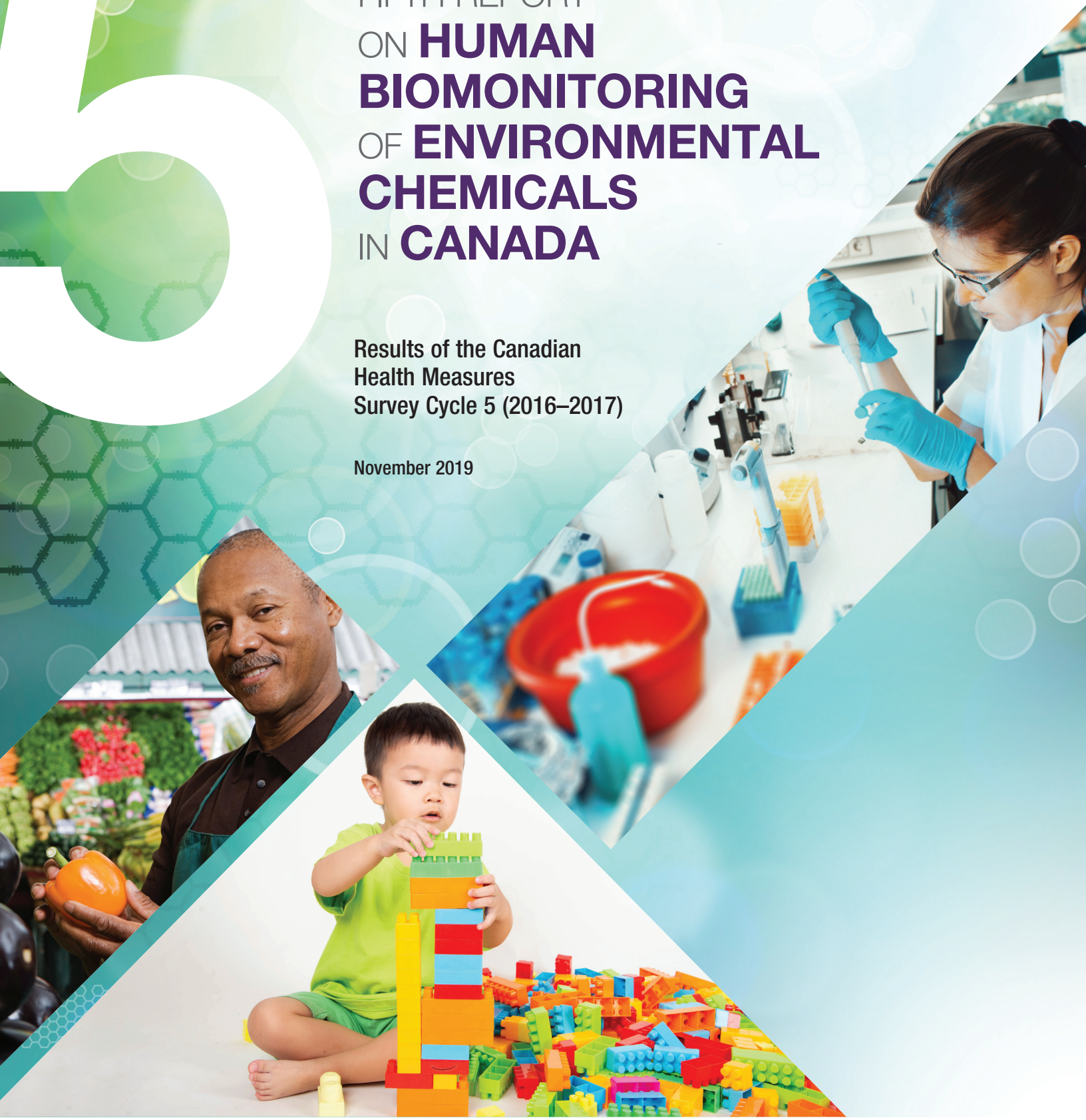
FIFTH REPORT ON HUMAN BIOMONITORING OF ENVIRONMENTAL CHEMICALS IN CANADA

Results of the Canadian Health Measures Survey Cycle 5 (2016–2017)

November 2019



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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 an ongoing national direct health measures survey called the Canadian Health Measures Survey (CHMS). 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 the blood and urine of survey participants. Hair measures were also included for a subsample of participants in cycle 5. 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, or consumer products.

Data from previous cycles have been published in four Health Canada reports, the most recent of which, the *Fourth Report on Human Biomonitoring of Environmental Chemicals in Canada,* was published in August 2017 (Health Canada, 2017). During the first four cycles, data were collected for 164 environmental chemicals in individual samples.

Data for cycle 5 were collected between January 2016 and December 2017 from approximately 5,800 Canadians aged 3–79 years at 16 sites across Canada. Cycle 5 included 99 environmental chemicals, 64 of which were also measured in previous cycles.

A summary of the environmental chemicals measured in the blood and/or urine of individual respondents in the first five cycles of the CHMS is presented in Table 1.1.

Table 1.1

Summary of chemical groups measured in blood and/or urine of individual respondents in the Canadian Health Measures Survey between 2007 and 2017

Chemical group	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Flame retardants	■				
Organochlorines	■				
Polychlorinated biphenyls	■				
Chlorophenols	■	■			
Per- and polyfluoroalkyl substances	■	■			■
Plasticizers	■	■			■
Polycyclic aromatic hydrocarbons		■	■	■	
Acrylamide			■	■	■
Volatile organic compounds			■	■	■
Metals and trace elements	■	■	■	■	■
Self-care and consumer product chemicals	■	■	■	■	■
Nicotine	■	■	■	■	■
Pesticides	■	■	■	■	■

Collection for cycle 6 of the CHMS began in January 2018 and will be completed in late 2019. Planning for future cycles is under way.

This report describes the general CHMS survey design and implementation, 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 and health effects, Canadian 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 measured in hair can be found in Appendix D. For chemicals only measured in previous cycles, data can be found in previous reports (Health Canada, 2010; Health Canada, 2013; Health Canada, 2015; Health Canada, 2017). Downloadable tables are available in XLS format through the Government of Canada's Open data 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 help them assess exposure to environmental chemicals and develop policies to reduce Canadians' exposure to toxic chemicals for the protection of their health.

Some specific uses of the CHMS biomonitoring data include:

- establishing baseline concentrations of chemicals in Canadians that allow for comparisons with subpopulations in Canada and with populations in other countries
- establishing baseline concentrations of chemicals to track trends in Canadians over time
- providing information for setting priorities and taking action to reduce Canadians' exposure to environmental chemicals and protect their health
- assessing the effectiveness of health and environmental risk management actions intended to reduce exposures and health risks from specific chemicals
- supporting future research on the potential links between exposure to certain chemicals and specific health effects
- contributing 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 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 5 have been published (Beck et al., 2018; Statistics Canada, 2019).

3.1 TARGET POPULATION

Cycle 5 of the CHMS targets the population aged 3–79 years living in one of the 10 provinces. The following groups were excluded from the survey: persons living in the three territories; persons living on reserves and other Indigenous 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 3% 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 (AFN) 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 Nations communities across Canada. The complete report has been published by the AFN.

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 Indigenous organizations, was established in 1991 to respond to concerns about human exposure to contaminants in the traditional diets of Northern Indigenous peoples. The NCP provides funding for numerous individual studies undertaken in various regions of the North, including the Northwest Territories, Nunavut, and Nunavik (Québec'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 5 of the CHMS required a minimum sample of at least 5,700 participants over a two-year period. The participants were distributed among 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 3–5-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 multistage 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 it within a reasonable period of time. For cycle 5, Census geography was used to create 379 collection sites across the country. A geographic area with a population of at least 10,000 and a maximum participant travel distance of 50 km in urban areas and 75 km in rural areas was required for the location of collection sites. Areas not meeting these criteria were excluded.

Including a larger number of collection sites with few respondents 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 (the Atlantic provinces, Québec, Ontario, the Prairies, and British Columbia) and were allocated to these regions in proportion to population size. Although not every province in Canada had a collection site, the CHMS sites were chosen to represent the Canadian population in all 10 provinces, including larger and smaller population densities. The collection sites selected for cycle 5 of the CHMS are listed in Table 3.3.1.1.

Table 3.3.1.1
Canadian Health Measures Survey cycle 5 (2016–2017) collection sites

Atlantic	Quebec	Ontario	Prairies	British Columbia
• Montague, P.E.I. • Saint John, N.B.	• Montréal Centre • Rimouski • Sherbrooke • West Longueuil/ Boucherville	• Brampton • Cambridge • Petawawa/Pembroke • Peterborough • Pickering/Ajax • Toronto West	• Calgary South, Alta. • Humboldt, Sask.	• Coquitlam • Trail

3.3.2 Dwelling and Participant Sampling

Within each site, the most recent version of the Household Survey Frame, as well as more current information from other administrative sources, was used to select dwellings and identify the birth dates of household members. Dwellings with known household composition at the time of the sample selection were stratified by age of household residents at the time of the survey, with the six age-group strata corresponding to the CHMS cycle 5 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

A series of formal and informal consultations were carried out to determine the set of environmental chemicals measured in cycle 5 of the CHMS. The consultations included stakeholders with expertise or interest in human biomonitoring of environmental chemicals. Key participants were 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.

The following criteria were used as general guides 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
- known data gaps
- commitments under national and international treaties, conventions, and agreements
- current and anticipated health policy development and implementation
- volume of biospecimens available from survey

A full list of the chemicals measured in the blood and/or urine of individual respondents in CHMS cycle 5 is presented in Table 3.4.1. Also included in this report are data for four organophosphate pesticide-related chemicals, namely 3,5,6-trichloro-2-pyridinol, malathion dicarboxylic acid, acephate, and methamidophos. While these chemicals were not measured in cycle 5, newly available data from cycle 3 are presented for them. Newly available data from cycle 3 for parabens are also presented. These data were not available for inclusion in previous reports due to delays in laboratory analyses.

Metals and trace elements were also measured in hair from a subsample of participants in cycle 5. Refer to Appendix D for a complete list of hair analytes.

Table 3.4.1

Chemicals measured in blood and/or urine of individual respondents in the Canadian Health Measures Survey cycle 5, 2016–2017[a] (includes new chemicals and chemicals carried forward from previous cycles)

Chemical group	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Metals and trace elements[b]					
Lead	■	■	■	■	■
Boron					■
Cadmium	■	■	■	■	■
Chromium (VI)[c]					■
Selenium	■	■			■
Arsenic (speciated)					
Inorganic-related arsenic species		■	■	■	■
Arsenite		■	■	■	■
Arsenate		■	■	■	■
Monomethylarsonic acid (MMA)		■	■	■	■
Dimethylarsinic acid (DMA)		■	■	■	■
Arsenocholine and arsenobetaine		■	■	■	■
Mercury					
Mercury (total)	■	■	■	■	■
Methylmercury			■	■	■
Mercury (inorganic)	■		■	■	■
Self-care and consumer product chemicals					
Bisphenol A (BPA)	■	■	■	■	■
Parabens					
Methyl paraben			■[d]	■	■
Ethyl paraben			■[d]	■	■
Propyl paraben			■[d]	■	■
Butyl paraben			■[d]	■	■
Nicotine					
Cotinine	■	■	■	■	■
Acrylamide					
Acrylamide haemoglobin adduct			■	■	■
Glycidamide haemoglobin adduct			■	■	■
Per- and polyfluoroalkyl substances					
Perfluorobutanoic acid (PFBA)		■			■
Perfluorobutane sulfonate (PFBS)		■			■
Perfluorohexanoic acid (PFHxA)		■			■
Perfluorohexane sulfonate (PFHxS)	■	■			■
Perfluorooctanoic acid (PFOA)	■	■			■
Perfluorooctane sulfonate (PFOS)	■	■			■
Perfluorononanoic acid (PFNA)		■			■
Perfluorodecanoic acid (PFDA)		■			■
Perfluoroundecanoic acid (PFUnDA)		■			■

Chemical group	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Pesticides					
Organophosphate pesticides					
Dimethylphosphate (DMP)	■	■			■
Dimethylthiophosphate (DMTP)	■	■			■
Dimethyldithiophosphate (DMDTP)	■	■			■
Diethylphosphate (DEP)	■	■			■
Diethylthiophosphate (DETP)	■	■			■
Diethyldithiophosphate (DEDTP)	■	■			■
3,5,6-Trichloro-2-pyridinol (TCPy)			■ [d]	■	
Malathion dicarboxylic acid (DCA)			■ [d]	■	
Acephate			■ [d]		
Methamidophos			■ [d]		
Ethylene bisdithiocarbamates					
Ethylene thiourea (ETU)					■
ortho-Phenylphenol (OPP)					
OPP-glucuronide					■
OPP-sulfate					■
Pyrethroids					
3-Phenoxybenzoic acid (3-PBA)	■	■			■
4-Fluoro-3-phenoxybenzoic acid (4-F-3-PBA)	■	■			■
cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA)	■	■			■
cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA)	■	■			■
trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA)	■	■			■
Plasticizers					
Monomethyl phthalate (MMP)	■	■			■
Monoethyl phthalate (MEP)	■	■			■
Mono(3-carboxypropyl) phthalate (MCPP)	■	■			■
Mono-*n*-butyl phthalate (M*n*BP)	■	■			■
Monoisobutyl phthalate (MiBP)		■			■
Mono-3-hydroxy-*n*-butyl phthalate (3OH-MBP)					■
Monocyclohexyl phthalate (MCHP)	■	■			■
Monobenzyl phthalate (MBzP)	■	■			■
Mono[2-(carboxymethyl)hexyl] phthalate (MCMHP)					■
Mono(2-ethylhexyl) phthalate (MEHP)	■	■			■
Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP)					■
Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP)	■	■			■
Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)	■	■			■
Mono-carboxy-*n*-heptyl phthalate (MCHpP)					■
Mono-*n*-octyl phthalate (MOP)	■	■			■

Chemical group	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Mono(carboxyisooctyl) phthalate (MCiOP)					■
Monoisononyl phthalate (MiNP)	■	■			■
Monocarboxyisononyl phthalate (MCiNP)					■
Monooxoisononyl phthalate (MOiNP)					■
Monohydroxyisononyl phthalate (MHiNP)					■
Monoisodecyl phthalate (MiDP)					■
Monooxoisodecyl phthalate (MOiDP)					■
Monohydroxyisodecyl phthalate (MHiDP)					■
Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH)					
trans-Cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH)					■
Cyclohexane-1,2-dicarboxylic mono oxo-isononyl ester (oxo-MINCH)					■
Cyclohexane-1,2-dicarboxylic mono hydroxy-isononyl ester (OH-MINCH)					■
cis-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH)					■
trans-Cyclohexane-1,2-diarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH)					■
Cyclohexane-1,2-dicarboxylic acid (CHDA)					■
2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB)					
2,2,4-Trimethyl-1,3-pentanediol (TMPD)					■
2,2,4-Trimethyl-3-hydroxy valeric acid (HTMV)					■
Tri-(2-ethylhexyl) trimellitate (TEHT)					
1-Mono(2-ethylhexyl)trimellitate (1-MEHTM)					■
2-Mono(2-ethylhexyl)trimellitate (2-MEHTM)					■
4-Mono(2-ethylhexyl)trimellitate (4-MEHTM)					■
Volatile organic compounds					
Benzene			■	■	■
Carbon tetrachloride					■
1,4-Dichlorobenzene					■
2,5-Dimethylfuran					■
Ethylbenzene			■	■	■
Isopropylbenzene					■
Methyl isobutyl ketone					■
Nitrobenzene					■
Styrene			■	■	■
1,1,1,2-Tetrachloroethane					■
Tetrachloroethylene			■	■	■
Tetrahydrofuran					■
Toluene			■	■	■
Trichloroethylene			■	■	■

Chemical group	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Trihalomethanes					
Bromodichloromethane					
Dibromochloromethane					
Tribromomethane (bromoform)					
Trichloromethane (chloroform)					
Xylenes					
m-Xylene and *p*-xylene					
o-Xylene					

a 3,5,6-Trichloro-2-pyridinol, malathion dicarboxylic acid, acephate, and methamidophos were not measured in cycle 5. However, new data for these chemicals from cycle 3 are presented in this report.

b Metals and trace elements were also measured in hair from a subsample of participants in cycle 5. Refer to Appendix D for a complete list of hair analytes.

c Chromium (VI) was measured indirectly as total chromium in red blood cells.

d New data from cycle 3 (2012–2013) are included in this report, as they were not available at the time of publication of the previous reports.

Owing to the high cost of laboratory analyses, some environmental chemicals were not measured for all CHMS participants in cycle 5. 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 selenium in blood, and cotinine in urine, were measured in all participants; methylmercury was measured in participants aged 3–19 years; volatile organic compounds were measured in 2,500 participants aged 12–79 years; and metals and trace elements in hair were measured in 2,000 participants aged 20–59 years. Further details on the subsampling for environmental chemicals are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 5* (Statistics Canada, 2019) and in *Sampling documentation for cycle 5 of the Canadian Health Measures Survey* (Beck et al., 2018). For the chemicals measured in cycle 3 for which new data are presented in this report, further details on the subsampling for environmental chemicals are available in the *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 3* (Statistics Canada, 2015) and in *Sampling documentation for cycle 3 of the Canadian Health Measures Survey* (Labrecque and Quigley, 2014).

