2002: 6). Among the general public, it is suggested that the degree of acceptance for risks related to health is low (Bouder, 2006). Environmental and breast cancer activists question the ethics around the acceptability of exposing populations to toxic substances which may result in detrimental health outcomes; exposing pregnant women to toxic substances which may result in their child developing health issues later in life; and exposing women to toxic substances which may result in the development of breast cancer.

The acceptable levels of risk associated with established guidelines and margins of exposure vary up to a million-fold (Health Canada, 1998: 9). Based on the notion that risk is unavoidable and to some degree, acceptable, there has been an emphasis on risk management over precaution (Scott and Lewis, forthcoming). Health Canada (1998: 2) concludes that risk management strategies provide a "high degree of health protection, based on the absence of observable health effects using epidemiological methodology." The risk management approach emphasizes control or reduction rather than elimination or substitution (CELA, 2007). As Scott (2009b: 70-71) notes:

> [i]t is increasingly clear that the central assumptions of our risk assessment models are completely ineffective at capturing the complexity that characterizes contemporary pollution harms. Advocates believe that 'precaution' demands allegiance to 'an entirely new set of assumptions', including the vulnerability of the 'environment' and 'bodies', and the serious limitations of our science with respect to the accurate prediction of the interactions between chemicals, between environments and bodies, and between chemicals and bodies. Most importantly,

> precaution assumes the availability of alternative, less harmful processes and products.

In this respect, precaution becomes particularly relevant in the case of exposure to endocrine disrupting chemicals, which are ubiquitous, display complex mechanisms and may result in detrimental health outcomes at low doses.

Endocrine Disrupting Chemicals

There has been growing concern from the public about the effects of endocrine disrupting chemicals on the environment and human health. The House of Commons Standing Committee on Environment and Sustainable Development received testimony about this issue as early as the legislative review of CEPA 1988. In their briefing to the Committee submitted in 1994, the Canadian Environmental Law Association raised concerns about the effects of endocrine disrupting chemicals.

> In recent years there has been growing concern that some chemicals – particularly persistent, bioaccumulative, chlorinated hydrocarbons – may be the cause of a variety of serious effects, including reproductive, developmental and behavioural abnormalities, in both humans and other species. The possible effects of such chemicals on the reproductive integrity of humans, particularly the suggested estrogenic properties of some pollutants, have now developed into a priority issue (House of Commons Standing Committee on Environment and Sustainable Development, 1995: 33).

Consistent with Beck's (1992) argument that toxic substances do not respect territorial boundaries, stakeholders expressed concern about pesticides and PCBs which originated in the southern industrial and agricultural regions of North America, Europe and Asia and are detected in wildlife in northern Canada and in the breast milk of northern aboriginal women (House of Commons Standing Committee on Environment and Sustainable Development, 1995).

It is suggested that the precautionary principle should be utilized in the management of endocrine disrupting chemicals (Servos et al., 2001; World Wildlife Fund Canada, 1998). Industries and sites of concern around endocrine disrupting chemicals include municipal effluents, agriculture, textile mill effluents, pulp and paper sector, mining and metal work, automotive, food canning, bars, casinos and racetracks, historically contaminated sites, identified areas of concern such as the Great Lakes, and contaminants in the Arctic including aboriginal food sources (Brophy et al., 2012; Servos et al., 2001). There is potential for risk assessment processes to "be used to identify effects produced via endocrine disrupting mechanisms, but subtle effects on growth, reproduction and development must also be considered" (Servos et al., 2001: 337). In order to adequately account for the impacts of endocrine disrupting chemicals, risk assessments need to consider sensitive life history stages, windows of susceptibility, the significance of delayed responses and effects, and the effects of mixtures and mixture interactions (Servos et al., 2001: 337).

Unlike the original Act, CEPA 1999 requires the Minister of the Environment and the Minister of Health to conduct research on hormone disrupting substances (Environment Canada, 2010e; Environment Canada and Health Canada, 2004).[61] Part 3, Section 44(4) under the Monitoring, Research and Publication requirements of CEPA 1999 involves research on the health and environmental impacts of endocrine disrupting chemicals. Specifically, the Minister of the Environment and Minister of Health must

[61] Hormone disrupting substances are defined under section 43 in CEPA 1999 as a "substance having the ability to disrupt the synthesis, secretion, transport, binding, action or elimination of natural hormones in an organism, or its progeny, that are responsible for the maintenance of homeostasis, reproduction, development or behaviour of the organism."

> conduct research or studies relating to hormone disrupting substances, methods related to their detection, methods to determine their actual or likely short-term or long-term effect on the environment and human health, and preventive, control and abatement measures to deal with those substances to protect the environment and human health.

Section 45 also requires the Minister of Health to i) conduct research and studies relating to the role of substances in illnesses or health problems; ii) collect, process, correlate and publish on a periodic basis data from any completed research or studies; and iii) distribute the available information to inform the public about the effects of substances on human health. This requirement has the potential to play an important role in preventing diseases such as breast cancer which are influenced by the role of endocrine disrupting substances. However, the ways in which the government is required to follow-through are unclear and the requirements in CEPA 1999 lack specificity with vague timelines such as a "periodic basis" that are open to interpretation. Ecojustice and the Canadian Environmental Law Association submitted a petition to the Office of the Auditor General of Canada in July 2012 seeking information about the federal research activities under Environment Canada and Health Canada on the effects of hormone disrupting substances as required by CEPA 1999. The petition inquires about how the data is collected on substances considered new under CEPA 1999; about the budget allocated to research and the involvement of Canada in international research initiatives; and how Environment Canada and Health Canada are using research results for risk assessment and risk management under CEPA 1999. Replies to the petition from Environment Canada and Health Canada are not yet available (Office of the Auditor General of Canada, 2012).

PBDE Flame Retardants and Phthalates

The House of Commons Standing Committee on Environment and Sustainable Development (2007) suggests that other federal governments have applied the precautionary principle more rigorously than Canada in cases such as polybrominated diphenyl ether (PBDE) flame retardants and phthalates in the European Union (House of Commons Standing Committee on Environment and Sustainable Development, 2007). PBDE flame retardants are lipophilic, bioaccumulative and have endocrine disrupting properties. These substances are an issue of global concern; their chemical structure is similar to PCBs and DDT and their distribution in the environment follows similar patterns as they are widespread moving beyond territorial boundaries (Rahman et al., 2001). The Restriction of the use of certain Hazardous Substances in electrical and electric equipment (RoHS) in the European Union bans new electrical and electronic equipment containing more than designated maximum allowable levels of PBDE flame retardants as of 2006 (Bromine Science and Environmental Forum, 2013; Steven Engineering Inc., 2013). This directive also requires that polybrominated diphenyl ether and polybrominated biphenyl flame retardants and heavy metals including lead, mercury, cadmium, and hexavalent chromium are substituted by safer alternatives (European Commission, 2013).

Exposure to endocrine disrupting phthalates is ubiquitous. The European Union ministers voted unanimously in 2004 to ban the use of di(2-ethylhexyl) (DEHP), di-n-butyl (DBP) and n-butyl benzyl (BBP) phthalates from use in children's toys in concentrations greater than 0.1 percent. This decision was grounded in the precautionary

principle and is viewed as protective of health and the environment (Euractiv, 2004). The European Union listed phthalates DEHP, DBP and BBP on the REACH Candidate List in October 2008, they were subsequently included on the Authorisation List in February 2011 and are scheduled to be phased-out by February 2015 (Plasticisers and Flexible PVC Information Centre, 2010). Danish Minister of the Environment announced a complete ban of DEHP, DBP, BBP, and DIBP (diisobutyl) phthalates in a wide range of consumer products and has also proposed banning the four phthalates at the EU level (Euractiv, 2012). Denmark has developed a progressive Phthalate Strategy which is grounded in the precautionary principle in its concern about the environmental health implications as a result of exposure to phthalates. It also recognizes the "cocktail effect" of the mixture of substances and proposes to address the class of substances rather than the substance-by-substance approach. "It is neither efficient nor enough to introduce legislation on phthalates one by one. With this long-term strategy, we take into account that several phthalates have the same effect on the body, and that we are often exposed to several phthalates at once" (Denmark Ministry of the Environment Environmental Protection Agency, 2013; McClenaghan et al., 2003). The current legislation around phthalates in the European Union and Denmark involves processes including

- *Classification*: Twelve phthalates have European Union-harmonized classification with eleven classified as toxic to reproduction;
- *Authorization*: Seven phthalates have been included in the European Union Candidate List of Substances of Very High Concern; and
- *Restrictions*: There are concentration limits for six phthalates in toys and childcare articles in the European Union. Four phthalates have been banned in Denmark in a wide range of products in concentrations higher than 0.1 percent, and all phthalates have been banned in Denmark in toys and childcare articles for

> children ages 0-3 years in concentrations higher than 0.05 percent (Danish Environmental Protection Agency, 2013: 7).

The aim of the Danish Phthalate Strategy is to generate new knowledge about the risks associated with these substances and will potentially result in the restriction of other phthalates (Ministry of the Environment Environmental Protection Agency, 2013). In order to adequately address endocrine disrupting substances within Canada's legislative and regulatory frameworks, a specific national risk management strategy would need to be developed (Servos et al., 2001: 337).

Applying Precaution in the Assessment and Management of Risk

The precautionary principle has not been meaningfully applied in the regulation of toxic substances through CEPA 1999 and the *Chemicals Management Plan*. If the regulatory regime were to truly utilize the precautionary principle, the focus would shift to being precautionary rather than reactionary and would not assume that humans are meant to possess a body burden of toxic substances (Lewis, 2010; Seager, 2003). "It is seen as acceptable for there to be delays in responding or refusals to act based on gaps in the research data…[The government] has rarely taken preventive measures in the face of these uncertainties and has thereby allowed existing exposures to continue" (Lewis, 2010: 25). The duty to act and to assess and manage risks associated with toxic substances does not adequately incorporate the precautionary principle which will be further explored in the following discussion about exposure and hazard.

Risk and Hazard Assessment

Concerns about the mandatory exposure requirement in the definition of CEPA-toxic were raised as early as the Parliamentary Review of CEPA 1988. The House of

Commons Standing Committee on Environment and Sustainable Development (1995) recommended changing the definition of CEPA-toxic to include both a risk assessment and a hazard assessment. A hazard assessment 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. This discussion continued as part of Pollution Probe's report on standard setting for toxic substances in Canada which was released in 2001. Non-governmental and non-industry participants promoted hazard assessment as either an alternative or in addition to risk assessment under CEPA 1999 that would be able to "trigger the use of the precautionary principle, which requires actions to be taken to remove or minimize the potential risk" (Ogilvie, 2001: 57). Despite being raised as significant concerns as early as 1995, there have been no changes to the exposure requirement of CEPA-toxic or the risk assessment process as part of CEPA 1999. As a result, a toxic substance in Canada "cannot be regulated merely for having the inherent potential to cause harm" (Cooper et al., 2000: 202; Scott and Lewis, forthcoming).

The Canadian Environmental Law Association and Canadian Institute for Environmental Law and Policy (1996) cite toluene as an example of a toxic substance that possesses inherently toxic properties but was found to be not toxic under CEPA because of the exposure requirement. Toluene is an aromatic hydrocarbon which is used in cosmetics including nail polish and as a petrochemical solvent and paint thinner. This substance is linked to numerous health concerns including developmental and reproductive toxicity, neurotoxicity, and organ system toxicity (Environmental Working Group, 2013; Office of Environmental Health Hazard Assessment, n/d). "Toluene is

listed in virtually every provincial hazardous waste and occupational health and safety regulation in the country" (CELA and CIELAP, 1996: 106). However, while toluene was originally included on the Priority Substances List, it was subsequently found to be not toxic under CEPA[62] and, as such, is not subject to risk management provisions (Environment Canada and Health Canada, 1992; Health Canada, 2007e).

In California, toluene falls under the risk management provisions of the *Safe Drinking Water and Toxic Enforcement Act* of 1986, 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). The list is currently comprised of approximately 800 chemicals, including toluene (Office of Environmental Health Hazard Assessment, 2013; State of California, 2013a). 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

[62] The Government of Canada addressed paragraph 11 under CEPA in assessing the potential impact of toluene exposure on the environment and human health. Paragraph 11(c) considers the effects on human life or health and it was concluded that the estimated total average daily intake of toluene for the Canadian population is between 50-670 times less than the tolerable daily intake derived from bioassays in animal studies and data from clinical studies (Environment Canada and Health Canada, 1992: 16-17). However, it is noted that the available epidemiological data are unable to adequately assess the carcinogenicity of toluene in humans (Environment Canada and Health Canada, 1992: 15). Based on these findings, the Ministers of Environment Canada and Health Canada concluded that "the current concentrations of toluene present in the environment do not constitute a danger in Canada to the environment or to the environment on which human life depends or to human life or health. Therefore, toluene is not considered to be 'toxic' as interpreted under section 11 of the *Canadian Environmental Protection Act*" (Environment Canada and Health Canada, 1992: v).

adequately controlled under other state or federal regulation. However, it is also noted that this law has 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, and the carcinogens trichloroethylene and methylene chloride are no longer used in most correction fluids and reformulated paint strippers (Office of Environmental Health Hazard Assessment, 2013).

The European Union placed restrictions on toluene in 2004 so that the substance "shall not be placed on the 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). Seventy-four percent of member states (twenty countries) carried out enforcement action on toluene by 2012 (Armstrong and Dupont, 2012). The European Union has also restricted the content of toluene in nail products to twenty-five percent and included conditions of use which require that the label must contain warnings that the products be kept out of reach of children and used by adults only (Verheugen, 2009).

Traditional risk assessment and management processes fall short in the risk society (Beck, 1992). The risk assessment process determines toxicological effects utilizing threshold values and the dose-response relationship. The use of threshold values in the risk assessment process suggests that threshold effects occur only at a specific level of exposure, whereas non-threshold effects occur at any level of exposure to a substance or product. Health Canada (2007c: 6) 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, 2007c: 6).

However, 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 effects or result in cumulative exposures. There is a significant critique from the field of epidemiology of the dose-response relationship which is utilized in risk assessment processes and is based on traditional toxicology.

The traditional dose-response relationship posits that toxicological effects increase with increased exposure and dose of a toxic substance (Health Canada, 2007c). 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[63] and the traditional dose- response relationship.

> Whether low doses of EDCs [(endocrine disrupting chemicals)] 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).

[63] Traditional monotonic dose response curves demonstrate the relationship between the concentration of a toxic substance and an adverse effect (i.e., "the dose makes the poison"). However, non-monotonic dose response curves demonstrate situations where the effect of a toxic substance may be greater at lower doses than at higher doses (USEPA, 2013b).

In demonstrating the continued debate and contested nature around low dose exposures to endocrine disrupting substances, the United States Environmental Protection Agency (2013a) has recently produced a *State of the Science Report* to evaluate the impact of potential non-monotonic dose response relationships to estrogen-, androgen-, and thyroid-based modes of action and the current risk assessment and management processes. This comes in response to the Vandenberg et al. (2012) article that reviewed hormones and endocrine disrupting chemicals and in particular, their low-dose effects and non-monotonic dose responses. The article "criticized the [US] government's decades old-strategy for testing the safety of many chemicals found in the environment and in consumer products" (Bienkowski, 2013b). The Environmental Protection Agency's draft report concludes that the current testing of endocrine disrupting chemicals is adequate for detecting low-dose effects of toxic substances; the "current testing strategies are unlikely to mischaracterize, as a consequence of NMDR [non-montonic dose responses], a chemical that has the potential for adverse perturbations of the estrogen, androgen or thyroid pathways" (USEPA, 2013c: 15). A trade association, the American Chemistry Council, praised these conclusions stating that this "affirms what mainstream scientists have expressed for years: the purported scientific evidence for non-monotonic low dose exposures leading to endocrine disruption and adverse effects is, at best, very weak" (Bienkowski, 2013b). However, lead author of the *Endocrine Reviews* article, Dr. Laura Vandenberg criticized the draft report and the suggestion that high dose testing cannot predict safety or a lack of risk at low doses as it "flies in the face of our knowledge of how hormones work…[Endocrine disrupting chemicals] are overtly toxic

at high doses but act like hormones, with completely different actions, at low doses" (Bienkowski, 2013b). She also suggests that the Environmental Protection Agency has used out-of-date studies on both atrazine and BPA (Bienkowski, 2013b). The draft report will be peer reviewed through the National Academies of Science and through a public comment process (USEPA, 2013d).

Endocrinologists and other environmental health researchers and advocates are raising competing paradigms to contest the reliance on toxicology and the dose-response relationship in risk assessment processes (Darbre and Fernandez, 2013; Grossman, 2012, 2013; Pesch et al., 2004; Ritter, 2011; Vandenberg et al., 2009, 2012). This debate is clearly demonstrated by the divergent positions in recently published editorials by Dietrich et al. (2013) and a subsequent response by Gore et al. (2013). Editors of toxicology and pharmacology journals prepared the Dietrich et al. (2013) editorial which demonstrates the contestation around issues of the risk assessment of endocrine disrupting chemicals, threshold values and the precautionary principle. They contend that detrimental effects associated with endocrine disrupting chemicals can be determined exclusively through toxicity studies. The authors argue that

> regulations that profoundly affect human activities, that legally impose significant fines and even detention, should not be based on irrelevant tests forced to be regarded as relevant by administrative dictates, and on arbitrary default assumptions of no thresholds. Such standards would be contrary not only to science, but to the very principles of an enlightened governance and social contract. Not only scientists but society itself would pay dearly if unscientific approaches were to undermine our everyday practice of science, and the stringency of data analysis and evaluation developed by scientific thinking over the past centuries (Dietrich et al., 2013: A1).

Gore et al. (2013) published a formal response refuting Dietrich et al. in *Endocrinology.*

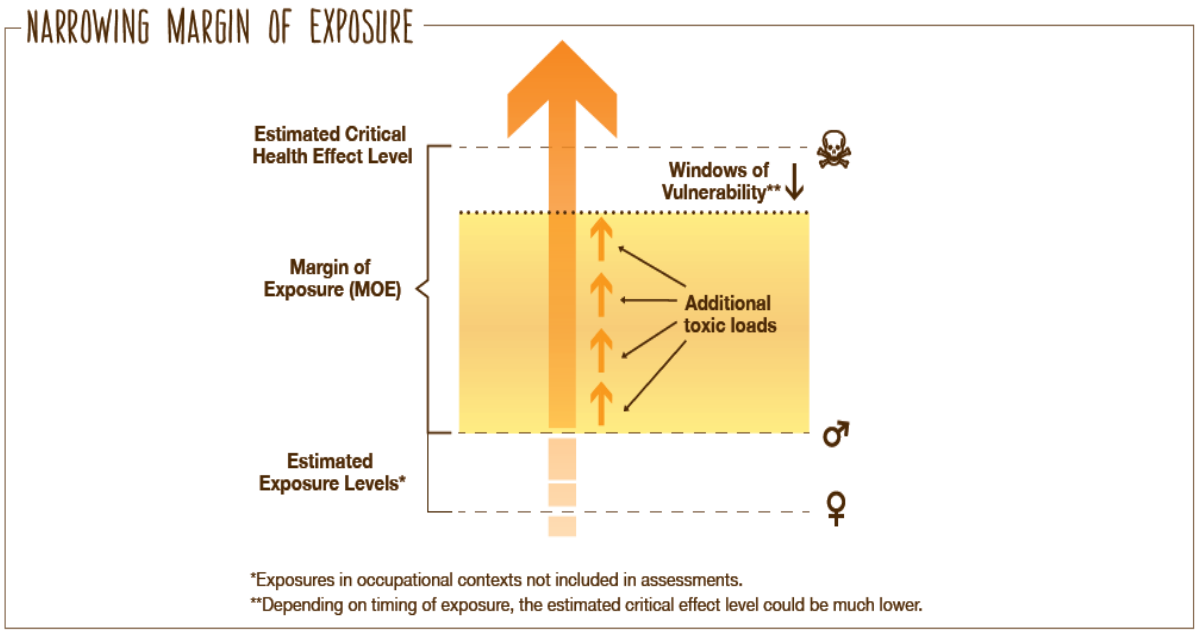
It is suggested that the latter article is "neither based on the fundamental principles of how the endocrine system works and how chemicals can interfere with its normal function, nor does it consider the consequences of that interference….[It] also ignores a growing and rigorous body of literature on both endogenous hormonal and exogenous EDC effects" (Gore et al., 2013: 3958). The authors contend that regulation of endocrine disrupting chemicals should be based on science and expertise from reproductive biology, endocrinology, medicine, genetics, behaviour, developmental biology, and toxicology (Gore et al., 2013).

The role of scientists in influencing law, policy and practice of the federal government has been diminishing in recent years in Canada. This is reflected in the 700 jobs cuts at Environment Canada in 2011 and repeated accusations of "muzzling" federal scientists from speaking publicly about peer-reviewed research results (CBC, 2011c, 2011d, 2013b; De Souza, 2013; Fitzpatrick, 2011; Gatehouse, 2013; Klinkenborg, 2013; Makuch, 2013; McLeod, 2013; Woods, 2013). However, the current regulatory regime remains highly dependent on very specific types of expertise, including the exclusive reliance on toxicology for the risk assessment processes associated with toxic substances. For instance, the Minister of the Environment appointed the representatives to the Board of Review for siloxane D5 in 2010 -- all three members are toxicologists (Government of Canada, 2011e).

The distinction between threshold and non-threshold effects is of particular concern with priority risks such environmental links to cancer (Saner, 2010). Lewis (2011: 21) notes that the margin of exposure assessment approach which is utilized in

determining whether toxic substances are "safe," involves determining the "difference between the estimated critical health effect level of the chemical (the threshold at which a chemical is considered harmful to human health or the environment), and its estimated exposure level." However, this approach does not account for the potential health effects of low dose and cumulative exposures or how sex and gender can affect the margin values. The current margins of exposure do not account for the timing of exposure, the impact of windows of susceptibility, and the role of gender in creating disproportionate exposure to toxic substances through domestic responsibilities (Lewis, 2011). Women 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 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).

It is likely that the margins of exposure used in risk assessments are vastly over-estimated in many cases. It is proposed that the margins of exposure should be narrowed in order to adequately account for the risk associated with exposure to toxic substances and specifically with the risk in relation to women's exposure and detrimental health outcomes (Lewis, 2011; Scott and Lewis, forthcoming). An approach which narrows the margins of exposure in order to adequately account for women's risk associated with exposure to toxic substances is reflected in the diagram below:



(Lewis, 2011: 21).

de Leon, Madray and Richardson (2010: 9) also recommend that risk management should be initiated by hazard rather than exposure data. There has been little emphasis on the elimination of toxic chemicals as an overall objective of the *Chemicals Management Plan* which is contradictory to the goal of pollution prevention as the cornerstone of CEPA 1999. The current practice of risk assessment in Canada enables widespread environmental contamination and detrimental health outcomes before the risks can be assessed and managed; the process is inherently reactionary rather than precautionary (de Leon, 2010: 6; Lewis, 2011). The regulatory process is unable to be truly precautionary as long as the exposure component is required as it becomes necessary for harm to occur before preventive measures can be established despite government endorsements of the precautionary principle.

Conclusions

There is a need for coherence across and among government policies and legislation in order to protect the health of Canadian citizens (Environment Canada and Health Canada, 2004: 13). This analysis indicates that Canada's environmental legislation is not capable of being protective with the exposure requirement in the assessment of risk. The use of an exposure-based approach in risk assessment processes rather than a hazard-based approach does not account for the inherent toxicity of a toxic substance. Despite the inclusion of the precautionary principle in CEPA 1999, the implementation of CEPA 1999 and the *Chemicals Management Plan* continues to be reactionary rather than precautionary in the assessment and management of toxic substances. The regulatory regime does not adequately account for low dose, cumulative exposures of toxic substances including endocrine disrupting chemicals. These gaps do not allow for the legislation to prevent detrimental health outcomes and enact a primary prevention approach to women's health. The following chapter will explore issues of sex, gender, risk and responsibility which emerge in Canadian law, policy and practice related to toxic substances.

Chapter 5

Toxics Regulation: Sex, Gender, Risk and Responsibility

Introduction

This chapter examines the relationship between theory and practice in an examination of the regulatory regime for toxic substances. It draws upon government publications, grey literature and media coverage in order to explore questions and tensions around risk, precaution, and prevention. The chapter begins with a discussion of the role of sex- and gender-based analysis in Canadian health policy. It asks where the burden of risk in preventing detrimental health outcomes is presumed to lie. It explores the responsibilization paradigm which places the onus of responsibility for assuming risks associated with everyday exposures on the individual, and the concept of precautionary consumption which encourages individuals to avoid everyday exposures to toxic substances through decision-making practices as consumers. The chapter then asks who is at risk of exposure to toxic substances and who the policies are designed to protect. Health Canada identifies children as a specific vulnerable population of concern, but women are not considered to be at-risk or a susceptible population. It provides an overview of the regulation of bisphenol A (BPA) in Canada and a critique of the limited scope of its regulation. It explores issues of occupational health exposures to toxic substances along with the difficulties related to accountability and compensation. Finally, this chapter also considers women's health and cancer organizations' messaging and campaigns around breast cancer which are influenced by or framed in response to government policies.

The Role of Sex- and Gender-Based Analysis in Canadian Health Policy

My focus in this research is on toxic substances management under CEPA 1999 and the *Chemicals Management Plan* and the primary prevention of breast cancer. There are, of course, broader policy areas in Canada related to breast cancer prevention, including those related to sex- and gender-based analysis. This section provides a brief overview of the policy history of health and sex- and gender-based analysis in Canada in order to provide context for a call to apply a gender lens in health research, and for the applied use of sex- and gender-based analysis within the development of regulation and policy relevant to environmental health.

In reviewing the history of breast cancer, it is evident that the disease is both highly sexed and gendered. The Women's Health Bureau of Canada was established in 1993 with a mandate of "enhancing Health Canada's capacity to promote equitable health outcomes for women and men, boys and girls in Canada" (Tudiver, 2009: 21). By signing the *Beijing Declaration and Platform for Action* at the Fourth World Conference on Women in 1995, Canada demonstrated its commitment to the recognition that both biology and social context have a significant influence on women's health. This document built upon the World Health Organization's definition of health as a "state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (WHO, 2003). The declaration utilized a broad definition which additionally identified women's health as involving "their emotional, social and physical well-being and [it] is determined by the social, political and economic context of their lives, as well

as biology" (Clow et al., 2009: 6). Just as sex and gender were used interchangeably during the 1990s, "women" and "gender" were used synonymously in discussions of health (Greaves, 2009). However, it is important to note that both men and women experience health effects related to gender.[64]

At the same time as the 1995 conference, the government published a federal plan entitled *Setting the Stage for the Next Century: The Federal Plan for Gender Equality* (Status of Women Canada, 1995). This report called for the implementation of gender-based analysis in federal departments and agencies including:

- the development and application of tools and methodologies to assist in the implementation of gender-based analysis;
- providing training on gender-based analysis of legislation and policies;
- the development of indicators to assess progress around gender equality;
- the collection and use of gender-disaggregated data;
- the use of gender-sensitive language across the federal government; and
- the evaluation of the effectiveness of gender-based analysis (Minister of Public Works and Government Services, 2009: 6).

The federal government also established the Women's Health Contribution Program in 1995 which was designed to address gaps in women's health research, as well as "improve the health status of women in Canada by enhancing the health system's understanding of, and responsiveness to, women's health issues" (Health Canada, 2010b). The Women's Health Contribution Program included the Atlantic Centre of Excellence

[64] There is a growing field of research on men's health which utilizes a gender lens (UBC, 2011). Greaves (2009: 17) attributes both the fields of gender and health, and men's health as positive by-products of feminist theory, activism and policy-making of the past forty years.

for Women's Health, the British Columbia Centre of Excellence for Women's Health, the Prairie Women's Health Centre of Excellence, and the Centre of Excellence for Women's Health - Consortium Université de Montréal. The Centres of Excellence[65] were established as multidisciplinary partnerships between academic researchers and community-based organizations to conduct policy-oriented research which investigates the way sex and gender interact with other determinants of health (Armstrong, 2012; Health Canada, 2003b). The Women's Health Contribution Program also funded the Canadian Women's Health Network[66], Réseau Québécois d'Action pour la Santé des

[65] The mandate of the Atlantic Centre of Excellence for Women's Health (ACEWH) includes contributing to research on women's health issues, promoting an understanding of gender as a critical variable in health outcomes, and enhancing the health system's responsiveness to the needs and concerns of women and girls. ACEWH's specific research focuses on health status and services; women's unpaid caregiving; HIV/AIDS; healthy living; obesity; sex- and gender-based analysis; gender mainstreaming; and social and economic inclusion (ACEWH, 2013). The mission of the British Columbia Centre for Excellence in Women's Health (BCCEWH) is to improve the health status of women by conducting innovative research and developing women-centred programs, practice and policies. Specific initiatives conducted by the BCCEWH include work around healthy choices in pregnancy; coalescing on women and substance use; pregnancy and smoking cessation; and developing *The Source*, a web-based resource of data sources, reports and synthesis documents related to women's health (BCCEWH, 2013).The mandate of the Prairie Women's Health Centre of Excellence (PCEWH) includes improving the health of women in Manitoba and Saskatchewan through making the health system and social systems more responsive to the health and well-being of women and girls. Program areas of the PCEWH include women and poverty; gender and health; rural, remote and Northern women's health; and Aboriginal women's health (PCEWH, 2013). Work conducted by the Centre of Excellence for Women's Health - Consortium Université de Montréal (CESAF) included caregiving, gender sensitive health indicators, experiences of immigrant and refugee women with the health care system, immigration and perinatal risk, and the health of Aboriginal women. The CESAF closed its office on August 31, 2001 (CWHN, 2012a).

[66] The Canadian Women's Health Network (CWHN) was created in 1993 as a voluntary national organization dedicated to improving the health and lives of girls and women in Canada by producing, sharing and distributing education and relevant information. In its commitment to women's health and equity, specific initiatives include establishing a visible national presence for women's health in Canada; providing user-friendly and reliable health information, resources and research; working to change inequitable health policies and practices by contributing women's voices and expertise; acting as a knowledge broker for researchers, clinicians, decision makers, media, and the public; encouraging and promoting community-based participatory research; monitoring emerging issues and trends affecting women's health; and acting as a form for debate on women's health research and policy issues (CHWN, 2012b).

Femmes[67], the National Network on Environments and Women's Health[68], specific research projects such as the Aboriginal Women's Health and Healing Research Group[69], and working groups including Women and Health Protection and the National Coordinating Group on Health Reform and Women[70] (Health Canada, 2003b, 2010b). While the individual organizations had distinct mandates, as a group they conducted research on issues which impact women's health including the role of social, economic and physical environments.

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[67] The Réseau Québécois d'Action pour la Santé des Femmes (RQASF) (Quebec Womens' Health Action Network) was founded in 1997 as a provincial, multi-disciplinary, non-profit organization. Their mission is to work closely with others in improving the physical and mental health of women, as well as their living conditions. Specific areas of interest of the RQASF include education campaigns around menopause; cognitive health and aging; homophobia and heterosexism; body image; and the medicalization of health (RQASF, 2013).

[68] The National Network on Environments and Women's Health (NNEWH) was founded in 1996 and is committed to producing policy-oriented research on the social, economic and physical environments that impact women's health. NNEWH seeks to improve the health of Canadian women through research which examines the ways in which environments impact the health status, beliefs and practices of women. NNEWH utilizes a sex-, gender- and diversity-based framework in its analysis of health research, policy development, and education materials. NNEWH benefits from expertise from academic research associates, community partners, service providers, and women's groups (NNEWH, 2013).

[69] The Aboriginal Women's Health and Healing Group represents a national network of First Nations, Métis and Inuit women researchers interested in community-based research focused on the health and healing of Aboriginal women, their families and communities. The group supports community-based health and healing research done by and with Aboriginal women, as well as developing policy recommendations (CWHN, 2012c).

[70] Women and Health Protection (WHP) is a coalition of community groups, researchers, journalists, and activists who are concerned with the safety of pharmaceutical drugs. WHP has focused on direct-to-consumer advertising, post-marketing surveillance, risk management and the precautionary principle, and the regulation of natural health products. WHP is particularly concerned with the impact of health protection legislation on Canadian women (WHP, 2010, 2012). Women and Health Care Reform (WHCR) is a multi-disciplinary, collaborative group that investigates and advises around the effects of health care reforms on women as providers, decision makers and users of health care systems. The mandate of WHCR is to coordinate research on health care reform in order to translate research into policy and practice. WHCR's areas of interest include ancillary health care work; environment; evidence about health and health care; gender and disaster management; gender and mental health of female health care workers; women and health care reform; home care; long-term care; maternity care; primary health care; principles of sex- and gender-based analysis of health care reform; private health insurance; privatization; quality of health care; and timely access to care (WHCR, 2013).

Health Canada's Women's Health's Strategy stated in 1999 that the department will "apply gender-based analysis to programs and policies in the areas of health system modernization, population health, risk management, direct services and research" (Health Canada, 1999; Health Canada, 2003b: 6). In *Exploring the Concepts of Gender and Health,* Health Canada (2003: 1) promotes the integrated use of gender-based analysis

> throughout the research, policy and program development processes [which] can improve our understanding of sex and gender as determinants of health, of their interaction with other determinants, and the effectiveness of how we design and implement sex- and gender-sensitive policies and programs.

It was during this time period that that there was a broader shift away from a focus on women's health and towards "gender and health." This shift is reflected in the federal government's adoption of the Gender-Based Analysis Policy in 2000. The intent of this policy was to attain gender equality through the use of gender-based analysis and fulfill the Government of Canada's domestic and international commitments to equality between men and women (Hankivsky, 2007a; Health Canada, 2010c). There was an attempt to mainstream gender issues, but some found these policies problematic as the focus on gender more broadly may be framed as a deliberate shift away from research and policy focused on women's health (Armstrong, 2012). The focus on gender may be framed as "less threatening to government than a woman-centred approach. Consequently, focusing on gender may be a way to avoid a focus on women and avoid funding women specific issues" (Saulnier et al., 1999: 7).

The Gender-Based Analysis Policy was replaced by the Health Portfolio Sex- and Gender-Based Analysis Policy in 2009 to develop, implement and evaluate research, programs and policies to address the different needs of men, women, boys, and girls. This

policy applies to the Health Portfolio of the Government of Canada which includes Assisted Human Reproduction Canada, Canadian Institutes of Health Research, Hazardous Materials Information Review Commission, Health Canada, Patented Medicine Prices Review Board, and the Public Health Agency of Canada (Health Canada, 2010c). The Health Portfolio is promoted as providing a

> comprehensive understanding of variations in health status, experiences of health and illness, health service use and interaction with the health system; the development of sound science and reliable evidence that addresses sex and gender health differences between men and women, boys and girls; and the implementation of rigorous and effective research, programs and policies that address sex and gender health differences between men and women, boys and girls (Health Canada, 2010c).

Advocates of sex- and gender-based analysis contend that it is essential for improving the health of Canadians in conducting health research and in the development and implementation of health programs and policies. Recognizing that "actions to reduce gender inequality will improve health for both women and men" (Clow et al., 2009: 8), the specific policy goals of the Sex- and Gender-Based Analysis Policy adopted by the Government of Canada in 2009 include:

- a comprehensive understanding of variations in health status, experiences of health and illness, health service use and interaction with the health system;
- the development of sound science and reliable evidence that addresses sex and gender health differences between men and women, boys and girls; and
- the implementation of rigorous and effective research, programs and policies that address sex and gender health differences between men and women, boys and girls (Health Canada, 2010c).

Based upon a recommendation from the House of Commons Standing Committee on the Status of Women in April 2008, the Auditor General of Canada conducted an audit

of the implementation of sex- and gender-based analysis policy by the federal government. The Auditor General of Canada conducted an audit of seven departments including the Department of Finance Canada, Health Canada, Human Resources and Skills Development Canada, Indian and Northern Affairs Canada, the Department of Justice Canada, Transport Canada, and Veterans Affairs Canada (Minister of Public Works and Government Services Canada, 2009). The audit found that "despite the government commitment to GBA [(gender based-analysis)] since 1995, there is no government-wide policy requiring that departments and agencies perform it" (Minister of Public Works and Government Services Canada, 2009: 2).

Importantly, sex- and gender-based analysis was found to be inadequately integrated into policy development. Only four of the sixty-eight initiatives integrated sex- and gender-based analysis into policy development including two at the Department of Finance Canada and two at Indian and Northern Affairs Canada. Despite a formal commitment to sex- and gender-based analysis in the Health Portfolio of the federal government, there were zero cases in Health Canada where sex- and gender-based analysis was performed and integrated into policy options development. In one case the department provided a rationale for not performing sex- and gender-based analysis, in three cases gender impacts were considered but not documented in the policy options developed, and in two cases there was no consideration of sex- and gender-based analysis (Minister of Public Works and Government Services Canada, 2009: 16). Additionally, the audit found that while Health Canada has a departmental policy and commitment in effect along with tools and methodologies readily available, training is not regularly

offered, a champion within the department has not been appointed, and sex- and gender-based analysis practices have not been evaluated (Minister of Public Works and Government Services Canada, 2009: 11).

> Sex- and gender-based analysis is argued to be:
>
> > vital to planning appropriate health programs and services, developing inclusive health policies and conducting research. It is effective because it requires policy-makers, scientists and researchers to think about who they are trying to serve and whose needs they are trying to meet (Lewis, 2011: 6).

There is also a need for an approach which incorporates intersectionality so that sex and gender are considered with other relevant factors including age, race, ethnicity, culture, geographic location, sexual orientation, and socioeconomic status (Hankivsky et al., 2010; Paterson, 2010; Tudiver, 2009). There is a call for challenging and transforming policy paradigms in the "process of engendering policy" (Hankivsky, 2005: 980; 2009: 116). Sex- and gender-based analysis should examine and critique the influence of the broader social, political and economic environments which impact health outcomes (Hankivsky, 2009).

The audit clearly demonstrates that sex-and gender-based analysis is not being adequately incorporated in health policy or legislation in Canada. Sex and gender must be accounted for in public health policy and legislation as the lack of implementation can have real implications for health outcomes among Canadian citizens (Butler-Jones, 2012). This discussion raises important questions about where the burden of risk and responsibility is presumed to lie in the prevention of disease. The Government of Canada (2011f) promotes risk as being within the control of Canadian citizens in suggesting that "we are all risk managers."

Where is the Burden of Risk Presumed to Lie?

Responsibilization Paradigm

The majority of discussions around disease prevention still focus on personal responsibility, accountability and modifiable risk factors. "The dominant view of cancer prevention has focused almost exclusively on individual lifestyle changes" (Chernomas and Donner, 2004: 3). There is a long history which focuses on the role of lifestyle and personal behaviours in health outcomes. The role of lifestyle emerged as a key area of research and aspect of health promotion in the Lalonde Report (Lalonde, 1974). The individualization of health and illness resulted in a responsibilization paradigm (Orsini, 2007: 349). This ideology places the onus of responsibility on the individual and suggests that 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. This approach to health promotion does not recognize other social determinants of health, particularly those outside of one's control such as environmental contaminants (Orsini, 2007; Simpson, 2000).

The role of lifestyle and personal responsibility as the primary factors in the development of cancer were established in part by Doll and Peto's foundational monograph which was published in 1981. Doll and Peto's work set the stage for the promotion of this paradigm in the medical and public health communities in its emphasis on personal responsibility around smoking, alcohol consumption, reproductive and sexual behaviours, and diet, while simultaneously downplaying the role of environmental and occupational risks to contributing to only two percent of cancer deaths from pollution and

four percent from occupational exposures (Clapp et al., 2006; Doll and Peto, 1981). These findings are continually cited by "commentators who argue that 'cleaning up the environment' is not going to make much difference in cancer rates" (Clapp et al., 2006: 62). The Harvard Center for Cancer Prevention published a report entitled *Human Causes of Cancer* in 1996 which built upon Doll and Peto's work in its focus on modifiable risk behaviours. It suggests that "the public can become overly concerned about minimal risks while losing sight of major cancer risk factors that can be controlled or modified, in particular, tobacco use, diet, exercise, and sun exposure" (Harvard Center for Cancer Prevention, 1996: S3; Clapp et al., 2006: 63). Colditz and Hunter (2000) further develop the Harvard Center for Cancer Prevention reports in later work which continues to focus primarily on lifestyle factors regarding cancer prevention.

> For major reductions in the burden of cancer to be achieved, we need broad scale interventions that will shift the behavior of the whole population. Rather than focus on individuals defined as being at "high risk", a shift in behavior by the whole population can achieve greater reductions in cancer (Colditz and Hunter, 2000: 325-26).

While there is value in public health policy and campaigns which promote healthy lifestyles, it is highly problematic when primary prevention efforts are focused solely on modifiable behaviours and lifestyle factors. It is important to acknowledge the distinction between primary prevention in the field of environmental health and the prevention efforts focused on modifiable behaviours in traditional cancer prevention which are grounded in and directly influenced by the Doll and Peto paradigm. For instance, the Canadian Partnership Against Cancer was established in 2006 as a federally funded non-governmental organization to implement Canada's cancer control strategy. The

prevention focus of the Canadian Partnership Against Cancer operates largely under the umbrella of healthy communities and lifestyles with a significant emphasis on modifiable behavioural factors around tobacco, nutrition, physical activity, alcohol, and ultraviolet radiation (Canadian Partnership Against Cancer, 2009a, 2009b, 2013a, 2013b).

The Cancer 2020 program was developed by Cancer Care Ontario in collaboration with the Canadian Cancer Society as a long-term plan for cancer prevention and screening. Cancer 2020 is grounded in an understanding that approximately fifty percent of cancers that will be diagnosed over a twenty year period can be either prevented or detected early and aims to provide a long-term provincial plan for cancer prevention in Ontario (Cancer 2020 Steering Committee, 2003a). The program focuses on well-established risk factors as an effective avenue for cancer prevention with specific efforts focused on methods to change the risk behaviours of Ontario citizens including promoting healthy eating and physical activity, and reducing alcohol consumption and cigarette smoking (Cancer 2020 Steering Committee, 2003a, 2003b, 2006). This program does include occupational and environmental carcinogens, though some of that focus still falls under behavioural practices such as reducing tobacco smoke and demonstrating sun-protective safety practices for workers. Occupational cancer surveillance programs are recommended and in broader environmental practices, setting standards around drinking water and reducing air pollution are suggested (Cancer 2020 Steering Committee, 2006). However, while the Cancer 2020 Program acknowledges occupational and environmental carcinogens as playing a role in the development of cancer, its focus for action remains

primarily on methods to change the risk behaviours of citizens (Cancer 2020 Steering Committee, 2003b: 17).

Precautionary Consumption

The individualization of risk is consistent with both the dominant epidemiological paradigm and the responsibilization paradigm, and is reflected in the promotion of "precautionary consumption" practices. MacKendrick (2010) observed a shift in media discourse around the bioaccumulation of chemicals and the body burden over a twenty year period from 1986 to 2006. These findings reflect a move away from collective forms of prevention around the risks associated with everyday exposures to toxic substances and towards personal responsibility through behavioural practices and precautionary consumption. The practice of precautionary consumption encourages individuals to take responsibility for protecting their health and to avoid exposure to toxic substances by purchasing "green" consumer products. Precautionary consumption promotes a sense of individual empowerment and control through the act of green consumption and chemical avoidance (MacKendrick, 2010: 127).

The ideology of this practice emphasizes the precautionary principle and the agency of individual consumers as the primary mechanisms for risk management around everyday exposures to toxic substances and the chemical body burden (MacKendrick, 2010: 43). Risks are now perceived as something that can be controlled by the individual. The risk frame "redistributes the responsibility for decision-making about risk from government agencies to self-governing 'consumer-citizens'" (Scott, 2007: 37; MacKendrick, 2010). Women's health and environmental organizations are influenced by

the individualization of risk and promote practices of precautionary consumption which encourage consumer-citizens to protect themselves from risks. The Women's Healthy Environments Network launched their "Wanna Be Toxic Free" campaign in 2010 to educate the public about the risks associated with toxic substances in consumer products and how to choose safer alternatives. Information about this program is made available through the organization's website, at fundraising events and as part of Community Environment Days which take place annually in neighbourhoods in Toronto from April to September. Specific substances that individuals are encouraged to avoid as part of this campaign include:

- parabens which are endocrine disrupting chemicals and suspected carcinogens, and are widely used as preservatives in the cosmetic and pharmaceutical industries;
- phthalates which are classified as reproductive toxicants in the European Union and are commonly used as plastic softeners or solvents in perfumes and fragrances;
- triclosan which is suspected to have carcinogenic and endocrine disrupting properties and is used as an antibacterial agent in toothpaste, mouth wash, deodorants, shaving creams, and hand sanitizers;
- butylated hydroxyanisole (BHA) and butylated hydroxytoluene (BHT) which are suspected endocrine disrupters and carcinogens used as preservatives in moisturizers and makeup;
- sodium laureth sulfate which is a skin irritant used in shampoos and bubble baths;
- polyethylene glycol compounds (PEGs) which are potentially carcinogenic and act as foaming agents in cleansing products;
- diethylalomines (DEAs) which are used in skin lotions, shampoos and sunscreens, and have demonstrated negative health effects in mice including inhibiting brain development and spontaneous abortion;

- petrolatum which is used as an emollient in hair products, lip balms, lip sticks, and moisturizers and may be contaminated with polycyclic aromatic hydrocarbons which are carcinogenic;
- coal tar dyes which are used in some hair dyes and dandruff treatments, are potentially carcinogenic, and may be contaminated with toxic heavy metals; and
- fragrance and parfum which are used in many personal care products and the ingredients are not available to the consumer because of proprietary interests (WHEN, 2013).

Campaigns such as Wanna Be Toxic Free[71] encourage individuals to exercise precaution in order to protect themselves from everyday exposures to toxic substances and subsequent health outcomes. However, these campaigns do not include a recognition that precautionary consumption is "undeniably women's work" (Kearns, 2011; Scott and Lee, n/d). These practices create a gendered and disproportionate burden on women who often have the primary responsibility for both the sustainability of the household and the health of their family members (Lee, 2011). "This practice reinforces women's socially prescribed roles as providers for the household, adding to their 'care burden' from both a physical and emotional perspective, and contributing to the gendered divisions of labour and exploitation of women's unpaid work in the home" (Scott and Lewis, forthcoming).

The measures of precautionary consumption that are encouraged by government and women's health and environmental organizations also make a number of assumptions about the women targeted by these campaigns. They assume a particular level of language proficiency, literacy and scientific understanding, as well as the economic ability to exercise choice, and a significant time commitment in encouraging women to

[71] While the primary focus of this campaign is on precautionary consumption, the Women's Healthy Environments Network does support the assessment and management of risk by the federal government and calls for phasing out chemicals that are carcinogens, reproductive toxins or mutagens in personal care products (WHEN, 2013).

read labels with long and complicated ingredients on cleaning, cosmetics and personal care products.

Altman et al. (2008: 426) describe the underlying flaw associated with precautionary consumption as a "consumption fallacy" which suggests that individuals can protect themselves from risks by attempting to avoid exposure to toxic substances through consumerism. Precautionary consumption practices can never truly eliminate exposure to toxic substances which are ubiquitous. For instance, Scott and Lee found that less toxic alternative products may not be available and that toxic substances such as phthalates and flame retardants are very difficult to avoid despite precautionary attempts to do so by individuals (Kearns, 2011; Scott and Lee, n/d). These substances are "so pervasive in consumer products, are rarely clearly labelled, and alternative products can be difficult or expensive to obtain, consumers are unlikely to be able to avoid exposures" (Scott and Lee, n/d).

Precautionary consumption is also unable to account for everyday exposures to toxic substances through other mechanisms including the air, soil and water, and which environmental justice research demonstrates are often unequally distributed (Brown et al., 2012b; Hoover et al., 2012; Scott and Lee, n/d). The body burden experienced by Canadian citizens may be framed as "evidence of the failure of…risk assessments to prevent universal exposure to bioaccumulative chemicals" (MacKendrick, 2010: 128). The body burden is portrayed as a "blameless phenomenon" and as a "social problem by portraying it as a personal or individual-level concern, rather than societal or collective concern" (MacKendrick, 2010: 140; 2011: 43). The emphasis on behaviour at the level of

the individual does not encourage political and collective action that may be targeted at long-term and more broadly focused solutions including regulatory reform (Kearns, 2011; Scott and Lee, n/d). This discussion of where the burden of risk is presumed to lie demonstrates that the public health of the Canadian population can never be truly protected if responsibility remains at an individual level rather than recognizing the role of government and industry in health outcomes as a result of exposure to toxic substances.

Who is at Risk and Who are the Policies Designed to Protect?

Children as a Vulnerable Population

An important question which emerges when researching environmental health legislation and public health policy asks *who* is at risk. The only "vulnerable population" specifically designated by Health Canada[72] are children. This is reflected in the *National Strategic Framework on Children's Environmental Health* (Health Canada, 2010c). Vulnerable populations are considered to be at-risk to environmental exposures due to "physical differences, behaviours, location and/or control over their environment" (Health Canada, 2011e). The rationale behind the focus on children as a vulnerable population of concern is framed as a result of environmental hazards disproportionately impacting children. Age-specific windows of susceptibility which impact infants and children include pre-conception, the embryonic, fetal and neonatal period during which maternal ingestion, inhalation, and dermal contact play a role, as well as the first three years of life, preschool and primary school-age, and adolescence when inhalation,

[72] Health Canada (2011e) does acknowledge Aboriginal peoples and senior citizens as vulnerable populations, but children are the only population specifically addressed through a strategic framework.

ingestion and dermal contact occurs through the child's body (Health Canada, 2007f). Cooper et al. (2011: 130) note that cancer latency periods can span 20 to 40 years and that industrial activities result in multiple exposures to known or suspected carcinogens and endocrine disrupting chemicals. Windows of susceptibility during the prenatal exposures and early stages of development can impact cancer development and other developmental and reproductive health issues later in life.

> …[C]hildren are extremely sensitive to exogenous sex steroids and endocrine disruptors with no apparent lower threshold below which hormonal effects in children and potentially severe effects in adult life, are not seen. Thus,…[it is] caution[ed] that unnecessary exposure of fetuses and children to such substances, even at very low levels, should be avoided (Cooper et al., 2011: 130).

However, focusing solely on early periods of susceptibility does not account for four critical windows of susceptibility women experience later in life including i) before menstruation, ii) menstruation to first full pregnancy, iii) first full pregnancy to menopause, and iv) after menopause (Brophy et al., 2012; Schwarzman and Janssen, 2010).[73]

The scope of the *National Strategic Framework on Children's Environmental Health* includes chemical, biological and physical hazards related to children's exposure through air, water, soil, dust, food, consumer products, and any other features of the physical environment through pre-conception, prenatal and childhood exposures. The framework focuses on the environment as a determinant of health while also recognizing that other

[73] The language around critical windows of vulnerability is currently undergoing a shift. I observed this when attending the Environmental Health 2013 Conference: Science and Policy to Protect Future Generations in Boston, Massachusetts in March 2013. Key note speakers and presenters at this conference have shifted from describing the key periods of development in human bodies as "vulnerable" and are now using "windows of susceptibility."

determinants of health such as genetics, socioeconomic status and culture may influence the susceptibility of children to environmental exposures and subsequent health outcomes (Health Canada, 2010d: 9). Health Canada (2010d: 7) points to specific concerns around children's environmental health including:

- children's physiology and critical windows of vulnerability during developmental stages which may affect the absorption, metabolism and elimination of toxic substances;
- the development of the immune system which may be suppressed by exposure to persistent toxic substances;
- early exposure pathways including trans-placental transfer and consumption of breast milk;
- increased exposures related to size and weight of children compared to adults, and as a result of childhood behaviours such as close-to-ground exposures;
- children's lack of awareness and control over their own environmental risks including second-hand smoke exposure, parental occupational exposures, radiation, and microbiological hazards; and
- lack of knowledge about how to reduce environmental risks for children by parents, caregivers and health professionals.

As the primary source of authority for the assessment and management of risk associated with toxic substances, CEPA 1999 is identified as the foundation for policy direction related to toxic substance exposure (Health Canada, 2008c). Environment Canada and Health Canada (2004: 11) promote human health risk assessment and management as including research related to the exposure of the most affected population groups to toxic substances. While CEPA 1999 does not include specific reference to children's health, it is a "vital component of those activities related to the identification and assessment of existing substances that may pose a risk to the health and well-being of

children and Canadians of all ages" (Health Canada, 2008a: 6). The specific goals of the *National Strategic Framework on Children's Environmental Health* include risk assessment in order to increase the understanding of the existing and emerging environmental health impacts on children associated with environmental contaminants; risk management in order to prevent and reduce exposure of children to environmental hazards; as well as increasing communication and capacity building related to environmental health issues and children in Canada (Health Canada, 2010d: 13-4).

Consistent with the inclusion of socioeconomic status in CEPA 1999, the strategic framework provides a compelling argument which proposes the protection of children's environmental health as a cost-saving measure for adulthood. In accounting for health promotion, prevention and protection, Health Canada (2010d: 10) argues that "it is easier and less expensive to prevent or minimize environmental exposures which may lead to adverse outcomes, rather than to identify treatment strategies after children have been exposed or adversely affected." While the focus on children's health is predicted to reduce health care costs in adulthood, it does not consider important windows of susceptibility later in life that can also impact health outcomes.

The Regulation of Bisphenol A (BPA)

A recent example of a national effort focused on protecting the health of infants and children is the removal of the endocrine disruptor BPA from baby bottles. BPA is a high-production-volume industrial chemical that is widely used in the production of polycarbonate plastics including food and drink packaging and in epoxy resin linings of food and drink containers, as well as other applications such as additives in polyvinyl

chloride plastics, medical devices, automotive parts, electronics devices, compact discs, cell phones, sporting equipment, glasses, and receipts (Breast Cancer UK, 2013; Food and Agriculture Organization of the United Nations and World Health Organization, 2010; Health Canada, 2012b). The production of BPA has increased by 500% over the past thirty years reaching more than three billion kilograms per year. It is estimated to be worth approximately $500,000 per hour to the global economy (Breast Cancer UK, 2013: 11). Exposure to BPA is ubiquitous and the chemical can be detected and measured in humans in blood, urine, amniotic fluid, follicular fluid, placental tissue, and umbilical cord blood (Soto and Sonnenschein, 2010; Vandenberg et al., 2007). Nudelman et al. (2009: 87) note that studies funded by the chemical industry contend that BPA is harmless, whereas non-industry research suggests that it is a powerful hormone disrupter with the potential to result in detrimental health outcomes. Research is raising concerns about the health implications of broader exposures to BPA, particularly the estrogenic and endocrine disrupting properties as a result of low-dose, cumulative exposures (CAPE et al., 2010). Specific health concerns associated with BPA include the development of breast, prostate and testicular cancers, reproductive and developmental disorders, fertility disorders, neurodevelopmental and behavioural impacts including attention deficit hyperactivity disorder and impaired learning, and obesity (Breast Cancer Fund, 2013a; CAPE et al., 2010; Diamanti-Kandarakis et al., 2009; Ikezuki et al., 2002; Vandenberg et al., 2009, 2012; vom Saal et al., 2012).

BPA was identified as a high priority during the categorization of the Domestic Substances List. Environment Canada and Health Canada conducted a screening

assessment in 2008 which concluded that BPA meets the criteria under paragraph 64(a) and (c) of CEPA 1999. It found that BPA is

> entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term effect on the environment or its biological diversity….[and] in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health (Environment Canada and Health Canada, 2008b: 76).

In 2009, the Government of Canada announced its intention establish regulations to "prohibit the advertisement, sale and importation of polycarbonate plastic baby bottles that contain BPA, to reduce newborn and infant exposure to this substance" (Health Canada, 2009c). The federal government concluded that exposure levels for newborns and infants up to 18 months are below those that could cause health effects, but intends to further limit exposure due to uncertainty raised in other studies about the potential effects of low levels of BPA. The proposed regulations are part of the *Chemicals Management Plan*[74] (Health Canada, 2009c).

Health Canada hosted an international meeting about BPA in November 2010 with the Food and Agriculture Organization and the World Health Organization. The controversy associated with BPA within the scientific community was acknowledged and the meeting was organized

> in light of uncertainties about the possibility of adverse human health effects at low doses of BPA, especially on reproduction, the nervous system and behavioural development, and considering the relatively higher exposure of very young children compared with adults (Food and Agriculture Organization of the United Nations and World Health Organization, 2010: vi).

[74] BPA was part of Batch 2 of the Challenge under the *Chemicals Management Plan*, along with siloxane D5 (Government of Canada, 2012e; Health Canada, 2008d).

The meeting considered acute and repeated dose toxicity, carcinogenicity, reproductive and developmental toxicity, and hazard characterization associated with exposure to BPA. It was suggested that establishing a "safe" level of exposure for BPA was complicated by a lack of data and experimental animal studies that are suitable for risk assessment. Recommendations included generating new information and studies in order to better understand the risks to human health posed by exposure to BPA (Food and Agriculture Organization of the United Nations and World Health Organization, 2010: 30, xi).

BPA concentrations were measured in Canadian citizens for the first time at a national level as part of the Canadian Health Measures Survey in 2007-2009. Statistics Canada partnered with Health Canada and the Public Health Agency of Canada to collect health and wellness data, as well as biological specimens in the most comprehensive health measures survey conducted in Canada. The Canadian Health Measures Survey is the cornerstone of the national biomonitoring component of the *Chemicals Management Plan* and is meant to be used as a tool in evaluating the success of risk management measures (Health Canada, 2010a, 2010e, 2010f). Biomonitoring involves the "direct measurements of environmental chemicals, their metabolites or reaction products in people" and are most often measured in blood or urine, as well as other tissues and fluids such as hair, nails and breast milk (Haines, 2010; Health Canada, 2007g). While CEPA 1999 requires the Minister of Health to conduct research and studies on the role of toxic substances and health outcomes, there is no specific mandate for biomonitoring (Environment Canada and Health Canada, 2004: 27). There has been limited

biomonitoring data on Canadians. The Canadian Health Measures Survey was launched in 2007 to collect and provide data on levels of environmental chemicals that "represent the overall Canadian population" (Health Canada, 2007g). The body burden associated with exposure to toxic substances that is revealed through biomonitoring processes can be viewed as representing the personalization of pollution (Altman et al., 2008).

The first cycle of the Canadian Health Measures Survey was conducted in 2007-2009 and collected blood and urine samples from approximately 5600 Canadians aged six to seventy-nine in fifteen sites across the country (Health Canada, 2010e). The second cycle of the Canadian Health Measures Survey was conducted in 2009-2011 and included blood and urine samples from approximately 6400 Canadian citizens aged three to seventy-nine from eighteen sites[75,76] (Health Canada, 2013b). This was the first study to include biomonitoring data for children aged three to five years (Haines, 2013; Health Canada, 2013b). Chemical groups[77] that were measured in the first cycle include polybrominated flame retardants, polychlorinated biphenyls and organochlorines; metals

[75] The cycle one data collection sites included Moncton, NB; Quebec City, Montreal, Montérégie and South Mauricie, QC; Clarington, North York, Don Valley, St. Catherine's-Niagara, Kitchener-Waterloo, and Northumberland Country, ON; Edmonton and Red Deer, AB; and Vancouver, Williams Lake and Quesnel, BC (Health Canada, 2010e). The cycle two data collection sites included Saint John's, NL; Colchester and Pictou Counties, NS; Laval, South Montérégie, Gaspésie, and North Shore Montreal, QC; Central and East Ottawa, South Brantford, Southwest Toronto, East Toronto, Kingston, and Oakville, ON; Edmonton and Calgary, AB; Winnipeg, MB; and Richmond, Central and East Kootenay, and Coquitlam, BC (Health Canada, 2013b).

[76] Cycle three data collection is currently underway (January 2012-December 2013) and planning is in progress for cycle four (2014-2015) and cycle 5 (2016-2017) (Haines, 2013; Health Canada, 2013c).

[77] The chemicals tested in the biomonitoring aspect of the Canadian Health Measures Survey are selected for reasons including known or suspected health effects; level of public concern; evidence of exposure in the Canadian population; new or existing requirements in public health policy; ability to detect or measure the toxic substance in humans; similarity to substances measured in national and international programs for comparison; and the costs associated with performing the analysis. The second cycle of the survey includes fifty-five percent new chemicals and forty-five percent of the same chemicals as the first cycle (Health Canada, 2013c).

and trace elements, environmental phenols, pesticides, nicotine metabolites, perfluoroalkyl substances, phthalate metabolites, and chlorophenols were measured in both cycles one and two; and cycle two also tested triclocarban with the environmental phenols, as well as benzene metabolites and polycyclic aromatic hydrocarbons (Health Canada, 2013d).

BPA was measured in both cycles of the Canadian Health Measures Survey under the "environmental phenols" group of chemicals. Ninety-one percent of Canadians aged six to seventy-nine were found to have detectable concentrations of BPA in their urine in the first cycle of the Canadian Health Measures Survey from 2007-2009 (Bushnik et al., 2010; Statistics Canada, 2011). The results of the second cycle were released in April 2013 and determined that BPA was detected in ninety-five percent of Canadian citizens (Health Canada, 2013c).

The results of the Canadian Health Measures Survey data are promoted as being used to establish national baseline levels to track trends over time and as a reference point for international comparison (Haines, 2013; Health Canada, 2012b). Health Canada's special medical advisor, Dr. Robert Cushman stated that the "latest collection of national biomonitoring data will build on the [previous] information collected…for future monitoring and research. It will improve our understanding of human chemical exposure and help with the development of policies to protect the health of Canadians" (CBC, 2013c). Additional potential uses of the data also include providing information for priority-setting and action to protect Canadian's health from exposure to environmental chemicals; assessing the effectiveness of health and environmental risk management

strategies related to exposures and health risks associated with environmental chemicals; supporting future research on the potential links between exposure to environmental chemicals and specific health outcomes; and contributing to international monitoring programs such as the Stockholm Convention on Persistent Organic Pollutants (Health Canada, 2013c: 3).

Health Canada (2013d) acknowledges limitations associated with the use of biomonitoring as part of the Canadian Health Measures Survey. Biomonitoring measures the amount of a specific chemical in the body, but the measurement determines exposure from any or all routes including ingestion, inhalation or dermal contact, as well as any or all sources including air, water, soil, food, and consumer products. As a result, biomonitoring cannot determine the source or route of exposure and the chemical may be the result of a single source or multiple sources of exposure. Biomonitoring in and of itself cannot determine what health effects may occur as a result of the exposure. Relevant factors in considering whether a detrimental health outcome may occur include the amount of the chemical a person was exposed to, the duration and timing of exposure, and the toxicity of the chemical. It is also important to take into account levels of susceptibility in populations at-risk such as pregnant women, developing fetuses, children, the elderly, or people with compromised immune systems. Finally, the absence of a chemical in biomonitoring results does not mean that a person has not been exposed as existing technology may not be capable of detecting small amounts, and it is also possible that the chemical may have been eliminated, or metabolized, before the measurement occurs (Health Canada, 2010f: 4, 2013d).

The target population of the Canadian Health Measures Survey involves people living at home and residing in the ten provinces and three territories aged six to seventy-nine in cycle one and aged three to seventy-nine in cycle two. It is important to note that "people living on reserves or in other Aboriginal settlements in the provinces, residents of institutions, full-time members of the Canadian forces, persons living in certain remote areas, and persons living with a low population density were excluded" (Health Canada, 2010e: 3; Health Canada, 2013c: 4). While Health Canada (2010e) promotes the survey data as intended to be a nationally representative sample of the Canadian population, its limiters also exclude people who may be at higher risk for exposure to environmental chemicals. For instance, Sarnia and the Aamjiwnaang First Nation in southwestern Ontario are bordered by forty percent of Canada's chemical industry. The area known as "Chemical Valley" is one of the most polluted hotspots in the country (MacDonald and Rang, 2007). Residents of the Aamjiwnaang First Nation are exposed to chronic pollution including endocrine disrupting chemicals and as a result have experienced a significantly skewed sex ratio in the number of male live births compared to female (Mackenzie et al., 2005).[78] Residents have also experienced increased incidences of cancer, reproductive and developmental disorders (MacDonald and Rang, 2007).

[78] The disproportionate exposure to endocrine disrupting chemicals in this community and the resulting skewed sex ratio has resulted in an effect which is both gendered and gendering. The pollution itself may be "actively 'producing' sex, and to the extent that it is related, gender" (Scott, 2012a: 61, 2013). For additional discussion around exposure to toxic substances, the skewed sex ratio, and other health outcomes in Aamjiwnaang, refer to Dhillon and Young (2010), Jackson (2010), Luginaah et al. (2010), MacDonald and Rang (2007), Scott (2008, 2012a), and Wiebe (2010). For a sample of the media coverage associated with this case, refer to Colihan (2008) and Mittelstaedt (2008a, 2008b).

Environmental Defence has conducted biomonitoring research on Canadian citizens including participants from the Aamjiwnaang First Nation.[79] Biomonitoring tests were conducted on eleven individuals and five families across the country. Laboratory results detected forty-six of the sixty-eight toxic substances tested for in the body burdens of participants including five polybrominated diphenyl ethers (PBDEs), thirteen polychlorinated biphenyls (PCBs), five perfluorinated chemicals (PFCs), nine organochlorine pesticides, four organophosphate insecticide metabolites, five polycyclic aromatic hydrocarbons (PAHs), and five heavy metals. The results of the study detected thirty-eight carcinogens, twenty-three hormone disruptors, twelve respiratory toxins, thirty-eight reproductive and developmental toxins, and nineteen neurotoxins in participants (Environmental Defence, 2006: 1). However, the body burden of toxic substances in members of the family from Aamjiwnaang was among the highest in the study. The grandfather had the highest concentration of PFOS, PCBs and organochlorine pesticides, and the father had the highest total number of chemicals detected (Environmental Defence, 2006: 26). Basu et al. (2013) conducted a biomonitoring study of forty-three mother-child pairs living in Aamjiwnaang. This study found that mothers and their children are exposed to numerous environmental pollutants including metals, PAHs, PFCs, brominated flame retardants (BFRs), PCBs, and organochlorine pesticides (Basu et al., 2013). Residents of Aamjiwnaang are exposed to a "chemical cocktail" of toxic substances and the results of this study found that for some substances, the "trends

[79] For more detailed results of the biomonitoring research, refer to Environmental Defence (2005, 2006, 2007).

revealed higher exposures on the reserve than among the general population" (Dobson, 2013). The increased exposures included cadmium, mercury, DDT, organochlorine pesticides, hexachlorohexane, and some PFCs and PCBs (Basu et al., 2013). Excluding populations such as the Aamjiwnaang First Nation from the Canadian Health Measures Survey does not adequately account for potentially highly exposed and at-risk populations, nor the "manifestation of the pervasive, diffuse, and body-altering pollution that the residents report" (Scott, 2008: 297).

The federal Minister of Health, Leona Aglukkaq said in October 2010 that "[o]ur science indicated that Bisphenol A may be harmful to both human health and the environment and we were the first country to take bold action in the interest of Canadians" (Reuters, 2010). Minister Aglukkaq announced that BPA would be declared toxic making Canada the first jurisdiction to do so (Mittelstaedt, 2010). In light of BPA being added to Canada's list of toxic substances, Minister of the Environment Jim Prentice stated that "[w]e are continuing our leadership on this issue and Canadians can rest assured that we are working hard to monitor and manage bisphenol A" (CBC, 2010a). Yet despite BPA being declared toxic under CEPA 1999, this action does not require mandatory regulatory action and risk management strategies. The risk management associated with this assessment was limited to considering the highest potential for exposure and the potential vulnerability of newborns and infants by prohibiting the advertisement, sale and importation of polycarbonate plastic baby bottles that contain BPA (Health Canada, 2010b). The limited regulatory action that has occurred

focuses solely on infants and baby bottles but does not include any of the other numerous consumer products that still contain BPA.

A major route of exposure to BPA is through diet and the epoxy resins lining the insides of canned foods and beverages, as well as the metal lids of glass containers (Mittelstaedt, 2008c). In considering exposures to BPA beyond that in polycarbonate baby bottles, Health Canada's Food Directorate concluded that the current dietary exposure through food packaging is not expected to pose a health risk to the general population, including newborns and infants (Health Canada, 2008d; 2010e). However, it is important to note that fetuses and infants are exposed to BPA as breast milk passes on the exposures of nursing mothers and pregnant women (Kuruto-Niwa et al., 2007; Mendonca et al., 2012; Scott, 2012b; Ye et al., 2006).

In the most recent *Snapshot of Environmental Health in Canada*, Health Canada (2012b) acknowledges laboratory studies which suggest that low levels of exposure to BPA during windows of susceptibility in animals can affect neural development and behaviour. Health Canada does not reference any other studies which suggest wide-ranging health outcomes in humans as a result of exposure to BPA, though it does "support the need for additional research in these areas, and scientists continue to evaluate new scientific evidence as it emerges" (Health Canada, 2012b: 15). Health Canada (2012c) conducted additional surveys since the initial 2008 risk assessment around BPA in food packaging to measure concentrations in canned drink products, bottled water products, canned food products, soft drink and beer products, and total diet samples. Based on a weight-of-evidence approach, the Food Directorate again concluded

that dietary exposure to BPA through food packaging does not pose a health risk to the general population, including newborns and young children (CBC, 2013c; Health Canada, 2012c: 4; Mittelstaedt, 2010).

The Canadian Cancer Society (2013b) and the David Suzuki Foundation (2013) encourage Canadians to reduce their exposure to BPA. The Canadian Cancer Society suggests that it may be possible to reduce, but not eliminate exposure to BPA because it is so widely used. The organization recommends individuals take steps to avoid exposure to the substance if they are concerned about potential health effects (Canadian Cancer Society, 2013b). The David Suzuki Foundation (2013) also encourages individual citizens to reduce their exposure to BPA in light of the regulatory actions being limited to polycarbonate baby bottles. While the David Suzuki Foundation does call for a more precautionary regulatory framework to protect health and the environment, both organizations are promoting individualized strategies in the form of precautionary consumption. These campaigns place the focus at the level of individual citizens and do not acknowledge the limitations of a precautionary consumption approach.[80]

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[80] The precautionary consumption practices recommended by the Canadian Cancer Society (2013c) include avoiding children's toys, bottles and dishes made with polycarbonate plastic; using food and drink containers made of stainless steel, glass or non-polycarbonate plastic; choosing fresh or frozen foods not stored in cans; and talking to dentists about the materials being used and the options available if having dental work. The David Suzuki Foundation (2013) recommends using glass, stainless steel or porcelain containers instead of plastic dishes, containers and kitchen appliances; choosing plastics #2, #4 and #5 instead of #3 and #7 which often contain bisphenol A; using parchment paper, glass jars, beeswax cotton wraps or recycled aluminum foil instead of plastic wrap; not using plastic containers in the freezer, microwave or dishwasher as BPA and phthalates leach at a higher rate in hot or cold temperatures; using glass or stainless steel kettles instead of plastic kettles; avoiding canned food and drinks; breastfeeding or using powdered baby formula as more BPA leaches into liquid than powdered formulas; avoiding thermal paper receipts which contain BPA; using wood and cloth toys for children instead of plastic toys; and speaking to dentists about options as dental sealants and composites can contain BPA.

Critique of the Limited Scope of Bisphenol A regulation

Despite the regulatory action around BPA and baby bottles, the scope of action remains limited and does not go far enough. Consistent with the dominant epidemiological paradigm, the onus of responsibility is still placed primarily on the public and at the level of the individual. For instance, Health Canada (2010d: 7) maintains that "[t]he environmental burden of disease, with its associated socioeconomic costs, can be reduced by both ensuring healthier environments and providing people with the information they need to protect themselves [and their children] from harmful exposures." As Theo Colborn argues in her letter to the President of the United States about endocrine disrupting chemicals, there is no safe level for many chemicals to which fetuses and children are exposed when they penetrate the body and the womb (Colborn, 2012; The Endocrine Disruption Exchange, 2013).[81] A newly published report in May 2013 by the Royal College of Obstetricians and Gynaecologists is designed to provide guidelines informing women who are pregnant or breastfeeding about the "sources and routes of chemical exposure in order for them to take positive action in regard to minimizing harm to their unborn child" (Royal College of Obstetricians and Gynaecologists, 2013a). The report recommends a "safety first approach" which assumes there is risk in exposure to toxic substances but places the onus of responsibility on the mother to reduce consumption of foods in cans and plastic containers, minimize the use of personal care products and cosmetics, avoid paint fumes and pesticides, and reduce the

[81] Dr. Theo Colborn is one of the authors of the highly influential *Our Stolen Future* which was published in 1996 and explored the environmental and health effects associated with hormone disrupters and "hormone mimics" (Colborn, Dumanoski and Myers, 1996).

purchase of household furniture, fabrics, non-stick frying pans and cars while pregnant or nursing (Khatter, 2013; Royal College of Obstetricians and Gynaecologists, 2013a, 2013b).[82]

Placing responsibility for protecting children's health at the level of the individual creates a burden on the parents and particularly on mothers. "[M]others, more than other actors, are considered primarily responsible for controlling children's exposure to chemicals, and this responsibility represents a new way mothers are held accountable for their children's well-being" (MacKendrick, 2011: 42). The precautionary consumption behaviour with which women are encouraged to engage reinforces "individualized approaches to managing new forms of risk and simultaneously reinforce[s] mothering as a singular, but total responsibility for children's well-being" (MacKendrick, 2011: 42). This critique demonstrates that limiting the focus of health promotion to behavioural factors does not account for broader determinants of health including social, structural and environmental factors (Brown, 2007; Nash, 2006)

In restricting the scope of regulation to infants, the federal government is effectively dismissing exposures for other populations at risk. Thirteen of Canada's

[82] The American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women and the American Society for Reproductive Medicine Practice Committee published a Committee Opinion on exposure to toxic environmental agents in October 2013. The statement acknowledges reducing exposure to toxic substances at the level of the individual. However, it goes further than the position of the Royal College of Obstetricians and Gynaecologists by calling for primary prevention. It notes that individuals can "do little about exposure to toxic environmental agents, such as from air and water pollution, and exposure perpetuated by poverty. The incorporation of the authoritative voice of health care professionals in policy arenas is critical to translating emerging scientific findings into prevention-oriented action on a large scale" (American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women and the American Society for Reproductive Medicine Practice Committee, 2013: 3). At this time, the Society of Obstetricians and Gynaecologists of Canada has not published a position regarding exposure to toxic substances (Society of Obstetricians and Gynaecologists of Canada, 2013).

health and environmental organizations[83] released a joint statement calling for the federal government to eliminate key sources of exposure after Canada's designation of BPA as toxic under CEPA 1999. Providing context for the statement and demonstrating the real life implications for the Canadian population, Kathleen Cooper, Senior Researcher with the Canadian Environmental Law Association said that "[r]obust scientific evidence links low-dose BPA exposure with increased risks for breast, prostate and testicular cancers, altered reproductive function, altered metabolism of sugars and fats linked to obesity and diabetes, and adverse effects on the developing brain" (Canadian Partnership for Children's Health and the Environment (CPCHE), 2010). The joint statement makes recommendations which call for: an elimination of all food- and beverage-related uses of BPA; legislative reforms to improve the testing and regulation of toxic substances which are known endocrine disrupting chemicals; and clear labelling of products which contain endocrine disrupting chemicals (CPCHE, 2010).

In light of the recent findings that ninety-five percent of Canadians have BPA in their urine, Environmental Defence[84] launched a letter writing campaign asking

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[83] The thirteen signatories of *Focus on Bisphenol A - Statement of Health and Environmental Organizations on Endocrine Disrupting Chemicals* are the Canadian Association of Physicians for the Environment, Canadian Child Care Federation, Canadian Environmental Law Association, Canadian Partnership for Children's Health and Environment, Environmental Health Clinic – Women's College Hospital, Environmental Health Institute of Canada, Health Nexus, Learning Disabilities Association of Canada, Ontario College of Family Physicians, Ontario Public Health Association, Pollution Probe, South Riverdale Community Health Centre, The Lung Association – Ontario, and Toronto Public Health (CAPE et al., 2010).

[84] Environmental Defence first raised concerns about the potential health effects of BPA in 2007 as part of efforts to remove the substance from baby bottles (Environmental Defence, 2013b). Environmental Defense's most recent research and call to federal regulatory action surrounds a report released in June 2013 entitled *Pre-Polluted: A report on toxic substances in the umbilical cord blood of Canadian newborns.* The organization tested the umbilical cord blood of three newborn babies and the results determined that each of the children was born with between 55 and 121 toxic substances resulting in a toxic

Canadians to contact the Minister of the Environment and the Minister of Health. The text of the letter recognizes the success of the regulations around polycarbonate baby bottles, but calls on the federal government to take action around the continued exposure to children and adults to BPA.

> International organizations, expert panels and more than 150 peer-reviewed studies have associated bisphenol A with a variety of health problems – obesity, attention deficit hyperactivity disorder, breast cancer and a wide range of developmental problems – often at low levels of exposure (Environmental Defence, 2013d).

This campaign calls on the federal government to demonstrate leadership and develop and implement regulatory action that will be protective of both children and adults and eliminate BPA from all food and beverage containers, as well as other sources of exposure such as cash register receipts (Environmental Defence, 2013e).

