Where Did All The Baby Bottles Go? Risk Perception, Interest Groups, Media Coverage and Institutional Imperatives in Canada's Regulation of Bisphenol A

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Introduction

On April 18, 2008, the federal ministers of Health and of the Environment announced that the common chemical bisphenol A (BPA) would be added to the list of toxic substances under criteria laid out in the *Canadian Environmental Protection Act, 1999* (CEPA) and that the manufacture, sale, import and advertising of polycarbonate baby bottles made from BPA would be prohibited. Thus, Canada became the first jurisdiction to determine that the existing evidence about BPA justified regulation. This decision was one of the first and most high-profile decisions in the context of the Chemicals Management Plan (CMP). This $816 million initiative was designed to assess the risks associated with thousands of chemicals and was a product of amendments to CEPA passed in 1999. At the time, Parliament had to decide what to do with substances on the Domestic Substances List (DSL), around 23,000 "legacy" substances that were in use between 1984 and 1986 and had never been subjected to any assessment. Under CEPA 1999, Parliament mandated the ministers of Health and the Environment to categorize each substance within seven years of the act's proclamation. Any substance that met particular criteria (specified in the legislation, see below) was sent for a full screening assessment to determine whether it should be deemed toxic and, therefore, subject to federal regulation. Thus, in December 2006, the federal government announced it had complied with the seven-year timeline, completed the

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categorization of the DSL and launched the CMP, a four-year endeavour to conduct full screening assessments on the substances that met criteria laid out in the legislation. Scientific staff within Health Canada and Environment Canada collaborated on each screening assessment which examined existing knowledge about the toxicity, uses and exposure levels of each substance. In cases where CEPA's criteria for toxicity were met, the two departments developed risk management proposals to regulate the use of each chemical.

This paper analyzes Canada's regulation of BPA and makes three major claims. First, it argues that the principles underlying the CMP represent a qualitative shift in Canadian risk management from a technocratic and closed system of risk management to a more pluralistic and open one where interest groups had a more significant role (Harrison and Hoberg, 1994). Second, the institutional context within which BPA was regulated interacted with interest group politics, public opinion and media coverage to create a decision that went beyond what scientific evidence could justify. Third, the legislative provisions that are embodied in CEPA to deal with the DSL reflect a widespread perception of risk from chemicals that is attributable to both psychological mechanisms and cultural values.

The first section of this paper outlines existing literature on the politics of risk management and risk perception to construct a theoretical framework. The central argument derived from these observations is that although the institutional and legislative framework in which risks are assessed interact with interest group politics and public opinion to create particular outcomes, as Harrison and Hoberg described, ultimately the framework of the CMP itself also reflects a widespread judgment that chemicals pose unjustifiable risks to social welfare. Thus, the institutional framework of regulation reflects a specific perception of risk. The second section describes the precise challenges in assessing and managing the risks associated with products made from BPA and argues that there is no scientific consensus that there is a risk to human health at current levels of exposure. The third section describes how the legislative provisions of CEPA and the institutional framework of Canadian federalism interacted with public opinion, interest group politics and media coverage to shape the policy outcome.[1]

Politics, Risk Perception and Values

Working from institutional theories of political science, Harrison and Hoberg (1994) examined the relationships between institutional design and differences in American and Canadian regulation of toxic substances. The pluralistic process in the United States differed from Canada's more technocratic process in several ways, but the difference with the most significant relevance for the case at hand lay in the legislation governing the

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Abstract. As part of a multi-year, $816 million initiative to assess the risks posed by thousands of commonly used chemicals and compounds, Canada became the first country in the world to declare that bisphenol A (BPA) was toxic and justified regulation in April 2008. The process set up to conduct this risk assessment differed from the previous Canadian experience with the regulation of hazardous substances in that it was more formal, systematic and more pluralistic with much greater participation from interest groups. This case study explores the politics and process behind this decision and argues that the government's decision went beyond what scientific evidence could justify. The decision resulted from long-term institutional factors such as the incentive structure of Canadian federalism and values embedded in legislation as well as short-term factors such as media coverage, public opinion and interest group pressure.

Résumé. Grâce à une initiative pluriannuelle de 816 millions de dollars pour examiner les risques posés par l'utilisation courante de milliers de produits et de composés chimiques, le Canada est devenu le premier pays dans le monde à déclarer que le bisphenol A (BPA) est un produit toxique et que de ce fait devrait s'en suivre des régulations quant à son utilisation à partir d'avril 2008.

De part le fait que le processus d'évaluation des risques suivi s'est fait sur une base plus formelle, systématique et pluraliste avec davantage de participation de groupes d'intérêt, il diffère de ce que le Canada avait l'habitude en matière de régulation de substances dangereuses. Cette étude de cas examine les politiques et processus qui ont servi à la prise de cette décision et met en avant l'argument qu'en fait le gouvernement ne disposait pas de suffisament de preuves scientifiques pour la justifier. La prise de décision résulterait de l'influence de facteurs institutionnels à long terme tels que des mesures d'incitation du fédéralisme canadien et des valeurs ancrées dans la législation, ainsi que de facteurs à court terme tels que la couverture médiatique, l'opinion publique et la pression des groupes d'intérêt.

risk assessment process. Due to the separation of powers, the legislation that Congress passed governing the regulation of toxic substances contained specific directions to executive agencies governing the substances to be examined, the timing of the risk assessment and provided for congressional oversight of executive rule making in the implementation of decisions. This provided interest groups substantial participation through formal notice-and-comment periods. In Canada, many of these decisions were left to cabinet ministers while regulations were developed internally with ad hoc consultation with affected stakeholders (Harrison and Hoberg, 1994: 8–10). In contrast, the CMP provided strict legislative oversight as to the timing of the regulatory process and the substances to be regulated. For example, the categorization of the DSL was to be completed within seven years while the criteria that governed which substances would be subjected to a full risk assessment were both expansive and written directly into the legislation. While there was no legislative requirement to govern the executive's rule making following the risk assessment process, the culture of Canadian policy making had evolved to such an extent that consultations with stakeholders have become de rigeur and they were an important part of the regulation of BPA (Roy, 2013).

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However, in four of the seven cases Harrison and Hoberg examined, Canada adopted more stringent regulations, while in the remaining cases it was the United States whose regulations were more stringent. Thus, one of the key outcomes of interest in the study of risk management—the stringency of regulation—cannot be predicted from the degree to which a risk management framework is technocratic or pluralistic. In addition to the institutional and legislative framework, Harrison and Hoberg also point to the influence of other variables such as interest groups, public opinion and party preferences.

> Rather than playing a determinative role in policy outcomes, the effects of institutions are contingent on a number of other variables that influence policy such as the balance of power between competing interest groups and the interests of the party in power. (Harrison and Hoberg, 1994: 181)

Two additional and complementary approaches to the study of risk perception can help explain how and why such variables shape risk management policies. The first, the psychometric approach, documents the irrational ways in which the brain perceives risk. Much of this stems from the structure of the brain itself, which has evolved to use two styles of thinking: one, quick and affective, the other slow, rational and deliberate (Gardner, 2009; Ropeik, 2010; Slovic et al., 1982). The former is often privileged over the latter. For example, the part of the brain that triggers the "fight or flight" sensation processes incoming stimuli before any other part of the brain, meaning that we experience the sensation of threats before we rationally process a response (Gardner, 2009: 57). While this was evolutionarily sound in a pre-civilizational context where failing to perceive, and react quickly to, imminent threats meant that one might quickly perish, in modern societies, this leads to distorted processing of risks and dangers. For example, our perceptions of the balance of risks and benefits are actually linked, suggesting an "affect heuristic" (Slovic et al., 2002). In other words, if people deem something to be beneficial, they are less likely to perceive it as risky and vice versa. However, if humans perceived risks in a strictly rational fashion, these evaluations would be distinct. Slovic and colleagues (1982) also found that humans are more likely to fear catastrophic, but improbable, events such as nuclear reactor meltdowns or airplane crashes than other less catastrophic and more routine and also dangerous events, such as driving a car. The same study found that humans tend to fear the unknown and unobservable phenomena, which has contributed to a widespread perception of risks from chemicals. Kraus and colleagues (1992) call this "intuitive toxicology." A 1995 survey of both the Canadian public and of toxicologists revealed a substantial difference in how citizens and experts view the risks posed by chemicals. Citizens were far more likely than toxicologists to agree that natural chemicals are

not as harmful as man-made chemicals, that most chemicals cause cancer and that a person exposed to a cancer-causing chemical would likely get cancer than were toxicologists. By contrast, toxicologists are much more likely than citizens to agree that fruits and vegetables contain natural substances that cause cancer and also that the use of chemicals in society had improved human health rather than harmed it (Slovic et al., 1995: 72). One last heuristic identified in this literature that bears on the case at hand is that humans continually demonstrate a heightened sense of risk perception towards anything or any activity that might involve children (Ropeik, 2010: 125).

