An Archaeological-Genealogical Analysis of Public Health Discourse on Lead: Reformulating lead-based paint as a problem in Canada

by

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ABSTRACT

Lead is a serious developmental neurotoxin with the capacity to interrupt brain development and impair functioning. Since at least 1930 numerous case studies in American, Canadian and Australian literature have identified lead based paint in the home as a source of poisoning for young children; and since at least 1990 evidence has shown that it is the lead dust from deteriorating paint in older homes and renovating activities that is the primary source of chronic exposure for young children today.

Not much is known about the extent of childhood lead poisoning in Canada. Gaps in our understanding include a lack of national survey data on childhood blood lead levels and an absence of reliable data to determine the era of housing that poses the greatest risk. This thesis posits that despite this paucity of research knowledge there is evidence to suggest that populations of vulnerable children continue to be harmed by exposure to historic sources of lead, such as lead-based paint found in older housing stock. This thesis examines the evidence to support this contention by critically analyzing the Canadian public health response to the issue of childhood lead poisoning. Specific attention is paid to discourse corresponding to lead-based paint, the putative major pathway of exposure for children ages 1 to 5 years.

Using Foucault’s genealogical/archaeological approach, as elaborated upon by Rawlinson (1987) this thesis discusses the socio-political and economical processes that shaped health care knowledge regarding childhood lead poisoning in Canada and influenced the way knowledge was produced and used by health care providers and policy makers. The analysis is assisted via a comparison of Canadian public health discourse with American discourse, with an emphasis on discourse appearing in the post leaded gasoline era (1990-2008). The strength of a Foucauldian archaeological/genealogical analysis for nursing research and particularly for this analysis is in its focus on discourse, surfaces of emergence, transformations, mutations, contingencies, events, recognition of power/knowledge strategies, descriptions of discipline technologies and consequences, and suggested possibilities of resistance.

This thesis proposes that surveillance data constituted both a product and a catalyst of the dominant view on childhood lead poisoning occurring from residential sources and posits that a lack of Canadian context specific surveillance data was the major “policing” factor limiting Canadian public health discourse. Further, privileged access to blood lead survey data maintained the view that childhood lead poisoning was a problem of the past or an American problem. Third, tensions among Canada’s two federal agencies which hold primary responsibility for lead, health and housing resulted in a weakened response whereby, to date, no legislation exists to protect vulnerable populations of Canadian children from exposure to historic sources of lead in residential dwellings.
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PART I: OVERVIEW OF THE STUDY

1.1. Statement of the problem

In August 2010 Health Canada released findings from the Canadian Health Measures Survey (CHMS), a nationally representative investigation, involving over 5,000 Canadians (Bushnik et al., 2010). The survey collected information on diet and lifestyle as well as biomonitoring data such as blood and urine samples to analyze levels of various contaminants typically found in Canadians. Lead was one of three metals sampled. Survey findings indicate that average blood lead levels of Canadians, aged 6 to 79 years have declined considerably in the thirty year span since the last nation-wide survey of blood lead levels occurred in Canada, in 1978 (Statistics Canada, 2008). These results, at first glance, suggest a public health victory and appear to support Canada’s policy approach regarding childhood lead poisoning, which to date has focused primarily on lead in consumer products. The Daily reports that fewer than 1% of Canadians now have blood lead concentrations above the Health Canada guidance value of 10 micrograms per decilitre (BPb ≥ 10 μg/dL) (Statistics Canada, 2010). The decline in blood lead values, described in the media as “dramatic” and “drastic” is attributed to the phase-out of leaded gasoline, lead-containing paints and lead solder in food cans since the 1970s (Bouzane, 2008; CBC News, 2008; Statistics Canada, 2008; Toronto Sun, 2008).

This thesis posits that while average blood lead levels of the population have declined in the last 30 years there is evidence to suggest that populations of vulnerable children continue to be affected by historic sources of lead found in older housing stock. This thesis examines the evidence to support this contention by critically analyzing the Canadian public health response to the issue of childhood lead poisoning. Specific attention is paid to discourse corresponding to
lead-based paint, the putative major pathway of exposure for children ages 1 to 5 years (American Academy of Pediatrics, 2005). The overarching purpose of this thesis is to illuminate the socio-political and economic influences that shape healthcare knowledge, and ultimately, influence how healthcare providers, researchers, and policy makers produce and use this information. This thesis uses the case of lead poisoning as an exemplar to study this process.

Lead is a serious developmental neurotoxin with the capacity to interrupt brain development and impair functioning. Twelve million children in developing countries suffer from some form of permanent brain damage from lead poisoning and about 3.5 percent of minor mental retardation worldwide is attributable to lead poisoning (Fewtrell et al., 2004; Prüss-Üstün, 2004). Children identified with blood lead levels at levels less than our current level of concern (10 g/dL) are more likely to suffer lower IQ scores, poorer school performance and be diagnosed with attention-deficit/hyperactivity disorder (ADHD) than less exposed peers (CDC, 2005; Nigg et al., 2008; Nigg et al., 2010). Lead is also harmful to kidneys, blood-forming systems, and neuroendocrine and reproductive systems (Health Canada, 2005; Lockitch, 1993). Other health effects of lead exposure are summarized in Table 1.

Since at least 1930, numerous case studies in American, Canadian and Australian literature have identified lead based paint in the home as a source of poisoning for young children (Rabin, 1989); and since at least 1990 evidence has shown it is the lead dust from deteriorating paint in older homes and renovating activities that is the primary source of chronic exposure for young children today (Levin et al., 2008). All lead painted surfaces deteriorate with time, creating lead dust (Marcus & Elias, 1995). Lead dust found on floors and surfaces is the most common pathway of exposure for young children who, as toddlers, explore their
environment through hand to mouth behaviour (Moya et al., 2004; Bellinger, 2004; CDC, 2005a; 2005b; AAP, 2005). Oftentimes one or two dwellings are responsible for multiple cases of lead poisoning as affected families move out and new, unsuspecting, families move in to lead contaminated homes (Reissman et al., 2001).

The problem is not self mitigating. Homes containing lead hazards require remediation by experts with specialized knowledge and training in the safe removal of lead. Remediation measures range from repainting to the complete removal of all lead painted surfaces from a dwelling (Dixon et al., 2005).

1.1.1. The concept of lead poisoning

One hundred years ago, the term “lead poisoning” described a condition wherein children’s blood lead levels were dangerously elevated, to the point where cases of lead poisoning were identified based on the appearance of overt clinical symptoms. The diagnosis of childhood lead poisoning in the early 1900’s was, by necessity, reliant on a physician’s informed assessment of symptoms such as pallor, convulsions and coma (English, 2001). Unfortunately, however, the cerebral manifestations of lead poisoning mimicked other diseases which made this illness prone to misidentification, a point in which Mitchell, a Montreal pediatrician, raised in his 1932 discussion:

During the last 36 to 48 hours girl had developed a squint. This led the family physician to strongly suspect tuberculous meningitis; she was also quite pale and the question of a co-existing blood dyscrasia was raised (p. 547). Today lead-poisoning is recognized as a “silent disease.” There are no clinical or overt symptoms to identify children at our current blood lead level of
concern (CDC, 1991a; Needleman, 1990). The definition of lead poisoning has evolved over time and values in Canada and the United States of what constitutes lead poisoning cases have been lowered as knowledge of the damaging cognitive effects of lead exposure associated with lower blood lead levels has become apparent.

The CDC (2005b) bases their determination of poisoning cases, in general, on three parameters: clinical description, laboratory criteria for diagnosis, and case classification. In Canada and the U.S., the determination of lead ‘poisoning’ is typically identified on the basis of only one criteria: a capillary or venous blood lead level equal to or exceeding a blood lead standard of 10 micrograms of lead per deciliter blood (PbB ≥ 10 μg/dL) (Health Canada, 2005; CDC, 2005a). Use of the term “lead poisoning” to represent an unhealthy dose-response relationship (rather than an overt set of symptoms) distinguishes ‘lead poisoning’ as separate from the classification of other chemical poisonings, where, for example abnormal laboratory parameters such as an imbalance in electrolyte levels, for example, or an abnormal renal function test must first be established before a case is categorized as suspected or probable for chemical poisoning (CDC, 2005b). In developed nations, blood lead sampling is the most widely employed indicator for identifying cases of lead poisoning, although other biomarkers of lead exposure have also been developed (McBride, 2008). Table 2 summarizes the various biomarkers of lead exposure and their associative value for predicting measurable health effects.

Pathways of lead exposure. Humans are exposed to lead primarily through four routes: ingestion, inhalation, skin absorption, and maternal transfer of lead during pregnancy and lactation (ATSDR, 2005). The most common pathway of
lead exposure for children today is through contact with, and ingestion of, interior and exterior dust which is derived from the natural and gradual deterioration of lead-based paint over time (CDC, 2007). Clinical manifestations of chemical poisonings, including lead poisoning, will vary according to individual differences (i.e. genetic make-up, gender, or age), route of exposure and amount and duration of exposure (CDC, 2005b). The degree to which lead will be absorbed from the intestine following ingestion depends on the age of the child, fasting status, and nutritional factors such as intake of calcium and iron (ATSDR, 2005). Children can also breathe in lead dust suspended in air. All lead, regardless of amount, once inhaled deeply into the lung is eventually absorbed (IARC, 2006).

**Toxicokinetics of lead.** The excretory half-life of lead in blood in adult humans is approximately 30 to 36 days (ATSDR, 2005). There is limited data examining the half-life of lead in blood in children (ATSDR, 2005). Blood lead levels generally peak between the years of 18 and 36 months of age, and declines thereafter (Dixon et al., 2009). Blood lead levels will decline from the time of initial exposure as lead moves into long-term storage in bone and soft tissue (CDC, 2007). Skeletal bone is the major long-term storage compartment for lead, with a half-life of approximately 27 years (ATSDR, 2005). Lead is released from bone and transferred to blood during pregnancy and lactation (ATSDR, 2005). The placental barrier does not inhibit lead transport during pregnancy and therefore maternal and fetal blood lead levels can be identical (Goyer, 1990). Maternal lead can also be transferred to breastmilk, presumably through plasma (Ettinger et al., 2004). Prenatal exposure to lead is particularly harmful to the developing fetus (Gomaa et al., 2002; Hernandez-Avila et al., 2002; Rothenberg et al., 1995; Schnaas et al., 2006; Zhu et al., 2010).
Health Canada’s blood lead standard. Recent research on the effects of lead taken from in vitro, animal cohort and cross-sectional studies indicate that lead is harmful at PbB ≤ 10 µg/dL (CDC, 2007). Table 3 chronicles revisions to Canada’s blood lead standard for non-industrially exposed adults and children as standards evolved between 1978 and 1994.

A “safe” lead exposure threshold for children related to impaired cognitive function and heme synthesis has not yet been established (Wigle & Lanphear, 2005). This is partially due to the fact that determination of a protective BPb standard, or threshold is fraught with uncertainty stemming from residual confounding, from, for example, social factors (i.e. parental intelligence, socioeconomic status, and quality of the home environment), which weakens the strength of association between blood lead values and endpoints such as cognitive function (CDC, 2007). Other factors such as differing methods of blood collection, age of child at time of collection, seasonal variation in blood lead levels and laboratory error also weaken the association (CDC, 2007). U.S. Federal regulations, for example, allow laboratories performing blood lead testing to operate within an allowable error rate (proficiency rate) of ±4 µg/dL or ±10% (CDC, 2007). Mandatory proficiency testing is not a requirement in Canada. Of added concern is that the relation of child’s age to vulnerability is not well understood and few studies consider the contribution of maternal blood lead values on infant/child IQ scores (Schnaas et al., 2006; Gomaa et al., 2002).

A blood lead standard of ‘10’ was originally intended as a guidance point at which public health intervention became practical. This level, however, is often misinterpreted as an acceptable exposure level or a definitive toxicological threshold below which harm does not occur (CDC, 2005; Wilson et al., 2005). In 2009, the German Commission on Human Biomonitoring bypassed the large
uncertainty that comes with setting what is now perceived as an arbitrary (and non-
protective) blood lead standard by establishing a reference PbB value for German
children ages 3 –14 years of 3.5 µg/dL, a level based on the 95th percentile for
national population values (Wilhelm et al., 2010).

For the purpose of this thesis “lead poisoning” is defined as PBb ≥ 10
µg/dL) which is consistent with Health Canada’s and the U.S. Center for Disease
Control and Prevention’s current blood lead intervention level (Health Canada,
2005b; CDC, 2005). Blood lead levels in Canada are sometimes reported
detically, as micromoles per litre of blood (µmol/l). (10 µg/dL = 0.48 µmol/l) (Health
Canada, 2005b). Although the metric system is the official system for
measurement in Canada, this thesis preferentially reports blood lead levels as
micrograms per deciliter of blood to facilitate comparisons between U.S. and
Canadian data.

1.1.2. The extent of childhood lead poisoning in Canada and abroad

The extent of childhood lead poisoning resulting from exposure to
residential sources of lead-based paint is unknown in Canada. Blood lead-
surveillance data for children living in non-industrial communities, in general, is
sparse and little is known about the extent of Canadian dwellings that contain lead-
based paint (OPHA, 2004). To date, Quebec is the only Canadian province that
requires mandatory reporting for children and adults identified with elevated blood
lead levels (≥ 10 µg/dL) (Plante et al., 2003). Reporting requirements came into
effect in Quebec in 2003, although blood lead data has only been systematically

1 To convert to metric, or SI units (µmol/L) blood lead concentrations in units of g/dL are
multiplied by a conversion factor of 0.0483 (i.e. 10 g/dL x 0.0483 = 0.483 µmol/L).
collected for children in that province since 2006. Between 2006 and 2009, 42 cases of children aged 0 to 4 years were identified with blood lead levels ranging between 10 - 49 µg/dL (Marlene Mercier, personal communication, July 2010). In many reported cases of poisoning in Quebec, anemia was the only symptom triggering suspicion (Bailey & Bussières, 2003; Brisson & Kossowski, 2006). While reporting of elevated blood lead levels is mandatory, routine blood lead screening is not, which renders it difficult to judge the true extent of the problem, even in Quebec.

The United States (U.S.) has been systematically collecting national statistics on children’s blood lead levels since 1976 through the National Health and Nutrition Examination Survey (NHANES) (Roberts et al., 1985). NHANES selects a representative sample of children and adults, and tests, among other health indices, blood lead levels. Between 1976 and 1991 blood lead levels of the U.S. population aged 1 to 74 years declined 78%, from an average of 12.8 to 2.8 micrograms per deciliter of blood (µg/dL) (CDC, 2005). Recent surveillance reports estimate that 1.4% of US children aged 1 to 5 years continue to demonstrate PbB levels ≥10 µg/dL (Jones et al., 2009). Exposure to lead paint and dust account for 70% of reported cases in the U.S. today (Levin et al., 2008).

Internationally, numerous European countries banned, or limited the use of lead-based paint decades earlier than either Canada or the United States (Rabin, 1989). Lockhart Gibson, was the first to publicly identify lead-based paint as a source of exposure for Australian children in the late 1800’s (Gibson, 2005). Laidlaw and Taylor (2010) contend that lead-based paint continues to pose a health risk for children in Australia today and describe how investigation of residential sources of lead exposure has been neglected in favour of research occurring in smelter communities. The Australian PbB guideline for children is 10
μg/dL, but unlike Canada’s guideline, the Australian standard is age specific - pertaining to children 1 to 4 years, with a higher guidance PbB value for adult populations (NHMRC, 2009). France enacted policy to limit lead concentrations in domestic sources of paint in the early 1900’s; although, Fassin and Naude (2004) note that even then, childhood lead poisoning occurring from exposure to residential sources of lead-paint in housing continued to be an issue in France well into the 1980’s. International prevalence rates for children in various industrialized nations is available (see for example, European Environment and Health Indicators, 2007; Mayan et al., 2001; Mañay et al., 2008; McBride, 2008; Rubin et al., 2002) although to date, Germany and the United States are the only two countries, world-wide, to conduct national, periodic blood lead monitoring for non-occupationally exposed populations (Krewski et al., 2006).

To date Canada has conducted two national blood lead surveys of children and adults, one occurring from 1978-1979 (Statistics Canada & Health and Welfare Canada, 1981) and the more recent Canadian Health Measures Survey (Bushnik et al., 2010). Canadian blood lead surveillance data is less abundant than in the U.S.; however, similar to the U.S., Ontario surveys conducted during the 1980’s witnessed a sharp decline in children’s blood lead levels following the elimination of lead in paint, gasoline and lead solder in canned food (Wang et al., 1997). Further discussion regarding Canada’s two national surveys is found in the second of two articles, “Reformulating lead-based paint as a problem in Canada.”

Other sources of lead exposure in Canada. In addition to lead-based paint, residential sources of lead exposure can include drinking water (from plumbing solder, brass faucets, and lead service connectors) and soil (Maas et al., 2005; Mielke et al., 2008). Non-residential sources of lead exposure for North American
children include folk remedies such as greta, azarcon and rueda which, ironically, are remedies commonly used to treat stomach ailments; lead glazed pottery and ceramics, Mexican candy, jewellery, toys containing or painted with lead, products containing polyvinyl chloride (PVC), and synthetic turf (CDC, 2004; CDC, 2006; Levin et al., 2008; CDC, 2009; Van Ulirsch et al., 2010). The ingestion of wild game procured using lead shot is an important source of exposure for Canadian populations, especially aboriginal populations (Levesque et al., 2003; Tsuji et al., 2003). The use of khôl, an eye cosmetic applied to darken the eyelids, and commonly used by Asian populations, is a product implicated in nine cases of lead poisoning for Quebec children between 2002 and 2005 (Brisson & Kossowski, 2006).

As discussed earlier, lead-based paint is now recognized as the major source of lead exposure for American children today (CDC, 2007; Levin et al., 2008). It is likely that a problem with lead-based paint similarly threatens Canadian children. The systematic review of Canadian research pertaining to domestic sources of lead exposure (Article 1) and the critical review of Canadian research pertaining to childhood lead poisoning found in Appendix A supports this contention. Discussion from Article 2 further supports this view. For this research analysis, therefore, discussion of childhood lead poisoning in the Canadian and American setting, predominantly focuses on discourse pertaining to lead-based paint.

1.1.3. Risk factors associated with lead-based paint

The age of the dwelling. Cases of childhood lead poisoning caused by domestic sources of exposure were first reported in North America in the late 1800’s (English, 2001). The earliest published account of Canadian pediatric cases
occurring from exposure to domestic sources were described by Earle in 1874. Hamilton’s account (1905) documents a family’s poisoning caused by the burning of lead contaminated barrel staves. In 1982 Paton and Cembrowski described cases of developmentally challenged children exposed to lead from a condition known as pica, the eating of non-food substances (i.e. lead-based paint). In 2010, the extent of lead-based paint applied to the interior and exterior of Canadian dwellings remains poorly understood. The manufacture and sale of interior lead-based paint was not regulated in Canada until 2005, although paint manufacturers voluntarily agreed to restrict concentrations of lead added to interior paint in 1991 (Health Canada, 2005). To date lead can still be added to exterior paint coatings if the product carries a warning label (Health Canada, 2010). An information booklet published by Health Canada and the Canada Mortgage and Housing Corporation (CMHC) advises consumers that many Canadian homes built prior to 1960 do contain lead paint (CMHC & Health Canada, 2004) although this date is arbitrary as it is not supported by any systematic survey of Canadian dwellings as occurred in the U.S. (OPHA, 2004). The United States recognizes homes constructed prior to 1978 as the era of housing that poses the greatest risk to children due to the presence of lead-based paint (Clickner et al., 2001; Clickner & Rogers, 1995; Jacobs et al., 2002). While a firmly delineated era of concern for Canadian dwellings has not been established, various authors have speculated on what they consider to be an appropriate cut-off date. Estimates range from homes constructed prior to 1940 (Levallois et al., 1991) to structures built as recently as 1990 (McElgunn, 2004). A recent Canada-wide survey identifies that Canadian dwellings built prior to 1983 are more likely to contain lead dust on floors at levels associated with elevated blood lead levels in children (≥ 10 μg/dL) than later built homes (McDonald et al., 2010).
Renovation history. Any disturbance of lead-painted surfaces can potentially create a serious lead dust hazard. The term “renovation” signifies any activity that disturbs a painted surface. The following activities generally produce large amounts of lead dust and thus require daily and methodical clean-up of the work area: removing paint from floors and woodwork, use of power sanders, hand sanders, scrapers, torches, heat guns and chemical strippers, ceiling and wall repair, wallpaper removal, removal of woodwork trim, window and door replacement, and tearing up old carpets (HUD et al., 2001). This information has relevance within our Canadian context. Canadian home owners, for example, are more likely to undertake major home renovations in older dwellings than newer dwellings (CMHC, 2010). In 2007, 49% of homes constructed prior to 1960 were renovated in the previous year (CMHC, 2007).

Socio-economic factors. Families with fewer economic resources have less choice in where they can live and are more likely to dwell in poorly maintained older housing (Chaudhuri, 1998). In the U.S., low-income children living in pre-1946 dwellings have a 16% prevalence rate of lead poisoning versus a 4% rate for middle-income children (CDC, 2003). Lead poisoning can occur across diverse social strata since, as discussed earlier, children can also be poisoned by the lead dust generated during home renovation or repair even in well maintained homes (Jacobs et al., 2002).

1.1.4. Canadian federal policy regulating lead

Similar to the U.S., many historic sources of childhood lead exposure have already been addressed through the elimination of lead in gasoline, restriction of lead in solder, and elimination of lead in solder of food cans. Where the two countries differ is in their response to a remaining and persistent source, lead-
based paint found in older dwellings. Laws to eliminate lead-based paint have existed in the U.S. since 1970 (Richardson, 2005). Furthermore, in 1992, U.S. Congress passed the *Residential Lead-Based Paint Hazard Reduction Act*, commonly known as Title X, which apportioned funding to state and local governments to remediate sources of lead-based paint in older housing stock (U.S. Congress, 1992). Recently adopted U.S. regulations require contractors, painters and trades-people working in dwellings constructed prior to 1978, to be certified in lead precautionary measures (EPA, 2010).

In Canada, while some federal initiatives have been directed towards reducing lead in consumer products such as toys and jewelry, public health advocates and policy makers have been slow to recognize and call for action on sources of lead in housing (OPHA, 2004). To date, Canada has not drafted any federal legislation or undertaken any initiative that prevents children’s exposure to historic sources of the lead-based paint that is potentially found in Canada’s 12 million residential dwellings (Spady, 2006). Given that Canada does not have regulations in place similar to the protective measures contained in American law (i.e. Title X) it is assumed that homeowners and even contractors are not following lead precautionary measures whenever renovating and occupational exposure is likely a health risk during these activities.
1.2. Purpose of the study

Lead, a neurotoxin, interferes with children’s natural brain development. Lead-based paint is potentially a major source of Canadian children’s lead exposure today. The extent of lead-based paint use in Canadian dwellings is poorly understood, although there is sufficient evidence to support the contention that Canadian children have been harmed from exposure to historic sources of lead-based paint and dust found in Canada’s many older residential dwellings (Article 2, Appendix A). As children’s average blood lead levels declined following the elimination of a major source of exposure (leaded gasoline) in Canada, new public health discourse on lead surfaced. The purpose of this study is to critically examine Canadian public health discourse on the topic of childhood lead poisoning. The analysis is assisted via a comparison of Canadian public health discourse with American discourse, with an emphasis on text appearing in the post leaded gasoline era (1990-2008). The overarching purpose of this study is to illuminate the socio-political and economic influences that shape health-care knowledge, and ultimately, influence how healthcare providers, researchers, and policy makers produce and use this information.

1.2.1. Research Questions

This research seeks to examine: What is known and understood about childhood lead poisoning in Canada, especially as it relates to residential sources of lead exposure (i.e. lead-based paint)? What has been the Canadian public health response to this knowledge? These questions are explored via an historical analysis of Canadian public health discourses pertaining to lead-based paint beginning from when this topic first emerged in Canadian health-related literature.
in the 1930’s. Two events mark important transition points in Canadian discourse on lead and are important to this study: the banning of leaded gasoline in North America circa 1990 and the release of preliminary findings from Canada’s second national blood lead survey (2008). The year 2008 thus marks a convenient endpoint for this analysis (1932-2008). Through critical examination this thesis further seeks to explore the following questions: What were the dominant discourses around childhood lead poisoning in Canada during this time frame? How were these shaped and supported and what effect did these dominant discourses have on the perception and management of childhood lead poisoning?

1.2.2. Research Objectives

Four objectives guide this study:

1. To examine the discourses around lead poisoning as a public health issue and highlight tensions amongst various discourses;

2. To identify gaps in research and policy on childhood lead poisoning;

3. To discuss socio-political and economical processes that shape health care knowledge and influence the way knowledge is produced and used by health care providers and policymakers;

4. To apply social critique as a means of determining possible social change and transformation.

The importance of this analysis is discussed in the following section.

1.2.3. Significance of the problem and of this study

The Canadian price of “environmental disease” is estimated to cost $46 billion to $62 billion per year (Muir & Zegarac, 2001) whereas environmental illnesses such as childhood lead poisoning, asthma, cancer, and neurobehavioral
disorders cost United States taxpayers are estimated to cost $54.9 billion annually (Hewitt et al., 2006).

As discussed, children less than 6-years are particularly vulnerable to lead exposure for developmental reasons (AAP, 2005). Although a BPb ≥10 g/dL is considered, by federal standards, elevated, Health Canada recognizes the absence of a lower threshold for some of the adverse effects occurring in lead exposed children. The “Regulatory Impact Analysis” statement (RIAS) which accompanies recent federal regulations pertaining to lead content in children’s jewellery acknowledges that the avoidance, in children, of BPb ≤ 5 µg/dL is justifiable based on a rudimentary cost benefit analysis which considers the additional costs to society of $6,000 -10,000 per child per year accrued from medical treatment and special education expenditures alone (Health Canada, 2005b). Importantly, note that Health Canada’s estimate does not include the added cost to society derived from criminal justice related measures, the increased need for mental health services, or the loss of lifetime earnings, as has been calculated elsewhere (see for example, Davies, 2005). Assuming a 5% prevalence rate (BPb ≥5 µg/dL) the annual cost to Canadian society resulting from lead exposure costs between $89 -148 million (CDN) (Table 4).

Cohen-Cole estimates that being a child raised in an “old vintage house” results in lost future earnings equivalent to missing 6½ years of school (Cohen-Cole, 2006, p.757). The Learning Disabilities Association of Canada (LDAC, 2002) estimates the direct costs to individual with learning disabilities, from birth to retirement, accrued from additional medical care expenditures, educational and criminal justice services and reduced lifetime earnings at $1.9 million (CDN) per person. The yearly cost per person with a learning disability, amounts to $455,208. Assuming a 5% rate of learning disability rate occurs in the Canadian population,
the LDAC conservatively estimates the cost to society, from birth to retirement, in
the range of $707 billion. For comparison, the prevalence rate of Fetal Alcohol
Syndrome (FAS) is estimated to fall somewhere between 0.005 and 0.02% of all
live births with a total lifetime cost of caring for a child with FAS of $1.4 million
(Goh et al., 2010).

The CDC, in an addendum to their 1991 Strategic plan for the elimination of
childhood lead poisoning, weighed the cost of lead poisoning in terms of additional
medical treatment and special education expenses against the benefits of
remediation achieved through the abatement of all U.S. housing containing lead-
based paint. The report concluded that benefits of prevention out-weighed the cost
to society occurring from childhood lead exposure. The CDC further advised that
even if evidence of an economic benefit had not been established, the prevention
of childhood lead poisoning (through lead-based paint abatement) was a practical
and worthwhile public health activity based on ethical reasons alone (CDC, 1991).

Nevin and Jacobs (2006) in a restrospective analysis, concluded that the
replacement of lead-painted windows in federally assisted units reduced the
incidence of childhood lead poisoning in the United States considerably but also
improved home energy efficiency and affordability. A similar cost-benefit analysis
for home remediation in Canada is not available, although a CMHC case study
determined that replacing single pane windows with energy efficient models in a
test residential unit resulted in an annual gas energy savings of $600 (CDN)
(CMHC, 2004). Gould (2009) estimates a savings of $17-221 (US) per child for
every dollar spent on lead-based paint remediation (i.e. paint stripping,
replacement and encapsulation). Clearly, the cost benefits of primary prevention
outweigh the costs derived from secondary and tertiary prevention strategies.
1.2.4. Relevance of this health issue to nursing

The discipline of nursing is said to be defined by the four domains of health, nursing, person, and environment (Fawcett, 1984), yet, many baccalaureate nursing programs include sparse content on the impact of the environment on health (Hewitt et al., 2006; Kjellstrom et al., 2008; Leman Stefanovic & Wiseman, 2008; Sweeney & de Peyster, 2005). This may be because traditionally, the concept of environment, as applied in nursing, has been conceptualized narrowly, in a physical sense, for example, referring to the patient’s hospital room; or, in its widest application, to the patient’s geographic surroundings (Kleffel, 1991).

Kleffel, in reviewing major nurse theorists’ perspectives regarding the domain of environment, concluded that “[n]ursing theorists have given little attention to developing the concept [of environment]; its definition is ambiguous and vague, and it lacks conceptual unity” (Kleffel, 1991, p.48). Some scholars regard nursing’s myopic view of the environment as the underlying reason why the profession has played such a minor role in effecting change in the social, political and economic realms (Chopoorian, 1986; Kleffel, 1991). Kleffel foresees a major paradigmatic shift in nursing leading to “a revolution in nursing’ consciousness of the environmental domain” (Kleffel, 1991, p.47). As early as 1991 Kleffel predicted a world:

confronted with a global ecological crisis that has placed humans’ fulfillment and even survival in jeopardy. It now seems clear that nursing will have to move ahead as an integral part of the global ecologic and environmental movement or get left behind. We must now view human life and health as a multidimensional unity with the environment (1991, p.48).

A recent policy paper by the Canadian Nurses Association suggests that the nursing conception of environment in Canada is expanding through its explicit description of the various chemical, physical, biological, and psychosocial agents
encountered by individuals at home, in school and at the workplace and which impact human health (2007).

Whereas nursing initiatives in the acute care setting have traditionally focused on interventions after illness has occurred, environmental health initiatives typically begin *before* the onset of illness (Hewitt et al., 2006). In environmental health, prevention is key. Ideally, initiatives are geared towards the protection of fetuses, infants, and children from exposure to environmental contaminants because these populations are particularly vulnerable to exposure to hazardous substances due to developmental immaturity, a proportionately greater dietary intake per body weight, greater hand to mouth behaviour; and a greater surface-to-body mass ratio (Hewitt et al., 2006). An expanded nursing environmental domain also recognizes the importance of occupational health and the need to protect adult workers and the elderly from environmental exposures.

Nurses working in the field of environmental health also recognize that prevention of illness cannot be maintained simply through awareness. Dixon and Dixon (2002) suggest an “upstream” approach which combines knowledge about biological systems with behavioural dynamics and policy. Chapman and Lupton (1994) advocate for an approach focused on modifying economic, political and environmental factors. Larsson and Butterfield (2002) devised a roadmap for positioning environmental health curricula into nursing schools and mainstream practice. Kleffel encourages nurses to:

> explain oppressive environmental effects on health...by uncovering the relation of dominance and by demystifying the ideology that rationalizes unequal power relations...which will allow nurses to analyze environmental constraints upon freedom and health (Kleffel, 1991, p.48).

Clark, Barton, and Brown (2002) suggest a parallel expansion of nursing knowledge in environmental health by applying critical theory to community
concerns regarding environmental exposures and contaminated sites. They reason that this approach can lead to the emancipation and empowerment of individuals and communities, and that social justice can therefore be achieved.

This thesis builds upon these initiatives and advances the discipline of nursing by further increasing knowledge and expertise in the area of environmental health. An important role for a primary care Advanced Practice Nurse (APN), as advocated by this author, will be to critically examine the discourse on lead poisoning in Canada, identify discrepancies between knowledge and practice, suggest reasons for those discrepancies, and make recommendations for change.
Table 1. Adverse effects of lead exposure on health (Adapted from Jin et al., 1997; CDC, 2005a, Wilhelm et. al, 2010)

<table>
<thead>
<tr>
<th>Category</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Increased blood pressure</td>
</tr>
<tr>
<td></td>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td></td>
<td>Electrocardiographic abnormalities</td>
</tr>
<tr>
<td>Endocrine/metabolic</td>
<td>Decreased thyroxine levels in adults</td>
</tr>
<tr>
<td></td>
<td>Decreased 1,25-dihydroxy vitamin D in serum in children</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Colic</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Increased ALA synthetase activity</td>
</tr>
<tr>
<td></td>
<td>Decreased ALA dehydratase activity</td>
</tr>
<tr>
<td></td>
<td>Increased ALA in blood, plasma or urine</td>
</tr>
<tr>
<td></td>
<td>Increased erythrocyte protoporphyrin</td>
</tr>
<tr>
<td></td>
<td>Increased zinc protoporphyrin in children</td>
</tr>
<tr>
<td></td>
<td>Increased urine coproporphyrin</td>
</tr>
<tr>
<td></td>
<td>Decreased hemoglobin</td>
</tr>
<tr>
<td></td>
<td>Decreased pyrimidine-5'-nucleotidase activity in children</td>
</tr>
<tr>
<td>Immune</td>
<td>Decreased cell-mediated immune function</td>
</tr>
<tr>
<td>Nervous</td>
<td>Encephalopathy</td>
</tr>
<tr>
<td></td>
<td>Neurological symptoms and signs</td>
</tr>
<tr>
<td></td>
<td>Impaired peripheral nerve conduction</td>
</tr>
<tr>
<td></td>
<td>Reduced neurobehavioral test performance</td>
</tr>
<tr>
<td></td>
<td>Reduced auditory acuity in children</td>
</tr>
<tr>
<td>Renal</td>
<td>Chronic nephropathy</td>
</tr>
<tr>
<td></td>
<td>Renal impairment with gout or hypertension</td>
</tr>
<tr>
<td></td>
<td>Aminoaciduria or Fanconi Syndrome in children</td>
</tr>
<tr>
<td>Reproductive</td>
<td>Increased frequency of stillbirth</td>
</tr>
<tr>
<td></td>
<td>Increased frequency of spontaneous abortion</td>
</tr>
<tr>
<td></td>
<td>Reduced sperm production or motility</td>
</tr>
<tr>
<td></td>
<td>Increased % abnormal spermatocytes</td>
</tr>
<tr>
<td>Developmental</td>
<td>Reduced growth in children</td>
</tr>
<tr>
<td></td>
<td>Impaired mental development in children</td>
</tr>
<tr>
<td></td>
<td>Decreased birth weight or head circumference</td>
</tr>
<tr>
<td></td>
<td>Decreased gestational age at birth</td>
</tr>
<tr>
<td></td>
<td>Increased neonatal death ratio</td>
</tr>
<tr>
<td></td>
<td>Tooth decay</td>
</tr>
</tbody>
</table>
Table 2. Biomarkers of exposure to lead related to human health effects (Adapted from Jin et al., 1997)

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Type of validity</th>
<th>Related health effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pb in whole blood</td>
<td>Predictive</td>
<td>All known health effects</td>
</tr>
<tr>
<td>Pb in deciduous tooth dentin</td>
<td>Predictive</td>
<td>Growth and mental development</td>
</tr>
<tr>
<td>Chelatable Pb (3 day urine collection)</td>
<td>Predictive</td>
<td>Renal function</td>
</tr>
<tr>
<td>Pb in maternal blood</td>
<td>Predictive</td>
<td>Child’s growth and mental development</td>
</tr>
<tr>
<td>Pb in umbilical cord blood</td>
<td>Predictive</td>
<td>Child’s growth and mental development</td>
</tr>
<tr>
<td>Pb in placenta</td>
<td>Predictive</td>
<td>Birth weight, head circumference</td>
</tr>
<tr>
<td>Pb in plasma or serum</td>
<td>Criterion</td>
<td>Pb in whole blood</td>
</tr>
<tr>
<td>Pb in tibial bone (in-vivo x-ray fluorescence)</td>
<td>Criterion</td>
<td>Chelatable Pb (3-day urine collection)</td>
</tr>
<tr>
<td>Pb in breast milk</td>
<td>Criterion</td>
<td>Pb in whole blood</td>
</tr>
</tbody>
</table>
Table 3. Timeline describing emergence of Canadian blood lead standards

<table>
<thead>
<tr>
<th>Year</th>
<th>Blood lead level of concern</th>
<th>Reference population</th>
<th>Reference Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>40 μg/dL</td>
<td>No age group specified</td>
<td>(Statistics Canada &amp; Health and Welfare Canada, 1981)</td>
</tr>
<tr>
<td>1987</td>
<td>30-35 μg/dL</td>
<td>Adult males</td>
<td>(Health and Welfare Canada, 1987)</td>
</tr>
<tr>
<td></td>
<td>20-25 μg/dL</td>
<td>Adult females</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20-25 μg/dL</td>
<td>Children (no age specified)</td>
<td></td>
</tr>
<tr>
<td>1994</td>
<td>10 μg/dL</td>
<td>All age groups except for those occupationally exposed.</td>
<td>(Health Canada, 2005)</td>
</tr>
</tbody>
</table>
Table 4. The estimated cost to Canadian society for children identified with BPb ≥5 µg/dL

<table>
<thead>
<tr>
<th>Construction period</th>
<th>Children 0-4 yrs living in dwelling</th>
<th>5% assumed “at risk”</th>
<th>Extra medical and special education cost (millions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1920 or before</td>
<td>23,806</td>
<td>1,190</td>
<td>7.1-11.9</td>
</tr>
<tr>
<td>1921-1945</td>
<td>29,385</td>
<td>1,469</td>
<td>8.8-14.6</td>
</tr>
<tr>
<td>1946-1960</td>
<td>52,756</td>
<td>2,638</td>
<td>15.8-26.3</td>
</tr>
<tr>
<td>1971-1980</td>
<td>73,936</td>
<td>3,697</td>
<td>22.1-36.9</td>
</tr>
<tr>
<td>1981-1985</td>
<td>27,823</td>
<td>1,391</td>
<td>8.3-13.9</td>
</tr>
<tr>
<td>1986-1990</td>
<td>34,874</td>
<td>1,744</td>
<td>10.4-17.4</td>
</tr>
<tr>
<td>Total</td>
<td>298,944</td>
<td>14,884</td>
<td>89-148</td>
</tr>
</tbody>
</table>
1.3. Literature review

Not much is known about the extent of childhood lead poisoning in Canada. Gaps in our understanding include a lack of national blood lead surveillance data and an absence of reliable survey information to determine the era of housing that poses the greatest risk of harm to children from lead-based paint. The first of two articles in this thesis, “Applying Dixon and Dixon’s Integrative Model for Environmental Health Research towards a critical analysis of childhood lead poisoning” summarizes 24 case studies, blood lead surveys and environmental investigations which support the contention that Canadian children have been exposed, and in some cases harmed, from exposure to residential sources of lead, predominantly from lead-based paint, paint dust and lead contaminated drinking water. A detailed critical review for 15 of these investigations is included in Appendix A and forms the basis for Article 1 and the subsequent critical analysis (Article 2).

The article was published in Advances in Nursing Science in March, 2010, in a special theme issue entitled Critique & Replication. A PDF copy of the article is included in Appendix B.
ARTICLE 1

Applying Dixon and Dixon’s Integrative Model for Environmental Health Research

Toward a Critical Analysis of Childhood Lead Poisoning in Canada
Applying Dixon and Dixon’s Integrative Model for Environmental Health Research Toward a Critical Analysis of Childhood Lead Poisoning in Canada

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Amélie Perron, RN, PhD, Assistant Professor, School of Nursing, Faculty of Health Sciences, University of Ottawa

Abstract

Occurrences of childhood lead poisoning resulting from exposure to residential sources of lead is an underresearched area in Canada. Dixon and Dixon’s Integrative Model for Environmental Health Research substantiates this claim by grouping Canadian research on this health topic into the model’s four domains: physiological, vulnerability, health protection, and epistemological. This process is useful for identifying research gaps within the Canadian context but in also setting the groundwork for a future critical analysis to illuminate the socio-political and economic influences that shape health-care knowledge, and ultimately, influence how healthcare providers and policy makers produce and use this information.

Key words

Canada, child health, critical analysis, English, environmental health, environmental toxicology, health screening, housing, infancy and childhood, lead poisoning, nursing theories, prevention and control, risk factors

2 The actual citation is as follows: Perron, A.& O’Grady, K. (2010). Applying Dixon and Dixon's Integrative Model for Environmental Health Research towards a critical analysis of childhood lead poisoning in Canada. Advances in Nursing Science, 33(1), E1-E16. K. O’Grady contributed to the conception and design of the article and to the acquisition of data, led the writing and completed the initial analyses. A. Perron contributed to the editing and revision of the manuscript and approved the final version. For purposes of correspondence, A. Perron was designated by Advances in Nursing Science editorial staff as the lead author.
Introduction

Environmental illnesses such as childhood lead poisoning, asthma, cancer, and neurobehavioral disorders cost United States taxpayers $54.9 (US) billion annually (Landrigan et al., 2002). Similarly in Canada, the price of “environmental disease” is estimated to cost upwards of $30 billion (CAN) per year (Muir & Zegarac, 2001). Lead is a serious environmental toxin with the capacity to interrupt brain growth and impair functioning in developing fetuses and children. Twelve million children in developing countries suffer from some form of permanent brain damage due to lead poisoning and, about 3.5 % of minor mental retardation is attributable to lead poisoning worldwide (Fewtrell et al., 2004). Affected children can suffer lower IQ scores, poorer school performance and negative behavioral effects such as increased aggression and later criminal behaviour (American Academy of Pediatrics, 2005).

Since at least 1930 numerous case studies in American, Canadian and Australian literature have identified lead based paint in the home as a source of poisoning for young children (Rabin, 1989); and since at least 1990 evidence has shown that it is the lead dust from deteriorating paint in older homes and renovating activities that is the primary source of chronic exposure for young children today (Gaitens et al., 2009). All lead painted surfaces deteriorate with time, creating lead dust (Marcus & Elias, 1995). Lead dust found on floors and surfaces is the most common pathway of exposure for young children who, as toddlers, explore their environment through hand to mouth behavior (American Academy of Pediatrics, 2005). Oftentimes one or two dwellings are responsible for multiple cases of lead poisoning as affected families move out and new, unsuspecting families move in to lead contaminated homes (Reissman et al., 2001).
The United States has been regularly collecting information nationally on children’s blood lead levels since 1976. Following the phase out of leaded gasoline, blood lead levels of the U.S. population aged 1 to 74 years dropped 78% between 1976 and 1991 (CDC, 2005f). The most recent surveillance data estimates that 1.4% of U.S. children aged 1 to 5 years continue to be affected by lead (Jones et al., 2009a). Exposure to lead paint and dust account for 70% of reported cases (Levin et al., 2008).

