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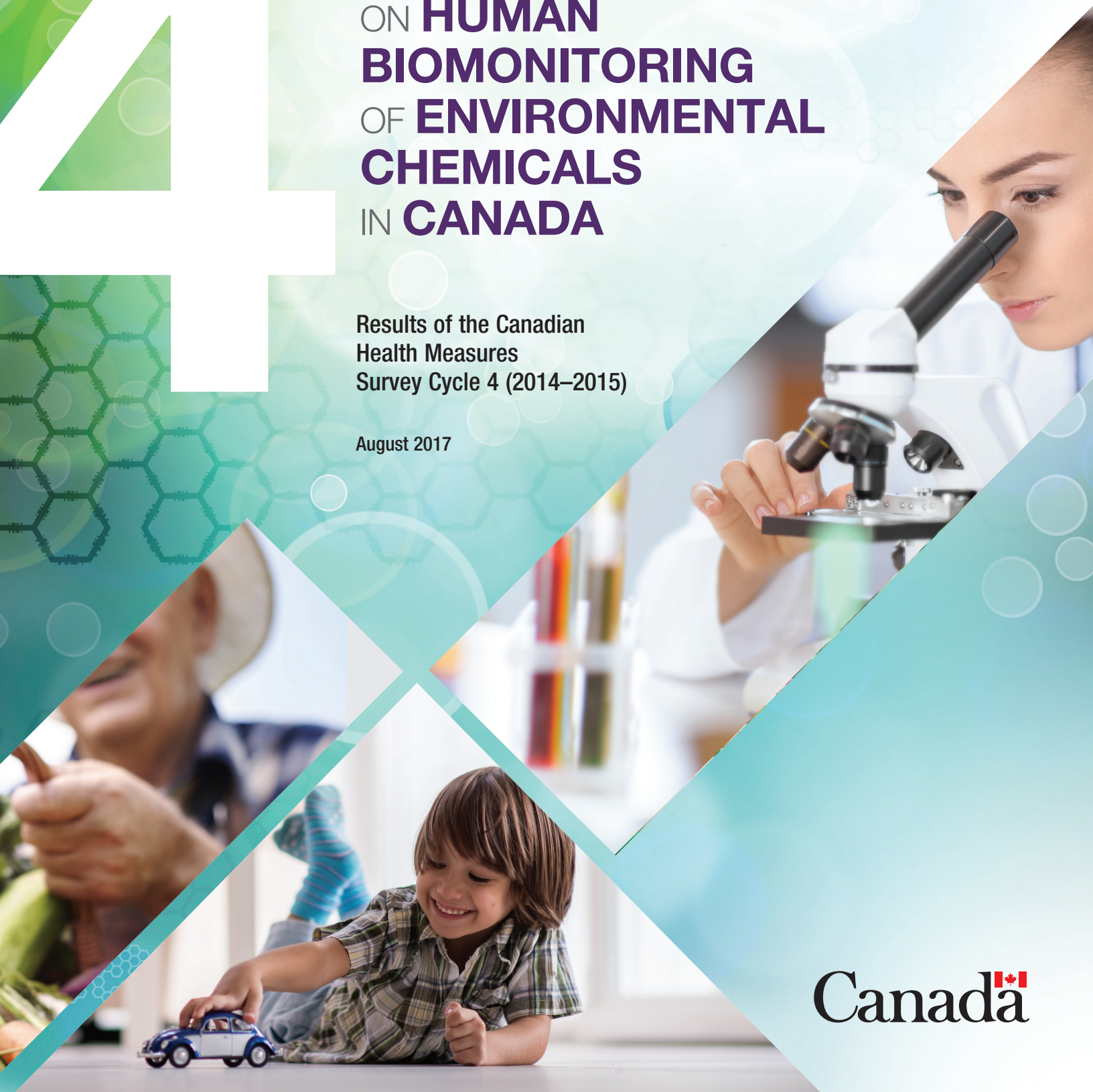
FOURTH REPORT ON HUMAN BIOMONITORING OF ENVIRONMENTAL CHEMICALS IN CANADA

Results of the Canadian Health Measures Survey Cycle 4 (2014–2015)

August 2017



Canada

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Fourth Report on Human Biomonitoring of Environmental Chemicals in Canada
is available on the Internet at the following address:
canada.ca/biomonitoring

Également disponible en français sous le titre :
Quatrième rapport sur la biosurveillance humaine des substances chimiques de l'environnement au Canada

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HC Pub.: 170058
Cat.: H128-1/10-601-4E-PDF
ISBN: 978-0-660-08527-2

Acknowledgements

The development and implementation of the biomonitoring component of the Canadian Health Measures Survey was achieved through extensive contributions of programs and staff across Health Canada and Statistics Canada. A special thank you goes out to the participants of the survey, without whom this study would not be possible.

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1 INTRODUCTION

These data tables present national data on concentrations of environmental chemicals in Canadians. These data were collected as part of the Canadian Health Measures Survey (CHMS), an ongoing national direct health measures survey. Statistics Canada, in partnership with Health Canada and the Public Health Agency of Canada, launched the CHMS in 2007 to collect health and wellness data and biological specimens on a nationally representative sample of Canadians. Biological specimens were analyzed for indicators of health status, chronic and infectious diseases, nutritional status, and environmental chemicals.

The CHMS biomonitoring component measures many environmental chemicals and/or their metabolites in blood and urine of survey participants. An environmental chemical can be defined as a chemical substance, either human-made or natural, that is present in the environment and to which humans may be exposed through media such as air, water, food, soil, dust, and consumer products.

The first *Report on Human Biomonitoring of Environmental Chemicals in Canada* was published in August 2010 and included baseline data for 92 environmental chemicals measured in cycle 1 (Health Canada, 2010). Data for cycle 1 of the CHMS were collected between March 2007 and February 2009 from approximately 5,600 Canadians aged 6–79 years at 15 sites across Canada.

The *Second Report on Human Biomonitoring of Environmental Chemicals in Canada* was published in April 2013 (Health Canada, 2013). Data for cycle 2 were collected between August 2009 and November 2011 from approximately 6,400 Canadians aged 3–79 years at 18 sites across Canada. Cycle 2 included 91 environmental chemicals, 42 of which were also measured in cycle 1.

The *Third Report on Human Biomonitoring of Environmental Chemicals in Canada* was published in July 2015 (Health Canada, 2015). Data for cycle 3 were collected between January 2012 and December 2013 from approximately 5,800 Canadians aged 3–79 years at 16 sites across Canada. Cycle 3 included 48 environmental chemicals, 32 of which were also measured in previous cycles.

Data for cycle 4 were collected between January 2014 and December 2015 from approximately 5,700 Canadians aged 3–79 years at 16 sites across Canada. Cycle 4 included 54 environmental chemicals.

A summary of the environmental chemicals measured in cycle 1, cycle 2, cycle 3, and cycle 4 of the CHMS is presented in the Table 1.1. Cycles 3 and 4 were paired so that the same chemicals were measured in both cycles.

Table 1.1

Summary of chemical groups measured in cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) of the Canadian Health Measures Survey

Chemical group	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Organochlorines	■			
Polybrominated flame retardants	■			
Polychlorinated biphenyls	■			
Chlorophenols	■	■		
Perfluoroalkyl substances	■	■		
Pesticides	■	■		
Phthalate metabolites	■	■		
Environmental phenols	■	■	■	■
Metals and trace elements	■	■	■	■
Nicotine metabolite	■	■	■	■
Polycyclic aromatic hydrocarbon metabolites		■	■	■
Volatile organic compounds: Benzene metabolites		■	■	■
Acrylamide			■	■
Parabens			NA[a]	■
Pesticides: Organophosphate pesticide metabolites			NA[a]	■
Volatile organic compounds			■	■

NA: Not available

a These chemicals were measured in cycle 3; however, the data are not yet available because ongoing quality assurance confirmation of the biospecimen analysis.

Collection for cycle 5 of the CHMS began in January 2016 and will be completed in late 2017. Planning for future cycles is under way.

In this report, the general CHMS survey design and implementation are described, with emphasis on the biomonitoring component. These sections are followed by descriptive summaries for each chemical, outlining the chemical's identity, common uses, occurrence in the environment, potential sources of exposure in the human population, toxicokinetics in the body, health effects, regulatory status, and existing Canadian biomonitoring data.

Data tables specific to each chemical are provided below the relevant text; the tables are broken down by age group and sex, and contain descriptive statistics on the distribution of blood and/or urine concentrations in the sample population. For chemicals that were also measured in previous cycles, data from all cycles are presented together in tables for ease of comparison. Data for chemicals that were only measured in cycle 1 and/or cycle 2 can be found in the first *Report on Human Biomonitoring of Environmental Chemicals in Canada* (Health Canada, 2010) or the *Second Report on Human Biomonitoring of Environmental Chemicals in Canada* (Health Canada, 2013). Downloadable tables are available through Canada's Open Government portal.

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2 OBJECTIVES

The primary purpose of the biomonitoring component of the Canadian Health Measures Survey (CHMS) is to provide human biomonitoring data to scientists and health and environment officials to aid in assessing exposure to environmental chemicals and in developing policies to reduce exposure to toxic chemicals for the protection of the health of Canadians.

Some specific uses of the CHMS biomonitoring data include the following:

- to establish baseline concentrations of chemicals in Canadians that could allow for comparisons with subpopulations in Canada and with other countries
- to establish baseline concentrations of chemicals to track trends in Canadians over time
- to provide information for setting priorities and taking action to protect the health of Canadians and to protect Canadians from exposure to environmental chemicals
- to assess the effectiveness of health and environmental risk management actions intended to reduce exposures and health risks from specific chemicals
- to support future research on the potential links between exposure to certain chemicals and specific health effects
- to contribute to international monitoring programs, such as the Stockholm Convention on Persistent Organic Pollutants

3 SURVEY DESIGN

The Canadian Health Measures Survey (CHMS) was designed as a cross-sectional survey to address important data gaps and limitations in existing health information in Canada. Its principal objective is to collect national-level baseline data on important indicators of Canadians' health status, including those pertaining to exposures to environmental chemicals. This information is important in understanding exposure to risk factors, detecting emerging trends in risk factors and exposures, and advancing health surveillance and research in Canada. Detailed descriptions of the CHMS rationale, survey design, sampling strategy, and mobile examination centre (MEC) operations and logistics for cycle 4 have been published (Labrecque and Quigley, 2016; Statistics Canada, 2017).

3.1 TARGET POPULATION

Cycle 4 of the CHMS targets the population aged 3–79 years living in one of the 10 provinces. The following groups are excluded from the survey's coverage: persons living in the three territories; persons living on reserves and other Aboriginal settlements in the provinces; full-time members of the Canadian Forces; the institutionalized population, and residents of certain remote regions. Altogether, these exclusions represent approximately 4% of the target population.

Although the CHMS is not able to provide representative data for the entire Canadian population, there are a number of surveys and research projects carried out in partnership with Health Canada that directly target some of these population gaps.

The First Nations Biomonitoring Initiative (FNBI) is a survey carried out by the Assembly of First Nations and Health Canada that seeks to establish baseline biomonitoring data for First Nations people living on-reserve south of the 60° parallel (AFN, 2013). Between 2009 and 2011, the FNBI measured the levels of 97 environmental chemicals in blood and urine samples collected from 503 participants living in 13 First Nation communities across Canada. The complete report has been published by the Assembly of First Nations (AFN, 2013).

In addition, numerous biomonitoring studies have been undertaken in Canada's North through the Northern Contaminants Program (NCP). The NCP, which is managed by federal government departments, provincial and territorial agencies, and Aboriginal organizations, was established in 1991 to respond to concerns about human exposure to contaminants in traditional diets of Northern Aboriginal peoples. The NCP provides funding for numerous individual studies undertaken in various regions of the North, including the Northwest Territories, Nunavut, and Nunavik (Quebec's North). More detailed information and results from these studies have been summarized in the Canadian Arctic Contaminants Assessment Reports and numerous scientific articles.

3.2 SAMPLE SIZE AND ALLOCATION

To meet the objective of producing reliable estimates at the national level by age group and sex, cycle 4 of the CHMS required a minimum sample of at least 5,700 participants. The participants were distributed among six age groups (3–5, 6–11, 12–19, 20–39, 40–59, and 60–79 years) and sex (except for 3–5 years), for a total of 11 groups. For the 3– to 5–year age group, the survey was not designed to provide estimates for the individual sexes.

3.3 SAMPLING STRATEGY

To meet the requirements of the CHMS, a multi–stage sampling strategy was used.

3.3.1 Sampling of Collection Sites

The CHMS required participants to report to a MEC and be able to travel to the centre within a reasonable period of time. For cycle 4, the 2011 Census geography was used to create 360 collection sites across the country. A geographic area with a population of at least 10,000 and a maximum participant travel distance of 75 kilometres (50 kilometres in urban areas and 75 kilometres in rural areas) were required for the location of collection sites. Areas not meeting these criteria were excluded.

A larger number of collection sites would have optimized the precision of the estimates. However, the logistical and cost constraints associated with the use of MECs restricted the number of collection sites to 16. The 16 collection sites were selected from within the five standard regional boundaries used by Statistics Canada (Atlantic, Quebec, Ontario, the Prairies, and British Columbia); they were allocated to these regions in proportion to the size of the population. Although not every province in Canada had a collection site, the CHMS sites were chosen to represent the Canadian population in all 10 provinces, east to west, including larger and smaller population densities. The collection sites selected for cycle 4 of the CHMS are listed in Table 3.3.1.1.

Table 3.3.1.1
Canadian Health Measures Survey cycle 4 (2014–2015) collection sites

Atlantic	Quebec	Ontario	Prairies	British Columbia
• Shelburne-Argyle, N.S. • South Fredericton, N.B.	• Saguenay • Sainte-Hyacinthe • West Laval • West Montréal	• Kitchener-Waterloo • Leeds-Grenville • North Toronto • Thunder Bay • West Hamilton • West Toronto	• Central and eastern Edmonton, Alta. • East Regina, Sask.	• Kelowna • Terrace-Kitimat

3.3.2 Dwelling and Participant Sampling

Within each site, dwellings with known household composition at the time of the 2011 Census, updated with the most recent information from administrative files, were stratified by age of household residents at the time of the survey, with the six age-group strata corresponding to the CHMS cycle 4 age groups (3–5, 6–11, 12–19, 20–39, 40–59, and 60–79 years). Within each site, a simple random sample of dwellings was selected in each stratum. Each selected dwelling was then contacted and asked to provide a list of current household members; this list was used to select the survey participants. One or two people were selected, depending on the household composition.

3.4 SELECTION OF ENVIRONMENTAL CHEMICALS

The process to determine the list of environmental chemicals to be included in cycles 3 and 4 of the CHMS built upon the existing consultation process used for cycle 2. The primary mechanism of consultation for cycle 2 was through a questionnaire distributed to key stakeholders with expertise or interest in human biomonitoring of environmental chemicals; the purpose was to define specifically what should be measured in blood and urine samples in the Canadian population. Key participants included various internal Health Canada branches and programs as well as a number of external groups, including other federal departments, provincial/territorial health and environment departments, industry groups, environment and health non-governmental organizations, and academics. Through this consultation, over 310 different chemicals and metabolites were nominated.

Selection was based on health risks; evidence of human exposure; existing data gaps; commitments under national and international treaties, conventions, and agreements; availability of standard laboratory analytical methods; and current and anticipated health policy development and implementations.

The following criteria were used as a general guide for identifying and selecting the environmental chemicals to include in the CHMS:

- seriousness of known or suspected health effects related to the substance
- need for public health actions related to the substance
- level of public concern about exposures and possible health effects related to the substance
- evidence of exposure of the Canadian population to the substance
- feasibility of collecting biological specimens in a national survey and associated burden on survey participants
- availability and efficiency of laboratory analytical methods
- costs of performing the test
- parity of selected chemicals with other national and international surveys and studies

Because fewer than 2 years had passed between the selection processes for cycle 2 and cycles 3 and 4, it was determined that an entirely new consultation was unnecessary; rather, the existing priority list from cycle 2 was used as the starting point for cycles 3 and 4. Chemicals that were included in the cycle 2 priority list, which could not previously be included for various reasons, were given highest priority for inclusion in cycles 3 and 4. In addition, environmental chemicals from cycles 1 and 2 considered to be high priorities were carried forward into cycles 3 and 4. Ultimately, the list was narrowed by the volume of biospecimens available from survey participants to conduct the analyses. Blood volume is generally limited; it is also required for analyses of chronic and infectious diseases and nutritional biomarkers. Thus, fewer environmental chemicals were measured in blood than in urine.

A full list of the chemicals measured in individual respondents in CHMS cycle 4 is presented in Table 3.4.1.

Table 3.4.1

Chemicals measured in individual respondents in the Canadian Health Measures Survey cycle 4, 2014–2015 (includes new chemicals and chemicals carried forward from previous cycles)

Chemical	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Acrylamide				
Acrylamide haemoglobin adduct				
Glycidamide haemoglobin adduct				
Environmental phenols				
Bisphenol A				
Triclosan				
Metals and trace elements				
Cadmium				
Fluoride				
Lead				
Mercury (inorganic)				
Mercury (total)				
Methylmercury				
Arsenic (speciated)				
Arsenate				
Arsenite				
Arsenocholine				
Arsenocholine and arsenobetaine				
Dimethylarsinic acid				
Monomethylarsonic acid				
Nicotine metabolite				
Cotinine				
Organophosphate pesticide metabolites				
Chlorpyrifos metabolite				
3,5,6-Trichloro-2-pyridinol			NA[a]	
Malathion metabolite				
Malathion dicarboxylic acid			NA[a]	
Parabens				
Methyl paraben			NA[a]	
Ethyl paraben			NA[a]	
Propyl paraben			NA[a]	
Butyl paraben			NA[a]	
Polycyclic aromatic hydrocarbon metabolites				
Benzo[*a*]pyrene metabolite				
3-Hydroxybenzo[*a*]pyrene				
Chrysene metabolites				
2-Hydroxychrysene				
3-Hydroxychrysene				
4-Hydroxychrysene				
6-Hydroxychrysene				
Fluoranthene metabolite				
3-Hydroxyfluoranthene				

Chemical	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Fluorene metabolites				
2-Hydroxyfluorene				
3-Hydroxyfluorene				
9-Hydroxyfluorene				
Naphthalene metabolites				
1-Hydroxynaphthalene				
2-Hydroxynaphthalene				
Phenanthrene metabolites				
1-Hydroxyphenanthrene				
2-Hydroxyphenanthrene				
3-Hydroxyphenanthrene				
4-Hydroxyphenanthrene				
9-Hydroxyphenanthrene				
Pyrene metabolite				
1-Hydroxypyrene				
Volatile organic compounds				
Benzene				
Ethylbenzene				
Styrene				
Tetrachloroethylene (perchloroethylene)				
Toluene				
Trichloroethylene				
Benzene metabolites				
trans,*trans*-Muconic acid				
S-Phenylmercapturic acid				
Trihalomethanes				
Bromodichloromethane				
Dibromochloromethane				
Tribromomethane (bromoform)				
Trichloromethane (chloroform)				
Xylenes				
m-Xylene and *p*-Xylene				
o-Xylene				

NA: Not available

a These chemicals were measured in cycle 3; however, the data are not yet available because of ongoing quality assurance confirmation of the biospecimen analysis.

Owing to the high cost of laboratory analyses, some environmental chemicals were not measured for all CHMS participants. The majority of the environmental chemicals were measured in a subsample of 2,500 participants aged 3–79 years, with the following exceptions: lead, cadmium, total mercury, and cotinine were measured in all participants; methylmercury was measured in 1,000 participants aged 20–79 years; and trihalomethanes and volatile organic compounds were measured in 2,500 participants aged 12–79 years. Further details on the subsampling for environmental chemicals are available in the Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4 (Statistics Canada, 2017) and in Sampling documentation for cycle 4 of the Canadian Health Measures Survey (Labrecque and Quigley, 2016).

Table 3.4.2
Environmental chemicals and chemical groups measured by age group

Measure	Matrix	Target sample size	Age (years)					
			3–5	6–11	12–19	20–39	40–59	60–79
Acrylamide	Blood	2,500	■	■	■	■	■	■
Environmental phenols	Urine	2,500	■	■	■	■	■	■
Metals and trace elements	Urine, blood	5,700	■	■	■	■	■	■
Metals and trace elements: Arsenic	Urine	2,500	■	■	■	■	■	■
Metals and trace elements Fluoride	Urine	2,500	■	■	■	■	■	■
Metals and trace elements: Methylmercury	Blood	1,000	—	—	—	■	■	■
Nicotine metabolite	Urine	5,700	■	■	■	■	■	■
Organophosphate pesticide metabolites	Urine	2,500	■	■	■	■	■	■
Parabens	Urine	2,500	■	■	■	■	■	■
Polycyclic aromatic hydrocarbon metabolites	Urine	2,500	■	■	■	■	■	■
Volatile organic compounds (VOCs)	Blood	2,500	—	—	■	■	■	■
VOCs: Benzene metabolites	Urine	2,500	■	■	■	■	■	■

3.5 ETHICAL CONSIDERATIONS

Personal information collected through the CHMS is protected under the federal *Statistics Act* (Canada, 1970-71-72). Under the Act, Statistics Canada is obliged to safeguard and to keep in trust the information it obtains from the Canadian public. Consequently, Statistics Canada has established a comprehensive framework of policies, procedures, and practices to protect confidential information against loss, theft, unauthorized access, disclosure, copying, or use; this includes physical, organizational, and technological measures. The steps taken by Statistics Canada to safeguard the information collected in the CHMS have been described previously (Day et al., 2007).

Ethics approval for all components of the CHMS was obtained from the Health Canada and Public Health Agency of Canada Research Ethics Board. Informed written consent for the MEC portion of the CHMS was obtained from participants older than 14 years of age. For younger children, a parent or legal guardian provided written consent, and the child provided assent. Participation in this survey was voluntary, and participants could opt out of any part of the survey at any time.

A strategy was developed to communicate results to survey participants with the advice and expert opinion of the CHMS Laboratory Advisory Committee, the Physician Advisory Committee, l'Institut national de santé publique du Québec (the reference laboratory performing some of the environmental chemical analyses), and Health Canada's Research Ethics Board (Day et al., 2007). For the environmental chemicals, only results for lead and mercury were actively reported to participants. However, participants could receive all other test results upon request to Statistics Canada.

More information on reporting to participants, including the ethical challenges encountered, can be found in Haines et al. (2011).

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4 FIELDWORK

Fieldwork for the Canadian Health Measures Survey (CHMS) cycle 4 took place over a period of 2 years from January 2014 to December 2015. Data were collected sequentially at 16 sites across Canada. The sites were ordered to take into account seasonality by region and the temporal effect, subject to operational and logistical constraints.

Statistics Canada mailed an advance letter and brochure to households that were selected as outlined in the Dwelling and Participant Sampling section. The mailing informed potential participants that they would be contacted for the survey's data collection.

Data were collected from consenting survey participants through a household personal interview, using a computer-assisted method, and a visit to a mobile examination centre (MEC) for physical measures and biospecimen collection. The field team consisted of household interviewers and the CHMS MEC staff, including trained health professionals who performed the physical measures testing (Statistics Canada, 2017).

Participants were first administered a household questionnaire in their home. Using a computer application, the interviewer randomly selected one or two participants and conducted separate 45- to 60-minute health interviews (Statistics Canada, 2017). The interviews collected demographic and socio-economic data and information about lifestyle, medical history, current health status, the environment, and housing conditions. At this time, the collection protocol for the tap water component of the survey was also initiated. Within approximately 2 weeks after the home visit, participants visited the MEC. Each MEC consisted of three trailers linked by enclosed pedestrian walkways. One trailer was for reception and contained an administration area and an examination room; the second trailer contained a laboratory, a phlebotomy (blood collection) room, and examination rooms; and the third trailer contained additional examination rooms. The MEC operated 7 days a week in order to complete approximately 350 visits at each site over 5 to 6 weeks and to accommodate participants' schedules (Statistics Canada, 2017). MEC appointments averaged about 2.5 hours. A parent or legal guardian accompanied children under 14 years of age. To maximize response rates, participants who were unable or unwilling to go to the MEC were offered the option of a home visit by members of the CHMS MEC staff to perform some of the physical measures and the biospecimen collection portion of the survey (Statistics Canada, 2017). At the end of the MEC visit, a subsample of households was asked to place a sampler in their home as part of the indoor air component of the survey.

At the start of the MEC visit, participants signed consent/assent forms prior to any testing and in most cases provided a urine sample immediately thereafter. For logistical purposes, spot samples were collected rather than 24-hour urine samples. The urine samples were collected using the first-catch urine, as opposed to the mid-stream urine collected in cycle 1. Guidelines were provided to participants asking them to abstain from urinating 2 hours prior to their MEC visit. Samples were collected in 120 mL urine specimen containers. Trained health professionals took physical health measurements such as height, weight, blood pressure, lung function, and physical fitness. A series of

screening questions were administered to participants to determine their eligibility for the various tests, including phlebotomy, based on pre-existing exclusion criteria (Statistics Canada, 2017). Blood specimens were drawn by a certified phlebotomist; the maximum amount depended upon the age of the participant. The approximate volume drawn from participants aged 3–5 years was 22.0 mL; 6–11 years, 28.5 mL; 12–13 years, 48.8 mL; 14–19 years, 52.8 mL; and 20–79 years, 72.8 mL.

All blood and urine specimens collected in the MEC were processed and aliquoted in the MEC. Biospecimens were stored temporarily in temperature-monitored freezers at –30°C until shipping, with the exception of blood samples collected for volatile organic compound analysis; these were refrigerated. Once a week, the specimens were shipped on dry ice or in monitored refrigerated conditions to the reference laboratory for analyses. Standardized operating procedures were developed for the collection of blood and urine specimens, processing and aliquoting procedures, as well as for shipping biospecimens to ensure adequate data quality and to standardize data collection. A priority sequence for laboratory analyses was established in the event that an insufficient volume of biospecimen was collected for complete analyses of the environmental chemicals as well as for analyses of infectious diseases, nutritional status, and chronic diseases. Details on the collection tubes, aliquot volumes, and priority testing are presented in Table 4.1.

Table 4.1

Urine and blood collection procedure for the environmental chemicals

Measure	Matrix	Collection Tube (size and type[a])	Optimal Volume[b]
Acrylamide	Whole Blood	4.0, 6.0, or 10mL[c] Lavender EDTA[d]	1.5 mL
Metals			1.0 mL
Methylmercury			1.8 mL[e]
Volatile organic compounds (VOCs)	Whole Blood	10 mL Washed Grey	10 mL
Creatinine	Urine	120 mL urine specimen container	0.5 mL
Fluoride			0.8 mL
Arsenic (speciated)			1.0 mL
Nicotine metabolite			0.8 mL
Inorganic mercury			1.5 mL
Environmental phenols			0.8 mL
Parabens			1.0 mL
Organophosphate pesticide metabolites			4.0 mL
Polycyclic aromatic hydrocarbon metabolites (PAHs) and benzene metabolites			12 mL
Specific gravity			0.3 mL

a Becton Dickinson Vacutainers were used for the collection of blood; VWR urine specimen containers were used for the collection of urine.

b Optimum sample volume sent to the reference laboratory

c 4.0 mL tubes used for respondents aged 3–5 years; 6.0 mL tubes used for respondents aged 6–11 years; 10 mL tubes used for respondents aged 20–79 years

d EDTA: ethylenediaminetetraacetic acid

e Methylmercury only collected in respondents aged 20–79 years

To maximize the reliability and validity of the data and to reduce systematic bias, the CHMS developed quality assurance and quality control protocols for all aspects of the fieldwork. Quality assurance for the MEC covered staff selection and training, instructions to respondents (pre-testing guidelines), and issues related to data collection. All staff had appropriate education and training for their respective positions. To ensure consistent measurement techniques, procedure manuals and training guides were developed in consultation with, and reviewed by, experts in the field. Quality control samples were done at each site, consisting of three field blanks per site (deionized water for most analytes), blind replicates (three pairs per site except blood VOCs and cotinine), and blind control samples (approximately six per site).

The quality control samples were sent to the laboratory with regular specimen shipments. Quality control sample results were sent to Statistics Canada's CHMS headquarters, along with all other respondent results, where they were assessed to determine the accuracy of the methodology based on the defined analyte concentration. The replicates were used to assess the precision of the analysis in pre-established acceptable ranges. If required, feedback was provided quickly to the reference laboratory for review and remedial action.

Beginning in cycle 2, a subsample of CHMS participants' households was selected for a component that involved sampling of indoor air over a 7-day period. A tap water sampling protocol was introduced in cycle 3 to complement the indoor air component. By sampling both indoor air and tap water in the home environment, where Canadians spend the majority of their time, two potential sources of exposure to environmental chemicals are captured.

Participants were asked to place the indoor air sampler in their household for 7 days in order to measure a number of VOCs. One indoor air sampler was given per selected household, along with a pencil, a postage-paid envelope, and an information sheet. After the 7-day collection period was over, participants mailed their indoor air sampler in the envelope provided to CASSEN Testing Laboratories where all indoor air analyses were performed.

The tap water sampling was carried out during the household interview by the interviewer and lasted for approximately 10 minutes. The objective of tap water collection was to determine the prevalence of and characterize the distribution of exposure to fluoride and to VOCs from tap water. Two samples were collected at each household and were shipped to the laboratories where the tap water analysis was performed. Fluoride samples were analyzed by the Laboratoire de santé publique du Québec whereas the VOC samples were analyzed by a Health Canada research laboratory.

Indoor air results were not reported back to respondents; however, the tap water test results were reported back to those who participated in both the household and the clinic portion of the survey. Reports included results for those chemicals for which either aesthetic quality or maximum acceptable concentration (MAC) guidelines have been established by the Federal-Provincial-Territorial Committee on Drinking Water. Aesthetic objectives address concentrations that could affect the taste, smell, or colour of water, while still being below the point at which health effects could appear whereas MACs are established on the basis of health considerations. Certain chemicals measured in the tap water sample do not have established guidelines; respondents were able to receive these results only upon request. If one or more of the tap water results was found to exceed a MAC, survey staff contacted the respondents to inform them of their result and to ask for their consent to share the result with provincial authorities.

A complete list of the substances measured in the indoor air and tap water samples is available in the *Canadian Health Measures Survey (CHMS) Content summary for cycles 1 to 8* (Statistics Canada, 2013). Further details on the indoor air study and tap water sampling are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Detailed descriptions of the CHMS MEC operations and logistics have been described previously in Bryan et al. (2007) and are presented in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle* 4 (Statistics Canada, 2017).

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5 LABORATORY ANALYSES

Laboratory analyses of environmental chemicals and creatinine were performed at analytical laboratories within Health Canada and l'Institut national de santé publique du Québec (INSPQ). Laboratories developed standardized operating procedures for the analytical methods used to measure environmental chemicals or their metabolites in biological samples. Analytical accuracy and precision of measurements were evaluated through rigorous method validation programs at each laboratory.

Internal quality control measures within each laboratory included the analysis of calibration standards, laboratory blanks, method blanks, and in-house quality control samples in each analytical batch. In addition, laboratories conducted periodical analyses of Standard Reference Materials/Certified Reference Materials when available. Quality assurance reviews were conducted on laboratory data on a regular basis to evaluate any issues in the batch processing and to identify inconsistencies in analytical results. Appropriate corrective measures were taken when required. As part of external quality control measures, laboratories participated in external quality control programs and inter-laboratory comparison studies when available. A table is provided with limits of detection for each method (Appendix A). The methods used in the analyses of the environmental chemicals and creatinine are described below.

5.1 ACRYLAMIDE

Whole blood was thawed at room temperature and reacted with modified Edman reagent (pentafluorophenyl isothiocyanate) for 2 hours at 55°C. The sample was purified using solid phase extraction on Isolute HM-N sorbent. Analytes were then eluted with diisopropyl ether/ethyl acetate/toluene (50/40/10 v/v/v) and the extract was evaporated under a stream of nitrogen. The sample was reconstituted in methanol/water (40/60 v/v) and analyzed using a Waters Acquity ultra performance liquid chromatograph (UPLC) system coupled to a Quattro Premier tandem mass spectrometer (Health Canada, 2014).

5.2 ENVIRONMENTAL PHENOLS

For the analysis of bisphenol A and triclosan, urine samples were subjected to enzymatic hydrolysis (ß-glucuronidase enzyme). The samples were then derivatized with pentafluorobenzyl bromide at 70°C for 2 hours. The derivatized products were extracted with a mixture of dichloromethane-hexane. Evaporated extracts were dissolved in the appropriate solvent and analyzed using an Agilent 6890 or 7890 gas chromatographic system coupled to a Waters Quattro Micro gas chromatograph (GC) tandem mass spectrometer. The mass spectrometer was operated in the negative ion chemical ionization mode and the analytes were quantified using multiple reaction monitoring (MRM) (INSPQ, 2014a). Free and hydrolyzed forms of bisphenol A were measured together by this procedure.

5.3 METALS AND TRACE ELEMENTS

5.3.1 Arsenic

Urine samples were diluted in ammonium carbonate solution and analyzed for arsenite (As3+), arsenate (As5+), monomethylarsonic acid, dimethylarsinic acid, and the sum of arsenobetaine and arsenocholine using a Waters Acquity UPLC coupled to a Varian 820-MS inductively coupled plasma-mass spectrometer (ICP-MS) system (INSPQ, 2009a). For arsenocholine, urine was diluted with formic acid and acetonitrile solution and analyzed on Waters Acquity UPLC coupled to a TQ-S tandem mass spectrometer (INSPQ, 2009a).

5.3.2 Cadmium, Lead, and Total Mercury

Blood samples were diluted in a basic solution containing octylphenol ethoxylate and ammonia. They were analyzed for cadmium, lead, and total mercury using a Perkin Elmer Sciex Elan DRC II ICP-MS. Matrix matched calibration was performed using blood from non-exposed individuals (INSPQ, 2010).

5.3.3 Fluoride

Urine samples were diluted with ionic adjustment buffer and analyzed using an Orion pH meter with a fluoride ion selective electrode (Orion Research Inc.) (INSPQ, 2009b).

5.3.4 Inorganic Mercury

Urine was digested in nitric acid at 50°C, diluted, and analyzed for inorganic mercury on a Perkin Elmer FIMS 100 (cold vapour system). Matrix matched calibration was performed using urine from non-exposed individuals (INSPQ, 2009c).

5.3.5 Methylmercury

Methylmercury was extracted by sonication from whole blood using an L-cysteine acid solution. After centrifugation, proteins were precipitated using acetonitrile. The supernatant was extracted using a micro coaxial solid phase extraction cartridge. The extract was evaporated to dryness, reconstituted in the appropriate solvent, and analyzed using a Waters Acquity UPLC coupled to a Varian 820-MS ICP-MS (INSPQ, 2011).

5.4 NICOTINE METABOLITE

Free cotinine was measured in participants aged 3–11 years (INSPQ, 2009d), and free cotinine and other tobacco biomarkers were measured in participants aged 12–79 years (INSPQ, 2015a). In both methods, free cotinine was recovered from urine samples by solid-phase extraction on an automated Janus workstation. Deuterated cotinine was used as the internal standard. The extract was redissolved in the mobile phase, analyzed using a Waters Acquity UPLC coupled to a Waters Quattro Premier XE tandem mass spectrometer with an electrospray ionization source operating in positive ion mode, and the analytes were quantified using MRM. Data from the two methods were combined and are presented together in tables for the total population aged 3–79 years.

5.5 ORGANOPHOSPHATE PESTICIDE METABOLITES

Malathion dicarboxylic acid (DCA) was measured in urine. The analyte was spiked with DCA analogue isotopically labeled with carbon 13. The two compounds were extracted on an ion-exchange cartridge, eluted, evaporated to dryness, resuspended in ethyl acetate, and then derivatized with MTBSTFA and analyzed using an Agilent 7890 GC coupled to an Agilent 7000B triple-quad tandem mass spectrometer with ions measured in MRM mode with a source in electron ionization mode (INSPQ, 2015b).

The total form (conjugated and free) of 3,5,6-trichloro-2-pyridinol (TCPy) was measured in urine. The urine sample was spiked with TCPy analogue isotopically labeled with carbon 13 and hydrolyzed at 37°C with the enzyme ß-glucuronidase/arylsulfatase. The compound and its isotopically labeled analogue were derivatized at 60°C in the presence of dansyl chloride. The compounds were then extracted with hexane. The extracts were dried, resuspended in an acetonitrile:MeOH:water mixture, and analyzed using a Waters Acquity ultrahigh performance liquid

chromatograph coupled to a Waters Xevo TQ-S tandem mass spectrometer in MRM mode with an electrospray source in positive mode (INSPQ, 2015c).

5.6 PARABENS

For the analysis of butyl paraben, ethyl paraben, methyl paraben, and propyl paraben, urine samples were subjected to enzymatic hydrolysis (ß-glucuronidase enzyme). The samples were then acidified and preconcentrated by Solid Phase Extraction. Evaporated extracts were dissolved in the appropriate solvent and analyzed using a Waters Acquity UPLC coupled to a Waters Quattro Premier XE tandem mass spectrometer. The mass spectrometer was operated in the negative ion electrospray ionization mode and the analytes were quantified using MRM. Free and hydrolyzed forms of parabens in urine were measured together by this procedure (Health Canada, 2016).

5.7 POLYCYCLIC AROMATIC HYDROCARBON METABOLITES

For the analysis of polycyclic aromatic hydrocarbon metabolites (3-hydroxybenzo[*a*] pyrene, 2-hydroxychrysene, 3-hydroxychrysene, 4-hydroxychrysene, 6-hydroxychrysene, 3-hydroxyfluoranthene, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxynaphthalene, 2-hydroxynaphthalene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3-hydroxyphenanthrene, 4-hydroxyphenanthrene, 9-hydroxyphenanthrene, and 1-hydroxypyrene), urine samples were hydrolyzed using ß-glucuronidase enzymatic solution and extracted with an organic solvent at neutral pH. The extracts were evaporated, derivatized with *N*-methyl-*N*-(trimethylsilyl)-trifluoroacetamide, and analyzed using an Agilent 7890 GC coupled to an Agilent 7000B triple-quad tandem mass spectrometer operating in electron impact ionization mode. Analytes were quantified using MRM (INSPQ, 2014b).

5.8 VOLATILE ORGANIC COMPOUNDS

Whole blood samples were withdrawn using a previously cleaned air-tight syringe, transferred to a glass vial, and a mixture of isotopically labelled analogs was added. The vial was crimp-sealed and placed in a temperature-controlled autosampler tray. The samples were maintained at 40°C with continuous mixing. The analytes (benzene, toluene, ethylbenzene, *m*-xylene, *o*-xylene, *p*-xylene, chloroform, bromoform, bromodichloromethane, dibromochloromethane, trichloroethane, tetrachloroethene, and styrene) were extracted by inserting a solid phase microextraction fiber into the vial headspace. After extraction, the fiber was transferred to a heated GC inlet where the analytes were rapidly desorbed off the fiber. The analytes were focused using a Thermo Fisher Scientific cryotrap (Cryotrap 915) and analyzed using a Thermo Fisher Scientific TRACE™ ultra GC coupled to a TSQ Quantum XLS mass spectrometer equipped with electron ionization source. The analytes were quantified in selected reaction monitoring mode (Aranda-Rodriguez, 2015; Health Canada, 2012).

5.8.1 Benzene Metabolites

Benzene metabolites (*trans*,*trans*-muconic acid and *S*-phenylmercapturic acid) were extracted from urine using a hydrophilic-lipophilic-balanced solid-phase extraction cartridge on an automated Janus workstation. The extracts were evaporated to dryness, reconstituted in the mobile phase, and analyzed using a Waters Acquity UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer operated in negative ion mode (INSPQ, 2014c).

5.9 CREATININE

Creatinine was measured in urine using the colorimetric end-point Jaffe method. An alkaline solution of sodium picrate reacts with creatinine in urine to form a red Janovski complex using Microgenics DRI® Creatinine-Detect® reagents (#917). The absorbance was read at 505 nm on a Hitachi 917 chemistry autoanalyzer (INSPQ, 2008).

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6 STATISTICAL DATA ANALYSES

Descriptive statistics on the concentrations of environmental chemicals in the blood and urine of Canadians, aged 3–79 years, were generated using the Statistical Analysis System software (SAS Institute Inc., version 9.2, 2008) and the SUDAAN® (SUDAAN Release 11.0.1, 2013) statistical software package.

The Canadian Health Measures Survey (CHMS) is a sample survey, meaning that the participants represent many other Canadians not included in the survey. In order for the results of the survey to be representative of the entire population, sample weights were generated by Statistics Canada and incorporated into all estimates presented in the data tables (e.g. geometric means). Survey weights were used to take into account the unequal probability of selection into the survey as well as non-response. Further, to account for the complex survey design of the CHMS, the set of bootstrap weights included with the data set was used to estimate the 95% confidence intervals (CIs) for all means and percentiles (Rao et al., 1992; Rust and Rao, 1996).

For each chemical measured in cycle 4, data tables are presented. Data from cycles 1, 2, and 3 are also provided within the tables for those substances measured in all cycles. In the first *Report on Biomonitoring of Environmental Chemicals in Canada*, results for cycle 1 were reported to two decimal places. For cycles 2, 3, and 4 of the CHMS, the reporting protocol changed and the results were reported to two significant digits. For consistency, cycle 1 data were adjusted to two significant digits before generating the descriptive statistics, and data from all cycles are presented to two significant digits. Therefore, the descriptive statistics presented for cycle 1 may differ from those presented in the first report. The differences are not significant and the values presented in the first report are still considered to be accurate.

The data tables include the sample size (n); percentage of results that fall below the limit of detection (LOD); geometric mean (GM); the 10th, 50th, 90th, and 95th percentiles; and associated 95% CIs. For each chemical, results are presented for the total population as well as by age group and sex. For each chemical that was measured in multiple cycles of the CHMS, a summary table is provided that compares results for the aggregate of all age groups common to all cycles and for that same aggregate population separated by sex. Measurements that fell below the LOD for the laboratory analytical method were assigned a value equal to half the LOD. If the proportion of results below the LOD was greater than 40%, GMs were not calculated. Percentile estimates that are less than the LOD are reported as <LOD. The appendices contain tables of LOD values for each chemical, specific to each cycle, and conversion factors to assist in the comparison of data from other studies that report different units (Appendices A and B).

Chemicals measured in whole blood are presented as weight of chemical per volume of whole blood (e.g. µg chemical/L blood).

For urine measurements, concentrations are presented as weight of chemical per volume of urine (e.g. µg chemical/L urine) and adjusted for urinary creatinine (e.g. µg chemical/g creatinine). Urinary creatinine is a chemical by-product generated from muscle metabolism; it is frequently used to adjust for urine concentration (or

dilution) in spot urine samples because its production and excretion are relatively constant over 24 hours owing to homeostatic controls (Barr et al., 2005; Boeniger et al., 1993; Pearson et al., 2009). If the chemical measured behaves similarly to creatinine in the kidney, it will be filtered at the same rate; thus, expressing the chemical per gram of creatinine helps adjust for the effect of urinary dilution as well as some differences in renal function and lean body mass (Barr et al., 2005; CDC, 2009; Pearson et al., 2009). Creatinine is primarily excreted by glomerular filtration; therefore, creatinine adjustment may not be appropriate for compounds that are excreted primarily by tubular secretion in the kidney (Barr et al., 2005; Teass et al., 2003). In addition, creatinine excretion can vary owing to age, sex, and ethnicity; therefore, it may not be appropriate to compare creatinine-adjusted concentrations among different demographic groups (e.g. children with adults) (Barr et al., 2005). Where urinary creatinine values were missing or <LOD, the estimate of that participant's creatinine-adjusted chemical was not calculated and was also set to missing.

Descriptive statistics are available for creatinine (mg/dL) (Appendix C). These include n; GM; the 10th, 50th, 90th, and 95th percentiles; and associated 95% CIs for the total population as well as by age group and sex.

Specific gravity was also measured in all urine samples immediately following sample collection at the mobile examination centre. Urinary specific gravity is the ratio of densities between urine and pure water and can be used to adjust for variations in urine output, similar to urinary creatinine adjustment. Urinary specific gravity adjustment has not been presented for any of the chemicals; however, specific gravity data are available upon request by contacting Statistics Canada at infostats@statcan.gc.ca should researchers wish to perform this adjustment for their own data analyses.

Under the *Statistics Act*, Statistics Canada is required to ensure participant confidentiality. Therefore, estimates based on a small number of participants are suppressed. Following suppression rules for the CHMS, any estimate based on fewer than 10 participants is suppressed in the data tables. To avoid suppression, estimates at the 95th percentile require at least 200 participants, estimates at the 10th and 90th percentiles require at least 100 participants, estimates at the 50th percentile require at least 20 participants, and estimates of the geometric mean require at least 10 participants.

Estimates from a sample survey inevitably include sampling errors. Measuring the possible scope of sampling errors is based on the standard error of the estimates drawn from the survey results. To get a better indication of the size of the standard error, it is often more useful to express the standard error in terms of the estimate being measured. The resulting measure, called the coefficient of variation (CV), is obtained by dividing the standard error of the estimate by the estimate itself, and it is expressed as a percentage of the estimate. This report employs the following Statistics Canada guidelines for releasing estimates based on their CV:

- When a CV is between 16.6% and 33.3%, an estimate can be considered for general unrestricted release but is accompanied by a warning cautioning subsequent users of the high sampling variability associated with the estimate. These estimates are identified by the superscript letter E.
- When a CV is greater than 33.3%, Statistics Canada recommends not releasing the estimate because conclusions based on these data will be unreliable and most likely invalid. These estimates will not be published and will instead be replaced by the letter F.

Further details on the sample weights and data analysis are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017).

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CONSIDERATIONS FOR INTERPRETING THE BIOMONITORING DATA 7

The Canadian Health Measures Survey (CHMS) was designed to provide estimates of environmental chemical concentrations in blood or urine for the Canadian population as a whole. The first cycle of the survey covered approximately 96% of the Canadian population aged 6–79 years. The second, third, and fourth cycles included children as young as 3 years of age and also covered approximately 96% of the Canadian population aged 3–79 years of age. The survey was not designed to permit breakdown of data by region, province, or collection site, although some analysis is possible if data from more than one cycle are combined (see *Instructions for Combining Multiple Cycles of Canadian Health Measures Survey [CHMS] Data* [Statistics Canada, 2015]). In addition, the CHMS design did not target specific exposure scenarios; consequently, it did not select or exclude participants on the basis of their potential for low or high exposures to environmental chemicals.

Biomonitoring can estimate how much of a chemical is present in a person, but it cannot say what health effects, if any, may result from that exposure. The ability to measure environmental chemicals at very low concentrations has advanced in recent years. However, the presence alone of a chemical in a person's body does not necessarily mean that it will cause a health effect. Factors such as the dose, the toxicity of the chemical, and the duration and timing of exposure are important to determine whether potential adverse health effects may occur. For chemicals such as lead or mercury, research studies have provided a good understanding of the health risks associated with different concentrations in blood. However, for many chemicals, further research is needed to understand the potential health effects, if any, from different blood or urine concentrations. Furthermore, small amounts of certain chemicals, such as manganese and zinc, are essential for the maintenance of good health and would be expected to be present in the body. In addition, the way in which a chemical will act in the body will differ among individuals and cannot be predicted with certainty. Certain populations (children, pregnant women, the elderly, or immuno-compromised people) may be more susceptible to the effects of exposure.

The absence of a chemical does not necessarily mean a person has not been exposed. It may be that the technology is not capable of detecting such a small amount, or that the exposure occurred at an earlier point in time allowing for the chemical to be eliminated from the person's body before measurement took place.

Biomonitoring cannot tell us the source or route of the exposure. The amount of chemical measured indicates the total amount that has entered the body through all routes of exposure (ingestion, inhalation, and skin contact) and from all sources (air, water, soil, food, and consumer products). The detection of the chemical may be the result of exposure to a single source or multiple sources. In addition, in most cases biomonitoring cannot distinguish between natural and anthropogenic sources. Many chemicals (lead, mercury, cadmium, and arsenic) occur naturally in the environment and are also present in human-made products.

While metals are measured in urine as the parent compounds, almost all other chemicals are measured as metabolites. For many chemicals, parent compounds may be broken down (i.e. metabolized) in the body into one or more metabolites. For example, the polycyclic aromatic hydrocarbon chrysene is broken down into several metabolites. Some metabolites are

specific to one parent compound whereas others are common to several parent compounds. Several urinary metabolites are also formed in the environment (e.g. chlorpyrifos metabolites). Their presence in urine does not necessarily mean that an exposure to the parent chemical has occurred; rather, exposure could be to the metabolite itself in media such as food, water, or air.

Factors that contribute to the concentrations of chemicals measured in blood and urine include the quantity entering the body through all routes of exposure, absorption rates, distribution to various tissues in the body, metabolism, and excretion of the chemical and/or its metabolites from the body. These processes, also called toxicokinetics, depend on both the characteristics of the chemical, including its solubility in fat (or lipophilicity), its pH, its particle size, and the characteristics of the individual being exposed, such as age, diet, health status, and ethnicity. For these reasons, the way in which a chemical will act in the body will differ among individuals and cannot be predicted with certainty.

The CHMS biomonitoring data currently available include temporal data for substances measured in cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Results from future cycles can be compared with the baseline data from the CHMS in order to begin to examine trends in Canadians' exposures to selected environmental chemicals. It is important to note that there were some sampling and analytical modifications between cycles that may have contributed some variation in results for those substances measured in multiple cycles. The limits of detection (LOD) for certain analytical methods have changed from cycle to cycle. Although the LOD values did not change by a large margin, this difference should be noted when comparing data from multiple cycles. A list of LOD values from cycles 1, 2, 3, and 4 is provided (Appendix A). In addition, the urine collection protocol and guidelines were changed in cycle 2, and this may have resulted in a shift in creatinine levels when cycle 1 data are compared with those from subsequent cycles. This, in turn, could affect creatinine-adjusted levels of some chemicals.

Urinary creatinine concentrations can also be affected by variables such as age, sex, and ethnicity resulting in differences among demographic groups within a single cycle (Mage et al., 2004). In particular, creatinine excretion per unit bodyweight increases substantially with increasing age in children (Aylward et al., 2011; Remer et al., 2002). As a result, it is acceptable to compare creatinine-adjusted concentrations among similar demographic groups (e.g. children with children, adults with adults, males with males) but not among two different demographic groups (e.g. children with adults, males with females) (Barr et al., 2005).

More in-depth statistical analyses of the CHMS biomonitoring data, including time trends, exploring relationships among environmental chemicals, other physical measures, and self-reported information are being published by researchers in the scientific literature. A bibliography of publications using CHMS data is available. CHMS data are available to scientists through Statistics Canada's Research Data Centres Program and are a resource for additional scientific analyses. Further information about the CHMS can be obtained by contacting Statistics Canada at infostats@statcan.gc.ca.

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SUMMARY AND RESULTS FOR ACRYLAMIDE 8

8.1 ACRYLAMIDE

Acrylamide (CASRN 79-06-1) is a chemical used primarily in the production of polymers such as polyacrylamides (ATSDR, 2012). Polyacrylamides are used to clarify drinking water and treat effluent from water treatment plants and industrial processes (ATSDR, 2012). They are also used as binding, thickening, or flocculating agents in grout, cement, pesticide formulations, cosmetics, food manufacturing, and soil erosion prevention (Environment Canada and Health Canada, 2009a). Polymers of acrylamide are also used in ore processing, food packaging, and plastic products (Environment Canada and Health Canada, 2009a). In Canada, polyacrylamide is used as a coagulant and flocculant for the clarification of drinking water, in potting soils, and as a non-medicinal ingredient in natural health products and pharmaceuticals (Environment Canada and Health Canada, 2009b). Acrylamide can also form naturally in certain foods during processing or cooking at high temperatures (Health Canada, 2009a). It is formed mainly in carbohydrate-rich plant-based foods such as potatoes and grains with the highest concentrations detected in potato chips and french fries (Health Canada, 2009a).

Entry into the environment may occur during production and industrial use (ATSDR, 2012). Residual monomers may be released to drinking water during polyacrylamide treatment processes and are the main source of drinking water contamination by acrylamide (ATSDR, 2012). Acrylamide is a component of cigarette smoke and may be released to indoor air as a result of smoking (NTP, 2005; Urban et al., 2006).

Acrylamide exposure in the general population occurs primarily through food and to a lesser degree through air, drinking water, and soil (Environment Canada and Health Canada, 2009a). Inhalation of tobacco smoke, including second-hand smoke, is also a major source of inhalation exposure for the general population (ATSDR, 2012). Animal studies indicate that acrylamide is readily absorbed via oral and pulmonary routes, and to a lesser degree following dermal exposure (ATSDR, 2012). Once absorbed, acrylamide is widely distributed throughout the body accumulating in red blood cells (ATSDR, 2012). Acrylamide is metabolized via glutathione conjugation to form a mercapturic acid acrylamide derivative or by oxidation to form the epoxide derivative, glycidamide, which can be further metabolized via conjugation with glutathione. Both acrylamide and glycidamide react with haemoglobin in red blood cells forming adducts (ATSDR, 2012). Absorbed acrylamide and its metabolites are rapidly eliminated in urine, primarily as mercapturic acid conjugates of acrylamide and glycidamide (ATSDR, 2012). Acrylamide and glycidamide haemoglobin adducts are considered markers of exposure over the previous 120 days, the average life span of red blood cells (ATSDR, 2012).

Exposure to acrylamide is known to cause a number of health effects in humans, including neurotoxicity. Inhalation exposure to acrylamide in occupational settings has been associated with peripheral neuropathy characterized by muscle weakness and numbness in hands and feet (Environment Canada and Health Canada, 2009b). Studies with laboratory animals have observed adverse reproductive and developmental

effects and shown that acrylamide is genotoxic and carcinogenic (Environment Canada and Health Canada, 2009b; FAO/WHO, 2006). Reviews of existing epidemiological studies have found that there is inadequate evidence in humans to establish an association between acrylamide exposure and carcinogenicity (Health Canada, 2008; IARC, 1994). However, on the basis of evidence in experimental animals, the International Agency for Research on Cancer (IARC) and the U.S. Environmental Protection Agency (U.S. EPA) have classified acrylamide as probably carcinogenic to humans (EPA, 2010; IARC, 1994). Further, on the basis of available evidence from animal studies, the Joint FAO/WHO Expert Committee on Food Additives determined that the estimated intake of acrylamide from certain foods may be a human health concern (FAO/WHO, 2006; FAO/WHO, 2011).

Health Canada and Environment Canada (now referred to as Environment and Climate Change Canada) concluded, on the basis of carcinogenic potential, that acrylamide in Canada may constitute a danger to human life or health (Environment Canada and Health Canada, 2009b). Acrylamide is listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The Act allows the federal government to control the importation, manufacture, distribution, and use of acrylamide in Canada (Canada, 1999; Canada, 2011). Health Canada's risk management strategy for acrylamide in food is focused on reducing foodborne exposure to acrylamide (Health Canada, 2009b). To reduce exposure to acrylamide from food sources, Health Canada suggests following the recommendations provided in *Canada's Food Guide*, thereby limiting consumption of carbohydrate-rich foods that are high in fat (such as potato chips and french fries), sugar, or salt (Health Canada, 2009a). However, occasional consumption of these products is not likely to be a health concern. Other suggestions for reducing exposure to acrylamide from certain foods include paying careful attention to oil and baking temperatures, following the manufacturer's cooking instructions, storing potatoes at a temperature above 8°C, washing or soaking cut potatoes in water prior to frying, and toasting bread or baked goods to the lightest colour acceptable (Health Canada, 2009a). Health Canada regularly reviews data on the concentrations of acrylamide in foods sold on the Canadian market; these results may be shared with industry, particularly if elevated levels of acrylamide are identified in certain products. Health Canada continues to encourage the food industry to further pursue reduction efforts for acrylamide in processed foods (Health Canada, 2012). In order to obtain additional information demonstrating successful acrylamide reduction strategies on the part of food manufacturers, Health Canada initiated a one-year call for data in September 2013 seeking submissions of published and unpublished technical information on the occurrence of acrylamide in foods available for sale in Canada (Health Canada, 2013a). Health Canada has also amended the Food and Drug Regulations to permit the use of asparaginase in certain food products to reduce the formation of acrylamide during cooking (Canada, 2012; Health Canada, 2013b). Acrylamide is included as a prohibited ingredient on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Health Canada, 2015).

Because acrylamide-containing polymers are used in drinking water treatment, most Canadian jurisdictions have requirements to meet health-based standards for additives that limit the amount of acrylamide present in the treated drinking water (NSF International, 2016a; NSF International, 2016b). Health Canada has also set a maximum level for acrylamide in polyacrylamide-containing formulations used in natural health products in Canada (Environment Canada and Health Canada, 2009a; Health Canada, 2016).

In a study carried out on Montreal Island to assess the levels of acrylamide in 195 non-smoking teenagers aged 10–17 years, the geometric mean concentrations of haemoglobin adducts of acrylamide and glycidamide were 45.4 pmol/g haemoglobin and 45.6 pmol/g haemoglobin, respectively (Brisson et al., 2014).

Acrylamide and its metabolite glycidamide were analyzed as adducts in whole blood of CHMS participants aged 3–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented in blood as pmol/g haemoglobin (Hb). Finding a measurable amount of acrylamide or glycidamide haemoglobin adducts in blood is an indicator of exposure to acrylamide and does not necessarily mean that an adverse health effect will occur.

Table 8.1.1

Acrylamide haemoglobin adducts — Geometric means and selected percentiles of whole blood concentrations (pmol/g Hb) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2492	0	73 (65–82)	35 (30–40)	64 (57–70)	190 (160–230)	240 (190–290)
4 (2014–2015)	2529	0.04	67 (62–73)	38 (35–41)	60 (55–66)	150 (130–180)	200 (180–230)
Males, 3–79 years							
3 (2012–2013)	1225	0	79 (69–90)	36 (31–40)	68 (61–75)	200 (150–260)	270[E] (160–380)
4 (2014–2015)	1267	0.08	70 (62–79)	37 (33–42)	64 (57–71)	170[E] (110–230)	220 (180–250)
Females, 3–79 years							
3 (2012–2013)	1267	0	68 (59–78)	35 (29–41)	60 (51–69)	180 (130–230)	210 (180–250)
4 (2014–2015)	1262	0	65 (58–72)	38 (36–41)	58 (53–62)	140 (100–180)	180 (140–220)
3–5 years							
3 (2012–2013)	471	0	59 (55–64)	39 (35–43)	59 (55–63)	87 (73–100)	100 (82–120)
4 (2014–2015)	484	0	60 (56–65)	37 (32–43)	61 (55–66)	96 (84–110)	100 (83–120)
6–11 years							
3 (2012–2013)	505	0	61 (57–65)	37 (34–41)	62 (58–67)	100 (88–110)	110 (98–120)
4 (2014–2015)	507	0	62 (59–66)	42 (39–45)	62 (58–66)	90 (83–96)	100 (94–110)
12–19 years							
3 (2012–2013)	507	0	63 (59–67)	37 (31–42)	57 (53–61)	110 (87–130)	170[E] (96–240)
4 (2014–2015)	505	0	63 (55–72)	37 (33–42)	60 (51–70)	100 (83–120)	120 (91–160)
20–39 years							
3 (2012–2013)	348	0	80 (65–97)	34 (24–43)	74 (59–89)	190 (130–260)	260 (190–340)
4 (2014–2015)	363	0	70 (60–80)	37 (33–41)	61 (53–70)	170 (120–220)	210 (170–250)
40–59 years							
3 (2012–2013)	311	0	83 (67–100)	35 (24–47)	66 (49–82)	230 (180–290)	330 (210–450)
4 (2014–2015)	312	0.32	71 (62–80)	38 (34–42)	60 (50–70)	180 (130–230)	250 (170–330)
60–79 years							
3 (2012–2013)	350	0	63 (59–68)	34 (29–40)	62 (59–65)	130 (100–150)	160 (130–190)
4 (2014–2015)	358	0	63 (56–71)	34 (26–43)	59 (53–65)	150 (110–190)	190 (170–210)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 8.1.2

Glycidamide haemoglobin adducts — Geometric means and selected percentiles of whole blood concentrations (pmol/g Hb) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2492	0.76	68 (62-75)	36 (34-38)	65 (59-70)	150 (120-180)	190 (150–220)
4 (2014–2015)	2529	1.86	60 (54-67)	34 (30-37)	57 (52-62)	120 (100-140)	170 (150–200)
Males, 3–79 years							
3 (2012–2013)	1225	1.14	69 (62–77)	37 (35–38)	66 (58–74)	170 (120–210)	210 (160–260)
4 (2014–2015)	1267	2.37	61 (53–70)	33 (27–39)	58 (50–66)	130 (100–160)	170 (130–200)
Females, 3–79 years							
3 (2012–2013)	1267	0.39	67 (60–74)	36 (32–40)	64 (57–71)	130 (100–160)	160 (120–200)
4 (2014–2015)	1262	1.35	59 (53–67)	34 (31–37)	56 (51–62)	110 (81–140)	170 (110–240)
3–5 years							
3 (2012–2013)	471	0	80 (75–85)	51 (43–59)	78 (74–81)	120 (110–130)	140 (120–150)
4 (2014–2015)	484	0.21	76 (69–84)	49 (44–53)	73 (65–82)	120 (100–130)	140 (110–180)
6–11 years							
3 (2012–2013)	505	0	73 (70–77)	47 (45–48)	74 (68–81)	110 (97–120)	130 (110–150)
4 (2014–2015)	507	0.39	70 (65–74)	44 (41–48)	66 (60–73)	100 (95–110)	120 (110–130)
12–19 years							
3 (2012–2013)	507	1.18	62 (59–65)	35 (32–37)	60 (57–62)	110 (95–130)	160 (120–200)
4 (2014–2015)	505	2.38	58 (51–67)	34 (27–41)	55 (49–62)	99 (83–120)	120[E] (58–180)
20–39 years							
3 (2012–2013)	348	0.86	72 (60–86)	38 (30–46)	74 (62–86)	160 (130–190)	210 (160–260)
4 (2014–2015)	363	2.20	62 (52–74)	34 (29–39)	57 (49–66)	170 (110–230)	190 (170–220)
40–59 years							
3 (2012–2013)	311	1.29	71 (58–86)	36 (31–42)	62 (50–74)	180 (140–220)	230 (170–290)
4 (2014–2015)	312	1.92	63 (55–71)	35 (30–39)	58 (50–65)	130 (97–160)	160[E] (57–260)
60–79 years							
3 (2012–2013)	350	1.71	60 (53–67)	34 (29–39)	60 (50–70)	100 (90–110)	120 (110–130)
4 (2014–2015)	358	5.03	50 (44–57)	25 (<LOD–33)	50 (44–56)	98 (87–110)	120 (93–150)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

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SUMMARIES AND RESULTS FOR ENVIRONMENTAL PHENOLS 9

9.1 BISPHENOL A

Bisphenol A (BPA; CASRN 80-05-7) is a synthetic chemical used as a monomer in the production of some polycarbonate plastics and as a precursor for monomers of certain epoxy-phenolic resins (EFSA, 2007). Polycarbonate is used in the manufacture of food and beverage containers such as repeat-use water bottles and storage containers; it was also used in infant bottles in Canada prior to 2010. Epoxy resins are used as an interior protective lining for food and beverage cans. Additional end-use products containing polycarbonate plastics and resins include medical devices, some dental fillings and sealants, sporting and safety equipment, electronics, and automotive parts (EFSA, 2007; NTP, 2007). BPA is also used in the paper industry to produce thermal paper used for various products including receipts, prescription labels, airline tickets, and lottery tickets (Geens et al., 2011).

BPA does not occur naturally in the environment (Environment Canada and Health Canada, 2008a). Entry into the environment may occur from industrial sources or from product leaching, disposal, and use (CDC, 2009).

The primary route of exposure to BPA for the general public is through dietary intake as a result of various sources, including migration from food packaging and repeat-use polycarbonate containers (Health Canada, 2008). Health Canada has recently updated its dietary exposure estimates for BPA following the completion of a number of specific food surveys, including canned foods and beverages, liquid infant formula, and Total Diet samples (Health Canada, 2012). Exposure can also occur from contact with environmental media, including ambient and indoor air, drinking water, soil, and dust, and from the use of consumer products (Environment Canada and Health Canada, 2008a). BPA exposure from dental fillings and sealants is short term and considered unlikely to contribute substantially to chronic exposure (WHO, 2011). However, further clinical research would help to answer questions about the potential harms caused by the exposure to BPA from dental composite materials (CADTH, 2015).

In humans, BPA is readily absorbed and undergoes extensive metabolism in the gut wall and the liver (WHO, 2011). Recent studies have also suggested that it may be absorbed and metabolized by the skin following dermal exposure to free BPA in products such as those made from thermal printing papers (Mielke et al., 2011; Zalko et al., 2011). Glucuronidation has been recognized as a major metabolic pathway for BPA, resulting in the BPA-glucuronide conjugate metabolite (EFSA, 2008; FDA, 2008). Conjugation of BPA to BPA-sulphate has been shown to be a minor metabolic pathway (Dekant and Völkel, 2008). The BPA-glucuronide metabolite is rapidly excreted in urine with a half-life of less than 2 hours (WHO, 2011). Urinary levels of total BPA, including both conjugated and free unconjugated forms, are commonly used as biomarkers to assess recent exposures (Ye et al., 2005).

Characterization of the potential risk to human health from exposure to BPA includes key effects on the liver, kidney and on reproduction, including fertility and developmental effects (EFSA CEF Panel, 2015; Environment Canada and Health Canada, 2008a; EU, 2010). The potential role of BPA and other

environmental estrogens in the prevalence of obesity and related metabolic diseases, as well as certain types of cancer, is under intensive debate and investigation among scientific communities (Ben-Jonathan et al., 2009; Carwile and Michels, 2011; Newbold et al., 2009; Song et al., 2014; Soto et al., 2008).

The Government of Canada has conducted a scientific screening assessment of the impact of human and environmental exposure to BPA and determined that it is toxic to human health and the environment as per the criteria set out under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment Canada and Health Canada, 2008a). Because of the uncertainty raised by the results of some laboratory animal studies relating to the potential effects of low levels of BPA, a precautionary approach was applied when characterizing risk. Considering the highest potential exposure and subpopulations with potential vulnerability due to potential differences in the toxicokinetics and metabolism of BPA identified in the assessment, the risk management strategy for health focused on decreasing exposure to newborns and infants (Environment Canada and Health Canada, 2008b).

Health Canada has concluded that current dietary exposure to BPA through food packaging uses is not expected to pose a health risk to the general population, including newborns and young children (Health Canada, 2012). However, the general principle of as low as reasonably achievable (ALARA) was applied to continue efforts on limiting BPA exposure from food packaging applications to infants and newborns, specifically from pre-packaged infant formula products as a sole source food. As part of this ALARA approach, Health Canada committed to supporting industry to reduce levels of BPA in infant-formula can linings (Health Canada, 2010). In addition, Health Canada has assessed a number of proposed industry alternatives to BPA and deemed them acceptable for packaging of liquid infant-formula. Infant-formula manufacturers have abandoned or phased out the use of BPA-containing packaging materials for liquid infant formula, and the can-coating industry has developed various BPA-free alternatives for can coatings currently available on the market (Health Canada, 2014). Health Canada will continue to review pre-market submissions for infant-formula packaging to ensure the lowest levels of BPA achievable (Health Canada, 2010). As of March 2010, under the *Canada Consumer Product Safety Act*, Health Canada has prohibited the manufacturing, advertisement, sale, or import of polycarbonate baby bottles that contain BPA (Canada, 2010). BPA is also included as a prohibited ingredient on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Health Canada, 2015). Risk management actions also have been developed under CEPA 1999 with the objective of minimizing releases of BPA in industrial effluents (Canada, 2012).