Risk management policies can also be shaped by values and political culture, not just cognitive processes. The cultural theory of risk developed by Douglas and Wildavsky (1983) suggests that conflicts over the existence and management of risks in society are less questions of science than they are questions values. The process of weighing the costs and benefits of any given activity are influenced by commitments that have been previously made to a form of social organization. Recent empirical tests of this theory have found that value commitments toward egalitarianism and communitarianism are related to one's perception of the risks posed by global warming, controlling for demographics (e.g. education, gender and income), partisan affiliation, left-right ideology and trust in government (Kahan et al., 2007: 4). The combination of these cognitive and cultural forces has led to a widespread suspicion of chemicals and chemistry (Hartings and Fahy, 2011; Laszlo, 2007). Building on the insights that Harrison and Hoberg developed about the way that institutions interact with public opinion, interest groups and political parties, it will be shown below how this widespread opinion shaped the regulation of BPA in Canada.

Understanding this case matters because making risk management more pluralistic and less technocratic opens up the possibility that decisions will be made that are not warranted by the state of scientific knowledge but rather that cater to cognitive and cultural forces that shape our perceptions of risks (Harrison and Hoberg, 1994: 183; Sunstein 2004: 294). This is precisely what occurred in this case. When the federal government declared BPA to be toxic according to the criteria of CEPA, it did so on evidence that was—in its own words—"limited" (Canada. Health Canada and Environment Canada, 2008: 69).

Allowing cognitive and cultural forces to shape risk management policies can create new risks. For example, a society might dedicate inordinate resources trying to manage or eliminate risks that are objectively inconsequential but which are perceived to be more significant. Canada's decision on BPA gave momentum to activists and scientists in other countries concerned about possible adverse effects from BPA. The United States invested another $30 million to further investigate BPA (United States. National

Institute of Environmental Health Sciences, 2010). It is worth asking the utilitarian question of whether further investments will deliver a corresponding payoff in terms of human safety (Sunstein, 2004: 5; United States. Environmental Protection Agency, 1990: 2; Vogel, 2012: 262–63; Wildavsky, 1997: 434–35). In this regard, the toxicologist Richard Sharpe has written:

> Fundamental, repetitive work on bisphenol A has sucked in tens, probably hundreds, of millions of dollars from government bodies and industry which, at a time when research money is thin on the ground, looks increasingly like an investment with a nil return. All it has done is to show that there is a huge price to pay when initial studies are adhered to as being correct when the second phase of scientific peer review, namely, the inability of other laboratories to repeat the initial studies, says otherwise. (Sharpe, 2010: 3)

Moreover, ceding legitimacy to fears that outstrip scientific evidence, without recognizing how biased the perception of risks can be, could complicate efforts to promote important—but often controversial—public health interventions. For example, opposition to municipal fluoridation has been increasing in Canada; Calgary, Waterloo, Windsor, St. John and Quebec City have all recently stopped the practice. One of the arguments opponents to fluoridation make is that municipalities usually do not add pure fluoride to water supplies but the compound hydrofluorosilicic acid (see Waterloo Watch, 2010). This compound dissolves into fluoride and other non-toxic elements (Haneke and Carson, 2001: 4). However, opponents of fluoridation strategically emphasize the use of the more complex compound name in their campaign materials, thereby capitalizing on the cognitive heuristics that lead us to fear the unknown, not to mention the culturally dominant assumption that chemicals are intrinsically harmful substances (Gardner, 2009; Ropeik, 2010; Vogel, 2012; Wildavsky, 1997). Without a more balanced discourse about the costs and benefits of chemicals in general, public health initiatives can suffer (Entine, 2011).

Assessing the Risks from BPA

A voluminous amount of published scientific research has been dedicated to the study of BPA (Chapin et al., 2008; Hengstler et al., 2011; vom Saal and Welshons, 2006; vom Saal et al., 2007; Teeguarden and Hanson-Drury, 2013; Vandenberg and Maffini, 2009; Welshons and Nagel, 2006; Welshons et al., 2003). Thus, a comprehensive literature review here is beyond the scope of this paper. The purpose here is to describe the key scientific debates that relate to the risk assessment process and to demonstrate that there is no scientific consensus that humans face adverse health effects

at current levels of exposure from BPA. Instead, Canada's policy decision was as much about the institutional and legislative framework, interest group politics, media coverage and public opinion as it was about scientific evidence.

BPA is a common chemical in the contemporary marketplace. Its primary benefit is its capacity to harden plastic, preventing breaks. It is used in the protective epoxy linings of food jars and cans, as a protective lining for safety equipment and in medical tubing. Although estrogenic effects have been known for decades, concerns of possible adverse effects reached a broader audience in 1997 when Colborn and colleagues (1997) popularized the thesis that it could function as an endocrine disruptor in their influential book *Our Stolen Future*. The central allegation was that BPA mimics the female hormone estrogen and, at very low levels of exposure, is implicated in a range of adverse health effects—the "low dose hypothesis" (Vandenberg et al., 2012; vom Saal and Welshons, 2006; Welshons et al., 2003; Welshons and Nagel, 2006). Initially, attention focused on the potential of BPA to reduce sperm quality and increase the size of the prostate in male mice, findings that were reported by Frederick vom Saal (Nagel et al. 1997; vom Saal et al., 1997; vom Saal et al., 1998). Since then vom Saal has become the leading scientific proponent of the allegation that, at current levels of exposure, BPA poses a risk to human health.

Currently, US and European regulatory agencies maintain the position that the dose at which adverse health effects start to appear (the "no observable effects level" or NOAEL) is 5 mg per kilogram of body weight per day (mg/kg/bw.day). This is based on two industry-funded, multi-generation, peer-reviewed studies that exposed rodents to a wide range of doses of BPA (Tyl et al., 2002; Tyl et al., 2008). In keeping with standard risk assessment procedure, and to account for the possibility that humans and rodents might metabolize BPA differently, both the European Food Safety Authority (EFSA) and the Food and Drug Administration (FDA) reduced that level by a factor of 100 to derive a safe exposure for humans (tolerable daily intake, TDI) of 50 micrograms per unit of body weight per day (mcg/kg/bw.day). Those values have become standard across regulatory agencies, although in 1996, Canada set a provisional TDI of 25 mcg/kg/bw.day. However, in the 2008 screening assessment of BPA, Health Canada estimated that average adults were exposed to about 0.08 mcg/kg/bw.day, while formula-fed infants were exposed to between 0.92 and 4.3 mcg/kg/bw.day, with the higher levels being attributed primarily to using boiling water to prepare formula in polycarbonate baby bottles (Canada. Health Canada and Environment Canada, 2008: 53). Thus, even the highest exposure rates (4.3 mcg/kg/bw.day) were still five times lower than Health Canada's provisional TDI, 10 times lower than the internationally accepted TDI and more

than 1000 times lower than the level at which adverse effects start to appear in rodents.