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
Metals and trace elements	Blood	5,700	■	■	■	■	■	■
Metals and trace elements	Hair	2,000	—	—	—	■	■	—
Arsenic	Urine	2,500	■	■	■	■	■	■
Cadmium and boron	Urine	2,500	■	■	■	■	■	■
Chromium (VI)[a]	Red blood cells	2,500	■	■	■	■	■	■
Methylmercury and inorganic mercury	Blood	2,500	■	■	■	—	—	—
Bisphenol A (BPA)	Urine	2,500	■	■	■	■	■	■
Parabens	Urine	2,500	■	■	■	■	■	■
Cotinine	Urine	5,700	■	■	■	■	■	■
Acrylamide	Blood	2,500	■	■	■	■	■	■
Per- and polyfluoroalkyl substances	Plasma	2,500	■	■	■	■	■	■
Organophosphate pesticides	Urine	2,500	■	■	■	■	■	■
Pyrethroids	Urine	2,500	■	■	■	■	■	■
Ethylene thiourea (ETU)	Urine	2,500	■	■	■	■	■	■
ortho-Phenylphenol (OPP)	Urine	2,500	■	■	■	■	■	■
Phthalates	Urine	2,500	■	■	■	■	■	■
Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) and tri-(2-ethylhexyl) trimellitate (TEHT)	Urine	2,500	■	■	■	■	■	■
2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB)	Urine	2,500	■	■	■	■	■	■
Volatile organic compounds	Blood	2,500	—	—	■	■	■	■

a Chromium (VI) was measured indirectly as total chromium in red blood cells.

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 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 children aged 6–13 years of age 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 cadmium, lead and mercury were actively reported to participants from all sites. 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 5 took place over a period of two years from January 2016 to December 2017. 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 advance letters and brochures to households that were selected as outlined in Section 3.3.2, Dwelling and Participant Sampling. The mailing informed potential participants that they would be contacted for the survey's data collection.

Data were collected from each consenting survey participant through a household personal interview, using a computer-assisted method, and through 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, 2019).

Participants were first administered a household questionnaire in their homes. Using a computer application, the interviewer randomly selected one or two participants and conducted separate 45- to 60-minute health interviews (Statistics Canada, 2019). The interviews collected demographic and socio-economic data, including information about lifestyle, medical history, current health status, smoking status and electronic cigarette use, and neighbourhood environment. Participants were also informed that Statistics Canada would link the information collected during the interview to information from the tax data of all members of their household. Within approximately two weeks of 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) area, and examination rooms; and the third trailer contained additional examination rooms. The MEC operated for five to six weeks at each site to complete approximately 350 visits (Statistics Canada, 2019). MEC appointments averaged 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 CHMS MEC staff members to perform some of the physical measures and the biospecimen collection portion of the survey; there were seven home visits in total in cycle 5 (Statistics Canada, 2019).

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 first-catch urine. (To note, as an exception, mid-stream urine was collected in cycle 1.) Guidelines were provided to participants asking them to abstain from urinating two hours prior to their MEC visit. Samples were collected in 120 mL urine specimen containers.

Trained health professionals collected hair samples and took physical health measurements, such as for height, weight, blood pressure, and physical fitness. A series of screening questions were administered to participants to determine their eligibility for the various tests, including phlebotomy and hair sampling, based on pre-existing exclusion criteria (Statistics Canada, 2019). A minimum natural hair length of 2 cm was required; approximately 100 strands of full-length hair were collected from the back of the scalp. Blood specimens were drawn by a certified phlebotomist; the maximum amount depended upon the age of the participant and consent to storage. The approximate volume drawn with and without consent to storage from participants aged 3–5 years was 25.5 mL and 22.5 mL; 6–11 years, 40.0 mL and 34.0 mL; 12–13 years, 59.0 mL and 39.0 mL; 14–19 years, 77.0 mL and 49.0 mL; and 20–79 years, 83.0 mL and 53.0 mL.

Standardized operating procedures were developed for the collection of blood, urine and hair specimens, processing and aliquoting procedures, and the shipping of biospecimens to ensure adequate data quality and standardize data collection. All blood and urine specimens collected in the MEC were processed and aliquoted in the MEC. Blood and urine specimens were stored in the MEC in either the refrigerator or the freezer, depending on the test, while hair specimens were stored at room temperature. All specimens were stored as soon as processing was complete to maintain sample integrity. A four-hour time limit from the point of collection was set for blood samples to be processed and stored; however, for most samples, this was completed within two hours. Given specific pre-analytical requirements for chromium (VI) in red blood cells, a time limit of three hours was set for processing and storing the samples. Once a week, the specimens were shipped on dry ice or in monitored refrigerated conditions to the reference laboratory for analyses. 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 matrix, collection tubes, and aliquot volumes are presented in Table 4.1 in order of testing priority.

Table 4.1

Urine and blood collection procedure for the environmental chemicals

Measure	Matrix	Collection Tube (size and type[a])	Optimal Volume[b]
Volatile organic compounds (VOCs)	Whole blood	5 mL baked grey top	5 mL
Per- and polyfluoroalkyl substances	Plasma	4.0, 6.0, or 10 mL[c] lavender EDTA	1.3 mL
Chromium (VI)	Red blood cells	4.0, 6.0, or 10 mL[c] lavender EDTA	1.2 mL
Cadmium and boron	Urine	120 mL urine specimen container	1.8 mL
Specific gravity			0.3 mL
Arsenic (speciated)			1.0 mL
Acrylamide	Whole blood	4.0, 6.0, or 10 mL[c] lavender EDTA	1.0 or 1.5 mL[d]
Metals and trace elements			1.0 or 1.8 mL[d]
Ethylene thiourea (ETU)	Urine	120 mL urine specimen container	1.0 mL
ortho-Phenylphenol (OPP)			1.8 mL
Creatinine			0.5 mL
2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB)			1.8 mL
Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) and tri-(2-ethylhexyl) trimellitate (TEHT)			2.0 mL
Cotinine			1.8 mL
Phthalates			2.5 mL
Parabens			1.0 mL
Pyrethroids			3.0 mL
Bisphenol A (BPA) and organophosphate pesticides			1.3 mL
Metals and trace elements	Hair	Ziploc bag	3 cm in length

EDTA: ethylenediaminetetraacetic acid

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

b Optimum sample volume sent to the reference laboratory.

c Collection tube size was dependent upon respondent age and whether or not they consented to storage of samples in the biobank.

d Collection volume was dependent upon respondent age.

To maximize the reliability and validity of the data and 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 evaluated for each site and consisted of field blanks, blind replicates, and blind controls. Three field blanks (deionized water) were analyzed per site for all analytes except acrylamide and VOCs in blood, and creatinine and cotinine in urine. Three pairs of blind replicates were assessed per site for all analytes. Approximately six blind control samples were evaluated per site for all analytes except chromium (VI) in red blood cells.

Field blanks were sent to the reference laboratories at the start of each site and results were expeditiously returned directly to the laboratory coordinators at Statistics Canada. Blind replicate and blind control samples were sent to the reference laboratories with regular specimen shipments. Quality control sample results were sent to Statistics Canada's CHMS headquarters, along with all other respondent results. If required, feedback was provided quickly to the relevant reference laboratory for review and remedial action.

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 5* (Statistics Canada, 2019).

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LABORATORY ANALYSES 5

Laboratory analyses of environmental chemicals and creatinine were performed at analytical laboratories within Health Canada and at 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 the precision of measurements were evaluated through rigorous method validation programs at each laboratory.

To ensure ongoing accuracy and precision of results, several quality control measures were employed as part of the Canadian Health Measures Survey (CHMS). Field blanks were used to confirm that samples had not been contaminated during collection, processing, storage, or shipping. Blind replicate samples were used as indicators of the precision of sample analysis, while blind control samples were used as indicators of the accuracy of sample analysis. Laboratories also participated in external quality control programs and interlaboratory comparison studies, as outlined in the sections below. The methods used in the analyses of the environmental chemicals and creatinine are described below.

5.1 METALS AND TRACE ELEMENTS

5.1.1 Blood Analyses

5.1.1.1 Lead, Cadmium, Selenium, and Total Mercury

Lead, cadmium, selenium, and mercury analyses in whole blood were performed at the Centre de toxicologie du Québec (CTQ), INSPQ (INSPQ, 2018p). Briefly, whole blood samples were diluted in a basic solution containing octylphenol ethoxylate and ammonium hydroxide and analyzed for lead, cadmium, selenium, and mercury using inductively coupled plasma mass spectrometry (ICP-MS). The ICP-MS method employed a Perkin Elmer Sciex Elan DRC II with an ESI SC-4 autosampler and an Elan workstation version 3.0. Matrix-matched calibration was performed using blood from non-exposed individuals. Internal quality control was ensured by analyzing two different reference materials from the Québec Multielement External Quality Assessment Scheme (QMEQAS) in each analysis sequence. The external quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the internal CTQ Programme de comparaisons interlaboratoires pour les métaux en milieu biologique (PCI); QMEQAS; the German External Quality Assessment Scheme (G-EQUAS); the U.S. Centers for Disease Control and Prevention's Lead and Multielement Proficiency Program (LAMP); and the New York State Department of Health's Proficiency Program for Trace Elements in Whole Blood.

5.1.1.2 Chromium (VI)

Chromium (VI) analyses in red blood cells were performed at the CTQ, INSPQ(INSPQ, 2018f). The analysis was an indirect measurement of chromium (VI) and was based on the fact that chromium (VI) is the only form of inorganic chromium to penetrate cells. As such, chromium measured in red blood cells is attributed specifically to chromium (VI) exposure (Devoy et al., 2016).

Briefly, red blood cells were purified shortly after collection via a saline wash. Purified red blood cells were digested with concentrated nitric acid and hydrogen peroxide and diluted in water to reduce viscosity. The samples were then analyzed using inductively coupled plasma tandem mass spectrometry (ICP-MS-MS). The ICP-MS-MS method employed an Agilent Technologies 8800 ICP-QQQ with a CETAC ASX-500 autosampler and a MassHunter 4.2 workstation version C.01.02. Terbium was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium and high) in each analysis sequence.

5.1.1.3 Methylmercury and Inorganic Mercury

Methylmercury and inorganic mercury analyses in whole blood were performed at the CTQ, INSPQ (INSPQ, 2018k). Briefly, whole blood samples were digested with tetramethylammonium hydroxide and mercury species were derivatized into volatile compounds by sodium tetra-n-propylborate. Mercury was extracted in the gas phase by solid-phase microextraction with polydimethylsiloxane/divinylbenzene fibre. Ultimately, mercury species were analyzed using isotopic dilution in tandem gas chromatography and inductively coupled plasma mass spectrometry (ID-GC-ICP-MS). The ID-GC-ICP-MS method employed a Perkin Elmer Clarus 580 gas chromatograph with a Zebron ZB-5 column (Phenomenex), a CTC Analytics CombiPAL autosampler, and an Empower chromatograph workstation version 3 alongside a Perkin Elmer NexION 350s ICP-MS with a Syngistix workstation version 1.1. Quantification was obtained by isotope dilution calculation. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.1.2 Urine Analyses

5.1.2.1 Arsenic

Speciated arsenic analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018j). The analyses measured arsenite (As3+), arsenate (As5+), monomethylarsonic acid, dimethylarsinic acid, and the sum of arsenobetaine and arsenocholine. Briefly, urine samples were diluted tenfold in an ammonium carbonate solution (dilution solvent) compatible with the initial eluent, then analyzed on the high-performance liquid chromatography system, used in high pressure mode only, combined with inductively coupled plasma mass spectrometry (HPLC-ICP-MS). The HPLC-ICP-MS method employed a Waters ACQUITY HPLC with an Empower chromatograph workstation version 3 and a Perkin Elmer NexION 350s ICP-MS with a Syngistix workstation version 1.1. Methylseleno-L-cysteine was used as an internal standard. Internal quality control was ensured by analyzing three non-certified, in-house reference materials in each analysis sequence. External quality and the accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the G-EQUAS.

5.1.2.2 Boron

Boron analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018e). Briefly, urine samples were diluted in 0.5% nitric acid and analyzed for boron using ICP-MS-MS. The ICP-MS-MS method employed an Agilent Technologies 8800 ICP-QQQ with a CETAC ASX-500 autosampler and a MassHunter 4.2 workstation version C.01.02. Beryllium was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.1.2.3 Cadmium

Cadmium analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018q). Briefly, urine samples were diluted in 0.5% nitric acid and analyzed for cadmium using ICP-MS. The ICP-MS method employed a Perkin Elmer Sciex Elan DRC II with an ESI SC-4 autosampler and an Elan workstation version 3.0. Matrix-matched calibration was performed using urine from non-exposed individuals. Correction of molybdenum-based interference on cadmium concentrations was performed mathematically using equations derived following the addition of molybdenum to urine samples. Internal quality control was ensured by analyzing three different

reference materials from the QMEQAS in each analysis sequence. The external quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the internal CTQ PCI, QMEQAS, the G-EQUAS, and the New York State Department of Health's Proficiency Program for Trace Elements in Urine.

5.2 SELF-CARE AND CONSUMER PRODUCT CHEMICALS

5.2.1 Bisphenol A

Bisphenol A analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018d). Briefly, urine samples were hydrolyzed using ß-glucuronidase and derivatized with pentafluorobenzyl bromide. The derivatized products were then extracted with a mixture of dichloromethane and hexane. Extracts were then evaporated and redissolved, and the sum of free and conjugated forms of bisphenol A was analyzed by gas chromatography coupled with tandem mass spectrometry (GC-MS-MS). The GC-MS-MS method employed an Agilent 6890 gas chromatograph with an Agilent 7683 automatic injector and sampler coupled to a Waters Quattro Micro-GC tandem quadrupole mass spectrometer and a workstation equipped with Waters MassLynx software version 4.1; measurements were carried out in multiple reaction monitoring (MRM) mode with a source in negative chemical ionization mode. Carbon-13–labelled bisphenol A analogues were used as internal standards. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence. The external quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the G-EQUAS.

5.2.2 Parabens

Paraben analyses in urine were performed at the Food Program Western Region Laboratory, Health Canada, British Columbia, Canada (Health Canada, 2017) using a method adapted from the U.S. Centers for Disease Control and Prevention (CDC, 2011). In these analyses, free and conjugated forms of butyl paraben, ethyl paraben, methyl paraben, and propyl paraben were measured together. Briefly, urine samples were hydrolyzed using ß-glucuronidase/sulfatase (Helix pomatia type H1). After enzymatic hydrolysis, samples were acidified with formic acid and preconcentrated using solid-phase extraction (Waters Oasis HLB SPE tubes). The sum of free and conjugated parabens was detected and quantified using ultra-performance liquid chromatography coupled with tandem mass spectrometry (UPLC-MS-MS). The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Quattro Premier XE tandem mass spectrometer; data were collected as MRM data in electrospray ionization negative mode. Deuterated parabens (D_4-methyl paraben, D_4-ethyl paraben, D_4-propyl paraben, and D_4-butyl paraben) were used as the internal standards. Internal quality control was ensured by analyzing two in-house quality control pools (low and high) in each batch of analyses.

5.3 COTININE

Free cotinine analyses in urine were performed at the CTQ, INSPQ. One method was used for participants aged 3–11 years (INSPQ, 2018a) and another for participants aged 12–79 years (INSPQ, 2018c). Data from the two methods were combined and are presented separately for smokers aged 12–79 years and non-smokers aged 3–79 years. Briefly, for both methods, free cotinine was extracted from urine samples by solid-phase extraction via mixed cation-exchange and reverse phase support on an automated Perkin Elmer JANUS automated liquid-handling workstation. The extracts were redissolved in the mobile phase and analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S or Quattro Premier XE tandem mass spectrometer and a workstation equipped with Waters MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray source positive mode. For participants aged 12–79 years, within each analysis sequence, samples from non-smokers were analyzed first, followed by samples from smokers, to avoid contamination between samples. Deuterated cotinine was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence. The external quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the G-EQUAS.

5.4 ACRYLAMIDE

Acrylamide and glycidamide hemoglobin adduct analyses in whole blood were performed at the Ontario Food Laboratory, Health Canada, Ontario, Canada (Health Canada, 2014). Briefly, whole blood samples were reacted with modified Edman reagent (pentafluorophenyl isothiocyanate) and purified using solid-phase extraction on a column of ISOLUTE HM-N sorbent with a diisopropyl ether/ethyl acetate/toluene (50/40/10 v/v/v) eluent. The extract was evaporated, reconstituted, and analyzed using UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY ultra-performance liquid chromatograph system coupled to a Waters Quattro Premier tandem mass spectrometer and a workstation equipped with MassLynx software; measurements were carried out in MRM mode with an Atmospheric Pressure Chemical Ionization (APCI) positive ion mode. Carbon-13 labelled acrylamide octapeptide was used as an internal standard. Internal quality control was ensured by analyzing two different in-house reference materials (low and high) in each analysis sequence. Hemoglobin was also measured in whole blood using a commercial HemoCue assay kit; the hemoglobin value was used to adjust the acrylamide and glycidamide hemoglobin adduct results.