The Maternal-Infant Research on Environmental Chemicals (MIREC) Study is a national-level prospective biomonitoring study carried out in pregnant women aged 18 years and older from 10 sites across Canada (Arbuckle et al., 2013). In the MIREC Study of 1,936 participants in their first trimester of pregnancy, the geometric mean and 95th percentile for total BPA in urine were 0.80 µg/L and 5.40 µg/L, respectively (Arbuckle et al., 2014). The Plastics and Personal-care Products use in Pregnancy (P4) Study is a targeted biomonitoring study carried out in 80 pregnant women aged 18 years and older from the Ottawa area. The geometric mean and 95th percentile for total BPA in urine were 1.1 µg/L and 6.4 µg/L, respectively, based on analyses of multiple samples per woman (Arbuckle et al., 2015). The First Nations Biomonitoring Initiative (FNBI) is a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprises 13 randomly selected First Nation communities in Canada with 503 First Nations participants aged 20 years and older. The geometric mean and 95th percentile for total BPA in urine were 1.55 µg/L and 11.27 µg/L, respectively.

Urinary total BPA (including both free and conjugated forms) was analyzed in the urine of Canadian Health Measures Survey participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Data from these cycles are presented as both µg/L and µg/g creatinine. Finding a measurable amount of BPA in urine is an indicator of exposure to BPA and does not necessarily mean that an adverse health effect will occur.

Table 9.1.1

Bisphenol A (BPA) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2560	5.04	1.2 (1.1–1.3)	0.27 (0.22–0.31)	1.2 (1.1–1.3)	4.5 (4.0–5.0)	6.7 (4.8–8.6)
3 (2012–2013)	5670	7.80	1.1 (1.0–1.2)	0.29 (0.27–0.32)	1.1 (0.95–1.2)	4.2 (3.6–4.8)	6.6 (5.8–7.5)
4 (2014–2015)	2560	7.30	1.0 (0.95–1.1)	0.26 (<LOD–0.33)	1.0 (0.94–1.1)	4.0 (3.2–4.8)	6.0 (5.0–7.1)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1281	4.84	1.3 (1.1–1.5)	0.27 (<LOD–0.36)	1.3 (1.1–1.5)	4.6 (4.1–5.2)	7.9[E] (4.3–11)
3 (2012–2013)	2826	6.97	1.2 (1.1–1.4)	0.35 (0.25–0.46)	1.2 (0.99–1.4)	4.4 (3.7–5.0)	6.4 (5.2–7.7)
4 (2014–2015)	1273	5.89	1.2 (1.0–1.3)	0.35 (0.28–0.43)	1.2 (0.97–1.3)	4.3 (3.0–5.6)	6.2 (4.3–8.0)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	5.24	1.2 (1.0–1.3)	0.26 (0.21–0.32)	1.1 (0.98–1.3)	4.1 (3.0–5.1)	6.6 (4.9–8.4)
3 (2012–2013)	2844	8.61	1.0 (0.88–1.2)	0.29 (<LOD–0.39)	1.0 (0.91–1.1)	4.1 (3.3–4.9)	6.9 (5.4–8.4)
4 (2014–2015)	1287	8.70	0.92 (0.79–1.1)	<LOD	0.98 (0.82–1.1)	3.4 (2.8–4.0)	5.4 (3.6–7.3)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	524	4.20	1.4 (1.1–1.8)	0.30[E] (<LOD–0.46)	1.3 (1.1–1.5)	5.4[E] (1.9–9.0)	9.9[E] (5.5–14)
3 (2012–2013)	521	5.76	1.2 (0.87–1.6)	0.29[E] (<LOD–0.47)	1.2 (0.95–1.5)	4.0 (2.6–5.4)	6.0 (4.3–7.7)
4 (2014–2015)	511	7.83	1.2 (1.0–1.4)	0.28[E] (<LOD–0.44)	1.2 (1.0–1.3)	4.0 (3.5–4.5)	6.4[E] (2.9–9.9)
6–11 years							
1 (2007–2009)	1031	6.79	1.3 (1.2–1.4)	0.28 (<LOD–0.37)	1.3 (1.1–1.6)	4.5 (3.8–5.1)	7.1 (5.5–8.7)
2 (2009–2011)	516	5.81	1.4 (1.1–1.7)	0.25[E] (<LOD–0.41)	1.3 (0.94–1.7)	4.6[E] (2.6–6.6)	F
3 (2012–2013)	1004	5.58	1.2 (1.1–1.4)	0.39 (0.30–0.49)	1.2 (1.0–1.3)	3.8 (2.8–4.8)	5.3[E] (3.0–7.6)
4 (2014–2015)	511	6.46	1.1 (0.90–1.4)	0.29 (<LOD–0.40)	1.1 (0.83–1.4)	3.5 (2.6–4.4)	5.0 (4.0–6.0)
12–19 years							
1 (2007–2009)	980	6.22	1.5 (1.3–1.8)	0.29 (0.22–0.36)	1.6 (1.3–1.9)	5.9 (4.8–7.0)	8.3 (6.2–10)
2 (2009–2011)	512	4.69	1.3 (1.1–1.6)	0.35 (0.23–0.47)	1.3 (0.99–1.6)	4.4 (2.9–5.9)	7.6[E] (4.3–11)
3 (2012–2013)	992	6.15	1.3 (1.1–1.6)	0.30[E] (<LOD–0.46)	1.4 (1.3–1.6)	4.8 (3.4–6.2)	8.0[E] (4.1–12)
4 (2014–2015)	505	4.95	1.1 (1.1–1.2)	0.26 (<LOD–0.35)	1.2 (1.0–1.3)	3.8 (3.1–4.6)	5.5 (4.5–6.5)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	1165	8.84	1.3 (1.2–1.5)	F	1.4 (1.2–1.6)	4.8 (4.1–5.4)	7.3 (5.2–9.5)
2 (2009–2011)	357	2.80	1.3 (1.1–1.5)	0.32 (0.21–0.42)	1.3 (0.92–1.6)	4.6 (3.7–5.5)	F
3 (2012–2013)	1040	7.88	1.1 (0.92–1.4)	0.29 (<LOD–0.39)	1.1 (0.81–1.3)	5.5 (3.9–7.0)	6.7 (5.1–8.3)
4 (2014–2015)	362	7.73	1.1 (0.93–1.4)	<LOD[E] (<LOD–0.35)	1.2 (0.97–1.4)	5.6[E] (3.3–7.8)	7.4 (5.1–9.7)
40–59 years							
1 (2007–2009)	1219	12.06	1.0 (0.96–1.1)	<LOD	1.2 (1.1–1.4)	4.4 (3.5–5.3)	6.6 (4.8–8.4)
2 (2009–2011)	360	6.11	1.2 (0.97–1.5)	0.25[E] (<LOD–0.37)	1.2 (0.98–1.4)	4.3[E] (2.7–6.0)	6.7[E] (2.6–11)
3 (2012–2013)	1075	9.86	1.1 (1.0–1.3)	0.30 (<LOD–0.36)	1.1 (0.94–1.2)	4.2 (3.1–5.3)	7.5[E] (4.3–11)
4 (2014–2015)	311	7.72	0.86 (0.74–1.0)	0.28 (<LOD–0.38)	0.94 (0.77–1.1)	2.4 (1.9–2.9)	4.2[E] (2.4–5.9)
60–79 years							
1 (2007–2009)	1081	11.66	0.90 (0.81–0.99)	<LOD	0.99 (0.87–1.1)	3.7 (3.3–4.2)	5.2 (3.8–6.6)
2 (2009–2011)	291	7.22	1.0 (0.84–1.3)	0.21[E] (<LOD–0.31)	0.99 (0.76–1.2)	4.4[E] (2.5–6.2)	6.3 (4.4–8.1)
3 (2012–2013)	1038	10.31	0.88 (0.77–1.0)	<LOD	0.88 (0.76–1.0)	3.3 (2.8–3.7)	5.5 (4.2–6.7)
4 (2014–2015)	360	10.28	1.1 (0.96–1.2)	<LOD	1.0 (0.84–1.2)	4.2 (3.1–5.3)	5.5[E] (2.3–8.7)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

F Data is too unreliable to be published.

Table 9.1.2

Bisphenol A (BPA) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2550	5.04	1.2 (1.1–1.3)	0.39 (0.35–0.44)	1.0 (0.92–1.1)	4.1 (3.6–4.6)	6.9 (5.1–8.7)
3 (2012–2013)	5667	7.80	1.1 (1.0–1.2)	0.40 (0.36–0.45)	0.99 (0.94–1.0)	3.6 (3.0–4.2)	5.9 (4.4–7.5)
4 (2014–2015)	2559	7.30	0.93 (0.87–0.99)	0.32 (<LOD–0.36)	0.87 (0.80–0.94)	3.1 (2.6–3.5)	4.5 (3.9–5.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	4.84	1.1 (0.96–1.2)	0.36 (<LOD–0.48)	0.99 (0.93–1.1)	3.7 (2.7–4.8)	6.2[E] (3.5–8.8)
3 (2012–2013)	2826	6.97	1.1 (0.96–1.2)	0.38 (0.32–0.45)	0.98 (0.90–1.1)	3.1 (2.8–3.4)	5.1 (3.9–6.4)
4 (2014–2015)	1272	5.89	0.92 (0.83–1.0)	0.30 (0.24–0.36)	0.87 (0.76–0.98)	2.8 (2.2–3.5)	4.1 (3.2–4.9)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	5.24	1.3 (1.2–1.5)	0.48 (0.40–0.57)	1.1 (0.95–1.3)	4.5 (3.5–5.5)	6.9 (4.5–9.4)
3 (2012–2013)	2841	8.61	1.2 (1.1–1.4)	0.42 (<LOD–0.46)	1.0 (0.91–1.1)	4.0 (3.1–5.0)	7.1[E] (4.4–9.9)
4 (2014–2015)	1287	8.70	0.94 (0.85–1.0)	<LOD	0.88 (0.78–0.97)	3.4 (2.5–4.3)	5.0 (4.2–5.8)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	4.20	2.4 (1.9–3.1)	0.88[E] (<LOD–1.2)	2.0 (1.8–2.3)	10[E] (4.6–15)	13 (8.6–17)
3 (2012–2013)	520	5.76	2.3 (1.8–2.9)	0.86[E] (<LOD–1.2)	2.1 (1.4–2.7)	5.9 (4.1–7.8)	8.4 (6.7–10)
4 (2014–2015)	511	7.83	2.0 (1.7–2.4)	0.64[E] (<LOD–0.90)	1.8 (1.5–2.2)	6.7 (4.7–8.7)	13[E] (4.4–21)
6–11 years							
1 (2007–2009)	1028	6.79	2.0 (1.8–2.2)	0.68 (<LOD–0.82)	2.0 (1.8–2.1)	5.8 (4.8–6.9)	9.8 (7.4–12)
2 (2009–2011)	514	5.81	1.5 (1.2–1.9)	0.44[E] (<LOD–0.68)	1.4 (1.1–1.7)	F	10[E] (3.0–18)
3 (2012–2013)	1004	5.58	1.5 (1.3–1.7)	0.58 (0.46–0.69)	1.4 (1.1–1.6)	3.9 (2.6–5.2)	5.3[E] (2.0–8.6)
4 (2014–2015)	510	6.46	1.2 (1.0–1.5)	0.41 (<LOD–0.54)	1.1 (0.94–1.3)	3.2 (2.6–3.8)	F
12–19 years							
1 (2007–2009)	978	6.22	1.3 (1.2–1.4)	0.40 (0.30–0.50)	1.2 (0.99–1.4)	4.2 (3.3–5.0)	6.4[E] (4.0–8.8)
2 (2009–2011)	510	4.69	1.0 (0.83–1.2)	0.30[E] (0.17–0.43)	0.94 (0.79–1.1)	3.4[E] (1.5–5.2)	5.0 (3.8–6.3)
3 (2012–2013)	991	6.15	1.0 (0.85–1.2)	0.35 (<LOD–0.44)	0.95 (0.82–1.1)	3.0 (2.3–3.8)	5.4[E] (2.6–8.2)
4 (2014–2015)	505	4.95	0.83 (0.74–0.93)	0.30 (<LOD–0.35)	0.74 (0.61–0.87)	2.7 (2.1–3.3)	3.9 (2.6–5.1)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	1161	8.84	1.5 (1.4–1.6)	0.44 (<LOD–0.55)	1.4 (1.2–1.6)	4.4 (3.4–5.4)	6.8 (5.9–7.7)
2 (2009–2011)	355	2.80	1.1 (0.89–1.3)	0.39 (0.27–0.50)	0.99 (0.85–1.1)	2.8 (1.8–3.7)	F
3 (2012–2013)	1040	7.88	1.0 (0.90–1.2)	0.36 (<LOD–0.43)	0.93 (0.80–1.1)	3.3 (2.6–3.9)	5.4[E] (2.7–8.1)
4 (2014–2015)	362	7.73	0.91 (0.80–1.0)	<LOD	0.87 (0.75–0.99)	3.5[E] (1.7–5.3)	4.6[E] (2.0–7.1)
40–59 years							
1 (2007–2009)	1214	12.06	1.3 (1.2–1.5)	<LOD	1.2 (1.0–1.4)	4.7 (3.8–5.7)	7.5 (6.1–8.8)
2 (2009–2011)	358	6.11	1.2 (0.99–1.4)	0.39 (<LOD–0.50)	1.1 (0.86–1.3)	4.2[E] (2.3–6.2)	6.9[E] (3.4–10)
3 (2012–2013)	1074	9.86	1.2 (1.1–1.3)	0.47 (<LOD–0.52)	0.99 (0.90–1.1)	3.8 (2.9–4.6)	6.1[E] (3.7–8.5)
4 (2014–2015)	311	7.72	0.78 (0.70–0.86)	0.33 (<LOD–0.40)	0.71 (0.64–0.78)	1.9[E] (0.95–2.9)	3.8[E] (2.2–5.4)
60–79 years							
1 (2007–2009)	1081	11.66	1.2 (1.1–1.4)	<LOD	1.1 (0.94–1.3)	4.3 (3.0–5.6)	7.6 (5.4–9.8)
2 (2009–2011)	290	7.22	1.2 (0.99–1.4)	0.29[E] (<LOD–0.45)	1.0 (0.89–1.1)	4.7 (3.3–6.0)	6.8[E] (2.9–11)
3 (2012–2013)	1038	10.31	1.0 (0.97–1.1)	<LOD	0.99 (0.94–1.0)	3.0 (2.7–3.4)	4.7[E] (2.7–6.7)
4 (2014–2015)	360	10.28	1.0 (0.92–1.2)	<LOD	0.99 (0.89–1.1)	3.5 (2.5–4.4)	4.8[E] (2.1–7.4)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

F Data is too unreliable to be published.

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9.2 TRICLOSAN

Triclosan (CASRN 3380-34-5) is a synthetic chemical with wide application since 1972 as an antimicrobial agent and as a preservative (Jones et al., 2000). It is used as a medicinal ingredient in non-prescription drug products and as a non-medicinal ingredient in cosmetics, natural health products, and drug products. Cosmetic products containing triclosan have been imported or manufactured for sale in Canada, including skin moisturizers (body, face, and hands), face and eye makeup, deodorant sticks/sprays, fragrances, tanning products, skin cleansers, shaving preparations, and shampoos (Environment and Climate Change Canada and Health Canada, 2016a). In addition, a number of products containing triclosan as an active medicinal ingredient are regulated as non-prescription drug products in Canada, including anti-bacterial hand sanitizers and soaps (Health Canada, 2016a). Triclosan has also been used to control the spread of bacteria in household items such as cleaners, textiles, carpets, cutting boards and other food contact materials, and in medical devices (Jones et al., 2000). As of December 31, 2014, triclosan is no longer registered as a pesticide for material preservative uses under the *Pest Control Products Act* (Canada, 2006; Environment and Climate Change Canada and Health Canada, 2016a). Triclosan does not occur naturally in the environment (Environment and Climate Change Canada and Health Canada, 2016a). The use of triclosan-containing products results in its release to waste-water systems and subsequently surface water (Environment and Climate Change Canada and Health Canada, 2016a). The potential routes of exposure for the general public are oral and dermal contact with products such as toothpastes and cosmetics that contain triclosan, ingestion of triclosan-contaminated drinking water, breast milk, or ingestion of household dust (Environment and Climate Change Canada and Health Canada, 2016a).

Following oral exposures, triclosan is rapidly absorbed and distributed in humans, with plasma levels increasing rapidly within 1 to 4 hours (Environment and Climate Change Canada and Health Canada, 2016a). Absorption following dermal exposure to triclosan-containing products ranges from 11% to 17% in humans (Maibach, 1969; Queckenberg et al., 2010; Stierlin, 1972). Only limited absorption (approximately 5% to 10%) occurs under normal conditions of toothpaste use (SCCP, 2009). Following

all routes of administration, absorbed triclosan is nearly totally converted to glucuronic and sulfuric acid conjugates (Fang et al., 2010). Triclosan is rapidly eliminated after metabolism with an observed half-life in humans ranging from 13 to 29 hours following oral administration (SCCP, 2009). About 24% to 83% of absorbed triclosan is excreted in urine, mostly as the glucuronide conjugate (Fang et al., 2010; Sandborgh-Englund et al., 2006). Excretion of triclosan in feces is as the free unchanged compound and represents a smaller portion of the administered dose (10% to 30%) (Environment and Climate Change Canada and Health Canada, 2016a). Currently, there is no evidence of bioaccumulation potential in humans (SCCP, 2009). The concentration of total triclosan in urine (conjugated and free) can be used as a biomarker of exposure to triclosan (Calafat et al., 2007).

Triclosan is not acutely toxic to mammals, but it can interact with a cellular receptor and several enzymes (Calafat et al., 2007). The potential effects of these interactions remain unknown. In rodents, there have been observations of adverse effects of triclosan on thyroid hormone homeostasis resulting from liver toxicity; however, the overall weight of evidence does not currently support effects of triclosan on thyroid function as a critical effect for risk characterization in humans (Environment and Climate Change Canada and Health Canada, 2016a). To date, triclosan has not been assessed for carcinogenic potential by the International Agency for Research on Cancer; the United States Environmental Protection Agency has classified triclosan as not likely to be carcinogenic to humans (EPA, 2008).

Health Canada and Environment and Climate Change Canada have jointly reviewed triclosan in an assessment and have concluded that it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health (Environment and Climate Change Canada and Health Canada, 2016a). However, at current environmental levels, triclosan is concluded to be an ecological concern and it was found to meet the definition of toxic under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada 2016a).

Currently, Health Canada regulates triclosan under the *Food and Drugs Act* for its inclusion in personal care products such as cosmetics, natural health products, and non-prescription drugs. Triclosan is included as a restricted ingredient on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Health Canada, 2015). The Hotlist indicates concentration limits of triclosan in mouthwash and other cosmetic products (Health Canada, 2015). In addition, the Hotlist indicates that oral cosmetics containing triclosan shall include a label statement indicating that the product is not to be used by children under 12 years of age (Health Canada, 2015). The Hotlist also indicates that mouthwashes include a label statement to the effect of "avoid swallowing" (Health Canada, 2015). Consistent with the Hotlist, Health Canada has also set concentration limits for non-medicinal use of triclosan as an antimicrobial preservative in natural health products and for use as an active medicinal ingredient in non-prescription drug products in Canada (Health Canada, 2016a; Health Canada, 2016b). As of December 31, 2014, triclosan is no longer registered in Canada as a pest control product because of voluntary withdrawal from the market (Canada, 2006; Environment and Climate Change Canada and Health Canada, 2016a). A risk management approach, including a requirement for the preparation and implementation of Pollution Prevention Plans, has also been proposed under CEPA 1999 with the objective of reducing releases of triclosan to the aquatic environment as a result of the use by consumers of triclosan-containing products (Environment and Climate Change Canada and Health Canada, 2016b).

The Maternal-Infant Research on Environmental Chemicals (MIREC) Study is a national-level prospective biomonitoring study carried out in pregnant women aged 18 years and older from 10 sites across Canada (Arbuckle et al., 2013). In the MIREC Study of 1,861 participants in their first trimester of pregnancy, the geometric mean and 95th percentile for triclosan in urine were 12.64 µg/L and 697.58 µg/L, respectively (Arbuckle, Marro et al., 2015). The Plastics and Personal-care Products use in Pregnancy (P4) Study is a targeted biomonitoring study carried out in 80 pregnant women aged 18 years and older from the Ottawa area.

The geometric mean and 95th percentile for triclosan in urine were 21.6 µg/L and 833.4 µg/L, respectively, based on analysis of multiple urine samples per woman (Arbuckle, Weiss et al., 2015).

Total triclosan (including both free and conjugated forms) was analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and is presented as both µg/L and µg/g creatinine. Finding a measurable amount of triclosan in urine is an indicator of exposure to triclosan and does not necessarily mean that an adverse health effect will occur.

Table 9.2.1

Triclosan — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2550	28.20	16 (13–20)	<LOD	9.5[E] (5.8–13)	400 (280–520)	710 (540–880)
3 (2012–2013)	5645	34.47	17 (15–19)	<LOD	9.9 (8.4–11)	350 (270–430)	720 (460–980)
4 (2014–2015)	2558	44.57	—	<LOD	5.9 (<LOD–7.6)	310[E] (160–460)	660[E] (370–940)
Males, 3–79 years							
2 (2009–2011)	1274	26.77	18 (13–26)	<LOD	12[E] (5.3–18)	510 (330–690)	790[E] (350–1200)
3 (2012–2013)	2815	34.03	17 (14–21)	<LOD	10 (7.4–13)	330[E] (180–480)	760[E] (380–1100)
4 (2014–2015)	1273	44.85	—	<LOD	6.1[E] (<LOD–8.5)	270[E] (120–430)	640[E] (320–970)
Females, 3–79 years							
2 (2009–2011)	1276	29.62	14 (11–18)	<LOD	7.5[E] (3.1–12)	310[E] (140–470)	680[E] (410–960)
3 (2012–2013)	2830	34.91	17 (13–22)	<LOD	9.6 (7.8–12)	390[E] (220–550)	700[E] (280–1100)
4 (2014–2015)	1285	44.28	—	<LOD	5.7 (<LOD–7.4)	360[E] (120–590)	880[E] (320–1400)
3–5 years							
2 (2009–2011)	523	29.45	8.9 (7.3–11)	<LOD	7.3 (4.9–9.6)	50 (40–61)	120[E] (68–160)
3 (2012–2013)	518	36.29	9.5 (7.4–12)	<LOD	7.7[E] (<LOD–11)	78[E] (43–110)	110[E] (47–170)
4 (2014–2015)	511	49.12	—	<LOD	4.8 (<LOD–6.5)	33[E] (13–53)	F
6–11 years							
2 (2009–2011)	515	33.98	8.5 (6.7–11)	<LOD	3.8[E] (<LOD–5.9)	130[E] (54–210)	250[E] (82–410)
3 (2012–2013)	1001	36.26	11 (8.4–16)	<LOD	7.2[E] (<LOD–10)	F	340[E] (190–500)
4 (2014–2015)	510	49.22	—	<LOD	5.2 (<LOD–6.6)	F	170[E] (93–250)
12–19 years							
2 (2009–2011)	510	19.02	20 (14–27)	<LOD	13[E] (7.7–18)	350[E] (230–480)	640[E] (400–870)
3 (2012–2013)	984	28.35	19 (14–26)	<LOD	10 (7.2–13)	510[E] (220–800)	840 (580–1100)
4 (2014–2015)	504	38.89	13 (8.7–18)	<LOD	6.4[E] (<LOD–9.8)	F	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	19.26	21[E] (13–32)	<LOD	17[E] (9.1–25)	470[E] (180–760)	910[E] (430–1400)
3 (2012–2013)	1035	27.44	24 (18–30)	<LOD	15 (11–19)	420[E] (250–580)	F
4 (2014–2015)	361	34.63	12[E] (7.2–20)	<LOD	5.4[E] (<LOD–8.0)	420[E] (190–650)	880[E] (480–1300)
40–59 years							
2 (2009–2011)	359	28.97	19[E] (12–29)	<LOD	12[E] (4.3–20)	470[E] (200–740)	740[E] (290–1200)
3 (2012–2013)	1072	37.22	16 (12–22)	<LOD	8.9 (6.6–11)	380[E] (140–620)	910[E] (250–1600)
4 (2014–2015)	312	41.03	—	<LOD	F	F	F
60–79 years							
2 (2009–2011)	290	41.72	—	<LOD	4.8[E] (<LOD–6.8)	360[E] (160–560)	590 (430–750)
3 (2012–2013)	1035	41.84	—	<LOD	6.9 (6.0–7.7)	260[E] (140–380)	580[E] (270–890)
4 (2014–2015)	360	52.50	—	<LOD	<LOD	F	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 9.2.2

Triclosan (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2540	28.20	15 (11–19)	<LOD	9.0 (7.5–10)	370 (260–480)	610 (400–830)
3 (2012–2013)	5642	34.47	17 (15–20)	<LOD	9.9 (9.2–11)	350 (310–390)	640 (510–770)
4 (2014–2015)	2557	44.57	—	<LOD	6.1 (<LOD–8.1)	250 (160–340)	540[E] (310–770)
Males, 3–79 years							
2 (2009–2011)	1270	26.77	15 (10–21)	<LOD	8.7 (6.4–11)	390 (280–490)	700[E] (360–1000)
3 (2012–2013)	2815	34.03	15 (12–18)	<LOD	8.7 (7.1–10)	310[E] (190–440)	470 (340–610)
4 (2014–2015)	1272	44.85	—	<LOD	5.2 (<LOD–7.0)	190[E] (80–290)	410[E] (220–610)
Females, 3–79 years							
2 (2009–2011)	1270	29.62	14 (11–19)	<LOD	9.9 (8.4–11)	320[E] (150–480)	570[E] (340–800)
3 (2012–2013)	2827	34.91	21 (16–27)	<LOD	11 (8.0–13)	390 (260–520)	810 (560–1100)
4 (2014–2015)	1285	44.28	—	<LOD	7.2 (<LOD–9.2)	320[E] (120–510)	810[E] (380–1300)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
2 (2009–2011)	522	29.45	14 (12–17)	<LOD	10[E] (3.9–16)	84 (58–110)	180 (140–230)
3 (2012–2013)	517	36.29	18 (15–23)	<LOD	13 (<LOD–17)	110[E] (47–180)	260 (170–350)
4 (2014–2015)	511	49.12	—	<LOD	9.2 (<LOD–11)	70[E] (38–100)	120[E] (46–200)
6–11 years							
2 (2009–2011)	513	33.98	8.5 (6.2–12)	<LOD	4.4[E] (<LOD–6.9)	150[E] (57–250)	270[E] (82–470)
3 (2012–2013)	1001	36.26	14 (11–17)	<LOD	8.8 (<LOD–11)	F	340[E] (160–530)
4 (2014–2015)	509	49.22	—	<LOD	5.4 (<LOD–7.4)	F	190[E] (60–330)
12–19 years							
2 (2009–2011)	508	19.02	14 (10–19)	<LOD	8.9[E] (5.3–13)	280[E] (150–420)	490[E] (280–710)
3 (2012–2013)	983	28.35	14 (11–19)	<LOD	8.7 (7.0–11)	350[E] (160–540)	530 (380–680)
4 (2014–2015)	504	38.89	9.1 (6.7–12)	<LOD	5.1[E] (<LOD–7.2)	F	440 (290–590)
20–39 years							
2 (2009–2011)	351	19.26	17[E] (11–27)	<LOD	11[E] (6.2–15)	410[E] (220–600)	680[E] (290–1100)
3 (2012–2013)	1035	27.44	22 (16–29)	<LOD	11 (7.6–15)	350 (270–430)	560[E] (320–810)
4 (2014–2015)	361	34.63	9.7[E] (5.4–17)	<LOD	5.8[E] (<LOD–8.8)	270[E] (71–460)	740[E] (350–1100)
40–59 years							
2 (2009–2011)	357	28.97	17[E] (11–28)	<LOD	9.7[E] (2.7–17)	410[E] (230–590)	820[E] (440–1200)
3 (2012–2013)	1071	37.22	17 (13–22)	<LOD	9.4 (7.1–12)	400[E] (240–560)	900[E] (410–1400)
4 (2014–2015)	312	41.03	—	<LOD	8.2[E] (<LOD–13)	F	F
60–79 years							
2 (2009–2011)	289	41.72	—	<LOD	6.6 (<LOD–9.0)	370[E] (200–550)	600[E] (280–910)
3 (2012–2013)	1035	41.84	—	<LOD	9.5 (8.2–11)	340[E] (200–480)	720[E] (430–1000)
4 (2014–2015)	360	52.50	—	<LOD	<LOD	F	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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SUMMARIES AND RESULTS FOR METALS AND TRACE ELEMENTS 10

10.1 ARSENIC

Arsenic (CASRN 7440-38-2) is a naturally occurring element making up a small fraction (0.00015%) of the Earth's crust (ATSDR, 2007; Emsley, 2001). It is classified as a metalloid, exhibiting properties of both a metal and a non-metal. Arsenic is commonly found as an inorganic sulphide complexed with other metals (CCME, 1997). Arsenic also forms stable organic compounds in its trivalent (+3) and pentavalent (+5) states. Common organic arsenic compounds include monomethylarsonic acid (MMA), dimethylarsinic acid (DMA), arsenobetaine, and arsenocholine (WHO, 2001).

Arsenic may enter lakes, rivers, or groundwater naturally through erosion and weathering of soils, minerals, and ores (Health Canada, 2006). The primary anthropogenic sources of arsenic are the smelting of metal ores, the use of arsenical pesticides, and the burning of fossil fuels (WHO, 2001).

Arsenic is used in the manufacture of transistors, lasers, and semi-conductors, and in the processing of glass, pigments, textiles, paper, metal adhesives, ceramics, wood preservatives, ammunition, and explosives. Historical uses of arsenic include application of lead arsenate as a pesticide in apple orchards and vineyards and arsenic trioxide as a herbicide (ATSDR, 2007; Health Canada, 2006). Chromated copper arsenate was formerly used as a wood preservative in residential construction projects, such as playground structures and decks; however, it is now used only for industrial purposes and for domestic wood foundations (Health Canada, 2005). Organic arsenical herbicides, such as MMA and DMA, are no longer registered for use in Canada (Environment Canada, 2008; Health Canada, 2016a).

The public can be exposed to arsenic through food, drinking water, soil, and ambient air (Environment Canada and Health Canada, 1993). Food is the major source of exposure with total arsenic concentrations being highest in seafood (IARC, 2012). Organic forms of arsenic, including arsenobetaine and arsenocholine, make up the majority of arsenic in seafood (Ackley et al., 1999; Leufroy et al., 2011; Ruttens et al., 2012). In other foods, there is growing evidence to suggest that inorganic arsenic may represent the predominant form of arsenic (Batista et al., 2011; CFIA, 2013; Conklin and Chen, 2012; FDA, 2016; Health Canada, 2014; Huang et al., 2012). Exposure to arsenic may also arise from indoor house dust; levels in dust can exceed levels in soil (Rasmussen et al., 2001). Further, exposure to arsenic may be elevated in populations residing in areas where industrial or natural sources occur.

Inorganic arsenic and organic arsenic are readily absorbed via the oral and inhalation routes of exposure; arsenic in all its forms is not readily absorbed via the dermal route. Absorption of arsenic is much lower for highly insoluble forms of arsenic such as arsenic sulfide, arsenic triselenide, and lead arsenate (ATSDR, 2007). Following absorption, arsenic appears rapidly in blood circulation where it binds primarily to haemoglobin. Within 24 hours, it is found in the liver, kidney, lung, spleen, and skin. Skin, bone, and muscle represent the major storage organs. In cases of chronic exposure, arsenic will preferentially accumulate in tissues rich in keratin or sulfhydryl functional groups, such as

hair, nails, skin, and other protein-containing tissues (HBM Commission, 2003). Metabolism of inorganic arsenic involves an initial reduction of pentavalent to trivalent arsenic followed by oxidative methylation to monomethylated, dimethylated, and trimethylated products, including MMA and DMA (WHO, 2011). Methylation facilitates the excretion of inorganic arsenic from the body because the end-products MMA and DMA are water soluble and readily excreted in urine (WHO, 2001). Absorbed organic arsenic species do not undergo significant metabolism and are predominantly and rapidly eliminated in urine (WHO, 2001).

Biomarkers of arsenic exposure include the levels of arsenic or its metabolites in blood, hair, nails, and urine (WHO, 2001). Measurements of speciated metabolites in urine expressed either as inorganic arsenic or as the sum of metabolites (inorganic arsenic + MMA + DMA) are generally accepted as the most reliable indicator of recent arsenic exposure (ATSDR, 2007; WHO, 2001). Measurements of arsenic in urine have been used to identify recent arsenic ingestion or above-average exposures in populations living near industrial point sources of arsenic (ATSDR, 2007).

Acute oral arsenic exposure may cause gastrointestinal effects in humans as well as pain to the extremities and muscles (Health Canada, 2006). These symptoms are often followed by numbness and tingling of the extremities and muscular cramping and may progress into burning paraesthesias of the extremities, palmoplantar hyperkeratosis, and deterioration in motor and sensory responses (Health Canada, 2006).

Chronic exposure to inorganic arsenic has been associated with decreased lung function, non-cancer skin effects, and cardiovascular effects including increased incidence of high blood pressure and circulatory problems (ATSDR, 2007; Environment Canada and Health Canada, 1993). In addition, increased incidences of skin cancer and various cancers of the internal organs have been associated with chronic ingestion of inorganic arsenic-contaminated drinking water (Health Canada, 2006). Much of the evidence on the carcinogenicity of arsenic in humans comes from epidemiological studies conducted in populations consuming high levels of inorganic arsenic through drinking water including those from Taiwan, Chile, and Bangladesh (Health Canada 2006, 2016b). Arsenic and inorganic arsenic compounds are classified as carcinogenic to humans by Health Canada and other international agencies (EPA, 1998; Health Canada, 2006; IARC, 2012). More recently, a growing body of evidence suggests that in-utero and childhood exposure to high levels of inorganic arsenic may affect fetal and childhood health and development (EFSA CONTAM Panel, 2009; FAO/WHO, 2011; FDA, 2016; NRC, 2013). Although the current amount of information regarding developmental effects in humans is relatively limited and presents some conflicting results, the available data do raise concerns surrounding exposure to inorganic arsenic during critical windows of early development (Health Canada, 2016b). Although the majority of assessments on the toxicity of arsenic have concentrated on the inorganic forms, recent studies have highlighted the potential for organic arsenic compounds, in particular the pentavalent DMA, to exert carcinogenic effects (Cohen et al., 2006; IARC, 2012; Schwerdtle et al., 2003). The International Agency for Research on Cancer (IARC) has classified the methylated arsenic metabolites MMA and DMA as Group 2B, possibly carcinogenic to humans, based on evidence from experimental animals (IARC, 2012). IARC has also evaluated arsenobetaine and other organic arsenic compounds and found them to be not classifiable as to their carcinogenicity to humans (Group 3) (IARC, 2012).

Health Canada and Environment Canada concluded that arsenic and its inorganic compounds in Canada may be harmful to the environment and may constitute a danger to human life or health (Environment Canada and Health Canada, 1993). Inorganic arsenic compounds are listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The Act allows the federal government to control the importation, manufacture, distribution, and use of inorganic arsenic compounds in Canada (Canada, 1999; Canada, 2000). Risk management actions under CEPA 1999 have been developed to control releases of arsenic from thermal electric power generation, base-metal smelting, wood preservation, and steel manufacturing processes (Environment Canada, 2010). Arsenic and its compounds are included as prohibited ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section

16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Canada, 1985; Health Canada, 2015). The Food and Drug Regulations prohibit the sale in Canada of drugs for human use containing arsenic or any of its salts or derivatives (Canada, 2012). Further, the leachable arsenic content in a variety of consumer products is regulated under the *Canada Consumer Product Safety Act* (Canada, 2010a). These regulated consumer products include paints and other surface coatings on cribs, toys, and other products for use by a child in learning or play situations (Canada, 2010b; Canada, 2011).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for arsenic in drinking water (Health Canada, 2006). The guideline was developed based on the incidence of internal (lung, bladder, and liver) cancers in humans and the ability of currently available treatment technologies to remove arsenic from drinking water at or below the guideline level (Health Canada, 2006). Arsenic is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply. The concentration of arsenic in some foods is regulated by Health Canada under the Food and Drug Regulations; the existing maximum levels for arsenic in a variety of beverages including apple juice and bottled water are in the process of being updated (Canada, 2012; Health Canada, 2014). The existing maximum levels for arsenic in other foods and beverages are also scheduled for review and update.

In a study carried out in British Columbia to assess the levels of trace elements in 61 non-smoking adults aged 30–65 years, the geometric mean concentration and 95th percentile of total arsenic in urine were 27.8 µg/g creatinine and 175.5 µg/g creatinine, respectively (Clark et al., 2007). In a biomonitoring study carried out in the region of the city of Québec with 500 participants aged 18–65 years, the geometric mean of total arsenic in urine was 12.73 µg/L and in whole blood was 0.95 µg/L (INSPQ, 2004).

Arsenite (+3), arsenate (+5), and methylated metabolites of arsenic (MMA and DMA) were analyzed individually in the urine of Canadian Health Measures Survey (CHMS) cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years. The data from these cycles are presented as both µg As/L and µg As/g creatinine. The organoarsenic compounds, arsenobetaine and arsenocholine, were analyzed together in the urine of CHMS cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years and arsenocholine was also analyzed alone in cycles 3 and 4. The data are presented as both µg As/L and µg As/g creatinine. Finding a measurable amount of arsenic in urine is an indicator of exposure to arsenic and does not necessarily mean that an adverse health effect will occur.

Table 10.1.1

Inorganic arsenic species, sum of arsenate, arsenite, dimethylarsinic acid and monomethylarsonic acid[a] — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years						
2 (2009–2011)	2537	5.3 (4.7–6.0)	2.1 (2.0–2.3)	4.8 (4.2–5.4)	14 (11–18)	22[E] (12–33)
3 (2012–2013)	2535	5.4 (4.9–6.0)	2.2 (2.0–2.5)	4.6 (4.2–5.0)	14 (10–18)	21[E] (12–31)
4 (2014–2015)	2567	5.3 (4.9–5.9)	2.2 (2.1–2.4)	4.7 (4.2–5.3)	14 (12–16)	20 (15–25)
Males, 3–79 years						
2 (2009–2011)	1271	5.5 (4.8–6.4)	2.2 (1.8–2.5)	5.0 (3.9–6.1)	15 (11–19)	22[E] (12–32)
3 (2012–2013)	1250	5.6 (5.0–6.3)	2.4 (1.9–3.0)	5.1 (4.4–5.8)	13 (10–15)	19[E] (7.9–29)
4 (2014–2015)	1275	5.6 (4.9–6.4)	2.2 (2.0–2.4)	4.9 (4.1–5.7)	15 (12–19)	25[E] (15–35)
Females, 3–79 years						
2 (2009–2011)	1266	5.1 (4.5–5.8)	2.1 (1.8–2.4)	4.7 (4.2–5.2)	14 (10–18)	22[E] (8.9–36)
3 (2012–2013)	1285	5.2 (4.5–6.1)	2.2 (2.0–2.3)	4.3 (3.9–4.7)	16[E] (8.2–23)	F
4 (2014–2015)	1292	5.1 (4.6–5.7)	2.3 (2.1–2.5)	4.5 (3.9–5.1)	13 (10–16)	17 (12–23)
3–5 years						
2 (2009–2011)	516	5.2 (4.6–5.9)	2.5 (2.3–2.7)	4.6 (4.1–5.1)	11 (7.4–15)	16[E] (10–22)
3 (2012–2013)	500	5.0 (4.6–5.4)	2.2 (1.9–2.5)	4.5 (4.0–5.1)	13 (10–16)	19[E] (11–26)
4 (2014–2015)	512	5.0 (4.5–5.6)	2.3 (2.0–2.6)	4.6 (4.0–5.1)	12 (9.5–14)	15[E] (9.6–21)
6–11 years						
2 (2009–2011)	511	5.5 (5.1–6.0)	2.6 (2.3–2.9)	5.4 (4.8–6.1)	12 (9.7–14)	17 (11–23)
3 (2012–2013)	506	5.2 (4.5–6.0)	2.2 (1.7–2.7)	4.9 (4.2–5.6)	11 (7.8–14)	17[E] (9.1–25)
4 (2014–2015)	514	5.5 (4.9–6.3)	2.5 (2.0–2.9)	5.0 (4.3–5.7)	13 (8.9–18)	20[E] (8.1–32)
12–19 years						
2 (2009–2011)	510	5.5 (4.6–6.6)	2.3 (1.9–2.7)	4.8 (3.6–6.0)	15 (11–19)	22[E] (12–32)
3 (2012–2013)	510	5.4 (4.7–6.3)	2.4 (2.0–2.9)	4.7 (3.5–5.9)	13 (8.4–17)	20[E] (7.7–31)
4 (2014–2015)	506	5.5 (4.7–6.4)	2.4 (1.9–2.8)	4.6 (3.8–5.5)	14 (9.3–18)	19 (14–24)
20–39 years						
2 (2009–2011)	355	5.6 (4.6–6.8)	2.1 (1.8–2.4)	5.1 (3.8–6.3)	F	28[E] (16–41)
3 (2012–2013)	355	5.8 (5.0–6.6)	2.4 (1.7–3.1)	4.8 (4.1–5.5)	15[E] (5.6–25)	31[E] (9.7–52)
4 (2014–2015)	362	5.5 (4.9–6.1)	2.2 (1.8–2.6)	4.9 (4.2–5.7)	14 (12–16)	16 (13–20)
40–59 years						
2 (2009–2011)	356	4.9 (4.2–5.7)	2.0 (1.6–2.5)	4.2 (3.6–4.9)	12 (9.2–15)	15 (12–19)
3 (2012–2013)	312	5.3 (4.3–6.4)	2.2 (1.8–2.6)	4.5 (3.7–5.3)	15[E] (5.6–23)	F
4 (2014–2015)	312	5.1 (4.4–6.0)	2.2 (2.0–2.4)	4.3 (3.4–5.1)	14[E] (4.8–23)	23[E] (13–32)

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years						
2 (2009–2011)	289	5.4 (4.4–6.6)	2.2 (1.9–2.4)	4.7 (4.1–5.4)	16[E] (8.9–24)	F
3 (2012–2013)	352	5.3 (4.6–6.2)	2.2 (2.0–2.3)	4.7 (3.8–5.5)	14 (11–17)	22[E] (14–31)
4 (2014–2015)	361	5.4 (4.5–6.5)	2.3 (1.9–2.6)	4.8 (3.7–6.0)	15 (10–19)	18[E] (6.2–29)

a For each individual within a cycle, the sum of arsenate, arsenite, dimethylarsinic acid, and monomethylarsonic acid is calculated. If the value of a species is less than the limit of detection (LOD), then the imputed value calculated as LOD divided by 2 is used. If all four arsenic species are reported as less than the LOD, then the sum will be the sum of the four imputed values.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.2

Inorganic arsenic species, sum of arsenate, arsenite, dimethylarsinic acid and monomethylarsonic acid[a] (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years						
2 (2009–2011)	2527	5.3 (4.6–6.0)	2.3 (2.1–2.5)	4.7 (4.0–5.4)	13 (9.1–17)	20 (13–27)
3 (2012–2013)	2534	5.5 (4.8–6.3)	2.2 (2.0–2.5)	4.9 (4.4–5.5)	14[E] (7.8–21)	26[E] (12–39)
4 (2014–2015)	2566	4.8 (4.3–5.4)	2.1 (1.9–2.3)	4.3 (3.8–4.7)	12 (8.7–16)	18 (14–22)
Males, 3–79 years						
2 (2009–2011)	1267	4.7 (4.1–5.5)	2.2 (2.0–2.5)	4.2 (3.4–4.9)	10 (8.0–13)	15[E] (5.8–24)
3 (2012–2013)	1250	4.6 (4.2–5.1)	2.0 (1.7–2.3)	4.4 (3.7–5.1)	9.6 (7.7–12)	17[E] (9.2–24)
4 (2014–2015)	1274	4.4 (3.9–5.0)	2.0 (1.8–2.3)	3.9 (3.5–4.4)	10 (7.3–13)	15 (11–19)
Females, 3–79 years						
2 (2009–2011)	1260	5.8 (5.1–6.8)	2.4 (2.1–2.8)	5.3 (4.5–6.1)	15 (10–21)	22[E] (14–30)
3 (2012–2013)	1284	6.6 (5.5–8.0)	2.5 (2.2–2.9)	5.8 (4.8–6.7)	19[E] (5.6–33)	33[E] (18–49)
4 (2014–2015)	1292	5.3 (4.5–6.1)	2.4 (2.0–2.7)	4.7 (4.1–5.4)	14 (9.0–18)	20 (15–25)
3–5 years						
2 (2009–2011)	515	9.1 (8.1–10)	4.6 (4.0–5.2)	8.0 (7.0–8.9)	19 (15–24)	29[E] (13–45)
3 (2012–2013)	499	9.6 (8.8–10)	4.7 (4.2–5.2)	8.7 (7.9–9.5)	20 (15–25)	29[E] (13–45)
4 (2014–2015)	512	8.7 (8.0–9.5)	4.2 (3.6–4.8)	7.9 (7.2–8.6)	19 (15–23)	26 (18–34)
6–11 years						
2 (2009–2011)	509	6.4 (5.8–7.1)	3.2 (2.9–3.5)	5.9 (5.2–6.5)	14 (10–17)	23[E] (14–31)
3 (2012–2013)	506	6.6 (5.8–7.5)	3.4 (3.1–3.7)	5.9 (5.3–6.5)	13 (9.2–17)	17[E] (9.8–25)
4 (2014–2015)	513	6.1 (5.5–6.7)	3.0 (2.8–3.3)	5.5 (4.9–6.0)	14 (9.9–18)	18[E] (11–25)

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years						
2 (2009–2011)	508	4.2 (3.6–5.0)	1.9 (1.6–2.2)	3.6 (3.0–4.2)	12[E] (6.7–16)	17[E] (9.4–26)
3 (2012–2013)	510	4.1 (3.3–5.0)	1.9 (1.7–2.1)	3.5 (2.8–4.1)	10[E] (5.5–15)	17[E] (9.4–24)
4 (2014–2015)	506	4.0 (3.5–4.5)	1.7 (1.4–2.0)	3.6 (3.0–4.2)	9.1 (6.3–12)	13[E] (8.0–18)
20–39 years						
2 (2009–2011)	353	4.8 (3.8–5.9)	2.3 (1.9–2.6)	3.9 (2.7–5.1)	12[E] (4.2–21)	21[E] (12–31)
3 (2012–2013)	355	4.4 (3.8–5.1)	1.8 (1.3–2.3)	3.8 (3.0–4.5)	F	F
4 (2014–2015)	362	4.4 (3.8–5.1)	2.0 (1.8–2.3)	3.9 (3.3–4.5)	10 (6.6–14)	15[E] (7.5–22)
40–59 years						
2 (2009–2011)	354	5.0 (4.5–5.6)	2.3 (2.0–2.5)	4.6 (3.8–5.5)	10 (7.6–13)	14[E] (9.2–20)
3 (2012–2013)	312	6.2 (5.1–7.6)	2.5 (2.2–2.9)	5.7 (4.7–6.8)	F	F
4 (2014–2015)	312	4.7 (3.9–5.5)	2.1 (1.7–2.4)	4.2 (3.8–4.6)	11[E] (5.1–17)	19[E] (9.6–29)
60–79 years						
2 (2009–2011)	288	6.4 (5.2–7.8)	2.5 (2.1–3.0)	6.0 (4.7–7.3)	16[E] (6.2–25)	26[E] (8.6–43)
3 (2012–2013)	352	6.0 (4.9–7.2)	2.6 (2.1–3.2)	5.1 (4.0–6.2)	F	27[E] (15–40)
4 (2014–2015)	361	5.2 (4.5–6.1)	2.3 (2.1–2.5)	4.5 (3.5–5.5)	13 (9.2–16)	19[E] (10–28)

a For each individual within a cycle, the sum of arsenate, arsenite, dimethylarsinic acid, and monomethylarsonic acid is calculated. If the value of a species is less than the limit of detection (LOD), then the imputed value calculated as LOD divided by 2 is used. If all four arsenic species are reported as less than the LOD, then the sum will be the sum of the four imputed values.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.3

Arsenate — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	99.49	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2536	99.25	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2567	98.95	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1271	99.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1251	99.04	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1275	98.82	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1267	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1285	99.46	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1292	99.07	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	516	98.84	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	500	98.60	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	98.44	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	511	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	507	99.61	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	514	98.64	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	510	99.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	510	98.82	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	98.42	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	355	99.44	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	355	99.72	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	99.45	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	357	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	99.04	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	99.68	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	289	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	352	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	99.72	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 10.1.4

Arsenate (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	99.49	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2535	99.25	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2566	98.95	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1267	99.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1251	99.04	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1274	98.82	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1261	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1284	99.46	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1292	99.07	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	515	98.84	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	499	98.60	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	98.44	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	509	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	507	99.61	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	513	98.64	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	508	99.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	510	98.82	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	98.42	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	353	99.44	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	355	99.72	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	99.45	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	355	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	99.04	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	99.68	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	288	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	352	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	99.72	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 10.1.5

Arsenite — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2537	75.60	—	<LOD	<LOD	1.7 (1.1–2.3)	2.7[E] (1.3–4.0)
3 (2012–2013)	2535	73.96	—	<LOD	<LOD	1.7[E] (0.92–2.5)	F
4 (2014–2015)	2567	70.63	—	<LOD	<LOD	1.9 (1.5–2.3)	2.7 (2.1–3.4)
Males, 3–79 years							
2 (2009–2011)	1271	72.54	—	<LOD	<LOD	1.7 (1.1–2.3)	2.8[E] (0.88–4.7)
3 (2012–2013)	1250	71.20	—	<LOD	<LOD	1.4 (1.0–1.8)	F
4 (2014–2015)	1275	69.02	—	<LOD	<LOD	2.2 (1.7–2.6)	3.0 (2.3–3.8)
Females, 3–79 years							
2 (2009–2011)	1266	78.67	—	<LOD	<LOD	1.5[E] (0.72–2.3)	2.4[E] (1.1–3.7)
3 (2012–2013)	1285	76.65	—	<LOD	<LOD	F	F
4 (2014–2015)	1292	72.21	—	<LOD	<LOD	1.5 (1.1–2.0)	2.4[E] (1.3–3.5)
3–5 years							
2 (2009–2011)	516	84.50	—	<LOD	<LOD	0.79[E] (<LOD–1.2)	1.3[E] (0.74–1.9)
3 (2012–2013)	500	81.80	—	<LOD	<LOD	0.94 (<LOD–1.2)	1.9[E] (0.75–3.0)
4 (2014–2015)	512	80.47	—	<LOD	<LOD	1.1 (0.84–1.3)	1.8[E] (1.0–2.5)
6–11 years							
2 (2009–2011)	511	78.86	—	<LOD	<LOD	1.0[E] (<LOD–1.4)	1.8[E] (1.1–2.4)
3 (2012–2013)	506	76.09	—	<LOD	<LOD	1.1 (0.81–1.4)	1.6[E] (0.82–2.5)
4 (2014–2015)	514	75.88	—	<LOD	<LOD	1.5[E] (0.92–2.0)	2.6[E] (1.2–4.0)
12–19 years							
2 (2009–2011)	510	72.35	—	<LOD	<LOD	1.9[E] (1.2–2.7)	F
3 (2012–2013)	510	68.43	—	<LOD	<LOD	1.5[E] (<LOD–2.3)	2.6[E] (1.1–4.0)
4 (2014–2015)	506	62.25	—	<LOD	<LOD	2.1[E] (1.2–3.0)	3.2 (2.1–4.4)
20–39 years							
2 (2009–2011)	355	69.86	—	<LOD	<LOD	1.9[E] (<LOD–3.1)	F
3 (2012–2013)	355	70.70	—	<LOD	<LOD	F	F
4 (2014–2015)	362	62.71	—	<LOD	<LOD	2.3 (1.7–2.8)	3.0 (2.3–3.8)
40–59 years							
2 (2009–2011)	356	70.51	—	<LOD	<LOD	1.3[E] (0.75–1.8)	2.0[E] (1.0–2.9)
3 (2012–2013)	312	70.19	—	<LOD	<LOD	F	F
4 (2014–2015)	312	70.83	—	<LOD	<LOD	1.6[E] (1.0–2.3)	2.3[E] (1.5–3.2)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	289	73.01	—	<LOD	<LOD	1.9[E] (1.1–2.7)	F
3 (2012–2013)	352	74.43	—	<LOD	<LOD	1.8 (1.1–2.4)	3.2[E] (1.3–5.2)
4 (2014–2015)	361	68.70	—	<LOD	<LOD	1.8 (1.2–2.3)	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.6

Arsenite (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2527	75.60	—	<LOD	<LOD	2.0 (1.6–2.3)	2.9 (1.9–3.9)
3 (2012–2013)	2534	73.96	—	<LOD	<LOD	1.9[E] (1.2–2.7)	F
4 (2014–2015)	2566	70.63	—	<LOD	<LOD	1.6 (1.3–1.9)	2.2 (1.5–2.9)
Males, 3–79 years							
2 (2009–2011)	1267	72.54	—	<LOD	<LOD	1.4[E] (0.85–1.9)	F
3 (2012–2013)	1250	71.20	—	<LOD	<LOD	1.2 (0.94–1.5)	F
4 (2014–2015)	1274	69.02	—	<LOD	<LOD	1.5 (1.0–1.9)	2.0 (1.4–2.6)
Females, 3–79 years							
2 (2009–2011)	1260	78.67	—	<LOD	<LOD	2.2 (1.6–2.8)	3.0 (2.1–3.9)
3 (2012–2013)	1284	76.65	—	<LOD	<LOD	2.4[E] (<LOD–3.9)	F
4 (2014–2015)	1292	72.21	—	<LOD	<LOD	1.7 (1.2–2.1)	2.6[E] (1.4–3.9)
3–5 years							
2 (2009–2011)	515	84.50	—	<LOD	<LOD	1.9 (<LOD–2.2)	2.9 (1.9–3.9)
3 (2012–2013)	499	81.80	—	<LOD	<LOD	2.5[E] (<LOD–3.7)	4.3[E] (2.6–6.1)
4 (2014–2015)	512	80.47	—	<LOD	<LOD	2.1 (1.8–2.5)	3.0[E] (1.8–4.2)
6–11 years							
2 (2009–2011)	509	78.86	—	<LOD	<LOD	1.6[E] (<LOD–2.2)	2.2[E] (1.2–3.1)
3 (2012–2013)	506	76.09	—	<LOD	<LOD	1.7 (1.1–2.2)	2.5[E] (1.3–3.6)
4 (2014–2015)	513	75.88	—	<LOD	<LOD	1.6 (1.2–2.0)	2.2[E] (0.77–3.7)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	508	72.35	—	<LOD	<LOD	1.4[E] (0.85–2.0)	2.9[E] (1.4–4.5)
3 (2012–2013)	510	68.43	—	<LOD	<LOD	1.4[E] (<LOD–2.0)	1.9[E] (1.0–2.8)
4 (2014–2015)	506	62.25	—	<LOD	<LOD	1.4 (1.0–1.8)	2.0[E] (1.2–2.8)
20–39 years							
2 (2009–2011)	353	69.86	—	<LOD	<LOD	1.9[E] (<LOD–3.0)	2.6[E] (<LOD–4.3)
3 (2012–2013)	355	70.70	—	<LOD	<LOD	F	F
4 (2014–2015)	362	62.71	—	<LOD	<LOD	1.6 (1.0–2.1)	2.1[E] (1.2–3.0)
40–59 years							
2 (2009–2011)	354	70.51	—	<LOD	<LOD	1.9 (1.3–2.6)	2.0[E] (1.2–2.8)
3 (2012–2013)	312	70.19	—	<LOD	<LOD	F	F
4 (2014–2015)	312	70.83	—	<LOD	<LOD	1.4 (0.93–1.9)	F
60–79 years							
2 (2009–2011)	288	73.01	—	<LOD	<LOD	2.3[E] (1.2–3.3)	F
3 (2012–2013)	352	74.43	—	<LOD	<LOD	2.3[E] (0.79–3.8)	3.7[E] (1.7–5.6)
4 (2014–2015)	361	68.70	—	<LOD	<LOD	1.7 (1.3–2.0)	2.6[E] (<LOD–4.0)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.7

Arsenocholine — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2536	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2566	100	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
3 (2012–2013)	1251	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1275	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
3 (2012–2013)	1285	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1291	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
3 (2012–2013)	500	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
3 (2012–2013)	507	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	514	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	510	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	355	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	312	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	311	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	352	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 10.1.8

Arsenocholine (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2535	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2565	100	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
3 (2012–2013)	1251	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1274	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
3 (2012–2013)	1284	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1291	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
3 (2012–2013)	499	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
3 (2012–2013)	507	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	513	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	510	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	355	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	312	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	311	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	352	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 10.1.9

Arsenocholine and arsenobetaine — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	48.50	—	<LOD	1.4[E] (<LOD–2.2)	28[E] (18–39)	48[E] (30–67)
3 (2012–2013)	2536	48.15	—	<LOD	1.4[E] (<LOD–2.1)	24[E] (11–36)	56 (37–75)
4 (2014–2015)	2564	47.85	—	<LOD	1.2[E] (<LOD–1.7)	28[E] (13–44)	49 (33–65)
Males, 3–79 years							
2 (2009–2011)	1271	46.34	—	<LOD	1.5[E] (<LOD–2.5)	29[E] (14–43)	F
3 (2012–2013)	1251	47.40	—	<LOD	1.4[E] (<LOD–2.0)	21[E] (13–29)	38 (25–51)
4 (2014–2015)	1273	47.68	—	<LOD	1.6[E] (<LOD–2.6)	33[E] (12–54)	44 (30–59)
Females, 3–79 years							
2 (2009–2011)	1267	50.67	—	<LOD	<LOD	28[E] (15–41)	49[E] (29–69)
3 (2012–2013)	1285	48.87	—	<LOD	1.5[E] (<LOD–2.6)	F	58[E] (33–83)
4 (2014–2015)	1291	48.02	—	<LOD	F	F	52[E] (18–86)
3–5 years							
2 (2009–2011)	516	59.69	—	<LOD	<LOD	F	34[E] (19–49)
3 (2012–2013)	500	57.40	—	<LOD	<LOD	12[E] (6.3–17)	F
4 (2014–2015)	512	59.96	—	<LOD	<LOD	16[E] (5.4–26)	F
6–11 years							
2 (2009–2011)	511	58.12	—	<LOD	<LOD	F	F
3 (2012–2013)	507	59.57	—	<LOD	<LOD	F	27[E] (14–39)
4 (2014–2015)	512	59.77	—	<LOD	<LOD	15[E] (5.2–25)	39[E] (13–64)
12–19 years							
2 (2009–2011)	510	57.65	—	<LOD	<LOD	12[E] (4.5–19)	38[E] (16–59)
3 (2012–2013)	510	51.18	—	<LOD	<LOD	16[E] (7.2–24)	37[E] (17–56)
4 (2014–2015)	506	49.80	—	<LOD	0.75[E] (<LOD–1.2)	16[E] (9.4–22)	26[E] (13–39)
20–39 years							
2 (2009–2011)	355	38.59	2.3[E] (1.5–3.6)	<LOD	F	33[E] (15–52)	68[E] (20–110)
3 (2012–2013)	355	44.51	—	<LOD	F	19[E] (11–28)	35[E] (12–58)
4 (2014–2015)	361	37.12	1.9 (1.5–2.5)	<LOD	1.5[E] (<LOD–2.4)	32[E] (17–47)	46[E] (24–67)
40–59 years							
2 (2009–2011)	357	30.81	1.8 (1.4–2.4)	<LOD	1.4[E] (<LOD–2.5)	F	35[E] (19–52)
3 (2012–2013)	312	34.29	2.2[E] (1.3–3.8)	<LOD	F	F	57[E] (30–84)
4 (2014–2015)	312	38.14	1.8 (1.3–2.6)	<LOD	1.3[E] (<LOD–1.9)	F	37[E] (18–56)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	289	29.41	3.6[E] (2.2–5.9)	<LOD	3.6[E] (1.4–5.8)	40[E] (21–59)	74[E] (33–120)
3 (2012–2013)	352	30.11	2.6[E] (1.8–3.8)	<LOD	2.1[E] (0.86–3.4)	F	67[E] (29–100)
4 (2014–2015)	361	30.19	2.8[E] (1.7–4.7)	<LOD	2.5[E] (0.91–4.0)	F	88[E] (49–130)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.10

Arsenocholine and arsenobetaine (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	48.50	—	<LOD	1.5[E] (<LOD–2.5)	22 (16–28)	44[E] (18–71)
3 (2012–2013)	2535	48.15	—	<LOD	1.6 (<LOD–2.1)	25[E] (12–39)	44[E] (24–63)
4 (2014–2015)	2563	47.85	—	<LOD	1.2 (<LOD–1.5)	23[E] (12–34)	46[E] (27–65)
Males, 3–79 years							
2 (2009–2011)	1267	46.34	—	<LOD	F	18[E] (9.4–27)	F
3 (2012–2013)	1251	47.40	—	<LOD	1.2 (<LOD–1.6)	16[E] (7.3–24)	34 (25–43)
4 (2014–2015)	1272	47.68	—	<LOD	1.3[E] (<LOD–1.8)	20[E] (9.8–30)	37[E] (19–55)
Females, 3–79 years							
2 (2009–2011)	1261	50.67	—	<LOD	<LOD	25 (19–32)	61[E] (20–100)
3 (2012–2013)	1284	48.87	—	<LOD	2.1[E] (<LOD–3.3)	33[E] (9.5–56)	F
4 (2014–2015)	1291	48.02	—	<LOD	1.1 (<LOD–1.4)	F	62[E] (36–89)
3–5 years							
2 (2009–2011)	515	59.69	—	<LOD	<LOD	F	F
3 (2012–2013)	499	57.40	—	<LOD	<LOD	21[E] (11–31)	F
4 (2014–2015)	512	59.96	—	<LOD	<LOD	26[E] (14–38)	57[E] (15–98)
6–11 years							
2 (2009–2011)	509	58.12	—	<LOD	<LOD	F	F
3 (2012–2013)	507	59.57	—	<LOD	<LOD	F	40[E] (12–69)
4 (2014–2015)	511	59.77	—	<LOD	<LOD	17[E] (8.4–27)	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	508	57.65	—	<LOD	<LOD	9.3[E] (4.0–15)	24[E] (10–38)
3 (2012–2013)	510	51.18	—	<LOD	<LOD	10[E] (3.8–17)	F
4 (2014–2015)	506	49.80	—	<LOD	0.72[E] (<LOD–1.0)	9.9[E] (5.4–14)	F
20–39 years							
2 (2009–2011)	353	38.59	1.9[E] (1.2–2.8)	<LOD	F	22[E] (7.8–37)	F
3 (2012–2013)	355	44.51	—	<LOD	1.4[E] (<LOD–1.9)	12[E] (5.5–19)	21[E] (9.8–32)
4 (2014–2015)	361	37.12	1.6 (1.2–2.1)	<LOD	1.1[E] (<LOD–1.6)	20 (13–27)	29[E] (7.7–50)
40–59 years							
2 (2009–2011)	355	30.81	1.8 (1.3–2.5)	<LOD	1.9[E] (<LOD–3.1)	17[E] (10–24)	24[E] (9.8–39)
3 (2012–2013)	312	34.29	2.6[E] (1.6–4.4)	<LOD	F	33[E] (14–52)	F
4 (2014–2015)	312	38.14	1.7 (1.2–2.4)	<LOD	1.1[E] (<LOD–1.5)	F	F
60–79 years							
2 (2009–2011)	288	29.41	4.2[E] (2.6–6.8)	<LOD	4.6[E] (1.7–7.5)	47[E] (13–80)	84[E] (43–120)
3 (2012–2013)	352	30.11	2.9[E] (1.9–4.4)	<LOD	F	35[E] (<LOD–57)	F
4 (2014–2015)	361	30.19	2.8[E] (1.7–4.4)	<LOD	2.2[E] (0.93–3.5)	F	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.11

Dimethylarsinic acid (DMA) — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	3.78	3.5 (3.0–4.0)	0.93 (0.89–0.97)	3.6 (3.1–4.1)	11 (8.3–13)	16[E] (6.6–25)
3 (2012–2013)	2536	3.86	3.6 (3.2–4.0)	1.1 (0.89–1.4)	3.4 (3.0–3.8)	11 (7.8–13)	16[E] (7.4–25)
4 (2014–2015)	2567	2.65	3.5 (3.1–3.9)	1.1 (1.0–1.3)	3.4 (3.0–3.8)	10 (8.2–12)	15 (11–20)
Males, 3–79 years							
2 (2009–2011)	1271	3.15	3.6 (3.1–4.3)	0.95 (<LOD–1.3)	3.7 (2.8–4.5)	11 (7.9–14)	16[E] (7.7–24)
3 (2012–2013)	1251	2.96	3.8 (3.3–4.4)	1.3[E] (0.75–1.8)	3.8 (3.3–4.3)	9.8 (7.8–12)	14[E] (4.8–23)
4 (2014–2015)	1275	2.27	3.6 (3.1–4.3)	1.1 (0.81–1.3)	3.6 (3.0–4.3)	11 (8.2–14)	19[E] (9.8–28)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1267	4.42	3.3 (2.8–3.9)	0.92 (0.75–1.1)	3.5 (3.0–3.9)	11 (7.5–14)	18[E] (7.3–29)
3 (2012–2013)	1285	4.75	3.4 (2.9–4.1)	1.0 (0.85–1.2)	3.1 (2.7–3.5)	12 (8.4–16)	F
4 (2014–2015)	1292	3.02	3.4 (3.0–3.9)	1.2 (1.1–1.4)	3.3 (2.9–3.7)	9.8 (7.7–12)	13 (9.0–17)
3–5 years							
2 (2009–2011)	516	3.68	3.6 (3.1–4.3)	1.4[E] (0.89–1.9)	3.5 (3.0–4.0)	9.4 (6.9–12)	13[E] (8.5–18)
3 (2012–2013)	500	3.00	3.3 (3.0–3.8)	1.1 (0.83–1.4)	3.4 (2.8–3.9)	10 (7.9–12)	16[E] (9.9–21)
4 (2014–2015)	512	2.34	3.4 (3.0–4.0)	1.2 (0.94–1.4)	3.4 (3.0–3.9)	9.2 (7.3–11)	13 (9.1–16)
6–11 years							
2 (2009–2011)	511	2.74	3.9 (3.5–4.4)	1.5 (1.0–1.9)	4.1 (3.5–4.7)	9.8 (8.4–11)	14[E] (7.7–20)
3 (2012–2013)	507	2.76	3.6 (3.1–4.1)	1.1[E] (<LOD–1.6)	3.7 (3.0–4.4)	9.1 (6.6–12)	14[E] (6.9–22)
4 (2014–2015)	514	2.14	3.8 (3.2–4.5)	1.3 (0.89–1.7)	3.9 (3.3–4.5)	10 (6.4–14)	16[E] (5.7–26)
12–19 years							
2 (2009–2011)	510	2.75	3.6 (2.9–4.6)	0.94[E] (<LOD–1.5)	3.5 (2.5–4.4)	11 (7.5–14)	17[E] (9.3–25)
3 (2012–2013)	510	3.53	3.6 (3.0–4.3)	1.3 (0.88–1.7)	3.4 (2.6–4.2)	9.9 (6.6–13)	F
4 (2014–2015)	506	1.38	3.6 (3.0–4.3)	1.2[E] (0.77–1.7)	3.3 (2.8–3.9)	10 (7.9–13)	13 (8.6–18)
20–39 years							
2 (2009–2011)	355	5.63	3.6 (2.9–4.5)	0.92 (0.72–1.1)	3.9 (3.0–4.8)	F	22[E] (11–33)
3 (2012–2013)	355	4.79	3.8 (3.3–4.5)	1.2[E] (<LOD–1.9)	3.5 (2.9–4.1)	12[E] (4.4–20)	24[E] (8.5–40)
4 (2014–2015)	362	3.31	3.6 (3.1–4.1)	1.1 (<LOD–1.4)	3.4 (2.7–4.0)	9.9 (8.4–11)	12 (9.3–15)
40–59 years							
2 (2009–2011)	357	5.32	3.2 (2.6–3.8)	0.91[E] (<LOD–1.2)	3.1 (2.5–3.8)	9.0 (7.4–11)	12 (8.8–15)
3 (2012–2013)	312	6.09	3.5 (2.8–4.4)	1.1 (0.77–1.5)	3.4 (2.7–4.1)	12[E] (6.0–17)	F
4 (2014–2015)	312	4.17	3.3 (2.8–4.0)	1.1 (0.89–1.3)	3.1 (2.4–3.8)	10[E] (4.7–16)	18[E] (8.6–27)
60–79 years							
2 (2009–2011)	289	3.46	3.6 (2.8–4.5)	0.92 (0.82–1.0)	3.6 (2.9–4.3)	13[E] (5.8–20)	21[E] (6.5–35)
3 (2012–2013)	352	4.26	3.5 (3.0–4.2)	1.0 (0.86–1.2)	3.4 (2.6–4.2)	10 (7.4–13)	18[E] (10–26)
4 (2014–2015)	361	3.60	3.6 (2.9–4.5)	1.2 (0.87–1.5)	3.6 (2.7–4.6)	11 (7.5–14)	14[E] (5.3–23)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.12