Issues of Occupational Health Exposures, Accountability and Compensation

There has also been no move to address the occupational health exposures for workers who are exposed to BPA in their workplaces, such as the food canning industry. The occupational risks for breast cancer have traditionally been a neglected area of research (DeMatteo et al., 2012). A Canadian case-control study recently published by Brophy et al. (2012) found an increased risk of breast cancer among women working in

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body burden at birth. Of the 137 chemicals detected, 132 are reported to be carcinogenic, 110 are considered to be toxic to the brain and nervous system, and 133 result in developmental and reproductive problems. This study has a small sample size but is consistent with other umbilical cord studies conducted in the United States. Demonstrating the extreme persistence of some chemicals, 96 PCBs were found among the three samples. PCBs cross international boundaries, are bioaccumulative and carcinogenic and toxic to the immune, reproductive and neurological systems; they have also been banned in Canada since 1977. Organochlorine pesticides including DDT which are highly toxic and persistent were also found in the cord blood, despite being banned by the federal government in 1985 and banned from agricultural use by the Stockholm Convention in 2004. While the chemicals found in these children were in low doses, there is still significant cause for concern around windows of susceptibility, as well as additive, cumulative and synergistic effects of chemicals (Environmental Defense, 2013c). For more information, refer to CTV (2013), Environmental Defence, (2013b, 2013c), MacDonald, (2013), and Ubelacker, (2013).

occupations including farming, automotive, food canning, metal working, and bars, casinos and racetracks. Women who worked for ten years in occupations classified as "highly exposed" to cancer-causing substances and endocrine disrupting chemicals were found to have a higher risk for developing breast cancer. Specific findings included:

- *Farming:* A 36 percent increased breast cancer risk was found in the farming sector. Research has established that several pesticides act as mammary carcinogens and many are endocrine disrupting chemicals. Employment in farming and exposure to pesticides often begins earlier in women's lives than other occupations and may play a role in the development of breast cancer during subsequent windows of susceptibility.

- *Automotive:* A statistically significant more than two-fold increased breast cancer risk was found in the automotive plastics industry sector. The increase rose to an almost 5-fold excess among women who were pre-menopausal. Many plastics have been found to release estrogenic and carcinogenic chemicals and cumulative exposures to mixtures of these chemicals are a particularly significant concern.

- *Food Canning:* A statistically significant 2-fold breast cancer risk was found in the food canning sector. The increase rose to more than 5-fold among women who were pre-menopausal. Exposures to chemicals in the food canning industry may include pesticide residues and emissions from the polymer linings of cans including BPA.

- *Metalworking:* A statistically significant 73 percent increased breast cancer risk was found in the metalworking sector. Women working in tooling, foundries and metal parts manufacturing are exposed to a variety of potentially hazardous metals and chemicals. The cumulative exposures to mixtures of toxic substances are of concern.

- *Bars, Casinos and Racetracks:* A 2-fold increased breast cancer risk approaching statistical significance was found in bars, casinos and racetracks. The elevated risk of developing breast cancer may be linked to second-hand smoke exposure and night work which has been found to disrupt the endocrine system (Sweeney, 2012b).

This research has a number of important implications which are detailed by Brophy et al. (2013). This work challenges the paradigm promoted by Doll and Peto by demonstrating

the importance of occupational and environmental factors in the development of cancer. The predominant focus on lifestyle and behavioural factors in cancer research and public health policy has resulted in gaps including the prevention of exposure to toxic substances and the resulting detrimental health outcomes such as the development of breast cancer in environmental and occupational settings.

Seriously considering the role of endocrine disrupting chemicals in the development of breast cancer undermines the established orthodoxy in traditional toxicology where the "dose makes the poison." These chemicals can have effects at low doses. The majority of exposure standards for occupational, environmental and consumer health and safety are still based on the toxicology model which is insufficient in accounting for endocrine disrupting chemicals and low-dose cumulative exposures. Brophy et al. (2013) suggest that "[i]f there are no 'thresholds' for certain substances at which no effects are observed, no 'safe' limit can be established."

Finally, these research findings present clear challenges to the workers' compensation system (Brophy et al., 2013). Definitively assessing and managing occupational diseases is a complex and highly problematic process which is influenced by social, cultural and political issues, as well as scientific and medical knowledge and theories (Watterson, 1999). The difficulties in establishing a direct and causal link between a particular substance and a specific health outcome are complicated by a variety of factors. For example, lengthy latency periods are often required in order to establish a statistically significant correlation between an exposure to a toxic substance and an increased incidence of disease in a particular population. The contested nature of

environmental health outcomes may mean that it is not possible to establish a connection conclusively and to the satisfaction of the entire scientific community (Markowitz and Rosner, 2002: 6).

Health outcomes as a result of environmental and occupational exposures have traditionally been framed as contested and are surrounded by questions of uncertainty and accountability. The risks that arise as a result of new and continually evolving technologies are unique to the risk society and there are difficulties with compensation and accountability where the risks are not limitable, either spatially or temporally; may not be accountable according to the prevailing rules of causality, guilt and liability; and may not be compensable nor insurable (Beck, 1995: 2; 1996: 31). Issues of accountability are wrought with difficulties, "far from being caused by individuals who…[can] be held accountable, these risks…[are] caused by the system of high technology itself" (Richter et al., 2006: 7).

In examining examples of accountability and compensation, Richter et al. (2006: 6) note that the traditional compensation system is most often unable to adequately account for those affected by environmental health issues such as those impacted by the Chernobyl nuclear disaster. This is especially evident in cases where the illness is contested, both in its existence and in cases where there are issues around determining causality. For instance, health care workers in Nova Scotia claimed to be suffering from heavy metal poisoning as a result of exposure to toxic dust during renovations at the New Waterford Consolidated Hospital in 2001-2002. These claims have not been universally accepted and their illnesses and the events in this community constitute an environmental

health controversy with opposing viewpoints carried out in both public and private realms.[85] The health care workers at the New Waterford Consolidated Hospital encountered resistance in three specific ways including belief in the existence of the illness; conflict in a diagnosis; and conflict regarding an appropriate treatment (Sweeney, 2006a). Affected employees filed a workers' compensation claim which was supported by local MLA Frank Corbett who stated that "there is no excuse left for the [Workers' Compensation Board] to further delay the compensation owed to eligible workers who became sick while they were employed [at the New Waterford Consolidated Hospital]" (Corbett, 2004; Sweeney, 2006a). The Workers' Compensation Board rejected the claim from thirty-six employees reasoning that they could not find a link between the employees' illnesses and the Hospital (CBC, 2005). The approval of the Workers Compensation Board claim not only would have assisted workers financially, but would have formally legitimized their claims. An oral surgeon who was affected by this case filed a statement of claim at the Supreme Court against the Cape Breton District Health Authority contending that occupational health and safety rules were violated and resulted in hospital workers being exposed to excessive levels of toxic dust (Richer, 2004; Sweeney, 2006a). The lawsuit concluded in 2009 when the Supreme Court judge found

[85] Those affected in this case engaged in Brown's (1992: 267-9) stages of popular epidemiology which are based on his research surrounding communities affected by toxic waste "where lay persons gather and direct and marshall the knowledge and resources of experts in order to understand the epidemiology of disease, treat existing and prevent future disease, and remove the responsible contaminants." A case study of the events surrounding the environmental health controversy at the New Waterford Consolidated Hospital amends the stages of popular epidemiology to include biographical disruption in an examination of the experiences of health care workers who become ill with environment-related illnesses, when they encounter resistance from their peers and the health care system to which they belong (Sweeney, 2006a).

in favour of the Cape Breton District Health Authority. In a 113-page decision, Justice MacLellan found that

> [t]he plaintiff here has suffered a great deal. His life has been torn apart by his illness. He is a good man and a skilled dental surgeon. The court finds no joy in denying his claim. However, the legal system requires that a plaintiff prove his claim based on certain legal principles, including proof of causation (Camus, 2009).

The plaintiff has since filed an appeal which suggests that the Supreme Court failed to address the original claim that the Cape Breton District Health Authority breached its leasing contract by exposing employees to hazardous substances including heavy metals and toxic gases (CBC, 2010b). It also suggests that the Supreme Court erred by placing the burden of proof on the plaintiff to "prove hazardous materials were present in the hospital while the renovations were going on, when the Occupational Health and Safety Act requires the health authority to determine what hazardous materials were present before starting the renovations" (Hayes, 2010). The controversy around issues of accountability and compensation in this case involves the affected health care workers, the Hospital Administration, health care system, and the Workers' Compensation Board, and has yet to be resolved. This case demonstrates the intrinsic link between risk, environmental health controversies and contested illnesses, where the affected health care workers have continued to suffer physically, financially and emotionally.

In cases concerning occupational exposures and breast cancer, the existence of the disease itself is not contested but its causation and issues of accountability are continually surrounded by scrutiny and debate. Many of the endocrine disrupting chemicals and mammary carcinogens of concern in the development of breast cancer have come into

widespread use over the past thirty years and women in Canada are exposed to these toxic substances on a regular basis.

> Based on the mounting evidence, this widespread introduction of toxic chemicals into various work environments, and particularly new pesticides into agriculture and plastics into automotive manufacturing, will likely result in escalating numbers of claims for work place compensation for women who have developed breast cancer from these new technologies (Brophy et al., 2013).

To date there have been no workers' compensation claims upheld in Canada in cases of toxic exposures linked to breast cancer (Keith, 2013). Manitoba became the first jurisdiction in Canada to "enact a firefighter's disease presumption" when it added breast cancer to its list of compensable diseases for firefighters in 2011 (Government of Manitoba, 2010). Ten primary-site cancers were listed in the original legislation in 2002 including brain, bladder, kidney, lung, ureter, colorectal, esophageal, and testicular cancers, non-Hodgkin's lymphoma, and leukemia. The amendments proposed in 2010 apply to volunteer, part-time and full-time firefighters and included four additional cancers including multiple myeloma, primary site prostate, skin, and breast cancer (Government of Manitoba, 2010). The risk of a female firefighter developing breast cancer is three to five times higher than the general population as a result of exposure to more than 200 known carcinogens connected to breast cancer at every fire (CBC, 2010c; Kusch, 2010).[86] Thus far, other provinces and territories do not have this category for

[86] Janette Neves Rivera is a California firefighter who was diagnosed with breast cancer that is believed to be linked to her exposure to toxic substances as a result of her occupation (Fire Engineering, 2013). Rivera maximized her sick time and applied to San Francisco's catastrophic illness program which enables employees to donate their sick time to each other, but the city's Department of Public Health denied her claim, stating that her current condition was not considered to be "life threatening" (KTVU, 2012). Rivera recently filed a petition along with the Center for Environmental Health to the Chairman of the Consumer Product Safety Commission which is currently considering a federal flammability standard that would restrict the use of flame retardants in furniture and other products across the United States. The federal standard being considered will undermine a new California standard (TB 117-2013) which would require

firefighters or any other specific occupational group. A workers' compensation claim was initially granted to health care workers who experienced a breast cancer cluster in a hospital laboratory in British Columbia and claimed they were exposed to carcinogens. However, this claim was appealed by the employer who argued that there was insufficient evidence to demonstrate that the claimants' cases of breast cancer were caused by occupational factors. The claim was overturned by the provincial Supreme Court, though the case has been left open if new evidence becomes available in the future (BC Justice, 2013; Keith, 2013).

Meek and Armstrong (2007: 593) note that the definition of environment in CEPA 1999 is broad enough to encompass the occupational environment. However, the federal regulatory regime that is designed to protect human health including CEPA 1999 and the *Chemicals Management Plan* does not encompass occupational health which instead falls under provincial and territorial legislation in the form of Occupational Health and Safety Acts. The research conducted by Brophy et al. (2012) linking increased incidence rates of breast cancer to occupational exposures of toxic substances raises important questions about the adequacy of existing chemical testing protocols in workplaces under provincial occupational health and safety standards. The Association of Workers' Compensation Boards of Canada recently listed breast cancer as an emerging issue citing the Brophy et al. (2012) study and its findings that the risk of breast cancer is

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companies to make products with improved fire safety and without hazardous flame retardants. The petition calls on the Consumer Product Safety Commission to join California and research other fire safety approaches that would provide greater fire safety without the use of toxic chemicals that are hazardous to human health and the environment (Fire Engineering, 2013; Rivera, Janette Neves and the Center for Environmental Health, 2013; State of California, 2013b, 2013c).

higher in workers in automotive plastic manufacturing and food canning industries (Association of Workers' Compensation Boards of Canada, 2013; Keith, 2013). The growing body of epidemiological and laboratory research has the potential to impact the workers' compensation system and frame breast cancer as a compensable occupational disease (Brophy et al., 2013).

Neither the federal regulatory or provincial occupational health and safety regimes adequately protect women and prevent detrimental health outcomes including the development of breast cancer as a result of exposure to toxic substances. The Government of Canada contends that "[n]ational consistency secures the same level of environmental and human health protection for all Canadians" (Environment Canada and Health Canada, 2006: 18). However, the federal regulatory regime does not account for women as a susceptible population who are at risk as a result of everyday exposures to toxic substances. Health Canada's focus on infants and children as the only vulnerable population at risk from exposure to environmental contaminants does not account for either a lifecourse approach to health or windows of susceptibility that occur through a woman's life. The risk assessment and management frameworks do not adequately account for the effects of low-dose, cumulative and synergistic effects of exposure to complex mixtures of toxic substances. These frameworks do not currently account for the emerging understandings of the long-term health effects of endocrine disrupting chemicals, despite the "abundant scientific evidence of the harmful effects by EDCs [which] has accumulated to support a swift change in public health and environmental policies aimed at protecting the public in general, and, in particular, the developing fetus

and women of reproductive age" (Soto and Sonnenschein, 2010: 7).

Women's Health and Cancer Organizations' Response

The majority of health and environmental organizations operate within the framework and discourse established by the government and public health sector. The more mainstream cancer and women's health organizations have traditionally and primarily promoted the lifestyle and behavioural risk factors in the development of cancer with the individualization of risk and responsibilization paradigm. It is also suggested that this approach has been promoted by mainstream organizations because of partnerships with and funding from pharmaceutical organizations. Batt (2010: 69) demonstrates that "[s]urveys based on annual reports, websites, and interviews confirm the prevalence of pharmaceutical company donors as well as concerns about disclosure, not only in Canada but in the U.S., Europe, Australia, and New Zealand." Health-related organizations receive tens to hundreds of thousands of dollars annually from pharmaceutical companies for conferences, publications, websites, and advocacy training (Batt, 2010: 69).[87] However, there is emerging evidence that points to a shift in mainstream women's health and cancer organizations which acknowledges the environment as a determinant of health and exposure to toxic substances as hazardous to women's health.

[87] Indeed, the Canadian Breast Cancer Foundation lists GlaxoSmithKline and the Roche Group as "Pink Ribbon Partners" of its Ontario branch with donations greater than $50,000 (CBCF, 2013). The Canadian Breast Cancer Network lists GlaxoSmithKline, the Roche Group, Novartis, and Amgen as sponsors (CBCN, 2013a).

The Canadian Cancer Society is perhaps the most well-known cancer organization in the country with a Nationwide Strategic Plan for 2010-2015 which aims to "deter, defeat and defy cancers" through the reduction of cancer incidence and cancer mortality rates for Canadians (Canadian Cancer Society, 2013d). The Canadian Cancer Society has traditionally focused its public education campaigns on lifestyle and behavioural risk factors. The Canadian Cancer Society published the "Seven Steps to Health" which were widely utilized in public educational campaigns of various cancer organizations including the Canadian Breast Cancer Foundation, Halifax Breast Cancer Screening Clinic, Cancer Care Nova Scotia, and the Canadian Cancer Society itself. For instance, the Seven Steps to Health appear prominently in public education pamphlets including "Breast Self-Examination: How to Check Your Breasts" (1997); "Facts on Breast Cancer" (2000); "Cancer Facts for Women" (2000); "Breast Health: What You Can Do" (2002); and "Breast Self-Examination: What You Can Do" (2002) (Sweeney, 2006b). The Seven Steps to Health are framed around the knowledge that "some cancers can be prevented," and the suggestion that members of the general public should take responsibility for their health and use these steps to reduce their risk of developing breast cancer:

> 1) Be a non-smoker and avoid second hand-smoke;
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> 2) Eat 5 to 10 servings of vegetables and fruit a day. Choose high fibre, lower fat foods. If you drink alcohol, limit your intake to 1 or 2 drinks a day;
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> 3) Be physically active on a regular basis: this will also help you maintain a healthy body weight;
>
> 4) Protect yourself and your family from the sun. Reduce sun exposure between 11a.m. and 4 p.m. Check your skin regularly and report any changes to your doctor;

> 5) Follow cancer screening guidelines. For women, discuss mammography, Pap tests and breast exams with a health professional. Both men and women should also discuss screening for colon and rectal cancers.
>
> 6) Visit your doctor or dentist if you notice a change in your normal state of health; and
>
> 7) Follow health and safety instructions both at home and work when using, storing and disposing of hazardous materials (Sweeney, 2006b: 82).

The influence of the Doll and Peto paradigm, the individualization of risk, and promotion of responsibilization is evident in the emphasis on lifestyle and behavioural factors in the public education literature. Breast cancer is primarily framed as a problem of behavioural practices as women are encouraged to live a healthy lifestyle which includes not smoking, limiting alcohol consumption, eating healthy, exercising, and engaging in cancer screening practices (Steingraber, 2000).

Step seven of the Canadian Cancer Society's approach is the only one to acknowledge an environmental role in the development of cancer. The language used in step seven has evolved over time. A public education pamphlet published in 1997 suggests that "[a]t home and work, follow health and safety instructions when using hazardous materials." There was a minor revision in 2000 to read "[f]ollow health and safety instructions at home and at work when using, storing and disposing of hazardous materials" (Sweeney, 2006b: 84). An article published in *This Magazine* questioned the Chief Executive Officer of the Canadian Cancer Society about why this step offers advice on handling hazardous materials and does not urge citizens to avoid known carcinogens altogether. The question in itself reflects the lifestyle and behavioural component in framing the responsibility for avoiding exposure to carcinogens as a problem to be

addressed at the individual level. The Chief Executive Officer replied that "[i]t's sort of wussy," and that all public education materials using the Seven Steps to Health should be updated within the year (Murphy, 2002: 32). The interview was published in March 2002, and two of the pamphlets that were revised and printed in July 2002 contained a re-wording of step seven: "[f]ollow health and safety instructions both at home and at work when using, storing and disposing of hazardous materials." This was nearly identical to the text from seven years prior and the Canadian Cancer Society's website reflected the same information in 2003 (Sweeney, 2006b: 84-85). Chernomas and Donner (2004) note that in 2004 the Canadian Cancer Society endorsed the precautionary principle as an effective tool for preventing cancer. However, the authors critique the organization's approach as "inconsistent in acknowledging the importance of primary prevention" in relation to the precautionary principle as the public education literature on breast cancer does not contain reference to possible environmental risk factors (Chernomas and Donner, 2004: 17).

More recently, the Canadian Cancer Society (2008, 2013b) suggests that the current scientific evidence has not been able to confirm or eliminate a causal link to environmental contaminants in the development of cancer and list environmental exposures under unknown risk factors, but does allow that "people who are continually exposed to cancer-causing substances at high levels or over long periods of time may have a higher risk of developing cancers" (Canadian Cancer Society, 2008: 3). *The Environment, Cancer and You* is an underwhelming publication as it still places a significant amount of responsibility on the individual and only fully confirms asbestos

and radon as cancer-causing, while other substances such as flame retardants, phthalates and electromagnetic fields are classified as a concern (Canadian Cancer Society, 2008). The organization maintains that more research is needed in order to clearly understand how toxic or environmental substances may be linked to cancer, but suggest that substances that are known to cause cancer should be replaced with safer alternatives and if that is not possible, then exposure to the substance should be reduced as much as possible (Canadian Cancer Society, 2013e). The Canadian Cancer Society relies on classification material from international sources including the International Agency for Research on Cancer, the United States National Toxicology Program; and the United States Environmental Protection Agency (Canadian Cancer Society, 2013e).[88]

The Canadian Cancer Society announced in November 2012 that it will be funding three new prevention-focused projects. Director of Research, Dr. Mary Argent-Katwala stated that

> [t]hese new prevention grants are a unique opportunity for the Society to use the findings to inform our advocacy and policy agenda. For example, the results…could be used for educational campaigns, advocacy activities to urge governments to enact prevention regulations, and to set priorities for our prevention activities (Canadian Cancer Society, 2012b).

[88] The International Agency for Research on Cancer (IARC) is an intergovernmental agency and part of the World Health Organization. It uses four classes in its conclusions around carcinogenicity including Group 1 (carcinogenic to humans); Group 2A (probably carcinogenic to humans); Group 2B (possibly carcinogenic to humans); Group 3 (not classifiable as to its carcinogenicity to humans); and Group 4 (probably not carcinogenic to humans) (Canadian Cancer Society, 2013f; IARC, 2013). The National Toxicology Program (NTP) is part of the United States Department of Health and Human Services. The defining criteria it uses for determining carcinogenicity includes known to be a human carcinogen; and reasonably anticipated to be a human carcinogen (Canadian Cancer Society, 2013f; NTP, 2011). The United States Environmental Protection Agency (USEPA) classifies toxic substances in five categories including carcinogenic to humans; likely to be carcinogenic to humans; suggestive of carcinogenicity but not sufficient to assess human carcinogenic potential; data are inadequate for an assessment of human carcinogenic potential; and not likely to be carcinogenic to humans (Canadian Cancer Society, 2013f; USEPA, 1999; 2012).

The first study will quantify the number of new cancer cases and deaths in Canada that can be attributed to workplace factors and determine its economic impact. This multi-disciplinary research will receive $1 million over four years to examine the human and economic impact of workplace exposure to forty-four known or suspected carcinogens based on the guidelines from the International Agency for Research on Cancer, and toxic substances links to twenty-seven types of cancer. The substances that will be considered include industrial chemicals benzene, formaldehyde and 1.3-butadiene; metals such as chromium, nickel and arsenic; and other factors including exposure to sunlight, asbestos, paint, diesel fumes, and shift work. The research will also estimate direct costs such as medical care, indirect costs such as lost work time, and quality of life costs related to occupationally-related cancers; estimate the human and economic burden of occupational cancer by province, industry, sector, and gender (sex); and utilize the estimates in order to determine potential benefits of cancer prevention such as toxics use reduction (Canadian Cancer Society, 2012b).

The second prevention-focused research project funded by the Canadian Cancer Society involves $928,000 over four years to study how public health agencies can collaborate to reduce cancer rates in Northern British Columbia which has higher rates of smoking, obesity and cancer-related deaths than the rest of the province. The third project involves $970,000 over four years to create a smoking cessation intervention program for youth in Quebec involving general practitioners and nurses, follow-up counselling, and peer support in order to target youth who smoke and prevent cancer incidence related to smoking-related illnesses such as lung cancer (Canadian Cancer Society, 2012b). While

the first study has a broader focus on occupational exposure to toxic substances, the two remaining studies continue to focus primarily on lifestyle and behavioural factors in their focus on smoking cessation.

The Canadian Breast Cancer Foundation (2012d) acknowledges the potential of toxic substances in increasing the risk of developing breast cancer and points to key areas of concern such as the health effects of endocrine disrupting chemicals, low-level cumulative exposures and mixtures of toxic substances, and critical periods of development and susceptibility including infancy, puberty and pregnancy. Research on indoor air and household exposures to toxic substances suggests that low-level exposures may result in triggers for the development of breast cancer including disruptions in the hormonal system, early puberty and altered mammary gland development. The Canadian Breast Cancer Foundation endorses the use of the precautionary principle as a way to "apply evolving breast cancer prevention evidence in our daily lives. By following the precautionary principle in your life, when scientific evidence is inconclusive you put your health first and err on the side of caution" (CBCF, 2012d).

The Canadian Breast Cancer Foundation encourages people to limit their daily exposure to toxic substances at home and in the workplace (CBCF, 2012d, 2012e). Specifically, the organization suggests that people can reduce their exposure to toxic substances in food, plastics, personal care and cleaning products, and products for children in order to reduce chemical exposures in the home. In order to reduce exposures through food, people are encouraged to wash fruits and vegetables to remove traces of pesticides; buy local, pesticide-free or organic food; and minimize exposure to BPA by

avoiding canned foods and using glass containers instead of plastic to prevent leaching. Avoiding plastics that contain polyvinyl chloride, polystyrene and polycarbonate is also suggested. In order to reduce or limit exposure, the organization recommends using fewer cosmetics and personal care products or ones with less ingredients. They also encourage consumers to read the label for ingredients and avoid products that contain fragrance or parfum or toxic substances such as phthalates, parabens, alkylphenols, and placental extracts. To reduce exposures through the use of household products, consumers are encouraged to use non-toxic household products, avoid products containing bleach, and use pesticide-free and non-toxic products on yards and gardens and in particular avoid products which contain 2,4-D or malathion. Finally, the organization suggests limiting children's exposure to toxic substances by using glass food containers instead of plastic and buying toys that do not contain phthalates (CBCF, 2012f). These recommendations all fall under practices of precautionary consumption and seem to target a highly educated and affluent demographic with recommendations to purchase organic food and read labels for cosmetics, personal care products, and cleaning products.

The Canadian Breast Cancer Foundation also acknowledges concerns around workplace exposures to toxic substances and the risk of developing breast cancer. Occupations with increased breast cancer risks include agriculture and manufacturing of textiles, paper, microelectronics, metals, food canning, and automotive plastics as a result of exposure to toxic chemicals; health care where workers are exposed to ionizing radiation; work with heavy traffic such as border service agents where there are high levels of exposure to diesel exhaust; and long-term night-shift workers whose exposure to

artificial light can reduce melatonin levels which can play a role in suppressing the growth of breast tumours (CBCF, 2012f). The Canadian Breast Cancer Foundation cites the Brophy et al. (2012) work as a "landmark study" in occupational breast cancer research which found that women who were exposed to carcinogens and endocrine disrupting chemicals over a ten year period had a forty-two percent increased average risk and women working in the food canning and automotive plastics had a five times higher risk of developing breast cancer before menopause. The precautionary steps recommended for workers exposed to toxic substances include knowing workplace health and safety rights; learning about how to best protect yourself from risks such as how to properly use protective equipment or clothing; as well as raising concerns with supervisors, Occupational Health and Safety Committees, the local ministry of labour office, or the Canadian Centre for Occupational Health and Safety. In addition to the precautionary measures recommended for individual workers, the Canadian Breast Cancer Foundation argues that there is a need for regulators and employers to take precautionary action in order to protect workers and reduce the risk of negative health outcomes (CBCF, 2012g).

The Breast Cancer Society of Canada (2013c) has also moved towards acknowledging the environment as playing a role in "healthy living" and in the development of breast cancer. The Breast Cancer Society of Canada acknowledges both environmental and occupational exposures as issues of concern, although their recommendations for "living green" fall into precautionary consumption behaviours and are vague at best. The organization suggests that the cost of making extreme

modifications to our homes or changing careers in order to live a completely green lifestyle without exposures to toxic substances is not feasible, while still placing responsibility on the individual by recommending that "there are many things that can be done in your daily life and if you integrate these changes gradually in the areas where you are most vulnerable, you'll be living green before you know it" (BCSC, 2013c). The Breast Cancer Society of Canada acknowledges that Canadian citizens come into contact with chemicals that have been linked to breast cancer including polychlorinated biphenyls, dioxin, pesticides, phthalates, BPA, polyvinyl chloride, fire retardants, and ingredients in cosmetics. However, there is no background or context provided as part of this discussion, rather they provide a link to the Environmental Working Group's Cosmetics Database where people can "verify how safe your brands are," and a link to an alphabetized list of carcinogens compiled by Health Canada. Farm workers are the only occupation included as increasing the risk of developing breast cancer and the recommendations are to follow warnings and handling procedures in material safety data sheets for chemicals and to work with employers to ensure the workplace has good air quality and that chemicals are properly handled (BCSC, 2013c). This information lacks any level of detail and specificity while placing the onus of responsibility for behavioral change on the individual rather than any acknowledgement or call for regulatory reform around environmental and occupational exposures.

The Canadian Breast Cancer Network finds that some organizations categorize environmental risk factors with a very broad understanding of environment to encompass all determinants that are not genetic or hereditary including lifestyle and behavioral

factors such as smoking. This framework is grounded in the responsibilization paradigm and the individualization of risk which are promoted in public health policy. At the other end of the spectrum are organizations who attempt to shift the discourse and approach to health outcomes by defining environmental risk factors as limited primarily to industrial chemical pollutants. The Canadian Breast Cancer Network itself recognizes the wide variation between the definitions as well as the political implications involved in categorical decisions. As such, the organization chooses to work with a middle ground by recognizing the impact of the environment on health outcomes and by providing a general selection of resources in its educational materials "relating to environmental concerns with the hope that a cleaner, healthier, conscientious environment is conducive to all facets of health, not least of all breast health" (CBCN, 2013b). The Canadian Breast Cancer Network acknowledges that toxic chemicals and radiation play a role in the risk a woman has of developing breast cancer and suggests that our exposure to chemicals and radiation is something that can be controlled through personal, corporate and political action. They do not provide any detailed information or suggestions as to what the corporate or political action may involve, but suggest precautionary consumption practices in "get[ting] to know the chemicals that have been linked to breast cancer and tak[ing] action to reduce your risk" (CBCN, 2013b).

While the Canadian Breast Cancer Network does acknowledge environmental risks, they categorize occupational hazards as a "lifestyle risk." The occupational information provided includes automotive combustion with an article published in 2000 specific to male breast cancer incidence as a result of exposure to gasoline and vehicular

combustion product; and chemical exposure in female firefighters providing links to an American study from Cornell University and the International Agency for Research on Cancer Monographs with "recent evaluations of occupational carcinogens" which provides a broken link to the Canadian Cancer Society website (CBCN, 2010). There is no substantive discussion for concerns around automotive combustion or chemical exposure, though there is slightly more information provided about hazards associated with working night shifts and the role of melatonin in tumour development and a higher incidence of breast cancer among women who work night shifts (CBCN, 2010). Recognizing the risks associated with occupational exposures and developing breast cancer is of great importance. However, categorizing occupational exposures as a lifestyle risk is problematic in a number of ways. First, framing occupational exposures and health outcomes in this manner clearly implies that individuals can control these exposures and subsequent health outcomes through behavioural practices when in the majority of cases this is beyond the capacity of the individual. It is also important to consider the social determinants of health such as socioeconomic status when discussing occupational exposures. There is often apprehension about being labelled a "troublemaker" for raising concerns around occupational health issues. In addition to this stigma, workers face the very real threat and fear of workplace closure and subsequent job loss in cases with environmental, contested or occupational exposures and health outcomes. This is similar to threats around mine closures when respiratory illnesses were linked to miners who were exposed to contaminated air (Rosner and Markowitz, 1987). The threat of job loss is particularly relevant in rural communities where there are few

major employers (Sweeney, 2006a). Brophy et al. (2012) also point to a class and gender bias around the issue of occupational exposures and that breast cancer is a neglected issue. Finally, framing occupational exposures as a lifestyle risk effectively dismisses the role of the employer and the government in regulating exposures and protecting workers' health.

Despite the Canadian Cancer Society, Canadian Breast Cancer Foundation, Canadian Breast Cancer Society, and the Canadian Breast Cancer Network making some move towards acknowledging the environment and toxic substances as influencing women's health and cancer outcomes, their primary focus remains on lifestyle and behavioural factors which places the onus of responsibility for preventing breast cancer on the individual and which is consistent with the mainstream organizations in the United States. Separate from these mainstream organizations, there are those which are more progressive in taking action through educational campaigns and lobbying government, and which can be classified as part of the culture of cancer prevention and environmental risk. The Breast Cancer Fund and Breast Cancer Action are two organizations that emerged as part of this culture of action in the San Francisco Bay Area, and Breast Cancer Action Montreal represents a similar organization in Canada as part of the broader environmental breast cancer movement. All of these organizations have similar mandates in their commitment to advancing and protecting women's health through public education and advocacy campaigns and a refusal to accept funding from organizations and corporations that conflict with their mandate such as pharmaceutical

companies, chemical manufacturers, oil companies, tobacco companies, and health treatment facilities.

The Breast Cancer Fund works towards translating scientific evidence which links environmental exposures to the development of breast cancer into public education and advocacy campaigns around reducing breast cancer risk and protecting women's health (Breast Cancer Fund, 2013b). The organization points to the emerging body of scientific evidence which indicates that exposures to toxic substances such as chemicals and radiation are contributing to the increased breast cancer incidence rates in industrialized countries. The Breast Cancer Fund's *State of the Evidence: The Connection Between Breast Cancer and the Environment* is now in its sixth edition. This widely cited report examines the links between exposure to environmental chemicals and radiation and the development of breast cancer within a broad context that recognizes the social determinants of health and susceptible populations who may be at higher risk of developing the disease. The organization argues for the importance of the timing and duration of exposures to toxic substances, low-dose exposures at environmentally significant levels, patterns and mixtures of exposures, and the complexity of interactions between environmental and other risk factors for breast cancer (Gray et al., 2009; Gray, 2010). The organization points to the role of windows of susceptibility when "mammary cells are more susceptible to the carcinogenic effects of hormones, chemicals and radiation including early stages of development, from the prenatal period through puberty, adolescence and on until the first full-term pregnancy" (Nudelman et al., 2009: 80).

The broad categories of concerns addressed by the Breast Cancer Fund include hormones in personal care products; endocrine disrupting chemicals; hormones in food; non-endocrine disrupting industrial chemicals; light-at-night and melatonin; and radiation. Specifically, there is a focus on air and water contaminants; chemicals used in cosmetic and personal care products; chemical ingredients in household cleaning products; chemicals in plastics; exposures in health care settings; and pesticides. The chemicals are considered in terms of the source of exposure and whether they are carcinogenic, a mammary carcinogen, or an endocrine disrupting compound (Gray et al., 2009; Gray, 2010; Nudelman et al., 2009; Nudelman and Engel, 2010). The everyday exposures associated with these toxic substances are widespread and cannot be controlled through measures of precautionary consumption. The Breast Cancer Fund advocates for increased research and regulatory change in order to "decrease human exposures to toxic substances implicated in the high rates of breast cancer, thereby decreasing the incidence of this disease" (Nudelman et al., 2009: 97).

Advocacy organization Breast Cancer Action operates with three priority areas including i) advocating for more effective and less toxic breast cancer treatments; ii) decreasing involuntary environmental exposures that increase the risk of developing breast cancer; and iii) creating awareness that social injustices, including political, economic and racial inequities lead to disparities in breast cancer outcomes (BCA, 2013a). Breast Cancer Action promotes the use of the precautionary principle in its commitment to advancing women's health. "While many breast cancer organizations offer advice on how individuals can reduce their voluntary exposures to carcinogens, the

policy changes needed to eliminate these exposures for everyone require a broader social justice approach" (BCA, 2013b).

Breast Cancer Action launched the "Think Before You Pink" campaign in 2002 which calls for increased levels of transparency and accountability by companies who participate in breast cancer fundraising, and encourages people to ask critical questions about pink ribbon products and promotions (BCA, 2013c). The "What the Cluck?" campaign in 2010 noted the health hypocrisy of "Buckets for the Cure" which was a partnership between KFC and Susan G. Komen for the Cure, one of the largest and most well-funded breast cancer organizations. "Raise a Stink" also targeted Susan G. Komen for the Cure in 2011 to demand the recall of Promise Me perfume and that the highest precautionary standards be adopted to protect women's health. The specific concerns with Promise Me Perfume centre around the chemicals it contains that i) are categorized as toxic and hazardous; ii) have not been adequately evaluated for human safety; and iii) have demonstrated negative health effects. Ingredients in the perfume include galaxolide which is a synthetic musk that acts as a hormone disruptor detected in blood and breast milk, and toluene which is a neurotoxin with a variety of negative health effects and is banned by the International Fragrance Association. This campaign resulted in a victory for Breast Cancer Action with Susan G. Komen for the Cure ending their partnership to produce Promise Me perfume as of May 2012 (BCA, 2011b, 2011c). The 2012 Think Before You Pink campaign was entitled "It's An Epidemic, Stupid" and argued that "after three decades of 'awareness' campaigns and billions of dollars raised, breast cancer remains a public health crisis of epidemic proportions." This campaign called for a

mandate for government action and for meaningful prevention efforts (BCA, 2012). The most recent Think Before You Pink campaign is entitled "Toxic Time is Up!" and was launched in October 2013. It calls for an end to pinkwashing and asks that chemical substances be proven to be safe before they are placed on the market and subsequently, into women's bodies (BCA, 2013d). Breast Cancer Action is calling for a systemic change in advocating for the legislation, regulation, research, and education which would reduce and ultimately eliminate involuntary everyday exposures to toxic substances (BCA, 2013c).

Breast Cancer Action Montreal is a non-profit activist and advocacy organization which works to i) educate the public about environmental toxicants and widespread exposures linked to breast cancer, the precautionary principle, the benefits and risks associated with various treatments for breast cancer, and current cancer research, treatment and services; ii) advocate for policies that would decrease the amount of toxic substances in the environment and allocate increased funding for research on environmental causes of breast cancer; iii) provide support for efforts to improve services, health care and health policies, as well as for individuals to have a strong voice in decisions about their diagnosis and treatment; and iv) network to create a resource-sharing community of women around the issue of breast cancer, and encourage other breast cancer organizations to join the fight for prevention of the disease, as well as for improvements in diagnosis and treatment (BCAM, 2013a).

Breast Cancer Action Montreal has a similar critique about the use of the pink ribbon and launched a campaign called "Little Pink Lies" which counters the mythology

around Breast Cancer Awareness Month (Cohen, 2012[89]). The little pink lies included in this educational campaign include:

- "*We're close to finding a cure*." This message is repeated at fundraising events and printed in media coverage around Breast Cancer Awareness Month. However, the treatment options including surgery, radiation and chemotherapy have not changed in the past forty years, and a cure or cures have not been found despite the significant amount of funding for research in this area. With the mainstream organizational, corporate and media attention focused on the "cure," there has been far less focus on prevention and less than five percent of research funding goes to prevention;

- *"Rates of breast cancer are decreasing.*" While mortality rates among women diagnosed in Canada and the United States are decreasing, this is attributed to detection measures and the reduced use of hormone replacement therapy. The incidence of breast cancer is increasing in developing countries which may be linked with the proliferation of toxic substances in the environment and is not reflected in pink ribbon campaigns;

- "*Pink ribbons mean companies care.*" There is a general perception that purchasing pink ribbons products contributes to the "cause" and that the company's practice is grounded in philanthropy. However, there is very little transparency about what amount of money will be contributed, if there is a cap on donations, or what organization the money will be donated to and what work will be done with the donation. Breast Cancer Action Montreal points to the pinkwashing associated with companies who "support breast cancer" with pink ribbon products while simultaneously manufacturing products which contain ingredients that may be linked to the disease such as automobile manufacturers whose vehicles emit PAHs, cosmetic companies whose products contain carcinogens or endocrine disrupting chemicals, food producers whose cans are lined with BPA, and multinational pharmaceutical companies who produce and sell carcinogenic pesticides as well as medications that are used in cancer treatment;

- "*Government regulations prohibit the use of known or suspected carcinogens in consumer products.*" The *Consumer Product Safety Act* which was introduced in 2011 was designed to protect the public by "addressing or preventing dangers to human health or safety posed by consumer products in Canada." However,

[89] This research project received ethics approval from York University's Office of Research Ethics. Participation was completely voluntary and participants received information describing the goals of the study and signed an informed consent form.

despite meeting the Act's definition of a "danger to human health," it does not include carcinogens. Breast Cancer Action Montreal created a petition calling for regulatory changes asking Health Canada to prohibit the use of any chemicals that are inherently carcinogenic or mutagenic, as well as those that have been identified as reproductive toxicants in products sold in Canada; mandate that manufacturers of consumer products supply full and complete safety data tests for all chemical ingredients used in their products; and require that producers submit complete environmental and health data to Health Canada on each chemical used as most chemicals lack comprehensive testing information; and

- "*We lack evidence that environmental factors affect breast cancer.*" Breast Cancer Action Montreal points to the substantial and growing body of evidence on how chemicals, radiation and other environmental factors contribute to the development of the disease. Over the past sixty years there have been at least 100,000 new chemicals introduced into the environment and breast cancer incidence in Canada rose from one in forty women to one in nine. Recognizing that only five to ten percent of breast cancers are as a result of genetic and family history, the organization points to important findings in considering the everyday environmental exposures women experience including: synthetic chemicals mimicking the action of estrogen; a woman's risk of breast cancer increasing with her lifetime exposure to estrogen; and that estrogen-like chemicals including BPA, PVCs and phthalates are found in consumer goods and personal care products. Women are consistently exposed to a "chemical soup" which can have cumulative and synergistic effects, as well as effects as a result of low-dose, long-term and chronic exposures (BCAM, 2013b).

Breast Cancer Action Montreal is particularly interested in the involuntary risk factors which are inherent in everyday exposures to environmental toxicants and grounds its work in the precautionary principle (Cohen, 2012). In addition to the Little Pink Lies campaign, Breast Cancer Action Montreal has a campaign for safe cosmetics which calls on Health Canada to prohibit the use of chemicals in cosmetics sold in Canada that are inherently carcinogenic, mutagenic or act as a reproductive toxin. The campaign also seeks to mandate that cosmetic companies supply complete safety data tests for all chemical ingredients in their products, and demands that producers supply full environmental and health data on all chemicals used in their products to Health Canada

(BCAM, 2013c, 2013d). FemmeToxic is a related initiative and educational campaign which focuses on young women and toxic substances found in cosmetic and personal care products that are detrimental to human health and may increase the risk of breast cancer.

> The average woman uses 12 cosmetic and personal care products every day, exposing her to 126 unique chemicals. Canada's weak cosmetic regulations, and the influence of the powerful $5.4 billion Canadian cosmetic industry, allow compounds such as carcinogens, mutagens and reproductive toxins, for example, to be used in our cosmetic products. It has reached a point where the financial costs of reformulating outweigh and undermine the impacts and concerns these chemicals have on our health. Marketing schemes have been successful in skewing a woman's perspective on true beauty. The small dose, long-term exposure from these cosmetics toxins accumulate and add to the body burden of women who have already been overloaded with other environmental contaminants that pollute our bodies (FemmeToxic, 2013).

The FemmeToxic campaign engages with youth aged twelve to twenty-five and advocates for stronger regulations from Health Canada including labelling and the substitution and removal of toxic substances in cosmetics and personal care products (BCAM, 2013e; FemmeToxic, 2013).

The final campaign from Breast Cancer Action Montreal is framed around the loopholes in the federal regulatory system for consumer products. "Becoming a Chemical Detective" provides education and resources with practical solutions and safer alternatives in order to help reduce exposure to toxic substances and provides low-cost and affordable solutions, recognizing potential socioeconomic barriers. The campaign is focused on new and potential parents in its emphasis on the risks associated with exposure to toxic substances and susceptibility during pregnancy, infancy, early childhood and adolescence (BCAM, 2013f). Breast Cancer Action Montreal

demonstrates that the focus of breast cancer research must "move beyond its current emphasis on treatment to also embrace a serious search for the causes of the disease and its prevention" (BCAM, 2013g).

As a response to the critiques around Breast Cancer Awareness Month and the pink ribbon campaigns, the Canadian Women's Health Network launched a postcard campaign in 2012 focused on breast cancer prevention. The Canadian Women's Health Network utilized messaging in this educational campaign which included:

- *Think Before You Pink*: Drawing on Breast Cancer Action's campaign, the Canadian Women's Health Network notes the history behind Charlotte Haley's peach ribbon and the lack of funding for primary prevention;

- *Prevent the Root Causes:* This postcard calls for the prevention of the root causes of breast cancer, lung cancer and cardiovascular disease and their impacts on women's health and mortality;

- *An Ounce of Prevention:* Questioning the predominant focus on research for a cure for breast cancer, this postcard notes organizations who are working on prevention including Breast Cancer Action Montreal, the Women's Healthy Environments Network, the Breast Cancer Fund, Breast Cancer Action, and the Alliance for Cancer Prevention;

- *Breast Cancer is Preventable:* This postcard notes that breast cancer is preventable and points to the toxic substances and environmental exposures to the disease, calling for both personal and political action; and

- *Link Our Environments with Prevention*: This postcard speaks to occupational exposures and calls for the need to link both home and work environments with breast cancer prevention efforts (Canadian Women's Health Network, 2012d).

The Canadian Women's Health Network is attempting to shift the dominant discourse on breast cancer and acknowledge the role of the environment, risk and prevention in women's health outcomes (CWHN, 2012d). While the burden of responsibility most often falls onto the individual through precautionary consumption practices, it is

ultimately legislation and public health policy that can play a role in truly protecting women's health and exposure to toxic substances (Cohen, 2012).

The longstanding organizations that operated with funding from Health Canada as part of the Women's Health Contribution Program produced research with a commitment to sex- and gender-based analysis and advancing women's health and well-being. This program was "critical to funding innovative social policy research, building community partnerships and providing important mentorship opportunities in women's health" (CWHN, 2012e). The Women's Health Contribution Program lost its funding as part of the 2012 federal budget. It was anticipated that Health Canada needed to cut more than $200 million from its budget and would save $2.85 million per year in eliminating the Women's Health Contribution Program (Rabson, 2012; Smith, 2012). The budget cuts resulted in four research centres and two communications networks losing their federal funding as of March 31, 2013, including the Atlantic Centre of Excellence for Women's Health, the British Columbia Centre of Excellence for Women's Health, the Canadian Women's Health Network, the Prairie Women's Health Centre of Excellence, the National Network on Environments and Women's Health, and the Réseau Québécois d'Action pour la Santé des Femmes. Steven Outhouse, communications director for the Minister of Health said that Health Canada was prioritizing front-line services (Rabson, 2012). However, Anne Rochon Ford, Executive Director of the Canadian Women's Health Network stated that the biggest loss associated with these cuts will be "how the

groups went beyond clinical research to focus on how particular government policies and regulations affect the health of women" (Smith, 2012).[90]

The Atlantic and Prairie Centres of Excellence for Women's Health have already closed as a result of the budget cuts, while the remaining centres and organizations such as the Canadian Women's Health Network and the National Network on Environments and Women's Health are searching for other sources of funding in the hope of remaining open and continuing to conduct research (CWHN, 2013). Chi Nguyen, Chair of the Board of the Canadian Women's Health Network criticized the funding cuts and remarked that

> [t]he effect of this decision by Health Canada is yet another sign that the federal government is pulling away from its responsibility to gender equality. The work funded through the WHCP [(Women's Health Contribution Program)] has been critical to ensuring that Canadian women have had access to the best evidence and policy advice on women's health issues, through research that recognized social

[90] The Canadian Federation of University Women compiled a list of women's organizations and programs whose funding has been cut or eliminated by the federal government since the Conservative government was elected in 2006. These organizations and programs include Aboriginal Healing Foundation (cuts affected several healing centres that focused on providing support to abused women, such as the Native Women's Shelter of Montreal), Action travail des femmes, Alberta Network of Immigrant Women, Association féminine d'éducation et d'action sociale, Atlantic Centre of Excellence for Women's Health, British Columbia Centre of Excellence for Women's Health, Canadian Child Care Federation, Canadian Research Institute for the Advancement of Women, Canadian Women's Health Network, Centre de documentation sur l'éducation des adultes et la condition féminine, Child Care Advocacy Association of Canada, Childcare Resource and Research Unit, SpeciaLink the National Centre for Child Care Inclusion, Conseil d'intervention pour l'accès des femmes au travail, Elspeth Heyworth Centre for Women Toronto, Feminists for Just and Equitable Public Policy, First Nations Child and Family Caring Society, International Planned Parenthood Federation, Kelowna Women's Resource Centre, Marie Stopes International (a maternal health agency that has received only a promise of "conditional" funding if it avoids any and all connection with abortion), MATCH International, National Association of Women and the Law, National Network on Environments and Women's Health, Native Women's Association of Canada, New Brunswick Coalition for Pay Equity (lost funding for advocacy and research), Older Women's Network, Ontario Association of Interval and Transition Houses, Ontario Coalition for Better Child Care, Pauktuutit, Intuit Women of Canada, Prairie Women's Health Centre of Excellence, Réseau action femmes, Réseau des tables régionales de groupes de femmes du Québec, Le Réseau québécois d'action pour la santé des femmes, Riverdale Immigrant Women's Centre, Toronto, Sisters in Spirit, South Asian Women's Centre, Tri-Country Women's Centre Society, Womanspace Resource Centre, Women and Health Protection, Women for Community Economic Development in Southwest Nova Scotia, Women's Innovative Justice Initiative – Nova Scotia, and Workplace Equity/Employment Equity Program (Canadian Federation of University Women, 2012).

> and environmental determinants of health are key (Institute for Feminist Legal Studies, 2012).

These funding cuts have resulted in losing organizations that conduct important research, policy and advocacy work on women's health with a critical feminist lens (Armstrong et al., 2012). Dayna Scott, Director of the National Network on Environments and Women's Health reflected upon the funding cuts and that organizations will no longer be able to apply their expertise and offer important critiques to policy issues. "It is difficult to reconcile it with a genuine need or desire to protect health in the long-term and to take preventive strategies that move towards health promotion for Canadians" (Scott, 2012c).

Federal funding cuts also seriously impacted the Canadian Environmental Network which is one of the country's oldest and largest environmental groups. It was established in 1977 and represents over 640 diverse environmental organizations with support for networking, communication and coordination services (Canadian Environmental Network, 2013). The Canadian Environmental Network provided opportunities for representatives from environmental non-governmental organizations to "participate in federal government meetings, conferences, workshops and consultations on environmental policy issues through a transparent, bilingual and democratic delegate selection process" (Canadian Environmental Network, 2013). The federal government eliminated the $547,000 in core funding to the Canadian Environmental Network in 2011 (Canadian Environmental Network, 2011; CTV, 2011).

The level of engagement as part of the Canadian Environmental Network falls under the public participation requirements of CEPA 1999 and with the funding cuts, there is no longer an organizational process to coordinate feedback from non-

governmental organizations (Lewis, 2011). The federal government cited responsible spending and sound management of tax dollars as the basis for their decision and suggested that Environment Canada will be moving towards web-based public consultation (Environmental Hansard, 2011). However, there was no explanation provided about the possible limitations of this process including barriers around socioeconomic status and geographic location, access to computers, and levels of computer literacy. Megan Leslie, MP for Halifax criticized this decision stating that

> The cancellation of this funding is forcing the closure of one of the most critical environment networks Canada has. Environment Canada has senselessly ended a 34 year partnership with a respected organisation. The government has told [the network]…that this decision is part of a cost-efficiency plan, but given the important role this organization plays, cancelling their funding will likely have expensive, long-term consequences. This decision just doesn't make sense financially, and it will endanger the health and sustainability of our environment (Leslie, 2011).

Conclusions

This chapter continued to examine the relationship between theory and practice in its examination of Canada's regulatory regime for toxic substances. In doing so, the gaps which exist in Canadian law, policy and practice are revealed. It demonstrates the ways in which women are not adequately protected from detrimental health outcomes as a result of exposure to toxic substances, in part because the regime places the onus of responsibility on individual citizens, and in part because it does not recognize women as a susceptible population who are at risk as a result of exposure to toxic substances. The final chapter will synthesize the findings of the dissertation and provide recommendations based on the research findings including policy implications related to environmental health, breast cancer and disease prevention.

Chapter 6

Conclusions –

A Paradigm Shift: From A Reactionary to a Preventative Approach to Health Policy

Through my examination of the history of Canada's regulatory regime, it becomes clear that this research is steeped in politicized debates as it engages with issues central to women's health, risk and the environment. Davies and Sadler (1997: 19) found that "[p]ublic policy to achieve 'health for all' has yet to be translated into institutionalized processes that systematically address health issues at the policy, program and plan levels for decision-making." Despite substantive regulatory changes since 1997, including a revised *Canadian Environmental Protection Act* and the *Chemicals Management Plan*, these findings remain consistent sixteen years later with Canadian efforts for pollution prevention and precaution surrounding environmental health falling short. The significance of my research findings lies between the promise of precaution grounded in the regulatory regime and promoted by the federal government, and in exposing the gaps which exist in practice. These gaps result in an uneven protection which places women at risk for developing breast cancer. Women are not considered to be a susceptible population at risk as a result of exposure to toxic substances, and the influence of sex- and gender-related determinants of health are not adequately considered. At the same time, there is a gendered burden which places the onus of responsibility for preventing disease on individual women. My findings clearly demonstrate that despite being framed by the precautionary principle, Canadian law, policy and practice is not truly precautionary and does not enact a primary prevention approach.

Only a truly precautionary approach can be effective in protecting women's health. This approach would require shifting debates around causation upstream to focus on everyday exposures to toxic substances, while concurrently shifting the focus away from individual-level factors (Brown et al., 2006: 529). This concluding chapter will explore how primary prevention might be best reflected in Canadian health policy and its potential for positive health outcomes. By engaging with issues of risk, environmental justice, and viewing breast cancer as a multi-faceted social movement, the overarching theoretical framework and interdisciplinary approach allowed for an analysis which involved issues of gender, risk and precaution. The overarching goal of my research was not only to determine what legislation and policies exist within Canada's regulatory regime for toxic substances, but to examine how the issues are communicated and understood, where the burden of risk is presumed to lie, who the policies are designed to protect, and if the policies capture the need for prevention and action related to protecting women's health.

A paradigm shift is required in how the issues around breast cancer are communicated and understood, and where the burden of responsibility and risk is presumed to lie. The responsibilization paradigm and the trend towards individualization of risk clearly place the onus of responsibility in determining health outcomes on the individual. The dominant epidemiological paradigm is utilized by the biomedical community and the mainstream breast cancer movement to promote individual-level approaches to prevention, detection and treatment. This approach is promoted by government departments and mainstream cancer organizations and focuses solely on

modifiable risk factors such as tobacco and alcohol use, a lack of physical activity, and a healthy diet. The clear messaging in health promotion and public education campaigns is that breast cancer is preventable if individuals engage and participate in a "healthy lifestyle." However, it is not made clear that the modifiable risk factors account for only a fraction of breast cancer incidence. The focus on lifestyle and behavioural risk factors excludes the possibility of other approaches and dismisses the importance of other social, structural, political, economic, and environmental factors that influence the disease.

The promotion of precautionary consumption practices acknowledges the potential role of toxic substances in health outcomes. However, risk is still framed as something that can be controlled by individual citizens through acts of green consumption in order to avoid everyday exposures to toxic substances. This practice is also highly problematic in placing the onus of responsibility at the level of the individual and in dismissing other social determinants of health including socioeconomic status and education, as well as creating a gendered and disproportionate burden on women. In both the dominant epidemiological paradigm and practices of precautionary consumption, individual citizens are encouraged to act and are framed as the "risk managers" that the federal government promotes (Government of Canada, 2011f).

Women would benefit from a repoliticization of breast cancer in order to shift away from the fundamental emphasis on lifestyle and behavioural risk factors, as well as from the widespread and consumption-based pink ribbon campaigns which are designed to raise a very specific type of "awareness." Pink ribbon campaigns have resulted in the commercialization of breast cancer which presents the disease through a very restricted

and narrow lens. The efforts to raise awareness about breast cancer present a particular framing of the disease and do not encourage a more critical examination around the messaging of the campaigns, a lack of transparency in donated funds, and instances of pinkwashing. These pink ribbon campaigns divert attention from the realities of the disease, environmental links to breast cancer, and calls for primary prevention.

Environmental law, policy and practice are designed to protect and be representative of the entire Canadian population, but they are inadequate in protecting populations at risk including those who are highly exposed through occupational settings and in geographic areas that are highly polluted. Children's health emerged as a particular area of concern in the late 1990s, but this has not been reflected in the legislation as CEPA 1999 does not specifically address any populations of concern. Health Canada (2010c) does consider children as a vulnerable population in the *National Strategic Framework on Children's Environmental Health.* However, this method is contradictory as it does not include a lifecourse approach which allows for understanding the causal links between determinants of health throughout a person's life and health outcomes. It also does not account for windows of susceptibility that occur across the lifespan where the timing of exposure to toxic substances and stage of biological development can impact the development of diseases such as breast cancer.

Lalonde (1974: 18) noted with great foresight that "all the foregoing environmental conditions create risks which are a far greater threat to health than any present inadequacy of the health care system." *A New Perspective on the Health of Canadians* would benefit from a revised second edition with updated information on

environmental contaminants as determinants of health, the impact of sex and gender on health outcomes, and the role of primary prevention. Despite a provision in section 44(1)(f) of CEPA 1999 that requires the publication of reports on the state of the Canadian environment, this practice appears to have ceased since publications in 1991 and 1996. Both Standing Committees raised concerns about this during the CEPA 1999 legislative review and it would be advantageous to reinstate the practice of monitoring, reporting and disseminating the results about the state of the Canadian environment (House of Commons Standing Committee on Environment and Sustainable Development, 2007; House of Commons Standing Senate Committee on Energy, the Environment and Natural Resources, 2008). This process is a requirement for the Minister of the Environment, but it would be beneficial for Environment Canada to collaborate with Health Canada in order to address the current state of the Canadian environment, as well as the impacts of toxic substances on the environment and on human health.

Despite CEPA 1999 and the *Chemicals Management Plan* emphasizing the precautionary principle and the prevention of pollution, it is clear that the risk assessment and risk management processes are reactionary rather than precautionary. Firstly, the mandatory exposure requirement in determining the toxicity of a substance does not recognize inherent toxicity, and a toxic substance cannot be regulated for having the potential to cause harm. This process allows for harm to occur before a risk can be appropriately managed whereas a hazard assessment instead of or along with a risk assessment would offer room to evaluate the inherent toxicity and hazard of the substance, and its potential to cause harm in and of itself. Secondly, the risk assessment

and risk management process continues to be based on threshold values and the traditional dose-response relationship which suggest that threshold effects occur only at a specific level of exposure and that there is a toxicological threshold below which adverse effects do not occur. The basis of this aspect of risk assessment is problematic because it does not adequately account for the timing of exposure, windows of susceptibility, or the effects of low dose, cumulative and synergistic exposures to toxic substances including endocrine disrupting chemicals which have impacts below the traditional dose-response curve and threshold values. It also does not consider how sex and gender may impact the margins of exposure and how women may be a susceptible population.

Despite a formal commitment by the federal government, the Auditor General's report clearly demonstrates that sex- and gender-based analysis is not adequately integrated into policy development (Minister of Public Works and Government Services Canada, 2009). The role of sex and gender in influencing health outcomes is not reflected in Canadian law, policy and practice, and women are not considered to be a susceptible population of concern under CEPA 1999 or the *Chemicals Management Plan*. Public health policy and the regulatory regime for toxic chemicals lack the application of sex- and gender-based analysis which implies a gendered preference in their implementation and in the ability to protect women from health outcomes, such as breast cancer which is influenced by exposure to mammary carcinogens and endocrine disrupting chemicals. The budget cuts which formally eliminated federal funding for the Canadian Environmental Network and the Women's Health Contribution Program demonstrate a

continued devaluing of public engagement around environmental issues and of a commitment to women's health research and policy.

My dissertation concludes that Canadian law, policy and practice are not truly precautionary and do not capture the need for prevention and action related to women's health. Unless these gaps are adequately addressed in the federal regulatory regime, women will continue to be placed at risk. The importance of primary prevention in breast cancer cannot be overstated, and the current regulatory regime does not enact primary prevention. There is a need to bring the environment into public health discourse in a meaningful way which recognizes and fully understands the risks associated with exposure to toxic substances. An approach to health which embodies primary prevention must include sex- and gender-based analysis, as well as shifting the burden of risk and responsibility away from individual women. As Seager (2003: 957) suggests

> [i]t has taken (and still takes) relentless pressure from environmental justice and women's health advocates to shift paradigms—to put human health issues on the mainstream environmental movement agenda and to put environmental issues on the health map. Even now, virtually all assertions of causality between health disruptions and environmental assaults are fiercely contested, all the more so when women are the primary proponents of linkage.

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Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Dose Responses

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For decades, studies of endocrine-disrupting chemicals (EDCs) have challenged traditional concepts in toxicology, in particular the dogma of "the dose makes the poison," because EDCs can have effects at low doses that are not predicted by effects at higher doses. Here, we review two major concepts in EDC studies: low dose and nonmonotonicity. Low-dose effects were defined by the National Toxicology Program as those that occur in the range of human exposures or effects observed at doses below those used for traditional toxicological studies. We review the mechanistic data for low-dose effects and use a weight-of-evidence approach to analyze five examples from the EDC literature. Additionally, we explore nonmonotonic dose-response curves, defined as a nonlinear relationship between dose and effect where the slope of the curve changes sign somewhere within the range of doses examined. We provide a detailed discussion of the mechanisms responsible for generating these phenomena, plus hundreds of examples from the cell culture, animal, and epidemiology literature. We illustrate that nonmonotonic responses and low-dose effects are remarkably common in studies of natural hormones and EDCs. 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. We conclude that when nonmonotonic dose-response curves occur, the effects of low doses cannot be predicted by the effects observed at high doses. Thus, fundamental changes in chemical testing and safety determination are needed to protect human health. (***Endocrine Reviews*** **33: 378–455, 2012)**

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Abbreviations: A4, Androstenedione; AhR, aryl hydrocarbon receptor; BPA, bisphenol A; CDC, Centers for Disease Control and Prevention; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; DES, diethylstilbestrol; EDC, endocrine-disrupting chemical; EPA, Environmental Protection Agency; ER, estrogen receptor; FDA, Food and Drug Administration; GLP, good laboratory practices; LOAEL, lowest observed adverse effect level; mER, membrane-associated ER; NHANES, National Health and Nutrition Examination Survey; NIS, sodium/iodide symporter; NMDRC, nonmonotonic dose-response curve; NOEL, no observed effect level; NOAEL, no observed adverse effect level; NTP, National Toxicology Program; PIN, prostatic intraepithelial neoplasias; POP, persistent organic pollutants; ppb, parts per billion; SERM, selective ER modulator; TCDD, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; WoE, weight of evidence.

I. Introduction

This review focuses on two major issues in the study of endocrine-disrupting chemicals (EDCs): low-dose exposures and nonmonotonic dose-response curves (NMDRCs). These concepts are interrelated, and NMDRCs are especially problematic for assessing potential impacts of exposure when nonmonotonicity is evident at levels of exposure below those that are typically used in toxicological assessments. For clarity of presentation, however, we will first examine each of the concepts separately.

A. Background: low-dose exposure

It is well established in the endocrine literature that natural hormones act at extremely low serum concentrations, typically in the picomolar to nanomolar range. Many studies published in the peer-reviewed literature document that EDCs can act in the nanomolar to micromolar range, and some show activity at picomolar levels.

1. What is meant by low dose?

In 2001, at the request of the U.S. Environmental Protection Agency (EPA), the National Toxicology Program (NTP) assembled a group of scientists to perform a review of the low-dose EDC literature (1). At that time, the NTP panel defined low-dose effects as any biological changes 1) occurring in the range of typical human exposures or 2) occurring at doses lower than those typically used in standard testing protocols, *i.e.* doses below those tested in traditional toxicology assessments (2). Other definitions of low dose include 3) a dose below the lowest dose at which a biological change (or damage) for a specific chemical has been measured in the past, *i.e.* any dose below the lowest observed effect level or lowest observed adverse effect level (LOAEL) (3), or 4) a dose administered to an animal that produces blood concentrations of that chemical in the range of what has been measured in the general human population (*i.e.* not exposed occupationally, and often referred to as an environmentally relevant dose because it creates an internal dose relevant to concentrations of the chemical measured in humans) (4, 5). This last definition takes into account differences in chemical metabolism and pharmacokinetics (*i.e.* absorption, distribution, and excretion of the chemical) across species and reduces the importance of route of exposure by directly comparing similar blood or other tissue concentrations across model systems and experimental paradigms. Although these different definitions may seem quite similar, using just a single well-studied chemical like bisphenol A (BPA) shows how these definitions produce different cutoffs for exposure concentrations that are considered low dose (Table 1). For many chemicals, including EDCs, a large number of studies meet the criteria for low-dose studies regardless of whether the cutoff point for a low dose was based on the range of typical human exposures, doses used in traditional toxicology, or doses that use an internal measure of body burden.

Whether low doses of EDCs influence disease is a question that now extends beyond the laboratory bench, because epidemiological studies show that environmental exposures to these chemicals are associated with disorders in humans as well (see for examples Refs. 6–16). Although disease associations have historically been observed in individuals exposed to large concentrations of EDCs after

TABLE 1. Low-dose definitions and cutoff doses: BPA and DEHP as examples

Chemical	Estimated range of human exposures	Doses below the NOAEL	Doses below the LOAEL	Administered doses (to animals) that produce blood levels in typical humans
BPA	0.4–5 μg/kg · d (679)	No NOAEL was ever established in toxicological studies (38)	<50 mg/kg · d (38)	~400 μg/kg · d to rodents and nonhuman primates (4, 253)
DEHP	0.5–25 μg/kg · d (680)	<5.8 mg/kg · d (681, 682)	<29 mg/kg · d (681, 682)	Unknown

Estimates of human exposure are made from consumer product consumption data but do not take into account that there are unknown sources of these chemicals.
DEHP, Bis(2-ethylhexyl) phthalate.

industrial accidents (17–19) or via occupational applications (20–22), recent epidemiological studies reveal links between environmentally relevant low concentrations and disease prevalence. With the extensive biomonitoring studies performed by the U.S. Centers for Disease Control and Prevention (CDC) (23, 24) and similar environmental surveys performed in Europe (25) and elsewhere (www.statcan.gc.ca/concepts/hs-es/measures-mesures-eng.htm), knowledge about environmental exposures to EDCs and their associations with human health disorders has increased substantially.

Low-dose effects have received considerable attention from the scientific and regulatory communities, especially when examined for single well-studied chemicals like BPA (4, 27–32). The low-dose literature as a whole, however, has not been carefully examined for more than a decade. Furthermore, this body of literature has been disregarded or considered insignificant by many (33, 34). Since the NTP's review of the low-dose literature in 2001 (2), a very large body of data has been published including 1) additional striking examples of low-dose effects from exposures to well-characterized EDCs as well as other chemicals, 2) an understanding of the mechanisms responsible for these low-dose effects, 3) exploration of nonmonotonicity in *in vivo* and *in vitro* systems, and 4) epidemiological support for both low-dose effects and NMDRCs.

2. Is the term low dose a misnomer?

Endogenous hormones are active at extremely low doses, within and below the picomolar range for endogenous estrogens and estrogenic drugs, whereas environmental estrogen mimics are typically active in the nanomolar to micromolar range (for examples, see Refs. 35–38), although some show effects at even lower concentrations (39–41). Importantly, the definitions above do not take into account the potency or efficacy of the chemical in question, a topic that will be discussed in greater detail below. Instead, low dose provides an operational definition, in which doses that are in the range of human exposure, or doses below those traditionally tested in toxicological studies, are considered low. To be clear, none of these definitions suggest that a single concentration can be set as a low dose cutoff for all chemicals. Using the above definitions, for some chemicals, low doses could potentially be in the nanogram per kilogram range, but for most chemicals, doses in the traditional micro- and milligram per kilogram range could be considered low doses because traditional approaches to testing chemicals typically did not examine doses below the milligram per kilogram dose range.

B. Background: NMDRCs

We have defined low-dose studies according to the definitions established by the NTP panel of experts (2). However, because the types of endpoints that are typically examined at high doses in toxicological studies are often different from the types of endpoints examined in low-dose studies, one cannot assume that an effect reported in the low-dose range is necessarily different from what would be observed at higher doses. For example, low doses of a chemical could affect expression of a hormone receptor in the hypothalamus, an endpoint not examined in high-dose toxicology testing, and high doses could similarly affect this same endpoint (but are likely to be unreported because high doses are rarely tested for these types of endpoints). Thus, the presence of low-dose effects makes no assumptions about what has been observed at higher concentrations. (As discussed elsewhere, for the majority of chemicals in commerce, there are no data on health effects and thus no established high- or low-dose range.) Therefore, low-dose effects could be observed at the lower end of a monotonic or linear dose-response curve.

In contrast, the definition of a NMDRC is based upon the mathematical definition of nonmonotonicity: that the slope of the dose-response curve changes sign from positive to negative or vice versa at some point along the range of doses examined (42). Often NMDRCs have a U- or inverted U-shape (43); these NMDRCs are thus also often referred to as biphasic dose-response curves because responses show ascending and descending phases in relation to dose. Complex, multiphasic curves have also been observed (41, 44, 45). NMDRCs need not span from true low doses to high (pharmacologically relevant) doses, although experiments with such a broad dose range have been performed for several EDCs; the observation of nonmonotonicity makes no assumptions about the range of doses tested. Examples of NMDRCs from *in vitro* cell culture and *in vivo* animal experiments, as well as epidemiological examples, are presented in detail later in this review (see *Sections III.C.1–3*). Additional examples of NMDRCs are available in studies examining the effects of vitamins and other essential elements on various endpoints (see for example (46); these will not be examined in detail in this review due to space constraints.

NMDRCs present an important challenge to traditional approaches in regulatory toxicology, which assume that the dose-response curve is monotonic. For all monotonic responses, the observed effects may be linear or nonlinear, but the slope does not change sign. This assumption justifies using high-dose testing as the standard for assessing chemical safety. When it is violated, high-dose testing regimes cannot be used to assess the safety of low doses.

It should be noted that both low dose and nonmonotonicity are distinguished from the concept of hormesis, which is defined as a specific type of response whereby "the various points along [the dose response] curve can be interpreted as beneficial or detrimental, depending on the biological or ecological context in which they occur" (47). Estimations of beneficial or adverse effects cannot be ascertained from the direction of the slope of a dose-response curve (48–50). In their 2001 Low Dose Peer Review, the NTP expert panel declined to consider whether any effect was adverse because "in many cases, the long-term health consequences of altered endocrine function during development have not been fully characterized" (2). There are still debates over how to define adverse effects (51–53), so for the purposes of this review, we consider any biological change to be an effect. Importantly, most epidemiological studies are by definition examining low doses (unless they are focusing on occupationally exposed individuals), and these studies typically focus on endpoints that are accepted to be adverse for human health, although some important exceptions exist (54–56).

Finally, it is worth noting that any biological effect, whether it is observed to follow linear relationships with administered dose or not, provides conclusive evidence that an EDC has biological activity. Thus, other biological effects are likely to be present but may remain undetected or unexamined. Many EDCs, including those used as pesticides, were designed to have biological effects (for example, insecticides designed to mimic molting hormone). Thus, the question of whether these chemicals have biological effects is answered unequivocally in their design; the question is what other effects are induced by these biologically active agents, not whether they exist.

C. Low-dose studies: a decade after the NTP panel's assessment

In 2000, the EPA requested that the NTP assemble a panel of experts to evaluate the scientific evidence for low-dose effects and dose-response relationships in the field of endocrine disruption. The EPA proposed that an independent and open peer review of the available evidence would allow for a sound foundation on which the EPA could "determine what aspects, if any, of its standard guidelines for reproductive and developmental toxicity testing [would] need to be modified to detect and characterize low-dose effects" (2). The NTP panel verified that low-dose effects were observed for a multitude of endpoints for specific EDCs including diethylstilbestrol (DES), genistein, methoxychlor, and nonylphenol. The panel identified uncertainties around low-dose effects after exposure to BPA; although BPA had low-dose effects on some endpoints in some laboratories, others were not found to be consistent, leading the panel to conclude that it was "not persuaded that a low-dose effect of BPA has been conclusively established as a general or reproducible finding" (2).

Since the NTP's review of low-dose endocrine disruptor studies, only a few published analyses have reexamined the low-dose hypothesis from a broad perspective. In 2002, R. J. Witorsch (57) analyzed low doses of xenoestrogens and their relevance to human health, considering the different physiologies associated with pregnancy in the mouse and human. He proposed that low doses of endocrine disruptors would not likely affect humans because, although low-dose effects had been observed in rodents, the hormonal milieu, organs controlling hormonal release, and blood levels of estrogen achieved are quite different in humans. There are, of course, differences in hormones and hormone targets between rodents and humans (58), but the view that these differences negate all knowledge gained from animal studies is not supported by evolutionary theory (59–61). This human-centered stance argues against the use of animals for any regulatory testing (62) and runs counter to the similarities in effects of EDCs on humans and animals; rodents proved to be highly predictive of the effects of DES on humans (63, 64). In a striking example, studies from mice and rats predicted that gestational exposure to DES would increase mammary cancer incidence decades before women exposed *in utero* reached the age where this increase in risk was actually observed (65–67).

In 2007, M. A. Kamrin (68) examined the low-dose literature, focusing on BPA as a test case. He suggested that three criteria were required to support the low-dose hypothesis. First is reproducibility, which he defined as "the same results are seen from the same causes each time a study is conducted." Furthermore, he proposed that the dose response for the effects must be the same from study to study. Second is consistency, which he defined as the results all fitting into a pattern, whereby the results collected from multiple species and under variable conditions all show the same effect. And third is proper conduct of studies, which he defined as including the appropriate controls and performance under suitable experimental conditions as well as the inclusion of multiple doses such that a dose-response curve can be obtained.

Although we and others (69–72) agree with the use of these criteria (reproducibility, consistency, and proper experimental design), there are significant weaknesses in the logic Kamrin employed to define these factors. First, suggesting that reproducibility is equivalent to the same results obtained each time a study is conducted is unrealistic and not a true representation of what is required of replication. As has been discussed in other fields, "there is no

end to the ways in which any two experiments can be counted as the same — or different . . . All experiments are the same in respect of their being experiments; they are all different by virtue of being done at different places, at different times, by different people, with different strains of rat, training regime, and so on" (73).

Furthermore, according to the Bradford-Hill criteria, a set of requirements accepted in the field of epidemiology to provide adequate evidence of a causal relationship between two factors, a single negative result (or even several studies showing negative results) cannot negate other studies that show adverse effects (74). Essentially, all scientists know that it is very easy for an experiment to find no significant effects due to a myriad of reasons; it is more difficult to actually find effects, particularly when using highly sophisticated techniques (69).

Second, the concept of consistency as a pattern that can be derived from all results is one we will use below, using a weight-of-evidence (WoE) approach and several specific examples. However, Kamrin's proposed idea that every study must show the same effect has the same weaknesses as discussed for the proposed definition of reproducibility and does not acknowledge the obvious differences in many species and strains. It also suggests that the identification of a single insensitive strain could negate any number of positive studies conducted with appropriate animal models (75).

And finally, Kamrin suggested that only studies with appropriate controls should be used for analyses, a criterion we agree should be followed. However, his own scrutiny of the low-dose animal literature fails to do so (68). He also suggested that studies use multiple doses so that a dose-response curve can be obtained. Although studies using a single dose can be informative, we agree that dose-response relationships provide important information to researchers and risk assessors alike. However, this requirement is not helpful if there is an insistence on observing a linear response; as we discuss in depth in this review, there are hundreds of examples of nonmonotonic and other nonlinear relationships between dose and endpoint. These should not be ignored.

In 2004, Hayes (76) reviewed the available literature concerning the effects of atrazine on amphibian development, with a specific focus on the effect of ecologically relevant doses of this EDC on malformations of the gonads and other sexually dimorphic structures; in the case of aquatic exposures, it can be difficult to determine what a cutoff for a low dose would be; thus, Hayes focused on studies examining the effects of atrazine at levels that had been measured in the environment. He reviewed the results produced by several labs, in which it was independently demonstrated that low concentrations of atrazine produced gonadal abnormalities including hermaphroditism, males with extra testes, discontinuous gonads, and other defects. Hayes' work also clearly addressed the so-called irreproducibility of these findings by analyzing the studies that were unable to find effects of the pesticide; he noted that the negative studies had multiple experimental flaws, including contamination of the controls with atrazine, overcrowding (and therefore underdosing) of experimental animals, and other problems with animal husbandry that led to mortality rates above 80%.

In 2006, vom Saal and Welshons (77) examined the low-dose BPA literature, identifying more than 100 studies published as of July 2005 that reported significant effects of BPA below the established LOAEL, of which 40 studies reported adverse effects below the 50 μg/kg · d safe dose set by the EPA and U.S. Food and Drug Administration (FDA); all of these studies would be considered low dose according to the NTP's definition (2). The authors proposed that these examples should be used as evidence to support the low-dose hypothesis. Furthermore, this publication detailed the similarities among the studies that were unable to detect any effects of low doses of BPA and established a set of criteria required to accept negative studies. We have adapted the criteria detailed by Hayes (76) and vom Saal and Welshons (77) to produce a set of requirements for low-dose studies; these criteria are described in some detail below.

D. Why examine low-dose studies now?

The developmental origins of health and disease hypothesis originated from studies showing that fetal DES exposure could cause severe malformations and cancers of the reproductive tract, and other studies demonstrating that fetal malnutrition could lead to adult diseases including metabolic syndrome, diabetes, and increased stroke incidence (78–81). Since that time, the developmental origins of health and disease hypothesis has been extended to address whether diseases that are increasing in prevalence in human populations could be caused by developmental exposures to EDCs (67, 82–85). Evidence from the animal literature has been tremendously informative about the effects of EDC exposures early in development and has driven new hypotheses to be tested in epidemiology studies (86). Studies including several discussed in this review provide supportive evidence that the fetal and neonatal periods are specifically sensitive to chemicals that alter endocrine signaling and that EDCs could be contributing to a range of diseases.

Strong, reliable, and reproducible evidence documents the presence of low concentrations of EDCs and other chemicals in human tissues and fluids, as well as in environmental samples (28, 87–89). These studies indicate

that samples collected from humans and the environment typically contain hundreds of contaminants, usually in the parts-per-billion (ppb) range (90, 91). The obvious question with potentially large public health implications is whether these concentrations are so low as to be irrelevant to human health. The fact that epidemiological analyses (reviewed in *Section III.C.3*) repeatedly find associations between the measured concentrations in human samples and disease endpoints suggests it is inappropriate to assume the exposures are too low to matter. That is especially the case given the empirical data (reviewed in *Section II.A*) from animal and cell culture experiments showing effects can be caused by concentrations comparable (and sometimes below) what is measured in humans and also the detection of NMDRCs in some of those same experiments.

