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Abstract

Background
Lead is a known toxicant that occurs naturally in the environment. Bisphenol A (BPA) is an industrial chemical used primarily in polycarbonate plastic and epoxy resins. It has been 30 years since lead exposure was measured at a national level, and it is the first time for a national assessment of BPA exposure.

Data and methods
Data are from the 2007-2009 Canadian Health Measure Survey. Lead in whole blood (PbB) and urinary BPA were measured in 5,319 and 5,476 respondents aged 6 to 79, respectively. Geometric means (GMs) are presented by age group and sex for PbB (µg/dL), volume-based BPA (µg/L), and creatinine-standardized BPA (µg/g creatinine). Adjusted least squares geometric means (LSGMs) for PbB and BPA are presented by selected covariates.

Results
PbB was detected in 100% of the population, with a GM concentration of 1.34 µg/dL. Adults aged 60 to 79 and males had significantly higher GM PbB concentrations. Lower household income, being born outside Canada, living in a dwelling at least 50 years old, current or former smoking, and drinking alcohol at least once a week were associated with higher PbB concentrations. Urinary BPA was detected in 91% of the population, with a GM concentration of 1.16 µg/L (1.40 µg/g creatinine). Children aged 6 to 11 had significantly higher GM creatinine-standardized BPA concentrations than did other age groups.

Interpretation
Although PbB concentrations have declined dramatically since the 1970s, socio-demographic characteristics, the age of dwellings, and certain lifestyle behaviours are associated with higher levels. Given the short half-life of orally ingested BPA and the high frequency of detection, the CHMS data suggest continual widespread exposure in the Canadian population.

Keywords
biomonitoring, blood lead, detection, environmental exposure

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Every day, people are exposed to natural and man-made chemicals—in the air, food and water, and consumer products.[1] These chemicals can enter the body through ingestion, inhalation, and/or dermal contact. They may be essential nutrients or toxic compounds.[2] Human biomonitoring is an effective way to provide baseline information about levels of exposure to environmental chemicals, and can help determine usual exposure and changes over time.[1,3,4] It involves the direct measurement of chemicals or their metabolites in blood, urine, other bodily fluids or tissues. From March 2007 through February 2009, the Canadian Health Measures Survey (CHMS) collected biomonitoring data from a nationally representative sample of the population.

The CHMS was conducted by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada. As part of the physical examination component of the survey,[5] blood and urine samples were collected and analyzed for chronic and infectious diseases, nutritional biomarkers, general health, and a wide range of environmental chemicals and their metabolites.

This study presents estimates of concentrations of two environmental chemicals in the population aged 6 to 79 years: lead and bisphenol A (BPA). Lead is a metal with a relatively long half-life the use of which has declined steadily since the 1970s. BPA is a synthetic organic chemical with a relatively short half-life whose use has increased over the same period.

Canadians' exposure to lead has not been measured at the national level in 30 years.[6] Lead, a known toxicant,[7] occurs naturally in rock and soil, although exposure is often related to human activity. It is no longer added to

automotive gasoline or used as solder in food cans, and lead limits in paint have been reduced. However, it continues to be used in the refining and manufacturing of products such as lead acid car batteries and electronic equipment.[7] It is also found in consumer products such as plastic mini-blinds, toys and jewellery.[8-10]

Lead that has been released into the environment does not degrade to other substances, so its past use continues to contribute to human exposure.[7] Potential sources include ingestion of dust from lead-based paint on older buildings, ingestion of water distributed through lead pipes, and certain occupations or hobbies (soldering, renovation, pottery or ceramics, etc.).[11,12] Lifestyle factors such as smoking and alcohol consumption may also increase exposure.[7,13,14]

The amount of lead that is absorbed depends on its physical and chemical form and on the individual's characteristics including age and sex.[15] Once absorbed, lead circulates in the bloodstream and accumulates in tissues and bone, or is excreted, primarily in urine and feces.[16]

Blood lead (PbB) concentrations, which are commonly used to evaluate human exposure,[15] reflect recent exposure and may also represent past exposure as a result of lead mobilization from bone into blood.[17] The half-life of PbB is approximately one month, but the half-life of lead in bone can be decades long.[18]

High lead levels can increase the risk of brain[19,20] and kidney damage.[21,22] A PbB concentration at or above 10 µg/dL (≥0.48µmol/L) is considered an intervention level.[23] However, recent studies have found health effects among children at lower concentrations,[24-26] suggesting that there may be no obvious threshold.[25-27] Other researchers have suggested that the intervention level be reduced to 5 µg/dL (0.24 µmol/L)[28] or 2 µg/dL (0.10 µmol/L).[29]

The CHMS is the first national survey to measure Canadians' BPA exposure. BPA does not occur naturally in the environment; it is an industrial chemical used primarily in polycarbonate plastic (such as food containers and water bottles) and epoxy resins (protective linings for canned food and beverages, the inner coating on metal lids for glass jars and bottles, and dental sealants[16,30-32]). Exposure is mainly dietary, although BPA is in drinking water, soil, dust, air and consumer products.[16] It can migrate from food packaging, particularly when heated,[33,34] as well as from repeat-use containers.[16]

Orally ingested BPA is quickly absorbed from the gastrointestinal tract and metabolized in the liver to BPA-glucuronide (BPA-G), which is excreted in urine with a half-life of less than six hours.[35,36] Free BPA is considered to be the biologically active form,[37] and together with BPA-G, makes up total BPA.[38] However, because of the effectiveness of initial metabolism, studies suggest low systemic availability of free BPA after oral exposure.[39] Urine is considered the appropriate body fluid to assess exposure to total BPA.[40] Measuring total BPA may provide valuable information for exposure and risk assessment.[41]

BPA is recognized as an endocrine disruptor,[37] although its estrogenic potency is under debate.[42] Reproductive toxicity, including effects on fertility and development, has been identified as a key health effect of exposure to high concentrations.[32] Some studies suggest that repeated maternal exposure could elevate BPA exposure in utero[43] or in the newborn.[44] Although BPA may constitute a health risk,[37] no guidance values are currently available in Canada for urinary BPA.