Dimethylarsinic acid (DMA) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	3.78	3.5 (3.0–4.0)	1.4 (1.2–1.6)	3.0 (2.6–3.4)	9.5 (7.1–12)	15[E] (9.1–21)
3 (2012–2013)	2535	3.86	3.7 (3.2–4.3)	1.4 (1.3–1.5)	3.4 (3.0–3.8)	11[E] (5.6–16)	20[E] (11–30)
4 (2014–2015)	2566	2.65	3.2 (2.8–3.6)	1.3 (1.1–1.4)	2.8 (2.5–3.2)	9.1 (6.7–12)	13 (10–16)
Males, 3–79 years							
2 (2009–2011)	1267	3.15	3.1 (2.7–3.6)	1.3 (<LOD–1.5)	2.9 (2.5–3.3)	7.7 (5.3–10)	10[E] (4.4–16)
3 (2012–2013)	1251	2.96	3.1 (2.8–3.6)	1.3 (1.1–1.4)	3.0 (2.4–3.5)	7.2 (5.4–9.1)	13[E] (7.1–19)
4 (2014–2015)	1274	2.27	2.9 (2.5–3.4)	1.1 (0.93–1.3)	2.5 (2.1–2.9)	8.4 (6.3–11)	12 (8.4–15)
Females, 3–79 years							
2 (2009–2011)	1261	4.42	3.9 (3.3–4.5)	1.6 (1.3–1.8)	3.3 (2.8–3.9)	11[E] (5.9–16)	18[E] (11–24)
3 (2012–2013)	1284	4.75	4.3 (3.6–5.3)	1.5 (1.3–1.7)	3.8 (3.1–4.4)	15[E] (5.2–25)	24[E] (15–33)
4 (2014–2015)	1292	3.02	3.5 (3.0–4.1)	1.4 (1.1–1.7)	3.0 (2.4–3.5)	10 (7.4–13)	15 (11–19)
3–5 years							
2 (2009–2011)	515	3.68	6.4 (5.6–7.3)	3.0 (2.7–3.3)	5.6 (4.7–6.5)	16 (11–20)	23[E] (10–36)
3 (2012–2013)	499	3.00	6.5 (5.9–7.1)	2.8 (2.1–3.4)	6.1 (5.5–6.8)	14 (11–17)	24[E] (13–36)
4 (2014–2015)	512	2.34	6.0 (5.4–6.6)	2.7 (2.3–3.1)	5.3 (4.8–5.8)	15 (11–18)	21[E] (12–30)
6–11 years							
2 (2009–2011)	509	2.74	4.5 (4.1–5.0)	2.1 (1.9–2.3)	4.2 (3.8–4.7)	11 (7.9–13)	17[E] (10–24)
3 (2012–2013)	507	2.76	4.5 (3.9–5.2)	2.2 (<LOD–2.4)	4.1 (3.7–4.4)	9.9 (6.7–13)	14[E] (7.2–21)
4 (2014–2015)	513	2.14	4.2 (3.7–4.8)	1.9 (1.6–2.2)	3.7 (3.3–4.2)	11 (7.6–14)	14[E] (7.7–21)
12–19 years							
2 (2009–2011)	508	2.75	2.8 (2.3–3.5)	1.1 (<LOD–1.4)	2.4 (1.9–3.0)	8.5[E] (4.5–13)	13[E] (7.6–19)
3 (2012–2013)	510	3.53	2.7 (2.2–3.4)	1.2 (1.1–1.4)	2.3 (1.7–2.9)	7.4[E] (2.9–12)	12[E] (5.9–17)
4 (2014–2015)	506	1.38	2.6 (2.3–3.1)	1.1 (0.92–1.3)	2.4 (2.0–2.8)	7.3 (4.7–9.9)	10 (6.8–13)
20–39 years							
2 (2009–2011)	353	5.63	3.1 (2.5–3.9)	1.3 (0.97–1.6)	2.6 (1.9–3.3)	9.1[E] (5.8–12)	14[E] (7.2–21)
3 (2012–2013)	355	4.79	2.9 (2.6–3.3)	1.1[E] (<LOD–1.6)	2.7 (2.3–3.0)	F	17[E] (4.7–29)
4 (2014–2015)	362	3.31	2.9 (2.5–3.4)	1.2 (<LOD–1.4)	2.5 (2.0–3.0)	8.4 (6.4–10)	11[E] (6.4–15)
40–59 years							
2 (2009–2011)	355	5.32	3.3 (2.9–3.7)	1.6 (<LOD–1.8)	3.0 (2.7–3.2)	7.7 (5.5–9.9)	11[E] (6.1–15)
3 (2012–2013)	312	6.09	4.1 (3.3–5.2)	1.5 (1.2–1.7)	3.8 (3.1–4.5)	F	24[E] (<LOD–40)
4 (2014–2015)	312	4.17	3.1 (2.5–3.7)	1.2 (1.0–1.4)	2.9 (2.3–3.5)	8.5[E] (3.3–14)	15[E] (7.1–22)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	288	3.46	4.2 (3.4–5.3)	1.5[E] (0.88–2.1)	4.1 (3.1–5.0)	F	F
3 (2012–2013)	352	4.26	4.0 (3.2–4.9)	1.5 (1.2–1.9)	3.6 (2.9–4.3)	11[E] (4.6–18)	20[E] (10–30)
4 (2014–2015)	361	3.60	3.5 (2.9–4.2)	1.4 (1.1–1.7)	2.9 (2.0–3.8)	11 (7.2–14)	14[E] (7.3–20)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 10.1.13

Monomethylarsonic acid (MMA) — Geometric means and selected percentiles of urine concentrations (µg As/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009—2011)	2538	73.01	—	<LOD	<LOD	1.2 (1.0–1.4)	1.6 (1.1–2.0)
3 (2012–2013)	2536	71.53	—	<LOD	<LOD	1.2 (1.1–1.4)	1.5 (1.3–1.7)
4 (2014–2015)	2567	71.25	—	<LOD	<LOD	1.2 (1.0–1.4)	1.6 (1.3–1.9)
Males, 3–79 years							
2 (2009–2011)	1271	69.63	—	<LOD	<LOD	1.3 (0.92–1.6)	1.8 (1.3–2.4)
3 (2012–2013)	1251	68.35	—	<LOD	<LOD	1.2 (1.0–1.4)	1.5 (1.3–1.7)
4 (2014–2015)	1275	69.65	—	<LOD	<LOD	1.3 (1.1–1.6)	1.7 (1.3–2.1)
Females, 3–79 years							
2 (2009–2011)	1267	76.40	—	<LOD	<LOD	1.1 (0.84–1.3)	1.3 (1.0–1.5)
3 (2012–2013)	1285	74.63	—	<LOD	<LOD	1.2 (0.88–1.5)	1.5 (1.3–1.8)
4 (2014–2015)	1292	72.83	—	<LOD	<LOD	1.1 (0.89–1.3)	1.5 (1.1–1.9)
3–5 years							
2 (2009–2011)	516	77.91	—	<LOD	<LOD	0.98 (0.79–1.2)	1.3 (1.1–1.5)
3 (2012–2013)	500	79.20	—	<LOD	<LOD	0.91 (<LOD–1.2)	1.5 (1.1–1.9)
4 (2014–2015)	512	79.10	—	<LOD	<LOD	0.89 (0.81–0.98)	1.1 (0.94–1.3)
6–11 years							
2 (2009–2011)	511	76.52	—	<LOD	<LOD	0.97[E] (<LOD–1.3)	1.6 (1.1–2.1)
3 (2012–2013)	507	72.58	—	<LOD	<LOD	1.0 (0.84–1.2)	1.3 (1.1–1.4)
4 (2014–2015)	514	72.57	—	<LOD	<LOD	1.2 (0.89–1.4)	1.5 (1.2–1.8)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	510	62.94	—	<LOD	<LOD	1.3 (0.97–1.6)	1.7 (1.2–2.2)
3 (2012–2013)	510	62.16	—	<LOD	<LOD	1.3 (1.1–1.6)	1.6 (1.3–1.8)
4 (2014–2015)	506	57.71	—	<LOD	<LOD	1.3 (0.88–1.8)	1.8 (1.3–2.4)
20–39 years							
2 (2009–2011)	355	70.14	—	<LOD	<LOD	1.2 (0.89–1.6)	1.7[E] (0.94–2.5)
3 (2012–2013)	355	66.48	—	<LOD	<LOD	1.3 (1.0–1.5)	1.5 (1.3–1.7)
4 (2014–2015)	362	63.54	—	<LOD	<LOD	1.3 (1.1–1.6)	1.6 (1.3–1.9)
40–59 years							
2 (2009–2011)	357	71.43	—	<LOD	<LOD	1.2 (0.92–1.5)	1.4[E] (0.87–1.9)
3 (2012–2013)	312	72.76	—	<LOD	<LOD	1.1 (0.84–1.4)	1.6 (1.1–2.2)
4 (2014–2015)	312	74.36	—	<LOD	<LOD	1.2[E] (<LOD–1.7)	1.9[E] (<LOD–3.0)
60–79 years							
2 (2009–2011)	289	81.31	—	<LOD	<LOD	1.0 (0.70–1.3)	1.4[E] (0.73–2.0)
3 (2012–2013)	352	76.70	—	<LOD	<LOD	1.1 (0.79–1.5)	1.4 (1.2–1.6)
4 (2014–2015)	361	82.27	—	<LOD	<LOD	1.1 (0.84–1.3)	1.3 (0.99–1.6)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 10.1.14

Monomethylarsonic acid (MMA) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg As/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	73.01	—	<LOD	<LOD	1.3[E] (0.75–1.8)	2.0 (1.8–2.1)
3 (2012–2013)	2535	71.53	—	<LOD	<LOD	1.2 (1.1–1.4)	1.7 (1.5–1.9)
4 (2014–2015)	2566	71.25	—	<LOD	<LOD	1.2 (0.97–1.3)	1.4 (1.2–1.7)
Males, 3–79 years							
2 (2009–2011)	1267	69.63	—	<LOD	<LOD	1.0 (0.87–1.1)	1.6[E] (1.0–2.2)
3 (2012–2013)	1251	68.35	—	<LOD	<LOD	1.0 (0.87–1.1)	1.3 (1.0–1.6)
4 (2014–2015)	1274	69.65	—	<LOD	<LOD	1.0 (0.91–1.2)	1.3 (1.0–1.6)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1261	76.40	—	<LOD	<LOD	1.9 (1.4–2.4)	2.0 (1.4–2.5)
3 (2012–2013)	1284	74.63	—	<LOD	<LOD	1.6 (1.3–1.9)	2.1 (1.8–2.5)
4 (2014–2015)	1292	72.83	—	<LOD	<LOD	1.3 (0.97–1.6)	1.7 (1.2–2.2)
3–5 years							
2 (2009–2011)	515	77.91	—	<LOD	<LOD	1.9 (1.8–2.0)	2.7 (1.8–3.6)
3 (2012–2013)	499	79.20	—	<LOD	<LOD	2.0 (<LOD–2.5)	3.0 (2.0–4.0)
4 (2014–2015)	512	79.10	—	<LOD	<LOD	1.8 (1.3–2.2)	2.2 (1.9–2.5)
6–11 years							
2 (2009–2011)	509	76.52	—	<LOD	<LOD	1.2 (<LOD–1.6)	1.9 (1.7–2.1)
3 (2012–2013)	507	72.58	—	<LOD	<LOD	1.3 (1.1–1.5)	1.8 (1.5–2.0)
4 (2014–2015)	513	72.57	—	<LOD	<LOD	1.2 (1.0–1.3)	1.4 (1.2–1.5)
12–19 years							
2 (2009–2011)	508	62.94	—	<LOD	<LOD	0.99 (0.84–1.1)	1.3[E] (0.74–1.9)
3 (2012–2013)	510	62.16	—	<LOD	<LOD	0.99 (0.75–1.2)	1.5 (1.0–2.0)
4 (2014–2015)	506	57.71	—	<LOD	<LOD	0.98 (0.82–1.1)	1.1 (0.87–1.4)
20–39 years							
2 (2009–2011)	353	70.14	—	<LOD	<LOD	F	1.8[E] (0.89–2.8)
3 (2012–2013)	355	66.48	—	<LOD	<LOD	0.97 (0.73–1.2)	1.3 (0.87–1.8)
4 (2014–2015)	362	63.54	—	<LOD	<LOD	1.1 (0.96–1.2)	F
40–59 years							
2 (2009–2011)	355	71.43	—	<LOD	<LOD	1.2[E] (0.55–1.8)	1.9 (1.4–2.4)
3 (2012–2013)	312	72.76	—	<LOD	<LOD	1.3 (0.92–1.6)	1.7 (1.3–2.0)
4 (2014–2015)	312	74.36	—	<LOD	<LOD	1.3 (<LOD–1.7)	1.5 (<LOD–1.9)
60–79 years							
2 (2009–2011)	288	81.31	—	<LOD	<LOD	1.7 (1.3–2.2)	1.9 (1.6–2.2)
3 (2012–2013)	352	76.70	—	<LOD	<LOD	1.4[E] (0.87–1.9)	2.1[E] (1.3–2.9)
4 (2014–2015)	361	82.27	—	<LOD	<LOD	1.0 (0.75–1.3)	1.3 (1.1–1.6)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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10.2 CADMIUM

Cadmium (CASRN 7440-43-9) is among the least abundant metals in the Earth's crust at an average concentration of approximately 0.00001% (Emsley, 2001). It is a naturally occurring soft, silvery white,

blue-tinged metal. Cadmium often occurs in zinc ores (Health Canada, 1986). Common forms include soluble and insoluble species that may also be found as particulate matter in the atmosphere (ATSDR, 2012; CCME, 1999).

Cadmium is released to the environment as a result of natural processes, including forest fires, volcanic emissions, and weathering of soil and bedrock (Morrow, 2000). The main anthropogenic sources of atmospheric cadmium are industrial base-metal smelting and refining processes, and combustion processes such as coal-fired electrical plants and waste incineration where cadmium is released as a by-product (CCME, 1999).

Cadmium is primarily used in the manufacture of nickel-cadmium batteries (USGS, 2012). It is also used in industrial coatings and electroplating, in pigments, and as a stabilizer in polyvinyl chloride plastics. Cadmium is present in metal alloy sheets, wires, rods, solders, and shields for various industrial applications (Environment Canada and Health Canada, 1994). It is also sometimes used in costume jewellery and as a pigment in ceramic glazes. Cadmium may also be present in fertilizers as the result of recycling of by-products and waste materials for land application. It is frequently an impurity in galvanized pipes and as a constituent of solders used in plumbing and distributions systems and can leach into drinking water (Health Canada, 1986; WHO, 2011).

In smokers, inhalation of cigarette smoke is a major source of cadmium exposure (Environment Canada and Health Canada, 1994; IARC, 2012). For non-smoking adults and children, the largest source of cadmium exposure is through the ingestion of food (Environment Canada and Health Canada, 1994; IARC, 2012). Ambient air is a minor source of exposure with intakes estimated to be two to three orders of magnitude lower than food, although cadmium compounds are more readily absorbed following inhalation than ingestion (Friberg, 1985). Other potential sources of exposure include ingestion of drinking water, soil, or dust (ATSDR, 2012; Environment Canada and Health Canada, 1994; Rasmussen et al., 2013).

Absorption of dietary cadmium into the bloodstream depends on one's nutritional status and the levels of other components of the diet such as iron, calcium, and protein. The average gastrointestinal absorption of dietary cadmium is estimated at 5% in adult men and 10% or higher in women (CDC, 2009). About 25% to 60% of inhaled cadmium is absorbed through the lungs (ATSDR, 2012). Absorbed cadmium accumulates mainly in the kidney and liver, with approximately one–third to one-half of the total body burden accumulating in the kidney (CDC, 2009). The biological half-life of cadmium in the kidney has been estimated to be approximately 10 to 12 years (Amzal et al., 2009; Lauwerys et al., 1994). Only a small proportion of absorbed cadmium is eliminated, mainly in the urine and feces with small amounts also eliminated through hair, nails, and sweat.

Cadmium can be measured in blood, urine, feces, liver, kidney, and hair among other tissues. Cadmium concentrations in urine best reflect cumulative exposure and the concentration of cadmium in the kidney, although slight fluctuations occur with recent exposures (Adams and Newcomb, 2014). Concentrations in blood reflect more recent exposures (Adams and Newcomb, 2014). Blood cadmium concentrations are about twice as high in smokers compared with non-smokers; concentrations can also be elevated following occupational exposures (ATSDR, 2012).

Oral exposure to high doses of cadmium may cause severe gastrointestinal irritation and kidney effects (ATSDR, 2012). Chronic exposure via inhalation has been associated with effects in the lungs, including emphysema, and in the kidneys (ATSDR, 2012). The kidney is considered the critical organ that exhibits the first adverse effects following both oral and inhalation exposure (EFSA, 2009; FAO/WHO, 2011).

Cadmium and its compounds have been classified as probably carcinogenic to humans by inhalation by Environment Canada and Health Canada (Environment Canada and Health Canada, 1994). More recently, the International Agency for Research on Cancer has classified cadmium and its compounds as carcinogenic to humans (Group 1) based on various data, including associations between occupational inhalation exposure and lung cancer (IARC, 2012). There is insufficient evidence to determine whether or not cadmium is carcinogenic following oral exposure (ATSDR, 2012).

Health Canada and Environment Canada concluded that inorganic cadmium compounds are a concern for human health based on its carcinogenic potential and effects on the kidneys (Environment Canada and Health Canada, 1994). Inorganic cadmium compounds

are listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The Act allows the federal government to control the importation, manufacture, distribution, and use of inorganic cadmium compounds in Canada (Canada, 1999; Canada, 2000). Risk management actions under CEPA 1999 have been developed to control releases of cadmium from thermal electric power generation, base-metal smelting, and steel manufacturing processes (Environment Canada, 2013).

In Canada, the leachable cadmium content in a variety of consumer products is regulated under the *Canada Consumer Product Safety Act* (Canada, 2010a). Consumer products regulated for leachable cadmium content include glazed ceramics and glassware, as well as paints and other surface coatings on cribs, toys, and other products for use by a child in learning or play situations (Canada, 1998; Canada, 2010b; Canada, 2011; Health Canada, 2009). In addition, because children's jewellery items containing high levels of cadmium have been found on the Canadian marketplace, a new guideline limit for total cadmium in children's jewellery was proposed in 2016 as part of the Children's Jewellery Regulations under the *Canadian Consumer Product Safety Act* (Canada, 2016). Cadmium and its compounds are included as prohibited ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist) (Health Canada, 2015). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations. On the basis of health considerations, Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for cadmium in drinking water (Health Canada, 1986; Health Canada, 2014). Cadmium is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply.

In a biomonitoring study carried out in the region of the city of Québec with 500 participants aged 18–65 years, the geometric means for cadmium in whole blood was 0.69 µg/L (INSPQ, 2004). The First Nations Biomonitoring Initiative (FNBI) is a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprises 13 randomly selected First Nation communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the geometric mean and 95th percentile for cadmium in blood were 0.96 µg/L and 4.65 µg/L, respectively. In Northern Canada, the contaminant component of the Inuit Health Survey (2007–2008) has measured the body burden of cadmium for 2,172 Inuit participants from 36 communities in Nunavut, Nunatsiavut, and the Inuvialuit Settlement Region (Laird et al., 2013). The geometric mean blood concentration of cadmium for all participants (18 years and older) was 1.6 µg/L. The Maternal-Infant Research on Environmental Chemicals (MIREC) Study is a national-level prospective biomonitoring study carried out in pregnant women aged 18 years and older from 10 sites across Canada, 2008–2011 (Arbuckle et al., 2013). In the MIREC Study of 1,938 participants in their first trimester of pregnancy, the geometric mean and 95th percentile for cadmium in blood were 0.2197 µg/L and 1.124 µg/L, respectively (Arbuckle et al., 2016).

Cadmium was analyzed in the whole blood of all Canadian Health Measures Survey participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Data from these cycles are presented in blood as µg/L. Finding a measurable amount of cadmium in blood is an indicator of exposure to cadmium and does not necessarily mean that an adverse health effect will occur.

Table 10.2.1

Cadmium — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	6070	5.16	0.29 (0.26–0.32)	0.083 (0.074–0.093)	0.26 (0.24–0.29)	1.7 (1.3–2.0)	2.6 (2.1–3.0)
3 (2012–2013)	5538	11.48	0.33 (0.30–0.36)	<LOD	0.27 (0.25–0.29)	2.0 (1.4–2.6)	3.4 (2.5–4.3)
4 (2014–2015)	5497	10.88	0.31 (0.29–0.32)	<LOD	0.25 (0.23–0.26)	1.9 (1.5–2.4)	3.3 (2.6–4.0)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	5.78	0.26 (0.24–0.29)	0.079 (0.070–0.089)	0.23 (0.20–0.26)	1.7 (1.5–2.0)	2.4 (2.0–2.9)
3 (2012–2013)	2769	12.35	0.29 (0.27–0.32)	<LOD	0.22 (0.19–0.25)	2.1 (1.5–2.7)	3.3 (2.5–4.2)
4 (2014–2015)	2753	12.42	0.28 (0.27–0.30)	<LOD	0.20 (0.19–0.21)	2.0 (1.4–2.6)	3.3 (2.5–4.2)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	4.57	0.32 (0.28–0.36)	0.089 (0.080–0.098)	0.30 (0.27–0.33)	1.5[E] (0.92–2.1)	2.7 (2.1–3.4)
3 (2012–2013)	2769	10.62	0.37 (0.33–0.41)	<LOD	0.32 (0.28–0.37)	1.7[E] (0.62–2.8)	3.4[E] (1.8–5.0)
4 (2014–2015)	2744	9.33	0.33 (0.31–0.35)	0.099 (0.095–0.10)	0.28 (0.25–0.30)	1.8[E] (1.1–2.5)	3.1 (2.3–4.0)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	15.15	0.073 (0.065–0.081)	<LOD	0.078 (0.069–0.087)	0.099 (0.098–0.10)	F
3 (2012–2013)	471	43.52	—	<LOD	0.091 (<LOD–0.11)	0.16 (0.11–0.20)	0.18[E] (<LOD–0.29)
4 (2014–2015)	479	36.95	0.082 (<LOD–0.091)	<LOD	0.093 (0.084–0.10)	0.16 (0.14–0.18)	0.19 (0.15–0.24)
6–11 years							
1 (2007–2009)	910	9.12	0.091 (0.082–0.10)	<LOD[E] (<LOD–0.053)	0.092 (0.090–0.094)	0.20 (0.18–0.21)	0.22 (0.19–0.26)
2 (2009–2011)	961	14.05	0.083 (0.076–0.090)	<LOD	0.090 (0.087–0.094)	0.17[E] (0.088–0.25)	0.20 (0.18–0.23)
3 (2012–2013)	944	27.44	0.095 (0.085–0.11)	<LOD	0.10 (0.099–0.10)	0.18 (0.16–0.20)	0.21 (0.18–0.24)
4 (2014–2015)	925	26.92	0.094 (0.086–0.10)	<LOD	0.10 (0.096–0.10)	0.16 (0.14–0.19)	0.19 (0.17–0.21)
12–19 years							
1 (2007–2009)	945	3.92	0.16 (0.13–0.20)	0.066 (0.045–0.086)	F	F	F
2 (2009–2011)	997	5.72	0.13 (0.12–0.15)	0.062 (0.040–0.084)	0.096 (0.095–0.097)	0.48[E] (0.27–0.70)	0.82[E] (0.45–1.2)
3 (2012–2013)	977	12.49	0.17 (0.15–0.20)	<LOD	0.12[E] (<LOD–0.17)	0.82[E] (0.31–1.3)	1.7[E] (0.91–2.4)
4 (2014–2015)	974	12.22	0.14 (0.13–0.15)	<LOD	0.12 (0.12–0.13)	0.29 (0.25–0.33)	0.54[E] (0.15–0.94)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	1165	1.55	0.34 (0.30–0.38)	0.091 (0.084–0.098)	0.24 (0.21–0.27)	2.6 (2.0–3.1)	3.4 (3.1–3.7)
2 (2009–2011)	1313	2.21	0.28 (0.24–0.34)	0.090 (0.066–0.11)	0.24 (0.20–0.29)	1.7[E] (1.0–2.3)	2.7 (2.1–3.2)
3 (2012–2013)	1032	3.68	0.31 (0.24–0.41)	0.10 (0.084–0.12)	0.25 (0.20–0.29)	2.0[E] (0.71–3.3)	F
4 (2014–2015)	1074	2.33	0.33 (0.28–0.38)	0.10 (0.090–0.11)	0.22 (0.17–0.26)	2.9 (1.9–3.9)	4.2[E] (2.5–5.9)
40–59 years							
1 (2007–2009)	1220	0.90	0.48 (0.43–0.54)	0.098[E] (0.054–0.14)	0.36 (0.32–0.41)	3.1 (2.3–3.9)	4.2 (3.7–4.7)
2 (2009–2011)	1222	0.98	0.41 (0.37–0.46)	0.095 (0.090–0.10)	0.34 (0.31–0.37)	2.2 (1.5–2.8)	3.1 (2.3–3.8)
3 (2012–2013)	1071	1.12	0.50 (0.43–0.57)	0.11 (0.084–0.13)	0.39 (0.30–0.48)	3.0 (2.3–3.7)	4.6 (3.7–5.5)
4 (2014–2015)	1050	1.81	0.41 (0.37–0.45)	0.12 (0.097–0.15)	0.33 (0.26–0.39)	2.1[E] (1.2–3.0)	3.4 (2.3–4.4)
60–79 years							
1 (2007–2009)	1079	0.56	0.45 (0.42–0.49)	0.19 (0.18–0.20)	0.39 (0.37–0.41)	1.7 (1.2–2.2)	2.7 (2.2–3.2)
2 (2009–2011)	1082	0.46	0.45 (0.41–0.50)	0.18 (0.13–0.23)	0.40 (0.35–0.44)	1.6 (1.3–2.0)	2.4 (1.9–2.8)
3 (2012–2013)	1043	0	0.48 (0.43–0.54)	0.19 (0.17–0.20)	0.41 (0.35–0.46)	1.5 (1.3–1.8)	2.6 (1.9–3.3)
4 (2014–2015)	995	0.90	0.44 (0.41–0.48)	0.17 (0.16–0.18)	0.37 (0.34–0.40)	1.6 (1.1–2.2)	2.8 (2.0–3.6)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.
b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).
E Use data with caution.
F Data is too unreliable to be published.

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10.3 FLUORIDE

Fluorine (CASRN 16984-48-8) is the 13th most abundant element, occurring naturally in the Earth's crust at an average concentration of about 0.09% (ATSDR, 2003). It is widely distributed and naturally occurring, but it is rarely found in nature because it reacts readily with most organic and inorganic substances. Fluorides are formed when fluorine reacts with metals. Four inorganic fluorides of environmental importance are calcium fluoride (fluorspar and fluorite), sodium fluoride, sulphur hexafluoride, and hydrogen fluoride (Cotton and Wilkinson, 1988; Mackay and Mackay, 1989).

Fluorides are found in rocks, coal, clay, and soil. Gases and particles produced from volcanic eruptions and minerals leached from bedrock release inorganic fluorides into the environment (ATSDR, 2003; CCME, 2002). In addition to these natural sources, inorganic fluorides are released through human activities such as phosphate fertilizer production, chemical production, and aluminum smelting (Environment Canada and Health Canada, 1993).

Hydrogen fluoride is one of the most commonly used fluoride compounds; it is a component in the production of refrigerants, herbicides, pharmaceuticals, aluminum, plastics, high-octane gasoline, electrical components, and fluorescent light bulbs (ATSDR, 2003). In water, hydrogen fluoride becomes hydrofluoric acid, which is used in the metal and glass manufacturing industries (ATSDR, 2003). Calcium fluoride is used in the production of steel, aluminum, glass, and enamel, and as the raw material for the production of hydrofluoric acid and hydrogen fluoride (CCME, 2002). Fluoride-containing compounds are often added to drinking water and dental products to prevent dental cavities. Toothpastes are the most commonly used dental product that contain fluoride (Health Canada, 2010a). Other fluoride-containing dental products available to consumers include fluoride supplements, fluoride mouth rinses, and dental floss. Fluoride is also used by professionals in some dental filling material, sealants, and fluoride varnishes. Sodium fluoride is also used as a preservative in wood and glues and in the production of glass, enamel, steel, and aluminum (CCME, 2002). Sulphur hexafluoride is used extensively in electrical switch gear such as power circuit breakers, compressed gas transmission lines, and various components in electrical substations (CCME, 2002).

Fluoride compounds are ubiquitous in the environment; however, the major sources of exposure to the general population are water, food, beverages, and dental products (Health Canada, 2010a). Following ingestion of soluble fluoride salts and inhalation of gaseous hydrogen fluoride, fluoride is rapidly and efficiently absorbed (ATSDR, 2003). Once absorbed, fluoride is rapidly distributed throughout the body via the bloodstream (ATSDR, 2003). In infants, about 80% to 90% of the total absorbed fluoride is retained in bones and teeth with the level dropping to about 60% in adults (Fawell et al., 2006). The remaining fluoride in adults and infants is excreted through urine (ATSDR, 2003). The biological half-life of fluoride is on the order of several hours (ATSDR, 2003; NRC, 2006). Urine

and blood analyses are the most common tests for fluoride exposure (ATSDR, 2003).

The primary adverse effects associated with chronic excess fluoride intake are dental and skeletal fluorosis (IOM, 1997). Exposure to excessive levels of fluoride over a very long period of time can lead to skeletal fluorosis characterized by dense bones, joint pain, and limited range of joint movement (ATSDR, 2003). Dense bones are often more brittle or fragile than normal bones and there is an increased risk of bone fractures in older adults. Dental fluorosis is a dose-response effect caused by fluoride ingestion during tooth formation that becomes apparent upon eruption of the teeth. The effects of dental fluorosis can range from mild discolouration of the tooth surface to severe staining, enamel loss, and pitting (NRC, 2006).

Health Canada found that the weight of evidence from existing scientific data does not support an association between fluoride and increased risks of cancer, and has classified fluoride in Group VI, unclassifiable with respect to carcinogenicity in humans (Health Canada, 2010a). Similarly, the International Agency for Research on Cancer has classified fluorides (inorganic, used in drinking water) as Group 3, not classifiable as to its carcinogenicity to humans (IARC, 1987).

Health Canada and Environment Canada have reviewed and assessed inorganic fluorides under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999). The screening assessment concluded that levels of inorganic fluorides normally found in the Canadian environment are not considered harmful to human health but are a concern for the environment (Environment Canada and Health Canada, 1993). Inorganic fluorides are listed on Schedule 1, List of Toxic Substances, under CEPA 1999. The Act allows the federal government to control the importation, manufacture, distribution, and use of inorganic fluorides in Canada (Canada, 1999; Canada, 2000).

Health Canada recommends that fluoride requirements be based on the beneficial effect on dental caries (Health Canada, 2010a). Young children tend to swallow toothpaste during brushing, so guidelines have been established that strive to balance the health risks with the health benefits of fluoride use. In general, toothpaste use is not recommended for children under the age of 3, unless recommended by a health professional. For children 3 to 6 years old, Health Canada recommends supervision during brushing and use of only a small amount of fluoridated toothpaste (Health Canada, 2010b). Fluoride-containing substances are included as restricted ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist) and are not permitted in oral products (Health Canada, 2015). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations.

Health Canada completed a review of the health risks associated with fluoride in drinking water in which moderate dental fluorosis was chosen as the endpoint of concern for fluoride (Health Canada, 2010a). Although moderate dental fluorosis is not a health concern and is not considered to be a toxicological endpoint, Health Canada considers it to be an adverse effect based on its potential aesthetic concern. The current guideline for Canadian drinking water quality developed by Health Canada in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water established the maximum acceptable concentration for fluoride in drinking water (Health Canada, 2010a). This guideline is considered to be protective against all potential adverse health effects, including those related to cancer, immunotoxicity, reproductive/developmental toxicity, genotoxicity, and/or neurotoxicity (Health Canada, 2010a). For communities wishing to fluoridate their water supply, Health Canada has determined an optimal concentration of fluoride in drinking water to promote dental health while protecting against adverse effects (Health Canada, 2010b). Tolerable upper intake levels for fluoride that account for its potential toxicity have been developed by the Institute of Medicine and adopted by Health Canada (Health Canada, 2010c; IOM, 1997).

The concentration of fluoride in some foods and prepackaged water and ice is regulated by Health Canada under the Food and Drug Regulations (Canada, 2012). Food tolerances, or maximum levels, for fluoride currently exist for edible bone meal and fish protein as well as prepackaged ice or water, including those represented as mineral or spring water (Canada, 2012).

The first cycle (2007–2009) of the Canadian Health Measures Survey (CHMS) included a National Oral Health Component supported by Health Canada (Health Canada, 2010d). In addition to many other dental considerations, dental fluorosis was measured in children ranging from 6 to 12 years old. The results from cycle 1 of the CHMS found that 60% of children had teeth considered normal, 24% had enamel with white flecks or spots where the cause was questionable, 12% had one or more teeth with fluorosis classified as very mild, and 4% had fluorosis classified as mild. The prevalence of moderate or severe fluorosis was too low to allow reporting (less than 0.3%).

Fluoride was analyzed in the urine of CHMS cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and is presented as both mg/L and mg/g creatinine. Finding a measurable amount of fluoride in urine is an indicator of exposure to fluoride and does not necessarily mean that an adverse health effect will occur.

Table 10.3.1

Fluoride — Geometric means and selected percentiles of urine concentrations (mg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2530	0	0.50 (0.46–0.55)	0.19 (0.17–0.22)	0.48 (0.44–0.53)	1.2 (1.0–1.3)	1.5 (1.2–1.7)
3 (2012–2013)	2671	0	0.43 (0.39–0.48)	0.15 (0.14–0.17)	0.44 (0.39–0.49)	1.1 (0.97–1.3)	1.4 (0.99–1.7)
4 (2014–2015)	2574	0	0.47 (0.38–0.59)	0.18 (0.15–0.22)	0.45 (0.33–0.58)	1.2 (0.91–1.4)	1.4 (1.2–1.6)
Males, 3–79 years							
2 (2009–2011)	1267	0	0.53 (0.47–0.60)	0.23 (0.20–0.25)	0.51 (0.42–0.60)	1.3 (1.0–1.5)	1.6 (1.4–1.9)
3 (2012–2013)	1320	0	0.44 (0.39–0.49)	0.16 (0.13–0.19)	0.44 (0.39–0.49)	1.1 (0.92–1.3)	1.3[E] (0.81–1.8)
4 (2014–2015)	1246	0	0.47 (0.36–0.61)	0.18 (0.13–0.23)	0.45 (0.29–0.60)	1.1 (0.79–1.4)	1.5 (1.1–1.9)
Females, 3–79 years							
2 (2009–2011)	1263	0	0.47 (0.43–0.52)	0.17 (0.15–0.20)	0.47 (0.41–0.53)	1.1 (0.92–1.3)	1.3 (1.0–1.6)
3 (2012–2013)	1351	0	0.43 (0.38–0.48)	0.14 (0.11–0.16)	0.45 (0.37–0.53)	1.1 (0.92–1.4)	1.4 (1.0–1.8)
4 (2014–2015)	1328	0	0.48 (0.39–0.58)	0.18 (0.16–0.21)	0.46 (0.35–0.57)	1.2 (0.94–1.5)	1.4 (1.2–1.6)
3–5 years							
2 (2009–2011)	510	0	0.47 (0.42–0.52)	0.18 (0.13–0.23)	0.51 (0.44–0.58)	0.99 (0.88–1.1)	1.3 (0.92–1.7)
3 (2012–2013)	493	0	0.39 (0.32–0.48)	0.13 (0.098–0.17)	0.37 (0.25–0.50)	0.99 (0.77–1.2)	1.2 (0.97–1.5)
4 (2014–2015)	483	0	0.42 (0.33–0.54)	0.13[E] (0.071–0.19)	0.39 (0.25–0.53)	1.1[E] (0.68–1.6)	1.6[E] (0.66–2.5)
6–11 years							
2 (2009–2011)	514	0	0.50 (0.44–0.57)	0.20 (0.17–0.24)	0.49 (0.42–0.55)	1.1 (0.90–1.3)	1.5 (1.1–1.8)
3 (2012–2013)	549	0	0.40 (0.36–0.45)	0.18 (0.15–0.20)	0.38 (0.35–0.41)	0.85 (0.61–1.1)	1.1 (0.88–1.4)
4 (2014–2015)	533	0	0.47 (0.37–0.60)	0.19 (0.14–0.25)	0.46 (0.34–0.58)	1.1[E] (0.55–1.7)	1.6 (1.1–2.0)
12–19 years							
2 (2009–2011)	507	0	0.41 (0.37–0.46)	0.17 (0.15–0.19)	0.44 (0.36–0.52)	0.94 (0.82–1.1)	1.2 (0.98–1.3)
3 (2012–2013)	549	0	0.39 (0.35–0.44)	0.16 (0.13–0.20)	0.37 (0.33–0.41)	0.93 (0.71–1.2)	1.1 (0.85–1.3)
4 (2014–2015)	481	0	0.42 (0.35–0.50)	0.17 (0.14–0.20)	0.44 (0.36–0.53)	0.99 (0.75–1.2)	1.1 (0.91–1.2)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	354	0	0.53 (0.47–0.59)	0.23 (0.19–0.27)	0.50 (0.38–0.62)	1.2 (0.96–1.5)	1.4 (1.1–1.8)
3 (2012–2013)	371	0	0.43 (0.35–0.53)	0.15 (0.11–0.19)	0.47 (0.37–0.57)	1.1 (0.77–1.4)	1.3[E] (0.58–2.1)
4 (2014–2015)	369	0	0.46 (0.35–0.60)	0.18[E] (0.099–0.27)	0.42 (0.29–0.55)	1.2 (0.81–1.6)	1.4 (1.1–1.7)
40–59 years							
2 (2009–2011)	357	0	0.51 (0.44–0.61)	0.19 (0.13–0.25)	0.51 (0.40–0.61)	1.2 (0.93–1.6)	1.7 (1.3–2.2)
3 (2012–2013)	359	0	0.46 (0.42–0.50)	0.16 (0.13–0.20)	0.46 (0.41–0.50)	1.2 (1.0–1.3)	1.4 (0.88–1.9)
4 (2014–2015)	368	0	0.49 (0.36–0.66)	0.18 (0.15–0.21)	0.48[E] (0.26–0.70)	1.2 (0.90–1.5)	1.4 (1.1–1.7)
60–79 years							
2 (2009–2011)	288	0	0.50 (0.44–0.56)	0.19[E] (0.11–0.27)	0.48 (0.42–0.54)	1.2 (0.99–1.5)	1.6 (1.3–2.0)
3 (2012–2013)	350	0	0.43 (0.36–0.51)	0.13 (0.086–0.18)	0.45 (0.34–0.56)	1.3 (0.85–1.7)	1.7 (1.3–2.0)
4 (2014–2015)	340	0	0.51 (0.43–0.61)	0.19 (0.15–0.22)	0.49 (0.33–0.64)	1.2 (0.95–1.5)	1.8 (1.2–2.4)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 10.3.2

Fluoride (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (mg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2520	0	0.50 (0.45–0.55)	0.20 (0.18–0.22)	0.48 (0.41–0.54)	1.2 (0.99–1.4)	1.6 (1.3–2.0)
3 (2012–2013)	2669	0	0.46 (0.41–0.51)	0.18 (0.16–0.21)	0.45 (0.37–0.53)	1.0 (0.88–1.1)	1.4 (1.1–1.7)
4 (2014–2015)	2574	0	0.41 (0.34–0.48)	0.17 (0.14–0.19)	0.40 (0.32–0.48)	0.97 (0.84–1.1)	1.2 (1.0–1.5)
Males, 3–79 years							
2 (2009–2011)	1263	0	0.46 (0.40–0.52)	0.20 (0.16–0.24)	0.43 (0.36–0.50)	1.0 (0.85–1.2)	1.2 (0.86–1.6)
3 (2012–2013)	1320	0	0.40 (0.35–0.45)	0.16 (0.12–0.20)	0.41 (0.33–0.48)	0.87 (0.75–0.98)	1.1 (0.90–1.2)
4 (2014–2015)	1246	0	0.35 (0.30–0.42)	0.15 (0.13–0.17)	0.36 (0.27–0.45)	0.75 (0.62–0.89)	0.99 (0.84–1.1)
Females, 3–79 years							
2 (2009–2011)	1257	0	0.54 (0.49–0.60)	0.21 (0.18–0.24)	0.52 (0.45–0.60)	1.4 (1.2–1.6)	1.9 (1.5–2.3)
3 (2012–2013)	1349	0	0.53 (0.47–0.59)	0.22 (0.19–0.25)	0.53 (0.43–0.63)	1.2 (0.93–1.4)	1.6 (1.3–2.0)
4 (2014–2015)	1328	0	0.47 (0.39–0.56)	0.19 (0.15–0.23)	0.44 (0.33–0.54)	1.1 (0.90–1.2)	1.6 (1.1–2.1)
3–5 years							
2 (2009–2011)	509	0	0.81 (0.73–0.90)	0.40 (0.34–0.46)	0.78 (0.72–0.85)	1.7 (1.3–2.0)	2.8[E] (1.7–4.0)
3 (2012–2013)	492	0	0.76 (0.68–0.86)	0.37 (0.30–0.44)	0.73 (0.55–0.90)	1.5 (1.3–1.6)	1.7 (1.4–1.9)
4 (2014–2015)	483	0	0.75 (0.63–0.90)	0.34 (0.27–0.42)	0.68 (0.52–0.85)	1.8 (1.4–2.2)	3.1[E] (1.3–4.9)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
2 (2009–2011)	512	0	0.58 (0.53–0.63)	0.30 (0.28–0.32)	0.57 (0.50–0.63)	1.2 (0.96–1.4)	1.5 (1.0–2.0)
3 (2012–2013)	549	0	0.50 (0.43–0.57)	0.24 (0.19–0.28)	0.45 (0.39–0.50)	1.0 (0.91–1.1)	1.2 (0.86–1.5)
4 (2014–2015)	533	0	0.50 (0.42–0.59)	0.26 (0.22–0.31)	0.46 (0.36–0.56)	0.96 (0.67–1.2)	1.2 (1.0–1.5)
12–19 years							
2 (2009–2011)	505	0	0.32 (0.28–0.35)	0.15 (0.13–0.16)	0.32 (0.28–0.37)	0.62 (0.52–0.72)	0.75 (0.54–0.97)
3 (2012–2013)	548	0	0.29 (0.25–0.33)	0.14 (0.12–0.16)	0.27 (0.23–0.31)	0.61 (0.45–0.76)	0.76 (0.63–0.89)
4 (2014–2015)	481	0	0.29 (0.25–0.34)	0.15 (0.14–0.16)	0.29 (0.22–0.35)	0.57 (0.49–0.64)	0.63 (0.49–0.76)
20–39 years							
2 (2009–2011)	352	0	0.46 (0.39–0.55)	0.20 (0.17–0.24)	0.42 (0.33–0.51)	1.1 (0.74–1.4)	F
3 (2012–2013)	371	0	0.40 (0.35–0.46)	0.16 (0.12–0.19)	0.39 (0.30–0.49)	0.87 (0.71–1.0)	1.0 (0.76–1.2)
4 (2014–2015)	369	0	0.35 (0.28–0.44)	0.14[E] (0.078–0.20)	0.36 (0.26–0.45)	0.85 (0.68–1.0)	1.0 (0.95–1.1)
40–59 years							
2 (2009–2011)	355	0	0.53 (0.46–0.60)	0.22 (0.18–0.26)	0.54 (0.43–0.66)	1.2 (0.94–1.4)	1.6 (1.2–2.0)
3 (2012–2013)	359	0	0.52 (0.46–0.59)	0.22 (0.20–0.24)	0.55 (0.44–0.66)	1.0 (0.87–1.2)	1.2 (0.77–1.7)
4 (2014–2015)	368	0	0.41 (0.34–0.50)	0.17 (0.13–0.21)	0.42 (0.35–0.49)	0.92 (0.67–1.2)	1.2 (0.86–1.6)
60–79 years							
2 (2009–2011)	287	0	0.58 (0.50–0.68)	0.22 (0.16–0.27)	0.55 (0.44–0.67)	1.6 (1.3–1.9)	1.9 (1.4–2.5)
3 (2012–2013)	350	0	0.52 (0.43–0.62)	0.20 (0.16–0.24)	0.52 (0.45–0.59)	1.4[E] (0.86–1.9)	2.1[E] (1.1–3.0)
4 (2014–2015)	340	0	0.49 (0.40–0.59)	0.17 (0.13–0.20)	0.50 (0.37–0.63)	1.5 (0.93–2.0)	2.1[E] (1.3–2.9)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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10.4 LEAD

Lead (CASRN 7439-92-1) is a naturally occurring element present at a median natural background concentration of 0.0008% in Canada (Rencz et al., 2006). It is a base metal and can exist in various oxidation states and in both inorganic and organic forms (ATSDR, 2007). Inorganic forms include substances such as elemental lead, lead sulphate, lead carbonate and hydroxyl carbonate, lead oxides, lead chromate, and lead citrate (Rasmussen et al., 2014). Organic lead compounds include tetra-alkyl, trialkyl, and dialkyl lead compounds.

Lead is found in bedrock, soils, sediments, surface water, groundwater, and sea water (Health Canada, 2013a). It enters the environment from a variety of natural and anthropogenic sources. Natural processes include soil weathering, erosion, and volcanic activity (ATSDR, 2007; IARC, 2006). Lead released from industrial emissions can be a major source of environmental contamination, especially near point sources such as smelters or refineries (ATSDR, 2007). Historical use of leaded motor fuels has contributed to the ubiquitous distribution of lead throughout the globe (WHO, 2000).

In North America, tetraethyl and tetramethyl lead were added to motor vehicle fuels as an anti-knock agent up until the 1990s. Presently in Canada, the addition of lead to gasoline is prohibited, with the exception of fuels for piston engine aircraft and racing fuels for competition vehicles (Health Canada, 2013a). Lead is currently used in the refining and manufacturing of products such as lead acid automotive batteries, lead shot and fishing weights, sheet lead, lead solder, some brass and bronze products, and some ceramic glazes (ATSDR, 2007; WHO, 2000). Other uses of lead include dyes in paints and pigments. It is also used in scientific equipment, as a stabilizer in plastics, in military equipment and ammunition, and in radiation detection and medical equipment for radiation shielding (ATSDR, 2007; WHO, 2000). Lead is also used in the manufacturing of cable sheathing, circuit boards, chemical baths and storage vessel linings, chemical transmission pipes, electrical components, and polyvinyl chloride (Health Canada, 2013a).

Everyone is exposed to trace amounts of lead through food, drinking water, soil, household dust, air, and some consumer products. Over the past 30 years,

lead exposure has declined by approximately 75% in Canadians (Statistics Canada, 2013). The substantial decrease in exposure to lead is attributed mainly to the phase-out of leaded gasoline, reduction of lead content in paint and surface coatings, and the elimination of lead solder in food cans (Health Canada, 2013b). Today, the main route of exposure for the general adult population is from ingestion of food and drinking water (ATSDR, 2007; Health Canada, 2013a). For infants and children, the primary sources of exposure are food, drinking water, and the ingestion of non-food items containing lead such as house dust, paint, soil, and consumer products (Health Canada, 2013a). Lead can enter the water supply from lead service lines in older homes, brass plumbing fittings that contain lead, or lead solder in the plumbing in homes (Health Canada, 2016). Other potential sources of exposure include costume jewellery, art supplies, leaded crystal, and glazes on ceramics and pottery; having a hobby, or living with someone who has, that uses lead or lead solder, such as making stained glass, ceramic glazing, lead shot or lead fishing weights, and furniture refinishing; living near airports with piston aircraft activity; and behaviours such as smoking (Health Canada, 2013b). The Canadian House Dust Study reported that lead is enriched in house dust compared with the natural geochemical background, as a result of the use of lead in consumer products, paints and building materials, and infiltration from outdoor sources (Rasmussen et al., 2011; Rasmussen et al., 2013).

Approximately 3% to 10% of ingested lead is absorbed into blood in adults; the amount absorbed can increase to up to 40% to 50% in children (Health Canada, 2013a). Nutritional calcium and iron deficiencies in children appear to increase lead absorption and decrease lead excretion (Health Canada, 2013a). Once absorbed by the human body, lead circulates in the bloodstream where it accumulates in tissues, particularly bone, and is excreted from the body. Some lead may also be absorbed into soft tissues such as the liver, kidneys, pancreas, and lungs. Bones account for approximately 70% of the total body burden of lead in children and more than 90% of the total body burden in adults (EPA, 2006). Lead stored in bone can be remobilized and released back into circulating blood. Pregnancy, lactation, menopause, andropause, post-menopause, extended bed rest, hyperparathyroidism, and osteoporosis are all conditions that can increase remobilization of lead from bone, increasing blood lead levels (Health Canada, 2013a).

During pregnancy, lead stored in maternal bone becomes a source of exposure to both fetus and mother (Rothenberg et al., 2000). Lead can also be present in breast milk and is transferred from lactating mothers to infants (ATSDR, 2007; EPA, 2006). The half-life for lead in blood is approximately 30 days whereas the half-life for lead accumulated in the body, such as in bone, is around 10 to 30 years (ATSDR, 2007; Health Canada, 2009a; Health Canada, 2013a). Excretion of absorbed lead, independent of the route of exposure, occurs primarily in urine and feces (ATSDR, 2007). Blood lead is the preferred indicator of human exposure to lead, although other matrices such as urine, bone, and teeth also have been used (ATSDR, 2007; CDC, 2009).

Lead is considered a cumulative general poison, with developing fetuses, infants, toddlers, and children being most susceptible to adverse health effects (WHO, 2011). Following acute exposure, a variety of metabolic processes may be affected. Very high exposure may result in vomiting, diarrhea, convulsions, coma, and death. Cases of lead poisoning are rare in Canada (Health Canada, 2009a). Symptoms of chronic exposure to relatively low levels of lead are often not apparent (ATSDR, 2007). Chronic low-level exposure may affect both the central and peripheral nervous systems (Health Canada, 2013a). Chronic low-level exposure to lead has also been associated with developmental neurotoxicity, increases in blood pressure, decreases in renal functioning, and reproductive problems as well as other health endpoints (ATSDR, 2007; Bushnik et al., 2014; Health Canada, 2013a). Cognitive and neurobehavioural effects have been recognized as major concerns for children exposed to lead. In infants and children, neurodevelopmental effects are most strongly associated with lead exposure, specifically the reduction of intelligence quotient (Lanphear et al., 2005) and an increased risk of attention-related behaviours (Health Canada, 2013a). Based on available data, no threshold has yet been identified for the effects of lead exposure on cognitive function and neurobehavioural development (CDC, 2012; EPA, 2006; Health Canada, 2013a). Developmental neurotoxicity has been associated with the lowest levels of lead exposure measured to date (Health Canada, 2013a). The International Agency for Research on Cancer classifies inorganic lead compounds as Group 2A, probably carcinogenic to humans (IARC, 2006).

Lead is listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The Act allows the federal government to control the importation, manufacture, distribution, and use of lead and lead compounds in Canada (Canada, 1999; Health Canada, 2009a). CEPA 1999 prohibits the addition of lead in gasoline and controls its release from secondary lead smelters, steel manufacturing, and mining effluents (Environment Canada, 2010). The use of lead in toys, children's jewellery and other products intended for children, glazed ceramics and glass foodware, and other consumer products representing a potential risk of lead exposure is limited under the *Canada Consumer Product Safety Act* and its associated regulations (Canada, 2010a; Canada, 2010b; Health Canada, 2013a). These regulations include the Children's Jewellery Regulations which propose a new guideline limit for lead in children's jewellery (Canada, 2016a). In addition, the Consumer Products Containing Lead Regulations have been recently proposed with a total lead limit for an expanded scope of products (Canada, 2016b). Lead and its compounds are also included as prohibited ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist) (Health Canada, 2015). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations.

On the basis of health considerations, Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for lead (Health Canada, 1992). This guideline is currently under review by Health Canada in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water (Health Canada, 2017). Health Canada has also published guidance on controlling corrosion in drinking water distribution systems to help control the leaching of metals, including lead, from the corrosion of distribution system materials and components (Health Canada, 2009b). The concentration of lead in some foods is managed by Health Canada under the Food and Drug Regulations; the existing maximum levels are in the process of being updated, with focus first being placed on a variety of beverages, including bottled water and fruit juices (Canada, 2012; Health Canada, 2011; Health Canada, 2014). These regulatory updates are some of several Health Canada activities that are under way to ensure that dietary exposure to lead is as low as reasonably achievable (Health Canada, 2011). Lead is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013c). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply. From 1981 to 2000, the average dietary exposure to lead in Canadians decreased by approximately eight-fold (Health Canada, 2011).

In 1994, the Federal-Provincial-Territorial Committee on Environmental and Occupational Health recommended a blood lead intervention level of 10 µg/dL as guidance for low-level exposure to lead (CEOH, 1994). Recent scientific assessments indicate that chronic health effects are occurring in children at blood lead levels below 10 µg/dL and that there is sufficient evidence that blood lead levels below 5 µg/dL are associated with adverse health effects (Health Canada, 2013a). The current guidance for lead in blood (CEOH, 1994) is under review by the federal, provincial, and territorial Council of Chief Medical Officers of Health.

A number of biomonitoring studies measuring blood lead levels have been conducted in various locations in Canada over the years. The reported geometric mean blood lead levels ranged from 0.7 µg/dL to 5.6 µg/dL for various age groups within the Canadian population (Health Canada, 2013a). The highest concentrations were reported for communities with point sources of environmental lead such as smelting (Trail Health and Environment Committee, 2011). In Northern Canada, the contaminant component of the Inuit Health Survey (2007–2008) has measured the body burden of lead for 2,172 Inuit participants from 36 communities in Nunavut, Nunatsiavut, and the Inuvialuit Settlement Region (Laird et al., 2013). The geometric mean blood lead level for all participants (18 years and older) was 3.52 µg/dL. In 2008, a study conducted in Hamilton on 643 children aged 0–6 years reported a geometric mean blood lead level of 2.21 µg/dL (Richardson et al., 2011). The First Nations Biomonitoring Initiative (FNBI) is a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprises 13 randomly selected

First Nation communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the geometric mean and 95th percentile for lead in blood were 1.17 µg/dL and 3.27 µg/dL, respectively.

Lead was analyzed in the whole blood of all Canadian Health Measures Survey participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Data from these cycles are presented in blood as µg/dL.

Table 10.4.1

Lead — Geometric means and selected percentiles of whole blood concentrations (µg/dL) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	6070	0	1.2 (1.1–1.2)	0.54 (0.50–0.59)	1.1 (1.1–1.2)	2.5 (2.3–2.7)	3.2 (2.9–3.4)
3 (2012–2013)	5538	0.09	1.1 (1.0–1.1)	0.49 (0.46–0.52)	1.0 (0.95–1.1)	2.4 (2.3–2.5)	3.2 (2.9–3.4)
4 (2014–2015)	5498	0.13	0.95 (0.90–1.0)	0.43 (0.40–0.46)	0.92 (0.88–0.95)	2.1 (1.8–2.3)	2.7 (2.4–3.0)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	0	1.3 (1.3–1.4)	0.62 (0.56–0.67)	1.2 (1.2–1.3)	2.8 (2.5–3.1)	3.4 (3.1–3.7)
3 (2012–2013)	2769	0.07	1.2 (1.2–1.3)	0.56 (0.55–0.58)	1.1 (1.0–1.2)	2.6 (2.4–2.9)	3.6 (3.1–4.0)
4 (2014–2015)	2754	0.07	1.0 (0.98–1.1)	0.47 (0.45–0.49)	1.0 (0.97–1.0)	2.2 (1.9–2.4)	2.9 (2.3–3.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	0	1.1 (1.0–1.1)	0.50 (0.46–0.54)	1.0 (0.96–1.1)	2.3 (2.1–2.5)	2.8 (2.6–3.0)
3 (2012–2013)	2769	0.11	0.96 (0.90–1.0)	0.42 (0.37–0.47)	0.93 (0.87–1.0)	2.2 (2.1–2.3)	2.6 (2.2–3.1)
4 (2014–2015)	2744	0.18	0.87 (0.81–0.94)	0.40 (0.36–0.43)	0.83 (0.78–0.89)	2.0 (1.6–2.3)	2.6 (2.3–2.8)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	0	0.93 (0.87–1.0)	0.51 (0.44–0.58)	0.93 (0.86–1.0)	1.6 (1.5–1.8)	2.1 (1.8–2.4)
3 (2012–2013)	471	0	0.77 (0.73–0.82)	0.40 (0.33–0.47)	0.72 (0.68–0.77)	1.4 (1.0–1.8)	2.2 (1.4–2.9)
4 (2014–2015)	479	0	0.67 (0.61–0.73)	0.37 (0.32–0.42)	0.64 (0.60–0.69)	1.2 (0.90–1.5)	1.7 (1.4–2.0)
6–11 years							
1 (2007–2009)	910	0	0.90 (0.81–0.99)	0.53 (0.49–0.56)	0.87 (0.77–0.97)	1.6 (1.4–1.7)	1.9 (1.6–2.2)
2 (2009–2011)	961	0	0.79 (0.74–0.84)	0.44 (0.38–0.50)	0.74 (0.68–0.81)	1.4 (1.2–1.6)	1.7 (1.5–1.9)
3 (2012–2013)	944	0	0.71 (0.67–0.76)	0.39 (0.36–0.42)	0.67 (0.64–0.71)	1.3 (1.1–1.5)	1.6 (1.3–1.9)
4 (2014–2015)	925	0.22	0.59 (0.55–0.62)	0.33 (0.31–0.35)	0.56 (0.52–0.59)	1.0 (0.89–1.1)	1.3 (1.0–1.5)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	945	0	0.80 (0.74–0.85)	0.47 (0.44–0.50)	0.76 (0.70–0.82)	1.3 (1.1–1.5)	1.6 (1.4–1.8)
2 (2009–2011)	997	0	0.71 (0.68–0.75)	0.39 (0.35–0.43)	0.68 (0.63–0.72)	1.2 (1.1–1.2)	1.6 (1.3–1.8)
3 (2012–2013)	977	0.10	0.64 (0.60–0.69)	0.34 (0.32–0.36)	0.60 (0.56–0.64)	1.2 (1.1–1.4)	1.5 (1.3–1.6)
4 (2014–2015)	974	0.31	0.54 (0.50–0.57)	0.30 (0.28–0.33)	0.51 (0.47–0.54)	0.98 (0.91–1.0)	1.1 (0.94–1.2)
20–39 years							
1 (2007–2009)	1165	0.09	1.1 (1.0–1.2)	0.57 (0.52–0.61)	1.0 (0.95–1.1)	2.3 (2.0–2.6)	3.1 (2.7–3.4)
2 (2009–2011)	1313	0	0.98 (0.88–1.1)	0.50 (0.43–0.57)	0.94 (0.87–1.0)	1.8 (1.5–2.1)	2.2 (1.6–2.9)
3 (2012–2013)	1032	0.19	0.90 (0.79–1.0)	0.44 (0.36–0.53)	0.88 (0.79–0.97)	1.7 (1.5–2.0)	2.1 (1.8–2.4)
4 (2014–2015)	1074	0.19	0.80 (0.74–0.88)	0.43 (0.39–0.47)	0.78 (0.67–0.88)	1.5 (1.2–1.7)	2.0 (1.6–2.5)
40–59 years							
1 (2007–2009)	1220	0	1.6 (1.5–1.8)	0.82 (0.69–0.94)	1.5 (1.4–1.6)	3.1 (2.6–3.6)	3.8 (3.1–4.5)
2 (2009–2011)	1222	0	1.4 (1.3–1.5)	0.70 (0.61–0.79)	1.4 (1.3–1.4)	2.7 (2.4–3.0)	3.2 (2.9–3.5)
3 (2012–2013)	1071	0.09	1.3 (1.3–1.4)	0.61 (0.55–0.68)	1.3 (1.2–1.4)	2.6 (2.2–2.9)	3.5 (2.9–4.2)
4 (2014–2015)	1051	0	1.2 (1.0–1.3)	0.58 (0.53–0.63)	1.1 (1.0–1.1)	2.4 (1.9–2.9)	3.2 (2.3–4.0)
60–79 years							
1 (2007–2009)	1079	0	2.1 (1.9–2.3)	1.0 (0.92–1.1)	2.0 (1.8–2.2)	4.1 (3.5–4.8)	5.2 (4.2–6.2)
2 (2009–2011)	1082	0	1.9 (1.8–1.9)	1.0 (0.94–1.1)	1.7 (1.7–1.8)	3.5 (3.2–3.8)	4.2 (3.8–4.6)
3 (2012–2013)	1043	0.10	1.6 (1.6–1.7)	0.81 (0.78–0.85)	1.6 (1.4–1.7)	3.3 (3.0–3.5)	4.0 (3.6–4.4)
4 (2014–2015)	995	0	1.5 (1.4–1.6)	0.74 (0.66–0.81)	1.4 (1.3–1.5)	2.9 (2.5–3.3)	3.8 (3.0–4.6)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

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10.5 MERCURY

Mercury (CASRN 7439-97-6) is a naturally occurring soft, silvery white metal present in the Earth's crust at an average concentration of approximately 0.000005% (Emsley, 2001). It is the only metal that is a liquid at room temperature. Mercury exists in elemental, inorganic, and organic forms (CCME, 1999). Elemental and certain organic forms of mercury have sufficiently high vapour pressures to be present as vapour in air (ATSDR, 1999; ATSDR, 2013). The most common organic mercury compounds in nature are methylmercury (monomethylmercury) and dimethylmercury. Mercury can be converted among its elemental, inorganic, and organic forms by a variety of processes, including biological transformation (Environment Canada, 2010).

Mercury is found throughout the environment, including remote Arctic regions because of its persistence, mobility, and tendency to accumulate in colder climates. Natural sources include volcanic activity and natural erosion of mercury-containing deposits (Environment Canada and Health Canada, 2010). Metabolism of inorganic mercury by micro-organisms in the environment creates organic mercury (methylmercury) that often bioaccumulates in terrestrial and aquatic food chains (ATSDR, 1999; ATSDR, 2013). Anthropogenic sources of inorganic mercury include metal mining and smelting; combustion of fossil fuels, particularly coal; incineration of municipal wastes; cement production; and sewage sludge and waste water (UNEP, 2002). Inorganic mercury may also be released to the environment following disposal of products containing mercury.

Mercury has unique properties that have made it useful in certain products such as wiring devices, switches, and scientific measuring devices, including vacuum gauges and thermometers (ATSDR, 1999; ATSDR, 2013). Today the manufacture and import of most mercury-containing products are prohibited in Canada. Exemptions include certain essential products such as certain medical and research applications, dental amalgams, and fluorescent and other types of lamps (Canada, 2014). Use of mercury-containing light bulbs is increasing because of widespread replacement of incandescent bulbs with compact fluorescent bulbs. Mercury is also used as an industrial catalyst and in laboratory reagents, disinfectants, embalming solutions, and some pharmaceuticals. A significant use of inorganic mercury is in dental amalgam, which is composed of approximately 50% mercury (IMERC, 2010; SCENIHR, 2015). Based on data collected as part of the Canadian Health Measures Survey (CHMS) cycle 1 (2007–2009), it was estimated that approximately 63.95% of the Canadian population age 6 and over had one or more amalgam-restored tooth surfaces (Richardson, 2014).

Mercury exposure in the general population is primarily through the consumption of fish and seafood in which methylmercury is the predominant form (Health Canada, 2007). To a lesser extent, the general population is exposed to inorganic mercury from such sources as dental amalgams (Health Canada, 1996, Health Canada, 2004; SCENIHR 2015). The general population may also be exposed to elemental mercury via inhalation of vapours in ambient air, ingestion, or through dental and medical treatments (ATSDR, 1999).

Approximately 95% of organic mercury is absorbed from the gastrointestinal tract following oral ingestion (ATSDR, 1999; ATSDR, 2013). Following absorption, organic mercury is distributed to all tissues, including hair, with highest accumulation in the kidneys. It readily passes the blood-brain barrier and enters the brain, and in pregnant women can easily cross the placental barrier into the fetus (Health Canada, 2004). Absorbed organic mercury is demethylated in the body to inorganic mercury that accumulates primarily in the liver and kidneys. The biological half-life of methylmercury is approximately 50 days. The majority of mercury in the body is excreted via feces, with a small amount excreted as inorganic mercury in urine (ATSDR, 1999; ATSDR, 2013).

Generally less than 10% of inorganic mercury is absorbed through the intestinal tract (Health Canada, 2004). Absorbed inorganic mercury accumulates readily in the kidneys (IPCS, 2003). It also accumulates in placental tissues but does not cross placental or blood-brain barriers as easily as elemental or methylmercury (Health Canada, 2004). Excretion of elemental and inorganic mercury compounds occurs mainly in urine and feces with an absorbed dose half-life of approximately 1 to 2 months (IPCS, 2003).

Elemental mercury is absorbed across the lungs and gastrointestinal tract with absorption rates of about 80% and 0.01%, respectively (Health Canada, 2004). Once absorbed, elemental mercury enters the bloodstream and is rapidly transported to other parts of the body, including the brain and kidneys. As with organic mercury, it readily crosses the blood-brain and placental barriers (Health Canada, 2004). Once in the body, elemental mercury is oxidized in the tissues to inorganic forms and can remain for weeks or months with an estimated half-life of approximately 60 days (Sandborgh-Englund et al., 1998).

Long-term exposure to elemental and inorganic mercury is commonly evaluated using mercury concentrations in urine (IPCS, 2003). Hair also may be used as a biomarker of chronic exposure, although inorganic forms of mercury are not excreted to any significant amount in scalp hair, making it an inappropriate biomarker of inorganic mercury exposure (ATSDR, 1999; ATSDR, 2013; IPCS, 2003). Total blood mercury concentrations primarily reflect recent dietary exposure to organic forms of mercury, particularly methylmercury (ATSDR, 1999; ATSDR, 2013; IPCS, 2003). The concentration of total mercury in blood is accepted as a reasonable measure of methylmercury exposure; however, methylmercury itself may also be measured directly in blood. Based on a review of existing data from other countries, the World Health Organization has estimated that the average total blood mercury concentration for the general population is approximately 8 µg/L (WHO, 1990). In individuals who consume fish daily, methylmercury concentrations in blood can be as high as 200 µg/L (WHO, 1990).