5.5 PERFLUOROALKYL AND POLYFLUOROALKYL SUBSTANCES

Perfluoroalkyl substance analyses in plasma were performed at the CTQ, INSPQ (INSPQ, 2018i). The analyses measured perfluorobutanoic acid, perfluorobutane sulfonate, perfluorohexanoic acid (PFHxA), perfluorohexane sulfonate (PFHxS), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), and perfluoroundecanoic acid (PFUnDA). Briefly, plasma samples were extracted by solid-phase extraction with a WAX support on an automated Perkin Elmer JANUS automated liquid-handling workstation. The extracts were redissolved in the mobile phase and analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization negative mode. Internal quality control was ensured by analyzing four different reference materials — three in-house (low, medium, and high) and one commercial — in each analysis sequence. External quality and accuracy of the analytical method was assessed by participating in interlaboratory comparison programs, including the internal CTQ Arctic Monitoring and Assessment Program (AMAP) ring test interlaboratory comparison program for persistent organic pollutants in human serum (PFHxA, PFHxS, PFNA, PFOA, PFOS, PFDA, PFUnDA) and the G-EQUAS for PFOS and PFOA.

5.6 PESTICIDES

5.6.1 Ethylene Bisdithiocarbamates

Ethylene thiourea (ETU) analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018g). Briefly, urine samples were hydrolyzed and derivatized with 2,3,4,5,6-pentafluorobenzyl bromide. The derivatized products were then extracted with hexane. The extracts were analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with electrospray ionization positive mode. Deuterated ETU was used as an internal standard. Internal quality control was ensured by analyzing four different reference materials — three in-house (low, medium, and high) and one commercial — in each analysis sequence.

5.6.2 *ortho*-Phenylphenol

ortho-Phenylphenol analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018r). In these analyses, glucuronide- and sulfate-conjugated forms of *ortho*-phenylphenol were measured. Briefly, urine samples were extracted on an ion-exchange cartridge, eluted, and evaporated to dryness. The extracts were redissolved in a mixture of methanol and demineralized water (25:75), then analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization negative mode.

Carbon-13 labelled *ortho*-phenylphenol was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.6.3 Organophosphate Pesticides

5.6.3.1 Dialkyl Phosphates

Dialkyl phosphate metabolite analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018h). The analyses measured dimethylphosphate (DMP), dimethylthiophosphate (DMTP), dimethyldithiophosphate (DMDTP), diethylphosphate (DEP), diethylthiophosphate (DETP), and diethyldithiophosphate (DEDTP). Briefly, urine samples were hydrolyzed using ß-glucuronidase and derivatized with pentafluorobenzyl bromide. The derivatized products were then extracted with a mixture of dichloromethane and hexane. Extracts were redissolved and analyzed by GC-MS-MS. The GC-MS-MS method employed an Agilent 6890 gas chromatograph with an Agilent 7683 automatic injector and sampler coupled to a Waters Quattro Micro-GC tandem quadrupole mass spectrometer and a workstation equipped with Waters MassLynx software version 4.1; measurements were carried out in MRM mode with a source in negative chemical ionization mode. Isotopically labelled dialkyl phosphate metabolite analogues were used as internal standards. Internal quality control was ensured by analyzing four different reference materials — three in-house (low, medium, and high) and one commercial — in each analysis sequence. External quality and accuracy of the analytical method was assessed by participating in interlaboratory comparison programs, including the G-EQUAS.

5.6.3.2 3,5,6-Trichloro-2-pyridinol

3,5,6-Trichloro-2-pyridinol (TCPy) analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2015c). In these analyses, free and conjugated forms of TCPy were measured together. Briefly, urine samples were hydrolyzed withß-glucuronidase/arylsulfatase and derivatized with dansyl chloride. The derivatized products were then extracted with hexane. The extracts were redissolved in a mixture of acetonitrile, methanol, and water, then analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization positive mode. Carbon-13 labelled TCPy was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence. The external quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the G-EQUAS.

5.6.3.3 Malathion Dicarboxylic Acid

Malathion dicarboxylic acid (DCA) analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2015b). Briefly, urine samples were extracted on an ion-exchange cartridge, eluted, and evaporated to dryness. Extracts were redissolved in ethyl acetate, derivatized with *N*-tert-butyldimethylsilyl-*N*-methyltrifluoroacetamide (MTBSTFA) and analyzed by gas chromatography coupled with tandem mass spectrometry (GC-MS-MS). The GC-MS-MS method employed an Agilent 7890A gas chromatograph with an Agilent 7693 automatic injector and sampler coupled to an Agilent 7000B tandem mass spectrometer and a workstation equipped with Waters MassHunter software; measurements were carried out in MRM mode with a source in electron ionization (EI) mode. Carbon-13 labelled DCA was used as an internal standard. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.6.3.4 Acephate and Methamidophos

Acephate and methamidophos analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2015a). Briefly, urine samples were extracted with dichloromethane under acidic conditions. The extracts were analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization positive mode. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.6.4 Pyrethroids

Pyrethroid analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018m). The analyses measured 3-phenoxybenzoic acid (3-PBA), 4-fluoro-3-phenoxybenzoic acid (4-F-3-PBA), *cis*-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA), *cis*-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA), and *trans*-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA). Briefly, urine samples were hydrolyzed using ß-glucuronidase and then acidified and extracted with hexane. The extracts were derivatized with hexafluoro-2-propanol (HFIP) and diisopropylcarbodiimide (DIC), and re-extracted with hexane. Extracts were then analyzed by GC-MS. The GC-MS method employed an Agilent 6890 network gas chromatograph with an Agilent 7683B automatic injector and sampler coupled to an Agilent 5975 mass spectrometer and a workstation equipped with Waters MassHunter software version B.07.01 build 7.1.524.0 and ChemStation G1701EA software version E02.01.1177; measurements were carried out in single ion monitoring (SIM) modes following negative chemical ionization. Carbon-13 labelled *trans*-DCCA, 4-F-3-PBA, and 3-PBA analogues were used as internal standards; the isotopically labelled *trans*-DCCA analogue was used as an internal standard for *cis*-DCCA, *trans*-DCCA, and *cis*-DCBA. Internal quality control was ensured by analyzing four different reference materials, three in-house (low, medium, and high) and one commercial, in each analysis sequence. External quality and accuracy of the analytical method was assessed by participating in interlaboratory comparison programs, including the G-EQUAS, for *cis*-DBCA, *cis*-DCCA, *trans*-DCCA, and 3-PBA.

5.7 PLASTICIZERS

5.7.1 Phthalates

Phthalate metabolite analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018l). The analyses measured 23 phthalate metabolites (see Table 3.4.1 for complete analyte list). Briefly, urine samples were hydrolyzed using ß-glucuronidase and the analytes were extracted using liquid–liquid extraction with a hexane:ethyl acetate solution (50:50) on an automated Perkin Elmer JANUS automated liquid-handling workstation. The extracts were analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization negative mode. Various internal standards were used including deuterated monoisobutyl phthalate (MiBP) and carbon-13 labelled monobenzyl phthalate (MBzP), monocyclohexyl phthalate (MCHP), monoisononyl phthalate (MiNP), monoethyl phthalate (MEP), monomethyl phthalate (MMP), mono-*n*-butyl phthalate (M*n*BP), mono-*n*-octyl phthalate (MOP), mono(2-ethylhexyl) phthalate (MEHP), mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), mono(2-ethyl-5-oxohexyl) phthalate (MEOHP), mono(3-carboxypropyl) phthalate (MCPP), mono(2-ethyl-5-carboxypentyl) phthalate (MECPP) analogues and deuterated mono[2-(carboxymethyl)hexyl] phthalate (MCMHP), monoisodecyl phthalate (MiDP), and mono-3-hydroxy-n-butyl phthalate (3OH-MBP). In addition to MEHHP, the isotopically labelled MEHHP was used as an internal standard for mono-carboxy-n-heptyl phthalate (MCHpP), monocarboxyisononyl phthalate (MCiNP), mono(carboxyisooctyl) phthalate (MCiOP), monohydroxyisodecyl phthalate (MHiDP), monohydroxyisononyl phthalate (MHiNP), monooxoisodecyl phthalate (MOiDP) and monooxoisononyl phthalate (MOiNP). Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence. External quality and accuracy of the analytical method was assessed by participating in interlaboratory comparison programs including the G-EQUAS, for MEHHP, MEOHP, MECPP, MEHP, M*n*BP, MiBP, and MBzP.

Due to issues during the peak integration process, results were reported for MCiOP, MiNP and MCiNP semi-quantitatively. Results for all other analytes were reported quantitatively.

5.7.2 Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) and Tri-(2-ethylhexyl) Trimellitate (TEHT)

Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) and tri-(2-ethylhexyl) trimellitate (TEHT) metabolite analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018n). The analyses measured the DINCH metabolites *trans*-cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH), cyclohexane-1,2-dicarboxylic mono oxoisononyl ester (oxo-MINCH), cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (OH-MINCH), *cis*-cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH), and *trans*-cyclohexane-1,2-diarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH). The analyses also measured the TEHT metabolites 1-mono(2-ethylhexyl)trimellitate (1-MEHTM), 2-mono(2-ethylhexyl)trimellitate (2-MEHTM), and 4-mono(2-ethylhexyl)trimellitate (4-MEHTM). Briefly, urine samples were hydrolyzed using ß-glucuronidase and the analytes were extracted using liquid–liquid extraction with a hexane:ethyl acetate solution (50:50) on an automated Perkin Elmer JANUS automated liquid-handling workstation. The extracts were taken up with a mixture of acetonitrile and demineralized water and analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization negative mode. Deuterated *trans*-cx-MINCH was used as an internal standard for *trans*-cx-MINCH, *cis*-cx-MINCH, oxo-MINCH, 1-MEHTM, 2-MEHTM, and 4-MEHTM. Deuterated *trans*-cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (*trans*-OH-MINCH) was used as an internal standard for OH-MINCH and deuterated *trans*-MINCH was used as an internal standard for *trans*-MINCH. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.7.3 2,2,4-Trimethyl-1,3-pentanediol Diisobutyrate (TXIB) and Cyclohexane-1,2-dicarboxylic Acid (CHDA)

Cyclohexane-1,2-dicarboxylic acid (CHDA) and 2,2,4-trimethyl-1,3-pentanediol diisobutyrate (TXIB) metabolite analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018o). The analyses measured the di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) metabolite CHDA and the TXIB metabolites 2,2,4-trimethyl-1,3-pentanediol (TMPD) and 2,2,4-trimethyl-3-hydroxy valeric acid (HTMV). Briefly, urine samples were hydrolyzed using ß-glucuronidase and arylsulfatase, acidified and extracted with ethyl acetate on an automated Perkin Elmer JANUS automated liquid-handling workstation. The extracts were taken up with a mixture of methanol and water and analyzed by UPLC-MS-MS. The UPLC-MS-MS method employed a Waters ACQUITY UPLC coupled to a Waters Xevo TQ-S tandem mass spectrometer and a workstation equipped with MassLynx software version 4.1; measurements were carried out in MRM mode with an electrospray ionization negative mode for HTMV and CHDA and positive mode for TMPD. Deuterated 2,2-bis(hydroxymethyl)pentane was used as an internal standard for TMPD. Deuterated HTMV and CHDA analogues were used as internal standards for HTMV and CHDA, respectively. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in each analysis sequence.

5.8 VOLATILE ORGANIC COMPOUNDS

Volatile organic compound (VOC) analyses in blood were performed at the Exposure and Biomonitoring Division Laboratory, Health Canada (Aranda-Rodriguez et al., 2015; Health Canada, 2018). The analyses measured benzene, carbon tetrachloride, 1,4-dichlorobenzene, 2,5-dimethylfuran, ethylbenzene, isopropylbenzene, methyl isobutyl ketone, nitrobenzene, styrene, 1,1,1,2-tetrachloroethane, tetrachloroethylene, tetrahydrofuran, toluene, trichloroethylene, bromodichloromethane, dibromochloromethane, tribromomethane, trichloromethane, *m*-xylene, *p*-xylene, and *o*-xylene. Briefly, blood samples were extracted by solid-phase microextraction, focused in a cryotrap and analyzed by GC-MS-MS. The method employed a

Thermo Fisher Scientific 915 cryotrap, TRACE™ ultra GC coupled to a TSQ Quantum XLS mass spectrometer and a workstation equipped with Xcalibur software version 3.1; measurements were carried out in selected reaction monitoring mode with an electron ionization source. Carbon-13 or deuterated analogues of the VOC analytes were used as internal standards. Internal quality control was ensured by analyzing three different in-house reference materials (low, medium, and high) in duplicate for each batch of analysis.

5.9 CREATININE

Creatinine analyses in urine were performed at the CTQ, INSPQ (INSPQ, 2018b) using the colorimetric end point Jaffe method. Briefly, urine samples were reacted with an alkaline solution of sodium picrate to form a red Janovski complex. The complex was analyzed by spectrophotometry at 510 nm. The method employed a Thermo Fischer Scientific Indiko Plus automatic analyzer and a workstation equipped with Indiko software version 5.3; measurements were carried out in kinetic mode. Internal quality control was ensured by analyzing two commercial reference materials in each analysis sequence. External quality and accuracy of the analytical method were assessed by participating in interlaboratory comparison programs, including the College of American Pathologists Forensic Urine Drug Testing (Confirmatory) Survey.

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6 STATISTICAL DATA ANALYSES

Descriptive statistics on the concentrations of environmental chemicals in the blood, urine, and hair of Canadians were generated using the Statistical Analysis System software (SAS Institute Inc., version 9.4, 2014) and the SUDAAN® (SUDAAN Release 11.0.1, 2013) statistical software package.

The Canadian Health Measures Survey (CHMS) is a sample survey. This means 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. 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, percentiles and detection frequencies (Rao et al., 1992; Rust and Rao, 1996).

Data tables are presented for each chemical measured in cycle 5. When available, data from previous cycles are also provided within the tables. In the first *Report on Biomonitoring of Environmental Chemicals in Canada*, all results were reported to two decimal places. For subsequent cycles 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); the percentage of the population with concentrations at or above the limit of detection (LOD), termed detection frequency; the geometric mean (GM); and the 10th, 50th, 90th, and 95th percentiles with associated 95% CIs. For each chemical, results are presented for the total population as well as by age group and 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. LOD values for each chemical are provided alongside their respective data tables and in Appendix A. Conversion factors to assist in the comparison of data from other studies that report different units are provided in Appendix B.

Chemicals measured in either whole blood or plasma are presented as weight of chemical per volume of whole blood or plasma (µg chemical/L blood or plasma). Data for hemoglobin adducts are presented as the amount of hemoglobin adduct per weight of hemoglobin (pmol adduct/g hemoglobin). Chromium (VI) measures in red blood cells are presented as weight of chromium per volume of red blood cells (µg/L red blood cells).

For urine measurements, concentrations are presented as weight of chemical per volume of urine (µg chemical/L urine) and adjusted for urinary creatinine (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 based on age, sex, and ethnicity; therefore, it may not be appropriate to compare creatinine-adjusted concentrations among different demographic groups (e.g., children and 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 listed as missing.

Descriptive statistics are available for creatinine (mg/dL) (Appendix C). These include n; detection frequency; GM; the 10th, 50th, 90th, and 95th percentiles; and associated 95% CIs for the total population as well as by age group and sex. Measurements that fell below the LOD for the laboratory analytical method were assigned a value equal to half the LOD.

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@canada.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 GM require at least 10 participants.

Estimates from a sample survey will 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; it is expressed as a percentage of the estimate. This report employs the following Statistics Canada guidelines for releasing estimates based on their CVs:

- 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 that cautions 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 5* (Statistics Canada, 2019).

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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 subsequent cycles included children as young as three years of age and covered approximately 96% to 97% of the Canadian population aged 3–79 years of age. For cycle 5, hair measures were added for a subset of the adult population aged 20–59 years. 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, associated with different blood, urine, or hair concentrations. Furthermore, small amounts of certain chemicals, such as selenium, 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 immunocompromized 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 the 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 most metals are measured as the parent compounds, many 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 pyrethroid pesticide deltamethrin is broken down into several metabolites. Some metabolites are specific to one parent compound, whereas others are common to several parent compounds. As well, several metabolites found in urine are also found in the environment as a result of other processes (e.g., dialkyl phosphate 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, urine and hair 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), cycle 4 (2014–2015), and cycle 5 (2016–2017). Results from future cycles can be compared with the baseline data from the CHMS in order to examine trends in Canadians' exposures to selected environmental chemicals. It is important to note that some sampling and analytical modifications between cycles may have contributed some variation in results for those substances measured in multiple cycles. The limits of detection (LODs) for certain analytical methods have changed from cycle to cycle (Appendix A). Although the LOD values did not change by a large margin, this difference should be noted when comparing data from multiple cycles. 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 of body weight 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 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@canada.ca.