Methods

Data source

The Canadian Health Measures Survey (CHMS) covers the household population aged 6 to 79 years. Data for this study are from cycle 1, which was conducted from March 2007 through February 2009 at 15 sites across the country. The sample represented 96.3% of the population. Full-time members of the Canadian Forces and residents of Crown lands, Indian reserves, institutions and certain remote regions were excluded.

The CHMS consisted of a face-to-face household interview and a subsequent visit to a mobile examination centre (MEC). The household interview gathered general demographic and socio-economic data and detailed health, nutrition and lifestyle information. Direct measurements were taken at the MEC, including the collection of blood and urine samples.[5] About 92% of respondents who attended the MEC had limited themselves to water for at least two hours before their appointment.

Of the households selected for the CHMS, 69.6% agreed to participate. Of these, 88.3% responded to the household questionnaire. And of those who completed that questionnaire, 84.9% reported to the MEC for the direct measurements, resulting in a total sample size of 5,604 respondents (Appendix Table A).

The overall response rate was 51.7%. Because two people were selected in some households, this overall rate is not the result of multiplying the household and person response rates. The survey weights produced by the CHMS were used to account for the different stages of non-response.[45]

Blood and urine collection

Blood for the lead analysis was taken from 5,319 respondents. A 6.0 mL lavender K2-EDTA vacutainer of whole blood specimen was collected by venipuncture. Urine for the BPA analysis was taken from 5,476 respondents. The spot midstream urine samples were collected at the beginning of each appointment in a 120 ml container. About 59% of respondents provided a urine sample before noon, and 41%, at noon or later.

Standardized procedures were developed for the collection of blood and urine specimens, processing and aliquoting and for shipping biospecimens to the testing laboratory at the Institut national de santé publique du Québec (INSPQ). The INSPQ is accredited under ISO 17025 and followed standardized procedures that were developed for

every assay and technique performed in its laboratory.

Field blanks were performed at all 15 sites to prevent baseline contamination from the collection, handling procedures, site environment or material used. Contamination in the field blanks was found at site 7 (0.08 µg/dL) for PbB. No significant levels of contamination for BPA were found at any site.

PbB analysis

Whole blood samples were diluted in a basic solution containing octylphenol ethoxylate and ammonia. They were analysed for PbB by inductively coupled plasma mass spectrometry (ICP-MS), Perkin Elmer Sciex, Elan DRC II. The accuracy of the analytical testing of PbB was monitored by sending blind reference quality control materials as a form of proficiency testing to the reference laboratory. Three levels of various acceptable ranges (7.45-11.39/ 21.95-32.92/ 39.96-60.04 µg/dL) of PbB controls were sent from each of the 15 sites. All results were within the acceptable ranges.

The limit of detection (LOD) for PbB, as determined by INSPQ, was established at 0.021 µg/dL (0.001 µmol/L).

Urinary BPA analysis

After collection at the MEC, urine samples were frozen at -20°C and shipped on dry ice to the laboratory. One hundred microlitres of urine were fortified with $^{13}C_{12}$-BPA and buffered to a pH 5. Samples were hydrolyzed with β-glucuronidase (Helix Pomatia type HP-2 of Sigma # G-7017 with activity >100000 units/ml and sulfatase activity of 7500 units/ml) for three hours at 37°C, then derivatized with pentafluorobenzyl bromide at 70ºC for 2 hours. The derivatized products were extracted with a mixture of dichloromethane-hexane. Evaporated extracts were re-dissolved and analyzed by gas chromatography (Agilent 6890 or 7890) coupled to tandem mass spectrometry detector (Waters Quattro Micro-GC), operating in MRM mode following negative chemical ionization (NCI). Total BPA was measured by this procedure. Special precautions were taken to minimize BPA contamination throughout the laboratory analysis. Contamination in the laboratory blanks (deionized water, derivatized) was subtracted from each analytical sequence. The LOD for BPA, as determined by INSPQ, was 0.2 µg/L.