Canadians witnessed a similar decline in children’s blood lead levels following the phasing out of lead in gasoline, new paint, and solder; but unlike the United States, surveillance in this country has been sporadic (Environmental Health Workgroup, 2004). In November 2008 Health Canada released the preliminary findings from the Canadian Health Measures Survey (Statistics Canada, 2008) a national investigation analyzing samples of blood from over 2,000 Canadians, measuring concentrations of lead, cadmium and mercury - three metals that can deleteriously affect human health, brain development and overall functioning. Findings from the survey’s first phase indicate that mean blood lead levels of Canadians, aged 6 to 79 years, have declined considerably in the 30-year span since the last nation-wide survey of blood lead levels in Canada, conducted in 1978 (Statistics Canada, 2008). The Daily, reporting on the survey’s findings declares that “[f]ewer than 1% of Canadians now have blood lead concentrations above the Health Canada guidance value of 10µg/dL” (Statistics Canada, 2008). These findings, at first glance, suggest a public health victory and appear to justify Canada’s phlegmatic response to childhood lead poisoning, which to date has focused primarily on lead in consumer products. Is there evidence to support the thesis that childhood lead poisoning related to housing sources exists in Canada? For this special Advances in Nursing Science (ANS) issue, we explore this
question using Dixon and Dixon Integrative Model for Environmental Health Research (IMEHR) (2002) to inform a literature review with a specific focus on Canadian data sources. And, as Dixon and Dixon intended their model as a “working hypothesis that may be useful in guiding investigations or suggesting needed policies” (2002, p. 44) we test the model’s utility in facilitating the detection of gaps in Canadian research and policy on the topic of childhood lead poisoning related to housing sources.

**The Dixon and Dixon Model**

Starting with the first international conference of health promotion, the Ottawa Health Charter (World Health Organization et al., 1986) with its explicit recognition of a stable eco-system, sustainable resources and social justice as basic prerequisites for improving health, and later the Jakarta Declaration which affirmed a commitment to social justice, equity and sustainability (World Health Organization, 1997), there has been a refreshing shift in the way health is viewed, moving the focus away from the individual and towards the social, economic and physical environmental contexts in which people live. Today a number of ecological models consider a wide range of determinants of health that include physical and social environmental factors, and that also highlight awareness of persistent health disparities. The Community as Partner Model developed by Anderson and McFarlane (2008) for example, recognizes the influence of 8 subsystems on health - physical environment, education, safety and transportation, politics and government, health and social services, communication, economics, and recreation - that can be applied to diverse community health problems of health, sociopolitical and environmental origin. Hancock and Perkins’ Mandala of Health model (1985) understands human ecology as the intersection between
culture and the natural environment, with a focus on equity and sustainability. Lavery and colleagues’ Community Action Model (2005) provides communities with a step by step framework to investigate and solve health disparities through environmental change. The United States Department of Health and Human Services’ Healthy People 2010 strategy establishes the domains of behavior, biology, physical and social environment, and individual factors as mediating factors in its Determinants of Health Model (2008); and, the Healthy People 2020 “overarching goals” includes an action piece on policy, programs and health communication (2009). Falk-Raphael’s Model of Empowered Caring (2005) promotes the use of knowledge development to empower people to action on behalf of health, social justice, equity, and lasting change, including policy; and, Giddings’ Theoretical Model of Social Consciousness is designed to reform social injustices underlying health disparities through social critique and active resistance (2005).

In an earlier issue of ANS, Jane and John Dixon presented their IMEHR (2002), developed to assist communities and individuals cope with exposure to environmental hazards. The model was built, in part, out of frustration by the fact that there was a preponderance of information on contaminants and their physiological impact on human health, but little research on how the public could access that information and use it to ameliorate their situation (Dixon & Dixon, 2002). Dixon and Dixon deduced their model from the growing body of research providing evidence that environmental agents such as lead, mercury, pesticides and other contaminants (i.e. poor air quality) can cause “premature mortality, morbidity, functional limitations … and affect the quantity and quality of life ” (2002, p. 43).
Similar to models mentioned earlier, the Dixon and Dixon model (2002) promotes an ecological perspective, recognizing a broad array of "environmental" influences on health such as political, economic, sociocultural and biological factors, and encourages an interdisciplinary approach to problem solving. Like Anderson and McFarlane's (2008) approach, the primary focus of the Dixon and Dixon model is on outcome – the control or elimination of environmental health hazards, although the route or means to achieving this outcome is not necessarily restricted to action on the part of the health care professional. What distinguishes the Dixon and Dixon model from other ecological models is the inclusion of environmental factors as major determinants of health with an emphasis on the assessment of multiple chemical exposures as opposed to reviewing the effects of just one chemical at a time.

The model explicitly assumes that:

(a) prevention, or “thinking upstream” can eliminate the precursors of illness;
(b) agents are potential causes of disease and can include infectious microbes, chemical and physical agents, the accumulation of which causes health hazards;
(c) an agent is only a health risk if there is a potential route for exposure to occur;
(d) risks to health due to environmental health hazards are not distributed equally among populations; and,
(e) change is required at the policy level as well as at the individual level.

The IMEHR (Dixon & Dixon, 2002) views phenomena through 4 domains: the physiological domain, the vulnerability domain, the epistemological domain, and the health protection domain. The physiological domain is explored primarily
through a review of toxicological and epidemiological studies; the vulnerability domain encompasses the “broad array of individual and community characteristics” that lead to variations in risk and health disparities (p. 44); the epistemological domain concerns “processes of personal thought and social knowledge” by which people make judgments on how to respond (p. 44); and, the health protection domain encompasses the actions that people take to reduce their risk from exposure to environmental agents. Authors Dixon and Dixon did not intend for researchers to study all domains simultaneously as each on its own is a “large area to cover” (2002, p.51). They recommend that researchers focus on one or two domains but they also note how researchers tend to gravitate towards the more “objective” physiological and vulnerability domains and neglect the “softer” but equally important exploration of the epistemological and health protection domains (how we know what we know and what we should do about it). A schematic of the relationships among the Dixon and Dixon model’s domains is provided in Figure 1. The domains are “closely interrelated, such that changes in any one domain lead to changes in other domains” (p. 44). The model does allow for overlap although each domain stresses “a different lens by which a complex, multifaceted problem may be examined” (p.44).

Given its emphasis on environmental agents and primary prevention, the Dixon and Dixon model is appropriate for the exploration of childhood lead poisoning in Canada. The primary purpose of this article is to examine the usefulness of the IMEHR in guiding environmental investigations and policy changes.
Literature Review

Dixon and Dixon’s IMEHR was used to structure a literature review on the topic of childhood lead poisoning in Canada. The purpose of the review was to identify (1) Research conducted to date on the topic of childhood lead poisoning, (research falling within the rubric of the physiological and vulnerability domains), (2) evidence to support the thesis that childhood lead poisoning due to residential sources of exposure exists in Canada (epistemological domain), and, (3) measures taken within Canada to remediate the problem (health protection domain). A primary focus of this review is on the epistemological domain: studies that inform Canadians that a problem with lead in housing exists in Canada.

Method

Seven databases were searched for relevant titles in this order: Ovid MEDLINE, CINAHL, HealthSTAR, Embase, PsychInfo, Environmental Sciences and Pollution Management, and GreenFILE). Four concepts were used to conduct the search: (1) lead poisoning; (2) child(ren); (3) housing; residential sources of exposure; and,(4) Canada. Appropriate search terms for each database were identified through mapping. The terms “Canada” and “Canadian” “Ontario” “Quebec” “New Brunswick” “Newfoundland” “Nova Scotia” “Prince Edward Island” “Manitoba” “Saskatchewan” “Alberta” “British Columbia” “Yukon” “Northwest Territories” and “Nunavut” were added as text words. No search limits were applied. Reference lists of retrieved articles were scanned for additional relevant titles. Hand searches were completed for relevant titles from dissertation abstracts, books, and peer reviewed journals (e.g. the Canadian Nurse, the Canadian Medical Association Journal and the Canadian Public Health Association Journal). Reports such as Health Canada’s 1994 and 2005 Update of Evidence for Low-
Level Effects of Lead and Blood Lead Intervention Levels and Strategies were scanned to identify key authors (Health Canada, 1994; Health Canada, 2005b). Several Web sites were searched for relevant publications: Health Canada, Canada Mortgage and Housing (CMHC), the Ontario Ministry of Health and Long term Care; the Canadian Nurse Association; the Canadian Medical Association, the Canadian Pediatric Society; and, the Canadian Partnership for Children's Health and Environment. Retrieved articles spanned the years from 1932 to 2008. Duplicate abstracts were excluded from the final set, leaving a total of 136 titles identified as relevant.

In phase I, abstracts of the 136 titles were further screened for relevance using the following inclusion criteria: (a) the survey or case study related to childhood lead poisoning within the Canadian context, (b) vulnerable populations (children ages prenatal to 6 years) were included as participants in the study and findings were disseminated by age group, (c) researchers examined the issue in relation to residential sources of lead exposure, (d) blood lead levels of participants or cases were assessed, and (e) researchers collected at least one of the following environmental media to identify sources of exposure: paint chip samples, interior lead dust samples, and water samples. Grey literature such as unpublished blood lead studies, case studies reported in the media, and personal communications (ie, unpublished community blood lead surveys) were included in the final selection if they met the criteria. Occupational exposure studies, animal studies, and blood lead surveys conducted in primary or secondary smelter communities were excluded, leaving a total of 24 articles.

The 24 articles included in the final screen were ranked by using a 2-point scale (1 = meets all of the inclusion criteria; 2 = meets some of the inclusion criteria, ie, measured residential sources of lead in housing but did not record or
collect children’s blood lead information). The final analysis resulted in the identification of only four studies in a 77 year span that examined childhood lead poisoning in relation to housing sources in Canada and which also collected environmental data such as paint chip samples, interior lead dust, and water samples to track exposure sources. Further discussion on findings from the literature search are discussed under the heading “Epistemological Domain.” Table 1 summarizes the 24 clinical, environmental and case studies pertaining to the issue of childhood lead poisoning in Canada.

In phase II, findings from the literature review were further grouped into the three remaining domains (physiological, vulnerability, and health protection) with a particular focus on Canadian data within the health protection domain. In this review, policy documents, position papers, systematic reviews, legislation, and guidelines were included if they pertained to federal jurisdictions. This grouping aids in the identification of process gaps – areas where problems have been identified through case studies or community surveys (epistemological domain) but where follow-up action such as primary or secondary prevention is not evident (health protection domain).

Results

1. Physiological domain. Research on the topic of the physiological effects of lead poisoning has been conducted both nationally and internationally. A complete listing of research on lead within this domain would be unfeasible as lead is one of the most widely studied environmental health topics worldwide (Wigle, 2003). Information regarding the chemical and physiological processes through which lead affects the environment and health has been thoroughly synthesized in various American publications such as the “Draft Toxicological Profile for Lead”

2. **Vulnerability domain.** In the United States, children of low-income families, living in pre-1946 dwellings have a 16% prevalence rate of lead poisoning versus a 4% rate for children from middle-income families (CDC, 2003). Families with fewer economic resources have less choice in where they can live and are more likely to dwell in poorly maintained older housing (Chaudhuri, 1998). For some time, researchers have also been aware of the vast and detrimental difference in impact between exposing a mature adult to lead through, for example, occupational exposure and chronically exposing a developing fetus to maternal sources, or a child to lead in drinking water or floor dust. A June 2000 supplemental edition of the journal *Environmental Health Perspectives* introduced the concept of “critical windows of exposure,” that is, periods of vulnerability or times when fetal and infant brain development are particularly susceptible to exposure to toxins such as lead, mercury, PCBs, and pesticides (Weiss & Landrigan, 2000).

Lidsky and Schneider (2006) reviewed in detail the neurological changes one can expect to see in a lead poisoned child. Of further importance is an
awareness that blood lead levels tend to peak in children between the ages of 18 and 36 months and decline thereafter (Tong, 2000). The implications for research within the Canadian context are clear: Investigations of childhood lead poisoning should include vulnerable populations, that is, children younger than 6 years and belonging to low income families.

3. Epistemology domain. How do people know if childhood lead poisoning is even a problem in Canada? A review of Canadian research spanning more than seven decades reveals that the investigation of childhood lead poisoning in relation to housing sources is an under researched area in Canada. The review recovered only 4 top ranked studies that described cases or conducted community blood lead surveys and which also collected environmental samples to verify the source of exposure (Table 1). Despite the paucity of data, there is sufficient evidence to support the thesis that childhood lead poisoning related to housing sources exists in Canada. Alder and colleagues (1993) reviewed the medical records of children admitted to or visiting as day patients’ at a psychiatric facility, to investigate the association between elevated blood lead levels in children living in certain districts of London, Ontario, containing older housing. The authors found a significant relationship between the age of the dwelling, family income, and children’s blood lead levels. Children living in dwellings built prior to or before 1945 were more likely to have an elevated blood lead level than their peers residing in more recently constructed homes. The article describes the cases of 4 children under the age of 6 years lead poisoned from residential sources.

A 1990 issue of the *Canadian Medical Association Journal* recounts 2 cases of lead poisoning occurring in toddlers: a 32-month old girl with a “17-month history of a voracious appetite for the paint peeling from the walls and window sills of her inner city home” (p.40) and a 26-month-old boy, who after many repeat visits
to hospital for persistent vomiting was eventually diagnosed and medically treated for lead encephalopathy (Tenenbein, 1990). In both instances, the source of exposure was traced to residential paint although residential samples were not collected to verify the exposure source (Tenenbein, 1990). A Quebec City study comparing rural children’s blood lead levels with urban children’s levels reported that 10.8% of children in that study had lead poisoning (considered blood lead levels $\geq 10 \mu g/dL$) with 6 of 10 cases attributed to housing sources (Levallois et al., 1991b). A New Brunswick survey of children ages 1 to 3 years discovered 11.3% of children with blood lead levels $\geq 10 \mu g/dl$ (unpublished data, C. Balram & S.C. Giffin, 1993). Sources of exposure were traced to residential paint, drinking water and other sources. The New Brunswick study is one of the few Canadian studies that examined childhood lead poisoning in relation to housing sources post leaded gasoline use. A 2001 investigation by Health Canada documented high levels of lead in floor dust in samples collected from 50 Ottawa homes. Unfortunately, blood lead samples of home occupants were not assessed to determine whether lead poisoning had occurred (Rasmussen et al., 2001). A 2004 issue of the Canadian Medical Association Journal reports the case of a 4-year old Montreal boy seriously poisoned from eating paint chips peeled off the bathroom trim in his family home (Lavoie & Bailey, 2004).

4. Health protection domain. In the United States 3 major federal agencies inform the public on the issue of childhood lead poisoning: EPA, the Centers for Disease Control and Prevention (CDC), and the Department of Housing and Urban Development (HUD). In addition, the National Lead Information Centre, a clearing house for lead information, operates under joint funding from EPA, CDC, and HUD. National not-for-profit organizations such as the National Center for Healthy Housing, the Alliance for Healthy Homes, and parent based organizations such as
United Parents Against Lead disseminate lead awareness information to parents and policy makers. The CDC publishes guidance documents for health professionals, such as *Preventing Lead Poisoning in Children* (CDC, 2005). The United States has also enacted legislation that requires disclosure of sources of lead in residential dwellings. Under the Residential Lead-Based Paint Hazard Reduction Act of 1992, also known as *Title X*, landlords and homeowners are required to disclose to the buyer and tenant any known existing lead hazards contained in housing built before 1978. EPA and HUD provide a national training program for the inspection of residential lead hazards. In addition, HUD provides millions in annual funding for the control of lead-based paint hazards in eligible communities (2009).

Two federal agencies in Canada hold the primary responsibility for disseminating information on childhood lead poisoning and housing. Under the Department of Health Act, (1996) the federal Minister of Health, (through Health Canada) has the duty to investigate and research public health, including the monitoring of diseases and the collection, analysis, interpretation, publication and distribution of information relating to public health. As mentioned, Health Canada is conducting a national bio-monitoring study, the Canadian Health Measures Survey (Statistics Canada, 2008) of adults and children, which does include measures of blood lead; however, vulnerable populations (children younger than 6 years) have been excluded from the sample population to date. And while Health Canada has developed a “lead strategy” its major focus is on lead in consumer products, a relatively minor source when compared to the potential of housing for childhood lead exposure (Health Canada, 2002). Under the National Housing Act (1985) CMHC is assigned the statutory responsibility to distribute information on housing and living conditions. Even so, in Canada, the extent of lead
contamination in housing is poorly understood and can only be estimated (OPHA, 2004).

The United States has been a leader in advancing knowledge in the field of childhood lead poisoning, and true to Dixon and Dixon's assertion, much of the research on this health topic falls within the physiological domain. Dixon and Dixon advise that if gains are to be made in environmental health then an interdisciplinary approach that increases knowledge development in all four domains is necessary (2002). The literature review was particularly useful in revealing deficiencies within the epistemological and health protection domains in Canadian research and policy.

**Toward a Critical Analysis of Childhood Lead Poisoning in Canada**

Despite a lack of case and cross-sectional studies, there is documentation of harm occurring to Canadian children from exposure to residential sources of lead – enough, surely, to warrant immediate action within the rubric of the health protection domain. *Are Canadians adequately protected? If not, why?* These questions can best be answered through critical analysis. Over the past two decades, critical theory has emerged as an important means for nurses to identify and understand the manner by which sociopolitical and cultural environments shape nursing practice (Powers, 1996; Wells, 1995). Holmes and colleagues (2008) note the following:

Research that aims to be critical seeks, as its purpose of inquiry, a confrontation of the injustices in society as well as a questioning of the status quo, while giving a voice to vulnerable persons (including marginal/ized discourses). Critical researchers believe that the knowledge developed in their research may serve as a first step toward addressing such injustices (p.43).
While Dixon and Dixon hint at the significant amount of literature in critical theory on public health discourses and its sociopolitical, historical and economic implications, this approach is not investigated further or explored via their model. In a later article Dixon and colleagues (2006) do suggest a series of research questions that can be adapted for future critical analysis: How does everyone know about this? What is the common knowledge and level of public concern? What messages come from corporations, environmental organizations, the media, health professionals, and other groups? What appears to be the dominant messages? How do these messages relate to what is known or not known through science? And, how might affected people acquire their sense of what is true? An important next step will be to critically examine the response of Canadian agencies to evidence of childhood lead poisoning related to residential sources. Studies have shown that there is an unequal distribution of health across society and that much of it can be linked to economic and social determinants of health (Falk-Rafael, 2006). As housing is an environmental factor with strong ties to social economic factors it will be important to explore this topic as a social justice issue. Giddings' Theoretical Model of Social Consciousness recognizes how “health disparities...are sustained by the institutionalizing of discriminatory healthcare polices and practices” and may be useful for designing a future critical analysis of this topic (2005, p.224).

Relevance of the Dixon and Dixon Model for research in nursing

Dixon and Dixon first published their model in ANS 8 years ago. It seems fitting to review what has been done with the model and re-assess its relevance for nursing. The Dixon and Dixon model exhibits a blend of natural and social
sciences and acknowledges multiple ways of knowing including tradition, authority, group loyalty, experience, intuition, and science. While not explicitly stated, this stance locates the model within a realism paradigm. Realism “proposes a common ontology and epistemology for the natural and social sciences” (Wainwright, 1997, p.1267). The model also reflects a persistence worldview, one that emphasizes equilibrium and balance as opposed to growth and progress (Fawcett, 1989a) as evidenced by Dixon and Dixon’s goal of “protect[ing] the well being …of members” and its emphasis on balance (2002p. 43). As it promotes “upstream” healthcare, it reflects a systems category that involves the “identification of actual and potential problems in the function of systems and delineation of intervention strategies that maximize efficient and effective system operation” (Fawcett, 1989b, p.14).

According to Fawcett, conceptual models are broad, abstract, and evolve from empirical observations and intuitive insights of scholars. They can be induced when specific situations exemplify more general events. Conceptual models also provide direction on how to observe and interpret phenomena (Fawcett, 1989b, p.16). Fawcett’s (1984) metaparadigm concepts of nurse, person, health and environment are incorporated into the Dixon and Dixon model. Dixon and Dixon note that nurses “are well positioned to participate in and provide leadership for interdisciplinary efforts to improve environmental health” (2002, p.54) although their approach facilitates a view that goes beyond the traditional realm of nursing. Similar to Anderson and McFarlane’s Community as Partner Model (2008) interventions are not constrained to established health care institutions. Public participation in health decision making is encouraged, in fact, imperative. In the article titled, “Kids Need Clean Air: Air Pollution and Children’s Health:
Environmental Issues in the Health of Children” (Dixon, 2002), nurses build the capacity of community members through knowledge development and leadership.

In the Dixon and Dixon model, “person” has individual, developmental, gender-based and physiological characteristics that make some people more vulnerable than others to exposures. Person in this model can also become an expert in the particular health issue that is of concern. In this respect the IMEHR shares a characteristic with Falk-Rafael’s Model for Empowered Caring in that both incorporate the expectation that as individuals and communities increase their knowledge of issues concerning health, the expertise gained will have a direct impact on health protection (Dixon & Dixon, 2002; Falk-Raphael, 2005). “Health” is seen as the outcome, or the result of exposure to an environmental agent. Outcomes can include but are not limited to increased mortality, morbidity, functional limitations, and symptom experience affecting quantity and quality of life. “Environment” can be the exposure agent, but it can also be the community characteristics, sociodemographic and cultural characteristics, public policies, location of residence, and occupation.

Butterfield, a contemporary of Jane Dixon and one of the reviewers of the Dixon and Dixon model devised a roadmap for positioning environmental health curricula into nursing schools and mainstream practice (Larsson & Butterfield, 2002). Harnish and colleagues (2006) tested Dixon and Dixon’s model by examining whether community perceptions of environmental risk were congruent with or diverged from the model. Home visit data from 11 parents were analyzed thematically according to the Dixon and Dixon model concepts. Through this research, they uncovered barriers to preventive action. Parents, for example, were aware of risks but were unsure of what to do about them. Harnish and colleagues
found the model was a “useful intermediate step for researchers involved in multiyear studies” (2006, p.465).

Macdonald (2004) used the Dixon and Dixon model to illuminate the Toronto severe acute respiratory syndrome (SARS) experience. Macdonald's research question was, “[W]hat combination of factors has the potential to lead to a problem, and what can be done upstream so the world is not dealing with emerging diseases like SARS?” (2004, p. 546). Macdonald found the epistemological domain the most helpful in gaining greater understanding of the Toronto experience. In exploring the question, “How does everyone know?” Macdonald uncovered weaknesses in provincial microbiology laboratory resources, as well as poor communication links between provincial public health units and between the public health department and hospitals (2004).

Im (2005) notes the model’s social utility in generating situation specific theory. A situation specific theory that incorporates the concepts of Dixon and Dixon's conceptual model may be appropriate for further investigation of health issues such as childhood lead poisoning, at the local or community level.

A minor shortfall of this model, which is strongly rooted in primary prevention, is its lack of guidance for secondary and tertiary prevention within the health protection domain. It is likely that populations of “at-risk” children (i.e. children with elevated blood lead levels) will be identified through future community research in Canada, and there will be a need for case management. Blending Dixon and Dixon’s model with one such as Anderson and McFarlane’s Community as Partner Model (2008) might address this problem as the latter does incorporate the concepts of secondary and tertiary prevention. Even so, Greving and Santacroce (2005) were able to demonstrate how the Dixon and Dixon model can be applied after harm has occurred. In this application, children who are survivors
of cancer continue to be at risk because the chemotherapy itself is toxic - it is especially damaging to the cardiovascular system. The nursing approach in this situation is both tertiary and primary. That is, nurses are treating cancer (tertiary prevention); but also, when nurses promote cardiovascular health for these children, they are practicing primary prevention.

The Community Action Model (Lavery et al., 2005) might also enhance the Dixon and Dixon health protection domain. It employs a detailed 5-step process for building community capacity for confronting health disparities. The steps include (1) training of participants, (2) defining and designing a community diagnosis, (3) analysis, (4) implementing an action or activity, and (5) maintenance or enforcement of the activity. The model involves participatory action research and was designed to have a “lasting impact … by creating environmental policy change” (2005, p. 616).

With its upstream focus on primary prevention, and its goal of facilitating optimal level wellness to protect the well-being of society’s most vulnerable members, the Dixon and Dixon model meets expectations for a nursing model in that nurses are required to view health in its larger framework, as linked to pathology in the environment. This model contributes to the discipline of nursing by viewing the concept of health through the lens of physiology, vulnerability, epistemology, and health protection. Perhaps what is most satisfying about the Dixon and Dixon model is that it does not privilege health professional action over action on the part of the community or individual members. “Person” is acknowledged for their gained expertise and ability to affect change. Shortcomings within Dixon and Dixon’s health protection domain can be addressed by incorporating elements of other ecological models such as the Community as Partner Model (2008) or the Community Action Model (Lavery et al., 2005).
The IMEHR was employed to structure a literature review on the topic of childhood lead poisoning in Canada and was useful in identifying gaps in areas of research and policy within the 4 primary domains: physiological, vulnerability, epistemological, and health protection. This exercise appears to support Dixon and Dixon’s claim that the physiological and vulnerability domains impact the development of knowledge (epistemological domain) which in turn forms the basis for the health protection domain – although, the model is limited in its ability to explain why knowing a problem exists does not necessarily lead to doing something about it. In future, a critical analysis that examines the interplay between the epistemological and health protection domains will assist in identifying power relations and underlying social conditions that have prevented knowledge of this problem from translating into requisite action.
References


CDC (2005). *Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control and Prevention* Atlanta, Ga..


Table 1. Canadian blood lead surveys, case studies and environmental studies related to housing as a source of Pb exposure

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Rank</th>
<th>Environmental media collected from residential sources</th>
<th>% EBLL or #Cases &amp; age range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alder et al., 1993 (1) London, Ontario</td>
<td>1</td>
<td>Paint, dust, first draw cold tap water</td>
<td>Case study: 6-y male (49.7 µg/dL); 2-y female (39.3 µg/dL); 2-y female (11.4 µg/dL) &amp; 1-y male (15.5 µg/dL).</td>
</tr>
<tr>
<td>Balram &amp; Giffin, 1993 (unpublished) St. John, New Brunswick</td>
<td>1</td>
<td>Paint, paint dust, tap water, soil, household food, blood lead levels</td>
<td>11.3% ± 3 y</td>
</tr>
<tr>
<td>Lavoie &amp; Bailey, 2004 (3) Montreal, Quebec</td>
<td>1</td>
<td>Paint chips, blood lead levels</td>
<td>Case study: Male 4–y male (97 µg/dL)</td>
</tr>
<tr>
<td>Levallois et al., 1991 (4) Quebec City, Quebec</td>
<td>1</td>
<td>Household dust samples, paint flakes, tap water, blood lead levels</td>
<td>10.8% urban population; 1.3% rural population 1-6 y</td>
</tr>
<tr>
<td>Hamilton BLL survey (5) (in progress)</td>
<td>1</td>
<td>616 children &lt; 7 y Capillary BL, survey to determine age of housing, tap water, dust and soil (192/254 homes)</td>
<td>12.5% &gt; ‘action level’ 4µg/dL 1% (6 children) ≥ 10 µg/dL 3% children 12-13 mos ≥ 10 µg/dL</td>
</tr>
<tr>
<td>Albert, 2000 (6) Ottawa, Ontario</td>
<td>2</td>
<td>1 well water sample, questionnaire, blood lead levels</td>
<td>1.7% children &lt; 7 y prenatal to adult</td>
</tr>
<tr>
<td>T. Bell, 2004 (unpublished) St. John’s Newfoundland</td>
<td>2</td>
<td>Soil samples some of which were collected in residential areas</td>
<td>Blood lead levels not sampled</td>
</tr>
<tr>
<td>Canada Health Survey, 1981 (7) National</td>
<td>2</td>
<td>blood lead levels</td>
<td>25% &gt;3 y</td>
</tr>
<tr>
<td>Canada Mortgage and Housing Corporation, 1993 (8) Medicine Hat, Alberta</td>
<td>2</td>
<td>Soil, housedust (vacuum samples), paint chips, lead swabs (post abatement), drinking water (standing sample).</td>
<td>Blood lead levels not sampled although 2 cases reported previously in national media report. (See CBC 1991(9))</td>
</tr>
<tr>
<td>Canada Mortgage and Housing Corporation, 1995 (10) St. John, New Brunswick</td>
<td>2</td>
<td>Environmental survey investigated post hoc the relationship between blood lead levels and drinking water. The sub-sample of 100 homes was chosen from a previous sample of 500 St. John residences (unpublished, R. Scott, 1995).</td>
<td></td>
</tr>
<tr>
<td>Study/Source</td>
<td>Region</td>
<td>Methodology</td>
<td>Findings</td>
</tr>
<tr>
<td>--------------</td>
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<tr>
<td>Canadian Broadcasting Corporation (CBC), 1991 (11) Medicine Hat, Alberta</td>
<td>National news coverage of two Medicine Hat children lead poisoned through home renovating activities (See also CMHC 1993(8)).</td>
<td>2 cases: 1-y male (30 μg/dL) 3-y female (22.9 μg/dL)</td>
<td></td>
</tr>
<tr>
<td>Cushing, 1934 (12) Children's Memorial Hospital, Montreal, Quebec</td>
<td>Clinical symptoms i.e. basophilic stippling, seizures</td>
<td>17 cases: 1-3 y</td>
<td></td>
</tr>
<tr>
<td>Canadian Health Measures Survey, 2008 (13) National</td>
<td>Blood and urine</td>
<td>&lt;1% 6-79 y GM =1.37 g/dL</td>
<td></td>
</tr>
<tr>
<td>Health Canada, The Canadian housedust study, 2007 (14). National</td>
<td>Residential dwellings. Vacuum samples collection (concentration) and floor swab samples (lead loading).</td>
<td>Blood lead levels not sampled</td>
<td></td>
</tr>
<tr>
<td>Health Canada, Maternal-infant research on environmental chemicals, 2007 (15) National</td>
<td>Measures of environmental contaminants in breast milk, blood, urine, hair and cord blood and meconium of infants. Information collected on construction date of housing through survey</td>
<td>Results not yet available</td>
<td></td>
</tr>
<tr>
<td>Hukowich, 1997 (unpublished), Port Hope, Ontario</td>
<td>Blood lead levels Elevated blood lead levels traced to residential sources of exposure although environmental samples not collected to confirm source</td>
<td>4% 3-6 y</td>
<td></td>
</tr>
<tr>
<td>Jin et al., 1995 (16) Vancouver, British Columbia</td>
<td>Blood lead levels, questionnaire</td>
<td>8% 24-36 mo</td>
<td></td>
</tr>
<tr>
<td>Mitchell, 1932 (17) Children's Memorial Hospital, Montreal, Quebec</td>
<td>Clinical symptoms such as bilateral wrist and foot drop</td>
<td>4 cases: 1-9 y</td>
<td></td>
</tr>
<tr>
<td>O'Heany et. al (1988) (18) Toronto, Ontario</td>
<td>BLLs and measures of lead in air, tap water, soil and gasoline. &quot;Housing condition&quot; entered into regression analysis. Soil samples collected from school yard. No description of water sampling method (i.e. flushed or standing).</td>
<td>4.3% &gt; 20 μg/dL ≤7 y</td>
<td></td>
</tr>
<tr>
<td>Sahni et. al, 2007)(19) New Brunswick</td>
<td>Investigated environmental sources of manganese but not lead. Exhibited progressive behavioral and neurologic symptoms, loss of balance, co-ordination, and fine motor skills, developed a high steppage &quot;cock-like&quot; gait.</td>
<td>1 case 6 yr old female</td>
<td></td>
</tr>
<tr>
<td>Poon et al., 1989 (2) London, Ontario</td>
<td>No environmental sampling conducted however researchers noticed an initial increase and then a decline in children's BLL following extensive renovations to remove lead painted surfaces.</td>
<td>25/1000 (2.5%) with BLLs &gt; 25 μg/dL. Overall mean value 8 μg/dL 6 mo-20 y</td>
<td></td>
</tr>
<tr>
<td>Study/Author/Year</td>
<td>Location</td>
<td>Description</td>
<td>Outcome</td>
</tr>
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</tr>
<tr>
<td>Rasmussen, Subramanian et al., 2001 (20)</td>
<td>Ottawa, Ontario</td>
<td>Residential dwellings. Concentrations of lead, among other metals, sampled in house dust using vacuum collection method. Range: 50.20 μg/g to 3225.66 μg/g</td>
<td>Blood lead not sampled</td>
</tr>
<tr>
<td>Ross and Brown, 1935 (21)</td>
<td>Sick Children’s Hospital, Toronto, Ontario</td>
<td>Clinical symptoms i.e. presence of basophilic stippling, seizures</td>
<td>23 cases, ages not identified</td>
</tr>
<tr>
<td>Tenenbein, 1990 (22)</td>
<td>Canadian location</td>
<td>Sources of lead exposure were, in both cases traced to residential paint (Winnipeg) but it is unclear if actual environmental samples were collected.</td>
<td>2 cases: 26 mo and 36 mo</td>
</tr>
<tr>
<td>Valiquette and Kosatsky, 1995 (23)</td>
<td>Montreal, Quebec</td>
<td>Review of laboratory records. Paint identified as principal source of exposure.</td>
<td>12 cases: (defined as &gt;25 g/dL). BLL Range: 27-94 g/dL &lt;15 y</td>
</tr>
</tbody>
</table>
Figure 1. Dixon and Dixon’s Integrative Model for Environmental Health Research.
1.4 Epistemological stance

Environmental health risks are not distributed equally among populations. It is often communities with the least political or economic power that are most affected; but, there are also individual and community characteristics that increase vulnerability (Dixon & Dixon, 2002). Socioeconomic factors such as poverty along with substandard housing combine to make the public health issue of childhood lead poisoning disproportionate. The multi-year [United States] National Health and Nutrition Examination Surveys (NHANES), for example, shows consistently that blood lead levels are higher for younger children (than older children), older adults (than younger adults), males more than females, black race more than white race, and inner city residents (Meyers et al., 2003b). U.S. Children with the highest blood lead levels tend to be economically disadvantaged and reside in older housing (Meyers et al., 2003c). Immigrants and children with developmental delays are also disproportionately affected (Woolf et al., 2007).

Clearly, lead poisoning is a health issue predominantly affecting children of disadvantaged families. Due to the numerous social justice implications this health topic presents, there is large opportunity for critical analysis. Foucault’s writings on discourse are particularly germane to nursing research (Gastaldo & Holmes, 1999; Powers, 1996) and relevant for a critical analysis of the problem of childhood lead poisoning in Canada. By applying Foucault’s archaeological-genealogical approach to critical discourse analysis, as described in various text (Foucault, 1972; 1980a; Hunt & Wickham, 1994; Kendall & Wickham, 1999), and further elaborated upon by Rawlinson (1987), this thesis proposes to identify and critically examine the conditions supporting dominant public health discourses on childhood lead poisoning in Canada.
1.4.1. Exploring Critical Theory

Critical Theory encompasses many perspectives and evolved primarily from Marxist based scholarship (Jorgensen & Phillips, 2002). It was developed by the Frankfurt School in Germany in the 1930s and is opposed to some of the central ideas of positivism such as the belief that causal laws are universal truths. At the same time Critical Theory accepts that empirical analysis is a necessary part of scientific inquiry (Threadgold, 2003). Habermas, an early proponent of Critical Theory, argued that scientific approaches to the study of social sciences require a variety of methods and classified inquiry into three categories:

1) Empirical-analytic sciences, which are interested in prediction and control;
2) Historical-hermeneutic, which understands human social science through ordinary language; and,
3) Critical Theory, which involves self reflection and an emancipatory interest in gaining release from domination (Habermas, 1987).

Critical Theory maintains that “truth” or evidence is always socially constructed. The aim of inquiry, therefore, is to identify the social, political, cultural, and economic structures that shape and govern thoughts, practices and beliefs. Meaning and truth is always interpreted within the context of history (Denzin & Lincoln, 1998). Critical research conceives of a better world and attempts to confront injustice in society. Advocacy and activism, then, are key objectives of the critical process (Denzin & Lincoln, 1998). Kleffel positions “critical social nursing,” as a viable, economically advantageous, upstream approach to health protection due to its ability to uncover oppressive and unequal “environmental constraints”

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3 Ruth Wodak (2002) provides a useful comparison of the differences between scientific theories and critical theories.
upon health (Kleffel, 1991, p.48). Critical analysis is often the first step towards political action (Kincheloe & McLaren, 1998; Holmes et al., 2008).

In critical theory, much of the focus is on an analysis and exploration of texts, their various representations, and how they come to be, with a focus on historicity, possibility and contextuality (Cheek, 2000; Powers, 2001). The Frankfurt School supported the view that claims to true knowledge were not value free but “tied to certain social projects, values, interest, gender, races, classes, agendas” but was itself criticized for its assumptions of universal, ahistorical and transcendental notions such as oppression, empowerment and emancipation (Powers, 2001, p.6)

Under the umbrella of critical theory, post-structuralist philosophy emerged in the 1950s. Post-structuralists view the world as an open system shaped by human agency. Similar to critical theorists, post-structuralists reject universal explanations and totalizing theories of a structured and orderly world (Powers, 2001). At the same time they reject the assumption that theoretical constructs (such as oppression) exist outside of specific human situations (Powers, 2001). In the context of nursing, Cheek offers post-structuralist methods, such as discourse analysis, as a “way of thinking deeply about nursing and health care” (Cheek, 2000, p.11). Post-structuralists also challenge the assumption that language is neutral or value free. The language of public health, for example, is regarded as “highly political and socially contextual” (Lupton, 1995, p.2).

A critical approach to the study of a public health issue implies an emancipatory function and because language is recognized as a form of social practice the researcher attempts to correct social injustices as practiced through language (discursive practices), through development of more equal power relations in communication processes. Critical analysts often “take the side of
oppressed social groups” (Jorgensen & Phillips, 2002, p.64). Deborah Lupton, a well known and outspoken critic of public health policies describes what is meant by critical:

A critical approach to health promotion should involve an explicit questioning of whose voices are being heard and privileged, the alliances to conflicts involved, what body of expertise is cited in support, what counts as knowledge, how it is organized, controlled authenticated and disseminated, who has access to knowledge and how this knowledge is taken up, justified and used by health promotion practitioners (Lupton, 1995, p.49).

Lupton used critical analysis to illustrate how the previously unseen problems of “dirt, miasma, odour, sexuality, reproduction, childhood and the family” became matters worthy of public health’s attention (Lupton, 1995, p.18). Clark, Barton and Brown applied critical theory to their research on contaminated communities. Their analysis, which focused on low income Hispanic populations, uncovered competing and incongruent agendas within various discourses (Clark et al., 2002). Fassin and Naude (2004) critically reviewed the response of French public health authorities to the problem of childhood lead poisoning occurring predominantly in immigrant occupied deteriorating housing districts of France during the 1980’s.

These authors demonstrate that using a critical theory approach to the study of childhood lead poisoning is not only relevant, but also useful. Although the properties of lead have been known to be toxic to humans for centuries, this metal continued to be widely marketed and promoted for public use and consumption in North America well into the 20th century (see for example, Markowitz & Rosner, 2002). The promotion of lead in consumer products continues today. Various American writers have been openly critical of U.S. government policy (or lack of it), corporate influence on research, industry promotion of a known and undisputed health hazard, and public health ‘foot dragging’ on prevention of childhood lead poisoning. See, for example, the insightful writings of Bellinger, (2004; 2006);

Critical analyses on the topic of childhood lead poisoning in Canada has been less abundant. Boothe and Harrison (2009) critically reviewed the Canadian response to environmental health issues, including lead regulations, via a comparison with American policy and advocacy. The Ontario Public Health Association Environmental Work Group summarized the Canadian federal response to residential sources of lead and issued a series of recommendations to address identified deficiencies (OPHA, 2004). Perhaps the most vocal among “lead” critics has been Kathleen Cooper, a long-time researcher with the Canadian Environmental Law Association (Cooper, 2000; Wallace & Cooper, 1986). What is lacking, to date, is a critical-historical analysis of the way in which childhood lead poisoning has been constructed in Canada in the post-leaded gasoline era.

1.4.2. Discourse

The concept of discourse has been described by various authors, with little consensus on the definition of this term. In its most basic sense discourse refers to language (Jorgensen & Phillips, 2002). Others define it as social practice which describes a way of speaking, giving meaning to experiences from a particular perspective (Laclau & Mouffe, 1985). Lupton defines discourse as “a group of ideas or patterned way of thinking which can be identified in textual and verbal communications [which] can also be located in wider social structures” (Lupton, 1992, p. 145). Parker describes discourses as “‘representations’ of the world which have a reality almost as coercive as gravity, and like gravity, we know of the objects through their effects” (1992, p. 8).
One of the earliest uses of the term is by the ancient philosopher Aristotle. Aristotle perceived discourse as containing three elements: the speaker, the topic, and the audience. He was perhaps the first to begin the tradition of discourse analysis with his observation that discourse consisted of three genres: forensic (legal), deliberative (legislative) and epideictic (ceremonial) (Hill, 2005). For Aristotle, the steps towards analyzing a discourse included constructing the context in which the speech occurred; constructing the audience, describing the source of the message, and then analyzing the message (Hill, 2005).

More recent representations of the term can be traced to the teachings of Swiss linguist Ferdinand de Saussure through later published accounts of his work by his students in the early 20th Century (Sanders, 2004). Saussure, widely regarded as the father of linguistics as well as of structuralism, formalized principles and methods for the “scientific” study of language by developing a rigorous methodology for the identification of replicable patterns in language (Sanders, 2004). Noam Chomsky’s work on language and textual analysis has also been identified as influential in the creation and recognition of “linguistics” as a separate and specialized academic discipline (Robins, 1997). Two distinct branches form the discipline of linguistics: the formalist (or cognitive) paradigm, and the functionalist paradigm (Schiffren, 1994).

While the study of language (linguistics) marked an important turning point in social theory (Robins, 1997) it also came under attack for its lack of concern with the way in which external processes shaped language, and its view of language as fixed, unchangeable and neutral. French theorist Jacques Derrida challenged Saussure’s “structuralist” and “positivistic repression” of language (Martin, 1992, p.7) and instead favoured a post-structuralist view of language, seen as fundamentally fluid and dynamic (Jorgensen & Phillips, 2002). Derrida perceived
language as “a system of relations and oppositions” whereby the “positive nature” of a term was less important than its function in a system (Aune, 1990, p.256). Derrida’s method of analysis - deconstruction, identified gaps and silences, rules, and oppositions in text, as well as their role in shaping meaning and understanding (Aune, 1990).