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SUMMARIES AND RESULTS FOR METALS AND TRACE ELEMENTS 8

8.1 LEAD

Lead (CASRN 7439-92-1) is a naturally occurring element present at a median natural background concentration of 0.0008% in soil and till 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 world (WHO, 2000).

In North America, tetraethyl and tetramethyl lead were added to motor vehicle fuels as an anti-knock agent until the 1990s. Today 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 (Canada, 1990; 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, the 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, 2016c). 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 sequestered in soft tissues, such as the liver, kidneys, 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 in the range of 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). Chronic low-level exposure may affect both the central and peripheral nervous systems; however, the symptoms of relatively low exposure levels are often not apparent (ATSDR, 2007; Health Canada, 2013a). Chronic low-level exposure to lead has also been associated with developmental neurotoxicity, cardiovascular disease, decreases in renal functioning, and reproductive problems, as well as other health responses (ATSDR, 2007; Bushnik et al., 2014; Health Canada, 2013a; Lanphear et al., 2018). Cognitive and neurobehavioural effects have been recognized as major concerns for exposed children. In infants and children, exposure to lead is most strongly associated with neurodevelopmental effects, specifically the reduction of intelligence quotient (IQ) (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, although there is uncertainty associated with effects observed at these levels (Health Canada, 2013a). Most recently, research has shown a prenatal and gender-specific association between cord blood lead concentrations and IQ in Canadian preschool children (Desrochers-Couture et al., 2018). The International Agency for Research on Cancer (IARC) 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). Lead is subject to numerous federal risk management initiatives in Canada directed toward consumer products, cosmetics, drinking water, food, natural health products, therapeutic products, tobacco, and environmental media, including household dust, soil, and air. CEPA 1999 prohibits the addition of lead in gasoline and controls its release from secondary lead smelters, steel manufacturing, and mining effluents (Environment and Climate Change Canada, 2018). The use of lead in toys,

children's jewellery, clothing and accessories, and other products intended for children — along with consumer paints and surface coatings, glazed ceramics and glass foodware, and other consumer products that represent 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 include the Children's Jewellery Regulations, which establish a new guideline limit for lead in children's jewellery (Canada, 2016a). In addition, the Consumer Products Containing Lead Regulations have recently been put in place to limit the total lead content in an expanded scope of consumer products intended for use by a child or an adult in caring for a child (Canada, 2016b). Lead and its compounds are also identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

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, 2019). Health Canada has also published guidance on controlling corrosion in drinking water distribution systems to help control the leaching of metals, including lead, from system materials and components (Health Canada, 2009b). The concentration of lead in specific foods is managed by Health Canada under the Food and Drug Regulations (Canada, 1978); the existing maximum levels for lead in foods are found in the *List of Contaminants and Other Adulterating Substances in Foods*. Health Canada has updated the maximum level for lead in fruit juice, fruit nectar and water in sealed containers (Health Canada 2017a). Maximum levels for other foods and beverages are scheduled for review and update. These regulatory updates are among several Health Canada activities that are under way to ensure that dietary exposure to lead is as low as reasonably achievable (Health Canada, 2017b). Lead is also included in the list of trace elements analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2016a). The food items analyzed represent those that are most typical of the Canadian diet; 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 eightfold (Health Canada, 2016b).

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). Despite some uncertainties, the evidence for an association between neurodevelopmental effects in children and blood lead levels in the lower range of exposure is of concern. 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 (GM) 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 GM 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 who are up to six years old reported a GM blood lead level of 2.21 µg/dL (Richardson et al., 2011). The First Nations Biomonitoring Initiative (FNBI) was a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprised 13 randomly selected First Nation communities in Canada, with 503 First Nations participants aged 20 years and older. In 2011, the GM 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 (CHMS) participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). Data from these cycles are presented in blood as µg/dL. Lead was also analyzed in hair from CHMS participants aged 20–59 in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Table 8.1.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	100	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	99.8 (98.7–100)	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	99.9 (99.7–100)	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)
5 (2016–2017)	4517	99.7 (98.6–99.9)	0.93 (0.85–1.0)	0.39 (0.36–0.42)	0.92 (0.82–1.0)	2.0 (1.8–2.1)	2.5 (2.1–2.9)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	100	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	99.9 (99.1–100)	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	100 (99.4–100)	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)
5 (2016–2017)	2257	100	1.0 (0.93–1.2)	0.48 (0.43–0.52)	1.0 (0.89–1.1)	2.2 (1.9–2.5)	2.8 (2.1–3.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	100	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	99.6 (97.1–100)	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	99.9 (99.8–100)	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)
5 (2016–2017)	2260	99.4 (97.3–99.9)	0.82 (0.77–0.88)	0.34 (0.32–0.36)	0.82 (0.72–0.92)	1.8 (1.6–1.9)	2.2 (2.0–2.4)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	100	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	100	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	100	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)
5 (2016–2017)	473	99.9 (99.2–100)	0.56 (0.42–0.73)	0.31 (0.26–0.35)	0.52 (0.40–0.65)	1.0[E] (0.43–1.6)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	910	100	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	100	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	100	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	99.9 (99.0–100)	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)
5 (2016–2017)	511	100	0.54 (0.48–0.59)	0.28 (0.26–0.30)	0.51 (0.45–0.58)	0.99 (0.76–1.2)	1.3 (1.0–1.5)
12–19 years							
1 (2007–2009)	945	100	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	100	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	100 (99.5–100)	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	99.7 (98.6–99.9)	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)
5 (2016–2017)	521	100	0.48 (0.43–0.52)	0.25 (0.22–0.29)	0.46 (0.42–0.49)	0.90 (0.75–1.1)	1.0 (0.70–1.3)
20–39 years							
1 (2007–2009)	1165	100 (99.6–100)	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	100	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	99.4 (96.0–99.9)	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	99.9 (98.9–100)	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)
5 (2016–2017)	1038	99.8 (99.5–99.9)	0.78 (0.70–0.86)	0.35 (0.29–0.40)	0.82 (0.68–0.97)	1.5 (1.3–1.6)	1.9 (1.5–2.3)
40–59 years							
1 (2007–2009)	1220	100	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	100	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	99.9 (98.4–100)	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	100	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)
5 (2016–2017)	990	99.2 (95.3–99.9)	1.0 (0.94–1.2)	0.50 (0.44–0.55)	1.0 (0.90–1.1)	2.1 (1.8–2.4)	2.6 (1.7–3.5)
60–79 years							
1 (2007–2009)	1079	100	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	100	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	99.9 (98.8–100)	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	100	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)
5 (2016–2017)	984	100	1.4 (1.3–1.5)	0.69 (0.62–0.76)	1.4 (1.3–1.5)	2.5 (2.3–2.7)	3.1 (2.6–3.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 0.02, 0.1, 0.16, 0.16, and 0.17 µg/dL, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

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8.2 ARSENIC

Arsenic (CASRN 7440-38-2) is a naturally occurring element that makes 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). It 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 in the environment 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 semiconductors, 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 an herbicide (ATSDR, 2007; Health Canada, 2006). Chromated copper arsenate has been used as a wood preservative in residential construction projects, such as playground structures and decks; however, it is now approved only for industrial purposes and domestic wood foundations (Health Canada, 2011). In 2004, the wood-treatment industry in the U.S. and Canada began to transition away from chromated copper arsenate for most residential uses. Organic arsenical herbicides, such as MMA and DMA, are no longer registered for use in Canada (Health Canada, 2019).

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), while in other foods, inorganic arsenic may represent the predominant form (Batista et al., 2011; CFIA, 2013; Conklin and Chen, 2012; FDA, 2016; Huang et al., 2012). Exposure may also arise from indoor house dust; levels of arsenic 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 can be absorbed via oral and inhalation routes; arsenic in all its forms is not readily absorbed via skin contact. 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 begins with a 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; Health Canada, 2016b). Arsenic and inorganic arsenic compounds are classified as carcinogenic to humans by Health Canada and other international agencies (EPA, 2002; 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). While the majority of assessments of the toxicity of arsenic have focused on the inorganic forms, recent studies have highlighted the potential for organic arsenic compounds, in particular the pentavalent DMA, to be carcinogenic (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 not be classifiable with respect to their carcinogenicity in humans (Group 3) (IARC, 2012).

As part of a risk assessment conducted under the mandate of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), 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 CEPA 1999, which 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, metal mining, wood preservation, and steel manufacturing processes (Environment and Climate Change Canada, 2017). Arsenic and its compounds are identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with the requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Canada, 1985; Health Canada, 2018). 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 children in learning or play situations (Canada, 2010b; Canada, 2011). The sale and use of arsenical pesticides, such as chromated copper arsenate,

are regulated in Canada by the Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002).

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 trace elements analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2016a). 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. Health Canada has established maximum levels for arsenic in some foods which are found in the List of Contaminants and Other Adulterating Substances in Foods, incorporated by reference into Division 15 of the Food and Drug Regulations. Health Canada has updated the maximum level for total arsenic in bottled water (Health Canada, 2017a; Health Canada, 2017b); maximum levels for 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 (GM) 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 Québec City with 500 participants aged 18–65 years, the GM of total arsenic in urine was 0.17 µmol/L (12.73 µg/L) and in whole blood was 12.71 nmol/L (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), cycle 4 (2014–2015), and cycle 5 (2016–2017) 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), cycle 4 (2014–2015) and cycle 5 (2016–2017) 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. In addition, total arsenic was analyzed in hair from CHMS participants aged 20–59 in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Table 8.2.1

Inorganic-related arsenic species[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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

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)
5 (2016–2017)	2615	4.3 (3.5–5.4)	1.3 (0.98–1.6)	4.1 (3.2–5.1)	14[E] (8.4–19)	20[E] (9.5–30)
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)
5 (2016–2017)	1299	4.3 (3.5–5.3)	1.3 (0.83–1.7)	4.0 (3.2–4.8)	14 (9.2–20)	20[E] (12–29)
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)
5 (2016–2017)	1316	4.4 (3.4–5.7)	1.3 (0.98–1.6)	4.4 (3.2–5.5)	13[E] (6.4–19)	F
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)
5 (2016–2017)	535	4.5 (3.7–5.4)	1.4 (0.92–1.9)	4.5 (3.6–5.5)	14 (9.8–18)	23[E] (13–33)
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)
5 (2016–2017)	513	4.4 (4.0–4.8)	1.7 (1.4–1.9)	4.3 (3.8–4.9)	9.7 (8.6–11)	14 (10–18)
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)
5 (2016–2017)	517	4.5 (3.8–5.3)	1.5 (1.1–2.0)	4.5 (3.9–5.1)	12 (9.1–16)	17[E] (11–24)

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
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)
5 (2016–2017)	357	4.6 (3.2–6.6)	1.5[E] (0.85–2.2)	3.8 (2.6–5.0)	17[E] (6.1–29)	F
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)
5 (2016–2017)	345	4.5 (3.4–6.0)	1.2 (0.90–1.5)	4.7 (3.2–6.3)	13[E] (8.2–19)	F
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)
5 (2016–2017)	348	3.8 (3.0–4.7)	1.1 (0.84–1.4)	3.3 (2.3–4.3)	13 (9.0–18)	18 (14–22)

CI: confidence interval; GM: geometric mean

a For each individual within a cycle, the sum of arsenate, arsenite, DMA, and MMA 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 are too unreliable to be published.

Table 8.2.2

Inorganic-related arsenic species[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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

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)
5 (2016–2017)	2605	4.3 (3.5–5.2)	1.6 (1.3–1.9)	3.6 (2.9–4.3)	12 (8.0–16)	18 (12–24)
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)
5 (2016–2017)	1296	3.7 (3.1–4.5)	1.4 (1.1–1.8)	3.2 (2.7–3.7)	12 (7.7–16)	17 (13–21)
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)
5 (2016–2017)	1309	4.9 (3.9–6.1)	1.9 (1.5–2.4)	4.3 (3.4–5.3)	12[E] (7.2–17)	F
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)
5 (2016–2017)	532	7.5 (6.1–9.1)	3.5 (2.8–4.2)	6.8 (5.3–8.3)	17[E] (9.7–24)	27 (18–35)
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)
5 (2016–2017)	509	5.1 (4.5–5.7)	2.3 (1.7–2.9)	4.9 (4.4–5.3)	11 (8.9–13)	14 (8.7–19)
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)
5 (2016–2017)	515	3.4 (3.0–3.9)	1.5 (1.1–1.9)	3.0 (2.6–3.4)	8.1 (6.0–10)	13[E] (6.1–20)

Cycle	n	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
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)
5 (2016–2017)	357	4.2[E] (2.9–6.1)	1.4 (0.96–1.8)	3.4 (2.2–4.6)	13[E] (4.1–21)	F
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)
5 (2016–2017)	345	4.1 (3.3–5.1)	1.6 (1.2–2.1)	3.4 (2.5–4.3)	12 (8.2–16)	20 (13–26)
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)
5 (2016–2017)	347	4.4 (3.6–5.3)	1.8 (1.6–2.1)	3.9 (3.0–4.8)	11 (7.4–15)	15 (13–18)

CI: confidence interval; GM: geometric mean

a For each individual within a cycle, the sum of arsenate, arsenite, DMA, and MMA 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 are too unreliable to be published.

Table 8.2.3

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2537	27.4 (21.0–34.9)	—	<LOD	<LOD	1.7 (1.1–2.3)	2.7[E] (1.3–4.0)
3 (2012–2013)	2535	25.7 (22.7–29.0)	—	<LOD	<LOD	1.7[E] (0.92–2.5)	F
4 (2014–2015)	2567	31.9 (27.0–37.2)	—	<LOD	<LOD	1.9 (1.5–2.3)	2.7 (2.1–3.4)
5 (2016–2017)	2615	60.9 (51.2–69.8)	—	<LOD	0.36 (0.25–0.48)	2.2[E] (0.91–3.4)	F
Males, 3–79 years							
2 (2009–2011)	1271	31.7 (23.8–40.7)	—	<LOD	<LOD	1.7 (1.1–2.3)	2.8[E] (0.88–4.7)
3 (2012–2013)	1250	29.1 (23.9–34.8)	—	<LOD	<LOD	1.4 (1.0–1.8)	F
4 (2014–2015)	1275	36.0 (29.3–43.4)	—	<LOD	<LOD	2.2 (1.7–2.6)	3.0 (2.3–3.8)
5 (2016–2017)	1299	62.2 (50.8–72.5)	—	<LOD	0.37 (0.26–0.47)	1.9[E] (0.69–3.0)	F
Females, 3–79 years							
2 (2009–2011)	1266	23.0 (17.2–30.1)	—	<LOD	<LOD	1.5[E] (0.72–2.3)	2.4[E] (1.1–3.7)
3 (2012–2013)	1285	22.3 (16.4–29.7)	—	<LOD	<LOD	F	F
4 (2014–2015)	1292	27.8 (21.7–34.8)	—	<LOD	<LOD	1.5 (1.1–2.0)	2.4[E] (1.3–3.5)
5 (2016–2017)	1316	59.5 (49.9–68.5)	—	<LOD	0.36[E] (<LOD–0.50)	2.5[E] (1.0–4.0)	F
3–5 years							
2 (2009–2011)	516	14.0 (9.7–19.6)	—	<LOD	<LOD	0.79[E] (<LOD–1.2)	1.3[E] (0.74–1.9)
3 (2012–2013)	500	13.9 (10.7–17.9)	—	<LOD	<LOD	0.94 (<LOD–1.2)	1.9[E] (0.75–3.0)
4 (2014–2015)	512	17.3 (13.1–22.5)	—	<LOD	<LOD	1.1 (0.84–1.3)	1.8[E] (1.0–2.5)
5 (2016–2017)	535	49.1 (37.4–60.9)	—	<LOD	<LOD	1.3 (0.96–1.5)	1.8[E] (0.57–3.1)
6–11 years							
2 (2009–2011)	511	20.6 (15.7–26.4)	—	<LOD	<LOD	1.0[E] (<LOD–1.4)	1.8[E] (1.1–2.4)
3 (2012–2013)	506	21.2[E] (13.3–32.1)	—	<LOD	<LOD	1.1 (0.81–1.4)	1.6[E] (0.82–2.5)
4 (2014–2015)	514	25.6 (19.0–33.6)	—	<LOD	<LOD	1.5[E] (0.92–2.0)	2.6[E] (1.2–4.0)
5 (2016–2017)	513	53.6 (44.3–62.6)	—	<LOD	0.26[E] (<LOD–0.38)	1.3 (0.87–1.7)	1.7 (1.4–2.1)
12–19 years							
2 (2009–2011)	510	29.2 (21.1–38.9)	—	<LOD	<LOD	1.9[E] (1.2–2.7)	F
3 (2012–2013)	510	28.2 (21.2–36.5)	—	<LOD	<LOD	1.5[E] (<LOD–2.3)	2.6[E] (1.1–4.0)
4 (2014–2015)	506	34.6 (26.6–43.5)	—	<LOD	<LOD	2.1[E] (1.2–3.0)	3.2 (2.1–4.4)
5 (2016–2017)	517	64.4 (53.0–74.4)	0.40 (0.31–0.53)	<LOD	0.42 (0.29–0.55)	F	4.2[E] (1.1–7.3)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	31.8 (21.8–43.8)	—	<LOD	<LOD	1.9[E] (<LOD–3.1)	F
3 (2012–2013)	355	28.9 (22.1–36.9)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	34.5 (28.5–41.1)	—	<LOD	<LOD	2.3 (1.7–2.8)	3.0 (2.3–3.8)
5 (2016–2017)	357	68.5 (53.6–80.4)	0.50[E] (0.31–0.78)	<LOD	0.44[E] (0.27–0.61)	F	F
40–59 years							
2 (2009–2011)	356	25.2 (17.6–34.8)	—	<LOD	<LOD	1.3[E] (0.75–1.8)	2.0[E] (1.0–2.9)
3 (2012–2013)	312	24.5[E] (16.6–34.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	29.4 (20.1–40.8)	—	<LOD	<LOD	1.6[E] (1.0–2.3)	2.3[E] (1.5–3.2)
5 (2016–2017)	345	63.2 (44.6–78.6)	0.43[E] (0.29–0.65)	<LOD	0.37[E] (<LOD–0.59)	2.5[E] (1.2–3.7)	F
60–79 years							
2 (2009–2011)	289	28.1 (19.5–38.6)	—	<LOD	<LOD	1.9[E] (1.1–2.7)	F
3 (2012–2013)	352	26.2 (18.9–35.1)	—	<LOD	<LOD	1.8 (1.1–2.4)	3.2[E] (1.3–5.2)
4 (2014–2015)	361	35.2 (26.2–45.4)	—	<LOD	<LOD	1.8 (1.2–2.3)	F
5 (2016–2017)	348	49.5 (39.8–59.3)	—	<LOD	<LOD	1.6[E] (0.77–2.4)	3.4[E] (1.5–5.4)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2, 3, 4, and 5 are 0.8, 0.75, 0.75, and 0.25 µg As/L, respectively.