Creatinine was measured using the Jaffé reaction.[46] Specimens with urine creatinine concentrations below 0.05 g/L (0.44 mmol/L) were excluded.[47]

Measures

PbB concentrations were calculated in conventional units (µg/dL) and in Système International (SI) units (µmol/L). Creatinine-standardized BPA concentrations (µg/g creatinine) were calculated by dividing the volume-based BPA measure (µg/L) by the urinary creatinine measure (g/L).[48] This resulted in a valid creatinine-standardized BPA measure for 5,462 respondents. Volume-based and creatinine-standardized BPA data are presented to allow comparisons with a wider range of results in the literature. While BPA may not be excreted through the same renal pathway as creatinine, creatinine standardization may still offer a reasonable biomarker for diuresis/ dilution.[49] Such a standardization procedure likely allows better comparison than volume concentrations in a reasonably homogenous group of individuals. However, comparisons between children and adults, or between men and women should be made with caution.[49]

Individuals whose PbB or urinary BPA concentration fell below the LOD were assigned a value of LOD/2.[50]

Covariates

In addition to age group and sex, household education level, household income and country of birth were examined in association with PbB and BPA concentrations (Appendix Table B). Five age groups were specified: 6 to 11, 12 to 19, 20 to 39, 40 to 59, and 60 to 79 years. The highest level of education for the household was determined based on the highest level attained by each household member and categorized into: less than secondary graduation, secondary graduation, some postsecondary, and postsecondary graduation. Household income quartiles ($25,000 or less, more than $25,000 to $41,000, more than $41,000 to $64,000, more than $64,000) were derived from the reported total household income adjusted for the number of people in the household. Country of birth was categorized as Canada or outside Canada. The association between PbB concentrations and age of the respondent's home, smoking status and alcohol use were examined. Age of dwelling was categorized as less than 20 years, 20 to less than 50 years, and 50 years or more. Smoking status was based on the respondents' reported smoking habits and categorized into never, former or current. Frequency of alcohol consumption was based on respondents' reported consumption in the past 12 months and categorized into: less than once a week, 1 to 3 times a week, 4 to 6 times a week and every day. Children younger than 12 years were not asked about smoking or alcohol consumption and were, therefore, not assigned to a category.

Four categories of PbB concentration were evaluated: 0 to less than 2 µg/dL; 2 to less than 5 µg/dL; 5 to less than 10 µg/dL; and 10 µg/dL or more. These cutpoints correspond to PbB concentrations that recent studies have associated with various health effects.[24-29]

The association between BPA and body mass index (BMI) was examined. At the MEC, each respondent's height and weight were measured. BMI was calculated as weight in kilograms divided by height in metres squared (kg/m^2). Respondents aged 18 years or older were classified as obese (30 kg/m^2 or more), overweight (25 to 29.9 kg/m^2), or neither overweight nor obese (less than 25 kg/m^2).[51] Children aged 6 to 17 years were classified into the same BMI categories based on definitions proposed by the International Obesity Task Force.[52]

Along with creatinine concentration, the time of day of urine collection (before noon; noon or later) was used as a control in the multivariable analysis of BPA.

Analytical techniques

Analyses were weighted using the CHMS survey weights generated by Statistics Canada.[45] The data were analyzed with SAS[53] and SUDAAN[54] software, using DDF=11 in the SUDAAN procedure statements. Proportions, geometric means (GMs), adjusted least squares geometric means (LSGMs) and their confidence intervals were calculated. A separate regression was run for each covariate, controlling for age group and sex, to estimate adjusted LSGM PbB concentrations. A similar approach was used to estimate adjusted LSGM BPA concentrations, with time of day of urine collection and creatinine concentration added as controls. Adjusting for time of urine collection helped address differences in BPA concentrations over the day based on the respondent's last meal or water consumption; controlling for creatinine addressed respondent differences in urine concentration/dilution.[49]

Because the concentrations of PbB, BPA and creatinine were not normally distributed, their log transformations were used in the regression models. Given the 11 degrees of freedom available for variance estimation, Satterwaite-adjusted statistics were used to test the significance of each regression model's coefficients. T-tests were used to compare GMs and LSGMs between categories. Statistical significance was set at $p < 0.05$, but was Bonferroni-adjusted depending on the number of comparisons.[55]

Table 1
Weighted geometric means of blood lead and urinary bisphenol A concentrations, by sex and age group, household population aged 6 to 79 years, Canada, March 2007 to February 2009