In the human biomonitoring field, large databases such as the CDC's National Health and Nutrition Examination Survey (NHANES) have allowed researchers to make comparisons between groups of individuals with various exposure criteria; some of these studies will be addressed in detail in subsequent sections of this review. Although by definition these databases examine low-dose exposures, their use has been the subject of significant debate. Because of the large number of chemicals that have been measured (>300 in the most recent NHANES by the CDC) and the large number of health outcomes and other disease-related data collected from the individuals that donated biological samples, it has been argued that the number of possible associations that could be made would lead to a significant number of false positives (92); thus, associations could be found simply because of extensive data dredging. This has led some to suggest that these studies as a whole should be rejected (93, 94).

In response to these criticisms, epidemiologist Jan Vandenbroucke (95) notes, "researchers do not mindlessly grind out one analysis after another"; the examination of these databases for associations between chemical exposures and health effects does not entail the statistical comparison between all possible factors, calculated as some 8800 comparisons in the CDC's NHANES database (92). Instead, epidemiologists typically focus on a select number of comparisons that address relationships between chemicals and diseases identified *a priori* (96, 97), often because of mechanistic data obtained in laboratory animals or *in vitro* work with human and animal cells and tissues. Repeated findings of links between EDC exposures and diseases in epidemiological analyses of biomonitoring data based on *a priori* hypotheses suggests these relationships should not be rejected as a statistical artifact and, instead, should be the basis for significant concern that low-dose effects can be detected in the general population (85, 98).

E. Mechanisms for low-dose effects

The endocrine system is particularly tuned to respond to very low concentrations of hormone, which allows an enormous number of hormonally active molecules to coexist in circulation (38). As a ligand-receptor system, hormones act by binding to receptors in the cell membrane, cytosol, or the nucleus. The classical effects of nuclear hormone receptors influence gene expression directly, although rapid nongenomic actions at membrane-associated receptors are now well documented and accepted. Membrane receptors are linked to different proteins in the cell, and binding to these receptors typically changes cellular responses in a rapid fashion (99), although the consequence of a rapid signaling event could be the activation of a nuclear transcription factor, leading to responses that take longer to detect. Peptide hormones can also influence gene expression directly (see Refs. 100 and 101 for examples).

There are several means by which the endocrine system displays specificity of responses to natural hormones. Many hormone receptors are expressed specifically in a single or a few cell types (for example, receptors for TSH are localized to the thyroid), whereas some (like thyroid hormone receptors) are found throughout the body (102). For receptors that are found in multiple cell types, different effects are produced in part due to the presence of different coregulators that influence behaviors of the target genes (103–105). And finally, some hormones have multiple receptors [for example estrogen receptor (ER)α and ERβ], which are expressed in different quantities in different cell types and organs and can produce variable effects on gene expression or cellular phenomena (cell proliferation *vs.* apoptosis) (102, 106).

The typical physiological levels of the endogenous hormones are extremely low, in the range of 10–900 pg/ml for estradiol, 300–10,000 pg/ml for testosterone, and 8–27 pg/ml for T_4 (see Table 2). Importantly, steroid hormones in the blood are distributed into three phases: free, representing the unconjugated, unbound form; bioavailable, representing hormones bound to low-affinity carrier proteins such as albumin; and inactive, representing the form that is bound to high-affinity binding proteins such as SHBG or α-fetoprotein (38) (Fig. 1A). When the circulating levels in blood are corrected for the low fraction of the hormones that are not bound to serum binding proteins, the free concentrations that actually bring about effects in cells are even lower, for example 0.1–9 pg/ml for estradiol. Concentrations of active hormones will vary based on the age and physiological status of the individual (*i.e.* plasma testosterone levels are less than 1 ng/ml in male children but increase to approximately 5–7 ng/ml in adulthood; during menses, estradiol levels are typically less than 100

TABLE 2. Ranges of endogenous hormones in humans (from Ref. 108)

Hormone	Free concentration (females)	Total concentration (females)	Free concentration (males)	Total concentration (males)
Cortisol	20–300 ng/ml		20–300 ng/ml	
Estradiol	0.5–9 pg/ml (adult female)	<20 pg/ml (prepubertal) 20–800 pg/ml (premenopausal) <30 pg/ml (postmenopausal)		10–60 pg/ml (adult)
Progesterone		0.2–0.55 ng/ml (prepubertal) 0.02–0.80 ng/ml (follicular phase) 0.90–4 ng/ml (luteal phase) <0.5 ng/ml (postmenopausal)		0.1–0.4 ng/ml (prepubertal) 0.2–2 ng/ml (adult)
Insulin		0–250 pmol/liter		0–250 pmol/liter
GH		2–6 ng/ml		2–6 ng/ml
Prolactin		0–15 ng/ml		0–10 ng/ml
Testosterone	9–150 pg/ml (adult)		0.3–250 ng/ml	
Thyroid hormone	8–30 pg/ml (10–35 pM)		8–30 pg/ml (10–35 pM)	
TSH	0.5–5 μU/ml		0.5–5 μU/ml	

pg/ml, but just before ovulation, they spike to 800 pg/ml; *etc.*) (107, 108). Of course, it should be noted that active concentrations of natural hormones vary somewhat from species to species and can even vary between strains of the same species (109).

There are several reasons why endogenous hormones are able to act at such low circulating concentrations: 1) the receptors specific for the hormone have such high affinity that they can bind sufficient molecules of the hormone to trigger a response, 2) there is a nonlinear relationship between hormone concentration and the number of bound receptors, and 3) there is also a nonlinear relationship between the number of bound receptors and the strongest observable biological effect. Welshons and colleagues (38) describe how hormone concentration influences receptor occupancy: "receptor occupancy is never determined to be linear in relation to hormone concentration . . . At concentrations above the K_d [the dissociation constant for receptor-ligand binding kinetics], saturation of the response occurs first, and then at higher concentrations, saturation of receptors is observed." What this means is that at low doses of hormone, a 10-fold increase in hormone concentration can have a 9-fold increase in receptor occupancy, whereas at high doses of hormone, a 10-fold increase in hormone concentration produces a less than 1.1-fold increase in receptor occupancy (38) (Fig. 1B). Thus, even moderate changes in hormone concentration in the low-dose range can produce substantial changes in receptor occupancy and therefore generate significant changes in biological effects. Welshons *et al.* (38) also note that a near-maximum biological response can be observed without a high rate of receptor occupancy, a situation that was previously termed the spare receptor hypothesis (110, 111); that is, the response mechanism saturates before all of the receptors are saturated. The presence of spare receptors is the basis for saying that these receptor systems are tuned to detect low concentrations that lead to occupancy of 0.1–10% of total receptors. Within this range of low receptor occupancy, there is high proportionality between changes in the free hormone concentration and changes in receptor occupancy, and a change in receptor occupancy by a ligand for the receptor is required to initiate changes in receptor-mediated responses (38).

There are additional reasons why natural hormones are active at low doses: 4) hormones have a strong affinity for their receptors (relative to affinity for other receptors) because many hormones are secreted from a single gland or site in the body but must have effects throughout the body in multiple tissues and 5) blood concentrations of hormones are normally pulsatile in nature, with the release of one hormone often controlled by the pulsatile release of another hormone (112, 113), and both the frequency and the amplitude of pulses modulate the biological response; hormones are also influenced by circadian rhythms, with dramatic differences in hormone secretion depending on the time of day (114, 115).

For many years, the mechanisms by which some environmental chemicals acted at low doses were not well understood. In 1995, the National Research Council appointed the Committee on Hormonally Active Agents in the Environment to address public concerns about the potential for adverse effects of EDCs on human health (116). At the time, work on understanding the mechanisms by which EDCs exert their effects was in its infancy, and in the executive summary, the committee stated, "Lack of knowledge about a mechanism does not mean that a reported effect is unconfirmed or unimportant, nor does demonstration of a mechanism document that the resulting effects are unique to that mechanism or are pervasive

Figure 1.

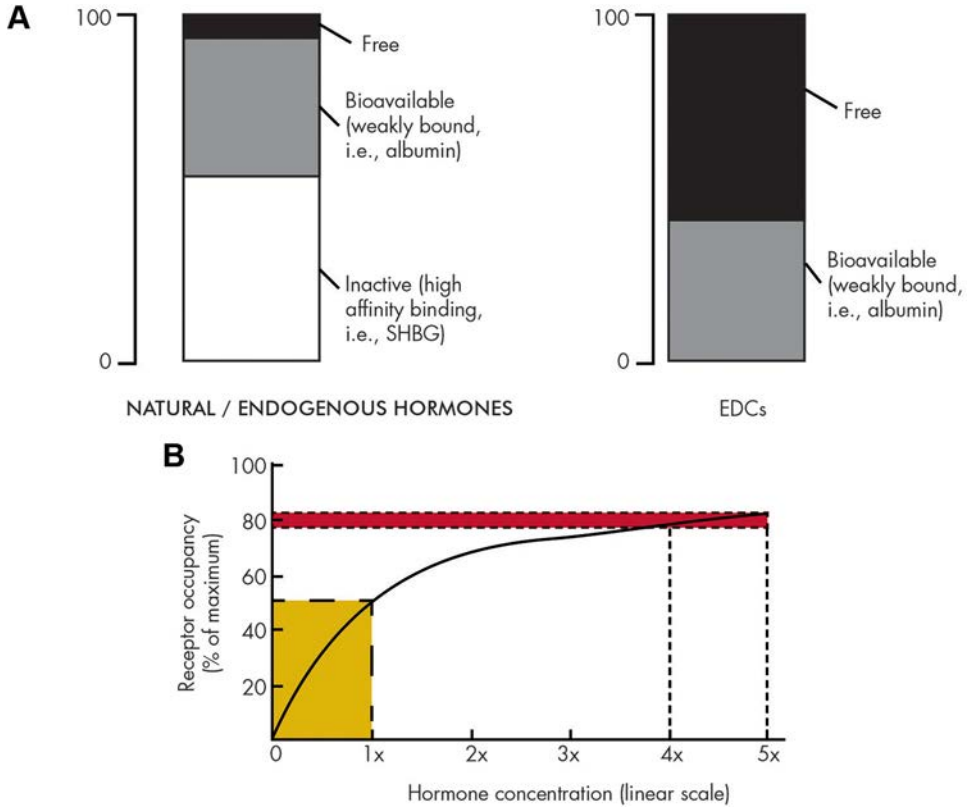

Figure 1. Characteristics and activities of natural hormones. A, This schematic depicts a typical relationship of three phases of circulating hormones: free (the active form of the hormone), bioavailable (bound weakly to proteins such as albumin), and inactive (bound with high affinity to proteins such as SHBG). These three phases act as a buffering system, allowing hormone to be accessible in the blood, but preventing large doses of physiologically active hormone from circulating. With EDCs, there may be little or no portion maintained in the inactive phase. Thus, the entirety or majority of a circulating EDC can be physiologically active; the natural buffering system is not present, and even a low concentration of an EDC can disrupt the natural balance of endogenous hormones in circulation. B, Schematic example of the relationship between receptor occupancy and hormone concentration. In this theoretical example, at low concentrations, an increase in hormone concentration of x (from 0 to 1x) causes an increase in receptor occupancy of approximately 50% (from 0 to 50%, see *yellow box*.) Yet the same increase in hormone concentration at higher doses (from 4x to 5x) causes an increase in receptor occupancy of only approximately 4% (from 78 to 82%, see *red box*).

in natural systems." Since that time, a tremendous amount of work has been dedicated to understanding the molecular mechanisms of action of EDCs, and in particular the mechanisms responsible for low-dose effects.

1. General mechanisms for EDC action

As discussed above, the endocrine system evolved to function when unbound physiologically active ligands (hormones) are present at extremely low doses (117). Because of shared receptor-mediated mechanisms, EDCs that mimic natural hormones have been proposed to follow the same rules and therefore have biological effects at low doses (38, 118). Similarly, EDCs that influence in any way the production, metabolism, uptake, or release of hormones also have effects at low doses, because even small changes in hormone concentration can have biologically important consequences (38, 119).

The estrogen-response mechanisms have been extensively studied with regard to the effects of endogenous estrogens and estrogenic drugs. In classical, genomic estrogen action, when endogenous estrogens bind to ER, those receptors bind to estrogen response element sequences or to a number of other response element sites adjacent to the genes directly responsive to estrogens; this binding influences transcription of estrogen-sensitive genes (120). Xenoestrogens produce the same reactions; these chemicals bind to ERs, which then initiate a cascade of molecular effects that ultimately modify gene expression. Therefore, for the actions of estrogenic EDCs, molecular mechanisms and targets are already known in some detail. Similar mechanisms are induced by the binding of androgens to the androgen receptor, or thyroid hormone agonists to the thyroid hormone receptor, among others.

Additionally, there are EDCs that act as antagonists of these hormone systems, binding to a receptor, but not activating the receptor's typical response, and preventing the binding or activity of the endogenous ligand. Finally, many EDCs bind to the receptor and trigger a response that is not necessarily the same as that triggered by the endogenous estrogens; these are termed selective ER modulators (SERMs). Ultimately, all of these actions occur at the level of the receptor.

Many studies have been dedicated to the understanding of which EDCs bind to which nuclear hormone receptors and how the binding affinities compare to the natural steroid. Thus, many of these chemicals have been classified as weak hormones. Yet studies have shown that, for example, the so-called weak estrogens like BPA can be equally potent as endogenous hormones in some systems, causing biological effects at picomolar levels (30, 38, 41, 121). Both endogenous estrogens and EDCs can bind to ER associated with the cell membrane [membrane-associated ER (mER)α and mERβ] that are identical to the nuclear ER (122–124), and a transmembrane ER called G-protein coupled receptor 30 that is structurally dissimilar to the nuclear ER and encoded by a distinct gene (125, 126). In many cells, 5–10% of total ERα and ERβ are localized to the plasma membrane (124); these membrane-associated receptors are capable of nongenomic steroid action in various cell types (30, 121, 127); thus, rapid and potent effects are well documented for many EDCs including BPA, DES, endosulfan, dichlorodiphenyldichloroethylene (DDE), dieldrin, and nonylphenol, among others (41, 128–130).

Finally, EDCs have other effects that are not dependent on binding to either classical or membrane-bound steroid hormone receptors. EDCs can influence the metabolism of natural hormones, thus producing differences in the amount of hormone that is available for binding either because more (or less) hormone is produced than in a typical system or because the hormone is degraded faster (or slower) than is normal. Other EDCs influence transport of hormone, which can also change the amount of hormone that is available for receptor binding. And EDCs can also have effects that are independent from known endocrine actions. One example is the effect of endogenous hormones and EDCs on ion channel activity. BPA, dichlorodiphenyltrichloroethane (DDT), DES, nonylphenol, and octylphenol have all been shown to disrupt Ca^{2+} channel activity and/or Ca^{2+} signaling in some cell types (131–134). This example illustrates how both natural hormones and EDCs can have hormonal activity via binding to nuclear hormone receptors but may also have unexpected effects via receptor-mediated actions outside of the classical endocrine system.

2. Mechanisms of EDC-induced low-dose actions

The various mechanisms by which EDCs act *in vitro* and *in vivo* provide evidence to explain how these chemicals induce effects that range from altered cellular function, to abnormal organ development, to atypical behaviors. Just as natural hormones display nonlinear relationships between hormone concentration and the number of bound receptors, as well as between the number of bound receptors and the maximal observable biological effect, EDCs obey these rules of binding kinetics (38). Thus, in a way, EDCs exploit the highly sensitive endocrine system and produce significant effects at relatively low doses.

To gain insight into the effects of natural hormones and EDCs on gene expression profiles, it is possible to calculate doses that produce the same effect on proliferation of cultured cells, *i.e.* the quantitative cellular response doses, and determine the effect of those doses on transcriptomal signature profiles. When this is done for estradiol and EDCs with estrogenic properties, the affected estrogen-sensitive genes are clearly different (135). However, an interesting pattern emerges: comparing profiles among only the phytoestrogens shows striking similarities in the genes up- and down-regulated by these compounds; profile comparisons between only the plastic-based estrogens also show similarities within this group. Yet even more remarkable is what occurs when the doses are selected not based on cell proliferation assays but instead on the ability of estradiol and estrogen-mimics to induce a single estrogen-sensitive marker gene. When doses were standardized based on marker gene expression, the transcriptomal signature profiles were very similar between estradiol and estrogen mimics (135). Taken together, these results suggest that the outcomes of these experiments are contextual to the normalization parameter and that marker gene expression and cell proliferation are not superimposable. This indicates that the biological level at which the effects of chemicals are examined (*i.e.* gene expression, cellular, tissue, organ, or organismal) can greatly impact whether low-dose effects are observed and how these effects are interpreted.

There are several other mechanisms by which low-dose activities have been proposed. One such possibility is that low doses of EDCs can influence the response of individuals or organs/systems within the body to natural hormones; thus, the exposed individual has an increased sensitivity to small changes in endogenous steroids, similar to the effects of intrauterine position (see Ref. 136 and *Section I.F*). In fact, several studies have shown that exposure to EDCs such as BPA during perinatal development can influence the response of the mammary gland to estrogen (137, 138) and the prostate to an estrogen-testosterone

mixture similar to the concentrations produced in aging men (139–142). There is also evidence that EDCs work additively or even synergistically with other chemicals and natural hormones in the body (143–145). Thus, it is plausible that some of the low-dose effects of an EDC are actually effects of that exogenous chemical plus the effects of endogenous hormone.

Finally, it should be noted that during early development, the rodent fetus is largely, but not completely (146), protected from estrogen via the binding activity of α-fetoprotein, a plasma protein produced in high levels by the fetal liver (147). Some estrogen-like EDCs, however, bind very weakly to α-fetoprotein, and therefore, it is likely that this protein does not provide protection to the fetus during these sensitive developmental periods (36, 148). Furthermore, because EDCs may not bind to α-fetoprotein or other high-affinity proteins in the blood (148–150) and can have a higher binding affinity to proteins like albumin (compared with natural estrogens) (36, 149), the balanced buffer system in place for endogenous hormones may be disturbed (Fig. 1A). Thus, whereas only a portion of endogenous hormones are bioavailable, the entirety of a circulating EDC could be physiologically active.

The effects of hormones and EDCs are dependent on dose, and importantly, low (physiological) doses can be more effective at altering some endpoints compared with high (toxicological) doses. There are many well-characterized mechanisms for these dose-specific effects including signaling via single *vs.* multiple steroid receptors due to nonselectivity at higher doses (30), receptor down-regulation at high doses *vs.* up-regulation at low doses (151, 152), differences in the receptors present in various tissues (153, 154), cytotoxicity at high doses (155), and tissue-specific components of the endocrine-relevant transcriptional apparatus (104, 105). Some of these factors will be addressed in *Section III.B* in the section dedicated to NMDRCs.

F. Intrauterine position and human twins: examples of natural low-dose effects

Hormones have drastically different effects at different periods of development. In a now classical *Endocrinology* paper, Phoenix and colleagues (156) showed that hormone exposures during early development, and in particular fetal development, had organizational effects on the individual, whereby the developing organs were permanently reorganized by exposure to steroids. Permanent, nonreversible masculinization of the developing body plan by androgen exposure *in utero* is an example. These organizational effects are in contrast to the effects of the same hormones, at similar or even higher doses, on adults. The effects of steroids on individuals after puberty have been termed activational, because the effects on target organs are typically transient; withdrawal of the hormone returns the phenotype of the individual to the preexposed state (157), although this is not always the case (158).

One of the most striking examples of the ability of low doses of hormones to influence a large repertoire of phenotypes is provided by the study of intrauterine positioning effects in rodents and other animals. The rodent uterus in particular, where each fetus is fixed in position along a bicornate uterus with respect to its neighbors, is an excellent model to study how hormones released from neighboring fetuses (159) can influence the development of endocrine-sensitive endpoints (31). Importantly, differences in hormonal exposures by intrauterine position are relatively small (see Fig. 2) (160). Thus, even a small magnitude in differences of hormonal exposures is sufficient to generate effects on behavior, physiology, and development.

The earliest studies of intrauterine position compared behavioral characteristics of females relative to their position in the uterus (161–164); male behavior was also affected by intrauterine position (161, 165–167). Subsequent studies of intrauterine position showed that position in the uterus influenced physiological endpoints (157, 160–162, 168–174) as well as morphological endpoints in female rodents (160, 161, 163, 164, 175–177). Male physiology and morphological endpoints were similarly affected by intrauterine position (165, 167, 177–179).

The endocrine milieu of the uterine environment has been implicated in these effects because differences in hormonal exposure have been observed based on intrauterine position (Fig. 2). The production of testosterone in male mice starting at approximately d 12 of gestation allows for passive transfer of this hormone to neighboring fetuses (159, 160, 180). Thus, fetuses positioned between two male neighbors have slightly higher testosterone exposures compared with fetuses positioned between one male and one female or two female neighbors (168, 181–183). These data indicate that very small differences in hormone exposures during fetal development are capable of influencing a variety of endpoints, many of which become apparent only during or after puberty. Furthermore, small differences in hormone exposures may be compounded by other genetic variations such as those normally seen in human populations.

Intrauterine effects have been observed in animals with both large litters and singleton or twin births including ferrets, pigs, hamsters, voles, sheep, cows, and goats (136, 184, 185). But perhaps the most compelling evidence for intrauterine effects comes from human twin studies. Many

Figure 2.

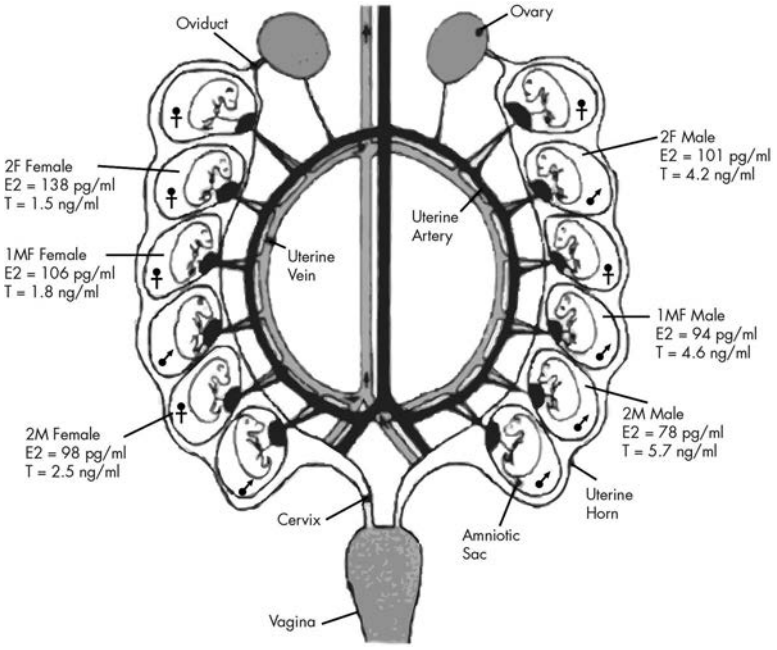

Figure 2. Intrauterine position produces offspring with variable circulating hormone levels. Fetuses are fixed in position in the bicornate rodent uterus, thus delivery via cesarean section has allowed for study of the influence of intrauterine position on behaviors, physiology, and organ morphology. Illustrated here are the differences in estradiol (E2) and testosterone (T) concentrations measured in male and female fetuses positioned between two male neighbors (2M), two female neighbors (2F), or neighbors of each sex (1MF). Direction of blood flow in the uterine artery (dark vessel) and vein (light vessel) is indicated by an *arrow* (159).

studies have found that the sex of the fetuses impacts the phenotype of one or more of the twins, with significant evidence suggesting that male twins strongly influence a female co-twin; endpoints including sensation seeking (186), ear superiority (187, 188), brain and cerebellum volume (189), masculine/feminine behaviors and aggression levels (190–192), handedness (193, 194), reproductive fitness (192, 195), finger length ratios (196), risk for developing eating disorders (197), and birth weight (198) were all affected in females with a male twin. From these studies, many authors have concluded that testosterone from male fetuses influences developmental parameters in female twins; typically, male same-sex twins do not display altered phenotypes for these endpoints. Yet importantly, limited studies indicate that female twins can influence their uterine pairs, with some behaviors affected in male co-twins (191); breast cancer incidence in women and testicular cancer in men have also been shown to be influenced by having a female co-twin (83, 199, 200).

Although the mechanisms for these intrauterine effects are not completely understood, very small differences in hormone exposures have been implicated, making the effects of twin gestations a natural example of low-dose phenomena. In the human fetus, the adrenals produce androgens that are converted to estrogen by the enzyme aromatase, specifically in the placenta. In a human study designed to compare hormone levels in the amniotic fluid, maternal serum, and umbilical cord blood of singleton male and female fetuses, significant differences were observed in the concentrations of testosterone, androstenedione (A4), and estradiol (201). Specifically, amniotic fluid concentrations of testosterone and A4 were approximately twice as high in male fetuses, whereas estradiol concentrations were slightly, but significantly, higher in female fetuses. Yet, interestingly, there were no differences for any of the hormones in maternal serum, similar to findings in mice that litters with a high proportion of males or females did not impact testosterone, estradiol, or progesterone serum levels in mothers (180). In umbilical cord serum, concentrations of A4 and estradiol were higher in males compared with females (201), although it must be noted that these samples were collected at parturition, long after the fetal period of sexual differentiation of the reproductive organs.

Several studies have specifically compared steroid hormone levels in maternal and umbilical cord blood samples collected from same-sex and opposite-sex twins. Male twins, whether their co-twin was a male or a female, had higher blood concentrations of progesterone and testosterone compared with female twins (202). Furthermore, for both sexes, dizygotic twins had higher levels of these hormones, as well as estradiol, compared with monozygotic twins. Fetal sex had no effect on maternal concentrations of testosterone, progesterone, or estrogen, suggesting that any differences observed in fetal samples are due to contributions from the fetuses' own endocrine systems and the placental tissue (203). Yet an additional study conducted in women carrying multiple fetuses (more than three) indicates that both estradiol and progesterone concentrations in maternal plasma increase with the number of fetuses, and when fetal reduction occurs, these hormone levels remain elevated (204).

It has been proposed that low-dose effects seen in different intrauterine positions in litter-bearing animals could be an evolutionary adaptation, whereby the genotypes of the fetuses are relatively similar but a range of phenotypes can be produced via differential hormone exposures (136, 168). For example, female mice positioned between two females are more docile and thus have better

reproductive success when resources are plentiful, but females positioned between two males are more aggressive and therefore are more successful breeders under stressful conditions (161, 171, 175). In this way, a mother produces offspring with variable responses to environmental conditions, increasing the chances that her own genetic material will continue to be passed on. Yet although there is evidence to suggest that a variable intrauterine environment is essential for normal development (171), intrauterine positional effects appear to have little effect on offspring phenotypes in inbred rodent strains (168, 205). This result may be related to the link between genetic diversity and hormone sensitivity (206, 207), suggesting that outbred strains are the most appropriate for studying endocrine endpoints and are also most similar to the effects of low doses of hormones on human fetuses.

Finally, it has been proposed that similar mechanisms are used by the developing fetus in response to natural hormones via intrauterine position and EDCs with hormonal activity (136). To this end, several studies have examined the effects of both exposure to an EDC and intrauterine position or have considered the effect of intrauterine position on the response of animals to these chemicals (174, 176, 181, 208, 209). For example, one study found that intrauterine position affected the morphology of the fetal mammary gland, yet position-specific differences were obliterated by BPA exposure (176). Additional studies suggest that prostate morphology is disrupted by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) exposure in males positioned between two females, but this chemical does not affect prostate morphology in males positioned between two males (181). Finally, male rodents positioned between two males have higher glucose intolerance than males positioned between two females, yet when these males are given a diet high in phytoestrogens, glucose tolerance is dramatically improved in the males positioned between two males, whereas their siblings positioned between two females do not benefit (209). What is clear from these studies is that low doses of natural hormones are capable of altering organ morphology, physiology, and reproductive development, similar to the effects of EDCs.

It has been suggested that the endocrine system allows for homeostatic control and that the aim of the endocrine system is to "maintain normal functions and development in the face of a constantly changing environment" (210). Yet studies from intrauterine position, together with studies of EDCs (see *Sections II.C–F*), clearly indicate that the fetal endocrine system cannot maintain a so-called homeostasis and is instead permanently affected by exposures to low doses of hormones.

II. Demonstrating Low-Dose Effects Using a WoE Approach

A. Use of a WoE approach in low-dose EDC studies

In 2001, the NTP acknowledged that there was evidence to support low-dose effects of DES, genistein, methoxychlor, and nonylphenol (2). Specifically, the NTP expert panel found that there was sufficient evidence for low-dose effects of DES on prostate size; genistein on brain sexual dimorphisms, male mammary gland development, and immune responses; methoxychlor on the immune system; and nonylphenol on brain sexual dimorphisms, thymus weight, estrous cyclicity, and immune responses. Using the NTP's definitions of low dose (*i.e.* effects occurring in the range of typical human exposures or occurring at doses lower than those typically used in standard testing protocols), we propose that most if not all EDCs are likely to have low-dose effects. Yet an important caveat of that statement is that low-dose effects are expected for particular endpoints depending on the endocrine activity of the EDC, and not for any/all endocrine-related endpoints. For example, if a chemical blocks the synthesis of a hormone, blood levels of the hormone are expected to decline, and the downstream effects should then be predicted from what is known about the health effects of low hormone levels. In contrast, if a chemical binds a hormone receptor, the effects are expected to be very complex and to be both tissue specific and dose specific. Finally, most EDCs interact with multiple hormone pathways, or even multiple hormone receptors, making the expected effects even more complex and context specific (211–213).

Table 3 summarizes a limited selection of chemicals that have evidence for low-dose effects, with a focus on *in vivo* animal studies. As seen by the results presented in this table, low-dose effects have been observed in chemicals from a number of classes with a wide range of uses including natural and synthetic hormones, insecticides, fungicides, herbicides, plastics, UV protection, and other industrial processes. Furthermore, low-dose effects have been observed in chemicals that target a number of endocrine endpoints including many that act as estrogens and antiandrogens as well as others that affect the metabolism, secretion, or synthesis of a number of hormones. It is also clear from this table that the cutoff for low-dose effects is not only chemical specific but also can be effect dependent. And finally, although this table is by no means comprehensive for all EDCs or even the low-dose effects of any particular chemical, the affected endpoints cover a large range of endocrine targets.

Several EDCs have been well studied, and the number of publications focusing on low-dose effects on a particular developmental endpoint is high; however, other

TABLE 3. EDCs with reported low-dose effects in animals (or humans, where stated)

Chemical	Use	EDC action	Low-dose cutoff	Affected endpoint	Refs.
Aroclor 1221 (PCB mixture)	Coolants, lubricants, paints, plastics	Mimics estrogens, antiestrogenic activity, *etc.*	0.1–1 mg/kg (produces human blood levels)	Brain sexual dimorphisms	683, 684
Atrazine	Herbicide	Increases aromatase expression	200 μg/liter (334, 335)	Male sexual differentiation/development	See this review
BPA	Plastics, thermal papers, epoxy resins	Binds ER, mER, ERRγ, PPARγ, may weakly bind TH receptor and AR	400 μg/kg · d (produces human blood concentrations)	Prostate, mammary gland, brain development and behavior, reproduction, immune system, metabolism	See this review
Chlordane	Insecticide	Binds ER	100 ng/g (produces human blood levels)	Sexually dimorphic behavior	685
Chlorothalonil	Fungicide, wood protectant	Aromatase inhibitor	164 μg/liter (environmental concentrations, EPA)	Corticosterone levels (amphibians)	686
Chlorpyrifos	Insecticide	Antiandrogenic	1 mg/kg · d (EPA)	Acetylcholine receptor binding (brain)	687
DDT	Insecticide	Binds ER	0.05 mg/kg (EPA)	Neurobehavior	688
DES	Synthetic hormone	Binds ER	0.3–1.3 mg/kg · d (dose typically administered to pregnant women)	Prostate weight	689
Dioxin (TCDD)	Industrial byproduct	Binds AhR	1 μg/kg · d (397)	Spermatogenesis, immune function and oxidative stress, tooth and bone development, female reproduction, mammary gland, behavior	See this review
Genistein	Phytoestrogen	Binds ER	50 mg/kg (EPA)	Brain sexual dimorphisms	690
Heptachlor	Insecticide	Induces testosterone hydroxylases	0.15 mg/kg · d (EPA)	Immune responses	691
Hexachlorobenzene	Fungicide	Modulates binding of ligand to TRE, weakly binds AhR	0.08 mg/kg · d (EPA)	Anxiety and aggressive behaviors	692
Maneb	Fungicide	Inhibits TSH release, may bind PPARγ	5 mg/kg · d (EU Commission)	Testosterone release	693
Methoxychlor	Insecticide	Binds ER	5 mg/kg · d (WHO)	Immune system	694, 695
4-Methylbenzylidine camphor	UV screen	Weakly estrogenic	10 mg/kg · d (Europa)	Sexual behavior	696
Methyl paraben	Preservative	Estrogenic	1000 mg/kg · d (EFSA)	Uterine tissue organization	697
Nicotine	Natural alkaloid in tobacco	Binds acetylcholine receptors, stimulates epinephrine	Human use of nicotine substitutes	Incidence of cryptorchidism (humans)	698
Nonylphenol	Detergents	Weakly estrogenic	15 mg/kg · d (EPA)	Testosterone metabolism	699
Octylphenol	Rubber bonding, surfactant	Weakly binds ER, RXR, PRGR	10 mg/kg · d (700)	Testes endpoints	701
Parathion	Insecticide		0.2 mg/kg · d (WHO)	Cognitive and emotional behaviors	702
PBDE-99	Flame retardant	Alters TH synthesis	0.3 mg/kg · d (EPA)	TH levels in blood	703
PCB180	Industrial lubricant, coolant	Impairs glutamate pathways, mimics estrogen	Examined normal human populations	Diabetes (humans)	704
PCB mixtures	Coolants, lubricants, paints, plastics	Binds AhR, mimic estrogens, antiestrogenic activity, etc.	Each at environmentally relevant levels	TH levels	705
Perchlorate	Fuel, fireworks	Blocks iodide uptake, alters TH	0.4 mg/kg · d (436)	TSH levels (humans)	See this review
Sodium fluoride	Water additive (to prevent dental caries), cleaning agent	Inhibits insulin secretion, PTH, TH	4 mg/liter water (EPA standard)	Bone mass and strength	706
Tributyltin oxide	Pesticide, wood preservation	Binds PPARγ	0.19 mg/kg · d (EPA)	Obesity	707
Triclosan	Antibacterial agent	Antithyroid effects, androgenic and estrogenic activity	12 mg/kg · d (Europe SCCP)	Altered uterine responses to ethinyl estradiol	708
Vinclozolin	Fungicide	Antiandrogenic	1.2 mg/kg · d (EPA)	Male fertility	709

EDC action indicates that for some chemicals, an effect is observed (*i.e.* estrogenic, androgenic), but for many EDCs, complete details of receptor binding are unavailable or incomplete. Low-dose cutoff means the lowest dose tested in traditional toxicology studies, or doses in the range of human exposure, depending on the data available. Affected endpoint means at least one example of an endpoint that shows significant effects below the low-dose cutoff dose. This list is not comprehensive, and the lack of an endpoint on this table does not suggest that low doses do or do not affect any other endpoints. AR, Androgen receptor; EFSA, European Food Safety Authority; ERR, estrogen related receptor; PCB, polychlorinated biphenyl; PPARγ, peroxisome proliferator-activated receptor-γ; PRGR, progesterone receptor; RXR, retinoid X receptor; SCCP, Scientific Committee on Consumer Products; TH, thyroid hormone; TRE, thyroid response element; WHO, World Health Organization.

chemicals are less well studied with fewer studies pointing to definitive low-dose effects on a given endpoint. In fact, there are a significant number of EDCs for which high-dose toxicology testing has been performed and the no observed adverse effect level (NOAEL) has been derived, but no animal studies in the low-dose range have been conducted, and several hundred additional EDCs where no significant high- or low-dose testing has been performed (see Table 4 for examples). Balancing the large amount of data collected from some well-studied chemicals like BPA and atrazine with the relative paucity of data about other chemicals is a difficult task.

TABLE 4. Select examples of EDCs whose potential low-dose effects on animals remain to be studied

Chemical	Use	EDC action	Low-dose cutoff
Antiseptics and preservatives			
Butyl paraben	Preservative (cosmetics)	Estrogenic, antiandrogenic	2 mg/kg · d (EPA)
Propyl paraben	Antimicrobial preservative found in pharmaceuticals, foods, cosmetics, and shampoos	Estrogenic activity	LOAEL 10 mg/kg · d, NOEL 6.5 mg/kg · d (Europa)
Cosmetics and personal care products			
2,4-Dihydroxybenzophenone	UV absorber in polymers, sunscreen agent	Estrogenic activity	Not identified
3-Benzylidene camphor	UV blocker used in personal care products	Estrogenic activity	0.07 mg/kg · d (710)
4,4′-Dihydroxybenzophenone	UV light stabilizer used in plastics, cosmetics, adhesives, and optical fiber	Estrogenic activity	Not identified
Benzophenone-2	Used in personal care products such as aftershave and fragrances	Estrogenic activity, changes in T_4, T_3, and TSH levels, alterations in cholesterol profile	NOEL 10–333 mg/kg · d (711)
Benzophenone-3	UV filter	Estrogenic, PPARγ activator	200 mg/kg · d (Europa)
Multiple use (other)			
Melamine	Flame-retardant additive and rust remover; used to make laminate, textile, and paper resins; metabolite of cyromazine	Affects voltage-gated K^+ and Na^+ channels and Ca^{2+} concentrations in hippocampal neurons	63.0 mg/kg · d (FDA)
Resorcinol	Used in the manufacturing of cosmetics, dyes, flame retardants, hair dye formulations, pharmaceuticals, skin creams, and tires	Alters T_4 and TSH levels	80.00 mg/kg · d (Europa)
Pesticides			
Aldrin[a]	Insecticide	Estrogenic activity	0.025 mg/kg · d (Health Canada)
Alachlor	Herbicide	Decreases serum T_4, binds PR, weakly binds ER	1 mg/kg · d (EPA)
Amitrole	Herbicide	Decreases thyroid hormone	0.12 mg/kg · d (FAO)
Bitertanol	Fungicide	Alters aromatase	30 mg/kg · d (EPA)
Carbendazim	Fungicide	Affects FSH, LH, and testosterone levels; alters spermatogenesis and Sertoli cell morphology	8 mg/kg · d (712)
Diazinon	Insecticide	Alters glucocorticoids	0.065 mg/kg · d (CDC)
Endrin[a]	Insecticide	Stimulates glucocorticoid receptor	0.025 mg/kg · d (CDC)
Fenoxycarb	Insecticide	Alters acetylcholinesterase	260 mg/kg · d (CDC)
Mirex[a]	Insecticide	Decreases testosterone levels	0.075 mg/kg · d (CDC)
Zineb	Fungicide	Alters T_4 and dopamine levels	LOAEL 25 mg/kg · d (EPA)
Ziram	Fungicide	Alters norepinephrine levels	1.6 mg/kg · d (EPA)
Resins			
Bisphenol F	Used in polycarbonates	Alters T_4, T_3, and adiponectin levels, has estrogenic activity	LOAEL 20 mg/kg · d (713)
Styrene	Precursor to polystyrene	Alters dopamine	200 mg/kg · d (EPA)

PPARγ, peroxisome proliferator-activated receptor-γ; PR, progesterone receptor.

[a] These chemicals were identified in the 1990s as part of the dirty dozen, 12 chemicals that were acknowledged to be the worst chemical offenders because of their persistence in the environment, their ability to accumulate through the food chain, and concerns about adverse effects of exposures to wildlife and humans. These chemicals were banned by the Stockholm convention and slated for virtual elimination. Yet there is still very little known about the low-dose effects of these chemicals, likely in the range of past and current human and/or wildlife exposures.

WoE approaches have been used in a large number of fields to determine whether the strength of many publications viewed as a whole can provide stronger conclusions than any single study examined alone. Although the term 'weight of evidence' is used in public policy and the scientific literature, there is surprisingly little consensus about what this term means or how to characterize the concept (214). Historically, risk assessors have used qualitative ap-

proaches (*i.e.* professional judgment to rank the value of different cases) and quantitative approaches (*i.e.* scoring methods to produce statistical and mathematical determinations of chemical safety), but it has been argued that these methods lack transparency and may produce findings that are unrepeatable from one risk assessor to another (215, 216). Whatever the method used, when EDCs are being assessed, it is important to use the principles of endocrinology to establish the criteria for a WoE approach. We do this in *Section II.B*, identifying three key criteria for determining whether a study reporting no effect should be incorporated into a WoE approach. It also should be noted that in epidemiology, the term 'weight of evidence' is typically not used, but the concept is actuated by meta-analysis, formally and quantitatively combining data across studies, including a plot of individual and pooled study findings and also a measure of heterogeneity of findings between studies.

For some well-studied chemicals, there are large numbers of studies showing both significant effects, and additional studies showing no effects, from low-dose exposures. In these cases, extensive work is needed to deal with discordant data collected from various sources; studies showing no effect of low-dose exposures must be balanced in some way with those studies that do show effects. As stated by Basketter and colleagues (217), "it is unwise to make a definitive assessment from any single piece of information as no individual assay or other assessment . . . is 100% accurate on every occasion . . . This means that from time to time, one piece of conflicting data has to be set aside." WoE approaches in EDC research have typically dealt with datasets that have some conflicting studies, and these conflicts are even more difficult to sort out when studies have attempted to directly replicate published findings of adverse effects (see for example Refs. 218–221).

Most previously published WoE analyses have examined chemicals broadly (asking questions such as, "Does BPA produce consistent adverse effects on any endpoint?") (see Ref. 222). This can lead to problems including those encountered by the NTP expert panel, which found that there was some evidence for low-dose effects of BPA on certain endpoints but mixed findings for other endpoints. For example, the panel noted that some studies found low-dose effects of BPA on the prostate, but other studies could not replicate these findings. In *Section II.B*, we address criteria that are needed to accept those studies that are unable to detect low-dose effects of chemicals; these criteria were not used by the NTP in 2001, but they are essential to address controversies of this sort and perform WoE analyses using the best available data. In the sections that follow, we employed a WoE approach to examine the evidence for low-dose effects of single chemicals on selected endpoints or tissues, also paying attention to when in development the EDCs in question were administered.

B. Refuting low-dose studies: criteria required for acceptance of studies that find no effect

Over the past decade, a variety of factors have been identified as features that influence the acceptance of low-dose studies (69, 71, 76, 77, 90, 205, 223, 224). In fact, the NTP low-dose panel itself suggested that factors such as strain differences, diet, caging and housing conditions, and seasonal variation can affect the ability to detect low-dose effects in controlled studies (2). In particular, three factors have been identified; when studies are unable to detect low-dose effects, these factors must be considered before coming to the conclusion that no such effects exist.

1. Negative controls confirm that the experimental system is free from contamination

Although all scientific experiments should include negative (untreated) controls, this treatment category is particularly important for EDC research. When a study fails to detect low-dose effects, the observed response in control animals should be compared with historical untreated controls; if the controls deviate significantly from typical controls in other studies, it may indicate that these animals were, in fact, treated or contaminated in some way or that the endpoint was not appropriately assessed (77, 205, 225). For example, if an experiment was designed to measure the effect of a chemical on uterine weight, and the control uteri have weights that are significantly higher than is normally observed in the same species and strain, these animals may have been inadvertently exposed to an estrogen source, or the uteri may not have been dissected properly by the experimenters. In either case, the study should be examined carefully and likely cannot be used to assess low-dose effects; of course, untreated controls should be monitored constantly because genetic drift and changes in diet and housing conditions can also influence these data, thus explaining changes from historical controls. Importantly, several types of contamination have been identified in studies of EDCs including the leaching of chemicals from caging or other environmental sources (226, 227), the use of pesticide-contaminated control sites for wildlife studies and contaminated controls in laboratory studies (76), and even the use of food that interferes with the effects of EDCs (224, 228). It is also important to note that experiments must consider the solvent used in the administration of their test chemical, and thus good negative controls should test for effects of the solvent itself. Using solvent negative controls helps prevent false posi-

tives as well as the possibility that the vehicle could mask the effects of the chemical being studied.

2. Positive controls indicate that the experimental system is capable of responding to low doses of a chemical acting on the same pathway

Many studies do not include a positive control, either because of the size and cost of the experiment when including an additional treatment or because an appropriate positive control has not been identified for the endpoint being examined. If the experiment detects an effect of the chemical in question, the exclusion of a positive control does not necessarily affect the interpretation of the results; instead, it can be appropriately concluded that the test chemical is significantly different from unexposed (but similarly handled/treated) negative controls. However, if the study fails to detect low-dose effects of a test chemical, no convincing conclusion can be made; in this case, a positive control is required to demonstrate that the experimental system was capable of detecting such effects (71, 75, 77, 205).

Several issues must be considered when addressing whether the positive control confirms the sensitivity of the assay. First, an appropriate chemical must be selected, and it must be administered via the appropriate route, *i.e.* if the test chemical is administered orally, a positive control that is orally active, such as ethinyl estradiol, should be used; if the test chemical is administered sc, a positive control that is active via this route, such as 17β-estradiol, is most appropriate. The use of 17β-estradiol in studies that use oral exposures is particularly inappropriate (see Ref. 229) for example) because this hormone, like most natural steroids, has very low oral activity (77). Second, the positive control chemical must be examined, and effective, at appropriately low doses. Thus, if the test chemical is 100 times less potent than the positive control, a dose of the positive control 100 times lower than the test compound must produce effects (69, 71, 205). For example, studies that report effects of ethinyl estradiol only at doses that are hundreds of times higher than the dose that is effective in contraceptives (230) are not capable of detecting low-dose effects of test chemicals. Without appropriate and concurrent positive and negative controls, studies that fail to detect low-dose effects of test chemicals should be rejected.

3. Species and animal strains that are responsive to EDCs must be used

The NTP expert panel specifically noted that "because of clear species and strain differences in sensitivity, animal-model selection should be based on responsiveness to endocrine-active agents of concern (*i.e.* responsive to positive controls), not on convenience and familiarity" (2). An analysis of the BPA literature clearly showed that many of the studies that failed to detect effects of low doses used the Charles River Sprague-Dawley rat (75); this strain was specifically bred to have large litters (231), and many generations of inbreeding have rendered the animal relatively insensitive to estrogens (205). The NTP expert panel noted the lack of effects of BPA on Sprague-Dawley rats and concluded that there were clear differences in strain sensitivity to this chemical (2). Importantly, this may not be true for Sprague-Dawley rats that originate from other vendors, indicating that animal origin can also influence EDC testing.

Many studies in mice (138, 206, 207, 232–234) and rats (232, 235–239) have described differences displayed between two (or more) animal strains to a natural hormone or EDC. Often these differences can be traced to whether a strain is inbred or outbred. Genetically diverse strains are generally found to be more sensitive to estrogens (206). Importantly, well-controlled studies demonstrate that strain differences in response to estrogen treatment may be organ dependent or may even differ between levels of tissue organization within the same organ. For example, the Sprague-Dawley rat is more sensitive to ethinyl estradiol than other strains when measured by uterine wet weight. However, when other endpoints were measured, *i.e.* height of cells in the uterine epithelium, the Sprague-Dawley rat was indistinguishable from the DA/Han rat; instead, the Wistar rat had the most heightened response (237). Additionally, there are data to indicate that strain differences for one estrogen may not be applicable for all estrogenic chemicals. In comparing the responses of DA/Han, Sprague-Dawley, and Wistar rats to other xenoestrogens, additional differences were observed including a greater increase in uterine wet weight of DA/Han and Sprague-Dawley rats but not Wistar rats after exposure to 200 mg/kg BPA; increased uterine epithelium thickness was observed in Wistar and Sprague-Dawley rats but not DA/Han rats after exposure to 200 mg/kg octylphenol (237). Attempts have been made, at times successfully, to map the differences in strain response to genetic loci (240). However, it appears that strains with differences in response that manifest in some organs do not have divergent responses in other organs, a phenomenon that is not explained by genetic differences alone. For these reasons, the NTP's recommendation that scientists use animals that are proven responsive to EDCs (2) must be observed.

4. Additional factors?

Additional factors have also been identified as influential in the ability (or inability) to detect low-dose effects in

EDC studies. Although these factors must be considered when interpreting studies and using a WoE approach, some issues that were previously identified as essential factors in the design of studies (*i.e.* route of administration) have more recently been disputed (241).

The first factor is the use of good laboratory practices (GLP) in the collection of data. When assessing the EDC literature for risk assessment purposes, the FDA and European Food Safety Authority (EFSA) have given special prominence to studies that complied with GLP guidelines, essentially giving scientific priority to industry-funded studies because that group typically conducts GLP guideline studies (33, 242). Because GLP guidelines are designed only to control data collection, standards for animal care, equipment, and facility maintenance, and they do not ensure that studies were designed properly with the appropriate controls, it has been argued that the use of GLP methods is not appropriate or required for EDC studies (69).

GLP studies are typically large, with dozens of animals studied for each endpoint and at each time point. Thus, it has been concluded that these studies are better simply because they are larger. Yet small studies designed with the use of power analysis, statistical tools that allow researchers to determine *a priori* the number of animals needed to determine significant differences based on effect size, are equally capable of detecting effects while reducing the number of animals used (69). GLP studies also typically (but not necessarily) rely upon standardized assays, which are not generally considered contemporary tools and are often shown to be incapable of detecting adverse effects on endpoints that employ modern tools from molecular genetics and related disciplines. Furthermore, some fields of EDC research have no GLP studies (243). Finally, there is no published evaluation of whether studies performed under GLP are more capable of providing accurate results. The priority given to GLP studies therefore does not appear to have been justified based on any comparative analysis. Thus, as long as studies include appropriate measures of quality assurance, they need not be performed under GLP standards to provide reliable and valuable information, and many GLP studies are inadequate to assess important and relevant endpoints. Instead, the most valuable studies consider the factors presented above, along with appropriate dose selections and choice of endpoint.

The second factor worth considering is the source of funding for studies. In several fields, significant controversy has been produced based on the results obtained from independent scientists compared with results obtained from scientists affiliated with the chemical industry (75, 76). Funding source *per se* should not dictate the outcome of a research study, but that does not mean that researchers are not subject to underlying biases. In our own WoE analyses, presented in *Sections II.C–G*, we do not discount studies merely because they were conducted with industry funds, nor do we lend higher weight to studies conducted in independent or government laboratories; if a study, regardless of funding, finds no effect of a chemical, it is given weight only if the three criteria described in *Sections II.B.1–3* (successful and appropriate negative and positive controls and appropriate choice of animal model) were met.

To perform a WoE evaluation, we identified some basic information about the chemical in question, the dose that would be considered a low-dose cutoff, and the studies in support of and against low-dose effects. We then considered whether the majority of studies found effects of low doses of a chemical on a single endpoint in question. If studies did not find low-dose effects, we considered whether they adhered to the criteria discussed above for proper design of an EDC low-dose study. In particular, we considered whether appropriate animal strains as well as positive and negative controls were used. With regard to animal strain, as discussed briefly in *Section II.B.3*, there is variability between animal strains that can significantly influence the ability to detect effects of EDCs; using insensitive strains to produce negative data cannot refute positive data in a sensitive strain. In several cases, it was easy to conclude that there was a strong case for low-dose effects because there were no studies finding no effects at low doses or because all of the negative studies were inappropriately designed. For other chemicals, a significant number of studies found effects on the endpoint being considered, but other (adequately designed) studies refuted those findings. Under those circumstances, we determined whether the findings of harmful effects came from multiple laboratories; when they did, we cautiously concluded that there was evidence for low-dose effects. Below (*Sections II.C–G*), we present five examples where a significant number of studies were available examining low-dose effects of an EDC on a single particular endpoint.

C. BPA and the prostate: contested effects at low doses?

As discussed briefly above, BPA is one of the best-studied EDCs, with more than 200 published animal studies, many of which focused on low doses (29, 31). The effects of this chemical on wildlife species have also been described in detail (28). BPA is found in a myriad of consumer products, and it leaches from these items under normal conditions of use (4). It has also been regularly detected in air, water, and dust samples. The majority of individuals in industrialized countries have BPA metabolites in their urine, and trends indicate increasing expo-

sures in developing nations like China (87, 244). Although it was long suspected that most human exposures originate from BPA contamination of food and beverages, a study comparing the excretion of BPA metabolites with the length of time spent fasting suggests that there are also likely to be significant exposures from sources other than food and beverages (245). BPA has recently been shown to be used in large quantities in thermal and recycled papers and can enter the skin easily via dermal absorption (246–248). Thus, despite the large amount of information available on BPA sources, our understanding of how these sources contribute to total human exposures remains poor; these studies also point to significant gaps in current knowledge about BPA metabolism in humans (243).

BPA binds to the nuclear and membrane ER, and thus most of the effects of this chemical have been attributed to its estrogenic activity (27). However, there is evidence that it can activate a number of additional pathways, including thyroid hormone receptor, androgen receptor, as well as peroxisome proliferator-activated receptor-γ signaling pathways (249–252). The cutoff for a low dose has been set at several different concentrations depending on which studies and definitions are used (see Table 1). The EPA calculated a reference dose for BPA of 50 μg/kg · d based on a LOAEL of 50 mg/kg · d (38). More recent pharmacokinetic scaling experiments have estimated that exposures to approximately 400 μg/kg · d produce blood concentrations of unconjugated BPA in the range of human blood concentrations (4). Thus, for the two WoE analyses of the BPA literature we conducted, doses of 400 μg/kg · d or lower were considered low dose; pharmacokinetic studies from nonhuman primates support the appropriateness of this dose for approximating human exposure levels (253). Furthermore, because this dose is below the toxicological LOAEL, it is a conservative cutoff for low-dose studies (see Refs. 3 and 38 and Table 1).

One of the most well studied and hotly debated examples of a low-dose effect comes from the BPA literature; regulatory agencies and scientists have addressed several times whether low doses of BPA during fetal and perinatal development affect the rodent prostate (118, 205, 254, 255). In 1997, the first study on BPA and the prostate determined that fetal exposure to low doses (2 and 20 μg/kg · d administered orally to pregnant mice) increased the weight of the adult prostate compared with unexposed male offspring (256). Since that time, several additional studies have verified that prostate weight is affected by fetal exposure to similar low doses (257–259). Studies have also shown that low doses of BPA affect androgen receptor binding activity in the prostate (257), tissue organization, and cytokeratin expression in the gland (260–262) as well as the volume of the prostate and the number and size of dorsolateral prostate ducts (208). Several recent studies have also examined whether low doses of BPA (10 μg/kg · d) influence the incidence of adult-onset prostatic intraepithelial neoplasia (PIN) lesions. Perinatal BPA exposure, whether administered orally or sc to pups, increases the incidence of PIN lesions in response to a mixture of testosterone and estradiol in adulthood (139, 141, 263); this hormonal cocktail was designed to mimic the endocrine changes associated with aging in men that also typically accompany the onset of prostate cancer. In addition to the effects of BPA on PIN lesions, these low doses also produced permanent alterations in the epigenome of exposed males, with prostates displaying completely unmethylated sequences in genes that are hypermethylated in unexposed controls (140, 263). In examining these studies, although the same effects of BPA on the prostate were not observed in all studies, there is an obvious trend demonstrating that low doses of BPA during early development significantly affect several aspects of prostate development.

Since the initial report showing effects of low doses on the prostate, approximately nine studies, including several designed specifically to replicate the original positive study, have shown no effects of low doses on the prostate (264–272); every one of these studies examined the prostate weight, and Ichihara *et al.* (264) also examined the effects of BPA on PIN lesions (without hormonal treatment) and the response of the prostate to a chemical carcinogen. Three of these studies failed to include a positive control of any kind (264, 268, 270); three studies used DES as a positive control but found no effect from exposure to this potent xenoestrogen (265–267) (*i.e.* the positive control failed); another study used 17β-estradiol as a positive control, inappropriately administered orally, and found no effects of this hormone on the prostate (271); and two studies used an estrogenic positive control (ethinyl estradiol) and found effects from its exposure, but only at inappropriately high doses (269, 272). These two studies clearly showed that the positive control dose was too high, because rather than increase the weight of the prostate (as seen after low doses of estrogens in other studies), the positive control decreased the weight of the adult prostate (269, 272).

Although this topic was once considered controversial, using a WoE approach, it is clear that there is strong evidence in support of low-dose effects of BPA on the development of the prostate. The evidence clearly shows that several endpoints, including prostate weight, were affected in similar ways in multiple studies from several different labs at doses below 400 μg/kg · d; most effects were seen at doses below 50 μg/kg · d. Furthermore, PIN lesions were reported after neonatal exposure to 10 μg/kg · d with

hormonal treatment in adulthood. No appropriately conducted studies contest this evidence. Therefore, the WoE analysis demonstrates that low doses of BPA significantly alter development of the rodent prostate. The NTP's review of the BPA literature in 2008 indicated that this agency agrees that there is now significant evidence that low-dose BPA adversely affects development of the prostate (273).

D. BPA and the mammary gland: undisputed evidence for low-dose effects

The mammary gland is a conspicuous choice to examine the effects of estrogenic compounds because this organ depends on estrogen for proper development at several critical periods in life (274). The fetal gland expresses ER in the mesenchymal compartment, and just before birth, the epithelium becomes ER positive as well (275). At puberty, estrogen is responsible for ductal elongation and overall development of the gland, allowing the epithelium to fill the stromal compartment in preparation for pregnancy and lactation. Although BPA is an example of a chemical that has been classified as a weak estrogen because it binds with a much lower affinity to ERα compared with 17β-estradiol, even weak estrogens are known to affect the development of the mammary gland during early development (276).

In the first study to examine the effects of BPA on the mammary gland, prepubertal rats were exposed to relatively high doses (100 μg/kg · d or 54 mg/kg · d) for 11 d. After even this short exposure, mammary gland architecture was affected in both dose groups, with increased numbers of epithelial structures and, in particular, structures that suggest advanced development (277). BPA exposure also altered proliferation rates of mammary epithelium and cell cycle kinetics, with an increased number of cells in S-phase and a decreased number of cells in G1. Although relatively high doses of BPA were examined, this initial study indicated that the prepubertal and pubertal gland could be sensitive to BPA.

Many additional studies have examined another critical period, the fetal and neonatal periods, which are sensitive to environmental estrogens (78, 276, 278). Mice exposed prenatally to low doses of BPA via maternal treatment (0.25 μg/kg · d) displayed altered development of both the stromal and epithelial compartments at embryonic d 18, suggesting that exposures affect tissue organization during the period of exposure (176). In addition, similar low doses produced alterations in tissue organization observed in puberty and throughout adulthood, long after exposures ended, and even induced pregnancy-like phenotypes in virgin females (137, 279–282). Female mice exposed to BPA *in utero* displayed heightened responses to estradiol at puberty, with altered morphology of their glands compared with animals exposed to vehicle *in utero* (138). Another study demonstrated that perinatal BPA exposure altered the mammary gland's response to progesterone (283). Remarkably, all of these effects were observed after maternal exposures to low doses (0.025–250 μg/kg), suggesting that the gland is extremely sensitive to xenoestrogen exposures. These studies are in contrast to one that examined the effects of higher doses (0.5 and 10 mg/kg · d) when BPA was administered for 4 d to the dam, which reported advanced development of BPA-exposed glands before puberty but no effects in adulthood (284).

Adult exposure to BPA is only now being examined in the mouse mammary gland model. A recent study examined the effects of BPA on mice with mutations in the *BRCA1* gene. This study reported that 4 wks of exposure to a low dose of BPA altered the tissue organization of the mammary gland in ways that are similar to the effects observed after perinatal exposure (285). This study focused on altered development of the gland during exposure; additional studies are needed to determine whether these effects are permanent or whether normal mammary morphology could be achieved by cessation of BPA exposure.

Another obvious endpoint is the effect of BPA exposure on mammary cancer incidence. Several studies indicate that exposure to BPA *in utero* produces preneoplastic (281, 286, 287) and neoplastic lesions (286) in the gland in the absence of any other treatment. Additionally, other studies show that females exposed to BPA during the perinatal period are more sensitive to mammary carcinogens, decreasing tumor latency and increasing tumor incidence (287–290). These studies are also supported by subsequent studies examining gene and protein expression, which show that low-dose BPA specifically up-regulates expression of genes related to immune function, cell proliferation, cytoskeletal function, and estrogen signaling and down-regulates apoptotic genes (282, 288, 289, 291).

Postnatal BPA exposures also influence mammary cancer incidence; animals exposed lactationally to BPA from postnatal d 2 until weaning displayed decreased tumor latency and increased tumor multiplicity after treatment with DMBA [7,12-dimethylbenz(a)anthracene], a carcinogen (292). This study suggested that BPA exposure led to increased cell proliferation and decreased apoptosis in the gland and shifted the period where the gland is most susceptible to mammary carcinogens, a result that has important implications for human breast cancer. Finally, an additional study examined the effects of adult BPA exposure on mammary cancer; this study demonstrated that low doses of BPA accelerate the appearance of mammary tumors in a tumor-prone mouse strain (293). Interestingly,

high doses did not have this effect; thus, this study is also an excellent example of a NMDRC.

Two studies of BPA and the mammary gland seem to contradict this body of literature, but both examined extremely high doses. In the first study, Nikaido *et al.* (294) exposed female mice to 10 mg/kg BPA from postnatal d 15–18. Mammary glands from these animals were examined at 4, 8, and 24 wk of age, and no differences were observed in the exposed animals relative to controls. Although the lack of effects reported in this study could be due to the high dose employed, they could also be related to the relatively short exposure period during the preweaning phase. In the second study, Yin and colleagues (295) examined the effects of BPA during the first few days after birth (0.1 or 10 mg BPA, equivalent to approximately 10 and 1000 mg/kg) on the incidence of mammary tumors after exposure to a mammary carcinogen at puberty. Similar to the study described above, this one also examined the effects of BPA after a relatively short period of exposure (only three injections administered between postnatal d 2 and 6). Although the study showed that BPA affected tissue organization, there was no change in the incidence of tumors in BPA-exposed females. Because both of these studies examined both high doses and relatively short periods of exposure, it is difficult to compare them directly to the studies finding effects of BPA on the mammary gland after longer exposures to lower doses; at the very least, they cannot refute studies suggesting that BPA alters development of this gland.

In summary, the WoE clearly shows that low-dose BPA exposure affects development of the mammary gland, mammary histogenesis, gene and protein expression in the gland, and the development of mammary cancers. In fact, this example of low-dose effects produced remarkably similar effects across more than a dozen studies conducted in several different labs. These results are also consistent with the effects of low-dose BPA exposure on mammary epithelial cells in culture (reviewed in Ref. 30). Although epidemiology studies examining the influence of BPA on breast cancer rates have proven to be inconclusive at best (296), to replicate the animal studies discussed above, epidemiologists must collect information about prenatal and neonatal exposures and relate them to adult breast cancer incidence. These types of studies would take decades to conduct (67) and should take into consideration the effects of other estrogens, because their effects can be additive or even synergistic (143, 144, 297).

Although our analyses of BPA have focused on its effects on the mammary gland and prostate (see *Sections II.C–D*), it is worth noting that several other endpoints have strong data to support the hypothesis that BPA has low-dose effects. In a recent review using similar WoE approaches, Hunt and colleagues (298) focused on those studies that examined the effects of BPA on the oocyte, specifically scrutinizing studies that reported effects, or no effects, on meiotic aneuploidy and other alterations in the intracellular organization and chromosome abnormalities. Similar to what has been observed with the prostate and mammary gland, the effects observed in the oocyte are variable from study to study, but overall consistent, and suggest that BPA exposure produces defects in these cells.

A large number of studies have also focused on the effects of BPA on the brain and behavior, with the most significant effects on sexually dimorphic regions of the brain and behaviors (299–307). Other affected behaviors include social behaviors, learning and anxiety, and maternal-neonate interactions (reviewed in Refs. 29 and 308). The NTP expert panel statement concluded that there were significant trends in these behavioral data and wrote that there was some concern that BPA could have similar effects in humans (273). Low-dose effects have also been reported for BPA in the female reproductive tract (309, 310), immune system (311, 312), maintenance of body weight and metabolism (313, 314), fertility (315–317), and the male reproductive tract (259, 318) (see Refs. 29 and 319 for comprehensive reviews).

E. Another controversial low-dose example: atrazine and amphibian sexual development

Atrazine is an herbicide that is applied in large volumes to crops, and there is concern that agricultural runoff of this chemical can affect nontarget animal species, especially amphibians that live and reproduce in small ponds and streams where significant amounts of atrazine have been regularly measured (320–322). It is the most commonly detected pesticide in ground and drinking water. Atrazine induces aromatase expression in cells and animals after exposure (323); this ultimately causes an increase in the conversion of testosterone to estrogen (324, 325). This effect has been reported in all vertebrate classes examined: fish, amphibians, reptiles, birds, and mammals, including human cell lines (see Ref. 326 for review). Another well-documented effect of atrazine is that it decreases androgen synthesis and activity, again, in every vertebrate class examined (326). In addition, endocrine-disrupting effects of atrazine occur through a number of other mechanisms, including antiestrogenic activity (327), altered prolactin release (328), and increased glucocorticoid release from the adrenal glands (329, 330), among others (327).

Because of atrazine's indirect effect on estrogen levels, one relevant endpoint that has been given attention is the effect of this chemical on gonad differentiation in various amphibian species. The early gonad is bipotential, and in

mammals, the expression of genes on the Y-chromosome is needed to masculinize the undifferentiated gonad; when this does not occur, the gonad develops into ovarian tissue. In *Xenopus laevis* frogs (and some other animals like birds), the opposite is true: females are heterogametic (*i.e.* ZW-chromosomes) and males have two of the same chromosomes (*i.e.* ZZ). In *X. laevis*, the W-chromosome is the dominant one, containing a gene, DM-W, which induces aromatase expression (331). Thus, having a W-chromosome is needed to produce estrogen; without the conversion of testosterone to estrogen, the frog develops as a male (332). Changes in sex ratio and gonadal morphology are therefore good indicators that an estrogen, or a chemical that up-regulates aromatase and indirectly increases estrogen levels, is present (76).

Determining a low-dose cutoff for atrazine is not a simple task. Although the safe limit of 3 μg/liter in drinking water was set by the EPA, actual levels in the environment often exceed this concentration (333), and levels in ponds and streams can reach 100 μg/liter (322) or more. In traditional toxicology studies examining several amphibian species, the LOAEL was set at 1.1 mg/liter, and the no observed effect level (NOEL) was 200 μg/liter (334, 335). Thus, using the definitions of low dose established by the NTP (2), we consider any treatment at or below 200 μg/liter to be a low dose.

In 2002, one of the first published studies to connect atrazine exposures to altered gonadal morphology examined *X. laevis* frogs exposed to 0.01–200 μg/liter throughout larval development (336). All doses from 0.1–200 μg/liter produced gonadal malformations including the presence of multiple gonads and hermaphroditism. Several other reports showed similar effects of low doses on gonadal phenotypes including studies that report the production of hermaphrodites and intersex frogs, males with ovotestes, and males with testicular oocytes (337–343). Additional studies showed that low-dose atrazine exposure (0.1–200 μg/liter in the water) during sexual differentiation caused testicular dysgenesis, testicular resorption, and testicular aplasia in male frogs (343, 344), and others indicated effects on sex ratios (339, 342, 345, 346). Importantly, these effects were not all observed at the same atrazine concentration, and the studies were conducted in several different species, with some reporting effects at low doses but no effects at higher doses (341) and others reporting effects in some but not all species (339). Examining these studies as a whole, there is clearly a pattern of effects that are reproducible from study to study, and they collectively support the hypothesis that atrazine disrupts sex hormone concentrations.

To date, five peer-reviewed studies have reported no effects of atrazine on sex ratios, gonadal morphology, the incidence of testicular abnormalities or testicular oocytes, gonad size, or the incidence of intersex phenotypes (347–351). Little can be ascertained from these negative studies, however, because four did not include any positive control, suggesting that the frogs used in those studies may have been incapable of responding to atrazine or any other hormonal treatment (347–350). Additionally, one of those studies reported testicular oocytes in the control frogs, suggesting either that the negative control population was contaminated with atrazine (or another EDC or hormone), or that an inappropriate strain of *X. laevis* was selected for the experiments (347). Only one study remains that did not find any effects of atrazine; this study used an appropriate positive control (17β-estradiol) and found effects of that hormone on sex ratios and the incidence of intersex gonads (351). An EPA expert panel noted, however, that this study used a strain of *X. laevis* that was obtained from a new, unexamined population of frogs from Chile and suggested that this strain may be insensitive to environmental chemicals. Furthermore, the panel called for additional analysis of the data in this study, including the statistical approaches; they suggested that an independent laboratory should evaluate the histopathological results; and they requested that atrazine metabolites be measured (352). The panel also proposed that these experiments should be repeated with an established *X. laevis* strain. Taking together the results of those studies that found effects of atrazine on sexual differentiation, and this one negative study, the WoE for the case of low-dose atrazine on sexual differentiation is clearly in support of adverse effects of this chemical.

Just as epidemiological studies have found links between EDCs and human diseases, ecological field studies have examined whether exposure to atrazine in natural environments affects the development of wild amphibians (343, 353–358). These studies have many of the same constraints as those observed in epidemiology: a paucity of data on early life exposures (including exposure levels of controls), limitations on the total number of EDCs that can be measured in environmental and biological samples, and a lack of causative relationships that can be established between exposures and effects. For these reasons, studies that found relationships between atrazine exposure (or concentrations in environmental samples) and effects on one or more aspect of sexual differentiation (343, 353–355) are considered weak, but significant, evidence for low-dose effects. The presence of several studies suggesting a relationship between low-dose exposure to atrazine in the wild and altered sexual differentiation indicates a plausible causal relationship. Because the ecological and laboratory data show similar effects of atrazine on go-

nadal development, this strengthens the conclusions of our WoE that low doses of atrazine cause harm to amphibians.

Feminization of males after atrazine exposure is not restricted to amphibians; exposure of zebrafish to low doses increased the ratio of female to male fish and increased expression of aromatase (359). Close to a dozen additional studies also report that environmentally relevant doses of atrazine can up-regulate aromatase, decrease testosterone, and/or increase estrogen levels in a large number of species (reviewed in Ref. 119), suggesting that low-dose effects of atrazine may be more widespread than their effects on the gonads of amphibians. Other studies indicate that low-dose atrazine affects the immune system and stress responses of salamanders (360–362), survivorship patterns of several frog species (363), and thyroid hormone and plasma ion concentrations in salmon (364).

An important factor to consider when examining the effects of atrazine on different animal models is the difficulty in identifying an appropriate low, environmentally relevant dose for all species. Aquatic animals can be housed in water containing levels of atrazine found in wild habitats, yet no toxicokinetic studies are available to determine what administered dose produces the levels of atrazine metabolites, typically in the parts-per-million or ppb range (365, 366), measured in human samples. There are also no blood or urine measurements in exposed rodents to compare with human levels; thus, extrapolations across species are estimates at best.

Keeping this qualification in mind, exposures in the range of 25–100 mg/kg · d during development have been shown to alter mammary gland development (367, 368), estrous cyclicity (369), serum and intratesticular testosterone concentrations (370), timing of puberty in males and prostate weight (371), and immune function (372) in rodents. Lower doses of atrazine metabolites (0.09–8.73 mg/kg · d) altered development of the mammary gland (373), male pubertal timing and prostate development (374). Identifying the range of doses administered to animals that produce the levels of atrazine and its metabolites measured in human blood and urine is an essential research need to pursue low-dose studies in rodents and other mammals.

F. Dioxin and spermatogenesis: low-dose effects from the most potent endocrine disruptor?

Dioxin, or TCDD, is formed as a byproduct of industrial processes as well as during waste incineration. Because TCDD is extremely toxic to some animals, with 1 μg/kg capable of killing 50% of guinea pigs, it has been labeled the most toxic chemical on earth (375). But interestingly, other animals are less sensitive to lethal effects of TCDD, with an LD_{50} of approximately 1000 μg/kg in hamsters, and studies also suggest that humans are not a hypersensitive species for lethality (376). Additionally, there are differences in the half-life of TCDD in different animals; in rodents, the half-life is 2–4 wks, but in humans, the half-life is approximately 10 yrs, and additional factors influence TCDD pharmacokinetics including the exposure level and the amount of body fat present (377–379). In cell cultures, doses as low as 10^{-11} M are toxic, with decreased viability observed even in cells maintained in nonproliferative states (380).

TCDD binds to the aryl hydrocarbon receptor (AhR), and differences in the affinity for the receptor may be responsible for differences in sensitivity between species (381). The K_d (dissociation constant for receptor-ligand binding kinetics) in human samples typically ranges from 3–15 nM, but in samples from rodents, the K_d is less than 1 nM (382). Importantly, there are also nongenomic pathways affected by TCDD that are mediated by AhR that are typically altered within minutes of TCDD exposure and therefore without changes in transcription (383). Yet many studies suggest that important differences exist between species regarding binding affinity of TCDD for AhR and the toxicity of this chemical, but that other adverse effects, including those related to the endocrine-disrupting activities of TCDD, occur at similar doses (or body burdens) across animal species (384, 385). Thus, it is plausible that AhR affinity alone can predict some, but not all, effects of TCDD and related chemicals.

The mechanisms responsible for many of the endocrine-disrupting activities of TCDD are currently not well understood. Knocking out AhR disrupts morphogenesis of several organ systems even in the absence of a ligand like TCDD, suggesting that this receptor plays important roles in early development (386). AhR is translocated to the nucleus after loss of cell-cell contacts and is often localized to the nucleus in embryonic cells, suggesting that it could have ligand-independent effects on development and/or that endogenous ligands could be present during early development (387). When TCDD is present, AhR translocates to the nucleus and dimerizes with ARNT, the aromatic hydrocarbon receptor nuclear translocator (388). Although the (currently unidentified) physiological activators of AhR are likely to induce rapid on/off signaling via AhR, TCDD and related compounds appear to maintain activation of AhR, and the presence of TCDD prevents the normal action of the AhR signaling pathway in the maintenance of homeostasis (389). This induces changes in the expression of genes and promotes the production of toxic metabolites. These effects may be responsible for some of the endocrine-related endpoints affected by TCDD exposure. Additionally, recent studies have shown complex and intricate interactions between the

AhR and ER signaling pathways (390), suggesting that dioxin may also have indirect effects on some ER-mediated endpoints via AhR signaling.

Teratogenic effects of TCDD have been well documented after high-dose (391, 392) and low-dose exposures (393). These studies show that almost every organ and system in the body is affected by this chemical. High doses that did not produce lethality caused severe weight loss, intestinal hemorrhaging, alopecia, chloracne, edemas, and severe liver damage. Sadly, there are now several examples in humans of accidental exposures after the industrial release of TCDD where a number of individuals have been exposed to large doses (389, 394) as well as a few documented intentional poisonings (395). The tolerated daily intake level was set at 1–4 pg/kg · d, although the doses consumed by nursing infants are likely to exceed these levels by a factor of 10 (375). Adult exposures usually result from the consumption of contaminated foods, and because TCDD is lipophilic, it is concentrated in the fat component of breast milk and therefore passed in large quantities from a nursing mother to her infant.

Using classical toxicology methods, the effects of single TCDD doses were examined in adult male rats, specifically focusing on the effects of this chemical on the number of spermatids per testis and the integrity of the testicular germinal epithelium (396). In one of the earliest studies, Chahoud and colleagues (397) determined a LOAEL of 3 μg/kg · d and set the NOAEL at 1 μg/kg · d for effects on the testes. Because there are significant differences in the toxicity of TCDD between animal models, and different endpoints have different identified NOAELs, we have selected the 1 μg/kg · d identified by Chahoud *et al.* as the cutoff for low-dose studies of this compound. This cutoff is based on the NTP's definition of low dose as occurring at doses lower than those tested in traditional toxicology assessments (2). However, it is important to acknowledge that body burdens that mimic those observed in human populations are likely the best indicators of low doses for TCDD (384), and thus we recommend that future studies determine body burdens after administration of TCDD for the specific strain, origin, and species of animal being tested to ensure that truly low doses, relevant to human populations, are being tested.

Several recent epidemiological studies have indicated that relatively high exposures to TCDD during early life (due to industrial release of high amounts of the chemical) can permanently affect semen quality and sperm count in men (398). Yet epidemiology studies also clearly show that the timing of TCDD exposure can vastly influence the effect of this chemical on spermatogenesis; exposures during perinatal life significantly reduced sperm parameters, but exposures during puberty increased sperm counts; exposures in adulthood had no effect on sperm parameters (399). Thus, it is also important for animal studies to focus on exposures during critical periods for development of the male reproductive tract and spermatogenesis in particular.

We are aware of 18 studies that have examined the effects of low doses ($\leq$1 μg/kg · d) of TCDD during perinatal development on male fertility endpoints in adulthood. The endpoints assessed vary, including epididymal sperm counts, ejaculated sperm number, daily sperm production, sperm transit rate, and percent abnormal sperm, and the sensitivity of these endpoints appears to impact the ability to detect low-dose effects in different studies (400, 401) (Table 5). In total, 16 rodent studies examined the effect of low-dose TCDD on epididymal sperm count; 12 showed significant effects on this endpoint (402–413), whereas the other four did not (414–417). Of the five studies that examined ejaculated sperm counts, four studies (404, 405, 408), including one examining rhesus monkeys (418), showed effects of low-dose TCDD, *i.e.* a significant decrease in sperm counts; one study found no effect (417). Daily sperm production was a less-sensitive endpoint, with four studies showing significant decreases after prenatal exposure to low doses (402, 403, 407, 409) and four studies showing no effects (406, 412, 413, 416); sperm transit rate was examined in only two studies, although both showed significant decreases in sperm tranfer rates (403, 410); and finally, three studies determined that low-dose TCDD produced abnormalities in sperm appearance or motility (414, 415, 419), but one study was not able to replicate these findings (417).