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 are too unreliable to be published.

Table 8.2.4

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2527	27.4 (21.0–34.9)	—	<LOD	<LOD	2.0 (1.6–2.3)	2.9 (1.9–3.9)
3 (2012–2013)	2534	25.7 (22.7–29.0)	—	<LOD	<LOD	1.9[E] (1.2–2.7)	F
4 (2014–2015)	2566	31.9 (27.0–37.2)	—	<LOD	<LOD	1.6 (1.3–1.9)	2.2 (1.5–2.9)
5 (2016–2017)	2605	60.9 (51.2–69.8)	—	<LOD	0.35 (0.28–0.42)	1.7[E] (0.71–2.7)	3.3[E] (1.7–5.0)
Males, 3–79 years							
2 (2009–2011)	1267	31.7 (23.8–40.7)	—	<LOD	<LOD	1.4[E] (0.85–1.9)	F
3 (2012–2013)	1250	29.1 (23.9–34.8)	—	<LOD	<LOD	1.2 (0.94–1.5)	F
4 (2014–2015)	1274	36.0 (29.3–43.4)	—	<LOD	<LOD	1.5 (1.0–1.9)	2.0 (1.4–2.6)
5 (2016–2017)	1296	62.2 (50.8–72.5)	—	<LOD	0.31 (0.25–0.38)	1.6[E] (0.91–2.3)	2.9[E] (1.6–4.1)
Females, 3–79 years							
2 (2009–2011)	1260	23.0 (17.2–30.1)	—	<LOD	<LOD	2.2 (1.6–2.8)	3.0 (2.1–3.9)
3 (2012–2013)	1284	22.3 (16.4–29.7)	—	<LOD	<LOD	2.4[E] (<LOD–3.9)	F
4 (2014–2015)	1292	27.8 (21.7–34.8)	—	<LOD	<LOD	1.7 (1.2–2.1)	2.6[E] (1.4–3.9)
5 (2016–2017)	1309	59.5 (49.9–68.5)	—	<LOD	0.41 (<LOD–0.55)	F	F
3–5 years							
2 (2009–2011)	515	14.0 (9.7–19.6)	—	<LOD	<LOD	1.9 (<LOD–2.2)	2.9 (1.9–3.9)
3 (2012–2013)	499	13.9 (10.7–17.9)	—	<LOD	<LOD	2.5[E] (<LOD–3.7)	4.3[E] (2.6–6.1)
4 (2014–2015)	512	17.3 (13.1–22.5)	—	<LOD	<LOD	2.1 (1.8–2.5)	3.0[E] (1.8–4.2)
5 (2016–2017)	532	49.1 (37.4–60.9)	—	<LOD	<LOD	1.7 (1.2–2.2)	2.3[E] (1.3–3.3)
6–11 years							
2 (2009–2011)	509	20.6 (15.7–26.4)	—	<LOD	<LOD	1.6[E] (<LOD–2.2)	2.2[E] (1.2–3.1)
3 (2012–2013)	506	21.2[E] (13.3–32.1)	—	<LOD	<LOD	1.7 (1.1–2.2)	2.5[E] (1.3–3.6)
4 (2014–2015)	513	25.6 (19.0–33.6)	—	<LOD	<LOD	1.6 (1.2–2.0)	2.2[E] (0.77–3.7)
5 (2016–2017)	509	53.6 (44.3–62.6)	—	<LOD	0.34 (<LOD–0.44)	1.2 (0.92–1.4)	1.7 (1.1–2.3)
12–19 years							
2 (2009–2011)	508	29.2 (21.1–38.9)	—	<LOD	<LOD	1.4[E] (0.85–2.0)	2.9[E] (1.4–4.5)
3 (2012–2013)	510	28.2 (21.2–36.5)	—	<LOD	<LOD	1.4[E] (<LOD–2.0)	1.9[E] (1.0–2.8)
4 (2014–2015)	506	34.6 (26.6–43.5)	—	<LOD	<LOD	1.4 (1.0–1.8)	2.0[E] (1.2–2.8)
5 (2016–2017)	515	64.4 (53.0–74.4)	0.31 (0.24–0.40)	<LOD	0.30 (0.24–0.36)	1.0 (<LOD–1.4)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	31.8 (21.8–43.8)	—	<LOD	<LOD	1.9[E] (<LOD–3.0)	2.6[E] (<LOD–4.3)
3 (2012–2013)	355	28.9 (22.1–36.9)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	34.5 (28.5–41.1)	—	<LOD	<LOD	1.6 (1.0–2.1)	2.1[E] (1.2–3.0)
5 (2016–2017)	357	68.5 (53.6–80.4)	0.46[E] (0.29–0.74)	<LOD	0.41[E] (0.24–0.58)	F	F
40–59 years							
2 (2009–2011)	354	25.2 (17.6–34.8)	—	<LOD	<LOD	1.9 (1.3–2.6)	2.0[E] (1.2–2.8)
3 (2012–2013)	312	24.5[E] (16.6–34.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	29.4 (20.1–40.8)	—	<LOD	<LOD	1.4 (0.93–1.9)	F
5 (2016–2017)	345	63.2 (44.6–78.6)	0.40 (0.29–0.56)	<LOD	0.35 (<LOD–0.46)	1.9[E] (1.1–2.6)	2.8[E] (1.3–4.3)
60–79 years							
2 (2009–2011)	288	28.1 (19.5–38.6)	—	<LOD	<LOD	2.3[E] (1.2–3.3)	F
3 (2012–2013)	352	26.2 (18.9–35.1)	—	<LOD	<LOD	2.3[E] (0.79–3.8)	3.7[E] (1.7–5.6)
4 (2014–2015)	361	35.2 (26.2–45.4)	—	<LOD	<LOD	1.7 (1.3–2.0)	2.6[E] (<LOD–4.0)
5 (2016–2017)	347	49.5 (39.8–59.3)	—	<LOD	<LOD	1.4 (0.98–1.8)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 8.2.5

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2536	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2567	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2615	12.7 (10.0–15.9)	—	<LOD	<LOD	0.17 (<LOD–0.20)	0.23 (0.19–0.27)
Males, 3–79 years							
2 (2009–2011)	1271	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1251	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1275	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1299	13.7 (9.6–19.1)	—	<LOD	<LOD	0.18 (<LOD–0.22)	0.25 (0.20–0.31)
Females, 3–79 years							
2 (2009–2011)	1267	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1285	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1292	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1316	11.7 (8.3–16.2)	—	<LOD	<LOD	0.15 (<LOD–0.21)	0.21 (0.18–0.24)
3–5 years							
2 (2009–2011)	516	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	500	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	535	25.1 (19.2–32.2)	—	<LOD	<LOD	0.29 (0.19–0.40)	0.38 (0.34–0.43)
6–11 years							
2 (2009–2011)	511	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	507	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	514	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	513	15.0[E] (8.2–26.0)	—	<LOD	<LOD	0.18 (<LOD–0.24)	0.24[E] (<LOD–0.41)
12–19 years							
2 (2009–2011)	510	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	510	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	517	18.0 (12.5–25.2)	—	<LOD	<LOD	0.20 (0.15–0.25)	0.27 (0.18–0.36)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	355	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	357	14.7[E] (8.8–23.5)	—	<LOD	<LOD	0.18 (<LOD–0.24)	0.21[E] (<LOD–0.30)
40–59 years							
2 (2009–2011)	357	0	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	345	10.6[E] (6.1–17.8)	—	<LOD	<LOD	F	0.26 (0.18–0.34)
60–79 years							
2 (2009–2011)	289	0	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	352	0	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	348	7.7[E] (5.0–11.7)	—	<LOD	<LOD	<LOD	0.18 (<LOD–0.23)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2, 3, 4, and 5 are 0.8, 0.75, 0.75, and 0.14 µg As/L, respectively.

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 are too unreliable to be published.

Table 8.2.6

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	2535	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2566	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2605	12.7 (10.0–15.9)	—	<LOD	<LOD	0.25 (<LOD–0.30)	0.36 (0.33–0.39)
Males, 3–79 years							
2 (2009–2011)	1267	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1251	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1274	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1296	13.7 (9.6–19.1)	—	<LOD	<LOD	0.20 (<LOD–0.23)	0.28 (0.19–0.38)
Females, 3–79 years							
2 (2009–2011)	1261	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	1284	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1292	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1309	11.7 (8.3–16.2)	—	<LOD	<LOD	0.32 (<LOD–0.38)	0.37 (0.35–0.40)
3–5 years							
2 (2009–2011)	515	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	499	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	512	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	532	25.1 (19.2–32.2)	—	<LOD	<LOD	0.50 (0.41–0.58)	0.73 (0.51–0.94)
6–11 years							
2 (2009–2011)	509	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	507	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	513	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	509	15.0[E] (8.2–26.0)	—	<LOD	<LOD	0.26[E] (<LOD–0.38)	0.37 (<LOD–0.43)
12–19 years							
2 (2009–2011)	508	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	510	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	506	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	515	18.0 (12.5–25.2)	—	<LOD	<LOD	0.19 (0.15–0.23)	0.28 (0.18–0.38)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	F	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	355	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	362	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	357	14.7[E] (8.8–23.5)	—	<LOD	<LOD	0.24[E] (<LOD–0.37)	0.36 (<LOD–0.42)
40–59 years							
2 (2009–2011)	355	0	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	312	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	312	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	345	10.6[E] (6.1–17.8)	—	<LOD	<LOD	<LOD	0.33 (0.27–0.38)
60–79 years							
2 (2009–2011)	288	0	—	<LOD	<LOD	<LOD	<LOD
3 (2012–2013)	352	0	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	361	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	347	7.7[E] (5.0–11.7)	—	<LOD	<LOD	<LOD	0.35 (<LOD–0.43)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 8.2.7

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	28.5 (22.7–35.2)	—	<LOD	<LOD	1.2 (1.0–1.4)	1.6 (1.1–2.0)
3 (2012–2013)	2536	26.4 (23.9–29.1)	—	<LOD	<LOD	1.2 (1.1–1.4)	1.5 (1.3–1.7)
4 (2014–2015)	2567	30.6 (26.1–35.4)	—	<LOD	<LOD	1.2 (1.0–1.4)	1.6 (1.3–1.9)
5 (2016–2017)	2615	81.7 (68.3–90.2)	0.35 (0.27–0.45)	<LOD	0.40 (0.31–0.48)	1.1 (0.81–1.4)	1.7 (1.1–2.2)
Males, 3–79 years							
2 (2009–2011)	1271	34.7 (27.4–42.8)	—	<LOD	<LOD	1.3 (0.92–1.6)	1.8 (1.3–2.4)
3 (2012–2013)	1251	31.8 (27.7–36.2)	—	<LOD	<LOD	1.2 (1.0–1.4)	1.5 (1.3–1.7)
4 (2014–2015)	1275	34.0 (28.1–40.6)	—	<LOD	<LOD	1.3 (1.1–1.6)	1.7 (1.3–2.1)
5 (2016–2017)	1299	83.9 (68.1–92.8)	0.37 (0.28–0.49)	<LOD	0.42 (0.33–0.52)	0.99[E] (0.58–1.4)	1.7 (1.2–2.2)
Females, 3–79 years							
2 (2009–2011)	1267	22.3 (16.6–29.3)	—	<LOD	<LOD	1.1 (0.84–1.3)	1.3 (1.0–1.5)
3 (2012–2013)	1285	21.0 (16.2–26.7)	—	<LOD	<LOD	1.2 (0.88–1.5)	1.5 (1.3–1.8)
4 (2014–2015)	1292	27.1 (22.0–32.8)	—	<LOD	<LOD	1.1 (0.89–1.3)	1.5 (1.1–1.9)
5 (2016–2017)	1316	79.4 (64.9–88.9)	0.33 (0.26–0.43)	<LOD	0.38 (0.29–0.47)	1.2 (0.96–1.5)	1.6[E] (0.95–2.3)
3–5 years							
2 (2009–2011)	516	19.7 (14.2–26.5)	—	<LOD	<LOD	0.98 (0.79–1.2)	1.3 (1.1–1.5)
3 (2012–2013)	500	18.2 (14.2–23.1)	—	<LOD	<LOD	0.91 (<LOD–1.2)	1.5 (1.1–1.9)
4 (2014–2015)	512	21.6[E] (14.4–31.2)	—	<LOD	<LOD	0.89 (0.81–0.98)	1.1 (0.94–1.3)
5 (2016–2017)	535	83.4 (70.1–91.5)	0.33 (0.25–0.44)	<LOD	0.37 (0.30–0.44)	0.92[E] (0.49–1.3)	1.1[E] (0.30–1.9)
6–11 years							
2 (2009–2011)	511	27.6 (21.9–34.1)	—	<LOD	<LOD	0.97[E] (<LOD–1.3)	1.6 (1.1–2.1)
3 (2012–2013)	507	24.2[E] (16.2–34.6)	—	<LOD	<LOD	1.0 (0.84–1.2)	1.3 (1.1–1.4)
4 (2014–2015)	514	27.6 (22.3–33.7)	—	<LOD	<LOD	1.2 (0.89–1.4)	1.5 (1.2–1.8)
5 (2016–2017)	513	87.4 (72.2–94.9)	0.34 (0.28–0.41)	<LOD	0.36 (0.30–0.43)	0.81 (0.74–0.88)	0.99 (0.90–1.1)
12–19 years							
2 (2009–2011)	510	33.6 (25.1–43.4)	—	<LOD	<LOD	1.3 (0.97–1.6)	1.7 (1.2–2.2)
3 (2012–2013)	510	40.7 (31.5–50.6)	—	<LOD	<LOD	1.3 (1.1–1.6)	1.6 (1.3–1.8)
4 (2014–2015)	506	37.3 (29.7–45.6)	—	<LOD	<LOD	1.3 (0.88–1.8)	1.8 (1.3–2.4)
5 (2016–2017)	517	88.7 (73.6–95.7)	0.43 (0.36–0.52)	<LOD	0.52 (0.45–0.60)	1.1 (0.80–1.4)	1.5 (1.2–1.7)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	34.3 (25.2–44.6)	—	<LOD	<LOD	1.2 (0.89–1.6)	1.7[E] (0.94–2.5)
3 (2012–2013)	355	32.1 (24.2–41.1)	—	<LOD	<LOD	1.3 (1.0–1.5)	1.5 (1.3–1.7)
4 (2014–2015)	362	38.1 (32.2–44.4)	—	<LOD	<LOD	1.3 (1.1–1.6)	1.6 (1.3–1.9)
5 (2016–2017)	357	77.1 (58.5–89.0)	0.36[E] (0.24–0.54)	<LOD	0.42[E] (0.27–0.58)	1.4[E] (0.67–2.1)	2.0[E] (1.2–2.7)
40–59 years							
2 (2009–2011)	357	27.8 (20.7–36.3)	—	<LOD	<LOD	1.2 (0.92–1.5)	1.4[E] (0.87–1.9)
3 (2012–2013)	312	20.9 (14.9–28.5)	—	<LOD	<LOD	1.1 (0.84–1.4)	1.6 (1.1–2.2)
4 (2014–2015)	312	28.4 (20.6–37.8)	—	<LOD	<LOD	1.2[E] (<LOD–1.7)	1.9[E] (<LOD–3.0)
5 (2016–2017)	345	85.4 (69.3–93.8)	0.38 (0.28–0.52)	<LOD	0.41 (0.28–0.53)	1.1 (0.84–1.3)	1.7[E] (1.0–2.3)
60–79 years							
2 (2009–2011)	289	19.2[E] (11.8–29.7)	—	<LOD	<LOD	1.0 (0.70–1.3)	1.4[E] (0.73–2.0)
3 (2012–2013)	352	23.9 (18.4–30.5)	—	<LOD	<LOD	1.1 (0.79–1.5)	1.4 (1.2–1.6)
4 (2014–2015)	361	21.7 (14.9–30.4)	—	<LOD	<LOD	1.1 (0.84–1.3)	1.3 (0.99–1.6)
5 (2016–2017)	348	77.2 (64.8–86.2)	0.28 (0.22–0.36)	<LOD	0.33 (0.27–0.39)	0.85 (0.67–1.0)	1.1[E] (0.60–1.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2, 3, 4, and 5 are 0.8, 0.75, 0.75, and 0.13 µg As/L, respectively.