This critical turn in linguistic analysis was further assisted by the work of feminist authors who, among other things, highlighted the ways in which language disadvantaged women through, for example, the uncritical use of male biased terminology (see for example, Penelope, 1990; Spender, 1985). The work of writers and theorists such as Michel Foucault, Norman Fairclough, Pierre Bourdieu, Antonio Gramsci, Michael Halliday, Jurgen Habermas, Roy Bhaskar, Anthony Giddings, Ernesto Laclau and Chantal Mouffe, and other neo-Marxist social theorists also challenged non-critical approaches to discourse analysis by examining the ways language shaped and was shaped, by underlying social, political and power relations (Jorgensen & Phillips, 2002; Threadgold, 2003).
1.5 Methods

1.5.1. Study design

This research study asks: What is known about childhood lead poisoning in Canada? And, How might this knowledge, or more specifically, how might limitations in knowledge been effective in maintaining dominant public health discourses on childhood lead poisoning in Canada? This thesis examines the historical formation of authority and its right to pronounce truth using Michel Foucault’s approach to critical discourse analysis as as a guide (Foucault, 1966; 1972; Rawlinson, 1987; Kendall & Wickham, 1999)

The roots of critical discourse analysis are located in postmodernism implying a concern for “context rather than universality, specificity rather than generalization, uniqueness rather than sameness, and relativism rather than absolutism” (Holmes & Warelow, 2000 p. 89). By qualitatively exploring what Foucault terms the “power/knowledge” perspective the critic reveals underlying transformations in discourse, previously unseen but influential historical events and the supporting contingencies which shaped knowledge concerning the issue of childhood lead poisoning in Canada. The strength of a Foucauldian archaeological/genealogical analysis for nursing research and particularly for this analysis is in its focus on discourse, surfaces of emergence, transformations, mutations, contingencies, events, recognition of power/knowledge strategies, descriptions of discipline technologies and subsequent consequences, and suggested possibilities of resistance.

The analysis is assisted via a comparison of Canadian public health discourse on lead poisoning and lead-based paint with American public health discourse. This method of analysis is transformative in that while it demystifies the
discourse of power, it also lays out the basis for future actions regarding identified problems (Powers, 1996).

1.5.2. Data collection

The method used for data retrieval served two purposes: 1) To identify key Canadian authors writing academically on the topic of lead poisoning in Canada with a view to identifying discourse and/or events which provided models or ideas that influenced the function of the discourse under analysis; and 2) To identify dominant non-academic discourses found within the public domain on the topic of childhood lead poisoning in Canada.

i. Canadian researchers/authors’ names were extracted from the database contained in Appendix C. Retrieval methods for this review are previously described in Article 1.

ii. The 322 authors’ names supplied in the database (Appendix C) were cross referenced with those names listed in three systematic reviews (CMHC, 1992; Tsekrekos & Buka, 2005; Smith et al., 2008). This cross referencing led to the identification of five additional articles on lead (Belles-Isles et al., 2002; Bussières et al., 2004; Butler Walker et al., 2006; Lucas & Dewailly, 2004; Nadon et al., 2002) and five additional authors (Belles-Isles, Bussières, Lucas, Nadon and Przybysz).

iii. To identify prominent authors and therefore dominant discourses on lead poisoning in Canada, a rank order was established using a frequency count of the number of times an author’s name appears in the database (Appendix D).

iv. To further identify “authoritative” voices on lead in Canada, a Google Scholar and PubMed search using the top 30 ranked authors’ names (i.e.
authors associated with 3 or more articles) was executed to retrieve publicly accessible documents such as academic affiliations, government affiliations, authors’ involvement in the writing or reviewing of policy papers and reports or committee involvement with respect to lead.

v. To identify marginal discourses (i.e. authors with two or less publications, a criteria which captures those authors previously ranked in Article 1 as “1s”), author names were searched using Google Scholar and PubMed to further identify grey literature and other research contributions by these authors with respect to lead. A total of 40 author names were searched.

vi. The author search resulted in the identification of two additional lead poisoning “cases”:

1. A case report identifying pediatric manganism in a 6 year old New Brunswick girl. Although this is a case study of manganese poisoning the child is described as having a BPb of 17.6 μg/dL, a levels is considered elevated according to current standards. The source of lead exposure was not investigated (Sahni et al., 2007);

2. A case report identifying a 6 year old Quebec youth with acute lead poisoning (>186 μg/dL) (Bailey & Bussières, 2003). In the second case, investigators did not identify the source of exposure.

vii. Internet search for dominant discourses on lead poisoning in Canada. The internet is becoming increasingly popular as a source of health information for the public (Hesse et al., 2005) therefore the following four search engines were used for this review: Google, Bing, Yahoo and Ask. Where available, the option to limit results to Canadian websites was applied (Google and Bing) so that a total of 6 searches were executed. Content from the top 14 Web sites was also used to inform the analysis.
A full description of methods and results for the Web site review is included in Appendix E.

As Health Canada and Canada Mortgage and Housing Corporation (CMHC) are the two federal agencies assigned primary jurisdiction for the protection of children from environmental sources of lead, web content from these two agencies’ public information sites also informs this analysis. Content was retrieved using the following search terms: “lead,” “lead poisoning,” “lead paint,” “housing,” “renovations,” “children,” and “environmental health.” Content from Statistics Canada online edition of The Daily was also included.

Federal reports such as Health Canada’s 1994 and 2005 Update of Evidence for Low-Level Effects of Lead and Blood Lead Intervention Levels, the Canadian Environmental Law Association’s report Environmental Standard Setting and Children’s Health (Cooper, 2000), Health Canada’s online Lead Strategy (Health Canada, 2002), the report Children’s Health and the Environment in North America: A first report on Environmental Indicators and Measures (2006) and the Canadian Task Force on the Periodic Health Examination Membership (Feldman & Randel, 1994) contain detailed discussion on the issue of childhood lead poisoning in Canada and thus also enriched this analysis.

Although discussion for this analysis was predominantly concerned with federal public health discourse, Quebec-based research and policy has contributed substantially to the body of Canadian knowledge on lead poisoning and contrasts with English public health discourses on lead in Canada. The Institut national de santé publique du Québec policy paper, D finition nosologique d’une maladie à déclaration obligatoire ou d’une intoxication et d’une exposition significative: Le plomb (Plante et al., 2003) therefore, also provided important insights for this analysis. Additionally, many pieces of commentary inform this study such as letters
featured in the *Canadian Medical Association Journal*, the *Canadian Public Health Association Journal, Pediatrics and Child Health*, and *Clinical Chemistry*.

The early 1990s was a pivotal transitional point in public health discourse on childhood lead poisoning in both Canada and the U.S. Leaded gasoline, for example, was, for decades characterized as the predominant source of exposure for North American children. This view changed with leaded gasoline’s virtual elimination and the identification of “new” sources. Particularly relevant to this discussion are the data and findings on childhood lead poisoning rates for U.S. children derived from the multi-year U.S. National Health and Nutrition Examination Surveys (NHANES). NHANES began collecting information on American children’s blood lead values beginning in 1976 (1976–1980); and for the years 1988–1991, 1991–1994, and 1999–2004 (Jones et. al, 2009; Lin-Fu, 1985; Meyers et al., 2003a). Subsequent discourses emanating from these findings played a major role in repositioning historic sources of lead paint found in older homes as the new primary source of exposure, as evidenced in American public health discourse. Pirkle and colleagues (1994), for example, demonstrated graphically, the continued existence of residual populations of American children identified with lead poisoning and thus, was used, successfully, to challenge emerging public health discourses that childhood lead poisoning had been defeated and therefore a problem of the past (Jacobs, 1995).

What influence, if any, did the extensive American surveillance data have on Canadian public health discourse? The Google Scholar [cited by] tool was used to gauge strength of interest in American discourse, but also to identify Canadian authors who themselves cited various influential NHANES related American discourses and less influential Canadian sources. Also fruitful for this discussion were the proceedings from a 1995 international conference on lead, organized by
the American Society for Testing and Materials (ASTM, 1995), as it provides an understanding of what was known about lead-based paint, dust and its contribution to children’s blood lead levels during the early 1990’s.

Inclusion criteria. Content from Web sites, journal articles and other text was included for the analysis if it met the following criteria: a) it pertained to the Canadian context; b) it pertained to childhood lead poisoning; and, c) it played a strategic role in transforming the public health discourse on childhood lead poisoning in Canada. A total of 214 items were screened for relevance. The 139 items which met inclusion criteria were included for further analysis. A complete list of titles, including material not meeting inclusion criteria (75 items) can be found in Appendix F.

Content from sources of information meeting criteria was extracted using a guide adapted for this inquiry. The guide is based on Rawlinson’s (1987) three axes of inquiry (Appendix G). Following this, content was further synthesized into a cohesive analysis and discussion identifying dominant and non-dominant discourses, structures or apparatuses that support and maintain dominant discourses on childhood lead poisoning in Canada, the technologies that suppressed conflicting/dissenting discourses, and an examination of the various contingencies that enabled certain “truths” concerning childhood lead poisoning to emerge.

1.5.3. Data analysis: Critical Discourse Analysis

Critical discourse analysis distinguishes itself from discourse analysis by its critical component. The underlying assumption is that language shapes, not merely reflects, the world in which people live. As discussed in Article 2, much of the research in Canada on lead poisoning occurred in smelter communities or
occurred in the form of clinical investigations. The majority of blood lead surveys that did involve children occurred during the era of leaded gasoline usage. Article 1 describes how childhood lead poisoning resulting from exposure to residential sources of lead is an under-researched area in Canada. While these gaps in data make it difficult to define the extent of this problem, rather than use this as grounds to dismiss the existence of a problem, this thesis contends that evidence from case, cross sectional and environmental studies supports an alternative view: that childhood lead poisoning from residential source exposure has been a problem in Canada in the past and, untreated, continues to be a problem today.

The intention of this critical analysis is not to “prove” that one interpretation is correct or accurate but to search for insights, and in the process, attempt a restructuring of the social relations of domination that shape current understandings of childhood lead poisoning (Kincheloe & McLaren, 1998; Lupton, 1995). A critical approach requires researchers to perceive the world in ways that call into question obvious assumptions through “careful and thoughtful analyses of the political implications of knowledge development, the inter-textual relationships of objects, and decentralization strategies” (Holmes et al., 2006, p. 95-96). A critical approach can uncover the many unseen or unmeasured metaphysical factors that contribute to this problem. Lead, for one, is elusive in blood. Blood lead levels tend to peak in children between the ages of 1 and 3 years and diminish thereafter (Meyers et al., 2003d). The definition of “elevated blood lead level” itself is based on the ability of state-of-the-art equipment to accurately detect low levels of blood lead, and the human resource capacity for dealing with populations defined as “lead poisoned.”

While traditional methods of qualitative research attempt to capture “reality” through the gathering of individual stories and lived experience in language, critical
discourse analysis is more concerned with the way historical and social context shape individual experiences, as expressed through text (Crowe, 2005). Critical discourse analysis draws attention to power imbalances, social inequities, non-democratic practices, and other injustices in hopes of “spurring people to corrective actions” (Fairclough, 1995, p. 132). Critical discourse analysis is founded on three key premises: knowledge of the world should not be taken as “objective” truth; all knowledge is historically and culturally located; and, knowledge of the world is created and maintained through competing social interactions (Jorgensen & Phillips, 2002).

This thesis proposes critical discourse analysis as a radical and appropriate method for the topic of childhood lead poisoning in Canada. A discourse analysis using Foucault’s archaeological-genealogical approach offers what McHoul and Grace (2003, p.27) term a “counter reading” of history by extending an appreciation of the social political and economic conditions that shaped Canadian public health discourse on lead.

1.5.4. Foucault’s approach

French philosopher and historian Michel Foucault conceived of discourse analysis in broader terms than did Aristotle’s approach to analyzing rhetoric or the linguistic approach to analysis (described earlier). Foucault’s method of analysis requires more than simply judging whether or not a discourse is well received (McHoul & Grace, 2003). For Foucault an important distinction of discourse is the recognition that language is not passive. Discourses have the ability to shape or constitute, for example, what is considered ‘normal’ or ‘standard’ (McHoul & Grace, 2003). Discourses are produced and supported by hidden rules defining or delimiting what can be said on a particular topic as well as who gets to say it.
While Fairclough’s (1992) method of critical discourse analysis concerns itself with hegemonic ideology, oppression, consciousness-raising and emancipation, a Foucauldian approach implies not so much finding blame or the source of oppression, as finding interconnected pieces of history or archives that impart knowledge or understanding of the relationship between what is “sayable” and what is “visible” (Kendall & Wickham, 1999). Foucault’s approach to history is to select a problem rather than a historical period for investigation, to look for contingencies rather than causation (Kendall & Wickham, 1999). The researcher is specifically concerned with the way in which knowledge gets translated into ‘being’ through, for example, forms of institutionalization, surveillance systems and privileging (Kendall & Wickham, 1999).

The archaeological approach, described by Foucault in, *The Archaeology of Knowledge* (1972) concentrates attention on the “systems of unwritten rules which produces, organizes and distributes the ‘statement’” (p. 130). This method of analysis involves an archival examination of emergent discourse by paying close attention to the necessary conditions that allow certain discourses to emerge and prevail whereas others do not. Foucault’s genealogical approach complements this method by employing an explicit questioning of whose voices are being heard and privileged, which body of expertise is cited in support, what counts as knowledge, who has access to knowledge and how this knowledge is justified and used. Disciplinary techniques which maintain or support dominant discourses are also examined (Rawlinson, 1987).

1.5.4.1. *Foucault’s archaeology*

Archaeology is the analysis of systems of hidden rules which produce and organize the ‘statement’ (Foucault, 1972b, p. 130). Whereas traditional historical
examinations aim to create a “total history” by searching for overarching distinguishing characteristics and principles, an archaeological analysis is dismissive of all generalizations and totalizing theories and instead concentrates on describing, historically, emerging differences, transformations, continuities, mutations and possible relations between events (Kendall & Wickham, 1999). The focus of archaeological research is on the archive, the collected texts from a given period and the set of relations and institutions that enable statements to exist (Foucault, 1972b). All meaning and all truth are interpreted within the context of history. The archival researcher holds no assumptions concerning the legitimacy of knowledge and suspends all acceptance of ‘truth.’

Truth is a major consideration of Foucault’s approach to discourse analysis. Foucault considers truth to be materially produced and socially situated (Rawlinson, 1987). He uses the term “regime of truth” to denote the hidden rules in society that govern the designation of statements as true or false:

Each society has its regime of truth, its general politics of truth: that is, the type of discourse which it accepts and makes function as true; the mechanisms and instances which enable one to distinguish true and false statements, the means by which each is sanctioned; the techniques and procedures accorded value in the acquisition of truth; the status of those who are charged with saying what counts as true (Foucault, 1980b, p. 131).

Foucault also challenges the idea that knowledge is “value-free, progressive and universal” (Cheek, 2000, p.21) and encourages an examination of the rules that allow phenomena to be viewed in certain ways. The role of the critical researcher, then, is not to search for truth, but to make explicit the variety of constraints and restrictions which delimit what can be said or constituted as “true.” These underlying rules or limits are not always apparent. As Mills describes it, although we think we can “potentially utter an infinite variety of sentences... what is
surprising, in fact, [is that] we choose to speak within very narrowly confined limits” (Mills, 2003, p. 56). Rawlinson (1987) further explains:

[U]nder the "archaeological" aspect of Foucault's method all commitments on truth and falsity with respect to specific objectivities are suspended in favor of an analysis of the system of concepts, rules for the formation of meaningful statements, and procedures for determining truth and falsity in the discourse for which those objectivities are constituted as real (p. 376).

The critical researcher, through archaeological analysis attempts to provide a non-judgemental account of the institutional origins of statements by uncovering rules and systematic shaping factors which form the épistémé, the common knowledge representations of the world (Foucault, 1966). Knowledge is seen as an unstable, complex set of discursive relations that make it possible for statements to qualify as something that is 'known' (Kendall & Wickham, 1999). The researcher identifies, for example, how disciplinary boundaries serve to limit our view on a subject area by proscribing what can be counted as possible. Analyzing the relationship between statements and the rules for their formation is an integral part of archaeological research:

...when natural history becomes biology, when the analysis of wealth becomes economics”, when above all, reflection upon language becomes philology and classical discourse, in which being a representation found their common locus, is eclipsed, then, in the profound upheaval of such archaeological, man appears in his ambiguous position as an object o knowledge and as a subject that knows (Foucault, 1966, p.312).

Foucault's archaeological method of discourse analysis employs a multi-dimensional approach for locating statements within their historical and social context. The statement serves as the basic unit of discourse analyzed in the archeological method and can be anything from a scientific graph to a sentence in a novel (Kendall & Wickham, 1999). The determination of whether or not a written
or spoken word counts as knowledge (i.e. scientific fact) depends on a wide range of discursive conditions such as the formation of specific 'objects' of knowledge or 'strategies' which deploy competing theories against one another (Foucault, 1972b).

1.5.4.2. Foucault's genealogy

A genealogical analysis is similar to the archaeological method in that it also describes the various contingencies or material conditions that must be in place for events to occur. Its focus, however, is not on the historical context, but rather on the underlying power structures which allow certain statements to be perceived as 'true' (Kendall & Wickham, 1999). Rawlinson describes Foucault's genealogical approach in this way:

A genealogical analysis …reveals that the epistemological and the political, knowledge and power, are ineluctably intertwined, so that truth is not so much discovered — as if it lay ready-made in an objective reality patiently awaiting the articulate voice of science — as produced according to regular and identifiable procedures that determine in any given historical situation what it is possible to say, who is authorized to speak, what can become an object of scientific inquiry, and how knowledge is to be tested, accumulated, and dispersed (Rawlinson, 1987, p.372).

Foucault developed the phrase power/knowledge to underscore the way in which these two phenomena, knowledge and power, are mutually dependent (Kendall & Wickham, 1999). All knowledge is inextricably linked to power, and discourses always function in relation to power relations:

We should admit...that power produces knowledge (and not simply by encouraging it because it serves power or by applying it because it is useful); that power and knowledge directly imply one another; that there is no power relations without the correlative constitution of a field of knowledge, nor any knowledge that does not presuppose and constitute at the same time power relations (Foucault, 1977, p.27).
Foucault developed various “ordering” tools that can be used to “flush out assumptions” (Kendall & Wickham, 1999, p. 30). In the *Archaeology of Knowledge* (1972) Foucault distinguishes three dimensions wherein objects become subjects in a discourse: a) surfaces of emergence, b) authorities of delimitation, and c) grids of specification. *Surfaces of emergence* relates to the point in which discourses appear. Kendall and Wickham refer to discourse and the subsequent emergent content as the “sayable” and the “visible” (Kendall & Wickham, 1999). Discourses always arise in relation to other ways of thinking and not independently. Cooper’s (2000) discourse, for example, identifying the potential risk for children resulting from exposure to consumer products (the “sayable”) emerged as a response to the prevailing view that childhood lead poisoning from major sources such as leaded gasoline had been eliminated in Canada. Cooper’s discourse (2000) resulted in the formation of new federal laws and regulations governing the lead content in children’s products manufactured in Canada (the “visible”) and was itself generative of new discourses and subjects. Once childhood lead poisoning in Canada became identified as a problem related to lead in consumer products, for example, a whole new regulatory dialectic for identifying what cut-off age constitutes children’s jewellery, as opposed to adult jewellery, emerged.

*Authorities of delimitation* provides limits for the identification of subjects regarding a particular discourse. Canadian public health discourse on childhood lead poisoning is “shaped” in relation to American public discourse on the topic. Foucault, in the *Archaeology of Knowledge* refers to discursive “police” that also delimit what can be said (Foucault, 1972a). As Mills explains:

Thus, even if we are asserting something which as afar as we know it is ‘the truth,’ our statement will only be judged to be ‘true’ if they accord with, and fit in with, all of the other statements which are authorized within our society (Mills, 2003, p.58).
“Policing” of unauthorized discourse is accomplished through various means including commentary, by, for example, writing about another’s statements; or, by establishing disciplinary boundaries and institutions which proscribe limits on what can be said, by whom, and counted as “true” (Mills, 2003); or, through bio-power (also termed power/knowledge) via the production of willing and able bodies that support the status quo (Powers, 2001). Foucault uses the term “repressive hypothesis” to refer to the widely held and uncritical assumption that science is progressive, producing value-free and objective truth. Scientific knowledge then, positioned uncritically and perceived as freely accessible and progressive, is viewed as adequate on its own for preventing dominant authorities from applying “unchecked power over ordinary people” (Powers, 2001, p.18). “Radical ideas” resisting or challenging this established faith in the integrity of science while “unfettered” are unheard because they “seem so illogical, irrational, nonsensical, disorderly, uncivilized, and unscientific” (Powers, 2001, p. 16). Rejecting science is perceived as “rejecting rationality” (Powers, 2001, p.19).

Foucault’s term grids of specification refers to taxonomies used in the classifications of subjects, such as the medically derived definition of lead poisoning which is based on physiological measures such as blood lead levels. The reference or standard “normal” blood lead level is derived from nationally representative blood lead surveys of adults and children. As children’s average blood lead levels declined, the reference value for what should be considered a “normal” blood lead level was also lowered.

The nexus between discourse (what is sayable) and discursive practice (what is visible) is what Foucault terms power. Power is productive, but Foucault shuns the idea that power is solely repressive:
We must cease once and for all to describe the effects of power in negative terms: it “excludes”, it “represses”, it “censors”, it “abstracts”, it “masks”, it “conceals”. In fact, power produces, it produces reality, it produces domains of objects and rituals of truth (Foucault, 1977, p. 194).

The notion that scientific knowledge is ‘discontinuous’ rather than progressive is central to a genealogical analysis.

What [genealogy] really does is to entertain the claims to attention of local, discontinuous, disqualified, illegitimate knowledges against the claims of a unitary body which would filter hierarchies and order them in the name of some true knowledge …genealogies are therefore not positivistic returns to a more careful or exact form of science. They are precisely anti-sciences (Foucault, 1980c, p.83).

Foucault conceives of all knowledge as being the product of a vast and complex web of discursive and institutional relationships rather than a neutral phenomenon that is value free and objectively derived from, for example, scientific study (Foucault, 1972b). The aim of a genealogical analysis is to uncover the intricate web of power relations which shape current understanding. Fontana (2004) describes the emancipatory benefit derived from this type of analysis:

By historicizing the sources and motives of prevailing ideas and practices, it critiques ideology while remaining intensely skeptical of surface appearances. Digging beneath the surface provides a means by which people can radically alter any false self-conceptions (p.95).

Knowledge in any discourse will be whatever is considered to be true as defined by scientific peers (Powers, 1996). A genealogical analysis of public health discourse pertaining to childhood lead poisoning, then, might start by examining the way in which public health discourse is situated within the discipline of science. Thus positioned, public health brings with it a system of elaborate scientific rules which dictate what counts as knowledge, who is authorized to speak, and what objects will come under study (Rawlinson, 1987). From this the researcher might
describe how the field of public health is philosophically value laden and how these values are reflected in the overall conceptual approach to inquiry (Fontana, 2004). Public health discourse, for example, valorizes epidemiological knowledge, the setting of norms and standards and statistical generalizations over other forms of knowledge such as anecdotal accounts or case studies (Powers, 1996). The artificial ranking of certain types of knowledge then, authorizes certain practices within public health, such as dividing the ‘normal’ population from the ‘abnormal.’ A genealogical examination highlights these practices and through this process challenges established measures such as the concept of ‘normalcy’ (Rawlinson, 1987).

Genealogy further explores the series of épistêmés through which a given science has ‘progressed’ via prevailing modes of knowledge and investigations. Foucault uses the term épistémé to represent knowledge, albeit selective knowledge produced within a given historical period or archive, and governed by rules which specify or delimit its application and generation (Mills, 2003). Foucault later expanded the definition of this term (épistémé) to describe how knowledge can be strategically used to support and maintain dominant discourses:

…the épistémé is the strategic ‘apparatus’ which permits of separating out from among all the statements which are possible those that will be acceptable within...a field of scientificity, and which it is possible to say are true and false. The épistémé is the ‘apparatus’ which makes possible the separation, not of the true from the false, but of what may from what may not be characterized as scientific (Foucault, 1980a, p.197).

Genealogy allows the researcher to access certain historical events which might otherwise be overlooked as inconsequential by critically examining the interplay between epistemology (what we know on a subject) with how this knowledge is used to maintain dominant views (knowledge/power) (Hunt & Wickham, 1994).
Deborah Lupton in, *The Imperative of health* (1995), for example, describes how public health and health promotion serve as government apparatuses to dispense wisdom and expert advice “directed at improving individual’s health through self-regulation” (p. 10).

An archaeological analysis identifies the conditions or contingencies that establish, sustain, and advance a particular discourse (Kendall & Wickham, 1999) whereas a genealogical analysis concerns itself with the concepts of power and knowledge and systems of authority and constraint. Aspects of these methodologies and their implications for this research analysis are further explored in the following section.

1.5.5. Rawlinson’s Three Axes of Inquiry

In her paper entitled *Foucault’s strategy: Knowledge, power, and the specificity of truth* (1987), Rawlinson traces the progress of Foucault’s concept of power “from its initial formulation … as a merely repressive force” to its “mutation” into “a positive and productive force that engenders both knowledge and pleasure” (p. 374). Apart from summarizing Foucault’s many writings, Rawlinson (1987) promotes Foucault’s work as an effective means for health professionals to challenge the “normalizing practices of the human sciences” (p.373). She poses the following for reflection:

> What is being done when actual human subjects take up as their own the sanctioned problems and methods of formalized disciplines and professions, and deploy upon others the practices and procedures authorized by them?” (p. 374).

Rawlinson (1987) offers aspects of Foucault’s writings as a means of casting out “all commitments on truth and falsity” (p.376). She facilitates the process for “objectifying discourses” by organizing Foucault’s archaeological and
genealogical approach for critical analysis along three axes: 1) The axis of knowledge, which investigates assumptions concerning true and false; 2) The axis of authority, which examines systems of power; and 3) The axis of value/justification, which concerns itself with technologies of power as found in disciplinary measures. These three axes of inquiry guide this critical analysis and are explicated in more detail below.

1.5.5.1. The axis of knowledge

Rawlinson’s first line of inquiry, the axis of knowledge, involves an examination of the rules governing the formation of statements and the determination of what is considered to be true and false. Analysis along this axis involves identification of rules of evidence, rules concerning what can be addressed and what cannot, and rules for the repeatability or use of statements. The researcher examines the way, for example, public health policy/statements are created by describing the rules governing the use of language, the way in which statements are distributed and read, and the audience who reads them. Every discourse has an internal system of truth that can be recognized by its rules (Powers, 1996). This axis might also be termed rules for “the sayable.”

The analysis (Article 2, Part III), along this axis, involves a careful examination of the public health discourses as found in various text such as peer reviewed journals, government and non-governmental reports, and federal Web sites including Health Canada, Canada Mortgage and Housing, the Canadian Pediatric Society and the Canadian Environmental Law Association. The following questions are explored: What are the dominant public health discourses on childhood lead poisoning in Canada? What are the rules for what can be spoken?
What engenders evidence? What determines the difference between true and false; and, who is accorded the power to pronounce truth?

Dixon and Dixon’s model (2002) gave “shape” to the épistème on childhood lead poisoning in Canada (Article 1), and from this exercise, major gaps in Canadian research were identified. Limited as the research has been, this thesis posits that there is adequate evidence to support the contention that childhood lead poisoning occurring from residential sources is a health issue worthy of attention in Canada. As power/knowledge is productive agencies could be expected to produce tools and instruments for the management of childhood lead poisoning, and, accordingly, knowledge of this health problem should have triggered protective action in Canada such as occurred in the United States (i.e. establishment of Title X, a law that requires landlords and home sellers to disclose that their dwellings contain lead paint) or case management guidelines for children identified with lead poisoning such as those developed by the U.S. Centers for Disease Control and Prevention.

Many historic sources of childhood lead exposure have already been addressed in Canada and the United States, through the elimination of lead in gasoline; restriction of lead in lead solder; and elimination of lead in solder of food cans. Where these two countries differ is in the approach to remaining sources. The United States recognizes pre-1978 housing sources as the major contributor to childhood lead poisoning (American Academy of Pediatrics, 2005; CDC, 2005b) and American agencies have been working aggressively for over two decades to remediate this source of lead. Canada’s lead strategy, in sharp contrast, focuses exclusively on lead in consumer products. While Dixon and Dixon’s model was useful in identifying gaps in research and policy it was not equipped to explain the disjunction between knowledge (epistemological domain) and action (health
protection domain), or in Kendall and Wickham's (1999) terms - between the “sayable” and the “visible.”

1.5.5.2. The axis of authority

Foucault understands truth as being supported by a whole range of practices and institutions including universities, government departments, publishing houses, scientific bodies and so on. Only ‘true’ statements become circulated (Mills, 2003, p. 58). Rawlinson’s (1987) second line of inquiry, termed the axis of authority, investigates and describes “the system of power” or what she also calls, the “bureaucratic dimension of discourse” (p.378). Analysis along this plane focuses on “the visible.” As Rawlinson explains, “[t]here can be no truths or falsehoods about quarks or schizophrenia outside of the sciences that posit them” (p. 377).

The analysis using Rawlinson’s second axis examines what Foucault (1972a) terms “surfaces of emergence,” that is, points in history where various discourses on childhood lead poisoning emerged. The analysis is assisted via a comparison of Canadian public health discourses with that of U.S. public health discourse. Contingencies such as the elimination of leaded gasoline transformed the public health discourse on childhood lead poisoning in the early 1990s, in both Canada and the United States. How did the transformed American public health discourse differ from that of Canadian public health discourse? What contingencies peculiar to the U.S. and Canadian experiences allowed these variations in discourse to occur?
1.5.5.3. The axis of value/justification

The third axis is the *axis of value or justification*. Also referred to by Rawlinson as the “technologies of power,” this axis examines systems for the establishment of authority (1987, p.378). Mechanisms of power are enabled via “the production of efficient instruments for the formation and accumulation of knowledge – methods of observation, techniques of registration, procedures for investigation and research, apparatuses of control” (Smart, 2002, p.78).

At this level it is particularly important to be aware of the social and historical context surrounding the textual medium. A portion of this analysis was accomplished via the literature review in (Article 1) which collected and reviewed the “scientific” and “public health” discourse on childhood lead poisoning in Canada extending to 1932. The 24 case studies, blood lead surveys and environmental surveys form the épistème on childhood lead poisoning in Canada. The analysis (Article 2) explores how this knowledge (épistème) was used as an apparatus to shape the Canadian public health response to lead/lead poisoning.

1.5.6. Rigour criteria

Methodological rigour is a particularly important consideration in assessing the strength of a qualitative analysis. Loiselle and colleagues (2004) suggest four measures of rigour for use in qualitative analysis: credibility, dependability, confirmability and transferability. Criterion such as dependability (the measure of data stability), however, is not suitable to this analysis due to the fact that critical discourse analysis applies a method which is context dependent and open to multiple interpretations (Crowe, 2005). The criteria of credibility and confirmability do have application for this purpose and are used to establish methodological rigour.
1.5.6.1. Credibility

Credibility is the degree of confidence one has in the data and its subsequent analysis. Consideration is given to such things as the researcher’s length of engagement with the data, and whether sufficient time was given towards data collection to afford an in-depth understanding of the issue at hand. Also considered is the researcher’s previous experience with the issue. As a Registered Nurse (BScN), I have extensive experience in the field of children’s environmental health, and growing expertise on childhood lead poisoning as it relates to exposure occurring from residential sources. I was the lead author for the peer reviewed Ontario Public Health Association (OPHA) position paper, *Childhood lead exposure and housing sources: Does a problem exist in Ontario?* (2004) and the policy paper by The Association of Community Organizations for Reform Now (ACORN), *Paint Poisoned Homes: An Action Plan to Eliminate Childhood Lead Poisoning in Canada by 2020* (2007). In 2005 I was contracted as an external reviewer for the report: *Update of Evidence for Low-Level Effects of Lead and Blood Lead Intervention Levels and Strategies* (Health Canada, 2005) and I was the contracted writer for Canada Mortgage and Housing Corporations’ webpage series *About the House* on lead. Research for this current thesis, in addition to building upon the work begun through the OPHA and ACORN position papers, occurred over a period of two years. A variety of resources from historical, clinical and political text were accessed for this research project using methods described earlier.
1.5.6.2. Confirmability

Confirmability infers the reviewer’s ability to find a plausible linkage between the discourse and the interpretation. Crowe describes discourse analysis as an “interpretative process” that “acknowledges the multiple interpretations that can emerge from the text” (Crowe, 2005, p.61). Due to these characteristics, concern has been raised that researchers, approaching a topic from a certain ideological perspective may elect to include for analysis only those texts which support their position (Blackledge, 2005). Wherever possible I have constructed tables which summarize the text used as the basis for competing arguments or contentions (i.e. Appendix A). I make no claim for the absolute truth of this discourse analysis. I openly acknowledge that competing claims are also possible regarding these same discourses; however, as noted by Powers (2001) this limitation applies equally to other methods of inquiry as well.

Triangulation involves the use of multiple perspectives and multiple methods which can also be used to establish confirmability. Blackledge’s (2005) method of triangulation is specific to critical discourse analysis and involves four levels of analysis: a) a reading of the immediate text; b) an analysis of the inter-textual and inter-discursive relations between discourse; c) a discussion of the extra-linguistic social/sociological variables and institutional frames within a context; and, d) an examination of the broader socio-political and historical contexts within which the discursive practices situated. Rawlinson’s (1987) three part approach to critical discourse analysis, which is based on the genealogical-archaeological writings of Foucault, is similar to Blackledge’s (2005) method and engendered an in-depth multi-level analysis.
1.5.6.3. Issues of bias in qualitative inquiry

The unique methods of discourse analysis leave it perhaps more vulnerable than other “traditional” or empirical research methods to challenges over concerns with research bias, objectivity and rigour. The concern with bias in research is longstanding. In critical theory, an approach rooted in its response to positivism, the researcher openly acknowledges the set of social, political and ethical values he or she brings to the table and holds no pretense of objectivity. In critical discourse analysis, the researcher is aware that research findings are always open to alternative, even conflicting interpretations. Even so, critical theorists maintain that all research, including empiricist research, is inherently biased – beginning, for example, with the way research questions are formulated. Critical theorists view the need for science to uncover an objective truth as itself a dominant discourse and one that should be challenged (Denzin & Lincoln, 1998; Fairclough, 1992). Blackledge (2005) views selection as a necessary part of the analysis because a “complete” analysis would be “unmanageable” (p.17). Rather than view research bias as a negative component, and something to be eliminated, critical researchers recognize the richness that inherent values and experiences bring to investigation of a topic (Olesen, 1998).

The complete list of retrieved research, journal articles, and technical documents used for this analysis are included in Appendix C and D for use as an audit trail. Additional sources of text used for this analysis are listed in Appendices A and F.

1.5.6.4. Reflexivity

Reflexivity is another important tool for establishing credibility in data (Kincheloe & McLaren, 1998; Lupton, 1995; Denzin & Lincoln, 1998). Admittedly
this research topic presents ample opportunity for me as researcher to impose my own system of values, standards, and beliefs into the process of analysis. For one, I bring a personal interest to this topic which began with the discovery that one of my children was lead poisoned. The source of exposure, I later learned, was our hundred year old home. Analysis revealed lead-based paint in the painted trim, porch boards, window sills and wallpaper. The anger I initially felt – mainly for not being better informed – energized my quest for answers, but it also caused me to be one-sided in my appraisal of the situation. Over ten years has passed since this event and I have since developed the skills to enlist a more systematic approach to examining this health issue. Even so, the opportunity for injecting bias into this critical discourse analysis exists, as it does with any kind of research - no matter what the method design may be. Accepting bias as an inherent part of the research process therefore obliges researchers to openly acknowledge, through reflection, any “deep-seated but poorly recognized views on issues” including an awareness of one’s privileged position as researcher (Olesen, 1998, p.314). Instead of a striving for total objectivity, then, I openly acknowledge my position as a “mother of a lead poisoned child” but I also acknowledge the richness this experience imparts to my understanding of this health issue. I also recognize that my position as “nurse” brings with it related values as a member of an educated, privileged, predominantly white, female, middle-class group. Bias in research is problematic if one is not consciously aware of one’s “subjectivity” (Fairclough, 1992). Reflexivity brings one to be consciously aware of one’s “self-location” in what Kincheloe and McLaren (1998) term “the net of larger and overlapping social, cultural, and economic contexts” (p. 279).
1.5.7. Ethical considerations

Ethical considerations are established to protect individuals involved in research, and to ensure that guidelines for ethical research are followed. All documents obtained for the purpose of this research are publicly available and can be accessed without special permission; therefore, this thesis was not subject to formal ethical approval. Further, key values upon which ethical principles for this thesis were based, were adapted from the Public Health Leadership Society, *Principles of the ethical practice of public health* (2002).

Ethical considerations also entail an examination and evaluation of potential harm occurring from research activities as well as benefits. The benefits of this analysis, apart from contributing new knowledge (i.e. a database consisting of Canadian public health research pertaining to lead), lies in its potential to critically examine the ways in which childhood lead poisoning has been represented in Canada, thus raising consciousness by introducing alternate speaking positions on this health topic. The ethical implications of conducting an examination of childhood lead poisoning, and its potential for harm, are discussed in further detail, in Part III, Discussion.
PART II: RESULTS

Introduction

The aim of critical discourse analysis is “explanatory critique” – the identification of a problem in society or the disclosure of a misrepresentation; or, a disconnect between reality and the public perception of reality (Chouliaraki & Fairclough, 1999, p.33). Sabbattini and Crosby describe the process in this way:

Where others see smooth blandness, we peek beneath the surfaces to examine all the hidden roughness and irregularities. We listen for the dog that does not bark and watch the lips that do not speak. Conscious of the silences and knowing that the way one asks a question can determine the kind of answer one gets, we are suspicious of data *qua* data...[w]e ought to distrust anyone that cautions us to ‘let the facts speak for themselves.’ If you find a speaking fact, look right away for the ventriloquist (2003, p.265)

A Foucauldian discourse analysis typically entails the identification of how and where, historically, issues, behaviour, or phenomena become identified as problems while other equally concerning behaviour or phenomena are neglected. Foucault’s characterization of the emergence of the discourse on “madness,” for example, exemplifies this process (Foucault & Michel, 2001, p.171). Powers (1996), using concepts from Foucault’s text *The Archaeology of Knowledge* (1972), provides further examples of the ways psychiatric pathologies have become “problematized.”

This thesis uses the case study of lead poisoning to critically examine the responses of Canadian agencies accountable to the issue of childhood lead poisoning occurring from exposure to residential sources of lead found in paint and paint dust. Article 1 began this analysis via an examination of the process gaps located between Dixon and Dixon’s “epistemological domain” (what we know about the problem of childhood lead poisoning in Canada) and the “health protection
domain” (what was done to ‘fix’ the problem). Limited as the research has been, the article established that adequate evidence exists to support the contention that children in Canada have been harmed from exposure to lead-based paint. Accordingly, knowledge of this health problem should have stimulated a protective health response in Canada such as occurred in the United States (i.e. establishment of Title X) - but this did not occur. While Dixon and Dixon’s model was useful for identifying gaps in research and policy it was not equipped to explain the disjuncture occurring between knowledge and action.

The goal of the analysis that follows is to provide interpretative claims describing power relation within a specific historical context. The strength of a Foucauldian genealogical analysis for this analysis is its focus on discourse, recognition of power/knowledge strategies, descriptions of discipline technologies, subsequent consequences, and suggested possibilities of resistance (Powers, 2001). Key to this approach is an analysis of the historical formation of authority and its unchallenged right to pronounce truth. In critical discourse analysis there are no claims to objectivity. Powers describes a genealogical approach in this way:

No assumption is made to the effect that it can be determined exactly how people came to think and talk and act this way in some objective fashion. A genealogy is an interpretation openly arising from a postmodern, antifoundational, feminist orientation concerning the operation of power in a specifically situated context (2001, p. 54).

The analysis contained in this second article seeks to identify dominant public health discourses on childhood lead poisoning in Canada by comparing public health discourses on lead between Canada and the U.S. with a view to identifying “transformations” and “surfaces of emergence” in discourse; and, through careful review of the historical contingencies that permitted dominant discourses to be created, circulated and maintained. A description of Foucault’s
Archaeological/Genealogical methods is briefly introduced in Article 2, which follows the American Journal of Public Health (AJPH) author guidelines pertaining to the genre of analytical essay known as “Framing Health Matters.”
ARTICLE 2
Reformulating lead-based paint as a problem in Canada

Paper to be submitted to the
American Journal of Public Health
Reformulating lead-based paint as a problem in Canada

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Abstract

The post leaded gasoline era marks an important transition point in Canadian public health discourse. Leaded gasoline was officially removed from the Canadian market December 1990. The removal of a major lead source and the subsequent decline in children's blood lead levels sparked the emergence of new discourse on lead. Today, in Canada, childhood lead poisoning is viewed as a problem of the past or a problem of the United States. Sparse Canadian surveillance data supports this view. Further, tensions among federal agencies evolved into a power struggle with Health Canada ultimately succeeding to become the dominant authority, thereby relegating to obscurity important research initiatives, but also shaping a vastly weaker response to lead than occurred in the United States.

Key words

lead poisoning; critical discourse, Canada
Introduction

Major differences occur in the way childhood lead poisoning is constructed between Canada and the United States. Canadian public health discourse portrays this issue as a problem of the past or an American problem. American public health discourse portrays lead poisoning as a major and preventable disease, and one requiring concerted and sustained effort to eradicate all sources of lead exposure for young children.

Rawlinson (1987) promotes aspects of Foucault’s archaeological and genealogical strategies towards discourse analysis as an effective means of casting out “all commitments on truth and falsity” (p.376). She facilitates Foucault’s process for objectifying discourses by organizing his approach to inquiry along 3 axes: The axis of knowledge investigates assumptions concerning true and false, the axis of authority examines systems of power, and the axis of value/justification concerns itself with technologies of power as found in disciplinary measures (Rawlinson, 1987). This analysis adapts Rawlinson’s (1987) approach to offer what McHoul and Grace (2003) term a “counter reading” of the history of childhood lead poisoning in Canada, with a specific focus on discourses related to lead-based paint in the post leaded gasoline era. Our analysis seeks to identify dominant public health discourses on lead poisoning in Canada, highlighting the socio-political processes that shaped health care knowledge and influenced the way knowledge on lead was produced and used by health care providers and policymakers. This analysis is assisted via a comparison with American public health discourses on lead, with a view to identifying the historical contingencies that permitted dominant discourses in Canada to be created and maintained.