Thus, the entire scientific and regulatory debate revolves around the validity of findings of adverse effects at very low doses. To date, regulatory agencies have rejected the studies that do show low dose effects. First, many rely on a narrow range of doses. But to fully understand the dose-response curve for toxic substances and to guard against the possibility of false positives, it is crucial to conduct experiments with a wide range of doses (Bell, 2012; Shelby, 2008: 10). Four studies that formed the bedrock of Health Canada's decision to label BPA toxic relied on single doses (Gioiosa et al., 2007; Laviola et al., 2005; Nishizawa et al., 2003; Palanza et al., 2002), and one relied on two doses (Kawai et al., 2003). Second, many studies that purported to show adverse effects relied on experiments where rodents were exposed by sub-cutaneous injection, rather than oral exposure (Shelby 2008: 15). Such exposures bypass the liver, which, in humans, processes BPA by glucuronidation and turns it into bisphenol A monoglucoride, which does not have endocrine activity. This compound is quickly excreted from the system by urination, with a half-life of six hours (Canada. Health Canada and Environment Canada 2008; Shelby, 2008: 6; Völkel et al., 2002). Third, some studies, including vom Saal's original experiments have not been replicated (Purchase, 2004), although attempts have been made, including with publicly funded research (see Ashby et al., 1999).

Fourth, low-dose studies have often relied on small sample sizes, which limit their statistical power (Shelby, 2008: 14). For example, the industry-funded study that has served as the bedrock of regulatory decisions stretched over three generations, starting with 30 males and 30 female rodents per dose (Tyl et al., 2002: 123). By contrast, Nagel and colleagues (1997) had groups of five to six rodents tested at each dose. Lastly, even those studies that have been classed as "low dose" studies mostly examine doses above levels of human exposure. For example, a recent meta-analysis found that only 16 of 123 low-dose studies examined the effects of doses in the range of adult human exposures and only 24 examined the range of infant and child exposures (Teeguarden and Hanson-Drury, 2013: 14). Thus, most low-dose studies are still not relevant to assessing risks to humans, because they are above the levels at which humans are exposed. Moreover, those studies that are in the range of human exposures often suffer from the limitations noted above.

The mixed body of scientific evidence is made more complicated by a correlation between study funding sources and outcomes. Vom Saal and Welshons (2006) analyzed 130 peer-reviewed studies, finding that 109 government funded studies (90%) found some evidence of harmful effects. By contrast, all (11) industry-funded studies and 10 government studies found no harmful effects and no industry-funded studies found harmful studies.

However, while the financial source of scientific research properly invites scrutiny it does not invalidate conclusions (Conrad and Becker, 2011; Rowe et al., 2009; Sutton et al., 2011). Moreover, there have been several publicly funded, robust studies that have shown that BPA does not produce adverse effects at current levels of exposure (Ryan et al. 2010; Delclos et al. 2014).

Additionally, the correlation between funding source and conclusion is also treated skeptically because of the bias toward publishing positive findings in scientific literature (Young and Karr, 2011; Nosek et al., 2012). This is an alarming threat to the validity of scientific findings. Ioannidis (2005) examined the fates of 49 highly cited studies describing the effects of various medical treatments (each cited more than 1000 times) and found that 32 per cent of those treatments were later found to have no effect or a weaker effect than initially postulated. Less than half of the original 49 treatments had ever been fully replicated. Thus, even a dataset characterized by predominantly publicly funded studies that show some effect must be interpreted with caution in light of the bias to the publication of positive results. A recent review of the scientific literature on BPA raised precisely this warning (Hengstler et al., 2011: 270).

In short, the scientific debate about BPA is tremendously complex and displays a conflicted body of evidence. However, the case that there are adverse health effects to humans at current levels of exposure had clearly not been established at the time of Health Canada's screening assessment and this remains the case today. Scientists at Health Canada were aware of this as the screening assessment was being prepared. On April 14, 2008, just days before the screening assessment and the decision to label BPA as toxic were to be published, the Director of the Bureau of Chemical Safety in Health Canada wrote to colleagues: "At this stage, any risk related to BPA exposure is hypothetical."

Institutions, Interest Groups and Public Opinion

Legislative framework

CEPA 1999 mandated Health Canada and Environment Canada to evaluate substances on the DSL as to whether they should be subjected to a risk assessment process and then, if criteria were met, regulated by the federal government. Two provisions inserted into CEPA in 1999 influenced the BPA risk assessment process.[2] First, Parliament created very loose and expansive criteria a substance would have to meet to trigger a screening assessment. Second, it inserted a clause into the legislation that required screening assessments of chemicals to apply both a weight-of-evidence approach and the precautionary principle. How each provision shaped the

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regulation BPA is outlined below. Fully understanding the expansive criteria for a screening assessment first requires distinguishing between "hazard-based" regulation and "risk-based" regulation. In toxicology, "hazard" is synonymous with toxicity—the intrinsic capacity of a substance to do harm to an organism or an ecosystem. "Risk" by contrast is the product of both toxicity and exposure; if humans or ecosystems are not exposed to a given substance that is highly toxic, then it does not pose a risk. CEPA's criteria that substances must meet to qualify for regulation are risk-based (hazard plus exposure), but the legislation labels these as toxic substances in schedule 1 of the legislation. There is widespread agreement that this definition is problematic because it is so confusing (Leiss, 2001). Later it will be shown that this mixed terminology contributed to the way Canada regulated BPA, particularly as it developed its risk management strategy of banning polycarbonate baby bottles.

In its 1995 deliberations on CEPA, the House of Commons' Standing Committee on the Environment and Sustainable Development wanted to shift the basis for regulating substances from the risk-based approach to a hazard-based approach and remove the exposure criterion as a requirement for regulating substances. The motivation was the impression that not enough substances were being regulated. "Our overriding wish, however, is that a larger number of substances of concern should become subject to the regulatory process under CEPA" (Canada. House of Commons. 1995: 68). To clear up the confusing use of the term "toxic" substances, the report recommended the concept of "inherent toxicity" as a replacement, meaning that substances could be regulated only based on the capacity of a substance to do harm without regard to exposure.

However, the federal government's response only made matters worse. The federal government accepted this notion of "inherent toxicity" but in a peculiar and limited way. It insisted on maintaining the old, risk-based definition of toxicity, including the exposure criterion, as a basis for regulation. Yet it accepted the concept and term "inherent toxicity" and suggested making it, along with persistence and /or bioaccumulation, as the criterion a substance had to meet to trigger a full screening or risk assessment. To further meet the committee's concern that a "larger number of substances of concern" be subjected to regulation, the federal government agreed to another criterion, that of the "greatest potential for exposure" (GPE). Thus, substances on the DSL were to be sent for a full screening assessment if they represented the GPE to humans or if they were inherently toxic and persistent or inherently toxic and bioaccumulative to either humans or ecosystems. However, they would only be *regulated* if they met the previous definition of toxicity (capacity to do harm plus exposure).

Both the GPE and the inherent toxicity provisions affected the regulation of BPA, but in different ways. The first was the formal justification for a screening assessment, but the inherent toxicity criterion coloured public

debate in such a way so as to amplify the threat to human health posed by BPA. Given its ubiquity, BPA was deemed to have met the criteria for GPE, and this finding alone was sufficient to trigger a screening assessment. However, it was deemed to have not met the inherently toxic criterion for human health. By contrast, in a parallel process examining BPA's impacts on ecosystems and wildlife, Environment Canada did determine BPA to be inherently toxic for aquatic organisms. However, because it did not persist in ecosystems or accumulate in the food chain, it did not meet the environmental criteria for a full screening assessment (Canada. Environment Canada, 2010). But this nuance was completely lost in public debate. On April 7, 2007, as the screening assessment process was beginning, the *Globe and Mail* published a long article which labeled BPA as "inherently toxic" in both the article and the headline writing; "government scientists classified bisphenol A as 'inherently toxic,' and companies making it will be challenged by the assessment to prove that continued use is safe" (Mittelstaedt, 2007). This, however, ignored the fact that the categorization process had only defined BPA as "inherently toxic" for aquatic organisms, not for humans, and that the same process found that it did not meet the criteria of persistence or bioaccumulation, meaning that BPA did *not* meet the necessary legislative criteria for a screening assessment on the grounds of inherent toxicity. In this way, ambiguous concerns about the threats posed by unnamed chemicals expressed by the standing committee in 1995 and enshrined in legislation in 1999 influenced public debate in 2007 by introducing a crucial concept in a confusing fashion, allowing journalists and interest groups to seize on it in hyperbolic and misleading ways.