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 8.2.8

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	28.5 (22.7–35.2)	—	<LOD	<LOD	1.3[E] (0.75–1.8)	2.0 (1.8–2.1)
3 (2012–2013)	2535	26.4 (23.9–29.1)	—	<LOD	<LOD	1.2 (1.1–1.4)	1.7 (1.5–1.9)
4 (2014–2015)	2566	30.6 (26.1–35.4)	—	<LOD	<LOD	1.2 (0.97–1.3)	1.4 (1.2–1.7)
5 (2016–2017)	2605	81.7 (68.3–90.2)	0.35 (0.27–0.45)	<LOD	0.34 (0.29–0.39)	0.97 (0.73–1.2)	1.3 (0.97–1.7)
Males, 3–79 years							
2 (2009–2011)	1267	34.7 (27.4–42.8)	—	<LOD	<LOD	1.0 (0.87–1.1)	1.6[E] (1.0–2.2)
3 (2012–2013)	1251	31.8 (27.7–36.2)	—	<LOD	<LOD	1.0 (0.87–1.1)	1.3 (1.0–1.6)
4 (2014–2015)	1274	34.0 (28.1–40.6)	—	<LOD	<LOD	1.0 (0.91–1.2)	1.3 (1.0–1.6)
5 (2016–2017)	1296	83.9 (68.1–92.8)	0.32 (0.24–0.43)	<LOD	0.32 (0.27–0.37)	0.77 (0.56–0.98)	1.0 (0.76–1.2)
Females, 3–79 years							
2 (2009–2011)	1261	22.3 (16.6–29.3)	—	<LOD	<LOD	1.9 (1.4–2.4)	2.0 (1.4–2.5)
3 (2012–2013)	1284	21.0 (16.2–26.7)	—	<LOD	<LOD	1.6 (1.3–1.9)	2.1 (1.8–2.5)
4 (2014–2015)	1292	27.1 (22.0–32.8)	—	<LOD	<LOD	1.3 (0.97–1.6)	1.7 (1.2–2.2)
5 (2016–2017)	1309	79.4 (64.9–88.9)	0.37 (0.29–0.48)	<LOD	0.37 (0.31–0.43)	1.1 (0.73–1.5)	1.4 (1.1–1.8)
3–5 years							
2 (2009–2011)	515	19.7 (14.2–26.5)	—	<LOD	<LOD	1.9 (1.8–2.0)	2.7 (1.8–3.6)
3 (2012–2013)	499	18.2 (14.2–23.1)	—	<LOD	<LOD	2.0 (<LOD–2.5)	3.0 (2.0–4.0)
4 (2014–2015)	512	21.6[E] (14.4–31.2)	—	<LOD	<LOD	1.8 (1.3–2.2)	2.2 (1.9–2.5)
5 (2016–2017)	532	83.4 (70.1–91.5)	0.56 (0.47–0.68)	<LOD	0.60 (0.47–0.72)	1.3 (1.1–1.5)	1.5 (1.3–1.6)
6–11 years							
2 (2009–2011)	509	27.6 (21.9–34.1)	—	<LOD	<LOD	1.2 (<LOD–1.6)	1.9 (1.7–2.1)
3 (2012–2013)	507	24.2[E] (16.2–34.6)	—	<LOD	<LOD	1.3 (1.1–1.5)	1.8 (1.5–2.0)
4 (2014–2015)	513	27.6 (22.3–33.7)	—	<LOD	<LOD	1.2 (1.0–1.3)	1.4 (1.2–1.5)
5 (2016–2017)	509	87.4 (72.2–94.9)	0.39 (0.31–0.49)	<LOD	0.41 (0.35–0.47)	0.80 (0.65–0.95)	1.0 (0.88–1.2)
12–19 years							
2 (2009–2011)	508	33.6 (25.1–43.4)	—	<LOD	<LOD	0.99 (0.84–1.1)	1.3[E] (0.74–1.9)
3 (2012–2013)	510	40.7 (31.5–50.6)	—	<LOD	<LOD	0.99 (0.75–1.2)	1.5 (1.0–2.0)
4 (2014–2015)	506	37.3 (29.7–45.6)	—	<LOD	<LOD	0.98 (0.82–1.1)	1.1 (0.87–1.4)
5 (2016–2017)	515	88.7 (73.6–95.7)	0.33 (0.27–0.40)	<LOD	0.36 (0.31–0.40)	0.71 (0.59–0.82)	0.97[E] (0.59–1.4)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	34.3 (25.2–44.6)	—	<LOD	<LOD	F	1.8[E] (0.89–2.8)
3 (2012–2013)	355	32.1 (24.2–41.1)	—	<LOD	<LOD	0.97 (0.73–1.2)	1.3 (0.87–1.8)
4 (2014–2015)	362	38.1 (32.2–44.4)	—	<LOD	<LOD	1.1 (0.96–1.2)	F
5 (2016–2017)	357	77.1 (58.5–89.0)	0.33[E] (0.22–0.51)	<LOD	0.34 (0.26–0.42)	1.1[E] (0.46–1.6)	1.5[E] (0.83–2.1)
40–59 years							
2 (2009–2011)	355	27.8 (20.7–36.3)	—	<LOD	<LOD	1.2[E] (0.55–1.8)	1.9 (1.4–2.4)
3 (2012–2013)	312	20.9 (14.9–28.5)	—	<LOD	<LOD	1.3 (0.92–1.6)	1.7 (1.3–2.0)
4 (2014–2015)	312	28.4 (20.6–37.8)	—	<LOD	<LOD	1.3 (<LOD–1.7)	1.5 (<LOD–1.9)
5 (2016–2017)	345	85.4 (69.3–93.8)	0.35 (0.27–0.45)	<LOD	0.33 (0.26–0.40)	0.99 (0.71–1.3)	1.4[E] (0.62–2.1)
60–79 years							
2 (2009–2011)	288	19.2[E] (11.8–29.7)	—	<LOD	<LOD	1.7 (1.3–2.2)	1.9 (1.6–2.2)
3 (2012–2013)	352	23.9 (18.4–30.5)	—	<LOD	<LOD	1.4[E] (0.87–1.9)	2.1[E] (1.3–2.9)
4 (2014–2015)	361	21.7 (14.9–30.4)	—	<LOD	<LOD	1.0 (0.75–1.3)	1.3 (1.1–1.6)
5 (2016–2017)	347	77.2 (64.8–86.2)	0.33 (0.26–0.41)	<LOD	0.32 (0.25–0.40)	0.75 (0.58–0.93)	1.1 (0.69–1.4)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 8.2.9

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	95.5 (93.4–96.9)	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	95.7 (93.7–97.1)	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	95.7 (93.5–97.2)	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)
5 (2016–2017)	2615	99.6 (96.3–100)	3.2 (2.6–4.1)	0.98 (0.75–1.2)	3.1 (2.5–3.7)	10 (7.1–14)	15[E] (7.6–22)
Males, 3–79 years							
2 (2009–2011)	1271	96.0 (92.7–97.8)	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	96.4 (92.2–98.3)	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	94.7 (90.4–97.1)	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)
5 (2016–2017)	1299	99.8 (98.2–100)	3.2 (2.6–3.9)	0.94 (0.62–1.3)	3.1 (2.5–3.7)	11 (7.8–15)	15[E] (8.6–22)
Females, 3–79 years							
2 (2009–2011)	1267	95.0 (91.5–97.1)	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	95.1 (92.3–96.9)	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	96.7 (94.2–98.2)	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)
5 (2016–2017)	1316	99.4 (94.2–99.9)	3.3 (2.5–4.3)	0.98 (0.78–1.2)	3.2 (2.5–3.9)	9.9E (5.5–14)	F
3–5 years							
2 (2009–2011)	516	97.5 (95.7–98.6)	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	95.2 (91.4–97.4)	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	97.9 (93.9–99.3)	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)
5 (2016–2017)	535	97.6 (82.6–99.7)	3.4 (2.7–4.3)	1.0[E] (0.58–1.5)	3.6 (3.2–4.0)	12 (9.0–14)	20[E] (9.3–32)
6–11 years							
2 (2009–2011)	511	98.4 (97.4–99.0)	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	96.4 (91.1–98.6)	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	97.9 (95.1–99.1)	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)
5 (2016–2017)	513	99.7 (98.4–100)	3.5 (3.2–3.8)	1.3 (1.1–1.5)	3.5 (3.0–3.9)	7.6 (6.9–8.3)	10 (7.8–13)
12–19 years							
2 (2009–2011)	510	97.2 (92.8–99.0)	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	97.2 (93.1–98.9)	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	98.0 (95.3–99.2)	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)
5 (2016–2017)	517	99.4 (94.1–99.9)	3.3 (2.8–3.9)	1.2 (0.77–1.5)	3.4 (2.8–3.9)	8.3E (5.2–11)	14 (9.8–18)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	94.9 (89.7–97.5)	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	94.0 (83.4–98.0)	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	94.3 (86.1–97.7)	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)
5 (2016–2017)	357	99.9 (99.2–100)	3.3[E] (2.3–4.8)	1.0[E] (0.44–1.6)	3.0 (2.2–3.9)	13[E] (4.9–20)	F
40–59 years							
2 (2009–2011)	357	94.3 (88.0–97.3)	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	95.4 (90.9–97.8)	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	94.8 (91.3–97.0)	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)
5 (2016–2017)	345	99.6 (92.7–100)	3.4 (2.5–4.5)	0.88 (0.62–1.1)	3.7 (2.6–4.8)	11 (7.5–14)	F
60–79 years							
2 (2009–2011)	289	96.1 (92.7–98.0)	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	97.4 (94.8–98.7)	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	96.9 (93.9–98.4)	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)
5 (2016–2017)	348	99.6 (96.0–100)	2.9 (2.3–3.6)	0.84 (0.61–1.1)	2.5 (2.0–3.1)	10 (6.9–14)	15 (11–19)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2, 3, 4, and 5 are 0.8, 0.75, 0.75, and 0.14 µg As/L, respectively.

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 are too unreliable to be published.

Table 8.2.10

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	95.5 (93.4–96.9)	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	95.7 (93.7–97.1)	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	95.7 (93.5–97.2)	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)
5 (2016–2017)	2605	99.6 (96.3–100)	3.2 (2.6–3.9)	1.2 (0.98–1.3)	2.6 (2.1–3.2)	9.6 (7.0–12)	15 (10–20)
Males, 3–79 years							
2 (2009–2011)	1267	96.0 (92.7–97.8)	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	96.4 (92.2–98.3)	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	94.7 (90.4–97.1)	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)
5 (2016–2017)	1296	99.8 (98.2–100)	2.8 (2.3–3.3)	1.0 (0.85–1.2)	2.4 (2.1–2.7)	9.2 (6.8–12)	14 (11–17)
Females, 3–79 years							
2 (2009–2011)	1261	95.0 (91.5–97.1)	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	95.1 (92.3–96.9)	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	96.7 (94.2–98.2)	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)
5 (2016–2017)	1309	99.4 (94.2–99.9)	3.7 (2.9–4.6)	1.4 (1.1–1.6)	3.3 (2.5–4.1)	9.9[E] (5.7–14)	F
3–5 years							
2 (2009–2011)	515	97.5 (95.7–98.6)	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	95.2 (91.4–97.4)	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	97.9 (93.9–99.3)	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)
5 (2016–2017)	532	97.6 (82.6–99.7)	5.8 (4.5–7.4)	2.8 (2.2–3.4)	5.4 (4.2–6.6)	14[E] (8.3–20)	23 (15–31)
6–11 years							
2 (2009–2011)	509	98.4 (97.4–99.0)	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	96.4 (91.1–98.6)	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	97.9 (95.1–99.1)	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)
5 (2016–2017)	509	99.7 (98.4–100)	4.0 (3.6–4.5)	1.8 (1.5–2.2)	3.8 (3.4–4.1)	9.0 (7.4–11)	11 (7.6–15)
12–19 years							
2 (2009–2011)	508	97.2 (92.8–99.0)	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	97.2 (93.1–98.9)	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	98.0 (95.3–99.2)	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)
5 (2016–2017)	515	99.4 (94.1–99.9)	2.5 (2.2–2.9)	1.1 (0.88–1.3)	2.2 (1.9–2.5)	6.2 (4.1–8.3)	10[E] (5.6–15)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	94.9 (89.7–97.5)	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	94.0 (83.4–98.0)	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	94.3 (86.1–97.7)	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)
5 (2016–2017)	357	99.9 (99.2–100)	3.1[E] (2.1–4.5)	1.0 (0.83–1.2)	2.5[E] (1.5–3.4)	9.9[E] (3.6–16)	F
40–59 years							
2 (2009–2011)	355	94.3 (88.0–97.3)	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	95.4 (90.9–97.8)	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	94.8 (91.3–97.0)	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)
5 (2016–2017)	345	99.6 (92.7–100)	3.1 (2.5–3.8)	1.2 (0.82–1.5)	2.4 (1.8–3.1)	9.4 (6.1–13)	15 (10–20)
60–79 years							
2 (2009–2011)	288	96.1 (92.7–98.0)	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	97.4 (94.8–98.7)	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	96.9 (93.9–98.4)	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)
5 (2016–2017)	347	99.6 (96.0–100)	3.4 (2.8–4.1)	1.3 (1.1–1.5)	3.0 (2.2–3.9)	9.4 (7.2–12)	12 (9.8–14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 8.2.11