Mercury is known to be toxic to humans, with the effects depending on the mercury form, the route of exposure, the timing of exposure, and the absorbed concentration. Chronic oral exposure to low levels of methylmercury may not result in any observable

symptoms (Health Canada, 2007). The primary effects associated with oral exposure to organic mercury compounds are neurological effects and developmental neurotoxicity (ATSDR, 2013; EFSA CONTAM Panel, 2012; FAO/WHO, 2011; Health Canada, 2007). Symptoms of organic mercury toxicity include a tingling sensation in the extremities; impaired peripheral vision, hearing, taste, and smell; slurred speech; muscle weakness and an unsteady gait; irritability; memory loss; depression; and sleeping difficulties. Exposure of a fetus or young child to organic mercury can result in effects on the development of the nervous system, affecting fine-motor function, attention, verbal learning, and memory (ATSDR, 2013; Health Canada, 2007). Exposure to elemental mercury may be hazardous, depending upon the levels of exposure, because the vapour that can be released from this form is readily absorbed into the body through inhalation. Inhalation of mercury vapour may cause respiratory, cardiovascular, kidney, and neurological effects. In 1996, Health Canada concluded that mercury exposure from dental amalgams does not pose a health impact for the general population (Health Canada, 1996). Subsequent studies since this report have concurred that exposure to inorganic mercury from dental amalgams has not been associated with neurologic effects in children or adults (Bates et al., 2004; Bellinger et al., 2007; DeRouen et al., 2006; Factor-Litvak et al., 2003; SCENIHR, 2015).

The International Agency for Research on Cancer (IARC) determined that methylmercury compounds are possibly carcinogenic to humans (Group 2B), based on animal data showing a link to certain cancers, particularly renal cancer (IARC, 1993). Elemental mercury and inorganic mercury compounds were classified by IARC as Group 3, not classifiable as to their carcinogenicity to humans (IARC, 1993).

The United Nations Environment Programme (UNEP) *Global Risk Assessment for Mercury* concluded that there was sufficient evidence of adverse impacts from mercury to warrant further international action to reduce the risks to human health and the environment (UNEP, 2002). International negotiations under UNEP resulted in the signing of the Minamata Convention on Mercury, a global legally binding agreement to prevent mercury emissions and releases (UNEP, 2013). The Minamata Convention is intended to reduce global atmospheric emissions, supply, trade, and demand for mercury, and to find environmentally sound solutions for storage of mercury and mercury-containing wastes.

In Canada, mercury and its compounds are listed as toxic substances on Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Canada, 2012a). Existing and planned actions to manage the risks from mercury are summarized in the Government of Canada's *Risk Management Strategy for Mercury* (Environment Canada and Health Canada, 2010). These risk management actions include several Canada-wide standards that have been established to reduce the releases of mercury to the environment (CCME, 2000; CCME, 2005; CCME, 2006; CCME, 2007). The Products Containing Mercury Regulations came into force in 2015, and prohibit the manufacture and import of products containing mercury or any of its compounds as well as provide content limits for exempted products (Canada, 2014). The Surface Coating Materials Regulations, in effect under the *Canada Consumer Product Safety Act*, restrict the level of mercury in all surface coating materials advertised, sold, or imported into Canada (Canada, 2005). In addition, the Toys Regulations prohibit any compound of mercury in the surface coating material that is applied to a product that is used by a child in learning or play situations (Canada, 2011). Mercury and its compounds are also included as prohibited ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Canada, 1985; Health Canada, 2015). The Food and Drug Regulations prohibit sale in Canada of drugs for human use containing mercury or any of its salts or derivatives except in some specific instances, including those where it is present as a preservative (Canada, 2012b).

Health Canada has established a methylmercury blood guidance value of 20 µg/L for the general adult population, for which levels below this value are considered in the normal acceptable range (Health Canada, 2004). For children (under 18 years of age), pregnant women, and women of childbearing age (under 50 years of age), a provisional methylmercury blood guidance value of 8 µg/L has been proposed for the protection of the developing nervous system (Legrand et al., 2010). On the basis of health considerations, Health Canada, in collaboration

with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for mercury in drinking water (Health Canada, 1986; Health Canada, 2014). Health Canada has also established maximum levels for mercury in retail fish (Health Canada, 2012), and provided consumption advice for consumers of certain types of fish (Health Canada, 2008). *Eating Well with Canada's Food Guide* recommends eating at least two food guide servings each week of fish that are low in mercury and high in omega-3 fatty acids (Health Canada, 2011). Mercury is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply.

During cycle 1 (2007–2009) of the CHMS, the geometric mean total mercury level in blood of the Canadian population aged 6–79 years was 0.69 µg/L (Lye et al., 2013). The majority (97.8%) of Canadian women aged 16–49 years, including pregnant women, had blood mercury values below the provisional Health Canada blood guidance value of 8 µg/L (Lye et al., 2013). The geometric mean urinary inorganic mercury concentration in dental amalgam-free participants from cycle 1 of the CHMS was 0.10 µg/L compared with the geometric mean concentration for all participants of 0.22 µg/L (Nicolae et al., 2013). In general, mean urinary inorganic mercury concentrations tended to increase with the number of amalgam surfaces and appeared to be influenced by age and sex (Nicolae et al., 2013). The population coverage of the CHMS excludes persons living on reserves and other Aboriginal settlements in the provinces of Canada. However, this subpopulation has been surveyed as part of the First Nations Biomonitoring Initiative (FNBI), a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprises 13 randomly selected First Nation communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the geometric mean and 95th percentile for total mercury in blood were 0.95 µg/L and 9.28 µg/L, respectively. For inorganic mercury in urine, the geometric mean and 95th percentile were 0.26 µg/L and 1.98 µg/L, respectively.

Total mercury was analyzed in the whole blood of all CHMS participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Methylmercury was analyzed in the whole blood of CHMS participants aged 20–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Inorganic mercury was analyzed in the urine of all CHMS participants aged 6–79 years in cycle 1 (2007–2009) and 3 to 79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data from these cycles are presented in blood as µg/L and in urine as both µg/L and µg/g creatinine. Finding a measurable amount of mercury in blood or urine is an indicator of exposure to mercury and does not necessarily mean that an adverse health effect will occur.

Table 10.5.1

Mercury (total) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	6070	15.55	0.69 (0.56–0.87)	<LOD	0.74 (0.55–0.93)	3.4 (2.4–4.5)	5.5[E] (3.3–7.6)
3 (2012–2013)	5538	37.02	0.79 (0.64–0.97)	<LOD	0.79 (0.62–0.96)	3.2[E] (1.5–4.9)	5.2[E] (3.0–7.5)
4 (2014–2015)	5498	44.82	—	<LOD	0.59 (0.47–0.72)	2.5 (1.9–3.1)	3.5 (2.9–4.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	16.16	0.72 (0.56–0.91)	<LOD	0.76 (0.53–0.99)	3.9 (2.7–5.1)	6.1[E] (2.7–9.5)
3 (2012–2013)	2769	37.63	0.76 (0.60–0.97)	<LOD	0.74 (0.54–0.94)	3.2[E] (1.3–5.0)	5.6[E] (3.4–7.8)
4 (2014–2015)	2754	44.99	—	<LOD	0.58 (0.45–0.71)	2.8 (2.0–3.6)	3.7 (2.6–4.8)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	14.98	0.67 (0.54–0.83)	<LOD	0.71 (0.53–0.88)	3.0 (2.0–4.0)	5.1[E] (3.0–7.1)
3 (2012–2013)	2769	36.40	0.81 (0.67–0.99)	<LOD	0.82 (0.67–0.97)	3.2[E] (1.4–4.9)	5.1[E] (2.4–7.8)
4 (2014–2015)	2744	44.64	—	<LOD	0.60 (0.47–0.74)	2.2 (1.6–2.8)	3.3 (2.7–4.0)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	29.90	0.27 (0.20–0.36)	<LOD	0.19[E] (<LOD–0.29)	1.4[E] (0.44–2.3)	3.0[E] (1.7–4.3)
3 (2012–2013)	471	59.45	—	<LOD	<LOD	1.3 (1.0–1.7)	1.7[E] (0.88–2.5)
4 (2014–2015)	479	71.19	—	<LOD	<LOD	0.85[E] (<LOD–1.3)	1.3[E] (0.54–2.1)
6–11 years							
1 (2007–2009)	910	24.84	0.26 (0.22–0.32)	<LOD	0.24 (0.18–0.29)	1.3 (1.0–1.6)	2.1[E] (1.3–2.9)
2 (2009–2011)	961	29.03	0.28 (0.22–0.34)	<LOD	0.21[E] (0.11–0.30)	1.2 (0.84–1.5)	2.0 (1.3–2.6)
3 (2012–2013)	944	54.77	—	<LOD	<LOD	1.2 (0.78–1.7)	1.9[E] (0.91–2.9)
4 (2014–2015)	925	59.57	—	<LOD	<LOD	1.1 (0.84–1.3)	1.5 (0.96–2.0)
12–19 years							
1 (2007–2009)	945	20.85	0.30 (0.23–0.40)	<LOD	0.28 (0.20–0.37)	1.3[E] (0.47–2.2)	2.2[E] (0.88–3.5)
2 (2009–2011)	997	26.58	0.27 (0.21–0.35)	<LOD	0.19[E] (<LOD–0.30)	1.3 (0.84–1.7)	2.4[E] (1.3–3.5)
3 (2012–2013)	977	52.61	—	<LOD	<LOD	1.6[E] (0.62–2.6)	2.8[E] (1.3–4.4)
4 (2014–2015)	975	61.33	—	<LOD	<LOD	1.3 (0.92–1.7)	2.2[E] (1.2–3.2)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	1165	8.76	0.65 (0.52–0.81)	<LOD	0.76 (0.61–0.91)	3.0[E] (1.9–4.1)	4.9[E] (2.4–7.4)
2 (2009–2011)	1313	10.05	0.64 (0.47–0.85)	<LOD	0.65 (0.43–0.86)	2.9 (2.0–3.9)	5.2[E] (2.6–7.8)
3 (2012–2013)	1032	30.91	0.82 (0.65–1.0)	<LOD	0.77 (0.57–0.96)	4.1[E] (1.5–6.6)	6.0[E] (3.6–8.3)
4 (2014–2015)	1073	40.54	—	<LOD	0.48 (<LOD–0.65)	2.0 (1.6–2.4)	2.9 (2.0–3.8)
40–59 years							
1 (2007–2009)	1220	3.52	1.0 (0.80–1.3)	0.21[E] (0.12–0.30)	1.1 (0.83–1.3)	3.6 (2.3–4.9)	6.4[E] (3.0–9.8)
2 (2009–2011)	1222	5.16	1.0 (0.79–1.3)	0.15 (0.11–0.20)	1.0 (0.84–1.2)	4.1[E] (2.4–5.8)	7.3[E] (2.5–12)
3 (2012–2013)	1071	20.54	0.96 (0.74–1.2)	<LOD	0.99 (0.78–1.2)	3.4[E] (1.5–5.4)	5.2[E] (2.8–7.6)
4 (2014–2015)	1051	27.69	0.77 (0.65–0.92)	<LOD	0.80 (0.63–0.98)	3.1 (2.2–4.1)	3.7 (2.9–4.6)
60–79 years							
1 (2007–2009)	1079	4.73	0.87 (0.64–1.2)	F	0.96 (0.75–1.2)	3.4 (2.4–4.4)	4.8[E] (2.7–6.9)
2 (2009–2011)	1082	5.27	1.1 (0.86–1.5)	0.17[E] (<LOD–0.28)	1.2 (0.89–1.5)	4.3 (3.1–5.5)	6.5[E] (3.9–9.1)
3 (2012–2013)	1043	19.18	1.0 (0.82–1.3)	<LOD	0.99 (0.71–1.3)	3.8[E] (2.2–5.3)	6.7[E] (1.9–11)
4 (2014–2015)	995	24.92	0.88 (0.73–1.1)	<LOD	0.92 (0.76–1.1)	3.3 (2.6–4.0)	4.6 (3.1–6.1)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

Table 10.5.2

Mercury (inorganic) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	5696	50.42	—	<LOD	<LOD	1.3 (1.1–1.5)	2.0 (1.7–2.3)
4 (2014–2015)	5595	56.35	—	<LOD	<LOD	1.3 (0.94–1.6)	2.2 (1.7–2.7)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	2842	49.37	—	<LOD	0.20[E] (<LOD–0.27)	1.2 (0.92–1.5)	1.9 (1.3–2.4)
4 (2014–2015)	2809	55.68	—	<LOD	<LOD	1.3 (0.87–1.7)	2.2 (1.5–2.9)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	2854	51.47	—	<LOD	<LOD	1.4 (0.97–1.8)	2.1 (1.5–2.8)
4 (2014–2015)	2786	57.04	—	<LOD	<LOD	1.3 (0.82–1.7)	2.2[E] (1.3–3.2)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	520	74.81	—	<LOD	<LOD	0.28[E] (<LOD–0.48)	0.59[E] (0.35–0.84)
4 (2014–2015)	512	83.98	—	<LOD	<LOD	0.25[E] (<LOD–0.36)	0.41[E] (0.20–0.61)
6–11 years							
1 (2007–2009)	1028	66.05	—	<LOD	<LOD	0.99[E] (0.56–1.4)	1.8[E] (0.99–2.7)
3 (2012–2013)	1010	61.29	—	<LOD	<LOD	0.93[E] (0.50–1.4)	F
4 (2014–2015)	1008	69.94	—	<LOD	<LOD	0.58[E] (0.35–0.80)	1.3 (0.93–1.8)
12–19 years							
1 (2007–2009)	975	57.54	—	<LOD	<LOD	1.2 (0.76–1.6)	2.2 (1.5–3.0)
3 (2012–2013)	997	59.58	—	<LOD	<LOD	0.55[E] (0.33–0.78)	1.1[E] (0.53–1.6)
4 (2014–2015)	988	65.49	—	<LOD	<LOD	0.53 (0.38–0.68)	0.96 (0.65–1.3)
20–39 years							
1 (2007–2009)	1166	46.23	—	<LOD	0.22 (0.16–0.28)	1.4 (1.0–1.7)	2.3 (1.8–2.7)
3 (2012–2013)	1048	45.13	—	<LOD	0.20[E] (<LOD–0.28)	1.1 (0.87–1.3)	1.9[E] (0.89–3.0)
4 (2014–2015)	1057	50.99	—	<LOD	<LOD	1.1[E] (0.50–1.6)	2.1[E] (1.1–3.1)
40–59 years							
1 (2007–2009)	1207	36.04	0.31 (0.25–0.37)	<LOD	0.37 (0.28–0.47)	2.5 (1.8–3.2)	3.5 (2.3–4.7)
3 (2012–2013)	1080	36.20	0.31 (0.26–0.39)	<LOD	0.30 (0.20–0.40)	1.7 (1.2–2.2)	2.2 (1.7–2.6)
4 (2014–2015)	1037	40.31	—	<LOD	0.26 (0.21–0.32)	1.9 (1.3–2.5)	3.5[E] (2.0–5.1)
60–79 years							
1 (2007–2009)	1068	45.69	—	<LOD	0.25[E] (0.15–0.35)	2.0 (1.4–2.5)	3.0 (2.5–3.5)
3 (2012–2013)	1041	39.00	0.26 (0.23–0.30)	<LOD	0.24 (0.18–0.30)	1.4 (0.98–1.8)	2.3 (1.6–2.9)
4 (2014–2015)	993	41.69	—	<LOD	0.26 (0.20–0.32)	1.5 (1.1–1.9)	2.4 (2.0–2.7)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

F Data is too unreliable to be published.

Table 10.5.3

Mercury (inorganic) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	5694	50.42	—	<LOD	<LOD	1.0 (0.94–1.1)	1.6 (1.3–1.9)
4 (2014–2015)	5594	56.35	—	<LOD	<LOD	0.94 (0.78–1.1)	1.5 (1.2–1.8)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	2842	49.37	—	<LOD	0.21 (<LOD–0.24)	0.86 (0.63–1.1)	1.2 (1.1–1.4)
4 (2014–2015)	2808	55.68	—	<LOD	<LOD	0.82 (0.61–1.0)	1.3 (1.0–1.6)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	2852	51.47	—	<LOD	<LOD	1.2 (0.87–1.5)	1.9 (1.5–2.3)
4 (2014–2015)	2786	57.04	—	<LOD	<LOD	1.1 (0.85–1.3)	1.7 (1.3–2.1)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
3 (2012–2013)	519	74.81	—	<LOD	<LOD	0.73[E] (<LOD–1.0)	1.0 (0.72–1.3)
4 (2014–2015)	512	83.98	—	<LOD	<LOD	0.46 (<LOD–0.56)	0.72[E] (0.46–0.99)
6–11 years							
1 (2007–2009)	1025	66.05	—	<LOD	<LOD	1.3[E] (0.62–1.9)	2.0 (1.3–2.7)
3 (2012–2013)	1010	61.29	—	<LOD	<LOD	0.99[E] (0.55–1.4)	1.9[E] (0.84–3.0)
4 (2014–2015)	1007	69.94	—	<LOD	<LOD	0.58[E] (0.32–0.84)	1.3 (0.83–1.7)
12–19 years							
1 (2007–2009)	975	57.54	—	<LOD	<LOD	0.79 (0.55–1.0)	1.3[E] (0.79–1.8)
3 (2012–2013)	997	59.58	—	<LOD	<LOD	0.42[E] (0.27–0.58)	0.73[E] (0.42–1.0)
4 (2014–2015)	988	65.49	—	<LOD	<LOD	0.33 (0.27–0.40)	0.52 (0.35–0.70)
20–39 years							
1 (2007–2009)	1162	46.23	—	<LOD	0.21 (0.18–0.24)	1.1 (0.89–1.4)	1.9 (1.5–2.2)
3 (2012–2013)	1048	45.13	—	<LOD	0.22 (<LOD–0.26)	0.85 (0.56–1.2)	1.2 (0.97–1.3)
4 (2014–2015)	1057	50.99	—	<LOD	<LOD	0.66[E] (0.38–0.94)	0.92 (0.60–1.2)
40–59 years							
1 (2007–2009)	1202	36.04	0.39 (0.33–0.48)	<LOD	0.43 (0.33–0.52)	2.1 (1.5–2.7)	3.0 (2.3–3.7)
3 (2012–2013)	1079	36.20	0.33 (0.29–0.38)	<LOD	0.33 (0.27–0.40)	1.3 (0.91–1.7)	1.7 (1.5–2.0)
4 (2014–2015)	1037	40.31	—	<LOD	0.24 (0.21–0.27)	1.3 (0.93–1.8)	2.1[E] (1.1–3.1)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	1068	45.69	—	<LOD	0.29[E] (0.17–0.42)	2.0 (1.7–2.3)	2.7 (2.1–3.4)
3 (2012–2013)	1041	39.00	0.32 (0.28–0.36)	<LOD	0.32 (0.27–0.37)	1.3 (0.95–1.6)	2.2 (1.6–2.8)
4 (2014–2015)	993	41.69	—	<LOD	0.27 (0.23–0.31)	1.2 (1.1–1.4)	1.6 (1.4–1.9)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

Table 10.5.4

Methylmercury — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 20–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–79 years							
3 (2012–2013)	1032	18.70	0.69 (0.52–0.91)	<LOD	0.78 (0.54–1.0)	3.3[E] (1.3–5.3)	5.6[E] (2.9–8.2)
4 (2014–2015)	1043	18.12	0.59 (0.51–0.68)	<LOD	0.57 (0.45–0.68)	2.8 (1.9–3.7)	4.1 (3.5–4.6)
Males, 20–79 years							
3 (2012–2013)	502	18.33	0.68[E] (0.41–1.1)	<LOD	0.68[E] (0.26–1.1)	4.6[E] (1.3–7.8)	8.1[E] (4.2–12)
4 (2014–2015)	512	18.16	0.62 (0.53–0.71)	<LOD	0.56 (0.41–0.71)	2.9 (1.9–4.0)	4.0 (3.2–4.8)
Females, 20–79 years							
3 (2012–2013)	530	19.06	0.70 (0.58–0.85)	<LOD	0.89 (0.74–1.0)	2.8[E] (1.4–4.1)	4.7[E] (3.0–6.4)
4 (2014–2015)	531	18.08	0.57 (0.46–0.70)	<LOD	0.57 (0.43–0.72)	2.5[E] (0.99–4.0)	4.4 (3.2–5.7)
20–39 years							
3 (2012–2013)	359	24.51	0.61 (0.45–0.82)	<LOD	0.65 (0.42–0.87)	F	5.0[E] (1.9–8.1)
4 (2014–2015)	361	25.76	0.42 (0.34–0.52)	<LOD	0.48 (0.35–0.61)	1.8 (1.4–2.2)	2.2 (1.7–2.6)
40–59 years							
3 (2012–2013)	313	19.17	0.65[E] (0.44–0.96)	<LOD	0.71[E] (0.27–1.2)	3.2[E] (0.85–5.5)	5.8[E] (2.3–9.3)
4 (2014–2015)	316	17.41	0.66 (0.51–0.84)	<LOD	0.56[E] (0.33–0.79)	3.7 (2.5–4.9)	4.3 (3.3–5.3)
60–79 years							
3 (2012–2013)	360	12.50	0.94 (0.67–1.3)	<LOD	1.0[E] (0.65–1.4)	3.4[E] (2.0–4.8)	F
4 (2014–2015)	366	11.20	0.83 (0.63–1.1)	<LOD	0.78[E] (0.49–1.1)	3.8 (2.7–5.0)	5.1 (3.3–6.9)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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SUMMARY AND RESULTS FOR NICOTINE METABOLITE 11

11.1 COTININE

Cotinine (CASRN 486-56-6) is the major primary metabolite of nicotine, a chemical found naturally in the tobacco plant and present in tobacco products such as cigarettes, cigars, and smokeless tobacco products (e.g. chewing tobacco and snuff) (Benowitz and Jacob, 1994). Nicotine is also incorporated into nicotine replacement therapies such as the nicotine gum, patch, lozenge, inhaler, e-cigarettes, and buccal spray.

Human exposure to nicotine occurs primarily through the use of tobacco products, exposure to environmental tobacco smoke, and the use of nicotine replacement therapies (HSDB, 2009). In addition, infants breast fed by women who smoke may be exposed to nicotine in breast milk (HSDB, 2009).

Inhalation is the most effective intake route with on average 60% to 80% of nicotine absorbed through the lungs (Iwase et al., 1991). Nicotine can also be absorbed through the skin and gastrointestinal tract, but at a much lower efficiency (Karaconji, 2005). Once inside the body, approximately 70% to 80% of nicotine is metabolized into cotinine. It has a half-life of 10 to 20 hours and can remain in the body at detectable levels for up to 7 days (Benowitz and Jacob, 1994; Curvall et al., 1990; Hecht et al., 1999). Cotinine is considered to be the most relevant biomarker for exposure to tobacco products and tobacco smoke (Brown et al., 2005; CDC, 2009; Seaton and Vesell, 1993).

Tobacco smoke is a combination of gases, liquids, and breathable particles, some of which are harmful to human health. It contains over 4,000 chemicals, including at least 70 that cause, initiate, or promote cancer, and has been classified by the International Agency for Research on Cancer (IARC) as Group 1, carcinogenic to humans (Health Canada, 2011; IARC, 2004). Exposure to these chemicals also contributes directly to other diseases, such as emphysema and heart disease, and an increased risk of asthma (CDC, 2004). During pregnancy, smoking may lead to miscarriages, low-birth-weight infants and less breast milk (WHO, 2010). Most of these chemicals are formed during the combustion of tobacco; others are found naturally in tobacco and are released as the tobacco burns (CDC, 2004). Smokeless tobaccos, including chewing tobacco and snuff, contain 28 known cancer-causing chemicals and, similar to the tobaccos used in cigarettes, pipes, and cigars, can lead to nicotine dependence and addiction (Health Canada, 2010; IARC, 2007). Smokeless tobacco use causes oral, pancreatic, and esophageal cancer and has been classified by IARC as Group 1, carcinogenic to humans (IARC, 2007). It can also cause serious dental health problems, including recession of the gums, tooth loss, and discolouration of the teeth and gums (Walsh and Epstein, 2000). Levels of cotinine in the blood and urine of non-smokers have been correlated with some adverse health effects related to tobacco smoke exposure, and cotinine itself may contribute to the neuropharmacological effects of tobacco smoking (Benowitz, 1996; Crooks and Dwoskin, 1997).

As a result of the adverse health effects associated with tobacco use, the Government of Canada, along with provincial and territorial governments and various

municipalities, has taken several steps to reduce the prevalence of tobacco use as well as exposure to tobacco smoke. These steps include prohibitions on the sale of tobacco to youth, requirements to apply health warnings on tobacco packaging, and restrictions on the promotion of tobacco products, including the display of tobacco products at retail outlets (Health Canada, 2006). Additional steps include the offer of cessation help along with initiatives to eliminate smoking in workplaces and enclosed public locations (Health Canada, 2006).

The First Nations Biomonitoring Initiative (FNBI) is a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprises 13 randomly selected First Nation communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the 50th percentile concentrations of cotinine in urine from smokers and non-smokers were 315.79 µg/L and <1.1 µg/L, respectively. Data from cycle 1 (2007–2009) of the Canadian Health Measures Survey (CHMS) demonstrated that a substantial proportion of the Canadian population was exposed to secondhand smoke. The study found that certain non-smoking subpopulations, including children, adolescents, and those exposed to secondhand smoke in the home, had higher percentages with detectable cotinine concentrations (≥1.1 µg/L), indicating secondhand smoke exposure (Wong et al., 2013). A study of occupationally exposed non-smoking bar workers in the Toronto area examined the effects of a 2004 smoke-free workplace bylaw; the study showed a 1-month post-ban decline in the geometric mean of urinary cotinine from 10.3 µg/L to 3.10 µg/L (Repace et al., 2013). A concentration of 50 µg/L urine for cotinine is recommended for determining smoking status; levels greater than this concentration are attributed to smokers (SRNT Subcommittee on Biochemical Verification, 2002). Using this concentration, a study assessed the validity of self-reported cigarette smoking status among Canadians using urinary cotinine data from cycle 1 (2007–2009) of the CHMS (Wong et al., 2012). Compared with estimates based on urinary cotinine concentration, smoking prevalence based on self-reporting was only 0.3 percentage points lower, indicating that accurate estimates of the prevalence of cigarette smoking among Canadians can be derived from self-reported smoking status data.

Cotinine was analyzed in the urine of all CHMS participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015). Data from these cycles are presented as both µg/L and µg/g creatinine for non-smokers and smokers. Survey participants aged 3–11 years were assumed to be non-smokers. In this survey, a smoker is defined as someone who is a current daily or occasional smoker and a non-smoker is defined as someone who does not currently smoke and has either never smoked or who was previously a daily or occasional smoker. Finding a measurable amount of cotinine in urine is an indicator of exposure to nicotine.

In addition to free cotinine, nicotine and several other metabolites (cotinine-*N*-glucuronide, nicotine-*N*-glucuronide, *trans*-3-hydroxycotinine, *trans*-3-hydroxycotinine-*O*-glucuronide, and anabasine) were analyzed in cycle 1 (2007–2009) and cycle 3 (2012–2013) of the CHMS. Free and total 4-(methylnitrosoamino)-1-(3-pyridyl)-1-butanol (NNAL), a metabolite of a tobacco-specific *N*-nitrosamine found only in tobacco and products derived from tobacco, were also analyzed in cycle 1 (2007–2009) and cycle 3 (2012–2013) of the CHMS. Data on these tobacco chemicals and their metabolites are available from Statistics Canada through the Research Data Centres Program.

Table 11.1.1

Cotinine (non-smokers) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	5468	86.85	—	<LOD	<LOD	2.6[E] (<LOD–4.4)	F
3 (2012–2013)	4978	88.59	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	4907	88.95	—	<LOD	<LOD	F	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2594	84.93	—	<LOD	<LOD	F	F
3 (2012–2013)	2444	87.11	—	<LOD	<LOD	F	F
4 (2014–2015)	2446	86.84	—	<LOD	<LOD	F	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2874	88.59	—	<LOD	<LOD	1.5[E] (<LOD–2.5)	F
3 (2012–2013)	2534	90.02	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	2461	91.06	—	<LOD	<LOD	<LOD	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	573	86.56	—	<LOD	<LOD	F	F
3 (2012–2013)	522	88.12	—	<LOD	<LOD	F	F
4 (2014–2015)	512	87.11	—	<LOD	<LOD	2.3 (1.7–3.0)	F
6–11 years							
1 (2007–2009)	1045	83.83	—	<LOD	<LOD	3.9[E] (1.9–5.8)	10[E] (5.7–14)
2 (2009–2011)	1061	83.79	—	<LOD	<LOD	4.9[E] (1.9–7.9)	12[E] (6.3–18)
3 (2012–2013)	1007	86.79	—	<LOD	<LOD	F	7.1[E] (2.7–11)
4 (2014–2015)	1008	89.88	—	<LOD	<LOD	<LOD	F
12–19 years							
1 (2007–2009)	882	80.27	—	<LOD	<LOD	8.3[E] (3.8–13)	19[E] (8.3–30)
2 (2009–2011)	928	80.06	—	<LOD	<LOD	F	F
3 (2012–2013)	889	82.56	—	<LOD	<LOD	F	13[E] (7.6–19)
4 (2014–2015)	901	84.79	—	<LOD	<LOD	F	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	874	85.35	—	<LOD	<LOD	F	F
2 (2009–2011)	1009	86.22	—	<LOD	<LOD	F	F
3 (2012–2013)	792	90.53	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	785	87.26	—	<LOD	<LOD	F	F
40–59 years							
1 (2007–2009)	947	88.81	—	<LOD	<LOD	F	F
2 (2009–2011)	972	91.56	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	851	91.19	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	827	90.69	—	<LOD	<LOD	<LOD	F
60–79 years							
1 (2007–2009)	956	90.69	—	<LOD	<LOD	<LOD	F
2 (2009–2011)	925	93.08	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	917	92.58	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	874	93.14	—	<LOD	<LOD	<LOD	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).

E Use data with caution.

F Data is too unreliable to be published.

Table 11.1.2

Cotinine (non-smokers) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	5455	86.85	—	<LOD	<LOD	3.3 (<LOD–4.4)	F
3 (2012–2013)	4976	88.59	—	<LOD	<LOD	<LOD	6.1[E] (<LOD–10)
4 (2014–2015)	4906	88.95	—	<LOD	<LOD	2.6 (<LOD–3.5)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2588	84.93	—	<LOD	<LOD	3.9[E] (<LOD–5.9)	F
3 (2012–2013)	2444	87.11	—	<LOD	<LOD	2.4[E] (<LOD–3.3)	F
4 (2014–2015)	2445	86.84	—	<LOD	<LOD	2.6[E] (<LOD–4.3)	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2867	88.59	—	<LOD	<LOD	3.0 (<LOD–3.9)	F
3 (2012–2013)	2532	90.02	—	<LOD	<LOD	<LOD	5.2[E] (<LOD–7.8)
4 (2014–2015)	2461	91.06	—	<LOD	<LOD	<LOD	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	572	86.56	—	<LOD	<LOD	F	F
3 (2012–2013)	521	88.12	—	<LOD	<LOD	5.6[E] (<LOD–7.7)	F
4 (2014–2015)	512	87.11	—	<LOD	<LOD	3.7[E] (2.2–5.2)	F
6–11 years							
1 (2007–2009)	1042	83.83	—	<LOD	<LOD	6.2[E] (1.9–10)	F
2 (2009–2011)	1059	83.79	—	<LOD	<LOD	5.2[E] (1.9–8.5)	12[E] (5.4–18)
3 (2012–2013)	1007	86.79	—	<LOD	<LOD	3.5[E] (<LOD–5.8)	7.7[E] (2.6–13)
4 (2014–2015)	1007	89.88	—	<LOD	<LOD	<LOD	F
12–19 years							
1 (2007–2009)	881	80.27	—	<LOD	<LOD	7.9[E] (4.6–11)	F
2 (2009–2011)	926	80.06	—	<LOD	<LOD	F	F
3 (2012–2013)	889	82.56	—	<LOD	<LOD	3.2[E] (<LOD–5.5)	F
4 (2014–2015)	901	84.79	—	<LOD	<LOD	F	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
1 (2007–2009)	871	85.35	—	<LOD	<LOD	4.5[E] (<LOD–7.4)	F
2 (2009–2011)	1007	86.22	—	<LOD	<LOD	F	F
3 (2012–2013)	792	90.53	—	<LOD	<LOD	<LOD	3.3[E] (<LOD–5.2)
4 (2014–2015)	785	87.26	—	<LOD	<LOD	F	F
40–59 years							
1 (2007–2009)	944	88.81	—	<LOD	<LOD	4.6[E] (<LOD–6.4)	F
2 (2009–2011)	970	91.56	—	<LOD	<LOD	<LOD	4.7[E] (<LOD–7.8)
3 (2012–2013)	850	91.19	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	827	90.69	—	<LOD	<LOD	<LOD	F
60–79 years							
1 (2007–2009)	956	90.69	—	<LOD	<LOD	<LOD	F
2 (2009–2011)	921	93.08	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	917	92.58	—	<LOD	<LOD	<LOD	4.1[E] (<LOD–6.8)
4 (2014–2015)	874	93.14	—	<LOD	<LOD	<LOD	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.
b Data not available as participants under the age of 6 years were not included in cycle 1 (2007–2009).
E Use data with caution.
F Data is too unreliable to be published.

Table 11.1.3

Cotinine (smokers) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
1 (2007–2009)	805	4.22	590 (420–820)	F	1000 (810–1200)	2200 (2000–2400)	2600 (2300–2900)
2 (2009–2011)	819	5.74	490 (340–700)	F	1000 (810–1200)	2200 (1900–2500)	2600 (2100–3100)
3 (2012–2013)	701	5.14	490 (410–590)	F	990 (900–1100)	2000 (1600–2300)	2300 (2000–2600)
4 (2014–2015)	667	6.00	550 (420–710)	F	1000 (830–1200)	2300 (1900–2700)	2800 (2400–3200)
Males, 12–79 years							
1 (2007–2009)	406	4.43	660[E] (400–1100)	F	1200 (920–1500)	2300 (2000–2600)	2800 (2400–3300)
2 (2009–2011)	425	4.47	470[E] (280–770)	F	1000 (780–1200)	2300 (1900–2700)	2900 (2300–3500)
3 (2012–2013)	387	5.17	460 (340–630)	F	990 (820–1100)	2100 (1700–2500)	2400 (2100–2600)
4 (2014–2015)	359	4.46	610 (470–800)	F	980 (830–1100)	2200 (1800–2500)	2600 (1800–3400)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 12–79 years							
1 (2007–2009)	399	4.01	520 (390–700)	F	860 (640–1100)	2100 (1900–2300)	2500 (2300–2700)
2 (2009–2011)	394	7.11	510[E] (320–810)	F	1000 (720–1300)	2100 (1800–2400)	2400 (1900–2900)
3 (2012–2013)	314	5.10	550 (380–790)	F	990 (760–1200)	1700 (1200–2300)	2100 (1700–2500)
4 (2014–2015)	308	7.79	470[E] (250–870)	F	1100 (820–1400)	2500 (1900–3100)	2800 (2500–3100)
12–19 years							
1 (2007–2009)	102	10.78	160[E] (78–330)	<LOD	F	1600 (1400–1900)	x
2 (2009–2011)	102	11.76	F	<LOD	F	1700 (1200–2300)	x
3 (2012–2013)	98	14.29	F	x	F	x	x
4 (2014–2015)	73	19.18	F	x	430[E] (260–610)	x	x
20–39 years							
1 (2007–2009)	300	3.00	500[E] (300–850)	F	930 (620–1200)	2000 (1800–2200)	2500 (2100–2900)
2 (2009–2011)	311	9.00	400[E] (260–630)	F	850 (570–1100)	2200 (1600–2900)	2900 (2200–3600)
3 (2012–2013)	254	5.12	310[E] (190–520)	F	700[E] (350–1100)	1600 (1300–1900)	2000 (1600–2400)
4 (2014–2015)	271	6.27	360[E] (220–600)	F	970 (620–1300)	2400 (1600–3200)	2900 (2200–3500)
40–59 years							
1 (2007–2009)	275	3.27	830 (610–1100)	F	1200 (910–1500)	2500 (2200–2800)	2800 (2400–3100)
2 (2009–2011)	253	1.58	800[E] (480–1300)	F	1400 (1000–1700)	2200 (1900–2600)	2600 (2000–3300)
3 (2012–2013)	228	2.63	770 (550–1100)	340[E] (150–530)	1000 (890–1200)	2100 (1700–2600)	2300 (2000–2700)
4 (2014–2015)	208	2.88	880 (770–1000)	360[E] (190–540)	1100 (870–1400)	2600 (1900–3200)	2900 (2400–3300)
60–79 years							
1 (2007–2009)	128	3.91	650[E] (430–980)	F	860 (600–1100)	2200 (1900–2400)	x
2 (2009–2011)	153	1.96	F	F	980 (720–1200)	1800 (1500–2000)	x
3 (2012–2013)	121	2.48	940 (800–1100)	390[E] (240–540)	990 (830–1200)	2100 (1400–2700)	x
4 (2014–2015)	115	2.61	920 (720–1200)	440[E] (250–630)	990[E] (620–1400)	1900 (1500–2200)	x

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

x Suppressed to meet the confidentiality requirements of the *Statistics Act*.

Table 11.1.4

Cotinine (smokers) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
1 (2007–2009)	803	4.22	650 (480–890)	F	1000 (830–1200)	3000 (2500–3500)	4400 (3500–5300)
2 (2009–2011)	816	5.74	430[E] (290–630)	F	840 (620–1100)	2700 (1800–3700)	3800[E] (2300–5300)
3 (2012–2013)	701	5.14	440 (340–570)	F	750 (590–900)	2600[E] (1600–3700)	3900[E] (2100–5800)
4 (2014–2015)	666	6.00	480 (360–630)	F	780 (650–910)	2500 (1700–3300)	3300 (2900–3700)
Males, 12–79 years							
1 (2007–2009)	405	4.43	560[E] (360–880)	F	930 (680–1200)	2300 (1900–2700)	3200 (2300–4200)
2 (2009–2011)	425	4.47	370[E] (210–620)	F	730 (480–980)	2700[E] (1600–3700)	3700[E] (2300–5100)
3 (2012–2013)	387	5.17	360[E] (250–520)	F	710 (500–920)	2300 (1500–3100)	3000[E] (1900–4100)
4 (2014–2015)	358	4.46	500 (410–610)	F	770 (630–900)	2900[E] (1600–4200)	3300 (2500–4200)
Females, 12–79 years							
1 (2007–2009)	398	4.01	780 (590–1000)	F	1100 (900–1400)	3700 (2900–4500)	5500 (4300–6600)
2 (2009–2011)	391	7.11	520[E] (300–890)	F	1000 (650–1400)	F	4800[E] (2300–7400)
3 (2012–2013)	314	5.10	600 (420–850)	F	860[E] (510–1200)	3200[E] (1000–5300)	4900 (3300–6400)
4 (2014–2015)	308	7.79	450[E] (240–850)	F	830[E] (440–1200)	2500 (1800–3100)	F
12–19 years							
1 (2007–2009)	102	10.78	120[E] (58–250)	<LOD	290[E] (<LOD–470)	1400[E] (600–2200)	x
2 (2009–2011)	102	11.76	F	<LOD	F	1300 (990–1500)	x
3 (2012–2013)	98	14.29	F	x	F	x	x
4 (2014–2015)	72	19.18	F	x	F	x	x
20–39 years							
1 (2007–2009)	299	3.00	510[E] (310–840)	F	850 (560–1100)	2200 (1900–2600)	2500 (1900–3000)
2 (2009–2011)	311	9.00	330[E] (200–530)	F	710 (470–940)	2300 (1500–3000)	3200[E] (1700–4700)
3 (2012–2013)	254	5.12	230[E] (120–410)	F	520[E] (310–720)	1500[E] (830–2200)	2100[E] (1300–2900)
4 (2014–2015)	271	6.27	300[E] (170–520)	F	600 (390–800)	2300[E] (1200–3400)	3200 (2300–4200)
40–59 years							
1 (2007–2009)	275	3.27	1000 (810–1300)	F	1300 (920–1600)	4100 (2900–5400)	5500 (4400–6600)
2 (2009–2011)	251	1.58	710[E] (400–1200)	F	990[E] (560–1400)	3400[E] (1400–5400)	4900[E] (2800–7000)
3 (2012–2013)	228	2.63	840[E] (520–1300)	390[E] (190–580)	940[E] (570–1300)	3500[E] (1500–5500)	5200[E] (2500–7800)
4 (2014–2015)	208	2.88	780 (610–1000)	210[E] (120–300)	1000 (740–1300)	3000 (2200–3700)	3300 (2700–4000)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	127	3.91	840[E] (530–1300)	F	1300 (1000–1500)	3200 (2100–4300)	x
2 (2009–2011)	152	1.96	F	F	1000 (700–1400)	3000[E] (1700–4300)	x
3 (2012–2013)	121	2.48	960 (730–1200)	390 (270–500)	960[E] (530–1400)	3100[E] (1600–4700)	x
4 (2014–2015)	115	2.61	980 (780–1200)	400[E] (250–560)	1100 (820–1400)	2100 (1700–2500)	x

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

x Suppressed to meet the confidentiality requirements of the *Statistics Act*.

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SUMMARIES AND RESULTS FOR ORGANOPHOSPHATE PESTICIDE METABOLITES 12

12.1 CHLORPYRIFOS METABOLITE

The chemical 3,5,6-trichloro-2-pyridinol (TCPy; CASRN 6515-38-4) is a metabolite of chlorpyrifos and chlorpyrifos-methyl, both organophosphate insecticides, and triclopyr, a carboxylic acid herbicide. Chlorpyrifos was first introduced in 1965 to control pests associated with turfgrass, ornamentals, and indoor environments (CCME, 2008). It is also used in agricultural applications to control insects on food crops such as grains, fruit, nuts, and vegetables. The use of chlorpyrifos in Canada has changed drastically since the late 1990s owing to the discontinued use of most residential/homeowner applications and commercial applications in and around residential areas (CCME, 2008). Chlorpyrifos is currently used in agricultural, non-agricultural (e.g. structural), forestry and residential settings, as well as for mosquito control (Health Canada, 2007). There are over 25 chlorpyrifos-containing acaricide and insecticide products registered in Canada (Health Canada, 2016a). Chlorpyrifos-methyl has not been registered for use in Canada, but is used agriculturally in other countries around the world (CDC, 2009; Health Canada, 2016a). Triclopyr is a selective herbicide used to kill unwanted broadleaf plants in agricultural (non-food areas) and forest environments. There are 19 products currently registered in Canada that contain the triclopyr ester and amine salt formulations (Health Canada, 2016a). However, in humans, triclopyr is excreted almost completely unchanged in the urine, indicating that TCPy is not a useful biomarker of exposure to triclopyr (Carmichael et al., 1989). Therefore, this summary will focus only on TCPy as a biomarker of exposure to chlorpyrifos and chlorpyrifos-methyl.

Chlorpyrifos binds tightly to soil particles and is not expected to leach significantly (ATSDR, 1997). Dispersion generally occurs via volatilization from moist soils or surface waters. Chlorpyrifos is moderately persistent in terrestrial and aquatic environments, with a half-life ranging from less than 1 week to more than 24 weeks (Eisler, 2000; Jarvinen and Tanner, 1982). In water and soil, TCPy is one of the major transformation products of chlorpyrifos (CCME, 2008).

The primary routes of exposure are oral, dermal, and inhalation through ingestion of food and drinking water, handling chlorpyrifos-containing pesticide products, and environmental exposure in insecticide-treated areas (ATSDR, 1997). Following entry into the body, chlorpyrifos is rapidly absorbed, metabolized, and excreted, predominantly in urine and to a lesser degree in feces (FAO/WHO, 2000). Accumulation in tissues is low owing to an elimination half-life of 27 hours (Nolan et al., 1984). Chlorpyrifos is rapidly metabolized by oxidative desulfuration and hydrolysis to form TCPy and dialkyl phosphate metabolites (diethylphosphate and diethylthiophosphate) (ATSDR, 1997). Diethylphosphate and diethylthiophosphate are semi-specific organophosphate pesticide metabolites associated with several pesticides (e.g. coumaphos, diazon, phorate, phosalone, terbufos) in addition to chlorpyrifos. TCPy is a more specific metabolite and is the major chlorpyrifos metabolic product. TCPy can be found in blood and urine (ATSDR, 1997). Urine is the principal route of excretion for TCPy and levels in

urine are correlated with the degree of recent (within 48 hours) exposure to chlorpyrifos (ATSDR, 1997). The presence of TCPy in urine may also be the result of exposure to chlorpyrifos-methyl. In the environment, TCPy can also be found as a result of the breakdown of parent compounds. As TCPy is more persistent than its parent compounds in the environment, urinary levels may reflect direct environmental exposure to the metabolite (EPA, 2006). TCPy and the dialkly phosphate metabolites are considered to be less toxic than their parents, chlorpyrifos and chlorpyrifos-methyl.

Chlorpyrifos and chlorpyrifos-methyl, as with other organophosphates pesticides, are cholinesterase inhibitors that act on the nervous system of insects and mammals by interfering with the transmission of nerve impulses (EPA, 1999). Failure to properly metabolize acetylcholine results in an overstimulation of the nervous system. Symptoms of acute overexposure may include headache, dizziness, fatigue, irritation of the eyes or nose, nausea, vomiting, salivation, sweating, and changes in heart rate. Organophosphate-induced delayed polyneuropathy has also been observed in humans following acute-duration exposure to chlorpyrifos (ATSDR, 1997). Very high exposures can have effects such as paralysis, seizures, loss of consciousness, or even death (ATSDR, 1997). However, typical exposure through the ingestion of chlorpyrifos in food is estimated to be low (ATSDR, 1997). Further, there is a growing body of evidence of adverse neurodevelopmental outcomes in infants and children following low-dose gestational and/or early postnatal exposure to chlorpyrifos (EPA, 2016). Chlorpyrifos is not considered to be mutagenic or carcinogenic (EPA, 2006; Health Canada, 1986; NTP, 1992).

The sale, use, and maximum food residue limits (MRLs) of chlorpyrifos are regulated in Canada by Health Canada's Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2006). PMRA evaluates the toxicity of pesticides and potential exposure to determine whether a pesticide should be registered for a specific use. As part of the registration process, PMRA establishes MRLs of pesticides in food, including chlorpyrifos (Health Canada, 2016b). In 1999, PMRA commenced a re-evaluation of the 27 organophosphate pesticides, including chlorpyrifos, that were registered for use at that time in Canada (Health Canada, 1999). The re-evaluation of chlorpyrifos has been carried out in three phases. The first phase comprised a phase-out of most residential uses, discontinued use on tomatoes, and lowered MRLs for residues in imported apples and grapes (Health Canada, 2000). The second phase focused on the remaining agricultural and forestry uses of chlorpyrifos (Health Canada, 2007). The third phase will identify and implement, where warranted, measures to reduce environmental exposures (Health Canada, 2007).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for chlorpyrifos in drinking water using an acceptable daily intake for chlorpyrifos derived by the Food and Agriculture Organization of the United Nations and the World Health Organization (FAO/WHO, 1982; Health Canada, 1986). Chlorpyrifos has also been analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply.

TCPy was analyzed in the urine of Canadian Health Measures Survey participants aged 3–79 years in cycle 4 (2014–2015). Data are presented as µg/L and µg/g creatinine. TCPy was also measured in cycle 3 (2012–2013); however, the data are not yet available because of ongoing quality assurance confirmation of the biospecimen analysis. Finding a measurable amount of TCPy in urine is an indicator of exposure to chlorpyrifos or chlorpyrifos-methyl and does not necessarily mean that an adverse health effect will occur.

Table 12.1.1

3,5,6-Trichloro-2-pyridinol — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2422	1.28	1.4 (1.2–1.5)	0.37 (0.30–0.44)	1.3 (1.1–1.4)	5.7 (4.4–7.0)	9.3 (6.5–12)
Males, 3–79 years							
4 (2014–2015)	1209	1.41	1.5 (1.3–1.8)	0.47 (0.31–0.63)	1.4 (1.3–1.6)	6.4[E] (4.0–8.8)	9.9[E] (5.0–15)
Females, 3–79 years							
4 (2014–2015)	1213	1.15	1.2 (1.0–1.4)	0.31 (0.23–0.40)	1.1 (0.94–1.3)	5.2 (3.7–6.7)	7.8 (5.3–10)
3–5 years							
4 (2014–2015)	479	1.67	1.3 (1.1–1.5)	0.39 (0.28–0.49)	1.1 (0.81–1.4)	4.4 (3.4–5.4)	7.3[E] (4.5–10)
6–11 years							
4 (2014–2015)	489	0.41	1.6 (1.3–2.1)	0.45 (0.36–0.53)	1.4 (1.0–1.8)	6.3 (4.3–8.4)	F
12–19 years							
4 (2014–2015)	478	0	1.5 (1.3–1.7)	0.35[E] (0.19–0.50)	1.2 (0.99–1.5)	7.0 (4.4–9.6)	11[E] (6.3–15)
20–39 years							
4 (2014–2015)	336	1.49	1.3 (1.1–1.5)	0.36 (0.26–0.45)	1.2 (1.0–1.5)	6.0 (4.4–7.6)	8.4 (5.9–11)
40–59 years							
4 (2014–2015)	299	3.01	1.3 (1.1–1.7)	0.39[E] (0.19–0.59)	1.3 (1.0–1.6)	5.0[E] (2.5–7.5)	F
60–79 years							
4 (2014–2015)	341	2.05	1.4 (1.2–1.7)	0.35 (0.22–0.48)	1.2 (1.0–1.5)	6.0[E] (3.5–8.5)	9.7[E] (3.8–16)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 12.1.2

3,5,6-Trichloro-2-pyridinol (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2421	1.28	1.2 (1.1–1.4)	0.40 (0.32–0.49)	1.0 (0.92–1.1)	4.1 (3.4–4.8)	8.0[E] (4.9–11)
Males, 3–79 years							
4 (2014–2015)	1208	1.41	1.2 (1.0–1.4)	0.38 (0.26–0.50)	1.0 (0.91–1.1)	4.3 (3.3–5.3)	9.4[E] (4.3–15)
Females, 3–79 years							
4 (2014–2015)	1213	1.15	1.2 (1.1–1.5)	0.42 (0.34–0.50)	1.0 (0.84–1.2)	3.8 (2.7–4.9)	6.9[E] (3.5–10)
3–5 years							
4 (2014–2015)	479	1.67	2.2 (1.9–2.5)	0.87 (0.72–1.0)	1.9 (1.4–2.3)	6.8 (5.4–8.2)	10 (6.8–13)
6–11 years							
4 (2014–2015)	488	0.41	1.8 (1.4–2.2)	0.58 (0.46–0.70)	1.5 (1.0–1.9)	5.5[E] (3.2–7.7)	F
12–19 years							
4 (2014–2015)	478	0	1.0 (0.93–1.2)	0.34 (0.27–0.42)	0.96 (0.84–1.1)	4.3 (2.8–5.8)	6.6[E] (3.2–9.9)
20–39 years							
4 (2014–2015)	336	1.49	1.1 (0.87–1.3)	0.37 (0.26–0.48)	0.99 (0.73–1.3)	3.3 (2.4–4.2)	4.4 (3.3–5.6)
40–59 years							
4 (2014–2015)	299	3.01	1.2 (0.92–1.5)	0.41[E] (0.26–0.57)	0.96 (0.79–1.1)	F	9.7[E] (<LOD–16)
60–79 years							
4 (2014–2015)	341	2.05	1.4 (1.2–1.6)	0.44 (0.33–0.54)	1.2 (0.96–1.4)	4.5 (3.2–5.8)	7.4[E] (3.7–11)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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12.2 MALATHION METABOLITE

Malathion dicarboxylic acid (CASRN 1190-28-9) is a metabolite of malathion, which is a broad-spectrum organophosphate insecticide that has been registered for use in Canada since 1953 (Health Canada, 2003a). Malathion is registered for the control of crawling and flying insects in agricultural, non-agricultural (e.g. structural), and residential settings, as well as for mosquito abatement (Health Canada, 2012). Malathion is found in 27 acaricide and insecticide products registered in Canada (Health Canada, 2016a).

Malathion binds moderately to soil particles and undergoes significant biodegradation and hydrolysis such that it is generally not expected to leach into groundwater (ATSDR, 2003). Dispersion from moist soils or surface waters generally occurs via volatilization. In air, malathion can be oxidized resulting in the formation of its active metabolite, malaoxon. Malathion is moderately persistent in terrestrial and aquatic environments, staying in the environment from a few days to several months (ATSDR, 2003).

The primary routes of exposure are oral, dermal, and inhalation, and may occur through ingestion of food and drinking water, handling malathion-containing pesticide products and environmental exposure in insecticide-treated areas (ATSDR, 2003). Following ingestion, malathion is rapidly absorbed from the gastrointestinal tract and metabolized in the liver; the metabolites are excreted, predominantly in urine (ATSDR, 2003). Accumulation in tissues is low owing to an elimination half-life of less than 24 hours (Bouchard et al., 2003; Vasilic et al., 1999). Malathion is rapidly metabolized by oxidation, hydrolysis, and the elimination of a methyl group catalyzed by glutathione *S*-transferase (ATSDR, 2003). Malathion metabolism results in the formation of malaoxon, malathion monocarboxylic acid, malathion dicarboxylic acid, dialkyl phosphate metabolites, and other metabolites. Dialkyl phosphate metabolites are semi-specific organophosphate pesticide metabolites associated with several pesticides (e.g. azinophos-methyl, dimethoate, phosmet) in addition to malathion. Malathion dicarboxylic acid is a major urinary metabolite of malathion (ATSDR, 2003). Malathion dicarboxylic acid can also be found in the environment as a result of the breakdown of the parent compound. Both dialkyl phosphate metabolites and malathion dicarboxylic acid are considered to be less toxic than their parent, malathion.

Malathion, as with other organophosphates pesticides, is a cholinesterase inhibitor that acts on the nervous system of insects and mammals by interfering with the transmission of nerve impulses (EPA, 1999). The majority of the systemic effects observed following exposure to malathion are due to the action of its active metabolite, malaoxon, on the nervous system (ATSDR, 2003). Inhibition of acetylcholine metabolism results in an overstimulation of the nervous system. Symptoms of acute overexposure may include headache, dizziness, fatigue, irritation of the eyes or nose, nausea, vomiting, salivation, sweating, and changes in heart rate. Very high exposures can have effects such as difficulty breathing, dizziness, loss of consciousness, or even death (ATSDR, 2003). However, compared with other organophosphate pesticides, malathion has low acute toxicity and Health Canada's Pest Management Regulatory Agency (PMRA) has concluded that risk to health from dietary exposure is not of concern (CDC, 2009; Health Canada, 2012). Human health effects from malathion at low environmental levels are unknown, although some animal studies have suggested that there is a potential for toxic effects resulting from chronic low-dose exposure to organophosphates (Ray and Richards, 2001). Health Canada determined that malathion was unlikely to pose a carcinogenic risk to humans, nor was it found to be genotoxic or teratogenic in animal studies (Health Canada, 2010). More recently, malathion was classified as Group 2A, probably carcinogenic to humans, by the International Agency for Research on Cancer on the basis of evidence in experimental animals (IARC, 2017).

The sale, use, and maximum food residue limits (MRLs) of malathion are regulated in Canada by Health Canada's PMRA under the *Pest Control Products Act* (Canada, 2006). PMRA evaluates the toxicity of pesticides and potential exposure in order to determine whether a pesticide should be registered for a specific use. As part of the registration process, PMRA establishes MRLs of pesticides in food, including malathion (Health Canada, 2016b). In 1999, PMRA commenced a re-evaluation of the 27 organophosphate pesticides, including malathion, that were registered for use at that time in Canada (Health Canada, 1999). The re-evaluation of malathion was carried out in two phases. The first phase comprised an assessment of malathion use as an adulticide in mosquito abatement programs and resulted in mitigation measures including label changes for related end-use products (Health Canada, 2003b). The second phase included all

registered uses and the metabolite malaoxon in the re-evaluation of malathion (Health Canada, 2012). The re-evaluation found that most uses of malathion, including commercial products applied in agricultural, non-agricultural, and residential settings, do not pose unacceptable risks to human health.

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for malathion using an acceptable daily intake derived by the Food and Agriculture Organization of the United Nations and the World Health Organization (FAO/WHO, 1973; Health Canada, 1986). Malathion is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2013). The food items analyzed represent those that are most typical of the Canadian diet, and the surveys are used to provide dietary exposure estimates for chemicals that Canadians in different age-sex groups are exposed to through the food supply.

Malathion dicarboxylic acid was analyzed in the urine of all Canadian Health Measures Survey participants aged 3–79 years in cycle 4 (2014–2015). Data are presented as both µg/L and µg/g creatinine. Malathion dicarboxylic acid was also measured in cycle 3 (2012–2013); however, the data are not yet available because of ongoing quality assurance confirmation of the biospecimen analysis. Finding a measurable amount of malathion dicarboxylic acid in urine is an indicator of exposure to malathion and does not necessarily mean that an adverse health effect will occur.

Table 12.2.1

Malathion dicarboxylic acid — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2519	82.93	—	<LOD	<LOD	0.46[E] (0.23–0.69)	0.95[E] (0.46–1.4)
Males, 3–79 years							
4 (2014–2015)	1257	83.13	—	<LOD	<LOD	0.31[E] (<LOD–0.47)	F
Females, 3–79 years							
4 (2014–2015)	1262	82.73	—	<LOD	<LOD	0.68[E] (0.25–1.1)	1.3[E] (0.58–2.0)
3–5 years							
4 (2014–2015)	499	80.76	—	<LOD	<LOD	0.70 (0.44–0.95)	1.8[E] (0.62–3.0)
6–11 years							
4 (2014–2015)	510	79.41	—	<LOD	<LOD	F	F
12–19 years							
4 (2014–2015)	501	82.83	—	<LOD	<LOD	0.31[E] (<LOD–0.53)	0.89 (0.62–1.2)
20–39 years							
4 (2014–2015)	357	89.08	—	<LOD	<LOD	F	0.78[E] (0.23–1.3)
40–59 years							
4 (2014–2015)	302	83.77	—	<LOD	<LOD	F	F
60–79 years							
4 (2014–2015)	350	84.29	—	<LOD	<LOD	F	1.4[E] (0.52–2.3)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 12.2.2

Malathion dicarboxylic acid (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2518	82.93	—	<LOD	<LOD	0.42 (0.37–0.48)	0.95[E] (0.40–1.5)
Males, 3–79 years							
4 (2014–2015)	1256	83.13	—	<LOD	<LOD	0.35 (<LOD–0.45)	F
Females, 3–79 years							
4 (2014–2015)	1262	82.73	—	<LOD	<LOD	0.46 (0.31–0.61)	F
3–5 years							
4 (2014–2015)	499	80.76	—	<LOD	<LOD	1.1[E] (0.68–1.5)	F
6–11 years							
4 (2014–2015)	509	79.41	—	<LOD	<LOD	F	2.1[E] (0.87–3.2)
12–19 years							
4 (2014–2015)	501	82.83	—	<LOD	<LOD	0.32 (<LOD–0.42)	0.48 (0.31–0.65)
20–39 years							
4 (2014–2015)	357	89.08	—	<LOD	<LOD	0.32 (<LOD–0.42)	0.50[E] (0.19–0.80)
40–59 years							
4 (2014–2015)	302	83.77	—	<LOD	<LOD	0.42[E] (<LOD–0.64)	F
60–79 years							
4 (2014–2015)	350	84.29	—	<LOD	<LOD	F	1.7[E] (0.82–2.6)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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SUMMARY AND RESULTS FOR PARABENS 13

13.1 PARABENS

Parabens are a group of *para*-hydroxybenzoic (*p*-hydroxybenzoic) acid esters, four of which were measured in cycle 4 of the Canadian Health Measures Survey (CHMS): methyl, ethyl, propyl, and butyl paraben.

Table 13.1.1
Parabens measured in the Canadian Health Measures Survey cycle 4 (2014–2015).

Paraben	CASRN
Methyl paraben	99-76-3
Ethyl paraben	120-47-8
Propyl paraben	94-13-3
Butyl paraben	94-26-8

Parabens are widely used as preservatives in cosmetic and personal care products owing to their antibacterial and antifungal properties (Health Canada, 2016a). These products include makeup, moisturizers, sunscreens, hair-care products, facial and skin cleansers, and shaving products. Methyl, propyl, and butyl paraben are the most common ones used in cosmetic products (FDA, 2007). Typical concentrations of parabens in cosmetic products are generally 0.3% or less (Health Canada, 2016a). Parabens are also used as preservatives in food products, beverages, and pharmaceutical drugs (Ye et al., 2008). Propyl paraben and butyl paraben are classified as natural health products (NHPs) as they can be used as a source of *p*-hydroxybenzoic acid, a major metabolite of parabens (Health Canada, 2016b).

Although parabens in commercial use are synthetically produced, some parabens also occur naturally as preservatives in certain fruits and vegetables, such as blueberries and carrots (Health Canada, 2016a). Production and use of paraben-containing products can result in their release to the environment through various waste streams. A potential route of exposure for the general public is dermal contact with products such as moisturizers and cosmetics that contain parabens. Approximately 50% of cosmetics in the United States contain parabens with methylparaben being the most commonly used and lipstick having the highest concentrations (Cosmetic Ingredient Review Expert Panel, 2008; Yazar et al., 2011). Oral exposure to parabens can also occur via consumption of foods or pharmaceuticals containing parabens, ingestion of breast milk, and ingestion of house dust (CDC, 2009; Fan et al., 2010; Ye et al., 2008).

Dermal exposure may result in small amounts of parabens being absorbed. Following oral exposure, parabens are rapidly absorbed from the gastrointestinal tract (NTP, 2005). Once absorbed, parabens are mainly hydrolyzed to *p*-hydroxybenzoic acid that can then be conjugated with glycine, glucuronide, and sulphate for excretion in urine (Soni et al., 2005). Currently, there is no evidence of bioaccumulation potential in humans. In laboratory animals, complete elimination of orally ingested ethyl and propyl paraben was observed within 72 hours (Soni et al., 2005). Data are limited in humans, with one study of premature infants

observing excretion, primarily in the conjugated form, of approximately 10% to 90% of a methyl paraben dose (Hindmarsh et al., 1983). A recent study of parabens in urine of 100 adults found that parabens in urine appear predominantly in their conjugated forms (Ye et al., 2006). The concentration of parabens in urine (conjugated and free) can be used as a biomarker of exposure to parabens. As *p*-hydroxybenzoic acid is a nonspecific metabolite of all parabens, it may not be an optimal biomarker of exposure for specific parabens.

Health effects from low-level exposures to parabens are unknown; no acute, subchronic, or chronic toxicity has been observed (Cosmetic Ingredient Review Expert Panel, 2008). Animal studies have found parabens to be non-allergenic, however, sporadic human cases of anaphylactic reactions have been reported following paraben exposure. Parabens have been found to weakly mimic estrogens in animal studies. However, safety assessments of maximum estimated paraben exposure have concluded that estrogenic effects are unlikely in humans. Parabens have not been found to be animal carcinogens and neither Health Canada nor the International Agency for Research on Cancer has evaluated parabens with respect to human carcinogenicity.

Methyl, ethyl, propyl, and butyl paraben were included in the categorization of the Domestic Substances List carried out under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Canada, 2007). None of the four parabens was categorized as a priority substance for future assessment based on environmental or human health criteria; however, a screening level risk assessment is planned (Environment Canada, 2013). Health Canada has reviewed the current available evidence on risk posed by parabens when used in cosmetics and does not have any health or safety concerns with these ingredients in their present practices of use (Health Canada, 2016a). Health Canada continues to monitor and review any new scientific data on parabens (Health Canada, 2016a).

Parabens were measured in a 2011 biomonitoring study carried out in Alberta with 39 participants aged 12–67 years who were patients at a primary care clinic specializing in environmental health sciences (Genuis et al., 2013). The 50th percentile urinary concentrations measured in this study were 25.95, 10.30, 2.80, and 0.32 µg/L for methyl, ethyl, propyl, and butyl paraben, respectively.

Methyl, ethyl, propyl, and butyl paraben were analyzed in the urine of Canadian Health Measures Survey participants aged 3–79 years in cycle 4 (2014–2015). Data are presented as µg/L and µg/g creatinine. Parabens were also measured in cycle 3 (2012–2013); however, the data are not yet available because of ongoing quality assurance confirmation of the biospecimen analysis. Finding a measurable amount of parabens in urine is an indicator of exposure to parabens and does not necessarily mean that an adverse health effect will occur.