We argue that differences between the two countries exist primarily due to the fact that blood lead surveillance data for U.S. children was readily available
and easily accessible; whereas surveillance data for Canadian children was sparse
and accessible to a privileged few. This contingency, sparse Canadian surveillance
data set against a backdrop of abundant American surveillance data, justified the
insertion of Ontario’s limited and sporadic blood lead surveys as the surrogate
indicator for Canada’s children. As leaded gasoline was gradually phased out, and
as average children’s blood lead levels began to decline, limited blood lead survey
data and selective references to American survey data helped shape the
construction of a dominant view in Canada, that childhood lead poisoning was a
problem of the past, an American problem, or a problem mainly confined to smelter
communities. Conversely, this same contingency, sparse surveillance data
disadvantaged and disqualified equally vocal discourse proposing that lead
poisoning from exposure to residential sources did occur in Canadian children, at
similar rates to U.S. children.

Lastly, we suggest that tensions arising between Health Canada and
Canada Mortgage and Housing Corporation (CMHC), Canada’s two federal
agencies assigned responsibility for health, housing and lead, evolved into a power
struggle in the mid 1990s, with Health Canada ultimately succeeding to become
the dominant voice, thereby diminishing CMHC’s role and authority, but also
vanquishing important CMHC research projects, technical reports and initiatives on
lead in housing.

These contingencies combined to produce a vastly weaker public health
response to lead in Canada than the U.S., with the result that today no legislation
exists at the federal level to protect Canadian children living in lead contaminated
older dwellings. Current discourse resulting from the Canadian Health Measures
Survey, which emphasizes declining average blood lead levels, further breeds
support for current policy despite important limitations.
Formulating the problem of lead-based paint in Canada (1930-1980)

Lead is important to study for its population health effects. Evidence suggests that blood lead levels lower than our current level of concern ($\text{PbB} \leq 10$ g/dL) harm children's renal and blood-forming systems and neuroendocrine and reproductive systems (CDC, 2005; Health Canada, 2008). Lead’s properties as a developmental neurotoxin also deserve attention for its sociological implications. Lead exposed children are more likely, than less exposed peers, to suffer lower IQ scores manifested as poorer school performance and behavioral effects diagnosed as attention-deficit/hyperactivity disorder (CDC, 2005; Nigg et al., 2008; Nigg et al., 2010).

Canadian discourse on lead-based paint first emerged in the 1930’s in two of Canada’s oldest health related journals: the Canadian Medical Association Journal (CMAJ) and the Canadian Journal of Public Health (CJPH). Mitchell, in a 1932 issue of the CMAJ described two cases of lead poisoning occurring in Montreal children: a 9 year old girl who “stumbled when she walked, and could not use her hands properly to feed herself” and a three year old girl hospitalized “in generalized convulsions of sudden onset” (Mitchell, 1932, p.547). The ability to diagnose lead poisoning improved in the 1930’s with the advent of new x-ray technology, which could reveal lead lines in the growth plate of children’s long bones (Rabin, 1989). Using this method Cushing (1933) identified 14 more lead poisoned children admitted to Montreal’s Memorial Hospital (Montreal Medico-Chirurgical Society, 1933; Cushing, 1934). In a 1935 issue of CJPH, Ross and Brown described another 23 cases of lead poisoning in children from Toronto’s Hospital for Sick Children. Collectively, Mitchell (1932), Cushing (1934), Ross and Brown (1935) reported 39 cases of childhood lead poisoning within 3 years. Seven of these cases were fatal (Editorial, 1933; Ross & Brown, 1935). Cases were
attributed to lead-based paint used on furniture, toys and interior woodwork (Cushing, 1934; Ross & Brown, 1935). At least two American authors credit the research of these early Canadian authors with influencing the American public health response to lead (Williams et al., 1952; English, 2001). Paradoxically their influence within Canada appears to have been minimal. Only one Canadian source cites Cushing and Mitchell’s early research (Jephcott, 1937) and to date, apart from this article, only American sources cite Ross and Brown (Williams et al., 1952; Rabin, 1989; English, 2001).

By 1926, fifteen separate U.S. publications described lead based paint as a major source of poisoning, and by the late 1950’s American health authorities had identified over 6000 lead poisoning cases (Jacobs, 1995). In sharp contrast, discourse on lead-based paint appeared only sporadically in the CJPH and the CMAJ over the next 60 years. In 1947, the topic first resurfaced in the CMAJ, but only peripheral to discussion. Penfield and Paine identified lead-based paint as the underlying cause of focal epilepsy in 2 children: “Ingestion of lead-containing paint was followed by coma of two days’ duration with generalized convulsions. Habitual seizures began a few months later” (Penfield & Paine, 1947, p. 523). The same year, Childe (1947) described lead poisoning as a possible cause of bone lesions in infancy and cautioned: “Plumbism is not so uncommon as is sometimes supposed and if kept in mind will be easily diagnosed” (p. 294). A 1952 CMAJ editorial observed that 23 cases of lead poisoning in children were admitted to Toronto’s Hospital for Sick Children (1951-1952) and offered that “[r]epainted furniture causes much lead poisoning as young children nibble the wood through the layers of paint, which contains 7 to 27% of lead” (p.73). A CMAJ editorial appearing just 3 years later, however, inferred the problem was already resolving:
At the Hospital for Sick Children, Toronto, there has been a progressive decline in the number of lead poisoning admissions over the past 15 years. Before 1940 there was an average of 25 to 30 cases admitted each year; this has now decreased to two to four admissions per year. This decline is partly due to the education of parents by public health nurses and others as to the dangers of pica in small children, and partly to the decrease in the use of lead-containing house paints in the home. Legislation in Canada now prohibits the use of lead-containing paints on children's toys and children's furniture (1955, p.611).

Perhaps Toronto children benefited from an education campaign; but lead-based paint cannot be corrected only through better hygiene practices (Brown et al., 2006; Lanphear et al., 1998; Wilson et al., 2006). And contrary to what the editorial expressed, Canada did not restrict the amount of lead added to interior paints until 1976 (Surface Coating Material Regulations, 2003). Even then the restriction permitted a lead concentration of 5000 parts per million (ppm), an amount 8 times higher than the U.S. restriction of 600 ppm established in 1978 (Consumer Products Safety Commission, 1977). Figure 1 illustrates the appearance of Canadian against American lead-paint regulations.

Until 1990 Canadian paint manufacturers added lead to both interior and exterior paint sold in Canada (Surface Coating Material Regulations, 2003) although less toxic alternatives such as zinc sulphate and lithopone were also available (Canadian Paint and Coatings Association, 1991). Prior to 1976 some paints manufactured in Canada contained as much as 50% lead by weight (500,000 ppm) (Canada Mortgage and Housing Corporation, 1993). The Canadian Paint and Coatings Association agreed in 1990 to a voluntary restriction on lead in paints (Health Canada, 2010). Market surveys of new interior and exterior consumer paints sold in Canada identify that 29% of paints (1988-1999) exceeded the U.S. standard of 600 ppm. From 2005-2006, a further 1.4% of paints still had lead concentrations >600 ppm (Surface Coating Material Regulations, 2003). The
voluntary limit did not extend to lead concentrations in exterior paint. A 1991 survey uncovered that 39% of consumer paints marketed for “interior/exterior” use and 8% of paint designated for “exterior” use had been applied to the inside of dwellings (Surface Coating Material Regulations, 2003).

American researchers estimate that 38 million (32%) American dwellings contain lead-based paint somewhere in the home. Of these, 24 million (20.7%) contain significant lead-based paint hazards. A further 3% of newer dwellings, those constructed between 1978/1998 also contain significant hazards (Jacobs et al., 2002). Hypothetically speaking Canada has a similar problem with historic sources of lead-based paint in residential housing as the U.S. The difficulty with estimating the extent of the lead-based paint problem in Canadian dwellings is that there is no clear definition of the construction era that poses the greatest risk. An information booklet published by Health Canada and CMHC advises consumers that many Canadian homes built prior to 1960 do contain lead paint (Health Canada, 1978) although this date is not supported by any systematic survey of Canadian dwellings as occurred in the U.S. (OPHA, 2004).

American health authorities identify housing constructed prior to 1978 as the putative “risk” era (Jones et al., 2009). Using 1960, as the ‘Canadian’ era of concern identifies that 27% of housing stock is potentially at risk, although some argue that a cut-off date of 1990 is justified based on Canada’s late response in regulating lead in consumer paint (McElgunn, 2004). Using 1990 as the era of concern, then, identifies 78% of dwellings potentially at risk. Using 1980 as the era of concern brings the Canadian problem closer to American estimates. Figure 2 illustrates these various “risk” scenarios. A recent survey identifies that Canadian dwellings built prior to 1983 are more likely than later built homes, to contain lead
dust on floors at levels associated with elevated PbB levels in children (≥ 10 μg/dL) (McDonald et al., 2010).

In 1990 Tenenbein described two cases of lead poisoning occurring in Winnipeg children: a 32 month old girl with a “17-month history of a voracious appetite for the paint peeling from the walls and window sills of her inner city home” and a 26-month-old boy, with persistent vomiting, eventually diagnosed with lead encephalopathy (p.40). Paint sampled from both homes revealed lead concentrations ranging from 25,000 to 122,000 ppm (Tenenbein, 1990). Tenenbein (1990) considered that other children in Canada might similarly be affected, although his struggle to locate supporting survey data is clearly evident:

The prevalence of neurotoxic lead levels in asymptomatic children is unknown in Canada. Sound epidemiologic studies of blood lead concentrations involving children living in Canadian inner city areas are needed to define the extent of lead poisoning from lead-based paint (p.41).

Tenenbein’s two cases establish that lead-based paint continued to harm Canadian children well after the 1955 editorial predicted the lead-problem was on its way out.

Construction of the lead as an American problem (circa 1980)

Boothe and Harrison (2009) suggest that the relative indifference paid to lead in Canada stems from an historic lack of national blood monitoring data. We expand on this thesis and suggest that while national surveillance data was sparse, there was ample evidence from case studies, community surveys and environmental investigations to support the position that lead-poisoning constituted a major health threat facing children in Canada. Rather than a misunderstanding framed by a lack of knowledge, we suggest that Canada’s lead problem was advantageously constructed as an American problem. We argue that prior to 1988,
a lack of publicly accessible surveillance data supported the view that lead was an American problem and privileged access to surveillance data made it difficult to counter this view. Thus, the few cases studies reported in the literature could be dismissed as anomalies. The pre and post leaded gasoline era marks an important transition point in Canadian public health discourse and makes a good starting point in which to examine this thesis.

Discourse is always shaped in relation to other discourses. Canadian discourse on lead is “shaped” in relation to the American experience. In 1976, the National Health and Nutrition Examination Survey (NHANES) began gathering national blood lead monitoring data for the U.S. population and identified that 12.2% of black children and 2% of white children had PbB ≥ 30 µg/dL (Mahaffey et al., 1982). In Canada, during the 1970’s, blood lead screening programs were occurring in four cities (Environment Canada, 1984). A 1984 report (Table 1) identified that in 1973 4.8% of Montreal children (0-6 years) had PbB levels ≥ 40 µg/dL, and 5.1% of Halifax children (0-5 years) had PbB levels ≥ 30 µg/dL. Clearly, evidence supported the view that lead poisoning was a serious problem in Canada. By 1980, however, the first of many discourses began surfacing which characterized lead poisoning as an American problem. A 1980 letter to the CMAJ, by C.J. Mackenzie, Head of the Department of Epidemiology, University of British Columbia provides an example of how discourse contrasting sparse Canadian data against the comparatively well-defined American experience was used to downplay concern:

Lead poisoning due to ingestion of lead-based paint reached epidemic proportions in older housing areas in the United States...There is no evidence of a serious health threat to Canadian children from environmental lead exposure. However, epidemiologic data confirming this assumption are scanty and largely limited to hospital records (p.1347-1348).
A 1986 report by the Royal Society of Canada (RCS) employed a similar comparison:

In striking contrast with U.S. experience, the Commission received no hard evidence that paint was a significant source of lead uptake. Indoor flaking paint has been seen as a main cause of high blood levels in U.S. inner cities (and some isolated rural cases). No evidence of such effects exists for Canada, though several briefs to the Commission have suggested that an impact must exist, especially in the older cities (p.265-266).

The Ontario government had been routinely monitoring children’s PbB levels since the 1970’s (Wang et al., 1989) although results were not made public until 1988, and then only for the years 1984-1987, which made claims such as the above difficult to refute. In 1988, when findings from the Ontario 1984-1987 surveys were published (O’Heany et al., 1988), the view that lead poisoning was only an American problem could no longer be supported. In 1984, 5.6% of urban Ontario children ages 1-6 years had PbB levels ≥ 20 μg/dL; 64% had PbB levels ≥ 10 μg/dL (O’Heany et al., 1988). These rates compared well to U.S. figures for the same period. In 1984, 5.2% of American children ages 6 months to 5 years had PbB levels ≥ 20 μg/dL; 17% had PbB levels ≥ 15 μg/dL (U.S. Department of Housing and Urban Development, 1990).

**Reconstructing the problem in the post-leaded gasoline era**

Since lead has been eliminated from gasoline and in the absence of clinical and laboratory evidence to the contrary, we believe that no serious threat of lead exposure to children exists in Canada (Godolphin et al., 1993a, p. 518-519).

Leaded gasoline was officially banned in Canada December 1990 (Health and Welfare Canada, 1992). The removal of a major lead source and the subsequent decline in children’s blood lead levels sparked the emergence of new
discourse on lead. As the epigraph suggests, in the *post-leaded gasoline era*, childhood lead poisoning continued to be viewed as a problem of the past, or a problem of the U.S. - not a problem in Canada. Contradicting this view, however, was the residual 4% of urban Ontario children (1990-1992) identified with PbB levels ≥ 10μg/dL (Fleming & Ursitti, 1994). Clearly the problem had not been entirely eliminated. Smith, a senior consultant with the Ontario Public Health Branch involved with the Ontario surveys suggested, in a 1993 commentary, that exposure to “paint chips, paint dust during renovations, accumulated leaded dust in old houses” and other sources accounted for the remaining portion of affected children, and “parents should be warned” (Smith, 1993, p.214). Two years later, however, this cautionary message had changed:

The Centers for Disease Control and Prevention has described lead poisoning as one of the most common and preventable pediatric health problems in the United States. In Canada, however, blood lead levels have been consistently lower than in the United States: provincial and community surveys show blood lead levels in Canadian children have declined markedly over the past decade as lead has been phased out of gasoline (Smith & Rea, 1995, p.373).

U.S. health authorities had access to data from a nationally representative sample from which to calculate a national prevalence rate of childhood lead poisoning - the proportion of children ages 1-5 years with PbB ≥ 10μg/dL (Pirkle et al., 1994); whereas, at the time of Smith and Rea’s article (1995), national prevalence rates for Canadian children in the *post-leaded-gasoline era* were not available.

Canada’s first nation-wide blood lead screening investigation (Statistics Canada & Health and Welfare Canada, 1981) occurred between 1978/79 - a peak period for leaded gasoline use in Canada (Nriagu, 1998). Unleaded gasoline was introduced in 1975 but sales of leaded fuel did not start to drop until the mid-1980’s (Wang et al., 1997). The 1978/79 survey (Table 2) identified that 100% of males
and 96% of females ages 3 to 5 years in Canada had PbB levels sampling below 10 g/dL (Statistics Canada & Health and Welfare Canada, 1981) - a peculiar finding given the era in which the survey took place. Environment Canada charged that the blood lead results were not "log normal" as one would expect as 27% of observations were ≤ 1 μg/dL, "an unusual characteristic which cast doubt on the validity of the results" (Environment Canada, 1984, p.16). Health Canada’s own “Lead” Working Group advised the use of “caution” when interpreting the results (Health Canada, 1994, p. 22).

The absence of national data permitted the insertion of Ontario’s blood lead surveys as a surrogate indicator for Canada’s children. In 1994 the “Lead Working Group” used a patchwork of community investigations to estimate a national rate for Canada:

In assessing the present status of blood lead levels in the Canadian population overall, it is necessary to rely on only a few substantial studies, mainly from Ontario, with some uncertainty existing as to just how representative these are of the situation across Canada (Health Canada, 1994, p. ii).

The Working Group estimated that 5 to 10% of urban Canadian children had PbB levels exceeding 10 μg/dL (Health Canada, 1994, p. ii). Smith and Rea (1995) used the prevalence rate for children ages 1-5 years living in an isolated area of Northern Ontario for this same purpose:

Based on this survey, blood lead levels in Canadian children can be as low as about 3 μg/dL (geometric mean), with a 4% prevalence of levels over 10 μg/dL and no levels greater than 20μg/dL. (Smith & Rea, 1995, p.375)

Smith and Rea further compared findings from their 1995 survey with U.S. survey data from 1984:

The relatively low lead levels seen in this survey and others in Canada contrast with the situation reported from the United States
where an estimated 17% of preschool children had blood lead levels over 15 µg/dL in 1984 (Smith & Rea, 1995, p.375).

Jin and colleagues (1995) used a similar comparison to downplay findings from their 1995 Vancouver investigation which identified 8.1% of children (2-3 years) with PbB ≥10µg/dL:

The blood lead levels we found were much lower than those found in the Canada Health Survey, in the Ontario survey and in the United States National Health and Nutrition Examination Survey (1976-80) (Jin et al., 1995, p.1084).

Jin and colleagues’ discourse ignored more recent prevalence data from NHANES (1988–1991) which identified 8.6% of American children (1 - 5 years) with PbB levels ≥ 10 µg/dL (Brody et al., 1994), a rate much closer to the Vancouver study.

Average blood lead levels of the U.S. population began declining in 1977 (Annest et al., 1983). Average blood lead levels of Ontario children did not start to fall until 1984 (Wang et al., 1997). Table 3 summarizes available prevalence data for Canadian communities during the post-leded gasoline era (1990 to 2008). Prevalence rates ranged from 1.3% to 16.9%. Although limited, evidence supported the view that Canadian children continued to be affected by lead, even as average blood lead levels declined (Alder et al., 1993; Balram & Giffin, 1993 Fleming & Ursitti, 1994; Jin et al., 1995; Levallois et al., 1991; Leung et al., 1993; Smith, 1993; Smith & Rea, 1995; Scott, 1995). Sparse national surveillance data, however, authorized the insertion of Ontario survey data as the proxy indicator for Canadian trends. Contrasting ‘Canadian’ survey data, selectively, with dated American surveys sustained the view that the U.S. lead-problem was indeed greater than Canada’s. Further, privileged access to Ontario survey data made it difficult to challenge this view.
We use the term “privileged” to describe a type of discourse wherein authors identify, for example, prevalence rates, yet do not supply a supporting reference in which to trace the source of information. The omission of relevant reference material is particularly evident in government discourse (see for example, Smith & Rea, 1995; Smith, 1993; Wang et al., 1997; Wang et al., 1989). In two cases editorials referencing, un-cited, statistical data were also published anonymously (CMAJ editorials, 1952, 1955). This discursive technique implied that authors had special access to non-public data but also rendered it difficult for the reader to track and validate the information.

In the post-leaded gasoline era a second contingency emerged, one which supported the view that Canada’s lead-problem, if it existed, was mainly confined to children living in smelter communities. A review of lead’s “history” in the archives of the Canadian Journal of Public Health (CJPH), for example, reveals a parallel but equally influential discourse. Between 1935 and 2008 the CJPH published a total of fourteen articles on the topic of lead poisoning: a discussion of screening methods (Peter & Bourdeau, 1983); a case description of occupational exposure (Nosal & Wilhelm, 1990); four commentaries (Kosatsky, 1992; Lees & Langlois, 1994; Levallois et al., 1994; Rice, 1998); six field investigations of lead exposure pertaining to point source communities (Hertzman et al., 1991; Létourneau G. & Gagné, 1992; Kosatsky & Boivin, 1994; Kelly et al., 1994; Gagné, 1994; Hilts et al., 1995); and, two articles related to investigations of children living in non-industrial settings: Ross and Brown’s early commentary (1935) and Smith and Rea’s 1995 survey entitled, “Low blood lead levels in Northern Ontario – what now?” We suggest that the over-representation of smelter community investigations in one of Canada’s high profile public health journals supported the view, espoused by various authors (Daws et al., 1993; Dimock & Santori, 1993; Godolphin et al.,
The Medicine Hat Cases and media influence

By 1990, a growing body of international research, including Canadian research (Rice & Gilbert, 1985), demonstrated that lead was harmful to children’s developing brain and nervous system at blood lead levels ≤10 μg/dL. In 1991, in response to this evidence, the U.S. Centers for Disease Control and Prevention (CDC) lowered their PbB intervention level for children from 25 to 10 g/dL (CDC, 1991). Canada followed suit in 1994 by adopting the same standard (Health Canada, 1994).

Following the CDC’s revision (1991), U.S. media reports began characterizing lead poisoning as an endemic problem rather than an isolated concern (Shibley & Prosterman, 1998). A 1991 edition of Newsweek featured the stories of children from economically advantaged families who had been poisoned - not from eating paint chips, but from ingesting fine particles of dust (Waldman, 1991). The burning, sanding and scraping of lead paint produced fine lead particles that clung to surfaces, including children’s fingers. Symptoms of lead poisoning that were commonly described were increased irritability and stomach complaints – symptoms resembling the flu, not symptoms one typically associated with “poisoning” (Waldman, 1991).

The Newsweek story flowed into Canada and triggered the realization by a family from Medicine Hat, Alberta, that their children, too, might be lead poisoned. The family, given a copy of the article, recognized the same symptoms of irritability and stomachaches in their own two children (Canadian Press, 1991). The Alberta
couple had also been renovating their century old home, stripping paint off exterior walls and sanding storm windows. Their one year old son’s PbB was 30 μg/dL. Their three year old daughter’s level was 22.9 μg/dL (Canadian Press, 1991). Interior wall coatings contained lead concentrations as high as 140,000 ppm. Exterior wall paint had lead concentrations as high as 120,000 ppm. New exterior paint applied over old coatings contained 39,000 ppm (CMHC, 1993a).

The Medicine Hat case garnered national media attention in Canada. A 1991 broadcast of Marketplace featured an interview with the family as well as experts from Canada and the U.S. Host Bill Paul invited Roger Walker, the Senior Project Officer with Consumer and Corporate Affairs Canada, to explain Canada’s laggard response to lead paint in contrast with vigorous American initiatives. He responded that:

At this time we had no indication that the problem that the Americans were finding were indeed a problem in Canada. And the age of the housing stock in the United States is in many cities very much significantly older than Canada. There’s a significantly higher percentage of Americans that are housed in what are referred to as lower income urban areas with often, in the United States, deteriorating conditions (CBC, 1991).

Thomas Spitler, a Senior Researcher with the U.S. Environmental Protection Agency, also a guest, was asked for his opinion. Research in the U.S. had accumulated extensive evidence establishing an association between lead-based paint (and dust) exposure and elevated blood lead levels in children (U.S. Department of Housing and Urban Development, 1990). U.S. laws to eradicate domestic sources of lead-based paint were first established in 1970 (Richardson, 2005), although American advocates too, had encountered their own set of obstacles delaying action (Needleman, 1991; Richardson, 2005). Spitler responded:
I’m afraid that’s about a 20 or 30 year old myth that lead paint is an inner city problem. Lead poisoning is a problem anywhere where you have a disturbance of high levels of lead paint and children exposed to the dust that results from that disturbance or renovation (CBC, 1991).

Marketplace estimated that millions of homes in Canada contained lead-based paint, but most Canadians were unaware of the problem. Some cities however were aware of the issue. For example, in St. John, New Brunswick, Canada’s oldest city, a 1991 investigation identified that 53% of the 91 adults and children involved in a preliminary screening had PbB levels $\geq 10 \mu g/dL$ (Scott, 1995). A follow-up investigation identified 11.3% of children ages 1-3 years living in St. John with PbB levels $\geq 10 \mu g/dL$ (Balram & Giffin, 1993a). The highest blood lead concentrations were found in families living in the city’s older housing districts (Balram & Giffin, 1993a). Richard Scott, a toxicologist involved with the study, and also a guest on Marketplace, offered his explanation for Canada’s lethargy:

The level of awareness in the Canadian population would probably be close to zero. The reason being we have a perception, a generally held perception that we have legislated lead out of gasoline, out of paints, out of solder and so because of that, people will imagine that lead is no longer a problem. What they forget however, is that we’re dealing with a legacy of the use in the past (CBC, 1991).

In the end, the Medicine Hat family, overwhelmed by the projected cost to make their home “lead-safe” opted to abandon their property (CBC, 1991). The home was signed over to the bank’s mortgage insurer, Canada Mortgage and Housing Corporation (CMHC).

CMHC has two basic functions, to administer the National Housing Act (1985) and to provide discounting facilities for loan and mortgage companies. After the Marketplace story aired, the program received over 5000 requests for its
“lead” fact sheet. A repeat broadcast ran the following year. Burgeoning interest in lead must have worried Canadian authorities. The CMHC had a number of housing units funded through its agency. How many of these contained lead-based paint? What if another family were poisoned? What if more Canadians started abandoning their homes? A “Lead Swat Team” was formed in 1992 consisting of representatives from four federal agencies: Health and Welfare Canada, CMHC, Consumer and Corporate Affairs Canada and Environment Canada (Health and Welfare Canada et al., 1992). Following the Medicine Hat case, lead-based paint started getting political attention.

Canada’s unfinished business with lead-based paint

Harrison and Hoberg (1991) identify three steps involved in regulating a health issue: Information on the problem is collected; decisions about appropriate actions (including inaction) are made; and agency decisions are implemented. We suggest that tensions between federal agencies evolved into a power struggle with Health Canada ultimately emerging as the dominant organisation, thereby diminishing the CMHC’s role and authority but also preventing important research initiatives from being implemented.

Lead poisoning is a multi-faceted problem which crosses the boundaries of public health, housing and environment. The Public Health Agency of Canada Act (2006) authorizes Health Canada to safeguard the health of Canadians (Krewski et al., 2006). The Department of Health Act (1996) equally obliges Health Canada to protect and monitor the physical, mental and social well-being of Canadians. Lead itself is recognized as a priority substance under Schedule 1, Toxic Substances of the Canadian Environmental Protection Act (1999) which further requires Health Canada to manage health risks related to lead (Tyshenko et al., 2007). As
mentioned, responsibility for the investigation of housing falls within the mandate of Canada Mortgage and Housing Corporation under the *National Housing Act* (1985).

In 1992, U.S. Congress passed the *Residential Lead Based Paint Hazard Reduction Act, or Title X*, which authorized the development of a national strategy to eliminate lead based paint in all residential housing. Responsibility for implementation of the act was divided among three federal agencies: the U.S. Environmental Protection Agency (EPA), the Centers for Disease Control and Prevention (CDC) and the Department of Housing and Urban Development (HUD). HUD was designated as the lead agency (Richardson, 2005). By 1992 HUD had examined the various methods, costs and dangers involved in lead-based paint abatement - the primary strategy for the elimination of lead poisoning in American children (U.S. Department of Housing and Urban Development, 1990). A Baltimore study estimated that measures ranging from repainting and cleaning to full abatement cost between $1,650 and $6,000 US (Farfel & Lim, 1995). The only abatement study occurring in Canada had taken place in a secondary smelter community (Boehnke, 1999).

From 1991 to 1995, CMHC undertook various research initiatives related to lead-based paint: an abatement study of the Medicine Hat dwelling (CMHC, 1993a); an investigation of best practices for clean-up following renovation (CMHC, 1995a); development of a national training program geared to professionals involved in abatement, renovation and repair activities (CMHC, 1993b); publication of a series of technical reports related to lead-based paint, (CMHC, 1992b; 1993c; 1994); and, compilation of a resource compendium listing Canadian research initiatives and federal/ provincial guidelines pertaining to lead (CMHC, 1992a).
CMHC also co-directed an environmental investigation of the St. John, New Brunswick dwellings that were associated with elevated blood lead levels in adults and children (CMHC, 1995b).

The problem of lead-based paint was close to being regulated. Information on the problem was collected and decisions for appropriate actions were made. But lead-based paint was a housing problem, a medical problem and an environmental problem. Whose jurisdiction was it? Canada did not have a similar law to the U.S. *Title X* (1992) which mandated shared responsibility for lead among federal authorities. CMHC, on its own, lacked the necessary authority to move its action plan into the crucial and final phase, implementation. In 1994 a ‘lead’ Working Group was appointed by Health Canada to review evidence supporting a lowering of Canada’s PbB standard from 25 to 10 g/dL. As discussed, a general lack of surveillance data frustrated the group’s ability to make an informed appraisal for which they compensated by piecing together limited survey data to estimate a national prevalence rate (Health Canada, 1994). Table 4 summarizes various recommendations made by Canadian agents, including the Working Group (Health Canada, 1994) to address Canada’s monitoring issues. Table 5 summarizes the federal response thus far. To date, only one of the Working Group’s recommendations has been implemented: the routine screening of children living in smelter communities.

Early CMHC research activities demonstrated an appropriate level of concern by recognizing the importance of lead-based paint as a serious health issue; and yet, none of CMHC’s research activities resulted in policy change. The proposed national lead-abatement training program never came to fruition and no further research reports on lead-based paint were produced by CMHC after 1995.
Following a brief surge of interest, lead-based paint disappeared from the federal agenda. Currently there are no regulations at the federal level to protect Canadian children from exposure to historic sources of lead paint (Spady, 2006). Health Canada’s response to remediation of this persistent source has been described as a series of risk communiqués (Krewski et al., 2006).

Boothe and Harrison (2009) suggest that U.S. policy is further ahead than Canada in all aspects pertaining to children’s environmental health due to legislative differences which advantage American advocates:

[U.S.] Congress tends to write detailed statutes in an attempt to ensure faithful execution of its will. In Canada, the fusion of executive and legislative functions in parliamentary government means that Cabinet both drafts laws and implements them. Not surprisingly, parliamentary governments typically produce laws that grant considerable discretion to the executive. This contrast shapes interest group strategies in two ways. With the advantage of environmental statutes that not only establish explicit mandates for the executive, but back them up with citizen suit provisions, U.S. interest groups contest virtually every major regulatory decision in court (p. 293).

This argument has merit. In Canada, federal regulations limiting lead content in consumer products were justified on the basis of two case reports occurring in 1998. Neither case was fatal (Health Canada, 2005a). In 2004 Lavoie and Bailey reported the case of a 4 year old Montreal child who was “eating paint stripped from the walls of [his] new home” (Lavoie & Bailey, 2004, p.956). A PbB level of 98 µg/dL confirmed acute lead poisoning. A follow-up visit to the boy’s home by health authorities identified “lead-free” paint (< 0.5% dry weight or 5000 ppm) scraped off trim as the source of exposure (Lavoie & Bailey, 2004, p.956). Canadian regulations limiting lead-concentrations in new consumer paints came into effect in 2005, 17 years after U.S. regulations, and coincidentally, close on the heels of the Montreal case.
What meager concern regarding lead-based paint that had started to grow in the early 1990’s was diverted by the debate which followed the CDC’s 1991 decision to implement universal blood lead screening (CDC, 1991). Canadian discourse favored targeted screening of high risk groups (Cole & Rosen, 1993; Daws et al., 1993; Dimock & Santori, 1993; Godolphin et al., 1993a, 1993b; Levallois et al., 1994; Lockitch, 1993). Others proffered case finding as a solution (Balram & Giffin, 1993a; Levallois et al., 1994; Health Canada, 1994; Walters, 1993) a strategy U.S. health authorities had long ago rejected. Lead-poisoning was a “silent disease.” There were no clinical symptoms to identify children at the new, lower blood lead level of concern (CDC, 1991; Needleman, 1990). Without relevant blood lead surveillance data to justify either approach, however, debate subsided, as did interest. In 2002 Sanborn observed that Canada had adopted neither targeted nor universal blood lead screening. Today Québec is the only province requiring mandatory reporting of PbB ≥ 10μg/dL identified through case finding (Plante et al., 2003).

New federal initiatives related to investigations of human lead exposure in Canada

Following a brief but inconsequential hiatus the topic of lead poisoning appears to be the subject of renewed interest in Canada. Beginning in 2005 Health Canada produced a series of reports describing the health implications for children and adults occurring from “low level” lead exposure (Health Canada, 2005b; Health Canada, 2008; Wilson et al., 2005). In a previous review we submit that these reports, while useful, add to the already large body of knowledge describing the physiological effects of lead exposure. What is missing is Canadian context specific information describing the extent of the lead-problem (Perron & O’Grady,
2010). How do people know, for example, if childhood lead poisoning is even a problem in Canada? Although our analysis originally intended to focus on discourse occurring between 1932 and 2008 we cannot ignore new discourse emerging from three recent federal initiatives, all of which appear ready to answer this question.

In August 2010, findings from the *Canadian Health Measures Survey* (CHMS), a national investigation which measured concentrations of lead and other contaminants in blood and urine, indicate that mean blood lead levels of Canadians, aged 6 to 79 years, have declined considerably in the thirty year span since the last nation-wide survey of blood lead levels occurred in Canada in 1978 (Statistics Canada, 2008). The decline is attributed to the phase-out of leaded gasoline, lead-containing paints and lead solder in food cans since the 1970s (Statistics Canada, 2008). The CHMS reports that fewer than 1% of Canadians have blood lead concentrations above the Health Canada guidance value of 10\(\mu\)g/dL (Statistics Canada, 2008). What should be noted, however, is that children less than 6 years of age were excluded from this study. Children’s blood lead levels peak between 18 and 36 months and decline thereafter; therefore a blood lead level of ‘10’ is more readily found in children 1 to 5 years than children in the older age-range that was tested (Dixon et al., 2009; Jones et al., 2009; Meyer et al., 2003). In our view, the exclusion of a highly vulnerable group represents a serious flaw in this survey’s design and challenges the legitimacy of using this survey to describe a national prevalence rate for lead poisoning in Canada.

Complementing this survey is the *Maternal Infant Research on Environmental Chemicals* (MIREC) which is tracking approximately 2000 women from the first trimester of pregnancy until the 8 weeks following birth. Lead is one of
the five metals being tested. Results will be available by 2012 (Health Canada, 2007a). Again, children ages 1 to 5 years are excluded from this investigation.

Of special interest will be findings from a third national survey, the Canadian House Dust Study, Canada’s first large scale investigation of lead dust levels found normally on floors of residential non-industrial dwellings. Preliminary findings already yield important information regarding the construction era that poses the greatest risk to children from lead contaminated dust (McDonald et al., 2010). Full results were expected to be publicly available by the end of 2010. The final results have not yet been released. Further discussion of public health knowledge produced from these national reports is briefly discussed in the final chapter.

**Final remarks**

Early discourse from the 1930’s identified lead-based paint as a serious concern for Canadian children, mirroring similar concern in the U.S. And yet, the public health response to lead in Canada followed a different trajectory than occurred in the U.S. The topic of lead-based paint had barely taken root in Canada, when in 1950 lead poisoning was already being constructed as a declining concern. By 1980 the issue had almost completely transformed into an American problem. The emergence, in 1988, of publicly accessible surveillance data momentarily prevented Canada’s lead problem from being cast exclusively as an American problem. For a short time, childhood lead poisoning was given audience in Canada. Attention waned as average blood lead levels of Ontario children declined. National media coverage of the Medicine Hat cases, in 1991, rekindled public concern by drawing attention to the lead-based paint problem that was potentially affecting a large portion of Canada’s housing stock. For a while
federal agencies in Canada seemed sincere in their efforts to eradicate this remaining and persistent source. And yet, as average blood lead levels declined further, concern too shifted. By 1995, tensions among federal agencies stalled all activity on lead-based paint. To date, there are no federal initiatives protecting Canadian children living in lead contaminated dwellings. Discourse emerging from Canada’s latest national surveys will likely further thwart public concern.

We have argued that privileged access to surveillance data, more so than sparse surveillance data allowed the construction of childhood lead poisoning as an American problem, and was the major contingency shaping the Canadian public health response to lead. There are unexplained ‘holes’ in this thesis. We cannot explain, for example, why some public health authorities in Canada chose to diffuse concern, or why Quebec public health discourse varies from discourse emerging from other provinces, and consequently produced a distinctly different response. A further limitation of this study is its lack of discussion pertaining to the federal-provincial political relationship underlying the Canadian healthcare system. Federalism (shared power between the federal government and the provinces) is viewed by Maioni (2002) as one of the most important contingencies shaping Canadian health care policy. These are matters for future research. This much is known. Lead based interior and exterior paint was manufactured and sold in Canada until at least 1990, although legislation fully banning lead additives in paints did not come into effect in Canada until 2005. Lead can still be legally added to exterior paints if the product carries the warning: “Danger contains lead. Do not apply to surfaces accessible to children or pregnant women” (Health Canada, 2007b). Weaker Canadian regulations, a general lack of public awareness and a dampening of public concern by agencies perceived with authority on this issue
support the contention that lead-based paint is potentially a *greater* problem in Canada than in the U.S.

We have offered a “counter reading” of lead’s history by examining the contingencies which shaped the Canadian public health response to lead-based paint. Through this analysis we hope to bring Canada’s attention back to this serious but preventable health concern.
References


Ref Type: Journal (Full)


ou d'une intoxication et d'une exposition significative: Le plomb
Institut national de santé publique du Québec.


Table 1. Blood lead surveillance data for children living in 4 Canadian communities 1973-1977

<table>
<thead>
<tr>
<th>Location; Date</th>
<th>Population Studied</th>
<th>Mean</th>
<th>% ≥20μg/dL</th>
<th>% ≥30μg/dL</th>
<th>% ≥40μg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Montreal</td>
<td>712 children (0-6 yr) attending the Pediatric Outpatients Dept. of Montreal Children’s Hospital</td>
<td>4.8%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Halifax</td>
<td>184 children (0-5 yr)</td>
<td>4%</td>
<td>1.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nelson, B.C</td>
<td>55 children (1-3 yr)</td>
<td>14</td>
<td>0</td>
<td>2.1%</td>
<td></td>
</tr>
<tr>
<td>May 1975</td>
<td>103 grade 9 children</td>
<td>10</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kamloops, B.C.</td>
<td>109 children 2-3 yr</td>
<td>2.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May-Nov, 1977</td>
<td>240 children 4-5 yr</td>
<td>4.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>283 children 9 yr</td>
<td>4.4</td>
<td>0.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Prevalence rates for Canadian males and females ages 3-5 years with blood lead levels \( \geq 10 \, \mu g/dL \). Adapted from *Canada Health Survey*: Health and Welfare Canada, Statistics Canada (1981) [Table 83, p.157]

<table>
<thead>
<tr>
<th>Blood lead levels ( \mu g/dL )</th>
<th>Total</th>
<th>&lt; 10</th>
<th>10 to 19</th>
<th>20 to 39</th>
<th>( \geq 40 )</th>
<th>Results designated as “Unknown”</th>
<th>Total % accounted for (%)</th>
<th>Total data unaccounted for (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 to 5</td>
<td>No.</td>
<td>521</td>
<td>212</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>100</td>
<td>40.8</td>
<td>--</td>
<td>--</td>
<td>40.8</td>
<td>38.4</td>
<td>79.2</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 to 5</td>
<td>No.</td>
<td>474</td>
<td>332</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>123</td>
<td></td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>100</td>
<td>70</td>
<td>4</td>
<td>-</td>
<td>70</td>
<td>26</td>
<td>100</td>
</tr>
</tbody>
</table>

Key
(Health & Welfare Canada, 1981)
- means nil or zero
-- too small to be expressed

<table>
<thead>
<tr>
<th>Location/ Date</th>
<th>Sample size/age in years</th>
<th>% ≥10μg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quebec, QC</td>
<td>93 urban children (1 to 6 y)</td>
<td>10.8</td>
</tr>
<tr>
<td>(Levallois et al., 1991)</td>
<td>149 rural children</td>
<td>1.3</td>
</tr>
<tr>
<td>London, ON</td>
<td>164 (0-17 y)</td>
<td>7.3</td>
</tr>
<tr>
<td>(Alder et al., 1993)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>London, ON</td>
<td>726 children (0-16 y)</td>
<td>16.8</td>
</tr>
<tr>
<td>(Leung et al., 1993)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>St. John, NB</td>
<td>97 children (1-3 y)</td>
<td>11.3</td>
</tr>
<tr>
<td>(Balram &amp; Giffin, 1993b)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vancouver, BC</td>
<td>172 children (1-3 y)</td>
<td>8.1</td>
</tr>
<tr>
<td>(Jin et al., 1995)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northern Ontario</td>
<td>395 children (1-6 y)</td>
<td>4</td>
</tr>
<tr>
<td>(Smith &amp; Rea, 1995)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 4. Summary of recommendations made to address childhood lead poisoning in Canada

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Author/Year</th>
<th>Outcome - Decision implemented?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interventions and evaluation</td>
<td>(Balram &amp; Giffin, 1993)</td>
<td>none</td>
</tr>
<tr>
<td>Further research into sources of lead</td>
<td>(Health Canada, 1994) (Tsekrekos &amp; Buka, 2005)</td>
<td>Market surveys of jewellery/consumer products, food by Health Canada</td>
</tr>
<tr>
<td>Blood lead proficiency testing</td>
<td>(Locklitch, 1993)</td>
<td>Proficiency testing is not mandatory for laboratories in Canada although some agencies subscribe to a program voluntarily. none</td>
</tr>
<tr>
<td>Awareness training for physicians and other health care providers</td>
<td>(Health Canada, 1994) (Balram &amp; Giffin, 1993)</td>
<td>none</td>
</tr>
<tr>
<td>Training of contractors, trades people who renovate older homes</td>
<td>(Balram &amp; Giffin, 1993)</td>
<td>none</td>
</tr>
<tr>
<td>Cost effective measures to reduce lead exposure</td>
<td>(Health Canada, 1994) (Walters, 1993) (Tsekrekos &amp; Buka, 2005)</td>
<td>none</td>
</tr>
<tr>
<td>Federal funding to assist/establish remediation programs</td>
<td>(Balram &amp; Giffin, 1993)</td>
<td>none</td>
</tr>
</tbody>
</table>
Table 5. Federal Canadian policy and legislation regarding protection of human health from lead

<table>
<thead>
<tr>
<th>Agencies with legislated authority over public health or housing</th>
<th>Governance of toxic substances such as Lead</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health Canada</td>
<td>Canada Mortgage and Housing Corp</td>
</tr>
<tr>
<td>Public Health Agency of Canada Act, 2006</td>
<td>National Housing Act (1938)</td>
</tr>
<tr>
<td>Health Canada Activities</td>
<td>Canadian Environmental Protection Act (CEPA, 1999)</td>
</tr>
<tr>
<td>National Lead Strategy</td>
<td>Hazardous Products Act and Regulations</td>
</tr>
<tr>
<td>Addresses lead in consumer products only</td>
<td></td>
</tr>
<tr>
<td>Risk communication materials</td>
<td></td>
</tr>
<tr>
<td>Webpages, booklets</td>
<td></td>
</tr>
<tr>
<td>CMHC Activities</td>
<td></td>
</tr>
<tr>
<td>Various research reports on lead abatement and training</td>
<td></td>
</tr>
<tr>
<td>Canadian Environmental Protection Act (CEPA, 1999)</td>
<td></td>
</tr>
<tr>
<td>Health Canada Activities</td>
<td></td>
</tr>
<tr>
<td>National Lead Strategy</td>
<td></td>
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<td>Addresses lead in consumer products only</td>
<td></td>
</tr>
<tr>
<td>Risk communication materials</td>
<td></td>
</tr>
<tr>
<td>Webpages, booklets</td>
<td></td>
</tr>
<tr>
<td>Federal Guidelines - Environmental</td>
<td></td>
</tr>
<tr>
<td>Lead in drinking water</td>
<td></td>
</tr>
<tr>
<td>[Federal-Provincial-Territorial Committee on Drinking Water]</td>
<td></td>
</tr>
<tr>
<td>Lead in exterior residential soil</td>
<td></td>
</tr>
<tr>
<td>[Canadian Council of Ministers of the Environment]</td>
<td></td>
</tr>
<tr>
<td>Federal Provincial Guidelines – Public Health</td>
<td></td>
</tr>
<tr>
<td>Establishes blood lead “level of concern” of 10 µg/dL (1994)</td>
<td></td>
</tr>
</tbody>
</table>

**Additional Information:**
- **Lead in drinking water**
  - [Federal-Provincial-Territorial Committee on Drinking Water]
- **Federal Guidelines – Public Health**
  - Establishes blood lead “level of concern” of 10 µg/dL (1994)
Figure 1. Timeline comparing lead paint regulations in Canada and the United States
Figure 2. The percentage of Canadian “at risk” dwellings according to date of construction compared with the U.S. rate
PART III: DISCUSSION

Introduction

Foucault uses the term épistèmé to represent what is known about a subject/object during a particular historical time frame or context. He also uses the term to describe how knowledge itself can be used as an apparatus to support and maintain dominant discourses (Foucault, 1972a). This thesis posits that the major contingency separating the American and Canadian public health response to lead/lead-based-paint has been the limited episteme, more specifically, national surveillance data describing the extent of childhood lead poisoning in Canada. The systematic literature review (Article 1) based on Dixon and Dixon’s ecological model (2002) supports the contention that childhood lead poisoning occurring from exposure to residential sources of lead (i.e. lead-based paint) is an under-researched area in Canada. Building on this review, the critical analysis in Article 2 illustrates how this contingency (limited épistèmé) supports the current view in Canada, that childhood lead poisoning is a problem of the past or a problem of the United States. The analysis (Article 2) was conducted using Rawlinson’s (1987) 3-part mode of inquiry, as summarized below and presented in the Methods section (Part I).