When examining the TCDD literature as a whole, the WoE strongly suggests that prenatal exposure to low doses of TCDD affects sperm-related endpoints in adulthood (Table 5). In all, only two studies were unable to detect any effect of TCDD on the sperm endpoints assessed, although both studies found effects of TCDD on other endpoints including the weight of the adult prostate (416) and the timing of puberty (417). No study on TCDD used a positive control, likely due to a paucity of information on the mechanisms of dioxin action, but this raises obvious questions about the ability of these experimental systems to detect effects on spermatogenesis. Finally, some of the inability to detect effects of TCDD could be due to the use of insensitive strains, because 1000-fold differences in sensitivity have been reported for different rodent strains (420).

Even though we have focused the majority of our attention on the effects of low-dose TCDD exposure on spermatogenesis, it should be noted that low doses of this chemical affect a multitude of endpoints in animals, altering immune function (421, 422), indicators of oxidative

TABLE 5. Summary of low-dose animal studies examining the effects of TCDD on spermatogenesis endpoints

Study	Administered dose (time of administration)	Animal	Epididymal sperm count	Ejaculated sperm no.	Daily sperm production	Sperm transit rate	% abnormal sperm
Mably *et al.* (409)	0.064–1 μg/kg (gestational d 15)	Rat	Decreased	NA	Decreased	NA	NA
Bjerke and Peterson (402)	1 μg/kg (gestational d 15)	Rat	Decreased	NA	Decreased	NA	NA
Gray *et al.* (404)	1 μg/kg (gestational d 8)	Rat	Not significant	Decreased	NA	NA	NA
	1 μg/kg (gestational d 15)	Rat	Decreased	Decreased	NA	NA	NA
	1 μg/kg (gestational d 11)	Hamster	Decreased	Decreased	NA	NA	NA
Sommer *et al.* (408)	1 μg/kg (gestational d 15)	Rat	Decreased	Decreased	Decreased	Not significant	Not significant
Wilker *et al.* (410)	0.5, 1 or 2 μg/kg (gestational d 15)	Rat	Decreased	NA	Unaffected	Increased	NA
Gray *et al.* (405)	0.05–1 μg/kg (gestational d 15)	Rat	Decreased	Decreased	Decreased	NA	NA
Faqi *et al.* (403)	0.025–0.3 μg/kg (before mating, then 0.005–0.06 μg/kg weekly [to dams])	Rat	Decreased	NA	Decreased	Increased	Increased
Loeffler and Peterson (412)	0.25 μg/kg (gestational d 15)	Rat	Decreased	NA	Unaffected	NA	NA
Ohsako *et al.* (416)	0.0125–0.8 μg/kg (gestational d 15)	Rat	Not significant	NA	Unaffected	NA	NA
Ohsako *et al.* (406)	1 μg/kg (gestational d 15)	Rat	Decreased	NA	Unaffected	NA	NA
	1 μg/kg (gestational d 18)	Rat	Unaffected	NA	Unaffected	NA	NA
	1 μg/kg (postnatal d 2 [to pups])	Rat	Unaffected	NA	Unaffected	NA	NA
Simanainen *et al.* (407)	0.03–1 μg/kg (gestational d 15)	Rat	Decreased	NA	Decreased	NA	NA
Yonemoto *et al.* (417)	0.0125–0.8 μg/kg (gestational d 15)	Rat	Unaffected	Unaffected	NA	NA	Unaffected
Yamano *et al.* (714)	0.3 or 1 μg/kg (postnatal d 1 and then every week [to dams])	Rat	Not significant	NA	NA	NA	NA
Ikeda *et al.* (715)	0.4 μg/kg (before mating, then 0.08 μg/kg weekly [to dams])	Rat	Unaffected	NA	NA	NA	NA
Bell *et al.* (414)	0.05–1 μg/kg (gestational d 15)	Rat	Increased (at certain ages)	NA	NA	NA	Increased
Bell *et al.* (415)	0.0024–0.046 μg/kg (d 12 weeks before pregnancy through parturition)	Rat	Unaffected	NA	NA	NA	Increased
Arima *et al.* (418)	0.03 or 0.3 μg/kg (gestational d 20, then 5% of dose monthly [to dams])	Rhesus monkey	Decreased	Not significant	NA	NA	Not significant
Yamano *et al.* (419)	0.3 or 1 μg/kg (weekly to dams then pups [all postnatal])	Rat	NA	NA	NA	NA	Increased
Jin *et al.* (411)	1 μg/kg · d (postnatal days 1–4 [to dams])	Mouse	Decreased	NA	NA	NA	NA
Rebourcet *et al.* (413)	0.01–0.2 μg/kg (gestational d 15)	Rat	Decreased (at some ages)	NA	Not significant	NA	NA

Not significant indicates trend for effect but did not reach statistical significance. Unaffected means assessed, but no differences were observed relative to controls. Here, low doses were considered any at or below 1 μg/kg · d (see text for discussion of how this cutoff was established for rodent studies). NA, Not assessed.

stress (423–425), bone and tooth development (426, 427), female reproduction and timing of puberty (428–430), mammary gland development and suceptibility to cancers (431), behaviors (432, 433), and others. In several cases, lower doses were more effective at altering these endpoints than higher ones (423, 424, 426, 433). Epidemiology studies of nonoccupationally exposed individuals also indicate that serum TCDD levels may be linked to diseases in humans as well (434). Mean serum TCDD levels have decreased by a factor of 7 over a 25-yr period (1972–97) in several industrial nations (435), but results from both animal and epidemiological studies suggest that even the low levels detected now could have adverse effects on health-related endpoints.

G. Perchlorate and thyroid: low-dose effects in humans?

A significant challenge with observing low-dose effects of EDCs in the human population is that human chemical exposures are multivariate along the vectors of time, space, and sensitivities. In addition, chemicals can exert effects on several systems simultaneously. Therefore, associations in human studies between exposures and disease are difficult to reconcile with experimental studies in animal model systems. For this reason, the literature describing the potential impacts of perchlorate contamination on the human population is potentially clarifying because to the best of our knowledge, perchlorate exerts only a single effect, and the pharmacology of perchlorate exposures has been studied in human volunteers (436). This

literature offers a unique perspective into the issue of low-dose effects, perhaps providing important hypotheses to explain mechanistically why high-dose, short-term experiments can fail to predict the outcome of low-dose, lifetime exposures.

In the 2001–2002 NHANES dataset, perchlorate was detected in the urine of each of the 2820 samples tested (437). This widespread exposure means that the human population is being continuously exposed because perchlorate has a half-life in the human body of about 8 h (438). Human exposures to perchlorate are likely attributed to both contaminated drinking water and food (439); in fact, a recent analysis concludes that the majority of human exposure to perchlorate comes from food (440).

The predominant theory proposed to explain the source of perchlorate contamination in the United States is that it has been employed for many decades as the principal oxidant in explosives and solid rocket fuels (441). Perchlorate is chemically stable when wet and persists for long periods in geological systems and in ground water. Because of disposal practices during the 1960s through 1990s, perchlorate became a common contaminant of ground water in the United States (441, 442). Perchlorate is also formed under certain kinds of natural conditions (443), although the relative contributions to human exposure of these different sources is not completely understood. As a result of perchlorate contamination of natural waters, the food supply has become contaminated through irrigation in part because both aquatic and terrestrial plants can concentrate perchlorate more than 100-fold over water levels (444).

This exposure profile in the human population is important because high doses of perchlorate are known to reduce functioning of the thyroid gland, and poor thyroid function is an important cause of developmental deficits and adult disease (445). The primary question is: at what dose does perchlorate inhibit thyroid function sufficiently to cause disease? The current literature, reviewed below, supports the view that background exposure may affect thyroid function in adult women. These exposure levels, however, are considerably lower than predicted by early toxicology experiments in humans.

Perchlorate reduces thyroid function by inhibiting iodide uptake by the sodium/iodide symporter (NIS) (446), which is the only known effect of perchlorate on human physiology (438). NIS is responsible for transporting iodide into the thyroid gland, which is required for the production of thyroid hormone (447). However, NIS is also expressed in the gut (448, 449), in lactating breast (448, 450, 451), and in placenta (452), presumably all as a delivery mechanism for iodide to the developing and adult thyroid gland. Because the NIS transports perchlorate (450), the pathway by which humans take up and concentrate perchlorate is the same as the pathway by which humans take up and concentrate iodide. Interestingly, NIS expression in the human fetal thyroid gland is the rate-limiting step in production of thyroid hormone (453). Moreover, NIS transport of perchlorate explains why high levels of perchlorate are found in human amniotic fluid (454, 455) and breast milk (456–459).

This effect of perchlorate on thyroid function is important because thyroid hormone is essential for normal brain development, body growth as well as for adult physiology (445, 460). Moreover, it has become clear that even small deficits in circulating thyroid hormone in pregnant women (461, 462) or neonates (463) have permanent adverse outcomes. In fact, recent work indicates that very subtle thyroid hormone insufficiency in pregnant women is associated with cognitive deficits in their children (461). Because of the importance of thyroid hormone in development and adult physiology, and because perchlorate is a potent inhibitor of iodide uptake and thyroid hormone synthesis, identifying the dose at which these events occur is critical.

Perchlorate was used medically to reduce circulating levels of thyroid hormone in patients with an overactive thyroid gland in the 1950s and 1960s (reviewed in Ref. 446); therefore, it was reasonable to examine the dose-response characteristics of perchlorate on the human thyroid gland. Because perchlorate inhibits iodide uptake, several studies were performed to evaluate the effect of perchlorate exposure on iodide uptake inhibition in human volunteers (438, 464–466). In one study, 0.5 or 3 mg/d (approximately 0.007 and 0.04 mg/kg · d) perchlorate was administered to healthy volunteers (n = 9 females and 5 males, age 25–65 yr), and no effects were observed (466). Of course, it is important to note that the 2 wk of administration tested in this study is not sufficient to see any effect on serum concentrations of T_4 or TSH; the healthy thyroid can store several months' worth of thyroid hormone in the gland (467). Another small study also found no effects of administering 3 mg/d (approximately 0.04 mg/kg · d) on any thyroid endpoint assessed (n = 8 adult males) (464).

In contrast, two studies examining adult volunteers administered perchlorate found effects of this chemical on at least one endpoint. The first found that radioactive iodide uptake was affected by 2 wk of exposure to 10 mg/d (0.13 mg/kg · d), but other measures of thyroid function were not altered (n = 10 males) (465). The second examined adults (n = 37) given doses ranging from 0.007–0.5 mg/kg · d; all but the lowest dose altered radioactive iodide uptake, and only the highest dose altered TSH levels (438). These studies were interpreted to suggest that adults would have to consume 2 liters of drinking water daily that

was contaminated with at least 200 ppb (200 μg/liter) perchlorate to reach a level in which iodide uptake would begin to be inhibited. Yet, these administered doses are high and relatively acute, so the derivation of a safe dose from these studies, applied to vulnerable populations such as those with low iodide intake, has been strongly disputed (471).

Studies of occupational exposures have also been used to examine the effects of exposure to relatively high levels of perchlorate. In the first such study, more than 130 employees were separated into eight groups based on exposure estimates from airborne perchlorate in the workplace (472). The authors found that individuals with longer daily exposures to perchlorate, due to longer work shifts, had significant decreases in TSH levels compared with individuals with shorter exposures. But this study was hampered because actual exposure levels were not measured via urine or blood samples. A second study examined 37 employees exposed to perchlorate and 21 control employees from an azide factory; actual exposure measures were not conducted, but estimates were calculated based on exposures to perchlorate dust and air samples (473). This study found no effects of perchlorate exposures on any thyroid endpoint, although the sample size examined was small. In the final occupational exposure study, serum perchlorate levels were measured and compared with several measures of thyroid function in workers (n = 29) who had spent several years as employees in a perchlorate production plant (474). In this study, the most complete because of the biomonitoring aspect of the exposure measures, higher perchlorate levels were associated with lower radioactive iodide uptake, higher urinary iodide excretion, and higher thyroid hormone concentrations.

Although iodide uptake was often inhibited in these studies, serum thyroid hormones were typically not altered, perhaps because of sufficient stored hormone. Based on these observations, the National Academy Committee to Assess the Health Implications of Perchlorate Ingestion (467) estimated that perchlorate would have to inhibit thyroid iodide uptake by about 75% for several months to cause a reduction in serum thyroid hormones. Moreover, the drinking water concentration of perchlorate required for this kind of inhibition was estimated to be over 1,000 ppb (438). Therefore, the National Academy of Sciences committee recommended a reference dose of 0.0007 mg/kg · d (467), based on the dose at which perchlorate could inhibit iodide uptake, and the EPA used this value to set a provisional drinking water standard of 15 ppb.

Considering these data and general knowledge about the thyroid system, it was unexpected that Blount *et al.* (475) would identify a positive association between urinary iodide and serum TSH in adult women in the NHANES 2001–2002 dataset. Yet several features of this dataset were consistent with a causal action of perchlorate on thyroid function. First, in the general population of adult women, urinary perchlorate was positively associated with serum TSH. In the population of adult women who also had low urinary iodide, however, urinary perchlorate was more strongly associated with serum TSH and was negatively associated with serum T_4. The strength of this association was such that the authors calculated that women at the 50th percentile of perchlorate exposure experienced a 1 μg/dl T_4 reduction (reference range = 5–12 μg/dl). Should this magnitude of reduction in serum T_4 occur in a neonate, measurable cognitive deficits would also be present (476). Finally, Steinmaus *et al.* (477), using the same NHANES dataset, showed that women with low urinary iodide who smoke had an even stronger association between urinary perchlorate and measures of thyroid function. Tobacco smoke delivers thiocyanates, which also inhibit NIS-mediated iodide uptake (446).

The NHANES dataset suggests that perchlorate exposures of 0.2–0.4 μg/kg · d (440) are associated with depressed thyroid function, even when urinary iodide is not reduced. This is a considerably lower dose than the 7 μg/kg · d dose required to suppress iodide uptake in the Greer *et al.* (438) study or the 500 μg/kg · d the NAS estimated would be required for several months to actually cause a decline in serum T_4. Therefore, it is reasonable to question whether these associations represent a causative relationship between perchlorate and thyroid function.

A number of epidemiological studies have been published to test for a relationship between perchlorate exposure and thyroid function. Early work used neonatal screening data for T_4 as a measure of thyroid function, and the city of birth (Las Vegas, NV, compared with Reno, NV) as a proxy measure of exposure (478, 479). The reported findings were negative, but we now know that all Americans are exposed to perchlorate, so there was considerable misclassification of exposure, and no relationship should have been observed. Several additional studies using similar flawed designs also found no relationship between proxy measures of perchlorate exposures and clinical outcomes (480–484).

A recent study of the neonatal screening data from 1998 in California identified a strong association between neonatal TSH and whether or not the mother resided in a contaminated area (485). This study included over 497,000 TSH measurements and 800 perchlorate measurements. In addition, they used as a cutoff a variety of TSH levels (as opposed to the 99.9th percentile used for the diagnosis of congenital hypothy-

roidism), indicating that perchlorate exposure is not associated with congenital hypothyroidism. Two additional studies have shown similar relationships between perchlorate and TSH levels, particularly in families with a history of thyroid disease (486, 487).

Several studies in pregnant women have failed to identify a relationship between perchlorate exposure and measures of thyroid function (488–490). Although these are important studies that need to be carefully scrutinized, they do not replicate or refute the NHANES dataset. It thus remains important to conduct additional studies exploring the relationship between background exposure to perchlorate and thyroid function in adults, pregnant women, neonates, and infants. This effort will be challenging because of the different characteristics of thyroid function and hormone action at different life stages (460). In addition, it will be important to obtain individual measurements of exposures to perchlorate and other NIS inhibitors (thiocyanate and nitrate), and iodide itself as well as individual measures of thyroid function (free and total T_4 and TSH).

If background levels of perchlorate affect thyroid function in any segment of the population, it will be challenging to explain how the high-dose, short-term experiments of Greer *et al.* (438) completely underestimated the sensitivity of the human thyroid gland to perchlorate exposure. One possibility is that physiological systems respond to short durations of robust stress with compensatory mechanisms that reset during periods of long-term stress.

When these data are examined together, several important issues are raised. First, this example illustrates the difficulties inherent in studying human populations; epidemiology yields associations, not cause-effect relationships, in many cases using surrogate markers for perchlorate, and is not able to distinguish short- *vs.* long-term exposure duration. Second, our WoE analysis suggests that there is weak evidence for low-dose effects of perchlorate; further research is needed. The relationship between low-dose perchlorate exposures and thyroid endpoints would be strengthened by the addition of studies that measure biological concentrations of perchlorate and compare them with thyroid endpoints in neonates and other vulnerable populations. Third, the published studies that reported low-dose effects of perchlorate typically examined very specific populations, with several focusing on women with low iodine intake. This observation suggests that some groups may be more vulnerable to low doses of perchlorate than others (491).

H. Low-dose summary

These examples, and the examples of low-dose effects in less well-studied chemicals (Table 3), provide evidence that low-dose effects are common in EDC research and may be the default expectation for all chemicals with endocrine activity. Many known EDCs have not been examined for low-dose effects, but we predict that these chemicals will have effects at low doses if studied appropriately. Although studies unable to detect effects at low doses have received attention, including some studies designed to replicate others that reported low-dose effects, the majority of these studies contain at least one major design flaw. Thus, a WoE approach clearly indicates that low-dose effects are present across a wide span of chemical classes and activities.

III. Nonmonotonicity in EDC Studies

A concept related to low dose is that of nonmonotonicity. As noted in *Section I.B*, in a monotonic response, the observed effects may be linear or nonlinear, but the slope does not change sign (Fig. 3, A and B). In contrast, a dose-response curve is nonmonotonic when the slope of the curve changes sign somewhere within the range of doses examined (Fig. 3C). NMDRCs are often U-shaped (with maximal responses of the measured endpoint observed at low and high doses) or inverted U-shaped (with maximal responses observed at intermediate doses) (Fig. 3C, *top panels*). Some cases are more complicated, with multiple points along the curve at which the slope of the curve reverses sign (Fig. 3C, *bottom left*). Nonmonotonicity is not synomymous with low dose, because there are low-dose effects that follow monotonic dose-response curves. Thus, it is not required that a study include doses that span from the true low-dose range to the high toxicological range to detect nonmonotonicity. The consequence of NMDRCs for toxicity testing is that a safe dose determined from high doses does not guarantee safety at lower, untested doses that may be closer to current human exposures.

Examples of NMDRCs from the cell culture, animal, and epidemiological literature will be discussed in detail in *Section III.C*. Importantly, our review of the literature finds that NMDRCs are common in the endocrine and EDC literature. In fact, it is plausible that, considering the mechanisms discussed below, NMDRCs are not the exception but should be expected and perhaps even common.

A. Why is nonmonotonicity important?

NMDRCs in toxicology and in the regulatory process for EDCs are considered controversial. In addition to discussions of whether NMDRCs exist, there is also discussion of whether those that do exist have relevance to

Figure 3.

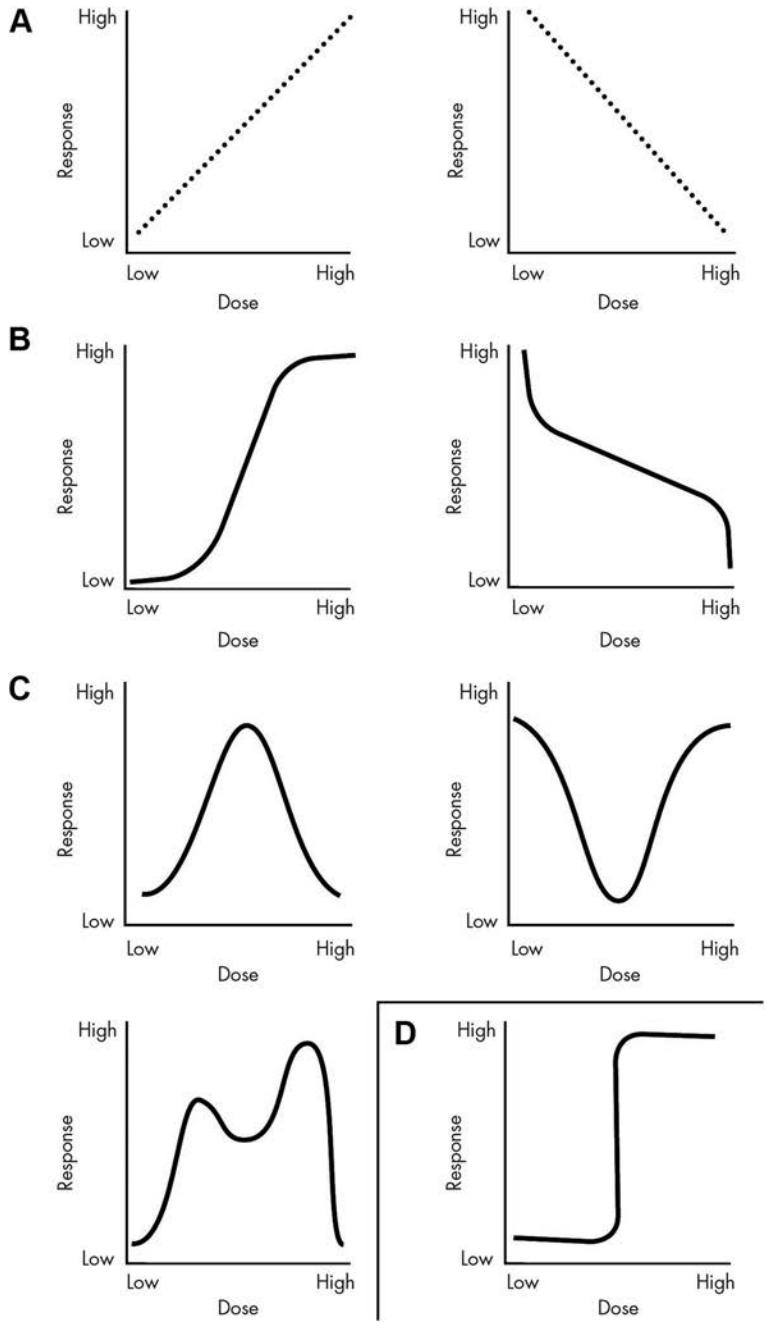

Figure 3. Examples of dose-response curves. A, Linear responses, whether there are positive or inverse associations between dose and effect, allow for extrapolations from one dose to another. Therefore, knowing the effects of a high dose permits accurate predictions of the effects at low doses. B, Examples of monotonic, nonlinear responses. In these examples, the slope of the curve never changes sign, but it does change in value. Thus, knowing what happens at very high or very low doses is not helpful to predict the effect of exposures at moderate doses. These types of responses often have a linear component within them, and predictions can be made within the linear range, as with other linear responses. C, Displayed are three different types of NMDRCs including an inverted U-shaped curve, a U-shaped curve, and a multiphasic curve. All of these are considered NMDRCs because the slope of the curve changes sign one or more times. It is clear from these curves that knowing the effect of a dose, or multiple doses, does not allow for assumptions to be made about the effects of other doses. D, A binary response is shown, where one range of doses has no effect, and then a threshold is met, and all higher doses have the same effect.

toxicological determination of putative safe exposures. In the standard practice of regulatory toxicology, the calculated safe dose, also called a reference dose, is rarely tested. In a system that is responding nonmonotonically, it is not appropriate to use a high-dose test to predict low-dose effects. Unfortunately, all regulatory testing for the effects of chemical exposures assume that this is possible. All current exposure standards employed by government agencies around the world, including the FDA and EPA, have been developed using an assumption of monotonicity (492, 493). The low-dose range, which presumably is what the general public normally experiences, is rarely, if ever, tested directly.

The standard procedure for regulatory testing typically involves a series of tests to establish the lowest dose at which an effect is observable (the LOAEL), then a dose beneath that at which no effect is observable (the NOAEL). Then a series of calculations are used to acknowledge uncertainty in the data, species differences, age differences, *etc.*, and those calculations, beginning with the LOAEL or the NOAEL, produce a reference dose that is presumed to be a safe exposure for humans (Fig. 4). Typically, the reference dose is 3- to 1000-fold lower than the NOAEL. That reference dose then becomes the allowable exposure and is deemed safe, even when it is never examined directly. For chemicals with monotonic linear dose-response curves (Fig. 3A), this may be appropriate. But for chemicals that display nonmonotonic patterns, it is likely to lead to false negatives, *i.e.* concluding that exposure to the reference dose is safe when in fact it is not.

As described above, there are other nonlinear dose-response curves that are monotonic (Fig. 3B). These curves may also present problems for extrapolating from high doses to low doses because there is no linear relationship that can be used to predict the effects of low doses. Equally troubling for regulatory purposes are responses that have a binary response rather than a classical dose-response curve (Fig. 3D). In these types of responses, one range of doses has no effect on an endpoint, and then a threshold is met, and all higher doses have the same effect. An example is seen in the atrazine literature, where doses below 1 ppb had no effect on the size of the male larynx but doses

at or above 1 ppb produced a significant decrease in size of approximately 10–15% (336). Even doses of 200 ppb, the toxicological NOEL, produce the same effect. Thus, this all-or-none effect is observed because atrazine does not shrink the larynx; instead, it removes the stimulatory agent (*i.e.* androgens). In the absence of some threshold dose of androgen, the larynx simply remains at the unstimulated (female) size. The EPA's assessment of this study and others was that the lack of a dose-dependent response negates the importance of this effect (352). The lack of a dose response for a threshold effect like larynx size does not mean that the effects are not dose dependent; thus, understanding these types of effects and their implications for risk assessments is essential for determining the safe levels of chemicals.

It is important to mention here that the appropriateness of determining NOAEL concentrations, and therefore calculating reference doses, from exposures to endogenous hormones or EDCs has been challenged by several studies (Fig. 4A) (494–496). These studies show that hormonally active agents may still induce significant biological effects even at extremely low concentrations and that presently available analytical methods or technologies might be unable to detect relatively small magnitudes of effects. Previous discussions of this topic have shown that as the dose gets lower (and approaches zero) and the effect size decreases, the number of animals needed to achieve the power to detect a significant effect would have to increase substantially (497). Even more importantly, the assumption of a threshold does not take into account situations where an endogenous hormone is already above the dose that causes detectable effects and that an exogenous chemical (whether an agonist or antagonist) will modulate the effect of the endogenous hormone at any dose above zero (Fig. 4B). There can thus be no threshold or safe dose for an exogenous chemical in this situation. Forced identification of NOAEL or threshold doses based on the assumption that dose-response curves are always monotonic without considering the background activity of endogenous hormones and the limitations of analytical techniques supports the misconception that hormonally active agents do not have any significant biological effects at low doses. Thus, the concept that a toxic agent has a safe dose that can be readily estimated from the NOAEL derived from testing high, acutely toxic doses is overly simplistic and contradicted by data when applied to EDC (5, 497, 498).

B. Mechanisms for NMDRCs

Previously, the lack of mechanisms to explain the appearance of NMDRCs was used as a rationale for ignoring these phenomena (492, 493). This is no longer acceptable because there are several mechanisms that have been identified and studied that demonstrate how hormones and EDCs produce nonmonotonic responses in cells, tissues, and animals. These mechanisms include cytotoxicity, cell- and tissue-specific receptors and cofactors, receptor selectivity, receptor down-regulation and desensitization, receptor competition, and endocrine negative feedback loops. These mechanisms are well understood, and by providing detailed biological insights at the molecular level into the etiology of NMDRCs, they strongly negate the presumption that has been central to regulatory toxicology that dose-response curves are by default monotonic.

1. Cytotoxicity

The simplest mechanism for NMDRCs derives from the observation that hormones can be acutely toxic at high doses yet alter biological endpoints at low, physiologically relevant doses. Experiments working at concentrations that are cytotoxic are incapable of detecting responses that are mediated by ligand-binding interactions. For example, the MCF7 breast cancer cell line proliferates in response to estradiol in the low-dose range (10^{-12} to 10^{-11} M) and in the pharmacological and toxicological range (10^{-11} to 10^{-6} M), but toxic responses are observed at higher doses (38). Thus, when total cell number is graphed, it displays an inverted U-shaped response to estrogen. But cells that do not contain ER, and therefore cannot be affected by the hormonal action of estradiol, also display cytotoxic responses when treated with high doses of hormone. These results clearly indicate that the effects of estradiol at high doses are toxic via non-ER-mediated mechanisms.

2. Cell- and tissue-specific receptors and cofactors

Some NMDRCs are generated by the combination of two or more monotonic responses that overlap, affecting a common endpoint in opposite ways via different pathways. For example, *in vitro* cultured prostate cell lines demonstrate a nonmonotonic response to increasing doses of androgen where low doses increase cell number and higher doses decrease cell number, thus producing an inverted U-shaped curve (499, 500). Although the parental cell expressed an inverted U-shaped dose-response curve, after a long period of inhibition, the effects on cell number could be segregated by selecting two populations of cells: one that proliferated in the absence of androgens and other cells that proliferated in the presence of high androgen levels (501). Thus, the observed inverted U-shaped response is due to actions via two independent pathways that can be separated from each other in an experimental setting (502). Similarly, estrogens have been shown to induce cell proliferation and inhibit apoptosis in several cell populations, but inhibit proliferation and induce apopto-

Figure 4.

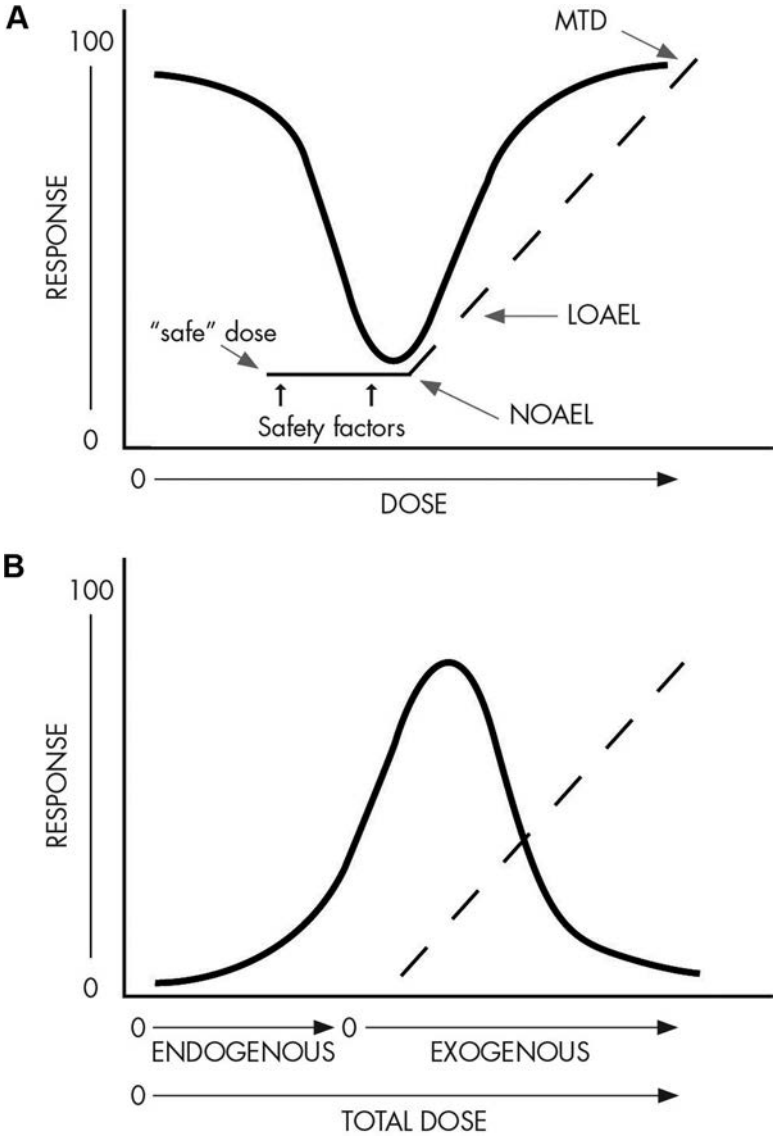

Figure 4. NOAEL, LOAEL, and calculation of a safe reference dose. A, In traditional toxicology testing, high doses are tested to obtain the maximum tolerated dose (MTD), the LOAEL, and the NOAEL. Several safety factors are then applied to derive the reference dose, *i.e.* the dose at which exposures are presumed safe. This reference dose is rarely tested directly. Yet when chemicals or hormones produce NMDRCs, adverse effects may be observed at or below the reference dose. Here, the doses that would be tested are shown by a *dotted line*, and the calculated safe dose is indicated by a *thick solid line*. The actual response, an inverted U-shaped NMDRC, is shown by a *thin solid line*. B, Experimental data indicate that EDCs and hormones do not have NOAELs or threshold doses, and therefore no dose can ever be considered safe. This is because an exogenous hormone (or EDC) could have a linear response in the tested range (*dotted line*), but because endogenous hormones are present (*thin solid line*), the effects of the exogenous hormone are always observed in the context of a hormone-containing system.

sis in others (503, 504), with the combined effect being an inverted U-shaped curve for cell number (505).

Why does one single cell type have different responses to different doses of the same hormone? The case of the prostate cell line described above is reminiscent of the results described from the transcriptome of MCF7 cells, whereby a discrete global response like cell proliferation manifests at significantly lower estrogen doses than the induction of a single marker gene (135). That a response like cell proliferation requires a significantly lower dose of hormone than the dose needed to induce a given target gene is counterintuitive but factual; it may be interpreted as consistent with the notion that metazoan cells, like cells in unicellular organisms, are intrinsically poised to divide (503, 506, 507) and that quiescence is an induced state (508, 509). The biochemical details underlying these different responses are largely unknown; however, recent studies showed that steroid receptors control only a portion of their target genes directly via promoter binding. The majority of the changes are indirect, through chromatin rearrangements (510, 511).

Why do different cell types (*in vitro* and *in vivo*) have different responses to the same hormone? One answer is that they may express different receptors, and these receptors have different responses to the same hormone. For example, some tissues express only one of the two major ER (ERα and ERβ), and actions via these receptors are important not just for responsiveness to hormone but also for cellular differentiation and cross talk between tissue compartments (512). Yet other tissues express both ERα and ERβ, and the effects of signaling via these two receptors often oppose each other; *i.e.* estrogen action via ERα induces proliferation in the uterus, but ERβ induces apoptosis (154). Complicating the situation further, different responses to a hormone can also be obtained due to the presence of different cofactors in different cell and tissue types (513, 514); these coregulators influence which genes are transcriptionally activated or repressed in response to the presence of hormone. They can also influence ligand selectivity of the receptor and DNA-binding capacity, having tremendous impact on the ability of a hormone to have effects in different cell types (105, 515, 516).

Although much of these activities occur on a biochemical level, *i.e.* at the receptor, there is also evidence that nonmonotonicity can originate at the level of tissue organization. The mammary gland has been used as a model to study inter- and intracompartmental effects of hormone treatment: within the ductal epithelium, estro-

gen has distinct effects during puberty, both inducing proliferation, which causes growth of the ductal tree, and inducing apoptosis, which is required for lumen formation (517, 518); in cell culture, the presence of stromal cells can also enhance the effects of estrogen on epithelial cells (519, 520), suggesting that stromal-epithelial compartmental interactions can mediate the effects of estrogen.

3. Receptor selectivity

NMDRCs can occur because of differences in receptor affinity, and thus the selectivity of the response, at low *vs.* high doses. For example, at low doses, BPA almost exclusively binds to the ER (including mER), but at high doses it can also bind weakly to other hormone receptors, like androgen receptor and thyroid hormone receptor (249, 521). This type of receptor nonselectivity is quite common for EDCs, and it has been proposed that binding to different receptors may be an explanation for the diverse patterns of disease observed after EDC exposures (522). In fact, several of the chemicals shown to have low-dose effects are known to act via multiple receptors and pathways (Table 3). Thus, the effects seen at high doses can be due to action via the binding of multiple receptors, compared with the effects of low doses, which may be caused by action via only a single receptor or receptor family.

4. Receptor down-regulation and desensitization

When hormones bind to nuclear receptors, the ultimate outcome is a change in the transcription of target genes. When the receptor is bound by ligand, an increase in response is observed; as discussed previously in this review, the relationship between hormone concentration and the number of bound receptors, as well as the relationship between the number of bound receptors and the biological effect, is nonlinear (38). After the nuclear receptor is bound by hormone and transcription of target genes has occurred (either due to binding of the receptor at a DNA response element or the relief of a repressive event on the DNA), the reaction eventually must cease; *i.e.* the bound receptor must eventually be inactivated in some way. Thus, nuclear hormone receptors are ubiquitinated and degraded, usually via the proteasome (523). Importantly, the role of the hormone in receptor degradation differs depending on the hormone; binding of estrogen, progesterone, and glucocorticoid mediates the degradation of their receptors (524–526), whereas the presence of hormone may actually stabilize some receptors and prevent degradation (527), and other receptors are degraded without ligand (528). As hormone levels rise, the number of receptors being inactivated and degraded also rises, and eventually the number of receptors being produced cannot maintain the pace of this degradation pathway (523). Furthermore, the internalization and degradation of receptors can also influence receptor production, leading to an even stronger down-regulation of receptor (529). In the animal, the role of receptor down-regulation is actually quite complex, because signaling from one hormone receptor can influence protein levels of another receptor; *i.e.* ER signaling can promote degradation of the glucocorticoid receptor by increasing the expression of enzymes in the proteasome pathway that degrade it (530).

There is also the issue of receptor desensitization, a process whereby a decrease in response to a hormone is not due to a decrease in the number of available receptors but instead due to the biochemical inactivation of a receptor (531). Desensitization typically occurs when repeated or continuous exposure to ligand occurs. Normally seen with membrane-bound G protein-coupled receptors, the activation of a receptor due to ligand binding is quickly followed by the uncoupling of the activated receptor from its G proteins due to phosphorylation of these binding partners (532). Receptor desensitization has been observed for a range of hormones including glucagon, FSH, human chorionic gonadotropin, and prostaglandins (533). Importantly, desensitization and down-regulation can occur in the same cells for the same receptor (534), and therefore, both can play a role in the production of NMDRCs.

5. Receptor competition

Mathematical modeling studies suggest that the mixture of endogenous hormones and EDCs establishes a natural environment to foster NMDRCs. Using mathematical models, Kohn and Melnick (42) proposed that when EDC exposures occur in the presence of endogenous hormone and unoccupied hormone receptors, some unoccupied receptors become bound with the EDC, leading to an increase in biological response (*i.e.* increased expression of a responsive gene, increased weight of an organ, *etc.*). At low concentrations, both the endogenous hormone and the EDC bind to receptors and activate this response, but at high doses, the EDC can outcompete the natural ligand. The model predicts that inverted U-shaped curves would occur regardless of the binding affinity of the EDC for the receptor and would be abolished only if the concentration of natural hormone were raised such that all receptors were bound.

6. Endocrine negative feedback loops

In several cases, the control of hormone synthesis is regulated by a series of positive- and negative feedback loops. Several hormones are known to control or influence their own secretion using these feedback systems. In one example, levels of insulin are known to regulate glucose uptake by cells. Blood glucose levels stimulate insulin pro-

duction, and as insulin removes glucose from circulation, insulin levels decline. Thus, NMDRCs can occur as the free/available ligand and receptor concentrations are influenced by one another. In another example, thyroid hormone secretion is stimulated by TSH, and thyroid hormone suppresses TSH; thus, feedback between these two hormones allows thyroid hormone to be maintained in a narrow dose range.

Several studies indicate that these negative feedback loops could produce NMDRCs when the duration of hormone administration is changed (535). For example, short exposures of estrogen induce proliferation in the uterus and pituitary, but longer hormone regimens inhibit cell proliferation (236, 536). Thus, the outcome is one where exposure to a single hormone concentration stimulates an endpoint until negative feedback loops are induced and stimulation ends (537).

7. Other downstream mechanisms

Removing the variability that can come from examining different cell types, or even single cell types in the context of a tissue, studies of cultured cells indicate that different gene profiles are affected by low doses of hormone compared with higher doses. In a study of the genes affected by low *vs.* higher doses of estrogen, researchers found that there were a small number of genes in MCF7 breast cancer cells with very high sensitivity to low doses of estradiol (10 pM) compared with the total number of genes that were affected by higher (30 or 100 pM) exposures (538). But the surprising finding was the pattern of estradiol-induced *vs.* estradiol-suppressed gene expression at high and low doses; when 10 pM was administered, the number of estradiol-suppressible genes was approximately three times higher than the number of estradiol-inducible genes. However, the overall profile of the number of estradiol-suppressible genes was approximately half the total number of estradiol-inducible genes. This observation suggests that low doses of estrogen selectively target a small subset of the total number of estrogen-sensitive genes and that the genes affected by low doses are most likely to be suppressed by that treatment. The mechanisms describing how low doses of estrogen differently affect the expression of genes compared with higher doses have yet to be elucidated, but low doses of estradiol inhibit expression of apoptotic genes (539), indicating that which genes are affected by hormone exposure is relevant to understand how low doses influence cellular activities.

C. Examples of nonmonotonicity

1. Examples of NMDRCs from cell culture

A tremendous amount of theoretical and mathematical modeling has been conducted to understand the production of nonlinear and nonmonotonic responses (42, 540). These studies and others suggest that the total number of theoretical response curves is infinite. Yet this does not mean that the occurrence of NMDRCs is speculative; these types of responses are reported for a wide variety of chemicals. Cell culture experiments alone provide hundreds of examples of nonmonotonic responses (see Table 6 for examples). In the natural hormone category, many different hormones produce NMDRCs; this is clearly not a phenomenon that is solely attributable to estrogen and androgen, the hormones that have been afforded the most attention in the dose-response literature. Instead, NMDRCs are observed after cells are treated with a range of hormones, suggesting that this is a fundamental and general feature of hormones.

Chemicals from a large number of categories with variable effects on the endocrine system also produce NMDRCs in cultured cells. These chemicals range from components of plastics to pesticides to industrial chemicals and even heavy metals. The mechanisms for nonmonotonicity discussed in *Section III.B* are likely explanations for the NMDRCs reported in a range of cell types after exposure to hormones and EDCs. Table 6 provides only a small number of examples from the literature, and it should be noted that because these are studies of cells in culture, most of these studies typically examined only a few types of outcomes: cell number (which could capture the effects of a chemical on cell proliferation, apoptosis, or both), stimulation or release of another hormone, and regulation of target protein function, often examined by measuring the phosphorylation status of a target.

2. Examples of NMDRCs in animal studies

Some scientists suggest that nonmonotonicity is an artifact of cell culture, however, a large number of NMDRCs have been observed in animals after administration of natural hormones and EDCs, refuting the hypothesis that this is a cell-based phenomenon only. Similar to what has been observed in cultured cells, the NMDRCs observed in animals also span a large range of chemicals, model organisms, and affected endpoints (Table 7). These results underscore the biological importance of the mechanisms of nonmonotonicity that have been largely worked out *in vitro*.

Although NMDRCs attributable to estrogen treatment are well documented, the induction of NMDRCs is again observed to be a general feature of hormone treatment; a wide range of hormones produce these types of responses in exposed animals. Importantly, a number of pharmaceutical compounds with hormone-mimicking or endocrine-disrupting activities also produce NMDRCs. Finally, as expected from the results of cell culture

TABLE 6. Examples of NMDRCs in cell culture experiments

Chemicals by chemical class	Nonmonotonic effect	Cell type	Refs.
Natural hormones			
17β-Estradiol	Cell number	MCF7 breast cancer cells	135, 716
	Dopamine uptake	Fetal hypothalamic cells (primary)	717
	pERK levels, prolactin release	GH3/B6/F10 pituitary cells	41, 718, 719
	β-Hexosaminidase release	HMC-1 mast cells	720
	Cell number	Vascular smooth muscle cells	721
	Production of L-PGDS, a sleep-promoting substance	U251 glioma cells	722
5α-Dihydrotestosterone	Cell number	LNCaP-FGC prostate cancer cells	499
	Cell number, kinase activity	Vascular smooth muscle cells	721
5α-Androstenedione	Cell number	LNCaP-FGC prostate cancer cells	499
Corticosterone	Mitochrondrial oxidation, calcium flux	Cortical neurons (primary)	723
Insulin	Markers of apoptosis (in absence of glucose)	Pancreatic β-cells (primary)	724
Progesterone	Cell number	LNCaP-FGC prostate cancer cells	499
Prolactin	Testosterone release	Adult rat testicular cells (primary)	725
hCG	Testosterone release	Adult rat testicular cells (primary)	725
T_3	Rate of protein phosphorylation	Cerebral cortex cells (primary, synaptosomes)	726
	LPL mRNA expression	White adipocytes (rat primary)	727
GH	*IGF-I* expression	Hepatocytes (primary cultures from silver sea bream)	728
Pharmaceutical hormones			
DES	Cell number	MCF7 breast cancer cells	716
	Prolactin release	GH3/B6/F10 pituitary cells	41
Ethinyl estradiol	CXCL12 secretion	MCF7 breast cancer cells, T47D breast cancer cells	729
R1881 (synthetic androgen)	Cell number	LNCaP-FGC cells	499
Trenbolone	Induction of micronuclei	RTL-W1 fish liver cells	730
Plastics			
BPA	Cell number	MCF7 breast cancer cells	135, 716
	Dopamine efflux	PC12 rat tumor cells	40
	pERK levels, intracellular Ca^{2+} changes, prolactin release	GH3/B6/F10 pituitary cells	41, 718
	Cell number	LNCaP prostate cancer cells	731
DEHP	Number of colonies	*Escherichia coli* and *B. subtilis* bacteria	732
Di-*n*-octyl phthalate	Number of colonies	*E. coli* and *B. subtilis* bacteria	732
Detergents, surfactants			
Octylphenol	Cell number	MCF7 breast cancer cells	716
	Dopamine uptake	Fetal hypothalamic cells (primary)	717
	pERK levels	GH3/B6/F10 pituitary cells	718
	hCG-stimulated testosterone levels	Leydig cells (primary)	733
Propylphenol	pERK levels	GH3/B6/F10 pituitary cells	718
Nonylphenol	pERK levels, prolactin release	GH3/B6/F10 pituitary cells	41, 718
	β-Hexosaminidase release	HMC-1 mast cells	720
	Cell number	MCF7 breast cancer cells	135
PAH			
Phenanthrene	All-trans retinoic acid activity	P19 embryonic carcinoma cells	734, 735
Benz(a)acridine	All-trans retinoic acid activity	P19 embryonic carcinoma cells	734
Naphthalene	hCG-stimulated testosterone	Pieces of goldfish testes	736
B-naphthoflavone	hCG-stimulated testosterone	Pieces of goldfish testes	736
Retene	hCG-stimulated testosterone	Pieces of goldfish testes	736
Heavy metals			
Lead	Estrogen, testosterone, and cortisol levels	Postvitellogenic follicles (isolated from catfish)	737
Cadmium	Expression of angiogenesis genes	Human endometrial endothelial cells	738

(*Continued*)

TABLE 6. Continued

Chemicals by chemical class	Nonmonotonic effect	Cell type	Refs.
Phytoestrogens and natural antioxidants			
Genistein	Cell number	Caco-2BBe colon adenocarcinoma cells	739
	CXCL12 secretion, cell number	T47D breast cancer cells	729
	Cell number, cell invasion, MMP-9 activity	PC3 prostate cancer cells	740
	pJNK levels, Ca^{2+} flux	GH3/B6/F10 pituitary cells	719
Coumesterol	Prolactin release, pERK levels	GH3/B6/F10 pituitary cells	719
Daidezin	Prolactin release, pERK levels	GH3/B6/F10 pituitary cells	719
	Cell number	MCF7 breast cancer cells	135
	Cell number	LoVo colon cancer cells	741
Resveratrol	Expression of angiogenesis genes	Human umbilical vein endothelial cells	742
Trans-resveratrol	pERK levels, Ca^{2+} flux	GH3/B6/F10 pituitary cells	719
Artelastochromene	Cell number	MCF7 breast cancer cells	743
Carpelastofuran	Cell number	MCF7 breast cancer cells	743
Biochanin A	Induction of estrogen-sensitive genes in the presence of testosterone	MCF7 breast cancer cells	744
Licoflavone C	Induction of estrogen-sensitive genes	Yeast bioassay	745
Quercetin	Aromatase activity	H295R adrenocortical carcinoma cells	746
	Cell number	SCC-25 oral squamous carcinoma cells	747
Dioxin			
TCDD	Cell number, gene expression	M13SV1 breast cells	748
PCB			
PCB-74	Cell viability, GnRH peptide levels	GT1-7 hypothalamic cells	749
PCB-118	Cell viability, GnRH peptide levels	GT1-7 hypothalamic cells	749
Aroclor 1242 (PCB mixture)	β-Hexosaminidase release	HMC-1 mast cells	720
POP mixture	Apoptosis of cumulus cells	Oocyte-cumulus complexes (primary, isolated from pigs)	750
Herbicides			
Glyphosphate-based herbicide (Round-Up)	Cell death, aromatase activity, ERβ activity	HepG2 liver cells	751
Atrazine	Cell number	IEC-6 intestinal cells	752
Insecticides			
Endosulfan	Cell number	IEC-6 intestinal cells	752
	β-Hexosaminidase release	HMC-1 mast cells	720
	ATPase activity of P-glycoprotein	CHO cell extracts	753
Diazinon	Cell number	IEC-6 intestinal cells	752
Dieldrin	β-Hexosaminidase release	HMC-1 mast cells	720
DDT	Cell number	MCF7 breast cancer cells	144
DDE	β-Hexosaminidase release	HMC-1 mast cells	720
	Prolactin release	GH3/B6/F10 pituitary cells	41
3-Methylsulfonyl-DDE	Cortisol and aldosterone release, expression of steroidogenic genes	H295R adrenocortical carcinoma cells	754
Fungicides			
Hexachlorobenzene	Transcriptional activity in the presence of DHT	PC3 prostate cancer cells	755
Prochloraz	Aldosterone, progesterone, and corticosterone levels; expression of steroidogenic genes	H295R adrenocortical cells	756
Ketoconazole	Aldosterone secretion	H295R adrenocortical cells	757
Fungicide mixtures	Aldosterone secretion	H295R adrenocortical cells	757
PBDE			
PBDE-49	Activation of ryanodine receptor 1	HEK293 cell (membranes)	758
PBDE-99	Expression of *GAP43*	Cerebral cortex cells (primary)	759

Due to space concerns, we have not elaborated on the shape of the curve (U, inverted U, or other nonmonotonic shape) or the magnitude of observed effects in this table. CXCL12, Chemokine (C-X-C motif) ligand 12; DEHP, bis(2-ethylhexyl) phthalate; DHT, dihydrotestosterone; hCG, human chorionic gonadotropin; MMP, matrix metalloproteinase; PAH, polyaromatic hydrocarbons; PBDE, polybrominated diphenyl ethers; PCB, polychlorinated biphenyl; pERK, phospho-ERK; PGDS, prostaglandin-D synthase; pJNK, phospho-c-Jun N-terminal kinase.

TABLE 7. Examples of NMDRCs in animal studies

Chemicals by chemical class	Nonmonotonic effect	Organ/sex/animal	Refs.
Natural hormones			
17β-Estradiol	Morphological parameters	Mammary gland/female/mice	138, 541
	Accumulation of cAMP	Pineal/female/rats	760
	Prostate weight	male/mice	689
	Uterine weight	female/mice	761
	Antidepressant effects, measured by immobility assay	Behavior/male/mice	762
	Nocturnal activity, gene expression in preoptic area	Brain and behavior/female/mice	763
Corticosterone	Spatial memory errors	Behavior/male/rats	764
	Cholinergic fiber loss in cortex after treatment with neurodegenerative drugs	Brain/male/rats	765
	Mitochondrial metabolism	Muscle/male/rats: strain differences	766
	Contextual fear conditioning	Behavior/male/rats	767
	Locomotor activity	Behavior/male/captive Adelie penguins	768
Glucocorticoid	Na^+/K^+-ATPase activity	Brain/tilapia (fish)	769
Testosterone	Na^+/K^+-ATPase activity	Brain/tilapia (fish)	769
	Gonadotropin subunit gene expression	Pituitary/sexually immature goldfish	770
11β-Hydroxyandrosterone	Gonadotropin subunit gene expression	Pituitary/sexually immature goldfish	770
T_4	Bone growth	Tibia/male/rats with induced hypothyroidism	771
Leptin	Insulin production (in the presence of glucose)	Pancreas/male/rats	560
Oxytocin	Infarct size, plasma LDH levels, creatine kinase activity after ischemia/ reperfusion injury	Brain and blood/male/rats	772
	Memory retention	Behavior/male/mice	773
Melatonin	Brain infarction and surviving neuron number after injury	Brain/female/rats	774
Dopamine	Memory	Brain/both/rhesus monkey	775
	Neuronal firing rate	Brain/male/rhesus monkey	776
Pharmaceutical			
DES	Sex ratio, neonatal body weight, other neonatal development	Mice	777
	Adult prostate weight	Male/mice	689
	Uterine weight	Female/mice	761
	Expression of PDGF receptor	Testes/male/rats	778
	Morphological parameters	Mammary gland/male and female/ mice	779
Estradiol benzoate	Dorsal prostate weight, body weight	Male/rats	780
	Sexual behaviors, testes morphology	Male/zebra finches (birds)	781
Ethinyl estradiol	GnRH neurons	Brain/zebrafish	782
Tamoxifen	Uterine weight	Female/mice	761
Fluoxetine (antidepressant)	Embryo number	*Potamopyrgus antipodarum* (snails)	783
Fadrozole (aromatase inhibitor)	Aromatase activity	Ovary/female/fathead minnows	784
Plastics			
BPA	Fertility	Reproductive axis /female/mice	316
	Reproductive behaviors	Behavior/male/rats	785
	Protein expression	Hepatopancreas/male/*Porcellio scaber* (isopod)	786
	Timing of vaginal opening, tissue organization of uterus	Reproductive axis/female/mice	577
	Expression of receptors in embryos	Brain and gonad/both/ mice	787
DEHP	Aromatase activity	Hypothalamus/male/rats	788
	Cholesterol levels	Serum/male/rats	569
	Timing of puberty	Reproductive axis /male/rats	789
	Body weight at birth, vaginal opening, and first estrous	Female/rats	790
	Seminal vesicle weight, epididymal weight, testicular expression of steroidogenesis genes	Male/rats	791
	Responses to allergens, chemokine expression	Skin/male/mice	792

(*Continued*)

TABLE 7. Continued

Chemicals by chemical class	Nonmonotonic effect	Organ/sex/animal	Refs.
Detergents, surfactants			
Nonylphenol ethoxylate	Fecundity	*Biomphalaria tenagophila* (snails)	793
Octylphenol	Embryo production	*P. antipodarum* (snails)	794
	Spawning mass and egg numbers	*Marisa cornuarietis* (snails)	795
Semicarbazide	Timing of preputial separation, serum DHT	Male/rats	796
Antimicrobial			
Triclocarban	Fecundity	*P. antipodarum* (snails)	797
PCB			
Mixture of PCB	Corticosterone levels	Male/kestrels (birds)	798
Environmental PCB mixture	Corticosterone levels	Female/tree swallows (birds)	799
UV filters			
Octyl methoxycinnamate	Activity, memory	Behavior/both/rats	800
Aromatic hydrocarbons			
B-naphthoflavone	Testosterone	Plasma/male/goldfish	736
Toluene	Locomotor activity	Behavior/male/rats	801
Dioxins			
TCDD	Cell-mediated immunity	Immune system/male/ rats	802
	Proliferation after treatment with chemical carcinogen	Liver/female/rats	803
Heavy metals			
Cadmium	Expression of metallothionein, *pS2/TFF1*	Intestine and kidney/ female/rats	804
	Activity of antioxidant enzymes	Earthworms	805
	Size parameters, metamorphic parameters	*Xenopus laevis*	806
Lead	Growth, gene expression	*Vicia faba* seedlings (plant)	807
	Retinal neurogenesis	Eye and brain/female/rats	808
Selenium	DNA damage, apoptotic index	Prostate/male/dogs	809
	Hatching failure	Eggs/red-winged blackbirds (wild population)	810
Phytoestrogens			
Genistein	Aggressive, defensive behaviors	Behavior/male/mice	811
	Retention of cancellous bone after ovariectomy	Tibia bones/female/rat	812
	Expression of *OPN*, activation of Akt	Prostate/male/mice	740
Resveratrol	Angiogenesis	Chorioallantoic membrane/chicken embryos	742
	Ulcer index after chemical treatment, expression of gastroprotective genes	Stomach/male/mice	813
Phytochemicals			
Phlorizin	Memory retention	Behavior/male/mice	814
Herbicides			
Atrazine	Time to metamorphosis	Thyroid axis/*Rhinella arenarum* (South American toad)	815
	Survivorship patterns	Four species of frogs	363
	Growth parameters	*Bufo americanus*	816
Pendimethalin	Expression of *AR*, *IGF-I*	Uterus/female/mice	817
Commercial mixture with mecoprop, 2,4-dichlorophenoxyacetic acid and dicamba	Number of implantation sites, number of live births	Female/mice	818
Simazine	Estrous cyclicity	Reproductive axis/female/rat	819
Insecticides			
Permethrin	Dopamine transport	Brain/male/mice	820
Heptachlor	Dopamine transport	Brain/male/mice	820
DDT	Number of pups, sex ratios, neonatal body weight, male anogenital distance	Mice	777
Methoxychlor	Number of pups, anogenital distance (males and females), neurobehaviors (males and females)	Mice	777
Chlorpyrifos	Body weight	Male/rats	821
	Antioxidant enzyme activity	*Oxya chinensis* (locusts)	822
Malathion	Antioxidant enzyme activity	*O. chinensis* (locusts)	822

(*Continued*)

TABLE 7. Continued

Chemicals by chemical class	Nonmonotonic effect	Organ/sex/animal	Refs.
Fungicides			
Carbendazim	Liver enzymes, hematology parameters	Blood and liver/male/rats	823
Chlorothalonil	Survival, immune response, corticosterone levels	Several amphibian species	686
Vinclozolin	Protein expression	Testes/male/*P. scaber* (isopod)	786

Due to space concerns, we have not elaborated on the shape of the curve (U, inverted U, or other nonmonotonic shape) or the magnitude of observed effects in this table. DEHP, Bis(2-ethylhexyl) phthalate; DHT, dihydrotestosterone; LDH, lactate dehydrogenase; PCB, polychlorinated biphenyl; PDGF, platelet-derived growth factor.

experiments, chemicals with many different modes of action generate NMDRCs in treated animals.

Perhaps most striking is the range of endpoints affected, from higher-order events such as the number of viable offspring (which could be due to alterations in the reproductive tissues themselves or the reproductive axis), to behavioral effects, to altered organ weights, and to lower-order events such as gene expression. The mechanisms responsible for these nonmonotonic phenomena may be similar to those studied in cell culture systems, although additional mechanisms are likely to be operating *in vivo* such as alterations in tissue organization (541) and the interactions of various players in the positive and negative feedback loops of the endocrine system.

3. Examples of NMDRCs in the epidemiology literature

Perhaps not surprisingly, natural hormones produce NMDRCs in human populations as well (Table 8). Although the methods needed to detect NMDRCs in humans are specific to the field of epidemiology, these results sup-

TABLE 8. NMDRCs for natural hormones identified in the epidemiology literature

Hormone	Affected endpoint	NMDRC	Study subjects	Refs.
Testosterone (free)	Incidence of coronary events	Incidence of 25% at extremes of exposure, 16% at moderate exposure	Rancho Bernardo Study participants, women aged 40+ (n = 639)	824
	Depression	Hypo- and hypergonadal had higher depression scores than those with intermediate free testosterone	Androx Vienna Municipality Study participants, manual workers, men aged 43–67 (n = 689)	825
PTH	Mortality	~50% excess risk for individuals with low or high iPTH	Hemodialysis patients (n = 3946)	826
	Risk of vertebral or hip fractures	~33% higher for low or high iPTH compared to normal levels	Elderly dialysis patients (n = 9007)	827
TSH	Incidence of Alzheimer's disease	About double the incidence in lowest and highest tertile in women (no effects observed in men)	Framingham Study participants (elderly) (n = 1864, 59% women)	828
Leptin	Mortality	Mortality ~10% higher for lowest and highest leptin levels	Framingham Heart Study participants (elderly) (n = 818, 62% women)	563
Insulin	Coronary artery calcification	Higher for low and high insulin area under the curve measures.	Nondiabetic patients with suspected coronary heart disease, cross-sectional (n = 582)	829
	Mortality (noncardiovascular only)	Relative risk ~1.5 for highest and lowest fasting insulin levels	Helsinki Policemen Study participants, men aged 34–64 (n = 970)	830
Cortisol	BMI, waist circumference	Low cortisol secretion per hour for individuals with highest and lowest BMI, waist circumference	Whitehall II participants, adults, cross-sectional (n = 2915 men; n = 1041 women)	831
	Major depression (by diagnostic interview)	Slight increases at extremes of cortisol	Longitudinal Aging Study Amsterdam participants, aged 65+, cross-sectional (n = 1185)	832

BMI, Body mass index; iPTH, intact PTH; PTH, parathyroid hormone.

port the idea that NMDRCs are a fundamental feature of hormones. Importantly, it should be noted that most of the individuals surveyed in studies examining the effects of natural hormones have a disease status or are elderly. This of course does not mean that natural hormones induce NMDRCs in only these select populations but may instead be a reflection of the types of individuals available for these studies (for example, there are very few clinical events in younger people).

NMDRCs observed in the epidemiology literature from human populations exposed to EDCs are now starting to receive attention (Table 9). Here, most reports of NMDRCs come from studies of healthy individuals exposed to persistent organic pollutants POPs, chemicals that do not easily degrade and consequently bioaccumulate in human and animal tissues (542). These POPs do encompass a range of chemical classes including components of plastics, pesticides, and industrial pollutants. A large number of these studies have focused on endpoints that are relevant to metabolic disease, and together, these studies show that there is a recurring pattern of NMDRCs related to POPs and disease. Of course, not every study of POPs shows NMDRCs, and this is probably due to the distribution of EDCs in the populations examined.

In addition to the studies that show strong evidence for NMDRCs in human populations, there is also a subset of studies that provide suggestive evidence for nonmonotonic relationships between EDCs and human health endpoints (Table 9). In fact, the authors of many of these papers clearly identify U- or inverted U-shaped dose-response curves. However, when authors do not perform the appropriate statistical tests to verify the presence of a NMDRC, there is some ambiguity in their conclusions. The usual cross-sectional *vs.* prospective design dichotomy in epidemiology also is a factor that can influence the strength of a NMDRC, or prevent the detection of one at all. This disjunction in design is often incongruous with EDC exposure studies because we often know very little about clearance rates of the chemical, interactions with adiposity, and changes to these factors with age and gender. Yet regardless of any possible weaknesses in these studies, they provide supportive evidence that NMDRCs are observed in human populations.

Because these reports of NMDRCs in human populations are relatively new, few mechanisms have been proposed for these phenomena. Why would risk curves be nonmonotonic over the dose distribution observed in human populations? Why would individuals with the highest exposures have less severe health outcomes compared with individuals with more moderate exposures? One plausible explanation is that the same mechanisms for NMDRCs in animals and cell cultures operate in human populations: chronic exposures to high doses can activate negative feedback loops, activate receptors that promote changes in different pathways that diverge on the same endpoint with opposing effects, or produce some measure of toxicity. Accidental exposures of very large doses may not behave the same as background doses for a variety of reasons, including the toxicity of high doses; these large doses tend to occur over a short time (and therefore more faithfully replicate what is observed in animal studies after controlled administration).

Another explanation is that epidemiology studies, unlike controlled animal studies, examine truly complex mixtures of EDCs and other environmental chemicals. Some chemical exposures are likely to be correlated due to their sources and their dynamics in air, water, soil, and living organisms that are subsequently eaten. Therefore, intake of these chemicals may produce unpredicted, likely nonlinear outcomes whether the two chemicals act via similar or different pathways.

The design of observational epidemiological studies is fundamentally different from studies of cells or animals, in that the EDC exposure distributions are given, rather than set by the investigator. In particular, as shown in Fig. 5, different epidemiological populations will have different ranges of exposure, with the schematic example showing increasing risk in a population with the lowest exposures (labeled group A), an inverted U-shaped risk in a moderate dose population (labeled group B), and an inverse risk in a population with the highest exposures (labeled group C). An additional example is provided (labeled group D) in which an industrial spill shows high risk, but the comparison with the entire unaffected population with a wide variety of risk levels due to differential background exposure could lead to a high- or a low-risk reference group and a wide variety of possible findings.

It is reasonable to suggest that even though epidemiological studies are an assessment of exposures at a single time point, many of these pollutants are persistent, and therefore a single measure of their concentration in blood may be a suitable surrogate for long-term exposures. The movement of people from relatively low- to higher-exposure groups over time depend on refreshed exposures, clearance rates, and individual differences in ability to handle exposures (*i.e.* due to genetic susceptibilities, amount of adipose tissue where POPs can be stored, *etc.*).

Figure 5 therefore further illustrates that observational epidemiological studies yield the composite effect of varying mixtures of EDCs at various exposure levels for various durations, combining acute and chronic effects. These studies are important, however, in that they are the only way to study EDC effects in the long term in intact humans, as opposed to studying signaling pathways, cells,

TABLE 9. NMDRCs for EDCs identified in the epidemiology literature

Chemicals by chemical class	Affected endpoint	NMDRC	Study subjects	Refs.
Insecticides				
Trans-nonachlor	Diabetes incidence	Highest risk in groups with intermediate exposures (quartile 2)	CARDIA participants, case-control study (n = 90 cases and n = 90 controls)	833
	Telomere length in peripheral leukocytes	Increased length in intermediate exposures (quintile 4)	Adults aged 40+ (Korea, n = 84)	591
p,p′-DDE	BMI, triglyceride levels, HDL cholesterol	Highest risk in groups with intermediate exposures (quartile 3)	CARDIA participants (n = 90 controls from nested case control study)	590
	Risk of rapid infant weight gain	For infants born to women of normal weight prepregnancy, risk is highest with intermediate exposures.	Infants from Childhood and the Environment project, Spain (n = 374 from normal prepregnancy weight mothers; n = 144 from overweight mothers)	834
	Telomere length in peripheral leukocytes	Increased length with intermediate exposures (quintile 4)	Adults aged 40+ (Korea, n = 84)	591
Oxychlordane	Bone mineral density of arm bones	With low exposures, fat mass had inverse associations with bone mineral density; with high exposures, fat mass had positive associations with bone mineral density.	NHANES 1999–2004 participants, aged 50+ (n = 679 women, n = 612 men)	835
Plastics				
Mono-methyl phthalate (MMP)	Atherosclerotic plaques	Increased risk in intermediate exposure groups (quintiles 2–4)	Adults aged 70, living in Sweden (n = 1016)	836
Perfluorinated compounds				
PFOA	Arthritis (self-reported)	Increased risk in intermediate exposure groups (quartile 2)	NHANES participants, aged 20+ (both sexes, n = 1006)	837
Fire retardants				
PBB-153	Blood triglyceride levels	Increased risk in intermediate exposure groups (quartile 2)	NHANES participants, aged 12+ (n = 637)	604
PBDE-153	Prevalence of diabetes,	Prevalence of diabetes highest in intermediate groups (quartiles 2–3 relative to individuals with undetectable levels)	NHANES participants, aged 12+ (n = 1367)	604
	Prevalence of metabolic syndrome, levels of blood triglycerides	Prevalence of metabolic syndrome highest in intermediate exposure groups (quartile 2 relative to individuals with undetectable levels); blood triglycerides highest in low exposure groups (quartile 1 relative to individuals with undetectable levels)	NHANES participants, aged 12+ (n = 637)	604
PCB				
PCB-74	Triglyceride levels	Lowest levels are observed in intermediate groups (quartile 2)	CARDIA participants (n = 90 controls from nested case-control study)	590
PCB-126	Bone mineral density in right arm	With low exposures, fat mass had inverse associations with bone mineral density; with high exposures, fat mass had positive associations with bone mineral density	NHANES participants, aged <50 (n = 710 women, n = 768 men)	835
PCB-138	Bone mineral density in right arm	With low exposures, fat mass had inverse associations with bone mineral density; with high exposures, fat mass had positive associations with bone mineral density	NHANES participants, women aged 50+ (n = 679 women, n = 612 men)	835
PCB-153	Telomere length in peripheral leukocytes	Increased length with intermediate exposure groups (quintile 4)	Adults aged 40+ (Korea, n = 84)	591
PCB-170	Diabetes incidence	Highest risk in groups with intermediate exposures (quartile 2)	CARDIA participants, case-control study (n = 90 cases and n = 90 controls)	833
	Endometriosis	Decreased risk in groups with intermediate exposures (quartile 3)	Participants from the Women at Risk of Endometriosis (WREN) study, 18–49 yr old, case-control study (n = 251 cases; n = 538 controls)	838
PCB-172	DNA hypomethylation (by Alu assay)	Highest levels of hypomethylation in groups with lowest and highest exposures	Adults aged 40+ (Korea, n = 86)	839
PCB-180[a]	BMI	Highest BMI with intermediate exposures (quartile 2)	CARDIA participants (n = 90 controls from nested case control study)	590
PCB-187[a]	HDL cholesterol levels	Lowest levels with intermediate exposures (quartile 2)	CARDIA participants (n = 90 controls from nested case control study)	590
PCB 196–203	Diabetes incidence	Highest risk in groups with intermediate exposures (quartile 2)	CARDIA participants, case-control study (n = 90 cases and n = 90 controls)	833
PCB-196	Endometriosis	Decreased risk in groups with intermediate exposures (quartile 3)	Participants from the Women at Risk of Endometriosis (WREN) study, 18–49 yr old, case-control study (n = 251 cases; n = 538 controls)	838

(*Continued*)

TABLE 9. Continued

Chemicals by chemical class	Affected endpoint	NMDRC	Study subjects	Refs.
PCB-199[a]	Triglyceride levels	Highest risk in groups with intermediate exposures (quartiles 2–3)	CARDIA participants (n = 90 controls from nested case control study)	590
PCB-201	Endometriosis	Decreased risk in groups with intermediate exposures (quartiles 2–3)	Participants from the Women at Risk of Endometriosis (WREN) study, 18–49 yr old, case-control study (n = 251 cases, n = 538 controls)	838
Heavy metals				
Selenium	Fasting glucose levels (by modeled exposure)	Intermediate exposures have highest fasting glucose levels	NHANES 2003- 2004 participants, aged 40+ (n = 917)	840
	Glycosylated hemoglobin (by modeled exposure)	Intermediate exposures have highest % glycosylated hemoglobin	NHANES 2003- 2004 participants, aged 40+ (n = 917)	840
	Diabetes incidence (by modeled exposure)	Intermediate exposures have highest risk for diabetes	NHANES 2003- 2004 participants, aged 40+ (n = 917)	840
	Blood triglyceride levels	Intermediate exposures have highest triglyceride levels	NHANES participants, aged 40+ (n = 1159)	841
Arsenic	Cytokines in umbilical cord blood	Lower inflammatory markers at intermediate exposures (quartile 2)	Pregnant women in Bangladesh (n = 130)	842
Manganese	Mental development scores in infants and toddlers	Intermediate exposures had highest mental development scores at 12 months of age; association lost in older toddlers	12-month-old infants, Mexico (n = 301)	843
	Sperm count, motility and morphology	Intermediate doses had lowest sperm counts and motility; intermediate doses also had the worst sperm morphologies	Men aged 18–55 (infertility clinic patients, n = 200)	844
Mixtures				
31 POP	Diabetes incidence	Highest incidence in intermediate groups (sextiles 2–3)	CARDIA participants, case-control study (n = 90 cases and n = 90 controls)	833
16 POP	Diabetes incidence	Highest incidence in intermediate groups (sextiles 2–3)	CARDIA participants, case-control study (n = 90 cases and n = 90 controls)	833
Non-dioxin-like PCB (mix)	Metabolic syndrome	Highest incidence in intermediate groups (quartile 3)	NHANES 1999–2002 participants, aged 20+ (n = 721)	845
Dioxin-like PCB (mix)	Triacylglycerol levels by quartile of exposure	Highest levels in intermediate groups (quartile 3)	NHANES 1999–2002 participants, aged 20+ (n = 721)	845
Additional supportive evidence for NMDRC in the epidemiology literature				
Insecticides				
Heptachlor epoxide	Prevalence of newly diagnosed hypertension	Highest risk in intermediate groups (quartile 2); other endpoints do not have NMDRC	NHANES participants, women aged 40+, cross-sectional (n = 51 cases, n = 278 total)	26
β-Hexachloro-cyclohexane	Triacylglycerol levels by quartile of exposure	Highest risk in intermediate group (quartile 2)	NHANES participants, aged 20+ (n = 896 men, 175 with metabolic syndrome)	845
Plastics				
Mono-*N*-butyl phthalate (MBP)	BMI, age-specific effects	Effects seen only in elderly participants (age 60–80); risk is lowest in quartile 3	NHANES male participants (n = 365; age 60–80)	470
Mono-benzyl phthalate (MBzP)	BMI, age-specific effects	Effects seen only in young participants (age 6–11); risk is highest in quartiles 2–3	NHANES participants (both sexes, n = 329 males; n = 327 females)	470
Flame retardants				
PFOA	Thyroid disease (self-reported)	Lowest risk in intermediate groups (quartile 3)	NHANES 1999–2000, 2003–2006 participants, males aged 20+ (n = 3974)	837
Dioxin and related compounds				
TCDD	Age at natural menopause	Highest for intermediate exposure group (quintile 4)	Highly exposed women; Seveso Women's Health Study participants (n = 616)	468
HCDD	Bone mineral density in right arm by quintile of fat mass	With low exposures, fat mass had inverse associations with bone mineral density; with high exposures, fat mass had positive associations with bone mineral density	NHANES participants, women aged 50+ (n = 679 women, n = 612 men)	835
Heavy metals				
Selenium	Prevalence of peripheral artery disease	Disease prevalence decreased in intermediate doses, then increased gradually with higher doses	NHANES participants, aged 40+ (n = 2062)	469

BMI, Body mass index; HCDD, hexachloro-dibenzo-p-dioxin; HDL, high-density lipoprotein; PCB, polychlorinated biphenyls; PFOA, perfluorooctanoic acid; PBB, polybrominated biphenyl; PBDE, polybrominated diphenyl ethers; POP, persistent organic pollutants.

[a] In many cases, multiple chemicals in the same class had similar effects. A few chemicals were selected to illustrate the observed effect. This list is not comprehensive.

organs, or animal models over limited periods of time. Causal inference is not done directly from the epidemiological study results; instead, it is done via combining information from the epidemiological observations with findings from the detailed studies of pathways and animals.

We have suggested that NMDRCs are a fundamental and general feature of hormone action in cells and animals.

Figure 5.

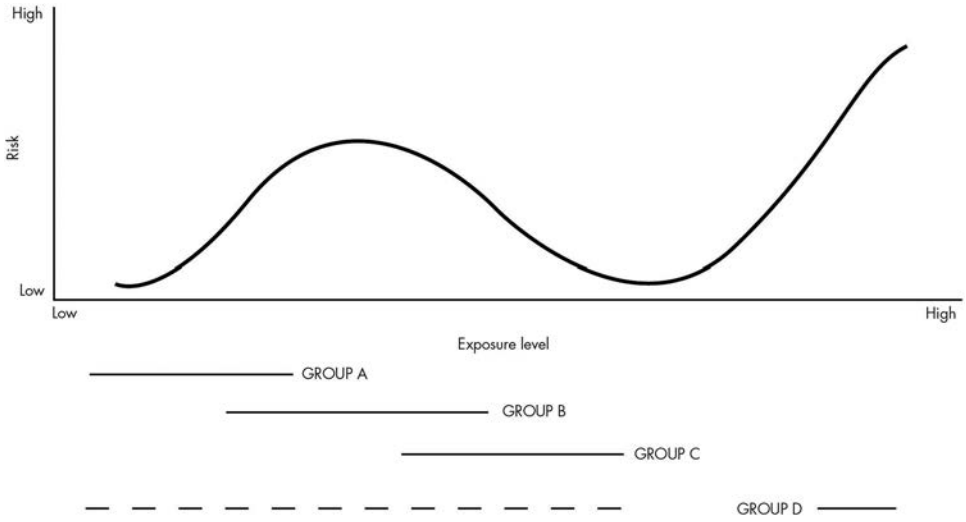

Figure 5. Example of a NMDRC in humans and the sampling populations that could be examined in epidemiology studies. This schematic illustrates a theoretical NMDRC in a human population. If a study were to sample only group A, the conclusion would be that with increasing exposures, risk increases monotonically. Sampling group B would allow researchers to conclude that there is a nonmonotonic relationship between exposure level and risk. If a study included only group C, the conclusion would be that with increasing exposures, there is decreased risk of disease. Group D represents a population that was highly exposed, *i.e.* due to an industrial accident. This group has the highest risk, and there is a monotonic relationship between exposures and risk, although risk is high for all individuals. In the group D situation, there is generally a background population with which high-dose exposure is compared (*dotted line*); relative risk for group D would depend on whether that background population resembles group A, B, or C. From this example, it is clear that the population sampled could strongly influence the shape of the dose-response curve produced as well as the conclusions reached by the study.