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2538	59.6 (52.5–66.4)	—	<LOD	1.4[E] (<LOD–2.2)	28[E] (18–39)	48[E] (30–67)
3 (2012–2013)	2536	60.0 (54.8–65.0)	—	<LOD	1.4[E] (<LOD–2.1)	24[E] (11–36)	56 (37–75)
4 (2014–2015)	2564	56.6 (51.8–61.3)	—	<LOD	1.2[E] (<LOD–1.7)	28[E] (13–44)	49 (33–65)
5 (2016–2017)	2615	82.8 (73.6–89.3)	1.3[E] (0.80–2.1)	<LOD	1.3[E] (0.58–1.9)	29[E] (14–44)	56[E] (27–86)
Males, 3–79 years							
2 (2009–2011)	1271	61.4 (53.2–69.0)	—	<LOD	1.5[E] (<LOD–2.5)	29[E] (14–43)	F
3 (2012–2013)	1251	60.9 (53.1–68.2)	—	<LOD	1.4[E] (<LOD–2.0)	21[E] (13–29)	38 (25–51)
4 (2014–2015)	1273	60.1 (52.7–67.1)	—	<LOD	1.6[E] (<LOD–2.6)	33[E] (12–54)	44 (30–59)
5 (2016–2017)	1299	83.3 (74.0–89.8)	1.2[E] (0.75–1.8)	<LOD	1.2[E] (0.58–1.8)	18[E] (11–25)	F
Females, 3–79 years							
2 (2009–2011)	1267	57.8 (48.9–66.3)	—	<LOD	<LOD	28[E] (15–41)	49[E] (29–69)
3 (2012–2013)	1285	59.1 (50.7–67.1)	—	<LOD	1.5[E] (<LOD–2.6)	F	58[E] (33–83)
4 (2014–2015)	1291	53.1 (44.9–61.2)	—	<LOD	F	F	52[E] (18–86)
5 (2016–2017)	1316	82.3 (71.6–89.6)	1.4[E] (0.83–2.5)	<LOD	1.3[E] (0.53–2.2)	37[E] (19–56)	65[E] (23–110)
3–5 years							
2 (2009–2011)	516	42.7 (34.7–51.0)	—	<LOD	<LOD	F	34[E] (19–49)
3 (2012–2013)	500	35.8 (30.2–41.8)	—	<LOD	<LOD	12[E] (6.3–17)	F
4 (2014–2015)	512	35.8 (26.7–46.0)	—	<LOD	<LOD	16[E] (5.4–26)	F
5 (2016–2017)	535	66.6 (55.0–76.5)	0.41[E] (0.25–0.69)	<LOD	0.18[E] (<LOD–0.30)	F	F
6–11 years							
2 (2009–2011)	511	40.7 (33.8–47.9)	—	<LOD	<LOD	F	F
3 (2012–2013)	507	44.2 (34.1–54.8)	—	<LOD	<LOD	F	27[E] (14–39)
4 (2014–2015)	512	37.1 (30.1–44.8)	—	<LOD	<LOD	15[E] (5.2–25)	39[E] (13–64)
5 (2016–2017)	513	64.9 (53.6–74.8)	0.38 (0.27–0.52)	<LOD	0.18[E] (<LOD–0.28)	13[E] (7.3–19)	F
12–19 years							
2 (2009–2011)	510	42.3 (34.1–51.0)	—	<LOD	<LOD	12[E] (4.5–19)	38[E] (16–59)
3 (2012–2013)	510	48.6 (39.0–58.3)	—	<LOD	<LOD	16[E] (7.2–24)	37[E] (17–56)
4 (2014–2015)	506	50.3 (41.1–59.5)	—	<LOD	0.75[E] (<LOD–1.2)	16[E] (9.4–22)	26[E] (13–39)
5 (2016–2017)	517	72.2 (57.8–83.1)	0.50[E] (0.31–0.80)	<LOD	F	10[E] (6.1–14)	24[E] (11–36)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	355	62.6 (51.2–72.8)	2.3[E] (1.5–3.6)	<LOD	F	33[E] (15–52)	68[E] (20–110)
3 (2012–2013)	355	59.3 (50.1–67.9)	—	<LOD	F	19[E] (11–28)	35[E] (12–58)
4 (2014–2015)	361	57.1 (47.3–66.4)	1.9 (1.5–2.5)	<LOD	1.5[E] (<LOD–2.4)	32[E] (17–47)	46[E] (24–67)
5 (2016–2017)	357	81.8 (70.0–89.7)	1.4[E] (0.72–2.8)	<LOD	1.5[E] (0.55–2.5)	F	F
40–59 years							
2 (2009–2011)	357	62.1 (51.8–71.4)	1.8 (1.4–2.4)	<LOD	1.4[E] (<LOD–2.5)	F	35[E] (19–52)
3 (2012–2013)	312	63.2 (52.9–72.5)	2.2[E] (1.3–3.8)	<LOD	F	F	57[E] (30–84)
4 (2014–2015)	312	58.2 (48.7–67.2)	1.8 (1.3–2.6)	<LOD	1.3[E] (<LOD–1.9)	F	37[E] (18–56)
5 (2016–2017)	345	88.2 (74.9–95.0)	2.0[E] (1.1–3.7)	<LOD	F	37 (24–50)	59[E] (30–87)
60–79 years							
2 (2009–2011)	289	71.1 (60.1–80.1)	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	70.3 (60.8–78.3)	2.6[E] (1.8–3.8)	<LOD	2.1[E] (0.86–3.4)	F	67[E] (29–100)
4 (2014–2015)	361	67.0 (57.8–75.1)	2.8[E] (1.7–4.7)	<LOD	2.5[E] (0.91–4.0)	F	88[E] (49–130)
5 (2016–2017)	348	89.7 (82.9–93.9)	1.7[E] (1.1–2.8)	<LOD	1.7[E] (0.59–2.8)	19[E] (6.5–32)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2, 3, 4, and 5 are 0.8, 0.75, 0.75, and 0.10 µg As/L, respectively.

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 are too unreliable to be published.

Table 8.2.12

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2528	59.6 (52.5–66.4)	—	<LOD	1.5[E] (<LOD–2.5)	22 (16–28)	44[E] (18–71)
3 (2012–2013)	2535	60.0 (54.8–65.0)	—	<LOD	1.6 (<LOD–2.1)	25[E] (12–39)	44[E] (24–63)
4 (2014–2015)	2563	56.6 (51.8–61.3)	—	<LOD	1.2 (<LOD–1.5)	23[E] (12–34)	46[E] (27–65)
5 (2016–2017)	2605	82.8 (73.6–89.3)	1.3[E] (0.79–2.1)	<LOD	1.1[E] (0.43–1.8)	23[E] (9.2–37)	62[E] (21–100)
Males, 3–79 years							
2 (2009–2011)	1267	61.4 (53.2–69.0)	—	<LOD	F	18[E] (9.4–27)	F
3 (2012–2013)	1251	60.9 (53.1–68.2)	—	<LOD	1.2 (<LOD–1.6)	16[E] (7.3–24)	34 (25–43)
4 (2014–2015)	1272	60.1 (52.7–67.1)	—	<LOD	1.3[E] (<LOD–1.8)	20[E] (9.8–30)	37[E] (19–55)
5 (2016–2017)	1296	83.3 (74.0–89.8)	1.0[E] (0.65–1.6)	<LOD	1.0[E] (0.50–1.5)	16 (12–21)	F
Females, 3–79 years							
2 (2009–2011)	1261	57.8 (48.9–66.3)	—	<LOD	<LOD	25 (19–32)	61[E] (20–100)
3 (2012–2013)	1284	59.1 (50.7–67.1)	—	<LOD	2.1[E] (<LOD–3.3)	33[E] (9.5–56)	F
4 (2014–2015)	1291	53.1 (44.9–61.2)	—	<LOD	1.1 (<LOD–1.4)	F	62[E] (36–89)
5 (2016–2017)	1309	82.3 (71.6–89.6)	1.6[E] (0.93–2.8)	<LOD	F	F	92[E] (35–150)
3–5 years							
2 (2009–2011)	515	42.7 (34.7–51.0)	—	<LOD	<LOD	F	F
3 (2012–2013)	499	35.8 (30.2–41.8)	—	<LOD	<LOD	21[E] (11–31)	F
4 (2014–2015)	512	35.8 (26.7–46.0)	—	<LOD	<LOD	26[E] (14–38)	57[E] (15–98)
5 (2016–2017)	532	66.6 (55.0–76.5)	0.69[E] (0.40–1.2)	<LOD	0.33[E] (<LOD–0.56)	F	F
6–11 years							
2 (2009–2011)	509	40.7 (33.8–47.9)	—	<LOD	<LOD	F	F
3 (2012–2013)	507	44.2 (34.1–54.8)	—	<LOD	<LOD	F	40[E] (12–69)
4 (2014–2015)	511	37.1 (30.1–44.8)	—	<LOD	<LOD	17[E] (8.4–27)	F
5 (2016–2017)	509	64.9 (53.6–74.8)	0.44 (0.31–0.64)	<LOD	0.23[E] (<LOD–0.36)	F	F
12–19 years							
2 (2009–2011)	508	42.3 (34.1–51.0)	—	<LOD	<LOD	9.3[E] (4.0–15)	24[E] (10–38)
3 (2012–2013)	510	48.6 (39.0–58.3)	—	<LOD	<LOD	10[E] (3.8–17)	F
4 (2014–2015)	506	50.3 (41.1–59.5)	—	<LOD	0.72[E] (<LOD–1.0)	9.9[E] (5.4–14)	F
5 (2016–2017)	515	72.2 (57.8–83.1)	0.38[E] (0.24–0.62)	<LOD	0.25[E] (<LOD–0.40)	8.0[E] (4.0–12)	17[E] (5.6–28)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
2 (2009–2011)	353	62.6 (51.2–72.8)	1.9[E] (1.2–2.8)	<LOD	F	22[E] (7.8–37)	F
3 (2012–2013)	355	59.3 (50.1–67.9)	—	<LOD	1.4[E] (<LOD–1.9)	12[E] (5.5–19)	21[E] (9.8–32)
4 (2014–2015)	361	57.1 (47.3–66.4)	1.6 (1.2–2.1)	<LOD	1.1[E] (<LOD–1.6)	20 (13–27)	29[E] (7.7–50)
5 (2016–2017)	357	81.8 (70.0–89.7)	F	<LOD	F	F	F
40–59 years							
2 (2009–2011)	355	62.1 (51.8–71.4)	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	63.2 (52.9–72.5)	2.6[E] (1.6–4.4)	<LOD	F	33[E] (14–52)	F
4 (2014–2015)	312	58.2 (48.7–67.2)	1.7 (1.2–2.4)	<LOD	1.1[E] (<LOD–1.5)	F	F
5 (2016–2017)	345	88.2 (74.9–95.0)	1.8[E] (1.0–3.2)	<LOD	F	F	76[E] (38–110)
60–79 years							
2 (2009–2011)	288	71.1 (60.1–80.1)	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	70.3 (60.8–78.3)	2.9[E] (1.9–4.4)	<LOD	F	35[E] (<LOD–57)	F
4 (2014–2015)	361	67.0 (57.8–75.1)	2.8[E] (1.7–4.4)	<LOD	2.2[E] (0.93–3.5)	F	F
5 (2016–2017)	347	89.7 (82.9–93.9)	2.0[E] (1.3–3.1)	<LOD	2.5[E] (1.5–3.5)	24[E] (9.9–39)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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8.3 BORON

Boron (CASRN 7440-42-8) is a naturally occurring element that is present in the Earth's crust at an average concentration of 0.0008% (ATSDR, 2010). It is a metalloid exhibiting properties intermediate between those of typical metals and nonmetals. Elemental boron exists in a crystalline or amorphous form; however, it is never found in nature in the free elemental form (Ince et al., 2017; WHO, 2009; ATSDR, 2010). Boron is always found in the environment combined with oxygen as borate compounds, including boric acid, sodium tetraborate (or Borax) and boron oxide (ATSDR, 2010).

Boron is widely distributed in nature and can be released by both natural and anthropogenic processes. Volcanic emissions, sea salt aerosol, soil dust, plant aerosols, and weathering of soil and rocks containing borates are important sources of natural borates

released into the environment (Canada, 2016; Health Canada, 1991; Health Canada, 2016). Anthropogenic sources include the manufacture, import and use of boric acid, its salts and its precursors in manufactured products and applications such as fibreglass insulation, oil and gas extraction, fertilizers, cellulose insulation, gypsum boards, engineered wood products, pulp and paper manufacturing, rubber manufacturing, chemical manufacturing, metallurgical applications, and cleaning products. Other anthropogenic sources include the incidental production and subsequent release of boric acid as a result of activities such as coal-fired power generation, metal mining, smelting and refining, coal mining, oil sands extraction and processing, oil and gas extraction, wastewater treatment, and waste disposal (Environment and Climate Change Canada and Health Canada, 2016).

Exposure to boron occurs primarily through the ingestion of food (mainly fruit and vegetables) and water (ATSDR, 2010; Canada, 2016). The range of boron concentrations in these media varies widely across the world (WHO, 2009; Canada, 2016). Boron is generally not present at significant levels in air because of the low volatility of borate compounds (WHO, 2009). Exposure to borates can also occur through consumer products such as cosmetics, arts and craft materials, toys, natural health products, cleaning products, and swimming pool products, as well as through the use of household pest control products (Canada, 2016; Environment and Climate Change Canada and Health Canada, 2016; Health Canada, 2016).

Inorganic borates are readily absorbed across mucous membranes; gastrointestinal absorption has been estimated at approximately 81% to 92% (ATSDR, 2010; Devirian and Volpe, 2003; Dourson et al., 1998). Significant absorption can also occur through inhalation (Ince et al., 2017). Dermal absorption is generally low in healthy skin (~0.5% to 10%), but can be significantly increased in damaged skin (Environment and Climate Change Canada and Health Canada, 2016); Ince et al., 2017). Boron is mostly present in the body as boric acid; borates are rapidly converted to boric acid in the mucosal layer before rapid absorption and distribution (Devirian and Volpe, 2003). Animal studies show that absorbed boric acid is equally distributed to liver, kidneys, genital tissue, brain, adrenals, muscles, and blood (Ince et al., 2017). Boron can also cross the placental barrier; some animal toxicology studies have reported accumulation in bone over long-term oral exposure (Ince et al., 2017). Boric acid is not further metabolized in the bodies of humans or animals because substantial energy is required to break the oxygen and boron bond (Ince et al., 2017). Consequently, orally absorbed boric acid is rapidly eliminated unchanged, mainly in urine, with a half-life of less than 24 hours (Ince et al., 2017). A small amount is found in feces (2%) and a smaller amount in sweat, saliva, bile, and breath (Devirian and Volpe, 2003; Health Canada, 1991). Measurement of inorganic borates in urine reflects boron intake, and is an indicator of human exposure (Devirian and Volpe, 2003). Boron in blood can also be used to estimate human exposure (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016).

Although boron plays important roles in human health — being involved in functions such as bone growth, regulation of sex hormones, and anti-inflammatory, and anti-cancer effects — it is not considered an essential trace element in humans at this time (Devirian and Volpe, 2003; IOM, 2001; Pizzorno, 2015). The acute oral toxicity of boron is generally low (Hubbard, 1998). Acute toxicity is more likely in children, the elderly, and people with kidney problems. Symptoms may include vomiting, nausea, digestive disorders, skin flushing, ataxia, headache, seizure, depression, vascular collapse, and death (Devirian and Volpe, 2003; Environment and Climate Change Canada and Health Canada, 2016; Health Canada, 1991; Ince et al., 2017). Acute inhalation toxicity marked by irritation of the respiratory tract and eyes has been reported in boron production workers following occupational exposure to borate dusts (ATSDR, 2010).

Chronic exposure to boron has been associated with digestive problems (nausea, vomiting, and loss of appetite) as well as nervous system irritation and convulsion. Subchronic and chronic experimental animal studies suggest that high-dose exposure to boron compounds leads to reproductive and developmental toxicity, particularly affecting the male reproductive system (Devirian and Volpe, 2003; Health Canada, 1991; Hubbard, 1998; Ince et al., 2017). There is no conclusive evidence for mutagenic or genotoxic effects of boron (Hubbard, 1998; Ince et al., 2017), and consequently boron is not classified as a carcinogen by the International Agency for Research on Cancer (IARC) or other agencies (ATSDR, 2010).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether boric acid, its salts, and its precursors present or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2016). The assessment proposes to conclude that boric acid, its salts, and its precursors are toxic under CEPA 1999 as they are considered harmful to the environment and human health (Environment and Climate Change Canada and Health Canada, 2016).

The sale and use of pesticides are regulated in Canada by the Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002). Based on a re-evaluation by the PMRA in 2016, most pesticides containing boric acid and its salts continue to be approved, as they pose no unacceptable risk for humans or the environment when they are used according to revised label directions (Health Canada, 2016). However, a number of pesticide products that contain boric acid for use in and around the home that are in powder form or in other formulations carrying a potential risk for overexposure will be phased out of the marketplace (Health Canada, 2016). Boric acid and its salts are identified as being prohibited on the List of Prohibited and Restricted Cosmetics Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018). Canada's Food and Drug Regulations specify that a cautionary statement must appear on the label of drug products containing boric acid or sodium borate to prevent administration to children under three years of age (Canada, 1985). Toy Regulations under the *Canada Consumer Product Safety Act* prohibit the presence of boron in children's toys (Canada, 2016).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has developed a Canadian drinking water quality guideline that establishes a maximum acceptable concentration for boron in drinking water (Health Canada, 1991). The guideline was developed based on reproductive effects in animal toxicology studies, and takes into account the treatment technology and analytical methods available to reduce boron in water (Health Canada, 1991).

Boron was analyzed in the urine of Canadian Health Measures Survey (CHMS) cycle 5 (2016–2017) participants aged 3–79 years. Data from this cycle are presented in urine as both µg/L and µg/g creatinine. Finding a measurable amount of boron in urine is an indicator of exposure to boron and does not necessarily mean that an adverse health effect will occur.

Table 8.3.1

Boron — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2715	98.2 (95.1–99.4)	960 (880–1000)	360 (310–410)	990 (940–1000)	2300 (2100–2400)	2900 (2500–3200)
Males, 3–79 years							
5 (2016–2017)	1351	98.0 (93.4–99.4)	920 (840–1000)	360 (300–430)	960 (910–1000)	2300 (2000–2600)	2900 (2200–3600)
Females, 3–79 years							
5 (2016–2017)	1364	98.5 (93.4–99.7)	990 (870–1100)	350 (290–410)	1100 (920–1200)	2200 (2100–2400)	2800 (2100–3500)
3–5 years							
5 (2016–2017)	553	99.8 (87.1–100)	1300 (1100–1500)	490 (310–670)	1200 (950–1500)	3000 (2700–3300)	3400 (3100–3700)
6–11 years							
5 (2016–2017)	538	99.2 (97.8–99.7)	1100 (1000–1200)	380 (290–480)	1200 (970–1300)	2500 (2100–2800)	3000 (2200–3900)
12–19 years							
5 (2016–2017)	534	98.5 (94.9–99.6)	980 (890–1100)	350 (260–430)	1000 (970–1000)	2200 (1800–2500)	2600 (2400–2900)
20–39 years							
5 (2016–2017)	375	97.9 (87.2–99.7)	860 (680–1100)	330 (210–440)	860 (630–1100)	2200 (1800–2700)	2500 (1800–3200)
40–59 years							
5 (2016–2017)	360	97.8 (90.3–99.5)	930 (780–1100)	390 (260–520)	970 (760–1200)	1900 (1500–2300)	2600[E] (1200–4000)
60–79 years							
5 (2016–2017)	355	98.5 (95.3–99.5)	1000 (960–1100)	370 (320–410)	1100 (920–1300)	2400 (1700–3100)	3500 (2800–4100)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 160 µg/L.