The second provision was the inclusion of the weight-of-evidence approach and the precautionary principle. Both are established principles of risk management but represent very different attitudes toward the role that scientific evidence should play in the regulatory process and where the burden of proof should lie. The former seeks to provide decision makers and scientists with guidance for interpreting evidence that is conflicting, or when a substance has an unclear relationship to human health (Linkov et al., 2009). The latter is also an approach for evaluating conflicting evidence that argues that the absence of full scientific certainty should not be used as a reason for not pursuing regulatory action (Canada. Privy Council Office, 2003). In the field of environmental or human health risk assessment, this principle puts the burden of proof on the polluter or agent responsible for a new technology to prove its safety (Edge and Eyles, 2013; Scott, 2009).

While not contradictory, these two principles do provide different ways of interpreting conflicting scientific evidence. The former involves the evaluation of the quality of different lines of evidence; the latter lends greater weight to evidence that shows some adverse effects. The tension

between the two provided a great deal of flexibility to decision makers to interpret conflicting evidence, creating a space for political pressure to be applied.[3] Using the weight-of-evidence approach, the screening assessment concluded that BPA was neither carcinogenic nor genotoxic at current levels of exposure (Canada. Health Canada and Environment Canada, 2008: 59–60). The assessment also acknowledged that the evidence for BPA's reproductive effects was so mixed that it could not draw a conclusion (60–63). Thus, the conclusion about the risk posed rested solely on the evaluation of the evidence related to neurological development. However, applying a weight-of-evidence approach to this dataset, the screening assessment noted that:

> Overall, taking into consideration rigour, power, corroboration/consistency, and biological plausibility/coherence, the weight of evidence supporting neurobehavioral effects in rodents following exposures to bisphenol A at exposures below established NOAELs for reproductive/developmental toxicity is limited. (69)

There were eight studies in this dataset that tested doses in the ranges to which Canadians are exposed to and seven of them found some sort of adverse effect. However, four of them were single-dose studies (three of them from the same lab) (Gioiosa et al., 2007; Laviola et al., 2005; Nishizawa et al., 2003; Palanza et al., 2002), one of them tested only two doses (Kawai et al., 2003) and none of them involved more than one generation. Two that studied a wide range of doses displayed unclear dose-response curves (Nishizawa et al., 2005a; Nishizawa et al., 2005b). Lastly, Ema and colleagues (2001) reported an industry-funded two-generation study, which tested the effects of four doses of BPA and found no adverse effects.

Thus, the decision to label BPA as toxic in the face of this limited evidence rested on utilizing the other principle mandated by the CEPA: the precautionary principle. Immediately following the screening assessment's characterization of the limitations in the neurodevelopmental toxicity dataset, it argued:

> The neurodevelopmental and behavioural dataset in rodents, though highly uncertain, is suggestive of potential effects at doses at the same order of magnitude to 1–2 orders of magnitude higher than exposures. Given that toxicokinetic and metabolism data indicate potential sensitivity to the pregnant woman/fetus and infant; and that animal studies suggest a trend towards heightened susceptibility during stages of development in rodents, it is considered appropriate to apply a precautionary approach when characterizing risk. As such, it is concluded that bisphenol A be considered as a substance that may be entering the environment in a quantity or concentration or under conditions that constitute or may constitute a

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> danger in Canada to human life or health. (Canada. Health Canada and Environment Canada, 2008: 73)

One effect of including both approaches in the legislation was that it created confusion that could best be clarified by adopting a dramatic risk management strategy that was more aggressive than warranted by this uncertain scientific evidence, namely, by banning polycarbonate baby bottles. On the one hand, the department had concluded that the evidence about the existence of a risk was limited. On the other, it had also concluded that it wanted to interpret this limited evidence in a precautionary fashion and thus declared BPA to be toxic. Communicating these two conclusions was a significant challenge. One official involved in the risk management process for BPA noted the problems caused by the terminology in the legislation, referring specifically to the improper use of the term toxic: "toxic is about the worst choice of language the law could have chosen. As a label, it's a nightmare for communication because you can't tell someone it's toxic but not toxic as you know it."

Because the greatest exposure to humans was to infants via polycarbonate baby bottles, the risk management strategy revolved centered on reducing exposure from this route. Initially, bureaucrats recommended a warning to avoid putting boiling liquids in polycarbonate baby bottles to prevent leeching of BPA into the formula. Reflecting the fact that humans are not exposed BPA at levels at which adverse effects occur, a draft news release of April 12, 2008, read:

> Science indicates that newborn and infant exposures are below the levels that cause health effects. However, the government has determined that levels should be even lower and is taking action to further reduce bisphenol A exposure in infants and newborns.

However, after an interdepartmental conference call that involved officials from the Prime Minister's Office, the Privy Council Office, Health Canada and Environment Canada on Friday, April 11, and another on April 13, a new, much more dramatic risk management proposal appeared, the prohibition of polycarbonate baby bottles made with BPA. On Sunday, April 13, the Associate Assistant Deputy Minister in charge of Health Canada's Health Products and Food Branch, Siddika Mithani, reported on these developments to her colleagues as follows: "[Samuel Godefroy, Director of the Bureau of Chemical Safety] and I have been on both conference calls, a lot of this is about possible polycarbonate baby bottles being banned if at the end of the 60 days there is no additional data. Our input is talking to industry to establish stringent migration targets and work on codes of practice." It appears that this strategy was rejected in favour of a more dramatic strategy as a draft news release dated April 14 reads:

> Science indicates that bisphenol A exposure to newborns and infants is below levels that may pose a risk; however, the gap between exposure and effect is not large enough. Health Canada is taking a series of actions to further reduce bisphenol A exposure in infants and newborns including a proposed ban on polycarbonate baby bottles and stringent migration targets for bisphenol A in infant formula cans and other canned foods.[4]

However, banning polycarbonate baby bottles opened up yet another conundrum. Namely, the government had to explain why it was banning this product made from BPA, but not banning the use of BPA in infant formula cans. An email dated April 12, 2008, from a Health Canada civil servant to the office of the Chief Public Health Officer, Dr. David Butler-Jones described this problem:

> HC [Health Canada] has told us we can't announce a ban on BPA in formula cans because that would effectively mean taking baby formula off the market...We would like to know whether [Dr. Butler-Jones] is prepared to state unequivocally that the benefits of feeding formula to babies outweigh the risks of exposure to BPA contained in cans of formula. Otherwise we have a significant comms [communications] and policy challenge.

Although Butler-Jones responded that he was comfortable with the dual strategies on the grounds that there were no alternatives to BPA in lining cans of infant formula, this exchange raises the question raised about why Health Canada was taking action on baby bottles in the first place. Prima facie, if a substance could be construed to be causing harm—and its declaration of "toxicity" and banning of polycarbonate baby bottles certainly implied that this was the government's opinion—whether or not there are alternatives would seem to be of secondary importance. Health Canada's incoherent policy choice was partly determined by the fact that there were no alternatives to BPA as a sealant in canned infant formula, partly by the fact that the exposure levels could potentially be higher in heated polycarbonate baby bottles, rather than from canned formula and partly by the fact that its own decision to label BPA toxic had created changed market conditions such that no retailers were offering the products anyway, making a prohibition on baby bottles a costless endeavour (Canada. Health Canada. 2008c).

At the end, Health Canada was left claiming multiple things simultaneously. First, it found that Canadians were fundamentally safe because there was only "limited" evidence that humans were being adversely affected. Second, it was interpreting this evidence in a precautious fashion and therefore declaring BPA to be toxic. Third, because BPA was toxic, but did not pose a risk to human health, it was banning polycarbonate baby bottles.

Lastly, despite labeling BPA toxic while declaring that it saw no evidence of effects on human health, it adopted a risk management strategy that had the objective of minimizing infant exposure (Canada. Health Canada. 2008c). Given that the policy instrument of a prohibition on polycarbonate baby bottles, rather than just warning labels, emerged after an interdepartmental conference call with high-level officials from the Prime Minister's Office, the Privy Council Office, Health Canada and Environment Canada suggests it was designed with at least some consideration of the communications challenges documented above as well as the political concerns (see the next section), even though this strategy created additional contradictions that had to be managed by ensuring that the Chief Public Health Officer was in agreement and would provide public support.