Age group (years)		% above limit of detection	Total			Males			Females		
			Geometric mean	95% confidence interval		Geometric mean	95% confidence interval		Geometric mean	95% confidence interval	
				from	to		from	to		from	to
Blood lead											
Total 6 to 79	**µg/dL**	**100.0**	**1.34**	**1.24**	**1.44**	**1.51**	**1.40**	**1.63**	**1.18‡**	**1.08**	**1.30**
	µmol/L	**100.0**	**0.06**	**0.06**	**0.07**	**0.07**	**0.07**	**0.08**	**0.06‡**	**0.05**	**0.06**
6 to 11†	µg/dL	100.0	0.90	0.81	0.99	0.92	0.85	0.99	0.87	0.77	0.99
	µmol/L	100.0	0.04	0.04	0.05	0.04	0.04	0.05	0.04	0.04	0.05
12 to 19	µg/dL	100.0	0.80*	0.74	0.85	0.88	0.82	0.96	0.71‡	0.66	0.77
	µmol/L	100.0	0.04*	0.04	0.04	0.04	0.04	0.05	0.03‡	0.03	0.04
20 to 39	µg/dL	99.9	1.12*	1.04	1.21	1.41	1.28	1.55	0.89‡	0.81	0.98
	µmol/L	99.9	0.05*	0.05	0.06	0.07	0.06	0.07	0.04‡	0.04	0.05
40 to 59	µg/dL	100.0	1.60*	1.46	1.75	1.74	1.57	1.92	1.47‡	1.31	1.65
	µmol/L	100.0	0.08*	0.07	0.08	0.08	0.08	0.09	0.07‡	0.06	0.08
60 to 79	µg/dL	100.0	2.08*	1.90	2.29	2.31	2.08	2.57	1.89‡	1.69	2.12
	µmol/L	100.0	0.10*	0.09	0.11	0.11	0.10	0.12	0.09‡	0.08	0.10
Urinary bisphenol A											
Total 6 to 79	**µg/L**	**90.7**	**1.16**	**1.08**	**1.24**	**1.29**	**1.20**	**1.38**	**1.04‡**	**0.94**	**1.16**
	µg/g creatinine	**90.7**	**1.40**	**1.32**	**1.49**	**1.28**	**1.18**	**1.38**	**1.54‡**	**1.44**	**1.64**
6 to 11†	µg/L	93.2	1.30	1.17	1.45	1.27	1.07	1.52	1.33	1.09	1.61
	µg/g creatinine	93.2	2.00	1.79	2.23	1.93	1.75	2.13	2.08	1.77	2.45
12 to 19	µg/L	93.8	1.50*	1.28	1.77	1.44	1.15	1.81	1.57	1.29	1.92
	µg/g creatinine	93.8	1.31*	1.17	1.46	1.22	1.02	1.45	1.41	1.28	1.56
20 to 39	µg/L	91.2	1.33	1.18	1.49	1.40	1.24	1.58	1.26	1.06	1.49
	µg/g creatinine	91.1	1.49*	1.41	1.57	1.30	1.17	1.45	1.70‡	1.53	1.89
40 to 59	µg/L	87.9	1.04*	0.96	1.12	1.25	1.13	1.39	0.86‡	0.77	0.96
	µg/g creatinine	87.9	1.33*	1.20	1.47	1.23	1.08	1.40	1.43‡	1.27	1.62
60 to 79	µg/L	88.3	0.90*	0.82	0.99	1.08	0.94	1.24	0.76‡	0.65	0.88
	µg/g creatinine	88.3	1.26*	1.14	1.40	1.14	1.02	1.27	1.39‡	1.23	1.56

† reference category
* significantly different from estimate for reference category ($p < 0.05$ adjusted for number of comparisons)
‡ significantly different from estimate for males ($p<0.05$)
Notes: The limit of detection for blood lead was 0.02072 µg/dL or 0.001 µmol/L. The limit of detection for urinary bisphenol A was 0.2 µg/L.
Source: 2007 to 2009 Canadian Health Measures Survey.

Results

Lead exposure

Lead (PbB) was detected in 100% of people aged 6 to 79 years; the geometric mean (GM) concentration was 1.34 µg/dL (Table 1). (Estimates in µmol/L are also presented in Table 1.)

The GM PbB concentration rose through the adult years from 1.12 µg/dL at ages 20 to 39 years to 2.08 µg/dL at ages 60 to 79 years. By contrast, concentrations were 0.9 µg/dL for children aged 6 to 11 years and 0.8 µg/dL at ages 12 to 19 years. The concentration at the 95th percentile ranged from 2.0 µg/dL for children aged 6 to 11 years to 5.2 µg/dL at ages 60 to 79 years (Figure 1).

Males had significantly higher GM PbB concentrations than did females in all age groups except 6 to 11 years.

Within their separate regression models and controlling for age group and sex, household income (p=0.005) and country of birth (p=0.001) were each associated with PbB concentration (Table 2). Residents of households in the lowest income quartile had a significantly higher least squares geometric mean (LSGM) PbB concentration (1.49 µg/dL) than did those in the highest income quartile (1.27 µg/dL). People born outside Canada had a significantly higher concentration (1.54 µg/dL) than did the Canadian-born (1.29 µg/dL). Household education (p=0.077) was not significantly associated with PbB concentration, although residents of households with the lowest level of education had a higher LSGM PbB concentration, compared with their reference group.

Age of dwelling (p=0.006), smoking status (p=0.000) and frequency of alcohol consumption (p=0.000) were each associated with PbB concentration, controlling for age group and sex. Residents of dwellings 50 or more years old had higher LSGM PbB concentrations than did residents of dwellings less than 20 years old. Concentrations were higher among current and former smokers than never smokers (excluding children aged 6 to 11 years). People who drank alcohol at least once a week had higher concentrations than did those who drank less frequently (excluding children aged 6 to 11 years).

Three-quarters (74%) of the population had PbB concentrations below 2 µg/dL, and 23% had concentrations from 2 to less than 5 µg/dL (Table 3). Another 2% had concentrations from 5 to less than 10 µg/dL, and fewer than 1% had concentrations greater than 10 µg/dL.