Rawlinson’s (1987) adaptation of Foucault’s methods for archival analysis organizes Foucault’s archaeological and genealogical analysis along three axes: The axis of knowledge, investigates assumptions concerning what is considered ‘true’ or ‘false;’ the axis of authority examines systems of power; and the axis of value/justification concerns itself with technologies of power as found in disciplinary measures. Rawlinson’s approach (1987) was relevant for guiding this examination, although it should be understood that these three axes of inquiry should not be
perceived as mutually exclusive but seen as overlapping, allowing a multi-focal examination of systems of knowledge, what makes such systems possible, and their mediated effects. Foucault (1966; 1972) describes the complex interplay between knowledge, discourse and power, and emphasises the difficulty in discussing one ‘theme’ without discussing the others. For example, public health discourse, or any discourse, is always shaped by transecting layers of historical, political and social contingencies. The analysis (Article 2) therefore, could not be neatly compartmentalized along Rawlinson’s three planes of inquiry. With this in mind, discussion in the following section is ‘artificially’ divided into discussions of power, discourse, and knowledge using Rawlinson’s three axes to highlight the intersecting and complex relationships occurring between knowledge and power.

3.1 Rawlinson’s 3 axes of inquiry

The axis of knowledge. Rawlinson’s first line of inquiry involves an examination of the rules governing the formation of statements and the determination of what is considered to be true and false, rules of evidence, rules concerning what can be addressed and what cannot, and rules for the repeatability or use of statements. The analysis sought to identify: What are the dominant public health discourses on childhood lead poisoning in Canada? What are the rules for what can be spoken? What engenders evidence? What determines the difference between true and false; and, who is accorded the power to pronounce truth?

Article 2, began the analysis by describing the early discourse of Mitchell, Cushing, Ross and Brown (Mitchell, 1932; Cushing et al., 1932; Ross & Brown, 1935) and how this discourse influenced the American public health response, yet paradoxically, did not trigger the same vigorous response in Canada as occurred in the United States. As determined by the review of the two Canadian journals in
which these early discourses were originally published (text summarized in Appendix G) further discussion of lead poisoning and lead-based paint disappeared from the public view after 1932. That is to say, discussion on lead in the *Canadian Medical Association Journal* (CMAJ), for example, did not resurface again until 1947, and then, only peripherally, never central to the discussion (See for example, Rabinowitch, 1934; Penfield & Paine, 1947; Childe, 1947; Paton & Cembrowski, 1982). The issue of childhood lead poisoning did not arise in the *Canadian Public Health Journal* (CJPH) as a topic of concern again until 1983 (Peter & Bourdeau, 1983) and then only non-critically in a discussion of screening methods. Discussion of lead-based paint and its potential harm to Canadian children did not reappear in the CJPH again until a 1994 commentary by Levallois, Gaudreault, Rhains and Weber in a discussion weighing the pros and cons of universal screening. Authors are dismissive of Canada’s need for universal screening, yet at the same time acknowledge the lack of research evidence to support their position: “Old lead-based paint seems to be an important cause of the continuous prevalence of lead poisoning in the U.S.’ however, since this situation has not been investigated carefully in Canada, it is possibly underestimated” (Levallois et al., 1994, p. 168). Remarkably, this brief commentary and Ross and Brown’s earlier article (1935) are the only times when lead-based paint, in terms of its impact on children’s health, appears in discussion in Canada’s leading public health journal.

As Powers (1996) notes, every discourse carries an internal system of truth that can be recognized by its rules. Kendall and Wickham describe the effect of such system of rules as “the sayable.” Exploration along this axis led to the identification of various ‘rules’ which made possible a certain kind of discourse about childhood lead poisoning in Canada. These rules are explicated below.
Rule 1. The first ‘rule’ concerns the way the Canadian Journal of Public Health (CJPH) privileged the publication of investigations pertaining to lead poisoning cases occurring in children living in smelter or point-source communities. After Ross and Brown’s paper (1935), the CJPH published only one other investigation of childhood lead poisoning in a non-industrial community setting (Smith & Rea, 1995). The “CJPH” rule enabled the representation of childhood lead poisoning in Canada as a problem concerning smelter communities rather than a problem caused from exposure to residential sources of lead (i.e. lead-based paint). Similarly, the Canadian Medical Association Journal (CMAJ) published only one investigation of childhood lead poisoning in a non-industrial community setting (Jin et al., 1995); although, on the whole, the CMAJ published more articles pertaining to discussions of lead than the CPJH (Appendix G), including two cases studies describing children poisoned from exposure to lead-based paint (Tenenbein, 1990; Lavoie & Bailey, 2004). CMAJ discourse on lead also differed from CJPH discourse in that the CMAJ favoured the genre ‘commentaries’ over systematic investigations. The appearance of various letters/commentaries debating whether Canada should adopt universal versus targeted screening appeared predominantly in the CMAJ, particularly in the 1990’s; but as Article 2 demonstrates, the screening debate faded from discussion in both journals by 1995, so that by 2002, Canada had adopted neither targeted nor universal screening. To date, children living in [Canadian] smelter communities are the only “at risk” population routinely monitored.

Rule 2. The second rule of discourse concerns the way authors deemed to have expert authority on the topic of lead poisoning could cite Canadian blood lead surveillance data without providing the supporting reference that was necessary to validate these statements. The following examples demonstrate this point.
Example 1.

A 1952 CMAJ editorial observed that 23 cases of lead poisoning in children were admitted to Toronto’s Hospital for Sick Children (1951-1952) and offered that “[r]epainted furniture causes much lead poisoning as young children nibble the wood through the layers of paint, which contains 7 to 27% of lead” (1952, p.73).

A later CMAJ editorial published in 1955 observed:

At the Hospital for Sick Children, Toronto, there has been a progressive decline in the number of lead poisoning admissions over the past 15 years. Before 1940 there was an average of 25 to 30 cases admitted each year; this has now decreased to two to four admissions per year. This decline is partly due to the education of parents by public health nurses and others as to the dangers of pica in small children, and partly to the decrease in the use of lead-containing house paints in the home. Legislation in Canada now prohibits the use of lead-containing paints on children's toys and children's furniture (1955, p.611)

Note that both editorials do not include a reference/citation for surveillance figures.

Further, note that both editorials were published anonymously which renders this information difficult to trace or validate.

Example 2:

In 1980, CJG Mackenzie, professor and acting head, Department of Healthcare and Epidemiology, University of British Columbia, Vancouver, published the following commentary in the CMAJ:

Lead poisoning due to ingestion of lead-based paint reached epidemic proportions in older housing areas in the United States…There is no evidence of a serious health threat to Canadian children from environmental lead exposure. However, epidemiologic data confirming this assumption are scanty and largely limited to hospital records. (Mackenzie, 1980, p.1347-1348)
Mackenzie’s letter to the CMAJ listed 21 references. Note that 18 of these citations pertain to American sources. The 3 Canadian sources Mackenzie cited were:


Observe that the third reference (Mehkeri et. al, 1976) is the only Canadian survey pertaining to an investigation of children living in a non-smelter community. Melkeri, Romanowski and Smallbone’s survey determined that 5-8% of Montreal children screened had BPb ≥ 40 μg/dL (Mackenzie, 1980, p.1347). Even so, Mackenzie’s discourse is dismissive of a problem, as evidenced by the following statement: “There is no evidence of a serious health threat to Canadian children from environmental lead exposure” (p. 1347). The second citation in Mackenzie’s commentary (1980) references a conference proceeding (Neri et. al, 1978), a source one assumes was relatively inaccessible in 1980, a time before internet access became widely available. Mackenzie does not include a citation for information pertaining to “hospital records.”

Example 3:

Authors Wang, Pizzolato, and Demshar report that the Ontario Ministry of Health “had been routinely determining lead in blood since 1970” (Wang et al.,
1989, p.251). Again, no citation is provided for the 1970 Ontario blood lead survey data alluded to.

**Example 4:**

In a 1997 article, Wang, Pizzolato, Demshar, and Smith reported:

Since 1980, the Ontario government has conducted several blood lead screening surveys in children in several cities and regions of Ontario, Canada. The blood lead concentrations in Ontario children has declined in both rural and urban areas over the past decade, this decline coinciding with the complete phasing out of lead in gasoline in 1990 (Wang et al., 1997)

The authors cited data from the following surveys:

The blood lead screening and surveys evaluated were done in the Toronto Western Health Unit in 1984, 1985, and 1988; the Peel Region, 1987; the Niagara Region, 1987; Southern Ontario, covering more-urban areas (Toronto, Windsor, etc.), 1984 and Northern Ontario, covering the less-settled areas (e.g., Thunder Bay and Moosonee), 1987 and 1992 (Wang et al., 1997)

Note that none of the above-mentioned survey data is publicly accessible (i.e. published in peer reviewed journals). Furthermore, Smith, Wang, Pizzolato, and Demshar, were employed with the *Ontario Ministry of Health*. Smith was a senior consultant with the Ontario Public Health Branch. The authors’ stated positions within the Ontario government conveys authority. This text example describes that average blood lead levels of Ontario children had declined since leaded gasoline was phased out. Importantly, what is missing from Wang and colleagues’ 1997 report is a discussion of prevalence rates - the proportion of Ontario children who continued to be identified with PbB ≥10 μg/dL in the post leaded gasoline era (circa 1990-1997).
Example 5

In a 1993 article, found in the trade journal, *Public Health and Epidemiology Report Ontario* (PHERO) Smith refers to four smaller Ontario blood lead surveys of Toronto children (ages 4-6 years) conducted during 1984, 1987, 1990, 1992 (Smith, 1993, p.214). Again, Smith does not provide a citation for the Toronto surveys – which might infer that access to this information was privileged (i.e. not published in a peer reviewed or publicly accessible journal). Again, Smith’s discussion does not provide a description of prevalence rates - the proportion of Ontario children who continued to be identified with PbB ≥10 μg/dL. The PHERO (1993) article lists one citation:


Koren and colleagues’ investigation (1990) reported that the majority of cord blood lead levels sampled in Toronto, Ontario infants were below the laboratory’s detection limit, a point which appears to support Smith’s position that childhood lead poisoning was no longer a problem in Ontario. Note however, that infants are not generally considered as a high risk group for lead poisoning. That is, children do not typically display elevated blood lead levels until they begin to crawl and explore their environment, usually around age 6 months (Lanphear et. al, 2002; CDC, 2007).

Example 6

In a 1995 issue of the CJPH Smith and Rea reported:

The Centers for Disease Control and Prevention has described lead poisoning as one of the most common and preventable pediatric
health problems in the United States. In Canada, however, blood lead levels have been consistently lower than in the United States: provincial and community surveys show blood lead levels in Canadian children have declined markedly over the past decade as lead has been phased out of gasoline (5-8) (Smith & Rea, 1995, p.373). [emphasis added]

The supporting citations for the statement: “blood lead levels in Canadian children have declined markedly over the past decade as lead has been phased out of gasoline (5-8)” were listed as follows:


Note that references 5, 6 and 7 pertain to investigations of smelter communities and thus might be deemed invalid for estimating prevalence rates for non-industrial exposed Canadian children. Further, note that Smith and Rea provide a reference (8) to a government report even though this same information was available in a separate, publicly accessible document (O'Heany et al., 1988). Smith and Rea’s choice to cite a relatively inaccessible government source advantaged the view that childhood lead poisoning was a problem of the past, or an American problem because the reader’s ability to substantiate this information was limited. Recall that Article 2 demonstrated that the 1984 prevalence rates for Ontario children (O'Heany et al., 1988) were comparable to prevalence rates from the same era for American children. Further to this, Smith and Rea’s discussion
lacked balance as it omitted the findings from two contemporary blood lead surveys occurring in Ontario circa 1993 (Alder et al., 1993 and Leung et al., 1993). These latter two investigations, both occurring in London Ontario, identified prevalence rates (PbB ≥ 10 µg/dL) in children ranging from 8 to 17% respectively, a finding which contradicted Smith and Rea’s position that lead poisoning was a problem of the past or predominantly an American problem.

The axis of authority. Rawlinson’s second line of inquiry investigates and describes “the system of power” or what she also refers to as the “bureaucratic dimension of discourse” (Rawlinson, 1987, p.378). The American response to lead poisoning is described at length in several documents published by the U.S. Centers for Disease Control and Surveillance (CDC, 1991b; CDC, 1991a; CDC, 2002; CDC, 2007). These documents served as a reference for what might be an expected public health response in Canada if a similar problem was viewed as “authorized” discourse. The following questions guided this reflection: What Canadian institutions were established to monitor any putative problems related to children’s exposure to lead-based paint? What (health) agencies were assigned to monitor children identified with lead poisoning? What funding was provided to remediate homes identified as containing lead-based paint? How did Canadian regulatory agencies define a residential lead-based paint hazard? What training programs were established to teach contractors and trades-people the safe repair and management of lead-based paint in residential dwellings?

Analysis along this axis began with the systematic review of public health discourse (Article 1) to identify gaps in Canada’s “health protection domain.” Article 2 extended this analysis by reviewing Canadian policy, rules and regulations which assign responsibility to federal agencies regarding health and housing; and also by
reviewing the Canadian response to lead, particularly lead-based paint, as evidenced in federal policy and legislation.

Kendall and Wickham (1999) use the terms “sayable” and “visible” to denote how discourse (the sayable) within a given context is productive (the visible). American public health discourse describing the “problem” of childhood lead poisoning, for example, led to the creation of federal laws such as Title X (1992), the provision of federal funding for home remediation, the development of a national lead strategy, the creation of mandatory screening guidelines for “at-risk” populations, training programs for health professionals and those involved in home repair and renovation, and evaluation programs to measure the effectiveness of these programs. Importantly, emerging new discourse on lead poisoning created new subjects such as vulnerable populations and specialized areas of research and sub-disciplines (i.e. children’s environmental health) and new terminology (i.e. critical windows of exposure and developmental neurotoxins).

Using the American experience for comparison, the analysis (Article 2) critically reviewed the Canadian public health response to lead poisoning. Three reference documents were germane to this review: 1) the 1992 compendium developed by CMHC, “A reference guide to lead research in Canada: A compilation of scientific publications, expertise and current legislation”; 2) Cooper’s critical analysis, “Environmental Standard Setting and Children’s Health” (2000) which critically reviewed federal regulations related to lead in consumer products; and; 3) “Health policy approaches to children’s environmental health: A research report to Health Canada” a recent systematic review of Canadian laws and regulations pertaining to children’s environmental health (Krewski et al., 2006). These documents established that Health Canada and CMHC were the two agencies deigned with primary jurisdiction over health, housing and lead. Also
useful for this analysis was the 2003 Regulatory Impact Analysis Statement (RIAS) that accompanied changes to federal regulations pertaining to lead in new paint and consumer products as it provided background information pertaining to lead paint regulations including the results of earlier market surveys.

Comparing Canada’s situation with the U.S. experience highlighted a third contingency: Canada does not possess a federal law similar to Title X which mandates shared responsibility among federal agencies. Article 2 described how tensions created by a lack of legal directive resulted in tensions between Health Canada and CMHC so that Health Canada eventually assumed the lead role with the end result that CMHC’s important research on lead-based paint never became translated into policy/regulation. Witness, for example, the absence of CMHC activities related to lead-based paint after 1995 and the absence today of legislation pertaining to historic sources of lead-based paint. Note also that Health Canada’s national “Lead Risk Reduction Strategy” (2002) lacks any discussion of historic sources of lead-based paint.

The axis of value or justification. Rawlinson’s (1987) third axis of investigation, also referred to as the “technologies of power,” examines systems which establish authority such as the apparatuses which maintained the dominant public health discourse on lead poisoning and the social practices and power arrangements that were necessary for discourse to maintain this dominant position.

As discussed, this thesis proposes that privileged access to blood lead surveillance data was a major contingency shaping what was known about lead poisoning. This contingency proscribed limits on what could be said or discussed on childhood lead poisoning in Canada and served as the major policing function
by disadvantaging the counter view proposing that a problem with lead-based paint did exist in Canada. Restrictions on who was authorized to speak were also established. Nurses, for example, played a central role in community investigations by recruiting participants, for example, (Levallois et al., 1991b) and collecting demographic information (Jin et al., 1995). As a group, nurses were possibly the study partners most intimately involved with families affected by lead poisoning. And yet nurses’ voices were noticeably absent from “official” public health discourse. Nurses rarely described their experience, for example, at least publicly (i.e. in Canadian health related journals).

A rare example of public “nursing” discourse is found in the book “Turning lead into Gold.” Written by Nancy Hallaway, nurse and mother, and co-authored with Zigurts Strauts, a physician who practised alternative medicine, authors describe, anecdotally, Ms Hallaway’s and her twin boys’ experience with heavy metal poisoning. The following excerpt from Tenebein’s review of Hallaway and Strauts’ book (1996) illustrates the way in which nursing speech was viewed as possessing little or no authority to speak on the topic of childhood lead poisoning and thus liable to “censorship:”

Turning Lead into Gold is written in a colloquial style, heavily spiced with vernacular, such as "Cadmium is one bad ass supertoxic heavy metal… (CMAJ, 1996, p.1208)

And,

This book belongs on the shelf that I discovered [it] at the library. All of us should read one of these books, but only one. The experience provides insight (CMAJ, 1996, p.1208).

Tenebein further laid out the ‘unspoken’ rules for who had the right to speak on this issue and who did not:

As a pediatrician and a toxicologist, a card-carrying member of the untrustworthy and despicable mainstream, I find it easy to poke

Recall from Article 2 that Tenenbein’s 1990 case study was one of the few discourses that problematized the issue of lead poisoning/lead-based paint in Canada; and further, how Tenenbein struggled to find relevant prevalence data to support his argument that the cases he described were not isolated appearances. Foucault describes how “willing bodies” that support the status quo are produced through power/knowledge contingencies (Powers, 2001). Tenenbein’s volte-face in perspective in his 1996 critical review illustrates his transformation into Foucault’s “willing body” when he discredits Hallaway and Strauts’ view “from the trenches” by attacking, for example, Hallaway’s vernacular.

What remains puzzling, and what cannot be explained, is that advocacy groups in Canada were equally slow to respond to the issue of lead-based paint. Canadian research pointed to a problem (Article 1, Appendix A), and yet, even well informed agents seemed unconcerned. The following excerpt provides an example of this paradox:

Since the paint used during the 20th Century (up until at least the 1960s) has been equally loaded with lead, the difference between the U.S. and Canada can reasonably be attributed to two general factors: the much lower prevalence of old, tenement-style housing in Canada and the social safety net that has ensured much lower levels of overall, and especially childhood poverty in Canada (Cooper, 2000, p. 272).

Cooper’s appraisal of Canada’s lead problem is surprisingly naïve given her thorough and critical analysis of Canada’s laggard regulatory response to leaded gasoline, (new) paint, and consumer products (Cooper, 2000). Stone’s suggestion (1989) that dominant beliefs are more likely to be adopted if they correspond with widespread and deeply held cultural values helps to partially
explain this phenomena. Perhaps a deeply rooted pride in Canada's publicly funded healthcare system curbed any normal skepticism that should have arisen in response to reasoned and thoughtful discourse emanating from a small but informed group of voices, as noted by Cooper's reference to Canada's "social safety net." Other than this excerpt however, no other Canadian text expressing a similar "cultural" belief was found. Due to a lack of supporting text, therefore, discussion of this latter contingency was excluded from the final analysis (Article 2).

It might also be suggested that the preferential weighting of certain types of knowledge (i.e. scientific, empirical, objective, statistically driven data) over other types of knowledge (i.e. opinions, observations, anecdotal evidence, case studies) was equally influential in shaping the Canadian response to lead. Tenenbein's discourse (1996), dismissive of Hallaway and Strauts (1995) anecdotal evidence, for example, supports this thesis; and yet, the Canadian response to lead was shaped by more than just the privileging of certain 'knowledges' over others. Clearly, even epidemiological evidence emanating from "authorized" sources identified a problem. Children's prevalence rates in London, Ontario; Quebec City, Quebec; Vancouver, British Columbia; Montreal, Quebec and St. John, New Brunswick pointed to a problem. This comparatively "strong" evidence however, was either ignored (i.e. Alder et. al, 1995; Leung et. al, 1993) or downplayed by focusing attention on declining average values (i.e. Wang et. al, 1997); or, by contrasting dated American surveillance data with more recent Canadian survey data (i.e. Levallois et al., 1991a; Smith, 1993; Jin et al., 1995).
3.2 Final reflections

Critical analysis, by its nature, is transformative, enabling new insights, and laying out the basis for new actions regarding identified problems (Blackledge, 2005). Discourses can be effective in bringing dissenting agents to change their positions - or to endorse the dominant discourse. This aspect of critical analysis - the manner in which critical researchers attempt to "restructure social relations of domination," is rife with political and ethical considerations (Kincheloe & McLaren, 1998, p. 279). For this reason, critical discourse analysis does not end with the demystification of a dominant discourse; rather, it is expected that the emergent discourse will be itself subject to the same rigorous critique and analysis applied to the preceding discourse. Powers, for example, identifies the concern that naming and describing a resistance discourse might have a co-opting effect on marginalized discourses (Powers, 1996). Assigning 'vulnerability' status is also extremely powerful and comes with social, ethical, and political implications (Hall et al., 1994). Further, Schneider and Ingram note that social constructions can be "manipulated" and used by public officials, the media, and advocacy groups themselves to create "new target groups" (Schneider & Ingram, 1993, p.342). Other have expressed the concern that the "[s]ocial relations which emerge from a reaction to a critique are themselves simply new forms of power and [therefore] subject to renewed skepticism" (McKerrow, 1989, p. 96). Or, that new causal theories can be used as an instrument of social control to maintain existing patterns of dominance (Stone, 1989a, p.296). These considerations underscore the need for further reflection: What are some other possible representations of childhood lead poisoning? How do these alternative representations position policymakers, decision-makers and health care providers with regard to this issue?
One possible outcome of critiquing the Canada Health Measures Survey (2010) is that other important research findings from this investigation might also be summarily dismissed. A complete dismissal of the CHMS findings was not an intended purpose, or hoped for outcome from this critical analysis. The CHMS report identified important associations: blood lead concentrations tended to be higher in adults than in children; older adults (aged 60 to 79) had the highest concentrations (Bushnik et al., 2010). These findings raise important policy implications for Canada’s aging population. Older adults, raised as children in Canada during the leaded-gasoline era would have been more highly exposed to environmental sources of lead than younger Canadians - those born post 1990 and this information should be used to inform health care decisions and strategies for health promotion and trigger further study. It will be important to know, for example, how ‘ancient’ lead stored in bone affects the mental and physical well being of Canadian women as they experience menopause, a period when bone lead can be remobilized into blood as bone tissue undergoes hormonally driven adsorption.

Other interesting associations were also identified in the CHMS survey. Higher concentrations of blood lead were found in individuals of lower income, those born outside Canada, those living in a dwellings at least 50 years old, current and former smokers, and in individuals drinking alcohol at least once a week (Bushnik et al., 2010). These factors too deserve further investigation. This analysis demonstrates that clearly, lead poisoning is an under researched area in Canada. Findings from the CHMS should provoke further research into the impact of environmental lead exposure on the health of Canadian populations.

One last note of caution: Because childhood lead poisoning is associated with the lead dust found on floors of family dwellings, there is the danger that emerging new discourse describing the “problem of lead” could well be framed as
a hygiene issue. Lead poisoning might be portrayed, for example, as a health issue mainly confined to poor uneducated families with lax knowledge and awareness regarding the domestic skills necessary to prevent this problem from occurring. This framing shifts the blame from the underlying problem – lead-based paint, and the industry that produced and promoted its use, to the victims.

Canadian populations would be better served if this problem were viewed as a housing issue, with the focus on the unhealthy condition of the home and the unhealthy dust created as lead-based paint *naturally* deteriorates – rather than the hygienic practices of families affected by lead. It is important to establish this distinction early on so as to avoid affixing public health’s gaze on, as Lanphear describes it, “passing out brochures and mop buckets” rather than promulgating regulations to protect children from residential lead hazards (2005, p.2274).

### 3.3 Implications for nursing

#### 3.3.1 Implications for nursing practice

Public health and community health nurses, moreso than other health professionals, are oftentimes granted privileged and intimate access to families’ home environments, a work-place characteristic that lends an advantage to nurses in that they have an opportunity to play a leading role in bringing about change; by witnessing, for example, deleterious living conditions, by documenting these concerns, and then advocating, politically, for better housing conditions. Part 1 discussion illustrates how primary prevention – the remediation of lead sources in residential dwellings, is cost-effective. Historically, public health nurses have influenced public health policy. Baltimore public health nurses, for example, through home visits, were the first to identify lead-based wall paint (and not toys) as the primary source of exposure for young children (Williams, 1952). Advanced
practice nurses such as Nurse Practitioners, also have an important role to play by identifying cases through targeted blood lead screening of “at risk” populations – children under the age of 6 years living in pre-1990 housing and by writing about these cases in peer reviewed journals.

3.3.2 Implications for nursing research

This thesis used lead as a case study, to demonstrate the effectiveness of poststructuralist research in tracing the connections between historical text, institutions and regulations and by providing a model for the analysis of power relations by using Foucault’s archaeological/genealogical approach to critical analysis. This method demonstrated a novel approach to nursing research by considering, and laying out for the reader, the numerous ‘historical’ events that might otherwise have been overlooked had a more traditional type of analysis been applied. The utility of this study, apart from serving as an exemplar for radical investigations, lies in its potential to “smooth the path” for future research into lead poisoning by presenting examples which can be used to support the conviction that more research into lead poisoning is needed in Canada. Hopefully the arguments offered from this analysis will adequately justify future requests for funding to investigate what has historically been constructed as, and viewed in Canada as, a “non-problem” or a problem of the past.

It is clear, from American research and now Canadian research, that higher blood lead levels in children and adults, are associated with socioeconomic disadvantage. This, perhaps, more than any other factor, should pique the interest and sharpen the attention of Canadian social scientists including nurse researchers. Discourse describing the health disparities occurring between economically advantaged individuals and less advantaged populations is abundant
in the literature, including Canadian discourse. What is needed now, and what has been missing from public health discussion thus far, is the ‘hidden’ yet powerful impact of physical environmental factors on health outcome. As noted previously, lead is one of the few factors with a clear “cause and effect” etiology and a clear link to socio-economic factors (Richardson, 2005). One might argue that research should account for poverty (and not lead) as a confounding factor in community investigations.

3.3.3. Implications for nursing education

One of the underlying aims of this analysis was to highlight the association between socio-economic factors (i.e. poverty), vulnerable populations (children <6 years) and childhood lead exposure. True, discussion of environmental factors such as housing condition more commonly enter into public health discourse (Dunn et al., 2003; Public Health Agency of Canada, 2004; 2006; Raphael, 2004). And yet, discussion of housing’s importance to health, unfortunately, is narrowly limited to only the socio-economic aspects, such as affordability (Hays et al., 2006). While economic factors can and do play a role in shaping health outcome, physical environmental factors such as chemical and biological hazards found within the home can also lead to poor health.

Using lead as an exemplar, and Dixon and Dixon’s model (2002) as the framework, this analysis expands the traditional view of nursing ‘purview’ by bringing to light the often unseen influence of environmental factors, particularly in terms of their importance as root causes of adverse health events. While poverty is now firmly entrenched as a determinant of health nurse educators are well positioned to challenge this association: Should poverty be considered as a root determinant of health outcome – or should poverty more accurately be considered
in terms of its association with an elevated risk for lead poisoning? Consider that neighbourhoods where low income families commonly reside are the same neighbourhoods which contain high soil and dust lead levels (Wakefield & Baxter, 2010; Kjellstrom et. al, 2008). Public health nurse educators are well positioned to take a lead role in discussions which challenge the ‘accepted’ view of poverty as a determinant of health, by incorporating into the classroom discussions of environmental factors such as lead poisoning and their longterm and detrimental health consequences.

3.3.4 Implications for nursing administration/management

The question facing nursing administrators today is how to use scarce public resources effectively to maintain optimum population health. This thesis proposes a focus on more economically viable efforts such as the identification of the root causes of disease/illness by recognizing the detrimental impact environmental factors such as lead, mercury, PCBs, dioxins and pesticides have on health outcome; and by recognizing how environmental health is oftentimes linked inextricably with social justice issues. In light of this ‘new’ knowledge, agencies such as the Canadian Nurses Association (CAN) have responded through endorsement of a broad systems approach, one which focuses attention on the spectra of social political and economic forces that shape health outcome in policy (2007). By thoughtful reform of nursing practice guidelines (i.e. professional standards and code of ethics) the CNA can further bring about real change by explicitly calling upon nurses to improve the physical living conditions for the economically disadvantaged through political action. By addressing poverty and malnutrition, by eliminating sources of children’s environmental exposure to
developmental neurotoxins such as lead, nurses will be truly promoting an upstream and cost effective approach to maintaining health.

3.4 Limitations of this study

The content for this analysis was derived mainly from text found in peer reviewed journals, web sites, media and policy papers. Using text as the sole basis for this analysis may have contributed to a one-sided understanding of this issue. To counteract any ‘subjective’ limitations imposed on the interpretation of the literature, this examination strove to include, in all discussions, a description of the context in which articles and other texts were written by seeking out related media reports and discussions found in letters and commentary. To further strengthen the analysis, American public health discourse on lead was extensively reviewed and then contrasted with Canadian public health discourse. The review encompassed public health discourse extending from the 1930’s onwards to facilitate the identification of surfaces of emergence, that is, points in time where discourse transformed events, but also, examples in which Canadian discourse diverged from American discourse in time.

In analyzing the texts used in this thesis, another contingency became apparent - the exploration of which was not possible. It concerns the way Canadian research on lead poisoning is typically funded through either government agencies or, in the case of investigations in smelter communities such as Trail British Columbia, by a combination of provincial government funding and industry. Concerns have been raised regarding the way corporate funding may influence the types of results produced (Bekelman et al., 2003). How might a funding contingency have shaped events? How might this factor have affected the ability of Canadian researchers, for example, to publicly discuss findings? And how might
this contingency frustrated alternative views from surfacing? How does the Canadian experience, in this regard, compare with the American experience? While addressing these questions at length would have been difficult in the context of this study, they are important for understanding the various ways public health discourse on lead was shaped over the years in Canada. These questions need to be explored further, from a feminist perspective perhaps, by examining the social, economic, and political contingencies that shaped research on childhood lead poisoning.

Another limitation of this study is its general lack of discussion pertaining to the federal-provincial relationship underlying the Canadian healthcare system. Jurisdiction over environment in Canada is shared between federal, provincial and territorial governments. Provincial laws in Ontario include standards for lead in drinking water, labour laws (i.e. occupational exposure) and programming guidelines for health units; whereas, municipalities assume responsibility for providing clean drinking water to consumers (Krewski et. al, 2006). While primary jurisdiction over health care rests within the provincial domain, federal government policy plays an important role both politically and financially, a factor which can lead to tensions among federal and provincial powers (Maioni, 2002). Importantly, the federal government allocates monies for provincial healthcare initiatives (i.e. shared-cost hospital and medical insurance) (Maioni, 2002). This contingency (shared power) is viewed by Maioni (2002) as one of the most influential factors shaping health care in Canada. A review of the provincial response to the public health issue of childhood lead poisoning might have revealed other important and equally influential contingencies. Certainly this factor deserves further attention. This field of investigation, however, fell outside the scope of this thesis, which aimed to review the federal response to lead poisoning and lead-based paint.
CONCLUSION

Is childhood lead poisoning a problem in Canada? Or is it a problem of the past? How do we know? Epistemology, the philosophy of knowledge, is what guides methodology in any research endeavour and is what guided this analysis. Epistemology concerns itself with questions of what can be known and who can be a knower. Epistemology also dictates what criteria must be met in order to consider ‘beliefs’ as ‘knowledge.’ The philosophy of knowledge is what justifies or authorizes how “truth” will be measured within a given historical context (i.e. common sense, statistical significance, scientific proof, an educated guess). A primary objective of this research analysis was to examine the underlying beliefs or criteria which constitute or authorize what is known about childhood lead poisoning in Canada. The following statement found on Health Canada’s website, for example, suggests that lead poisoning is a rare occurrence in Canada:

Very few cases of lead poisoning are documented in Canada each year. However, since low-level lead poisoning is often unrecognized, it is difficult to determine the number of Canadians affected by exposure to low levels of lead (Health Canada, 2009, p.6)

A closer inspection however, reveals a clever tautology which renders this statement difficult to dispute or even verify. The phrase “Very few cases of lead poisoning are documented in Canada each year” uses value laden terms such as “very few” and leaves this statement open to wide interpretation. What is meant by “very few”? A supporting reference for this “figure” is not supplied.

An examination of the Canadian public health archives reveals that discourse describing childhood lead poisoning predominantly follows three themes: Lead poisoning is a problem; a non-problem; or, a potential problem requiring more
investigation. The following excerpts illustrate the way these three perspectives are represented in the literature:

* A problem.

Lead poisoning is a preventable disease in children and lead-containing paints are the chief source of the poison. In order to adequately prevent this condition, the elimination of these paints from the immediate environment of the child during the second and third years of life is essential. Lead-free paints are readily available (Ross & Brown, 1935, p.242).

Assuming there are about 1,325,705 urban children in Canada (an estimate), if 5% exceed 0.05 $\text{mol/l}$ (10 $\mu$/dL), there may be as many as 66,285 urban children with blood lead levels greater than 0.05 $\text{mol/l}$ (10 $\mu$/dL) (Health Canada, 1994, p. iii)

A 1989 study in Vancouver found that about 7% of preschool children had a blood lead level of [10 $\mu$/dL] or more. If this is typical of all Canadian preschool children, 150,000 of them likely have such levels (Godolphin et al., 1993, p. 517).

Lead based paint continues to cause occasional problems in older houses, a situation whose abatement is long overdue (Godolphin et al., 1993, p. 518).

* A non-problem.

Lead poisoning due to ingestion of lead-based paint reached epidemic proportions in older housing areas in the United States….There is no evidence of a serious health threat to Canadian children from environmental lead exposure. However, epidemiologic data confirming this assumption are scanty and largely limited to hospital records (Mackenzie, 1980, p.1347-1348).

There are a number of avenues of exposure to lead in the environment. Lead-based paint has been cited as the most common source of lead exposure for children in the United States. It has not been established that this is the case in Canada (OMOH & MOEE, 1985, p.1)

In striking contrast with the U.S. experience, the Commission received no hard evidence that paint was a significant source of lead uptake. Indoor flaking paint has been seen as a main cause of high blood levels in U.S. inner cities (and some isolated rural cases). No evidence of such effects exists for Canada, though several briefs to the Commission have suggested that an impact must exist, especially in the older cities (Royal Society of Canada, 1986, p.265-266)
Since lead has been eliminated from gasoline and in the absence of clinical and laboratory evidence to the contrary, we believe that no serious threat of lead exposure to children exists in Canada (Godolphin et al., 1993, p. 518-519).

A potential problem requiring more investigation.

The prevalence of neurotoxic lead levels in asymptomatic children is unknown in Canada. Sound epidemiologic studies of blood lead concentrations involving children living in Canadian inner city areas are needed to define the extent of lead poisoning from lead-based paint (Tenenbein, 1990, p.41).

Little data are available on childhood lead exposure from paint in Canada. The present descriptive data suggest that more research into this potential problem in Canada is warranted (Alder et al., 1993, p.300).

In Canada, other than in areas associated with point source lead emission, lead poisoning has not been perceived to be a problem. There has been no coordinated effort to determine the epidemiology of childhood lead exposure in Canada (Lockitch, 1993, p.377).

Three seemingly disparate views; which one is ‘true’? Foucault recognizes the political and social nature of epistemology, and directs the critical researcher not in a search for truth but in a search to uncover the variety of constraints and restrictions which delimit what can be said or constituted as ‘true’ (Cheek, 2000).

A secondary aim of this research study was to determine how it is that certain agencies, Health Canada for example, are given the authority to pronounce ‘truth’ on the subject of childhood lead poisoning, i.e. who can speak on the issue and who can not, and who is deemed as possessing authority on this matter. Further examples were provided in the discussion (Part III).

A third objective of this study was to “problematize” the issue of childhood lead poisoning in Canada, chiefly as it applies to cases occurring from exposure to lead-based paint. This objective was achieved by deconstructing the problem of lead poisoning using an adaptation of Foucault’s method of
archaeological/genealogical analysis as described by Rawlinson (1987). Rawlinson’s axes of inquiry facilitated a Foucauldian analysis towards discerning knowledge, power, and “truth” via an historical (archival) examination of “surfaces of emergence” and the underlying contingencies which supported the construction of the “problem” or “non-problem” in Canada.

The first of two articles, “Applying Dixon and Dixon’s Integrative Model for Environmental Health Research Towards a Critical Analysis of Childhood Lead Poisoning in Canada,” (Article 1) entailed a systematic review of Canadian research on childhood lead poisoning. Dixon and Dixon’s framework (2002) was used to group Canadian research pertaining to the concepts of lead poisoning, housing, and children into the model’s four domains: physiological, vulnerability, health protection, and epistemological. This process was useful for solidly identifying research gaps within the Canadian context but in also setting the groundwork for the subsequent critical analysis. Article 1 established that the issue of lead was an under-researched area of investigation in Canada. Gaps in our current understanding include a lack of recent national blood lead surveillance data specifically for children ages 1 to 5 years and the absence of systematic surveys mapping the presence of lead-based paint in residential dwellings. This systematic review was essential to bring this analysis to the next ‘step’ - to critically review the issue of childhood lead poisoning by exploring what historic events led such ‘gaps’ to exist.

Part I discussion explored the roots of critical theory and described how critical discourse analysis, based on Foucault’s method of archaeological/genealogical investigation (Foucault, 1966; 1972a) was an appropriate method for this research study due to this method’s emancipatory qualities, but also for its ability to root out the underlying contingencies which
support and maintain current understandings of lead poisoning; and equally important, for its utility in expanding the field of nursing research, especially as it pertains to the domain of environment.

Foucault’s power/knowledge dyad produces various technologies that prove effective in maintaining and circulating dominant views about particular phenomena. The second article, “Reformulating lead-based paint as a problem in Canada” (Article 2) illuminated the socio-political and economic constraints that shaped health-care knowledge on childhood lead poisoning as it pertained to lead-based paint, and how, ultimately, this discourse influenced the way healthcare providers and policy makers in Canada produced and used this information.

A counterview to this thesis could well be that a general lack of attention paid to lead in Canada, in general, occurred simply because there was no evidence of a problem and therefore, no need for a response. Simply put, the American response to lead was a reflection of a problem in that country, the Canadian response to lead was a reflection of the non-problem in this country. Discussion in Article 2 counters this argument, for example, by recounting CMHC’s sedulous response to lead-based paint following the discovery of the Medicine Hat cases. The expenditure, by CMHC, of considerable resources towards investigating lead-based paint over a period of at least four years, supports this thesis’ contention that the comparatively weak Canadian response to lead-based paint was not just an artefact produced by a lack of supporting evidence.

New discourse emerging from the Canadian Health Measures Survey (CHMS) does not offer a way to counter this gap because the survey design failed to investigate the true extent of any potential problem by excluding a vulnerable population, children ages 1 to 5 years. Article 2 describes this contingency as a
“serious flaw.” This limitation, at the very least, should have been addressed in the accompanying literature disseminated by Health Canada.

This thesis describes evidence which supports the view that a problem with lead-based paint exists and proposes that surveillance data constituted both a product and a catalyst of the dominant view on childhood lead poisoning and further posits that a lack of Canadian context specific surveillance data was the major “policing” factor limiting knowledge and thus Canada’s public health response. Secondly, privileged access to primarily government surveillance data maintained the view that childhood lead poisoning was a problem of the past or an American problem. Third, tensions among Canada’s two federal agencies which held primary responsibility for lead, health and housing, produced a vastly weaker response to lead, as contrasted with the American public health response whereby to date, no legislation exists to protect vulnerable populations of Canadian children from exposure to historic sources of lead found in residential dwellings.

In the final analysis, the implications of this research analysis, particularly as it pertains to the discipline of nursing, were discussed. Consideration was given to implications for policy change as well as the potential harm arising from emerging ‘new discourse.’ The importance of framing this health issue as a problem generated by lead paint and not an outcome of poor hygiene practices was discussed. Lastly, suggestions for areas of future research were described.
REFERENCES


Ref Type: Journal (Full)


CDC (2005a). *Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control and Prevention* Atlanta, Ga..


1. *Environmental Health Perspectives, 89*, 101-105.