Interest Groups

This was the formal decision-making process that led to designating BPA as toxic. Informal interest group politics played an important role as well (see Edge and Eyles, 2013). On the environmental side, a variety of groups, including Pollution Probe, the Canadian Environmental Law Association, the Parks and Wilderness Society and Environmental Defence (ED), were organized into a public umbrella campaign with a website and a common position. Within this coalition, ED played a leading role. Throughout 2006, as the categorization of substances on the DSL was being finalized and substances were being prioritized in terms of the order in which they would be examined, ED ran a high-profile lobbying and media campaign around the issue of chemical pollution from common substances. One Conservative Party strategist, Tim Powers, described the government's positive reaction to ED's lobbying campaign as follows: "Toxic Nation fit with the main street ethos of the new government. You can see a plastic water bottle; you can't see a greenhouse gas" (quoted in Smith and Lourie, 2010: 224). In February 2006, ED's executive director, Rick Smith, coauthored an article in the *Globe and Mail*, with prominent Conservative, Adam Daifallah, suggesting the new government could find common ground with other parties in a minority Parliament by emphasizing environmental issues (Smith and Daifallah, 2006). In June 2006, ED released the blood test results of seven Canadian children who had volunteered to have their blood examined, which showed trace amounts of a number of chemicals. The results received significant media attention, including the lead story on *The National*. As a part of the response to that challenge, Environment Minister Rona Ambrose and Health Minister Tony Clement agreed to have their blood tested as well.

Did these measures affect the regulatory process? Given the broad criteria enshrined in CEPA in 1999, BPA was likely going to be subjected to a full screening assessment independent of any lobbying pressure in 2006.

However, a Health Canada official interviewed claimed that there was an agreement between ED and Health Canada to ensure that BPA was assigned a higher priority and screened early in the process. "There had been a negotiated agreement with Rick Smith of Environmental Defence that BPA would be part of the second batch of chemicals that were examined." While these are serious charges and not implausible, there is no mention of any agreement to that effect between ED and the federal government in Smith's written account of ED's lobbying efforts and the informant could not specifically name the source of his information. Smith neither denies nor confirms this charge but said this in an interview: "We made our position very clear that BPA should be given a high priority, and that's what ended up happening." And even if there was nothing so untoward as an explicit quid pro quo as the informant suggested, it is at least plausible that the lobbying and subsequent media coverage generated by ED had an influence on the *standards* that were developed and possibly played a role in ensuring that BPA was prioritized for quick action. One independent toxicologist interviewed and who provided advice to Health Canada on the assessment process suggested that this may have been the case.

During the preparation of the actual risk assessment, ED stepped up its campaign and organized a rally and meetings with Ontario Premier Dalton McGuinty in October 2007 which led to a public commitment by the premier to introduce provincial regulations (Smith and Lourie, 2010: 222). In this way, ED exploited an advantage that federal decision-making structures offer to environmental groups. Because environmental protection policies tend to be popular with the public, federal structures can lead to stricter environmental protection measures because governments compete with each other to claim credit (Harrison, 1996; Harrison, 2011). In 2007 and 2008, environmental issues were high on the public agenda; public opinion surveys noted that 16 to 21 per cent of Canadians listed the environment as the most important problem during 2007 (Environics, 2007, 2008). Also, the Liberal leader, Stéphane Dion, was defining himself as being particularly committed to environmental protection. Moreover, the existence of a minority parliament meant that politicians had to be particularly sensitive to public opinion. In this context, the federal government had a strong incentive to move aggressively to prevent the provincial government from claiming credit on this high-profile issue.

ED also pressured Canadian retailers into withdrawing polycarbonate water bottles from circulation (Smith and Lourie, 2010: 224). This had the dual effect of increasing public concern about the issue and simultaneously minimizing the potential regulatory costs associated with banning polycarbonate baby bottles, since they were already off the market by the time the government was called upon to develop regulations. Lastly, ED released its own non-peer reviewed study measuring BPA leeching into water stored in

polycarbonate baby bottles in extreme conditions (Environmental Defence, 2008: 7). Although this was not published in a peer-reviewed journal, Health Canada included it in its assessment of Canadians' exposure to BPA (Canada. Bureau of Chemical Safety, 2008: 40).

The environmental lobby was also matched by some lobbying by business groups including Dow Chemical, the Canadian Plastic Industry Association, BPA Global Group and the American Chemistry Council (see Edge and Eyles, 2013, for details). However, in contrast to the very public environmental campaign, there was less publicity by the chemical industry. The president of the American Chemistry Council wrote some letters to the editor and some representatives of the restaurant, grocery and packaging industries made written representations to Health Canada, but there is no evidence of any significant political activity. This is partly attributable to the fact that no company in Canada manufactures BPA. The bulk of the commercial activity on this issue was relegated to those retailers who voluntarily withdrew bottles made with BPA in response to pressure from ED.

Media Coverage, Issue Salience and Public Opinion

Although the scientific debate on BPA has been carried out in many countries, the issue attracted far more media and public attention in Canada than in other countries. Moreover, it is clear from Figure 1 that Canadian newspaper coverage had an impact on public salience of the BPA issue.[5] Figure 1 shows the correlation between the frequency of newspaper stories about BPA and the Canadian Google search traffic for the term over the same time frame, suggesting that newspaper coverage drove citizens to seek out information. In nearly every weekly period where there was an increase in media coverage, there was a corresponding increase in internet search traffic in Canada for the term "bisphenol A." This correlation between media coverage and public interest in the issue is consistent with findings from Brewer and Ley (2011) who conducted a telephone survey of Milwaukee residents, a city where one of the local newspapers ran a long, in-depth feature about BPA. That study found that respondent newspaper use was correlated with familiarity about the issue and behavioural change in response to it.

While this is not direct evidence that media coverage *changed* public opinion, it seems to have at least played a role in making the issue more salient amongst the general population. Smith and Lourie (2010: 252) also note that blogs and social media contributed to making it possible for citizens to mobilize public opinion on the issue. In addition, Kiss (2013) has found that levels of newspaper coverage about BPA in a state's daily newspapers in any given year was positively correlated with

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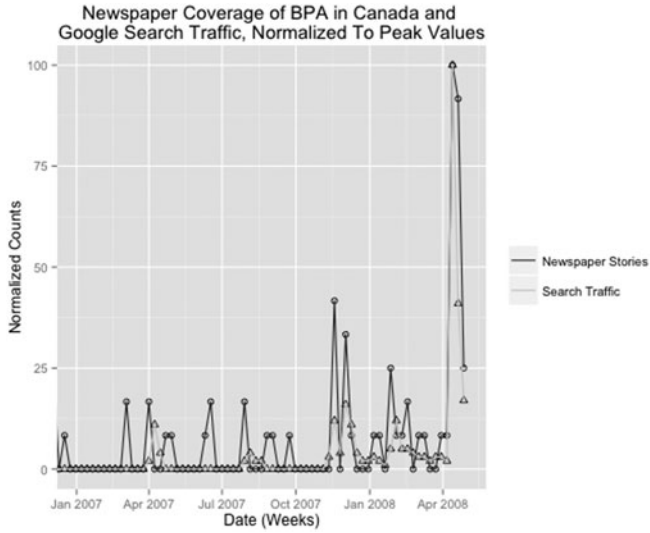
FIGURE 1
Google search traffic and newspaper frequency by week in Canada. These data are both normalized to the peak in April 2008 for maximum comparability.



the likelihood that the state's legislature would take up, or reconsider (and, in some cases, adopt) legislation banning products made with BPA the following year, controlling for state public opinion and the partisan character of the legislature. Pralle (2006) has also documented the ways in which elevating the media and public salience of pesticides in public opinion was integral to advancing the cause of regulation in Canada. It seems media coverage played a role in making the issue more salient to voters and, subsequently, to politicians.