Table 13.1.2

Methyl paraben — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2564	8.39	17 (13–22)	<LOD	15 (9.8–20)	270 (190–340)	490 (340–640)
Males, 3–79 years							
4 (2014–2015)	1275	10.51	9.4 (6.9–13)	<LOD	6.8[E] (4.2–9.4)	F	F
Females, 3–79 years							
4 (2014–2015)	1289	6.28	30 (21–43)	1.8 (1.3–2.4)	F	310[E] (170–440)	510[E] (170–850)
3–5 years							
4 (2014–2015)	511	6.26	12 (9.3–15)	1.9[E] (<LOD–2.7)	8.0 (5.9–10)	110[E] (50–170)	330[E] (110–560)
6–11 years							
4 (2014–2015)	514	9.73	7.6 (6.4–9.1)	1.4 (<LOD–1.8)	6.1 (4.0–8.2)	43 (30–57)	F
12–19 years							
4 (2014–2015)	505	8.12	14[E] (9.1–21)	<LOD	9.7 (6.4–13)	300[E] (130–470)	520[E] (250–780)
20–39 years							
4 (2014–2015)	362	7.18	16[E] (9.3–28)	1.3[E] (<LOD–1.9)	F	300[E] (170–430)	390[E] (180–610)
40–59 years							
4 (2014–2015)	312	11.22	21[E] (11–38)	<LOD	F	270[E] (93–440)	550[E] (250–860)
60–79 years							
4 (2014–2015)	360	8.61	20 (16–26)	1.4[E] (<LOD–2.0)	22[E] (8.2–36)	F	680[E] (210–1200)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.3

Methyl paraben (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2563	8.39	15 (11–21)	<LOD	13[E] (6.5–19)	230 (180–290)	340 (230–440)
Males, 3–79 years							
4 (2014–2015)	1274	10.51	7.4 (5.4–10)	<LOD	5.3[E] (3.3–7.2)	99[E] (<LOD–150)	230[E] (130–340)
Females, 3–79 years							
4 (2014–2015)	1289	6.28	31[E] (21–46)	2.1 (1.5–2.7)	37[E] (17–56)	290[E] (180–400)	480[E] (250–700)
3–5 years							
4 (2014–2015)	511	6.26	21 (16–27)	3.7 (<LOD–4.6)	13[E] (8.2–19)	210[E] (72–360)	430[E] (200–660)
6–11 years							
4 (2014–2015)	513	9.73	8.4 (7.1–9.8)	1.8 (<LOD–2.2)	7.1 (5.3–8.8)	41 (30–52)	F
12–19 years							
4 (2014–2015)	505	8.12	9.9[E] (6.7–15)	<LOD	7.2 (4.9–9.5)	180[E] (66–290)	370[E] (100–640)
20–39 years							
4 (2014–2015)	362	7.18	13[E] (6.9–25)	0.90[E] (<LOD–1.4)	F	230 (150–310)	280[E] (94–460)
40–59 years							
4 (2014–2015)	312	11.22	19[E] (10–35)	<LOD	F	250[E] (140–370)	310[E] (130–490)
60–79 years							
4 (2014–2015)	360	8.61	20 (16–23)	1.2[E] (<LOD–1.7)	22[E] (13–31)	320[E] (<LOD–510)	620[E] (340–890)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.4

Ethyl paraben — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2564	64.86	—	<LOD	<LOD	27[E] (14–39)	73[E] (33–110)
Males, 3–79 years							
4 (2014–2015)	1275	71.69	—	<LOD	<LOD	11 (6.9–14)	F
Females, 3–79 years							
4 (2014–2015)	1289	58.11	—	<LOD	<LOD	39[E] (14–64)	F
3–5 years							
4 (2014–2015)	511	65.95	—	<LOD	<LOD	F	F
6–11 years							
4 (2014–2015)	514	78.79	—	<LOD	<LOD	2.0[E] (1.1–2.9)	3.4[E] (1.3–5.5)
12–19 years							
4 (2014–2015)	505	68.91	—	<LOD	<LOD	F	28[E] (11–45)
20–39 years							
4 (2014–2015)	362	54.42	—	<LOD	<LOD	F	F
40–59 years							
4 (2014–2015)	312	52.56	—	<LOD	<LOD	F	98[E] (44–150)
60–79 years							
4 (2014–2015)	360	58.89	—	<LOD	<LOD	38[E] (22–55)	78[E] (44–110)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.5

Ethyl paraben (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2563	64.86	—	<LOD	<LOD	25[E] (8.9–42)	59[E] (23–95)
Males, 3–79 years							
4 (2014–2015)	1274	71.69	—	<LOD	<LOD	6.5 (4.4–8.6)	F
Females, 3–79 years							
4 (2014–2015)	1289	58.11	—	<LOD	<LOD	54[E] (22–86)	120[E] (54–190)
3–5 years							
4 (2014–2015)	511	65.95	—	<LOD	<LOD	F	F
6–11 years							
4 (2014–2015)	513	78.79	—	<LOD	<LOD	2.0[E] (1.2–2.8)	4.6[E] (2.2–7.1)
12–19 years							
4 (2014–2015)	505	68.91	—	<LOD	<LOD	F	F
20–39 years							
4 (2014–2015)	362	54.42	—	<LOD	<LOD	F	F
40–59 years							
4 (2014–2015)	312	52.56	—	<LOD	<LOD	41[E] (<LOD–70)	F
60–79 years							
4 (2014–2015)	360	58.89	—	<LOD	<LOD	44[E] (26–62)	70[E] (29–110)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.6

Propyl paraben — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2564	20.83	2.5 (1.8–3.5)	<LOD	2.0[E] (1.2–2.7)	59[E] (34–85)	130[E] (67–180)
Males, 3–79 years							
4 (2014–2015)	1275	26.51	1.3 (0.96–1.8)	<LOD	0.77 (0.55–0.99)	F	F
Females, 3–79 years							
4 (2014–2015)	1289	15.21	4.9[E] (3.2–7.6)	<LOD	5.6[E] (1.9–9.4)	83[E] (38–130)	170[E] (58–280)
3–5 years							
4 (2014–2015)	511	20.16	1.5 (1.1–2.0)	<LOD	1.2[E] (0.67–1.7)	16[E] (7.3–24)	F
6–11 years							
4 (2014–2015)	514	22.76	1.2 (0.99–1.6)	<LOD	0.95[E] (0.58–1.3)	11 (7.8–14)	F
12–19 years							
4 (2014–2015)	505	18.42	2.3[E] (1.6–3.3)	<LOD	1.8[E] (1.1–2.4)	55[E] (17–92)	110[E] (56–170)
20–39 years							
4 (2014–2015)	362	17.68	F	<LOD	F	F	F
40–59 years							
4 (2014–2015)	312	19.87	F	<LOD	F	F	F
60–79 years							
4 (2014–2015)	360	26.39	3.0[E] (2.0–4.6)	<LOD	F	F	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.7

Propyl paraben (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2563	20.83	2.3[E] (1.6–3.3)	<LOD	1.5[E] (0.85–2.1)	63[E] (30–96)	110 (73–140)
Males, 3–79 years							
4 (2014–2015)	1274	26.51	1.0 (0.74–1.4)	<LOD	0.70 (0.46–0.93)	F	F
Females, 3–79 years							
4 (2014–2015)	1289	15.21	5.1[E] (3.0–8.5)	<LOD	F	87[E] (51–120)	160[E] (91–230)
3–5 years							
4 (2014–2015)	511	20.16	2.6 (2.0–3.4)	<LOD	1.8[E] (1.0–2.6)	30[E] (17–43)	68[E] (20–120)
6–11 years							
4 (2014–2015)	513	22.76	1.4 (1.1–1.7)	<LOD	1.1 (0.74–1.4)	9.1 (6.4–12)	F
12–19 years							
4 (2014–2015)	505	18.42	1.7 (1.2–2.4)	<LOD	1.1[E] (0.64–1.6)	F	85[E] (42–130)
20–39 years							
4 (2014–2015)	362	17.68	F	<LOD	F	F	F
40–59 years							
4 (2014–2015)	312	19.87	F	<LOD	F	F	96[E] (29–160)
60–79 years							
4 (2014–2015)	360	26.39	2.9[E] (2.0–4.2)	<LOD	2.6[E] (0.95–4.2)	F	190[E] (110–280)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.8

Butyl paraben — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2564	83.15	—	<LOD	<LOD	F	4.3[E] (2.0–6.6)
Males, 3–79 years							
4 (2014–2015)	1275	90.12	—	<LOD	<LOD	<LOD	F
Females, 3–79 years							
4 (2014–2015)	1289	76.26	—	<LOD	<LOD	F	F
3–5 years							
4 (2014–2015)	511	83.37	—	<LOD	<LOD	F	F
6–11 years							
4 (2014–2015)	514	90.08	—	<LOD	<LOD	<LOD	1.1[E] (0.30–1.8)
12–19 years							
4 (2014–2015)	505	80.40	—	<LOD	<LOD	F	F
20–39 years							
4 (2014–2015)	362	81.77	—	<LOD	<LOD	F	F
40–59 years							
4 (2014–2015)	312	81.09	—	<LOD	<LOD	F	F
60–79 years							
4 (2014–2015)	360	80.00	—	<LOD	<LOD	F	6.8 (4.4–9.1)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 13.1.9

Butyl paraben (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
4 (2014–2015)	2563	83.15	—	<LOD	<LOD	F	4.2[E] (1.5–6.8)
Males, 3–79 years							
4 (2014–2015)	1274	90.12	—	<LOD	<LOD	<LOD	0.79[E] (<LOD–1.2)
Females, 3–79 years							
4 (2014–2015)	1289	76.26	—	<LOD	<LOD	F	9.2[E] (<LOD–15)
3–5 years							
4 (2014–2015)	511	83.37	—	<LOD	<LOD	F	3.1[E] (<LOD–5.1)
6–11 years							
4 (2014–2015)	513	90.08	—	<LOD	<LOD	<LOD	0.81[E] (0.30–1.3)
12–19 years							
4 (2014–2015)	505	80.40	—	<LOD	<LOD	F	F
20–39 years							
4 (2014–2015)	362	81.77	—	<LOD	<LOD	F	F
40–59 years							
4 (2014–2015)	312	81.09	—	<LOD	<LOD	F	F
60–79 years							
4 (2014–2015)	360	80.00	—	<LOD	<LOD	4.2[E] (<LOD–6.5)	6.7[E] (2.1–11)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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SUMMARIES AND RESULTS FOR POLYCYCLIC AROMATIC HYDROCARBON METABOLITES 14

14.1 OVERVIEW

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds characterized by the presence of two or more fused aromatic rings. The United States Environmental Protection Agency has prioritized 16 PAHs because of their potential human health and/or ecological toxicity (EPA, 2013). Table 14.1.1 lists seven of these priority PAHs and their metabolites measured in cycle 4 of the Canadian Health Measures Survey (CHMS).

Table 14.1.1
Hydroxylated polycyclic aromatic hydrocarbon (PAH) metabolites measured in the Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), cycle 4 (2014–2015) and their parent PAH compounds.

PAH	CASRN	Hydroxylated PAH metabolites	CASRN
Benzo[*a*]pyrene	50-32-8	3-Hydroxybenzo[a]pyrene	13345-21-6
Chrysene	218-01-9	2-Hydroxychrysene	65945-06-4
		3-Hydroxychrysene	63019-39-6
		4-Hydroxychrysene	63019-40-9
		6-Hydroxychrysene	37515-51-8
Fluoranthene	206-44-0	3-Hydroxyfluoranthene	17798-09-3
Fluorene	86-73-7	2-Hydroxyfluorene	2443-58-5
		3-Hydroxyfluorene	6344-67-8
		9-Hydroxyfluorene	1689-64-1
Naphthalene	91-20-3	1-Hydroxynaphthalene	90-15-3
		2-Hydroxynaphthalene	135-19-3
Phenanthrene	85-01-8	1-Hydroxyphenanthrene	2433-56-9
		2-Hydroxyphenanthrene	605-55-0
		3-Hydroxyphenanthrene	605-87-8
		4-Hydroxyphenanthrene	7651-86-7
		9-Hydroxyphenanthrene	484-17-3
Pyrene	129-00-0	1-Hydroxypyrene	5315-79-7

PAHs are released to the environment from both natural and anthropogenic sources; the contribution from anthropogenic sources is substantially higher than from natural sources (ATSDR, 1995). In Canada, forest fires are the largest natural source of PAHs in the environment (Environment Canada, 2010). PAHs also have been measured in emissions from crude oil, coal, and volcanoes. Anthropogenic PAH emissions are predominantly due to the incomplete combustion of organic substances from residential wood combustion, industrial emissions, as well as from waste incineration, tobacco smoke, cooking, vehicle exhaust, oil spills, and the use of creosote-treated products (ATSDR, 1995; ATDSR 2005; Environment Canada and Health Canada, 1994; Environment and Climate Change Canada, 2016).

For the general population, the major routes of exposure to PAHs are ingestion and inhalation from sources including diet, smoking, and ambient and indoor air (IARC, 2010; WHO, 2011). Levels in food depend on the source of the food and the method of cooking (ATSDR, 1995). PAHs can be formed when food is charbroiled, grilled, roasted, fried, or baked. Drinking water is considered to be a negligible source of exposure in Canada (Environment Canada and Health Canada, 1994). Vehicle exhaust, tobacco smoke, emissions from wood and charcoal-fired stoves, house dust, and ambient air all contribute to inhalation exposure. Human exposure to PAHs may also occur through skin contact with soot and tars (ATSDR, 1995).

PAHs can be absorbed following inhalation, oral, and dermal exposure. They undergo multi-step metabolism leading to several types of metabolites, including hydroxylated PAHs (Strickland et al., 1996). Elimination occurs through urine and feces, with urinary hydroxylated PAH metabolites observed within a few days of exposure (Viau et al., 1995). These metabolites are excreted both in the free form and as glucuronic acid and sulphate conjugates (Castaño-Vinyals et al., 2004).

Several approaches exist to assess human exposure to PAHs. The analysis of urinary hydroxylated PAH metabolites is the most common approach, and has been used in several biomonitoring studies (Becker et al., 2003; CDC, 2009).

Evaluating health effects of exposure to individual PAH species in humans is difficult because exposure is generally to PAH mixtures. Studies in laboratory animals have shown that several PAHs are carcinogenic, mutagenic, and/or teratogenic (IARC, 2010; IARC, 2012). The carcinogenic potency of PAHs differs among PAH species, and can differ across routes of exposure (ATSDR, 1995). For some PAHs, formation of epoxides through metabolic activation is considered a key step in eliciting carcinogenic effects (Guillén and Sopelana, 2003).

Benzo[*a*]pyrene, PAH-containing mixtures such as soot and coal tar, as well as occupational PAH exposures during selected industrial activities (i.e. coal-tar distillation, coal gasification, coke production, aluminium production) have recently been classified as carcinogenic to humans (Group 1) by the International Agency for Research on Cancer (IARC) (IARC, 2012). However, IARC has also classified some PAHs, such as chrysene and naphthalene, as possibly carcinogenic to humans (Group 2B) and others, such as fluoranthene, fluorene, phenanthrene, and pyrene, as not classifiable as to their carcinogenicity to humans (Group 3) (IARC, 2010). PAHs also elicit adverse immunologic effects, developmental and reproductive effects, as well as hepatic and renal effects in laboratory animals, but generally at doses higher than those that elicit a carcinogenic response (ATSDR, 1995).

In Canada, PAHs are listed as toxic substances on Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), based on an evaluation of the environmental and health effects of several PAHs, including benzo[*a*]pyrene (Canada, 1999; Canada, 2000; Environment Canada and Health Canada, 1994). Several environmental performance agreements, codes of practice, and recommendations have been established to reduce releases of PAHs to the environment from the aluminum production, metal refining and founding, and wood preservation sectors (Environment Canada, 2010). Health Canada has established a maximum level for PAHs in a specific food product called olive-pomace oil (Health Canada, 2012).

In the following sections, some priority PAHs are discussed, and data on the levels of urinary hydroxylated PAH metabolites in the Canadian population are presented.

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14.2 BENZO[*a*]PYRENE

Benzo[*a*]pyrene (CASRN 50-32-8) is a polycyclic aromatic hydrocarbon (PAH) composed of five fused benzene rings. It is not manufactured in Canada and no industrial uses are known (Health Canada, 2016). In laboratory rats, 40% to 60% of benzo[*a*]pyrene was shown to be absorbed following exposure through gavage or diet (Faust, 1994). Based on laboratory studies, approximately 3% of benzo[*a*]pyrene is expected to be absorbed through skin after 24 hours (Kao et al., 1985), although an optimized absorption study using rats showed 46% absorption over 5 days (Yang et al., 1986). Benzo[*a*]pyrene absorption through skin may be facilitated by metabolism in the cellular epidermis and the formation of stable adducts of benzo[*a*] pyrene metabolites and cellular structures. Evidence for absorption of benzo[*a*]pyrene following inhalation comes from the presence of urinary metabolites in workers exposed to PAHs in air (ATSDR, 1995). Inhalation absorption of benzo[*a*]pyrene is highly dependent on the type of particles onto which it is adsorbed. After absorption, benzo[*a*]pyrene distributes to several organs, including lungs, liver, and intestines (Faust, 1994). Like other PAHs, benzo[*a*]pyrene can be metabolized by many tissues forming various reactive metabolites such as diol epoxides, radical cations, and *o*-quinones. Quinones also induce the generation of reactive oxygen species (EPA, 2017). In recent years, the metabolite 3-hydroxybenzo[*a*]pyrene has been used as a human urine biomarker for exposure to benzo[*a*]pyrene (Chien and Yeh, 2012).

Adverse health effects have been observed in laboratory animals following benzo[*a*]pyrene exposure via inhalation, oral, and dermal routes. Non-carcinogenic effects have been observed, in rodents, including neurological and neurodevelopmental effects (ATSDR, 1995; Health Canada, 2016; Jules et al., 2012). The diolepoxides formed during metabolism of benzo[*a*] pyrene are considered to be the primary carcinogenic agents (IARC, 2012). Occupational exposures to benzo[*a*] pyrene-containing mixtures have been associated with a series of cancers (IARC, 2012). Based on the strong evidence for the carcinogenicity of benzo[*a*]pyrene in many animal species, and supported by evidence from laboratory and human studies, the International Agency for Research on Cancer has classified benzo[*a*]pyrene as Group 1, a known human carcinogen (IARC, 2012). Prior to this, Environment Canada and Health Canada had classified benzo[*a*]pyrene as probably carcinogenic to humans (Group II) based primarily on results from studies with laboratory animals (Environment Canada and Health Canada, 1994).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for benzo[*a*]pyrene in drinking water (Health Canada, 2016). This guideline was developed based on carcinogenic effects in experimental animals and is considered protective of both cancer and non-cancer effects.

The benzo[*a*]pyrene metabolite, 3-hydroxybenzo[*a*] pyrene, was analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2016) participants aged 3–79 years, and is presented as both µg/L and µg/g creatinine. Finding a measurable amount of 3-hydroxybenzo[*a*] pyrene in urine is an indicator of exposure to benzo[*a*] pyrene and does not necessarily mean that an adverse health effect will occur.

Table 14.2.1

3-Hydroxybenzo[*a*]pyrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2294	99.91	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2378	99.96	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2431	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1163	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1188	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1220	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1131	99.82	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1190	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1211	99.83	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	420	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	453	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	450	99.78	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	466	99.79	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	468	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	494	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	473	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	486	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	488	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	328	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	340	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	340	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	300	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	303	99.67	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	267	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	331	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	344	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.2.2

3-Hydroxybenzo[*a*]pyrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2284	99.91	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2377	99.96	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2431	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1159	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1188	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1220	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1125	99.82	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1189	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1211	99.83	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	419	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	452	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	450	99.78	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	464	99.79	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	468	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	494	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	471	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	486	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	488	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	326	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	340	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	338	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	300	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	303	99.67	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	266	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	331	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	344	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

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14.3 CHRYSENE

Chrysene is a polycyclic aromatic hydrocarbon (PAH) composed of four fused benzene rings. There are no known uses of chrysene other than its use as a research chemical (ATSDR, 1995).

Chrysene is highly lipophilic. In animal pharmacokinetic studies, approximately 75% of chrysene was absorbed when administered through oral, inhalation, and dermal routes of exposure; after absorption, it preferentially distributed to adipose tissues (Borges, 1994). Chrysene is metabolized into several mono- and di-hydroxychrysene metabolites (CDC, 2009). Chrysene metabolites are excreted predominantly in the feces. However, PAH biomonitoring studies in humans have attempted to measure urinary levels of 1-, 2-, 3-, 4-, and 6-hydroxychrysene, and have been able to detect urinary 3- and 6-hydroxychrysene in a small proportion of samples (Nethery et al., 2012).

Data on the systemic toxicity of chrysene in animals and humans are limited (Borges, 1994). In mice, chrysene exposure resulted in an increased incidence of skin papillomas, and hepatic and lung tumours (Chang et al., 1983; Wislocki et al., 1986). Based on a limited amount of available carcinogenicity data, the International Agency for Research on Cancer has classified chrysene as Group 2B, possibly carcinogenic to humans (IARC, 2010).

The urinary chrysene metabolites 3- and 6-hydroxychrysene were measured for 73 non-smoking, non-occupationally exposed individuals (aged 16–64 years) living approximately 1 km from an aluminum plant in Baie-Comeau, Quebec. These chrysene metabolites were measured as part of a spectrum of PAH metabolites. Although the levels of some other urinary PAH metabolites were higher compared with a control group of 71 individuals living at least 11 km from the plant, the urinary concentrations of these chrysene metabolites were below the limit of detection (0.032 µg/L for 3-hydroxychrysene and 0.019 µg/L for 6-hydroxychrysene) for most samples (Bouchard et al., 2009). Similarly, 2-, 3-, 4-, and 6-hydroxychrysene concentrations were below the limit of detection when measured in urine of pregnant women living in Hamilton, Ontario (Nethery et al., 2012).

The chrysene metabolites, 2-, 3-, 4-, and 6-hydroxychrysene, were analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and are presented as both µg/L and µg/g creatinine. Given that chrysene metabolites are predominantly excreted in the feces, their urinary absence alone does not indicate that exposure to chrysene did not occur. Finding a measurable amount of chrysene metabolites in urine is an indicator of exposure to chrysene and does not necessarily mean that an adverse health effect will occur.

Table 14.3.1

2-Hydroxychrysene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2497	99.84	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2496	99.96	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	100	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1237	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1246	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1243	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1259	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	499	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	492	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	508	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	498	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	504	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	352	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	345	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	357	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	283	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.2

2-Hydroxychrysene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2487	99.84	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2495	99.96	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	100	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1250	99.92	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1237	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1246	100	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1237	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1258	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	498	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	491	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	506	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	496	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	504	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	350	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	345	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	355	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	282	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.3

3-Hydroxychrysene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2495	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2498	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1255	99.92	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1237	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1246	99.92	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1240	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1261	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	499	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	492	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
2 (2009–2011)	506	99.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	498	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	505	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	99.80	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	351	99.72	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	358	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	283	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	99.72	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.4

3-Hydroxychrysene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2485	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2497	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1251	99.92	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1237	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1246	99.92	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1234	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1260	99.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	498	99.60	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	491	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	504	99.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	496	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	505	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	99.80	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	349	99.72	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	356	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	282	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	99.72	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.5

4-Hydroxychrysene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2498	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2498	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	99.88	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1241	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1261	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1241	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1261	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	498	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	492	99.39	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	508	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	99.80	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	499	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	505	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	352	99.72	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	358	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	99.68	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	283	99.65	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	99.43	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.6

4-Hydroxychrysene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2488	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2497	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2500	99.88	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1253	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1237	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1246	99.84	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1235	99.76	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1260	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1254	99.92	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	497	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	491	99.39	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	506	99.61	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	496	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	509	99.80	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	497	99.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	505	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	498	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	350	99.72	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	356	100	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	356	100	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	311	99.68	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	282	99.65	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	99.43	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.7

6-Hydroxychrysene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2459	96.87	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2494	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2492	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1239	96.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1234	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1245	99.84	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1220	97.38	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1260	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1247	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	494	97.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	492	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
2 (2009–2011)	499	95.79	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	494	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	508	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	489	97.55	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	504	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	495	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	344	96.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	99.42	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	354	99.44	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	354	96.33	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	310	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	310	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	279	97.49	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.3.8

6-Hydroxychrysene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2449	96.87	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2493	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2492	99.92	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1235	96.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1234	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1245	99.84	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1214	97.38	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1259	99.84	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1247	100	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	493	97.37	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	491	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	473	100	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	497	95.79	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	494	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	508	100	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	487	97.55	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	504	99.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	495	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	342	96.80	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	346	99.42	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	354	99.44	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	352	96.33	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	310	100	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	310	100	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	278	97.49	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	348	99.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	352	100	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

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14.4 FLUORANTHENE

Fluoranthene, also known as benzo[*j*,*k*]fluorene, is a polycyclic aromatic hydrocarbon (PAH) with five fused aromatic rings. It is found naturally in the environment in some bacteria, algae, and plants, and as a result of anthropogenic releases from incomplete combustion of organic substances (EPA, 1980). Fluoranthene is used in the synthesis of dyes and in biomedical research (Wu et al., 2010).

Limited pharmacokinetic data for fluoranthene are available. Similar to other structurally related PAHs, fluoranthene may be absorbed following oral, inhalation, or dermal exposure (Faust, 1993; Storer et al., 1984). Because of its high lipophilicity, fluoranthene distributes to adipose tissue (EPA, 1980). Metabolism of fluoranthene produces hydroxylated metabolites, and urinary 3-hydroxyfluoranthene is considered an indicator of recent exposures.

Kidney and liver effects were observed in rats orally administered fluoranthene (Faust, 1993). Fluoranthene exposure in mice has resulted in lung tumours (Busby Jr. et al., 1989; IARC, 2010). Dermal exposure in mice to a combination of benzo[*a*]pyrene and fluoranthene significantly increased the incidence of skin tumours (IARC, 2010). Based on the limited data on fluoranthene carcinogenicity, the International Agency for Research on Cancer has classified fluoranthene as Group 3, not classifiable as to its carcinogenicity to humans (IARC, 2010).

The urinary fluoranthene metabolite, 3-hydroxyfluoranthene, was measured for 73 non-smoking, non-occupationally exposed individuals (aged 16–64 years) living approximately 1 km from an aluminum plant in Baie-Comeau, Quebec. The fluoranthene metabolite was measured as part of a spectrum of PAH metabolites. Although the levels of some other urinary PAH metabolites were higher compared with a control group of 71 individuals living at least 11 km from the plant, the concentration of

3-hydroxyfluoranthene was below the limit of detection (0.030 µg/L) for most samples (Bouchard et al., 2009).

The fluoranthene metabolite, 3-hydroxyfluoranthene, was analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and is presented as both µg/L and µg/g creatinine. Finding a measurable amount of 3-hydroxyfluoranthene in urine is an indicator of exposure to fluoranthene and does not necessarily mean that an adverse health effect will occur.

Table 14.4.1

3-Hydroxyfluoranthene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2265	98.23	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2263	98.37	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2313	98.53	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1145	98.25	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1133	98.41	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1158	98.96	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1120	98.21	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1130	98.32	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1155	98.10	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	428	97.20	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	435	97.93	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	438	98.17	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)	463	97.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	450	98.67	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	459	98.69	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	460	99.57	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	469	98.08	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	468	98.93	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	319	99.69	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	98.72	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	331	98.79	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	329	97.87	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	288	98.26	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	293	97.95	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	266	97.74	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	309	98.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	324	98.46	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 14.4.2

3-Hydroxyfluoranthene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2257	98.23	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2262	98.37	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2313	98.53	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)	1142	98.25	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1133	98.41	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1158	98.96	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)	1115	98.21	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1129	98.32	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1155	98.10	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)	428	97.20	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	434	97.93	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	438	98.17	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
2 (2009–2011)	462	97.41	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	450	98.67	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	459	98.69	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	458	99.57	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	469	98.08	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	468	98.93	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	317	99.69	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	98.72	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	331	98.79	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	327	97.87	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	288	98.26	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	293	97.95	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	265	97.74	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	309	98.71	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	324	98.46	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

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14.5 FLUORENE

Fluorene is a polycyclic aromatic hydrocarbon (PAH) with three fused aromatic rings. Fluorene and its derivatives are used in the manufacture of dyes, pharmaceuticals, polymer materials, photonics, and basic research (Belfield et al., 1999; Bernius et al., 2000; Mondal et al., 2009).

Animal studies indicate that fluorene is absorbed following oral, inhalation, and dermal exposure (ATSDR, 1995). Metabolism of fluorene produces several hydroxylated metabolites that are further conjugated with glucuronic or sulphonic acids and rapidly eliminated in the urine (ATSDR, 1995). Several urinary monohydroxy fluorene metabolites, including 2-, 3-, and 9-hydroxyfluorene, have been identified in humans and are considered indicators of recent PAH exposure (Becker et al., 2003; CDC, 2009; Nethery et al., 2012). Urinary 3-hydroxyfluorene may be a good predictive biomarker for specifically assessing inhalation exposure to fluorene (Nethery et al., 2012).

Hematological and liver effects were observed in laboratory animals exposed orally to fluorene (ATSDR, 1995). Data on the carcinogenicity of fluorene in humans have not been identified and the International Agency for Research on Cancer has classified fluorene as Group 3, not classifiable as to its carcinogenicity in humans (IARC, 2010).

In a pilot biomonitoring study carried out in Hamilton, Ontario, with 19 pregnant women aged 19–42 years, the geometric means for 2-, 3-, and 9-hydroxyfluorene in urine were 0.2157 µg/g creatinine, 0.04827 µg/g creatinine, and 0.3908 µg/g creatinine, respectively (Nethery et al., 2012).

The fluorene metabolites, 2-, 3-, and 9-hydroxyfluorene, were analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and are presented as both µg/L and µg/g creatinine. Finding a measurable amount of fluorene metabolites in urine is an indicator of exposure to fluorene and does not necessarily mean that an adverse health effect will occur.

Table 14.5.1

2-Hydroxyfluorene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2524	0	0.27 (0.24–0.29)	0.069 (0.058–0.080)	0.24 (0.21–0.27)	1.2 (0.95–1.5)	2.3 (1.7–2.8)
3 (2012–2013)	2514	0	0.26 (0.24–0.29)	0.063 (0.051–0.075)	0.22 (0.20–0.24)	1.6 (1.4–1.8)	2.2 (1.6–2.7)
4 (2014–2015)	2510	0	0.28 (0.25–0.31)	0.074 (0.060–0.088)	0.24 (0.20–0.28)	1.2[E] (0.51–1.9)	2.3[E] (1.3–3.3)
Males, 3–79 years							
2 (2009–2011)	1268	0	0.32 (0.27–0.38)	0.087 (0.071–0.10)	0.27 (0.23–0.32)	1.6[E] (0.95–2.3)	3.0 (2.1–4.0)
3 (2012–2013)	1243	0	0.32 (0.29–0.35)	0.079 (0.058–0.10)	0.26 (0.23–0.29)	1.8 (1.5–2.0)	2.5 (1.7–3.2)
4 (2014–2015)	1251	0	0.31 (0.25–0.37)	0.085 (0.064–0.11)	0.30 (0.24–0.36)	F	2.4[E] (0.92–4.0)
Females, 3–79 years							
2 (2009–2011)	1256	0	0.22 (0.21–0.25)	0.064 (0.051–0.076)	0.21 (0.17–0.26)	0.88[E] (0.54–1.2)	1.8 (1.3–2.3)
3 (2012–2013)	1271	0	0.21 (0.17–0.27)	0.050 (0.035–0.065)	0.18 (0.14–0.21)	1.4[E] (0.84–2.0)	2.0 (1.4–2.6)
4 (2014–2015)	1259	0	0.25 (0.22–0.29)	0.058 (0.041–0.074)	0.21 (0.18–0.25)	1.6[E] (0.62–2.5)	2.2[E] (1.3–3.1)
3–5 years							
2 (2009–2011)	506	0	0.17 (0.16–0.19)	0.069 (0.061–0.077)	0.18 (0.16–0.20)	0.37 (0.29–0.45)	0.47 (0.32–0.62)
3 (2012–2013)	496	0	0.16 (0.13–0.18)	0.045 (0.029–0.060)	0.17 (0.13–0.21)	0.41 (0.32–0.51)	0.61 (0.41–0.81)
4 (2014–2015)	478	0	0.16 (0.14–0.19)	0.057[E] (0.034–0.081)	0.16 (0.13–0.19)	0.43 (0.32–0.55)	0.55 (0.46–0.64)
6–11 years							
2 (2009–2011)	511	0	0.22 (0.18–0.25)	0.088 (0.077–0.10)	0.24 (0.19–0.29)	0.48 (0.40–0.56)	0.57 (0.38–0.76)
3 (2012–2013)	502	0	0.19 (0.17–0.21)	0.062 (0.045–0.078)	0.18 (0.16–0.21)	0.49 (0.38–0.60)	0.66 (0.53–0.78)
4 (2014–2015)	510	0	0.21 (0.18–0.23)	0.081 (0.068–0.094)	0.20 (0.17–0.24)	0.54 (0.44–0.64)	0.65 (0.57–0.72)
12–19 years							
2 (2009–2011)	506	0	0.26 (0.24–0.29)	0.098 (0.073–0.12)	0.26 (0.22–0.30)	0.73 (0.55–0.90)	1.1 (0.87–1.3)
3 (2012–2013)	506	0	0.28 (0.23–0.33)	0.079 (0.063–0.095)	0.25 (0.19–0.31)	1.0[E] (0.52–1.5)	F
4 (2014–2015)	498	0	0.24 (0.21–0.28)	0.070 (0.056–0.084)	0.26 (0.21–0.32)	0.73 (0.60–0.86)	0.93 (0.67–1.2)
20–39 years							
2 (2009–2011)	355	0	0.30 (0.25–0.35)	0.085 (0.061–0.11)	0.28 (0.22–0.33)	1.3 (0.88–1.7)	2.2 (1.5–3.0)
3 (2012–2013)	351	0	0.36 (0.29–0.46)	0.087 (0.066–0.11)	0.33 (0.27–0.39)	1.8 (1.3–2.4)	F
4 (2014–2015)	359	0	0.34 (0.28–0.40)	0.084[E] (0.044–0.12)	0.32 (0.27–0.37)	1.8[E] (0.93–2.7)	2.3[E] (1.3–3.4)
40–59 years							
2 (2009–2011)	359	0	0.30 (0.25–0.37)	0.066[E] (0.037–0.095)	0.25 (0.18–0.31)	1.9 (1.2–2.6)	3.3 (2.4–4.2)
3 (2012–2013)	312	0	0.27 (0.21–0.34)	0.062 (0.044–0.081)	0.21 (0.16–0.27)	1.6 (1.2–2.0)	2.0[E] (1.3–2.8)
4 (2014–2015)	312	0	0.31 (0.26–0.36)	0.066[E] (0.038–0.095)	0.26 (0.19–0.33)	1.7[E] (0.67–2.8)	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	287	0	0.21 (0.18–0.25)	0.054 (0.043–0.064)	0.18 (0.15–0.20)	1.1[E] (0.63–1.5)	2.3[E] (1.2–3.3)
3 (2012–2013)	347	0	0.21 (0.16–0.27)	0.041[E] (0.020–0.062)	0.17 (0.14–0.20)	1.7[E] (0.82–2.7)	3.0[E] (1.3–4.8)
4 (2014–2015)	353	0	0.23 (0.20–0.26)	0.065 (0.052–0.078)	0.19 (0.16–0.22)	1.2[E] (0.72–1.7)	2.6[E] (1.4–3.7)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.5.2

2-Hydroxyfluorene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2514	0	0.27 (0.24–0.29)	0.10 (0.091–0.11)	0.21 (0.19–0.22)	1.1 (0.82–1.4)	1.9 (1.4–2.4)
3 (2012–2013)	2513	0	0.27 (0.25–0.29)	0.095 (0.089–0.10)	0.20 (0.18–0.23)	1.4 (1.1–1.7)	2.0 (1.6–2.3)
4 (2014–2015)	2510	0	0.25 (0.22–0.28)	0.094 (0.086–0.10)	0.20 (0.17–0.22)	1.3 (0.91–1.6)	1.9 (1.6–2.2)
Males, 3–79 years							
2 (2009–2011)	1264	0	0.27 (0.23–0.32)	0.096 (0.089–0.10)	0.21 (0.18–0.23)	1.4[E] (0.81–1.9)	2.4[E] (1.3–3.5)
3 (2012–2013)	1243	0	0.27 (0.23–0.31)	0.083 (0.071–0.096)	0.20 (0.16–0.23)	1.5 (1.0–2.0)	1.9 (1.4–2.3)
4 (2014–2015)	1251	0	0.24 (0.20–0.30)	0.084 (0.067–0.10)	0.19 (0.14–0.24)	1.3[E] (0.51–2.0)	1.9 (1.3–2.5)
Females, 3–79 years							
2 (2009–2011)	1250	0	0.26 (0.24–0.28)	0.12 (0.10–0.13)	0.21 (0.19–0.22)	0.87[E] (0.46–1.3)	1.7 (1.3–2.1)
3 (2012–2013)	1270	0	0.27 (0.23–0.32)	0.10 (0.097–0.11)	0.21 (0.18–0.24)	1.4 (0.87–1.9)	2.0 (1.5–2.5)
4 (2014–2015)	1259	0	0.26 (0.23–0.30)	0.099 (0.089–0.11)	0.20 (0.17–0.22)	1.3[E] (0.61–2.0)	2.0[E] (1.1–2.9)
3–5 years							
2 (2009–2011)	505	0	0.31 (0.28–0.34)	0.16 (0.14–0.18)	0.30 (0.26–0.33)	0.62 (0.46–0.78)	0.75 (0.63–0.88)
3 (2012–2013)	495	0	0.30 (0.28–0.33)	0.16 (0.15–0.18)	0.28 (0.26–0.31)	0.57 (0.50–0.65)	0.77 (0.58–0.95)
4 (2014–2015)	478	0	0.29 (0.26–0.32)	0.15 (0.13–0.17)	0.27 (0.24–0.29)	0.57 (0.49–0.66)	0.66 (0.51–0.82)
6–11 years							
2 (2009–2011)	509	0	0.25 (0.22–0.28)	0.14 (0.12–0.16)	0.23 (0.19–0.26)	0.46 (0.37–0.55)	0.59 (0.41–0.78)
3 (2012–2013)	502	0	0.24 (0.21–0.27)	0.12 (0.11–0.13)	0.22 (0.18–0.26)	0.55 (0.44–0.66)	0.67 (0.58–0.75)
4 (2014–2015)	510	0	0.23 (0.21–0.25)	0.11 (0.10–0.13)	0.22 (0.20–0.24)	0.43 (0.36–0.50)	0.53 (0.46–0.61)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	504	0	0.20 (0.18–0.22)	0.099 (0.092–0.11)	0.17 (0.16–0.18)	0.44 (0.33–0.55)	F
3 (2012–2013)	506	0	0.21 (0.18–0.24)	0.099 (0.091–0.11)	0.17 (0.15–0.20)	0.58[E] (0.33–0.83)	1.1[E] (0.37–1.8)
4 (2014–2015)	498	0	0.18 (0.16–0.20)	0.083 (0.071–0.096)	0.16 (0.14–0.18)	0.40 (0.30–0.50)	0.60[E] (0.28–0.93)
20–39 years							
2 (2009–2011)	353	0	0.27 (0.22–0.33)	0.10 (0.080–0.12)	0.20 (0.17–0.24)	1.1[E] (0.56–1.6)	2.2[E] (1.0–3.3)
3 (2012–2013)	351	0	0.28 (0.23–0.35)	0.096 (0.080–0.11)	0.22 (0.16–0.29)	1.3[E] (0.50–2.1)	1.7 (1.1–2.2)
4 (2014–2015)	359	0	0.27 (0.22–0.33)	0.095 (0.083–0.11)	0.22 (0.16–0.28)	1.4 (0.88–1.8)	1.5 (1.0–2.0)
40–59 years							
2 (2009–2011)	357	0	0.30 (0.26–0.36)	0.10 (0.081–0.12)	0.22 (0.18–0.25)	1.6 (1.1–2.1)	2.5 (1.6–3.3)
3 (2012–2013)	312	0	0.31 (0.25–0.38)	0.090 (0.074–0.11)	0.23 (0.17–0.29)	1.7 (1.1–2.2)	2.2 (1.6–2.9)
4 (2014–2015)	312	0	0.28 (0.24–0.33)	0.095 (0.079–0.11)	0.19 (0.15–0.24)	1.9[E] (1.2–2.6)	2.2[E] (1.3–3.1)
60–79 years							
2 (2009–2011)	286	0	0.25 (0.22–0.28)	0.098 (0.092–0.10)	0.18 (0.16–0.20)	1.3[E] (0.78–1.8)	1.8 (1.3–2.2)
3 (2012–2013)	347	0	0.24 (0.19–0.30)	0.084 (0.069–0.099)	0.16 (0.12–0.20)	1.6[E] (0.92–2.2)	2.2[E] (1.2–3.1)
4 (2014–2015)	353	0	0.22 (0.19–0.26)	0.086 (0.072–0.099)	0.16 (0.14–0.19)	1.2[E] (0.64–1.9)	2.0[E] (1.2–2.8)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.5.3

3-Hydroxyfluorene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2523	0.04	0.096 (0.087–0.11)	0.022 (0.020–0.025)	0.081 (0.072–0.089)	0.64 (0.46–0.81)	1.3 (0.96–1.7)
3 (2012–2013)	2513	0	0.10 (0.090–0.11)	0.019 (0.016–0.022)	0.082 (0.072–0.091)	0.95 (0.70–1.2)	1.3 (1.0–1.7)
4 (2014–2015)	2508	0	0.10 (0.092–0.12)	0.024 (0.021–0.027)	0.082 (0.068–0.096)	0.72[E] (0.35–1.1)	1.3[E] (0.79–1.9)
Males, 3–79 years							
2 (2009–2011)	1266	0	0.12 (0.099–0.14)	0.028 (0.024–0.032)	0.096 (0.080–0.11)	0.79[E] (0.34–1.2)	1.7[E] (1.1–2.3)
3 (2012–2013)	1242	0	0.13 (0.11–0.15)	0.024 (0.016–0.031)	0.099 (0.093–0.11)	1.1 (0.75–1.4)	1.5 (1.1–1.9)
4 (2014–2015)	1251	0	0.12 (0.091–0.15)	0.030 (0.026–0.034)	0.10 (0.082–0.12)	F	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1257	0.08	0.078 (0.070–0.086)	0.019 (0.015–0.023)	0.071 (0.058–0.083)	0.38[E] (0.15–0.60)	0.99[E] (0.59–1.4)
3 (2012–2013)	1271	0	0.078 (0.061–0.10)	0.016 (0.012–0.020)	0.063 (0.043–0.083)	0.68[E] (0.39–0.98)	1.2 (0.84–1.6)
4 (2014–2015)	1257	0	0.092 (0.077–0.11)	0.020 (0.015–0.026)	0.069 (0.059–0.078)	0.85[E] (0.28–1.4)	1.3[E] (0.71–1.9)
3–5 years							
2 (2009–2011)	507	0	0.069 (0.063–0.077)	0.025 (0.020–0.030)	0.071 (0.061–0.080)	0.16 (0.10–0.22)	0.23[E] (0.099–0.37)
3 (2012–2013)	496	0	0.064 (0.054–0.075)	0.016[E] (0.0076–0.024)	0.068 (0.052–0.083)	0.18 (0.13–0.23)	0.26 (0.18–0.34)
4 (2014–2015)	478	0	0.065 (0.055–0.075)	0.022 (0.015–0.028)	0.068 (0.056–0.081)	0.15 (0.12–0.18)	0.22 (0.15–0.29)
6–11 years							
2 (2009–2011)	511	0	0.084 (0.069–0.10)	0.033 (0.028–0.038)	0.087 (0.066–0.11)	0.22 (0.17–0.27)	0.26 (0.19–0.32)
3 (2012–2013)	501	0	0.077 (0.067–0.089)	0.023 (0.016–0.031)	0.080 (0.073–0.087)	0.25[E] (0.16–0.35)	0.36[E] (0.21–0.52)
4 (2014–2015)	510	0	0.082 (0.074–0.092)	0.032 (0.028–0.037)	0.082 (0.069–0.094)	0.22 (0.17–0.27)	0.30 (0.25–0.34)
12–19 years							
2 (2009–2011)	506	0.20	0.093 (0.082–0.11)	0.029 (0.022–0.036)	0.093 (0.081–0.11)	0.31[E] (0.19–0.42)	0.53[E] (0.33–0.73)
3 (2012–2013)	506	0	0.10 (0.088–0.12)	0.028 (0.019–0.037)	0.088 (0.074–0.10)	0.59[E] (0.24–0.93)	0.94[E] (0.31–1.6)
4 (2014–2015)	498	0	0.091 (0.077–0.11)	0.024 (0.018–0.030)	0.089 (0.073–0.11)	0.31 (0.23–0.40)	0.44 (0.31–0.56)
20–39 years							
2 (2009–2011)	354	0	0.11 (0.092–0.13)	0.025 (0.018–0.032)	0.10 (0.079–0.12)	0.75[E] (0.39–1.1)	1.1[E] (0.70–1.5)
3 (2012–2013)	351	0	0.14 (0.11–0.18)	0.026[E] (0.014–0.038)	0.10 (0.089–0.11)	1.1 (0.77–1.4)	1.4[E] (0.72–2.0)
4 (2014–2015)	358	0	0.12 (0.099–0.15)	0.033 (0.025–0.040)	0.10 (0.082–0.12)	1.0[E] (0.48–1.6)	1.4[E] (0.74–2.0)
40–59 years							
2 (2009–2011)	358	0	0.11 (0.090–0.14)	0.020[E] (0.011–0.030)	0.080 (0.062–0.099)	1.2[E] (0.66–1.8)	2.2 (1.5–3.0)
3 (2012–2013)	311	0	0.11 (0.079–0.14)	0.018[E] (0.011–0.025)	0.078 (0.052–0.10)	0.99[E] (0.54–1.4)	1.3[E] (0.76–1.8)
4 (2014–2015)	311	0	0.12 (0.10–0.15)	0.024 (0.017–0.031)	0.092 (0.061–0.12)	1.1[E] (0.33–1.8)	F
60–79 years							
2 (2009–2011)	287	0	0.067 (0.056–0.079)	0.018 (0.014–0.022)	0.050 (0.043–0.057)	0.44[E] (0.24–0.64)	1.1[E] (0.58–1.7)
3 (2012–2013)	348	0	0.074 (0.057–0.095)	0.012[E] (0.0050–0.018)	0.052 (0.037–0.068)	0.95[E] (0.28–1.6)	1.8[E] (0.83–2.8)
4 (2014–2015)	353	0	0.075 (0.065–0.087)	0.017 (0.013–0.022)	0.057 (0.046–0.068)	0.69[E] (0.29–1.1)	1.2[E] (0.66–1.8)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.5.4

3-Hydroxyfluorene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2513	0.04	0.096 (0.087–0.11)	0.032 (0.031–0.034)	0.070 (0.063–0.077)	0.68[E] (0.41–0.95)	1.1 (0.84–1.3)
3 (2012–2013)	2512	0	0.10 (0.095–0.11)	0.029 (0.025–0.032)	0.072 (0.063–0.081)	0.83 (0.64–1.0)	1.2 (0.94–1.4)
4 (2014–2015)	2508	0	0.093 (0.082–0.10)	0.027 (0.023–0.031)	0.069 (0.060–0.078)	0.67 (0.51–0.84)	1.0 (0.76–1.3)
Males, 3–79 years							
2 (2009–2011)	1262	0	0.10 (0.086–0.12)	0.031 (0.029–0.033)	0.074 (0.060–0.088)	0.81[E] (0.47–1.1)	1.4[E] (0.88–1.9)
3 (2012–2013)	1242	0	0.11 (0.090–0.13)	0.026 (0.021–0.030)	0.077 (0.062–0.091)	0.84 (0.64–1.0)	1.0 (0.82–1.3)
4 (2014–2015)	1251	0	0.091 (0.070–0.12)	0.024 (0.020–0.028)	0.067 (0.055–0.080)	0.60[E] (0.19–1.0)	1.1[E] (0.63–1.6)
Females, 3–79 years							
2 (2009–2011)	1251	0.08	0.089 (0.081–0.098)	0.035 (0.031–0.038)	0.069 (0.063–0.074)	F	0.99 (0.73–1.3)
3 (2012–2013)	1270	0	0.10 (0.084–0.12)	0.032 (0.028–0.036)	0.071 (0.061–0.082)	0.79[E] (0.38–1.2)	1.2 (0.92–1.6)
4 (2014–2015)	1257	0	0.095 (0.080–0.11)	0.030 (0.025–0.035)	0.071 (0.061–0.080)	0.75[E] (0.34–1.2)	1.0[E] (0.49–1.5)
3–5 years							
2 (2009–2011)	506	0	0.12 (0.11–0.14)	0.061 (0.057–0.066)	0.11 (0.089–0.13)	0.25 (0.19–0.30)	0.32[E] (0.16–0.49)
3 (2012–2013)	495	0	0.12 (0.11–0.14)	0.062 (0.053–0.072)	0.11 (0.10–0.13)	0.27 (0.23–0.30)	0.40 (0.27–0.53)
4 (2014–2015)	478	0	0.12 (0.10–0.13)	0.060 (0.054–0.066)	0.11 (0.092–0.12)	0.24 (0.19–0.29)	0.34 (0.24–0.44)
6–11 years							
2 (2009–2011)	509	0	0.098 (0.085–0.11)	0.050 (0.042–0.057)	0.094 (0.080–0.11)	0.20 (0.15–0.25)	0.26 (0.20–0.33)
3 (2012–2013)	501	0	0.099 (0.083–0.12)	0.044 (0.038–0.050)	0.085 (0.070–0.10)	0.25[E] (0.11–0.38)	0.43[E] (0.23–0.62)
4 (2014–2015)	510	0	0.090 (0.084–0.097)	0.041 (0.033–0.049)	0.087 (0.078–0.097)	0.19 (0.17–0.22)	0.27 (0.22–0.31)
12–19 years							
2 (2009–2011)	504	0.20	0.071 (0.062–0.082)	0.031 (0.027–0.035)	0.063 (0.055–0.070)	0.22[E] (0.11–0.33)	F
3 (2012–2013)	506	0	0.079 (0.069–0.090)	0.033 (0.028–0.038)	0.065 (0.056–0.074)	F	0.61[E] (0.25–0.96)
4 (2014–2015)	498	0	0.066 (0.057–0.076)	0.029 (0.026–0.032)	0.056 (0.047–0.065)	0.17 (0.12–0.23)	0.28[E] (0.10–0.47)
20–39 years							
2 (2009–2011)	352	0	0.099 (0.081–0.12)	0.033 (0.027–0.038)	0.070 (0.056–0.083)	0.66[E] (0.28–1.0)	1.1[E] (0.47–1.7)
3 (2012–2013)	351	0	0.11 (0.083–0.14)	0.032 (0.026–0.037)	0.079 (0.060–0.098)	0.78[E] (0.36–1.2)	0.99 (0.69–1.3)
4 (2014–2015)	358	0	0.097 (0.075–0.13)	0.025[E] (0.015–0.034)	0.072 (0.055–0.090)	0.66 (0.44–0.88)	0.88 (0.67–1.1)
40–59 years							
2 (2009–2011)	356	0	0.11 (0.092–0.14)	0.033 (0.028–0.038)	0.072 (0.052–0.093)	1.0 (0.72–1.4)	1.6 (1.2–2.0)
3 (2012–2013)	311	0	0.12 (0.095–0.16)	0.027 (0.019–0.035)	0.078 (0.058–0.099)	1.1 (0.80–1.4)	1.5 (1.0–1.9)
4 (2014–2015)	311	0	0.11 (0.090–0.14)	0.027 (0.019–0.034)	0.073 (0.058–0.088)	1.1[E] (0.63–1.7)	1.6 (1.1–2.1)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	286	0	0.078 (0.067–0.090)	0.028 (0.025–0.031)	0.055 (0.047–0.063)	0.58[E] (0.23–0.92)	0.96 (0.81–1.1)
3 (2012–2013)	348	0	0.085 (0.067–0.11)	0.025 (0.022–0.028)	0.053 (0.041–0.064)	0.82[E] (0.48–1.2)	1.4 (1.0–1.8)
4 (2014–2015)	353	0	0.073 (0.061–0.086)	0.024 (0.019–0.029)	0.053 (0.048–0.059)	0.72[E] (0.34–1.1)	0.93 (0.75–1.1)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.5.5

9-Hydroxyfluorene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2514	0	0.16 (0.15–0.17)	0.051 (0.045–0.058)	0.16 (0.14–0.17)	0.46 (0.37–0.55)	0.66 (0.57–0.76)
3 (2012–2013)	2499	0	0.15 (0.13–0.17)	0.045 (0.037–0.053)	0.14 (0.13–0.16)	0.53 (0.44–0.61)	0.71 (0.60–0.82)
4 (2014–2015)	2501	0	0.15 (0.14–0.17)	0.050 (0.043–0.057)	0.14 (0.12–0.16)	0.48 (0.39–0.57)	0.72 (0.49–0.95)
Males, 3–79 years							
2 (2009–2011)	1260	0	0.17 (0.15–0.20)	0.057 (0.048–0.066)	0.17 (0.13–0.20)	0.58 (0.46–0.70)	0.73 (0.61–0.85)
3 (2012–2013)	1238	0	0.17 (0.15–0.18)	0.051 (0.037–0.065)	0.16 (0.15–0.17)	0.58 (0.46–0.71)	0.73 (0.62–0.85)
4 (2014–2015)	1247	0	0.16 (0.13–0.19)	0.052 (0.040–0.064)	0.15 (0.12–0.18)	0.53 (0.46–0.60)	0.78 (0.52–1.0)
Females, 3–79 years							
2 (2009–2011)	1254	0	0.15 (0.13–0.16)	0.048 (0.040–0.055)	0.14 (0.13–0.16)	0.39 (0.33–0.46)	0.49 (0.32–0.66)
3 (2012–2013)	1261	0	0.13 (0.11–0.16)	0.041 (0.031–0.050)	0.13 (0.10–0.16)	0.49 (0.36–0.63)	0.65 (0.43–0.86)
4 (2014–2015)	1254	0	0.15 (0.13–0.17)	0.049 (0.041–0.056)	0.14 (0.11–0.16)	0.45 (0.33–0.57)	0.66 (0.42–0.89)
3–5 years							
2 (2009–2011)	505	0	0.098 (0.088–0.11)	0.040 (0.032–0.048)	0.098 (0.086–0.11)	0.24 (0.19–0.29)	0.30 (0.25–0.34)
3 (2012–2013)	490	0	0.084 (0.070–0.099)	0.029 (0.022–0.036)	0.085 (0.065–0.10)	0.22 (0.15–0.28)	0.29 (0.21–0.37)
4 (2014–2015)	473	0	0.083 (0.071–0.097)	0.035 (0.029–0.041)	0.082 (0.066–0.098)	0.19 (0.14–0.24)	0.27 (0.19–0.36)
6–11 years							
2 (2009–2011)	509	0	0.11 (0.091–0.13)	0.042 (0.032–0.051)	0.11 (0.086–0.13)	0.29 (0.21–0.37)	0.38 (0.28–0.47)
3 (2012–2013)	498	0	0.091 (0.082–0.10)	0.038 (0.030–0.045)	0.081 (0.072–0.090)	0.24 (0.19–0.30)	0.34 (0.26–0.42)
4 (2014–2015)	510	0	0.094 (0.077–0.11)	0.034 (0.026–0.042)	0.086 (0.059–0.11)	0.26 (0.19–0.33)	0.40[E] (0.25–0.55)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	501	0	0.15 (0.13–0.17)	0.060 (0.047–0.073)	0.14 (0.12–0.17)	0.38 (0.32–0.45)	0.49 (0.35–0.62)
3 (2012–2013)	505	0	0.13 (0.12–0.15)	0.047 (0.039–0.055)	0.13 (0.11–0.15)	0.38[E] (0.23–0.54)	0.58 (0.40–0.77)
4 (2014–2015)	496	0	0.13 (0.11–0.14)	0.041 (0.033–0.050)	0.12 (0.099–0.14)	0.37 (0.30–0.44)	0.45 (0.38–0.52)
20–39 years							
2 (2009–2011)	355	0	0.17 (0.15–0.20)	0.058 (0.041–0.076)	0.18 (0.15–0.21)	0.52 (0.34–0.70)	0.66 (0.53–0.79)
3 (2012–2013)	351	0	0.20 (0.17–0.22)	0.065[E] (0.041–0.089)	0.19 (0.13–0.25)	0.54[E] (0.34–0.74)	0.77 (0.60–0.93)
4 (2014–2015)	359	0	0.19 (0.15–0.23)	0.062 (0.044–0.080)	0.17 (0.11–0.22)	0.54[E] (0.32–0.76)	0.80[E] (0.43–1.2)
40–59 years							
2 (2009–2011)	358	0	0.17 (0.15–0.20)	0.048[E] (0.029–0.066)	0.17 (0.13–0.20)	0.51 (0.34–0.68)	0.80 (0.55–1.1)
3 (2012–2013)	310	0	0.17 (0.13–0.20)	0.045 (0.035–0.056)	0.15 (0.11–0.19)	0.58 (0.49–0.68)	0.76 (0.56–0.95)
4 (2014–2015)	312	0	0.16 (0.13–0.19)	0.053 (0.036–0.069)	0.14 (0.10–0.19)	0.45[E] (0.19–0.71)	0.80 (0.55–1.0)
60–79 years							
2 (2009–2011)	286	0	0.16 (0.14–0.18)	0.052 (0.046–0.058)	0.15 (0.13–0.17)	0.48 (0.31–0.66)	0.73[E] (0.46–1.0)
3 (2012–2013)	345	0	0.13 (0.11–0.16)	0.037[E] (0.018–0.056)	0.12 (0.10–0.15)	0.49[E] (0.28–0.71)	0.71 (0.48–0.94)
4 (2014–2015)	351	0	0.16 (0.14–0.18)	0.051 (0.047–0.054)	0.14 (0.12–0.16)	0.50 (0.40–0.60)	0.81 (0.53–1.1)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 14.5.6

9-Hydroxyfluorene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2504	0	0.16 (0.15–0.17)	0.059 (0.053–0.066)	0.14 (0.13–0.16)	0.43 (0.35–0.52)	0.62 (0.51–0.72)
3 (2012–2013)	2498	0	0.16 (0.14–0.17)	0.055 (0.048–0.062)	0.14 (0.13–0.16)	0.48 (0.43–0.52)	0.72 (0.64–0.81)
4 (2014–2015)	2501	0	0.14 (0.12–0.15)	0.049 (0.041–0.057)	0.12 (0.10–0.14)	0.48 (0.37–0.59)	0.67 (0.56–0.79)
Males, 3–79 years							
2 (2009–2011)	1256	0	0.15 (0.13–0.17)	0.055 (0.047–0.064)	0.13 (0.11–0.15)	0.50 (0.35–0.64)	0.62 (0.50–0.73)
3 (2012–2013)	1238	0	0.14 (0.12–0.16)	0.049 (0.041–0.058)	0.13 (0.10–0.15)	0.46 (0.35–0.56)	0.62 (0.48–0.76)
4 (2014–2015)	1247	0	0.12 (0.10–0.15)	0.043 (0.034–0.053)	0.10 (0.077–0.12)	0.51 (0.38–0.65)	0.68 (0.54–0.82)
Females, 3–79 years							
2 (2009–2011)	1248	0	0.17 (0.15–0.19)	0.065 (0.058–0.071)	0.15 (0.13–0.18)	0.42 (0.34–0.50)	0.62 (0.44–0.80)
3 (2012–2013)	1260	0	0.17 (0.15–0.20)	0.061 (0.050–0.071)	0.15 (0.14–0.17)	0.57[E] (0.35–0.80)	0.83 (0.63–1.0)
4 (2014–2015)	1254	0	0.15 (0.13–0.17)	0.058 (0.049–0.068)	0.14 (0.11–0.16)	0.48 (0.34–0.62)	0.65 (0.46–0.85)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
2 (2009–2011)	504	0	0.17 (0.15–0.19)	0.068 (0.053–0.083)	0.17 (0.15–0.20)	0.41 (0.31–0.50)	0.62 (0.42–0.81)
3 (2012–2013)	489	0	0.16 (0.14–0.18)	0.065 (0.058–0.072)	0.15 (0.12–0.18)	0.44 (0.36–0.52)	0.54 (0.45–0.64)
4 (2014–2015)	473	0	0.15 (0.13–0.17)	0.056 (0.042–0.071)	0.13 (0.11–0.15)	0.38 (0.28–0.49)	0.51 (0.40–0.61)
6–11 years							
2 (2009–2011)	507	0	0.13 (0.11–0.15)	0.056 (0.049–0.064)	0.11 (0.090–0.13)	0.31 (0.22–0.40)	0.42[E] (0.20–0.64)
3 (2012–2013)	498	0	0.12 (0.10–0.13)	0.046[E] (0.029–0.063)	0.11 (0.096–0.13)	0.27 (0.22–0.31)	0.43 (0.30–0.56)
4 (2014–2015)	510	0	0.10 (0.086–0.12)	0.040 (0.026–0.055)	0.095 (0.079–0.11)	0.27 (0.21–0.33)	0.40 (0.29–0.51)
12–19 years							
2 (2009–2011)	499	0	0.11 (0.097–0.13)	0.048 (0.039–0.056)	0.10 (0.083–0.12)	0.27 (0.19–0.35)	0.37[E] (0.16–0.57)
3 (2012–2013)	505	0	0.10 (0.087–0.12)	0.042 (0.035–0.048)	0.088 (0.077–0.098)	0.25 (0.17–0.34)	0.37[E] (0.22–0.51)
4 (2014–2015)	496	0	0.092 (0.078–0.11)	0.037 (0.032–0.042)	0.087 (0.075–0.099)	0.25 (0.17–0.32)	0.41[E] (0.26–0.57)
20–39 years							
2 (2009–2011)	353	0	0.15 (0.13–0.18)	0.060 (0.049–0.070)	0.14 (0.11–0.16)	0.35[E] (0.15–0.54)	0.56 (0.39–0.73)
3 (2012–2013)	351	0	0.15 (0.13–0.18)	0.055 (0.038–0.072)	0.13 (0.092–0.16)	0.51 (0.33–0.69)	0.68 (0.52–0.84)
4 (2014–2015)	359	0	0.15 (0.12–0.18)	0.049 (0.034–0.063)	0.15 (0.11–0.18)	0.48[E] (0.25–0.71)	0.67[E] (0.42–0.92)
40–59 years							
2 (2009–2011)	356	0	0.17 (0.16–0.19)	0.064 (0.055–0.073)	0.16 (0.14–0.18)	0.52 (0.40–0.63)	0.64[E] (0.40–0.88)
3 (2012–2013)	310	0	0.19 (0.16–0.23)	0.063 (0.043–0.084)	0.20 (0.15–0.24)	0.62[E] (0.38–0.86)	0.81 (0.52–1.1)
4 (2014–2015)	312	0	0.14 (0.12–0.17)	0.053 (0.039–0.066)	0.11[E] (0.066–0.15)	0.56 (0.36–0.77)	0.76 (0.53–0.99)
60–79 years							
2 (2009–2011)	285	0	0.19 (0.16–0.22)	0.065 (0.044–0.086)	0.16 (0.13–0.19)	0.52 (0.34–0.70)	0.77 (0.56–0.98)
3 (2012–2013)	345	0	0.15 (0.13–0.18)	0.056 (0.038–0.073)	0.15 (0.13–0.17)	0.45 (0.31–0.60)	0.64 (0.52–0.76)
4 (2014–2015)	351	0	0.15 (0.13–0.18)	0.065 (0.046–0.085)	0.13 (0.11–0.15)	0.52 (0.36–0.69)	0.72 (0.49–0.96)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

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14.6 NAPHTHALENE

Naphthalene is a polycyclic aromatic hydrocarbon (PAH) with two fused benzene rings. Naphthalene is manufactured and imported into Canada for a wide variety of industrial uses (Environment Canada and Health Canada, 2008). The major consumer products made from naphthalene are moth repellents, in the form of mothballs or mothcrystals. Other commercial uses of naphthalene include components of polyvinyl chloride phthalate plasticizers, dyes, resins, and leather-tanning agents (EPA, 2008; IARC, 2002).

Naphthalene evaporates easily and is often found in the gaseous phase in ambient air (WHO, 2010). Although diet and smoking are the most important sources of intake for most PAHs, inhalation of ambient and indoor air is the main source of naphthalene exposure for the general population. In Canada, indoor air exposure accounts for more than 95% of the total daily exposure across age groups (Environment Canada and Health Canada, 2008). A recent study identified mothballs and some building materials and furnishings (vinyl and wooden furniture, and painted walls and ceilings) as significant contributors to indoor naphthalene concentrations in Canadian homes (Kang et al., 2012). Other sources of naphthalene in indoor and ambient air include migration of volatile organic compounds from attached garages, during cooking, and from kerosene space heaters and wood stoves (Batterman et al., 2007; Environment Canada and Health Canada, 2008). Food and drinking water are considered minor sources of exposure to naphthalene (NTP, 2002).

Naphthalene is rapidly absorbed and metabolized following oral and inhalation exposures in laboratory animals (Bagchi et al., 2002; NTP, 2002). Naphthalene is also absorbed following dermal application in humans and laboratory animals (Storer et al., 1984; Turkall et al., 1994). Like other PAHs, naphthalene undergoes multi-step metabolism, the result of which includes the production of the hydroxynaphthalene metabolites, 1- and 2-hydroxynaphthalene (WHO, 2010). Urinary levels of hydroxynaphthalene metabolites are reflective of recent exposure and have been measured in several human studies (Bouchard et al., 2009; CDC, 2009; Nethery et al., 2012). Urinary 2-hydroxynaphthalene is a unique biomarker of naphthalene metabolism (CDC, 2009). 1-Hydroxynaphthalene is a metabolite of both naphthalene and the insecticide carbaryl, making it difficult to distinguish between these exposures in the general population (Meeker et al., 2007).

In humans, the most serious effects of acute exposure to naphthalene are reported in individuals with glucose 6-phosphate dehydrogenase deficiency, where hemolytic anemia is the primary adverse effect (Health Canada, 2013; WHO, 2010). Reports from occupational exposure and animal studies suggest chronic exposure to naphthalene may lead to the development of lens opacities such as cataracts (Health Canada, 2013;

WHO, 2010). Respiratory tract lesions also have been observed in laboratory animals following acute and chronic exposures (WHO, 2010). Naphthalene has been observed to induce airway tumours in laboratory animals (NTP, 2002). Increased cell proliferation due to cytotoxicity (cell damage) is considered a key element in the development of airway tumours (WHO, 2010). The International Agency for Research on Cancer has classified naphthalene as Group 2B, possibly carcinogenic to humans (IARC, 2002). The carcinogenicity of naphthalene has been proposed to involve non-genotoxic mechanisms (IARC, 2002). Based upon an assessment of acute and chronic effects, Health Canada has established a long-term residential maximum exposure limit for naphthalene (Health Canada, 2013). The guideline is considered to protect against the carcinogenic and non-carcinogenic effects to the respiratory tract resulting from naphthalene exposure.

On the basis of its potential cancer risk, and the margin between levels to which Canadians might be exposed and the critical effect level for non-cancer effects, Health Canada and Environment Canada have concluded that naphthalene is a concern for human health (Environment Canada and Health Canada, 2008). As a result, naphthalene is listed as a toxic substance on Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Canada, 2010a). In an effort to reduce exposure to naphthalene, several risk management approaches have been taken (Canada, 2010b). In 2010, the Pest Management Regulatory Agency (PMRA) re-evaluated the insecticidal uses of naphthalene. PMRA concluded that pest control products containing naphthalene do not present unacceptable risks to human health when used according to label directions, and has granted continued registration (Health Canada, 2010). As a condition of the continued registration of naphthalene uses, Health Canada has introduced new packaging and labelling requirements for naphthalene-containing consumer products (mothballs and moth flakes) in order to minimize exposure (Health Canada, 2012). Naphthalene and 2-hydroxynaphthalene are included as prohibited ingredients and 1-hydroxynaphthalene and its salts as restricted ingredients on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist) (Health Canada, 2015). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Canada, 1985; Health Canada, 2015).

1-Hydroxynaphthalene and 2-hydroxynaphthalene have been measured in urine as biomarkers of exposure to naphthalene in several studies. For various adult populations in Quebec living near industrial facilities, the reported geometric means ranged from 0.80 µg/g to 3.17 µg/g creatinine for 1-hydroxynaphthalene and from 1.38 µg/g to 3.26 µg/g creatinine for 2-hydroxynaphthalene (Bouchard et al., 2001; Bouchard et al., 2009). These naphthalene metabolites were also measured in a pilot biomonitoring study carried out in Hamilton, Ontario, with 19 pregnant women aged 19–42 years. The geometric mean urinary concentrations were 1.411 µg/g creatinine for 1-hydroxynaphthalene and 2.605 µg/g creatinine for 2-hydroxynaphthalene (Nethery et al., 2012). A study aiming to assess naphthalene exposure in 63 pregnant Canadian women aged 20–47 years residing in Ottawa, Ontario, reported geometric mean concentrations in urine ranging from 1.04 µg/L to 1.32 µg/L and from 2.72 µg/g to 2.92 µg/L for 1-hydroxynaphthalene and 2-hydroxynaphthalene, respectively (Wheeler et al., 2014).

Naphthalene metabolites, 1-hydroxynaphthalene and 2-hydroxynaphthalene, were analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and are presented as both µg/L and µg/g creatinine. Finding a measurable amount of naphthalene metabolites in urine can be an indicator of exposure to naphthalene or carbaryl (for 1-hydroxynaphthalene) and does not necessarily mean that an adverse health effect will occur.