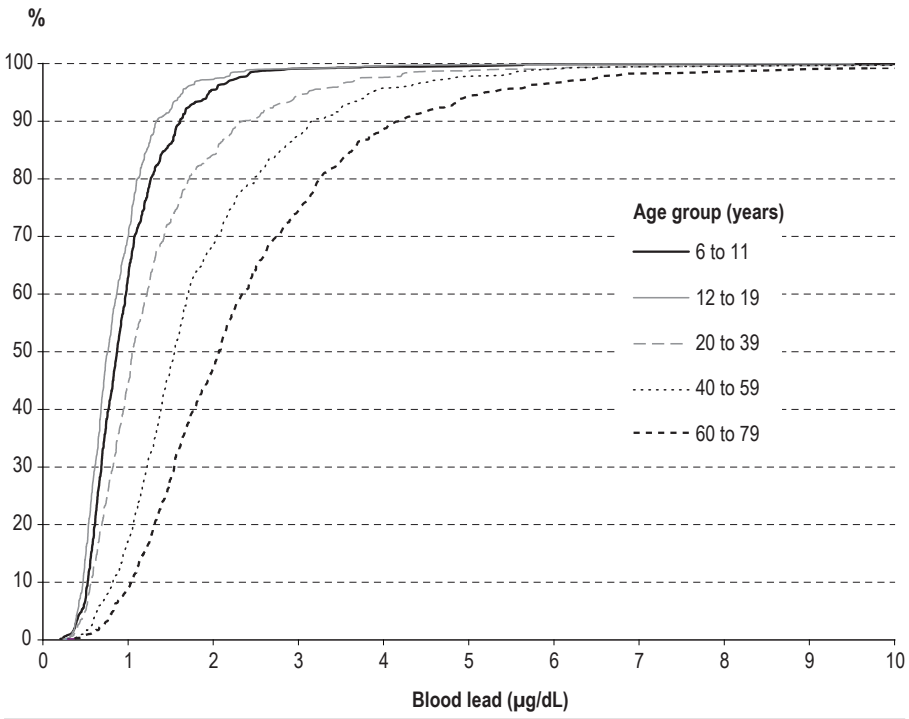
A large majority (at least 95%) of people younger than 20 years had PbB concentrations below 2 µg/dL. This percentage diminished to 47% by ages 60 to 79 years.

Bisphenol A exposure

Bisphenol A (BPA) was detected in 91% of people aged 6 to 79 years, with a volume-based GM concentration of 1.16 µg/L (Table 1). Compared with 6- to 11-year-olds, adolescents and teens (12 to 19) had a higher concentration, while adults aged 40 to 79 years had lower concentrations. Overall, males had a higher concentration (1.29 µg/L) than did females (1.04 µg/L), a difference largely attributable to higher concentrations among men aged 40 to 79. The distribution of BPA varied somewhat by age group: 12- to 19-year-olds generally had higher volume-based values than did people aged 60 to 79 years (Figure 2).

Standardizing BPA with urinary creatinine concentrations resulted in a GM BPA concentration of 1.40 µg/g creatinine for the total population aged 6 to 79 years. At 2.00 µg/g creatinine, 6- to 11-year-olds had a significantly higher concentration than did any other age group. Males generally had lower standardized GM BPA concentrations than did females, a difference that reached statistical significance at ages 20 to 79 years. Men's significantly higher concentrations of creatinine at ages 20 to 79 years (data not shown) helps explain their lower BPA-to-creatinine ratios. Conversely, 6- to-11-year-olds had the

Figure 1
Weighted cumulative distribution of blood lead concentrations, by age group, household population aged 6 to 79 years, Canada, March 2007 to February 2009



Source: 2007 to 2009 Canadian Health Measures Survey.

Table 2
Adjusted least squares geometric means (LSGMs) of blood lead concentrations, by selected characteristics, household population aged 6 to 79 years, Canada, March 2007 to February 2009

Characteristics	LSGM‡ (µg/dL)‡‡	95% confidence interval from	to
Highest level of education in household (p=0.077)			
Less than secondary graduation	1.49*	1.30	1.70
Secondary graduation	1.27	1.17	1.37
Some postsecondary	1.31	1.19	1.43
Postsecondary graduation†	1.32	1.22	1.43
Household income adjusted for household size†† (p=0.005)			
First quartile ($25,000 or less)	1.49*	1.35	1.63
Second quartile (more than $25,000 to $41,000)	1.32	1.17	1.48
Third quartile (more than $41,000 to $64,000)	1.27	1.19	1.35
Fourth quartile (more than $64,000)†	1.27	1.19	1.36
Country of birth (p=0.001)			
Outside Canada	1.54*	1.42	1.68
Canada†	1.29	1.19	1.39
Age of dwelling (p=0.006)			
Less than 20 years†	1.25	1.15	1.37
20 to less than 50 years	1.32	1.23	1.42
50 years or more	1.52*	1.36	1.70
Smoking status§ (p=0.000)			
Never†	1.27	1.16	1.39
Former	1.40*	1.30	1.50
Current	1.66*	1.52	1.80
Frequency of alcohol consumption in past 12 months§ (p=0.000)			
Less than once a week†	1.27	1.17	1.37
1 to 3 times a week	1.39*	1.28	1.52
4 to 6 times a week	1.72*	1.55	1.90
Daily	1.89*	1.75	2.03

† reference category
* significantly different from estimate for reference category (p < 0.05 adjusted for number of comparisons)
‡ adjusted for age group and sex
§ children younger than 12 excluded from model
†† adjusted using 40/30 formula; adjusted household incomes for all respondents ranked and divided into quartiles
‡‡ µg/dL converts to µmol/L by multiplying by 0.0483
Source: 2007 to 2009 Canadian Health Measures Survey.

lowest creatinine levels of all age groups (data not shown), which helps explain their higher creatinine-standardized BPA concentrations.

When the effects of age group, sex, time of day of urine collection and creatinine concentration were controlled, household education (p=0.293), household income (p=0.062), country of birth (p=0.473) and BMI status (p=0.311) were not significantly associated with BPA concentrations (Table 4). However, residents of households with the third highest level of education and those in the third income quartile had higher LSGM BPA concentrations, compared with their respective reference groups.