APPENDICES
APPENDIX A

Canadian case, cross sectional and environmental studies which incorporate residential sources of lead as a risk factor in the analysis
Key

BLL = blood lead levels
EBL = elevated blood lead level
GM = geometric mean
µg/dL = micrograms per decilitre
CLP = childhood lead poisoning

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<tr>
<td>Author(s):</td>
<td>Alder RJ, Dillon JA, Loomer S, Poon HC, Robertson JM</td>
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<tr>
<td>Title:</td>
<td>An analysis of blood lead data in clinical records by external data on lead pipes and age of household.</td>
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<tr>
<td>Year:</td>
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<td>Reference:</td>
<td>Journal of Exposure Analysis and Environmental Epidemiology 3(3):299-314</td>
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<td>Stratification (age, sex, etc.):</td>
<td>Participants not stratified by age however population identified as mean age 9.6 yrs (22% ≤ 5 yrs); 65.9% male; 4.9% native Indian; 4.6% immigrant</td>
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<td>Group selection method:</td>
<td>Children admitted to CPRI between 1984 &amp; 1989</td>
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<td>Association between BLLs and living in certain districts of London. Follow-up environmental investigation to identify sources of Pb exposure (see Poon et al., 1989)</td>
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<td>Data source format (e.g., questionnaire, medical records, etc.):</td>
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<td>--------------------------------------------------</td>
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<td>Exposure measurement</td>
<td>Age of dwelling – obtained from Municipal Property Tax Assessment File (used as a surrogate for lead-based paint use) BLL</td>
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<td>Toxicological/biological relevance of exposure measurement</td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
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<tr>
<td>Adjustments</td>
<td>Gender, year of lead test (surrogate for gasoline source), census tract prevalence of low family income, children in lead service area Logarithmic transformations, ages categorized; “year built” categorized as during or before 1945, 1946-1955, 1956-1965, 1966-1975, and &gt;1976</td>
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<td>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</td>
<td>Zeeman graphite furnace atomic absorption spectrophotometer. Precision was within 5%</td>
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<td>Results:</td>
<td>Among the 36 children ≤ 5 yrs GM was 4.2 μg/dL 3 children (8.3%) had a BLL ≥ 10 μg/dL Children living in industrialized areas of the city had 48.6% higher BLLs than those in less industrialized areas Mean BLL for children living in buildings constructed during or after 1945 was 62.3% higher than that of children in buildings constructed during or after 1976</td>
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<td>Procedures/tests:</td>
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<td>Significant findings:</td>
<td>Children diagnosed with conduct disorder tended to have higher lead levels than those without such a diagnosis (p &lt;0.003) “Using municipal tax assessment data on the age of each child’s home, those children living in homes built during or before 1945 (when interior paints were as much as 50% lead by dry weight) had an average blood lead level that was 62.3% higher (p = 0.011) than that of those in homes built since 1975 (when interior paints were limited to no higher than 0.5% lead by dry weight)”1, p 299-300</td>
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<td>Non-significant findings:</td>
<td>After adjusting for gender, year of lead test (a surrogate for gasoline source), and census tract prevalence of low family income, children in the lead service area (LSA) were found not to have higher blood lead levels (GM: LSA-4.7 μg/dL, Non-LSA – 4.8 μg/dL; p-0.839)</td>
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<td>Biases identified by the authors:</td>
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<td>Assumptions/limitations of the study:</td>
<td>The house at which the [6 year old boy with blood lead levels of 49.7 μg/dL ] residing for 10 months located in one of the oldest and lowest income inner-city neighborhoods in London Environmental samples not collected to verify lead in paint, drinking water</td>
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| Conclusions: | “A clear gradient was observed. This association with age of home remained significant after adjusting for gender, diagnosis, and year of lead tests” 1, p.300  
“The data suggest that with the removal of lead from gasoline, lead-based paint is a significant remaining source of lead exposure” 1, p.300.                                                                                                                                 |
<p>| Google Scholar cited by: (Canadian Sources) | 2-4                                                                                                                                                                                                                                                                                                                                 |
| Comments by reviewer: | 6 year old boy with BLLs of 49.7 μg/dL and a 2 year old sibling with BLLs of 39.3 μg/dL. BLLs of a 1 year old child the mother baby-sat were 11.4 μg/dL and 15.5 μg/dL respectively. One of few Canadian investigations that uses municipal property assessment data to identify construction date of dwelling |</p>
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<td>Balram, C. &amp; Giffin, S. C.</td>
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<td><strong>Title:</strong></td>
<td>A study of blood lead levels in children living in Saint John, New Brunswick. [unpublished]</td>
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<td><strong>Results:</strong></td>
<td>11.3% children had EBLs 7/11 children with EBLs lived in home built prior to 1976</td>
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| Significant findings: | Use of hot tap water in preparation of food \( (p \leq 0.05) \)  
Parent works with lead \( (p \leq 0.05) \)  
Pet in the home \( (p \leq 0.05) \)  
Residing in an apartment \( (p \leq 0.05) \)  
Living in the south end of the city \( (p \leq 0.05) \) |
| Non-significant findings: | Cleaning methods of household occupants assessed in survey but not identified as a risk factor. |
| Biases identified by the authors: | |
| Assumptions/limitations of the study: | Concern following public health investigations of the domestic water in the city which indicated Pb level > 50 ppm in 13% of homes. August 1991, 23 children and 68 adults were tested. 53% of those tested had elevated blood lead levels defined as \( \geq 10 \mu g/dL \). Between May 1991 and February 1992, an additional 205 individuals (age not disclosed) were tested for blood lead levels. 50 (24%) of these tests showed values above \( 10 \mu g/dL \). Highest BLLs found in homes containing lead service lines and/or brass plumbing.  
Environmental samples not collected to verify source of exposure.  
Reliant on homeowner to estimate age of dwelling – this information may not be accurate leading to bias |
| Conclusions: | Investigators identified the following risk factors: living in certain areas of the city, lead service, use of hot tap water for food preparation, peeling paint in the home, a parent employed in the oil refinery or chemical plant; a pet in the home, residing in an apartment complex. Recommend case finding based on identification of “at risk” children; adoption of mandatory reporting of elevated blood lead levels at the national level. |
| Google Scholar cited by: (Canadian Sources) | 4-6 |
| Comments by reviewer: | Neither Balram or Giffin went on to other investigations of CLP as it relates to residential sources of exposure i.e. lead-based paint.  
Balram co investigated a community contaminated by a lead smelter 7 and Giffin co- investigated a |
case of pediatric managanese poisoning.
Dr. Giffin interviewed by CBC 1991: “We reviewed 19 households [in St. John] and we found that out of those 19, 12 of them, recently, had renovations which could well have put lead dust into the air, could have involved removal of lead products.”
<table>
<thead>
<tr>
<th>3.</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Canada Mortgage and Housing Corporation.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Lead abatement protocol 307-7th Street, S.E. Medicine Hat, Alberta</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1993</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Canada Mortgage and Housing Corporation; Ottawa, ON Report No.: 930212.</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>yes</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Case study of a home where intensive renovation work conducted by previous owner disturbed a significant amount of lead in painted surfaces; creating residual dust containing lead. Two children living in the home</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>Sample size: n/a</td>
</tr>
<tr>
<td></td>
<td>Stratification (age, sex, etc.): n/a</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>Sample size: n/a</td>
</tr>
<tr>
<td></td>
<td>Stratification (age, sex, etc.): n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Safety and cost efficiency for the renovation of the property situated at 307-7th Street, Medicine hat, Alberta, to prepare a lead abatement protocol. Follow up environmental investigation following identification of 2 cases of lead poisoning in children living in this home 1-y male (BLL 30 g/dL); 3 y female (BLL 22.9 μg/dL).</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>Environmental sampling</td>
</tr>
<tr>
<td></td>
<td>Method for abatement and remediation follows HUD regulations and guidelines for abatement as well as findings from the Riverdale community study.</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Residential sources of lead</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>Paint, dust, soil, tap water</td>
</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td>BLL identified in media coverage</td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
</tr>
<tr>
<td><strong>Adjustments:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</td>
<td>n/a</td>
</tr>
<tr>
<td>-----------------</td>
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</tr>
<tr>
<td>Interaction assessment:</td>
<td>n/a</td>
</tr>
</tbody>
</table>
| Results: | 2 water samples < 20 µg/L  
8 soil samples from back and front property  
11 dust samples. Pb concentrations ranged from 14 - 39000 µg/g  
19 paint chip samples from interior and exterior surfaces. Pb concentrations ranged from 91 - 140,000 µg/g |
| RR, OR, CI and relevant tables with dose response etc: | n/a |
| Statistical analysis: | n/a |
| Procedures/tests: | n/a |
| Statistical significance: | n/a |
| Significant findings: | n/a |
| Non-significant findings: | n/a |
| Biases identified by the authors: | n/a |
| Assumptions/limitations of the study: | Doesn’t describe prevalence of EBLs in Canadian children, nor does it estimate extent of the problem in Canada. Identifies pre-1976 housing stock as era of concern for LBP use. |
| Conclusions: | Report does not provide recommendations |
| Google Scholar cited by: (Canadian Sources) | 4 |
| Comments by reviewer: | First Canadian environmental investigation of domestic sources of lead exposure related to renovating activities.  
This report not referenced by any other Canadian sources including Health Canada reports. |
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<td><strong>4.</strong></td>
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<tr>
<td><strong>Author(s):</strong></td>
<td>Canada Mortgage and Housing Corporation.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Residential sources of lead [St. John, New Brunswick].</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1995</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Canada Mortgage and Housing Corporation, Ottawa, ON.</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Cross sectional, environmental study</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>St. John, NB</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td>A sub-sample of homes chosen from a previous investigation of 500 homes (Unpublished, Richard Scott, 1995)</td>
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</tr>
<tr>
<td><strong>Sample size:</strong></td>
<td>100</td>
</tr>
<tr>
<td><strong>Stratification (age, sex, etc.):</strong></td>
<td>50 households from the “high blood lead” homes and 50 homes from the “low blood lead” homes</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
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<tr>
<td><strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
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<tr>
<td><strong>Group selection method:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>To examine, post hoc relationships between EBLs and sources such as drinking water, soil, paint, dust and food.</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>BL Questionnaire</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>High and low BLLs groups</td>
</tr>
<tr>
<td></td>
<td>High mean 17 μg/dL (Range 6.8 – 43.6 μg/dL)</td>
</tr>
<tr>
<td></td>
<td>Low mean 2 μg/dL (Range 1-3 μg/dL)</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Residential sources of lead</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>Tap water</td>
</tr>
<tr>
<td></td>
<td>Paint (XRF analysis)</td>
</tr>
<tr>
<td></td>
<td>Soil</td>
</tr>
<tr>
<td></td>
<td>Dust (Pb concentration)</td>
</tr>
<tr>
<td></td>
<td>Food</td>
</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td>Concurrent measure of BLL</td>
</tr>
<tr>
<td></td>
<td>Measured residency (years living in home)</td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
</tr>
<tr>
<td>Adjustments:</td>
<td>Distribution of variables (dust, soil, food &amp; paint) skewed and therefore log transformed</td>
</tr>
<tr>
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</tr>
<tr>
<td>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</td>
<td>Venous blood specimens, flushed water sample from previous study (unpublished, Richard Scott, 1995). BLL analysis at Metro Health Services Trace Element Laboratory Graphite furnace atomic absorption spectrometry, external proficiency testing</td>
</tr>
<tr>
<td>Interaction assessment:</td>
<td>n/a</td>
</tr>
<tr>
<td>Results:</td>
<td>Dust lead levels averaged 42 µg/g in entrance ways (range 1-522 µg/g), to 73 µg/g in living areas (range 1-722 µg/g), to 319 µg/g in master bedrooms (range 5-6601 µg/g)</td>
</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td></td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>Regression analysis – factors related to EBLLs</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td></td>
</tr>
</tbody>
</table>
| Statistical significance: | *1 tailed significance <0.001  
**1 tailed significance <0.01 |
| Significant findings: | *Soil  
*Food  
*Water flush  
*Age of participant  
**Paint  
**Home age |
| Non-significant findings: | |
| Biases identified by the authors: | Home age “strongly related to Log BL (r=0.449), resident age, log water flush and paint. Home age not included in final model (this does not imply that Home Age, as a substitute for Paint, could not be part of an equally good model.) p. 28 |
| Assumptions/limitations of the study: | No description of how age of home determined (i.e. questionnaire or municipal data) |
| Conclusions: | Age of resident, water flush lead content, length of stay, and lead in dust, soil, food and paint predict high or low BL categories ~89% correctly. Main contributing factors to elevated blood lead levels were resident’s age, household water lead levels, and lead in paint |
| Google Scholar cited by: (Canadian Sources) | No Canadian authors cite this report.  
Cited by one American source: 13, p.757 |
| Comments by reviewer: | This environmental investigation done in follow up to an earlier unpublished BL survey in St. John NB. BL clinic held April 1991 which was triggered following the detection of older couple with EBLs: male 48 µg/dL, female 41 µg/dL. Couple advised to not drink tap water, but continued. More water samples collected from residents living in 41 homes; 44 people screened in blood lead clinic (23 households). |
Findings: 84% had BLLs ≥ 10 μg/dL; 27/44 (61%) of participants had BLL > 15 μg/dL; 3 women > 40 μg/dL. Five children detected with EBLs: 4 yr female 18 μg/dL; 5 yr female 17 μg/dL; 5 yr female 12 μg/dL; 6 yr female 9 μg/dL; 6 yr male 12 μg/dL. Highest BLLs found in females > 60 yrs living in their home > 5 years. Tapwater Pb in one home measured 130 ppb after flushing for 2½ minutes. (Unpublished, Richard Scott, 1995).
<table>
<thead>
<tr>
<th>5.</th>
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<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Canadian Broadcasting Corporation (CBC)</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Case of lead poisoning in Medicine Hat Alberta</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>November 19 1991</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Marketplace</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
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</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>National media report</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Medicine Hat family</td>
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<tr>
<td><strong>Case identification/definition:</strong></td>
<td>Elevated BLL</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
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</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>n/a</td>
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<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
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<tr>
<td>• <strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Investigative consumer program. Hosts Bill Paul and Jacquie Perrin interview the parents of two lead poisoned Medicine Hat children. Show broadcast nationally in 1991. Dr. Richard Scott and Dr. Scott Giffin interviewed – conducting investigation into sources of lead poisoning in St. John NB at time.</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>Interview with family</td>
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<tr>
<td><strong>Exposure definition</strong></td>
<td>BLL ≥ 10µg/dL</td>
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<tr>
<td><strong>Exposure medium</strong></td>
<td>Residential sources of lead</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
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</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td>Concurrent measure of BLL</td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
</tr>
<tr>
<td><strong>Adjustments:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Interaction assessment:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>Results:</td>
<td>n/a</td>
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</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td>n/a</td>
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<tr>
<td>Statistical analysis:</td>
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<tr>
<td>Procedures/tests:</td>
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<tr>
<td>Statistical significance:</td>
<td>n/a</td>
</tr>
<tr>
<td>Significant findings:</td>
<td>DR. RICHARD SCOTT: The level of awareness in the Canadian population would probably be close to zero. The reason being we have a perception, a generally held perception that we have legislated lead out of gasoline, out of paints, out of solder and so because of that, people will imagine that lead is no longer a problem. What they forget however, is that we’re dealing with a legacy of the use in the past.</td>
</tr>
<tr>
<td>Non-significant findings:</td>
<td>n/a</td>
</tr>
<tr>
<td>Biases identified by the authors:</td>
<td>n/a</td>
</tr>
<tr>
<td>Assumptions/limitations of the study:</td>
<td>n/a</td>
</tr>
<tr>
<td>Conclusions:</td>
<td>Program estimates &gt; 7 million homes in Canada contain lead paint. Identify at risk homes as those built before 1980</td>
</tr>
<tr>
<td>Google Scholar cited by:</td>
<td>n/a</td>
</tr>
<tr>
<td>(Canadian Sources)</td>
<td></td>
</tr>
<tr>
<td>Comments by reviewer:</td>
<td>Medicine Hat Case significant – first publicly identified case of lead poisoning in Canada resulting from exposure to lead caused by home renovations. Triggered a host of follow-up investigations by CMHC</td>
</tr>
<tr>
<td>6.</td>
<td><strong>Author(s):</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Title:</strong></td>
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<td></td>
<td><strong>Year:</strong></td>
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<td><strong>Reference:</strong></td>
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<td><strong>Peer Reviewed:</strong></td>
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<td></td>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
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<td></td>
<td><strong>Population studied</strong></td>
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<td></td>
<td><strong>Case identification/definition:</strong></td>
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<td></td>
<td>• <strong>Sample size:</strong></td>
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<td></td>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
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<td></td>
<td><strong>Control identification/definition:</strong></td>
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<td></td>
<td>• <strong>Sample size:</strong></td>
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<td>• <strong>Stratification (age, sex, etc.)</strong></td>
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<td></td>
<td><strong>Group selection method:</strong></td>
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<td></td>
<td><strong>Outcome(s) studied:</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
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<tr>
<td></td>
<td><strong>Exposure definition</strong></td>
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<tr>
<td></td>
<td><strong>Exposure medium</strong></td>
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<tr>
<td></td>
<td><strong>Exposure measurement:</strong></td>
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<tr>
<td></td>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Adjustments:</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Interaction assessment:</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Results:</strong></td>
</tr>
</tbody>
</table>
8.1% (14/172) had BLLs ≥10 µg/dL. One child (0.6%) BLL > 15 µg/dL. GM 5.3 µg/dL (SD 1.56).

RR, OR, CI and relevant tables with dose response etc:

Statistical analysis:

Procedures/tests:

Statistical significance: (p< 0.05)

Significant findings: Soldering hobby
Aboriginal heritage
Dwelling built before 1921
Age of water service

Non-significant findings:

Biases identified by the authors: “We do not believe that the low levels found in our study were due to differences in methods between our study and the other Canadian studies. Of the three Canadian studies, ours had the youngest subjects (aged 24 to 36 months) and the highest proportion of city dwellers (100%). These factors would lead one to expect the results to show higher blood lead levels than those found in the 1984 Ontario survey or the Canada Health Survey. Similarly, time of year cannot explain the low levels we found. We collected blood specimens between Oct. 19 and Dec. 13, 1989, a period of mid-level exposure. The 1984 Ontario survey was conducted only slightly earlier in the year, from Sept. 12 to Nov. 21. The Canada Health Survey took place over 8 months, from July to March, that covers periods of high, mid-level and low exposure. Most important, we used a finger prick blood-collection method, which could have biased our measurements upwards as a result of contamination of the finger prick blood specimens by lead on the skin.” 14, p. 1084

“Because we did not conduct any concurrent testing of the environment (e.g., soil, water, house dust, food or house paint), we can only speculate about the reasons for the low levels of blood lead that we found. There was a marked decline in the use of leaded gasoline from 1979 to 1989. Other possible explanations include Vancouver’s relative lack of heavy industry and its mild climate, heavy rainfall and lush vegetation, which may reduce dust dispersion and limit children’s contact with soil” 14, p. 1084

Assumptions/limitations of the study: Reliant on homeowner to estimate age of dwelling – this information may not be accurate leading to bias
Did not collect soil, water, house dust, food or house paint which limits their ability to explain variations in BLLs and identify risk factors

Conclusions: “The study showed no clear risk factors for elevated blood lead levels: although a few factors had a statistically significant association with increased blood lead levels, the differences in levels were small
and unimportant” 14, p. 1077

“The blood lead levels we found were much lower than those found in the Canada Health Survey, in the Ontario survey and in the United States National Health and Nutrition Examination Survey 11 (1976-80). In fact, the levels were as low as those found in some studies of blood lead levels in non-industrialized societies” 14, p. 1084

<table>
<thead>
<tr>
<th>Comments by reviewer:</th>
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<tbody>
<tr>
<td>Discussion focuses on declining average BLLs:</td>
</tr>
<tr>
<td>“Our survey results were consistent with a quiet but momentous event: the marked decline in mean blood lead levels that appears to have occurred in North America during the 1980s” 22, p.1418.</td>
</tr>
<tr>
<td>It’s relevant that authors downplay prevalence rate of 8.1% and emphasize declining average GM BLLs. Prevalence rate of 6.1% similar to U.S. rate (8.6%) for children ages 1-5 yrs during the same era (NHANES III 1988-1991 Phase I)</td>
</tr>
<tr>
<td>7.</td>
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<tr>
<td><strong>Author(s):</strong></td>
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<td><strong>Year:</strong></td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
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<tr>
<td><strong>Peer Reviewed:</strong></td>
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<tr>
<td><strong>Population studied</strong></td>
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<tr>
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<tr>
<td><strong>Control identification/definition:</strong></td>
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<tr>
<td><strong>Group selection method:</strong></td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
</tr>
</tbody>
</table>
| **Data source format (e.g., questionnaire, medical records, etc.):** | Paint chips under child’s nails  
Paint chips seen in rectal effluent  
BL  
x-ray  
Follow-up public health visit to home noticed numerous paint scrapes on walls |
| **Exposure definition** | BLL ≥10μg/dL |
| **Exposure medium** | Residential sources of lead |
| **Exposure measurement:** | BL |
| **Duration of exposure applicable to measurement (i.e., acute, chronic):** | Concurrent measure of BLL |
| **Toxicological/biological relevance of exposure measurement:** | Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water).  
First publicly identified case of lead poisoning in Canada in a child from eating “lead free” paint |
| **Adjustments:** | n/a |
| **Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing** | n/a |
| **Interaction assessment:** | Serum iron level |
| **Results:** | Paint chips found to be within “legal limit” < 5000 ppm  
| Abundant radio-opaque material throughout the large bowel  
| BLL 97 μg/dL |
| **RR, OR, CI and relevant tables with dose response etc:** | n/a |
| **Statistical analysis:** | n/a |
| **Procedures/tests:** | n/a |
| **Statistical significance:** | n/a |
| **Significant findings:** | n/a |
| **Non-significant findings:** | n/a |
| **Biases identified by the authors:** | n/a |
| **Assumptions/limitations of the study:** | “lead poisoning is now rare in Canada” 23, p.956  
| Authors do not provide supporting BL surveillance data or prevalence data to support this contention.  
| In a follow-up letter 24 clarifies that “rare” refers to cases requiring chelation therapy.  
| States incorrectly that lead-based paint >5000 was legally banned for use in interior paints in Canada in 1975. Lead was not banned from paint manufactured in Canada until 2005  
| **Conclusions:** | Advise physicians to consider lead poisoning for children with pica or those with newly recognized microcytic anemia  
| **Google Scholar cited by:** | 4, 25-28 |
| **Comments by reviewer:** | A follow-up letter to CMAJ challenges use of term “rare” to describe this problem in Canada:  
<p>| “The reality is that there exists almost no data on the extent of lead poisoning in Canada. The last national pediatric blood lead survey of Canadian children took place in 1978. The few Canadian surveys which have since been conducted have revealed elevated blood lead levels in children ranging from 2% to 11% of the population tested…Physicians have a low “index of suspicion” for lead poisoning and environmental toxin effects in general, so the problem is rarely sought out.” 27, p.429 |</p>
<table>
<thead>
<tr>
<th>8.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Leung, F. Y., Bradley, C., &amp; Pellar, T. G.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Reference intervals for blood lead and evaluation of zinc protoporphyrin as a screening test for lead toxicity.</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1993</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Clinical Biochemistry, 26, 491-496</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>yes</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Cross-sectional</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Children and adults ages “1 day” to 100 years [Ontario residents]</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td></td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>2921 blood samples</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>1898 males&lt;br&gt;1023 females&lt;br&gt;585 children (1 day – 8 yrs)&lt;br&gt;141 children (&gt;8-16 yrs)</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>4,924 blood samples (age of sample donor not identified)</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td>Ontario children observed from 1983 to 1990</td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Relationship between urban and rural children’s BLLs. There was a significant decrease of average blood lead concentration from 1983 to 1990, both in rural and urban areas of Ontario, averaging about [1 μg/dL] per year. The decline in blood lead levels in Ontario children coincided with the gradual elimination of leaded gasoline in Ontario (Wang et al., 1992, p.150).</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>Blood samples submitted to University Hospital, London Ontario (1991-1993)</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>BLL ≥10 μg/dL</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Not identified</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>Reference limit 10-15 μg/dL.</td>
</tr>
<tr>
<td>Duration of exposure applicable to measurement (i.e., acute, chronic):</td>
<td>n/a</td>
</tr>
<tr>
<td>Toxico/technological relevance of exposure measurement:</td>
<td>Blood lead samples collected following the ban of leaded gasoline usage in Canada occurring December 1990</td>
</tr>
<tr>
<td>Adjustments:</td>
<td>n/a</td>
</tr>
<tr>
<td>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</td>
<td>Whole blood lead analyzed using flameless atomic absorption spectrometry (AAS) ZPP analyzed using Protofluor-Z hematofluorometer</td>
</tr>
<tr>
<td>Interaction assessment:</td>
<td>To evaluate the utility of zinc protoporphyrin (ZPP) as an indicator of lead poisoning</td>
</tr>
<tr>
<td>Results:</td>
<td>&gt;70% adult males &amp; &gt; 80% adult females BLLs within reference limits ZPP insensitive for detection of EBLs 10-15 μg/dL range. More suited to identifying occupationally exposed males</td>
</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td>Sensitivity/specificity analysis</td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>Median and average BLLs as well as prevalence rate of children with BLLs ≥10-15 μg/dL</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td>n/a</td>
</tr>
<tr>
<td>Statistical significance:</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Significant findings:  
Non-significant findings: n/a  
Biases identified by the authors: None identified  
Assumptions/limitations of the study: “Since 1988 we have used 0.48 μmol/L [10 μg/dL] as our upper reference value for blood lead in children and women” (p. 494).  
Conclusions: Average blood lead levels were similar [6 μg/dL] to average levels identified by Wang et. al, 1992.  
Google Scholar cited by: Cited 20 times internationally, none of these citations are from Canadian sources  
Comments by reviewer: Although this study did not investigate residential sources of exposure a critique of this investigation is included in this section as it is one of the few Canadian public health discourses to identify that a portion of Ontario children continued to exhibit EBLs in the post-leaded gasoline era (post-1990) – a finding which suggests that other sources of environmental exposure continued to pose a risk. Although this study was not intended as a “prevalence” study per se, the authors’ discussion does include commentary comparing Ontario “rates” with American prevalence rates: “In children ages 0-8 years, 17.3% of our cases presented with blood leads above 10 μg/dL. This is comparable to the estimated number of American children who are at neurotoxin risk using a cut-off of 10 μg/dL, above which community and then individual intervention is needed” (Leung et al., 1993, p.494).
<table>
<thead>
<tr>
<th>9.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Author(s):</td>
<td>Levallois P, Weber JP, Gingras S, Laliberté D, Lefèvre M.</td>
</tr>
<tr>
<td>Title:</td>
<td>Lead exposure of children living in the Quebec City area.</td>
</tr>
<tr>
<td>Year:</td>
<td>1991</td>
</tr>
<tr>
<td>Reference:</td>
<td>Environmental Geochemistry and Health; 13 (Supplement) (Trace substances in environmental health XXIV).</td>
</tr>
<tr>
<td>Peer Reviewed:</td>
<td>yes</td>
</tr>
<tr>
<td>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.):</td>
<td>Cross section</td>
</tr>
<tr>
<td>Population studied</td>
<td>Children ages 1 to 6 years living in Quebec City or nearby rural area. Found ten urban children 10.3% with elevated blood lead levels compared with 2 rural children 1.3% with BLLs ≥ 10μg/dL.</td>
</tr>
</tbody>
</table>

**Case identification/definition:**

- **Sample size:** 242 children ages 1 to 6 yrs
- **Stratification (age, sex, etc.):** 93 urban 149 rural

**Control identification/definition:**

- **Sample size:** n/a
- **Stratification (age, sex, etc.)** n/a

**Group selection method:** Relationship between urban and rural BLLs

**Outcome(s) studied:** Relationship between urban and rural BLLs

**Data source format (e.g., questionnaire, medical records, etc.):** Questionnaire

- Environmental investigation of 9 urban children with BLLs >10 μg/dL
- First-draw water sample
- Paint flakes

**Exposure definition** | BLL ≥10 μg/dL
---|---
**Exposure medium** | Residential sources of lead
**Exposure measurement:** Concurrent measure of BLL

**Duration of exposure applicable to measurement (i.e., acute, chronic):**

**Toxicological/biological relevance of exposure measurement:** Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)

**Adjustments:**
| Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing | Flameless atomic absorption spectrophotometry  
External proficiency testing |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Interaction assessment:</td>
<td>Hemoglobin, ferritin and transferring saturation measured</td>
</tr>
</tbody>
</table>
| Results: | Ten children (10.8%) urban >10 μg/dL  
Two children (1.3%) rural >10 μg/dL |

“Substantial lead exposure was found for six of these children. In three cases, lead in the first draw of drinking was above 20 μg/L; also in three cases the maximum of three dust samples taken in the house (on the floor or furniture) was found above 400 μg/g. For one house with elevated dust lead concentrations, the inside paint contained 16.2% of lead”29, p. 311

<table>
<thead>
<tr>
<th>RR, OR, CI and relevant tables with dose response etc:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Statistical analysis:</td>
<td>Compare GM BLLs between urban and rural children</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td>Odds ratio (urban vs rural BLLs)</td>
</tr>
<tr>
<td>Statistical significance:</td>
<td></td>
</tr>
</tbody>
</table>

| Significant findings: | Urban GM 5.6 μg/dL (95% Confidence Interval 5-6μg/dL)  
Rural GM 4.5 μg/dL (95% Confidence Interval 4-4.8 μg/dL)  
OR=6.7 (95% C.I.: 1.3-34.8) |
| Non-significant findings: | Occupational exposure (of parents) did not account for any difference |
| Biases identified by the authors: | Urban sample older (mean age 4.2 yrs vs 3.8); higher male/female ration (1.2 vs 0.9); more prone to pica (28% vs 18%) % mouthing toys (38% vs 23%). SES differences between two groups |
| Assumptions/limitations of the study: | Compare results with decades old survey from U.S. (NHANES II 1976–1980) which found a 77% prevalence rate (BLL >10 μg/dL) in children ages 1-5 yrs. Results from NHANES III 1988-1991 Phase I were closer to Quebec study with prevalence rates of 8.6% for children 1-5 yrs.  
“In comparison to what was found in the US in 1976-1980 the mean blood lead levels in this study are more than three times lower”29, p. 311.  
Also compares results to Ontario surveys – but Ontario surveys occurred during period of peak leaded gasoline consumption (1984-1987) whereas Quebec survey occurred as leaded gasoline being phased out:  
“The observed concentrations are also half those found in the province of Ontario in 1984 but the levels of that study could have been slightly overestimated due to the use of finger-prick sampling”29, p. 311. |
| Conclusions: | Automobile exhaust and municipal incinerator fumes seem the main environmental lead sources which |
could explain the excess of elevated blood levels in the urban children. Others sources as drinking water or inside painting could also have contributed it.:  

<table>
<thead>
<tr>
<th>Google Scholar cited by:</th>
<th>4, 5, 17, 30, 31</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Canadian sources)</td>
<td></td>
</tr>
</tbody>
</table>

**Comments by reviewer:**

Lead particulate in air not sampled whereas paint chip samples and water samples were. Study conclusions, therefore, do not match findings whereby 6 of 10 cases identified with EBLs traced back to domestic sources such as lead based paint, drinking water.
| Author(s): | McDonald, L. T., Rasmussen, P. E., Chenier, M., & Levesque, C. |
| Title: | Wipe sampling methodologies to assess exposures to lead and cadmium and soil tracer yttrium in urban Canadian homes |
| Year: | 2010 |
| Peer Reviewed: | Yes |
| Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.): | Cross-sectional |
| Population studied | 3 Ontario cities: Barrie (57 homes), Greater Sudbury (86 homes), and Thunder Bay (79 homes). Part of larger nation-wide study. |
| Case identification/definition: | Random selection |
| • Sample size: | 222 homes |
| • Stratification (age, sex, etc.): | 1372 wipe samples |
| Control identification/definition: | n/a |
| • Sample size: | n/a |
| • Stratification (age, sex, etc.): | n/a |
| Group selection method: | |
| Outcome(s) studied: | To quantify metal loadings in urban homes with the aim of establishing background levels for residential exposure in Canada |
| Data source format (e.g., questionnaire, medical records, etc.): | Questionnaire Interior floor swab samples for lead and cadmium in dust |
| Exposure definition | Dust lead loading per ASTM E 1728 protocol Sample locations using Wilson et al. 52 |
| Exposure medium | ICP-MS |
| Exposure measurement: | |
| Duration of exposure applicable to measurement (i.e., acute, chronic): | Concurrent measure of BLL for participants not assessed |
| Toxicological/biological relevance of exposure measurement: | Evidence of residential (non-industrial) lead dust contamination from lead-based paint. |
| Adjustments: | |
| Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing | USEPA (2000) regulation for Pb in floor dust 40 g ft⁻² |
Interaction assessment: The purpose of the present study is to quantify metal loadings in urban homes with the aim of establishing background levels for residential exposure in Canada. As such it represents the first published dataset of its kind for Canada. The only other published Canadian study to use wipes as the sampling medium was conducted by CMHC (1995).

Results: 3/186 homes within the elevated subpopulation had wipe samples exceeding the USEPA (2000) regulation for Pb in floor dust, i.e. $40 \text{ g ft}^{-2}$.

RR, OR, CI and relevant tables with dose response etc:

Statistical analysis: Spatial relations investigated using Spearman rank correlation coefficients, 50th and 95th percentiles used to summarize Cd and Pb loadings within rooms and within homes.

Procedures/tests:

Statistical significance:

Significant findings: Elevated interior Pb dust associated with:
- Bodywork on vehicle in driveway
- Recent renovations
- Evidence of chipping flaking paint
- Recent plumbing
- Tobacco use
- Home built prior to 1960

Non-significant findings:

Biases identified by the authors:

Assumptions/limitations of the study:

Conclusions: 99% homes in this study fell below the USEPA regulation of $40 \text{ g ft}^{-2}$ for Pb in floor dust (USEPA, 2000).

8/222 homes (3.6%) sampled $\geq 12 \text{ g ft}^{-2}$ [study threshold]; 3/222 homes (1.3%) sampled $\geq 40 \text{ g ft}^{-2}$ [EPA threshold]
Mean age of the 8 “threshold” homes 1954 ± 29 years

Google Scholar cited by: (Canadian Sources)

Comments by reviewer: Reliant on homeowner to estimate age of dwelling – this information may not be accurate leading to bias.
Using a threshold of $12 \text{ g ft}^{-2}$ more protective of children. Lanphear & colleagues report floor lead loading floor (PbD) $15 \text{ g/ft}^{2}$ (OR 2.2; 95% CI1.3, 3.8) associated with BLL $10 \text{ g/dL}$.
Dixon and colleagues’ model (2009) predicts that a floor PbD 12 g/ft² results in 4.6% of children with PbB ≥ 10 g/dL and 26.8% with PbB ≥ 5 g/dL, and a GM PbB of 3.9 g/dL. ⁵⁴

Gaitens et al (2009) identified 0.16% floor samples ≥ 40 g/ft² (n = 2,065) ⁵⁵
<table>
<thead>
<tr>
<th>11.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>O’Heany J, Kusiak R, Duncan CE, Smith JF, Smith LF, Spielberg L.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Blood lead and associated risk factors in Ontario children.</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1988</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>The Science of the Total Environment; 71:477-83.</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>yes</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Cross-section</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Ontario children aged 1-6 yrs</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td></td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>1314</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>Urban, suburban and rural Ontario communities</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>To determine BLLs in Ontario children; to identify risk factors associated with EBLs</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>Capillary BLL (\geq 20) (\mu)g/dL</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Exploratory investigation to identify sources of Pb exposure</td>
</tr>
<tr>
<td><strong>Exposure measurement :</strong></td>
<td>Questionnaire (random sub-set of 800 families) Provincial air monitoring data proximal to BL survey locations &amp; traffic density Gasoline Water sampling collected from cold water kitchen faucet Standing (random) sample Flushed sample Overnight standing sample – if lead service connector present Soil sampling Collected from child’s play area i.e. rear lawn, sandbox, mud driveway, vegetable garden “Do not sample within 2 feet of roadways, buildings or fences”</td>
</tr>
<tr>
<td>Duration of exposure applicable to measurement (i.e., acute, chronic):</td>
<td>Calculate % air lead levels attributed to automobile exhaust (range 67% - 85%)</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Toxicological/biological relevance of exposure measurement:</td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water) *Ontario BLL surveys often used as surrogate indicator of national trends for Canadian children</td>
</tr>
<tr>
<td>Adjustments:</td>
<td></td>
</tr>
<tr>
<td>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</td>
<td>Atomic absorption Hitachi Zeeman Graphite Furnace spectrophotometric method External proficiency testing</td>
</tr>
<tr>
<td>Interaction assessment:</td>
<td></td>
</tr>
<tr>
<td>Results:</td>
<td></td>
</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td>Authors do not report contribution of “paint condition”, age of dwelling, or water lead to variance in BLLs.</td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>Comparison of GM BLLs urban, suburban, rural children Regression analysis</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td>Initial univariate analysis – those significant at 10% level entered into regression analysis to identify risk factors, step-wise regression analysis</td>
</tr>
<tr>
<td>Statistical significance:</td>
<td>(p &lt; 0.001)</td>
</tr>
<tr>
<td>Significant findings:</td>
<td>Urban children had higher geometric mean blood lead levels than suburban children who in turn had higher blood lead levels than rural children (p &lt; 0.001). BLLs higher in males than females, younger children (&lt; 3 yrs) Significant risk factors for housing identified as: Condition of paint in child’s room (p=0.01); condition of paint in child’s favourite play area (p= 0.003)</td>
</tr>
<tr>
<td>Non-significant findings:</td>
<td>No difference among sites (urban, suburban, rural) for proportion of children with BLLs ≥ 20 µg/dL</td>
</tr>
<tr>
<td>Biases identified by the authors:</td>
<td></td>
</tr>
<tr>
<td>Assumptions/limitations of the study:</td>
<td>Age of housing included in the regression analysis but not identified by authors as statistically significant. Alder et. al notes that this may be due to fact that Ontario study did not derive age of dwelling from municipal property assessment data – which may have led to bias in the results: “The age of residence in these [Ontario] studies was obtained from questionnaire respondents, and therefore would be less accurate than the data from the present study which are based on the year of construction as documented in the local tax assessment file”(^1) p.312</td>
</tr>
</tbody>
</table>
**Conclusions:**
Urban children had higher geometric mean blood lead levels than suburban children who in turn had higher blood lead levels than rural children. Lead gasoline additives the most significant single environmental determinant of children’s blood lead levels.