It is therefore worth asking whether NMDRCs are expected in the epidemiology literature. The endpoints assessed in epidemiology studies are typically integrated effects, rather than short-term effects; therefore, the various cell- or organ-specific effects may cancel each other, particularly if they are NMDRCs (because they are unlikely to all have nonmonotonicity at the same dose and direction). Thus, NMDRCs are likely to be rarer in the epidemiology literature compared with studies examining the effects of a wide range of doses of an EDC on animals and cultured cells. Yet it is also important to ask what can be concluded if a NMDRC is detected in one epidemiology study but not in others examining the same chemical and outcome. There are several factors that must be considered. The first is that differences in the populations examined between the two studies could explain why a monotonic relationship is observed in one group and a nonmonotonic relationship in another (see Fig. 5). The second is that one or more studies may not be statistically designed to detect NMDRCs. Finally, it is plausible that the NMDRC is an artifact due to residual confounding or some other factor that was not considered in the experimental design. As more becomes known about the mechanisms operating in cells, tissues, and organs to generate NMDRCs, our ability to apply this information to epidemiology studies will increase as well.

4. Tamoxifen flare, a NMDRC observed in cells, animals, and human patients

Although there is controversy in toxicology and risk assessment for endocrine disruptors, NMDRCs are recognized and used in current human clinical practice, although under a different specific term, flare. Flare is often reported in the therapy of hormone-dependent cancers such as breast and prostate cancer. Clinically, failure to recognize the NMDRC that is termed a flare would be considered malpractice in human medicine.

Tamoxifen flare was described and named as a transient worsening of the symptoms of advanced breast cancer, particularly metastases to bone associated with increased pain, seen shortly after the initiation of therapy in some patients (543). If the therapy could be continued, the patients showing tamoxifen flare demonstrated a very high likelihood of subsequent response to tamoxifen, including arrest of tumor growth and progression of symptoms for some time.

The subsequent mechanism of the flare was described in basic lab studies in athymic mouse models of human hormone-dependent breast cancer xenografts (544) and in tissue culture of hormone-dependent human breast cancer cells (545–547). In these models, it was observed that although high, therapeutic concentrations of tamoxifen inhibited estrogen-stimulated proliferation of breast cancer cells, lower concentrations of tamoxifen actually stimulated breast can-

Figure 6.

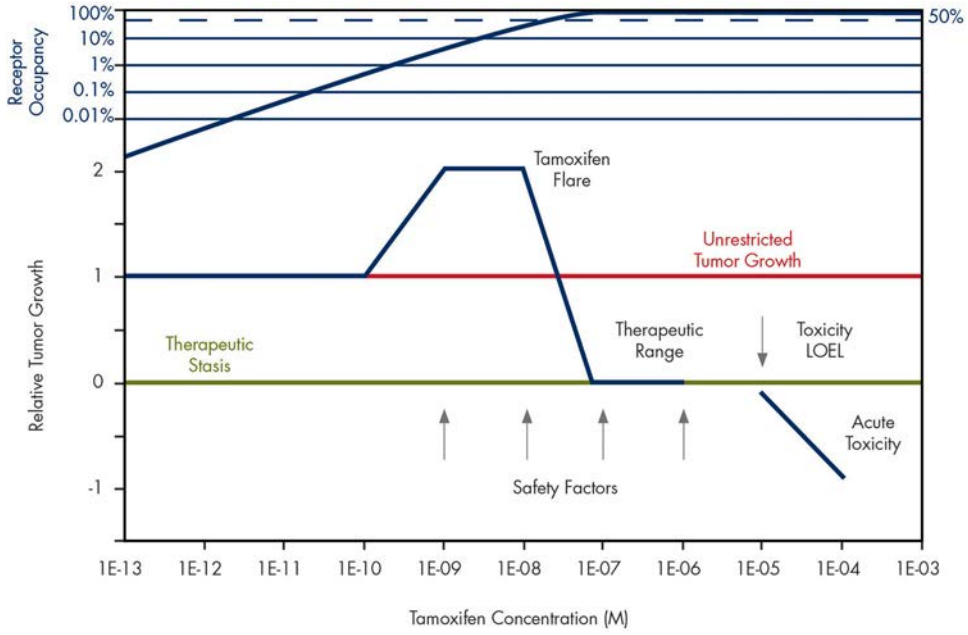

Figure 6. Dose-response ranges for tamoxifen in breast cancer therapy. This figure demonstrates the NMDRC, also called flare, in tamoxifen treatments. As the circulating dose of tamoxifen increases when treatment starts, patients initially experience flare, *i.e.* growth of the tumor (546), followed by a decrease in tumor size as the circulating levels of tamoxifen rise into the therapeutic range (676, 677). High doses of tamoxifen are acutely toxic (546). Starting from the highest concentrations, where acute toxicity is observed, and going to lower concentrations on the X-axis, the acute toxicity diminishes towards zero growth, *i.e.* therapeutic stasis (*green* baseline). This occurs at approximately 1E-05 m, the lowest observed effect level (LOEL) for toxicity. The vertical arrows show the results of applying three or four 10-fold safety factors to the LOEL for the high-dose toxicity of tamoxifen, and would calculate a safe or reference dose for tamoxifen in the region of flare, the least safe region of exposure in actual practice. Above the diagram of dose response ranges is estimated ER occupancy by tamoxifen. This was calculated from the affinity constant of tamoxifen for ERs determined in human breast cancer cells (Ki = 29.1 nM; Ref 678); flare appears to correspond to low receptor occupancy (*blue axis*), therapeutic range with mid and upper-range receptor occupancy, and acute toxicity well above 99% receptor occupancy. (678).

cer cell growth as long as the cells were estrogen dependent (548). Tamoxifen was also shown to disrupt tissue organization of the mammary gland, with specific effects on the stroma that may contribute to the observed effects on proliferation of epithelial cells (549, 550).

Tamoxifen therapy is administered as 10 mg twice per day (20 mg/d; approx 0.3 mg/kg body weight per day), but the target circulating levels are in the near submicromolar range (0.2–0.6 μM); these levels are reached slowly, after approximately 2 wks of therapy (551). In the initial period, where tamoxifen flare is observed, the circulating concentrations are ascending through lower concentrations, in the range below therapeutic suppression of growth, where breast cancer cell proliferation is actually stimulated by the drug, both in tissue culture, in animal xenograft studies, and in human patients (reviewed in Ref. 548). The recognition of this dual dose-response range for tamoxifen (low-dose, low-concentration estrogenic growth-stimulatory and higher-dose, higher-concentration estrogenic growth-inhibitory responses) led to the definition of the term selective estrogen response modulator, or SERM, activity (552–554). This SERM activity has since been observed for many or even most estrogenic EDCs, including BPA (3, 555–557).

These observations defined three separate dose-response ranges for the SERM tamoxifen in human clinical use. The lowest dose-response range, the range of flare, stimulated breast cancer growth and symptoms in some patients with hormone-dependent cancer. The next higher dose-response range is the therapeutic range where tamoxifen inhibits estrogen-dependent tumor growth. The highest dose range causes acute toxicity by the SERM (see Fig. 6).

Tamoxifen provides an excellent example for how high-dose testing cannot be used to predict the effects of low doses. For tamoxifen (as for other drugs), the range of acute human toxicity for tamoxifen was determined in phase I clinical trials. Phase I trials also defined an initial therapeutic range, the second dose-response range, as a dose below which acute toxicity was not observed. The therapeutic dose range was tested and further defined in phase II and later clinical trials to determine efficacy (see for example Ref. 558). Standard toxicological testing from

Figure 7.

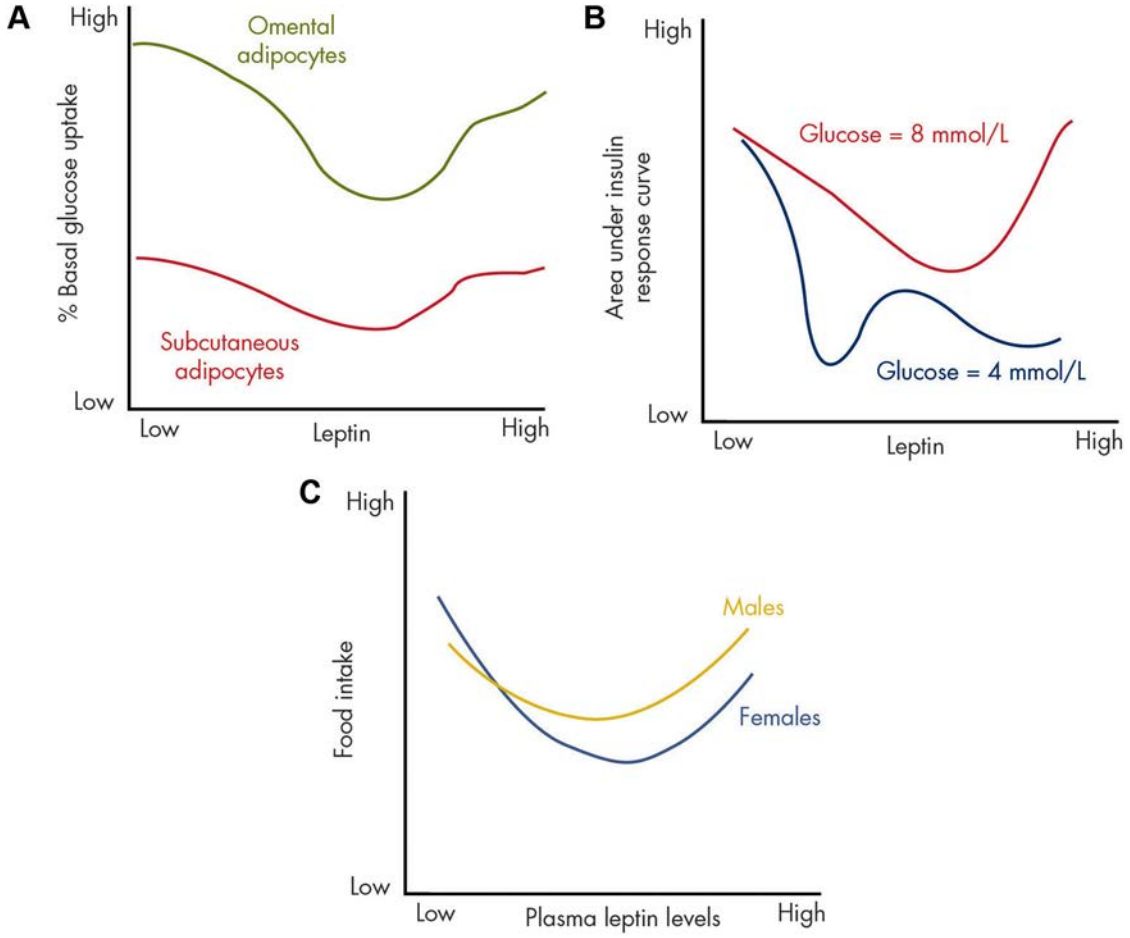

Figure 7. Leptin as an example of a NMDRC. Several studies report NMDRCs in response to leptin treatments. A, NMDRCs are observed in cultured primary adipocytes after leptin exposure. This graph illustrates the relationship between administered leptin dose and glucose uptake in two types of adipocytes, those isolated from omental tissue (*green*) and others from sc fat (*purple*) (schematic was made from data in Ref. 559). These data are on a log-linear plot. B, *Ex vivo* rat pancreas was treated with leptin and various doses of glucose, and the insulin response curves were examined. Area under the curve is a measure of the ability of the pancreas to bring glucose levels under control. Different dose-response curves were observed depending on the amount of glucose administered: a U-shaped curve when 8 mmol/liter was included (*pink*) or a multiphasic curve with 4 mmol/liter (*blue*) (schematic made from data in Ref. 560). These data are on a linear-linear plot. C, U-shaped NMDRCs were also observed when food intake was compared with leptin levels in the blood of rats administered the hormone. This response was similar in males (*orange*) and females (*cyan*) (schematic made from data in Ref. 562). These data are on a linear-linear plot.

high doses to define a LOAEL or NOAEL are equivalent to the phase I clinical testing, and in risk assessment, a safe dose or reference dose is calculated from these tests. However, the lowest dose range, with the highly adverse effects termed flare, was not detected in the phase I trials and was determined only for tamoxifen in breast cancer therapy at the therapeutic doses (543). The implication for risk assessment is that NMDRCs for EDCs, particularly those already identified as SERMs, would likely not be detected by standard toxicological testing at high doses. That is, the consequence of high-dose testing is the calculation of a defined but otherwise untested safe dose that is well within the range equivalent to flare, *i.e.* a manifestly unsafe dose of the EDC (Fig. 6).

5. Similarities in endpoints across cell culture, animal, and epidemiology studies: evidence for common mechanisms?

There are common trends in some findings of NMDRCs in cell, animal, and human studies and therefore evidence for related mechanisms for NMDRCs at various levels of biological complexity. Tamoxifen flare, discussed in *Section III.C.4*, is an informative example. Another illustrative example is that of the effect of the hormone leptin (Fig. 7). In cultured primary adipocytes, NMDRCs are observed after leptin exposure; moderate doses of leptin significantly reduce insulin-mediated glucose intake, whereas low and high doses maintain higher glucose intake in response to insulin (559). The rat pancreas shows a similar response to leptin; the amount of

secreted insulin has an inverted U-shaped response to leptin (560, 561). Even more striking is the relationship between leptin and food intake. Rats administered moderate doses of leptin consume less food compared to rats dosed with low or high levels of leptin (562); mechanistically, this lower food intake could be due to higher circulating glucose levels in these animals due to ineffective insulin action. And finally, in a human study, leptin levels were found to correlate with body mass index but have a U-shaped relationship with mortality (563). These results suggest that hormones can produce similar responses at several levels of biological complexity (cell, organ, animal, and population).

A large number of epidemiology studies with NMDRCs have found relationships between EDC exposures like POPs and metabolic diseases including obesity and diabetes (Table 9) (see also Ref. 564 for a review), and the mechanisms for these relationships have begun to be explored. Human and animal cells treated with EDCs in culture display NMDRCs that are relevant to these diseases: BPA has nonmonotonic effects on the expression of adipocyte proteins in preadipocytes and the release of adiponectin from mature adipocytes (565–567). Similarly, in female rodents, low doses but not high doses of BPA increased adipose tissue weight and serum leptin concentrations (568), and intermediate doses of phthalates decrease serum cholesterol levels (569). Thus, although understanding the mechanisms operating at the cellular level of organization has not yet led to definitive knowledge of the mechanisms producing NMDRCs in human populations, there appear to be strong similarities in cells, animals, and humans that support a call for continued work focusing on metabolic disease endpoints at each level of biological organization.

D. NMDRC summary

We have demonstrated that nonmonotonicity is a common occurrence after exposures to hormones and EDCs in cell culture and animals and across human populations. Because of the abundance of examples of NMDRCs, we expect that if adequate dose ranges are included in animal and cell culture studies, including the use of negative and well-chosen positive controls, NMDRCs may be observed more often than not. Here, we have focused mainly on studies that examined a wide range of doses, including many that examined the effects of doses that span the low-dose and toxicological ranges. We also discussed several mechanisms that produce NMDRCs. Each of these mechanisms can and does operate at the same time in a biological system, and this cooperative action is ultimately responsible for NMDRCs.

Understanding nonmonotonicity has both theoretical and practical relevance. When a chemical produces monotonic responses, all doses are expected to produce similar effects whose magnitude varies with the dose, but when a chemical produces a NMDRC, dissimilar or even opposite effects will be observed at different doses. Thus, monotonic responses can be modeled using the assumption that each step in a linear pathway behaves according to the law of mass action (43, 570); high doses are always expected to produce higher responses. In contrast, NMDRCs are not easy to model (although they are quite easy to test for), requiring detailed knowledge of the specific mechanisms operating in several biological components. From a regulatory standpoint, information from high doses cannot always be used to assess whether low doses will produce a biological effect (38).

IV. Implications of Low-Dose Effects and Nonmonotonicity

Both low-dose effects and NMDRCs have been observed for a wide variety of EDCs as well as natural hormones. Importantly, these phenomena encompass every level of biological organization, from gene expression, hormone production, and cell number to changes in tissue architecture to behavior and population-based disease risks. One conclusion from this review is that low-dose effects and NMDRCs are often observed after administration of environmentally relevant doses of EDCs. For both hormones and EDCs, NMDRCs should be the default assumption absent sufficient data to indicate otherwise. Furthermore, there are well-understood mechanisms to explain how low-dose effects and NMDRCs manifest *in vitro* and *in vivo*. Accepting these phenomena, therefore, should lead to paradigm shifts in toxicological studies and will likely also have lasting effects on regulatory science. Some of these aspects are discussed below. Additionally, we have briefly explored how this knowledge should influence future approaches in human and environmental health.

At a very practical level, we recommend that researchers publishing data with low-dose and nonmonotonic effects include key words in the abstract/article that identify them as such specifically. This review was unquestionably impeded because this has not been standard practice. We also strongly recommend that data showing nonmonotonic and binary response patterns not be rejected or criticized because there is no dose response.

A. Experimental design

1. Dose ranges must be chosen carefully

To detect low-dose effects or NMDRCs, the doses included for testing are of utmost importance. Most of the studies we examined here for nonmonotonicity tested

doses over severalfold concentrations. Unfortunately, regulatory guidelines only require that three doses be tested. Both low-dose effects and NMDRCs can be observed when examining only a few doses, but some studies may detect significant results purely by luck, because a small shift in dose can have a large impact on the ability to observe differences relative to untreated controls.

In the multitude of chemicals that have never been tested at low doses, or in the development of new chemicals, to determine whether a chemical has low-dose effects in laboratory animals, we suggest setting the NOAEL or LOAEL from traditional toxicological studies as the highest dose in experiments specifically designed to test endocrine-sensitive endpoints. We suggest setting the lowest dose in the experiment below the range of human exposures, if such a dose is known. Several intermediate doses overlapping the range of typical human exposures should be included also, bringing the total number in the range of five to eight total doses tested. Importantly, although the levels of many environmental chemicals in human blood and/or urine have been reported by the CDC and other groups responsible for population-scale biomonitoring, it is often not known what administered doses are needed to achieve these internal exposure levels in animals (4, 253); thus, toxicokinetic studies are often needed before the onset of low-dose testing. This is important because the critical issue is to determine what effects are observed in animals when circulating levels of an EDC match what is measured in the typical human. Due to differences in metabolism, route of exposure, and other factors, a relatively high dose may need to be administered to a rodent to produce blood concentrations in the range of human levels; however, this should not be considered a high-dose study.

It has also been suggested that animal studies that are used to understand the potential effects of a chemical on humans should use a relevant route of administration to recapitulate human exposures (571, 572) because there may be differences in metabolism after oral and nonoral administration. Many chemicals that enter the body orally undergo first-pass metabolism and are then inactivated via liver enzymes, whereas other routes (*i.e.* sc) can bypass these mechanisms and lead to a higher concentration of the active compound in circulation (573). Studies indicate, however, that inactivation of chemicals via first-pass metabolism is not complete and also that deconjugation of metabolites can occur in some tissues allowing the rerelease of the active form (574, 575). Additionally, for some chemicals, it is clear that route of administration has little or no impact on the availability of the active compound in the body (241, 384), and other studies show that route of administration has no impact on the biological effects of these chemicals; *i.e.* regardless of how it enters the body, dioxin has similar effects on exposed individuals (384), and comparable results have been observed for BPA (141). Although understanding the typical route of human exposure to each environmental chemical is an important task, it has been argued that any method that leads to blood concentrations of a test chemical in the range they are observed in humans is an acceptable exposure protocol, and this is especially true with gestational exposures, because fetuses are exposed to chemicals only via their mothers' blood (31, 576).

2. Timing of exposures is important

Rodent studies indicate that EDC exposures during development have organizational effects, with permanent effects that can manifest even in late adulthood, whereas exposures after puberty are for the most part activational, with effects that are abrogated when exposures cease. For example, the adult uterus requires relatively large doses of BPA (in the parts-per-million range) to induce changes associated with the uterotrophic assay (555, 577), whereas parts-per-trillion and ppb exposures during the fetal period permanently and effectively alter development of the uterus (279, 310, 578). Thus, the timing of exposures is profoundly important to detect low-dose effects of EDCs.

Human studies also support this conclusion. The 1976 explosion of a chemical plant in Seveso, Italy, which led to widespread human exposure to large amounts of TCDD, a particularly toxic form of dioxin, and the deposition of this chemical on the land surrounding the chemical plant, provided evidence in support of the organizational and activational effects of endocrine-active chemicals in humans (579). Serum TCDD concentrations showed correlations between exposure levels and several disease outcomes including breast cancer risk, abnormal menstrual cycles, and endometriosis (580–582), but individuals who were either infants or teenagers at the time of the explosion were found to be at greatest risk for developing adult diseases (583, 584). Importantly, many scientists have argued that organizational effects can occur during puberty, *i.e.* that the period where hormones have irreversible effects on organ development extends beyond the fetal and neonatal period (585), and for some endpoints this appears to be the case (586, 587).

It has also been proposed that the endocrine system maintains homeostasis in the face of environmental insults (210). The adult endocrine system does appear to provide some ability to maintain a type of homeostasis; when the pharmaceutical estrogen DES is administered to pregnant mice, the circulating estradiol concentrations in the dam respond by decreasing linearly (224). In contrast, fetal concentrations of estradiol respond nonmonotonically in

a way that is clearly not correlated with maternal levels. Similarly, there is evidence that BPA can induce aromatase and therefore increase estradiol levels *in situ* in the fetal urogenital sinus (588). This is an example of a feed-forward positive-feedback effect rather than a homeostatic response. The effects of EDCs on adult subjects, both animal and people, suggest that diseases often result from low-dose adult exposures (589–595); this argues against a view of the endocrine system as a means to maintain homeostatic control. Instead, individuals can be permanently changed, in an adverse way, after EDC exposures.

In one example, pregnant mice were exposed to low concentrations of BPA, and their male offspring had altered pancreatic function at 6 months of age (158). Surprisingly, however, the mothers (exposed only during pregnancy) were also affected, with altered metabolic machinery and body weight at 4 months postpartum, long after exposures had ended. The increased incidence of breast cancer in women that took DES during pregnancy also illustrates this point (596, 597). These studies suggest that even the adult endocrine system is not invariably capable of maintaining a so-called homeostatic state when exogenous chemicals affecting the endocrine system are present. Thus, although adult exposures to EDCs have been given some attention by bench scientists (29), more work of this kind is needed to better understand whether and how EDCs can have permanent organizational effects on adult animals.

At the beginning of this review, we justified the need to critically examine the low-dose literature because of recent epidemiological findings linking EDC exposures and diseases. Yet there is inherent difficulty in examining neonatal exposures to EDCs and their connection to diseases due to the length of time needed for these studies; thus, many studies of this type have examined high doses of pharmaceuticals (*i.e.* DES) or accidental exposures to industrial chemicals (*i.e.* dioxin) (66, 398, 399, 581, 597–601).

Only recently, with the availability of biomonitoring samples from large reference populations, have lower doses begun to receive widespread attention from epidemiologists. Many recent studies have examined adult exposures to EDCs and correlated exposures with disease statuses (see for example Refs. 15, 16, and 602–604). Human studies examining fetal/neonatal exposures to low-dose EDCs and early life effects have also begun to be studied (6, 333, 605–607), although studies linking these early life exposures to adult diseases are likely to be decades away. More than anything, these studies support our view that the effects of low-dose exposures should be considered when determining chemical safety.

3. Importance of endpoints being examined

Traditional toxicology testing, and in particular those studies performed for the purposes of risk assessment, typically adhere to guideline studies that have been approved by international committees of experts (608). The endpoints assessed in these guideline-compliant studies are centered around higher-order levels, including weight loss, mortality, changes in organ weight, and a limited number of histopathological analyses (609, 610). When pregnant animals are included in toxicological assessments, the endpoints measured typically include the ability to maintain pregnancies, the number of offspring delivered, sex ratios of surviving pups, and measures regarding maternal weight gain and food/water intake (610).

Yet low-dose EDCs are rarely toxic to the point of killing adult animals or causing spontaneous abortions, and traditional tests such as the uterotrophic assay have been shown to be relatively insensitive (72, 577). It has been argued that this type of testing is insufficient for understanding the effects of EDCs (31, 70, 495, 611). Many EDC studies have instead focused on examining newly developed, highly sensitive endpoints that span multiple levels of biological organization, from gene expression to tissue organization to organ systems to the whole animal (612), which may not be rapidly lethal but which nonetheless have enormous importance for health, including mortality. Thus, for example, studies designed to examine the effects of chemicals on obesity no longer focus on body weight alone but also analyze gene expression; fat content in adipose cells and the process of adipogenesis; inflammation, innervation, and vascularization parameters in specific fat pads; conversion rates of white and brown adipose tissues; systemic hormone levels and response to glucose and insulin challenges; and food intake and energy expenditures, among others (314, 613–615). As our knowledge of EDCs and the endocrine system continue to grow, the most sensitive endpoints should be used to determine whether a chemical is disrupting the development of organisms (70).

In moving beyond traditional, well-characterized health-related endpoints like mortality and weight loss, an important question has been raised: how do we define endpoints as adverse? This is an important point, because it has been suggested that the endpoints examined in independent EDC studies are not validated and may not represent adverse effects (609). There is also debate over whether the mechanism (or mode) of action must be explained for each effect to determine whether a relevant pathway is present in humans (616, 617). Yet, when originally assessing the low-dose literature, the NTP expert panel chose to examine all effects of EDC exposure, re-

gardless of whether the endpoint could be deemed adverse (2). From the perspective of developmental biology, any change in development should be seen as adverse, even if the change itself is not associated with a disease or dysfunction. Some of these developmental changes, in fact, may increase sensitivity or susceptibility to disease later on in life but will otherwise appear normal. Furthermore, studies of heavy metals have shown that small shifts in parameters like IQ may not have drastic effects on individuals but can have serious repercussions on the population level (618), and therefore changes in the variance/observable range of a phenotype should also be considered adverse (52).

4. Importance of study size

National Institutes of Health guidelines require that the number of vertebrate animals used in experiments be as small as possible to show statistically significant effects based on power analysis. Yet many traditional toxicology studies have used large numbers of animals to draw conclusions about chemical safety. When the endpoints being assessed have binary outcomes (*i.e.* animal has a tumor *vs.* animal does not have a tumor) and the incidence of the phenotype is not high, a large number of animals is required to reveal statistically significant effects. In contrast, many of the endpoints examined in the field of endocrine disruption are more complex and are not binary; thus, power analysis allows researchers to determine how many animals are needed to observe statistically significant (and biologically relevant) differences between control and exposed populations. For this reason, arbitrary numbers set as cutoffs for determining whether a study is acceptable or unacceptable for risk assessments are not appropriate. Instead, the number of animals required for a study to be complete is dependent on the effect size, precision/variance, minimal meaningful difference to be considered between populations, and the α-value set in statistical tests.

B. Regulatory science

For decades, regulatory agencies have tested, or approved testing, of chemicals by examining high doses and then extrapolating down from the NOAEL, NOEL, and LOAEL to determine safe levels for humans and/or wildlife. As discussed earlier, these extrapolations use safety factors that acknowledge differences between humans and animals, exposures of vulnerable populations, interspecies variability, and other uncertainty factors. These safety factors are informed guesses, not quantitatively based calculations. Using this traditional way of setting safe doses, the levels declared safe are never in fact tested. Doses in the range of human exposures are therefore also unlikely to be tested. This has generated the current state of science, where many chemicals of concern have never been examined at environmentally relevant low doses (see Table 4 for a small number of examples).

Assumptions used in chemical risk assessments to estimate a threshold dose below which daily exposure to a chemical is estimated to be safe are false for EDCs. First, experimental data provide evidence for the lack of a threshold for EDCs (619). More broadly, the data in this review demonstrate that the central assumption underlying the use of high doses to predict low-dose effects will lead to false estimates of safety. The use of only a few high doses is based on the assumption that all dose-response relationships are monotonic and therefore that it is appropriate to apply a log-linear extrapolation from high-dose testing to estimate a safe reference dose (Fig. 4). The Endocrine Society issued a position statement on EDCs (620) and urged the risk assessment community to use the expertise of their members to develop new approaches to chemical risk assessments for EDCs based on principles of endocrinology. Undertaking this mission will represent a true paradigm shift in regulatory toxicology (79). The Endocrine Society statement was then supported in March 2011 by a letter to *Science* from eight societies with relevant expertise representing over 40,000 scientists and medical professionals (621).

Studies conducted for the purposes of risk assessment are expected to include three doses: a dose that has no effects on traditional toxicological endpoints (the NOAEL), a higher dose with effects on traditional endpoints (the LOAEL), and an even higher dose that shows toxicity. Although reducing the number of animals used for these types of studies is an important goal, more than three doses are often needed for a true picture of a chemical's toxicity. The examination of a larger number of doses would allow for 1) the study of chemicals at the reference dose, *i.e.* the dose that is calculated to be safe; 2) examination of doses in the range of actual human exposures, which is likely to be below the reference dose; and 3) the ability to detect NMDRCs, particularly in the low-dose range. The impact of testing more doses on the numbers of animals required can be mitigated by use of power analysis, as suggested above. Because no amount of research will ever match the diversity and reality of actual human experience, there should be ongoing epidemiological study of potential adverse effects of EDCs even after safe levels are published, with periodic reevaluation of those safe levels.

One issue that has been raised by regulatory agencies is whether animal models are appropriate for understanding the effects of EDCs on humans. These arguments largely center around observed differences in hormone levels during different physiological periods in rodents and humans (57), and differences in the metabolic machinery and ex-

cretion of chemicals between species (622). To address the first issue, it should be noted that the FDA uses animals to test pharmaceuticals and other chemicals before any safety testing in humans because it is widely recognized that, although animals and humans do not have exactly the same physiologies, there is evolutionary conservation among vertebrates and specifically among mammals (62). Furthermore, animal studies proved to be highly predictive of the effects of DES on women, indicating that rodents are sufficiently similar to humans to reliably forecast affected endpoints in the endocrine system (64, 623). Thus, the default position must be that animal data are indicative of human effects until proven otherwise.

With regard to the second issue, BPA researchers in particular have examined species-specific differences in metabolism of this EDC. Interestingly, the pharmacokinetics of BPA in rodents, monkeys, and humans appear to be very similar (624), and regulatory agencies have subsequently concluded that rodents are appropriate models to assess the effects of this chemical (625, 626). Thus, researchers should select animal models that are sensitive to low doses of hormones and select appropriate species for the endpoints of interest. As the scope of our knowledge has broadened about how chemicals can alter the endocrine system, well beyond estrogens, androgens, and the thyroid, it is imperative that considerable thought be given to how to apply this for regulatory purposes.

C. Human health

As discussed several times throughout this review, there is now substantial evidence that low doses of EDCs have adverse effects on human health. Thus, although many epidemiological studies originally focused on occupationally exposed individuals and individuals affected by accidental exposures to high doses of environmental chemicals, these recent studies have suggested wide-ranging effects of EDCs on the general population.

Importantly, human exposures are examples of true mixtures; dozens if not hundreds of environmental chemicals are regularly detected in human tissues and fluids (91), yet very little is known about how these chemicals act in combination (627). Several studies indicate that EDCs can have additive or even synergistic effects (143, 323, 628–630), and thus these mixtures are likely to have unexpected and unpredictable effects on animals and humans. The study of mixtures is a growing and complex field that will require considerable attention in the years ahead as knowledge of EDCs in the laboratory setting are applied to human populations (631, 632).

How much will human health improve by testing chemicals at low, environmentally relevant doses and using the results to guide safety determinations? Current testing paradigms are missing important, sensitive endpoints; because they are often unable to detect NMDRCs, they cannot make appropriate predictions about what effects are occurring at low doses. At this time, it is not possible to quantify the total costs of low-dose exposures to EDCs. However, current epidemiology studies linking low-dose EDC exposures to a myriad of health problems, diseases, and disorders suggest that the costs of current low-dose exposures are likely to be substantial.

The weight of the available evidence suggests that EDCs affect a wide range of human health endpoints that manifest at different stages of life, from neonatal and infant periods to the aging adult. As the American population ages, healthcare costs continue to rise, and there are societal costs as well, with decreased quality of life concerns, decreases in work productivity due to illness or the need for workers to care for affected family members, and the psychological stresses of dealing with some outcomes like infertility. Thus, it is logical to conclude that low-dose testing, followed by regulatory action to minimize or eliminate human exposures to EDCs, could significantly benefit human health. This proposal effectively calls for greatly expanded research to give human communities feedback about themselves. It emanates from a view that human society benefits greatly from the many chemical compounds it uses but that extensive epidemiological surveillance and other focused research designs are needed to assure that the balance of risk/benefit from those chemicals is acceptable.

How much would human health benefit by a reduction in the use of EDCs? For some chemicals, minor changes in consumer habits or industrial practices can have drastic effects on exposures (633–636). Other chemicals like DDT that have been regulated in the United States for decades continue to be detected in human and environmental samples; the persistent nature of many of these agents suggests they may impact human health for decades to come. Even less-persistent chemicals like BPA are likely to remain in our environment long after a ban is enacted because of the large amounts of plastic waste leaching BPA (and other estrogenic compounds) from landfills into water sources (637) and its presence on thermal receipt paper and from there into recycled paper (638–640). Yet, despite these challenges, reducing human exposure to EDCs should be a priority, and one way to address that priority is to decrease the production and use of these chemicals. The Endocrine Society has called for such a reduction and the use of the precautionary principle, *i.e.* action in the presence of concerning information but in the absence of certainty to eliminate or cut the use of questionable chemicals even when cause-effect relationships are not yet established (620).

D. Wildlife

Much of the recent focus on EDCs has been on the impact of these chemicals on human health. Yet the earliest studies of EDCs that focused on the impact of these chemicals on wildlife should not be forgotten. Rachel Carson's work on DDT and other pesticides provided some of the earliest warning signs that there were unintended consequences of chemical use. Carson's work was ahead of its time; she understood that exceedingly small doses of these chemicals produced adverse effects, that the timing of exposures was critical, and that chemical mixtures produced compounded effects (641). Now, decades after some of the most dangerous EDCs have been regulated, they continue to be measured in environmental samples as well as the bodies of wildlife animals.

Furthermore, it should be pointed out that humans, like wildlife, are not insulated from the environment, and effects in wildlife, including nonmammalian species, are indicative of and mirror effects in humans. For example, BPA has estrogen-like effects in fish (642–644), amphibians (645, 646), and reptiles (647, 648). A recent review showed that demasculinizing and feminizing effects of atrazine have been demonstrated in fish, amphibians, reptiles, birds, and mammals, *i.e.* every vertebrate class examined (326); and in fact, the first report to suggest that atrazine induced aromatase was conducted in reptiles (649). Similarly, perchlorate affects fish (650–653), amphibians (654–658), and birds (659–661) via mechanisms consistent with those described for humans, and some of the earliest reports on perchlorate's effects on thyroid function were conducted in amphibians (661, 662). Finally, ecological studies of dioxin and dioxin-like chemicals reveal effects on a range of exposed wildlife including birds (663, 664), fish (665, 666), and invertebrates (667). Although these studies have highlighted some of the species-specific effects of dioxin (389), and orders of magnitude differences in toxic equivalency factors between species (668), they also indicate the conservation of mechanisms for the effects of dioxin on a range of biological endpoints in wildlife, laboratory animals, and humans (384). In fact, in many cases, nonmammalian species are much more sensitive to EDC effects, and wildlife species serve as sentinels for environmental and public health (669–673). Thus, the effects of these chemicals on wildlife populations are likely to continue; for this reason, the low-dose effects of these chemicals are particularly worth understanding (674, 675).

V. Summary

In conclusion, we have provided hundreds of examples that clearly show that NMDRCs and low-dose effects are common in studies of hormones and EDCs. We have examined each of these issues separately and provided mechanistic explanations and examples of both. These topics are related, but they must be examined individually to be understood. The concept of nonmonotonicity is an essential one for the field of environmental health science because when NMDRCs occur, the effects of low doses cannot be predicted by the effects observed at high doses. In addition, the finding that chemicals have adverse effects on animals and humans in the range of environmental exposures clearly indicates that low doses cannot be ignored.

In closing, we encourage scientists and journal editors to publish data demonstrating NMDRCs and low-dose effects, even if the exact mechanism of action has not yet been elucidated. This is important because the study of EDC is a growing specialty that crosses many scientific fields, and scientists that work on or regulate EDCs should appreciate and acknowledge the existence of NMDRCs and low-dose effects and have access to this important information. We further recommend greatly expanded and generalized safety testing and surveillance to detect potential adverse effects of this broad class of chemicals. Before new chemicals are developed, a wider range of doses, extending into the low-dose range, should be fully tested. And finally, we envision that the concepts and empirical results we have presented in this paper will lead to many more collaborations among research scientists in academic and government laboratories across the globe, that more and more sophisticated study designs will emerge, that what we have produced herein will facilitate those making regulatory decisions, that actions taken in light of this information will begin to abate the use of EDCs, and ultimately that health impacts in people and in wildlife will be averted.

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APPENDIX H-1

[redacted]

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---------- Forwarded message ---------
From: **Swain, Ken** <Ken.Swain@novascotia.ca>
Date: Mon, Jan 21, 2019 at 9:07 AM
Subject: RE: Test results
To: Jill Graham-Scanlan <jillgrahamscanlan@gmail.com>

Good morning, as attached, please see the sampling results for the raw effluent discharge ditch taken in October and November 2017. Note that the October sampling was during routine mill maintenance. Regards, Ken Swain

From: Jill Graham-Scanlan <jillgrahamscanlan@gmail.com>
Sent: Friday, January 18, 2019 3:54 PM
To: Swain, Ken <Ken.Swain@novascotia.ca>
Subject: Re: Test results

Thank you.

~Jill

On Thu, Jan 17, 2019 at 6:52 PM Swain, Ken <Ken.Swain@novascotia.ca> wrote:

Good evening. We are accessing the specific sampling results from our consultant and will forward them to you once we receive them. This will probably be first of next week.

Regards,

Ken Swain

From: Jill Graham-Scanlan <jillgrahamscanlan@gmail.com>
Sent: January 16, 2019 12:07 PM
To: Boat Harbour <BoatHarbour@novascotia.ca>
Subject: Test results

I understand that GHD had tests conducted to determine the nature of the raw effluent currently flowing into Boat Harbour. I would like a copy of these tests. Will you please forward them to me? If you are not able to forward them to me, where can I access or obtain them?

Thank you,

~Jill

Table 9

Surface Water Analytical Resutls - Marine
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:		**Current Raw Effluent Discharge Ditch (CRED)**	
Sample Location:		**CRED-EFF-1**	**CRED-EFF-1**
Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Field Parameters			
Conductivity, field	mS/cm	0.193	0.664
Dissolved oxygen (DO), field	µg/L	7480	6220
pH, field	s.u.	5.72	7.67
Temperature, field	Deg C	21.69	43.88
Turbidity, field	NTU	22.4	398
Metals			
Aluminum	µg/L	9400	2700
Antimony	µg/L	ND(1.0)	ND(1.0)
Arsenic	µg/L	2.1	1.4
Barium	µg/L	61	870[a]
Beryllium	µg/L	ND(1.0)	ND(1.0)
Bismuth	µg/L	ND(2.0)	ND(2.0)
Boron	µg/L	ND(50)	73
Cadmium	µg/L	0.35[a]	2.5[a]
Calcium	µg/L	20000	240000
Chromium	µg/L	2.8	6.9
Cobalt	µg/L	0.45	1.0
Copper	µg/L	9.7[a]	12[a]
Iron	µg/L	680	1300
Lead	µg/L	2.0	21[a]
Magnesium	µg/L	2800	7300
Manganese	µg/L	680	3200

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Mercury	µg/L	ND(0.013)	0.088[a]
Molybdenum	µg/L	5.0	ND(2.0)
Nickel	µg/L	3.8	5.4
Phosphorus	µg/L	160	1500
Potassium	µg/L	3300	6900
Selenium	µg/L	ND(1.0)	ND(1.0)
Silver	µg/L	0.22	0.56
Sodium	µg/L	180000	240000[c]
Strontium	µg/L	54	300
Thallium	µg/L	ND(0.10)	0.22
Tin	µg/L	ND(2.0)	ND(2.0)
Titanium	µg/L	15	79
Uranium	µg/L	0.17	1.0
Vanadium	µg/L	5.4	4.1
Zinc	µg/L	35[a]	190[a]
Polychlorinated Biphenyls (PCBs)			
Aroclor-1016 (PCB-1016)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1221 (PCB-1221)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1232 (PCB-1232)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1242 (PCB-1242)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1248 (PCB-1248)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1254 (PCB-1254)	µg/L	ND(0.050)	ND(0.50)
Aroclor-1260 (PCB-1260)	µg/L	ND(0.050)	ND(0.50)
Total PCBs	µg/L	ND(0.050)	ND(0.50)

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Petroleum Hydrocarbons (PHCs)			
Benzene	µg/L	ND(1)	ND(100)
Toluene	µg/L	ND(1)	ND(100)
Ethylbenzene	µg/L	ND(1)	ND(100)
Xylenes (total)	µg/L	ND(2)	ND(200)
Total Petroleum Hydrocarbons (C6-C10) Less BTEX	µg/L	ND(10)	ND(1000)
Total Petroleum hydrocarbons (>C10-C16)	µg/L	ND(50)	590
Total Petroleum Hydrocarbons (>C16-C21)	µg/L	92	960
Total Petroleum Hydrocarbons (>C21-C32)	µg/L	460	2400
Total Petroleum Hydrocarbons (Modified TPH)	µg/L	560[a]	4000[ac]
Polycyclic Aromatic Hydrocarbons (PAHs) and Phenols			
1-Methylnaphthalene	µg/L	ND(0.050)	ND(0.050)
2,3,4,5-Tetrachlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,3,4,6-Tetrachlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,3,4-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,3,5,6-Tetrachlorophenol	µg/L	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2,3,5-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2,3,6-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2,3-Dichlorophenol	µg/L	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2,4,5-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2,4,6-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,4+2,5-Dichlorophenol	µg/L	ND(0.50)	ND(0.50)
2,4-Dichlorophenol	µg/L	ND(0.1)	ND(0.1)
2,4-Dimethylphenol	µg/L	ND(0.50) / ND(1)	1.6 / 2
2,4-Dinitrophenol	µg/L	ND(1)	ND(1)

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Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
2,5-Dichlorophenol	µg/L	ND(0.1)	ND(0.1)
2,6-Dichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2-Chlorophenol	µg/L	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)
2-Methylnaphthalene	µg/L	ND(0.050)	ND(0.050)

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Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
2-Methylphenol	µg/L	0.55 / ND(0.5)	5.7 / 7.5
2-Nitrophenol	µg/L	ND(1) / ND(1.0)	ND(1.0) / ND(1)
3&4-Methylphenol	µg/L	0.9	2.1
3,4,5-Trichlorocatechol	µg/L	ND(0.50)	ND(0.50)
3,4,5-Trichloroguaiacol	µg/L	ND(0.50)	ND(0.50)
3,4,5-Trichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
3,4,5-Trichlorosyringol	µg/L	ND(0.50)	ND(0.50)
3,4,5-Trichloroveratrol	µg/L	ND(0.50)	ND(0.71)
3,4-Dichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.64) / ND(0.1)
3,5-Dichlorocatechol	µg/L	ND(0.50)	ND(0.50)
3,5-Dichlorophenol	µg/L	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
3/4-Chlorophenol	µg/L	ND(0.1)	ND(0.1)
3-Chlorophenol	µg/L	ND(0.50)	ND(0.50)
3-Methylphenol	µg/L	0.75	0.56
4,5,6-Trichloroguaiacol	µg/L	ND(0.50)	ND(0.50)
4,5-Dichlorocatechol	µg/L	ND(0.50)	ND(0.50)
4,5-Dichloroguaiacol	µg/L	ND(0.50)	ND(0.58)
4,5-Dichloroveratrol	µg/L	ND(0.50)	ND(0.50)
4,6-Dichloroguaiacol	µg/L	ND(1.7)	ND(0.50)
4,6-Dinitro-2-methylphenol	µg/L	ND(1)	ND(1)
4-Chloro-3-methylphenol	µg/L	ND(0.1)	ND(0.1)
4-Chloroguaiacol	µg/L	ND(0.50)	2.7
4-Chlorophenol	µg/L	ND(0.50)	0.68
4-Methylphenol	µg/L	0.69	ND(27)
4-Nitrophenol	µg/L	ND(1) / ND(5.0)	ND(9.2) / ND(1)
5,6-Dichlorovanilline	µg/L	ND(0.50)	ND(0.50)

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
6-Chlorovanilline	µg/L	ND(0.50)	14
Acenaphthene	µg/L	ND(0.010)	ND(0.010)
Acenaphthylene	µg/L	ND(0.010)	0.057
Acridine	µg/L	ND(0.050)	ND(0.050)
Anthracene	µg/L	ND(0.010)	ND(0.035)
Benzo(a)anthracene	µg/L	ND(0.010)	0.032
Benzo(a)pyrene	µg/L	ND(0.010)	ND(0.010)
Benzo(b)fluoranthene	µg/L	ND(0.010)	ND(0.010)
Benzo(b)pyridine (Quinoline)	µg/L	ND(0.050)	0.082
Benzo(g,h,i)perylene	µg/L	ND(0.010)	ND(0.010)
Benzo(j)fluoranthene	µg/L	ND(0.010)	ND(0.010)
Benzo(k)fluoranthene	µg/L	ND(0.010)	ND(0.010)
Benzo fluoranthenes	µg/L	ND(0.020)	ND(0.020)
Catechol	µg/L	0.61	6.3
Chlorocatechols	µg/L	ND(0.50)	ND(0.68)
Chrysene	µg/L	ND(0.010)	ND(0.033)
Dibenz(a,h)anthracene	µg/L	ND(0.010)	ND(0.010)
Eugenol	µg/L	ND(0.50)	19
Fluoranthene	µg/L	0.018	ND(0.015)
Fluorene	µg/L	ND(0.010)	0.18
Guaiacol	µg/L	19	2300
Indeno(1,2,3-cd)pyrene	µg/L	ND(0.010)	ND(0.010)
Isoeugenol	µg/L	ND(0.50)	1.1
Naphthalene	µg/L	ND(0.20)	ND(0.20)
Pentachlorophenol	µg/L	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
Perylene	µg/L	ND(0.010)	ND(0.010)

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Phenanthrene	µg/L	0.018	0.049
Phenol	µg/L	1.8 / 3.4c	52 / 120c
Phenolics (total)	µg/L	25	2500
Pyrene	µg/L	0.034[a]	ND(0.010)
Tetrachlorocatechol	µg/L	ND(0.50)	ND(0.50)
Tetrachloroguaiacol	µg/L	ND(0.50)	ND(0.50)
Tetrachloroveratrol	µg/L	ND(0.50)	ND(0.50)

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Resins and Fatty Acids			
-Chlorodehydroabietic acid	µg/L	ND(3.0)	ND(3.0)
-Chlorodehydroabietic acid	µg/L	ND(3.0)	ND(3.0)
9,10-Dichlorostearic acid	µg/L	ND(3.0)	6.9
Abietic Acid	µg/L	9.3	190
Dehydroabietic acid	µg/L	50	120
Hexadecanoic acid	µg/L	ND(30)	180
Isopimaric acid	µg/L	12	74
Levopimaric acid	µg/L	5.4 J	ND(3.0) J
Linoleic acid	µg/L	5.4	620
Linolenic acid	µg/L	ND(3.0)	7.4
Neoabietic acid	µg/L	ND(3.0)	12
Octadecanoic acid	µg/L	ND(30)	74
Oleic acid	µg/L	6.7	320
Palmitoleic acid	µg/L	ND(3.0)	ND(5.0)
Palustric acid	µg/L	ND(3.0)	21
Pimaric acid	µg/L	3.3	19
Sandarcopimaric acid	µg/L	3.9	21
Total of fatty acids detected	µg/L	ND(30)	1200
Total of resin acids detected	µg/L	83	460
Volitile Organic Compounds (VOCs)			
1,1,1-Trichloroethane	µg/L	ND(1.0)	ND(1.0)
1,1,2,2-Tetrachloroethane	µg/L	ND(0.50)	ND(0.50)
1,1,2-Trichloroethane	µg/L	ND(1.0)	ND(1.0)
1,1-Dichloroethane	µg/L	ND(2.0)	ND(2.0)

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Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
1,1-Dichloroethene	µg/L	ND(0.50)	ND(56)
1,2-Dibromoethane (Ethylene dibromide)	µg/L	ND(0.20)	ND(0.20)
1,2-Dichlorobenzene	µg/L	ND(0.50)	ND(0.50)
1,2-Dichloroethane	µg/L	ND(1.0)	ND(1.0)
1,2-Dichloropropane	µg/L	ND(0.50)	ND(0.50)
1,3-Dichlorobenzene	µg/L	ND(1.0)	ND(1.0)
1,4-Dichlorobenzene	µg/L	ND(1.0)	ND(1.0)
Benzene	µg/L	ND(1.0)	ND(1.0)
Bromodichloromethane	µg/L	2.7	ND(1.0)
Bromoform	µg/L	ND(1.0)	ND(1.0)
Bromomethane (Methyl bromide)	µg/L	ND(0.50)	ND(0.50)
Carbon tetrachloride	µg/L	ND(0.50)	ND(0.50)
Chlorobenzene	µg/L	ND(1.0)	ND(1.0)
Chloroethane	µg/L	ND(8.0)	ND(8.0)
Chloroform (Trichloromethane)	µg/L	22	25
Chloromethane (Methyl chloride)	µg/L	ND(8.0)	ND(8.0)
cis-1,2-Dichloroethene	µg/L	ND(0.50)	ND(0.50)
cis-1,3-Dichloropropene	µg/L	ND(0.50)	ND(0.50)
Dibromochloromethane	µg/L	ND(1.0)	ND(1.0)
Ethylbenzene	µg/L	ND(1.0)	ND(1.0)
m&p-Xylenes	µg/L	ND(2.0)	ND(2.0)
Methyl tert butyl ether (MTBE)	µg/L	ND(2.0)	ND(2.0)
Methylene chloride	µg/L	ND(3.0)	ND(3.0)
o-Xylene	µg/L	ND(1.0)	ND(1.0)
Styrene	µg/L	ND(1.0)	ND(1.0)
Tetrachloroethene	µg/L	ND(1.0)	ND(1.0)

Table 9

Surface Water Analytical Resutls - Marine
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:		**Current Raw Effluent Discharge Ditch (CRED)**	
Sample Location:		**CRED-EFF-1**	**CRED-EFF-1**
Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Toluene	µg/L	ND(1.0)	8.4
trans-1,2-Dichloroethene	µg/L	ND(0.50)	ND(0.50)
trans-1,3-Dichloropropene	µg/L	ND(0.50)	ND(0.50)
Trichloroethene	µg/L	ND(1.0)	ND(1.0)
Trichlorofluoromethane (CFC-11)	µg/L	ND(8.0)	ND(8.0)
Trihalomethanes	µg/L	25	25
Vinyl chloride	µg/L	ND(0.50)	ND(0.50)
Xylenes (total)	µg/L	ND(1.0)	ND(1.0)

Table 9

Surface Water Analytical Resutls - Marine
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:		**Current Raw Effluent Discharge Ditch (CRED)**	
Sample Location:		**CRED-EFF-1**	**CRED-EFF-1**
Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
General Chemistry			
%difference/ion balance	%	7.65	32.4
Alkalinity, bicarbonate (calculated)	µg/L	86000	270000
Alkalinity, carbonate (calculated)	µg/L	290000	1500
Alkalinity, total (as CaCO3)	µg/L	390000	270000
Ammonia-N	µg/L	88	2300
Anion sum	meq/L(me/L)	10.8	11.8
Cation sum	meq/L(me/L)	9.30	23.0
Chlorate	µg/L	520	51000[a]
Chloride (dissolved)	µg/L	91000	190000
Chlorite	µg/L	ND(100)	ND(100)
Color	TCU	68	620
Conductivity	uS/cm	860	1300
Cyanide (strong acid extractable)	µg/L	1.7[a]	8.2[a]
Hardness	µg/L	61000	620000
Hydrogen sulfide (calculated)	µg/L	120	150
Langelier saturated index @ 20C	none	2.30	1.05
Langelier saturated index @ 4C	none	2.05	0.801
Langelier saturated pH @ 20C	none	8.25	6.73
Langelier saturated pH @ 4C	none	8.50	6.98
Nitrate (as N)	µg/L	55	220
Nitrite (as N)	µg/L	ND(10)	60
Nitrite/Nitrate	µg/L	55	280
Orthophosphate	µg/L	19	810
pH, lab	s.u.	10.6	7.78
Silica, reactive	µg/L	5200	9500

Table 9

Surface Water Analytical Resutls - Marine
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:		**Current Raw Effluent Discharge Ditch (CRED)**	
Sample Location:		**CRED-EFF-1**	**CRED-EFF-1**
Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Sulfate (dissolved)	µg/L	21000	45000
Sulfide	µg/L	110	140
Total dissolved solids (TDS) (calculated)	µg/L	560000	900000
Total organic carbon (TOC)	µg/L	18000	230000
Turbidity	NTU	58	390
Dioxins and Furans			
1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)	pg/L	2.55	ND(1.60)
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin (OCDD)	pg/L	34.5 J	7.94
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	pg/L	ND(0.899)	ND(1.21)
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	pg/L	2.09	ND(1.48)
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	pg/L	ND(1.20)	ND(1.61)
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	pg/L	ND(0.531)	ND(1.50)
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	pg/L	ND(1.22)	ND(1.30)
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	pg/L	ND(0.516)	ND(1.46)
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	pg/L	ND(1.22)	ND(1.31)
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	pg/L	ND(0.635)	ND(1.80)
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	pg/L	ND(1.09)	ND(1.17)
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	pg/L	ND(1.01)	ND(1.58)
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	pg/L	ND(1.16)	ND(1.11)
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	pg/L	ND(0.580)	ND(1.64)
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	pg/L	ND(1.00)	ND(1.57)
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	pg/L	ND(0.968)	ND(1.61)
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	pg/L	ND(0.792)	ND(1.56)
Total heptachlorodibenzofuran (HpCDF)	pg/L	1.20	ND(1.38)
Total heptachlorodibenzo-p-dioxin (HpCDD)	pg/L	7.57	ND(1.48)

Table 9

Surface Water Analytical Resutls - Marine
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:		**Current Raw Effluent Discharge Ditch (CRED)**	
Sample Location:		**CRED-EFF-1**	**CRED-EFF-1**
Sample ID:		**CRED-EFF-1-1**	**CRED-EFF-1-2**
Sample Date:		**27-Oct-17**	**17-Nov-17**
Sample Matrix:		**Surface Water**	**Surface Water**
Sample Type:		**During Maintenance**	**During Operations**
Parameters	**Units**		
Total hexachlorodibenzofuran (HxCDF)	pg/L	ND(0.562)	ND(1.59)
Total hexachlorodibenzo-p-dioxin (HxCDD)	pg/L	ND(1.17)	ND(1.26)
Total pentachlorodibenzofuran (PeCDF)	pg/L	ND(1.01)	ND(1.58)
Total pentachlorodibenzo-p-dioxin (PeCDD)	pg/L	ND(1.16)	ND(1.11)
Total tetrachlorodibenzofuran (TCDF)	pg/L	ND(0.968)	ND(1.61)
Total tetrachlorodibenzo-p-dioxin (TCDD)	pg/L	ND(0.792)	ND(1.56)
Total Toxic Equivalency (TEQ)	pg/L	3.01	4.41

Table 10

Surface Water Analytical Results - Freshwater
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:						Background (BKGD) Chance Harbour Lake						Former Raw Effluent Discharge Ditch (FRED)	Former Settling Pond 1 (FSP1)	Former Settling Pond 2 (FSP2)		Former Settling Pond 3 (FSP3)	Sludge Disposal Cell (SDC)
Sample Location:						BKGD-SW-1	BKGD-SW-2	BKGD-SW-3	BKGD-SW-3	BKGD-SW-4	BKGD-SW-5	FRED-SW-1	FSP1-SW-1	FSP2-SW-1	FSP2-SW-2	FSP3-SW-1	SDC-SW-1
Sample ID:						BKGD-SW-1	BKGD-SW-2	BKGD-SW-3	BKGD-SW-DUP	BKGD-SW-4	BKGD-SW-5	FRED-SW-1	FSP1-SW-1	FSP2-SW-1	FSP2-SW-2	FSP3-SW-1	SDC-SW-1
Sample Date:						23-Nov-17	23-Nov-17	23-Nov-17	23-Nov-17	23-Nov-17	23-Nov-17	26-Oct-17	27-Oct-17	27-Oct-17	27-Oct-17	25-Oct-17	27-Oct-17
Sample Matrix:						Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water	Surface Water
Sample Type:						Original	Original	Original	Duplicate	Original	Original	Original	Original	Original	Original	Original	Original
Parameters	**Units**	**Provincial Ecological** a	**Freshwater Surface Water Criteria Federal Ecological** b	**Provincial Human Health** c	**Federal Human Health** d												
Field Parameters																	
Conductivity, field	mS/cm					0.07	0.071	0.071	0.071	0.071	0.111	0.1	0.109	0.119	0.118	0.073	0.057
Dissolved oxygen (DO), field	µg/L					11350	12200	11300	11300	11460	12110	8350	6570	8400	7190	8730	8810
pH, field	s.u.		6.5-9			6.56	6.89	6.34[b]	6.34[b]	6.15[b]	5.3[b]	6.94	7.59	7.92	7.88	6.53	7.6
Temperature, field	Deg C					5.71	5.62	5.72	5.72	5.72	5.64	15.93	14.21	16.27	16.33	15.71	14.53
Turbidity, field	NTU					--	--	--	--	--	--	5.6	2	1.9	1.7	2.1	0.7
Metals																	
Aluminum	µg/L	5				19[a]	20[a]	21[a]	20[a]	20[a]	26[a]	34[a]	9.3[a]	14[a]	12[a]	5.3[a]	ND(5.0)
Antimony	µg/L	20		6		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Arsenic	µg/L	5		10		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Barium	µg/L	1000		1000		170	170	170	170	170	180	420	140	150	150	120	53
Beryllium	µg/L	5.3		4		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Bismuth	µg/L					ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Boron	µg/L	1200		5000		ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)
Cadmium	µg/L	0.01		5		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Calcium	µg/L					4300	4300	4400	4400	4300	4300	26000	26000	28000	27000	17000	14000
Chromium	µg/L		8.9 (trivalent)	50		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Cobalt	µg/L	10		10		ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)	ND(0.40)
Copper	µg/L	2				ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Iron	µg/L	300				140	150	150	150	150	200	75	140	210	200	390[a]	ND(50)
Lead	µg/L	1		10		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Magnesium	µg/L					1900	1900	1900	2000	1900	1900	3300	2100	2400	2300	1800	3300
Manganese	µg/L	820				140	140	140	140	140	200	33	46	71	68	210	27
Mercury	µg/L	0.026		1		ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)	ND(0.013)
Molybdenum	µg/L	73		70		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Nickel	µg/L	25		100		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Phosphorus	µg/L					ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)
Potassium	µg/L					810	810	830	820	830	840	1300	990	1000	980	320	300
Selenium	µg/L	1		10		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Silver	µg/L	0.1		100		ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)
Sodium	µg/L			200000		6100	6200	6200	6400	6300	6300	10000	16000	19000	19000	11000	5600
Strontium	µg/L	21000		4400		15	16	15	16	16	15	85	45	49	47	37	41
Thallium	µg/L	0.8		2		ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)
Tin	µg/L			4400		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Titanium	µg/L					ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Uranium	µg/L	300		20		ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)	ND(0.10)
Vanadium	µg/L	6		6.2		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Zinc	µg/L	30				ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)
Petroleum Hydrocarbons (PHCs)																	
Benzene	µg/L	2100		5		ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)
Toluene	µg/L	770		24		ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)
Ethylbenzene	µg/L	320		1.6		ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)
Xylenes (total)	µg/L	330		20		ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)	ND(2)
Total Petroleum Hydrocarbons (C6-C10) Less BTEX	µg/L	see Modified TPH		see Modified TPH		ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)
Total Petroleum hydrocarbons (>C10-C16)	µg/L	see Modified TPH		see Modified TPH		ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)
Total Petroleum Hydrocarbons (>C16-C21)	µg/L	see Modified TPH		see Modified TPH		ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)
Total Petroleum Hydrocarbons >(C21-C32)	µg/L	see Modified TPH		see Modified TPH		ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)
Total Petroleum Hydrocarbons (Modified TPH)	µg/L	1500/100/100		4400/3200/7800		ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)
Polycyclic Aromatic Hydrocarbons (PAHs) and Phenols																	
1-Methylnaphthalene	µg/L	2		12		ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)
2,3,4,5-Tetrachlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,3,4,6-Tetrachlorophenol	µg/L				100	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2,3,4-Trichlorophenol	µg/L					ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2,3,5,6-Tetrachlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,3,5-Trichlorophenol	µg/L					ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2,3,6-Trichlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2,3-Dichlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,4,5-Trichlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2,4,6-Trichlorophenol	µg/L				5	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
2,4+2,5-Dichlorophenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
2,4-Dichlorophenol	µg/L				900	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)
2,4-Dimethylphenol	µg/L					ND(0.50) / ND(1)	ND(0.50) / ND(1)	ND(1) / ND(0.50)	ND(0.50) / ND(1)	ND(1) / ND(0.50)	ND(1) / ND(0.50)	ND(1) / ND(0.50)	ND(0.50) / ND(1)	ND(0.50) / ND(1)	ND(1) / ND(0.50)	ND(1) / ND(0.50)	ND(1) / ND(0.50)
2,4-Dinitrophenol	µg/L					ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)
2,5-Dichlorophenol	µg/L					ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)
2,6-Dichlorophenol	µg/L					ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
2-Chlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
2-Methylnaphthalene	µg/L	2		12		ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)
2-Methylphenol	µg/L					ND(0.5) / ND(0.50)	ND(0.5) / ND(0.50)	ND(0.5) / ND(0.50)	ND(0.5) / ND(0.50)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.5) / ND(0.50)	ND(0.50) / ND(0.5)
2-Nitrophenol	µg/L					ND(1.0) / ND(1)	ND(1.0) / ND(1)	ND(1) / ND(1.0)	ND(1) / ND(1.0)	ND(1) / ND(1.0)	ND(1.0) / ND(1)	ND(1.0) / ND(1)	ND(1.0) / ND(1)	ND(1) / ND(1.0)	ND(1) / ND(1.0)	ND(1) / ND(1.0)	ND(1) / ND(1.0)
3&4-Methylphenol	µg/L					ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)	ND(0.5)
3,4,5-Trichlorocatechol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3,4,5-Trichloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3,4,5-Trichlorophenol	µg/L					ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)
3,4,5-Trichlorosyringol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3,4,5-Trichloroveratrol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3,4-Dichlorophenol	µg/L					ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)
3,5-Dichlorocatechol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3,5-Dichlorophenol	µg/L					ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
3/4-Chlorophenol	µg/L					ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)
3-Chlorophenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
3-Methylphenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,5,6-Trichloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,5-Dichlorocatechol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,5-Dichloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,5-Dichloroveratrol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,6-Dichloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4,6-Dinitro-2-methylphenol	µg/L					ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)

Table 10

Surface Water Analytical Results - Freshwater
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:						Background (BKGD) Chance Harbour Lake						Former Raw Effluent Discharge Ditch (FRED)	Former Settling Pond 1 (FSP1)	Former Settling Pond 2 (FSP2)		Former Settling Pond 3 (FSP3)	Sludge Disposal Cell (SDC)
Sample Location:						**BKGD-SW-1**	**BKGD-SW-2**	**BKGD-SW-3**	**BKGD-SW-3**	**BKGD-SW-4**	**BKGD-SW-5**	**FRED-SW-1**	**FSP1-SW-1**	**FSP2-SW-1**	**FSP2-SW-2**	**FSP3-SW-1**	**SDC-SW-1**
Sample ID:						**BKGD-SW-1**	**BKGD-SW-2**	**BKGD-SW-3**	**BKGD-SW-DUP**	**BKGD-SW-4**	**BKGD-SW-5**	**FRED-SW-1**	**FSP1-SW-1**	**FSP2-SW-1**	**FSP2-SW-2**	**FSP3-SW-1**	**SDC-SW-1**
Sample Date:						**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**26-Oct-17**	**27-Oct-17**	**27-Oct-17**	**27-Oct-17**	**25-Oct-17**	**27-Oct-17**
Sample Matrix:						**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**
Sample Type:						**Original**	**Original**	**Original**	**Duplicate**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**
		Freshwater Surface Water Criteria															
		Provincial	**Federal**	**Provincial**	**Federal**												
		Ecological	**Ecological**	**Human Health**	**Human Health**												
		a	b	c	d												
Parameters	**Units**																
4-Chloro-3-methylphenol	µg/L					ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)	ND(0.1)
4-Chloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4-Chlorophenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4-Methylphenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
4-Nitrophenol	µg/L					ND(5.0) / ND(1)	ND(5.0) / ND(1)	ND(5.0) / ND(1)	ND(5.0) / ND(1)	ND(5.0) / ND(1)	ND(1) / ND(5.0)	ND(1) / ND(5.0)	ND(1) / ND(5.0)	ND(5.0) / ND(1)	ND(5.0) / ND(1)	ND(1) / ND(5.0)	ND(5.0) / ND(1)
5,6-Dichlorovanilline	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
6-Chlorovanilline	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Acenaphthene	µg/L	5.8		1400		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Acenaphthylene	µg/L	4.6		4.5		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Acridine	µg/L					ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)
Anthracene	µg/L	0.012				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(a)anthracene	µg/L	0.018				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(a)pyrene	µg/L	0.015		0.01		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(b)fluoranthene	µg/L	0.48				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(b)pyridine (Quinoline)	µg/L		3.4			ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)	ND(0.050)
Benzo(g,h,i)perylene	µg/L	0.17				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(j)fluoranthene	µg/L	0.48				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo(k)fluoranthene	µg/L	0.48				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Benzo fluoranthenes	µg/L					ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)	ND(0.020)
Catechol	µg/L	2000				ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Chlorocatechols	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Chrysene	µg/L	1.4				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Dibenz(a,h)anthracene	µg/L	0.26				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Eugenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Fluoranthene	µg/L	0.04				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Fluorene	µg/L	3		940		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Guaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Indeno(1,2,3-cd)pyrene	µg/L	0.21				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Isoeugenol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Naphthalene	µg/L	1.1		470		ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)
Pentachlorophenol	µg/L	0.5		30		ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)	ND(0.1) / ND(0.50)	ND(0.50) / ND(0.1)	ND(0.50) / ND(0.1)
Perylene	µg/L					ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Phenanthrene	µg/L	0.4				ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Phenol	µg/L	4000		0.8		ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.5) / ND(0.50)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.5) / ND(0.50)	ND(0.50) / ND(0.5)	ND(0.50) / ND(0.5)	ND(0.5) / ND(0.50)
Phenolics (total)	µg/L		0.004			ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)
Pyrene	µg/L	0.025		710		ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)	ND(0.010)
Tetrachlorocatechol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Tetrachloroguaiacol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Tetrachloroveratrol	µg/L					ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Resins and Fatty Acids																	
-Chlorodehydroabietic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
-Chlorodehydroabietic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
9,10-Dichlorostearic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Abietic Acid	µg/L					ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)
Dehydroabietic acid	µg/L					ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)
Hexadecanoic acid	µg/L					ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)
Isopimaric acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Levopimaric acid	µg/L					ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J	ND(3.0) J
Linoleic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Linolenic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Neoabietic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Octadecanoic acid	µg/L					ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)
Oleic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Palmitoleic acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Palustric acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Pimaric acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Sandarcopimaric acid	µg/L					ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
Total of fatty acids detected	µg/L					ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)	ND(30)
Total of resin acids detected	µg/L					ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)	ND(5.0)

Table 10

Surface Water Analytical Results - Freshwater
Phase 2 Environmental Site Assessment
Boat Harbour Effluent Treatment Facility
Pictou Landing, Nova Scotia

APEC:						Background (BKGD) Chance Harbour Lake						Former Raw Effluent Discharge Ditch (FRED)	Former Settling Pond 1 (FSP1)	Former Settling Pond 2 (FSP2)		Former Settling Pond 3 (FSP3)	Sludge Disposal Cell (SDC)
Sample Location:						**BKGD-SW-1**	**BKGD-SW-2**	**BKGD-SW-3**	**BKGD-SW-3**	**BKGD-SW-4**	**BKGD-SW-5**	**FRED-SW-1**	**FSP1-SW-1**	**FSP2-SW-1**	**FSP2-SW-2**	**FSP3-SW-1**	**SDC-SW-1**
Sample ID:						**BKGD-SW-1**	**BKGD-SW-2**	**BKGD-SW-3**	**BKGD-SW-DUP**	**BKGD-SW-4**	**BKGD-SW-5**	**FRED-SW-1**	**FSP1-SW-1**	**FSP2-SW-1**	**FSP2-SW-2**	**FSP3-SW-1**	**SDC-SW-1**
Sample Date:			Freshwater Surface Water Criteria			**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**23-Nov-17**	**26-Oct-17**	**27-Oct-17**	**27-Oct-17**	**27-Oct-17**	**25-Oct-17**	**27-Oct-17**
Sample Matrix:		**Provincial**	**Federal**	**Provincial**	**Federal**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**	**Surface Water**
Sample Type:		**Ecological** a	**Ecological** b	**Human Health** c	**Human Health** d	**Original**	**Original**	**Original**	**Duplicate**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**	**Original**
Parameters	**Units**																
Volitile Organic Compounds (VOCs)																	
1,1,1-Trichloroethane	µg/L	10		200		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
1,1,2,2-Tetrachloroethane	µg/L	70		1		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
1,1,2-Trichloroethane	µg/L	800		5		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
1,1-Dichloroethane	µg/L	200		5		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
1,1-Dichloroethene	µg/L	40		14		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
1,2-Dibromoethane (Ethylene dibromide)	µg/L	5		0.2		ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)	ND(0.20)
1,2-Dichlorobenzene	µg/L	0.7		200		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
1,2-Dichloroethane	µg/L	100		5		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
1,2-Dichloropropane	µg/L	0.7		5		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
1,3-Dichlorobenzene	µg/L	150		59		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
1,4-Dichlorobenzene	µg/L	26		5		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Benzene	µg/L	2100		5		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Bromodichloromethane	µg/L	200		100		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Bromoform	µg/L	60		100		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Bromomethane (Methyl bromide)	µg/L	0.9		0.89		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Carbon tetrachloride	µg/L	13.3		2		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Chlorobenzene	µg/L	1.3		30		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Chloroethane	µg/L	1100				ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)
Chloroform (Trichloromethane)	µg/L	1.8		100		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Chloromethane (Methyl chloride)	µg/L	700		38		ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)
cis-1,2-Dichloroethene	µg/L	200		20		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
cis-1,3-Dichloropropene	µg/L	7		0.5		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Dibromochloromethane	µg/L	40		100		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Ethylbenzene	µg/L	320		1.6		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
m&p-Xylenes	µg/L			20		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Methyl tert butyl ether (MTBE)	µg/L	10000		15		ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)	ND(2.0)
Methylene chloride	µg/L	98.1		50		ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)	ND(3.0)
o-Xylene	µg/L			20		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Styrene	µg/L	72		100		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Tetrachloroethene	µg/L	111		30		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Toluene	µg/L	770		24		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
trans-1,2-Dichloroethene	µg/L	200		20		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
trans-1,3-Dichloropropene	µg/L	7		0.5		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Trichloroethene	µg/L	21		5		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Trichlorofluoromethane (CFC-11)	µg/L					ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)	ND(8.0)
Trihalomethanes	µg/L					ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
Vinyl chloride	µg/L	600		2		ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)	ND(0.50)
Xylenes (total)	µg/L	330		20		ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)	ND(1.0)
General Chemistry																	
%difference/ion balance	%					1.49	1.49	1.49	0.740	0.00	0.740	7.73	6.75	3.70	4.16	7.32	3.61
Alkalinity, bicarbonate (calculated)	µg/L					16000	16000	16000	15000	15000	15000	84000	96000	97000	95000	65000	50000
Alkalinity, carbonate (calculated)	µg/L					ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)	ND(1000)
Alkalinity, total (as CaCO3)	µg/L					16000	16000	16000	15000	15000	15000	84000	97000	98000	96000	65000	50000
Ammonia-N	µg/L		pH and temp dependent			ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	54[a]
Anion sum	meq/L(me/L)					0.680	0.680	0.660	0.670	0.670	0.680	2.37	2.53	2.66	2.63	1.76	1.29
Cation sum	meq/L(me/L)					0.660	0.660	0.680	0.680	0.670	0.670	2.03	2.21	2.47	2.42	1.52	1.20
Chlorate	µg/L	30000				ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)
Chloride (dissolved)	µg/L		120000			9700	9600	9100	9800	9600	9900	20000	21000	23000	24000	16000	7700
Chlorite	µg/L					ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)	ND(100)
Color	TCU					27	26	26	27	26	28	11	14	17	15	16	17
Conductivity	uS/cm					76	76	77	76	76	77	210	220	240	240	160	120
Cyanide (strong acid extractable)	µg/L	5				1.1	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	ND(1)	1.1	ND(1)
Hardness	µg/L					19000	18000	19000	19000	19000	19000	77000	73000	80000	78000	50000	47000
Hydrogen sulfide (calculated)	µg/L					ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)	ND(21)
Langelier saturated index @ 20C	none					-2.41	-2.41	-2.38	-2.33	-2.37	-2.52	-0.0860	-0.0740	-0.0500	-0.0690	-1.09	-1.05
Langelier saturated index @ 4C	none					-2.66	-2.67	-2.63	-2.58	-2.62	-2.77	-0.336	-0.325	-0.301	-0.320	-1.34	-1.30
Langelier saturated pH @ 20C	none					9.47	9.47	9.47	9.47	9.48	9.48	8.03	7.96	7.92	7.94	8.29	8.49
Langelier saturated pH @ 4C	none					9.73	9.73	9.72	9.72	9.73	9.73	8.28	8.21	8.18	8.19	8.54	8.74
Nitrate (as N)	µg/L		13000		1000	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)
Nitrite (as N)	µg/L		60 (as NO2-N)		45000	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)
Nitrite/Nitrate	µg/L					ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)	ND(50)
Orthophosphate	µg/L					ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)	ND(10)
pH, lab	s.u.		6.5-9			7.07	7.06	7.09	7.14	7.11	6.96	7.94	7.88	7.87	7.87	7.20	7.44
Silica, reactive	µg/L					3100	3100	3100	3100	3200	3200	8000	ND(500)	ND(500)	ND(500)	2400	1200
Sulfate (dissolved)	µg/L					4500	4400	4400	4400	4400	4400	5500	ND(2000)	2200	2200	ND(2000)	3100
Sulfide	µg/L					ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)	ND(20)
Total dissolved solids (TDS) (calculated)	µg/L					40000	40000	40000	40000	40000	40000	130000	130000	140000	130000	89000	65000
Total organic carbon (TOC)	µg/L					6300	6300	6100	6200	6400	6200	2000	5100	6000	6100	5000	4800
Turbidity	NTU					0.79	0.70	0.73	0.72	1.1	1.5	0.43	0.80	1.5	1.4	1.7	0.56

Notes:

19[a] Exceeds applicable criteria; superscript identifies exceeded criteria

Provincial Ecological[a] Nova Scotia Contaminated Sites Regulation Table A2 References for Pathway Specific Standards for freshwater surface water

Federal Ecological[b] Canadian Council of Ministers of the Environment (CCME) Water Quality Guidelines for the protection of Aquatic Life for freshwater surface water

Provincial Human Health[c] Nova Scotia Contaminated Sites Regulation Table A3 References for Pathway Specific Standards for agricultural/residential land use and coarse-grained soil, potable groundwater drinking water pathway

Federal Human Health[d] Canadian Drinking Water Quality Guidelines

BTEX Benzene, Toluene, Ethylbenzene, and total Xylenes

ND (1.0) Not detected at the associated reporting limit

J The associated value is an estimated concentration

-- Parameter not analysed

APPENDIX H-2

DILLON
CONSULTING

January 26, 2000

CANSO CHEMICALS LIMITED
P.O. Box 484
New Glasgow, Nova Scotia
B2H 5E5

ATTENTION: Mr. Dan Currie
General Manager

Canso Chemicals Site Decommissioning Final Report

We are pleased to present six copies of our final report documenting decommissioning activities at the Canso Chemicals Limited site. The report also documents the results of an ecological and human health risk assessment relative to an area with residual mercury that did not meet the 1993 remedial action plan cleanup objectives. A Certificate of Compliance for the facility has been prepared, under the Nova Scotia Contaminated Sites Management Program, and is included with this report. App-F

It has been a pleasure to work with you on this project. If we can be of further assistance, please contact the undersigned at your convenience.