Naphthalene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015). Further details on the indoor air studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 14.6.1

1-Hydroxynaphthalene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2522	0.79	1.5 (1.3–1.7)	0.27 (0.18–0.37)	1.3 (1.1–1.5)	9.9 (7.7–12)	15 (12–19)
3 (2012–2013)	2511	2.27	1.0 (0.89–1.2)	0.19 (0.16–0.23)	0.85 (0.68–1.0)	7.8 (6.6–9.0)	11 (8.2–14)
4 (2014–2015)	2500	1.84	0.97 (0.86–1.1)	0.19 (0.16–0.22)	0.81 (0.70–0.92)	7.1 (5.0–9.2)	13 (8.8–18)
Males, 3–79 years							
2 (2009–2011)	1267	0.39	1.6 (1.3–2.0)	0.29[E] (0.16–0.42)	1.3 (1.0–1.7)	11 (9.5–14)	17 (13–21)
3 (2012–2013)	1241	1.69	1.3 (1.1–1.6)	0.24 (0.18–0.30)	1.1 (0.75–1.4)	8.3 (6.4–10)	12[E] (7.2–16)
4 (2014–2015)	1245	2.17	1.0 (0.83–1.2)	0.21 (0.14–0.27)	0.94 (0.79–1.1)	7.0[E] (4.3–9.8)	11[E] (5.1–17)
Females, 3–79 years							
2 (2009–2011)	1255	1.20	1.4 (1.2–1.6)	0.26[E] (0.14–0.37)	1.2 (1.1–1.4)	6.2[E] (2.4–10)	F
3 (2012–2013)	1270	2.83	0.80 (0.57–1.1)	0.18 (0.12–0.23)	0.72 (0.48–0.97)	6.6[E] (3.7–9.4)	9.9 (6.8–13)
4 (2014–2015)	1255	1.51	0.94 (0.73–1.2)	0.18 (0.15–0.20)	0.73 (0.60–0.87)	F	14[E] (6.5–22)
3–5 years							
2 (2009–2011)	506	0.40	1.4 (1.2–1.6)	0.43 (0.35–0.50)	1.2 (1.0–1.4)	5.4[E] (3.3–7.5)	F
3 (2012–2013)	495	1.82	0.69 (0.54–0.88)	0.19[E] (0.12–0.26)	0.68 (0.53–0.82)	2.9 (2.1–3.8)	4.2 (2.7–5.7)
4 (2014–2015)	474	2.53	0.64 (0.57–0.71)	0.18 (0.12–0.25)	0.62 (0.53–0.71)	2.0 (1.6–2.4)	F
6–11 years							
2 (2009–2011)	511	0.39	0.95 (0.79–1.1)	0.25[E] (<LOD–0.40)	0.92 (0.73–1.1)	2.8 (2.1–3.5)	4.0 (2.9–5.2)
3 (2012–2013)	502	2.19	0.75 (0.63–0.90)	0.23 (0.16–0.30)	0.68 (0.55–0.82)	3.0[E] (1.8–4.2)	F
4 (2014–2015)	508	2.56	0.62 (0.53–0.74)	0.18[E] (0.11–0.25)	0.57 (0.45–0.69)	2.2 (1.4–3.0)	3.7[E] (1.6–5.8)
12–19 years							
2 (2009–2011)	505	1.39	1.2 (0.98–1.4)	0.28[E] (0.14–0.41)	1.0 (0.83–1.2)	4.1[E] (2.1–6.1)	F
3 (2012–2013)	505	2.57	0.95 (0.70–1.3)	0.19[E] (0.12–0.26)	0.74 (0.50–0.98)	7.2 (4.8–9.6)	F
4 (2014–2015)	497	1.41	0.69 (0.57–0.84)	0.17[E] (0.10–0.23)	0.65 (0.54–0.76)	2.8 (1.9–3.6)	4.7[E] (1.6–7.8)
20–39 years							
2 (2009–2011)	354	1.13	1.4 (1.1–1.7)	0.29[E] (0.14–0.43)	1.4 (1.0–1.7)	7.1[E] (4.5–9.7)	13 (9.9–15)
3 (2012–2013)	350	2.29	1.2 (0.84–1.6)	0.20[E] (0.12–0.28)	0.91[E] (0.56–1.3)	8.4[E] (5.2–12)	12[E] (5.4–18)
4 (2014–2015)	358	1.40	1.1 (0.85–1.3)	0.18 (0.13–0.23)	0.97 (0.82–1.1)	7.1[E] (4.0–10)	11 (7.3–15)
40–59 years							
2 (2009–2011)	359	1.11	1.7 (1.3–2.2)	0.28[E] (0.11–0.44)	1.3 (0.97–1.7)	13 (10–15)	19[E] (11–27)
3 (2012–2013)	311	3.22	1.1[E] (0.74–1.6)	0.18[E] (0.10–0.25)	0.97[E] (0.27–1.7)	8.1 (5.2–11)	11 (7.1–15)
4 (2014–2015)	311	1.61	1.1 (0.87–1.4)	0.18[E] (0.11–0.25)	0.89[E] (0.43–1.3)	F	20[E] (7.4–32)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	287	0.35	1.7 (1.3–2.2)	0.25[E] (0.12–0.39)	1.6 (1.2–1.9)	F	F
3 (2012–2013)	348	1.72	0.99 (0.77–1.3)	0.21[E] (0.13–0.28)	0.86 (0.68–1.0)	7.0 (4.6–9.4)	13[E] (7.5–19)
4 (2014–2015)	352	1.14	1.1 (0.79–1.4)	0.21 (0.16–0.25)	0.79 (0.57–1.0)	7.6[E] (4.5–11)	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.6.2

1-Hydroxynaphthalene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2512	0.79	1.5 (1.3–1.7)	0.31[E] (0.20–0.42)	1.2 (1.0–1.4)	9.5 (7.0–12)	15 (12–18)
3 (2012–2013)	2510	2.27	1.1 (0.91–1.2)	0.20 (0.16–0.25)	0.99 (0.81–1.2)	6.9 (5.4–8.4)	10 (7.7–12)
4 (2014–2015)	2500	1.84	0.88 (0.78–0.99)	0.18 (0.15–0.21)	0.68 (0.56–0.81)	6.9 (5.4–8.4)	11 (7.1–14)
Males, 3–79 years							
2 (2009–2011)	1263	0.39	1.4 (1.1–1.7)	0.28 (0.19–0.37)	1.1 (0.84–1.3)	9.8[E] (6.0–14)	16 (12–20)
3 (2012–2013)	1241	1.69	1.1 (0.85–1.4)	0.20 (0.15–0.25)	1.0 (0.66–1.3)	7.6 (5.0–10)	9.2 (6.8–12)
4 (2014–2015)	1245	2.17	0.79 (0.65–0.97)	0.15 (0.12–0.18)	0.64 (0.49–0.79)	6.9[E] (3.9–10)	9.7 (6.7–13)
Females, 3–79 years							
2 (2009–2011)	1249	1.20	1.6 (1.4–1.8)	0.40 (0.30–0.51)	1.3 (1.2–1.5)	8.7 (5.6–12)	F
3 (2012–2013)	1269	2.83	1.0 (0.77–1.4)	0.23[E] (0.14–0.31)	0.97 (0.66–1.3)	6.5[E] (3.1–9.9)	12 (8.3–15)
4 (2014–2015)	1255	1.51	0.97 (0.74–1.3)	0.21 (0.15–0.28)	0.73 (0.57–0.90)	F	13[E] (7.0–20)
3–5 years							
2 (2009–2011)	505	0.40	2.5 (2.2–2.9)	0.74 (0.53–0.94)	2.2 (1.9–2.6)	8.4[E] (3.5–13)	16[E] (5.7–25)
3 (2012–2013)	494	1.82	1.3 (1.1–1.6)	0.46 (0.34–0.57)	1.2 (1.1–1.4)	4.9 (3.6–6.2)	6.7 (4.8–8.6)
4 (2014–2015)	474	2.53	1.1 (1.0–1.3)	0.37 (0.30–0.44)	1.0 (0.91–1.1)	3.8[E] (1.0–6.6)	7.5[E] (<LOD–11)
6–11 years							
2 (2009–2011)	509	0.39	1.1 (0.91–1.3)	0.34[E] (<LOD–0.49)	0.99 (0.86–1.1)	3.4 (2.5–4.2)	4.9 (3.4–6.5)
3 (2012–2013)	502	2.19	0.96 (0.76–1.2)	0.30 (0.26–0.34)	0.79 (0.61–0.98)	4.1[E] (2.5–5.8)	5.8[E] (3.2–8.5)
4 (2014–2015)	508	2.56	0.68 (0.60–0.78)	0.22 (0.18–0.27)	0.63 (0.51–0.75)	2.3 (1.6–3.0)	3.6[E] (1.8–5.4)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	503	1.39	0.89 (0.75–1.1)	0.27 (0.19–0.35)	0.83 (0.72–0.94)	3.5[E] (1.9–5.0)	F
3 (2012–2013)	505	2.57	0.72 (0.56–0.92)	0.15[E] (0.089–0.22)	0.67 (0.46–0.88)	4.7[E] (1.3–8.2)	F
4 (2014–2015)	497	1.41	0.51 (0.44–0.59)	0.16 (0.13–0.19)	0.46 (0.37–0.55)	1.8[E] (0.75–2.8)	5.2[E] (2.2–8.3)
20–39 years							
2 (2009–2011)	352	1.13	1.3 (0.97–1.6)	0.23[E] (0.13–0.32)	1.2 (0.79–1.7)	8.2[E] (3.8–13)	13[E] (7.2–18)
3 (2012–2013)	350	2.29	0.89 (0.62–1.3)	0.17[E] (0.074–0.26)	0.78[E] (0.40–1.2)	5.9[E] (2.1–9.6)	9.1[E] (5.7–13)
4 (2014–2015)	358	1.40	0.84 (0.66–1.1)	0.15 (0.11–0.19)	0.65[E] (0.34–0.95)	5.4[E] (3.2–7.6)	7.7 (6.0–9.5)
40–59 years							
2 (2009–2011)	357	1.11	1.7 (1.3–2.1)	0.39[E] (0.21–0.57)	1.3[E] (0.72–1.9)	13[E] (8.3–18)	18[E] (10–27)
3 (2012–2013)	311	3.22	1.3 (0.88–1.8)	0.23[E] (0.11–0.36)	1.3[E] (0.80–1.8)	9.8 (6.3–13)	13 (8.6–17)
4 (2014–2015)	311	1.61	1.0 (0.78–1.3)	0.20 (0.13–0.27)	0.76 (0.59–0.93)	9.9[E] (4.8–15)	14 (10–18)
60–79 years							
2 (2009–2011)	286	0.35	2.0 (1.6–2.5)	0.49 (0.33–0.65)	1.5 (1.1–1.8)	F	F
3 (2012–2013)	348	1.72	1.1 (0.93–1.4)	0.24[E] (0.13–0.36)	1.0 (0.70–1.3)	6.5[E] (4.1–8.9)	11 (8.0–14)
4 (2014–2015)	352	1.14	1.0 (0.76–1.4)	0.22 (0.15–0.30)	0.77 (0.57–0.96)	8.5[E] (4.4–13)	11[E] (6.9–15)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.6.3

2-Hydroxynaphthalene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2503	0	3.8 (3.4–4.4)	0.84 (0.68–1.0)	3.8 (3.2–4.4)	17 (14–20)	24 (18–30)
3 (2012–2013)	2506	0	4.1 (3.6–4.6)	0.93 (0.79–1.1)	4.0 (3.4–4.6)	18 (14–22)	26 (22–30)
4 (2014–2015)	2481	0	4.6 (4.2–5.0)	1.0 (0.91–1.1)	4.6 (4.0–5.1)	19 (16–21)	27 (17–37)
Males, 3–79 years							
2 (2009–2011)	1251	0	4.0 (3.3–4.9)	0.99 (0.81–1.2)	3.9 (3.1–4.8)	19 (13–26)	26 (19–32)
3 (2012–2013)	1239	0	4.6 (4.0–5.3)	0.96 (0.80–1.1)	4.4 (3.7–5.2)	20 (13–27)	30 (22–38)
4 (2014–2015)	1232	0	4.5 (4.0–5.0)	1.2 (1.1–1.4)	4.5 (3.8–5.2)	17 (12–21)	24[E] (13–36)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1252	0	3.7 (3.2–4.2)	0.63[E] (0.40–0.87)	3.5 (3.0–4.1)	17 (13–20)	23[E] (13–32)
3 (2012–2013)	1267	0	3.7 (2.9–4.7)	0.88 (0.65–1.1)	3.7 (2.8–4.6)	16 (12–20)	23 (18–28)
4 (2014–2015)	1249	0	4.7 (3.9–5.6)	0.94 (0.73–1.2)	4.8 (3.7–5.9)	20 (17–23)	37[E] (21–53)
3–5 years							
2 (2009–2011)	499	0	3.3 (2.8–3.8)	1.1 (0.91–1.2)	3.0 (2.4–3.6)	11[E] (5.8–15)	17[E] (8.9–24)
3 (2012–2013)	494	0	3.2 (2.6–4.0)	0.72 (0.50–0.93)	3.4 (2.7–4.1)	12[E] (6.7–16)	19[E] (12–26)
4 (2014–2015)	467	0	3.0 (2.8–3.3)	0.99 (0.73–1.2)	3.0 (2.5–3.5)	9.9 (8.6–11)	13 (8.7–16)
6–11 years							
2 (2009–2011)	509	0	3.2 (2.6–4.0)	1.1 (0.82–1.3)	3.0 (2.3–3.8)	8.8[E] (4.7–13)	F
3 (2012–2013)	498	0	3.2 (2.8–3.7)	0.84 (0.60–1.1)	3.2 (2.6–3.8)	10[E] (6.3–14)	14 (9.6–18)
4 (2014–2015)	503	0	3.8 (3.3–4.4)	1.0 (0.86–1.2)	3.6 (2.9–4.4)	14 (10–17)	19[E] (11–27)
12–19 years							
2 (2009–2011)	503	0	4.4 (3.8–5.0)	1.1 (0.93–1.3)	4.4 (3.5–5.3)	15 (9.6–20)	24 (19–29)
3 (2012–2013)	505	0	5.3 (4.6–6.2)	1.2 (0.77–1.7)	5.0 (3.8–6.2)	23 (15–32)	36 (30–43)
4 (2014–2015)	495	0	4.7 (4.3–5.2)	1.0 (0.80–1.2)	4.8 (4.3–5.4)	21 (14–29)	32 (26–38)
20–39 years							
2 (2009–2011)	352	0	4.4 (3.5–5.5)	0.88[E] (0.53–1.2)	4.8 (3.8–5.9)	17 (13–21)	22 (18–27)
3 (2012–2013)	350	0	5.2 (4.3–6.3)	1.2 (0.91–1.6)	5.6 (3.6–7.6)	18[E] (9.7–26)	26[E] (15–37)
4 (2014–2015)	354	0	5.6 (4.5–6.9)	1.3[E] (0.75–1.8)	5.4[E] (3.4–7.3)	20 (14–25)	29[E] (9.2–50)
40–59 years							
2 (2009–2011)	354	0	4.1 (3.1–5.4)	0.75[E] (0.27–1.2)	3.7[E] (2.1–5.2)	21[E] (13–30)	31 (20–42)
3 (2012–2013)	311	0	4.2 (3.5–5.2)	0.97 (0.77–1.2)	4.1 (2.7–5.5)	18[E] (9.6–26)	28 (21–35)
4 (2014–2015)	311	0	4.9 (4.5–5.4)	1.2 (0.83–1.6)	4.6 (3.6–5.6)	20 (15–24)	29[E] (11–47)
60–79 years							
2 (2009–2011)	286	0	2.8 (2.4–3.3)	0.58[E] (0.31–0.86)	2.5 (1.9–3.2)	12[E] (7.5–16)	22 (19–26)
3 (2012–2013)	348	0	3.0 (2.5–3.5)	0.73 (0.48–0.97)	2.9 (2.4–3.4)	14[E] (6.4–22)	24 (15–33)
4 (2014–2015)	351	0	3.3 (2.7–4.1)	0.92 (0.67–1.2)	3.1 (2.4–3.8)	14 (9.0–18)	24[E] (15–33)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.6.4

2-Hydroxynaphthalene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2493	0	3.8 (3.4–4.3)	1.2 (1.0–1.3)	3.4 (2.9–4.0)	15 (13–17)	20 (17–22)
3 (2012–2013)	2505	0	4.3 (3.9–4.7)	1.3 (1.1–1.5)	4.1 (3.4–4.7)	14 (12–17)	19 (16–21)
4 (2014–2015)	2481	0	4.1 (3.8–4.4)	1.3 (1.1–1.5)	3.8 (3.3–4.3)	13 (12–14)	17 (14–19)
Males, 3–79 years							
2 (2009–2011)	1247	0	3.5 (2.9–4.1)	1.0 (0.94–1.2)	2.9 (2.3–3.5)	14 (10–19)	20 (14–25)
3 (2012–2013)	1239	0	3.9 (3.3–4.5)	1.1 (0.91–1.4)	3.5 (2.6–4.4)	13 (9.7–17)	18 (15–21)
4 (2014–2015)	1232	0	3.5 (3.0–4.0)	1.1 (0.88–1.4)	3.1 (2.5–3.8)	12 (8.3–15)	15 (12–18)
Females, 3–79 years							
2 (2009–2011)	1246	0	4.3 (3.9–4.6)	1.4 (1.1–1.7)	3.8 (3.3–4.2)	15 (12–18)	20 (16–23)
3 (2012–2013)	1266	0	4.7 (4.0–5.5)	1.6 (1.3–1.8)	4.4 (3.4–5.3)	15 (13–17)	19 (16–23)
4 (2014–2015)	1249	0	4.8 (4.2–5.5)	1.5 (1.4–1.6)	4.5 (3.8–5.3)	14 (9.1–18)	19 (14–23)
3–5 years							
2 (2009–2011)	498	0	5.9 (5.1–6.8)	2.1 (1.9–2.2)	5.0 (4.2–5.9)	16 (11–21)	23[E] (13–33)
3 (2012–2013)	493	0	6.3 (5.4–7.3)	2.2 (1.7–2.7)	5.8 (4.8–6.8)	19 (13–25)	27[E] (17–37)
4 (2014–2015)	467	0	5.4 (4.9–6.0)	1.9 (1.7–2.0)	5.6 (4.9–6.2)	14[E] (7.9–21)	20 (14–26)
6–11 years							
2 (2009–2011)	507	0	3.8 (3.2–4.5)	1.5 (1.3–1.8)	3.6 (2.6–4.6)	9.4 (6.8–12)	12[E] (5.1–19)
3 (2012–2013)	498	0	4.1 (3.4–5.0)	1.5 (1.1–1.9)	3.9 (3.3–4.5)	11 (8.3–14)	13[E] (4.5–22)
4 (2014–2015)	503	0	4.2 (3.8–4.7)	1.5 (1.0–1.9)	4.0 (3.4–4.6)	12 (8.0–15)	16 (11–21)
12–19 years							
2 (2009–2011)	501	0	3.4 (3.0–3.9)	1.1 (1.0–1.3)	3.1 (2.6–3.6)	9.9 (7.6–12)	13 (10–16)
3 (2012–2013)	505	0	4.1 (3.7–4.4)	1.4 (1.1–1.7)	3.6 (3.2–4.0)	12 (9.6–15)	15 (11–20)
4 (2014–2015)	495	0	3.4 (3.1–3.8)	1.2 (0.91–1.5)	3.2 (2.8–3.6)	12 (8.8–15)	15 (12–18)
20–39 years							
2 (2009–2011)	350	0	3.9 (3.3–4.7)	1.2 (0.93–1.4)	3.4 (2.5–4.4)	15 (10–19)	19 (15–23)
3 (2012–2013)	350	0	4.0 (3.4–4.8)	1.3[E] (0.80–1.8)	4.0 (3.1–4.9)	11 (8.7–13)	14 (11–18)
4 (2014–2015)	354	0	4.5 (3.9–5.2)	1.3 (0.85–1.7)	4.9 (3.4–6.5)	12 (9.8–14)	14 (11–18)
40–59 years							
2 (2009–2011)	352	0	4.1 (3.3–5.2)	1.0[E] (0.64–1.4)	3.7 (2.8–4.7)	19 (14–25)	25 (20–30)
3 (2012–2013)	311	0	5.0 (4.1–6.0)	1.4 (1.1–1.8)	5.1 (3.4–6.7)	18 (14–21)	21 (17–25)
4 (2014–2015)	311	0	4.5 (4.1–4.9)	1.4 (1.2–1.7)	3.8 (3.2–4.5)	16 (13–20)	19 (13–25)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	285	0	3.3 (2.9–3.7)	1.1 (1.0–1.3)	2.6 (2.1–3.1)	14 (9.7–18)	18 (15–20)
3 (2012–2013)	348	0	3.4 (3.0–3.9)	1.0 (0.77–1.2)	3.2 (2.7–3.6)	12 (8.1–16)	17 (13–21)
4 (2014–2015)	351	0	3.2 (2.8–3.8)	1.1 (0.80–1.4)	2.6 (1.8–3.4)	12 (9.7–15)	14 (13–16)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

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14.7 PHENANTHRENE

Phenanthrene is a polycyclic aromatic hydrocarbon (PAH) with three fused benzene rings. It is used in the manufacture of dyes, polymer materials, and biomedical research (Mondal et al., 2009).

After oral administration in rats, phenanthrene was found to be absorbed from the gastrointestinal tract (Faust, 1993). It is also absorbed through the skin in humans following dermal exposure (Storer et al., 1984). Metabolism of phenanthrene proceeds through the formation of epoxides that rearrange to form hydroxy and dihydrodiol metabolites (Jacob and Seidel, 2002). Phenanthrene metabolites are primarily excreted in the urine (Faust, 1993).

Urinary hydroxylated phenanthrene metabolites (1-, 2-, 3-, 4-, and 9-hydroxyphenanthrene) have been assessed in several biomonitoring studies and are indicators of recent PAH exposure (Becker et al., 2003; CDC, 2009; Jacob and Seidel, 2002; Nethery et al., 2012). Their relatively high abundance in the urine

and the availability of validated analytical methods for their detection and quantification make them good biomarkers for assessing exposure. Additionally, urinary concentrations of monohydroxyphenanthrene metabolites are less sensitive to smoking status than other PAH metabolites; they are therefore better suited for assessing exposures where the study population comprises both smokers and non-smokers (Jacob et al., 1999; Rihs et al., 2005). Urinary 3-hydroxyphenanthrene may be a predictive biomarker for specifically assessing inhalation exposure to phenanthrene (Nethery et al., 2012).

In animal studies, phenanthrene did not elicit systemic or carcinogenic effects (ATSDR, 1995). The International Agency for Research on Cancer has classified phenanthrene as Group 3, not classifiable as to its carcinogenicity in humans (IARC, 2010).

In a pilot biomonitoring study carried out in Hamilton, Ontario, with 19 pregnant women aged 19–42 years, the geometric means for 1-, 2-, 3-, and 4-hydroxyphenanthrene in urine were 0.2439, 0.08240, 0.06989, and 0.0403 µg/g creatinine, respectively (Nethery et al., 2012).

Phenanthrene metabolites, 1-, 2-, 3-, 4-, and 9-hydroxyphenanthrene, were analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and are presented as both µg/L and µg/g creatinine. Finding a measurable amount of phenanthrene metabolites in urine is an indicator of exposure to phenanthrene and does not necessarily mean that an adverse health effect will occur.

Table 14.7.1

1-Hydroxyphenanthrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2522	0.04	0.15 (0.14–0.17)	0.049 (0.042–0.056)	0.15 (0.14–0.17)	0.47 (0.38–0.57)	0.69 (0.53–0.84)
3 (2012–2013)	2505	0.04	0.15 (0.13–0.16)	0.040 (0.028–0.051)	0.13 (0.12–0.15)	0.50 (0.38–0.62)	0.73 (0.58–0.88)
4 (2014–2015)	2506	0	0.16 (0.14–0.17)	0.051 (0.042–0.059)	0.15 (0.14–0.17)	0.52 (0.41–0.62)	0.70 (0.57–0.84)
Males, 3–79 years							
2 (2009–2011)	1268	0	0.16 (0.14–0.19)	0.054 (0.046–0.062)	0.16 (0.14–0.19)	0.50 (0.38–0.62)	0.73 (0.57–0.90)
3 (2012–2013)	1241	0.08	0.16 (0.14–0.18)	0.051 (0.038–0.064)	0.15 (0.13–0.17)	0.48 (0.37–0.59)	0.78 (0.54–1.0)
4 (2014–2015)	1249	0	0.15 (0.13–0.18)	0.060 (0.051–0.068)	0.14 (0.11–0.17)	0.46[E] (0.29–0.63)	0.66 (0.47–0.85)
Females, 3–79 years							
2 (2009–2011)	1254	0.08	0.14 (0.13–0.16)	0.041 (0.032–0.049)	0.14 (0.12–0.16)	0.42 (0.30–0.54)	0.66[E] (0.40–0.92)
3 (2012–2013)	1264	0	0.13 (0.11–0.16)	0.035 (0.026–0.044)	0.13 (0.096–0.15)	0.51 (0.35–0.66)	0.67 (0.56–0.79)
4 (2014–2015)	1257	0	0.16 (0.15–0.18)	0.045 (0.035–0.055)	0.16 (0.14–0.17)	0.54 (0.42–0.66)	0.76 (0.55–0.98)
3–5 years							
2 (2009–2011)	505	0	0.11 (0.097–0.13)	0.044 (0.037–0.051)	0.10 (0.094–0.12)	0.29 (0.23–0.36)	0.34 (0.26–0.42)
3 (2012–2013)	490	0	0.092 (0.079–0.11)	0.031 (0.021–0.042)	0.097 (0.086–0.11)	0.27 (0.22–0.31)	0.36 (0.30–0.42)
4 (2014–2015)	476	0	0.10 (0.086–0.12)	0.039 (0.027–0.051)	0.10 (0.079–0.12)	0.27 (0.21–0.34)	0.40 (0.29–0.52)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
2 (2009–2011)	510	0	0.12 (0.11–0.14)	0.046 (0.039–0.054)	0.12 (0.097–0.14)	0.30 (0.21–0.39)	0.42 (0.32–0.51)
3 (2012–2013)	501	0.20	0.11 (0.094–0.12)	0.031[E] (0.015–0.047)	0.11 (0.092–0.12)	0.26 (0.21–0.30)	0.36 (0.30–0.42)
4 (2014–2015)	510	0	0.13 (0.11–0.15)	0.046 (0.039–0.053)	0.12 (0.10–0.14)	0.36 (0.28–0.44)	0.47[E] (0.21–0.72)
12–19 years							
2 (2009–2011)	506	0	0.15 (0.14–0.17)	0.058 (0.044–0.073)	0.15 (0.13–0.18)	0.44 (0.29–0.59)	0.55 (0.43–0.67)
3 (2012–2013)	505	0	0.15 (0.13–0.18)	0.050 (0.035–0.066)	0.14 (0.11–0.17)	0.53 (0.40–0.65)	0.76[E] (0.41–1.1)
4 (2014–2015)	497	0	0.15 (0.13–0.18)	0.049 (0.036–0.063)	0.15 (0.13–0.17)	0.48 (0.34–0.61)	0.75 (0.53–0.97)
20–39 years							
2 (2009–2011)	355	0	0.16 (0.14–0.18)	0.049 (0.033–0.066)	0.17 (0.15–0.19)	0.49 (0.35–0.63)	0.64[E] (0.41–0.87)
3 (2012–2013)	350	0	0.18 (0.16–0.21)	0.052 (0.036–0.068)	0.20 (0.14–0.26)	0.46[E] (0.27–0.65)	0.70[E] (0.28–1.1)
4 (2014–2015)	358	0	0.17 (0.15–0.20)	0.050 (0.034–0.067)	0.17 (0.14–0.21)	0.57 (0.39–0.75)	0.76 (0.54–0.99)
40–59 years							
2 (2009–2011)	359	0	0.16 (0.14–0.19)	0.052[E] (0.031–0.073)	0.16 (0.12–0.19)	0.51[E] (0.32–0.71)	0.77 (0.58–0.97)
3 (2012–2013)	311	0	0.14 (0.12–0.17)	0.036[E] (0.018–0.055)	0.13 (0.095–0.16)	0.51[E] (0.30–0.73)	0.69[E] (0.43–0.94)
4 (2014–2015)	312	0	0.16 (0.13–0.18)	0.059 (0.048–0.069)	0.14 (0.12–0.17)	0.51[E] (0.33–0.70)	0.68[E] (0.26–1.1)
60–79 years							
2 (2009–2011)	287	0.35	0.15 (0.13–0.17)	0.038 (0.026–0.050)	0.16 (0.13–0.18)	0.50 (0.37–0.64)	0.81 (0.53–1.1)
3 (2012–2013)	348	0	0.14 (0.11–0.17)	0.032[E] (0.0094–0.054)	0.12 (0.085–0.15)	0.68 (0.53–0.82)	0.85[E] (0.37–1.3)
4 (2014–2015)	353	0	0.16 (0.14–0.19)	0.049 (0.036–0.062)	0.16 (0.13–0.18)	0.55 (0.46–0.65)	0.67[E] (0.41–0.94)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 14.7.2

1-Hydroxyphenanthrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2512	0.04	0.15 (0.14–0.16)	0.069 (0.064–0.075)	0.14 (0.12–0.15)	0.37 (0.31–0.44)	0.52 (0.41–0.63)
3 (2012–2013)	2504	0.04	0.15 (0.14–0.16)	0.059 (0.050–0.068)	0.14 (0.13–0.15)	0.39 (0.31–0.46)	0.59 (0.47–0.72)
4 (2014–2015)	2506	0	0.14 (0.13–0.16)	0.060 (0.052–0.068)	0.13 (0.11–0.14)	0.37 (0.31–0.43)	0.49 (0.42–0.57)
Males, 3–79 years							
2 (2009–2011)	1264	0	0.14 (0.13–0.16)	0.060 (0.051–0.069)	0.13 (0.11–0.15)	0.35 (0.27–0.43)	0.51 (0.39–0.64)
3 (2012–2013)	1241	0.08	0.13 (0.12–0.15)	0.052 (0.042–0.061)	0.13 (0.11–0.14)	0.34 (0.29–0.39)	0.53[E] (0.32–0.75)
4 (2014–2015)	1249	0	0.12 (0.10–0.14)	0.052 (0.043–0.060)	0.11 (0.090–0.13)	0.31 (0.24–0.37)	0.40 (0.33–0.47)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1248	0.08	0.17 (0.15–0.18)	0.078 (0.066–0.090)	0.15 (0.13–0.16)	0.39 (0.31–0.46)	0.55 (0.40–0.71)
3 (2012–2013)	1263	0	0.17 (0.15–0.19)	0.070 (0.058–0.081)	0.15 (0.13–0.17)	0.47 (0.36–0.57)	0.63 (0.52–0.74)
4 (2014–2015)	1257	0	0.17 (0.15–0.19)	0.074 (0.062–0.087)	0.15 (0.13–0.16)	0.41 (0.32–0.51)	0.61 (0.44–0.78)
3–5 years							
2 (2009–2011)	504	0	0.20 (0.17–0.22)	0.094 (0.077–0.11)	0.18 (0.16–0.21)	0.40 (0.32–0.47)	0.57 (0.39–0.75)
3 (2012–2013)	489	0	0.18 (0.17–0.19)	0.088 (0.073–0.10)	0.16 (0.14–0.18)	0.38 (0.33–0.43)	0.53 (0.42–0.64)
4 (2014–2015)	476	0	0.18 (0.16–0.21)	0.095 (0.082–0.11)	0.18 (0.15–0.21)	0.37 (0.29–0.45)	0.52 (0.42–0.63)
6–11 years							
2 (2009–2011)	508	0	0.14 (0.13–0.16)	0.079 (0.071–0.087)	0.12 (0.11–0.14)	0.31 (0.26–0.37)	0.44[E] (0.26–0.63)
3 (2012–2013)	501	0.20	0.13 (0.12–0.15)	0.068 (0.060–0.077)	0.12 (0.10–0.14)	0.29 (0.20–0.38)	0.37 (0.28–0.45)
4 (2014–2015)	510	0	0.14 (0.13–0.16)	0.068 (0.059–0.077)	0.12 (0.098–0.14)	0.31 (0.22–0.40)	0.50 (0.35–0.64)
12–19 years							
2 (2009–2011)	504	0	0.12 (0.11–0.13)	0.059 (0.055–0.063)	0.11 (0.095–0.12)	0.25 (0.18–0.32)	0.32 (0.25–0.40)
3 (2012–2013)	505	0	0.11 (0.097–0.13)	0.052 (0.040–0.064)	0.099 (0.090–0.11)	0.29 (0.22–0.35)	0.37[E] (0.10–0.64)
4 (2014–2015)	497	0	0.11 (0.094–0.13)	0.049 (0.043–0.055)	0.10 (0.091–0.11)	0.31[E] (0.19–0.43)	0.38 (0.25–0.51)
20–39 years							
2 (2009–2011)	353	0	0.14 (0.12–0.16)	0.057 (0.043–0.071)	0.12 (0.090–0.16)	0.35 (0.23–0.47)	0.49 (0.36–0.62)
3 (2012–2013)	350	0	0.14 (0.12–0.16)	0.060 (0.041–0.079)	0.13 (0.12–0.14)	0.34 (0.27–0.41)	0.50 (0.39–0.61)
4 (2014–2015)	358	0	0.14 (0.12–0.17)	0.064 (0.047–0.082)	0.13 (0.10–0.16)	0.31 (0.22–0.40)	0.43[E] (0.27–0.59)
40–59 years							
2 (2009–2011)	357	0	0.17 (0.15–0.18)	0.078 (0.067–0.088)	0.15 (0.12–0.17)	0.42 (0.36–0.47)	0.58 (0.45–0.70)
3 (2012–2013)	311	0	0.17 (0.15–0.19)	0.062[E] (0.038–0.085)	0.16 (0.13–0.19)	0.48[E] (0.30–0.67)	0.63 (0.44–0.82)
4 (2014–2015)	312	0	0.14 (0.12–0.16)	0.060 (0.046–0.073)	0.13 (0.11–0.14)	0.38 (0.31–0.46)	0.49 (0.32–0.66)
60–79 years							
2 (2009–2011)	286	0.35	0.18 (0.16–0.19)	0.075 (0.060–0.089)	0.16 (0.14–0.18)	0.38[E] (0.24–0.53)	0.62 (0.47–0.78)
3 (2012–2013)	348	0	0.16 (0.13–0.19)	0.059 (0.048–0.069)	0.13 (0.11–0.15)	0.46 (0.36–0.57)	0.81[E] (0.24–1.4)
4 (2014–2015)	353	0	0.16 (0.14–0.18)	0.061 (0.050–0.071)	0.14 (0.12–0.16)	0.42 (0.31–0.52)	0.53 (0.38–0.68)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 14.7.3

2-Hydroxyphenanthrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2520	0	0.067 (0.062–0.071)	0.027 (0.024–0.031)	0.065 (0.060–0.069)	0.17 (0.14–0.20)	0.23 (0.18–0.29)
3 (2012–2013)	2503	0	0.061 (0.054–0.068)	0.021 (0.016–0.025)	0.056 (0.048–0.064)	0.18 (0.16–0.20)	0.28 (0.24–0.33)
4 (2014–2015)	2506	0	0.062 (0.057–0.067)	0.020 (0.017–0.023)	0.061 (0.055–0.066)	0.17 (0.14–0.21)	0.26 (0.21–0.31)
Males, 3–79 years							
2 (2009–2011)	1265	0	0.074 (0.066–0.083)	0.029 (0.025–0.033)	0.070 (0.062–0.078)	0.18 (0.13–0.23)	0.26 (0.19–0.33)
3 (2012–2013)	1238	0	0.071 (0.065–0.078)	0.027 (0.023–0.031)	0.065 (0.058–0.071)	0.19 (0.15–0.23)	0.31 (0.21–0.41)
4 (2014–2015)	1249	0	0.066 (0.059–0.074)	0.022 (0.018–0.027)	0.067 (0.060–0.074)	0.18[E] (0.11–0.25)	0.27 (0.20–0.34)
Females, 3–79 years							
2 (2009–2011)	1255	0	0.060 (0.056–0.065)	0.024 (0.020–0.029)	0.058 (0.052–0.064)	0.14 (0.11–0.16)	0.19[E] (0.12–0.26)
3 (2012–2013)	1265	0	0.052 (0.044–0.063)	0.017 (0.013–0.021)	0.047 (0.035–0.059)	0.15 (0.11–0.18)	0.22[E] (0.10–0.33)
4 (2014–2015)	1257	0	0.057 (0.051–0.064)	0.018 (0.014–0.022)	0.055 (0.049–0.062)	0.17 (0.13–0.20)	0.24 (0.18–0.30)
3–5 years							
2 (2009–2011)	506	0	0.043 (0.038–0.049)	0.023 (0.019–0.027)	0.040 (0.033–0.046)	0.086 (0.060–0.11)	0.11 (0.077–0.15)
3 (2012–2013)	490	0	0.033 (0.028–0.038)	0.014 (0.010–0.018)	0.031 (0.028–0.035)	0.074 (0.067–0.081)	0.090 (0.076–0.10)
4 (2014–2015)	477	0	0.031 (0.026–0.036)	0.012 (0.0080–0.016)	0.029 (0.023–0.036)	0.073 (0.051–0.096)	0.11 (0.081–0.13)
6–11 years							
2 (2009–2011)	510	0	0.052 (0.046–0.059)	0.025 (0.021–0.030)	0.050 (0.045–0.056)	0.10 (0.076–0.13)	0.14 (0.11–0.17)
3 (2012–2013)	500	0	0.041 (0.036–0.045)	0.018 (0.014–0.022)	0.040 (0.034–0.047)	0.090 (0.073–0.11)	0.12 (0.085–0.17)
4 (2014–2015)	510	0	0.043 (0.039–0.048)	0.018 (0.015–0.021)	0.040 (0.035–0.045)	0.11 (0.094–0.13)	0.14 (0.12–0.17)
12–19 years							
2 (2009–2011)	506	0	0.067 (0.061–0.074)	0.033 (0.024–0.042)	0.064 (0.058–0.069)	0.16 (0.11–0.20)	0.19 (0.15–0.24)
3 (2012–2013)	505	0	0.064 (0.054–0.075)	0.024 (0.017–0.032)	0.061 (0.051–0.071)	0.16 (0.12–0.20)	0.26[E] (0.13–0.38)
4 (2014–2015)	497	0	0.059 (0.053–0.066)	0.020 (0.018–0.022)	0.059 (0.050–0.067)	0.16 (0.12–0.20)	0.22 (0.16–0.29)
20–39 years							
2 (2009–2011)	354	0	0.069 (0.060–0.078)	0.028 (0.023–0.033)	0.067 (0.059–0.074)	0.17 (0.13–0.21)	0.23[E] (0.13–0.32)
3 (2012–2013)	350	0	0.083 (0.072–0.095)	0.031 (0.026–0.036)	0.087 (0.068–0.11)	0.19[E] (0.12–0.26)	0.33[E] (0.17–0.49)
4 (2014–2015)	357	0	0.072 (0.061–0.084)	0.024 (0.017–0.030)	0.077 (0.056–0.098)	0.20 (0.16–0.25)	0.26 (0.22–0.30)
40–59 years							
2 (2009–2011)	359	0	0.073 (0.064–0.083)	0.027 (0.018–0.036)	0.071 (0.062–0.081)	0.20 (0.14–0.26)	0.27 (0.20–0.35)
3 (2012–2013)	311	0	0.057 (0.048–0.068)	0.019[E] (0.011–0.028)	0.050 (0.037–0.062)	0.16[E] (0.098–0.23)	0.27[E] (0.17–0.37)
4 (2014–2015)	312	0	0.063 (0.055–0.074)	0.020 (0.014–0.026)	0.065 (0.054–0.076)	0.17[E] (0.072–0.27)	0.30[E] (0.099–0.51)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	285	0	0.064 (0.058–0.071)	0.026 (0.018–0.034)	0.062 (0.055–0.070)	0.15 (0.11–0.19)	F
3 (2012–2013)	347	0	0.059 (0.049–0.073)	0.019[E] (0.012–0.027)	0.054 (0.043–0.064)	0.24[E] (0.12–0.36)	F
4 (2014–2015)	353	0	0.060 (0.054–0.067)	0.019 (0.016–0.022)	0.057 (0.048–0.067)	0.16 (0.14–0.19)	0.26 (0.20–0.33)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.4

2-Hydroxyphenanthrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2510	0	0.067 (0.062–0.072)	0.030 (0.027–0.034)	0.062 (0.057–0.067)	0.14 (0.12–0.16)	0.18 (0.16–0.20)
3 (2012–2013)	2502	0	0.063 (0.058–0.068)	0.028 (0.025–0.031)	0.056 (0.052–0.060)	0.16 (0.14–0.18)	0.21 (0.17–0.26)
4 (2014–2015)	2506	0	0.055 (0.051–0.060)	0.025 (0.023–0.027)	0.051 (0.047–0.054)	0.14 (0.11–0.17)	0.20 (0.17–0.22)
Males, 3–79 years							
2 (2009–2011)	1261	0	0.063 (0.058–0.070)	0.029 (0.027–0.031)	0.059 (0.052–0.066)	0.15 (0.12–0.18)	0.19 (0.15–0.23)
3 (2012–2013)	1238	0	0.059 (0.053–0.066)	0.028 (0.024–0.031)	0.054 (0.049–0.059)	0.17 (0.14–0.19)	0.20 (0.16–0.24)
4 (2014–2015)	1249	0	0.052 (0.045–0.059)	0.024 (0.022–0.026)	0.048 (0.043–0.053)	0.15 (0.11–0.19)	0.20 (0.14–0.26)
Females, 3–79 years							
2 (2009–2011)	1249	0	0.070 (0.062–0.078)	0.033 (0.028–0.039)	0.066 (0.060–0.072)	0.14 (0.11–0.17)	0.18 (0.12–0.23)
3 (2012–2013)	1264	0	0.067 (0.060–0.075)	0.031 (0.025–0.036)	0.061 (0.050–0.073)	0.16 (0.12–0.19)	0.22 (0.17–0.28)
4 (2014–2015)	1257	0	0.059 (0.054–0.064)	0.027 (0.023–0.032)	0.054 (0.050–0.057)	0.14 (0.091–0.18)	0.20 (0.15–0.24)
3–5 years							
2 (2009–2011)	505	0	0.077 (0.066–0.089)	0.040 (0.032–0.047)	0.076 (0.064–0.088)	0.15 (0.12–0.19)	0.18[E] (0.089–0.27)
3 (2012–2013)	489	0	0.063 (0.056–0.071)	0.034 (0.027–0.040)	0.058 (0.051–0.064)	0.13 (0.11–0.15)	0.17 (0.14–0.19)
4 (2014–2015)	477	0	0.055 (0.050–0.061)	0.030 (0.025–0.035)	0.052 (0.045–0.060)	0.10 (0.082–0.12)	0.13[E] (0.081–0.18)
6–11 years							
2 (2009–2011)	508	0	0.061 (0.055–0.067)	0.034 (0.029–0.039)	0.057 (0.052–0.061)	0.11 (0.080–0.15)	0.17 (0.12–0.22)
3 (2012–2013)	500	0	0.052 (0.045–0.060)	0.027 (0.020–0.034)	0.048 (0.039–0.058)	0.10 (0.085–0.12)	0.13 (0.10–0.16)
4 (2014–2015)	510	0	0.047 (0.044–0.051)	0.026 (0.024–0.029)	0.043 (0.040–0.047)	0.098 (0.082–0.11)	0.13 (0.090–0.17)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	504	0	0.051 (0.046–0.057)	0.027 (0.025–0.029)	0.047 (0.042–0.052)	0.10 (0.073–0.13)	0.13[E] (0.078–0.18)
3 (2012–2013)	505	0	0.048 (0.040–0.058)	0.024 (0.020–0.028)	0.045 (0.036–0.053)	0.099 (0.072–0.13)	0.13[E] (0.060–0.21)
4 (2014–2015)	497	0	0.043 (0.038–0.050)	0.020 (0.019–0.022)	0.041 (0.033–0.049)	0.096 (0.061–0.13)	0.15 (0.11–0.19)
20–39 years							
2 (2009–2011)	352	0	0.061 (0.053–0.071)	0.029 (0.026–0.032)	0.057 (0.048–0.066)	0.15 (0.10–0.19)	0.18 (0.14–0.22)
3 (2012–2013)	350	0	0.064 (0.055–0.073)	0.029 (0.024–0.033)	0.055 (0.049–0.062)	0.17 (0.14–0.20)	0.20 (0.14–0.26)
4 (2014–2015)	357	0	0.057 (0.050–0.066)	0.026 (0.022–0.029)	0.053 (0.043–0.063)	0.15 (0.11–0.18)	0.16 (0.13–0.19)
40–59 years							
2 (2009–2011)	357	0	0.074 (0.067–0.081)	0.033 (0.028–0.038)	0.071 (0.063–0.078)	0.17 (0.13–0.20)	0.18 (0.14–0.22)
3 (2012–2013)	311	0	0.067 (0.059–0.077)	0.029 (0.024–0.035)	0.066 (0.057–0.075)	0.17 (0.12–0.21)	0.22 (0.16–0.29)
4 (2014–2015)	312	0	0.058 (0.050–0.067)	0.026 (0.023–0.029)	0.052 (0.046–0.057)	0.17 (0.11–0.23)	0.22 (0.18–0.26)
60–79 years							
2 (2009–2011)	284	0	0.075 (0.067–0.084)	0.036 (0.029–0.043)	0.066 (0.061–0.072)	0.14[E] (0.085–0.19)	F
3 (2012–2013)	347	0	0.068 (0.057–0.082)	0.029 (0.022–0.037)	0.057 (0.048–0.066)	0.19 (0.13–0.24)	F
4 (2014–2015)	353	0	0.058 (0.051–0.067)	0.027 (0.021–0.032)	0.051 (0.046–0.056)	0.13[E] (0.066–0.19)	0.24[E] (0.12–0.35)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.5

3-Hydroxyphenanthrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2515	0	0.087 (0.080–0.095)	0.026 (0.023–0.029)	0.089 (0.080–0.098)	0.28 (0.22–0.35)	0.39 (0.31–0.46)
3 (2012–2013)	2505	0	0.083 (0.077–0.090)	0.021 (0.016–0.026)	0.081 (0.075–0.087)	0.29 (0.24–0.34)	0.48 (0.40–0.57)
4 (2014–2015)	2506	0	0.089 (0.081–0.097)	0.025 (0.021–0.029)	0.090 (0.079–0.10)	0.27 (0.20–0.35)	0.43 (0.36–0.50)
Males, 3–79 years							
2 (2009–2011)	1265	0	0.10 (0.087–0.12)	0.030 (0.026–0.035)	0.099 (0.085–0.11)	0.33 (0.25–0.42)	0.45 (0.30–0.60)
3 (2012–2013)	1241	0	0.099 (0.092–0.11)	0.029 (0.020–0.039)	0.098 (0.084–0.11)	0.33 (0.25–0.40)	0.54 (0.44–0.64)
4 (2014–2015)	1250	0	0.097 (0.085–0.11)	0.031 (0.025–0.037)	0.097 (0.090–0.10)	0.32[E] (0.20–0.44)	0.45 (0.36–0.54)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1250	0	0.075 (0.069–0.082)	0.022 (0.018–0.026)	0.078 (0.066–0.090)	0.22 (0.14–0.29)	0.35 (0.26–0.44)
3 (2012–2013)	1264	0	0.070 (0.060–0.082)	0.017 (0.013–0.021)	0.065 (0.051–0.079)	0.28 (0.22–0.34)	0.40 (0.27–0.52)
4 (2014–2015)	1256	0	0.081 (0.071–0.093)	0.022 (0.016–0.028)	0.081 (0.069–0.093)	0.27 (0.24–0.29)	0.39[E] (0.24–0.54)
3–5 years							
2 (2009–2011)	501	0	0.077 (0.068–0.086)	0.030 (0.026–0.034)	0.076 (0.064–0.088)	0.18[E] (0.10–0.25)	0.28 (0.20–0.35)
3 (2012–2013)	490	0	0.065 (0.058–0.074)	0.020 (0.015–0.024)	0.067 (0.059–0.075)	0.19 (0.16–0.22)	0.28 (0.22–0.34)
4 (2014–2015)	476	0	0.071 (0.060–0.084)	0.025[E] (0.015–0.034)	0.069 (0.051–0.088)	0.19 (0.12–0.25)	0.29 (0.21–0.36)
6–11 years							
2 (2009–2011)	509	0	0.084 (0.071–0.099)	0.029 (0.023–0.035)	0.092 (0.072–0.11)	0.21 (0.16–0.27)	0.28 (0.21–0.34)
3 (2012–2013)	501	0	0.069 (0.059–0.081)	0.021 (0.014–0.029)	0.073 (0.059–0.087)	0.17 (0.13–0.21)	0.22 (0.16–0.28)
4 (2014–2015)	510	0	0.085 (0.074–0.097)	0.030 (0.022–0.037)	0.083 (0.067–0.099)	0.21 (0.18–0.25)	0.33 (0.22–0.45)
12–19 years							
2 (2009–2011)	506	0	0.094 (0.084–0.11)	0.033 (0.025–0.042)	0.091 (0.077–0.10)	0.26 (0.18–0.33)	0.35[E] (0.20–0.50)
3 (2012–2013)	505	0	0.091 (0.081–0.10)	0.028[E] (0.015–0.040)	0.089 (0.075–0.10)	0.28 (0.19–0.38)	0.42 (0.30–0.55)
4 (2014–2015)	497	0	0.093 (0.083–0.11)	0.026 (0.022–0.030)	0.099 (0.090–0.11)	0.24 (0.20–0.29)	0.37 (0.24–0.51)
20–39 years							
2 (2009–2011)	355	0	0.091 (0.078–0.11)	0.027 (0.020–0.034)	0.099 (0.070–0.13)	0.30 (0.21–0.39)	0.38 (0.27–0.49)
3 (2012–2013)	350	0	0.11 (0.093–0.13)	0.035 (0.026–0.044)	0.10 (0.077–0.13)	0.30[E] (0.19–0.40)	0.58[E] (0.25–0.91)
4 (2014–2015)	358	0	0.095 (0.081–0.11)	0.029 (0.021–0.038)	0.099 (0.081–0.12)	0.30 (0.22–0.38)	0.44 (0.36–0.52)
40–59 years							
2 (2009–2011)	358	0	0.091 (0.078–0.11)	0.023[E] (0.014–0.032)	0.093 (0.082–0.10)	0.34 (0.27–0.41)	0.44[E] (0.27–0.60)
3 (2012–2013)	311	0	0.078 (0.066–0.092)	0.021 (0.015–0.027)	0.074 (0.060–0.088)	0.29 (0.22–0.35)	0.38 (0.26–0.50)
4 (2014–2015)	312	0	0.088 (0.074–0.10)	0.024 (0.018–0.029)	0.089 (0.070–0.11)	0.27[E] (0.085–0.46)	F
60–79 years							
2 (2009–2011)	286	0	0.073 (0.063–0.085)	0.020 (0.016–0.025)	0.073 (0.059–0.086)	0.24 (0.18–0.29)	0.33[E] (0.12–0.54)
3 (2012–2013)	348	0	0.072 (0.058–0.089)	0.015[E] (0.0058–0.024)	0.066 (0.050–0.083)	0.48 (0.32–0.65)	0.58 (0.39–0.76)
4 (2014–2015)	353	0	0.084 (0.075–0.093)	0.023 (0.017–0.029)	0.076 (0.063–0.089)	0.27 (0.19–0.34)	0.42 (0.34–0.50)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.6

3-Hydroxyphenanthrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2505	0	0.087 (0.080–0.094)	0.038 (0.035–0.041)	0.079 (0.073–0.085)	0.23 (0.18–0.27)	0.37 (0.29–0.46)
3 (2012–2013)	2504	0	0.086 (0.080–0.092)	0.032 (0.029–0.035)	0.082 (0.072–0.091)	0.26 (0.23–0.28)	0.35 (0.28–0.42)
4 (2014–2015)	2506	0	0.080 (0.072–0.088)	0.033 (0.030–0.036)	0.071 (0.059–0.082)	0.22 (0.19–0.26)	0.32 (0.27–0.37)
Males, 3–79 years							
2 (2009–2011)	1261	0	0.086 (0.076–0.098)	0.035 (0.031–0.039)	0.079 (0.069–0.090)	0.26 (0.18–0.35)	0.42 (0.29–0.55)
3 (2012–2013)	1241	0	0.083 (0.074–0.093)	0.031 (0.027–0.036)	0.078 (0.063–0.093)	0.26 (0.24–0.27)	0.35 (0.25–0.44)
4 (2014–2015)	1250	0	0.076 (0.065–0.088)	0.031 (0.027–0.036)	0.068 (0.056–0.081)	0.22 (0.14–0.31)	0.30 (0.23–0.36)
Females, 3–79 years							
2 (2009–2011)	1244	0	0.087 (0.079–0.095)	0.042 (0.037–0.046)	0.079 (0.072–0.086)	0.19 (0.15–0.23)	0.31[E] (0.15–0.47)
3 (2012–2013)	1263	0	0.090 (0.081–0.099)	0.036 (0.033–0.039)	0.084 (0.073–0.095)	0.26 (0.20–0.32)	0.35 (0.26–0.45)
4 (2014–2015)	1256	0	0.084 (0.074–0.095)	0.035 (0.030–0.041)	0.074 (0.061–0.088)	0.22[E] (0.13–0.32)	0.34 (0.26–0.42)
3–5 years							
2 (2009–2011)	500	0	0.14 (0.12–0.15)	0.067 (0.059–0.076)	0.13 (0.11–0.15)	0.29 (0.25–0.33)	0.36 (0.27–0.44)
3 (2012–2013)	489	0	0.13 (0.12–0.14)	0.065 (0.056–0.074)	0.11 (0.097–0.12)	0.28 (0.24–0.31)	0.41 (0.31–0.51)
4 (2014–2015)	476	0	0.13 (0.11–0.14)	0.068 (0.059–0.078)	0.11 (0.092–0.14)	0.24 (0.18–0.30)	0.34 (0.24–0.45)
6–11 years							
2 (2009–2011)	507	0	0.098 (0.087–0.11)	0.049 (0.039–0.058)	0.087 (0.075–0.10)	0.20 (0.14–0.25)	0.27[E] (0.098–0.44)
3 (2012–2013)	501	0	0.088 (0.076–0.10)	0.043 (0.038–0.048)	0.082 (0.064–0.10)	0.18 (0.13–0.24)	0.24 (0.17–0.30)
4 (2014–2015)	510	0	0.093 (0.084–0.10)	0.045 (0.037–0.052)	0.086 (0.076–0.096)	0.20 (0.15–0.25)	0.30 (0.20–0.40)
12–19 years							
2 (2009–2011)	504	0	0.072 (0.064–0.081)	0.037 (0.034–0.039)	0.068 (0.061–0.074)	0.14[E] (0.060–0.22)	0.23[E] (0.13–0.32)
3 (2012–2013)	505	0	0.069 (0.062–0.077)	0.034 (0.029–0.038)	0.060 (0.051–0.068)	0.15 (0.11–0.19)	0.24[E] (0.13–0.36)
4 (2014–2015)	497	0	0.068 (0.060–0.077)	0.034 (0.032–0.036)	0.062 (0.055–0.069)	0.15 (0.097–0.20)	0.22 (0.14–0.29)
20–39 years							
2 (2009–2011)	353	0	0.081 (0.071–0.093)	0.038 (0.033–0.042)	0.069 (0.055–0.082)	0.23[E] (0.12–0.33)	0.38[E] (0.23–0.52)
3 (2012–2013)	350	0	0.084 (0.070–0.10)	0.031 (0.025–0.038)	0.081 (0.065–0.098)	0.25 (0.20–0.31)	0.34 (0.22–0.47)
4 (2014–2015)	358	0	0.076 (0.064–0.091)	0.031 (0.026–0.037)	0.074 (0.053–0.095)	0.20 (0.15–0.24)	0.23[E] (0.13–0.32)
40–59 years							
2 (2009–2011)	356	0	0.092 (0.083–0.10)	0.037 (0.032–0.043)	0.086 (0.073–0.099)	0.27 (0.18–0.35)	0.45 (0.32–0.58)
3 (2012–2013)	311	0	0.092 (0.081–0.10)	0.033 (0.026–0.040)	0.097 (0.087–0.11)	0.28 (0.20–0.35)	0.34 (0.22–0.45)
4 (2014–2015)	312	0	0.080 (0.068–0.094)	0.032 (0.025–0.039)	0.065 (0.052–0.077)	0.30 (0.24–0.35)	0.34[E] (<LOD–0.48)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	285	0	0.085 (0.075–0.096)	0.039 (0.032–0.045)	0.077 (0.071–0.083)	0.18[E] (0.11–0.24)	0.30[E] (0.13–0.47)
3 (2012–2013)	348	0	0.083 (0.069–0.10)	0.030 (0.022–0.038)	0.069 (0.059–0.079)	0.33 (0.24–0.41)	0.43[E] (0.18–0.68)
4 (2014–2015)	353	0	0.081 (0.072–0.091)	0.035 (0.029–0.041)	0.071 (0.062–0.080)	0.25 (0.16–0.34)	0.40[E] (0.23–0.57)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 14.7.7

4-Hydroxyphenanthrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2519	0.08	0.025 (0.022–0.027)	0.0070 (0.0059–0.0081)	0.023 (0.020–0.026)	0.091 (0.074–0.11)	0.13 (0.11–0.15)
3 (2012–2013)	2495	4.33	0.021 (0.019–0.023)	0.0055 (0.0044–0.0065)	0.020 (0.016–0.023)	0.086 (0.068–0.10)	0.14 (0.11–0.18)
4 (2014–2015)	2504	2.52	0.023 (0.021–0.025)	0.0062 (0.0048–0.0076)	0.023 (0.019–0.027)	0.089 (0.078–0.10)	0.12 (0.084–0.15)
Males, 3–79 years							
2 (2009–2011)	1266	0	0.027 (0.023–0.031)	0.0075 (0.0057–0.0094)	0.026 (0.020–0.031)	0.10 (0.074–0.13)	0.15 (0.10–0.20)
3 (2012–2013)	1236	3.32	0.024 (0.021–0.027)	0.0067 (0.0047–0.0087)	0.023 (0.020–0.026)	0.097 (0.074–0.12)	0.15 (0.11–0.19)
4 (2014–2015)	1248	2.32	0.024 (0.020–0.028)	0.0068 (0.0052–0.0085)	0.024 (0.018–0.029)	0.084 (0.065–0.10)	0.13[E] (0.079–0.19)
Females, 3–79 years							
2 (2009–2011)	1253	0.16	0.023 (0.021–0.025)	0.0061 (0.0049–0.0073)	0.022 (0.019–0.024)	0.085 (0.065–0.10)	0.13 (0.095–0.16)
3 (2012–2013)	1259	5.32	0.018 (0.015–0.023)	0.0044 (0.0031–0.0058)	0.017 (0.011–0.022)	0.076 (0.052–0.10)	0.13[E] (0.079–0.18)
4 (2014–2015)	1256	2.71	0.022 (0.019–0.026)	0.0054 (0.0035–0.0073)	0.022 (0.017–0.026)	0.099 (0.078–0.12)	0.11[E] (0.066–0.16)
3–5 years							
2 (2009–2011)	505	0	0.017 (0.015–0.020)	0.0060 (0.0047–0.0072)	0.017 (0.014–0.020)	0.051 (0.042–0.061)	0.063[E] (0.032–0.093)
3 (2012–2013)	488	4.30	0.014 (0.012–0.016)	0.0045 (0.0031–0.0059)	0.013 (0.011–0.016)	0.047 (0.038–0.056)	0.062 (0.051–0.073)
4 (2014–2015)	475	3.16	0.013 (0.010–0.016)	0.0045 (<LOD–0.0061)	0.013 (0.0096–0.016)	0.040 (0.031–0.048)	0.056 (0.041–0.070)
6–11 years							
2 (2009–2011)	510	0	0.019 (0.016–0.023)	0.0067 (0.0057–0.0077)	0.019 (0.014–0.023)	0.057 (0.040–0.075)	0.074 (0.049–0.099)
3 (2012–2013)	500	4.40	0.014 (0.013–0.017)	0.0046 (0.0033–0.0059)	0.015 (0.012–0.018)	0.041 (0.032–0.050)	0.062 (0.050–0.073)
4 (2014–2015)	510	2.35	0.017 (0.015–0.020)	0.0052 (0.0039–0.0066)	0.017 (0.014–0.020)	0.052 (0.036–0.067)	0.074 (0.051–0.097)
12–19 years							
2 (2009–2011)	505	0.20	0.023 (0.020–0.025)	0.0079 (0.0057–0.010)	0.022 (0.020–0.025)	0.067 (0.053–0.081)	0.094 (0.062–0.12)
3 (2012–2013)	504	4.56	0.021 (0.018–0.024)	0.0068 (0.0053–0.0083)	0.018 (0.015–0.021)	0.077 (0.052–0.10)	0.11[E] (0.070–0.16)
4 (2014–2015)	497	2.62	0.021 (0.018–0.023)	0.0054[E] (0.0033–0.0075)	0.021 (0.019–0.024)	0.070 (0.052–0.089)	0.11 (0.077–0.13)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	0	0.026 (0.022–0.031)	0.0070 (0.0050–0.0090)	0.027 (0.019–0.035)	0.088 (0.057–0.12)	0.13 (0.086–0.18)
3 (2012–2013)	349	2.87	0.027 (0.022–0.034)	0.0072 (0.0054–0.0090)	0.028 (0.020–0.036)	0.095[E] (0.057–0.13)	0.16[E] (0.088–0.24)
4 (2014–2015)	358	3.07	0.027 (0.022–0.033)	0.0065 (0.0043–0.0088)	0.030 (0.023–0.037)	0.099 (0.081–0.12)	0.13 (0.090–0.17)
40–59 years							
2 (2009–2011)	357	0	0.027 (0.023–0.032)	0.0082 (0.0056–0.011)	0.024 (0.018–0.029)	0.11 (0.085–0.14)	0.15[E] (0.097–0.21)
3 (2012–2013)	308	4.55	0.021 (0.017–0.025)	0.0045[E] (<LOD–0.0067)	0.019 (0.014–0.025)	0.090[E] (0.050–0.13)	0.12[E] (0.075–0.17)
4 (2014–2015)	311	1.93	0.023 (0.020–0.027)	0.0064 (0.0045–0.0084)	0.022[E] (0.013–0.031)	0.079[E] (0.035–0.12)	F
60–79 years							
2 (2009–2011)	287	0.35	0.023 (0.020–0.026)	0.0060 (0.0047–0.0074)	0.021 (0.017–0.026)	0.086[E] (0.054–0.12)	0.14[E] (0.075–0.21)
3 (2012–2013)	346	5.20	0.020 (0.016–0.024)	0.0047[E] (<LOD–0.0071)	0.018 (0.013–0.022)	F	0.25[E] (0.12–0.39)
4 (2014–2015)	353	1.70	0.023 (0.020–0.028)	0.0060[E] (0.0038–0.0083)	0.021 (0.017–0.026)	0.099 (0.080–0.12)	0.14[E] (0.086–0.20)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.8

4-Hydroxyphenanthrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2509	0.08	0.024 (0.022–0.027)	0.0094 (0.0085–0.010)	0.022 (0.019–0.025)	0.077 (0.060–0.093)	0.11 (0.085–0.14)
3 (2012–2013)	2494	4.33	0.022 (0.020–0.024)	0.0077 (0.0066–0.0088)	0.019 (0.016–0.021)	0.076 (0.067–0.085)	0.10 (0.083–0.12)
4 (2014–2015)	2504	2.52	0.021 (0.019–0.023)	0.0071 (0.0060–0.0082)	0.018 (0.015–0.020)	0.076 (0.066–0.085)	0.099 (0.084–0.11)
Males, 3–79 years							
2 (2009–2011)	1262	0	0.023 (0.020–0.026)	0.0082 (0.0073–0.0091)	0.019 (0.015–0.023)	0.084 (0.061–0.11)	0.13 (0.087–0.17)
3 (2012–2013)	1236	3.32	0.020 (0.017–0.023)	0.0071 (0.0056–0.0087)	0.017 (0.014–0.019)	0.077 (0.063–0.091)	0.099 (0.081–0.12)
4 (2014–2015)	1248	2.32	0.019 (0.016–0.022)	0.0067 (0.0056–0.0077)	0.016 (0.013–0.018)	0.077[E] (0.047–0.11)	0.093 (0.075–0.11)
Females, 3–79 years							
2 (2009–2011)	1247	0.16	0.026 (0.023–0.029)	0.0099 (0.0091–0.011)	0.025 (0.020–0.029)	0.066 (0.048–0.083)	0.10 (0.072–0.13)
3 (2012–2013)	1258	5.32	0.023 (0.021–0.026)	0.0081 (0.0069–0.0094)	0.022 (0.019–0.025)	0.076 (0.061–0.090)	0.11 (0.079–0.14)
4 (2014–2015)	1256	2.71	0.023 (0.020–0.026)	0.0081 (0.0068–0.0093)	0.021 (0.018–0.025)	0.075 (0.054–0.097)	0.11 (0.074–0.15)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
2 (2009–2011)	504	0	0.031 (0.027–0.035)	0.011 (0.0083–0.015)	0.027 (0.024–0.031)	0.084 (0.068–0.10)	0.099 (0.073–0.13)
3 (2012–2013)	487	4.30	0.026 (0.024–0.029)	0.011 (0.010–0.012)	0.023 (0.019–0.027)	0.068 (0.062–0.074)	0.095 (0.075–0.12)
4 (2014–2015)	475	3.16	0.023 (0.020–0.027)	0.0096 (<LOD–0.012)	0.023 (0.017–0.029)	0.056 (0.040–0.072)	0.077 (0.063–0.091)
6–11 years							
2 (2009–2011)	508	0	0.023 (0.019–0.026)	0.010 (0.0085–0.011)	0.019 (0.016–0.023)	0.060 (0.041–0.079)	0.080 (0.061–0.099)
3 (2012–2013)	500	4.40	0.018 (0.016–0.021)	0.0078 (0.0066–0.0089)	0.017 (0.014–0.020)	0.045 (0.040–0.050)	0.052 (0.043–0.061)
4 (2014–2015)	510	2.35	0.019 (0.017–0.021)	0.0080 (0.0061–0.0099)	0.016 (0.013–0.018)	0.054 (0.035–0.072)	0.075 (0.058–0.092)
12–19 years							
2 (2009–2011)	503	0.20	0.017 (0.016–0.020)	0.0080 (0.0071–0.0088)	0.015 (0.014–0.017)	0.044 (0.032–0.055)	0.059 (0.040–0.079)
3 (2012–2013)	504	4.56	0.016 (0.014–0.018)	0.0070 (0.0060–0.0080)	0.013 (0.011–0.016)	0.036 (0.025–0.047)	F
4 (2014–2015)	497	2.62	0.015 (0.013–0.017)	0.0052 (0.0039–0.0065)	0.014 (0.011–0.016)	0.046 (0.033–0.059)	0.070 (0.047–0.094)
20–39 years							
2 (2009–2011)	353	0	0.023 (0.019–0.027)	0.0083 (0.0069–0.0096)	0.020 (0.015–0.024)	0.069[E] (0.041–0.097)	0.11[E] (0.066–0.16)
3 (2012–2013)	349	2.87	0.021 (0.017–0.026)	0.0080 (0.0064–0.0096)	0.017 (0.012–0.022)	0.077 (0.059–0.095)	0.093 (0.071–0.11)
4 (2014–2015)	358	3.07	0.021 (0.018–0.026)	0.0075 (0.0061–0.0089)	0.021 (0.017–0.026)	0.079 (0.055–0.10)	0.088 (0.071–0.10)
40–59 years							
2 (2009–2011)	355	0	0.027 (0.024–0.031)	0.0098 (0.0089–0.011)	0.025 (0.019–0.031)	0.091 (0.073–0.11)	0.14 (0.091–0.19)
3 (2012–2013)	308	4.55	0.024 (0.020–0.029)	0.0080 (<LOD–0.011)	0.024 (0.018–0.030)	0.083 (0.060–0.11)	0.11[E] (0.070–0.15)
4 (2014–2015)	311	1.93	0.021 (0.018–0.024)	0.0074 (0.0060–0.0088)	0.016 (0.011–0.021)	0.083 (0.055–0.11)	0.11[E] (<LOD–0.16)
60–79 years							
2 (2009–2011)	286	0.35	0.027 (0.023–0.031)	0.0095 (0.0080–0.011)	0.027 (0.021–0.032)	0.081[E] (0.043–0.12)	0.13[E] (0.073–0.19)
3 (2012–2013)	346	5.20	0.023 (0.019–0.027)	0.0074 (<LOD–0.0090)	0.020 (0.015–0.024)	0.096[E] (0.059–0.13)	0.14[E] (0.075–0.21)
4 (2014–2015)	353	1.70	0.023 (0.019–0.027)	0.0079 (0.0054–0.011)	0.018 (0.016–0.020)	0.087[E] (0.043–0.13)	0.16[E] (0.077–0.24)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.9