A large part of this coverage can be attributed to the *Globe and Mail's* environment reporter, Martin Mittelstaedt, who began publishing stories on BPA in 2006. He accounted for 67 of 146 stories about BPA that appeared in the *Globe and Mail* and the *National Post* between 2006 and 2010. Much of the newspaper coverage focused heavily on the risks and downplayed the scientific controversy and uncertainty in the literature. For example, on June 9th, 2006, Mittelstaedt wrote about a peer-reviewed study linking exposure to BPA with prostate cancer (Ho et al., 2006), although he neglected to point out that the study exposed rodents to BPA via injections in the skin, rather than orally, the limits of which have been discussed above. Often there was close collaboration with Environmental Defence. For example,

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a two-part series about the adverse health effects of BPA (and chemicals in general) appeared on May 31 and June 1, 2006, the same day that ED released its blood tests of children showing trace amounts of various chemicals. Similarly, on February 8, 2008, just weeks before the screening assessment was finished, Mittelstaedt wrote a story about the non-peer reviewed study by ED testing the leeching of BPA from polycarbonate baby bottles. One research scientist affiliated with Health Canada during the screening assessment process said, "I can't help but think that Martin Mittelstaedt was kind of in partnership with Rick Smith to get BPA banned... I don't think he was out to do objective reporting, I think he was out to help Rick Smith."

One episode in particular further politicized this issue and increased its importance on the government's agenda. While Health Canada was preparing the screening assessment, the departmental scientist overseeing the process, Mark Richardson, was involved in a heated exchange about the risks posed by BPA with vom Saal at a scientific conference. Richardson expressed the view that he was not convinced there was evidence of harm to human health at current levels of exposure. Mittelstaedt was able to view a recording of the exchange and questioned the office of the health minister about whether the risk assessment could be truly objective given Richardson's comments. In return, the minister's office demanded an investigation from the department which was conducted by Health Canada's chief scientist. Ultimately, Richardson was pulled from the file and Mittelstaedt published a story on June 20, 2007. Following this episode, ED capitalized on the publicity and increased its lobbying efforts of federal officials (Smith and Lourie, 2010). This was one more way in which the issue was moved higher up the decision-making hierarchy within the federal government. Moreover, it created the impression that Health Canada's process was suspect or biased, possibly increasing the pressure on the federal government to assign more priority to explicitly political—rather than strictly scientific—grounds in its final regulations.

Conclusion

In 2006, Canada launched the Chemicals Management Plan, a program originally mandated by Parliament in 1999 to screen thousands of substances in the marketplace for evidence of harm to human health. This process represented a shift in how Canada historically regulated toxic substances from a closed technocratic process that offered the executive a great deal of autonomy and integrated interest group participation in an ad hoc fashion at best. Instead, the CMP provided stricter guidelines on executive decision making and exhibited far greater and more regular interest group participation. The rules governing the CMP reflect a widespread perception that chemicals pose an unjustifiable risk to human health, which has its

roots in cognitive psychology and deep value commitments. Over the course of this program, Canada became the first jurisdiction in the world to declare the ubiquitous chemical BPA to be toxic and subsequently banned polycarbonate baby bottles. However, this decision cannot be explained with reference to compelling scientific evidence that Canadian are at risk of any harm. Instead, short-term factors such as high levels of critical media coverage, strong levels of interest group lobbying and a concomitant increase in the salience of the issue in public opinion, a high level of concern for environmental issues in general and a minority parliament all interacted with the rules of the CMP and the values underlying it to produce the decision that BPA was toxic according to CEPA.

Although the shift from a technocratic to a pluralistic regulatory regime was responsible for this decision, it is not asserted here that Canada should revert to its previous manner of risk management. Technocratic regimes, dominated by experts, are subject to manipulation by special interests as well (Conway and Oreskes, 2011; McGarity and Wagner, 2008). For Harrison and Hoberg (1994: 184) the answer to managing the disadvantages of a more open system is to improve the public discourse about risk. While this is no easy process and beyond the scope of this paper, it is hoped that this present contribution documenting how risk perception can shape policy outcomes, citizens, scholars and politicians can better evaluate controversies about.

Notes

1 These claims are substantiated with a variety of sources of evidence. The author conducted eight unstructured interviews with scientists, environmental activists and officials at Health Canada . Second, the paper draws on a unique set of documents released under the *Access to Information and Protection of Privacy Act* to the *Montreal Gazette* and provided to the author . These documents date from March and April 2008, as the screening assessment for BPA was being finalized. They include email messages between civil servants and internal documents, such as briefing notes. Citations to these documents are provided in text with the date of the document's production and the name of the author. In particular, they enable the reconstruction how the department's risk management strategy evolved as a response to political decisions and communications challenges. Third, the impact of the media's coverage on public opinion is documented by correlating newspaper coverage with Google Trends data reflecting public interest in the topic.

2 This section is informed by Leiss's description (2001) and analysis of the revisions in CEPA 1999.

3 For an overview of the conflict between the weight-of-evidence approach and the precautionary principle in this case, see Edge and Eyles (2013).

4 A news story in the *National Post* on February 24, 2010, quotes an associate assistant deputy minister of Health Canada as saying the final decision to ban polycarbonate baby bottles was made with officials from the departmental executive committee, the PMO and the PCO.

5 Figures reporting data supporting the assertion that this issue was higher on the Canadian media and public agendas than in other countries are available as supplementary data at the online version of this article.

References

Ashby, J., H Tinwell, and J. Haseman. 1999. "Lack of effects for low dose levels of bisphenol A and diethylstilbestrol on the prostate gland of CF1 mice exposed in utero." *Regulatory toxicology and pharmacology* 30 (2): 156–66.

Bell, David. 2012. "Nature of an effect: adverse or non-adverse." EFSA Scientific Colloquium no. 17 on low dose response in toxicology and risk assessment. Parma, Italy. http://www.efsa.europa.eu/en/events/documents/120614l-p04.pdf (June 13, 2013).

Brewer, P.R. and B.L. Ley. 2011. "Multiple Exposures: Scientific Controversy, the Media, and Public Responses to Bisphenol A." *Science Communication* 33 (1): 76–97.

Canada. Bureau of Chemical Safety. 2008. *Health Risk Assessment of Bisphenol A from Food Packaging Applications*. Ottawa: Health Canada. http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/securit/bpa_hra-ers-eng.pdf (June 30, 2013).

Canada. Environment Canada. 2010. *Domestic Substances List*. http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=5F213FA8-1 (June 6, 2013).

Canada. Health Canada and Environment Canada. 2008. *Screening Assessment for the Challenge Phenol, 4,4' -(1-methylethylidene)bis(Bisphenol A)*. Ottawa. http://www.ec.gc.ca/ese-ees/3C756383-BEB3-45D5-B8D3-E8C800F35243/batch2_80-05-7_en.pdf (June 13, 2013).

Canada. Health Canada. 2008. *Proposed Risk Management Approach for Phenol, 4,4'-(1-Methylethylidene) Bis (Bisphenol A)*. Ottawa: Queen's Printer.

Canada. House of Commons. Standing Committee on the Environment and Sustainable Development. 1995. *It's About Our Health: Towards Pollution Prevention*. Ottawa: Queen's Printer.

Canada. Privy Council Office. 2003. *A Framework for the Application of Precaution in Science-Based Decision Making About Risk*. Ottawa: Privy Council Office.

Colborn, T., D. Dumanoski and J.P. Myers. 1997. *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival? A Scientific Detective Story*. New York: Plume.

Conrad, James W., Jr., and Richard Becker. 2011. "Enhancing Credibility of Chemical Safety Studies." *Environmental Health Perspectives* 119 (6): 757–64.

Conway, E. M. and N. Oreskes. 2011. *Merchants of Doubt: How a Handful of Scientists Obscured the Truth on Issues from Tobacco Smoke to Global Warming*. New York: Bloomsbury.