Discussion

Lead

Although lead was detected in 100% of the population, concentrations have fallen dramatically over the past 30 years. In 1978/1979, the Canada Health Survey estimated a GM PbB concentration of 4.79 µg/dL among people aged 6 to 79 years (excluding the 5% whose PbB concentrations were recorded as 0 µg/dL on the datafile).[56] By 2007-2009, the overall GM PbB concentration was 1.34 µg/dL, about a third of the 1978/1979 concentration. Furthermore, in 1978/1979, about 27% of Canadians aged 6 to 79 years had a PbB concentration at or above the intervention level of 10 µg/dL; in 2007-2009, the figure was less than 1%, reflecting the removal of major lead sources from the environment.

Recent analyses of 2007-2008 National Health and Nutrition Examination Survey (NHANES) data for the United States[57] found similar concentrations of PbB. The American GM PbB concentrations for the 6-to-11 and 12-to-19 age groups were 1.00 and 0.80 µg/dL, respectively, compared with 0.90 and 0.80 µg/dL for the same age groups in Canada. The 2007-2008 Second Korean National Human Exposure and Bio-monitoring Examination reported a GM PbB concentration of 1.72 µg/dL for all respondents aged 18 years or older,[58] again similar to that in Canada.

In this study, people aged 60 to 79 years had the highest PbB concentrations. Seniors have been exposed to higher environmental lead concentrations in the past, and their PbB concentrations might be particularly influenced by bone turnover due to aging.[59,60]

The significantly higher PbB concentrations among males than females are consistent with findings from other studies and may be due to occupational or hobbies' exposure or to a higher hematocrit level in men.[61,62] Although small sample sizes prevented examination of relationships between occupation and PbB concentrations, people (predominantly males) who reported welding or soldering at least once a week as a leisure activity had significantly higher GM PbB concentrations than did those who engaged in such activities less than once a month (data not shown).

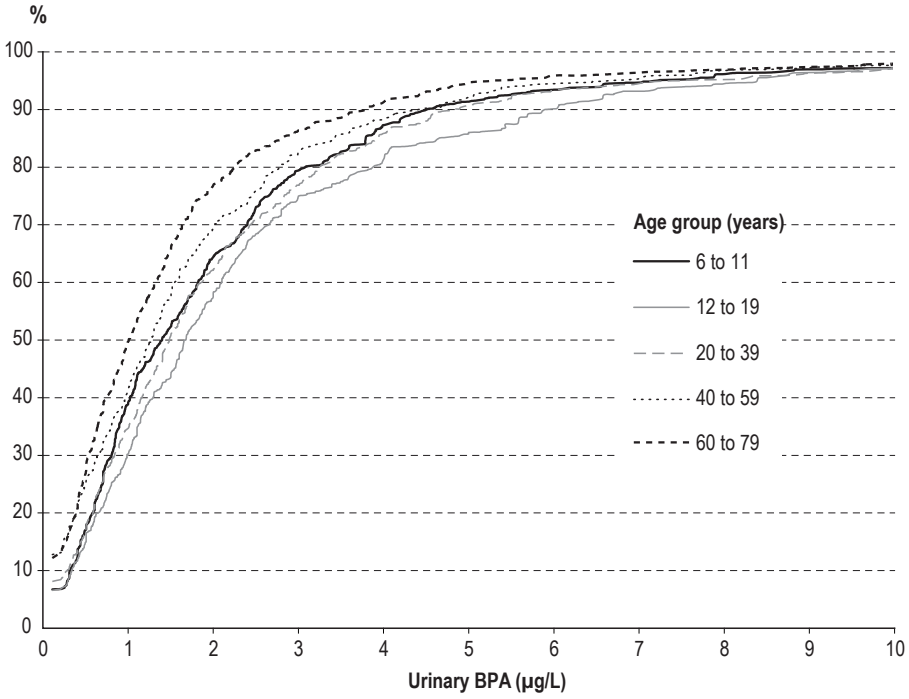
In this study, when age group and sex were taken into account, a higher LSGM PbB concentration was associated with lower household income. The association with socio-economic level has been observed in the United States and other countries,[61,63,64] and could be a consequence of higher exposure to environmental contamination and to greater absorption due to possible nutrition deficiencies.[64,65] The present study also found that people born outside Canada had a higher LSGM PbB concentration than did the Canadian-born.

Table 3
Percentage distribution by selected ranges of blood lead concentrations, by age group, household population aged 6 to 79 years, Canada, March 2007 to February 2009

Age group (years) and sex	0 to less than 2 µg/dL (0<0.10 µmol/L)			2 to less than 5 µg/dL (0.10<0.24 µmol/L)			5 to less than 10 µg/dL (0.24<0.48 µmol/L)			10 µg/dL or higher (≥0.48 µmol/L)		
	%	95% confidence interval from	to	%	95% confidence interval from	to	%	95% confidence interval from	to	%	95% confidence interval from	to
Total 6 to 79	**74.5**	**70.0**	**78.6**	**23.3**	**19.8**	**27.1**	**1.8[E]**	**1.1**	**3.0**	**< 1.0**	**...**	**...**
Age group												
6 to 11	95.5	91.3	97.7	< 8.6	...	...	< 1.4	...	...	< 0.7	...	...
12 to 19	97.2	94.9	98.5	2.5[E]	1.4	4.5	< 1.8	...	...	< 1.0	...	...
20 to 39	84.1*	79.7	87.6	14.7	11.4	18.7	< 3.3	...	...	< 2.9	...	...
40 to 59	68.4*	61.2	74.8	29.4	23.7	35.8	1.9[E]	1.0	3.6	< 1.9	...	...
60 to 79	47.0*	40.1	54.0	47.3	41.6	53.0	4.9[E]	2.8	8.4	< 2.0	...	...
Sex												
Males	69.7	64.2	74.7	27.2	23.0	31.8	2.5[E]	1.5	4.3	< 1.7	...	...
Females	79.4†	73.9	83.9	19.4†	15.1	24.5	1.2[E†]	0.6	2.3	< 0.8	...	...