Yours truly,

DILLON CONSULTING LIMITED

Andrew J. Blackmer, M.Sc.
Project Manager

AJB:keb
cc: Mr. Ralston Maloney, P.Eng.
Pioneer Chemicals Limited
Our File: 99-5519-0501

2701
Dutch Village Road
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Halifax
Nova Scotia
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Telephone
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ISO 9001 Registered

1.0 INTRODUCTION

1.1 Purpose

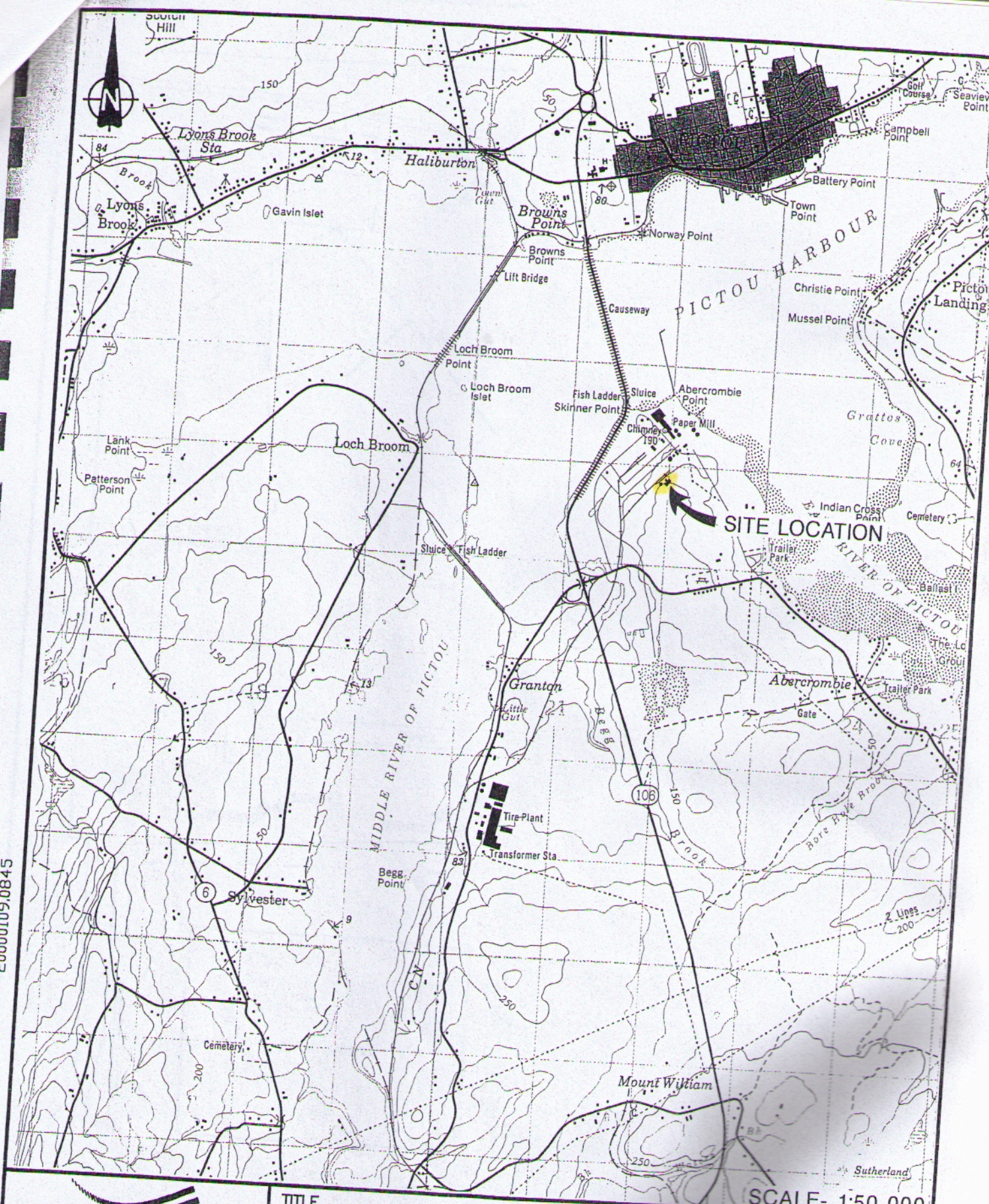
The purpose of this report is to document remedial activities carried out at Canso Chemicals Limited, located at Abercrombie Point (refer to Figure 1-1). Canso Chemicals Limited is a former chloro-alkali plant, which used mercury in its process to generate chlorine and caustic soda for the pulp and paper industry. Upon ceasing operations at the plant in 1992, a program was put in place to decommission the site, including remediation of environmental contaminants. Included in this report is a description of demolition activities relating to the process building (housing the cell room and brine basement) in 1999 (refer to Figure 1-2).

This report provides an overview of site remedial activities surrounding the processing and chlorate buildings, documents confirmatory sampling completed to demonstrate adherence to generic remedial objectives, and proposes site specific remedial objectives for a limited area of the site, based on an assessment of risk to human health and the environment. Recommendations are made for the long term monitoring of the site.

1.2 Background

Canso Chemicals Limited was formed in 1968. A chloro-alkali plant was commissioned in 1970 at Abercrombie Point to produce bleaching chemicals used in the production of pulp and paper (i.e., liquid chlorine and liquid caustic soda). In 1971 a chlorate plant (producing liquid sodium chlorate) was constructed on the site and was operated until 1981.

In 1992, the chloro-alkali plant was shut down. The site is currently used as a caustic soda shipping and receiving facility.



SCALE- 1:50 000

DILLON CONSULTING	TITLE: SITE LOCATION	PROJECT No. 98-5519
DATE: JAN. 2000	PROJECT: CANSO CHEMICALS SITE DECOMMISSIONING	FIGURE No. 1-1

20000109.0845

G:\CAD\985519\FIG1-1

The products made at Canso Chemicals Limited were the result of the electrolysis of a brine solution over mercury electrolytic cells. The primary contaminant of concern on the site is mercury. During 1973-1975, a series of equipment failures in the cell room resulted in high mercury consumption and mercury was lost to the floor (Canso Chemicals Limited, 1993). After that time a comprehensive mercury inventory was maintained, and mercury losses were more tightly controlled (pers. comm. D. Currie). Mercury was also found in process by-products (e.g., brine sludge), which were managed on-site.

Canso Chemicals Limited submitted a site remedial action plan to Nova Scotia Department of the Environment on June 21, 1993 (Canso Chemicals Limited, 1993). This plan outlined an approach to manage site contaminants, including mercury. The plan established an objective of meeting Canadian Council of Ministers of the Environment (CCME) industrial land use guidelines for soil.

1.3 Regulatory Context

The 1993 remedial action plan (RAP) committed to meeting CCME industrial land use guidelines for soil. The Interim Canadian Environmental Quality Criteria for Contaminated Sites (CCME, 1991) established a remedial objective of 10 mg/kg mercury in soil for commercial/industrial sites. The Recommended Canadian Soil Quality Guidelines (CCME, 1997) adopt 30 mg/kg as the soil quality guideline for mercury at industrial sites, superseding the 1991 guideline.

The Nova Scotia Department of the Environment have established guidelines for management of contaminated sites (NSDOE, 1996). These guidelines outline two approaches to contaminated site management. Tier I requires the owner to remediate the site to guideline concentrations appropriate for the future use of the property. Tier II identifies potential risk by comparing detected concentrations of contaminants with site specific criteria. Site specific criteria are determined by calculating potential adverse effects associated with exposure to contaminants at the concentrations detected on site.

The 1993 RAP adopted a Tier I approach, committing to cleanup the site to CCME industrial land use guidelines. Until June 1999, remedial activities were completed in accordance with the RAP. During final phases of demolition of the cell room/brine basement structure (in the process building), elemental mercury was found in an area that could not be excavated in the context of current site conditions. To address this area of concern, an assessment of risks to human health and the environment was completed to establish site specific remedial objectives. This report presents the findings of the assessment of these risks.

As an result of the decommissioning activities and the implementation of remedial actions, a Certificate of Compliance has been prepared, under the Nova Scotia Contaminated Sites Management Program, and is included with this report.

2.0 APPROACH AND METHODOLOGY

Site assessment and remedial activities have been ongoing at Canso Chemicals since cessation of chlorine/caustic production in 1992. An overview of these activities is presented in Section 3 of this report. Work completed between 1992 and 1997 has focussed on recovery of mercury and disposal of mercury contaminated materials without demolition of site buildings. Work completed in 1998/1999 has addressed demolition of the process (including cell room and brine basement) and chlorate buildings, and disposal of mercury contaminated materials previously not acceptable due to presence of the building structures. Site materials contaminated with mercury have been disposed in an on-site secure landfill. Section 4 documents groundwater sampling completed by Canso Chemicals Limited as part of its ongoing monitoring program, as well as additional investigations completed specifically for this assessment.

Remedial activities have been implemented to meet the objectives of the 1993 RAP. In June 1999, elemental mercury was identified in the bedrock beneath the area of the former cell room/brine basement. This material could not be excavated in the context of current site conditions (i.e., potential impact on remaining site buildings and potential for increasing the areal extent and depth of mercury impact due to mercury's physical properties). Section 5 of this report presents findings of ecological and human health risk assessments pertaining to the area of the site that has not been remediated to the generic RAP objectives. These risk assessments evaluate potential impacts associated with assumed levels of contaminants. Section 6 presents recommendations for long term monitoring at the site to evaluate the risk assessment assumptions over time.

site rainfall runoff. Mercury was not detected at depths greater than 0.5 metres, but in all cases, the ditches were excavated to a depth of 0.6 metres or more.

By 1996 decommissioning of the facility was essentially complete and the site stabilized for use as an industrial storage space. Little, if any, further remedial work was undertaken until 1998 when the process buildings were removed.

3.2 Remedial Activities 1998-1999

Remedial activities undertaken at Canso Chemicals during 1998 and 1999 have focussed on demolition of site process buildings (the process building and the chlorate building), excavation of remaining mercury contaminated soil (facilitated by removal of building footings) and disposal of mercury contaminated materials in the on-site secure landfill.

The requirements for demolition activities are outlined in the engineering drawings and specifications for the site (Porter Dillon Limited, 1998). Demolition was completed in two phases: building structures were demolished during the fall of 1998. Mercury contaminated debris was stockpiled for disposal in the secure landfill. Other materials were set aside for use as backfill, or disposed off-site. During the spring/early summer of 1999, building foundations and footings were removed and stockpiled for disposal in the on-site landfill. Mercury contaminated soil in the vicinity of the building foundations and footings, which was previously inaccessible, was also excavated for on-site disposal.

Excavation of the mercury contaminated soil was completed to meet the requirements of the 1993 RAP. The RAP objectives were achieved at all locations within the demolition excavation, with the exception of an area in the vicinity of the cell room/brine basement interface. Figure 3-1 presents locations of confirmatory soil sampling undertaken by Porter Dillon upon completion of excavation activities. These samples supplement sampling undertaken by both

5.0 ASSESSMENT OF RISK

5.1 Ecological Risk Assessment

5.1.1 Approach & Methodology

As described in Section 3, elemental mercury has migrated into the bedrock below the former cell room. Due to the practical limitations of excavation, the remedial program was unable to remove mercury below a depth of eight metres. The remaining mercury is located in bedrock, approximately five metres below the water table, and hence there is potential for it to dissolve into groundwater and migrate towards Pictou Harbour.

An ecological risk assessment was carried out to determine whether the mercury remaining in the ground below the former cell room could pose a risk to ecological receptors in the harbour. The assessment was carried out based on procedures described in CCME's Framework for Ecological Risk Assessment at Contaminated Sites (CCME, 1994). The assessment used a preliminary quantitative approach that involved contaminant fate and transport modelling to estimate the potential mercury concentration in the harbour water and sediment. The concentration estimates were compared to CCME Freshwater Aquatic Life Guidelines and Sediment Quality Guidelines for Aquatic Life to determine whether ecological receptors could be at risk.

The ecological risk assessment involved four main components as follows:

- **Receptor Characterization** - evaluation of potential contaminant receptors
- **Exposure Assessment** - evaluation of exposure pathways and exposure concentrations

- **Hazard Assessment** - evaluation of hazards and their possible effects on receptors

- **Risk Characterization** - evaluation of risk based upon the above three inputs

In addition to the above components, a uncertainty analysis was carried out to evaluate the effect of assumptions used in the risk assessment.

5.1.2 Receptor Characterization

A receptor is an ecosystem component that may be adversely affected by a contaminant. Receptors may include biological (e.g., aquatic life) or abiotic (e.g., water quality, sediment quality).

Groundwater flow data presented in Section 4 indicates that the horizontal direction of groundwater flow in the shallow and deep bedrock aquifer is northwest, towards the confluence of the Middle River and West River of Pictou in Pictou Harbour. The data indicate that the vertical direction of groundwater flow at the site is downwards.

There are no water supply wells located down-gradient of the former cell room where the source of mercury is located. The only down-gradient property between the mercury source and the harbour is the Kimberly-Clark paper mill which uses the Middle River of Pictou for process water and bottled water for drinking (pers. comm., Penny MacLeod). Therefore, the receptor of mercury-impacted groundwater is expected to be Pictou Harbour, and the plume is expected to eventually discharge to the harbour approximately 700 m northwest of the former cell room. Both water quality and sediment quality could be affected since dissolved mercury may adsorb to sediments as it discharges through bottom sediments into the harbour.

Aquatic plant life observed by Porter Dillon in July 1999 indicate the harbour can be considered an estuary to marine habitat in the area where the plume may discharge. An electrical conductivity (EC) measurement of the harbour water in this area (EC = 30,000 μS/cm) indicated that the water is in the estuary to marine range (EC = 800 to 55,000 as/cm).

There is also a small pond located adjacent to the harbour in the area where the plume could discharge. The pond is physically separated from the harbour by a causeway, but is hydraulically connected to the harbour by two culverts. Although the pond is a potential receptor of mercury impacted groundwater, is it likely that deep groundwater originating from the Canso Chemical site will bypass the pond and discharge to the harbour further from the shoreline. Shallow groundwater in the vicinity of the pond is more likely to discharge to the pond.

5.1.3 Exposure Assessment

Exposure assessment is the estimation of the exposure resulting from the presence of contaminants in air, soil/sediment or water. It often involves contaminant fate and transport modelling to assess the exposure associated with indirect pathways in which contaminants must travel some distance before they reach a receptor.

The data in Section 4 indicate that mercury-impacted groundwater is present down-gradient of the former cell room but the plume has not reached the northern property boundary, located approximately 100 m from the former cell room. Because the impacted groundwater is located at depth, and is not currently discharging to an ecological habitat, there are currently no complete exposure pathways for ecological receptors. However, there is potential for mercury-impacted groundwater to migrate and discharge to Pictou Harbour in the future.

A two-dimensional contaminant fate and transport model was used to predict mercury concentrations that could discharge to the harbour in the future. The

model assumes a constant uniform groundwater flow and accounts for the following contaminant transport features:

- advection (i.e., transport at the speed of groundwater);
- longitudinal and transverse dispersion (i.e., plume spreading), and
- retardation (slowing down of mercury migration due to sorption to aquifer material).

For modelling purposes, the source of mercury contamination below the former Cell Room was approximated as a rectangular mass measuring 18 m wide and 10 m deep. Although the width of the mercury-impacted zone has been measured during the remedial program, the vertical extent is uncertain. The maximum concentration of dissolved mercury in groundwater at the source was assumed to be 60 μg/L. Literature values for the solubility of mercury are in the range of 25 μg/L to 60 μg/L (Environmental Canada, 1984) and the maximum concentration measured in groundwater at the site is 45 μg/L (see Section 4). The mercury source in the bedrock was modelled as a long-term, continuous source that remains at a constant concentration of 60 μg/L (i.e., there is no source decay). Further details about the assumptions and input parameters used in the model are presented in Section 5.1.6 and Appendix E.

The potential concentration of mercury in the harbour was predicted by dividing the maximum predicted mercury concentration in groundwater discharging to the harbour by a dilution factor. The dilution factor was estimated by assuming complete mixing between the plume and the harbour. The flow rate in the harbour available for diluting the plume was estimated from reported flow rates for the Pictou Harbour, East River and Middle River of Pictou. A review of the watershed areas for the East, Middle and West Rivers of Pictou was also carried out. Further details about the assumptions and input parameters used for predicting potential mercury concentrations in harbour water are presented in Section 5.1.6 and Appendix E.

Table 5-1 Summary of Modelling Results	
Parameter	**Predicted Value**
Time for leading edge of Hg plume to reach the harbour (based on a leading edge plume concentration of 0.1 μg/L)	200 years
Maximum Hg concentration in groundwater discharging to harbour (prior to dilution in harbour)	6.3 μg/L
Hg concentration in harbour water (after dilution in harbour)	0.0001 μg/L
Total amount of Hg discharging to harbour	28 grams/year
Hg concentration in harbour sediment	0.0001 mg/kg

5.1.4 Hazard Assessment

A hazard assessment determines the existence of a hazard by investigating the relationship between a contaminant and the receptors exposed to the contaminant.

The fate and transport modelling in Section 5.1.3 indicated that there is potential for mercury to migrate from the site and discharge to the harbour. This may cause aquatic life to be exposed to elevated mercury concentrations in the harbour water and sediment. The main concern regarding elemental mercury in the environment is bacterial conversion to methyl mercury, which is highly toxic to aquatic life, and mercury's ability to bio-accumulate in the food chain. Aquatic organisms may accumulate mercury either directly from water or through the food web. The biological half-life for mercury in fish is approximately two years. Because of rapid uptake and slow cleansing rates, bio-accumulation factors for aquatic organisms are high (approximately 10,000) (CCME, 1987). Table 5-2 presents CCME ecological guidelines for mercury in soil and sediment.

5.1.6 *Uncertainty Analysis*

Risk characterization involves a number of assumptions. In particular, the models used to estimate exposure concentrations in the harbour use a number of simplifying assumptions to facilitate contaminant transport calculations.

A qualitative uncertainty analysis was carried out to assess the degree of uncertainty associated with the modelling assumptions and whether or not the uncertainty was acceptable. The uncertainty analysis evaluated the applicability of the major modelling assumptions and the confidence in key model input parameters. An assessment of whether the assumption could lead to an over-estimate or under-estimate of risk was also carried out.

The results presented in Table 5-4 indicate that exposure concentrations and risks are generally expected to be over-estimated. This is considered acceptable because it is protective of the environment. Some assumptions involving the amount of plume dilution in the harbour may lead to an under-estimation of risk. This is not considered to be an immediate concern because of the length of time estimated for the plume to reach the harbour (200 years) and the very low levels that are predicted to result in the harbour (i.e., three orders of magnitude below guidelines). However, as a precaution, it is recommended that a monitoring program be put in place to confirm the modelling predictions.

5.1.7 *Summary of Ecological Risk Assessment Findings*

The risk assessment indicated that there is potential for mercury-impacted groundwater at the site to migrate and discharge to Pictou Harbour and this could cause ecological receptors to be exposed to elevated mercury concentrations in harbour water and sediment. However, due to slow rate of mercury migration, groundwater plume dispersion, and dilution in the harbour, the mercury is expected to pose negligible risk to ecological receptors.

APPENDIX H-3

From: 20(1)
To: 20(1) (20(1) northernpulp.com); Porter, Gary S
Cc: 20(1) (20(1) northernpulp.com); 20(1) 20(1) @northernpulp.com); Ken Frei
Subject: Envoi électronique - Alt D 2D modelling results.pdf
Date: May 29, 2017 3:14:19 PM
Attachments: Alt D 2D modelling results.pdf

20(1)/ Gary,

Attached please find the superimposition of the 2D modelling results for Alternate outfall locations D (500m further and 0.5m deeper than Alt.C) and D2 (1,000m further and 1.0m deeper than Alt.C). Both D and D2 show no backwash of effluent to Boat Harbour while D2 shows no presence of effluent (I'm not calling it an impact…) in traditional lobster fisheries off of Chance Harbour and a minor residual in herring fisheries off of Cole Point and Bay View.

My recommendation would be to proceed with 3-D modelling using the furthest point (D2)

Let me know what you think and when you would be available to have a chat with Stantec on this.

20(1)

.....

20(1)

Conseiller Principal, Procédés et Environnement /
Principal Consultant, Process and Environment

KSH Consulting
1 Place Alexis Nihon
3400 de Maisonneuve O., bureau 1500
Montréal (Québec) Canada H3Z 3B8
T 20(1) **M** 20(1) **F** 514.939.5266

KSH

www.ksh.ca

mail in our communications with you.

APPENDIX H-4

Introduction

Mandate

1. KSH Consulting was requested to prepare a study benchmarking Canadian effluent discharge permits from chemical pulp mills in order to assess current industrial practices in terms of effluent treatment systems performance and how they compare to proposed new limits facing the Northern Pulp Nova Scotia (NPNS) facility in New Glasgow.
2. The study is required by NPNS for their discussions with government authorities (Nova Scotia Environment) to ascertain if proposed new effluent permits for the mill are comparable and achievable given the age of the mill, technologies employed and the nature of the effluent treatment facilities at NPNS.

Background

3. The Kraft mill located on Abercrombie Point, in the town of New Glasgow NS, has been in operation since 1967. The mill produces bleached (ECF) Kraft market pulp at a current rate of ~300,000 ADt/a. At the time of construction, the Government of Nova Scotia agreed to build and operate a treatment system for the mill's process effluent, which was constructed in the western portion of an area known as Boat Harbour. The system was refurbished in 1996, at which time the mill took over operation of the system. The effluent treatment system consists of constructed sedimentation basins followed by aeration in a natural basin with baffle curtains directing flow. A large, natural final polishing/stabilization basin follows prior to release to the Northumberland Straight.
4. While the system has met federal and provincial standards since 1996, there has been pressure to return some of the natural waterways to their original state and increasingly stringent environmental regulations are being proposed that may require modifications to the system or the effluent streams that feed it to continue operating in the years to come.

Total Suspended Solids (TSS)

1. TSS limits (along with BOD_5) are the longest established pulp and paper emission regulations on the books. From early on, even before organic load mitigation, mills would collect solids coming from their process that were found to be overloading local waterways. Mitigation has ranged from settling ponds to raked clarifiers.
2. As mills have evolved, more and more fibre (the main solid waste) has been retained in the process and sold as product instead of being lost to effluent. This has resulted in a steady reduction in TSS losses.
3. NPNS's proposed TSS daily limit of 4,100 kg/d and monthly limit of 2,460 kg/d equates to approximately 5 and 3 kg/tADt of production respectively, based on 800 ADt/d.
4. As can be seen in the following charts, this would place NPNS as the one of the most tightly restricted emitters of the mills compared for both the daily and monthly limits.
5. NPNS has been able to maintain levels just below this new limit performing at annual average levels of 3.26, 2.61 and 2.05 kg/ADt at Point C for 2011, 2012, and 2013 respectively. In fact, a marked downward trend can be seen.
6. Continued TSS compliance is in fact more difficult for NPNS since there is no final clarifier and lagoons can be prone to rising solids and upsets. Point C of the effluent treatment system also benefits from the settling effect of Boat Harbour prior to Point D, so the impact on marine environments is even less pronounced.
7. Historic data over the past two years has shown 10-15% better performance for TSS losses in the summer. This may be due in part to less solids degradation in the ASB in the winter when temperatures are lower. Seasonal differences in performance (winter to summer) are similar to those reported in the BOD section for NPNS.

APPENDIX H-5

From: 20(1)
To: Porter, Gary S
Cc: 20(1)
Subject: Metals in Effluent
Date: April 7, 2017 9:42:31 AM
Attachments: Wastewater Metals - Middle River, Point C and Port Hawkesbury.xlsx

---

Hi,
Port Hawkesbury contacted me after our meeting yesterday. They found an old metal analysis from 2001 and forwarded it to me. I asked 20(1) to add a column in our chart for PHP. Everything in yellow is above the CCME guideline. So basically, they are just the same as us. Neither of us can meet that standard.
Cheers,

20(1)
Technical Manager
Northern Pulp Nova Scotia
20(1)

Wastewater Metals Analysis

			Middle River	Middle River	NPNS Point C	NPNS Point C	NPNS Point C	Port Hawkesbury
Parameter (Total)	RDL	Guideline FWAL	Raw Apr 2016	Raw Jan 2016	2-15-2015	2-16-2016	2-17-2017	PHP 2001
Aluminum	5	5	420	448	2330	1710	1800	200
Antimony	2		<1	2	<2	<2	<1	<2
Arsenic	2	5	<1	<2	<2	<2	1.2	9
Barium	5		30	36	339	354	450	92
Beryllium	2		<1	<2	<2	0	<1	0
Bismuth	2		2	<2	<2	2	<2	
Boron	5	29,000	<50	7	58	61	94	<2
Cadmium	0.017	1.0,0.9	<0.010	<0.17	1.11	0.898	1.4	1.86
Chromium	1		<1	<1	3	2	2.1	2.8
Cobalt	1		<0.040	<1	<1	<1	0.67	1.3
Copper	1		<2	<1	6	5	7.5	55.6
Iron	50	300	480	367	439	718	530	1220
Lead	0.5	1	0.061	<0.5	3	2.7	2.8	8
Manganese	2		1800	70	2710	2490	2800	2510
Molybdenum	2	73	2	<2	4	2	2.7	<4
Nickel	2	25	<2	<2	5	4	2.9	7.5
Selenium	1	1	<1	<1	<1	2	<1	1.7
Silver	0.1	0.25	0.2	<0.1	0.2	0.2	0.37	<0.2
Strontium	5		30	38	163	144	160	118
Thallium	0.1	0.8	<0.1	<0.1	<0.1	<0.1	<0.1	0.8
Tin	2			<2	<2	<2		<2
Titanium	2			9	11	14		
Uranium	0.1	33,15		<0.1	0.9	0.7		3.95
Vanadium	2		5	<2	4	5	4.8	19.9
Zinc	5	30	<5	<5	127	76	160	184
Mercury	0.026	0.026	0.026	<0.026	0.037	0.026	0.028	<0.1

All units ug/L.
Lead FWAL guideline based on hardness of 26ppm,
Nickel FWAL guideline based on hardness of 26ppm.

APPENDIX H-6

NOVA SCOTIA
NOUVELLE-ÉCOSSE

Environment
Environnement

14/06/17

Northern Pulp Nova Scotia Corporation
PO Box 594, Station Main
New Glasgow, NS
B2H 5E8

Attn: 20(1)
General Manager

Dear 20(1):

RE: Minimum Requirements of a Receiving Water Study

Further to our meeting on June 7, 2017, at your request, the Department offers the following general guidance with respect to the terms of reference for receiving water studies and how they are used to establish end of pipe effluent discharge criteria:

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 must be designed to be as small as possible and it is only one factor to be considered in establishing effluent requirements.

The concept of mixing zones recognizes that the release of the aqueous component of adequately-treated municipal or industrial wastes to watercourses or water resources does occur. 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 or dangerous goods. Mixing zones also do not apply to bio-accumulative or persistence substances and despite the allowance of a mixing zone, effluent shall not be acutely toxic. It should be noted that in this particular case, a receiving water study must address all potential substances of concern not limited to those outlined in the Federal Pulp & Paper Effluent Regulations.

Conditions within a mixing zone must not result in irreversible environmental damage, risk to ecosystem integrity or risk to human health. Mixing zones cannot interfere with other water uses such as drinking water supply, active fisheries or recreation.

As effluent loading requirements are based on careful design, so too should mixing zones be carefully planned on a site-specific basis including consideration of water quality, seasonal streamflow and current patterns, physical factors, biotic communities and spawning areas in and adjacent to the mixing zone, nearby water uses such as public or private beaches and drinking water intakes as well as other wastewater discharges. This information should be provided to the Department.

As general guidance in the design of mixing zones, the following range of concerns should be adequately addressed:

1. In order to protect important aquatic communities (fish, invertebrates and plants) in the vicinity of mixing zones, no conditions within the mixing zone will be permitted which:
    a. are acutely lethal to aquatic life;
    b. cause irreversible responses which could result in detrimental post-exposure effects;
    c. result in bioconcentration of toxic materials which are harmful to the organism or its consumer;
    d. attract organisms to the mixing zones, resulting in a prolonged exposure;
    e. create a barrier to the migration of fish or other aquatic life.
2. To ensure the protection of acceptable aesthetic conditions, mixing zones should not contain:
    a. materials which form objectionable deposits (e.g. scums, oil or floating debris);
    b. substances producing objectionable colour, odour, taste or turbidity;
    c. substances which produce or contribute to the production of objectionable growths of nuisance plants and animals;
    d. substances that render the mixing zone aesthetically unacceptable.
3. Mixing zones should not impinge upon existing municipal and other water supply intakes, public or private beaches or important fish spawning and/or fishing areas. Conversely, new intakes or aquatic recreation areas should not be constructed within the boundaries of existing mixing zones.
4. Mixing zones may overlap unless the combined effects exceed acceptable conditions.
5. When background water quality conditions at a proposed mixing zone site are degraded, effluent discharge requirements established must ensure, at the very least, that background water quality is not further degraded.

The Department requires enough information to ensure each of the above concerns is adequately addressed. Specifically including but not limited to:

- information about the effluent (substances of potential concern, volumes, etc.);
- information about the receiving water (physical characteristics, size, upstream and downstream water quality);
- the location of the receiving water in relation to the facility, the treatment system and the outfall (including a drawing or plan); and
- location of any nearby receptors

With respect to derivation of effluent limits, effluent requirements can be established based on treatment-technology, receiving water concerns, aquatic habitat concerns as well as community concerns including recreational uses. An important factor in the assessment is the use of the principles of assimilative capacity and mixing zone, but it is not the only factor.

The information provided to the Department should include one year's worth of effluent characterization data. For contaminants which are not considered to be designated hazardous

contaminants, every watercourse/water resource has a definable assimilative capacity. Once the assimilative capacity of the receiving water has been determined, end of pipe discharge criteria can be calculated. Water quality considerations take precedence when contaminant discharges exceed the assimilative capacity of the receiving waters, even if the discharged loadings are within the treatment technology based effluent requirements based on the guidelines, regulations or policies. Receiving-water based effluent requirements also take precedence when ambient levels of contaminants are above acceptable levels. Biological effluent requirements may also be specified.

All effluent discharges must not be acutely lethal.

CCME has a guidance document, ***Guidance on the Site-Specific Application of Water Quality Guidelines in Canada: Procedure for Deriving Numerical Water Quality Objectives,*** which you can reference which may be of assistance to you. It can be obtained at the following link: http://ceqg-rcqe.ccme.ca/download/en/221 .

Should you have any questions or concerns regarding this information, please do not hesitate to contact Paul Keats, District Manager at 902-863-7600.

Regards,

Kathleen Johnson, P.Eng.
Engineering Specialist

cc: Sarah Jadot, P.Eng., Regional Engineer
Paul Keats, District Manager
Adrian Fuller, Executive Director
Frances Martin, Deputy Minister

APPENDIX H-7

From: 20(1)
To: 20(1) 20(1) 20(1) Porter, Gary S
Subject: Open houses - ideas for story boards
Date: November 15, 2017 6:57:49 PM
Attachments: image002.png

Hi All,

This is a combination of things that Mike and I have put together as things we should think about addressing through the EA process:

TV screens for 2-D videos. How to present at the meeting?

3 D story board – show effluent plume in relation the background mixing zone and point of outfall for major parameters

EEM summary

History of effluent – existing limits and data over the years (maybe production or maybe express in units/t)

History of effluent – Canso Chemicals, ECF bleaching

History of effluent – how BH came about, legal and historical timelines and possible etc upgrades over the years

Metals in effluent – recent facebook questions (NSE has a piece of this)

Water quality of middle river – not drinking water standards, has color is Michelin effluent. Spills over to harbour if not used.

Effluent parameters – difference from today. Jars made up for color (show historical) and possibly jars made up for TSS. – have several sets of jars made up

Cartoon diagram of AST system – dynamic model showing water going through it.

Aerial maps of areas

Air emissions - difference in odour from existing (septic settling stage, etc), burning of sludge will have to be identified. Possibly get samples of sludge from Port Hawkesbury.

Possibly bring microscope.

History of kraft mills in North America – details of what their systems are.

Ask Domtar is we can use their pictures

Should we bring lobster taste test studies from the 90s?

Where are the moreturium areas for shellfish in the strait?

Background samples of water – GHD ??

No backflow into BH after return to tidal

Show layout – federal water vs provincial water

In mill processes - o2 delig & process water cooling towers (non-contact & water recycle)

Show that fresh water goes over dam and into strait whether or not it goes through the mill or not (out at lighthouse beach). Concern over big fresh water influx.

Michelin effluent is our influent

Fisherman talking about lobster larvae and 30 ft depth that they float and then sink.

Explain toxicity testing – usually means low oxygen & explain how test is done with fish (how to visualize)

Biologist question about adding heat to the strait – RWS less than 1 C (will be higher temp than today however)

Fisherman concerned about stirring up Mercury while we build near the Canso property

Goldboro experience - increase in lobster habitat

Fisherman worried about build up of bottom sludge – specific reference to scallop fishing

Biologist worried about low water circulation in the strait – bottom warming (explain large study area)

Fisherman worried about contamination in BH now and when opened up – mixing up the 2 projects

Worried about oxygen in the effluent – dead zone for fish

Why is new AST better than ASB?

Facebook reference to Paul Klopping Supreme Court appeal letter – odour issues with ASTs too

Bring piece of HDPE outfall pipe – comparison to fiberglass line current

Fisherman concern about difference between PHP and NPNS is that strait of canso is ice fee and Northumberland is not – will effluent get trapped and not flow? How does ice cover affect outfall?

20(1) – right now mill has BH as large buffer zone, so effluent at point C is not comparable to new effluent. Need to compare to Point D or speak to difference between current point C and D.

20(1) - Why not closed loop system? Offering up a cheap solution, not a good solution

20(1) has 2012 KSH study that says AST quality not significantly different than ASB

20(1) – all toxic sludge will now be burned in the PB and end up as air emissions.

20(1)
Technical Manager
Northern Pulp Nova Scotia
P: 20(1)
C:
Paper Excellence Canada
www.paperexcellence.com

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APPENDIX H-8

From: 20(1)
To: 20(1)
Cc: 20(1) dillon.ca; Porter, Gary S; 20(1) 20(1) 20(1)
Subject: Re: Media Inquiry
Date: November 17, 2017 12:45:51 PM

Article is up from CBC

http://www.cbc.ca/news/canada/prince-edward-island/pei-peifa-wastewater-treatment-plant-1.4406788

20(1)
Dillon Consulting Limited
137 Chain Lake Drive Suite 100
Halifax, Nova Scotia, B3S 1B3
T - 20(1)
F - 902.450.2008
M - 902.240.7311
20(1) dillon.ca
www.dillon.ca

Please consider the environment before printing this email

On Wed, Nov 15, 2017 at 7:03 PM, 20(1) 20(1) @northernpulp.com> wrote:

> FYI. Late afternoon media inquiry from CBC PEI. Original questions plus reporters follow up below.
> 20(1)
>
> 20(1)
> Communications Director
> Paper Excellence
> Northern Pulp Nova Scotia Corporation
> 20(1)
> 20(1) (cell)
> 260 Granton Abercrombie Branch Rd
> Abercrombie, NS B2H 5C7
>
> Begin forwarded message:
>
> **From:** "20(1) paperexcellence.com" <20(1) paperexcellence.com>
> **Date:** November 15, 2017 at 3:59:38 PM AST
> **To:** 20(1) @CBC.CA"
> 20(1) @CBC.CA>
> **Subject: Re: Media Inquiry**
>
> Hi 20(1)
> See updated responses to your additional questions below
> 20(1)
>
> Thanks for getting back to me. I do have a few more questions.
>
> 1. Would the new facility fall under the current lease with the province?

The facility will be built on the mill site.

2. When would you expect a formal application to be made to the province for the plans?
Dillon Consulting has been hired to assist in the Environmental Assessment (EA) process. There is much work to be completed before the application can be filed. One of the first steps will be public consultations.

3. There are a lot of concerned fishermen that are worried about how this new treatment facility will affect the water in the Northumberland Strait. Are they right to be concerned?
Treated effluent has been flowing through Boat Harbour and into the Northumberland Strait for over 50 years. The new treatment facility and diffused outfall will reduce the impact on the Strait. For example, the new outfall is designed to meet the Canadian Council of Ministers of the Environment (CCME) guidelines for effluent discharges which stipulate that all effluent parameters of concern should meet background concentrations of the receiving water in less than 100 metres from the outfall.

4. If the plans are rejected by the province, what is the next step for the company?
We are working cooperatively with the Government of Nova Scotia to design a treatment facility that meets all environmental requirements.

On Wed, Nov 15, 2017 at 2:54 PM, <20(1) paperexcellence.com> wrote:
Hello 20(1)

Thank you for your email. Please see below bulleted information answering your query. Please feel free to email me if you have further specific questions and deadline you are working with. I am in meetings for the remainder of the day but will be able to access email periodically.

You may attribute the response to 20(1) Director of Communications, Paper Excellence
Kindly
20(1)

-KSH Solutions Inc has recently completed preliminary engineering where all available technologies were reviewed. Activated Sludge Treatment (AST) was recommended as the best and most reliable process for treatment for the new facility to be located on existing mill property.

-The proposed new treatment system will be a modern AST system that mills and other facilities (industrial, municipal, etcetera) throughout the world have in place.

-The current effluent treatment system is an ASB system. In North American Kraft mills, there are predominantly two systems used to biologically treat industrial wastewater, the aerated stabilization basin (ASB or aerated lagoon) and the activated sludge system (AST). These two systems have far more similarities than differences. Both processes are based on the utilization of dissolved oxygen by microorganisms in converting organic and inorganic matter into a settleable form. The ASB process uses a large aerated lagoon and long retention times (days) whilst the AST process treats the wastewater in hours and involves two stages – an aeration stage and a clarification and recycle stage. AST systems generally operate at higher removal efficiencies as compared to ASB systems.

-In early October, Dillon Consulting of Halifax was contracted to provide professional guidance throughout the entire Environmental Assessment process, including creation of the Environmental Impact Assessment (EIA) project registration document.

-Presentation of all scientific information as well as documentation of public and stakeholder concerns, complete with how they will be addressed, are an important and vital part of the consultation process and EIA document.

-The effluent pipeline (outfall) will be designed with an engineered diffuser which is also an improvement from the current system.

-The new outfall is designed to meet the Canadian Council of Ministers of the Environment (CCME) guidelines for effluent discharges which stipulate that all effluent parameters of concern should meet background concentrations of the receiving water in less than 100 metres from the outfall.

-Construction would begin upon registration and approval of the project by Nova Scotia Environment.

From: 20(1) [redacted] @CBC.CA]
Sent: Wednesday, November 15, 2017 1:50:37 PM
To: Media
Subject: Boat Harbour Plans
Auto forwarded by a Rule

Good afternoon, I am working on a story about the proposed plans for the new

wastewater treatment facility in Pictou, N.S.

I am hoping to speak to someone from Northern Pulp about how the wastewater system will work, and why that location and type of treatment system was chosen as well as what the next steps in the application process would be.

Feel free to shoot me an email or give me a call on my cell, the number is listed below.

Thanks very much.

--
20(1)
Video Journalist/ Web Writer CBC PEI
20(1)
20(1) @cbc.ca

--
20(1)
Video Journalist/ Web Writer CBC PEI
20(1)
20(1) @cbc.ca

APPENDIX H-9

CBC

Northern Pulp's plans for pipeline, effluent treatment plant now public

Environment minister has until March 29 to decide whether plan is acceptable

Jean Laroche · CBC News · Posted: Feb 07, 2019 1:15 PM AT | Last Updated: February 7

Northern Pulp has released details of its plan to build a new effluent treatment plant and discharge pipe. (Jeorge Sadi/CBC)

Nova Scotians now have access to the details of Northern Pulp's controversial plan to build a new effluent treatment plant and discharge pipeline that will empty into the Northumberland Strait.

The Pictou County pulp mill's 614-page document, including 18 appendices, was filed with Nova Scotia's Environment Department a week ago and was posted Thursday on the

department's website.

The plan put forward to the Environment Department is to build a "biological activated sludge" treatment facility purchased from a Paris-based multinational corporation called Veolia Water Technologies.

The corporate website says Veolia Water "specializes in water treatment solutions and provides the complete range of services required to design, build, maintain and upgrade water and wastewater treatment facilities for industrial clients and public authorities."

Nova Scotia Environment Minister Margaret Miller said the nearly 2,000-page submission was not a surprise.

"I think it's pretty much what the department was expecting," she said.

Safe drinking water a concern

The treatment facility would be located on Northern Pulp property not far from the existing plant.

The 15.5-kilometre pipeline would run from the new facility along the shoulder of Highway 106 to Caribou before entering Caribou harbour next to the Northumberland Ferries terminal. From there, it would discharge roughly four kilometres into the Northumberland Strait.

A boat doing survey work for the proposed Northern Pulp effluent pipe is tied to the wharf in Pictou, N.S. (Submitted by Ben Anderson)

That route is a concern for the town of Pictou. Mayor Jim Ryan said it means wastewater will be piped over the town's main watershed.

"This particular issue is about safe drinking water," he said in a telephone interview Thursday.

Ryan said he told Northern Pulp general manager Bruce Chapman in November that any plans for a pipe that carries treated or untreated effluent through the watershed would be unacceptable to the town.

Work would take 21 months

Northern Pulp's plan to discharge treated effluent into the strait has also been controversial.

Thousands protested last July over concerns it would hurt the environment. Fishermen had also prevented a survey crew from doing work for the company, but agreed last month to a court injunction ordering them not interfere.

Northern Pulp protesters outside a Supreme Court injunction hearing late last year. (Preston Mulligan/CBC)

Company owners have also sought a one-year extension of the provincial law requiring the mill's current treatment facility in Boat Harbour to close in January 2020. The company has argued it needs more time to build a replacement, but Premier Stephen McNeil has refused to extend the deadline.

According to company documents, the plan is to complete the work within 21 months, starting this spring. That means a working system would not be in place until 11 months — at the earliest — after the provincial government is legally mandated to turn off the tap to the provincially owned treatment plant.

Pipe would mostly be buried

The company has proposed using a polyethylene pipe that's 90 centimetres in diameter to carry the treated effluent from the plant to the dispersal site.

"The terminus of the effluent pipe consists of an outfall location with the three-port diffuser, situated at the depth of approximately 20 [metres]," says the project description.

The plan is to bury the pipe along most of the route, but the company is proposing suspending it to the exterior of the bridge that crosses the Pictou Causeway "due to the limited roadway width."

"The exposed area will be protected from damage by existing guard rails," says the document.

Northern Pulp's proposed route for the effluent pipe would go from a new treatment plant into the Northumberland Strait. (Nic Meloney/CBC)

The company has promised to mark the pipeline location with signs and post markings at public and private roads and water crossings. The system will also need a pumping station which the company states "will operate in a similar manner to municipal pumping station."

Serious impact on lobster 'highly unlikely'

The company said it looked at alternatives to the plan it has submitted for provincial approval, including simply shutting down or creating a closed wastewater recovery system, but none was feasible.

An indication of how much the company wants an extension is the people it has hired to lobby the governing Liberals on its behalf: Kirby McVicar, McNeil's former chief of staff; Stephen Moore, McNeil's former director of communications; and Trevor Floyd, a one-time executive assistant to Health Minister Randy Delorey when he held the environment portfolio.

- **Premier unmoved by Northern Pulp's ask for more time to close waste water facility**

As for concerns expressed by opponents to the plan, the company has included a response to 38 questions or comments, ranging from the possible harm to lobster stocks to heavy metal

contamination and the environmental review process.

The company stated "it is highly unlikely that there will be serious impact on lobster," and that heavy metals occurred naturally in the environment "and are released to the environment from a range of human and natural sources."

Public invited to submit comments

As for the review process, Northern Pulp noted it was a provincial process but the Canadian Environmental Assessment Agency would review the company's application to determine whether a federal environmental assessment was necessary.

Nova Scotia Environment Minister Margaret Miller has until March 29 to make a decision on Northern Pulp's plan. (CBC)

The public now has until March 9 to digest the information and submit their comments, either by mail or using an online form.

Miller has until March 29 to decide if the project will be granted conditional environmental assessment approval. Officials in her department will sift through the material to ensure it provides a complete picture of the plan and its potential impact on the environment.

If additional work is needed, they can ask the company to provide it. But Miller said the consultation period would not be extended beyond the 30 days if that were to happen.

She acknowledged the existing file could be a challenge for Nova Scotians to assess.

"I don't know that the public is really going to be able to fully digest everything that's been submitted."

APPENDIX H-10

From: 20(1)
To: 20(1) 20(1) 20(1) 20(1) 20(1) 20(1) 20(1) Porter, Gary S; 20(1) 20(1) 20(1) 20(1)
Cc: 176461
Subject: RE: Mill proposed slides - story we need to tell (updated with people in charge of slide preparation
Date: November 30, 2017 4:59:18 PM
Attachments: image002.png
image007.png
image008.png

Hi All – I am pairing these down to a story board, so don't put any time into these yet.

Northern Pulp Nova Scotia
P:
C:
Paper Excellence Canada
www.paperexcellence.com

Excellence… It is in our name, our vision, and our values

Since its inaugural year in 2010, Paper Excellence Canada (PEC) has evolved from a new entrant as an exporter to a leader in Canada's value-added natural resources (pulp) industry. Achieving sales in excess of $1 billion through its seven Canadian mills, PEC has accomplished this level of sales volume through innovation and best practices in all areas with a commitment to deliver premium quality products to all customers. Through its Richmond-based headquarters which hosts an employee base of 100 supporting over 2300 employees in Canada and Europe, PEC continues to lead by example operating through economic and environmentally sustainable best practices.

From: 20(1) [mailto:20(1) ksh.ca]
Sent: Thursday, November 30, 2017 2:52 PM
To: 20(1) <20(1) northernpulp.com>; 20(1) <20(1) dillon.ca>; 20(1) <20(1) dillon.ca>; 20(1) 20(1) r@northernpulp.com>; 20(1) <20(1) dillon.ca>; 20(1) <20(1) northernpulp.com>; 20(1) <20(1) northernpulp.com>; Gary Porter <gary.s.porter@novascotia.ca>; 20(1) <20(1) raycon.ca>; 20(1) <20(1) dillon.ca>; 20(1) <20(1) dillon.ca>; 20(1) 20(1) @dillon.ca>
Cc: 176461 <176461@dillon.ca>
Subject: RE: Mill proposed slides - story we need to tell (updated with people in charge of slide preparation

Hi 20(1)

Here's what I was able to put together, based on the comments in your e-mail. It's all under the same file (the zero-effluent file I sent yesterday), which is dated today and is in PowerPoint format (easier to extract)

I included some additional info that could be useful to others as well.

Let me know if you need anything else

20(1)

.....

20(1)
Conseiller Principal, Procédés et Environnement /
Principal Consultant, Process and Environment

KSH Consulting
1 Place Alexis Nihon
3400 de Maisonneuve O., bureau 1600
Montréal (Québec) Canada H3Z 3B8
T 20(1) **M** 20(1) **F** 514.939.5266

KSH

www.ksh.ca

From: 20(1) [mailto:20(1) northernpulp.com]
Sent: November 30, 2017 8:53 AM
To: 20(1) 20(1) 20(1) 20(1) 20(1) 20(1) Gary Porter; 20(1) 20(1) 20(1) 20(1) 20(1)
Cc: 176461
Subject: Mill proposed slides - story we need to tell (updated with people in charge of slide preparation

Hi – please see additional notes below.

20(1)
Technical Manager
Northern Pulp Nova Scotia
P: 20(1)
C:
Paper Excellence Canada
www.paperexcellence.com

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From: 20(1)
Sent: Wednesday, November 29, 2017 8:34 PM
To: '20(1) <20(1) dillon.ca>; 20(1) <20(1) dillon.ca>; 20(1) <20(1) 20(1) <20(1) dillon.ca>; 20(1) <20(1) northernpulp.com>; 20(1) <20(1) northernpulp.com>; Gary Porter <gary.s.porter@novascotia.ca>; 20(1) <20(1) raycon.ca>; 20(1)

<20(1) dillon.ca>; 20(1) <20(1) dillon.ca>; 20(1) <20(1) @dillon.ca>
Cc: 176461 <176461@dillon.ca>
Subject: Mill proposed slides - story we need to tell

This is the story I think we need to tell:

Simplified description of EA process – Class I and II (show timeline schematic on handouts that ends in June/July 2018, make a second schematic that starts at Registration and ends at decision and show steps or milestones, point out that other agencies involved (DNR, DFO, trans, ECCC) not just NSE. (Dillon)

What is effluent? Why treat it? What is in it? (20(1)

Map of existing ETC – point out pipeline, current process equipment and dam and outfall to Pictou Road (Dillon)

Water supply to the mill – aerial picture (attached) – will explain 3 rivers running to harbour, Michelin and NPNS water supply, unused water spills over dam, RV Anderson report on sustainability (incredible # of people worried we are going to dilute the Strait with fresh water – they need to know it goes there anyway) – (Dillon to identify 3 rivers, dam, pumping station, causeway underflow, Michelin, NPNS and existing and new outfall on the map) (mill– sample bottle and talking points)

- Sample bottle of color, doesn't meet CCME drinking water standards.

Options considered – how did we pick AST? BAETA methodology. Why not zero effluent, table (summary) of North American systems who has ASB, AST and marine or FW discharges? no mills in NA running anything else. Speak to Europe and asia . Closed loop build up of chloride in our boilers, corrosion control – cathodic protection on your boats. (20(1) to provide)

In mill changes to support ETF – O2 delig and cooling tower slide (and picture of CT) – run seasonally only to reduce summer cooling water flow, RWS did not consider O2 delig improved quality, not sure of start-up and commissioning times, ETC more conservatively designed for extra aeration. Cooling towers use more fresh water due to evaporation. Water and effluent flows not equal. (20(1) – moved after why we picked AST, improvement to support new AST)

New system – 3 pics - block diagram (20(1) aerial layout and pipeline aerial layout (Dillon 2 aerials) – point out that old pipeline was untreated and new pipeline is after treatment.

Differences between AST and ASB – why better process. more modern, confidence in best system. 20(1))

RWS - 30 day lunar cycle July 2016 – most challenging month for river inflow (conservative case) – show map of study area from stantec report. High effluent loading for 30 days straight with no biological degredation. (Dillon)

Why outfall selected where it was? Locations considered in RWS – both inside and outside harbour

Inside harbour could lead to build up of nutrients (nitrogen and phosphorous) – eutrophication. Had to be far enough from entrance to BH to ensure no eutrophication there either. (Dillon)

Design of line – marine geotech, ROV to confirm path not impacting sensitive habitat or marine protected areas. Picture or sample of HDPE pipeline for thickness. Rock mattress, diameter of line, 1 m estimated armour stone cover, create habitat. Why we show a corridor, not a line. Questions about top of line being hazard to navigation – someone said 9 ft minimum at entrance to harbour at low tide – check. Show we are OK? (Dillon)

Design of the diffuser – pictures of diffuser (schematic and photograph - attached), (Dillon)

6 ports chosen – modelled 1, 3 and 6

Meet guidelines for CCME – table of parameters and distances (include 20(1) note on salinity)

Aerial showing mixing zone

stantec sensitivity maps considered – show?

History of Effluent Quality – time/project/impact/effluent sample bottles. Last line will be predictions for the project (NPNS to provide mark up, Dillon to draw up) including 1992 and 1997 changes. Possibly by increments of decades. How do we explain difference between in and out of BH? ASB data from 2009 to present is into BH, data before 2009 is out of BH. Some say effluent quality will be worse than today because of all the polishing that is happening across the BH basin – and they are correct to some extent.

Summary of EEMs – chart of year/study/result (not sure if this is a good idea or not?) 20(1)

Effluent temperature – hotter than now (BH big basin provides a lot of natural cooling today). Give temperatures and explain CCME meeting 1C guideline (0.2 or 0.3 in 100 m this is in July when ocean the warmest. 3 dimensional plume. Mixing zone above the benthic layer. Add Table from Stantec report showing 0.2 C increase. (Dillon)

Solids – concern about large amount of solids (greater than 1000 kg/d). GUY – what percentage of solids is organic or bug bodies? Compare future to current – speak of high solids in history (especially pre 1990s). One of lower mills in Canada. Concern over build-up of bottom sludge and scallop fishing. 20(1) – will we speak to odour m??

Metals/Dioxans and Furans – don't know what to do with this. How to handle questions around burning of sludge that all other mills do? Are municipal ETC's burning sludge? (Dillon) metals from wood – also burned in wood stoves.

List EA studies completed, ongoing and planned. (Dillon)

Like the "anticipated change chart" in LaFarge Cement EA also Irving EA – could we make same? Include positive and negative and things that stay the same – odour, sludge burn vs landfill, color

and organic improvements, solids about same, rock cover, energy reduction, less CO2 emissions, economic, etc. – work on this together on Friday

Four studies to share: Timeframe?

1. RV Anderson – Middle River Sustainability
2. KSH Phase I
3. KSH Phase II ??
4. Stantec RWS

To add to frequently asked questions:

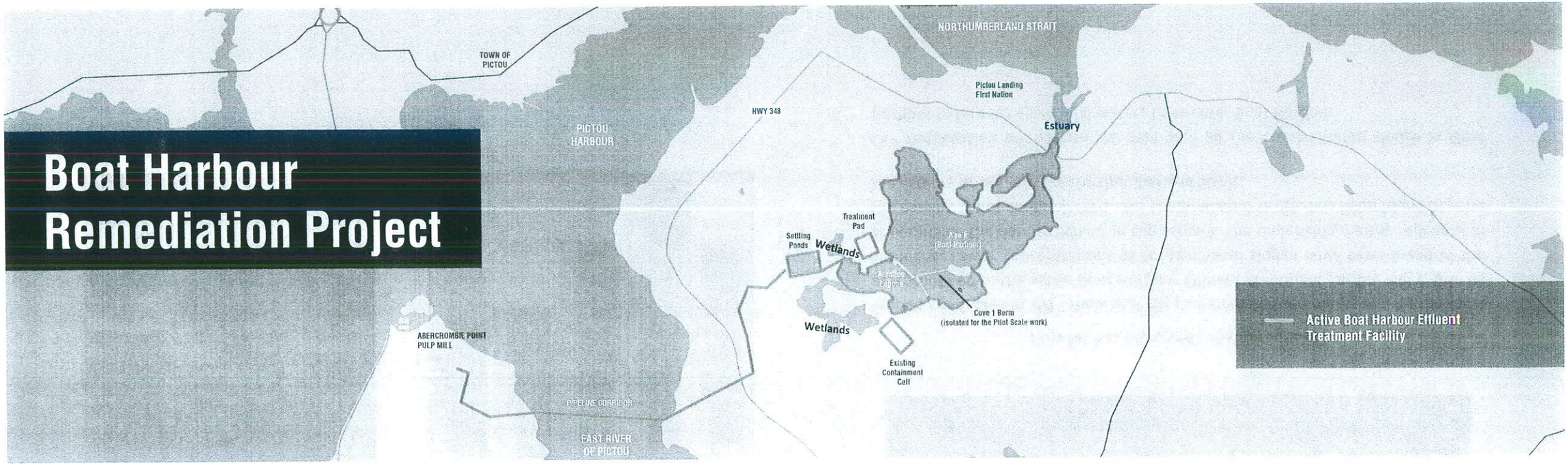
Is dilution the solution to pollution for Northern Pulp? Industry standard limits as well as current PPER limits are mass-based (ex. kg/day) and not based on volume. Therefore adding more fresh water to the effluent does not make effluent limits easier to meet. The RWS was conducted at design flow. Reducing effluent flow results in an improvement in meeting CCME guidelines compare to the higher design flow. Therefore, there is no benefit to adding dilution to the effluent.

20(1)
Technical Manager
Northern Pulp Nova Scotia
P: 20(1)
C:
Paper Excellence Canada
www.paperexcellence.com

Excellence... It is in our name, our vision, and our values

Since its inaugural year in 2010, Paper Excellence Canada (PEC) has evolved from a new entrant as an exporter to a leader in Canada's value-added natural resources (pulp) industry. Achieving sales in excess of $1 billion through its seven Canadian mills, PEC has accomplished this level of sales volume through innovation and best practices in all areas with a commitment to deliver premium quality products to all customers. Through its Richmond-based headquarters which hosts an employee base of 100 supporting over 2300 employees in Canada and Europe, PEC continues to lead by example operating through economic and environmentally sustainable best practices.

APPENDIX H-11



Boat Harbour Sludge – Our Problem

What contaminants are in Boat Harbour?

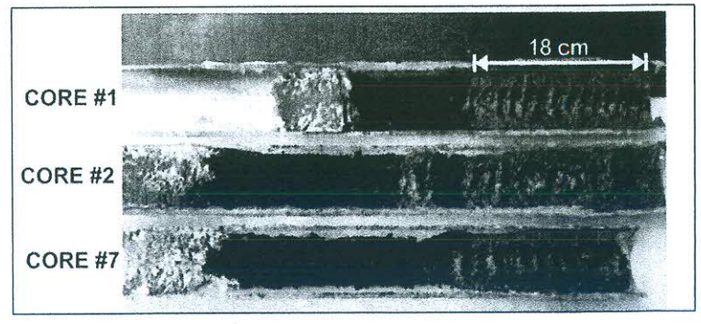
A layer of contaminated sludge has settled on top of the Boat Harbour bottom. This sludge has been accumulating since 1967. It has been sampled many times over the years. A full suite of testing was completed in 2017 which has confirmed the contaminants in the sludge. Over time, the contaminants have not changed.

We know the sludge contains:

- Dioxins and furans, the principal contaminants of concern are carcinogens which are residues of industrial processes
- Metals such as mercury, cadmium and zinc, which are residues of industrial processes
- Polycyclic aromatic hydrocarbons (PAHs), which can be produced by incomplete combustion of fossil fuels in engines and boilers or from forest fires
- Total petroleum hydrocarbons (TPH), a term used for any mixture of hydrocarbons that are found in crude oil and petroleum products
- Volatile organic compounds (VOCs), include human made residues from industrial processes and naturally occurring chemical compounds.

The contaminated sludge is generally less than a foot, or 30 centimeters, thick and is black in colour while the underlying marine sediment is brownish gray and is not contaminated.

The wetlands above Boat Harbour have also been impacted from the early years of Mill operations and contains contaminated sediments.



This image shows several core samples taken from Boat Harbour, the black contaminated sludge is clearly visible; the brownish gray is the clean Pre-industrial marine sediment and, the bentonite is a clay product put in the core during sampling as a plug - **Note:** The figure shows from left to right bentonite plug, black contaminated sludge, brownish gray pre-industrial marine sediment

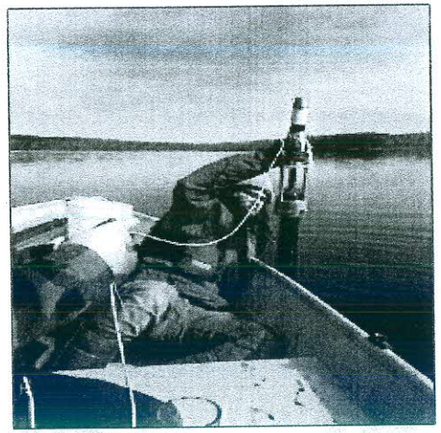
Boat Harbour Remediation Project

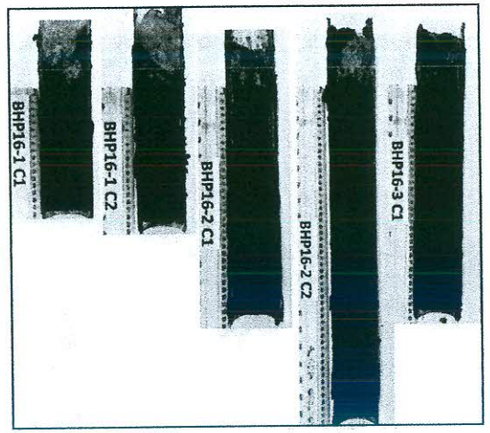
How much is there?

The contaminated sludge in Boat Harbour is of a soft, wet nature and is unevenly distributed along the harbour bottom. We have estimated the volume of material to be removed from Boat Harbour to be as much as 1,000,000 cubic meters. This number includes some of the marine sediment on the harbour bottom that will accompany removal of the contaminated sludge. To effectively remove the contaminated sludge, we need to take some of the marine sediment. Once the material is removed it will be treated, dewatered and its volume will be significantly reduced to about 500,000 cubic meters.

Sampling procedure

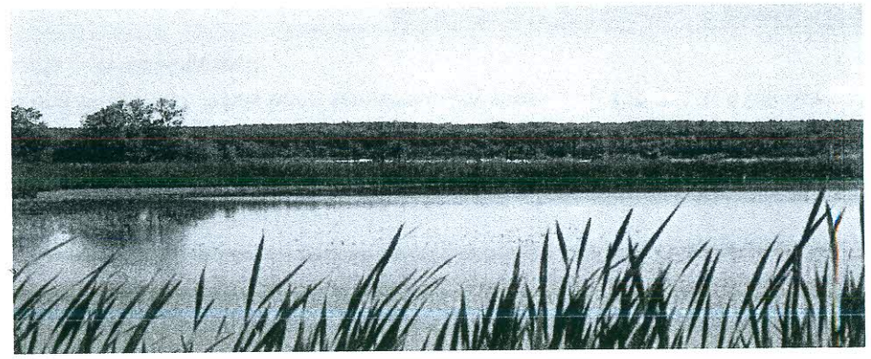
All sampling followed scientific protocols based upon generally accepted procedures. These protocols were vetted with Dr. Ian Spooner, a geoscientist and sampling expert from Acadia University.





The wetlands

Twenty-five wetland areas are around Boat Harbour and are identified as marsh and/or swamp complexes. One wetland of about 36 hectares in area, pictured below, is situated near the existing settling ponds and was impacted by early years of effluent discharge. The remediation planning is considering either removal of all the impacted sediments or a risk-based approach which may involve removal of areas of higher contamination and allowing monitoring and natural attenuation (treatment) for the balance of the wetland.



How far has the contamination spread?

Studies conducted in 2017 show that the contaminated sediments are, for the most part, confined to the active Boat Harbour Effluent Treatment Facility and within its shoreline. Lower concentrations of contaminated sludge have been found in the area outside the dam structure, in the estuary. The underlying marine sediment in the estuary is not contaminated. No contaminated sludge has been found beyond the estuary or out into the Northumberland Strait.

Our remediation will ensure we deal with all the contaminated sludge in Boat Harbour before we allow tidal waters to re-enter Boat Harbour.

APPENDIX H-12

Chapter 4: Environment – Environmental Assessments

Overall Conclusion:

- Poor monitoring of projects increases risks to the environment
- Monitoring of terms and conditions of project approvals is weak
- Department not evaluating whether terms and conditions are working
- Department meeting legislative requirements for issuing approvals

Why we did this audit:

- Protecting the environment is important to Nova Scotians
- Environmental impacts should be known before a project begins
- Steps should be taken to limit environmental impacts of projects
- Projects should be monitored to ensure the environment is protected

What we found in our audit:

Monitoring of approved projects

- Monitoring not done for almost half the project terms and conditions we tested
- Department not appropriately recording all approved projects in tracking system
- Department hasn't assessed if terms and conditions have decreased environmental risks
- Department taking steps to improve monitoring

Setting terms and conditions for project approval

- Some terms and conditions lack details such as deadlines and reporting requirements
- Approvals issued without consulting inspectors who know risks
- Lack of discussion with project owners on understanding terms and conditions
- Department doesn't follow up with project reviewers to ensure responses provided

Approving project applications

- Applications for approvals contained information required by legislation
- Minister given all required information to make project approval decision
- Terms and conditions of approvals can reduce risks, but are less useful because of poor monitoring

Recommendations at a Glance

Recommendation 4.1
Environment should develop and implement a process for entering approved projects and the associated terms and conditions into the Department's tracking system to help ensure regular monitoring is completed.

Recommendation 4.2
Environment should regularly review whether standard terms and conditions of approved projects are effective at addressing identified risks.

Recommendation 4.3
Environment should develop terms and conditions for approved projects in consultation with the inspectors responsible for ensuring they are met. Terms and conditions should include clear timeframes for completion and requirements to provide documentation to confirm terms and conditions have been satisfied.

Recommendation 4.4
Environment should provide relevant draft terms and conditions of approved projects that involve other departments to the respective departments for review and confirmation of their responsibility under the terms and conditions.

Recommendation 4.5
Environment should meet with project owners to discuss the terms and conditions once projects are approved. There should also be regular meetings between Nova Scotia Environment and project owners to discuss the status of terms and conditions of approved projects.

Recommendation 4.6
Environment should document and implement a process for using government reviewers on environmental assessment applications. The process should include how reviewers are selected, the Department's expectations of reviewers and a follow-up process if responses are not provided by the deadline.

Recommendation 4.7
Environment should complete and document a review of information sent to the Minister of Environment for deciding on whether to approve or reject a project.

4 Environment: Environmental Assessments

Background

4.1 Nova Scotia Environment uses environmental assessments to identify potential negative environmental impacts of proposed projects before they begin. The goal is to approve sustainable projects while also promoting the protection and appropriate use of the environment. The Minister of Environment decides whether a project can proceed if the results of the assessment determine the potential impacts can be properly managed.

4.2 An environmental assessment is not required for every proposed project. In Nova Scotia, the Environmental Assessment Regulations list the types of projects that require an environmental assessment. Projects requiring an environmental assessment can include wind farms, quarries, pipelines and energy facilities. From 2013 to 2016, 53 of 54 environmental assessments conducted were approved.

4.3 Terms and conditions that project owners must meet are attached to project approvals. The purpose of these is to address environmental risks identified through the assessment process and ensure steps are taken to reduce the impact of the project on the environment. A typical approval includes approximately 30 terms and conditions such as monitoring programs for wildlife, habitat and groundwater well surveys. Nova Scotia Environment is responsible for ensuring project owners are complying with the terms and conditions of the approval and determining whether risks to the environment are being reduced.

4.4 An environmental assessment approval allows a project owner to proceed with the proposed project. However, projects may not immediately start when the approval is received and for some types of projects, additional approvals, such as industrial approval or wetland approval issued by Nova Scotia Environment, are needed before the project can begin.

Significant Audit Observations

Monitoring of Environmental Assessment Approvals

Terms and conditions are not monitored

4.5 Nova Scotia Environment is not monitoring terms and conditions attached to approved projects. Terms and conditions are future actions added to

approved projects to address risks projects pose to the environment. These can include various wildlife and habit monitoring programs, or restricting construction during animal breeding seasons. Failure to properly monitor compliance with these requirements increases the risk that project owners are not protecting the environment. Without monitoring, Nova Scotia Environment does not know if the terms and conditions of approved projects are effective in reducing impacts on the environment.

4.6 We reviewed a sample of 22 approved projects which contained 672 terms and conditions. We then selected two to three terms and conditions from each approved project to determine if Nova Scotia Environment had evidence to confirm the term and condition had been satisfied. In total, we examined 53 of the 672 terms and conditions identified. For 23, the Department did not confirm the term and condition had been satisfied. For example, Nova Scotia Environment did not have evidence to confirm requirements such as groundwater well and wildlife surveys were completed by the project owner or work was completed outside of animal breeding seasons.

List of approved projects and terms and conditions not complete

4.7 Nova Scotia Environment did not record approved projects and their associated terms and conditions in its electronic tracking system. This meant inspectors were not assigned responsibility for monitoring terms and conditions of approved projects and were unable to take advantage of system features that help in monitoring. Features include reminders of when inspections and audits are due, and allows managers to monitor the work of inspectors.

4.8 Management uses the information included in the tracking system to ensure the required monitoring is completed. If approved projects are not in the tracking system, managers do not have access to complete information and may not know if terms and conditions are properly monitored.

4.9 Other approvals, such as industrial and wetland approvals issued by the Department, are automatically loaded into the Department's tracking system and assigned to an inspector. One division within Nova Scotia Environment is responsible for approving projects while another division oversees whether project owners are satisfying the terms and conditions of approved projects. Once the approval is issued, it must be manually entered by the division responsible for monitoring project owners for compliance with the terms and conditions. However, this did not happen.

4.10 Nova Scotia Environment conducted an internal review in 2015, finding that only 75 of the 276 environmental assessment approvals issued between 1989 and 2015 had been recorded in the tracking system. They also concluded for almost all the approved projects entered in the tracking system, the terms and conditions of the approval were not included. Until the Department identified

this issue there was nearly no monitoring of the terms and conditions attached to approved projects.

4.11 Nova Scotia Environment has worked to address these issues, but our work found that there are still problems. Four of the 22 approved projects we examined were not recorded in the tracking system. For 15 of the remaining 18 approvals, the terms and conditions were not added to the tracking system in a way which allowed the inspectors to use the system features for monitoring.

4.12 Information on terms and conditions was not stored in a single file for each approved project, making it difficult to confirm if terms and conditions had been met. When completing our work, we often had to look in several different locations to determine if terms and conditions had been satisfied.

4.13 For example, we identified several cases in which the project owner provided requirements such as wildlife surveys and confirmation of site restoration insurance to staff at Nova Scotia Environment or other government departments, but it was not passed on to the inspectors responsible for monitoring the approval. Inspectors did not know the information was provided nor did they follow up with the project owners to request the information.

4.14 All information related to the terms and conditions of approved projects should be kept in a central location so it can be quickly determined which ones have been satisfied and those that still require monitoring.

Recommendation 4.1
Environment should develop and implement a process for entering approved projects and the associated terms and conditions into the Department's tracking system to help ensure regular monitoring is completed.

Environment Response: *Agree. In February 2017, NSE implemented a System of Notification and Approval Processing (SNAP). Going forward, approvals will be captured in the system to enable terms and conditions to be tracked. Timing: Currently underway*

Regular assessments of terms and conditions not completed

4.15 Neither the Department nor project owners completed the required assessments for any of the projects we examined. Without this reporting, Nova Scotia Environment does not have the necessary information to ensure terms and conditions of approved projects were satisfied and environmental risks were properly managed.

4.16 Nova Scotia Environment's documented process for monitoring the terms and conditions of approved projects includes:

- a Department assessment of whether terms and conditions are met and effective at reducing risk; and
- a review of the project owner's assessment of whether terms and conditions are met along with comments or suggestions for future environmental assessments.

4.17 Nova Scotia Environment's evaluation of the effectiveness of the terms and conditions of approved projects for reducing risks to the environment is especially important. This evaluation considers things such as the results of water or wildlife monitoring completed by the project owner, results of Departmental inspections, and complaints received against the project. Without this information, the Department may not be aware of the need for additional monitoring or changes to the terms and conditions. This process also provides information that can be used by the Department when approving future projects.

4.18 Nova Scotia Environment staff acknowledged this process is not followed and noted it needs to be updated since it was developed and implemented in 2002. While we recognize the process is old and there have been changes within the Department since 2002, regularly assessing the status and effectiveness of the terms and conditions of approved projects is an important practice that should be completed.

Recommendation 4.2
Environment should regularly review whether standard terms and conditions of approved projects are effective at addressing identified risks.

Environment Response: *Agree. We have committed to reviewing and updating the Internal Guide to EA Follow-up Procedures. This guide will establish procedures for reviewing Environmental Assessment terms and conditions to ensure enforceability leading to better compliance. Timing: 2017-18*

Approval of Projects

Wording of terms and conditions attached to approvals not clear

4.19 Applications are reviewed by Nova Scotia Environment to identify risks to the environment posed by the project. Approvals are subject to owners satisfying the terms and conditions included with it. If terms and conditions are not properly developed it is possible risks to the environment may go unaddressed.

4.20 The terms and conditions attached to approved projects were not always clear and well defined. We found problems with 11 of the 53 terms and conditions we examined. Specific issues included no established timelines for when the project owner must comply, no requirement to submit documentation to confirm a term and condition has been satisfied, and no indication of who the supporting documentation must be submitted to.

4.21 For example, one term and condition stated *"The Approval Holder must develop a turbine lighting plan in consultation with CWS [Canadian Wildlife Services] and Transport Canada"*. There was no deadline for the development of the plan and no requirement for the plan to be submitted to Nova Scotia Environment. The Department did not know this was completed until we asked about it as part of the audit.

4.22 Nova Scotia Environment's ability to hold project owners accountable is limited when terms and conditions of approved projects are not clear. This increases risk to the environment. For example, if a project owner is not required to provide documentation to confirm a term and condition has been satisfied, it is difficult for the Department to act against the project owner if nothing is submitted. Inspectors told us they had concerns related to the enforceability of some terms and conditions. Inspectors believed their lack of involvement in the development of the terms and conditions contributed to this issue.

4.23 The Department's process states draft terms and conditions are to be given to inspectors for review and feedback before final approval. However, based on our work, this did not always happen. For 11 of the 22 approved projects we examined, the terms and conditions were not given to inspectors for review before the approval was issued. Inspector input on terms and conditions is an important step in setting clear and enforceable expectations for project owners and limiting impacts on the environment.

Recommendation 4.3
Environment should develop terms and conditions for approved projects in consultation with the inspectors responsible for ensuring they are met. Terms and conditions should include clear timeframes for completion and requirements to provide documentation to confirm terms and conditions have been satisfied.

Environment Response: *Agree. NSE is undertaking a larger project to review terms and conditions of its approvals including EA authorizations. The project is intended to update/review existing terms and conditions to ensure requirements are relevant, clear, consistent and enforceable. Timing: 2018-19*

Responsibility for some terms and conditions not established

4.24 Nova Scotia Environment did not review terms and conditions of approved projects with other departments before approval. Departments other than Nova Scotia Environment are often responsible for receiving information from project owners and confirming if terms and conditions have been satisfied. However, Nova Scotia Environment did not consult with other departments on the terms and conditions before issuing the approval. For example, the project owner might have to develop a moose monitoring program that is to the satisfaction of the Department of Natural Resources. If Nova Scotia Environment does not discuss terms and conditions assigned to other departments with those departments, those other departments may not be aware of their responsibility or understand of what is expected of them. This could result in terms and conditions not being properly monitored or information not passed on to Nova Scotia Environment.

Recommendation 4.4
Environment should provide relevant draft terms and conditions of approved projects that involve other departments to the respective departments for review and confirmation of their responsibility under the terms and conditions.

Environment Response: *Agree. The review and update of the EA Checklists will ensure a record of communication with other departments is clear and a record is maintained. Timing: June 2017*

Review of terms and conditions with project owners not done

4.25 The Department did not review terms and conditions of approved projects with the project owners as required. These meetings help ensure project owners fully understand what is required of them and the expectations of Nova Scotia Environment. For example, meetings can provide an opportunity to discuss periods during the year when project owners are not allowed to clear land or steps that must be taken to monitor wildlife within the project area.

4.26 The Department's process is to meet with the project owner within four weeks of the project being approved to review the terms and conditions. This meeting did not occur for three of the 22 approved projects we examined. For another seven projects, the meeting was not held within four weeks of the approval being granted. In two of these, approximately a year had passed before Nova Scotia Environment met with the project owner.

Recommendation 4.5
Environment should meet with project owners to discuss the terms and conditions once projects are approved. There should also be regular meetings between Nova Scotia Environment and project owners to discuss the status of terms and conditions of approved projects.

Environment Response: *Agree. The Internal Guide to EA Follow-up Procedures will be updated to ensure the initial meeting with the approval holder is completed. Subsequent meetings with the approval holder to review terms and conditions will be captured during an audit or inspection process. Timing: 2017-18*

Better processes needed for reviewers of environmental assessment applications

4.27 Government reviewers were used to assess each of the 22 approved projects we examined. As an example, applications were sent to divisions within Nova Scotia Environment to assess a proposed project's impact on groundwater, while the Department of Natural Resources was used to identify risks to wildlife. Comments from reviewers were considered in developing terms and conditions for approved projects.

4.28 While the use of reviewers provides valuable feedback on proposed projects, improvements are needed. Currently, applications are sent to a broad list of reviewers. For some of the environmental assessment applications we examined, the documentation was provided to over 40 individuals. The Department does not have a process to identify the specific reviewers that should be used. Furthermore, no guidance is provided to reviewers on what Nova Scotia Environment's expectations are for the review. For example, it isn't clear whether the individuals are to review the entire application or just sections.

4.29 Also, Nova Scotia Environment does not have a process to follow up with reviewers if a response is not received. While comments from some reviewers were provided for each of the applications we examined, not all reviewers responded. The Department did not know if a reviewer did not respond because the request was not received, not enough time was given to review the application, or if the reviewer had no comments to provide.

Recommendation 4.6
Environment should document and implement a process for using government reviewers on environmental assessment applications. The process should include how reviewers are selected, the Department's expectations of reviewers and a follow-up process if responses are not provided by the deadline.

Environment Response: *Agree. The review and update of the EA Checklists will ensure interactions with reviewers are identified, carried out and documented. Timing: June 2017*

Project owners are submitting required information

4.30 The Environmental Assessment Regulations outline the minimum information project owners are to include with their application for an approval and what

the Minister of Environment must consider in making the decision to approve or reject the project. For example, project owners are to provide information on the purpose and need for the project, along with details on the potential effects on groundwater, vegetation, and wildlife in the area. Factors the Minister is to consider in making a decision include the nature and sensitivity of the project area, along with any potential or known environmental impacts on species at risk and their habitats.

4.31 For each of the 22 approved projects selected we examined whether project owners provided the required information and whether the summary of the project included all factors to be considered by the Minister in making a decision. We did not find any significant instances in which the legislative requirements were not met for the applications we examined. In all cases, the information provided by the project owners met the legislative requirements and the Minister was provided with all required information to be used in deciding to approve the project.

4.32 Terms and conditions to manage the risks of the project identified during the application process were attached to each of the approved projects we examined. However, the lack of monitoring of the terms and conditions of approved projects previously discussed weakens the work done by Nova Scotia Environment in deciding to approve a project. The value of the terms and conditions can only be achieved if there is regular monitoring to ensure project owners comply.

4.33 One area in which Nova Scotia Environment can improve its process is the review of an application before it goes to the Minister for a decision. When staff within the Department complete their review, a summary of the project and associated risks is compiled and forwarded to the Minister. However, there is no review of the summary to ensure the details and risks of the project are accurately and completely captured.

Recommendation 4.7
Environment should complete and document a review of information sent to the Minister of Environment for deciding on whether to approve or reject a project.

Environment Response: *Agree. A routing sheet will be instituted to ensure a review of information is in place before it is sent to the Minister. Timing: June 2017*

Appendix I

Audit Objectives and Scope

In winter 2017, we completed a performance audit at Nova Scotia Environment on the Environmental Assessment program. The audit was conducted in accordance with sections 18 and 21 of the Auditor General Act, and auditing standards of the Chartered Professional Accountants of Canada.

The purpose of the audit was to determine whether Nova Scotia Environment is appropriately reviewing environmental assessments to ensure potential impacts to the environment are avoided or reduced, monitoring the risks identified, and taking action when necessary.

The objectives of the audit were to determine whether Nova Scotia Environment:

- conducted environmental assessments that are consistent with relevant legislation, policies and procedures;
- has processes to monitor compliance with terms and conditions of environmental assessment approvals;
- has a process to evaluate the effectiveness of the environmental assessment program in reducing the impact of adverse effects or significant environmental effects.