**Google Scholar cited by:**
(Canadian sources) 4, 14, 17-19, 32-35

**Comments by reviewer:**
Authors concluded that lead gasoline additives were “the most significant single environmental determinant of children’s blood lead levels” even though this variable accounted for only 19% of the variation in BLLs. Not well explained how variable “gasoline” measured. Authors do not report contribution of “paint condition” age of dwelling or water lead to variance in BLLs.
<table>
<thead>
<tr>
<th>12.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Poon HC, Carson R, Peter F, Goldberg B, Haust HL</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>The Lead Program at CPRI.</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1989</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>yes</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>11 year randomized blood survey</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Children and adolescents admitted to a Children’s Psychiatric Research Institute (CPRI), London Ontario</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td></td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>4188</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>Age of subjects not identified except for the one case</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Variables: Identify upper reference BL level</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>BL</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>BLLs for children upon admission, discharge and children visiting as outpatients</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Residential sources of lead</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>Concurrent measure of BLL</td>
</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
</tr>
<tr>
<td><strong>Adjustments:</strong></td>
<td>Exclusion of “discharge” group to eliminate repeat measures of same subject</td>
</tr>
<tr>
<td><strong>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</strong></td>
<td>Anodic stripping voltammeter until 1983 Zeeman graphite furnace atomic absorption spectrophotometer 1983-1987 AnP Model 4000 hematofluorometer Precision within 5%</td>
</tr>
<tr>
<td><strong>Interaction assessment:</strong></td>
<td>Erythrocyte protoporphyrin determined as zinc protoporphyrin</td>
</tr>
<tr>
<td>Results:</td>
<td>Upper reference BL level 18 µg/dl (95% confidence level)</td>
</tr>
<tr>
<td>-------------------</td>
<td>----------------------------------------------------------</td>
</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td></td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>Yearly mean BLLs</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td></td>
</tr>
<tr>
<td>Statistical significance:</td>
<td></td>
</tr>
</tbody>
</table>

| Significant findings: | No significant difference between admission, discharge and outpatient mean BLL or EP |
| Non-significant findings: |                                                                                      |
| Biases identified by the authors: |                                                                                     |
| Assumptions/limitations of the study: | Prevalence rate BLL ≥ 20µg/dL 14/1000 compared to 25/1000 for CPRI survey. Sample size 4x larger, longer study period, mixed rural/urban population. |
| Conclusions: | No recommendations made for further study other than to lower laboratory blood lead reference value from 30 to 18 µg/dL. |
| Google Scholar cited by: | 2, 4-6, 36 (Canadian Sources) |

| Comments by reviewer: | Survey extended from January 1977 to August 1987. During 1978-79, mean BLLs increased then fell to 1977 levels. There is no explanation provided for the increase. This writer notes that an increase may have been related to abatement work carried on at the facility during this time. For example, the authors note that "walls [were] stripped bare and repainted with Pb-free paint and toys and other items [were] replaced with Pb-free counterparts" 37, p.213 |


<table>
<thead>
<tr>
<th>13.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Rasmussen PE, Subramanian KS, Jessiman BJ., Canada.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>A multi-element profile of housedust in relation to exterior dust and soils in the city of Ottawa</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>2001</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Science of the Total Environment; 267(1-3):125-40</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Cross-sectional</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Ottawa (non-industrial community)</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td>Median construction age 1953 (range 1893 to 1997)</td>
</tr>
<tr>
<td><strong>Sample size:</strong></td>
<td>50 homes</td>
</tr>
<tr>
<td><strong>Stratification (age, sex, etc.):</strong></td>
<td>10 locations</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Stratification (age, sex, etc.)</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td>Random</td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Multi-element profile of street, soil, interior (domestic) dust</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>Questionnaire given to home occupants Lead concentrations (vacuuming method – no sampling protocol referenced)</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>Exploratory investigation to determine normal background levels of metals in interior dust/soil</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Residential sources of lead and other metals analysed (i.e. mercury &amp; arsenic)</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td>Concurrent measure of BLL not assessed for participants Environmental samples collected in 1993</td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of residential (non-industrial) lead dust contamination from lead-based paint.</td>
</tr>
<tr>
<td><strong>Adjustments:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</strong></td>
<td>ICP-MS</td>
</tr>
<tr>
<td><strong>Results:</strong></td>
<td>Interior dust Pb levels (Range50.20 - 3225.66 μg/g); arithmetic mean 405.56 μg/g.</td>
</tr>
<tr>
<td><strong>RR, OR, CI and relevant tables with dose response etc:</strong></td>
<td>Information on renovation history collected (questionnaire) but contribution of this variable to variance in interior dust lead levels not reported in findings</td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>Regression analysis</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td></td>
</tr>
<tr>
<td>Statistical significance:</td>
<td></td>
</tr>
<tr>
<td>Significant findings:</td>
<td>Housedust samples contain significantly higher concentrations of many elements, including lead, cadmium, antimony and mercury, than either street dust or garden soil. Sample concentrations of lead in housedust samples exceeded street dust lead concentrations in all 48 residences, and exceeded garden soil lead concentrations in all but one residence. In general, higher lead concentrations identified in dust samples obtained from older housing stock.</td>
</tr>
<tr>
<td>Non-significant findings:</td>
<td></td>
</tr>
<tr>
<td>Biases identified by the authors:</td>
<td>None</td>
</tr>
<tr>
<td>Assumptions/limitations of the study:</td>
<td></td>
</tr>
<tr>
<td>Conclusions:</td>
<td>“a city such as Ottawa with few industrial sources, a significant reduction in childhood exposure to lead and other elements of concern, such as mercury, cadmium and antimony, will not be accomplished through continued lowering of exterior soil cleanup criteria and guidelines, but through increased attention to indoor sources of exposure, and improved parental attention to personal hygiene and housekeeping practices” (p. 139)</td>
</tr>
<tr>
<td>Google Scholar cited by: (Canadian Sources)</td>
<td>Cited by 74 (international &amp; Cdn) 4, 38-44</td>
</tr>
<tr>
<td>Comments by reviewer:</td>
<td>Homes screened for various metals including Hg, As, Pb. Interior dust Pb range 50.20 - 3225.66 μg/g ,with an arithmetic mean 405.56 μg/g. While there are no Canadian standards for lead in interior dust, lead levels in at least half the samples exceeded guidelines provided by the Ministry of the [Ontario] Environment and the Canadian Council of Ministers of the Environment (200 μg/g lead and 140 μg/g lead for residential soil) respectively. No follow up blood lead survey of residents living within these homes; however, using the Environmental Lead to Blood-lead Slope Factors, developed by the U.S. (ATSDR,1999) and assuming that lead dust contributes 0.008 μg/dL in blood per μg of lead/g dust for children 1-6 years, a child living in a home with lead dust levels of 405.56 μg/g would have a corresponding BLL of 3.2 μg/dL from this source alone. At dust lead levels found in the 95th percentile (1,311.92 μg/g) a child age 6 years or under would have a BLL of 10 μg/dL; and a child living in the most contaminated home, with dust lead levels of 3,225.66 μg/g could conceivably have a BLL of 25 μg/dL.</td>
</tr>
<tr>
<td>14.</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Title:</td>
<td>Case report: A metabolic disorder presenting as pediatric manganism.</td>
</tr>
<tr>
<td>Year:</td>
<td>2007</td>
</tr>
<tr>
<td>Reference:</td>
<td>Environmental Health Perspectives; 115(12).</td>
</tr>
<tr>
<td>Peer Reviewed:</td>
<td></td>
</tr>
<tr>
<td>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.):</td>
<td>Case study</td>
</tr>
<tr>
<td>6 yr old child with symptoms (case)</td>
<td></td>
</tr>
<tr>
<td>7 yr old sibling (asymptomatic)</td>
<td></td>
</tr>
<tr>
<td>Immediate family and five extended family members (maternal grandparents, paternal grandfather, and the father’s two siblings in lieu of the deceased paternal grandmother) were tested for Mn</td>
<td></td>
</tr>
<tr>
<td>Population studied</td>
<td>New Brunswick</td>
</tr>
<tr>
<td>Case identification/definition:</td>
<td>“In August 2004, a previously healthy 6-year-old female presented with pica and emotional lability. Over the following months she developed progressive behavioral and neurologic symptoms: She became withdrawn and less verbal with repetitive stuttered speech, and her balance, co-ordination, and fine motor skills declined. By November, she could no longer stand independently, tended to fall backward, and developed a high steppage “cock-like” gait.”p. 1776</td>
</tr>
<tr>
<td>• Sample size:</td>
<td>n/a</td>
</tr>
<tr>
<td>• Stratification (age, sex, etc.):</td>
<td>n/a</td>
</tr>
<tr>
<td>Control identification/definition:</td>
<td>n/a</td>
</tr>
<tr>
<td>• Sample size:</td>
<td>n/a</td>
</tr>
<tr>
<td>• Stratification (age, sex, etc.)</td>
<td>n/a</td>
</tr>
<tr>
<td>Group selection method:</td>
<td>Immediate family</td>
</tr>
<tr>
<td>Outcome(s) studied:</td>
<td>Manganese poisoning, source of exposure, genetic predisposition to Mn poisoning</td>
</tr>
<tr>
<td>Data source format (e.g., questionnaire, medical records, etc.):</td>
<td>Questionnaire</td>
</tr>
<tr>
<td>Diet history (Mn)</td>
<td></td>
</tr>
<tr>
<td>Water (Mn)</td>
<td></td>
</tr>
<tr>
<td>Exposure definition</td>
<td></td>
</tr>
<tr>
<td>Exposure medium</td>
<td>Manganese</td>
</tr>
<tr>
<td>Exposure measurement:</td>
<td>Blood Mn Levels</td>
</tr>
<tr>
<td>Blood Pb levels (case only)</td>
<td></td>
</tr>
<tr>
<td>Duration of exposure applicable to measurement (i.e., acute, chronic):</td>
<td>Seasonal (visits to cottage)</td>
</tr>
<tr>
<td>Toxicological/biological relevance of exposure measurement:</td>
<td>Evidence of lead poisoning occurring in 6 yr old case, although environmental sources of Pb not investigated.</td>
</tr>
</tbody>
</table>
**Adjustments:**

<table>
<thead>
<tr>
<th>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</th>
<th>ICP MS (blood analysis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interaction assessment:</td>
<td>Serum ferritin</td>
</tr>
</tbody>
</table>

**Results:**

| Elevated plasma Mn ranging from 1.9 to 2.8 g/L (normal, 0.3–1.0 g/L) for everyone except case’s sister; normal (0.6 g/L) in March but elevated (2.3 g/L) when retested in May. BLL of 6 yr old (2.9 g/dL; 4.6 g/dL) in October 2004 but elevated (17.6 g/dL) in early August 2005. |

**RR, OR, CI and relevant tables with dose response etc:**

| Case: Mn in whole blood (39.7 g/L; normal, 4.3–15.9 g/L) |

<table>
<thead>
<tr>
<th>Statistical analysis:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedures/tests:</td>
</tr>
<tr>
<td>Statistical significance:</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Significant findings:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-significant findings:</td>
</tr>
<tr>
<td>Biases identified by the authors:</td>
</tr>
<tr>
<td>Assumptions/limitations of the study:</td>
</tr>
<tr>
<td>There currently is no “gold standard” biomarker for Mn exposure. Used plasma and RBC Mn as indicators. Note these are effective tools in measuring exposure differences between populations, but they are less reliable on an individual level. Variability in Mn blood levels: 3 consecutive blood samples for plasma Mn taken from the case on a single day showed Mn 0.6, 2.2, and 2.4 g/L. Comparing measurements between individuals may be subject to the same variability. A lack of appropriate population reference values. Reference ranges derived from population norms in southern Ontario. Absence of available reference levels for plasma and RBC Mn for populations exposed to higher natural background levels (e.g., through drinking water) makes the interpretation of such results difficult. The seemingly elevated plasma Mn levels among the patient’s family members may well be within a “normal” range.</td>
</tr>
</tbody>
</table>

| Conclusions: |
| The patient’s symptoms, biomonitoring results, and environmental assessment were consistent with a chronic, seasonal exposure pattern. |

<table>
<thead>
<tr>
<th>Google Scholar cited by: (Canadian Sources)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n/a</td>
</tr>
</tbody>
</table>

| Comments by reviewer: |
| Although this is a case study of manganese poisoning this case study reports a child with BLLs 17.6 µg/dL and “severe Fe deficiency”. Intriguingly, environmental sources of Pb exposure not |
investigated and BLL not investigated in sibling (at least not reported). Relevance of Pb exposure as contributing factor to neurological symptoms not discussed, does not appear to have been considered. This is curious as co-researcher and supervisor Scott Giffin involved in New Brunswick investigation of residential sources of lead.

The ICP/MS method has the capacity to measure lead levels at the parts per billion detection level.
<table>
<thead>
<tr>
<th>15.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Tenenbein M.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Does lead poisoning occur in Canadian children?</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1990</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>CMAJ;142(1):40-1.</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>Letter – to peer reviewed journal</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Commentary – describes two cases</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Winnipeg location</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td>BLL ( \geq 10 \mu g/dL )</td>
</tr>
<tr>
<td></td>
<td><strong>Sample size:</strong> 2 children (female, male)</td>
</tr>
<tr>
<td></td>
<td><strong>Stratification (age, sex, etc.):</strong></td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td><strong>Sample size:</strong> n/a</td>
</tr>
<tr>
<td></td>
<td><strong>Stratification (age, sex, etc.)</strong> n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td></td>
</tr>
</tbody>
</table>
| **Data source format (e.g., questionnaire, medical records, etc.):** | Clinical assessment (female case)  
Review of hospital record (male case) |
| **Exposure definition** | |
| **Exposure medium** | Residential sources of lead |
| **Exposure measurement:** | Concurrent measure of BLL |
| **Duration of exposure applicable to measurement (i.e., acute, chronic):** | |
| **Toxicological/biological relevance of exposure measurement:** | Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water) |
| **Adjustments:** | |
| **Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing** | |
| **Interaction assessment:** | Free erythrocyte protoporphyrin, iron deficiency, hemoglobin, basophilic stippling in blood smear, urine lead post chelation, lead lines in long bones (x-ray), developmental delays, psychological assessment |
| **Results:** | Case 1  
A 32-month-old girl with a “pigeon-toed gait” and a “17-month history of a voracious appetite for the
paint peeling from the walls and window sills of her inner city home” [p. 40] BLLs assessed two weeks later were 83.9 µg/dL.
“After inspecting the original residence city health officials found that samples of chipped plaster and peeling paint contained up to 122 000 ppm of lead (upper limit of acceptability 5000 ppm in Canada” (p. 40)

Case 2
A 26-month-old boy comatose after experiencing a generalized seizure. X-ray films showed lead lines in the long bones and numerous radiopaque densities in the abdomen. Basophilic stippling was found in his blood smear. Lead encephalopathy was diagnosed (p. 41).
“The sequelae of the encephalopathy have included moderate mental retardation and a chronic seizure disorder requiring therapy with multiple anticonvulsants. During the boy's hospital stay a history of pica for peeling paint was discovered. Analysis of paint chips from his home revealed a lead concentration of 25 000 ppm. The city health department issued a local improvement order” [p. 41]

<p>| RR, OR, CI and relevant tables with dose response etc: | n/a |
| Statistical analysis: | n/a |
| Procedures/tests: | n/a |
| Statistical significance: | n/a |
| Significant findings: | n/a |
| Non-significant findings: | n/a |
| Biases identified by the authors: | None |
| Assumptions/limitations of the study: | |
| Conclusions: | “The prevalence of neurotoxic lead levels in asymptomatic children is unknown in Canada. Sound epidemiologic studies of blood lead concentrations involving children living in Canadian inner city areas are needed to define the extent of lead poisoning from lead-based paint” p. 41 |
| Google Scholar cited by: (Canadian Sources) | Cited by 12 (international and Cdn) 4, 17, 31, 33, 36, 46, 47 |
| Comments by reviewer: | Unable to identify any other articles or research on lead by Tenenbein except for a scathing review of a book describing heavy metal poisoning 48 |</p>
<table>
<thead>
<tr>
<th>16.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author(s):</strong></td>
<td>Valiquette L, Kosatsky T.</td>
</tr>
<tr>
<td><strong>Title:</strong></td>
<td>Portrait of Montreal children with high blood lead levels identified through community-wide review of laboratory records.</td>
</tr>
<tr>
<td><strong>Year:</strong></td>
<td>1995</td>
</tr>
<tr>
<td><strong>Reference:</strong></td>
<td>Chronic Diseases in Canada;16(2).</td>
</tr>
<tr>
<td><strong>Peer Reviewed:</strong></td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Type of study (meta-analysis, cohort, case-control, cross-sectional, etc.)</strong></td>
<td>Retrospective archival analysis of hospital records</td>
</tr>
<tr>
<td><strong>Population studied</strong></td>
<td>Montreal</td>
</tr>
<tr>
<td><strong>Case identification/definition:</strong></td>
<td>Children &lt;15 yrs</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>12 cases identified</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Control identification/definition:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Sample size:</strong></td>
<td>n/a</td>
</tr>
<tr>
<td>• <strong>Stratification (age, sex, etc.):</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Group selection method:</strong></td>
<td>125 patients identified from hospital logs with BLL ≥ 25µg/dL. We were able to obtain medical record for 100 (80%) Case selected based on age(&lt; 15 yrs) and BLL (≥ 25µg/dL)</td>
</tr>
<tr>
<td><strong>Outcome(s) studied:</strong></td>
<td>Identify environmental source of exposure, audit case management/investigation, follow-up, reporting</td>
</tr>
<tr>
<td><strong>Data source format (e.g., questionnaire, medical records, etc.):</strong></td>
<td>Hospital log records for years (1981-1985, 1986-1989) Quebec provincial toxicological laboratory database (1987-1989)</td>
</tr>
<tr>
<td><strong>Exposure definition</strong></td>
<td>BLL ≥ 25µg/dL</td>
</tr>
<tr>
<td><strong>Exposure medium</strong></td>
<td>Environmental sources of lead exposure</td>
</tr>
<tr>
<td><strong>Exposure measurement:</strong></td>
<td>Blood lead</td>
</tr>
<tr>
<td><strong>Duration of exposure applicable to measurement (i.e., acute, chronic):</strong></td>
<td>n/a</td>
</tr>
<tr>
<td><strong>Toxicological/biological relevance of exposure measurement:</strong></td>
<td>Evidence of lead poisoning occurring from residential source (i.e. lead-based paint, drinking water)</td>
</tr>
<tr>
<td><strong>Adjustments:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Laboratory analysis: (i.e. AAS, GFAAS, ICP/MS), proficiency testing</strong></td>
<td>Not specified</td>
</tr>
<tr>
<td>Interaction assessment:</td>
<td>No</td>
</tr>
<tr>
<td>-------------------------</td>
<td>----</td>
</tr>
<tr>
<td>Results:</td>
<td>BLL Range: 27-94 g/dL</td>
</tr>
<tr>
<td>RR, OR, CI and relevant tables with dose response etc:</td>
<td>n/a</td>
</tr>
<tr>
<td>Statistical analysis:</td>
<td>n/a</td>
</tr>
<tr>
<td>Procedures/tests:</td>
<td>n/a</td>
</tr>
<tr>
<td>Statistical significance:</td>
<td>n/a</td>
</tr>
<tr>
<td>Significant findings:</td>
<td>Paint was the principal source of exposure identified. Lead poisoning was the direct cause of hospitalization for three children, two received chelation therapy</td>
</tr>
<tr>
<td>Non-significant findings:</td>
<td>n/a</td>
</tr>
<tr>
<td>Biases identified by the authors:</td>
<td>None</td>
</tr>
<tr>
<td>Assumptions/limitations of the study:</td>
<td></td>
</tr>
<tr>
<td>Conclusions:</td>
<td>BL screening triggered by symptoms of anemia (10 cases), neurological or behavioural problems (3 cases), abdominal pain (3 cases), vomiting (3 cases). Paint identified as principal source of exposure in majority of these cases, convulsions (1 case). Clinical follow-up to identify source of exposure in 8 cases, environmental sampling performed in 3 cases confirmed presence of lead in paint fragments. One case poisoned in utero (maternal ingestion of Asian folk remedy). Sibling/family follow-up occurred for 3 families resulting in identification of new case. Only 2 cases reported to community health department.</td>
</tr>
<tr>
<td>Google Scholar cited by:</td>
<td>4, 18, 49, 50</td>
</tr>
<tr>
<td>(Canadian Sources)</td>
<td></td>
</tr>
<tr>
<td>Comments by reviewer:</td>
<td>Quebec only province in Canada that requires mandatory reporting of EBLs. Legislation in place since 2003, but only started systematically collecting BLL data in 2006</td>
</tr>
</tbody>
</table>
APPENDIX B

“Applying Dixon and Dixon’s Integrative Model for Environmental Health Research towards a critical analysis of childhood lead poisoning”
Applying Dixon and Dixon’s Integrative Model for Environmental Health Research Toward a Critical Analysis of Childhood Lead Poisoning in Canada

Amélie Perron, PhD, RN; Kelly A. O’Grady, BScN

Occurrences of childhood lead poisoning resulting from exposure to residential sources of lead is an underresearched area in Canada. Dixon and Dixon’s Integrative Model for Environmental Health Research substantiates this claim by grouping Canadian research on this health topic into the model’s 4 domains: physiological, vulnerability, epistemological, and health protection. This process is useful not only for identifying research gaps within the Canadian context but also in setting the groundwork for a future critical analysis to illuminate the sociopolitical and economic influences that shape healthcare knowledge, and ultimately, influence how healthcare providers and policy makers produce and use this information.

Key words: Canada, child health, critical analysis, English, environmental health, environmental toxicology, health screening, housing, infancy and childhood, lead poisoning, nursing theories, prevention and control, risk factors

Environmental illnesses such as childhood lead poisoning, asthma, cancer, and neurobehavioral disorders cost United States taxpayers $54.9 billion (US) annually.1 Similarly, in Canada, the price of “environmental disease” is estimated to cost more than $30 billion (CAN) per year.2 Lead is a serious environmental toxin, with the capacity to interrupt brain growth and impair functioning in developing fetuses and children. Twelve million children in developing countries suffer from some form of permanent brain damage due to lead poisoning and, about 3.5% of minor mental retardation is attributable to lead poisoning worldwide.3 Affected children can suffer lower IQ scores, poorer school performance, and negative behavioral effects such as increased aggression and later criminal behavior.4

Since at least 1930, numerous case studies in American, Canadian, and Australian literature have identified lead-based paint in the home as a source of poisoning for young children5; and since at least 1990, evidence has shown that it is the lead dust from deteriorating paint in older homes and renovating activities that is the primary source of chronic exposure for young children today.6 All lead-painted surfaces deteriorate with time, creating lead dust.7 Lead dust found on floors and surfaces is the most common pathway of exposure for young children who, as toddlers, explore their environment through hand-to-mouth behavior.4 Oftentimes one or two
dwellings are responsible for multiple cases of lead poisoning as affected families move out and new, unsuspecting families move in to lead-contaminated homes.8

The United States has been regularly collecting information nationally on children’s blood lead levels since 1976. Following the phaseout of leaded gasoline, blood lead levels of the US population aged 1 to 74 years dropped 78% between 1976 and 1991.9 The most recent surveillance data estimate that 1.4% of US children aged 1 to 5 years continue to be affected by lead.10 Exposure to lead paint and dust account for 70% of reported cases.11

Canadians witnessed a similar decline in children’s blood lead levels following the phasing out of lead in gasoline, new paint, and solder; but unlike the United States, surveillance in this country has been sporadic.12 In November 2008, Health Canada released the preliminary findings from the Canadian Health Measures Survey,13 a national investigation analyzing samples of blood from more than 2000 Canadians, measuring concentrations of lead, cadmium, and mercury—three metals that can deleteriously affect human health, brain development, and overall functioning. Findings from the survey’s first phase indicate that mean blood lead levels of Canadians aged 6 to 79 years have declined considerably in the 30-year span since the last nationwide survey of blood lead levels in Canada, conducted in 1978.13 The Daily, reporting on the survey’s findings, declares that “[f]ewer than 1% of Canadians now have blood lead concentrations above the Health Canada guidance value of 10 μg/dL.” These findings, at first glance, suggest a public health victory and appear to justify Canada’s phlegmatic response to childhood lead poisoning, which to date has focused primarily on lead in consumer products. Is there evidence to support the thesis that childhood lead poisoning related to housing sources exists in Canada? For this special Advances in Nursing Science (ANS) issue, we explore this question by using Dixon and Dixon’s Integrative Model for Environmental Health Research (IMEHR)14 to inform a literature review with a specific focus on Canadian data sources. And as Dixon and Dixon intended their model as a “working hypothesis that may be useful in guiding investigations or suggesting needed policies,”14(p44) we test the model’s utility in facilitating the detection of gaps in Canadian research and policy on the topic of childhood lead poisoning related to housing sources.

THE DIXON AND DIXON MODEL

Starting with the first international conference of health promotion, the Ottawa Health Charter15 with its explicit recognition of a stable ecosystem, sustainable resources, and social justice as basic prerequisites for improving health, and later the Jakarta Declaration, which affirmed a commitment to social justice, equity, and sustainability,16 there has been a refreshing shift in the way health is viewed, moving the focus away from the individual and toward the social, economic, and physical environmental contexts in which people live. Today, a number of ecological models consider a wide range of determinants of health that include physical and social environmental factors and that also highlight awareness of persistent health disparities. The Community as Partner Model, developed by Anderson and McFarlane,17 for example, recognizes the influence of 8 subsystems on health—physical environment, education, safety and transportation, politics and government, health and social services, communication, economics, and recreation—that can be applied to diverse community health problems of health, sociopolitical, and environmental origin. Hancock and Perkins’ Mandala of Health model understands human ecology as the intersection between culture and the natural environment, with a focus on equity and sustainability.18 Lavery and colleagues’ Community Action Model19 provides communities with a step-by-step framework to investigate and solve health disparities through environmental change. The US Department of Health and Human Services’ Healthy People
2010 strategy establishes the domains of behavior, biology, physical and social environment, and individual factors as mediating factors in its Determinants of Health Model; and the Healthy People 2020 “overarching goals” includes an action piece on policy, programs, and health communication. Falk-Raphael’s Model of Empowered Caring promotes the use of knowledge development to empower people to action on behalf of health, social justice, equity, and lasting change, including policy; and Giddings’ Theoretical Model of Social Consciousness is designed to reform social injustices underlying health disparities through social critique and active resistance.

In an earlier issue of ANS, Jane and John Dixon presented their IMEHR, developed to assist communities and individuals cope with exposure to environmental hazards. The model was built, in part, out of frustration by the fact that there was a preponderance of information on contaminants and their physiological impact on human health but little research on how the public could access that information and use it to ameliorate their situation. Dixon and Dixon deduced their model from the growing body of research providing evidence that environmental agents such as lead, mercury, pesticides, and other contaminants can cause “premature mortality, morbidity, functional limitations ... and affect the quantity and quality of life.”

Similar to models mentioned earlier, the Dixon and Dixon model promotes an ecological perspective, recognizing a broad array of “environmental” influences on health, such as political, economic, sociocultural and biological factors, and encourages an interdisciplinary approach to problem solving. Like Anderson and McFarlane’s approach, the primary focus of the Dixon and Dixon model is on outcome—the control or elimination of environmental health hazards, although the route or means to achieving this outcome is not necessarily restricted to action on the part of the healthcare professional. What distinguishes the Dixon and Dixon model from other ecological models is the inclusion of environmental factors as major determinants of health, with an emphasis on the assessment of multiple chemical exposures as opposed to reviewing the effects of just one chemical at a time.

The model explicitly assumes that:
(a) prevention, or “thinking upstream” can eliminate the precursors of illness;
(b) agents are potential causes of disease and can include infectious microbes, chemical, and physical agents, the accumulation of which causes health hazards;
(c) an agent is a health risk only if there is a potential route for exposure to occur;
(d) risks to health due to environmental health hazards are not distributed equally among populations; and
(e) change is required at the policy level as well as at the individual level.

The IMEHR views phenomena through 4 domains: the physiological domain, the vulnerability domain, the epistemological domain, and the health protection domain. The physiological domain is explored primarily through a review of toxicological and epidemiological studies; the vulnerability domain encompasses the “broad array of individual and community characteristics” that lead to variations in risk and health disparities; the epistemological domain concerns “processes of personal thought and social knowledge” by which people make judgments on how to respond; and the health protection domain encompasses the actions that people take to reduce their risk from exposure to environmental agents. Authors Dixon and Dixon did not intend for researchers to study all domains simultaneously as each on its own is a “large area to cover.” They recommend that researchers focus on one or two domains, but they also note how researchers tend to gravitate toward the more “objective” physiological and vulnerability domains and neglect the “softer” but equally important exploration of
Figure 1. Dixon and Dixon’s Integrative Model for Environmental Health Research.

the epistemological and health protection domains (how we know, what we know, and what we should do about it). A schematic of the relationships among the domains in the Dixon and Dixon model is provided in Figure 1. The domains are “closely interrelated, such that changes in any one domain lead to changes in other domains.” 14(p44) The model does allow for overlap although each domain stresses “a different lens by which a complex, multifaceted problem may be examined.” 14(p44)

Given its emphasis on environmental agents and primary prevention, the Dixon and Dixon model is appropriate for the exploration of childhood lead poisoning in Canada. The primary purpose of this article is to examine the usefulness of the IMEHR in guiding environmental investigations and policy changes.

LITERATURE REVIEW

Dixon and Dixon’s IMEHR was used to structure a literature review on the topic of childhood lead poisoning in Canada. The purpose of the review was to identify (1) research conducted to date on the topic of childhood lead poisoning (research falling within the rubric of the physiological and vulnerability domains), (2) evidence to support the thesis that childhood lead poisoning due to residential sources of exposure exists in Canada (epistemological domain), and (3) measures taken within Canada to remediate the problem (health protection domain). A primary focus of this review is on the epistemological domain: studies that inform Canadians that a problem with lead in housing exists in Canada.

Method

Seven databases were searched for relevant titles in this order: Ovid MEDLINE, CINAHL, HealthSTAR, Embase, PsychInfo, Environmental Sciences and Pollution Management, and GreenFILE. Four concepts were used to conduct the search: (1) lead poisoning; (2) child(ren); (3) housing, residential sources of exposure; and (4) Canada. Appropriate search terms for each database were identified through mapping. The terms “Canada” and “Canadian,” “Ontario,” “Quebec,” “New Brunswick,” “Newfoundland,” “Nova Scotia,” “Prince Edward Island,” “Manitoba,” “Saskatchewan,” “Alberta,” “British Columbia,” “Yukon,” “Northwest Territories,” and “Nunavut” were added as text words. No search limits were applied. Reference lists of retrieved articles were scanned for additional relevant titles. Hand searches were completed for relevant titles from dissertation abstracts, books, and peer-reviewed journals (eg, the Canadian Nurse, the Canadian Medical Association Journal, and the Canadian Public Health Association Journal). Reports such as Health Canada’s 1994 and 2005 “Update of Evidence for Low-Level Effects of Lead and
Blood Lead Intervention Levels and Strategies” were scanned to identify key authors. Several Web sites were searched for relevant publications: Health Canada, Canada Mortgage and Housing Corporation (CMHC), the Ontario Ministry of Health and Long-Term Care, the Canadian Nurses Association, the Canadian Medical Association, the Canadian Pediatric Society, and the Canadian Partnership for Children’s Health and Environment. Retrieved articles spanned the years from 1932 to 2008. Duplicate abstracts were excluded from the final set, leaving a total of 136 titles identified as relevant.

In phase I, abstracts of the 136 titles were further screened for relevance, using the following inclusion criteria: (a) the survey or case study related to childhood lead poisoning within the Canadian context, (b) vulnerable populations (children ages prenatal to 6 years) were included as participants in the study and findings were disseminated by age group, (c) researchers examined the issue in relation to residential sources of lead exposure, (d) blood lead levels of participants or cases were assessed, and (e) researchers collected at least one of the following environmental media to identify sources of exposure: paint chip samples, interior lead dust samples, and water samples. Grey literature such as unpublished blood lead studies, case studies reported in the media, and personal communications (ie, unpublished community blood lead surveys) were included in the final selection if they met the criteria. Occupational exposure studies, animal studies, and blood lead surveys conducted in primary or secondary smelter communities were excluded, leaving a total of 24 articles.

The 24 articles included in the final screen were ranked by using a 2-point scale (1 = meets all of the inclusion criteria; 2 = meets some of the inclusion criteria, ie, measured residential sources of lead in housing but did not record or collect children’s blood lead information). The final analysis resulted in the identification of only 4 studies in a 77-year span that examined childhood lead poisoning in relation to housing sources in Canada and which also collected environmental data such as paint chip samples, interior lead dust, and water samples to track exposure sources. Further discussion on findings from the literature search is discussed under the heading “Epistemological Domain.” Table 1 summarizes the 24 clinical, environmental, and case studies pertaining to the issue of childhood lead poisoning in Canada.

In phase II, findings from the literature review were further grouped into the 3 remaining domains (physiological, vulnerability, and health protection), with a particular focus on Canadian data within the health protection domain. In this review, policy documents, position papers, systematic reviews, legislation, and guidelines were included if they pertained to federal jurisdictions. This grouping aids in the identification of process gaps—areas where problems have been identified through case studies or community surveys (epistemological domain) but where follow-up action such as primary or secondary prevention is not evident (health protection domain).

RESULTS

1. Physiological domain. Research on the topic of the physiological effects of lead poisoning has been conducted both nationally and internationally. A complete listing of research on lead within this domain would be unfeasible as lead is one of the most widely studied environmental health topics worldwide. Information regarding the chemical and physiological processes through which lead affects the environment and health has been thoroughly synthesized in various American publications such as the Draft Toxicological Profile for Lead from the Agency for Toxic Substances and Disease Registry and Air Quality Criteria for Lead from the US Environmental Protection Agency. The International Agency for Research on Cancer
Table 1. Epistemological domain: Canadian blood lead surveys, case studies and environmental studies related to housing as a source of Pb exposure

<table>
<thead>
<tr>
<th>Author/Location</th>
<th>Rank</th>
<th>Environmental media collected from residential sources</th>
<th>% EBL or #cases &amp; age range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alder et al(^{61}) London, Ontario</td>
<td>1</td>
<td>Paint, dust, first draw cold tap water Municipal tax assessment data used to determine the age of housing, blood lead data obtained from Children’s Psychiatric Research Institute medical charts between 1984 &amp; 1989. (See Poon et al(^{62}))</td>
<td>Case study: 6-y male (49.7 μg/dL); 2-y female (39.3 μg/dL); 2-y female (11.4 μg/dL) &amp; 1-y male (15.5 μg/dL).</td>
</tr>
<tr>
<td>Balram &amp; Giffin, 1993 (unpublished) St John, New Brunswick</td>
<td>1</td>
<td>Paint, paint dust, tap water, soil, household food, blood lead levels.</td>
<td>11.3% ≥ 3 y</td>
</tr>
<tr>
<td>Lavoie &amp; Bailey(^{41}) Montreal, Quebec, Canada</td>
<td>1</td>
<td>Paint chips, blood lead levels</td>
<td>Case study: Male 4-y male (97 μg/dL)</td>
</tr>
<tr>
<td>Levallois et al(^{39}) Quebec City, Quebec, Canada</td>
<td>1</td>
<td>Household dust samples, paint flakes, tap water, blood lead levels</td>
<td>10.8% urban population; 1.3% rural population</td>
</tr>
<tr>
<td>Albert(^{63}) Ottawa, Ontario, Canada</td>
<td>2</td>
<td>1 well water sample, questionnaire, blood lead levels</td>
<td>1.7% children &lt;7 y prenatal to adult</td>
</tr>
<tr>
<td>T. Bell, 2004 (unpublished) St. John’s Newfoundland</td>
<td>2</td>
<td>Soil samples, some of which were collected in residential areas</td>
<td>Blood lead levels not sampled</td>
</tr>
<tr>
<td>Canada Health Survey(^{64}) National</td>
<td>2</td>
<td>blood lead levels</td>
<td>25% &gt;3 y</td>
</tr>
<tr>
<td>Canada Mortgage and Housing Corporation(^{65}) Medicine Hat, Alberta</td>
<td>2</td>
<td>Soil, housedust (vacuum samples), paint chips, lead swabs (postabatement), drinking water (standing sample).</td>
<td>Blood lead levels not sampled although 2 cases reported previously in national media report. (See Canadian Broadcasting Corporation(^{66})) Blood lead levels reported in a previous study (unpublished, R. Scott, 1995).</td>
</tr>
<tr>
<td>Canada Mortgage and Housing Corporation(^{67}) St John, New Brunswick</td>
<td>2</td>
<td>Environmental survey investigated post hoc the relationship between blood lead levels and drinking water. The subsample of 100 homes was chosen from a previous sample of 500 St John residences (unpublished, R. Scott, 1995).</td>
<td></td>
</tr>
<tr>
<td>Canadian Broadcasting Corporation (CBC)(^{66}) Medicine Hat, Alberta</td>
<td>2</td>
<td>National news coverage of 2 Medicine Hat children lead poisoned through home renovating activities (See also Canada Mortgage and Housing Corporation(^{65}))</td>
<td>2 cases 1-y male (30 μg/dL) 3-y female (22.9 μg/dL)</td>
</tr>
</tbody>
</table>

(continues)
Table 1. Epistemological domain: Canadian blood lead surveys, case studies and environmental studies related to housing as a source of Pb exposure (Continued)

<table>
<thead>
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<th>Author/Location</th>
<th>Environmental media collected from residential sources</th>
<th>% EBLL or #cases &amp; age range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cushing^68</td>
<td>Clinical symptoms ie, basophilic stippling, seizures</td>
<td>17 cases</td>
</tr>
<tr>
<td>Children’s Memorial Hospital, Montreal, Quebec, Canada</td>
<td>1</td>
<td>1-3 y</td>
</tr>
<tr>
<td>Canadian Health Measures Survey^13</td>
<td>Blood and urine</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>National Health Canada, The Canadian housedust study^69</td>
<td>Residential dwellings. Vacuum samples collection (concentration) and floor swab samples (lead loading)</td>
<td>Blood lead levels not sampled</td>
</tr>
<tr>
<td>National Health Canada, Maternal-infant research on environmental chemicals^70</td>
<td>Measures of environmental contaminants in breast milk, blood, urine, hair and cord blood and meconium of infants. Information collected on construction date of housing</td>
<td>Results not available</td>
</tr>
<tr>
<td>Hukowich, 1997 (unpublished) Port Hope, Ontario, Canada</td>
<td>Blood lead levels</td>
<td>4%</td>
</tr>
<tr>
<td></td>
<td>Elevated blood lead levels traced to residential sources of exposure although environmental samples not collected to confirm source</td>
<td>3-6 y</td>
</tr>
<tr>
<td>Jin et al^71</td>
<td>Blood lead levels, questionnaire</td>
<td>8%</td>
</tr>
<tr>
<td>Vancouver, British Columbia, Canada</td>
<td>24-36 mo</td>
<td></td>
</tr>
<tr>
<td>Mitchell^72</td>
<td>Clinical symptoms such as bilateral wrist and foot drop</td>
<td>4 cases</td>
</tr>
<tr>
<td>Children’s Memorial Hospital, Montreal, Quebec</td>
<td>1-9 y</td>
<td></td>
</tr>
<tr>
<td>O’Heany et al^73</td>
<td>Blood lead levels and measures of lead in air, tap water, soil, and gasoline. “Housing condition” entered into regression analysis. Soil samples collected from school yard. No description of water sampling method (ie, flushed or standing)</td>
<td>4.3%</td>
</tr>
<tr>
<td>Toronto, Ontario, Canada</td>
<td>&gt; 20 μg/dL</td>
<td></td>
</tr>
<tr>
<td>Uplands and Rockcliffe Military Base, CFSU(O) Healthcare Centre 2002 (unpublished), Ottawa, Ontario, Canada</td>
<td>House dust sampling and Blood lead levels</td>
<td>Unknown. Both adults and children tested but results not distributed by age.</td>
</tr>
</tbody>
</table>

(continues)
Table 1. Epistemological domain: Canadian blood lead surveys, case studies and environmental studies related to housing as a source of Pb exposure (Continued)

<table>
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<th>Author/Location</th>
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<th>% EBL or #cases &amp; age range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poon et al\textsuperscript{62}</td>
<td>2</td>
<td>No environmental sampling conducted; however, researchers noticed an initial increase and then a decline in children’s Blood lead level following extensive renovations to remove lead painted surfaces.</td>
<td>25/1000 (2.5%) with Blood lead levels &gt; 25 μg/dL. Overall mean value 8 μg/dL 6 mo–20 y</td>
</tr>
<tr>
<td>London, Ontario, Canada</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rasmussen, Subramanian et al\textsuperscript{40}</td>
<td>2</td>
<td>Residential dwellings. Concentrations of lead, among other metals, sampled in house dust using vacuum collection method. Range: 50.20 μg/g to 3225.66 μg/g</td>
<td>Blood lead levels not sampled</td>
</tr>
<tr>
<td>Ottawa, Ontario, Canada</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ross and Brown\textsuperscript{54}</td>
<td>2</td>
<td>Clinical symptoms, ie, presence of basophilic stippling, seizures</td>
<td>23 cases, ages not identified</td>
</tr>
<tr>
<td>Sick Children’s Hospital, Toronto, Ontario, Canada</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tenenbein\textsuperscript{38}</td>
<td>2</td>
<td>Sources of lead exposure were in both cases traced to residential paint, but it is unclear if actual environmental samples were collected.</td>
<td>2 cases: 26 mo and 36 mo</td>
</tr>
<tr>
<td>Undisclosed Canadian location</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valiquette and Kosatsky, 1995\textsuperscript{75}</td>
<td>2</td>
<td>Review of laboratory records. Paint identified as a principal source of exposure.</td>
<td>12 cases: (defined as &gt; 25 μg/dL). Blood lead level range: 27–94 μg/dL &lt;15 y</td>
</tr>
<tr>
<td>Montreal, Quebec, Canada</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

classifies lead as a possible group 2B carcinogen.\textsuperscript{29} Health Canada has also contributed to this body of knowledge with the “2005 Update of Evidence for Low-Level Effects of Lead and Blood Lead Intervention Levels and Strategies—Final Report of the Working Group”\textsuperscript{25}; “Lead (Pb) Risk Assessment in Canada, Part I: Critical Review of Toxicity Reference Values”\textsuperscript{56}; and “Critical Review of Potential Health Effects Associated With Lead (Pb): Final Report.”\textsuperscript{51}

2. **Vulnerability domain.** In the United States, children of low-income families, living in pre-1946 dwellings have a 16% prevalence rate of lead poisoning compared with a 4% rate for children from middle-income families.\textsuperscript{52} Families with fewer economic resources have less choice in where they can live and are more likely to dwell in poorly maintained older housing.\textsuperscript{55} For some time, researchers have also been aware of the vast and detrimental difference in impact between exposing a mature adult to lead through, for example, occupational exposure and chronically exposing a developing fetus to maternal sources, or a child to lead in drinking water or floor dust. A June 2000 supplemental edition of the journal *Environmental Health Perspectives* introduced the concept of
“critical windows of exposure,” that is, periods of vulnerability or times when fetal and infant brain development are particularly susceptible to exposure to toxins such as lead, mercury, polychlorinated biphenyls, and pesticides. Lidsky and Schneider reviewed in detail the neurological changes one can expect to see in a lead-poisoned child. Of further importance is an awareness that blood lead levels tend to peak in children between the ages of 18 and 36 months and decline thereafter. The implications for research within the Canadian context are clear: investigations of childhood lead poisoning should include vulnerable populations, that is, children younger than 6 years and belonging to low-income families.

3. Epistemology domain. How do people know if childhood lead poisoning is even a problem in Canada? A review of Canadian research spanning more than 7 decades reveals that the investigation of childhood lead poisoning in relation to housing sources is an underresearched area in Canada. The review recovered only 4 top-ranked studies that described cases or conducted community blood lead surveys and which also collected environmental samples to verify the source of exposure (Table 1). Despite the paucity of data, there is sufficient evidence to support the thesis that childhood lead poisoning related to housing sources exists in Canada. The article describes the cases of 4 children younger than 6 years who were lead poisoned from residential sources.

A 1990 issue of the Canadian Medical Association Journal recounts 2 cases of lead poisoning occurring in toddlers: a 32-month-old girl with a “17-month history of a voracious appetite for the paint peeling from the walls and window sills of her inner city home” and a 26-month-old boy, who after many repeat visits to hospital for persistent vomiting was eventually diagnosed and medically treated for lead encephalopathy. In both instances, the source of exposure was traced to residential paint although residential samples were not collected to verify the exposure source. A Quebec City study comparing rural children’s blood lead levels with urban children’s levels reported that 10.8% of children in that study had lead poisoning (considered blood lead levels \( \geq 10 \mu g/dL \)), with 6 of 10 cases attributed to housing sources. A New Brunswick survey of children aged 1 to 3 years discovered 11.3% of children with blood lead levels \( \geq 10 \mu g/dL \) (C. Balram and S. C. Giffin, unpublished data, 1993). Sources of exposure were traced to residential paint, drinking water, and other sources. The New Brunswick study is one of the few Canadian studies that examined childhood lead poisoning in relation to housing sources post leaded gasoline use. A 2001 investigation by Health Canada documented high levels of lead in floor dust in samples collected from 50 Ottawa homes. Unfortunately, blood lead samples of home occupants were not assessed to
determine whether lead poisoning had occurred. A 2004 issue of the Canadian Medical Association Journal reports the case of a 4-year-old Montreal boy seriously poisoned from eating paint chips peeled off the bathroom trim in his family home.11

4. Health protection domain. In the United States, 3 major federal agencies inform the public on the issue of childhood lead poisoning: EPA, the Centers for Disease Control and Prevention (CDC), and the Department of Housing and Urban Development (HUD). In addition, the National Lead Information Centre, a clearinghouse for lead information, operates under joint funding from EPA, CDC, and HUD. National not-for-profit organizations such as the National Center for Healthy Housing, the Alliance for Healthy Homes, and parent-based organizations such as United Parents Against Lead disseminate lead awareness information to parents and policy makers. The CDC publishes guidance documents for health professionals, such as Preventing Lead Poisoning in Children.9 The United States has also enacted legislation that requires disclosure of sources of lead in residential dwellings. Under the Residential Lead-Based Paint Hazard Reduction Act of 1992, also known as Title X, landlords and homeowners are required to disclose to the buyer and the tenant any known existing lead hazards contained in housing built before 1978. EPA and HUD provide a national training program for the inspection of residential lead hazards. In addition, HUD provides millions in annual funding for the control of lead-based paint hazards in eligible communities.42

Two federal agencies in Canada hold the primary responsibility for disseminating information on childhood lead poisoning and housing. Under the Department of Health Act,43 the federal Minister of Health, (through Health Canada) has the duty to investigate and research public health, including the monitoring of diseases and the collection, analysis, interpretation, publication, and distribution of information relating to public health. As mentioned, Health Canada is conducting a national biomonitoring study, the Canadian Health Measures Survey13 of adults and children, which does include measures of blood lead; however, vulnerable populations (children younger than 6 years) have been excluded from the sample population to date. And while Health Canada has developed a “lead strategy,” its major focus is on lead in consumer products, a relatively minor source when compared with the potential of housing for childhood lead exposure.44 Under the National Housing Act,45 CMHC is assigned the statutory responsibility to distribute information on housing and living conditions. Even so, in Canada, the extent of lead contamination in housing is poorly understood and can only be estimated.12

The United States has been a leader in advancing knowledge in the field of childhood lead poisoning, and true to Dixon and Dixon’s assertion, much of the research on this health topic falls within the physiological domain. Dixon and Dixon advise that if gains are to be made in environmental health, then an interdisciplinary approach that increases knowledge development in all 4 domains is necessary.14 The literature review was particularly useful in revealing deficiencies within the epistemological and health protection domains in Canadian research and policy.