* significantly different from estimate for 6 to 11 age group ($p < 0.05$ adjusted for number of comparisons)
† significantly different from estimate for males ($p<0.05$)
[E] use with caution (coefficient of variation 16.6% to 33.3%)
... not applicable
Note: If coefficient of variation of estimate exceeds 33%, estimate is indicated as being less than upper limit of 95% confidence interval.
Source: 2007 to 2009 Canadian Health Measures Survey.

Figure 2
Weighted cumulative distribution of urinary bisphenol A concentrations, by age group, household population aged 6 to 79 years, Canada, March 2007 to February 2009



Source: 2007 to 2009 Canadian Health Measures Survey.

The former may have lived in countries where environmental lead levels were higher than in North America.[66]

Residents of older dwellings had higher PbB concentrations, an association that has been well documented in earlier research.[7,67,68] Older buildings are more frequently contaminated by lead paint and might be connected to water main distribution systems with lead pipes.[11,12]

Regardless of age and sex, PbB concentrations among Canadians aged 12 to 79 years were associated with smoking behaviour and alcohol consumption. Daily smokers had higher LSGM PbB concentrations than did former smokers and those who had never smoked. The link with smoking has been found in many studies,[7,13,14,61,62,69] possibly related to the lead content in cigarettes.[70]

People who reported drinking alcohol once a week or more had higher LSGM PbB concentrations than did those who drank less frequently. Alcohol consumption has repeatedly been associated with higher PbB concentrations.[7,13,14,62,64] Lead is present in various types of alcohol, particularly wine.[71,72] Containers in which alcohol is stored and/or served may further increase the lead content.[73-75]

Table 4
Adjusted least squares geometric means (LSGMs) of urinary bisphenol A concentrations, by selected characteristics, household population aged 6 to 79 years, Canada, March 2007 to February 2009

Characteristics	LSGM§ (µg/L)	95% confidence interval	
		from	to
Highest level of education in household (p=0.293)			
Less than secondary graduation	1.14	0.90	1.43
Secondary graduation	1.18	1.03	1.36
Some postsecondary	1.33*	1.20	1.47
Postsecondary graduation†	1.15	1.08	1.22
Household income adjusted for household size‡ (p=0.062)			
First quartile ($25,000 or less)	1.22	1.08	1.39
Second quartile (more than $25,000 to $41,000)	1.16	1.07	1.27
Third quartile (more than $41,000 to $64,000)	1.24*	1.12	1.37
Fourth quartile (more than $64,000)†	1.04	0.97	1.12
Country of birth (p=0.473)			
Outside Canada	1.12	0.99	1.26
Canada†	1.17	1.09	1.26
BMI status (p=0.311)			
Neither overweight nor obese†	1.12	1.00	1.26
Overweight	1.18	1.12	1.25
Obese	1.21	1.14	1.30

† reference category
* significantly different from estimate for reference category (p < 0.05 adjusted for number of comparisons)
‡ adjusted using 40/30 formula; adjusted household incomes for all respondents ranked and divided into quartiles
§ adjusted for age group, sex, time of day of urine collection, and creatinine concentration; age groups 40 to 59 and 60 to 79 combined
Source: 2007 to 2009 Canadian Health Measures Survey.

Bisphenol A

Urinary bisphenol A (BPA), the sum of BPA-G and free BPA, was detected in 91% of Canadians aged 6 to 79. This is similar to NHANES results, where BPA was detected in 93% of the American population aged 6 or older.[76] The German Environmental Survey detected it in 99% of 3- to 14-year-olds.[77] Given the short half-life of orally ingested BPA and the high frequency of detection, these data suggest continual and widespread exposure to BPA.[78]

Because of differences in sample populations, in age groups and in laboratory analytical methods, comparisons with other studies must be made cautiously. Nonetheless, the volume-based GM BPA concentration of 1.16 µg/L for Canadians aged 6 to 79 is consistent with results in other studies of reference populations reporting mean or median concentrations of 1 to 3 µg/L.[40]

In Canada, males had significantly higher volume-based BPA concentrations than did females, but significantly lower creatinine-standardized urinary concentrations of BPA. This reversal in the sex difference from BPA (µg/L) to creatinine-standardized BPA (µg/g creatinine) is attributed to the higher urinary creatinine concentrations in males,[49] which were also observed in this study. Differences between the sexes in urinary BPA concentrations may reflect differences in exposure and in pharmacokinetic factors, the relevance of which is not currently known.[76]

The higher volume-based GM BPA concentrations in 12- to-19-year-olds and the significantly higher creatinine-standardized GM BPA concentrations in children aged 6 to 11 in this analysis are similar to those from NHANES.[76] The higher GM BPA concentrations in children may be due to their greater food consumption in relation to their body weight.[32] They may also reflect differences in absorption, distribution, metabolism, or excretion of BPA,[79] creatinine metabolism and excretion, or the use of products containing BPA.[32]

When the influences of age, sex, time of day of urine collection, and creatinine concentrations were controlled, no associations were found between volume-based BPA concentrations and household education, household income, country of birth, or BMI. However, certain categories of household income and education level did have LSGM BPA values that differed significantly from their respective reference groups. The lack of association between BMI and BPA concentrations in the overall study population corresponds to similar results reported for NHANES.[76]

Limitations

The overall response rate to the CHMS was slightly above 50%. While the survey weights ensured that the sample was representative of the target population, bias might exist if the PbB or BPA concentrations of non-respondents differed systematically from those of respondents.