Generally accepted criteria consistent with the objectives of the audit did not exist. Audit criteria were developed specifically for this engagement. Criteria were accepted as appropriate by senior management of Nova Scotia Environment.

Our audit approach included an examination of documentation of systems and processes; examination of legislation, policies, guidelines, standards, and other documentation; and testing compliance with legislation, policies, guidelines, and standards. We interviewed management and staff at Nova Scotia Environment. Our main audit period included activities between January 2013 to August 2016. However, we examined activities outside of this period when necessary.

We did not comment on the accuracy of the information provided by project owners included in environmental assessment applications, nor did we comment on the technical feedback provided on applications by government reviewers. Our work focused on whether the required steps were followed in issuing environmental assessment approvals and whether the Department ensured project owners met the terms and conditions of the approvals.

APPENDIX H-13

PRINCE EDWARD ISLAND LEGISLATIVE ASSEMBLY

Speaker: Hon. Francis (Buck) Watts

Published by Order of the Legislature

Standing Committee on Agriculture and Fisheries

DATE OF HEARING: 1 FEBRUARY 2019

MEETING STATUS: PUBLIC

LOCATION: LEGISLATIVE CHAMBER, HON. GEORGE COLES BUILDING, CHARLOTTETOWN

SUBJECT: BRIEFING FROM REPRESENTATIVES OF ENVIRONMENT AND CLIMATE CHANGE CANADA RE: PROPOSED WASTE WATER TREATMENT PROJECT AT NORTHERN PULP

COMMITTEE:

Allen Roach, MLA Montague-Kilmuir [Chair] (replaces Hal Perry, MLA Tignish-Palmer Road)
Dr. Peter Bevan-Baker, Leader of the Third Party
Hon. Richard Brown, Minister of Communities Land and Environment (replaces Hon. Paula Biggar, Minister of Transportation, Infrastructure and Energy)
Jamie Fox, MLA Borden-Kinkora
Hon. Sonny Gallant, Minister of Workforce and Advanced Learning
Colin LaVie, MLA Souris-Elmira
Hon. Chris Palmer, Minister of Economic Development and Tourism

COMMITTEE MEMBERS ABSENT:

Alan McIsaac, MLA Vernon River-Stratford

MEMBERS IN ATTENDANCE:

Darlene Compton, MLA Belfast-Murray River

GUESTS:

Environment and Climate Change Canada (Caroline Blais; Geoff Mercer)

STAFF:

Ryan Reddin, Clerk Assistant (Research and Committees)

Edited by Hansard

The Committee met at 10:00 a.m.

Clerk Assistant (Reddin): Good morning everyone.

The Chair of the committee, Mr. Perry, is absent today, so, I'll open the meeting to ask for a nomination for a temporary Chair.

Mr. R. Brown: Allen Roach.

Clerk Assistant: Hearing Mr. Roach; any other nominations?

An Hon. Member: I'll second that motion.

Clerk Assistant: All in favour please signify by saying "aye".

Some Hon. Members: Aye!

Clerk Assistant: Any opposed say "nay".

Motion is carried.

Mr. Roach, please take the chair.

Chair (Roach): Good morning everyone.

First of all I'd like to welcome our guests that are here today; welcome to Prince Edward Island. Welcome to the cold weather, but I guess no matter where you go in Canada today you're going to get the same.

We're here today to talk about the Northern Pulp effluent treatment plan and what's taking place in Nova Scotia. Just before we get started I'd like to – anyone who's going to speak – the first time you speak I would ask you to identify yourself and that way our good folks over in the corner will be able to know who's speaking when they're doing their transcribing over there.

The other thing I've been doing at committee meetings is, when it comes time for questions later on, I'd like to give each minister and MLA the opportunity to ask three questions and then that way everybody in the room gets an equal opportunity to ask questions and at the end of that, if there's more time then we'll just start that process again. That seems to work quite well, so unless there're any objections, I'd like to continue it that way.

Having heard none; thank you.

I would invite whoever would like to speak to go ahead.

Could I also get an adoption of the agenda for today?

An Hon. Member: So moved.

Chair: Thank you.

Okay, Geoff and Caroline, whoever would like to go first?

Geoff Mercer: Thank you Mr. Chair, it's a pleasure to begin.

Good morning everyone, it's a pleasure to be here today in front of the Standing Committee on Agriculture and Fisheries. It's an absolute pleasure to be here in Charlottetown; of course on lovely Prince Edward Island despite the frigid Canadian winter.

Mr. Chair, my name is Geoff Mercer; I have the pleasure to be the Regional Director General for Atlantic and Quebec Regions of Environment and Climate Change Canada. I'm headquartered in Halifax, Nova Scotia and I'd like to introduce my colleague Ms. Caroline Blais. Ms. Blais is the director responsible for forest products and the policy administration of the pollution prevention provisions. You'll hear that term today, the pollution prevention provisions we refer to as subsection 36(3) to 36(6) of the *Fisheries Act*, with, and under, the administration of Environment and Climate Change Canada. I sincerely appreciate Caroline's travel last night from Ottawa. Thanks for being here, Caroline.

In follow up to my department's letter to this committee dated on the 19th of December, we are here today to provide more details on the Environment and Climate Change Canada's responsibilities under section 36(3) of the *Fisheries Act*, as they relate to the regulations for the release of effluents into waterways as was requested in the letter we received.

It's important to understand that Environment and Climate Change Canada is a science-based department. We develop policies, we deliver programs, we make

decisions based on scientific evidence. The departments programming focus is on minimizing threats to Canadians and their environment, including climate change, conserving and restoring Canada's natural environment and providing data and information to Canadians to make informed decisions on weather and weather conditions.

Secondly, please note that we are a regulator. As a regulator, the department is committed to maintaining a regulatory system in Canada that is evidence-based, effective, efficient, transparent and adaptable. We enforce Canada's environmental laws. Those laws include – just to name a few – the *Canadian Environmental Protection Act* which was recent – amended in 1999 last. We refer to it as CEPA (CEPA 99). The *Species at Risk Act*, you'll hear the acronym SARA used for that; the *Migratory Bird Convention Act, 1994*, just to name a few pieces of the legislation that we enforce. But we are here today to talk to you about the pollution prevention provisions, remember section 36(3) to 36(6) of the *Fisheries Act*, which is why we're here.

Ms. Blais will provide the committee with information on the department's rules and responsibilities for the pollution prevention provisions of the *Fisheries Act*, and specifically, the Pulp and Paper Effluent Regulations.

Following our presentation, we look forward to receiving any questions you may have on these regulations.

So, with further ado, Mr. Chair, I'd like to ask my colleague, Ms. Blais to begin with her presentation.

Caroline Blais: Thank you, Geoff.

Good morning everyone; Chair, members of the committee, thank you for the opportunity to appear here today. My name is Caroline Blais; I'm the Director of the Forest Product and *Fisheries Act* Division at Environment and Climate Change Canada. My team and I are responsible for implementing the Pulp and Paper Effluent Regulations.

So, I'd like to start with some information regarding the *Fisheries Act*. The *Fisheries Act* is the oldest piece of environmental legislation in Canada. It was first enacted in 1868. It applies to all Canadian fishing zones, territorial seas and inland waters, so that's our basis.

The Minister of Fisheries and Oceans is responsible for most of the act. The minister of environment is responsible for the pollution prevention provision for all activities, except aquaculture, the control and eradication of aquatic invasive species and aquatic pests. That stays with the Minister of Fisheries and Oceans.

The sharing of the responsibility was confirmed in 2014 through a designation Order in Council that gave the minister of the environment primary responsibilities for the pollution prevention provision and those are the section 36(3) to 36(6).

There are two of these subsections that are key: first the subsection 3, which really prohibits the deposit of any deleterious substance pollution in waters frequented by fish, or in any place under any circumstances where it may enter such water unless authorized by regulations. That's our basis.

Second, the Governor in Council may make a regulation under subsection 36(5) authorizing certain deposit under conditions. In 1992, the Government of Canada promulgated the Pulp and Paper Effluent Regulations also known by its acronym PPER; we love acronyms. The regulations apply to all pulp and paper mills in Canada by setting national effluent quality standards.

The purpose of the regulations is to manage threats to fish, fish habitat and the use of fish by humans, i.e. when we eat the fish, by limiting the deposit of deleterious substances into fish bearing waters. All 90 pulp and paper mills in Canada, across Canada, must comply with these regulations.

The regulations set maximum quantities of amount of biochemical oxygen demand matter and suspended solid that may be deposited by mills and prohibit the deposit of effluent that are acutely lethal to fish. I will explain some of these terms.

Biochemical oxygen demand matter; it represents the amount of matter that consumes oxygen dissolved in water. It is determined using biochemical oxygen demand test, which is the measure of dissolved oxygen in the water consumed by the organisms to break down the organic material present in the effluent. It is used as one of the measures of the degree of pollution in that effluent. The higher the value, the more pollution in the effluent. So it is important to limit BOD matter so that an effluent does not reduce the available oxygen in the receiving environment. If oxygen levels in water are too low, aquatic organism will be negatively impacted.

Total suspended solids are essentially solids that are not dissolved in the water and are also a measure of pollution. This is an important perimeter to control because high levels of suspended solid can harm fish gills, so those are two substances that are regulated under PPER.

Finally, acute lethality to fish is determined when more than 50% of rainbow trout die when they are placed in the effluent at 100% concentration level over a 96-hour period. This test is used to make sure that the effluent as a whole does not kill fish.

The maximum quantity set in the PPER are national standards based on what was achievable by pulp and paper mills using secondary wastewater treatment system at the time the regulations were developed. The regulations sets a number of conditions that pulp mills must meet in order to be authorized to deposit biochemical oxygen demand matter and suspended solids.

The regulatees must; install, maintain, calibrate monitoring equipment, monitor effluent and submit monthly report of the monitoring results and production information, notify an inspector without delay of any results of a test conducted that indicates failure or non-compliance, submit identifying information, prepare and update annually a remedial plan, prepare an emergency response plan, submit information on all outfall structures and deposit effluent only through these outfall structures, comply with the requirements of environmental effects monitoring studies and keep records on sites for inspections.

Environmental effects monitoring done by the mills is the ultimate performance measurement tool for the Pulp and Paper Effluent Regulations. It measures, directly in the receiving environment, the effects of effluent on fish, fish habitat and human use of fisheries resources to assess how well the regulations protect fish, fish habitat and the use of fish by human.

This is achieved by comparing a number of measures taken in the area exposed to the effluent and a similar area not exposed to the effluent. These measures include data on water, sediment, fish, fish tissue, fish habitat. Environmental effects monitoring also includes sub-lethal toxicity of final effluent. So, this test is performed in a laboratory to provide information on the potential effect of effluent on biological component in the receiving environment and it's a longer test.

Under the regulations, mills are required to identify and investigate potential effects on their effluent on fish, fish habitat and the use of fisheries resources. When environmental effect studies show effects as a result of the discharge of the pulp and paper effluent, mills are then required to conduct studies to understand the causes and identify potential solutions to mitigate these effects.

Now I'd like to explain the results that have been achieved by the Pulp and Paper Effluent Regulations since it came into force in 1992. Since then, mills across the country have made significant improvement in their effluent quality; in most cases by implementing secondary biological treatment system. Between 1987 and 1996 – so total discharge of suspended solids and biochemical oxygen demand matter decreased approximately by 60 and 90% – so that's across the country. Compliance rates with the regulations is high and based on the self-reported data, over 97% attest that mills across the country conduct are compliant with the regulations.

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.

So on the national basis, like the most prevalent impact of mill effluents are eutrophication, which is when there is too much nutrient in the body of water and reduced size of reproductive organs in fish. Eutrophication can lead to oxygen depletion and decrease in species diversity. Reduce reproductive organs in fish can lead to reproduction in species, diversities and imbalance in the fish communities.

In taking this information – in September 2017, the department launched a process to modernize the Pulp and Paper Effluent Regulations.

The modernization is focused on four key areas: 1) winding the scope of the regulations to capture new and innovative products being produced by the sector; 2) reviewing the regulatory limits for existing and new deleterious substances 3) improving the administration of the regulations and; 4) streamlining the reporting and administrative requirements.

A key area of the modernization is increasing environmental protection. The department has publicly proposed to reduce maximal allowable biochemical oxygen demand matter and suspended solids and to add new substances to regulate, such as nutrients.

A first round of engagement with interested parties occurred between fall of 2017 and spring 2018. A second round of engagement is planned for winter/spring 2019.

In closing, I would to emphasis that notwithstanding the evolution of the regulations effluent from pulp and paper mills across Canada are regulated and the effluent of any new treatment facility at a mill in Canada would be subject to these regulations.

That concludes my remarks and happy to answer any questions.

Chair: Thank you both very much.

First question is from MLA Compton.

Ms. Compton: Thank you, Chair. I want to thank both of you for coming in today.

I'm not actually on this committee but it's of great impact to my district and had a lot of constituents reach out. We've had a number of people come in, including Northern Pulp and it seems every question we asked it was someone else's department or no, we can't answer that, or no, that's environment, or no, that's fisheries.

I guess my first question would be – looking down the road, I mean 50 years ago they thought Boat Harbour was going to be the answer and we now know that it wasn't. Fifty years down the road what are we going to be saying about the Northumberland Strait and the impact that this is going to have on fishery? I don't know if you can comment.

Thank you for all that information; it was a lot to process and maybe we could have that given to us in written form or online, that would be great. But if you can just answer how we can be assured that 50 years down the road this process – which the only answer we've gotten is it's going to better than it was – is not going to be better than it was – or is going to impact the environment and the fisheries.

Geoff Mercer: I think I'll start general. Thanks very much MLA Compton for the question, first and foremost.

Environment and Climate Change Canada like I said in the beginning is a science-based department and we're a regulator. The development and implementation of the Pulp and Paper Effluent Regulations in 1992, as my colleague Ms. Blais indicated, markedly improved the performance of mills in protecting the environment, particularly, the decrease in toxins or furans in the environment. Toxins and furans are cancer-causing agents that were released up until then.

In today's world in environmental standards, we use the absolute best practices that are available and the leading class science that we can in this country to make sure that the regulations are written in a way and are applied consistently across this county, as Caroline has indicated, from coast to coast to coast in all 90 mills. When those regulations are applied consistently and with world class enforcement officers that we have through our environmental

enforcement officers program, then we have absolute certainty that those regulations are being applied as the laws are written in Canada.

Ms. Compton: I wondered if either of you could answer the question as to, if there was a new mill being built that was proposing the same effluent treatment, would that be approved through the regulations that both your departments have?

Caroline Blais: Thank you for the question.

As a regulator, a regulation does not approve single projects. So if there is a new mill, from the beginning, they will be subject to the effluent standard. We don't prescribe the technology; they just have to meet the effluent standard for biochemical oxygen demands, suspended solid acute lethality of the existing reg. When the new regs are coming into force they will be subject to those levels.

Ms. Compton: You did state that of the mills that are existing, 70% of those mills are having an impact on the fish that the effluent is going into the water. I'm not even sure I'd ask the question, but the concern that we have here is it's going to ruin the fishery in the Northumberland Strait. If right now 70% of the mills which supposedly meet the criteria by both the regulators that are represented here, there's still a problem and how do we move forward from that? If we take 70% of the fishing in our Strait or 70% of the mills, this is included I'm sure – and maybe can you answer: Is Northern Pulp one of the mills and the effluent coming out of there now is affecting the fisheries?

Caroline Blais: So effluent standards – okay, so I'll answer the question (Indistinct) two steps. The first part of the question is that we are aware that despite the high level of compliance with the effluent standard, effluents across Canada are having an impact on fish, which is why in September 2017, the department launched a process to modernize these regulations, so to update the effluents limit. The second part is, if Northern Pulp has an impact and our information looking at their (Indistinct) studies, we found that yes, they have an impact on fish habitat and the company is at the stage where they are looking for the cause of the impact and their last study is posted on their website. That's where they are in the process.

Ms. Compton: Do you want me to keep going?

Chair: No.

MLA Fox.

Mr. Fox: Thanks, Chair. Thank you very much for attending today.

In your submission there, you talk about installing and maintaining and calibrating and monitor equipment. I'm interested in: How often do the inspectors actually check and verify that the equipment they're using to test the water is actually calibrated?

Caroline Blais: Our colleagues in the enforcement branch have a prioritization exercise and on a yearly basis they kind of look at all of their regulations across the department and the regulatee and to set up a schedule to go and inspect a number of facilities, including pulp and paper mills. In terms of the exact frequency, I don't have that information here, but they do have a process to go and inspect and verify those documents.

Mr. Fox: Chair, a follow up to that, is it possible to get a list of how many times inspectors attended Northern Pulp and found them to be non-compliance to the act or the regulations, or the standards that are required?

Caroline Blais: We'll take that information back (Indistinct)

Mr. Fox: Thank you.

If we recognize that the effluent going into the Strait or into a waterway is above the temperature of the water and we know that that rise in temperature affects the oxygen levels in the water which has a negative effect on the species, any species of fish in the water – we also know that climate change is real – why would we allow any substance to go into a waterway that is above the average temperature of the water, which is actually worsening the climate effect change?

APPENDIX H-14

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Gunn's pulp mill permit lapses so land now for sale

31 August 2017


Andrea Dawkins

One of Tasmania's most controversial issues – a proposal for a $2.5 billion pulp mill at Bell Bay – is finally over. Sources: The Mercury, The Examiner, The Bendigo Advertiser

Permits to build the pulp mill, originally proposed by collapsed timber giant Gunns Limited, lapsed.

A spokesman for Gunns Limited receivers, Korda Mentha, said it would not contest the lapsing of the permits because there was no prospect of a developer being interested.

He said Korda Mentha would instead try to sell the land at Bell Bay and that they had been obliged to wait until the permits lapsed before proceeding with an attempt to sell the land.

"Receivers will now be able to start serious discussions with parties who expressed an interest," he said.

The land, with or without the permits, has been on the market for more than two years.

Gunns Ltd spent about $230 million on research and getting the pulp mill permits in place before collapsing in 2012.

The spokesman said conditions in the international pulp market meant a sale would not occur.

The pulp mill saga ran from 2007 and involved political drama over the approval processes and huge opposition from environmentalists.

Gunns bankers ANZ bank would not finance the mill and Gunns attempted to bring in partners without success.

Daily Timber News Archives

The Greens will table a bill when Parliament resumes next month to repeal the Pulp Mill Assessment Act and bury the project for good.

The previous Labor government, with support from the Liberals, extended the permit conditions under the act to this year

Bass Greens MHA Andrea Dawkins said the party would table legislation in the spring session of Parliament to repeal the entire act.

"The expiration of the pulp mill permits means the Tamar Valley community is one step closer to ending this sorry saga that has plagued them for fifteen years," Ms Dawkins said.

"People within that community were vilified and abused for standing up for their right to live in a place with clean air and water."

A previous move to repeal the Pulp Mill Assessment Act was defeated in 2011.

The state government have said it would not be offering the project any more support.

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APPENDIX H-15

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18(1)(a)

October 5, 2017

[redacted]General Manager
Northern Pulp Nova Scotia Corporation
PO Box 549, Station Main
New Glasgow, NS
B2H 5E8

Dear [redacted]

RE: Review of Preliminary Receiving Water Study for Northern Pulp Effluent Treatment Plant Replacement, Pictou Harbour, Nova Scotia

As you are aware, the Northern Pulp Effluent Treatment Plant replacement requires a Class I Environmental Assessment (EA). This letter provides results of a preliminary review done by the department of the above mentioned receiving water study. This study is expected to form part of the registration package that you would provide when Northern Pulp officially registers the project for EA. Please note, this represents only a preliminary examination of the report and does not preclude further assessment and consideration of information that is received as part of the EA.

As this review was undertaken only by Nova Scotia Environment (NSE), you are advised to seek input from Fisheries and Oceans Canada and Environment and Climate Change Canada, as well as other federal and municipal agencies, to ensure their requirements are addressed prior to final design of the treatment facility and selection of the discharge location. The intent of this input is to assist you in finalizing the design and preparing your documents for Environmental Assessment and does not in any way encumber the Minister's decision following the EA process.

Following are key comments on the report:

1.0 Based upon the modelling data provided, the report identifies the following maximum allowable concentrations:

- Maximum daily flow - 85,000 m^3
- BOD_5 – 48 mg/L
- TSS – 48 mg/L
- Colour – 750 TCU*
- AOX – 7.8 mg/L
- Nitrogen – 3.0 mg/L
- Phosphorous – 1.5 mg/L
- pH – 7-8.5

  * Note: color must achieve background concentrations prior to the effluent surfacing at 90 meters.

.../2

[redacted] 18(1)(a)

Department reviewers are generally supportive of these numbers. They make sense from a science perspective. As mentioned however, additional information may become available during the EA that will also need to be assessed. We are however comfortable with using these numbers for the design of the project.

2.0 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 in 1.0 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 submit a plan to the Department indicating what changes are required to the Mill to achieve the maximum concentrations. Should additions/modifications be required to the Mill itself, an application for amendment to the current Industrial Approval will be required prior to implementation of any modification.

3.0 With respect to the current Industrial Approval the maximum Chemical Oxygen Demand (COD) concentration discharged from the Kraft Pulp Mill into the effluent treatment facility is:

- COD – 950 mg/L (570 mg/L at point of discharge, assuming a 40% reduction in the SBR)

4.0 NSE Accepts that a flow of 85,000 m^3/day is appropriate for design of the new system. However, Northern Pulp must continue to meet the current maximum daily water intake requirement of 92,310 m^3 outlined within the existing approval that expires January 30, 2020.

Should you have any questions or concerns regarding this information, please do not hesitate to contact me at 902-483-2696 or via email at helen.macphail@novascotia.ca.

Regards,

Helen MacPhail

Helen MacPhail
Supervisor of Environmental Assessment

c. Paul Keats, Regional Director
Kathleen Johnson, P.Eng., Engineering Specialist
Stefan Furey, P.Eng., Engineering Specialist
Adrian Fuller, Executive Director, ICE
Frances Martin, Deputy Minister

APPENDIX H-16

Lack of public consultation ahead of Northern Pulp's submission of Environmental assessment sparks backlash

Brendan Ahern (brendan.ahern@ngnews.ca)
Published: Jan 16 at 2:13 p.m.

An aerial view of the Town of Pictou, with the Northern Pulp mill across the harbour. August 21, 2014. - Christian Laforce

Pictou

Northern Pulp's decision not to hold public consultations before filing its environmental assessment has received blowback from groups representing Northumberland fishermen.

In a statement issued from Friends of the Northumberland Strait, the group decries the company's decision, stating that, "Concerned citizens and fishermen say they are appalled that Northern Pulp

does not plan to hold any open houses or public consultation before filing for environmental assessment."

The statement emphasizes Northern Pulp's past promise to hold further open house events designed to keep the public up to date on the company's plan.

"We were told Northern Pulp were wanting to be transparent during this process," said Friends of the Northumberland Strait president Jill Graham-Scalan in an interview. "They had promised us during that time that as the studies they were conducting on the route and the receiving waters were completed that they would be making those studies available to the public."

An addendum to Northern Pulp's water receiving study was posted after the press release, but Graham-Scalan says that this is not the level of transparency that was promised form the company back in December 2017.

With the company's environmental assessment being submitted at the end of January, Graham-Scalan says that it could be difficult to properly analyze the information and provide feedback.

"Not only do we have to read it, we have to absorb and reply to it," she said. "That 30-day period is the only period of time that the public has to come up to speed on the proposal and respond to the province in a way that is complete."

Northern Pulp has cited 'significant' delays and a need to move forward under an impending deadline as it's reason for filing the assessment without holding the open houses.

"We've experienced a significant number of delays through the fall in attempting to get some of the information compiled," said Northern Pulp director of communications, Kathy Cloutier. "We're at a point now where we want the project to move forward."

Cloutier added that the blockades Pictou Harbour by strait fishermen of the company's survey boat was a major factor in the need to push ahead to make up for the lost time.

When asked if the incomplete survey of the sea-floor will impact the quality of environmental data submitted to the province, Cloutier said that the only gaps will have to do with the pipe's construction.

"There may be some constructability gaps, but the environmental assessment will be a complete document."

"Another major factor to delays is the number of studies and expanded studies that were done," said Cloutier. "When the process began there were seven studies that we were aware that we'd need to submit for an environmental assessment. Since then we've moved from seven to 17, to 20, we're now at 28."

The validity of that data revealed in those studies will then be determined by the provincial assessment.

After Jan 31. Cloutier says that that information will be in the hands of the public and the provincial government.

"I can't speak to a government process, but what I can say is that there will be time for people to submit comment, and that will help the minister."

The statement issued by Friends of the Northumberland Strait remains skeptical that Northern Pulp's assessment will accurately measure the outfall's effect on marine life in the strait.

"Knowing the composition of the treated effluent they plan to release is critical," wrote president of the Northumberland Fishing association Carl Allen. "We've asked for this information for almost a year, and have never received it. If the effluent is as harmless as Northern Pulp tells the public, why haven't they provided the information?"

APPENDIX H-20

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NOV 3 0 2017

Bruce Chapman, P.Eng., General Manager
Northern Pulp Nova Scotia Corporation
PO Box 594, Station Main
New Glasgow NS B2H 5E8

Dear Mr. Chapman,

Re: Industrial Approval Clarification

In response to your letter of November 3rd, 2017 and subsequent email of November 14th, 2017 to Deputy Martin and Helen MacPhail, the Inspection, Compliance and Enforcement Division would like to provide clarification on some of the issues raised in your correspondence. Final regulatory effluent discharge limits will be established once the environmental assessment process is complete.

Northern Pulp's receiving water study for flow, BOD_5, TSS, Colour, AOX, Nitrogen, Phosphorous and pH will be taken into consideration. Requirements of the current industrial approval for influent COD reductions from benchmark concentrations by 2020 as submitted by Northern Pulp on December 14, 2016 and accepted by NSE on February 27, 2017 will also be considered. The upcoming environmental assessment will also be used to establish those limits. Currently, NSE has indicated support for discharge concentrations as indicated in the October 5th, 2017 correspondence from Helen MacPhail. These effluent concentrations may be subject to change, depending on the outcome of the environmental assessment.

In general, NSE considers operational variations when determining compliance for items such as monitoring parameters through the industrial approval process. Industrial approval application submission requirements include a detailed engineering report. Northern Pulp will also need to submit plans for an effluent treatment plant design which will meet the effluent discharge requirements, taking all the factors listed above into consideration.

With respect to the regulation of COD, as you may recall, during early industrial approval discussions, Northern Pulp indicated it did not wish to have a study style approach to reduction of black liquor spills. Instead, the company requested the approval contain a compliance limit your company could work to achieve. Through discussions with third-party experts, it was recommended the best way to manage black liquor spills is through regulation of COD. NSE recognizes COD is not a widely-regulated parameter in North America for the pulp and paper industry, although there are individual facilities which do have COD limits within their individual operational permits. During discussions between Northern Pulp and NSE following Northern Pulp's appeal of the industrial approval, Northern Pulp agreed that an influent concentration of 1900 mg/L was a reasonable benchmark for COD reductions. Northern Pulp confirmed it could achieve a 50 per cent reduction from this benchmark by January 2020 would be achieved in the December 14, 2016 submission. That is why correspondence from the Environmental Assessment branch regarding effluent discharge limits references the current industrial approval requirements for COD rather than the receiving water study, as not to create a conflict between the two.

.../2

With respect to your comment from your email dated November14th on the renewal of the industrial approval and the ability to start discussions on a future IA, NSE offers the following comment. As you have stated and agree, there are many items in a future IA that will be determined once the EA process is completed. NSE agrees with this comment and recognizes that large industrial applications such as a pulp and paper approval can take several months of review and discussions between the proponent prior to any issuance of an approval. It is not uncommon for this size of an industrial file to take up to 12 to 24 months to complete a review process. Therefore, it would be prudent for both Northern Pulp and Nova Scotia Environment to begin discussions on a future industrial approval sometime soon. As stated above these discussions will be solely focused on the items in the industrial approval that are not impacted by the future environmental assessment process and those items will be dealt with after the environmental assessment. As this industrial approval process will be completed by the inspection, compliance and enforcement division of Nova Scotia Environment, you can contact me as the lead for this industrial file at your convenience.

Should you have any further questions or concerns, please do not hesitate to contact me at 902-863-7600.

Regards,

Paul Keats, B.Tech (Env), Eng.Tech
Eastern Regional Director NSE

c: Adrian Fuller, Executive Director ICE
Frances Martin, Deputy Minister
Tanya MacKenzie, District Manager ICE

APPENDIX H-21

NORTHERN PULP
NOVA SCOTIA CORPORATION
A PAPER EXCELLENCE COMPANY

April 9, 2015

The Honourable Randy Delorey
Minister of Environment
Nova Scotia Environment
PO Box 442
Halifax, NS B3J 2P8

Dear Minister Delorey:

Re: Industrial Approval No. 2011-076657-A01
Appeal pursuant to Section 137 of the *Environment Act*

Northern Pulp Nova Scotia Corporation ("Northern Pulp") appeals Industrial Approval No. 2011-076657-A01 (the "Approval") issued by Jay Brenton of the Nova Scotia Department of Environment (the "Department") on March 9, 2015 for the operation of Northern Pulp's mill in Abercrombie Point, Nova Scotia (the "Mill").

Northern Pulp makes this appeal (the "Appeal") to you, the Minister of Environment, pursuant to section 137 of the *Environment Act*, SNS 1994-1995, c 1 (the "Act"). In the sections below, Northern Pulp states its reasons for this Appeal, outlines its specific objections to various provisions in the Approval and sets out its requested relief.

While Northern Pulp considers it necessary to proceed with this Appeal to protect the long term economic viability of the Mill, Northern Pulp remains open to working with the Department and the Province to achieve a long term solution with respect to the Mill.

SUMMARY OF APPEAL

In summary, Northern Pulp requests that certain provisions (identified below) of the Approval be removed or revised on the grounds that these provisions:

(i) are in breach of the Province's contractual obligations to Northern Pulp under various agreements (which agreements the Department refused to consider in issuing the Approval) and impose conditions that in essence seek to achieve indirectly what the Province is not permitted to do directly, namely, require Northern Pulp to cease using its current effluent treatment facility ("Effluent Treatment Facility");

(ii) impose conditions that are impossible to meet and/or are prohibitory rather than regulatory in nature;

(iii) impose conditions that are too vague or uncertain;

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(iv) are unrealistic and otherwise unreasonable; and/or

(v) are inconsistent with the protection granted to Northern Pulp by the *Freedom of Information and Protection of Privacy Act* ("FOIPOP").

Northern Pulp respectfully requests that you exercise your powers under Section 137(4) of the Act to allow Northern Pulp's Appeal and remove or revise the provisions of the Approval as further outlined in this letter. Doing so would ensure that the Approval contains conditions that: (i) do not breach the Province's obligations under the various agreements between the Province and Northern Pulp and do not effectively require Northern Pulp to change the location of its current Effluent Treatment Facility; (ii) are not impossible to achieve and are regulatory rather than prohibitory in nature; (iii) are not vague or uncertain, (iv) are realistic and otherwise reasonable; and (v) are not inconsistent with the protection granted under FOIPOP.

GROUNDS OF APPEAL

I. Department's Refusal to Consider Province's Contractual Obligations - Section 8(2)(c) of the Act

As you are aware, Northern Pulp and the Province are parties to a number of agreements in respect of the operation of the Mill, including:

1) Memorandum of Understanding dated December 1, 1995 ("MOU");

2) Lease dated December 31, 1995 ("Lease");

3) License Agreement dated December 31, 1995;

4) Indemnity Agreement dated December 31, 1995 ("Indemnity Agreement");

5) Water Supply Agreement dated June 30, 1995 ("Water Supply Agreement");

6) Lease Extension Agreement dated October 22, 2002; and

7) Acknowledgement Agreement by the Province dated May 12, 2008 ("Acknowledgement Agreement").

(collectively, the "Agreements").

Northern Pulp wrote to you on December 18, 2014 in order to ensure that the Department reviewed the Approval in light of the Province's contractual obligations to Northern Pulp under the Agreements prior to issuing the final Approval. In addition, Northern Pulp requested a joint meeting with the Department of Environment and Department of Internal Services to discuss how a long term solution can be achieved in compliance with the terms of the Agreements and the Approval.

In a letter dated January 8, 2015, you responded: "While my department is aware of the existence of agreements between the Province and your company, the Province has maintained independence between the regulation of environmental issues and management of the various agreements with the mill." You encouraged Northern Pulp to meet with Internal Services to discuss the Agreements and any implications that may arise from the issuance of the Approval

by your Department, thereby refusing Northern Pulp's request to meet jointly with Northern Pulp and Internal Services.

Your express refusal to consider the Province's various contractual obligations in issuing the Approval and your refusal to meet jointly with Northern Pulp and Internal Services as requested by Northern Pulp is not in compliance with the mandatory requirement of Section 8(2)(c) of the Act which provides:

> 8 (2) The Minister, for the purposes of the administration and enforcement of this Act, and after engaging in such public review as the Minister considers appropriate, shall
>
> [...]
>
> (c) consult with and co-ordinate activities with other departments, Government agencies, municipalities, governments and other persons; [emphasis added]

Any provision in the Approval which is inconsistent with the provisions of the Agreements is necessarily the result of the Department's refusal to consider those Agreements when it issued the Approval. Northern Pulp submits that those provisions of the Approval are unreasonable due to the failure of the Department to comply with the process of consultation and co-ordination required by its own governing statute in issuing the Approval.

In the Acknowledgement Agreement, the Province acknowledged, agreed and confirmed that each of the Agreements and understandings between the Province and the Mill's prior owner Scott Maritimes Limited ("Scott") are in good standing and will continue in full force and effect for the benefit of Northern Pulp.

Northern Pulp's current owners acquired the shares of Northern Pulp and have continued to invest substantial amounts in the Mill on the understanding that the Province could and would comply with its obligations under the Agreements. It is not unreasonable to anticipate and expect that government will comply with the contracts which it has entered into. It is unreasonable for the Department to issue the Approval with a stated disregard for those contractual obligations. Northern Pulp does not waive any of its rights under any of the Agreements.

Northern Pulp submits that, taken together, the objectionable conditions included by the Department in the Approval that are in breach of the Province's obligations under the Agreements are an attempt by the Department to indirectly achieve the Province's stated goal of closing the Effluent Treatment Facility (see, e.g., Agreement in Principle between the Province and Pictou Landing First Nation dated June 16, 2014). These provisions would force Northern Pulp to apply for an approval for and build an alternative effluent treatment system that meets conditions that would be applicable to a substantially new Mill. This is a goal which cannot be achieved directly under the Agreements which the Province has entered into with the Mill's owners or under the Department's own regulatory jurisdiction. Government cannot do indirectly what it cannot do directly and it is unreasonable for the Department to seek to do so by imposing conditions as part of a regulatory approval process aimed at achieving that indirect goal.

These provisions of the Approval, in addition to being in breach of the Province's obligations under the Agreements, impose conditions which are unreasonable for a mill that was built in the 1960s to meet without requiring that a substantially new Mill be built. While Northern Pulp has

invested in upgrades at the Mill, such equipment modifications will not achieve the requirements of the Approval. The Approval therefore essentially prohibits Northern Pulp from operating the current Mill, and requires Northern Pulp to construct a substantially new Mill, in breach of the Agreements. The cumulative cost and uncertainty associated with the approval and construction of a new effluent treatment system jeopardize the long term economic viability of the Mill and is inconsistent with the Agreements.

Government cannot arbitrarily revoke Northern Pulp's contractual rights under the Agreements with the Province by way of an administrative approval process. Any re-negotiation of Northern Pulp's rights or the Province's obligations under the Agreements must necessarily be the result of written amendment to the Agreements. Northern Pulp approached the Department and the Province indicating that it was prepared to cooperatively work with the Province, as stated in Northern Pulp's December 18, 2014 letter to you:

> "We look forward to working with your department and Internal Services. We hereby request a meeting the first week of January with Internal Services and Nova Scotia Environment to discuss how a long term solution can be achieved in compliance with the terms of the above agreements and the Industrial Approval."

Absent any such cooperation between the Province and Northern Pulp, certain provisions of the Approval cannot reasonably stand given their clear breach of Northern Pulp's rights and the Province's obligations under the Agreements. Any provision in the Approval that is inconsistent with the Agreements must be removed or revised so that the Approval is consistent with the Agreements, including:

A. Definition of Effluent Treatment System – Clause 1(t)

The defined term "Effluent Treatment System" in Clause 1(t) of the Approval now includes the "former stabilization lagoon (known as Boat Harbour)". The defined term "Effluent Treatment System" is included as part of the definition of "Facility" and both terms are referred to throughout the Approval. Northern Pulp has never leased and has never been in control of Boat Harbour. The Mill discharges effluent into Boat Harbour at Point C in compliance with its obligations under applicable approvals and regulations, and has received a broad indemnification from the Province for claims and expenses related to Boat Harbour.

Under Section 4.01(g) of the MOU, the Province remained in possession of and responsible for the stabilization basin. The Lease definition of "Reconfigured Facility" or "Facility" makes it clear that the stabilization basin is not included as part of the Facility. Any terms in the Approval which impose responsibility on Northern Pulp for the stabilization basin known as Boat Harbour ("Boat Harbour") by virtue of:

- including the "former stabilization lagoon (known as Boat Harbour)" into the definition of Effluent Treatment System, including Clauses 5(h) and (i), 6 (h) and (i), 7(j) and (n), 12(au) and 16(b); or

- indirectly including the "former stabilization lagoon (known as Boat Harbour)" into the definition of the Facility, including Clauses 2(c), 3(a), 12(ac) and (am), 24(a) and 25(a),

are inconsistent with the terms of the Lease and Indemnity Agreement.

Northern Pulp therefore requests that the reference to the "former stabilization lagoon (known as Boat Harbour)" be removed from Clause 1(t) of the Approval.

B. Responsibility for Restoration of Stabilization Basin - Clause 7(n)

Clause 7(n) of the Approval requiring Northern Pulp to develop a plan for the long term environmental management and/or rehabilitation of Boat Harbour is expressly contrary to the Agreements. Under Sections 4.01(g) and 4.01(c)(ii) of the MOU, that responsibility falls upon the Province. Further, Section 4.01(f) of the MOU provides that Northern Pulp shall have no obligation or responsibility to restore the stabilization basin, which restoration will be undertaken by the Province. Northern Pulp is entitled to indemnification for any such costs under the broad provisions of the Indemnity Agreement.

Northern Pulp therefore requests that Clause 7(n) be removed from the Approval.

C. Spill Containment Plans – Clauses 14(m) and 14(n)

Clauses 14(m) and 14(n) require Northern Pulp to submit plans to the Department with respect to spill containment. However, Northern Pulp under the Lease is entitled to the use of the Effluent Treatment Facility until 2030 and presently uses that facility for spill containment, which use has been wholly adequate to date.

Requiring Northern Pulp to undertake a new spill containment system when Northern Pulp already has an adequate containment system available to it, which it is contractually entitled to use, provides no additional benefit to the environment and is in breach of the Province's obligations under the Agreements. As Northern Pulp's current system is adequate to protect the public and environment, Clauses 14(m) and 14(n) are inappropriate. Requiring Northern Pulp to undertake a new spill containment system would also be in breach of Northern Pulp's contractual rights under the Agreements. Northern Pulp requests that these Clauses be removed entirely.

D. Water Supply Agreement – Clauses 5(d) and 5(e)

Clause 5(d) of the Approval limits daily water consumption to a rate of 63,000 cubic meters per day by January 30, 2020. For comparison purposes, this represents an intensity of 72.2 m3/Adt. Clause 5(e) of the Approval also requires Northern Pulp to achieve certain water reduction milestones in the meantime. Clause 2.01 of the Water Supply Agreement says that the Province is obliged to continue to supply up to 25 million imperial gallons (or 113,652 m3) of fresh water per day to the Mill until 2021.

The intention of the parties in agreeing to the Water Supply Agreement is clearly contravened by the reductions required by Clauses 5(d) and 5(e) of the Approval. Northern Pulp remains willing to work cooperatively with the Department, as part of an overall negotiation with the Province with respect to the Agreements, to achieve water reductions that benefit the environment but are also practical and realistic. In the absence of such discussions, however, Northern Pulp requests that Clauses 5(d) and 5(e) be removed as they are clearly in breach of the Province's obligations under the Water Supply Agreement and are therefore unreasonable.

E. Surface Run-Off Diversion - Clause 6(c)

The AMEC Report[1] at p. 26 estimates that surface run-off accounts for 2.0 m3/ADt of effluent (rain water entering process sewers). See enclosed KSH Memo[2], at p. 6-8, where KSH has provided more extensive calculations that indicate a similar volume. As a very small fraction of the total flow (less than 1.5% of the total effluent flow for the highest month of the year, April), requiring Northern Pulp to divert surface run-off water into the Pictou Harbour is unreasonable.

Northern Pulp's current practice with respect to the surface run-off stream is to treat it with the Mill effluent, which system (as discussed above in Section I.C.) Northern Pulp is entitled to use until 2030. Clause 6(c) is improper as Northern Pulp's current system is adequate in protecting the public and environment and requiring Northern Pulp to undertake a new surface run-off water diversion program would be in breach of Northern Pulp's contractual rights under the Agreements.

Therefore, Northern Pulp requests that Clause 6(c) be removed.

F. Point D Monitoring – Clause 7(o)

Clause 7(o) requires monitoring at Point D for the parameters in Table 6 of Appendix A. As Northern Pulp's obligations cease at Point C under the Agreements, Northern Pulp cannot properly be responsible for monitoring at Point D, a location in the control of the Province. Northern Pulp therefore requests that Clause 7(o) be removed entirely.

G. Limits in Excess of Pulp & Paper Effluent Regulations – Clauses 6(e), 6(f), 8(d) and 8(f)

Section 4.01(k) of the MOU provides that the Province will impose operating limits on Northern Pulp that reflect and do not exceed or apply more stringently than the standards set out in the *Pulp and Paper Effluent Regulations* (Canada) ("PPER").

As the PPER does not contain limits on COD and TRS, the Approval under Clause 6(e), with respect to COD, and Clause 8(d), with respect to TRS, imposes more stringent operating limits on the Mill than are reflected in the PPER.

Further, to the extent that limits on COD have a limiting effect on BOD, the required reductions in COD under Clause 6(e) of the Approval result in more stringent conditions being imposed on the Mill, notwithstanding that Table 6 of the Approval indicates that limits for BOD are at the rate set by the PPER.

BOD_5 (Biochemical Oxygen Demand) is the amount of oxygen required to biologically oxidize the soluble components in an effluent sample incubated for a 5-day period. It is a measure of easily biodegradable organics that can be utilized as food by naturally occurring organisms. BOD_5 is an indirect measure of how the easily biodegradable organics in the effluent will consume dissolved oxygen in the receiving waters as natural microorganisms consume these

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[1] The Department indicated to Northern Pulp that AMEC was consulted during the development of the Approval. The April, 2010 AMEC Report entitled "Boat Harbour Return to Tidal Re-Evaluation Final Report" ("AMEC Report") is the latest report that was made available to Northern Pulp.

[2] Memo from KSH Solutions Inc. ("KSH") dated March 31, 2015 entitled "Technical Assistance with Appeal of Final Industrial Approval" ("KSH Memo").

organics, thus potentially affecting flora and fauna due to reduced dissolved oxygen concentrations. COD (Chemical Oxygen Demand) is the amount of oxygen required to chemically oxidize the soluble components in an effluent sample. COD is a measure of all organics (lower molecular weight biodegradable and higher molecular weight non-biodegradable) as well as oxidizable inorganics.

COD will always be the greater of the two, as BOD_5 is a percentage of COD. It is generally accepted that COD and BOD share an empirically demonstrated relationship. Fairly regular relationships exist between COD and BOD_5 for any given industrial or municipal wastewater stream. Once the average COD:BOD_5 ratio for a particular wastewater stream is established, a relatively simple and quick 1-hour COD test can be used to predict BOD_5 with relative reliability. This is why COD tests are often done, but rarely reported. Kraft mill lignin and cellulose contain both low and high molecular weight organics. Therefore, any measures undertaken to reduce COD at the Mill will undoubtedly reduce BOD_5 as well, in breach of the MOU. See enclosed Klopping report at p. 3-4.[3]

As a result of the inconsistency of these provisions with the MOU, Northern Pulp submits that Clauses 6(e) and 8(d) are unreasonable and therefore requests that these provisions, as well as the related provisions at Clauses 6(f) and 8(f), be removed.

H. Precluding Use of Boat Harbour Effluent Treatment Facility – Clauses 5(h), 5(i) and 5(j) and Clauses 6(h), 6(i) and 6(j) and Table 6A

Under the Lease, as extended, Northern Pulp has the right to exclusive possession and occupation of the Effluent Treatment Facility until 2030. The provisions of the Approval that effectively preclude Northern Pulp from using the Effluent Treatment Facility for the Mill's operations are inconsistent with the provisions of the Lease, including Clauses 5(b) and Clause 14 of the Lease.

The effect of Clauses 5(h), 5(i) and 5(j) and Clauses 6(h), 6(i) and 6(j) of the Approval is to require Northern Pulp to apply for an amendment to the Approval if Northern Pulp's modeling of water use reduction projects shows that the final wastewater effluent quality will not meet the requirements of Table 6 of the Approval, without modification or addition to the Effluent Treatment System as configured as of the date of the Approval. The application for an amendment must include a plan for alternative effluent management and/or treatment designed to meet the requirements of Table 6A of the Approval.

These provisions make it clear that the Department intends to require Northern Pulp to discharge its effluent at a treatment facility at a location which has yet to be determined if it is unable to comply with the conditions imposed. By specifically not contemplating or providing a means for compliance to be achieved through modifications or additions to the current Effluent Treatment System, the Province is in breach of the Agreements with Northern Pulp that entitle Northern Pulp to use the Effluent Treatment Facility until 2030. The estimated capital cost of constructing a new effluent treatment plant is in excess of $100 million.

Northern Pulp therefore requests that any Clause in the Approval which contemplates that Northern Pulp cease using the Effluent Treatment Facility prior to 2030, including Clauses 5(h), 5(i), 5(j), 6(h), 6(i) and 6(j) and Table 6A, be removed.

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[3] Report from Paul H. Klopping dated April 8, 2015 ("Klopping Report"). Paul Klopping has been auditing the Mill since 2005.

I. Rehabilitation Plans – Clause 24

Clause 24(a) of the Approval provides that Northern Pulp must submit a detailed closure plan to the Department one year prior to the decommissioning or closure of the Facility or any part thereof. To the extent that the definition of Facility in the Approval incorporates by reference to Effluent Treatment System the "former stabilization lagoon (known as Boat Harbour)", Northern Pulp refers to Sections I.A., I.B. and I.H. above and submits that any requirement under Clause 24 for Northern Pulp to cease using its current Effluent Treatment Facility, or submit any plan or undertake any costs or rehabilitation with respect to Boat Harbour is in breach of the Lease and the Indemnity Agreement. Northern Pulp is entitled to indemnification from the Province in respect of any cessation by Northern Pulp of the Effluent Treatment Facility and any costs for any rehabilitation of Boat Harbour imposed by the Province on Northern Pulp, under the Approval or otherwise.

Northern Pulp requests that Clause 24 be revised to acknowledge that any cessation of Northern Pulp's use of the Effluent Treatment Facility and any rehabilitation of Boat Harbour are the Province's responsibility and the plans and costs thereof will be assumed by the Province.

II. Prohibitory and/or Impossible Provisions

A. Capital Investment Required to Comply with Approval is Prohibitory - Clauses 5(i), 6(c), 6(g), 6(i), 14(m) and 14(n)

In addition to being in breach of the Agreements as discussed throughout Section I above, Northern Pulp's compliance with the Approval will require Northern Pulp to undertake significant capital expenditures estimated to be in excess of $90 million, to divert surface run-off water to the Pictou Harbour, to implement a new spill containment system, and to reduce COD. See enclosed EKONO Memo, p. 2.[4]

The estimated $90 million in capital expenditures referred to above does not include the capital expenditures to construct a new effluent treatment plant in order to comply with Clauses 5(i) and 6(i) of the Approval, estimated to be in excess of $100 million. This requirement is also in breach of Northern Pulp's rights under the Lease, as discussed in Section I. H. above

The cumulative effect of the capital cost associated with Clauses 5(i), 6(c), 6(g), 6(i), 14(m) and 14(n) of the Approval is effectively the prohibition of Northern Pulp's operation by the Department, instead of the Department's regulation of it.

With respect to COD, Clause 6(g) requires Northern Pulp to develop a plan for additional reductions to achieve a maximum effluent COD of 11,890 kg/day at Point C. This equates to a 77% reduction from the Mill's present operation.[5] Such a provision is not regulatory in nature. It is prohibitory. That is, the Approval through Clause 6(g) has the effect of prohibiting Northern Pulp's activities, rather than regulating them. As discussed further in Section II.B. below, the

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[4] Memo from Heikki Mannisto and Eva Mannisto dated April 6, 2015 entitled "Possibility to meet COD limit of 11,890 kg/day" ("EKONO Memo").

[5] The average annual COD at Point C for the period of January 1, 2010 to December 31, 2014 is 58.8 kg/Adt. At 873 Adt/day, that equates to 51,350 kg/day at the end of the Approval if the Mill is running at current levels. 11,890 kg/day represents an intensity of 13.6 kg/Adt at Point C.

COD reduction may not even be possible and any capital investments in an attempt to achieve this requirement would be prohibitory.

With respect to surface run-off diversion under Clause 6(c), the new segregated system would require a new sewer network, retention ponds, oil and sediment traps, sample collection, alarms and a mechanism to divert this stream back to the main Mill effluent stream if contamination was detected. This new non-contact outfall will require permits from the Department as well as Environment Canada and the Department of Fisheries and Oceans. Northern Pulp has no guarantee that the Mill will receive these required approvals from these departments.

A new spill containment system under Clause 6(g) is also unreasonable in that it imposes significant cost on Northern Pulp without additional environmental or public benefit and is in breach of the Agreements.

The high capital cost of complying with any or all of Clauses 6(c), 6(g), 14(m) and 14(n) are completely disproportionate to the benefit, if any, to the public and the environment of these measures. The surface run-off diversion requirement is disproportionate given the small fraction of the flow that surface run-off represents and the fact that it is presently treated with the Mill effluent. The spill containment requirement is not only disproportionate but unnecessary because Northern Pulp already has an adequate spill containment system available to it. The Department is aware through the AMEC Report that process technologies for reducing COD to the requirement of Clause 6(g), assuming those reductions are achievable, would be prohibitory. The timelines contained in the Approval for achieving these Clauses are too short for such major construction projects to be completed. Therefore, the conditions in the Approval are impossible for Northern Pulp to meet and are prohibitory.

Through the cumulative effect of these Clauses, and the capital cost associated therewith, the Department is prohibiting the Mill from operating. Instead of regulating the existing Mill, the Department is requiring that Northern Pulp build a substantially new Mill. Provisions of regulatory approvals that prohibit rather than regulate operations are unreasonable and ultra vires. Therefore, Northern Pulp requests that Clauses 5(i), 6(c), 6(g), 6(i), 14(m) and 14(n) be removed from the Approval.

B. COD Reduction is Prohibitory and Impossible to Meet – Clause 6(g)

As noted in Section II.A. above, the proposed COD reduction of 77% under Clause 6(g) of the Approval is prohibitory not only because of the capital investments in order to attempt to achieve this requirement but also because the proposed COD reduction is unrealistic and is essentially impossible for Northern Pulp to meet.

The AMEC Report provided an assessment of the status and operation of the existing treatment facility for the Mill as well as preliminary engineering for several effluent treatment alternatives. The assessment was conducted at the time that the regulatory discharge for the Mill was located at Point D. The AMEC Report concluded that the Mill's Aerated Stabilization Basin ("ASB") was operating well. Since the regulatory discharge point was moved to Point C on July 1st, 2010, there have been no incidences of daily or monthly BOD_5 or TSS being over the limits in the PPER. In fact, all tests have shown that the Mill is well below the regulated limits.

Even though the AMEC Report was based on outdated information and therefore could not recognize the current operational attainments of the Mill, the AMEC Report itself demonstrates that it is simply not possible for the Mill to achieve this COD reduction. As such, the

requirement that the Mill achieve the reduction required by Clause 6(g) amounts to a prohibition of the Mill's operations, rather than the regulation of it, and therefore is unreasonable.

Appendix 2 of the AMEC Report contains the preliminary engineering for the effluent treatment alternatives with mass balances (annual averages and not daily limits). The most stringent case in the AMEC Report, Case 3, completely abandons the Mill's effluent treatment facility and replaces it with a new Activated Sludge Treatment ("AST") system and includes "Significant Improvements & Water Use Reduction", as outlined in section 4.2.3 of the AMEC Report. Drawing #158229-100-6-923 is the mass balance for the New Treatment Plant – Case 3. This Case assumes a 50% reduction in liquor losses will be achieved (20,400 kg/day to 11,400 kg/day). It also assumes marginal increase in production with a design capacity of 908 Adt/day. Incineration of Waste Activated Sludge would occur in the Recovery Boiler, which at present is not permitted in Northern Pulp's Approval. The predicted effluent flow is 65,151 m3/d as an annual average; the new daily limit of 67,500 m3/d required in the Approval is even more stringent than the AMEC mass balance and does not consider seasonal variations.

The predicted COD for Case 3 is 70,900 kg/day. The typical COD removal for a new AST system would be in the neighbourhood of 50 – 60%. Even at the higher end of that removal efficiency (60%), the final COD discharge would be 28,360 kg/day (70,900 kg/day x 0.4). In summary, Case 3 demonstrates that even with significant in-Mill improvements and a brand new AST treatment system, the Mill would still exceed, by more than double, the COD daily limit of 11,890 kg/day at Point C as set out in the Approval at Clause 6(g). AMEC recognizes that other process technologies that were not included in Case 3 would be economically prohibitive to the Mill. The Klopping Report, EKONO Memo and McCubbin Report[6] all agree that the requirement contained in Clause 6(g) is unrealistic and that the associated costs would be significant.

Therefore, the requirement contained in Clause 6(g) of the Approval, as a daily limit representing a 77% reduction from the Mill's current annual average for the Mill's current operations, is impossible for Northern Pulp to meet. The Department included the requirement of Clause 6(g) in the Approval knowing that the AMEC Report says that the Mill cannot achieve that requirement even if the Mill adopted the strictures of Case 3 including installing a completely new treatment system. The requirement of Clause 6(g) is completely untenable, even according to the Report on which the Approval is based. The Department therefore must be considered to have included this provision in order to prohibit the Mill's operations.

The Approval contains conditions that treat the Mill as if it is a new mill and as such the Department has imposed regulatory standards on the Mill that are prohibitory to the existing Mill operating. Provisions of regulatory approvals that prohibit instead of regulate and that are impossible to meet are unreasonable and ultra vires. Therefore, Northern Pulp requests that Clause 6(g) be removed from the Approval.

C. Modeling Deadlines are Impossible to Meet - Clauses 5(h) and 6(h)

Clauses 5(h) and 6(h) of the Approval require Northern Pulp to submit to the Department modeling for all reduction stages along with a list of planned projects required under Clauses 5(e) and 6(e) no later than October 30, 2015.

Northern Pulp's consultants have indicated that it is impossible to complete in-Mill engineering studies and then adequately complete effluent quality modeling to predict Mill wastewater

[6] Report from Neil McCubbin dated April 6, 2015 ("McCubbin Report").

changes for submission to the Department by October 30, 2015. It is not reasonable to require such a deadline considering the impossibility of meeting that deadline as well as the fact that the modeling is for reductions that are not required until 2017.

In Northern Pulp's submission, October 30, 2016 is more reasonably attainable and appropriate. Therefore, Northern Pulp requests that Clauses 5(h) and 6(h) each be revised to change the deadline for this study from October 30, 2015 to October 30, 2016.

D. Regulatory Compliance without Modification or Addition - Clauses 5(i), 6(i) and 6(j)

Northern Pulp is also precluded through Clauses 5(i) and 6(i) from achieving the requirements of the Approval through modification or addition to the effluent treatment system as configured as of the date of the Approval, and through Clause 6(j) from reduction and prevention of liquor losses entering the effluent system. In addition to being contrary to the Agreements as discussed in Section I.H. above, requirements that a proponent achieve regulatory compliance without making improvements to the existing system are prohibitory on their face, and do not serve the public or benefit the receiving environment. Northern Pulp again requests that Clauses 5(i), 6(i) and 6(j) be removed.

III. Vague and Uncertain Provisions

A. New Treatment Facility - Clauses 5(h), 5(i) and 5(j) and Clauses 6(h), 6(i) and 6(j) and Table 6A

In addition to the objection noted in Section I.H. above, the majority of parameters and discharge limits in Table 6A in the event that Northern Pulp is required to discharge its effluent at a treatment facility at a different location have yet to be determined. Those limits are specifically contemplated as being determined based on an unknown location of a new treatment facility and on results of a receiving water study yet to be completed. That receiving water study must meet the satisfaction of the Department, yet the Approval does not indicate any criteria upon which such water study will be considered satisfactory. Northern Pulp considers the requirement to apply for and obtain approval of an as-yet-unidentified discharge location to be unreasonable.

At present, there is an absence of standards and guidelines in Clauses 5(h), 5(i), and 5(j) and Clauses 6(h), 6(i) and 6(j) and Table 6A. These provisions are too vague and uncertain to be considered reasonable. The conditions contain no limitation on the Department's discretion and as a result Northern Pulp has no fair notice of the conditions it must meet. Therefore, Northern Pulp requests that these provisions be removed.

B. COD Reductions - Clause 6(e)

The provisions in Clause 6(e) requiring reductions in COD do not specify or provide a means for calculating the base point for measuring such reductions. These provisions are therefore too vague to be reasonable and therefore must be removed.

IV. Unrealistic and Otherwise Unreasonable Provisions

It is clear that many provisions of the Approval are based on the AMEC Report. However, as noted in Section II.B. above, the AMEC Report is outdated and does not represent the Mill's

current operations and its operational achievements since the date of the AMEC Report. As such, certain provisions imposed by the Department in reliance of this Report are unreasonable.

The AMEC Report ranks Northern Pulp in the 95th – 100 percentile on water usage in Canada. In 2007, the year that Northern Pulp was benchmarked in this 2010 study, the Mill's regulated effluent discharge point was Point D exiting the Boat Harbour basin. In July 2010, the regulated effluent discharge point was moved back to Point C exiting the ASB. Point D is not representative of the Mill effluent flow when compared to other mills because it is downstream of the treatment plant. Effluent flow at Point D is historically 25% higher than at Point C due to surface run-off from the large Boat Harbour basin. Surface run-off was not taken into account in the AMEC Report, and therefore comparisons are not valid. Comparison of Point C data at the end of the treatment process is appropriate for benchmarking purposes. Table I shows the decrease in effluent flow from 2010 forward:

Table I: Effluent Flow Intensity (m3/Adt) – Annual Averages

Regulated Outfall Location	2007	2008	2009	2010	2011	2012	2013	2014
Point D	112	133	129					
Point C				94	89	87	86	88

As the Approval is clearly based on a lack of appreciation for the Mill's current operations, including an outdated report from AMEC which contains conclusions that are no longer correct, the Department has no basis for many provisions contained in the Approval. It is unreasonable for the Department to impose regulatory standards based on a report that relies on almost 10-year-old data and disregards the achievements of the Mill since that time.

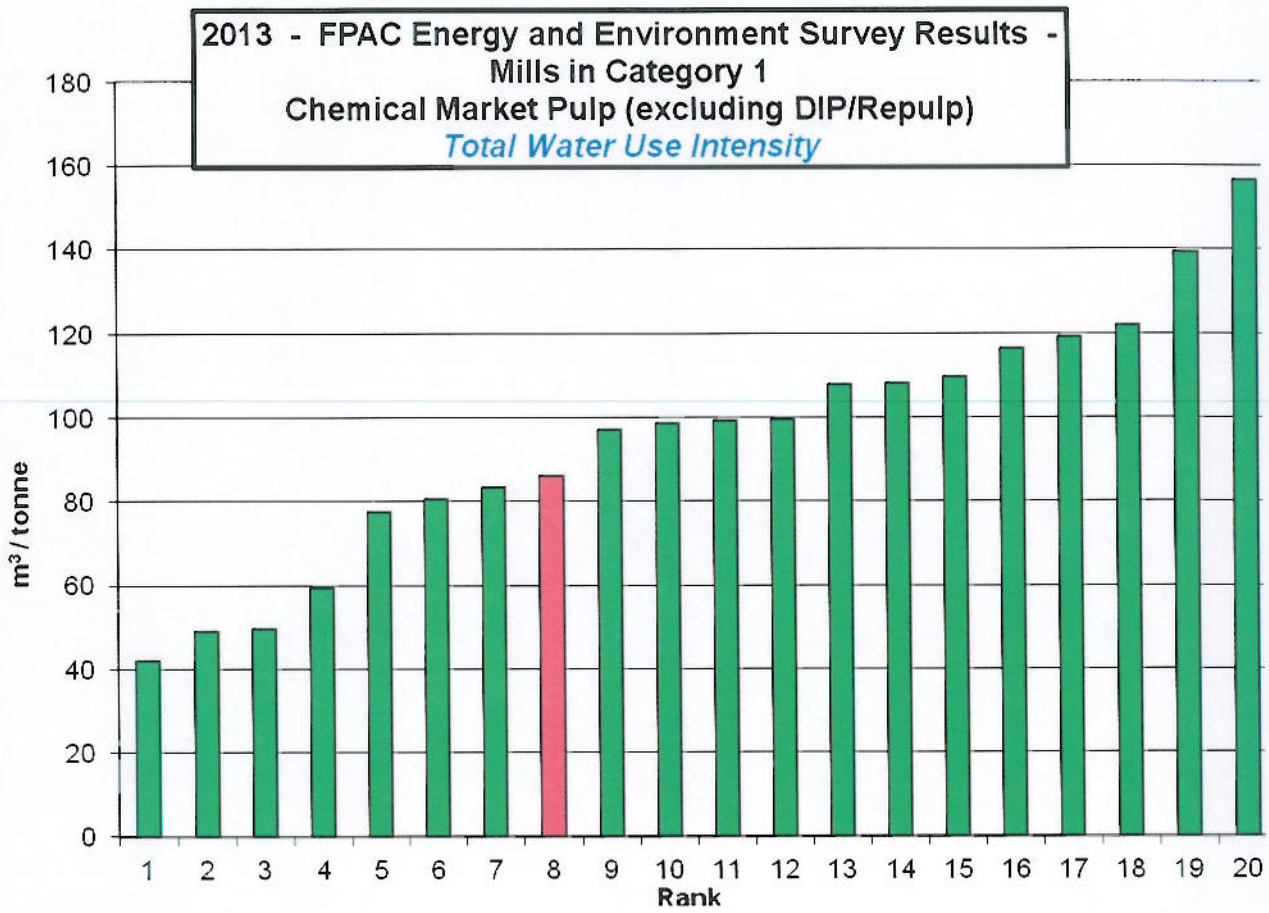
Other provisions in the Approval are unreasonable as they provide little or no additional environmental benefit but impose very significant costs on Northern Pulp and therefore are improper. As well, statements made by the Department in issuing the Approval make it clear that some provisions of the Approval are based on a misunderstanding of or lack of appreciation for the Mill's operations, which make those provisions unrealistic. Some provisions in the Approval are unreasonable as they fail to recognize industry norms or factors outside of Northern Pulp's control. Others are simply inaccurate. Those provisions must therefore be removed or revised as set out in the Sections below.

A. Water Reduction Requirement – Clauses 5(d) and 5(e)

As stated by all effluent experts (Ken Frei and Guy Martin of KSH, Paul Klopping, Neil McCubbin, and Heikki Mannisto and Eva Mannisto of EKONO), the daily limits for water intake and effluent contained in the Approval are highly unusual. Few jurisdictions place flow limits in general on intake and effluent; exceptions are generally where the mill makes up a large percentage of the waters (usually rivers) that it is drawing its water supply from or is discharging effluent into. Mills that ultimately discharge into marine environments do not typically have these restrictions: see KSH Memo, p. 4. The AMEC Report at p. 30 refers to the 2007 EKONO study which also recognizes that such limits will only be placed on mills in certain circumstances, which Northern Pulp notes are not applicable to the Mill.

The requirements related to water reductions in the Approval appear to be based on a mischaracterization of the Mill's water usage. A Departmental document accompanying the

release of the January 30, 2015 version of the Approval stated that "The Mill is a large consumer of water, especially compared to other mills." In fact, Northern Pulp's water usage is average by comparison to other Canadian mills. Enclosed is a copy of the Forest Products Association of Canada survey entitled "FPAC 2013 Energy and Environment Benchmarking Report – Pulp and Paper Sector" dated March 10th, 2015 ("FPAC Report"). The following graph is an excerpt from the FPAC Report. With 20 out of a possible 30 mills reporting, Northern Pulp ranks the 8th best in the survey for water use intensity:



Reductions of water flow of the magnitude contained in the Approval could have negative effects on the biological treatment process and ultimately the receiving water, increasing odour for residents who live in the vicinity of the effluent treatment centre.

As noted in Section I.D above, Clauses 5(d) and 5(e) should be removed from the Approval for being in breach of the Water Supply Agreement. Clauses 5(d) and 5(e) are also unreasonable as they ignore industry norms and are based on a mischaracterization of the Mill's water usage, as a result of the Department's reliance on an outdated report. Therefore, Northern Pulp repeats its requests that these Clauses be removed.

B. TRS Reduction Requirement – Clauses 8(d), 8(e) and 8(f)

The new requirement under Clause 8(d) for water-phase TRS measurements in the effluent are best described by the KSH Memo at p. 14-16. In summary, Clause 8(d) is modeled after

Ontario's new 2014 Air Pollution – Local Air Quality Regulation (which imposes certain air standards, including TRS) and the Ontario Technical Standards to Manage Air Pollution. To Northern Pulp's knowledge, Ontario is the only other province that is introducing this standard. Mills may comply with the Regulation by electing to register under the optional Technical Standard which has phased-in limits for TRS in water entering the wastewater treatment system. The Ontario Ministry of the Environment researched the relationship between TRS effluent loading and TRS in ambient air originally reported by the US EPA. Because the understanding of the relationships of TRS effluent loading and TRS in ambient air are still in their infancy and the influence of other parameters (including pH, temperature and the various types of odorous compounds present) are not well known, the Ontario government introduced this as an optional standard that mills could choose to apply for. Mills that choose to apply will, over time, provide data that will lead to improved understanding of the relationships between effluent concentrations and air quality. The optional nature of the standard gives Ontario the ability to modify the standard as is deemed necessary as information and data become available. By imposing Clause 8(d) in the Approval, the Department has applied the optional Ontario standards as a hard and fast requirement that must be abided by without regard for the outcome of the Ontario mill experiences. If the limits are deemed to be unrealistic, Ontario has allowed room for possible changes; the Department has not.

It is of particular note that Northern Pulp is regulated to fixed TRS limits, including 3-fold reductions in coming years, when the Mill does not have any baseline data. The limits in Clause 8(d) have been set by the Department before any data has been gathered and before it is known if there is even an environmental reason to impose such limits. Ontario has recognized that there is not enough known yet about the relationship between TRS effluent loading and TRS in ambient air to justify the regulation. What is an optional consideration in Ontario because the precise relationship between TRS effluent loading and TRS in ambient air has not yet been demonstrated, the Department has made a mandatory requirement of the Approval. It is unreasonable for the Department to impose a limit before knowing if there is a relationship necessitating that limit.

Northern Pulp has contacted a mill in Ontario and all of the major laboratories in Canada. It appears that no Canadian laboratories (after conferring with their satellite offices) are capable of performing the test required by Clause 8(d), NCASI Method RSC – 02.02. At this point, the closest capable lab that Northern Pulp has found is located in Simi Valley, California. This demonstrates that the Department is imposing a requirement on the Mill is entirely inconsistent with other Canadian jurisdictions.

As noted in Section I.G above, Clauses 8(d) and 8(f) should be removed from the Approval as they are in breach of the MOU. Clauses 8(d) and 8(f) should also be removed as there is no known scientific basis for the limit, which regulators elsewhere have recognized. Northern Pulp does not object to performing testing of TRS in accordance with Table 6 for the benefit of residents, as such monitoring is contemplated in Clause 8(e). However, the imposition of the regulatory requirement to achieve the TRS limits imposed in Clauses 8(e) to 8(f) is unreasonable.

Northern Pulp requests that Clauses 8(d) and 8(f) be removed entirely and that Clause 8(e) be revised to impose only a requirement that Northern Pulp test TRS in accordance with Table 6.

C. Effluent Flow Reduction Requirements – Clauses 5 and 6(a)

The reductions in Clauses 5 and 6 of the Approval are inconsistent. As water use and effluent flow are directly related in terms of the Mill's mass balance, the water use restrictions outlined in Clause 5 of the Approval effectively restrict the effluent limit contained in Clause 6(a) of the Approval to less than 67,500 m3/day or 77.3 m3/Adt. The effect of the water use restrictions in Clause 5 is to impose a greater effluent flow reduction requirement than the Department requires in Clause 6(a).

The AMEC Report (p. 25-26) indicates that consumptive water use (water taken but not returned as effluent) with the power boiler scrubber in operation is estimated at approximately 2 m3/ADt. In essence, from a mass balance perspective, Clause 5(d) is more stringent and practically translates to an effluent limit of 70.1 m3/Adt (72.2 – 2) or 61,200 m3/day. This represents a further reduction of 6,300 m3/day or an additional 9%. The McCubbin Report indicates that the net water consumption of 2 m3/Adt is very conservative and could be as high as 5 m3/Adt, which could lead to even further reduction requirements. KSH indicates that 8% of incoming water supply to the mill would be lost to evaporation. All three consultants (AMEC, Neil McCubbin and KSH) support this reduction of an additional 9-10%.

In addition to the arguments set out in Section IV.A above, the water use restrictions in Clause 5 are unreasonable because they impose conditions that are more onerous than the effluent control limits in Clause 6(a) of the Approval. Therefore, Northern Pulp requests that the water use restrictions in Clause 5 be removed.

D. COD Limits – Clauses 6(d) to 6(j)

The COD limits in Clauses 6(d) to 6(j) of the Approval are based on the outdated AMEC Report and are unreasonable.

Table II below summarizes the Mill COD data at several locations in the treatment plant.

Table II: Effluent COD Intensity (kg/Adt)

Location	2007	2008	2009	2010	2011	2012	2013	2014
Point A	154.6	124.7	103.5	123.4	99.6	92.2	82.5	87.8
Point C	**71.7**	**82.3**	**76.7**	**70.1**	**63.1**	**56.0**	**53.6**	**51.4**
Point D	74.0	78.6	71.3	65.1	62.8	56.0	55.2	50.8

The improvements in COD intensity beginning in 2011 reflected in Table II are in part the result of the Green Transformation Projects carried out by the Mill that year. Continuous improvement initiatives at the Mill have continued to provide COD reduction through to 2014.

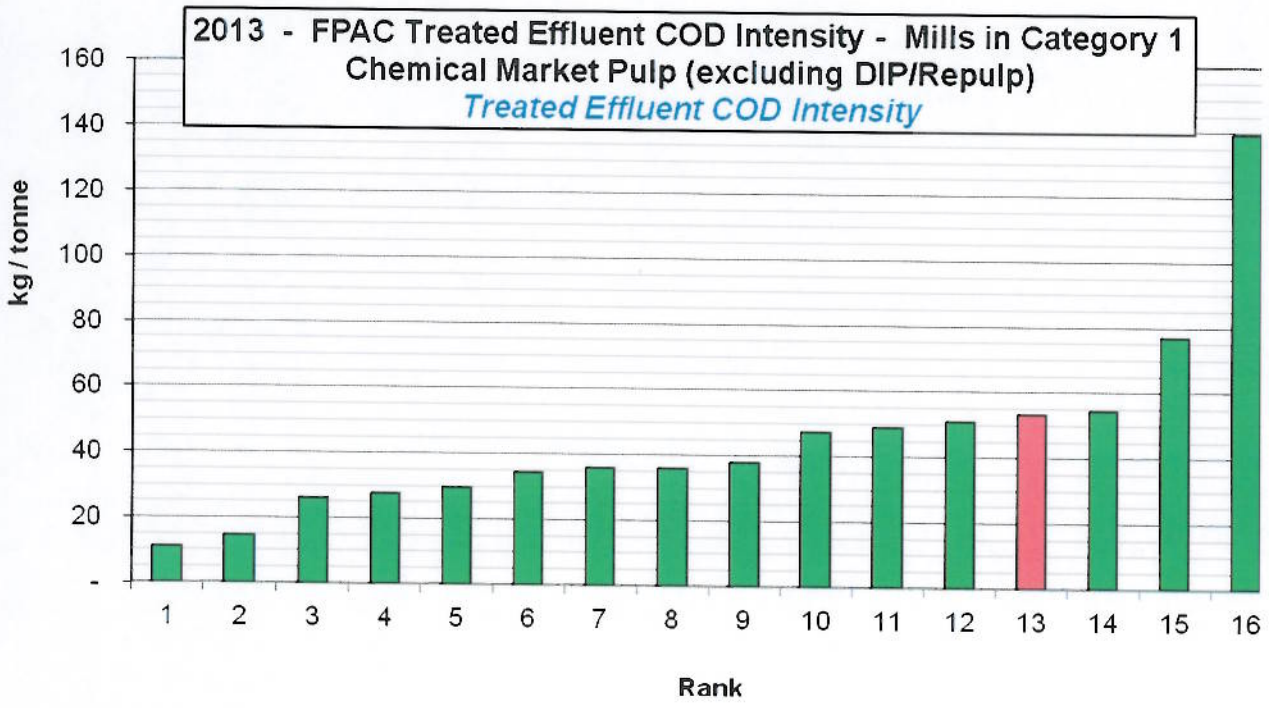
Since the AMEC Report was written, many of the proposed projects for effluent quality improvements identified in the Report have been completed, including: improvements to brown stock washing including two new washer drums and closing of the screen room with true counter-current washing, recycle of PM rejects, Stage 1 DNCG system, sludge removal plan, PB scrubber and a new emergency spill tank. A COD reduction of 30% has been achieved from the 2007 benchmark conducted in the AMEC Report.

Although not clearly stated, Northern Pulp has assumed that the COD reductions in Clause 6(e) of the Approval are based on the five year annual average COD at Point A that was reported pursuant to Clause 6(d).

Northern Pulp's average COD intensity at Point A for 2010 through 2014, as requested in Clause 6(d), is 97.1 kg/Adt. At 873 Adt/day[7], the daily discharge would be 84,800 kg/day. The 50% reduction in COD required by Clause 6(e) would mean a daily discharge of 42,400 kg/day at Point A.

There is very little published data for COD in North America, and virtually nothing from the United States. Data that is published, and all third party data presented in this Appeal, refer to "after treatment" and would be comparable to Point C in the above Table II, not point A (the inlet to treatment).

The Department has recently made statements in media interviews that Northern Pulp has four times greater COD than mills of similar size. This is clearly wrong. The following graph is an excerpt from the FPAC Report. With 16 out of a possible 30 mills in Canada reporting, this graph shows that Northern Pulp clearly does not have four times greater COD than mills of similar size, nor does it have the highest COD discharge.



The AMEC Report indicates (on p. 29) that benchmarking of effluent was done using the widely accepted EKONO study of September, 2007, which is based on 2005 data. The enclosed EKONO Memo compares COD and BOD data from Northern Pulp to the results found in the EKONO database. The EKONO benchmarking and analysis of the new COD limits concludes

[7] 310,000 Adt/year at 355 days is 873 Adt/day at the end of the Approval, assuming that the Mill is able to increase production to this limit by that time.

that the two lowest COD discharge mills are hardwood mills with oxygen delignification, modified cooking, condensate stripping, etc. and the lowest COD discharge in a Canadian bleached softwood kraft pulp mill in the database is 28-30 kg/Adt. The EKONO Memo also concludes that more than 40% of the North American bleached market kraft pulp in 2013 was produced by mills with BOD discharges higher than those at Northern Pulp's Mill. This proves once again that the Department is not working with current data and does not understand the influences of mill process equipment and wood species on COD.

It is clear that the Department was relying on outdated studies and analysis in setting the COD limits in Clause 6. This is based on outdated data and is unreasonable. Therefore, Northern Pulp requests that Clauses 6(d) to 6(j) be removed.

E. Production Limit – Clause 4(a)

Clause 4(a) of the Approval limits the maximum production rate of the Mill to 310,000 Adt/yr. Not only does this Clause prohibit the Mill's future expansion, it also offers no additional environmental benefit. The production capacity of the Mill is and should properly be limited by the existing equipment of the Mill and the other requirements of the Approval. So long as Northern Pulp is in compliance with the other requirements of the Approval, whatever the Mill's production limit happens to be when it complies with those requirements has no environmental relevance.

There is no additional environmental basis or additional public benefit for including in the Approval a provision that arbitrarily limits the production of the Mill. Northern Pulp therefore requests that Clause 4(a) be removed entirely.

F. Phenanthrene Testing – Clause 12(ag)

Clause 12(ag) requires semi-annual testing of Phenanthrene at Surface Water station SW12-3. The trend analysis requested is not warranted because the measurement was at or near the detection limit. See enclosed Dillon Consulting Memo[8]. The requirement of Clause 12(ag) therefore has no justification. It only adds cost to Northern Pulp with no corresponding additional benefit to the environment. The requirement in Clause 12(ag) is therefore unreasonable and Northern Pulp requests that it be removed.