Chapin, Robert E., J. Adams, K. Boekelheide, L.E. Gray , Jr., S.W. Hayward, P.S. Lees, B.S. McIntyre, K.M. Portier, T.M. Schnorr, S.G. Selevan, J.G. Vandenbergh and S.R. Woskie. 2008. "NTP-CERHR Expert Panel Report on the Reproductive and Developmental Toxicity of Bisphenol A." *Birth Defects Research Part B: Developmental and Reproductive Toxicology* 83 (3): 157–395.

Delclos, K. Barry, L. Camacho, S.M. Lewis, Michelle M. Vanlandingham, John R. Lattendresse, Greg R. Olson, Kelly J. Davis, Ralph E. Patton, Gonçalo Gamboa da Costa, Kellie A. Woodling, Matthew S. Bryant, Mani Chidambaram, Raul Trbojevich, Beth E. Juliar, Robert P. Felton, Brett T. Thorn. 2014. "Toxicity evaluation of bisphenol A administered by gavage to Sprague-Dawley rats from gestation day 6 through postnatal day 90", *Toxicological Sciences* 139 (1): 174–197.

Douglas, M. and A. Wildavsky. 1983. *Risk and Culture: An Essay on the Selection of Technical and Environmental Dangers*. Berkeley CA: University of California Press.

Edge, S. and J. Eyles. 2013. "Message in a bottle: claims disputes and the reconciliation of precaution and weight-of-evidence in the regulation of risks from Bisphenol A in Canada." *Health, Risk & Society* 15 (5): 432–48.

Ema, M., S. Fujii, M. Furukawa, M. Kiguchi, T. Ikka and A. Harazono. 2001. "Rat two-generation reproductive toxicity study of bisphenol A." *Reproductive toxicology* 15 (5): 505–23.

Entine, Jon. 2011. *Scared to Death: How Chemophobia Threatens Public Health*. New York: American Council on Science and Health.

Environmental Defence. 2008. *Toxic Baby Bottles in Canada*. Toronto.

Environics. *Environics Focus Canada 1985–2007*. http://www.queensu.ca/cora. (July 18, 2014).

Gardner, D. 2009. *Risk: The Science and Politics of Fear*. Toronto: McClelland & Stewart.

Gioiosa, Laura, E. Fissore, G. Ghirardelli, S. Parmigiani and P. Palanza. 2007. "Developmental exposure to low-dose estrogenic endocrine disruptors alters sex differences in exploration and emotional responses in mice." *Hormones and behavior* 52 (3): 307–16.

Haneke, Karen and Bonnie Carson. 2001. *Toxicological Summary for Sodium Hexafluorosilicate [16893-85-9] and Fluorosilicic Acid [16961-83-4]*. Washington: National Institute of Environmental Health Sciences

Harrison, K. 2011. *Racing to the Bottom? Provincial Interdependence in the Canadian Federation*. Vancouver: UBC Press.

Harrison, K. and G. Hoberg. 1994. *Risk, Science, and Politics: Regulating Toxic Substances in Canada and the United States*. Montreal and Kingtson: McGill-Queen's University Press.

Harrison, Kathryn. 1996. *Passing the Buck: Federalism and Canadian Environmental Policy*. Vancouver: UBC Press.

Hartings, M.R. and D. Fahy. 2011. "Communicating chemistry for public engagement." *Nature Chemistry* 3: 674–77.

Hengstler, J. G., H. Foth, T. Gebel, P.J. Kramer, W. Lilienblum, H. Schweinfurth, W. Völkel, K.M. Wollin and U. Gundert-Remy, 2011. "Critical evaluation of key evidence on the human health hazards of exposure to bisphenol A." *Critical Reviews in Toxicology* 41 (4): 263–91.

Ho, S.M., W.Y. Tang, J. Belmonte de Frausto and G.S. Prins. 2006. "Developmental exposure to estradiol and bisphenol A increases susceptibility to prostate carcinogenesis and epigenetically regulates phosphodiesterase type 4 variant 4." *Cancer research* 66 (11): 5624.

Ioannidis, John P.A. 2005. "Contradicted and Initially Stronger Effects in Highly Cited Clinical Research." *Journal of the American Medical Association* 294 (2): 218–28.

Kahan, Dan M., Donald Breman, Paul Slovic, John Gastil and Geoffrey L. Cohan. 2007. "The Second National Risk and Culture Study: Making Sense of and Making Progress in the American Culture War of Fact." http://www.culturalcognition.net (July 24, 2014).

Kahan, Dan M., Donald Braman, Geoffrey L. Cohen, John Gastil and Paul Slovic. 2010. "Who fears the HPV vaccine, who doesn't, and why? An experimental study of the mechanisms of cultural cognition." *Law and human behavior* 34 (6): 501–16.

Kawai, Keisuke, Takehiro Nozaki, Hiroaki Nishikata, Shuji Aou, Masato Takii and Chiharu Kubo. 2003. "Aggressive behavior and serum testosterone concentration during the maturation process of male mice: the effects of fetal exposure to bisphenol A." *Environmental Health Perspectives* 111 (2): 175.

Kiss, Simon. 2013. "Legislation by agenda-setting: Assessing the media's role in the regulation of BPA in the US states." *Mass Communication and Society* 16 (5): 687–712.

Kraus, Nancy, Torbjorn Malmfors and Paul Slovic. 1992. "Intuitive Toxicology: Expert and Lay Judgments of Chemical Risks." *Risk analysis*. 12 (2): 215–32.

Laszlo, P. 2007. "On the self-image of chemists, 1950–2000." *The Public Image of Chemistry*, ed. Joachim Schummer, Bernadette Bensaude Vincent and Brigitte Van Tiggelen. London: World Scientific Publishing.

Laviola, Giovanni, W. Adriani and P. Palanza. 2005. "D-amphetamine-related reinforcing effects are reduced in mice exposed prenatally to estrogenic endocrine disruptors." *Brain research bulletin* 65 (3): 235–40.

Leiss, W. 2001. *In the Chamber of Risks: Understanding Risk Controversies*. Montreal, Kingston: McGill-Queen's University Press.

Linkov, I., D. Loney, S. Cormier, S.K. Satterstrom and T. Bridges.. 2009. "Weight-of-evidence evaluation in environmental assessment: Review of qualitative and quantitative approaches." *Science of the Total Environment* 407 (19): 5199–5205.

McGarity, Thomas O. and Wendy Elizabeth Wagner. 2008. *Bending Science: How Special Interests Corrupt Public Health Research*. Cambridge MA: Harvard University Press.

Nagel, S.C., F.S. vom Saal, K.A. Thayer, M.G. Dhar, M. Boechler and W.V. Welshons. 1997. "Relative binding affinity-serum modified access (RBA-SMA) assay predicts the relative in vivo bioactivity of the xenoestrogens bisphenol A and octylphenol." *Environmental Health Perspectives* 105 (1): 70–76.

Nishizawa, Hanako, Noboru Manabe, M. Morita, M. Sugimoto, S. Imanishi and H. Miyamoto.2003. "Effects of in utero exposure to bisphenol A on expression of RARalpha and RXRalpha mRNAs in murine embryos." *The Journal of Reproduction and Development* 49 (6): 539–45.

Nishizawa, Hanako, Satoshi Imanishi and Noboru Manabe. 2005a. "Effects of exposure in utero to bisphenol a on the expression of aryl hydrocarbon receptor, related factors, and xenobiotic metabolizing enzymes in murine embryos." *The Journal of Reproduction and Development* 51 (5): 593–605.

Nishizawa, Hanako, Maki Morita, M. Sugimoto S. Imanishi and N. Manabe. 2005b. "Effects of in utero exposure to bisphenol A on mRNA expression of arylhydrocarbon and retinoid receptors in murine embryos." *The Journal of Reproduction and Development* 51 (3): 315–24.

Nosek, Brian A., Jeffrey R. Spies and Matt Motyl. 2012. "Scientific Utopia II. Restructuring Incentives and Practices to Promote Truth over Publishability" *Perspectives on Psychological Science* 7 (6): 615–31.

Palanza, Paola, Kembra L. Howdeshell, Stefano Parmigiani and Frederick vom Saal. 2002. "Exposure to a low dose of bisphenol A during fetal life or in adulthood alters maternal behavior in mice." *Environmental Health Perspectives* 110 (suppl 3): 415.