Logistical and cost constraints in using mobile examination centres restricted the number of collection sites to 15 in the first CHMS cycle.[80] Given this sample design, it was not possible to include all covariates of interest in a single model. As future CHMS cycles become available, exploration of these and other interrelationships might be possible.

Only a small number of covariates was examined in conjunction with PbB. Research suggests that dietary and nutritional factors[81,82] and industry and occupation[7,62] are associated with PbB concentrations. The current analysis did not examine diet, a topic that could be explored in future work with the CHMS, but curent sample sizes limited the exploration of other variables.

BPA concentrations may vary according to food intake, time between urine collection and food consumption, and urine production rate. As well, BPA has a short half-life and urinary excretion is rapid, so the single spot urine collected from CHMS respondents may reflect only very recent exposures and cannot characterize average BPA exposure for any individual.[40] Nonetheless, the large number of single spot samples analyzed in the CHMS likely averages out variations in urinary BPA concentrations

and allows for reasonably accurate population-level exposure estimates.[40]

Conclusion

It has been 30 years since blood lead has been measured in the Canadian population, and until the CHMS, urinary BPA had never been measured in a national survey. Results of this analysis show that exposure to both lead and BPA is widespread.

Lead exposure has declined dramatically in the last decades. Nevertheless, socio-demographic characteristics, the age of dwellings and certain lifestyle behaviours are associated with higher concentrations. Because of the toxicity of lead at even very low concentrations,[25] there is still a place for ongoing monitoring and public health interventions.

Concentrations of urinary BPA tend to be higher in younger age groups. Additional research is needed to better identify factors that contribute to these higher concentrations and to understand factors associated with socio-demographic characteristics that may affect exposure to BPA.

These results can serve as baseline data to track trends as subsequent cycles of the CHMS become available. Future data combined with the first cycle will permit more in-depth examination of factors related to exposure to these two chemicals and the exploration of the association between exposure and health outcomes. ■

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Appendix

Table A
Unweighted sample sizes for respondents with valid blood lead and urinary bisphenol A concentrations, by age group and sex, household population aged 6 to 79 years, Canada, March 2007 to February 2009

	Blood lead		Bisphenol A (µg/L)		Bisphenol A (µg/g creatinine)	
Age group (years)	Males	Females	Males	Females	Males	Females
Total 6 to 79	**2,576**	**2,743**	**2,659**	**2,817**	**2,650**	**2,812**
6 to 11	459	451	524	507	522	506
12 to 19	489	456	504	476	503	475
20 to 39	514	651	513	652	511	650
40 to 59	577	643	577	642	573	641
60 to 79	537	542	541	540	541	540

Source: 2007 to 2009 Canadian Health Measures Survey.

Table B
Weighted characteristics of sample with valid blood lead and/or urinary bisphenol A concentrations, household population aged 6 to 79 years, Canada, March 2007 to February 2009

	All respondents		
		95% confidence interval	
Characteristics	%	from	to
Sex			
Males	49.8	49.7	49.9
Females	50.2	50.1	50.3
Age group (years)			
6 to 11	7.3	7.2	7.5
12 to 19	11.4	11.2	11.6
20 to 39	30.9	30.7	31.2
40 to 59	33.5	33.3	33.6
60 to 79	16.9	16.8	17.0
Highest level of education in household			
Less than secondary graduation	5.7	4.3	7.6
Secondary graduation	11.6	8.9	15.0
Some postsecondary	6.6	5.0	8.7
Postsecondary graduation	76.1	69.6	81.5
Household income adjusted for household size†			
First quartile ($25,000 or less)	25.7	21.7	30.1
Second quartile (more than $25,000 to $41,000)	24.1	21.1	27.3
Third quartile (more than $41,000 to $64,000)	25.3	22.3	28.5
Fourth quartile (more than $64,000)	25.0	20.9	29.5
Country of birth			
Outside Canada	21.5[E]	14.1	31.3
Canada	78.5	68.7	85.9
Age of dwelling			
Less than 20 years	33.0	25.4	41.7
20 to less than 50 years	43.5	36.3	51.1
50 years or more	23.4	16.2	32.6
Smoking status‡			
Never	52.6	49.4	55.8
Former	27.0	24.3	30.0
Current	20.3	17.9	23.0
Frequency of alcohol consumption in past 12 months‡			
Less than once a week	54.9	51.0	58.7
1 to 3 times a week	30.2	27.3	33.3
4 to 6 times a week	7.0	5.6	8.6
Daily	7.9	6.2	10.0
BMI status			
Neither overweight nor obese	45.0	40.2	49.9
Overweight	33.6	30.8	36.6
Obese	21.4	18.4	24.6

† adjusted using 40/30 formula; adjusted household incomes for all respondents ranked and divided into quartiles
‡ excludes children younger than 12
[E] use with caution (coefficient of variation 16.6% to 33.3%)
Source: 2007 to 2009 Canadian Health Measures Survey.