9-Hydroxyphenanthrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2474	5.86	0.039 (0.034–0.044)	0.0075 (0.0066–0.0084)	0.036 (0.029–0.043)	0.24 (0.18–0.31)	0.41 (0.33–0.49)
3 (2012–2013)	2295	3.66	0.036 (0.033–0.040)	0.0080 (0.0059–0.010)	0.034 (0.029–0.038)	0.19 (0.14–0.23)	0.32 (0.21–0.43)
4 (2014–2015)	2361	0.04	0.045 (0.041–0.050)	0.013 (0.012–0.015)	0.037 (0.034–0.040)	0.20[E] (0.12–0.28)	0.33 (0.25–0.41)
Males, 3–79 years							
2 (2009–2011)	1249	5.44	0.043 (0.035–0.052)	0.0080 (0.0065–0.0094)	0.043 (0.033–0.053)	0.25[E] (0.15–0.35)	0.49 (0.32–0.65)
3 (2012–2013)	1155	2.60	0.040 (0.036–0.045)	0.0099 (0.0086–0.011)	0.037 (0.032–0.041)	0.19 (0.15–0.22)	0.26[E] (0.12–0.40)
4 (2014–2015)	1174	0.09	0.046 (0.039–0.055)	0.014 (0.012–0.016)	0.039 (0.031–0.047)	F	0.36[E] (0.23–0.50)
Females, 3–79 years							
2 (2009–2011)	1225	6.29	0.035 (0.031–0.040)	0.0070 (0.0053–0.0086)	0.032 (0.028–0.036)	0.23[E] (0.11–0.34)	0.38 (0.26–0.50)
3 (2012–2013)	1140	4.74	0.032 (0.026–0.039)	0.0070 (0.0045–0.0095)	0.032 (0.026–0.037)	0.19[E] (0.083–0.29)	0.39 (0.25–0.53)
4 (2014–2015)	1187	0	0.045 (0.039–0.051)	0.012 (0.010–0.015)	0.036 (0.032–0.040)	0.25[E] (0.14–0.36)	0.32 (0.25–0.39)
3–5 years							
2 (2009–2011)	490	11.43	0.018 (0.015–0.022)	<LOD	0.020 (0.017–0.023)	0.072 (0.054–0.089)	0.095[E] (0.041–0.15)
3 (2012–2013)	426	4.46	0.019 (0.017–0.022)	0.0069[E] (<LOD–0.0099)	0.019 (0.015–0.023)	0.057 (0.042–0.072)	0.072 (0.051–0.093)
4 (2014–2015)	431	0.23	0.025 (0.023–0.028)	0.011 (0.010–0.013)	0.024 (0.021–0.026)	0.056 (0.050–0.063)	0.086 (0.067–0.10)
6–11 years							
2 (2009–2011)	502	5.78	0.019 (0.015–0.023)	0.0044[E] (<LOD–0.0075)	0.022 (0.017–0.026)	0.056 (0.043–0.069)	0.076 (0.055–0.097)
3 (2012–2013)	447	4.92	0.019 (0.017–0.022)	0.0058[E] (<LOD–0.0091)	0.021 (0.019–0.023)	0.048 (0.041–0.054)	F
4 (2014–2015)	464	0	0.028 (0.025–0.031)	0.012 (0.011–0.014)	0.026 (0.024–0.029)	0.062 (0.046–0.079)	0.084[E] (0.051–0.12)
12–19 years							
2 (2009–2011)	499	5.41	0.027 (0.023–0.032)	0.0073 (0.0058–0.0089)	0.029 (0.023–0.035)	0.099 (0.076–0.12)	0.15[E] (0.092–0.20)
3 (2012–2013)	480	3.75	0.026 (0.021–0.031)	0.0070[E] (0.0040–0.0099)	0.022 (0.018–0.026)	0.10 (0.066–0.13)	F
4 (2014–2015)	477	0	0.030 (0.027–0.034)	0.012 (0.0096–0.014)	0.028 (0.022–0.033)	0.078 (0.065–0.092)	0.092[E] (0.040–0.14)
20–39 years							
2 (2009–2011)	348	3.45	0.041 (0.034–0.050)	0.0088[E] (0.0055–0.012)	0.040 (0.030–0.050)	0.23[E] (0.088–0.38)	0.39[E] (0.20–0.58)
3 (2012–2013)	331	2.11	0.040 (0.031–0.051)	0.0094 (0.0072–0.012)	0.036 (0.030–0.043)	0.19 (0.13–0.25)	0.23[E] (0.088–0.36)
4 (2014–2015)	344	0	0.048 (0.039–0.059)	0.014 (0.010–0.017)	0.045 (0.036–0.054)	0.24[E] (0.10–0.37)	0.32 (0.23–0.42)
40–59 years							
2 (2009–2011)	350	3.14	0.049 (0.040–0.059)	0.0089 (0.0071–0.011)	0.045 (0.034–0.056)	0.31 (0.23–0.38)	0.48 (0.40–0.56)
3 (2012–2013)	287	4.18	0.041 (0.031–0.054)	0.0080 (0.0052–0.011)	0.043 (0.031–0.055)	0.22[E] (0.086–0.35)	0.38[E] (0.22–0.53)
4 (2014–2015)	302	0	0.050 (0.041–0.061)	0.014 (0.012–0.016)	0.040 (0.032–0.048)	0.27[E] (0.11–0.44)	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	285	3.51	0.043 (0.033–0.056)	0.0065[E] (<LOD–0.0092)	0.035 (0.024–0.045)	0.31[E] (0.19–0.42)	0.60[E] (0.34–0.85)
3 (2012–2013)	324	1.85	0.041 (0.034–0.050)	F	0.040 (0.031–0.049)	0.28[E] (0.076–0.49)	F
4 (2014–2015)	343	0	0.054 (0.046–0.065)	0.014 (0.011–0.017)	0.040 (0.026–0.054)	0.30 (0.21–0.38)	0.41[E] (0.24–0.59)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.7.10

9-Hydroxyphenanthrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2464	5.86	0.039 (0.034–0.044)	0.010 (0.0091–0.011)	0.032 (0.026–0.038)	0.22 (0.16–0.28)	0.34 (0.26–0.42)
3 (2012–2013)	2294	3.66	0.037 (0.034–0.041)	0.011 (0.0095–0.012)	0.032 (0.028–0.037)	0.17 (0.13–0.21)	0.29 (0.20–0.38)
4 (2014–2015)	2361	0.04	0.041 (0.038–0.044)	0.014 (0.013–0.015)	0.032 (0.028–0.035)	0.19 (0.16–0.23)	0.29 (0.22–0.36)
Males, 3–79 years							
2 (2009–2011)	1245	5.44	0.037 (0.030–0.045)	0.0099 (0.0077–0.012)	0.029 (0.022–0.035)	0.23[E] (0.14–0.33)	0.38 (0.27–0.48)
3 (2012–2013)	1155	2.60	0.033 (0.028–0.040)	0.0097 (0.0079–0.012)	0.030 (0.023–0.037)	0.16 (0.11–0.21)	0.23 (0.16–0.30)
4 (2014–2015)	1174	0.09	0.036 (0.031–0.042)	0.012 (0.011–0.014)	0.029 (0.026–0.031)	0.19[E] (0.11–0.27)	0.25 (0.16–0.33)
Females, 3–79 years							
2 (2009–2011)	1219	6.29	0.041 (0.035–0.048)	0.010[E] (0.0060–0.014)	0.035 (0.027–0.043)	0.21[E] (0.13–0.29)	0.29[E] (0.18–0.39)
3 (2012–2013)	1139	4.74	0.041 (0.037–0.046)	0.012 (0.010–0.013)	0.035 (0.030–0.039)	0.21[E] (0.12–0.30)	0.39 (0.25–0.53)
4 (2014–2015)	1187	0	0.046 (0.040–0.054)	0.016 (0.014–0.017)	0.036 (0.030–0.041)	0.20[E] (0.11–0.29)	0.34[E] (0.18–0.51)
3–5 years							
2 (2009–2011)	489	11.43	0.032 (0.027–0.037)	<LOD	0.037 (0.030–0.043)	0.11 (0.086–0.13)	0.14[E] (0.086–0.20)
3 (2012–2013)	425	4.46	0.037 (0.035–0.040)	0.017 (<LOD–0.020)	0.036 (0.032–0.040)	0.081 (0.066–0.095)	0.10 (0.086–0.12)
4 (2014–2015)	431	0.23	0.046 (0.043–0.050)	0.024 (0.023–0.026)	0.043 (0.037–0.048)	0.094 (0.083–0.10)	0.11 (0.096–0.13)
6–11 years							
2 (2009–2011)	500	5.78	0.022 (0.018–0.027)	0.0068[E] (<LOD–0.010)	0.025 (0.021–0.029)	0.053 (0.044–0.063)	0.071 (0.050–0.093)
3 (2012–2013)	447	4.92	0.025 (0.022–0.028)	0.012 (<LOD–0.015)	0.023 (0.020–0.025)	0.057 (0.046–0.069)	0.082[E] (0.037–0.13)
4 (2014–2015)	464	0	0.031 (0.028–0.034)	0.016 (0.015–0.018)	0.028 (0.025–0.031)	0.060 (0.047–0.073)	F

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	497	5.41	0.021 (0.018–0.024)	0.0076 (0.0060–0.0092)	0.020 (0.017–0.022)	0.059[E] (0.037–0.082)	0.087[E] (0.043–0.13)
3 (2012–2013)	480	3.75	0.019 (0.017–0.022)	0.0087 (0.0071–0.010)	0.016 (0.014–0.018)	0.056[E] (0.030–0.082)	0.097[E] (0.042–0.15)
4 (2014–2015)	477	0	0.022 (0.020–0.025)	0.010 (0.0091–0.011)	0.020 (0.017–0.022)	0.047[E] (0.027–0.067)	0.086[E] (0.029–0.14)
20–39 years							
2 (2009–2011)	346	3.45	0.037 (0.029–0.047)	0.010[E] (0.0047–0.015)	0.028 (0.020–0.035)	0.26[E] (0.11–0.41)	0.36[E] (0.22–0.51)
3 (2012–2013)	331	2.11	0.030 (0.023–0.040)	0.010 (0.0066–0.013)	0.025[E] (0.012–0.038)	0.12[E] (0.048–0.20)	0.17[E] (0.093–0.25)
4 (2014–2015)	344	0	0.039 (0.031–0.049)	0.013 (0.011–0.015)	0.032 (0.021–0.042)	0.17[E] (0.089–0.25)	0.25[E] (0.14–0.36)
40–59 years							
2 (2009–2011)	348	3.14	0.049 (0.040–0.060)	0.012[E] (0.0067–0.018)	0.043 (0.030–0.056)	0.25 (0.19–0.31)	0.40[E] (0.25–0.55)
3 (2012–2013)	287	4.18	0.048 (0.038–0.060)	0.011 (0.0076–0.015)	0.042[E] (0.024–0.061)	0.24[E] (0.10–0.37)	0.39[E] (0.23–0.56)
4 (2014–2015)	302	0	0.046 (0.039–0.055)	0.014 (0.012–0.016)	0.033 (0.022–0.043)	0.25 (0.17–0.34)	0.33[E] (<LOD–0.56)
60–79 years							
2 (2009–2011)	284	3.51	0.051 (0.040–0.064)	0.013[E] (<LOD–0.019)	0.039[E] (0.023–0.056)	0.29[E] (0.18–0.40)	F
3 (2012–2013)	324	1.85	0.048 (0.040–0.058)	0.013 (<LOD–0.017)	0.041 (0.031–0.051)	0.25[E] (0.14–0.36)	0.37[E] (0.21–0.54)
4 (2014–2015)	343	0	0.053 (0.045–0.061)	0.017 (0.015–0.019)	0.039 (0.030–0.049)	0.23[E] (0.089–0.38)	0.47[E] (0.20–0.73)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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14.8 PYRENE

Pyrene is a polycyclic aromatic hydrocarbon (PAH) with four fused benzene rings. Pyrene is used as an intermediate in the synthesis of dyes and fluorescent molecular probes for biomedical research (WHO, 1998).

Pyrene absorption occurs rapidly in the respiratory tract, but more slowly through the gastrointestinal tract and skin (Faust, 1993). After oral administration in rats, pyrene has been found predominantly in the gastrointestinal tract (Mitchell and Tu, 1979). 1-Hydroxypyrene has been identified as the primary metabolite of pyrene (IARC, 2010). In humans, urinary elimination of 1-hydroxypyrene is triphasic with half-lives of 5, 22, and 408 hours (ACGIH, 2005). Monitoring studies of pyrene exposure can measure urinary levels of 1-hydroxypyrene to assess recent and chronic exposures (Becker et al., 2003; CDC, 2009; Hopf et al., 2009; Jongeneelen et al., 1985). Urinary 1-hydroxypyrene may also serve as a useful biomarker for total PAH exposure, given that pyrene is found in most PAH mixtures (Hopf et al., 2009; WHO, 1998).

Subchronic oral exposure to pyrene results in kidney and liver effects in laboratory animals, and the liver has been suggested as the main target organ for toxicity (Faust, 1993; TRL, 1989). Because the carcinogenic evidence is limited, the International Agency for Research on Cancer has classified pyrene as Group 3, not classifiable as to its carcinogenicity to humans (IARC, 2010).

1-Hydroxypyrene was measured in the urine of 73 non-smoking, non-occupationally exposed residents (aged 16–64 years) living approximately 1 km from an aluminum plant in Baie-Comeau, Quebec. The geometric mean levels ranged from 0.090 µg/g to 0.111 µg/g creatinine, compared with from 0.048 µg/g to 0.077 µg/g creatinine for 71 control individuals living at least 11 km from the plant (Bouchard et al., 2009). In a pilot biomonitoring study carried out in Hamilton, Ontario, with 19 pregnant women aged 19–42 years, the geometric means for 1-hydroxypyrene in urine was 0.1359 µg/g creatinine (Nethery et al., 2012).

1-Hydroxypyrene was analyzed in the urine of Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years, and is presented as both µg/L and µg/g creatinine. Finding a measurable amount of 1-hydroxypyrene in urine is an indicator of exposure to pyrene and does not necessarily mean that an adverse health effect will occur.

Table 14.8.1

1-Hydroxypyrene — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2422	0.04	0.11 (0.099–0.12)	0.031 (0.027–0.034)	0.10 (0.092–0.11)	0.35 (0.31–0.39)	0.57 (0.47–0.68)
3 (2012–2013)	2410	0.04	0.088 (0.078–0.10)	0.027 (0.020–0.035)	0.087 (0.078–0.096)	0.31 (0.26–0.35)	0.46 (0.38–0.55)
4 (2014–2015)	2409	0.04	0.096 (0.087–0.11)	0.028 (0.025–0.031)	0.093 (0.083–0.10)	0.29 (0.23–0.35)	0.51 (0.40–0.63)
Males, 3–79 years							
2 (2009–2011)	1206	0	0.12 (0.11–0.14)	0.040 (0.034–0.045)	0.12 (0.10–0.13)	0.38 (0.25–0.50)	0.59 (0.46–0.73)
3 (2012–2013)	1178	0.08	0.10 (0.090–0.12)	0.033 (0.023–0.042)	0.094 (0.088–0.10)	0.36 (0.30–0.43)	0.52 (0.45–0.60)
4 (2014–2015)	1186	0	0.10 (0.086–0.12)	0.034 (0.025–0.042)	0.093 (0.080–0.11)	0.32[E] (0.16–0.48)	0.52 (0.38–0.66)
Females, 3–79 years							
2 (2009–2011)	1216	0.08	0.095 (0.088–0.10)	0.026 (0.021–0.031)	0.095 (0.085–0.10)	0.33 (0.28–0.37)	0.48 (0.34–0.62)
3 (2012–2013)	1232	0	0.077 (0.064–0.092)	0.022[E] (0.014–0.031)	0.077 (0.061–0.093)	0.26 (0.21–0.30)	0.36 (0.25–0.48)
4 (2014–2015)	1223	0.08	0.090 (0.082–0.099)	0.024 (0.019–0.029)	0.093 (0.081–0.10)	0.29 (0.25–0.32)	0.49[E] (0.30–0.69)
3–5 years							
2 (2009–2011)	504	0	0.12 (0.11–0.13)	0.050 (0.041–0.059)	0.11 (0.10–0.12)	0.27 (0.20–0.34)	0.40 (0.30–0.51)
3 (2012–2013)	493	0	0.093 (0.077–0.11)	0.029 (0.023–0.036)	0.098 (0.081–0.12)	0.26 (0.21–0.31)	0.31 (0.25–0.37)
4 (2014–2015)	476	0	0.093 (0.078–0.11)	0.030 (0.020–0.041)	0.096 (0.078–0.11)	0.23 (0.19–0.27)	0.33[E] (0.21–0.46)
6–11 years							
2 (2009–2011)	507	0	0.13 (0.11–0.15)	0.049 (0.039–0.058)	0.12 (0.096–0.14)	0.34 (0.25–0.42)	0.47 (0.34–0.60)
3 (2012–2013)	501	0	0.092 (0.084–0.10)	0.032 (0.024–0.039)	0.097 (0.088–0.11)	0.21 (0.16–0.26)	0.28 (0.22–0.33)
4 (2014–2015)	510	0	0.099 (0.088–0.11)	0.035 (0.026–0.044)	0.097 (0.084–0.11)	0.25 (0.22–0.28)	0.33 (0.26–0.41)
12–19 years							
2 (2009–2011)	480	0	0.15 (0.14–0.17)	0.050[E] (0.031–0.069)	0.15 (0.13–0.17)	0.44 (0.36–0.52)	0.62 (0.45–0.79)
3 (2012–2013)	473	0	0.12 (0.097–0.14)	0.040[E] (0.024–0.057)	0.11 (0.092–0.13)	0.34 (0.24–0.43)	0.47 (0.34–0.60)
4 (2014–2015)	462	0.22	0.11 (0.087–0.13)	0.032 (0.021–0.042)	0.10 (0.083–0.12)	0.33 (0.21–0.44)	0.47 (0.31–0.63)
20–39 years							
2 (2009–2011)	327	0	0.13 (0.11–0.15)	0.041 (0.027–0.054)	0.12 (0.10–0.14)	0.35 (0.29–0.42)	0.48[E] (0.27–0.69)
3 (2012–2013)	308	0	0.12 (0.10–0.15)	0.037 (0.025–0.048)	0.11 (0.077–0.15)	0.36 (0.25–0.47)	F
4 (2014–2015)	329	0	0.12 (0.10–0.13)	0.030[E] (0.019–0.042)	0.12 (0.092–0.15)	0.31[E] (0.13–0.49)	0.55 (0.40–0.69)
40–59 years							
2 (2009–2011)	329	0.30	0.10 (0.084–0.12)	0.026[E] (0.012–0.039)	0.094 (0.076–0.11)	0.44[E] (0.22–0.65)	0.58 (0.47–0.69)
3 (2012–2013)	296	0	0.080 (0.067–0.095)	0.027[E] (0.016–0.039)	0.079 (0.063–0.095)	0.26[E] (0.14–0.38)	0.43[E] (0.26–0.60)
4 (2014–2015)	293	0	0.095 (0.082–0.11)	0.029 (0.026–0.033)	0.092 (0.072–0.11)	0.37[E] (0.22–0.52)	0.87[E] (0.28–1.5)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	275	0	0.067 (0.057–0.079)	0.024 (0.018–0.030)	0.062 (0.048–0.076)	0.20 (0.16–0.24)	F
3 (2012–2013)	339	0.29	0.064 (0.051–0.081)	0.015[E] (0.0086–0.022)	0.060 (0.041–0.079)	0.31 (0.20–0.41)	0.50[E] (0.29–0.71)
4 (2014–2015)	339	0	0.072 (0.064–0.081)	0.022 (0.019–0.025)	0.067 (0.060–0.075)	0.26 (0.20–0.31)	0.35[E] (0.22–0.48)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 14.8.2

1-Hydroxypyrene (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2412	0.04	0.11 (0.10–0.12)	0.045 (0.042–0.048)	0.099 (0.096–0.10)	0.28 (0.24–0.33)	0.40 (0.31–0.50)
3 (2012–2013)	2409	0.04	0.094 (0.084–0.10)	0.034 (0.028–0.040)	0.088 (0.079–0.096)	0.25 (0.20–0.29)	0.36 (0.24–0.47)
4 (2014–2015)	2409	0.04	0.088 (0.078–0.098)	0.036 (0.030–0.042)	0.078 (0.065–0.091)	0.24 (0.21–0.27)	0.34 (0.30–0.38)
Males, 3–79 years							
2 (2009–2011)	1202	0	0.11 (0.093–0.12)	0.042 (0.036–0.047)	0.098 (0.091–0.10)	0.31 (0.24–0.39)	0.43[E] (0.28–0.59)
3 (2012–2013)	1178	0.08	0.089 (0.076–0.10)	0.030 (0.020–0.039)	0.087 (0.075–0.099)	0.25 (0.18–0.31)	0.35 (0.26–0.44)
4 (2014–2015)	1186	0	0.082 (0.067–0.099)	0.034 (0.027–0.040)	0.071 (0.052–0.089)	0.23 (0.15–0.30)	0.33 (0.25–0.42)
Females, 3–79 years							
2 (2009–2011)	1210	0.08	0.11 (0.10–0.12)	0.048 (0.044–0.052)	0.099 (0.094–0.11)	0.26 (0.21–0.31)	0.38 (0.28–0.49)
3 (2012–2013)	1231	0	0.099 (0.087–0.11)	0.038 (0.027–0.049)	0.088 (0.071–0.11)	0.25 (0.19–0.30)	0.42[E] (0.23–0.60)
4 (2014–2015)	1223	0.08	0.094 (0.083–0.11)	0.041 (0.034–0.048)	0.085 (0.074–0.096)	0.24 (0.21–0.28)	0.36 (0.29–0.43)
3–5 years							
2 (2009–2011)	503	0	0.21 (0.20–0.23)	0.11 (0.089–0.12)	0.20 (0.18–0.23)	0.41 (0.35–0.46)	0.51 (0.42–0.60)
3 (2012–2013)	492	0	0.18 (0.16–0.20)	0.094 (0.078–0.11)	0.17 (0.15–0.20)	0.34 (0.28–0.41)	0.43 (0.38–0.49)
4 (2014–2015)	476	0	0.17 (0.15–0.19)	0.082 (0.074–0.091)	0.16 (0.14–0.19)	0.33 (0.27–0.40)	0.38[E] (0.24–0.52)
6–11 years							
2 (2009–2011)	505	0	0.15 (0.13–0.16)	0.074 (0.063–0.085)	0.14 (0.12–0.15)	0.28 (0.22–0.34)	0.37 (0.26–0.49)
3 (2012–2013)	501	0	0.12 (0.11–0.13)	0.066 (0.057–0.074)	0.11 (0.091–0.12)	0.21 (0.17–0.25)	0.27 (0.23–0.31)
4 (2014–2015)	510	0	0.11 (0.097–0.12)	0.055 (0.047–0.063)	0.097 (0.091–0.10)	0.22 (0.18–0.26)	0.33[E] (0.19–0.48)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	478	0	0.12 (0.10–0.13)	0.056 (0.051–0.062)	0.10 (0.091–0.12)	0.28 (0.20–0.37)	0.39[E] (0.21–0.57)
3 (2012–2013)	473	0	0.089 (0.073–0.11)	0.044 (0.031–0.057)	0.087 (0.074–0.10)	0.19[E] (0.12–0.26)	0.26[E] (0.15–0.37)
4 (2014–2015)	462	0.22	0.076 (0.063–0.092)	0.035 (0.028–0.041)	0.073 (0.059–0.087)	0.18[E] (0.10–0.26)	0.27[E] (0.17–0.37)
20–39 years							
2 (2009–2011)	325	0	0.12 (0.096–0.14)	0.050 (0.035–0.065)	0.10 (0.083–0.12)	0.28 (0.18–0.38)	0.41 (0.27–0.54)
3 (2012–2013)	308	0	0.10 (0.087–0.12)	0.036 (0.026–0.047)	0.099 (0.087–0.11)	0.26[E] (0.083–0.44)	0.54[E] (0.20–0.88)
4 (2014–2015)	329	0	0.096 (0.083–0.11)	0.048 (0.039–0.057)	0.090 (0.071–0.11)	0.20 (0.17–0.23)	0.23[E] (0.13–0.34)
40–59 years							
2 (2009–2011)	327	0.30	0.10 (0.090–0.12)	0.043 (0.039–0.047)	0.094 (0.085–0.10)	0.33 (0.25–0.41)	0.59[E] (0.24–0.94)
3 (2012–2013)	296	0	0.093 (0.080–0.11)	0.033[E] (0.020–0.046)	0.088 (0.074–0.10)	0.26[E] (0.15–0.37)	0.38[E] (0.20–0.56)
4 (2014–2015)	293	0	0.086 (0.075–0.099)	0.034 (0.025–0.043)	0.070 (0.057–0.084)	0.30 (0.22–0.38)	0.36[E] (0.12–0.60)
60–79 years							
2 (2009–2011)	274	0	0.079 (0.069–0.091)	0.035 (0.028–0.042)	0.078 (0.072–0.084)	0.16 (0.13–0.20)	0.23[E] (<LOD–0.36)
3 (2012–2013)	339	0.29	0.073 (0.060–0.090)	0.028 (0.023–0.034)	0.062 (0.050–0.075)	0.23 (0.16–0.30)	0.35 (0.23–0.48)
4 (2014–2015)	339	0	0.070 (0.058–0.084)	0.028 (0.023–0.034)	0.062 (0.052–0.072)	0.24[E] (0.15–0.33)	0.37[E] (0.19–0.54)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

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SUMMARIES AND RESULTS FOR VOLATILE ORGANIC COMPOUNDS 15

15.1 BENZENE AND BENZENE METABOLITES

Benzene (CASRN 71-43-2) is a colourless liquid and volatile organic compound (VOC) that is naturally present in ambient air at low concentrations (Health Canada, 2009). It was first isolated and synthesized in the early 1800s and presently is commercially recovered from both coal and petroleum sources for industrial applications (ATSDR, 2007).

Benzene is used widely in industry as a solvent and as an intermediate in the production of a variety of chemicals, with typical end-products including plastics and elastomers, phenol and acetone, and nylon resins (ATSDR, 2007; Environment Canada and Health Canada, 1993). Benzene is also used at various stages in the manufacturing of synthetic fibres, rubbers, lubricants, dyes, detergents, drugs, and pesticides (ATSDR, 2007).

Benzene is released to the environment from natural and anthropogenic sources. It is naturally present in crude oil, and is formed during the incomplete combustion of organic materials (Environment Canada and Health Canada, 1993). Benzene enters the environment as a result of natural processes including petroleum seepage, weathering of rock and soil, volcanic activity, forest fires, and releases from plant life (Environment Canada and Health Canada, 1993). Anthropogenic sources include the production, storage, use, and transport of isolated benzene, crude oil, and other petroleum products. Examples include evaporative releases from gasoline at service stations and combustion by-products in the form of motor vehicle exhaust (Health Canada, 2009). Natural sources are generally considered to contribute less benzene to the environment than anthropogenic sources (Environment Canada and Health Canada, 1993).

The general population is exposed to benzene mainly through inhalation of ambient air; higher exposures occur particularly in areas of heavy vehicle traffic and at gasoline service stations, and from tobacco smoke (ATSDR, 2007). Exposure to benzene in ambient air accounts for an estimated 98% to 99% of total benzene intake for Canadian non-smokers (Health Canada, 2009). Inside residences, benzene levels in air have been shown to be higher for homes with attached garages, or where smoking occurs (Héroux et al., 2008; Héroux et al., 2010; Wheeler et al., 2013). Various marketplace products containing benzene can also contribute to its presence in indoor air (Environment Canada and Health Canada, 1993). Although benzene has been detected in tap water and in certain foods and beverages, these are not considered to constitute major sources of exposure for the general population (ATSDR, 2007; Health Canada, 2009).

Following inhalation, benzene is readily absorbed into the blood and is distributed throughout the body, concentrating in adipose tissue (EPA, 2002). In the lung and liver, benzene is metabolized into several reactive metabolites including benzene oxide (EPA, 2002; McHale et al., 2012). Benzene metabolism can branch into several alternative metabolic pathways: spontaneous rearrangement of benzene oxide produces

phenol, a major product; reaction with glutathione ultimately forms *S*-phenylmercapturic acid (*S*-PMA); and an iron-catalyzed reaction leads to the formation of *trans*,*trans*-muconic acid (*t*,*t*-MA) (EPA, 2002). Excretion of benzene occurs via exhalation of benzene from the lungs and as conjugated metabolites in urine; all benzene metabolites may be conjugated with sulphate or glucuronic acid (EPA, 2002). Phenol, *S*-PMA, and *t*,*t*-MA are considered urinary biomarkers of recent benzene exposure (Boogaard and van Sittert, 1995; Qu et al., 2005; Weisel, 2010). Measurements of *t*,*t*-MA and *S*-PMA are more sensitive and reliable indicators of benzene exposure because urinary phenol may be a result of dietary or environmental exposure to phenol or other phenolic compounds (ATSDR, 2007). Benzene levels in blood are a reliable biomarker of benzene exposure and reflect recent exposure (Arnold et al., 2013; Weisel, 2010).

Benzene is known to cause a number of health effects in humans with the specific adverse effects dependent upon the concentration and duration of benzene exposure. Exposure to benzene can be hematotoxic in humans and laboratory animals, with bone marrow the principal target organ (EPA, 2002). Available data indicate that benzene metabolites produced in the liver may be carried to bone marrow where hematotoxicity occurs (EPA, 2002). In rodents, chronic inhalation exposure to benzene has been shown to cause leukemia (EPA, 2002). Epidemiologic studies and case studies provide strong evidence of an association between exposure to high levels of benzene and leukemia risk in occupationally exposed humans (EPA, 2002).

Benzene has been classified as carcinogenic to humans by Environment Canada and Health Canada (Group I) and the International Agency for Research on Cancer (Group 1) (Environment Canada and Health Canada, 1993; IARC, 2012). A common mode of action has not been established for hematotoxic and carcinogenic effects; however, it is generally accepted that acute myelogenous leukemia and non-cancer effects are caused by one or more reactive metabolites of benzene (ATSDR, 2007; McHale et al., 2012; Meek and Klaunig, 2010; Smith, 2010).

Globally, benzene has become one of the most intensively regulated substances (Capleton and Levy, 2005). In Canada, regulations have been put in place to limit the concentration of benzene in gasoline as well as emissions from vehicles (Canada, 1997; Environment Canada, 2014). Benzene is listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) and is a candidate for full life cycle management to prevent or minimize its release into the environment (Canada, 1999; Environment Canada and Health Canada, 1993). In 2000–2001, the Canadian Council of Ministers of the Environment endorsed the Canada-wide standard for benzene requiring industry reduction of total benzene emissions and use of best management practices (CCME, 2000; CCME, 2001). With the implementation of these standards, emissions of benzene from industry to ambient air fell by 71% between 1995 and 2008 (CCME, 2012). Benzene is also included as a prohibited ingredient on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Canada, 1985; Health Canada, 2015).

The Government of Canada has also taken a number of actions to address VOCs, a large class of compounds that includes benzene. As a class, they are environmental and health concerns because of their contribution to the formation of smog. The Government of Canada has taken and proposed a number of actions to address VOC emissions resulting from the use of consumer and commercial products in Canada (Canada, 2009a; Canada, 2009b; Environment Canada, 2002; Environment Canada, 2013).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for benzene in drinking water based on cancer endpoints, and that is considered protective of both cancer and non-cancer effects (Health Canada, 2009). Health Canada has identified benzene as a priority indoor air contaminant and has developed a guidance document for benzene in residential indoor air (Health Canada, 2013). On the basis of a low but non-negligible cancer risk at indoor exposure levels, the guidance recommends that individuals take actions to reduce exposure to benzene indoors as much as possible. In particular, exposure reduction strategies have been recommended targeting attached garages and indoor smoking as primary sources of benzene indoors.

Benzene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) cycle 3 (2012–2013) and cycle 4 (2014–2015) participants aged 12–79 years. Benzene metabolites, *t,t*-MA and *S*-PMA, were analyzed in the urine of CHMS cycle 2 (2009–2011) and cycle 3 (2012–2013) participants aged 3 to 79 years. Data are presented as µg/L blood for benzene and µg/L and µg/g creatinine for *t,t*-MA and *S*-PMA. Finding a measurable amount of benzene in blood or *t,t*-MA and *S*-PMA in urine can be an indicator of exposure to benzene and does not necessarily mean that an adverse health effect will occur.

Benzene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Statistics Canada, 2012; Wheeler et al., 2013; Zhu et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.1.1

Benzene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2488	12.58	0.036 (0.025–0.050)	<LOD	0.039 (0.030–0.049)	0.15 (0.12–0.19)	0.24 (0.18–0.29)
4 (2014–2015)	2354	7.48	0.034[E] (0.024–0.050)	0.0093[E] (<LOD–0.013)	0.033[E] (0.017–0.049)	0.14 (0.090–0.19)	0.21 (0.16–0.26)
Males, 12–79 years							
3 (2012–2013)	1245	11.57	0.037 (0.026–0.052)	<LOD	0.040 (0.030–0.049)	0.15 (0.13–0.18)	0.24 (0.18–0.30)
4 (2014–2015)	1164	6.44	0.037 (0.026–0.054)	0.0097[E] (<LOD–0.015)	0.036[E] (0.019–0.054)	0.16 (0.10–0.21)	0.23 (0.15–0.31)
Females, 12–79 years							
3 (2012–2013)	1243	13.60	0.035[E] (0.024–0.051)	<LOD	0.038 (0.028–0.049)	0.17[E] (0.093–0.24)	0.23[E] (0.11–0.35)
4 (2014–2015)	1190	8.49	0.032[E] (0.021–0.048)	0.0090[E] (<LOD–0.013)	0.030[E] (0.015–0.045)	0.13[E] (0.071–0.19)	0.19 (0.14–0.25)
12–19 years							
3 (2012–2013)	750	14.00	0.028 (0.019–0.040)	<LOD	0.034 (0.025–0.043)	0.084 (0.063–0.10)	0.12 (0.076–0.16)
4 (2014–2015)	663	9.65	0.028[E] (0.019–0.041)	0.0087[E] (<LOD–0.014)	0.029[E] (0.013–0.045)	0.087 (0.068–0.11)	0.12 (0.074–0.16)
20–39 years							
3 (2012–2013)	548	10.40	0.037[E] (0.023–0.059)	<LOD	0.040 (0.027–0.054)	0.13 (0.080–0.17)	0.18 (0.14–0.22)
4 (2014–2015)	568	6.69	0.033[E] (0.021–0.051)	0.0097[E] (<LOD–0.014)	0.031[E] (0.0097–0.052)	0.12 (0.074–0.16)	0.17[E] (0.11–0.24)
40–59 years							
3 (2012–2013)	598	8.70	0.040 (0.030–0.055)	<LOD	0.039 (0.028–0.050)	0.23 (0.16–0.31)	0.40[E] (0.24–0.56)
4 (2014–2015)	575	6.26	0.041[E] (0.027–0.062)	0.010[E] (<LOD–0.015)	0.037[E] (0.014–0.060)	0.18 (0.13–0.22)	0.29[E] (0.18–0.40)
60–79 years							
3 (2012–2013)	592	16.72	0.031[E] (0.021–0.047)	<LOD	0.038 (0.026–0.051)	0.13 (0.085–0.17)	0.20 (0.16–0.24)
4 (2014–2015)	548	6.93	0.031 (0.023–0.042)	0.0084[E] (<LOD–0.013)	0.030 (0.021–0.039)	0.13 (0.085–0.17)	0.24[E] (0.15–0.33)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 15.1.2

S-Phenylmercapturic acid (*S*-PMA) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2525	22.10	0.20 (0.18–0.23)	<LOD	0.12 (0.095–0.15)	1.3 (0.85–1.7)	3.5 (2.5–4.5)
3 (2012–2013)	2472	34.67	0.17 (0.14–0.21)	<LOD	0.10[E] (<LOD–0.16)	F	3.4 (2.3–4.5)
4 (2014–2015)	2484	36.59	0.17 (0.14–0.19)	<LOD	0.12 (0.10–0.14)	1.7[E] (0.86–2.5)	3.4 (2.1–4.6)
Males, 3–79 years							
2 (2009–2011)	1267	20.21	0.23 (0.20–0.26)	<LOD	0.13 (0.10–0.16)	F	3.9[E] (2.5–5.4)
3 (2012–2013)	1223	31.07	0.20 (0.16–0.25)	<LOD	0.19[E] (0.080–0.30)	1.9[E] (0.51–3.3)	4.0 (2.7–5.3)
4 (2014–2015)	1246	34.43	0.17 (0.13–0.23)	<LOD	0.12 (0.093–0.15)	1.6[E] (0.65–2.6)	3.1[E] (1.1–5.0)
Females, 3–79 years							
2 (2009–2011)	1258	24.01	0.18 (0.15–0.22)	<LOD	0.11 (<LOD–0.14)	1.1[E] (0.66–1.6)	2.5[E] (0.89–4.1)
3 (2012–2013)	1249	38.19	0.14 (0.10–0.19)	<LOD	0.099 (<LOD–0.12)	F	3.3[E] (1.4–5.2)
4 (2014–2015)	1238	38.77	0.16 (0.12–0.20)	<LOD	0.12 (0.089–0.14)	F	4.2[E] (2.3–6.1)
3–5 years							
2 (2009–2011)	507	20.32	0.15 (0.13–0.17)	<LOD	0.12 (0.094–0.14)	0.40 (0.29–0.52)	0.64[E] (0.40–0.88)
3 (2012–2013)	491	28.51	0.11 (0.10–0.12)	<LOD	0.099 (0.096–0.10)	0.32 (0.26–0.37)	0.51[E] (0.30–0.72)
4 (2014–2015)	487	34.50	0.11 (0.091–0.13)	<LOD	0.11 (0.087–0.13)	0.43 (0.32–0.55)	0.56 (0.41–0.71)
6–11 years							
2 (2009–2011)	511	25.24	0.14 (0.11–0.17)	<LOD	0.099 (0.083–0.12)	0.38 (0.28–0.49)	0.58[E] (0.33–0.82)
3 (2012–2013)	491	38.90	0.099 (0.084–0.12)	<LOD	0.099 (0.092–0.11)	0.31 (0.23–0.39)	0.41 (0.35–0.47)
4 (2014–2015)	501	42.51	—	<LOD	0.11 (0.095–0.12)	0.36 (0.28–0.45)	0.49 (0.37–0.62)
12–19 years							
2 (2009–2011)	506	18.97	0.17 (0.15–0.20)	<LOD	0.13 (0.094–0.16)	0.62 (0.45–0.79)	1.1[E] (0.53–1.6)
3 (2012–2013)	497	32.19	0.14 (0.11–0.19)	<LOD	0.10[E] (<LOD–0.15)	F	2.3[E] (0.74–4.0)
4 (2014–2015)	488	33.81	0.12 (0.11–0.13)	<LOD	0.11 (0.093–0.12)	0.65[E] (0.40–0.90)	0.80 (0.60–0.99)
20–39 years							
2 (2009–2011)	355	19.44	0.21 (0.17–0.27)	<LOD	0.12 (<LOD–0.16)	1.4 (1.1–1.8)	3.0[E] (1.5–4.5)
3 (2012–2013)	345	35.07	0.20[E] (0.14–0.30)	<LOD	0.17[E] (<LOD–0.29)	F	3.3[E] (1.4–5.3)
4 (2014–2015)	353	30.59	0.19 (0.14–0.27)	<LOD	0.13 (0.093–0.17)	F	3.3[E] (2.0–4.6)
40–59 years							
2 (2009–2011)	359	25.91	0.24 (0.18–0.30)	<LOD	0.13[E] (<LOD–0.20)	2.9[E] (1.1–4.7)	5.2[E] (3.2–7.3)
3 (2012–2013)	306	35.62	0.20[E] (0.14–0.30)	<LOD	0.17[E] (<LOD–0.29)	F	3.4[E] (1.8–5.0)
4 (2014–2015)	309	38.83	0.20 (0.15–0.27)	<LOD	0.12 (0.084–0.16)	3.1[E] (1.1–5.2)	5.4[E] (2.9–8.0)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	287	23.69	0.19 (0.15–0.23)	<LOD	0.12 (0.094–0.15)	1.1[E] (0.57–1.7)	3.4[E] (1.3–5.4)
3 (2012–2013)	342	39.77	0.14 (0.11–0.18)	<LOD	0.093 (0.087–0.099)	F	5.1[E] (2.0–8.3)
4 (2014–2015)	346	39.02	0.14 (0.12–0.18)	<LOD	0.10 (<LOD–0.14)	1.6[E] (0.44–2.8)	3.2[E] (1.3–5.1)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 15.1.3

S-Phenylmercapturic acid (*S*-PMA) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2515	22.10	0.20 (0.17–0.24)	<LOD	0.19 (0.12–0.26)	1.2[E] (0.62–1.8)	3.1 (2.0–4.2)
3 (2012–2013)	2471	34.67	0.18 (0.15–0.22)	<LOD	0.14 (<LOD–0.16)	1.4[E] (0.86–2.0)	2.9 (1.9–4.0)
4 (2014–2015)	2484	36.59	0.15 (0.13–0.17)	<LOD	0.11 (0.090–0.13)	1.6 (1.0–2.1)	2.7 (2.3–3.0)
Males, 3–79 years							
2 (2009–2011)	1263	20.21	0.19 (0.16–0.23)	<LOD	0.13[E] (0.061–0.20)	1.8[E] (0.84–2.8)	3.0[E] (1.0–5.0)
3 (2012–2013)	1223	31.07	0.17 (0.14–0.21)	<LOD	0.12 (0.089–0.16)	1.4 (0.94–1.9)	2.2[E] (1.2–3.3)
4 (2014–2015)	1246	34.43	0.14 (0.11–0.18)	<LOD	0.099 (0.084–0.11)	1.4[E] (0.38–2.4)	2.3[E] (1.4–3.3)
Females, 3–79 years							
2 (2009–2011)	1252	24.01	0.20 (0.16–0.26)	<LOD	0.19 (<LOD–0.24)	0.91[E] (0.57–1.2)	3.1[E] (1.1–5.2)
3 (2012–2013)	1248	38.19	0.18 (0.14–0.24)	<LOD	0.14 (<LOD–0.16)	F	3.4[E] (1.7–5.1)
4 (2014–2015)	1238	38.77	0.17 (0.13–0.21)	<LOD	0.13 (0.096–0.15)	1.7[E] (<LOD–2.8)	3.1[E] (1.6–4.6)
3–5 years							
2 (2009–2011)	506	20.32	0.26 (0.23–0.29)	<LOD	0.29 (0.19–0.39)	0.69 (0.56–0.82)	0.91 (0.71–1.1)
3 (2012–2013)	490	28.51	0.22 (0.20–0.24)	<LOD	0.20 (0.16–0.24)	0.52 (0.39–0.65)	0.79[E] (0.50–1.1)
4 (2014–2015)	487	34.50	0.19 (0.17–0.22)	<LOD	0.18 (0.16–0.21)	0.54 (0.44–0.64)	0.76 (0.62–0.89)
6–11 years							
2 (2009–2011)	509	25.24	0.15 (0.13–0.19)	<LOD	0.17[E] (0.057–0.28)	0.46 (0.31–0.61)	0.60 (0.40–0.80)
3 (2012–2013)	491	38.90	0.13 (0.11–0.15)	<LOD	0.13 (0.10–0.15)	0.32 (0.27–0.37)	0.41 (0.36–0.45)
4 (2014–2015)	501	42.51	—	<LOD	0.12 (0.10–0.14)	0.33 (0.25–0.42)	0.46 (0.39–0.54)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
2 (2009–2011)	504	18.97	0.13 (0.11–0.15)	<LOD	0.10 (0.091–0.11)	0.50 (0.34–0.66)	0.78[E] (0.50–1.1)
3 (2012–2013)	497	32.19	0.11 (0.087–0.14)	<LOD	0.092 (<LOD–0.11)	F	1.3[E] (0.59–2.0)
4 (2014–2015)	488	33.81	0.089 (0.080–0.10)	<LOD	0.077 (0.065–0.088)	0.33 (0.24–0.43)	0.52[E] (0.26–0.78)
20–39 years							
2 (2009–2011)	353	19.44	0.19 (0.14–0.25)	<LOD	F	1.6[E] (0.59–2.6)	2.9[E] (1.7–4.1)
3 (2012–2013)	345	35.07	0.15 (0.11–0.21)	<LOD	0.12 (<LOD–0.16)	1.1[E] (<LOD–1.8)	1.7[E] (0.86–2.6)
4 (2014–2015)	353	30.59	0.16 (0.11–0.23)	<LOD	0.13 (0.096–0.16)	1.9[E] (0.93–2.9)	2.4 (1.7–3.1)
40–59 years							
2 (2009–2011)	357	25.91	0.23 (0.17–0.31)	<LOD	0.19 (<LOD–0.24)	F	4.2[E] (1.5–7.0)
3 (2012–2013)	306	35.62	0.24 (0.17–0.34)	<LOD	0.17 (<LOD–0.23)	2.0[E] (<LOD–3.5)	3.5[E] (1.8–5.3)
4 (2014–2015)	309	38.83	0.18 (0.13–0.24)	<LOD	0.10 (0.068–0.13)	2.5 (1.7–3.4)	3.6[E] (1.3–5.8)
60–79 years							
2 (2009–2011)	286	23.69	0.21 (0.17–0.27)	<LOD	0.19 (0.15–0.23)	1.2[E] (0.55–1.9)	2.9[E] (1.3–4.5)
3 (2012–2013)	342	39.77	0.17 (0.13–0.23)	<LOD	0.12 (0.080–0.16)	2.1[E] (<LOD–3.5)	3.5[E] (2.2–4.9)
4 (2014–2015)	346	39.02	0.14 (0.11–0.19)	<LOD	0.098 (<LOD–0.13)	F	3.2[E] (1.6–4.7)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 15.1.4

trans,trans-Muconic acid (*t,t*-MA) — Geometric means and selected percentiles of urine concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2523	0.20	64 (57–71)	15 (12–19)	59 (52–66)	330 (260–390)	500 (330–680)
3 (2012–2013)	2492	0	56 (47–67)	14 (11–16)	53 (41–65)	250 (160–340)	400 (290–510)
4 (2014–2015)	2514	0	67 (61–74)	15 (12–18)	57 (53–61)	350 (260–450)	670[E] (410–930)
Males, 3–79 years							
2 (2009–2011)	1267	0.24	68 (58–81)	19 (13–25)	66 (54–78)	340 (260–420)	480 (330–630)
3 (2012–2013)	1231	0	64 (53–78)	17 (14–20)	59 (45–73)	260[E] (140–380)	400[E] (140–650)
4 (2014–2015)	1257	0	72 (62–84)	19 (16–23)	58 (50–66)	340 (220–450)	540[E] (280–790)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Females, 3–79 years							
2 (2009–2011)	1256	0.16	59 (51–70)	13 (9.2–17)	56 (47–64)	320 (220–420)	610[E] (330–890)
3 (2012–2013)	1261	0	49 (41–60)	11 (7.2–14)	46 (34–59)	230[E] (120–340)	430 (290–580)
4 (2014–2015)	1257	0	62 (51–74)	12 (8.3–15)	54 (41–66)	370 (240–510)	730[E] (460–1000)
3–5 years							
2 (2009–2011)	506	0.40	75 (63–90)	20 (15–24)	68 (52–83)	380[E] (220–540)	670 (510–840)
3 (2012–2013)	489	0	65 (57–75)	14 (12–17)	51 (41–61)	440 (390–490)	730[E] (440–1000)
4 (2014–2015)	494	0	73 (58–93)	16[E] (9.1–22)	63 (46–79)	380[E] (230–520)	750[E] (400–1100)
6–11 years							
2 (2009–2011)	511	0.20	71 (57–87)	17 (13–21)	63 (41–85)	380[E] (240–510)	540 (360–720)
3 (2012–2013)	496	0	61 (49–75)	12 (7.3–16)	55 (39–71)	330[E] (200–470)	740[E] (220–1300)
4 (2014–2015)	508	0	75 (60–93)	14 (9.6–17)	61 (50–73)	500 (340–660)	820[E] (470–1200)
12–19 years							
2 (2009–2011)	506	0	75 (61–92)	15[E] (8.4–21)	66 (47–85)	380 (290–480)	560 (440–680)
3 (2012–2013)	506	0	65 (48–88)	13 (8.3–18)	61 (45–77)	360[E] (220–510)	670[E] (350–1000)
4 (2014–2015)	495	0	74 (66–83)	17 (13–20)	71 (55–87)	350 (230–460)	740[E] (430–1100)
20–39 years							
2 (2009–2011)	355	0.56	62 (48–81)	13[E] (6.3–19)	70 (54–86)	310[E] (120–510)	610[E] (300–910)
3 (2012–2013)	347	0	66 (46–95)	15[E] (9.0–22)	66[E] (40–92)	270[E] (140–400)	380[E] (160–590)
4 (2014–2015)	356	0	75 (57–100)	17[E] (10–24)	58 (45–70)	680[E] (190–1200)	790[E] (450–1100)
40–59 years							
2 (2009–2011)	359	0	65 (54–80)	17 (14–20)	57 (41–73)	310[E] (170–460)	470[E] (200–750)
3 (2012–2013)	307	0	50 (39–65)	15[E] (8.4–21)	47 (32–61)	160[E] (73–240)	360[E] (130–590)
4 (2014–2015)	309	0	60 (52–70)	16 (11–21)	53 (40–66)	250 (170–330)	410[E] (220–600)
60–79 years							
2 (2009–2011)	286	0	54 (43–67)	14[E] (7.6–21)	52 (37–67)	240[E] (120–370)	400 (300–500)
3 (2012–2013)	347	0	50 (40–62)	10[E] (4.5–16)	44 (34–55)	260[E] (70–460)	550[E] (170–930)
4 (2014–2015)	352	0	59 (51–67)	14 (11–18)	55 (46–64)	240 (160–320)	390 (280–510)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

Table 15.1.5

trans,trans-Muconic acid (*t,t*-MA) (creatinine adjusted) — Geometric means and selected percentiles of urine concentrations (µg/g creatinine) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011), cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2513	0.20	63 (58–70)	19 (16–21)	54 (48–60)	280 (230–340)	450 (370–520)
3 (2012–2013)	2491	0	58 (51–66)	19 (17–21)	51 (43–58)	220 (160–280)	390 (270–510)
4 (2014–2015)	2514	0	60 (54–68)	17 (15–18)	52 (45–59)	280 (220–340)	460 (340–580)
Males, 3–79 years							
2 (2009–2011)	1263	0.24	59 (50–69)	17 (15–19)	52 (40–63)	230 (150–320)	380 (280–480)
3 (2012–2013)	1231	0	54 (48–60)	19 (16–21)	51 (44–57)	160[E] (97–210)	290[E] (110–470)
4 (2014–2015)	1257	0	57 (48–68)	17 (15–18)	49 (39–60)	250[E] (160–350)	400[E] (250–560)
Females, 3–79 years							
2 (2009–2011)	1250	0.16	69 (61–77)	20 (17–23)	55 (48–63)	320 (240–400)	490 (320–650)
3 (2012–2013)	1260	0	63 (54–74)	19 (16–22)	51 (39–62)	270 (190–340)	460 (300–630)
4 (2014–2015)	1257	0	64 (56–74)	16 (14–19)	54 (45–62)	310 (250–380)	490 (350–620)
3–5 years							
2 (2009–2011)	505	0.40	130 (110–160)	36 (31–41)	110 (87–130)	590 (420–750)	990[E] (580–1400)
3 (2012–2013)	488	0	130 (110–150)	34 (31–37)	87 (70–100)	910 (650–1200)	1500[E] (810–2100)
4 (2014–2015)	494	0	130 (110–160)	35 (32–38)	100[E] (63–140)	760 (510–1000)	1300[E] (690–1900)
6–11 years							
2 (2009–2011)	509	0.20	82 (68–99)	24 (20–28)	69 (52–87)	380 (290–470)	490 (360–620)
3 (2012–2013)	496	0	78 (65–93)	21 (18–23)	65 (55–75)	380[E] (220–530)	720[E] (260–1200)
4 (2014–2015)	508	0	81 (67–98)	21 (17–25)	61 (46–76)	470[E] (240–700)	760 (600–910)
12–19 years							
2 (2009–2011)	504	0	57 (48–69)	18 (15–20)	43 (29–57)	320 (230–410)	410 (340–490)
3 (2012–2013)	506	0	49 (40–61)	15 (13–17)	38 (27–49)	230[E] (140–310)	450 (290–600)
4 (2014–2015)	495	0	54 (47–63)	15 (12–18)	47 (36–58)	210 (140–280)	450 (330–560)
20–39 years							
2 (2009–2011)	353	0.56	55 (46–66)	16 (14–19)	48 (36–60)	270[E] (120–420)	430 (300–570)
3 (2012–2013)	347	0	50 (38–66)	16 (10–21)	46[E] (22–70)	160 (120–200)	240[E] (90–390)
4 (2014–2015)	356	0	61 (47–81)	17 (14–21)	53 (37–68)	400[E] (200–600)	580[E] (320–840)
40–59 years							
2 (2009–2011)	357	0	66 (53–82)	19 (14–23)	54 (37–71)	270 (200–340)	F
3 (2012–2013)	307	0	59 (47–75)	22 (19–24)	54 (41–66)	190[E] (95–280)	290[E] (120–450)
4 (2014–2015)	309	0	55 (49–62)	16 (13–19)	49 (41–57)	190 (150–230)	270 (190–350)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
2 (2009–2011)	285	0	63 (54–73)	20 (18–23)	55 (44–65)	220[E] (120–330)	400[E] (210–590)
3 (2012–2013)	347	0	57 (48–70)	20 (18–23)	43 (32–53)	F	490[E] (220–760)
4 (2014–2015)	352	0	57 (47–69)	16 (13–19)	50 (41–59)	240[E] (130–350)	300 (230–370)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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15.2 ETHYLBENZENE

Ethylbenzene (CASRN 100-41-4) is a colourless liquid and a volatile organic compound (VOC). It is a high-production volume industrial chemical produced commercially primarily by alkylating benzene with ethylene (ATSDR, 2010; IARC, 2000). The quantity of ethylbenzene manufactured in Canada has remained relatively stable since 1999 (Environment and Climate Change Canada and Health Canada, 2016).

Major uses of ethylbenzene include manufacturing of styrene and synthetic rubber (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016; IARC, 2000). It is also used in the production of diethylbenzene, acetophenone, and other chemicals, as a solvent in the semiconductor industry, and as a general solvent used in manufactured products (ATSDR, 2010). Ethylbenzene is a constituent of asphalt, naphtha, and automotive and aviation fuels, including gasoline that typically contains about 2% ethylbenzene by weight (ATSDR, 2010). Commercial mixed xylenes contain ethylbenzene at levels up to 25% and, as such, ethylbenzene may be present in some paints, including spray paints and primers, lacquers, printing inks, insecticides, and solvents containing xylenes (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016; IARC, 2000).

Ethylbenzene is released to the environment, primarily to the atmosphere, from natural and anthropogenic sources. It has been measured in emissions from volcanoes, forest fires, crude petroleum, and coal deposits (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016; IARC, 2000). Anthropogenic sources include the manufacture, processing, storage, use, transportation and disposal of fuels, solvents, petrochemicals, and polymers. Releases of ethylbenzene to air, especially as a product of fuel combustion, may be increasing as well, with increasing population and demand for energy (Environment and Climate Change Canada and Health Canada, 2016).

For the general population, most exposure to ethylbenzene originates from the inhalation of indoor air (Environment and Climate Change Canada and Health Canada, 2016; Health Canada, 2007). Inside residences, ethylbenzene levels in air have been shown to be higher for homes with an attached garage, with a higher number of occupants, with recent renovations, and in which fragrances and paint remover have been recently used (Wheeler et al., 2013). Use of consumer products such as lacquers, stains, varnishes, and concrete floor sealers can also result in inhalation exposures of short duration but potentially high concentration. Although cigarette smoke may contribute to the concentration of ethylbenzene in the home, it is unlikely a significant source (Environment and Climate Change Canada and Health Canada, 2016; Health Canada, 2010). Various other marketplace products containing ethylbenzene can also contribute to its presence in indoor air (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). Although ethylbenzene has been detected in outdoor air, drinking water, soil, and food, these are not considered to constitute major sources of exposure for the general population (Health Canada, 2007).

Following inhalation, oral, and dermal exposures, ethylbenzene is readily absorbed and distributed throughout the body (ATSDR, 2010; IARC, 2000). Absorption of ethylbenzene by inhalation is approximately 49% to 64%, in humans (ATSDR, 2010). Once absorbed, ethylbenzene is eliminated from the blood and body mostly in the urine with minor amounts exhaled in the breath, and has an elimination half-life ranging from fewer than 1 to 25 hours (ATSDR, 2010). Following oral exposure, absorption of ethylbenzene is approximately 72% to 92% in laboratory animals and elimination is rapid, occurring predominantly via urinary excretion (ATSDR, 2010). In contrast, following uptake through the skin only a small proportion of absorbed ethylbenzene is eliminated in the urine and none in exhaled air (ATSDR, 2010). Ethylbenzene levels in blood are the most accurate biomarker of ethylbenzene exposure and are reflective of recent exposures (ATSDR, 2010).

In humans, ethylbenzene can be irritating to the eyes, nose, throat, lungs, and skin, and it has been associated with symptoms of headaches, dizziness, vertigo, and feelings of intoxication (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). Acute inhalation exposure has been generally associated with reversible neurological symptoms and respiratory tract irritation whereas chronic exposure has been associated with impaired neurological function, including cognitive and neuromuscular performance (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). Studies in laboratory animals exposed by inhalation to ethylbenzene provide supporting evidence for central nervous system effects, neuromuscular and behavioural changes, and hearing loss (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). In laboratory animals, chronic exposure to high levels of ethylbenzene in air and via the oral route has been associated with kidney and liver damage, some minor developmental effects, and effects in blood, pituitary, thyroid, and respiratory tissues (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). Ethylbenzene is classified as possibly carcinogenic to humans (Group 2B carcinogen) according to the International Agency for Research on Cancer (IARC, 2000). However, the more recent evaluation by Health

Canada and Environment Canada concludes that ethylbenzene is likely to be a threshold carcinogen, indicating that there is a threshold below which tumour formation would not be expected (Environment and Climate Change Canada and Health Canada, 2016).

Health Canada and Environment Canada published a final screening assessment on ethylbenzene in 2016 and have concluded that it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health (Environment and Climate Change Canada and Health Canada, 2016).

The Government of Canada has also taken a number of actions to address VOCs, a large class of compounds that includes ethylbenzene. As a class, they are environmental and health concerns because of their contribution to the formation of smog. The Government of Canada has taken and proposed a number of actions to address VOC emissions resulting from the use of consumer and commercial products in Canada (Canada, 2009a; Canada, 2009b; Environment Canada, 2002; Environment Canada, 2013).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for ethylbenzene that is protective of human health, as well as an aesthetic objective for ethylbenzene based on its odour threshold (Health Canada, 2014). The guideline was developed based on cancer and non-cancer endpoints in the liver, kidney, and pituitary gland of experimental animals (Health Canada, 2014).

Ethylbenzene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood. Finding a measurable amount of ethylbenzene in blood can be an indicator of recent exposure to ethylbenzene and does not necessarily mean that an adverse health effect will occur.

Ethylbenzene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Statistics Canada, 2012; Wheeler et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle* 4 (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.2.1

Ethylbenzene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2441	17.90	0.026 (0.020–0.033)	<LOD	0.025 (0.017–0.033)	0.084 (0.070–0.098)	0.12 (0.095–0.15)
4 (2014–2015)	2505	11.90	0.026 (0.022–0.031)	<LOD	0.024 (0.018–0.029)	0.078 (0.061–0.094)	0.11 (0.089–0.13)
Males, 12–79 years							
3 (2012–2013)	1212	17.49	0.028 (0.022–0.034)	<LOD	0.026 (0.018–0.034)	0.088 (0.063–0.11)	0.14 (0.096–0.18)
4 (2014–2015)	1239	11.62	0.028 (0.023–0.035)	<LOD	0.027 (0.019–0.034)	0.088 (0.067–0.11)	0.12 (0.086–0.15)
Females, 12–79 years							
3 (2012–2013)	1229	18.31	0.025 (0.018–0.033)	<LOD	0.025 (0.016–0.033)	0.080 (0.057–0.10)	0.11 (0.076–0.14)
4 (2014–2015)	1266	12.16	0.024 (0.020–0.029)	<LOD	0.022 (0.018–0.026)	0.065 (0.046–0.084)	0.093 (0.068–0.12)

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
3 (2012–2013)	731	19.84	0.020 (0.016–0.027)	<LOD	0.021 (0.015–0.027)	0.064 (0.044–0.084)	0.081 (0.056–0.11)
4 (2014–2015)	709	15.37	0.022 (0.017–0.027)	<LOD	0.022 (0.016–0.027)	0.053 (0.044–0.061)	0.065 (0.052–0.077)
20–39 years							
3 (2012–2013)	532	17.29	0.026 (0.019–0.035)	<LOD	0.026[E] (0.012–0.041)	0.077[E] (0.040–0.11)	0.12[E] (0.058–0.17)
4 (2014–2015)	596	12.75	0.024 (0.019–0.032)	<LOD	0.023 (0.016–0.029)	0.062[E] (0.034–0.089)	F
40–59 years							
3 (2012–2013)	591	14.89	0.029 (0.024–0.037)	<LOD	0.027 (0.020–0.034)	0.10 (0.082–0.12)	0.14 (0.10–0.18)
4 (2014–2015)	622	8.36	0.029 (0.023–0.036)	0.012[E] (<LOD–0.016)	0.025 (0.017–0.033)	0.098 (0.070–0.13)	0.12 (0.10–0.14)
60–79 years							
3 (2012–2013)	587	19.08	0.025 (0.019–0.032)	<LOD	0.024 (0.016–0.032)	0.079 (0.064–0.094)	0.12[E] (0.062–0.17)
4 (2014–2015)	578	10.55	0.027 (0.024–0.030)	<LOD	0.026 (0.022–0.029)	0.087 (0.074–0.10)	0.12 (0.084–0.15)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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15.3 STYRENE

Styrene (CASRN 100-42-5) is a colourless liquid classified as a volatile organic compound (VOC) and a high-production volume industrial chemical. Styrene was first recovered by distillation of a natural resin (storax balsam), sapwood, and bark tissues of trees (ATSDR, 2010; IARC, 2002).

Styrene has been synthetically produced since the early 19th century and is a well-known impurity of coal tar industrial processing and petroleum cracking (IARC, 2002). Styrene is available as a commercial product and is used worldwide in the manufacture of plastics, glass fibre-reinforced resins, protective coatings, ion-exchange resins, and synthetic rubber (ATSDR, 2010; IARC, 2002). Commercial styrene contains several components, including benzene, ethylbenzene, xylene, and other VOCs (IARC, 2002). In Canada, industrial uses of styrene include the manufacture of polystyrene, styrene-butadiene latex and rubber, acrylonitrile-butadiene styrene resins, and unsaturated polyester resins (Environment Canada and Health Canada, 1993). Styrene-based polymer materials are used in the manufacturing of a wide range of products, most of which also contain a small amount of unlinked styrene monomer (ATSDR, 2010; Environment Canada and Health Canada, 1993). Examples of products made with or containing styrene include foam insulation, automobile tires, packaging materials, custom mouldings, waxes and surface coatings, adhesives, and metal cleaners (ATSDR, 2010; Environment Canada and Health Canada, 1993).

Styrene is released to the environment from natural and anthropogenic sources. Styrene releases to the environment are mainly atmospheric and occur as a result of the manufacture, use, and disposal of styrene-containing products, industrial releases, vehicle exhaust, incineration, and tobacco smoke (Environment Canada and Health Canada, 1993; ATSDR, 2010). Production, use, and disposal of styrene and styrene-containing products can also result in releases to the aquatic environment via wastewater. Natural sources of styrene releases to the environment include biodegradation of vegetation and organic material (ATSDR, 2010; Environment Canada and Health Canada, 1993).

The most common route of exposure to styrene in the general population is inhalation, with levels of styrene often higher in indoor air than outdoor (ATSDR, 2010; Environment Canada and Health Canada, 1993). Styrene is a minor and natural component of tobacco smoke, and tobacco smoke is the major contributor to the total styrene exposure in smokers (Environment Canada and Health Canada, 1993; Zhu et al., 2013). In addition to tobacco smoke, common sources of styrene present in air are automobile exhaust, the use and manufacturing of styrene, and the use of photocopiers and laser printers (ATSDR, 2010; Environment Canada and Health Canada, 1993). Further, it is not uncommon for short-term inhalation exposures to styrene to occur as a result of indoor air releases from new building materials made with polymer resins, synthetic rubbers, laminated materials, and from fresh adhesives and surface coatings. Additional exposures in the general population may occur through ingestion of food and beverages; however, most styrene associated with food is residue of styrene monomer leached from packaged food in polystyrene containers (ATSDR, 2010; Genualdi et al., 2014). Intake of styrene from drinking water is generally negligible (Environment Canada and Health Canada, 1993). Exposure through skin and eye contact can also occur when handling liquid styrene-containing products.

Styrene is readily absorbed and distributed throughout the body following inhalation, with the highest concentrations measured in adipose tissue (ATSDR, 2010; Environment Canada and Health Canada, 1993). Following oral exposure in laboratory animals, styrene absorption was rapid and complete with distribution to the kidney, liver, pancreas, adipose tissue and, to a lesser extent, the stomach, and small and large intestines

(ATSDR, 2010). Styrene absorbed into the body was rapidly eliminated from all tissues within 1 to 3 days (ATSDR, 2010). Half-lives are estimated to range between 1 and 13 hours depending on the phase of elimination; in adipose tissue, an elimination half-life of 2 to 5 days has been estimated (ATSDR, 2010). In humans, approximately 97% of the styrene absorbed is excreted as urinary metabolites, with the remainder eliminated unchanged in expired air (ATSDR, 2010; Environment Canada and Health Canada, 1993). The primary intermediate metabolite of styrene is styrene-7, 8-oxide, which is hydrolyzed to styrene glycol and further metabolized to mandelic and phenylglyoxylic acids, the principal urinary metabolites (ATSDR, 2010; Environment Canada and Health Canada, 1993). The major site of styrene metabolism is the liver. At high exposures that saturate metabolic enzymes, increased amounts of unchanged styrene are excreted in expired air (ATSDR, 2010; Environment Canada and Health Canada, 1993). In laboratory animals, following oral exposure styrene was rapidly excreted in urine with 90% eliminated within 24 hours, and less than 2% in the feces (ATSDR, 2010). The most reliable biomarkers of recent exposure to styrene are measurements of styrene in blood, urine, and breath (ATSDR, 2010).