G. Daily Limits for Water and Effluent Usage – Clauses 5(d), 5(e), 6(a) and 6(b)

The reference to water and effluent usage in the Approval in terms of daily limits is contrary to the way that effluent data is compared and benchmarked in the industry. See the AMEC Report and the enclosed EKONO Memo, FPAC Report, KSH Memo, McCubbin Report, and Klopping Report. As stated in the KSH Memo at p. 5, "Effluent flow restrictions are usually stated on an average basis, usually monthly or annually since there is no measurable risk to the environmental [sic] by one day of effluent flow above a certain amount."

The imposition of daily limits for water usage that do not consider seasonality are of particular concern to the Mill. Based on data from the last three years, the Mill's water intake has peaked at 87,000-91,000 m3/day (23-24 million gallons/day), even though the Mill's annual average water usage is 75,000 m3/day (20 million gallons/day). These peak days do not occur during the highest surface run-off months. Rather, they historically occur during the hottest days

[8] Letter from Dillon Consulting Limited dated February 11, 2015 ("Dillon Consulting Memo").

during the summer when the incoming Middle River water is at its warmest and cooling systems are most heavily loaded. Cooling towers will be an essential component of water reduction that will recycle "once-through" cooling water for re-use. Cooling towers are driven by temperature differential (water to ambient air) and relative humidity and are a very effective means of cooling. However, heat transfer during hot, humid spells can be challenging. The cooling towers offer significant water reduction on an annual basis, but daily reductions will be highly influenced by weather. It is therefore clear that the Mill will not be able to operate and comply with the daily water usage requirements of the Approval on certain days of the year. Therefore, daily limits on water usage are unreasonable as they effectively preclude the Mill from operating on a consistent basis.

There is no environmental reason to impose a daily limit in Northern Pulp's circumstances. Northern Pulp therefore requests that all water and effluent usage restrictions in the Approval, including Clauses 5(d), 5(e), 6(a) and 6(b), be revised to be based on yearly averages, not daily limits.

H. $PM_{2.5}$ Contributors – Clauses 9(a) to 9(e)

Clauses 9(a) to 9(e) of the Approval transform the voluntary objectives of the Canadian Ambient Air Quality Standards ("CAAQS") into mandatory compliance requirements without any consideration given to other local sources of $PM_{2.5}$, especially other local heavy industry, wood-burning stoves and other non-commercial sources, or the not insignificant contribution of interprovincial and international transport of $PM_{2.5}$ from other sources.

It is unreasonable to require the Mill's mandatory compliance with the limits in the CAAQS without taking into account other industrial facilities, residential sources and transportation that are also contributors to $PM_{2.5}$ in the area. See KSH Memo, p. 16-18. As the Mill's compliance with Clauses 9(a) to 9(e) is outside of the Mill's own control, such mandatory compliance requirements are unreasonable.

Northern Pulp is confident that the Mill will meet these limits as a contributor; however, imposing mandatory compliance on the Mill without taking into account other contributors is unreasonable. Northern Pulp therefore requests that Clauses 9(a) to 9(e) be revised to acknowledge that Northern Pulp is only required to demonstrate that its relative contribution to the local air quality meets the limits of the CAAQS.

I. Particulate Limit – Clause 9(h)

Clause 9(h) imposes a regulatory limit on the Recovery Boiler for particulate of 77 mg/Rm^3 @ 11% O_2. We understand, based on discussions with the Department, that this limit is based on the performance expected by the manufacturer of the new electrostatic precipitator ("ESP"). This limit does not properly take into consideration the variables that can affect actual boiler performance on a day-to-day basis, particularly in the case of a new ESP attached to an existing boiler. Contrary to North American regulatory convention, the approach taken by the Department in setting the particulate limit for the Recovery Boiler treats the installation of the new ESP as if it were a significant modification to an existing boiler and, as such, treats it as a new boiler and sets emission limits that are in line with such units: KSH Memo, p. 21-22. This is inconsistent with the approach taken in most North American jurisdictions. For example, under the U.S. Air Quality Regulations, the addition and/or replacement of pollution control equipment, such as a new ESP, is not, in itself, considered to be a modification to an existing boiler, and would not result in treating such existing boiler as if it were a new boiler for the purposes of

setting a particulate limit. As the particulate limit imposed by Clause 9(h) fails to recognize that the ESP is being connected to Northern Pulp's existing boiler system and the variables that can affect the performance of that boiler system on a day-to-day basis, Clause 9(h) is unreasonable.

The limit imposed on Quebec's existing recovery boilers of 153.5 mg/Rm^3 @ 11% O_2 represents a more reasonable and appropriate limit, as it represents state-of-the-art performance for a boiler and direct contact evaporator system of the same vintage as Northern Pulp's boiler. See KSH Memo at p. 18-22. Northern Pulp therefore requests that Clause 9(h) be revised accordingly.

J. Middle River Study – Clause 5(f)

Clause 5(f) makes Northern Pulp responsible for conducting a maximum sustainable yield study of the Middle River watershed. In the 47 years of the Mill's operation, neither the Department nor any other stakeholder has ever raised a concern about water usage impacts on the Middle River watershed. The Michelin, Granton facility also draws its fresh water supply from the Middle River and discharges its effluent back into Middle River upstream of the Mill's water supply. As well, the river is dammed to prevent salt water intrusion and the spillway dam is controlled by the Province. See KSH Memo at p. 2. No low-level warnings have ever been communicated to the Mill.

In addition, the December 15, 2015 deadline does not allow for a full year study to cover all seasons which would be normal for this type of assessment, implying that existing data must be used. The responsibility of Northern Pulp to conduct this maximum yield study of the watershed (which is usually directed towards new applicants for a water removal permit) is unreasonable, especially considering the Mill does not have access to or control of pertinent data that would be required to properly complete this study.

Northern Pulp therefore requests that Clause 5(f) be removed.

K. Waste Dangerous Goods – Clause 4(i)

Northern Pulp requests that Clause 4(i) be revised to replace the words "all wastes generated" with "all waste dangerous goods generated" in order to clarify this Clause and ensure its consistency with the preceding Clause 4(h), which deals only with waste dangerous goods.

L. TRS Reporting – Clause 7(m)(i)

The monthly reporting requirement for TRS in Clause 7(m)(i) is internally inconsistent with the reporting requirement for TRS in Table 6 of Appendix A. Clause 7(m)(i) should therefore be amended to include an exception for TRS reporting of every three months.

M. Cardlock Facility Wells – Clauses 12(a) and 12(g)

The Approval at Clause 12(a) requires Northern Pulp to maintain the Cardlock Facility Wells. The Cardlock Facility Wells are owned and operated by Parkland Fuels. Northern Pulp has permission from Parkland Fuels to sample the Wells, but the maintenance of the Wells is not under the control of Northern Pulp. Further, Clause 12(g) requires Northern Pulp to maintain records, including borehole logs, construction details and maintenance records, in respect of the Cardlock Facility Wells. This information is the property of Parkland Fuels and Parkland Fuels has no obligation to provide it to Northern Pulp.

Clauses 12(a) and 12(g) are unreasonable to the extent that these Clauses impose an obligation on Northern Pulp to maintain Wells which are not under Northern Pulp's ownership or control and require Northern Pulp to maintain with respect to those Wells records of information to which Northern Pulp is not entitled. Northern Pulp therefore requests that Clauses 12(a) and 12(g) be revised accordingly.

N. Surface Water Stations – Clause 12(ad)

Several of the Surface Water stations listed do not exist, while other new stations are not listed in Clause 12(ad). See enclosed Dillon Consulting Memo. Northern Pulp requests that this Clause be revised accordingly.

O. Stantec Report – Clause 12(au)

Clause 12(au) is inaccurate as it refers to the Stantec "Hydrogeological and Hydrological Evaluation of the Boat Harbour Treatment Facility Report" as being dated April 30, 2012. Rather, the Report is dated September 28, 2011. Northern Pulp requests that this Clause be revised accordingly.

V. Inconsistency with FOIPOP – Clause 22

The requirements in Clause 22 of the Approval that Northern Pulp provide information to the Pictou Landing First Nation ("PLFN"), including a copy of all reporting information and reports that are required to be submitted under the Approval, is inconsistent with the protection granted to Northern Pulp under FOIPOP. The public accessibility to information under the control of the Department under section 10 of the *Environment Act* is subject to the important restrictions of FOIPOP: section 10(2) of the Act.

Specifically, section 21 of FOIPOP provides that a head of a public body (such as the Minister and Administrator of the Department of Environment) must refuse to disclose information:

> (a) That would reveal
>
> (i) trade secrets of a third party, or
> (ii) commercial, financial, labour relations, scientific or technical information of a third party;
>
> (b) That is supplied, implicitly or explicitly, in confidence; and
>
> (c) The disclosure of which could reasonably be expected to
>
> (i) harm significantly the competitive position or interfere significantly with the negotiating position of the third party,
> [...]
> (iii) result in undue financial loss or gain to any person or organization, or

Much of the information required to be provided by Northern Pulp to the Department constitutes commercial, financial and technical information. That information is supplied to the Department in confidence. The disclosure of this information could reasonably be expected to harm both

Northern Pulp's competitive position as well as its negotiating position with respect to an action that has been brought by PLFN against Northern Pulp. The competitive position of Northern Pulp would be harmed because its competitors in a global market and others having interests adverse to Northern Pulp would become fully aware of the Northern Pulp's commercially sensitive information.

Requiring Northern Pulp to comply with Clause 22 of the Approval is contrary to Section 10 of the *Environment Act* and Section 21 of FOIPOP. The Department through the Approval is circumventing the due process provided for under FOIPOP and the procedural fairness afforded to Northern Pulp with respect to the release of Northern Pulp's confidential and commercially sensitive information to third parties. The Department cannot by administrative fiat overcome a legislative requirement. Clause 22 of the Approval therefore is an unreasonable and ultra vires condition imposed by the Department. Accordingly, Northern Pulp requests that Clause 22 of the Approval be removed entirely.

Northern Pulp hereby confirms that any documents submitted by Northern Pulp to the Department at any time, including but not limited to this Appeal and its enclosures, are being submitted with the expectation that the Department will observe the protections afforded to Northern Pulp by FOIPOP in respect of such documents.

CONCLUSION

In light of the above, Northern Pulp respectfully requests that you exercise your powers under Section 137(4) of the Act to allow Northern Pulp's Appeal and remove or revise the provisions of the Approval as outlined in this letter.

Yours truly,

Terri Fraser

Terri Fraser, Technical Manager
Northern Pulp Nova Scotia Corporation

Encl.[9]

1. KSH Memo, Ken Frei and Guy Martin Resumes
2. McCubbin Report, Neil McCubbin Resume
3. EKONO Memo, Heikki Mannisto and Eva Mannisto Resumes
4. Klopping Report, Paul Klopping Resume
5. Dillon Consulting Memo
6. FPAC Report

cc: Bruce Chapman, Northern Pulp (via email)
cc: Dave Davis, Northern Pulp (via email)

[9] The enclosures refer to Approval No. 2011-076657-R03 but remain valid with respect to the Appeal.

APPENDIX H-22

DILLON CONSULTING

NORTHERN PULP NOVA SCOTIA

Replacement Effluent Treatment Facility

Information Submission to CEAA

April 2018

4.1.3 Emissions, discharges and waste

Dewatered solids ('sludge') is collected as a waste material from the ETF. The sludge includes clay, sand, silt, organic matter, nutrients, microorganisms and metals. It is proposed that the sludge is mixed with the existing biomass feeding the NPNS Biomass Power Boiler. Combustion will occur as it does now, which is on a travelling grate at the bottom of the boiler, using the same controls and combustion temperatures as are currently being used. Quantity of sludge generated will be determined after detailed engineering is complete, but will be approximately 5 - 10% of the biomass currently fed to the boiler.

With the proposed future addition of sludge into the power boiler, potential for changes to air quality are being assessed using air dispersion modelling as part of the Environmental Assessment. Effluent quality is discussed under operation of the ETF in **Section 4.1.1** above.

The replacement ETF will significantly reduce odour emissions relative to existing conditions. Core to the treatment difference between the existing and proposed facility is how accumulated solids are handled. In the new system they will be continuously removed as underflow from the clarifiers. In the existing facility, solids settle over time in both the primary sedimentation basins and the aerated stabilization basin and decompose under anaerobic conditions, generating hydrogen sulfide, hence creating odour. Additionally, air needed in the process will be injected subsurface in the new facility. In the existing facility, aeration stages occur at the surface, by the action of the surface aerators that throw the effluent into the air to allow it to absorb oxygen. This action also allows sulphur compounds to be released to the atmosphere by volatilization. The new cooling stage was also designed with odour in mind. NPNS has opted to install indirect effluent cooling to completely eliminate any chance for odour release from this stage in the process.

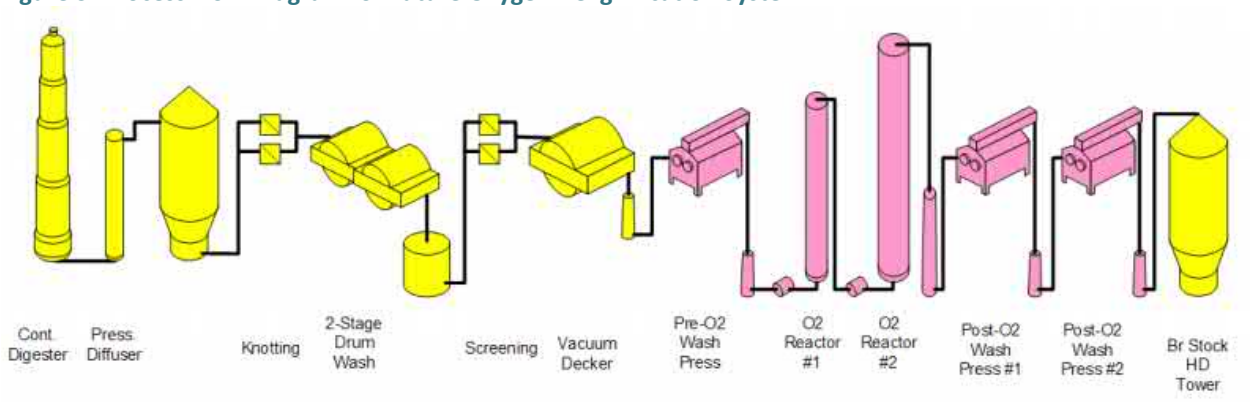
4.2 Proposed in-Plant Upgrades

There are several in-plant upgrades proposed subsequent to the ETF project which are anticipated to further improve effluent quality, including the addition of an oxygen delignification system and fresh-water cooling towers. The environmental improvements resulting from the proposed upgrades described are not included as part of the EA. The proposed in plant upgrades will occur subsequent to the commissioning of the Effluent Treatment Facility, and therefore, the ETF must be able to meet approvals for construction without the upgrades in place.

4.2.1 Oxygen Delignification System

Two stage oxygen delignification technology will be incorporated into the pulp making process. The system includes oxygen reactors and wash presses. It will be installed after the brown stock washing stage and before the existing bleaching stages. A process flow diagram is included in **Figure 3** below.

Figure 3: Process Flow Diagram for Future Oxygen Delignification System



Legend:
Existing equipment: Yellow
Future equipment: Pink

Oxygen delignification systems use oxygen gas to react with residual lignin that remains in the pulp after brown stock washing. The lignin removed in this new stage will result in the use of less bleaching chemicals to whiten the pulp in the existing bleach plant. It is a significant and well-proven process for Elemental-Chlorine-Free (ECF) pulp and as such it is often referred to as the first stage of bleaching (oxygen bleaching). Benefits of oxygen delignification include:

- Reduces chlorine dioxide bleaching chemicals by 30-40% leading to a decrease in effluent loading for BOD, COD and AOX;
- Improvement in aesthetics of effluent (colour);
- A reduction in wood losses;
- Increase in recovery of lignin that can be used in the boiler thus reducing carbon footprint; and
- A reduction in nutrients added to the effluent.

4.2.2 Water Cooling Towers

A significant portion of water consumption at the NPNS facility is using non-contact water systems for cooling and maintaining temperatures within the kraft pulp system. NPNS will install water cooling towers (subsequent to the ETF Project) to decrease the temperature of water used in the cooling systems. This will allow for water to be recycled within the system, as well as making the cooling itself more efficient. Water used in the cooling system, once too warm to be recycled, is fed into the effluent treatment facility. By making the cooling system more efficient, less volume of water in total will be used and ultimately less water will be transferred to the effluent treatment facility. The current cooling systems are once-through services with no segregation from the main effluent stream.

The water reduction as a result of the anticipated future cooling towers will be considered under the cumulative effects assessment in the EA.

APPENDIX H-23

Westhaver, Erna B

From: Martin, Frances R
Sent: Tuesday, November 14, 2017 9:01 PM
To: Keats, Paul J
Subject: Fwd: Phone call November 14
Attachments: image002.png

Sent from my iPhone

Begin forwarded message:

From: "Chapman, Bruce" [redacted]@northernpulp.com> 20(1)
Date: November 14, 2017 at 6:36:00 PM AST
To: "Martin, Frances R" <Frances.Martin@novascotia.ca>
Cc: "Fraser, Terri" [redacted]@northernpulp.com>
Subject: Phone call November 14 20(1)

During our telephone call today, you stated that your department is working on a response on my letter of November 3. I would like to clarify a few points:

Receiving Water Study and ETP Design
Your department has reviewed the Receiving Water Study (RWS) and have indicated that ENS is generally supportive of the concentrations used as inputs for the RWS and that these concentrations can be used as a basis for detailed design of the treatment plant. I would like to explain how those concentration inputs were derived. For normally regulated discharge limits (BOD, TSS and AOX), our consultants used the monthly limits as outlined in the Quebec regulations for existing pulp and paper mills. The Quebec regulations were reviewed in 2016. For effluent parameters that are generally not regulated, the consultants used the expected concentrations from the preliminary design. To recognize that there are daily variations in the operation, the Quebec regulations and most other regulations in Canada use the monthly limit and a multiplier to set daily limits.
Understanding that the EA outcome will be used to set the actual limits for a future IA, please confirm that the limits will allow for variations in daily operation as is the case in most regulations in Canada.

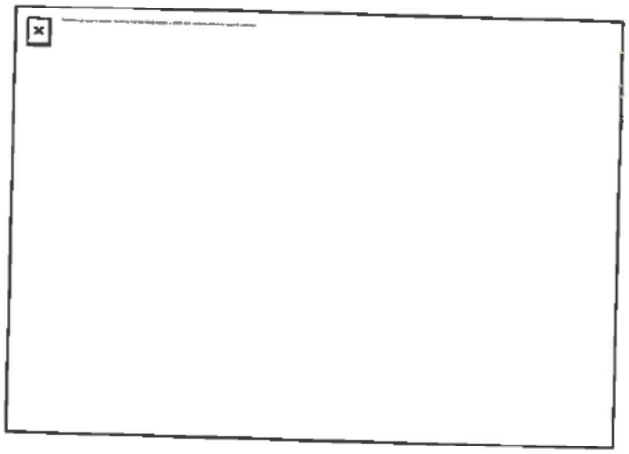
COD
We do not understand NSE's position on COD as outlined in the letter of October 5th. COD is normally not a regulated limit in North America. BOD is normally used to regulate oxygen demand in North America. Can you explain why COD is listed separately in the letter and why the concentrations listed do not match with those used in the RWS?

Water
NPNS understands that the we must continue to meet the current maximum daily water intake as outlined in the letter of October 5th.

Renewal of the Industrial Approval
As we discussed, regulatory certainty is important to the executives and the owners of PEC as the province and NPNS move forward on the Effluent Treatment Plant project. As such, NPNS would prefer for discussions on a future IA to begin as soon as possible. It is understood that there are many items in

a future IA that will be determined by the EA process, so only items not associated with the EA will need to be discussed.

Is there a time tomorrow that you will be available for a short telephone call on these items?



APPENDIX H-24

January 22, 2018

Jill Scanlan

Northern Pulp Nova Scotia, Effluent Treatment Facility Replacement Project Update

Thank you for engaging with the Environmental Assessment (EA) study for Northern Pulp's replacement Effluent Treatment Facility. The feedback we receive during the EA study process will guide the environmental planning for the project.

A Class 1 EA process is being followed for this project. Nova Scotia Environment's (NSE) Environmental Assessment Branch determined that the project is a modification of an existing undertaking.

Engagement activities will continue to occur throughout the EA study. Our next steps are described below. At the completion of the EA study, the EA Registration Document is submitted to NSE for review. NSE's review process includes making the Registration Document available to the public and seeking public comment.

The proposed effluent treatment system is being designed to Pulp and Paper Effluent Regulations. Additional consideration of the Canadian Council of Ministers of Environment (CCME) Marine Guidelines and other national and international standards has been made. The design engineers, KSH Solutions with support from Stantec, used complex hydrodynamic models to develop the appropriate system to meet the guidelines. This replacement system is similar in design to many other North American and international Kraft pulp mills. KSH Solutions is a Canadian Engineering company with worldwide experience in the design of effluent treatment systems and has based their design on industry best practice to serve Northern Pulp's operations.

We are following up with key stakeholders and Pictou Landing First Nation for feedback on limitations and environmental conditions related to the outfall pipeline route and diffuser location.

The scientific modelling completed predicts there will be minimal impact on water quality in the Northumberland Strait, Pictou Harbour, Boat Harbour, and the near shore areas.

We have heard many questions related to the interaction of the treated effluent and the valued marine environment. Further studies to identify potential impacts and necessary mitigation measures will be completed.

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Responses to Your Questions

Open Hause Format

Thank you for sharing feedback on how you found the Open House session. The format with the display panels allows for many voices to be heard through small groups or one-on-one conversations, facilitates a large number of participants, and respects different needs in communication. We will continue to try to create sessions where many people can engage in meaningful dialogue and consider this and other feedback received as we move forward.

Potential for Marine Enviranmental Impacts

The proposed effluent treatment system is designed to meet federal Pulp and Paper Effluent Regulations, similar to the existing effluent treatment system. Additional, more stringent, guidelines were also considered to achieve best practice in the design and operation of pulp and paper effluent treatment facilities. The development of monitoring programs will be an outcome of the regulatory process for approval of the facility. Under the Pulp and Paper Regulations, an Environmental Effects Monitoring Program is required. A copy of the Cycle 7 EEM report for the current treatment facility can be found on the project website. Additional monitoring programs will be designed based on approvals requirements.

i.e. monitoring won't be figured out until after approval

The Receiving Water Study, available on the project website, reports the modeled performance of how the treated effluent will mix into the Northumberland Strait through the outfall (the six port diffuser) and the resulting predicted water quality. One pipe will carry treated effluent from the treatment facility, located on the mill property, through Pictou Harbour to the outfall location. Six 'diffusers', or engineered outlets, will be installed at the end of the pipe. The Receiving Water Study also discusses how the diffuser design was determined.

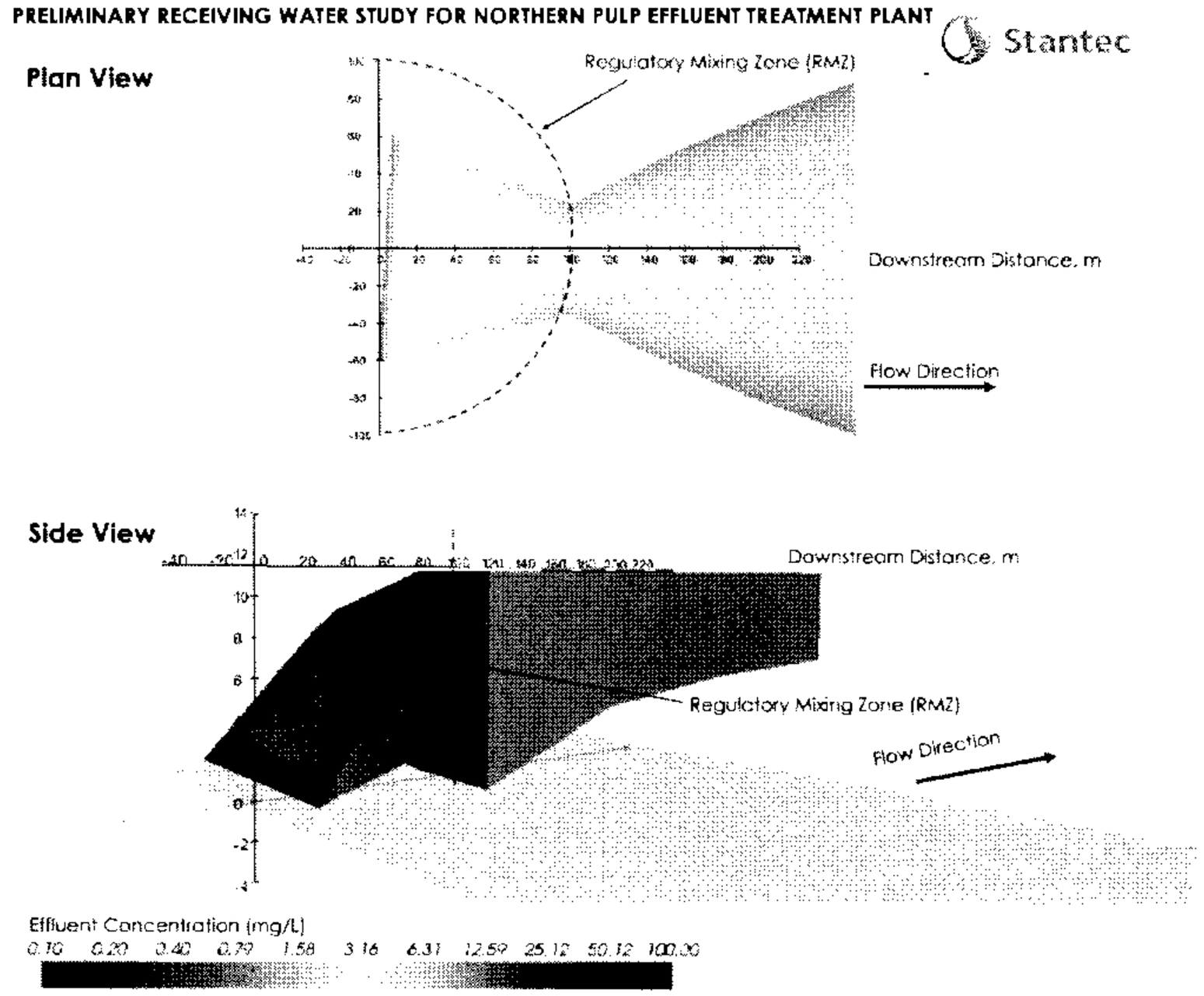
Available studies completed in the area will be reviewed, such as the Environmental Effects Monitoring of Northern Pulp, and the many studies being completed related to Boat Harbour.

The Mixing Zone

The water depth at the proposed location of the outfall pipe is 11.3 m (Mean Water Level). The mixing zone for the discharged effluent was defined as the lateral 100-m distance from the outfall following the Canadian Council of Ministers of the Environment (CCME) guidelines. To assess the impacts on the surrounding environment, be that lobsters at different life stages, or the plants on the bottom of the Strait which support the ecosystem, we must also understand where in the water column the mixing occurs. The effluent plume does not move upwards in a block, it expands from the diffusers upward but also laterally. Figure 3.7 of the Receiving Water Study, shown below, shows the shape of the effluent plume. The Receiving Water Study is available on the project website. The plume from the six port diffuser in the location chosen reaches the surface water at about 90 m from the diffuser. The dilution ratio in the receiving environment is 109 times at the end of the 100 m mixing zone as measured laterally from the point of discharge. Colour, for example, is

predicted to reach background levels within 40 m of the diffuser ports. At 40 m laterally from the diffuser, the effluent plume is still more than 1 m below the surface of the water.

Figure 3-7. Effluent Plume Scenario 6 - Six Ports Diffuser at Alt-D



Potential for Sediment Accumulation at the Outfall

The effluent is a liquid. Within the liquid will be an amount of 'suspended solids', similar to how silt can be suspended in a river. The amount of total suspended solids (TSS) discharged in the effluent in 2017 was 1,563 kg/day. By volume, this represents approximately 25 mg/L or 0.002S%.

The federal Pulp and Paper Effluent Regulations (PPER) manage threats to fish, fish habitat and human health from fish consumption by governing the deposit of deleterious substances from pulp and paper mills into waters frequented by fish for the protection of aquatic life. PPER specify a discharge limit of 11,500 kg/day for NPNS. The current discharge is significantly under the limit as significant improvements have been made over the years. The new ETF anticipates TSS discharges to remain low, in the range of 25 - 35 mg/L or 0.0025 - 0.0035%.

The solids which are present in the treated effluent will be very small particles left from the wood, and organics from the microorganisms used in the treatment process and are 98% biodegradable.

what are remaining 2%?

The Environmental Assessment (EA) will evaluate expected long term implications and potential environmental impacts related to the proposed replacement Effluent Treatment Facility. The replacement treatment facility design will incorporate the components needed to address regulations established with consideration of the longer term implications. As well, Environmental Effects Monitoring (EEM), which is a science-based performance measurement tool used to evaluate the adequacy of effluent regulations, will use baseline information to determine if any effects occur as a result of the TSS from the treated effluent discharge and to measure those effects, should they occur.

for where?

what about remediation?

Air Quality: Burning the Sludge

Being able to burn the sludge in the power boiler is the preferred approach as it increases power generation from green energy sources at Northern Pulp.

The dewatered solids include clay, sand, silt, organic matter, nutrients, microorganisms and metals. Sludge is typically either burned for fuel at treatment facilities, or used as soil or a soil additive as in all other Canadian provinces as well as in the USA, Europe, South America, Africa and Asia.

Changes to air quality will be assessed during the Environmental Assessment and will include alternative uses for the sludge.

Unrelated to the effluent treatment, Northern Pulp is separately addressing air quality concerns associated with its operations and working closely with the provincial government and regulatory authorities to continue to improve air quality.

Sharing Information

Results of specialist studies completed as part of the EA study will be made available upon their completion on the project website:
www.northernpulpeffluenttreatmentfacility.ca.

The study which looked at the initial siting of the outfall location and diffuser performance, and the study of treatment facility alternatives are posted on the project website now. Environmental monitoring (EEM) reporting of the existing Boat Harbour facility is also provided. The EEM is not directly applicable to the proposed replacement effluent treatment facility and the associated treatment effluent.

A "Frequently Asked Questions" list has also been added to the project website. Additions will be made to it as the project evolves.

Next Steps

Over the coming months, the project team will be continuing conversations with First Nations and key stakeholders, and completing environmental and engineering studies. Environmental plans to protect and/or mitigate potential impacts will be developed based on this information and input and dialogue.

In Spring 2018 we will return to the community with another series of Open House sessions which will provide answers to questions raised during the initial phase of engagement and present the recommended environmental plans and seek feedback.

If you have further questions or comments, please contact the project team at npns.effluenttreatmentfacility@dillon.ca, 1-877-635-8553 x 5050 and/or visit the project website www.northernpulpeffluenttreatmentfacility.ca.

Again, thank you for your time and interest. We look forward to continued engagement with you on this important project.

Sincerely,

DILLON CONSULTING LIMITED

Annamarie Burgess, LPP, P.Eng.
Project Coordinator
Northern Pulp Effluent Treatment Facility Environmental Assessment
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APPENDIX H-25

July 9, 2018

Jill Scanlan
Jerd.scanlan@ns.sympatico.ca

Northern Pulp Nova Scotia, Effluent Treatment Facility Replacement Project Update

Thank you for continuing to engage with the Environmental Assessment (EA) study for Northern Pulp's replacement Effluent Treatment Facility. The feedback we receive during the EA study process is important and will continue to guide the environmental planning for the project.

Responses to Your Questions

On page 2.22 of Stantec's Receiving Water Study, it is stated: "Characteristics of the expected treated effluent from the NPNS mill for the new wastewater treatment plant were provided by KSH (KSH, 2016) as summarized in Table 2-6."
How did KSH determine these characteristics?
KSH determined the effluent characteristics identified in Table 2-6 from an evaluation and benchmarking of the NPNS operations and the predicted effluent quality that will result from changing from the ASB (Aerated Stabilization Basin) process to the AST (Activated Sludge Treatment) process for effluent treatment.

Are these characteristics expected after the oxygen delignification system is installed, or prior to its installation?
The results of the Receiving Water Study (RWS) do not account for the improvements that the Oxygen Delignification system will provide.

The RWS was conducted without considering the benefits of the Oxygen Delignification system. This way, the EA is assessing a conservative or 'worst case' scenario. A brief description of Oxygen Delignification and its environmental benefits are available on the project website*(*www.northernpulpfuture.ca*, Project overview drop down menu/in-mill improvements tab)*.

Will the oxygen delignification system be installed and operating when the AST system is in place, or is the plan to install the oxygen delignification sometime in the future, after the AST system is in place? If the oxygen delignification will not be installed at the same time as the AST system is in place, when will the oxygen delignification be installed?
The Oxygen Delignification Project will start-up after the new AST system is operational.

Sharing Information
In fall 2018 we will return to the community with another Open House session which will provide answers to questions raised during the initial phase of engagement and

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902.450.2008

present the recommended environmental plans. Indigenous community and stakeholder engagement has also been ongoing through this phase of the project.

If you have further questions or comments, please contact the project team at npns.effluenttreatmentfacility@dillon.ca, 1-877-635-8553 x 5050 and/or visit the project website www.NorthernPulpFuture.ca. The website is updated as new information becomes available.

Again, thank you for your time and interest. We look forward to continued engagement with you on this important project.

Sincerely,

DILLON CONSULTING LIMITED

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APPENDIX H-26

Project Materials

Specialist Studies and Engagement Materials

Specialist Studies

Addendum Receiving Water Study for Outfall Location at Caribou Point

- During preliminary design, a discharge location for the marine outfall was selected in the Northumberland Strait as a preferred option. After subsequent marine geophysical and geotechnical field investigations, it was determined this outfall was not technically feasible. This addendum to the Receiving Water Study was completed to investigate two alternative outfall locations off of Caribou Point.

Brochure with information on the New Effluent Treatment Facility, Northern Pulp

- Brochure providing additional details on the new effluent treatment facility.

NPNS Global Market Study, Brian McClay & Associates Inc.

- The NPNS Global Market Study assesses the viability of converting the existing Pictou Northern Bleached Softwood Kraft (NBSK) mill to produce either Unbleached Kraft Pulp (UKP) or Bleached Chemi-Thermo-Mechanical Pulp (BCTMP). For the reasons outlined in the market report, it can be concluded that continuing to produce premium reinforcement NBSK is the most competitively viable option by far for Northern Pulp.

Receiving Water Study, Stantec Consulting Ltd.

- The Receiving Water Study was completed during preliminary design to (1) evaluate potential locations for a marine outfall and identified the recommended area, (2) evaluate and made recommendations for the design and performance of the diffuser at the end of the outfall, and (3) model how the treated effluent will mix with the water

at the end of the outfall, and (3) model how the treated effluent will mix with the water at the outlet in the Northumberland Strait.

2016 EEM Report, ECOMETRIX

- The Environmental Effects Monitoring (EEM) study gives the results of the environmental effects monitoring from the existing Boat Harbour Treatment Facility. This EEM study is not directly applicable to the proposed replacement effluent treatment facility and associated treated effluent, but was provided upon request.

Technology Selection Summary Report, KSH

- This report documents the Preliminary Engineering for which reviewed the technology alternatives when determining the approach for the treatment facility at Northern Pulp.

Middle River Water Availability Report

- Completed in 2015 by RV Anderson for the Government of Nova Scotia, this report reviewed the sustainability of the water intake used by Northern Pulp.

Engagement Materials

Materials used at project engagement meetings, and summaries of those meetings will be posted here. This way we will increase the transparency of our process, and allow as many people as possible to engage with the project.

Project Launch: Summary of Engagement - What We Heard

Project Launch Open House Materials (December 2017 & January 2018)

Project Launch: Initiation Newsletter

APPENDIX H-27

copied from NPRI - March 7, 2019

Company/Facility information: Northern Pulp Nova Scotia Corporation/Northern Pulp Nova Scotia Corporation (2017)

Information for Northern Pulp Nova Scotia Corporation

Company	Northern Pulp Nova Scotia Corporation
Facility	Northern Pulp Nova Scotia Corporation
NPRI ID	815
Address	260 Abercrombie Branch Road New Glasgow, NS B2H 5E8 Canada

(1) NOTE: as of the 2006 reporting year, the Disposal columns include information on tailings and waste rock disposals. Negative numbers are possible for on-site disposal of tailings and waste rock, which would reflect a net removal of the substances from the tailings or waste rock management area.

(2) NOTE: Off-site column under Disposal in this table includes 'Off-site Disposal' and 'Off-Site Treatment Prior to Final Disposal'

Acenaphthene (83-32-9)

Historical reports for Acenaphthene (83-32-9)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	224	-	-	224	-	-	-	kg
2016	216	-	-	216	-	-	-	kg
2015	208	-	-	208	-	-	-	kg
2014	227	-	-	227	0	-	-	kg
2013	227	-	-	227	-	-	-	kg
2012	224	-	-	224	-	-	-	kg
2011	202	-	-	202	-	-	-	kg
2010	230	-	-	230	-	-	-	kg
2009	200	-	-	200	-	-	-	kg
2008	209	-	-	209	-	-	-	kg
2007	214	-	-	214	-	-	-	kg
2006	214	-	-	214	-	-	-	kg

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Acenaphthylene (208-96-8)

Historical reports for Acenaphthylene (208-96-8)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	2,741	-	-	2,741	-	-	-	kg
2016	2,645	-	-	2,645	-	-	-	kg
2015	2,542	-	-	2,542	-	-	-	kg
2014	2,776	-	-	2,776	0	-	-	kg
2013	2,776	-	-	2,776	-	-	-	kg
2012	2,741	-	-	2,741	-	-	-	kg
2011	2,472	-	-	2,472	-	-	-	kg
2010	2,804	-	-	2,804	-	-	-	kg
2009	2,443	-	-	2,443	-	-	-	kg
2008	2,553	-	-	2,553	-	-	-	kg
2007	2,613	-	-	2,613	-	-	-	kg
2006	2,613	-	-	2,613	-	-	-	kg

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Acetaldehyde (75-07-0)

Historical reports for Acetaldehyde (75-07-0)

Year	On-Site Releases	Disposal(1)	Off-Site	Units

Year	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	Units
2017	21	0.03	-	21	-	-	-	tonnes
2016	21	0.032	-	21	-	-	-	tonnes
2015	21	0.03	-	21	-	-	-	tonnes
2014	23	0.033	-	23	0	-	-	tonnes
2013	24	0.034	-	24	-	-	-	tonnes
2012	24	0.031	-	24	-	-	-	tonnes
2011	21	0.03	-	21	-	-	-	tonnes
2010	24	0.035	-	24	-	-	-	tonnes
2009	21	0.037	-	21	-	-	-	tonnes
2008	23	0.045	-	23	-	-	-	tonnes
2007	25	0.035	-	25	-	-	-	tonnes
2006	21	0.035	-	21	-	-	-	tonnes
2005	18	0.268	-	19	0.005	-	-	tonnes
2004	18	0.539	-	19	0.005	-	-	tonnes
2003	18	0.529	-	18	0.005	-	-	tonnes
2002	18	0.61	-	19	0.005	-	-	tonnes
2001	18	0.54	-	18	0.005	-	-	tonnes
2000	17	0.57	-	18	0.005	-	-	tonnes
1999	13	0.57	-	13	0.005	-	-	tonnes

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Ammonia (total) (NA - 16)

Historical reports for Ammonia (total) (NA - 16)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	37	-	-	37	-	-	-	tonnes
2016	36	-	-	36	-	-	-	tonnes
2015	37	-	-	37	-	-	-	tonnes
2014	42	-	-	42	0	-	-	tonnes
2013	48	-	-	48	-	-	-	tonnes
2012	50	-	-	50	-	-	-	tonnes
2011	42	-	-	42	-	-	-	tonnes
2010	46	-	-	46	-	-	-	tonnes
2009	42	-	-	42	-	-	-	tonnes
2008	50	-	-	50	-	-	-	tonnes
2007	41	-	-	41	-	-	-	tonnes
2006	40	-	-	40	-	-	-	tonnes
2005	49	-	-	49	-	-	-	tonnes
2004	49	-	-	49	-	-	-	tonnes
2003	48	-	-	48	-	-	-	tonnes
2002	47	-	-	47	-	-	-	tonnes
2001	46	-	-	46	-	-	-	tonnes
2000	44	-	-	44	-	-	-	tonnes

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Benzo(a)anthracene (56-55-3)

Historical reports for Benzo(a)anthracene (56-55-3)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	9.7	-	-	9.7	1.9	-	-	kg
2016	9.4	-	-	9.4	1.7	-	-	kg
2015	9	-	-	9	0.719	-	-	kg
2014	9.8	-	-	9.8	0.797	-	-	kg
2013	9.9	-	-	9.9	0.753	-	-	kg
2012	9.7	-	0	9.7	0.753	-	-	kg
2011	8.8	-	-	8.8	0.694	-	-	kg
2010	10	-	-	10	0.665	-	-	kg
2009	8.7	-	-	8.7	0.665	-	-	kg
2008	9.1	-	-	9.1	0.434	-	-	kg
2007	9.3	-	-	9.3	0.68	-	-	kg
2006	9.3	-	-	9.3	0.68	-	-	kg
2005	6.4	-	-	6.4	0.616	-	-	kg
2004	8.9	-	-	8.9	0.301	-	-	kg
2003	8.6	-	-	8.6	0.311	-	-	kg
2002	8.3	-	-	8.3	0.26	-	-	kg
2001	8.1	-	-	8.1	0.666	-	-	kg
2000	8.1	-	-	8.1	0.65	-	-	kg

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Benzo(a)phenanthrene (218-01-9)

Historical reports for Benzo(a)phenanthrene (218-01-9)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	6.7	-	-	6.7	0.122	-	-	kg
2016	6.5	-	-	6.5	0.117	-	-	kg
2015	6.2	-	-	6.2	0.063	-	-	kg
2014	6.8	-	-	6.8	0.142	-	-	kg
2013	6.8	-	-	6.8	0.142	-	-	kg
2012	6.7	-	-	6.7	0.142	-	-	kg
2011	6	-	-	6	0.13	-	-	kg
2010	6.9	-	-	6.9	0.125	-	-	kg
2009	6	-	-	6	0.125	-	-	kg
2008	6.2	-	-	6.2	0.059	-	-	kg
2007	6.4	-	-	6.4	0.052	-	-	kg
2006	6.4	-	-	6.4	0.052	-	-	kg
2005	6.1	-	-	6.1	0.047	-	-	kg
2004	6.2	-	-	6.2	0.172	-	-	kg
2003	6	-	-	6	0.178	-	-	kg
2002	5.8	-	-	5.8	0.15	-	-	kg
2001	5.6	-	-	5.6	0.381	-	-	kg
2000	5.6	-	-	5.6	0.37	-	-	kg

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Benzo(a)pyrene (50-32-8)

Historical reports for Benzo(a)pyrene (50-32-8)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2006	3	-	-	3	0.657	-	-	kg
2005	0.582	-	-	0.582	0.595	-	-	kg
2004	0.584	-	-	0.584	1.6	-	-	kg
2003	0.565	-	-	0.565	1.6	-	-	kg
2002	0.55	-	-	0.55	1.3	-	-	kg
2001	0.53	-	-	0.53	3.5	-	-	kg
2000	0.53	-	-	0.53	3.4	-	-	kg

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Benzo(b)fluoranthene (205-99-2)

Historical reports for Benzo(b)fluoranthene (205-99-2)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2006	2.7	-	-	2.7	0.87	-	-	kg
2005	2.5	-	-	2.5	0.591	-	-	kg
2004	2.6	-	-	2.6	0.285	-	-	kg
2003	2.5	-	-	2.5	0.295	-	-	kg
2002	2.4	-	-	2.4	0.24	-	-	kg
2001	2.4	-	-	2.4	0.632	-	-	kg
2000	2.4	-	-	2.4	0.62	-	-	kg

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Benzo(e)pyrene (192-97-2)

Historical reports for Benzo(e)pyrene (192-97-2)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2006	0.127	-	-	0.127	-	-	-	kg

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Benzo(g,h,i)perylene (191-24-2)

Historical reports for Benzo(g,h,i)perylene (191-24-2)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2006	1.1	-	-	1.1	0.665	-	-	kg
2005	0.993	-	-	0.993	0.603	-	-	kg
2004	1	-	-	1	0.289	-	-	kg
2003	0.968	-	-	0.968	0.299	-	-	kg
2002	0.94	-	-	0.94	0.25	-	-	kg
2001	0.9	-	-	0.9	0.64	-	-	kg
2000	0.9	-	-	0.9	0.62	-	-	kg

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Benzo(j)fluoranthene (205-82-3)

Historical reports for Benzo(j)fluoranthene (205-82-3)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2006	0.148	-	-	0.148	-	-	-	kg

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Benzo(k)fluoranthene (207-08-9)

Historical reports for Benzo(k)fluoranthene (207-08-9)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2006	0.937	-	-	0.937	0.627	-	-	kg
2005	0.864	-	-	0.864	0.569	-	-	kg
2004	0.871	-	-	0.871	0.258	-	-	kg
2003	0.843	-	-	0.843	0.267	-	-	kg
2002	0.81	-	-	0.81	0.22	-	-	kg
2001	0.8	-	-	0.8	0.571	-	-	kg
2000	0.79	-	-	0.79	0.56	-	-	kg

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Carbon monoxide (630-08-0)

Historical reports for Carbon monoxide (630-08-0)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	2,498	-	-	2,498	-	-	-	tonnes
2016	787	-	-	787	-	-	-	tonnes
2015	831	-	-	831	-	-	-	tonnes
2014	987	-	-	987	-	-	-	tonnes
2013	890	-	-	890	-	-	-	tonnes
2012	878	-	-	878	-	-	-	tonnes
2011	829	-	-	829	-	-	-	tonnes
2010	800	-	-	800	-	-	-	tonnes
2009	897	-	-	897	-	-	-	tonnes
2008	824	-	-	824	-	-	-	tonnes
2007	3,816	-	-	3,816	-	-	-	tonnes
2006	3,796	-	-	3,796	-	-	-	tonnes
2005	3,316	-	-	3,316	-	-	-	tonnes
2004	3,341	-	-	3,341	-	-	-	tonnes
2003	3,321	-	-	3,321	-	-	-	tonnes
2002	3,291	-	-	3,291	-	-	-	tonnes

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Carbonyl sulphide (463-58-1)

Historical reports for Carbonyl sulphide (463-58-1)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2008	15	-	-	15	-	-	-	tonnes
2007	15	-	-	15	-	-	-	tonnes

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Chlorine (7782-50-5)

Historical reports for Chlorine (7782-50-5)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	2.9	-	-	2.9	-	-	-	tonnes
2016	2.9	-	-	2.9	-	-	-	tonnes
2015	3.1	-	-	3.1	-	-	-	tonnes
2014	3.2	-	-	3.2	0	-	-	tonnes
2013	3.6	-	-	3.6	-	-	-	tonnes
2012	3.6	-	-	3.6	-	-	-	tonnes
2011	3.9	-	-	3.9	-	-	-	tonnes

2010	3.9	-	-	3.9	-	-	-	tonnes
2009	3.5	-	-	3.5	-	-	-	tonnes
2008	3.6	-	-	3.6	-	-	-	tonnes
2007	3.6	-	-	3.6	-	-	-	tonnes
2006	3.9	-	-	3.9	-	-	-	tonnes
2005	3.8	-	-	3.8	-	-	-	tonnes
2004	3.8	-	-	3.8	-	-	-	tonnes
2003	3.3	-	-	3.3	-	-	-	tonnes
2002	3.4	-	-	3.4	-	-	-	tonnes
2001	3.5	-	-	3.5	-	-	-	tonnes
2000	3.7	-	-	3.7	-	-	-	tonnes
1999	2.6	-	-	2.6	-	-	-	tonnes
1998	15	-	-	15	-	-	-	tonnes
1997	47	-	-	47	-	-	-	tonnes
1996	50	-	-	50	-	-	-	tonnes
1995	78	-	-	78	-	-	-	tonnes
1994	97	-	-	97	-	-	-	tonnes
1993	123	0	0	123	0	-	0	tonnes

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Chlorine dioxide (10049-04-4)

Historical reports for Chlorine dioxide (10049-04-4)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	

2017		77	-	-		77	-	-	-	tonnes
2016		23	-	-		23	-	-	-	tonnes
2015		0	-	-		0	-	-	-	tonnes
2014	-	-	-	-		0	0	-	-	tonnes
2013	-	-	-	-		0	-	-	-	tonnes
2012		0	-	-		0	-	-	-	tonnes
2011		0	-	-		0	-	-	-	tonnes
2010		0	-	-		0	-	-	-	tonnes
2009		0	-	-		0	-	-	-	tonnes
2008		0	-	-		0	-	-	-	tonnes
2007		0	-	-		0	-	-	-	tonnes
2006		0	-	-		0	-	-	-	tonnes
2005		0	-	-		0	-	-	-	tonnes
2004		0	-	-		0	-	-	-	tonnes
2003		0	-	-		0	-	-	-	tonnes
2002		0	-	-		0	-	-	-	tonnes
2001		0	-	-		0	-	-	-	tonnes
2000		0	-	-		0	-	-	-	tonnes
1999		0	-	-		0	-	-	-	tonnes
1998		12	-	-		12	-	-	-	tonnes
1997		63	-	-		63	-	-	-	tonnes
1996		63	-	-		63	-	-	-	tonnes
1995		50	-	-		50	-	-	-	tonnes
1994		67	-	-		67	-	-	-	tonnes
1993		62		0	0	62		0 -	0	tonnes

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Chloromethane (74-87-3)

Historical reports for Chloromethane (74-87-3)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	16	0.04	-	16	-	-	-	tonnes
2016	15	0.05	-	15	-	-	-	tonnes
2015	15	0.04	-	15	-	-	-	tonnes
2014	16	0.05	-	16	0	-	-	tonnes
2013	16	0.05	-	16	-	-	-	tonnes
2012	16	0.05	-	16	-	-	-	tonnes
2011	15	0.05	-	15	-	-	-	tonnes
2010	17	0.06	-	17	-	-	-	tonnes
2009	15	0.07	-	15	-	-	-	tonnes
2008	16	0.07	-	16	-	-	-	tonnes
2007	15	0.03	-	15	-	-	-	tonnes
2006	15	0.03	-	15	-	-	-	tonnes
2005	15	0.03	-	15	-	-	-	tonnes
2004	15	0.03	-	15	-	-	-	tonnes
2003	14	0.03	-	14	-	-	-	tonnes
2002	14	0.028	-	14	-	-	-	tonnes
2001	14	0.029	-	14	-	-	-	tonnes
2000	13	0.25	-	14	-	-	-	tonnes

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Dibenzo(a,h)anthracene (53-70-3)

Historical reports for Dibenzo(a,h)anthracene (53-70-3)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2006	0.613	-	-	0.613	0.663	-	-	kg
2005	0.579	-	-	0.579	0.601	-	-	kg
2004	0.583	-	-	0.583	0.287	-	-	kg
2003	0.565	-	-	0.565	0.297	-	-	kg
2002	0.54	-	-	0.54	0.25	-	-	kg
2001	0.55	-	-	0.55	0.636	-	-	kg
2000	0.53	-	-	0.53	0.62	-	-	kg

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Dioxins and furans - total (NA - D/F)

Historical reports for Dioxins and furans - total (NA - D/F)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	0.008	-	-	0.008	-	-	-	g TEQ(ET)
2016	0.008	-	-	0.008	-	-	-	g TEQ(ET)
2015	0.008	-	-	0.008	-	-	-	g TEQ(ET)
2014	0.008	-	-	0.008	0	-	-	g TEQ(ET)

2013		0.008	-	-	0.008	-	-	g TEQ(ET)
2012		0.008	-	-	0.008	-	-	g TEQ(ET)
2011		0.008	-	-	0.008	-	-	g TEQ(ET)
2010	-	-	-	-	0	-	-	g TEQ(ET)
2009	-	-	-	-	0	-	-	g TEQ(ET)
2008	-	-	-	-	0	-	-	g TEQ(ET)
2007	-	-	-	-	0	-	-	g TEQ(ET)
2006		0.008	-	-	0.008	-	-	g TEQ(ET)
2005		0.009	-	-	0.009	-	-	g TEQ(ET)
2004		0.012	-	-	0.012	-	-	g TEQ(ET)
2003		0.012	-	-	0.012	-	-	g TEQ(ET)
2002		0.011	-	-	0.011	-	-	g TEQ(ET)
2001		0.012	0.538	-	0.55	-	-	g TEQ(ET)

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Fluoranthene (206-44-0)

Historical reports for Fluoranthene (206-44-0)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	95	-	-	95	1.7	-	-	kg
2016	91	-	-	91	1.6	-	-	kg
2015	88	-	-	88	0.722	-	-	kg
2014	96	-	-	96	1.1	-	-	kg
2013	96	-	-	96	1.1	-	-	kg

2012	95	-	-	95	1.1	-	-	kg
2011	86	-	-	86	1	-	-	kg
2010	97	-	-	97	0.973	-	-	kg
2009	85	-	-	85	0.947	-	-	kg
2008	88	-	-	88	0.53	-	-	kg
2007	91	-	-	91	0.648	-	-	kg
2006	91	-	-	91	0.648	-	-	kg
2005	85	-	-	85	0.588	-	-	kg
2004	86	-	-	86	0.43	-	-	kg
2003	84	-	-	84	0.444	-	-	kg
2002	81	-	-	81	0.37	-	-	kg
2001	79	-	-	79	0.952	-	-	kg
2000	78	-	-	78	0.93	-	-	kg

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Fluorene (86-73-7)

Historical reports for Fluorene (86-73-7)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	21	-	-	21	-	-	-	kg
2016	20	-	-	20	-	-	-	kg
2006	20	-	-	20	-	-	-	kg

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Fluorine (7782-41-4)

Historical reports for Fluorine (7782-41-4)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2015	19	-	-	19	-	-	-	tonnes
2014	21	-	-	21	0	-	-	tonnes
2013	21	-	-	21	-	-	-	tonnes
2012	21	-	-	21	-	-	-	tonnes
2011	19	-	-	19	-	-	-	tonnes

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Formaldehyde (50-00-0)

Historical reports for Formaldehyde (50-00-0)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	12	-	-	12	-	-	-	tonnes
2016	12	-	-	12	-	-	-	tonnes
2015	12	-	-	12	-	-	-	tonnes
2014	12	-	-	12	0	-	-	tonnes

2013	12	-	-	12	-	-	tonnes
2012	12	-	-	12	-	-	tonnes
2011	12	-	-	12	-	-	tonnes
2010	12	-	-	12	-	-	tonnes
2009	12	-	-	12	-	-	tonnes
2008	12	-	-	12	-	-	tonnes

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Hexachlorobenzene (118-74-1)

Historical reports for Hexachlorobenzene (118-74-1)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	1.6	-	-	1.6	-	-	-	grams
2016	1.5	-	-	1.5	-	-	-	grams
2015	1.5	-	-	1.5	-	-	-	grams
2014	1.5	-	-	1.5	0	-	-	grams
2013	1.6	-	-	1.6	-	-	-	grams
2012	1.6	-	-	1.6	-	-	-	grams
2011	1.4	-	-	1.4	-	-	-	grams
2010	1.6	-	-	1.6	-	-	-	grams
2009	1.4	-	-	1.4	-	-	-	grams
2008	1.5	-	-	1.5	-	-	-	grams
2007	1.4	-	-	1.4	-	-	-	grams
2006	1.4	-	-	1.4	-	-	-	grams

2005	-	-	-	0	-	-	grams
2004	-	-	-	0	-	-	grams
2003	-	-	-	0	-	-	grams
2002	-	-	-	0	-	-	grams
2001	-	-	-	0	-	-	grams
2000	-	-	-	0	-	-	grams

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Hydrochloric acid (7647-01-0)

Historical reports for Hydrochloric acid (7647-01-0)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	3.8	-	-	3.8	-	-	-	tonnes
2016	3.8	-	-	3.8	-	-	-	tonnes
2015	3.7	-	-	3.7	-	-	-	tonnes
2014	3.7	-	-	3.7	0	-	-	tonnes
2013	3.8	-	-	3.8	-	-	-	tonnes
2012	3.8	-	-	3.8	-	-	-	tonnes
2011	3.5	-	-	3.5	-	-	-	tonnes
2010	3.9	-	-	3.9	-	-	-	tonnes
2009	3.3	-	-	3.3	-	-	-	tonnes
2008	3.4	-	-	3.4	-	-	-	tonnes
2007	3.7	-	-	3.7	-	-	-	tonnes
2006	3.6	-	-	3.6	-	-	-	tonnes

2005	3.5	-	-	3.5	-	-	-	tonnes
2004	3.6	-	-	3.6	-	-	-	tonnes
2003	3.5	-	-	3.5	-	-	-	tonnes
2002	3.6	-	-	3.6	-	-	-	tonnes
2001	3.5	-	-	3.5	-	-	-	tonnes
2000	3.5	-	-	3.5	-	-	-	tonnes
1999	2.4	-	-	2.4	-	-	-	tonnes
1998	2.9	-	-	2.9	-	-	-	tonnes
1997	2.7	-	-	2.7	-	-	-	tonnes
1996	2.7	-	-	2.7	-	-	-	tonnes
1995	3.5	-	-	3.5	-	-	-	tonnes
1994	6.8	-	-	6.8	-	-	-	tonnes
1993	2.7	0	0	2.7	0	-	0	tonnes

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Hydrogen sulphide (7783-06-4)

Historical reports for Hydrogen sulphide (7783-06-4)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	24	-	-	24	-	-	-	tonnes
2016	23	-	-	23	-	-	-	tonnes
2015	21	-	-	21	-	-	-	tonnes
2014	27	0.41	-	27	0	-	-	tonnes
2013	35	0.42	-	35	-	-	-	tonnes

2012	23	0.39	-	24	-	-	-	tonnes
2011	21	0.4	-	21	-	-	-	tonnes
2010	24	0.48	-	24	-	-	-	tonnes
2009	21	0.58	-	21	-	-	-	tonnes
2008	52	0.61	-	52	-	-	-	tonnes
2007	60	0.53	-	61	-	-	-	tonnes
2006	32	0.54	-	32	-	-	-	tonnes
2005	31	0.58	-	31	-	-	-	tonnes
2004	58	0.53	-	58	-	-	-	tonnes
2003	56	0.49	-	56	-	-	-	tonnes
2002	55	0.5	-	55	-	-	-	tonnes
2001	53	0.43	-	54	-	-	-	tonnes
2000	52	0.43	-	53	-	-	-	tonnes
1999	176	0.43	-	176	-	-	-	tonnes

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Indeno(1,2,3-c,d)pyrene (193-39-5)

Historical reports for Indeno(1,2,3-c,d)pyrene (193-39-5)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2006	0.486	-	-	0.486	0.029	-	-	kg
2005	0.39	-	-	0.39	0.027	-	-	kg
2004	0.39	-	-	0.39	0.153	-	-	kg
2003	0.378	-	-	0.378	0.158	-	-	kg

2002	0.36	-	-	0.36	0.13	-	-	kg
2001	0.35	-	-	0.35	0.339	-	-	kg
2000	0.35	-	-	0.35	0.33	-	-	kg

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Methanol (67-56-1)

Historical reports for Methanol (67-56-1)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	69	-	-	69	-	-	-	tonnes
2016	69	-	-	69	-	-	-	tonnes
2015	98	-	-	98	-	-	-	tonnes
2014	218	-	-	218	0	-	-	tonnes
2013	381	-	-	381	-	-	-	tonnes
2012	334	-	-	334	-	-	-	tonnes
2011	268	-	-	268	-	-	-	tonnes
2010	220	-	-	220	-	-	-	tonnes
2009	217	0	-	217	-	-	-	tonnes
2008	228	0	-	228	-	-	-	tonnes
2007	472	0	-	472	-	-	-	tonnes
2006	168	0	-	168	-	-	-	tonnes
2005	173	0	-	173	-	-	-	tonnes
2004	177	0	-	177	-	-	-	tonnes
2003	172	0	-	172	-	-	-	tonnes

2002	172	0	-	172	-	-	-	tonnes
2001	169	0	-	169	-	-	-	tonnes
2000	110	0	-	110	-	-	-	tonnes
1999	36	-	-	36	-	-	-	tonnes
1998	42	-	-	42	-	-	-	tonnes
1997	213	44	-	258	-	-	-	tonnes
1996	220	43	-	263	-	-	-	tonnes
1995	227	46	-	272	-	-	-	tonnes
1994	230	-	-	230	-	2,667	-	tonnes
1993	217	0	0	217	0	1,953	0	tonnes

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Nitrate ion (NA - 17)

Historical reports for Nitrate ion (NA - 17)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2015	-	12	-	12	-	-	-	tonnes
2014	-	13	-	13	0	-	-	tonnes
2013	-	13	-	13	-	-	-	tonnes
2012	-	0.53	-	0.53	-	-	-	tonnes
2011	-	0.34	-	0.34	-	-	-	tonnes
2010	-	32	-	32	-	-	-	tonnes
2009	-	41	-	41	-	-	-	tonnes
2008	-	43	-	43	-	-	-	tonnes

2007	-	37	-	37	-	-	tonnes
2006	-	38	-	38	-	-	tonnes
2005	-	75	-	75	-	-	tonnes
2004	-	126	-	126	-	-	tonnes
2003	-	174	-	174	-	-	tonnes
2002	-	72	-	72	-	-	tonnes
2001	-	44	-	44	-	-	tonnes
2000	-	28	-	28	-	-	tonnes

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Nitrogen oxides (expressed as nitrogen dioxide) (11104-93-1)

Historical reports for Nitrogen oxides (expressed as nitrogen dioxide) (11104-93-1)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	436	-	-	436	-	-	-	tonnes
2016	331	-	-	331	-	-	-	tonnes
2015	364	-	-	364	-	-	-	tonnes
2014	436	-	-	436	-	-	-	tonnes
2013	405	-	-	405	-	-	-	tonnes
2012	478	-	-	478	-	-	-	tonnes
2011	770	-	-	770	-	-	-	tonnes
2010	676	-	-	676	-	-	-	tonnes
2009	688	-	-	688	-	-	-	tonnes
2008	524	-	-	524	-	-	-	tonnes

2007	585	-	-	585	-	-	tonnes
2006	505	-	-	505	-	-	tonnes
2005	539	-	-	539	-	-	tonnes
2004	527	-	-	527	-	-	tonnes
2003	528	-	-	528	-	-	tonnes
2002	214	-	-	214	-	-	tonnes

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PAHs, total unspeciated (NA - P/H)

Historical reports for PAHs, total unspeciated (NA - P/H)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2006	-	1.1	-	1.1	-	-	-	kg

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Perylene (198-55-0)

Historical reports for Perylene (198-55-0)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		

2006	0.023	-	-	0.023	-	-	-	kg

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Phenanthrene (85-01-8)

Historical reports for Phenanthrene (85-01-8)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	647	-	-	647	5.8	-	-	kg
2016	629	-	-	629	5.5	-	-	kg
2015	600	-	-	600	2.5	-	-	kg
2014	654	-	-	654	3.9	-	-	kg
2013	655	-	-	655	3.8	-	-	kg
2012	647	-	-	647	3.8	-	-	kg
2011	583	-	-	583	3.5	-	-	kg
2010	661	-	-	661	3.4	-	-	kg
2009	577	-	-	577	3.4	-	-	kg
2008	602	-	-	602	1.8	-	-	kg
2007	617	-	-	617	2.3	-	-	kg
2006	617	-	-	617	2.3	-	-	kg
2005	584	-	-	584	2	-	-	kg
2004	590	-	-	590	1.9	-	-	kg
2003	571	-	-	571	1.9	-	-	kg
2002	551	-	-	551	1.6	-	-	kg
2001	538	-	-	538	4.1	-	-	kg

2000	536	-	-	536	4	-	-	kg

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Total particulate matter (NA - M08)

Historical reports for Total particulate matter (NA - M08)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	255	-	-	255	-	-	-	tonnes
2016	250	-	-	250	-	-	-	tonnes
2015	1,263	-	-	1,263	-	-	-	tonnes
2014	2,255	-	-	2,255	-	-	-	tonnes
2013	1,248	-	-	1,248	-	-	-	tonnes
2012	1,500	-	-	1,500	-	-	-	tonnes
2011	1,477	-	-	1,477	-	-	-	tonnes
2010	1,914	-	-	1,914	-	-	-	tonnes
2009	1,828	-	-	1,828	-	-	-	tonnes
2008	2,418	-	-	2,418	-	-	-	tonnes
2007	695	-	-	695	-	-	-	tonnes
2006	520	-	-	520	-	-	-	tonnes
2005	966	-	-	966	-	-	-	tonnes
2004	947	-	-	947	-	-	-	tonnes
2003	968	-	-	968	-	-	-	tonnes
2002	896	-	-	896	-	-	-	tonnes

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PM10 (NA - M09)

Historical reports for PM10 (NA - M09)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	149	-	-	149	-	-	-	tonnes
2016	127	-	-	127	-	-	-	tonnes
2015	1,210	-	-	1,210	-	-	-	tonnes
2014	1,823	-	-	1,823	-	-	-	tonnes
2013	1,023	-	-	1,023	-	-	-	tonnes
2012	1,294	-	-	1,294	-	-	-	tonnes
2011	1,241	-	-	1,241	-	-	-	tonnes
2010	1,280	-	-	1,280	-	-	-	tonnes
2009	1,603	-	-	1,603	-	-	-	tonnes
2008	2,128	-	-	2,128	-	-	-	tonnes
2007	586	-	-	586	-	-	-	tonnes
2006	425	-	-	425	-	-	-	tonnes
2005	940	-	-	940	-	-	-	tonnes
2004	947	-	-	947	-	-	-	tonnes
2003	941	-	-	941	-	-	-	tonnes
2002	870	-	-	870	-	-	-	tonnes

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PM2.5 (NA - M10)

Historical reports for PM2.5 (NA - M10)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	1.6	-	-	1.6	-	-	-	tonnes
2016	110	-	-	110	-	-	-	tonnes
2015	823	-	-	823	-	-	-	tonnes
2014	1,291	-	-	1,291	-	-	-	tonnes
2013	734	-	-	734	-	-	-	tonnes
2012	1,011	-	-	1,011	-	-	-	tonnes
2011	986	-	-	986	-	-	-	tonnes
2010	937	-	-	937	-	-	-	tonnes
2009	1,274	-	-	1,274	-	-	-	tonnes
2008	1,689	-	-	1,689	-	-	-	tonnes
2007	457	-	-	457	-	-	-	tonnes
2006	325	-	-	325	-	-	-	tonnes
2005	828	-	-	828	-	-	-	tonnes
2004	834	-	-	834	-	-	-	tonnes
2003	829	-	-	829	-	-	-	tonnes
2002	632	-	-	632	-	-	-	tonnes

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Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (NA - D/F)

Historical reports for Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (NA - D/F)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2000	0.006	-	-	0.006	-	-	-	g TEQ(ET)

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Pyrene (129-00-0)

Historical reports for Pyrene (129-00-0)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	56	-	-	56	1.7	-	-	kg
2016	53	-	-	53	1.6	-	-	kg
2015	51	-	-	51	0.767	-	-	kg
2014	56	-	-	56	1.3	-	-	kg
2013	56	-	-	56	1.3	-	-	kg
2012	56	-	-	56	1.3	-	-	kg
2011	50	-	-	50	1.2	-	-	kg
2010	57	-	-	57	1.1	-	-	kg
2009	50	-	-	50	1.1	-	-	kg
2008	52	-	-	52	0.587	-	-	kg
2007	53	-	-	53	0.68	-	-	kg

2006	53	-	-	53	0.68	-	-	kg
2005	48	-	-	48	0.616	-	-	kg
2004	48	-	-	48	0.481	-	-	kg
2003	47	-	-	47	0.498	-	-	kg
2002	45	-	-	45	0.41	-	-	kg
2001	44	-	-	44	1.1	-	-	kg
2000	44	-	-	44	1	-	-	kg

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Sulphur dioxide (7446-09-5)

Historical reports for Sulphur dioxide (7446-09-5)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	92	-	-	92	-	-	-	tonnes
2016	45	-	-	45	-	-	-	tonnes
2015	182	-	-	182	-	-	-	tonnes
2014	135	-	-	135	-	-	-	tonnes
2013	141	-	-	141	-	-	-	tonnes
2012	129	-	-	129	-	-	-	tonnes
2011	96	-	-	96	-	-	-	tonnes
2010	89	-	-	89	-	-	-	tonnes
2009	246	-	-	246	-	-	-	tonnes
2008	761	-	-	761	-	-	-	tonnes
2007	457	-	-	457	-	-	-	tonnes

2006	18	-	-	18	-	-	tonnes
2005	18	-	-	18	-	-	tonnes
2004	18	-	-	18	-	-	tonnes
2003	18	-	-	18	-	-	tonnes
2002	17	-	-	17	-	-	tonnes

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Sulphuric acid (7664-93-9)

Historical reports for Sulphuric acid (7664-93-9)

Year	On-Site Releases				Disposal(1)		Off-Site Recycling	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)		
2017	4.2	-	-	4.2	-	-	-	tonnes
2016	4.5	-	-	4.5	-	-	-	tonnes
2015	4.3	-	-	4.3	-	-	-	tonnes
2014	5.1	-	-	5.1	0	-	-	tonnes
2013	4.9	-	-	4.9	-	-	-	tonnes
2012	4.7	-	-	4.7	-	-	-	tonnes
2011	5.1	-	-	5.1	-	-	-	tonnes
2010	5.5	-	-	5.5	-	-	-	tonnes
2009	7.3	-	-	7.3	-	-	-	tonnes
2008	8.8	-	-	8.8	-	-	-	tonnes
2007	7.7	-	-	7.7	-	-	-	tonnes
2006	7	-	-	7	-	-	-	tonnes
2005	14	-	-	14	-	-	-	tonnes

2004	10	-	-	10	-	-	-	tonnes
2003	12	-	-	12	-	-	-	tonnes
2002	13	-	-	13	-	-	-	tonnes
2001	8.5	-	-	8.5	-	-	-	tonnes
2000	10	-	-	10	-	-	-	tonnes
1999	7.9	-	-	7.9	-	-	-	tonnes
1998	-	-	-	0	-	-	-	tonnes
1997	0.98	-	-	0.98	-	-	-	tonnes
1996	1.9	-	-	1.9	-	-	-	tonnes
1995	1.5	-	-	1.5	-	-	-	tonnes
1994	73	-	-	73	-	-	-	tonnes
1993	0	0	0	0	0	-	0	tonnes

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Total reduced sulphur (expressed as hydrogen sulphide) (NA - M14) (3)

Historical reports for Total reduced sulphur (expressed as hydrogen sulphide) (NA - M14)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	84	-	-	84	-	-	-	tonnes
2016	43	-	-	43	-	-	-	tonnes
2015	160	-	-	160	-	-	-	tonnes
2014	165	-	-	165	-	-	-	tonnes
2013	210	0.42	-	211	-	-	-	tonnes
2012	241	-	-	241	-	-	-	tonnes

2011	294	-	-	294	-	-	tonnes
2010	249	0.48	-	249	-	-	tonnes
2009	267	0.58	-	267	-	-	tonnes
2008	293	0.61	-	293	-	-	tonnes
2007	318	0.53	-	318	-	-	tonnes

(3) NOTE: Total reduced sulphur consists of 6 substances. Three of these substances (hydrogen sulphide [H2S], carbon disulphide [CS2] and carbonyl sulfide [COS]) are also listed individually in the NPRI substance list. If a facility meets the 10 tonne reporting threshold for any of H2S, CS2 or COS, it should report total reduced sulphur and the individual substance(s). Therefore, there is a potential for "double counting" of total reduced sulphur and the individual substance(s).

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Volatile organic compounds (NA - M16)

Historical reports for Volatile organic compounds (NA - M16)

Year	On-Site Releases				Disposal(1)		Off-Site	Units
	Air	Water	Land	Total	On-Site	Off-Site(2)	Recycling	
2017	143	-	-	143	-	-	-	tonnes
2016	136	-	-	136	-	-	-	tonnes
2015	132	-	-	132	-	-	-	tonnes
2014	140	-	-	140	-	-	-	tonnes
2013	144	-	-	144	-	-	-	tonnes
2012	143	-	-	143	-	-	-	tonnes
2011	108	-	-	108	-	-	-	tonnes

2010	118	-	-	118	-	-	tonnes
2009	103	-	-	103	-	-	tonnes
2008	107	-	-	107	-	-	tonnes
2007	407	-	-	407	-	-	tonnes
2006	392	-	-	392	-	-	tonnes
2005	352	-	-	352	-	-	tonnes
2004	366	-	-	366	-	-	tonnes
2003	351	-	-	351	-	-	tonnes
2002	356	-	-	356	-	-	tonnes

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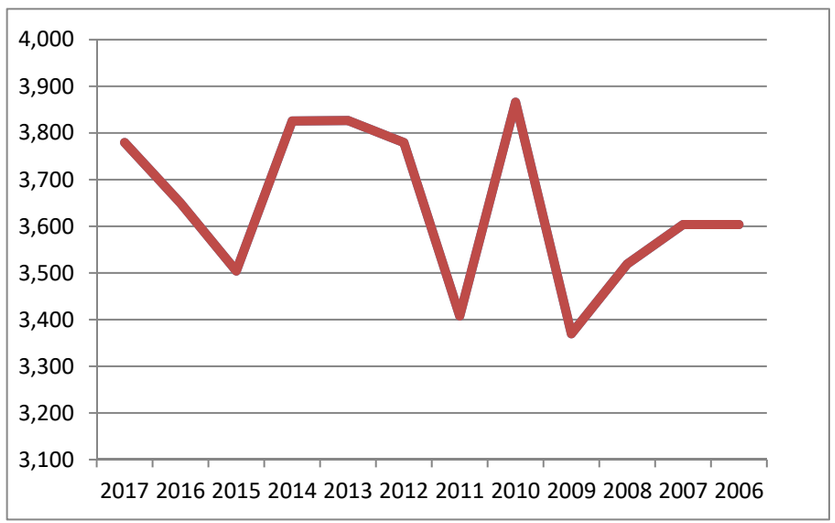
Units:

tonnes
g - grams
kg - kilograms
g TEQ - grams of Toxic Equivalent

NPRI air releases for Northern Pulp

Year	Dioxins and Furans		Acenaphthene		Acenaphthylene		Benzo(a)anthracene		Benzo(a)phenanthrene		Fluoranthene (206-44-0)		Phenanthrene (85-01-8)		Pyrene (129-00-0)		VOCs	
2017	0.008	TEQ (g)	224	kg	2,741	kg	9.7	kg	6.7	kg	95	kg	647	kg	56	kg	143	tonnes
2016	0.008		216		2,645		9.4		6.5		91		629		53		136	
2015	0.008		208		2,542		9		6.2		88		600		51		132	
2014	0.008		227		2,776		9.8		6.8		96		654		56		140	
2013	0.008		227		2,776		9.9		6.8		96		655		56		144	
2012	0.008		224		2,741		9.7		6.7		95		647		56		143	
2011	0.008		202		2,472		8.8		6		86		583		50		108	
2010	-		230		2,804		10		6.9		97		661		57		118	
2009	-		200		2,443		8.7		6		85		577		50		103	
2008	-		209		2,553		9.1		6.2		88		602		52		107	
2007	-		214		2,613		9.3		6.4		91		617		53		407	
2006	0.008		214		2,613		9.3		6.4		91		617		53		392	

Year	total PAHs	
2017	3,779	kg
2016	3,650	kg
2015	3,504	kg
2014	3,826	kg
2013	3,827	kg
2012	3,779	kg
2011	3,408	kg
2010	3,866	kg
2009	3,370	kg
2008	3,519	kg
2007	3,604	kg
2006	3,604	kg
Average	3,645	kg



APPENDIX H-28

Project Materials

Specialist Studies and Engagement Materials

Brochure with information on the New Effluent Treatment Facility, Northern Pulp

- Brochure providing additional details on the new effluent treatment facility.

NPNS Global Market Study, Brian McClay & Associates Inc.

- The NPNS Global Market Study assesses the viability of converting the existing Pictou Northern Bleached Softwood Kraft (NBSK) mill to produce either Unbleached Kraft Pulp (UKP) or Bleached Chemi-Thermo-Mechanical Pulp (BCTMP). For the reasons outlined in the market report, it can be concluded that continuing to produce premium reinforcement NBSK is the most competitively viable option by far for Northern Pulp.

Receiving Water Study, Stantec Consulting Ltd.

- The Receiving Water Study was completed during preliminary design to (1) evaluate potential locations for a marine outfall and identified the recommended area, (2) evaluate and made recommendations for the design and performance of the diffuser at the end of the outfall, and (3) model how the treated effluent will mix with the water at the outlet in the Northumberland Strait.

2016 EEM Report, ECOMETRIX

- The Environmental Effects Monitoring (EEM) study gives the results of the environmental effects monitoring from the existing Boat Harbour Treatment Facility. This EEM study is not directly applicable to the proposed replacement effluent treatment facility and associated treated effluent, but was provided upon request.

Technology Selection Summary Report, KSH

- This report documents the Preliminary Engineering for which reviewed the technology alternatives when determining the approach for the treatment facility at Northern Pulp.

Middle River Water Availability Report

- Completed in 2015 by RV Anderson for the Government of Nova Scotia, this report reviewed the sustainability of the water intake used by Northern Pulp.

Engagement Materials

Materials used at project engagement meetings, and summaries of those meetings will be posted here. This way we will increase the transparency of our process, and allow as many people as possible to engage with the project.

Project Launch: Summary of Engagement - What We Heard

Project Launch Open House Materials (December 2017 & January 2018)

Project Launch: Initiation Newsletter