Pralle, S. 2006. "The 'mouse that roared': Agenda setting in Canadian pesticides politics." *Policy Studies Journal* 34 (2): 781–99.

Purchase, Iain F.H. 2004. "Fraud, errors and gamesmanship in experimental toxicology." *Toxicology* 202 (1-2): 1–20.

Ropeik, David. 2010. *How Risky Is It, Really?* McGraw-Hill Professional.

Roy, Jeffrey. 2013. *From Machinery to Mobility: Government and Democracy in a Participative Age*. New York: Springer.

Ryan, Bryce C., Andrew K. Hotchkiess, Kevin M. Crofton and L. Earl Gray, Jr. 2010. "In utero and lactational exposure to bisphenol A, in contrast to ethinyl estradiol, does not alter sexually dimorphic behavior, puberty, fertility, and anatomy of female LE rats." *Toxicological sciences: An official journal of the Society of Toxicology* 114 (1): 133–48.

Rowe, Sylvia, Nick Alexander, Fergus M. Clydesdale, Rhona S. Applebaum, Stephanie Atkinson, Richard M. Black, Johanna T. Dwyer, Eric Hentges, Nancy A. Higley, Michael Lefevre, Joanne R. Lupton, Sanford A. Miller, Doris L. Tancredi, Connie M. Weaver, Catherine E. Woteki and Elaine Wedral. 2009. "Funding Food Science and Nutrition Research: Financial Conflicts and Scientific Integrity." *The American Journal of Clinical Nutrition* 89 (5): 1285–91.

Scott, Dayna Nadine. 2009. "Testing Toxicity: Proof and Precaution in Canada's Chemicals Management Plan." *Review of European Community & International Environmental Law* 18 (1): 59–76.

Sharpe, Richard M. 2010. "Is it time to end concerns over the estrogenic effects of bisphenol A?" *Toxicological sciences: An official journal of the Society of Toxicology* 114 (1): 1–4.

Shelby, M. 2008. *Potential Human Reproductive and Development Effects of Bisphenol A. NIH Publication no. 08-5994*. Washington: Department of Health and Human Services.

Slovic, P., T. Malmfors, Daniel Krewski, C.K. Mertz, Nancy Neil and Sheryl Bartlett. 1995. "Intuitive toxicology. II. Expert and lay judgments of chemical risks in Canada." *Risk Analysis* 15 (6): 661–75.

Slovic, Paul, Melissa Finucane, Ellen Peters and Donald G. MacGregor. 2002. "The Affect Heuristic." In *Heuristics and Biases: Intuitive Judgment*, ed. Thomas Gilovich, Daniel Kahneman, and Dale Griffin. Cambridge: Cambridge University Press.

Slovic, Paul, Baruch Fischhoff and Sarah Lichtenstein. 1982. "Why Study Risk Perception?" *Risk analysis* 2 (2): 83–93.

Smith, R. and A. Daifallah. 2006. "It's in Tory genes to go green". *Globe and Mail* (Toronto). February 6, 2006.

Smith, R. and B. Lourie. 2010. *Slow Death by Rubber Duck: How the Toxic Chemistry of Everyday Life Affects Our Health*. Toronto: Random House.

Sunstein, Cass. 2004. *Risk and Reason: Safety, Law, and the Environment*. Cambridge: Cambridge University Press.

Sutton, P., T.J. Woodruff and S. Vogel. 2011. "Conrad and Becker's '10 Criteria' Fall Short of Addressing Conflicts of Interest in Chemical Safety Studies." *Environmental Health Perspectives* 119 (12): 506–09.

Teeguarden, Justin G. and Sesha Hanson-Drury. 2013. "A Systematic Review of Bisphenol A 'Low Dose' Studies in the Context of Human Exposure: A Case for Establishing Standards for Reporting 'LowDose' Effects of Chemicals." *Food and Chemical Toxicology* 62: 935–48.

Tyl, R.W., C.B. Myers, M.C. Marr, B.F. Thomas, A.R. Keimowitz, D.R. Brine, M.M. Veselica, P.A. Fail, T.Y. Chang, J.C. Seely, R.L. Joiner, J.H. Butala, S S. Dimond, S. Z. Cagen, R.N. Shiotsuka, G.D. Stropp and J.M. Waechter. 2002. "Three-generation reproductive toxicity study of dietary bisphenol A in CD Sprague Dawley rats." *Toxicological Sciences: An official journal of the Society of Toxicology* 68 (1): 121–46.

Tyl, R., C. Myers, M. Marr and C. Sloan. 2008. "Two-generation reproductive toxicity study of dietary bisphenol A (BPA) in CD-1 (Swiss) mice." *Toxicological Sciences* 104.(2): 362–84.

United States. Environmental Protection Agency. 1990. *Reducing Risk: Setting priorities and strategies for environmental protection*. Washington DC.

United States. National Institute of Environmental Health Sciences. 2010. *Bisphenol A (BPA): Expanding Research to Impact Human Health*. http://www.niehs.nih.gov/research/supported/recovery/critical/bpa/ (July 18, 2014).

Vandenberg, L.N., T. Colborn, T.B. Hayes, J.J. Heindel, D.R. Jacobs, Jr., D.H. Lee, T. Shioda, A.M. Soto, F.S. vom Saal, W.V. Welshons, R.T. Zoeller and J.P. Myers 2012. "Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses." *Endocrine Reviews* (33) 1: 1–75.

Vandenberg, L.N., M.V. Maffini and C. Sonnenschein. 2009. Bisphenol-A and the great divide: A review of controversies in the field of endocrine disruption. *Endocrine Reviews* 30 (1): 75–95.

Völkel, W., T. Colnot, G.A. Csanády, J.G. Filser and W. Dekant. 2002. "Metabolism and kinetics of bisphenol A in humans at low doses following oral administration." *Chemical research in toxicology* 15(10): 1281–87.

Vogel, D. 2012. *The Politics of Precaution: Regulating Health, Safety, and Environmental Risks in Europe and the United States*. Princeton: Princeton University Press.

Vom Saal, F., B. Timms, and M. Montano. 1997. "Prostate enlargement in mice due to fetal exposure to low doses of estradiol or diethylstilbestrol and opposite effects at high doses." *Proceedings of the National Academy of Sciences* 94: 2056–61.

Vom Saal, F. and W. Welshons. 2006. "Large effects from small exposures II. The importance of positive controls in low-dose research on bisphenol A". *Environmental research*. 100: 50–76.

Vom Saal, F. Paul S. Cooke, D.L. Buchanan, P. Palanza, K.A. Thayer, S.C. Nagel, S. Parmigiani and W.V. Welshons. 1998. "A physiologically based approach to the study of bisphenol A and other estrogenic chemicals on the size of reproductive organs, daily sperm production, and behavior." *Toxicology and Industrial Health* 14 (1–2): 239–60.

Vom Saal, Frederick S. *et al*. 2007. "Chapel Hill Bisphenol A Expert Panel Consensus Statement: Integration of Mechanisms, Effects in Animals and Potential to Impact Human Health at Current Levels of Exposure." *Reproductive toxicology* 24(2): 131–38.

Waterloo Watch. 2010. "Fluoridation: It's just not worth the risk." http://waterloowatch.com (July 20, 2014).

Welshons, W.V., K.A. Thayer and B.M. Judy. 2003. "Large Effects from Small Exposures. I. Mechanisms for Endocrine-Disrupting Chemicals with Estrogenic Activity." *Environmental Health Perspectives* 111 (8): 994–1006.

Welshons, W., and S. Nagel. 2006. "Large Effects from Small Exposures. III. Endocrine Mechanisms Mediating Effects of Bisphenol A at Levels of Human Exposure." *Endocrinology* 147 (6): S56–S59.

Wildavsky, Aaron. 1997. *But Is It True? A Citizen's Guide to Environmental Health and Safety Issues*. Cambridge: Harvard University Press.

Young, S. Stanley and Alan Karr. 2011. "Deming, data and observational studies." *Significance* 8 (3): 116–20.