Acute exposure to styrene is irritating to the eyes, nose, and throat, and induces dermatitis (ATSDR, 2010; IARC, 2002). In humans, acute exposure to high levels of styrene in air is associated with central nervous system effects, including nausea, headache, tiredness, and concentration problems, similar to the narcotic effects of other organic solvents; effects are generally reversible after the source of exposure is eliminated (ATSDR, 2010; Environment Canada and Health Canada, 1993). Chronic exposure to styrene is associated with central and peripheral nervous system effects, slower reaction times, decreased colour discrimination, hearing problems, altered hand-eye coordination, and impairment of verbal learning skills (ATSDR, 2010; ATSDR, 2012; IARC, 2002). Whether chronic styrene exposure results in permanent damage to the nervous system in humans has not been determined (ATSDR, 2010). Data from studies in humans and laboratory animals exposed via inhalation and the oral route to high levels of styrene also suggest styrene can be immunosuppressive (ATSDR, 2010; Environment Canada and Health Canada, 1993; IARC, 2002). Chronic exposure to high levels of styrene in air in the presence of other chemicals, including carcinogens, has been weakly associated with lymphomas and other cancers and chromosomal alterations (ATSDR, 2010; IARC, 2002). Styrene has been classified as possibly carcinogenic to humans, on the basis of limited evidence in animals and humans, by Environment Canada and Health Canada (Group III) and the International Agency for Research on Cancer (IARC; Group 2B) (Environment Canada and Health Canada, 1993; IARC, 2002). The styrene primary intermediate metabolite styrene-7, 8-oxide is classified by IARC as a Group 2A carcinogen, probably carcinogenic to humans (IARC, 2002). Recently, the U.S. National Toxicology Program listed styrene as reasonably anticipated to be a human carcinogen based on human cancer studies, laboratory animal studies, and supporting mechanistic data (ATSDR, 2011; NTP, 2016).

Health Canada and Environment Canada concluded that levels of styrene normally found in the Canadian environment are not a concern to human health (Environment Canada and Health Canada, 1993). Styrene is also part of a larger class of VOCs that, as a group, are environmental and health concerns because of their contribution to the formation of smog. The Government of Canada has taken and proposed a number of actions to address VOC emissions resulting from the use of consumer and commercial products in Canada (Canada, 2009a; Canada, 2009b; Environment Canada, 2002; Environment Canada, 2013). Because styrene has not been detected in Canadian drinking water supplies, no guideline for Canadian drinking water quality has been established by the Federal-Provincial-Territorial Committee on Drinking Water.

Styrene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood. Finding a measurable amount of styrene in blood can be an indicator of exposure to styrene and does not necessarily mean that an adverse health effect will occur.

Styrene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015), and in tap water from households in cycle 3 and cycle 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle* 4 (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.3.1

Styrene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2063	7.61	0.043[E] (0.029–0.062)	F	0.043 (0.030–0.055)	0.12 (0.076–0.16)	0.17[E] (0.10–0.23)
4 (2014–2015)	2527	3.09	0.055 (0.043–0.070)	0.026[E] (0.013–0.040)	0.058 (0.047–0.069)	0.11 (0.094–0.13)	0.14 (0.12–0.15)
Males, 12–79 years							
3 (2012–2013)	1036	6.95	0.043[E] (0.029–0.064)	F	0.045 (0.033–0.057)	0.12 (0.079–0.15)	0.17[E] (0.099–0.24)
4 (2014–2015)	1251	3.28	0.056 (0.042–0.075)	0.026[E] (<LOD–0.042)	0.063 (0.049–0.077)	0.12 (0.097–0.14)	0.14 (0.12–0.16)
Females, 12–79 years							
3 (2012–2013)	1027	8.28	0.042[E] (0.028–0.061)	F	0.041 (0.028–0.055)	0.11[E] (0.062–0.17)	0.16[E] (0.092–0.23)
4 (2014–2015)	1276	2.90	0.053 (0.044–0.065)	0.027[E] (0.015–0.038)	0.055 (0.046–0.065)	0.10 (0.078–0.12)	0.13 (0.10–0.15)
12–19 years							
3 (2012–2013)	626	8.47	0.037[E] (0.024–0.057)	F	0.040 (0.029–0.052)	0.094[E] (0.029–0.16)	0.15[E] (0.063–0.24)
4 (2014–2015)	713	3.23	0.053 (0.041–0.068)	0.027[E] (0.014–0.041)	0.058 (0.045–0.070)	0.097 (0.086–0.11)	0.10 (0.087–0.11)
20–39 years							
3 (2012–2013)	435	7.36	0.043[E] (0.029–0.065)	<LOD	0.043[E] (0.024–0.061)	0.12[E] (0.055–0.18)	0.18[E] (0.10–0.26)
4 (2014–2015)	600	2.83	0.055 (0.043–0.070)	0.029[E] (0.014–0.044)	0.057 (0.047–0.068)	0.11 (0.085–0.13)	0.12 (0.10–0.15)
40–59 years							
3 (2012–2013)	493	5.68	0.045[E] (0.031–0.066)	0.016[E] (<LOD–0.026)	0.044 (0.032–0.056)	0.13 (0.090–0.16)	0.18[E] (0.11–0.25)
4 (2014–2015)	625	3.84	0.056 (0.042–0.075)	0.025[E] (<LOD–0.040)	0.064 (0.049–0.079)	0.12 (0.099–0.15)	0.15 (0.12–0.17)
60–79 years							
3 (2012–2013)	509	8.64	0.041[E] (0.027–0.063)	F	0.044 (0.029–0.058)	0.11 (0.069–0.15)	0.14[E] (0.049–0.24)
4 (2014–2015)	589	2.38	0.053 (0.043–0.065)	0.025[E] (0.012–0.038)	0.053 (0.042–0.064)	0.11 (0.086–0.13)	0.14 (0.11–0.17)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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15.4 TETRACHLORO-ETHYLENE (PERCHLORO-ETHYLENE)

Tetrachloroethylene (CASRN 127-18-4), commonly known as perchloroethylene, is a colourless liquid classified as a volatile organic compound (Canada, 2011a; Canada, 2011b; Environment Canada and Health Canada, 1993; IARC, 2014). It is an industrial chemical produced commercially by chlorination of other hydrocarbons, including acetylene, via trichloroethylene (IARC, 2014). The use of tetrachloroethylene has changed over the years. In the mid-20th century, tetrachloroethylene was primarily used in the dry-cleaning industry and was the primary organic solvent used for vapour degreasing in metal-cleaning operations (IARC, 2014). In the 1980s, changes in use coincided with the introduction of environmental regulations and improved technology controls in Canada and internationally (Canada, 2011a; Canada, 2011b; IARC, 2014). Since the 1990s, the most common use of tetrachloroethylene was as a feedstock for producing fluorocarbons (IARC, 2014). However, under the *Montreal Protocol on Substances that Deplete the Ozone layer*, the production of chlorofluorocarbons is being phased out by 2030 (IARC, 2014; UNEP, 2007). In Canada, tetrachloroethylene production ceased in 1992 and, since then, importation has continued primarily for domestic use as a chemical feedstock and as a solvent in the dry-cleaning and metal-cleaning industries (Environment Canada and Health Canada, 1993; Health Canada, 2015a).

Releases of tetrachloroethylene are mainly to the atmosphere by evaporative losses from anthropogenic sources (ATSDR, 1997; Environment Canada and Health Canada, 1993). Use and disposal of tetrachloroethylene and tetrachloroethylene-containing products can also result in releases to the environment via wastewater. A small amount of tetrachloroethylene is

produced naturally in the environment by marine algae (Abrahamsson et al., 1995).

The primary route of exposure to tetrachloroethylene for the general population is through inhalation of indoor air containing tetrachloroethylene emitted by freshly dry-cleaned clothes, automotive products, and other consumer products containing tetracholroethylene (Environment Canada and Health Canada, 1993). Tetrachloroethylene has been detected in drinking water; the ingestion of drinking water is, generally, a minor contributor to overall tetrachloroethylene exposure (Environment Canada and Health Canada, 1993). Exposure can also occur during the use of consumer products containing tetrachloroethylene, and from ambient air and food (ATSDR, 1997; Environment Canada and Health Canada, 1993). Living near a dry-cleaning facility may also increase the potential for exposure (ATSDR, 1997; CDC, 2009; IARC, 2014).

Tetrachloroethylene is rapidly absorbed into the blood and is distributed throughout the body with some concentration in adipose tissue (ATSDR, 1997; Environment Canada and Health Canada, 1993; IARC, 2014). Tetrachloroethylene is metabolized in the kidney, liver, and lungs forming the major metabolite trichloroacetic acid (TCA) and other minor metabolites including trichloroethanol (IARC, 2014). Absorbed tetrachloroethylene is rapidly eliminated unchanged from the body within minutes and hours via exhalation, followed by a slower excretion of metabolites in urine (IARC, 2014). The half-lives of tetrachloroethylene in vessel-rich tissue, muscle tissue, and adipose tissue are estimated to be 12 to 16, 30 to 40, and 55 hours, respectively (ATSDR, 1997). Tetrachloroethylene metabolites can be measured in urine whereas tetrachloroethylene can be measured in exhaled air and blood; the latter is considered the most reliable biomarker of recent exposure (ATSDR, 1997; IARC, 2014).

Exposure to tetrachloroethylene is known to cause a number of health effects in humans. Acute exposure via inhalation, ingestion, and skin contact can result in irritation of membranes (ATSDR, 1997). At very high concentrations, acute inhalation and oral exposure to tetrachloroethylene can induce atrophy of olfactory nerves, tremors, and central nervous system depression, as well as kidney and liver dysfunction in laboratory animals; these symptoms are similar to those observed in humans following accidental poisonings and solvent abuse (ATSDR, 1997; Environment Canada and Health Canada, 1993). Tetrachloroethylene exposure is also associated with narcotic and anesthetic effects increasing in severity with increasing exposure (ATSDR, 1997; Environment Canada and Health Canada, 1993; EPA, 2012). These neurological symptoms may be reversible following cessation of acute exposure; however, chronic exposures may result in more persistent neurological impairments (ATSDR, 1997; Environment Canada and Health Canada, 1993; IARC, 2014). Multiple cancer sites of interest have been evaluated by the International Agency for Research (IARC) on Cancer Expert Working Group and positive associations for cancer of the bladder in humans are consistently found (IARC, 2014). Tetrachloroethylene has been classified by IARC as probably carcinogenic to humans (Group 2A), on the basis of limited evidence in humans and sufficient evidence in laboratory animals, and as possibly carcinogenic to humans (Group III) by Environment Canada and Health Canada (Environment Canada and Health Canada, 1993; IARC, 2014).

The Government of Canada conducted a scientific assessment on the impact of tetrachloroethylene exposure on humans and the environment and concluded that it is toxic to the environment, but not to human health, as per criteria set out under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Environment Canada and Health Canada, 1993). Tetrachloroethylene is listed on Schedule 1, List of Toxic Substances, under CEPA 1999, and is a risk-managed substance involving a full life cycle management approach to prevent or minimize its release into the environment (Canada, 1999). In Canada, Regulations for Tetrachloroethylene Use in Dry Cleaning and Reporting Requirements have been introduced to reduce releases of tetrachloroethylene from dry-cleaning facilities (Canada, 2011a; Canada, 2011b). The Government of Canada has also introduced Solvent Degreasing Regulations to reduce total Canadian consumption of trichloroethylene and tetrachloroethylene used in solvent-degreasing operations (Canada, 2011b; Environment Canada, 2013). Tetrachloroethylene is included as a prohibited ingredient on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative

tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene the general prohibition found in section 16 of the *Food and Drugs Act* or a provision of the Cosmetic Regulations (Canada, 1985; Health Canada, 2015b). In addition, Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes the maximum acceptable concentration for tetrachloroethylene in drinking water that is protective of human health (Health Canada, 2015a). This guideline was developed based on neurological effects observed in humans and experimental animals and is considered protective of both cancer and non-cancer effects.

Tetrachloroethylene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood. Finding a measurable amount of tetrachloroethylene in blood can be an indicator of exposure to tetrachloroethylene and does not necessarily mean that an adverse health effect will occur.

Tetrachloroethylene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.4.1

Tetrachloroethylene (Perchloroethylene) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2453	60.82	—	<LOD	<LOD	0.10 (0.067–0.14)	0.17[E] (0.10–0.23)
4 (2014–2015)	2527	70.32	—	<LOD	<LOD	0.066[E] (0.022–0.11)	F
Males, 12–79 years							
3 (2012–2013)	1228	58.96	—	<LOD	<LOD	0.13 (0.086–0.17)	0.19 (0.13–0.25)
4 (2014–2015)	1251	70.02	—	<LOD	<LOD	F	F
Females, 12–79 years							
3 (2012–2013)	1225	62.69	—	<LOD	<LOD	0.096[E] (0.060–0.13)	0.13[E] (0.039–0.22)
4 (2014–2015)	1276	70.61	—	<LOD	<LOD	0.068[E] (<LOD–0.12)	F
12–19 years							
3 (2012–2013)	739	60.76	—	<LOD	<LOD	F	F
4 (2014–2015)	713	77.00	—	<LOD	<LOD	0.042[E] (<LOD–0.065)	F
20–39 years							
3 (2012–2013)	543	60.04	—	<LOD	<LOD	0.093[E] (0.052–0.13)	0.15[E] (0.080–0.23)
4 (2014–2015)	600	68.50	—	<LOD	<LOD	F	F
40–59 years							
3 (2012–2013)	587	65.08	—	<LOD	<LOD	0.10[E] (0.058–0.14)	0.13 (0.089–0.17)
4 (2014–2015)	625	70.08	—	<LOD	<LOD	0.061[E] (<LOD–0.10)	F
60–79 years							

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3 (2012–2013)	584	57.36	—	<LOD	<LOD	0.16[E] (0.062–0.25)	F
4 (2014–2015)	589	64.35	—	<LOD	<LOD	0.088[E] (0.028–0.15)	F

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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15.5 TOLUENE

Toluene (CASRN 108-88-3) is a colourless liquid and volatile organic compound (VOC). It is produced commercially, primarily through the conversion of petroleum to gasoline and other fuels or recovered as a by-product in the coke oven and styrene-manufacturing industries (ATSDR, 2000; Environment Canada and Health Canada, 1992).

Toluene is used widely as an industrial solvent and an intermediate in the production of a variety of chemicals. Major uses of toluene have included the manufacture of benzene, benzene derivatives, trinitrotoluene and toluene diisocyanate, and in the blending of gasoline fuels as octane boosters (ATSDR, 2000; CDC, 2009). It has also been widely used as a solvent in paints and finishes, adhesives, polymers and resins, dyes, automotive products, and some personal care products (ATSDR, 2000; Environment Canada and Health Canada, 1992; Health Canada, 2016). The use of toluene in solvent-based products and processes has decreased as alternative formulations with lower VOC content, as well as alcohol-based and water-based products and processes, are now available.

Toluene is released to the environment from natural and anthropogenic sources. It has been measured in emissions from volcanoes, forest fires, natural gas deposits, and crude oil (ATSDR, 2000; Environment Canada and Health Canada, 1992). Anthropogenic sources of atmospheric toluene include primarily the volatilization of petroleum fuels, toluene-based solvents and thinners, motor vehicle exhaust, and the off-gassing of toluene from some building materials, consumer, and automotive products (ATSDR, 2000; Environment Canada and Health Canada, 1992). Toluene can also be released to the environment in waste from manufacturing and processing facilities, from spills and accidental releases, and from the disposal of toluene-containing products (ATSDR, 2000; CCME, 2004; Environment Canada and Health Canada, 1992).

The general population is exposed to toluene mainly through the inhalation of indoor air (Health Canada, 2011). Cigarette smoking may significantly increase exposure and, in smokers, inhalation of cigarette smoke is expected to be a major contributor to the total toluene exposure (ATSDR, 2000; Health Canada, 2011; Health Canada, 2012). Inside residences, toluene levels in air have been shown to be higher in newer homes and homes with a garage on the property, and in homes where paint or paint remover has been used in the previous week (Wheeler et al., 2013). Toluene is also found in tobacco smoke, and regular smoking in the home is a significant predictor of toluene in indoor air (Health Canada, 2012). Although toluene has been detected in drinking water and in certain foods, these are not considered to constitute major sources of exposure for the general population (Environment Canada and Health Canada, 1992; Health Canada, 2014).

Following inhalation, toluene is readily absorbed and distributed throughout the body (ATSDR, 2000; Environment Canada and Health Canada, 1992). The majority of absorbed toluene is rapidly eliminated from the body with a small amount in adipose tissues eliminated more slowly (ATSDR, 2000). Up to 20% of absorbed toluene is exhaled unchanged and less than 1% is excreted unchanged in the urine (ATSDR, 2000; Donald et al., 1991). The elimination of toluene following inhalation has half-lives ranging from less than 3 minutes to 12 hours in blood and from 0.5 to 3 days in subcutaneous adipose tissues of humans (ATSDR, 2000). Toluene levels in blood are the most accurate biomarker of toluene exposure and are reflective of recent exposure (ATSDR, 2000; CDC, 2009).

Toluene exposure can be irritating to the eyes, nose, throat, lungs and skin, and has been associated with symptoms of headaches, dizziness, reduced coordination, and feelings of intoxication (ATSDR, 2000; CCOHS, 2013; Health Canada, 2011; Health Canada, 2012; IARC, 1999). Acute inhalation exposure has been generally associated with reversible neurological symptoms whereas chronic exposure is associated with impaired neurological function including cognitive and neuromuscular performance, as well as negative effects on colour vision and hearing (ATSDR, 2000; CCOHS, 2013; CDC, 2009; Health

Canada, 2011; IARC, 1999). Studies in laboratory animals exposed to toluene provide supporting evidence for behavioural changes, hearing loss and subtle changes in brain structure, brain electrophysiology, and brain chemistry (ATSDR, 2000; Bowen and Hannigan, 2006; Gospe and Zhou, 2000). Exposure to high levels of toluene in humans during pregnancy has been associated with fetal toxicity and developmental effects in children, at levels associated with potential maternal toxicity such as in solvent abuse (ATSDR, 2000; Bowen and Hannigan, 2006; Donald et al., 1991; Yücel et al., 2008). Toluene carcinogenicity to humans is not classifiable according to the International Agency for Research on Cancer (Group 3) and the U.S. Environmental Protection Agency (Group D) (EPA, 2005; IARC, 1999).

Under the *Canadian Environmental Protection Act, 1999* (CEPA 1999), Health Canada and Environment Canada concluded that at current environmental concentrations, toluene is not a concern for human life or health (Environment Canada and Health Canada, 1992). Toluene is also part of a larger class of VOCs that, as a group, are environmental and health concerns because of their contribution to the formation of smog. The Government of Canada has taken and proposed a number of actions to address VOC emissions resulting from the use of consumer and commercial products in Canada (Canada, 2009a; Canada, 2009b; Environment Canada, 2002; Environment Canada, 2013).

In 2011, Health Canada released a residential indoor air quality guideline for both short- and long-term exposure to toluene (Health Canada, 2011). Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for toluene that is protective of human health, as well as an aesthetic objective for toluene based on its odour threshold (Health Canada, 2014). The guideline was developed based on several neurological endpoints reported in human occupational studies.

Toluene was analyzed in the whole blood of Canadian Health Measures Survey participants aged 12 to 79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood. Finding a measurable amount of toluene in blood can be an indicator of recent exposure to toluene and does not necessarily mean that an adverse health effect will occur.

Toluene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Statistics Canada, 2012; Wheeler et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.5.1

Toluene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2449	0.69	0.096 (0.083–0.11)	0.036 (0.030–0.042)	0.079 (0.067–0.090)	0.39 (0.32–0.46)	0.58 (0.46–0.71)
4 (2014–2015)	2384	0.08	0.12 (0.094–0.16)	0.044 (0.028–0.059)	0.11 (0.076–0.14)	0.42 (0.27–0.58)	0.55 (0.39–0.71)
Males, 12–79 years							
3 (2012–2013)	1224	0.65	0.098 (0.081–0.12)	0.034 (0.025–0.043)	0.081 (0.066–0.095)	0.42 (0.33–0.51)	0.59 (0.42–0.77)
4 (2014–2015)	1182	0.17	0.13 (0.10–0.18)	0.044[E] (0.023–0.065)	0.12 (0.085–0.15)	0.46 (0.30–0.61)	0.65 (0.41–0.88)
Females, 12–79 years							
3 (2012–2013)	1225	0.73	0.093 (0.081–0.11)	0.037 (0.034–0.041)	0.077 (0.064–0.089)	0.35 (0.24–0.46)	0.55[E] (0.34–0.76)
4 (2014–2015)	1202	0	0.11 (0.086–0.15)	0.043 (0.030–0.055)	0.10[E] (0.058–0.14)	0.37[E] (0.17–0.57)	0.53 (0.43–0.64)
12–19 years							
3 (2012–2013)	732	0.55	0.074 (0.066–0.083)	0.034 (0.026–0.042)	0.070 (0.058–0.082)	0.19 (0.14–0.24)	0.26 (0.19–0.32)
4 (2014–2015)	681	0	0.096 (0.070–0.13)	0.039 (0.028–0.050)	0.097 (0.061–0.13)	0.22[E] (0.14–0.31)	0.30[E] (0.17–0.44)
20–39 years							
3 (2012–2013)	533	0.94	0.089 (0.069–0.11)	0.036 (0.028–0.045)	0.074 (0.050–0.098)	0.29[E] (0.16–0.43)	0.42[E] (0.23–0.61)
4 (2014–2015)	574	0	0.12 (0.094–0.16)	0.047[E] (0.027–0.067)	0.12[E] (0.076–0.17)	0.30[E] (0.19–0.41)	0.46 (0.30–0.61)
40–59 years							
3 (2012–2013)	594	0.51	0.12 (0.10–0.14)	0.041 (0.033–0.049)	0.085 (0.071–0.10)	0.58 (0.38–0.79)	0.86 (0.64–1.1)
4 (2014–2015)	580	0.17	0.13 (0.10–0.18)	0.045 (0.029–0.060)	0.11 (0.071–0.14)	0.51 (0.34–0.67)	0.72 (0.55–0.88)
60–79 years							
3 (2012–2013)	590	0.85	0.086 (0.070–0.11)	0.031 (0.024–0.039)	0.080 (0.065–0.096)	0.31 (0.22–0.40)	0.46 (0.39–0.53)
4 (2014–2015)	549	0.18	0.12 (0.089–0.16)	0.038[E] (0.023–0.054)	0.099[E] (0.061–0.14)	0.49 (0.34–0.64)	0.70 (0.46–0.94)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

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15.6 TRICHLOROETHYLENE

Trichloroethylene (CASRN 79-01-6) is a colourless liquid classified as a volatile organic compound. It has been produced commercially by chlorination of acetylene and ethylene since the 1920s (ATSDR, 1997; IARC, 1995). There has been a general decline in demand for trichloroethylene over the years (Health Canada, 2005; IARC, 2014). This decline may be due to several factors, including use of alternative solvents, an increase in solvent recovery/recycling by users, and the introduction of regulations and controls to address concerns about environmental, health, and safety implications of chlorinated solvents (Health Canada, 2005; IARC, 2014). In Canada, production of trichloroethylene stopped in 1985 (Health Canada, 2005). Since then, it continues to be imported for use primarily as a solvent in the vapour-degreasing and cold-cleaning of metal parts and, in smaller amounts, in dry-cleaning operations, specialty paints and paint removers, and various other household products (Environment Canada, 2013a; Environment Canada, 2013b; Health Canada, 2005). Trichloroethylene is also used as a chemical intermediate in the production of other chemicals (IARC, 2014).

Trichloroethylene enters the environment primarily through evaporation from anthropogenic sources (ATSDR, 1997; Environment Canada, 2013b). Although the majority of anthropogenic releases enter the atmosphere, production, use, and disposal of trichloroethylene and trichloroethylene-containing products can also result in releases to the environment via wastewater. A small amount of trichloroethylene is produced naturally in the environment by marine algae (Abrahamsson et al., 1995).

The most common exposure route to trichloroethylene for the general population is inhalation of indoor air containing trichloroethylene emitted from specialty paints, adhesives, and household products (CDC, 2009; Environment Canada and Health Canada, 1993). Canadians may also be exposed to trichloroethylene through its presence in drinking water, air, and food (Health Canada, 2005).

Following all routes of exposure, trichloroethylene is rapidly and nearly completely absorbed into the blood and distributed throughout the body (ATSDR, 1997; Environment Canada and Health Canada, 1993; EPA, 2011). Absorbed trichloroethylene is rapidly distributed mainly to the brain, kidney, liver, muscle, and adipose tissue (ATSDR, 1997). Trichloroethylene is metabolized in the kidney, liver, and lungs forming the major metabolites trichloroacetic acid (TCA) and trichloroethanol (TCOH) (ATSDR, 1997; EPA, 2011). Absorbed trichloroethylene is rapidly eliminated from the body, within minutes and hours, via exhalation of trichloroethylene and urinary excretion of the metabolites along with minimal amounts of unchanged trichloroethylene (ATSDR, 1997; EPA, 2011). The most reliable biomarker of recent exposure to trichloroethylene is its measurement in blood and breath (ATSDR, 1997; IARC, 1995). Measurements of the metabolites TCA and TCOH in blood or urine are less reliable because of intra-individual differences in urinary concentrations and a lack of specificity for trichloroethylene exposure (ATSDR, 1997; IARC, 1995).

Exposure to trichloroethylene is known to cause a number of health effects in humans. Acute exposure via inhalation, ingestion, and skin contact can result in irritation of membranes (ATSDR, 1997; Health Canada, 2005; IARC, 1995). Trichloroethylene exposure is also associated with narcotic and anesthetic effects increasing in severity with increasing exposure (Environment Canada and Health Canada, 1993; IARC, 1995). These neurological symptoms may be reversible following cessation of acute exposure; however, chronic exposures may result in more persistent neurological impairments (ATSDR, 1997; Environment Canada and Health Canada, 1993; EPA, 2011). Recently, the International Agency for Research on Cancer updated its classification for trichloroethylene to Group 1 carcinogenic to humans, on the basis of new and sufficient evidence for cancer of the kidney in humans, with strong support from studies in laboratory animals (IARC, 2014). A positive association has also been shown between trichloroethylene exposure and cancers of the liver and biliary tract, and non-Hodgkin lymphoma (EPA, 2011; IARC, 2014; WHO, 2000).

The Government of Canada conducted a scientific assessment on the impact of trichloroethylene exposure

on humans and the environment and concluded that it may enter the environment in quantities or under conditions that may constitute a danger in Canada to human life or health as per criteria set out under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Environment Canada and Health Canada, 1993). Trichloroethylene is listed on Schedule 1, List of Toxic Substances, under CEPA 1999 (Canada, 1999). Under CEPA 1999, the Government of Canada published Solvent Degreasing Regulations to reduce total Canadian consumption of trichloroethylene and tetrachloroethylene used in solvent-degreasing operations (Environment Canada, 2013c). Improvements have also been implemented in the commercial dry-cleaning industry to prevent and minimize releases of dry-cleaning solvents, particularly trichloroethylene and tetrachloroethylene (Canada, 2011a; Canada, 2011b; IARC, 2014). The current guideline for Canadian drinking water quality, developed by Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, establishes the maximum acceptable concentration for trichloroethylene in drinking water (Health Canada, 2005). The guideline was developed based upon developmental toxicity and is considered protective for both cancer and non-cancer effects.

Trichloroethylene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood. Finding a measurable amount of trichloroethylene in blood can be an indicator of exposure to trichloroethylene and does not necessarily mean that an adverse health effect will occur.

Trichloroethylene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.6.1

Trichloroethylene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2474	99.51	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2527	99.49	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1240	99.35	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	99.20	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1234	99.68	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1276	99.76	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	746	99.73	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	100	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	543	99.63	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	98.83	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	594	99.33	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	99.52	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	591	99.32	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	589	99.49	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

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15.7 TRIHALOMETHANES

Disinfection by-products are a group of chemical compounds formed when water disinfection agents (e.g. chlorine, chloramines, ozone, chlorine dioxide) interact with organic precursors or bromide naturally present in water (CCME, 1999; CDC, 2009; Health Canada, 2006). Disinfection by-products include, among others, trihalomethanes (THMs), haloacetic acids, haloacetonitriles, haloketones, and chlorophenols. THM formation increases as a function of the concentration of chlorine and organic matter; in the presence of bromide, brominated THMs are formed (Health Canada, 2006). In cycle 4 of the Canadian Health Measures Survey (CHMS), four THMs were measured: bromodichloromethane, dibromochloromethane, bromoform (tribromomethane), and chloroform (trichloromethane). Each of these compounds consists of three halogen groups attached to a single carbon atom and are classified as volatile organic compounds (VOCs) (CCME, 1999). Chloroform is the most common THM and the most frequently measured disinfection by-product in chlorinated drinking water in Canada (ATSDR, 2005; Health Canada, 2006).

Table 15.7.1
Trihalomethanes measured in the Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015)

Trihalomethanes	CASRN
Bromodichloromethane	75-27-4
Dibromochloromethane	124-48-1
Tribromomethane (Bromoform)	75-25-2
Trichloromethane (Chloroform)	67-66-3

The four THMs are also commercially produced chemicals (ATSDR, 1999; ATSDR, 2005). Chloroform and bromodichloromethane are used as chemical intermediates in the manufacturing of organic chemicals and as solvents, although chloroform has not been manufactured in Canada since 1978 (ATSDR, 2005; Health Canada, 2006). In Canada, the use of chloroform as an anaesthetic has been discontinued and its use in dentifrices, liniments, and antitussives has been banned (CCME, 1999; Environment Canada and Health Canada, 2001). Dibromochloromethane is used as an intermediate in the manufacture of refrigerants, pesticides, propellants, and other organic chemicals (Health Canada, 2006). Bromoform is used as a solvent in the synthesis of pharmaceuticals and in fire-resistant chemicals, as well as gauge fluid used in the aircraft and shipbuilding industries (Health Canada, 2006).

A small proportion of THMs present in the environment may be due to natural production by marine algae and by natural degradation and transformation processes (ATSDR, 1999; ATSDR, 2005). Anthropogenic sources are generally considered to be larger contributors of THMs in the environment than natural ones. In Canada, the major anthropogenic sources of THMs are disinfected water from drinking water treatment plants, chlorinated effluents from municipal wastewater treatment plants and industrial plants, and cooling waters from power plants and industrial plants (Environment Canada and Health Canada, 1993). Chlorine use in the treatment of drinking water has virtually eliminated waterborne diseases because of its ability to kill or inactivate most microorganisms commonly found in water (Health Canada, 2006). It is used in the majority of drinking water treatment plants in Canada to treat the water directly in the treatment plant and/or to maintain a chlorine residual in the distribution system to prevent bacterial regrowth (Health Canada, 2006). Effluent wastewaters are disinfected to protect downstream municipal water supplies, recreational waters, and shellfish-growing areas from bacterial contamination and other microorganisms causing water-borne disease (Environment Canada and Health Canada, 1993). In addition to drinking water, disinfection effluents, and cooling waters, anthropogenic sources of THMs include chemical manufacturing plants and industrial sites, swimming pools, hot tubs, and water parks (ATSDR, 2005; CCME, 1999; Health Canada, 2006).

The general population is exposed to THMs primarily by drinking chlorinated water (CDC, 2009; Environment Canada and Health Canada, 2001; Health Canada, 2006). Exposure also occurs through inhalation during showering and bathing, and by skin absorption during bathing and swimming (CDC, 2009; Health Canada, 2006). Minor exposures may occur from the consumption of food and beverages (Health Canada, 2006). Swimming pools and hot tubs are additional sources of THM exposure (Aggazzotti et al., 1998).

Following ingestion, all four THMs are rapidly absorbed into the blood and distributed throughout the body, primarily in the fat, blood, liver, kidney, lungs, and nervous system (ATSDR, 1989; Health Canada, 2006; WHO, 2004). THMs are well absorbed following both oral and inhalation exposure, with dermal exposure as another potentially significant route of exposure (ATSDR, 1989; Health Canada, 2006; IPCS, 2000; WHO, 2004). Estimated half-lives for THMs in the body generally range from 1.5 hours to 6 hours; about 95% of absorbed bromodichloromethane is eliminated from the body in 8 hours (ATSDR, 1989; Health Canada, 2006; WHO, 2004). Absorbed THMs are mainly eliminated from the body by exhalation of unchanged compounds and volatile metabolites, with only minor amounts excreted in the urine and less in the feces (Health Canada, 2006; IPCS, 2000). Unchanged disinfection by-products measured in blood are the most accurate biomarkers of exposure and reflect recent exposures (CDC, 2009).

Each of the four THMs is irritating to the eyes and respiratory tract, and acute inhalation exposure has been associated with reddening of the face (Health Canada, 2006; IPCS, 2000; WHO, 2004). Acute high-level inhalation and oral exposures to these disinfection by-products in laboratory animals induce general narcotic and anesthetic effects increasing in severity with exposure level and are generally reversible following cessation of exposure (Health Canada, 2006; IPCS, 2000; WHO, 2004). Some studies in laboratory animals indicate that THMs containing bromine, such as bromodichloromethane, may be more toxic than chloroform and other chlorine-containing disinfection by-products (Health Canada, 2006). Chronic exposures to THMs in drinking water are weakly and inconsistently associated with cancers of the liver, kidney, colon, rectal, brain, pancreas, and bladder (Health Canada, 2006; IPCS, 2000; WHO, 2004). Results of studies in laboratory animals chronically exposed by the oral route to high levels of individual THMs provide supporting evidence of an association among cancers of the kidney, liver, and intestines with exposures to disinfection by-products (ATSDR, 1989; Health Canada, 2006; WHO, 2004). Based upon available evidence in laboratory animals, chloroform and bromodichloromethane have been classified as possibly carcinogenic to humans (Group 2B) by the International Agency for Research on Cancer (IARC, 1999a; IARC, 1999b). There is insufficient evidence to determine whether or not bromoform, dibromochloromethane, and chlorinated drinking water are carcinogenic (IARC, 1991; IARC, 1999a).

Health Canada and Environment Canada have reviewed and assessed chlorinated wastewater effluents, defined as those effluents to which chlorine or chlorination agents are added for disinfection, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The screening assessment concluded that chlorinated wastewater effluents discharged to the Canadian environment by municipal wastewater treatment plants are a concern for the environment (Environment Canada and Health Canada, 1993). However, there is insufficient information to determine whether chlorinated wastewater effluents are harmful to human health. Chlorinated wastewater effluents are listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999). Under Canada's Food and Drugs Regulations, manufacturers are not permitted to import or sell a drug for human use in Canada that contains chloroform (Canada, 2012; Environment Canada and Health Canada, 2001).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for total THMs (defined as the sum of bromodichloromethane, dibromochloromethane, bromoform, and chloroform) in drinking water (Health Canada, 2006). The Canadian guideline states that utilities should make every effort to maintain concentrations as low as reasonably achievable without compromising the effectiveness of disinfection (Health Canada, 2006). The approach to reducing THM exposure is generally focused on reducing the formation of chlorinated disinfection by-products. This can be achieved by removing organic matter from the water before chlorine is added, by optimizing the disinfection

process or using alternative disinfection strategies, or by using a different water source.

Chloroform, bromoform, dibromochloromethane, and bromodichloromethane were analyzed in the whole blood of CHMS cycle 3 (2012–2013) and cycle 4 (2014–2015) participants aged 12–79 years. Data are presented as µg/L blood. Finding a measurable amount of THMs in blood can be an indicator of exposure to THMs and does not necessarily mean that an adverse health effect will occur.

Table 15.7.2

Bromodichloromethane — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2499	98.88	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2527	96.91	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1245	98.96	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	97.36	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1254	98.80	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1276	96.47	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	744	98.12	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	96.35	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	556	98.92	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	97.67	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	595	99.66	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	96.00	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	604	99.01	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	589	97.79	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 15.7.3

Dibromochloromethane — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2527	97.07	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2499	96.24	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1263	96.52	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1233	96.76	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1264	97.63	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1266	95.73	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	757	96.83	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	704	96.45	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	557	97.13	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	596	96.14	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	604	98.01	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	617	95.62	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	609	96.39	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	582	96.74	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

Table 15.7.4

Tribromomethane (Bromoform) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2496	94.79	—	<LOD	<LOD	<LOD	0.010[E] (<LOD–0.015)
4 (2014–2015)	2527	97.39	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1244	95.02	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	97.44	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1252	94.57	—	<LOD	<LOD	<LOD	<LOD[E] (<LOD–0.013)
4 (2014–2015)	1276	97.34	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	744	94.49	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	97.05	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	554	94.40	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	97.67	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	595	96.47	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	97.60	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	603	93.86	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	589	97.28	—	<LOD	<LOD	<LOD	<LOD

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 15.7.5

Trichloromethane (Chloroform) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2527	77.44	—	<LOD	<LOD	0.021 (0.016–0.026)	0.029 (0.019–0.038)
4 (2014–2015)	2527	75.78	—	<LOD	<LOD	0.028[E] (<LOD–0.043)	0.043[E] (0.022–0.064)
Males, 12–79 years							
3 (2012–2013)	1263	77.51	—	<LOD	<LOD	0.021 (0.015–0.027)	0.035[E] (0.018–0.052)
4 (2014–2015)	1251	77.22	—	<LOD	<LOD	F	0.046[E] (0.022–0.069)
Females, 12–79 years							
3 (2012–2013)	1264	77.37	—	<LOD	<LOD	0.021 (0.016–0.027)	0.028 (0.019–0.037)
4 (2014–2015)	1276	74.37	—	<LOD	<LOD	0.030[E] (0.016–0.045)	0.039[E] (0.016–0.062)
12–19 years							
3 (2012–2013)	757	77.81	—	<LOD	<LOD	0.020[E] (<LOD–0.028)	0.031[E] (<LOD–0.049)
4 (2014–2015)	713	75.32	—	<LOD	<LOD	0.028[E] (0.017–0.038)	0.040[E] (0.015–0.066)
20–39 years							
3 (2012–2013)	557	76.48	—	<LOD	<LOD	0.023 (0.016–0.029)	0.036[E] (0.015–0.058)
4 (2014–2015)	600	75.33	—	<LOD	<LOD	0.030[E] (0.016–0.045)	F
40–59 years							
3 (2012–2013)	604	78.81	—	<LOD	<LOD	0.019 (<LOD–0.025)	0.027 (0.019–0.036)
4 (2014–2015)	625	76.16	—	<LOD	<LOD	F	0.046[E] (0.024–0.067)
60–79 years							
3 (2012–2013)	609	76.52	—	<LOD	<LOD	0.020[E] (<LOD–0.027)	0.028[E] (<LOD–0.041)
4 (2014–2015)	589	76.40	—	<LOD	<LOD	0.027[E] (<LOD–0.040)	0.037[E] (0.019–0.056)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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15.8 XYLENES

Xylenes (CASRN 1330-20-7) are classified as volatile organic compounds (VOCs) (ATSDR, 2007; CCOHS, 2013; Environment Canada and Health Canada, 1993). The three isomers of xylene are *ortho*-xylene (*o*-xylene; CASRN 95-47-6), *meta*-xylene (*m*-xylene; CASRN 108-38-3) and *para*-xylene (*p*-xylene; CASRN 106-42-3); they differ from each other in the position of the two methyl group substitutions on the aromatic ring. The term "total xylenes" refers to all three isomers of xylene, whereas "mixed xylene" is a mixture of total xylenes and ethylbenzene (6% to 15%) (CCOHS, 2013). Xylenes are primarily produced either directly or as by-products of petroleum and coal refining and as by-products of olefin manufacturing (ATSDR, 2007; Environment Canada and Health Canada, 1993).

Xylenes have been extensively and increasingly used in a wide range of applications as a solvent, as a replacement for benzene in the solvent components of various commercial products, and as a mixture in gasoline (ATSDR, 2007). Xylenes may be widely used as a solvent in paint thinners, varnishes, lacquers, stains, concrete sealers, cleaning products, adhesives, inks, cleaning and degreasing agents, and in the production of dyes, perfumes, plastics, pharmaceuticals, and pesticides (ATSDR, 2007; Environment Canada and Health Canada, 1993; IPCS, 1997).

Xylenes are released to the environment from natural and anthropogenic sources. Xylenes have been measured in emissions from volcanoes, forest fires, and in volatiles from plants and vegetation (ATSDR, 2007; CCME, 2004). Anthropogenic sources of atmospheric xylenes include volatilization of petroleum fuels and xylene-based solvents and thinners, gasoline use and motor vehicle exhaust, and the off-gassing of xylenes from some building materials, and consumer and automotive products containing xylenes (ATSDR, 2007; Environment Canada and Health Canada, 1993). Xylenes are also released to the environment in waste from manufacturing and processing facilities, from spills and accidental releases, and from the disposal of xylene-containing products (ATSDR, 2007; CCME, 2004; Environment Canada, 2014). In the past, predominant sources of releases to the atmosphere included emissions from petroleum refineries and chemical manufacturing facilities of styrene-butadiene, rubber, solvents, paints, plastics, synthetic fabric polymers, and polyesters. As new emissions-free and low VOC technologies are being implemented, and changes in industrial and consumer use patterns and increases in fuel efficiency occur, releases of VOCs, including xylenes, are expected to continue their decline.

The general population is exposed to xylenes mainly through inhalation of indoor air (Environment Canada and Health Canada, 1993). Cigarette smoking may significantly increase levels in indoor air and, in smokers, inhalation of cigarette smoke is expected to be a major contribution to the total source of xylene exposure (ATSDR, 2007). In addition to smoking, xylene levels in air have been shown to be higher for homes with a garage on the property, with a higher number of occupants, with recent renovations, and in which fragrances and paint remover have been recently used (Wheeler et al., 2013). Additional exposure may result from the use of consumer products containing xylenes, from the use of gasoline-powered engines, such as lawn mowers and outboard motors, and from ambient air, water, soil, drinking water, and food (ATSDR, 2007; IARC, 1999; Wheeler et al., 2013). As xylenes are present as a mixture in gasoline and commercial products, the general population is expected to be primarily exposed to xylenes as a mixture, not to the separate xylene isomers (ATSDR, 2007).

Xylenes are rapidly absorbed by all routes of exposure and distributed throughout the body following exposure, primarily into adipose tissues and those tissues with higher lipid content, such as the liver and the brain (ATSDR, 2007; EPA, 2003; Health Canada, 2014). Elimination of xylenes from blood and most tissue compartments following inhalation is generally rapid, and in humans has a half-life ranging from about 1 to 20 hours (ATSDR, 2007). The major route of excretion of absorbed xylenes in the blood and body is excretion of metabolites in urine, with minor elimination by exhalation of unchanged chemical from the lungs (ATSDR, 2007). Xylene levels in the blood are the most accurate biomarker of xylene exposure and reflect recent exposure (ATSDR, 2007; IARC, 1999).

Adverse health effects have been observed in humans and laboratory animals following xylene exposure via inhalation, ingestion, and dermal routes. In humans, xylenes can be irritating to the eyes, nose, throat, lungs, and skin, and has been associated with symptoms of headaches, dizziness, reduced coordination, and feelings of intoxication (ATSDR, 2007; CCOHS, 2013). Acute inhalation exposure has been associated with reversible

neurological symptoms whereas chronic exposure is associated with impaired neurological function, including cognitive and neuromuscular performance, as well as hearing deficits and dermatitis in humans (ATSDR, 2007; IARC, 1999). In humans, acute exposure to xylenes by ingestion has been associated with stomach discomfort, and changes in liver and kidney function; ingestion of petroleum solvents can be fatal (ATSDR, 2007; IPCS, 1997). Exposure to high levels of mixed xylenes (and other solvents) in humans during pregnancy has been associated with fetal toxicity and developmental effects in children at levels associated with potential maternal toxicity, such as in solvent abuse (ATSDR, 2007; EPA, 2003; IPCS, 1997). Xylenes are not classifiable as to their carcinogenicity in humans according to Environment Canada and Health Canada (Group IV), the International Agency for Research on Cancer (Group 3) and the U.S. Environmental Protection Agency (Group D) (Environment Canada and Health Canada, 1993; EPA, 2003; IARC, 1999).

Under the *Canadian Environmental Protection Act, 1999* (CEPA 1999), Health Canada and Environment Canada concluded that xylenes are not entering the environment in quantities or under conditions that may constitute a danger to human life or health (Environment Canada and Health Canada, 1993). Xylenes also part of a larger class of VOCs that, as a group, are environmental and health concerns because of their contribution to the formation of smog. The Government of Canada has taken and proposed a number of actions to address VOC emissions resulting from the use of consumer and commercial products in Canada (Canada, 2009a; Canada, 2009b; Environment Canada, 2002; Environment Canada, 2013).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration for xylene that is protective of human health, as well as an aesthetic objective for xylenes based on their odour thresholds (Health Canada 2014).

Xylenes were analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). Data are presented as µg/L blood for *o*-xylene and the sum of *m*-xylene and *p*-xylene. Finding a measurable amount of xylenes in blood can be an indicator of recent exposure to xylene and does not necessarily mean that an adverse health effect will occur.

Xylenes were also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Statistics Canada, 2012; Wheeler et al., 2013), cycle 3 (2012–2013), and cycle 4 (2014–2015) and in tap water from households in cycles 3 and 4. Further details on the indoor air and tap water studies are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 4* (Statistics Canada, 2017). Indoor air and tap water data are available through Statistics Canada's Research Data Centres or upon request by contacting Statistics Canada at infostats@statcan.gc.ca.

Table 15.8.1

m-Xylene and *p*-xylene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2326	14.53	0.062 (0.050–0.079)	<LOD	0.063 (0.047–0.080)	0.20 (0.14–0.26)	0.30 (0.20–0.39)
4 (2014–2015)	2505	7.27	0.063 (0.053–0.075)	0.023 (<LOD–0.030)	0.061 (0.047–0.076)	0.18 (0.15–0.21)	0.26 (0.22–0.30)
Males, 12–79 years							
3 (2012–2013)	1172	13.31	0.065 (0.051–0.082)	<LOD	0.062 (0.045–0.080)	0.21 (0.15–0.28)	0.34[E] (0.19–0.49)
4 (2014–2015)	1239	6.86	0.069 (0.057–0.083)	<LOD	0.069 (0.055–0.084)	0.21 (0.15–0.27)	0.30 (0.22–0.39)
Females, 12–79 years							
3 (2012–2013)	1154	15.77	0.060 (0.047–0.078)	<LOD	0.064 (0.046–0.082)	0.19 (0.12–0.26)	0.27 (0.18–0.36)
4 (2014–2015)	1266	7.66	0.059 (0.049–0.069)	0.024 (<LOD–0.030)	0.056 (0.042–0.071)	0.16 (0.12–0.19)	0.21 (0.18–0.23)
12–19 years							
3 (2012–2013)	701	16.83	0.049 (0.037–0.065)	<LOD	0.055 (0.039–0.071)	0.14[E] (0.086–0.19)	0.18 (0.14–0.23)
4 (2014–2015)	709	7.90	0.054 (0.043–0.067)	0.024[E] (<LOD–0.033)	0.055 (0.044–0.066)	0.12 (0.092–0.14)	0.16 (0.12–0.20)
20–39 years							
3 (2012–2013)	500	14.00	0.058 (0.045–0.074)	<LOD	0.057[E] (0.026–0.088)	0.16 (0.11–0.22)	0.25 (0.17–0.32)
4 (2014–2015)	596	7.21	0.059 (0.046–0.076)	<LOD	0.055 (0.037–0.073)	0.16 (0.12–0.19)	F
40–59 years							
3 (2012–2013)	559	11.99	0.074 (0.056–0.096)	<LOD	0.068 (0.052–0.084)	0.28[E] (0.17–0.39)	0.42 (0.29–0.54)
4 (2014–2015)	622	6.43	0.067 (0.054–0.083)	<LOD[E] (<LOD–0.034)	0.069 (0.050–0.088)	0.21 (0.15–0.26)	0.27 (0.21–0.33)
60–79 years							
3 (2012–2013)	566	14.66	0.060 (0.045–0.079)	<LOD	0.061 (0.043–0.078)	0.18 (0.15–0.21)	0.25[E] (0.12–0.37)
4 (2014–2015)	578	7.44	0.071 (0.063–0.080)	0.025 (<LOD–0.034)	0.068 (0.057–0.079)	0.22 (0.17–0.27)	0.31 (0.23–0.39)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

Table 15.8.2

o-Xylene — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 12–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013) and cycle 4 (2014–2015).

Cycle	n	%<LOD[a]	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2336	41.05	—	<LOD	0.022[E] (0.010–0.034)	0.087 (0.061–0.11)	0.11 (0.083–0.14)
4 (2014–2015)	2428	29.57	0.015 (0.012–0.019)	<LOD	0.016 (0.011–0.020)	0.056 (0.045–0.066)	0.082 (0.063–0.10)
Males, 12–79 years							
3 (2012–2013)	1164	40.55	—	<LOD	0.022[E] (0.0097–0.033)	0.088 (0.061–0.11)	0.12 (0.075–0.16)
4 (2014–2015)	1198	27.71	0.017 (0.013–0.021)	<LOD	0.017 (0.012–0.023)	0.065 (0.047–0.082)	0.097[E] (0.044–0.15)
Females, 12–79 years							
3 (2012–2013)	1172	41.55	—	<LOD	0.022[E] (0.011–0.034)	0.081 (0.052–0.11)	0.11 (0.082–0.14)
4 (2014–2015)	1230	31.38	0.014 (0.011–0.017)	<LOD	0.015 (0.010–0.019)	0.049 (0.039–0.058)	0.064 (0.048–0.080)
12–19 years							
3 (2012–2013)	692	43.93	—	<LOD	F	0.057 (0.041–0.072)	0.075 (0.053–0.098)
4 (2014–2015)	687	32.17	0.013 (0.0099–0.017)	<LOD	0.014 (0.0090–0.019)	0.041 (0.028–0.053)	0.052 (0.038–0.067)
20–39 years							
3 (2012–2013)	515	42.14	—	<LOD	0.020[E] (0.0095–0.030)	0.077[E] (0.036–0.12)	0.11[E] (0.053–0.17)
4 (2014–2015)	580	32.41	0.012 (0.0090–0.017)	<LOD	0.012[E] (<LOD–0.018)	0.046 (0.036–0.057)	F
40–59 years							
3 (2012–2013)	565	38.94	0.022[E] (0.014–0.034)	<LOD	0.029[E] (0.012–0.045)	0.099 (0.075–0.12)	0.13 (0.095–0.17)
4 (2014–2015)	604	26.49	0.017 (0.014–0.021)	<LOD	0.018 (0.012–0.023)	0.060 (0.049–0.071)	0.087 (0.063–0.11)
60–79 years							
3 (2012–2013)	564	38.65	0.016[E] (0.010–0.023)	<LOD	0.016[E] (<LOD–0.027)	0.076 (0.055–0.098)	0.10[E] (0.030–0.17)
4 (2014–2015)	557	26.75	0.018 (0.016–0.021)	<LOD	0.019 (0.015–0.023)	0.077 (0.058–0.096)	0.096 (0.070–0.12)

a If >40% of samples were below the LOD, the percentile distribution is reported but means were not calculated.

E Use data with caution.

F Data is too unreliable to be published.

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APPENDIX A

LIMITS OF DETECTION

Laboratory analyses of environmental chemicals and creatinine were performed at analytical laboratories within Health Canada, l'Institut national de santé publique du Québec, and the ALS Laboratory Group. Laboratories developed standardized operating procedures for the analytical methods used to measure environmental chemicals or their metabolites in biological samples. The limit of detection (LOD) is defined as the lowest concentration of the analyte whose analytical response is measured to be greater than the noise level with 99% confidence and evaluated using U.S. Environmental Protection Agency methodology (EPA, 2015).

Limits of Detection

Chemical	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Acrylamide				
Acrylamide haemoglobin (Hb) adduct	—	—	11 pmol/g Hb	11 pmol/g Hb
Glycidamide haemoglobin (Hb) adduct	—	—	23 pmol/g Hb	23 pmol/g Hb
Environmental phenols				
Bisphenol A	0.2 µg/L	0.2 µg/L	0.23 µg/L	0.23 µg/L
Triclosan	—	3 µg/L	4.8 µg/L	4.8 µg/L
Metals and trace elements in blood				
Cadmium	0.04 µg/L	0.04 µg/L	0.080 µg/L	0.080 µg/L
Lead	0.02 µg/dL	0.1 µg/dL	0.16 µg/dL	0.16 µg/dL
Mercury (total)	0.1 µg/L	0.1 µg/L	0.42 µg/L	0.42 µg/L
Methylmercury	—	—	0.19 µg/L	0.19 µg/L
Metals and trace elements in urine				
Fluoride	—	0.02 mg/L	0.012 mg/L	0.012 mg/L
Mercury (inorganic)	0.1 µg/L	—	0.16 µg/L	0.16 µg/L
Arsenic (speciated)				
Arsenate	—	0.8 µg As/L[a]	0.75 µg As/L	0.75 µg As/L
Arsenite	—	0.8 µg As/L[a]	0.75 µg As/L	0.75 µg As/L
Arsenocholine	—	—	0.75 µg As/L	0.75 µg As/L
Arsenocholine and arsenobetaine	—	0.8 µg As/L[a]	0.75 µg As/L	0.75 µg As/L
Dimethylarsinic acid	—	0.8 µg As/L[a]	0.75 µg As/L	0.75 µg As/L
Monomethylarsonic acid	—	0.8 µg As/L[a]	0.75 µg As/L	0.75 µg As/L

Chemical	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Nicotine metabolite				
Cotinine	1 µg/L	1 µg/L	1.1 µg/L	1.1 µg/L
Organophosphate pesticide metabolites				
Chlorpyrifos metabolite				
3,5,6-Trichloro-2-pyridinol	—	—	NA[b]	0.13 µg/L
Malathion metabolite				
Malathion dicarboxylic acid	—	—	NA[b]	0.19 µg/L
Parabens				
Methyl paraben	—	—	NA[b]	1.3 µg/L
Ethyl paraben	—	—	NA[b]	0.90 µg/L
Propyl paraben	—	—	NA[b]	0.30 µg/L
Butyl paraben	—	—	NA[b]	0.30 µg/L
Polycyclic aromatic hydrocarbon metabolites				
Benzo[*a*]pyrene metabolite				
3-Hydroxybenzo[*a*]pyrene	—	0.002 µg/L	0.0029 µg/L	0.0029 µg/L
Chrysene metabolites				
2-Hydroxychrysene	—	0.004 µg/L	0.0054 µg/L	0.0054 µg/L
3-Hydroxychrysene	—	0.003 µg/L	0.0026 µg/L	0.0026 µg/L
4-Hydroxychrysene	—	0.003 µg/L	0.0023 µg/L	0.0023 µg/L
6-Hydroxychrysene	—	0.006 µg/L	0.0025 µg/L	0.0025 µg/L
Fluoranthene metabolite				
3-Hydroxyfluoranthene	—	0.008 µg/L	0.0080 µg/L	0.0080 µg/L
Fluorene metabolites				
2-Hydroxyfluorene	—	0.003 µg/L	0.0064 µg/L	0.0064 µg/L
3-Hydroxyfluorene	—	0.001 µg/L	0.0020 µg/L	0.0020 µg/L
9-Hydroxyfluorene	—	0.003 µg/L	0.0045 µg/L	0.0045 µg/L
Naphthalene metabolites				
1-Hydroxynaphthalene	—	0.1 µg/L	0.021 µg/L	0.021 µg/L
2-Hydroxynaphthalene	—	0.05 µg/L	0.031 µg/L	0.031 µg/L
Phenanthrene metabolites				
1-Hydroxyphenanthrene	—	0.005 µg/L	0.0024 µg/L	0.0024 µg/L
2-Hydroxyphenanthrene	—	0.003 µg/L	0.0025 µg/L	0.0025 µg/L
3-Hydroxyphenanthrene	—	0.003 µg/L	0.0021 µg/L	0.0021 µg/L
4-Hydroxyphenanthrene	—	0.001 µg/L	0.0031 µg/L	0.0031 µg/L
9-Hydroxyphenanthrene	—	0.004 µg/L	0.0040 µg/L	0.0040 µg/L
Pyrene metabolite				
1-Hydroxypyrene	—	0.002 µg/L	0.0029 µg/L	0.0029 µg/L
Volatile organic compounds				
Benzene	—	—	0.0070 µg/L	0.0070 µg/L
Ethylbenzene	—	—	0.011 µg/L	0.011 µg/L
Styrene	—	—	0.012 µg/L	0.012 µg/L
Tetrachloroethylene (perchloroethylene)	—	—	0.020 µg/L	0.020 µg/L
Toluene	—	—	0.011 µg/L	0.011 µg/L
Trichloroethylene	—	—	0.027 µg/L	0.027 µg/L

Chemical	Cycle 1	Cycle 2	Cycle 3	Cycle 4
Benzene metabolites				
S-Phenylmercapturic acid	—	0.08 µg/L	0.080 µg/L	0.080 µg/L
trans,*trans*-Muconic acid	—	0.8 µg/L	0.61 µg/L	0.61 µg/L
Trihalomethanes				
Bromodichloromethane	—	—	0.012 µg/L	0.012 µg/L
Dibromochloromethane	—	—	0.0070 µg/L	0.0070 µg/L
Tribromomethane (bromoform)	—	—	0.010 µg/L	0.010 µg/L
Trichloromethane (chloroform)	—	—	0.014 µg/L	0.014 µg/L
Xylenes				
m-Xylene and *p*-xylene	—	—	0.023 µg/L	0.023 µg/L
o-Xylene	—	—	0.0090 µg/L	0.0090 µg/L
Adjustment factor				
Creatinine	3 mg/dL	4 mg/dL	5.0 mg/dL	5.0 mg/dL

NA: Not available

a In the *Second Report on Human Biomonitoring of Environmental Chemicals in Canada*, all speciated arsenic was reported as µg of arsenic species per litre (e.g. µg arsenate/L). For this reason, the values presented in this report may differ from those in the Second Report.

b These chemicals were measured in cycle 3; however, the data are not yet available because of ongoing quality assurance confirmation of the biospecimen analysis.

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APPENDIX B

CONVERSION FACTORS

Units of measurement are important. Results are reported here using standard units; however, units can be converted using the conversion factors presented below for comparison of data with other data sets.

Definition of Units		
Unit	**Abbreviation**	**Value**
litre	L	—
decilitre	dL	10^{-1} L
millilitre	mL	10^{-3} L
microlitre	µL	10^{-6} L
gram	g	—
milligram	mg	10^{-3} g
microgram	µg	10^{-6} g
nanogram	ng	10^{-9} g
picogram	pg	10^{-12} g

Data can be converted from µg/L to µmol/L using the molecular weight (MW) of the chemical using the formula:

Y µmol/L = X µg/L × conversion factor (CF), where the CF is equivalent to 1/MW.

Conversion Factors

Chemical	MW (g/mol)	CF (µg/L → µmol/L)
Acrylamide		
Acrylamide haemoglobin adduct	—	NA
Glycidamide haemoglobin adduct	—	NA
Environmental phenols		
Bisphenol A	228.29	0.00438
Triclosan	289.54	0.00345
Metals and trace elements		
Cadmium	112.41	0.00896
Fluoride	19.00	0.05263
Lead	207.20	0.04826[a]
Mercury	200.59	0.00499
Methylmercury	215.63	0.00464
Arsenic (speciated)		
Arsenate	—	0.01335[b]
Arsenite	—	0.01335[b]
Arsenocholine	—	0.01335[b]
Arsenocholine and arsenobetaine	—	0.01335[b]
Dimethylarsinic acid	—	0.01335[b]
Monomethylarsonic acid	—	0.01335[b]
Nicotine metabolite		
Cotinine	176.22	0.00567
Organophosphate pesticide metabolites		
Chlorpyrifos metabolite		
3,5,6-Trichloro-2-pyridinol	198.43	0.00504
Malathion metabolite		
Malathion dicarboxylic acid	274.24	0.00365
Parabens		
Methyl paraben	152.15	0.00657
Ethyl paraben	166.18	0.00602
Propyl paraben	180.20	0.00555
Butyl paraben	194.23	0.00515
Polycyclic aromatic hydrocarbon metabolites		
Benzo[***a***]pyrene metabolite		
3-Hydroxybenzo[*a*]pyrene	268.31	0.00373
Chrysene metabolites		
2-Hydroxychrysene	244.29	0.00409
3-Hydroxychrysene	244.29	0.00409
4-Hydroxychrysene	244.29	0.00409
6-Hydroxychrysene	244.29	0.00409
Fluoranthene metabolite		
3-Hydroxyfluoranthene	218.25	0.00458

Chemical	MW (g/mol)	CF (µg/L → µmol/L)
Fluorene metabolites		
2-Hydroxyfluorene	182.22	0.00549
3-Hydroxyfluorene	182.22	0.00549
9-Hydroxyfluorene	182.22	0.00549
Naphthalene metabolites		
1-Hydroxynaphthalene	144.17	0.00694
2-Hydroxynaphthalene	144.17	0.00694
Phenanthrene metabolites		
1-Hydroxyphenanthrene	194.23	0.00515
2-Hydroxyphenanthrene	194.23	0.00515
3-Hydroxyphenanthrene	194.23	0.00515
4-Hydroxyphenanthrene	194.23	0.00515
9-Hydroxyphenanthrene	194.23	0.00515
Pyrene metabolite		
1-Hydroxypyrene	218.25	0.00458
Volatile organic compounds		
Benzene	78.11	0.01280
Ethylbenzene	106.17	0.00942
Styrene	104.15	0.00960
Tetrachloroethylene (perchloroethylene)	165.83	0.00603
Toluene	92.14	0.01085
Trichloroethylene	131.39	0.00761
Benzene metabolites		
trans,trans-Muconic acid	142.11	0.00704
S-Phenylmercapturic acid	239.29	0.00418
Trihalomethanes		
Bromodichloromethane	163.83	0.00610
Dibromochloromethane	208.28	0.00480
Tribromomethane (bromoform)	252.73	0.00396
Trichloromethane (chloroform)	119.38	0.00838
Xylenes		
m-Xylene and *p*-xylene	106.17	0.00942
o-Xylene	106.17	0.00942
Adjustment factor		
Creatinine	113.12	88.4[c]

NA: Not available

a For converting Pb from µg/dL to µmol/L

b For converting arsenic species from µg As/L to µmol As/L

c For converting creatinine from mg/dL to µmol/L

APPENDIX C

CREATININE

Creatinine — Geometric means and selected percentiles of urine concentrations (mg/dL) for the Canadian population aged 6–79 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 6–79 years						
1 (2007–2009)	5515	83 (78–89)	27 (23–30)	93 (86–99)	210 (200–220)	250 (240–260)
Males, 6–79 years						
1 (2007–2009)	2663	100 (97–110)	36 (28–43)	110 (100–110)	230 (220–240)	270 (250–280)
Females, 6–79 years						
1 (2007–2009)	2852	68 (62–74)	22 (18–25)	75 (66–84)	180 (160–190)	210 (200–230)
6–11 years						
1 (2007–2009)	1042	66 (60–72)	24 (18–29)	74 (67–81)	140 (130–150)	170 (160–180)
12–19 years						
1 (2007–2009)	992	120 (110–130)	39 (30–47)	130 (120–140)	250 (230–280)	300 (260–330)
20–39 years						
1 (2007–2009)	1172	90 (81–100)	29 (22–36)	99 (91–110)	230 (210–240)	280 (250–300)
40–59 years						
1 (2007–2009)	1221	78 (73–84)	24 (19–28)	86 (76–96)	210 (190–230)	240 (230–250)
60–79 years						
1 (2007–2009)	1088	72 (68–75)	26 (22–31)	81 (77–85)	150 (140–160)	190 (170–220)

Creatinine — Geometric means and selected percentiles of urine concentrations (mg/dL) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years						
2 (2009–2011)	6299	100 (100–110)	35 (33–38)	110 (110–120)	240 (230–260)	280 (270–300)
Males, 3–79 years						
2 (2009–2011)	3031	120 (120–130)	47 (42–53)	130 (120–150)	260 (240–280)	310 (280–340)
Females, 3–79 years						
2 (2009–2011)	3268	89 (85–94)	30 (27–32)	100 (96–100)	200 (180–230)	250 (240–270)
3–5 years						
2 (2009–2011)	572	59 (55–63)	26 (24–29)	61 (55–67)	110 (110–120)	140 (110–160)
6–11 years						
2 (2009–2011)	1059	88 (83–94)	37 (33–42)	98 (94–100)	170 (160–170)	190 (170–210)
12–19 years						
2 (2009–2011)	1042	130 (120–150)	52 (36–68)	150 (140–160)	270 (260–280)	300 (270–340)
20–39 years						
2 (2009–2011)	1322	120 (110–130)	37 (25–48)	140 (130–160)	260 (250–280)	330 (270–380)
40–59 years						
2 (2009–2011)	1223	100 (96–110)	33 (27–40)	110 (100–120)	240 (220–260)	280 (260–310)
60–79 years						
2 (2009–2011)	1081	85 (80–89)	32 (26–37)	96 (90–100)	180 (170–200)	230 (210–260)

Creatinine — Geometric means and selected percentiles of urine concentrations (mg/dL) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years						
3 (2012–2013)	5704	97 (93–100)	33 (29–37)	100 (100–110)	240 (220–250)	280 (250–300)
Males, 3–79 years						
3 (2012–2013)	2847	110 (110–120)	40 (35–46)	120 (110–130)	260 (230–280)	300 (260–340)
Females, 3–79 years						
3 (2012–2013)	2857	83 (76–90)	26 (21–30)	93 (81–110)	210 (190–240)	250 (220–270)
3–5 years						
3 (2012–2013)	521	51 (45–58)	19 (14–24)	58 (51–65)	110 (99–110)	120 (110–120)
6–11 years						
3 (2012–2013)	1013	84 (77–92)	35 (28–42)	93 (82–100)	160 (150–180)	200 (170–230)
12–19 years						
3 (2012–2013)	998	130 (120–150)	52 (37–66)	150 (140–160)	280 (260–300)	320 (290–360)
20–39 years						
3 (2012–2013)	1048	110 (98–120)	36 (26–45)	110 (97–130)	270 (220–320)	330 (290–380)
40–59 years						
3 (2012–2013)	1080	95 (86–110)	34 (24–44)	110 (98–110)	220 (200–250)	250 (230–280)
60–79 years						
3 (2012–2013)	1044	84 (76–91)	26 (19–32)	96 (89–100)	190 (170–210)	230 (210–240)

Creatinine — Geometric means and selected percentiles of urine concentrations (mg/dL) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 4 (2014–2015).

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years						
4 (2014–2015)	5603	110 (110–120)	40 (35–46)	110 (110–120)	250 (240–260)	290 (270–310)
Males, 3–79 years						
4 (2014–2015)	2815	130 (120–140)	50 (40–60)	140 (120–150)	270 (250–290)	320 (310–330)
Females, 3–79 years						
4 (2014–2015)	2788	98 (94–100)	35 (30–39)	100 (98–100)	230 (210–240)	260 (250–270)
3–5 years						
4 (2014–2015)	513	58 (51–65)	22 (15–29)	66 (58–73)	110 (99–120)	130 (120–150)
6–11 years						
4 (2014–2015)	1008	90 (84–98)	35 (24–45)	99 (94–100)	170 (150–190)	210 (170–250)
12–19 years						
4 (2014–2015)	991	140 (130–150)	54 (46–61)	150 (140–170)	280 (270–300)	350 (320–370)
20–39 years						
4 (2014–2015)	1059	130 (120–140)	41 (36–47)	140 (130–160)	290 (260–320)	350 (320–390)
40–59 years						
4 (2014–2015)	1037	110 (100–120)	41 (29–54)	110 (110–120)	240 (220–260)	270 (260–280)
60–79 years						
4 (2014–2015)	995	100 (97–110)	37 (32–42)	100 (100–110)	200 (180–220)	240 (210–270)