Toward a critical analysis of childhood lead poisoning in Canada

Despite a lack of case and cross-sectional studies, there is documentation of harm occurring to Canadian children from exposure to residential sources of lead—enough, surely,
to warrant immediate action within the rubric of the health protection domain. Are Canadians adequately protected? If not, why? These questions can best be answered through critical analysis. Over the past 2 decades, critical theory has emerged as an important means for nurses to identify and understand the manner by which sociopolitical and cultural environments shape nursing practice.\textsuperscript{46,47} Holmes and colleagues\textsuperscript{48} note the following:

Research that aims to be critical seeks, as its purpose of inquiry, a confrontation of the injustices in society as well as a questioning of the status quo, while giving a voice to vulnerable persons (including marginal/ized discourses). Critical researchers believe that the knowledge developed in their research may serve as a first step toward addressing such injustices.\textsuperscript{(p43)}

While Dixon and Dixon hint at the significant amount of literature in critical theory on public health discourses and its sociopolitical, historical, and economic implications, this approach is not investigated further or explored via their model. In a later article, Dixon and colleagues\textsuperscript{49} do suggest a series of research questions that can be adapted for future critical analysis: How does everyone know about this? What is the common knowledge and level of public concern? What messages come from corporations, environmental organizations, the media, health professionals, and other groups? What appears to be the dominant messages? How do these messages relate to what is known or not known through science? And, how might affected people acquire their sense of what is true? An important next step will be to critically examine the response of Canadian agencies to evidence of childhood lead poisoning related to residential sources. Studies have shown that there is an unequal distribution of health across society and that much of it can be linked to economic and social determinants of health.\textsuperscript{50} As housing is an environmental factor with strong ties to socioeconomic factors, it will be important to explore this topic as a social justice issue. Giddings’ Theoretical Model of Social Consciousness recognizes how “health disparities . . . are sustained by the institutionalizing of discriminatory healthcare policies and practices” and may be useful for designing a future critical analysis of this topic.\textsuperscript{23(p224)}

**RELEVANCE OF THE DIXON AND DIXON MODEL FOR RESEARCH IN NURSING**

Dixon and Dixon first published their model in ANS 8 years ago. It seems fitting to review what has been done with the model and reassess its relevance for nursing. The Dixon and Dixon model exhibits a blend of natural and social sciences and acknowledges multiple ways of knowing, including tradition, authority, group loyalty, experience, intuition, and science. While not explicitly stated, this stance locates the model within a realism paradigm. Realism “proposes a common ontology and epistemology for the natural and social sciences.”\textsuperscript{51(p1267)} The model also reflects a persistence worldview, one that emphasizes equilibrium and balance as opposed to growth and progress\textsuperscript{52} as evidenced by Dixon and Dixon’s goal of “protect[ing] the well being . . . of members and its emphasis on ‘balance.’”\textsuperscript{14(p43)} As it promotes “upstream” healthcare, it reflects a systems category that involves the “identification of actual and potential problems in the function of systems and delineation of intervention strategies that maximize efficient and effective system operation.”\textsuperscript{53(p14)}

According to Fawcett, conceptual models are broad, abstract, and evolve from empirical observations and intuitive insights of scholars. They can be induced when specific situations exemplify more general events. Conceptual models also provide direction on how to observe and interpret phenomena. Fawcett’s\textsuperscript{54} metaparadigm concepts of nurse, person, health, and environment are incorporated into the Dixon and Dixon model. Dixon and Dixon note that nurses “are well positioned to participate in and provide leadership for interdisciplinary efforts to improve environmental health”\textsuperscript{14(p54)} although their approach facilitates a view that goes beyond
the traditional realm of nursing. Similar to Anderson and McFarlane’s Community as Partner Model interventions are not constrained to established healthcare institutions. Public participation in health decision making is encouraged, in fact, imperative. In the article titled “Kids Need Clean: Air Pollution and Children’s Health: Environmental Issues in the Health of Children,” nurses build the capacity of community members through knowledge development and leadership.

In the Dixon and Dixon model, “person” has individual, developmental, gender-based and physiological characteristics that make some people more vulnerable than others to exposures. Person in this model can also become an expert in the particular health issue that is of concern. In this respect, the IMEHR shares a characteristic with Falk-Rafael’s Model for Empowered Caring, in that, both incorporate the expectation that as individuals and communities increase their knowledge of issues concerning health, the expertise gained will have a direct impact on health protection. “Health” is seen as the outcome or the result of exposure to an environmental agent. Outcomes can include but are not limited to increased mortality, morbidity, functional limitations, and symptom experience affecting quantity and quality of life. “Environment” can be the exposure agent, but it can also be the community characteristics, sociodemographic and cultural characteristics, public policies, location of residence, and occupation.

Butterfield, a contemporary of Jane Dixon and one of the reviewers of the Dixon and Dixon model, devised a road map for positioning environmental health curricula into nursing schools and mainstream practice. Harnish and colleagues tested the Dixon and Dixon model by examining whether community perceptions of environmental risk were congruent with or diverged from the model. Home visit data from 11 parents were analyzed thematically according to the Dixon and Dixon model concepts. Through this research, they uncovered barriers to preventive action. Parents, for example, were aware of risks but were unsure of what to do about them. Harnish and colleagues found that the model was a “useful intermediate step for researchers involved in multiyear studies.”

Macdonald used the Dixon and Dixon model to illuminate the Toronto severe acute respiratory syndrome (SARS) experience. Macdonald’s research question was, “[W]hat combination of factors has the potential to lead to a problem, and what can be done upstream so the world is not dealing with emerging diseases like SARS?” Macdonald found the epistemological domain the most helpful in gaining greater understanding of the Toronto experience. In exploring the question “How does everyone know?” Macdonald uncovered weaknesses in provincial microbiology laboratory resources, as well as poor communication links between provincial public health units and between the public health department and hospitals.

Im notes the model’s social utility in generating situation-specific theory. A situation-specific theory that incorporates the concepts of the Dixon and Dixon conceptual model may be appropriate for further investigation of health issues such as childhood lead poisoning, at the local or community level.

A minor shortfall of this model, which is strongly rooted in primary prevention, is its lack of guidance for secondary and tertiary prevention within the health protection domain. It is likely that populations of “at-risk” children (ie, children with elevated blood lead levels) will be identified through future community research in Canada, and there will be a need for case management. Blending the Dixon and Dixon model with one such as Anderson and McFarlane’s Community as Partner Model might address this problem as the latter does incorporate the concepts of secondary and tertiary prevention. Even so, Greving and Santacroce were able to demonstrate how the Dixon and Dixon model can be applied after harm has occurred. In this application, children who are survivors of cancer continue to be at risk because the
chemotherapy itself is toxic—it is especially
damaging to the cardiovascular system. The
nursing approach in this situation is both ter-
tiary and primary. That is, nurses are treat-
ing cancer (tertiary prevention); but also,
when nurses promote cardiovascular health
for these children, they are practicing primary
prevention.

The Community Action Model might also
enhance the Dixon and Dixon health protec-
tion domain. It employs a detailed 5-step pro-
cess for building community capacity for con-
fronting health disparities. The steps include
(1) training of participants, (2) defining and
designing a community diagnosis, (3) analy-
sis, (4) implementing an action or activity, and
(5) maintenance or enforcement of the activity.
The model involves participatory action
research and was designed to have a “lasting impact . . . by creating environmental policy
change.”

With its upstream focus on primary pre-
vention, and its goal of facilitating optimal
level wellness to protect the well-being of so-
ciety’s most vulnerable members, the Dixon
and Dixon model meets expectations for a
nursing model in that nurses are required to
view health in its larger framework, as linked
to pathology in the environment. This model
contributes to the discipline of nursing by
viewing the concept of health through the
lens of physiology, vulnerability, epistemol-
ogy, and health protection. Perhaps what is
most satisfying about the Dixon and Dixon
model is that it does not privilege health pro-
fessional action over action on the part of the
community or individual members. “Person”
is acknowledged for the gained expertise and
ability to affect change. Shortcomings within
the Dixon and Dixon health protection do-
main can be addressed by incorporating ele-
ments of other ecological models such as the
Community as Partner Model or the Com-
munity Action Model.

IMEHR was employed to structure a lit-
erature review on the topic of childhood
lead poisoning in Canada and was useful
in identifying gaps in areas of research and
policy within the 4 primary domains: phys-
iological, vulnerability, epistemological, and
health protection. This exercise appears to
support Dixon and Dixon’s claim that the
physiological and vulnerability domains im-
 pact the development of knowledge (episte-
 mological domain), which in turn forms the
basis for the health protection domain—
although the model is limited in its ability
to explain why knowing a problem exists
does not necessarily lead to doing something
about it. In future, a critical analysis that exam-
ines the interplay between the epistemologi-
cal and health protection domains will assist
in identifying power relations and underlying
social conditions that have prevented knowl-
edge of this problem from translating into req-
quisite action.

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JL. Estimating the global burden of disease of mild
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of U.S. children to residential dustlead, 1999–2004:
housing and demographic factors. Environ Health


16. Weiss B, Landrigan PJ. The developing brain and the...


APPENDIX C

Complete list of titles included and excluded for the analysis
A. Titles included in the analysis


McKeown D. Environmental threats to children understanding the risks, enabling prevention. Toronto, ON: Toronto Public Health; 2005.


B. Titles excluded from the analysis


Bussières D, Ayotte P, Levallois P, Dewailly E, Nieboer E, Gingras S, Côté S. Exposure of a Cree population living near mine tailings in northern Quebec (Canada) to metals and metalloids. Arch Environ Health. 2004 Dec;59(12):732-41


Canadian Association of Physicians for the Environment (CAPE): lead information
CDC (2005). Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control and Prevention Atlanta, Ga..


Health Canada: Maternal-Infant Research on Environmental Chemicals (MIREC); Managing Results of Maternal Lead, Mercury and Cadmium in Blood, October 2008


The Canadian Partnership for Children’s Health and Environment (CPCHE)
The Learning Disabilities Association of Canada (LDAC): children’s environmental health
The Public Health Agency of Canada, (PHAC)


APPENDIX D

Canadian ‘lead’ authors listed by frequency of appearance and then alphabetically
<table>
<thead>
<tr>
<th>Author name</th>
<th>Frequency</th>
<th>Author name</th>
<th>Frequency</th>
<th>Author name</th>
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APPENDIX E

Internet search strategy, methods and results
Purpose. To identify dominant publicly accessible discourses on the topic of childhood lead poisoning in Canada.

Method

The internet is becoming increasingly popular as a source of health information for the public (Hesse et al., 2005). The Google search engine is particularly well-liked, handling up to two-thirds of all Internet search queries in the United States (Stone, 2010). Other popular search engines include Yahoo!, MSN (which defaults to Bing for those with Canadian IP addresses) and Ask (Sullivan, 2006). Each search engine has a built-in system for ranking websites according to the number of times a link is activated. Google, for example, assigns a rank value of “1” to a website link each time it is “clicked” on. Each additional “click” increases a site’s value of “weight” by “1” so that the most popular pages appear at the top of the results page and pages with less weight appear lower or on subsequent pages (Evans, 2007). Website rankings tend to be stable over periods of months although popular search engines such as Google experience minor changes in ranked results over time (Bar-Ilan, Levene, & Mat-Hassan, 2006)

i. The following four search engines were used for this review: Google, Bing, Yahoo! and Ask. A search using the terms “lead poisoning” returned information exclusively from American sources for all four search engines including Google.ca; therefore the term “Canada” was added. Where available, the option to limit results to Canadian websites was applied (Google and Bing) so that a total of 6 searches were executed.

ii. Google.ca was searched using the terms “lead poisoning” [and] united states to provide a comparison between Canadian and American search results.

Search 1.
Terms: lead poisoning Canada
Date of search: April 22 2010
Search engines

Google
www.google.com (defaults to http://www.google.ca/)
www.google.us (defaults to http://www.google.com/webhp)

Yahoo!
www.yahoo.com (defaults to www.yahoo.ca)

Bing
www.bing.com (only from Canada)
www.bing.com (show all)

Ask
www.ask.com

Search 2.
Terms: lead poisoning united states
Date of search: April 22 2010
Search engines

Google
www.google.ca

Search 3 French discourse on lead poisoning.
Terms:
Étude de la prévalence de la plombémie chez Québec
La prévalence du saturnisme infantile Québec
en rénovation plombémie chez Québec
la poussière de plomb
Études de biosurveillance axés sur les populations ciblées Canadiens
l'exposition chronique au plomb chez les Canadiens
contamination par le plomb
la teneur sanguine en plomb des jeunes enfants Canadiens

Date of search: May 1 2010
Search engine
Google
www.google.ca

iii. All search engines used for this review had a default display setting of 10 results per page. Only the top 10 results were included for analysis since users do not typically inspect more than the first results page returned by a search engine (Bar-Ilan et al., 2006).
iv. In the next phase, a scoring system was developed to rank the 60 returned search items. Only those Web sites pertaining to lead poisoning and Canada were ranked. American based websites, news agencies and sponsored (commercial) links were excluded leaving a total of 31 Web sites. Because lay people assign credibility to Web site information based on source, professional design, scientific appearance, and ease of use (Eysenbach, 2002) a “weight” of “1” was given to agencies identified as “authorities,” i.e. government Web sites.

v. Web sites were assigned a score of 10 if they appeared first on the results list (i.e. 1=10; 2=0; 3=8, .... 10=1) for a possible total of a possible 60. Ranked results for the 31 Websites are displayed in Table 1.
Table E1. Top ranked discourses on “lead poisoning” and “Canada” by search engine

<table>
<thead>
<tr>
<th>Agency</th>
<th>Deemed as having authority</th>
<th>Yahoo!</th>
<th>Ask</th>
<th>Bing (Cdn)</th>
<th>Bing (generic)</th>
<th>Google (Cdn + Generic)</th>
<th>Google (Generic)</th>
<th>Total Score</th>
<th>Total Possible Score</th>
<th>Rank (%) Total score/ Possible score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health Canada: It's Your Health</td>
<td>1</td>
<td>10</td>
<td>8</td>
<td>9</td>
<td>5</td>
<td>9</td>
<td>9</td>
<td>51</td>
<td>60</td>
<td>85</td>
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<tr>
<td>Canadian Medical Association Journal</td>
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<td>0</td>
<td>7</td>
<td>10</td>
<td>8</td>
<td>11</td>
<td>11</td>
<td>48</td>
<td>60</td>
<td>80</td>
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<tr>
<td>Health Canada: Some commonly asked questions</td>
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<td>0</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>10</td>
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<td>30</td>
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<td>University of Northern BC</td>
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<td>0</td>
<td>8</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>17</td>
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<td>28</td>
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<td>7</td>
<td>15</td>
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<td>Abilities</td>
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<td>14</td>
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<td>0</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>11</td>
<td>60</td>
<td>18</td>
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<td>McGill University Health Centre</td>
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<td>0</td>
<td>4</td>
<td>6</td>
<td>0</td>
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<td>0</td>
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<td>2</td>
<td>60</td>
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</table>
The following Canadian agencies, although they contain information on lead poisoning, were not captured by the Web search strategy: the Canadian Association of Physicians for the Environment (CAPE), the Learning Disabilities Association of Canada (LDAC) and the Canadian Partnership for Children’s Health and Environment (CPCHE).

Inclusion criteria. Web site content from the above internet sites was included for analysis if it met the following inclusion criteria: a) the discourse pertained to the Canadian context; b) the discourse pertained to childhood lead poisoning; and c) the discourse played a strategic role in transforming the public health discourse on childhood lead poisoning in Canada, i.e. Cooper’s critical analysis of Health Canada’s policy on lead (2000).

RESULTS

Google Search 1&2

Search engine: www.google.ca and www.google.com/webhp
Search terms: lead poisoning Canada
Date of search: April 22 2010

1. Page 6 - Lead Information Package - Some Commonly Asked Questions ...
   Jump to Is lead poisoning a common problem in Canada? : However, since low-level lead poisoning is often unrecognized, it is difficult to ...
   www.hc-sc.gc.ca › ... › Environmental Contaminants › Lead - Cached - Similar
   (Health Canada = 10)

2. It’s Your Health - Effects of Lead on Human Health
   20 Nov 2008 ... Severe cases of lead poisoning are rare in Canada. However, ongoing exposure to even very small amounts of lead can be harmful, ...
   www.hc-sc.gc.ca › ... › Healthy Living › It’s Your Health › Environment -
   (Health Canada = 9)

3. Risks of Lead Poisoning - Abilities Canada - Abilities Magazine
   Risks of Lead Poisoning - Abilities Canada - Abilities Magazine. ... Lead poisoning is more pervasive than most Canadians think, and it usually happens
4. Screening Children for Lead Exposure in Canada
File Format: PDF/Adobe Acrobat - Quick View
by W Feldman - Cited by 4 - Related articles
Lead poisoning is insignificant when compared to poisoning by other drugs. In 1978, The Canada Health Survey, the only national survey of ... www.phac-aspc.gc.ca/publicat/clinic-clinique/pdf/s2c25e.pdf
(Public Health Agency of Canada = 7)

5. Lead poisoning in children -- Bell and O'Grady 171 (5): 429 ...
by W Bell - 2004 - Related articles
The authors assert that lead poisoning is now rare in Canada, but there are almost no current data on this problem. The last national pediatric blood lead ...
www.cmaj.ca/cgi/content/full/171/5/429
(CMAJ = 6)

6. Lead poisoning from "lead-free" paint -- Lavoie and Bailey 170 (6 ... 
by PM Lavoie - 2004 - Cited by 7 - Related articles
Although lead poisoning is now rare in Canada, exposure from a variety of possible environmental sources must be considered if a patient has an elevated ...
www.cmaj.ca/cgi/content/full/170/6/956 - Similar
(CMAJ = 5)

7. Yahoo! Canada Directory > Lead Poisoning
Working to educate the public and health care providers about childhood lead poisoning. Includes information and statistics about lead exposure and links to ca.dir.yahoo.com/Health/Environmental.../Lead_Poisoning/
(Excluded)

8. Fish Lead Free - Canadian Wildlife Service - Environment Canada
It is estimated that 20–30% of loon mortalities in eastern Canada are due to lead poisoning from the ingestion of lead-containing sinkers or jigs that are ...
www.cws-scf.ec.gc.ca/flf-psp/index_e.cfm - Cached - Similar
(Canadian Wildlife Service = 3)

9. The Franklin Expedition
Lead causes lead poisoning, which affects the body in many severe ways: ... Mysteries of Canada
http://www.mysteriesofcanada.com/Nunavut/franklin.htm
www.mysteriesofcanada.com/.../franklin_expedition.htm - Cached - Similar
(Excluded)

10. Lead Poisoning in Birds: How and Why Birds Die from Lead Poisoning
Symptoms of lead poisoning in birds. A Canada Goose huddles at the
lake's edge. It is weak and listless and has been eating poorly for days. ...
birds.suite101.com/article.cfm/lead_poisoning_in_birds - Cached - Similar
(Excluded)

-----------------------------------------------------------------------------------------------------

Bing Search 3

Search engine: www.bing.com (only from Canada)
Search terms: lead poisoning Canada
Date of search: April 22, 2010

1. Lead poisoning in children -- Bell and O'Grady 171 (5 ... 
Medical Association or its licensors All editorial matter in CMAJ represents the
opinions of the ... 
www.cmaj.ca/cgi/content/full/171/5/429
(CMAJ = 10)

2. It's Your Health - Effects of Lead on Human Health
2002 article from Health Canada and the Public Health Agency of Canada. Lead
occurs naturally in ... Severe cases of lead poisoning are rare in Canada. However,
ongoing exposure to ...
www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/lead-plomb-eng.php · Cached page
(Health Canada = 9)

3. OSH Answers: 2-Health Effects of Lead
What happens when lead comes into contact with my skin? Can lead hurt my eyes?
... www.ccohs.ca
www.ccohs.ca/oshanswers/chemicals/chem_profiles/lead/health_lead.html · Cached page
(Canadian Centre for Occupational Health and Safety = 8)

4. Lead and the Fall of Rome: A Bibliography
www.nipissingu.ca/department/history/muhlberger/orb/lead.htm · (Excluded)

5. No need to worry about lead poisoning - Cases of lead ...
6. Lead poisoning in cats dogs and birds - Article on Pets ...
Lead poisoning in cats dogs and birds - Pets.ca is Canada's source for info on pets including dogs cats birds and more. We have articles and information, pet store, free petsites ...
www.pets.ca/articles/article-leadpoisoning.htm · Cached page
(Excluded)

7. EnviroZine - Features
Studies in Ontario and Atlantic Canada suggest that lead poisoning resulting from swallowing lead sinkers and jigs is the leading cause of reported deaths of adult Common Loons.
www.ec.gc.ca/EnviroZine/english/issues/06/feature2_e.cfm · Cached page
(Environment Canada = 4)

8. Lead poisoning tough to diagnose
Lead poisoning tough to diagnose Fisher-Price recall prompts some parents to toss toys that might contain lead in paint August 03, 2007 TRISH CRAWFORD LIVING REPORTER Alicia Martell is ...
www.pcfk.on.ca/PDFs/Research_Ken/LeadPoisoningHardto%20Diagnose.pdf · (Excluded)

Lead Poisoning . In the section on poisoning, we mentioned getting your paint tested for poisonous lead. Lead is especially dangerous to pregnant women and ...
www.nald.ca/library/research/hudson/childsaf/page23.htm · (National Adult Literacy Database = 2)

10. CBC News - Consumer Life - Children's necklaces, key ...
Four children's metal pendant necklaces and key chains sold at jewelry stores Claire's and Ardène are being recalled because they could make children ill from lead poisoning ...
www.cbc.ca/consumer/story/2007/01/03/necklace-advisory.html
(Content included only)
1. Lead Poisoning

Lead Poisoning: Risk Factor: Metallic lead is a highly toxic metal that when ingested, affects many organs and organ systems. Wildlife are generally exposed to lead following ingestion ...

www.unbc.ca/nlui/wildlife_diseases_bc/lead_poisoning.htm · (UNBC = 10)

2. Childhood Lead Poisoning Prevention Branch

(Ver esta pagina en español.) Childhood Lead Poisoning Prevention Branch. California has enacted landmark legislation to prevent childhood lead poisoning.

www.dhs.ca.gov/childlead · (Excluded – non-Canadian)

3. Lead poisoning in children – Bell and O’Grady 171 (5 …

CMAJ • August 31, 2004; 171 (5). doi:10.1503/cmaj.1040573. © 2004 Canadian Medical Association or its licensors All editorial matter in CMAJ represents the opinions of the ...

www.cmaj.ca/cgi/content/full/171/5/429 · (CMAJ = 8)

4. Lead poisoning - Wikipedia, the free encyclopedia

Lead poisoning (also known as plumbism, colica pictonium, saturnism, Devon colic, or painter’s colic) is a medical condition caused by increased levels of the heavy metal lead in the ...

Classification · Signs and symptoms · Exposure routes · Pathophysiology

en.wikipedia.org/wiki/Lead_poisoning · Cached page · (Excluded)
5. Lead Poisoning: Rome-Lead Connection
Lead Rome & Lead Connection. Did lead poison the Roman Empire? Lead's discovery dates back to 3500 BC. Lead artifacts have been found throughout the ancient world, and some ...
ces.ca.uky.edu/energy/lead/rome_lead.htm ·
(Excluded – non Canadian)

6. It's Your Health - Effects of Lead on Human Health
2002 article from Health Canada and the Public Health Agency of Canada. Lead occurs naturally in ... Severe cases of lead poisoning are rare in Canada. However, ongoing exposure to ...
www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/lead-plomb-eng.php ·
(Health Canada = 5)

7. OSH Answers: 2-Health Effects of Lead
What happens when lead comes into contact with my skin? Can lead hurt my eyes? ... www.ccohs.ca
www.ccohs.ca/oshanswers/chemicals/chem_profiles/lead/health_lead.html ·
(Canadian Centre for Occupational Health and Safety = 4)

8. Lead poisoning in cats dogs and birds - Article on Pets ...
Lead poisoning in cats dogs and birds - Pets.ca is Canada's source for info on pets including dogs cats birds and more. We have articles and information, pet store, free petsites ...
www.pets.ca/articles/article-leadpoisoning.htm ·
(Excluded – animals)

9. Lead Poisoning
Lead Poisoning Symptoms of lead poisoning can range from lethargy and depression, inability to ... retails at $9.40 but is probably cheaper in the U.S. Gillian Willis willis@dpic.bc.ca
theaviary.com/lead.shtml ·
(Excluded – commercial)

10. Occupational Lead Poisoning Prevention Program
The Occupational Lead Poisoning Prevention Program (OLPPP) The Occupational Health Branch and most California Department of Public Health (CDPH) offices will be closed three ...
www.dhs.ca.gov/ohb/OLPPP ·
(Excluded – non-Canadian)
Ask search 5

Search engine: www.ask.com
Search terms: lead poisoning Canada
Date of search: April 22, 2010

1. News results for lead poisoning canada
   Nearly pure lead bracelet recalled - Vancouver Sun - April 16 3:24 PM Explore Story »
   news.ask.com · More news articles »
   (Excluded – media)

2. Page 6 - Lead Information Package - Some Commonly Asked Questions
   Is lead poisoning a common problem in Canada? ... Very few cases of lead poisoning are documented in Canada each year. However, since low-level lead poisoning is often unrecognized, it is difficult to determine the number of Canadians affected by exposure to low levels of lead.
   www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/exposu...
   (Health Canada = 9)

3. It's Your Health - Effects of Lead on Human Health
   Short-term exposure to high levels of lead can cause vomiting, diarrhea, convulsions, coma or even death. Severe cases of lead poisoning are rare in Canada.
   www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/lead-plomb-eng.ph...
   (Health Canada = 8)

4. Lead poisoning in children -- Bell and O'Grady 171 (5): 429
   The case history of lead poisoning presented by Pascal Lavoie and Benoit Bailey illustrates the surreal state of political responses to this issue in Canada today. ... The authors assert that lead poisoning is now rare in Canada, but there are almost no current data on this problem.
   www.cmaj.ca/cgi/content/full/171/5/429
   (CMAJ = 7)

5. Yahoo! Canada Directory > Lead Poisoning
   CDC Childhood Lead Poisoning Prevention Program - Working to educate the public and health care providers about childhood lead poisoning. Includes information and statistics about lead exposure and links to federal, state, and local lead poisoning prevention sites
   ca.dir.yahoo.com/Health/Environmental_Health/Environmen
   (Excluded)

6. Lead poisoning - Wikipedia, the free encyclopedia
   Lead poisoning (also known as plumbism, colica pictumnum, saturnism, Devon colic, or painter's colic) is a medical condition caused by increased levels of the heavy metal lead in the ...
   Classification ·
   Signs and symptoms ·
7. No need to worry about lead poisoning - Cases of lead poisoning in Canada are very rare. However, we strongly recommend that the recalled toys should be taken away from children immediately." For a complete list of the recalled toys please go to Mattel’s website (www.mattel.com).

8. Lead poisoning in Canada geese on Plum Island, Massachusetts
During December 1983 and early January 1984, about 200 Canada geese (Branta canadensis) died of lead poisoning at Parker River National Wildlife Refuge on Plum Island, Massachusetts

9. Paint Poisoned Homes: An Action Plan to Eliminate Childhood Lead Poisoning in Canada, in sharp contrast, has been doing little to address this issue. Not much is known about the extent of childhood lead poisoning in Canada

10. Home / World Wide / Canada / Lead Poisoning in Detroit - Medi
Lead poisoning is sometimes called the silent killer. It seems a fitting name for a substance that has destroyed the lives of millions of people across the country and tens of thousands in Detroit, ...
2. Canadian Wildlife Service (CWS), part of Environment Canada, handles wildlife matters that are the responsibility of the ... Canada are due to lead poisoning ... www.cws-scf.ec.gc.ca/flf-psp/index_e.cfm - (CWS = 9)

3. Lead Poisoning
Lead poisoning has been mainly reported in birds but has also been documented in ... Environment Canada / Canadian Wildlife Service - lead poisoning information ...www.unbc.ca/nlui/wildlife_diseases_bc/lead_poisoning.htm - (UNBC = 8)

4. Lead Poisoning In Cattle
A review of lead poisoning in Alberta cattle, with a discussion of the causes and symptoms, and prevention tips ... Lead poisoning can affect any cattle operation. ... www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/agdex791 - (Agriculture Alberta = 7)

5. CBC News - Canada - Children's necklaces, key chains recalled for lead ... ... make children ill from lead poisoning. Health Canada said in an advisory notice Wednesday. ... Health Canada: Effects of Lead on Human Health ... www.cbc.ca/canada/story/2007/01/03/necklace-advisory.html - (CBC News = 6)

Wildlife, Nature and Conservation in British Columbia, Canada: Lead Poisoning in Birds - on BritishColumbia.com ... and other birds die from lead poisoning. ...vancouverisland.com/.../information/Lead Poisoning of Water Birds.htm - Cached - More from this site

7. Cheap jewelry can give kids lead poisoning - Capital News Online
Health Canada warns kids who suck or chew on cheap jewelry risk lead poisoning ... lead poisoning, and a solution to the problem is still years away Health www.carleton.ca/jmc/cnews/19012001/n4.htm - Cached - More from this site

8. The Franklin Expedition
Lead causes lead poisoning, which affects the body in many severe ways: delusion, ... Mysteries of CanadaVIZCAN Systems Corporation - www.canadabooksonline.com
www.mysteriesofcanada.com/Nunavut/franklin_expedition.htm - Cached - More from this site


10. More from this siteVancouver Island eagles died from lead shot poisoning
Lead shot causing deaths in wildlife, plus discarded fishing sinkers and jigs ... year in Canada thousands of waterfowl contract lead poisoning from shotgun timescolonist.com/Vancouver+Island+eagles+died+from+lead+shot+poisoning...
References


APPENDIX F

“Rawlinson’s” Guide to Inquiry
Citation:

Screening criteria for inclusion of discourse/event for analysis

1. Does this discourse and/or events provide a model or ideas that influence the function of the discourse on childhood lead poisoning?
   __Yes - include
   __No - exclude

2. In what ways? For example, what words in the discourse have linguistic or social history that is significant for assessing the role of the discourse within current power relations?

3. Document genre [Highlight all that apply]:

Analysis Guide

A. The axis of knowledge: Describes systems of concepts and rules for the formation of statements, what determines the difference between true and false, what can be spoken about, and what engenders evidence

Foucault in The Archaeology of Knowledge (1972) identifies 3 dimensions along which objects become subject in a discourse: a) surfaces of emergence, b) authorities of delimitation, and c) grids of specification.

Surfaces of emergence describe the point in which discourses “surface.” Discourse arises in relation to other ways of thinking, not in some independent manner.

Authorities of delimitation describes limits for the identification of subjects regarding the discourse in question. The discipline of medicine, for example, defines limits for the discipline of nursing. How did US public discourse on childhood lead poisoning provide limits for the Canadian public health discourse on childhood lead poisoning?

Grids of specification concerns taxonomies, for example, the identification of “risk” populations of blood lead levels of concern)

How is CLP socially constructed in Canada? i.e. acute poisoning vs chronic poisoning.

What are the rules for the appearance of subjects?
What grids of specification are there? For example how are “lead poisoned children” classified, interrelated and placed in a taxonomy? (i.e. definition of “at risk”)

What are the surfaces of emergence? What historical context influenced the development of the discourse? How did this particular discourse arise (in relation to other discourses)?

What authorities of delimitation exist? For example, do authors describe findings of Canadian blood surveys reported in comparison to US findings or other geographic/political contexts?

B. Axis of authority: Describes the historical formation of the authority of the discourse and how it came to have the right to pronounce truth in some self-defined region of experience, how this right is preserved, exercised and reproduced. For example, what systems are allowed for education, association, and advancement of members of the discourse?

What kinds of practices or discourses had to be in place before the discourse of childhood lead poisoning could be constructed? (i.e. what were the contingencies that allowed this discourse to take place?)

What was known about childhood lead poisoning in Canada in the early 1990’s? Whose voice was heard, and whose voice was not heard in the formation of the discourse?

What are conflicting groups of people saying within the discourse? Who gets listened to most often and why?

Who is provided the status to speak on lead poisoning in Canada? (i.e. who developed the taxonomies?). How and where did institutions become awarded the right to pronounce truth and how is this right preserved, exercised and reproduced?

How are the opinions of those with authority defended?

What power struggles or turf battles occurred and what was the outcome?

Are there social economic issues involved in the evolution of the discourses?

Are there any marginalized ways of generating discourse?

How do they differ from the dominant way of generating discourse?

C. Axis of value or justification: Describes systems of regulation, organization, normalization, and punishment. The discourse constitutes something by describing it. Policing functions to maintain the range of normal that it has described, through power/knowledge control strategies.
What social practices and power arrangements are necessary for the discourse of CLP to continue?

What apparatuses maintain the dominant discourse? (i.e. epideictic rhetoric, gaps in the episteme, is the research cited by others)

Mills (2003, p. 58) identifies the following as “internal exclusions” to discourse. Does the discourse employ any of these devices?

   a) commentary – writing about another’s statement i.e. literary criticism
   b) author function allows us to group or classify discourse
   c) disciplinary boundary: limits our place on a subject area – proscribe what can be counted as possible, disciplines allow for the production of new propositions but within tightly defined limits
   d) the rarefaction of the speaking subject – less about imparting knowledge and more about institutionalization of discourse

How is the existence of the discourse necessary and to whom? For example, does the discourse reinforce belief in Canada’s social safety net?

How does the discourse justify the technologies of power that it constructs for its purposes?

How does the discourse justify suppressing other discourses that challenge its dominance in pronouncing truth?

How are the technologies of power/knowledge justified by the discourse?

What justification is provided for the punishment of participants?

How is the suppression of competing discourse justified?

What is the justification provided by the discourse for its position as a pronouncer of truth?

References


APPENDIX G

Titles from Canadian public health discourse on lead as retrieved from two journals: The *Canadian Journal of Public Health* and the *Canadian Medical Association Journal* (1930-2008)
<table>
<thead>
<tr>
<th>Genre</th>
<th>Author/Year</th>
<th>Title</th>
<th>Cited by</th>
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</thead>
<tbody>
<tr>
<td>Case studies</td>
<td>(Ross &amp; Brown, 1935)</td>
<td>Poisoning common in children</td>
<td>2</td>
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<tr>
<td>Occupational Exposure/Smelter Community Investigations</td>
<td>(Nosal &amp; Wilhelm, 1990)</td>
<td>Lead toxicity in the shipbreaking industry: the Ontario experience</td>
<td>3</td>
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<tr>
<td></td>
<td>(Hertzman et al., 1991)</td>
<td>Childhood lead exposure in Trail revisited</td>
<td>4,5,6,7,9</td>
</tr>
<tr>
<td></td>
<td>(Létourneau G. &amp; Gagné, 1992)</td>
<td>Blood lead level in children living close to a smelter area: 10 years later</td>
<td>3,8,9</td>
</tr>
<tr>
<td></td>
<td>(Kosatsky &amp; Boivin, 1994)</td>
<td>Blood lead levels in children and pregnant women living near a lead-reclamation plant</td>
<td>10</td>
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<td></td>
<td>(Kelly et al., 1994)</td>
<td>Trace element analysis of soils collected near a lead/zinc smelter</td>
<td>11</td>
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<tr>
<td></td>
<td>(Gagné, 1994)</td>
<td>Blood lead levels in Noranda children following removal of smelter-contaminated yard soil</td>
<td>8,9</td>
</tr>
<tr>
<td></td>
<td>(Hilts et al., 1995)</td>
<td>A controlled trial of the effect of HEPA vacuuming on childhood lead exposure</td>
<td>4</td>
</tr>
<tr>
<td>Cross sectional Investigations</td>
<td>(Peter &amp; Bourdeau, 1983)</td>
<td>Screening for undue lead absorption: Correlation between lead and erythrocyte protoporphyrin</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>(Smith &amp; Rea, 1995)</td>
<td>Low blood lead levels in Northern Ontario - what now?</td>
<td>12,13,14,15,16,9</td>
</tr>
<tr>
<td>Commentary/letters/review articles</td>
<td>(Kosatsky, 1992)</td>
<td>Blood lead levels in children. [Letter].</td>
<td>17,8,18</td>
</tr>
<tr>
<td></td>
<td>(Levallois et al., 1994)</td>
<td>Is there a need for systematic blood lead screening in Canadian children?</td>
<td>19</td>
</tr>
</tbody>
</table>

4 The CJPH was registered in 1910 as "The Public Health Journal". The title changed to the "Canadian Public Health Journal" in 1929, and to its current name, the "Canadian Journal of Public Health" in 1943
### Canadian Medical Association Journal

<table>
<thead>
<tr>
<th>Genre</th>
<th>Author/Year</th>
<th>Title</th>
<th>Cited by</th>
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<tbody>
<tr>
<td>Case Studies</td>
<td>Mitchell, 1932</td>
<td>Lead poisoning in children</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rabinowitch, 1934</td>
<td>Mercurial poisoning</td>
<td>58 cases of lead poisoning out of 309 total poisoning cases reported at Montreal General Hospital</td>
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<td></td>
<td>Wilcox, 1939</td>
<td>Lead poisoning from &quot;canned heat&quot;</td>
<td></td>
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<td></td>
<td>Penfield &amp; Paine, 1955</td>
<td>Results of surgery for focal epileptic seizures</td>
<td>2 cases of lead poisoning from ingesting lead-based paint – miscellaneous cause of epilepsy</td>
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<td></td>
<td>Wyllie, 1954</td>
<td>A family outbreak of lead poisoning from the burning of storage battery casings</td>
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<td></td>
<td>Harris, 1958</td>
<td>A comparison of intravenous calcium disodium versenate and oral penicillamine in promoting elimination of lead</td>
<td>22,23</td>
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<td>Commentary</td>
<td>Childe, 1947</td>
<td>Bone lesions encountered during infancy</td>
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<td>Ng &amp; Martin, 1977</td>
<td>Lead poisoning from lead-soldered electric kettles</td>
<td>24,25</td>
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<td></td>
<td>Tenenbein, 1990</td>
<td>Does lead poisoning occur in Canadian children?</td>
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<td></td>
<td>Lavoie &amp; Bailey, 2004</td>
<td>Lead poisoning from &quot;lead-free&quot; paint</td>
<td>30,2,31</td>
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<td></td>
<td>Ross, 1982</td>
<td>Gasoline sniffing and lead encephalopathy</td>
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<td>Pontifex &amp; Garg, 1985</td>
<td>Lead poisoning from an Asian Indian folk remedy</td>
<td>32,33,3</td>
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<tr>
<td>Case-control investigations</td>
<td>(Wilson &amp; Card, 1986)</td>
<td>Lead poisoning: unusual manifestation and unusual source</td>
<td></td>
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<td></td>
<td>(Paton, 1982)</td>
<td>Fluorometric assay of erythrocyte protoporphyrin: simple screening test for lead poisoning and iron deficiency</td>
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<tr>
<td>Cross-sectional investigations</td>
<td>(Jin et al., 1995a)</td>
<td>Blood lead levels in children aged 24 to 36 months in Vancouver</td>
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<tr>
<td></td>
<td>(Koren et al., 1990)</td>
<td>Lead exposure among mothers and their newborns in Toronto</td>
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<td>Occupational Exposure/Smelter</td>
<td>(Levallois et al., 1991)</td>
<td>Blood lead levels in children and pregnant women living near a lead-reclamation plant</td>
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<td>Community Investigations</td>
<td>(Editor, 1933)</td>
<td>Lead-poisoning in children [editorial]</td>
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<td>(Lynch, 1949)</td>
<td>The problem of lead poisoning</td>
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<td>(Editor, 1952)</td>
<td>Lead poisoning in childhood. [editorial]</td>
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<td>(McNeely, 1978)</td>
<td>Chemical monitoring of exposure to heavy metals</td>
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<td>(Schmitt et al., 1979)</td>
<td>Surface soil as a potential source of lead exposure for young children</td>
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<td>(Coodin et al., 1980)</td>
<td>Riposte to ‘Environmental lead and young children.’ [Letter]</td>
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<td>(Godolphin et al., 1993a)</td>
<td>Blood lead in Canadian children: a current perspective</td>
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<td>Letters generated by Godolphin et al., 1993a</td>
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<td>• (Godolphin et al., 1993b)</td>
<td>Blood lead levels in children: The authors respond [letter].</td>
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<td>• (Lockitch, 1993)</td>
<td>Blood lead levels in children. [Letter in response to]</td>
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Letters generated by Jin et al., 1995a

• (Gelpke, 1995) Blood lead levels in children: Poor methods or good news? [Letter in response to Jin et al., 1995]

• (Jin et al., 1995b) The authors respond [Letter in response to Gelpke & Johnstone]

• (Jin et al., 1995c) The authors respond [Letter in response to Rowe Larsen and Schmitt].

• (Rowe et al., 1995) Blood lead levels in children: conclusions questioned [Letter in response to Jin et al., 1995]

(Schmitt et al., 1996) Could zinc help protect children from lead poisoning?

(Tenenbein, 1996) Review of “Turning lead into gold: How heavy metal poisoning can affect your child and how to prevent and treat it”
(Winneke, 1996) Zinc to prevent lead poisoning

(Sanborn et al., 2002) Identifying and managing adverse environmental health effects: 3. Lead exposure

(Marshall et al., 2002) Identifying and managing adverse environmental health effects: 1. Taking an exposure history

Letters generated by Lavoie & Bailey, 2004

• (Bailey, 2004) Lead poisoning in children. [Response letter]
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(Brodkin et al., 2007) Lead and mercury exposures: interpretation and action

(Payne, 2008) Lead in drinking water

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(29) Valiquette L, Kosatsky T. Portrait of Montreal children with high blood lead levels identified through community-wide review of laboratory records. *Chronic Diseases in Canada* 1995;16(2).