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 8.3.2

Boron (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2691	98.2 (95.1–99.4)	930 (860–1000)	420 (360–480)	880 (800–960)	2300 (1900–2700)	2900 (2600–3300)
Males, 3–79 years							
5 (2016–2017)	1341	98.0 (93.4–99.4)	800 (740–860)	350 (280–430)	760 (700–830)	1800 (1500–2200)	2800 (2100–3500)
Females, 3–79 years							
5 (2016–2017)	1350	98.5 (93.4–99.7)	1100 (960–1200)	510 (450–570)	1000 (890–1100)	2500 (2100–3000)	3000 (2100–3900)
3–5 years							
5 (2016–2017)	545	99.8 (87.1–100)	2200 (1900–2400)	1000 (820–1200)	2200 (1900–2500)	4100 (3500–4700)	4800 (4200–5400)
6–11 years							
5 (2016–2017)	531	99.2 (97.8–99.7)	1300 (1200–1400)	630 (560–690)	1200 (1100–1400)	2500 (1900–3100)	3300 (2600–4000)
12–19 years							
5 (2016–2017)	530	98.5 (94.9–99.6)	750 (690–810)	370 (310–430)	760 (690–830)	1400 (1200–1500)	1700 (1500–1800)
20–39 years							
5 (2016–2017)	372	97.9 (87.2–99.7)	770 (690–870)	330 (230–430)	720 (600–840)	1800 (1300–2300)	2200[E] (1400–3100)
40–59 years							
5 (2016–2017)	359	97.8 (90.3–99.5)	860 (740–990)	430 (340–520)	780 (660–910)	1700[E] (870–2500)	2600 (1700–3500)
60–79 years							
5 (2016–2017)	354	98.5 (95.3–99.5)	1200 (1100–1300)	540 (490–580)	1100 (980–1300)	2800 (2300–3400)	3400 (2800–4000)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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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8.4 CADMIUM

Cadmium (CASRN 7440-43-9) is among the least abundant metals in the Earth's crust, with 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 (USGS, 2018). Common forms include soluble (e.g., cadmium chloride, cadmium sulphate) and insoluble (e.g., cadmium metal and its oxides) species that may also be found as particulate matter in the atmosphere (ATSDR, 2012; CCME, 1999).

Cadmium is released into 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, 2018). 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 applications. It is frequently found as an impurity in galvanized pipes and as a constituent of solders used in plumbing and distribution systems. It can leach into drinking water (Health Canada, 2019; 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).

Health Canada released dietary exposure estimates for cadmium using data collected as part of surveys conducted by the Canadian Food Inspection Agency and Health Canada, including the Total Diet Study (Health Canada, 2018a). Ambient air is usually a minor source of exposure, with intakes estimated to be two to three orders of magnitude lower than for food, although cadmium compounds are more readily absorbed following inhalation than through ingestion (Friberg, 1985). Other potential sources of exposure include ingestion of drinking water, soil, or dust (ATSDR, 2012; Health Canada, 2019; 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 majority of dietary cadmium is not absorbed; average gastrointestinal absorption is estimated at 5% in adult men and 10% or higher in adult 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 kidneys (ATSDR, 2012). The kidney is considered the critical organ that exhibits the first adverse effects after either oral or inhalation exposure, based on observations in both human epidemiology and animal toxicity studies (EFSA, 2009; FAO/WHO, 2011; ATSDR, 2012).

Inhaled cadmium and its compounds have been classified as probably carcinogenic to humans by Environment Canada and Health Canada (Environment Canada and Health Canada, 1994). More recently, the International Agency for Research on Cancer (IARC) 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 may be harmful to the environment and may constitute a danger to human life or health in Canada based on their 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).

Cadmium is included in the list of trace elements analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2016). 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 to which Canadians in different age-sex groups are exposed through the food supply. On the basis of data collected, as part the Total Diet Study surveys and surveys conducted by the Canadian Food Inspection Agency, Health Canada has concluded that dietary exposure to cadmium does not represent a health concern for the general population of Canadians (Health Canada, 2018a). 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 guideline limit for total cadmium in children's jewellery was finalized and published in 2018 as part of the Children's Jewellery Regulations under the *Canadian Consumer Product Safety Act* (Canada, 2018). Cadmium and its compounds are identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018b). 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, 2017; Health Canada, 2019).

In a biomonitoring study carried out in the region of Québec City with 500 participants aged 18–65 years, the geometric means (GMs) for cadmium in urine and whole blood were 4.79 nmol/L (0.54 µg/L) and 6.15 nmol/L (0.69 µg/L), respectively (INSPQ, 2004). The First Nations Biomonitoring Initiative (FNBI) was a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprised 13 randomly selected First Nations communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the GM 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) 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 GM 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 recruited from 10 sites across Canada between 2008 and 2011 (Arbuckle et al., 2013). In the MIREC study of 1,938 participants in their first trimester of pregnancy, the GM 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 (CHMS) participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). Data from these cycles are presented in blood as µg/L. Cadmium was analyzed in the urine of CHMS participants aged 6–79 years in cycle 1 (2007-2009), and 3–79 years in cycle 2 (2009-2011) and cycle 5 (2016–2017). Data from these cycles are presented in urine as both µg/L and µg/g creatinine. Finding a measurable amount of cadmium in blood or urine is an indicator of exposure to cadmium, and does not necessarily mean that an adverse health effect will occur. Cadmium was also analyzed in hair from CHMS participants 20–59 years old in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Table 8.4.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	97.1 (94.0–98.6)	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	94.4 (92.4–95.9)	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	94.9 (93.6–96.0)	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)
5 (2016–2017)	4517	87.9 (84.0–90.9)	0.28 (0.25–0.30)	<LOD	0.23 (0.21–0.26)	1.7 (1.1–2.3)	2.9 (2.4–3.3)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	97.0 (93.8–98.5)	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	92.6 (90.1–94.4)	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	93.7 (91.5–95.4)	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)
5 (2016–2017)	2257	84.7 (79.1–89.0)	0.26 (0.23–0.30)	<LOD	0.19 (0.18–0.20)	2.4[E] (1.5–3.3)	3.2 (2.7–3.7)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	97.2 (93.4–98.8)	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	96.3 (94.5–97.5)	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	96.2 (95.3–96.9)	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)
5 (2016–2017)	2260	91.1 (88.0–93.4)	0.29 (0.26–0.32)	<LOD	0.27 (0.24–0.29)	1.1[E] (0.59–1.6)	2.3[E] (1.4–3.2)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	87.9 (78.4–93.6)	0.073 (0.065–0.081)	<LOD	0.078 (0.069–0.087)	0.099 (0.098–0.10)	F
3 (2012–2013)	471	60.0 (49.8–69.4)	—	<LOD	0.091 (<LOD–0.11)	0.16 (0.11–0.20)	0.18[E] (<LOD–0.29)
4 (2014–2015)	479	65.9 (57.7–73.3)	0.082 (<LOD–0.091)	<LOD	0.093 (0.084–0.10)	0.16 (0.14–0.18)	0.19 (0.15–0.24)
5 (2016–2017)	473	32.4 (23.4–43.0)	—	<LOD	<LOD	0.13 (<LOD–0.16)	0.16 (0.13–0.19)
6–11 years							
1 (2007–2009)	910	91.3 (87.6–94.0)	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	89.1 (82.9–93.3)	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	77.1 (67.6–84.5)	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	76.7 (70.9–81.7)	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)
5 (2016–2017)	511	43.7 (32.8–55.3)	—	<LOD	<LOD	0.16 (0.13–0.19)	0.19 (0.14–0.24)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	945	97.0 (95.1–98.1)	0.16 (0.13–0.20)	0.066 (0.045–0.086)	F	F	F
2 (2009–2011)	997	95.0 (89.1–97.8)	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	88.5 (81.6–93.0)	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	88.8 (83.8–92.5)	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)
5 (2016–2017)	521	63.9 (53.5–73.2)	0.11 (0.098–0.13)	<LOD	0.11 (<LOD–0.12)	0.24[E] (0.15–0.34)	F
20–39 years							
1 (2007–2009)	1165	98.3 (95.4–99.4)	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	97.1 (89.8–99.2)	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	95.2 (91.9–97.2)	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	96.7 (93.9–98.3)	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)
5 (2016–2017)	1038	88.3 (81.3–92.9)	0.28 (0.23–0.33)	<LOD	0.19 (0.16–0.22)	2.3[E] (1.1–3.5)	3.1 (2.1–4.0)
40–59 years							
1 (2007–2009)	1220	99.6 (98.0–99.9)	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	98.6 (94.5–99.6)	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	99.1 (97.9–99.6)	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	98.9 (97.8–99.4)	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)
5 (2016–2017)	990	95.2 (89.2–98.0)	0.35 (0.31–0.38)	0.11 (<LOD–0.14)	0.27 (0.24–0.29)	2.0[E] (1.2–2.9)	2.8 (2.3–3.2)
60–79 years							
1 (2007–2009)	1079	99.2 (95.5–99.9)	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	99.7 (98.3–99.9)	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	100	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	99.1 (97.4–99.7)	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)
5 (2016–2017)	984	97.8 (93.5–99.3)	0.39 (0.34–0.44)	0.15 (0.13–0.17)	0.32 (0.28–0.36)	1.2[E] (0.70–1.7)	2.7 (1.7–3.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 0.04, 0.04, 0.080, 0.080, and 0.097 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 8.4.2

Cadmium — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)[c]	6311	94.4 (92.5–95.9)	0.38 (0.34–0.43)	0.092 (0.088–0.096)	0.41 (0.35–0.47)	1.2 (1.1–1.4)	1.8 (1.7–2.0)
5 (2016–2017)	2715	72.0 (65.3–77.9)	—	<LOD	0.16 (0.14–0.19)	0.91 (0.71–1.1)	1.4 (1.0–1.8)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	3036	94.2 (91.5–96.1)	0.38 (0.33–0.44)	0.092 (0.084–0.10)	0.41 (0.34–0.47)	1.2 (1.0–1.4)	1.6 (1.4–1.8)
5 (2016–2017)	1351	68.3 (58.9–76.4)	—	<LOD	0.15 (0.11–0.18)	0.67 (0.48–0.85)	0.99 (0.77–1.2)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	3275	94.6 (92.7–96.1)	0.39 (0.35–0.42)	0.092 (0.089–0.095)	0.41 (0.35–0.48)	1.3 (0.90–1.7)	2.0 (1.5–2.4)
5 (2016–2017)	1364	75.7 (70.3–80.3)	—	<LOD	0.19 (0.13–0.24)	1.1 (0.77–1.3)	1.5 (1.3–1.8)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	573	89.7 (82.1–94.3)	0.22 (0.18–0.28)	<LOD	0.26 (0.21–0.30)	0.62 (0.50–0.75)	F
5 (2016–2017)	553	19.6[E] (10.5–33.6)	—	<LOD	<LOD	0.12[E] (0.073–0.17)	0.17[E] (0.070–0.28)
6–11 years							
1 (2007–2009)[c]	1033	85.8 (80.3–90.0)	0.22 (0.18–0.25)	<LOD	0.25 (0.20–0.30)	0.58 (0.52–0.65)	0.72 (0.60–0.85)
2 (2009–2011)[c]	1062	91.8 (89.0–94.0)	0.24 (0.20–0.29)	0.077 (<LOD–0.095)	0.27 (0.22–0.32)	0.67 (0.48–0.86)	0.87 (0.65–1.1)
5 (2016–2017)	538	27.4[E] (18.0–39.5)	—	<LOD	<LOD	0.16 (0.11–0.20)	0.19 (0.16–0.22)
12–19 years							
1 (2007–2009)[c]	983	89.3 (85.2–92.4)	0.27 (0.23–0.31)	<LOD	0.32 (0.28–0.36)	0.68 (0.58–0.78)	0.89 (0.66–1.1)
2 (2009–2011)[c]	1041	94.2 (89.7–96.8)	0.26 (0.21–0.32)	0.090 (<LOD–0.11)	0.30 (0.24–0.36)	0.68 (0.56–0.79)	0.81 (0.67–0.94)
5 (2016–2017)	534	41.4 (27.7–56.6)	—	<LOD	<LOD	0.20 (0.16–0.23)	0.26 (0.20–0.32)
20–39 years							
1 (2007–2009)[c]	1169	86.5 (82.0–90.0)	0.27 (0.25–0.31)	<LOD	0.31 (0.27–0.36)	0.92 (0.83–1.0)	1.1 (0.99–1.3)
2 (2009–2011)[c]	1321	92.8 (88.3–95.6)	0.33 (0.28–0.38)	0.088[E] (<LOD–0.12)	0.36 (0.30–0.43)	0.99 (0.88–1.1)	1.2 (0.99–1.4)
5 (2016–2017)	375	67.9 (56.4–77.6)	0.13 (0.10–0.16)	<LOD	0.12[E] (0.077–0.17)	0.64 (0.44–0.83)	0.84[E] (0.32–1.4)
40–59 years							
1 (2007–2009)[c]	1223	92.4 (90.4–94.1)	0.42 (0.38–0.46)	0.093 (<LOD–0.10)	0.45 (0.40–0.51)	1.5 (1.3–1.6)	2.1 (1.7–2.4)
2 (2009–2011)[c]	1228	94.9 (92.2–96.7)	0.49 (0.43–0.56)	0.096 (0.084–0.11)	0.53 (0.44–0.62)	1.7 (1.5–2.0)	2.5 (2.0–3.0)
5 (2016–2017)	360	87.8 (82.1–91.9)	0.25 (0.22–0.29)	<LOD	0.28 (0.22–0.34)	1.0 (0.78–1.3)	1.5 (1.3–1.7)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)[c]	1083	96.2 (93.2–97.9)	0.50 (0.44–0.56)	0.099 (<LOD–0.13)	0.51 (0.46–0.56)	1.6 (1.4–1.8)	2.2 (1.9–2.6)
2 (2009–2011)[c]	1086	98.5 (97.2–99.1)	0.53 (0.47–0.61)	0.098 (0.078–0.12)	0.57 (0.50–0.65)	1.7 (1.3–2.1)	2.5 (2.0–2.9)
5 (2016–2017)	355	92.2 (87.8–95.1)	0.36 (0.30–0.44)	0.090 (<LOD–0.12)	0.39 (0.27–0.51)	1.5 (1.2–1.8)	2.2 (1.4–2.9)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.09 µg/L, 0.07 µg/L, and 0.066 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

c Urinary cadmium results from cycles 1 and 2 are not comparable with those from cycle 5 due to a change in analytical reporting based on molybdenum interference.

E Use data with caution.

F Data are too unreliable to be published.

Table 8.4.3

Cadmium (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)[c]	6291	94.4 (92.5–95.9)	0.37 (0.34–0.41)	0.14 (0.11–0.16)	0.36 (0.31–0.41)	0.99 (0.94–1.0)	1.4 (1.2–1.6)
5 (2016–2017)	2691	72.0 (65.3–77.9)	—	<LOD	0.16 (0.13–0.18)	0.77 (0.59–0.96)	1.2 (0.88–1.5)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	3028	94.2 (91.5–96.1)	0.31 (0.28–0.35)	0.12 (0.087–0.15)	0.31 (0.26–0.35)	0.83 (0.73–0.93)	1.1 (0.94–1.2)
5 (2016–2017)	1341	68.3 (58.9–76.4)	—	<LOD	0.11 (0.093–0.14)	0.48[E] (0.30–0.66)	0.84 (0.62–1.1)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	3263	94.6 (92.7–96.1)	0.44 (0.40–0.47)	0.17 (0.14–0.20)	0.42 (0.38–0.46)	1.2 (0.94–1.4)	1.8 (1.4–2.3)
5 (2016–2017)	1350	75.7 (70.3–80.3)	—	<LOD	0.23 (0.20–0.25)	1.0 (0.69–1.3)	1.4 (1.1–1.7)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	572	89.7 (82.1–94.3)	0.39 (0.33–0.46)	<LOD	0.41 (0.35–0.47)	0.92 (0.78–1.0)	F
5 (2016–2017)	545	19.6[E] (10.5–33.6)	—	<LOD	<LOD	0.21[E] (0.13–0.29)	0.29 (0.21–0.38)
6–11 years							
1 (2007–2009)[c]	1030	85.8 (80.3–90.0)	0.34 (0.30–0.38)	<LOD	0.32 (0.28–0.37)	0.69 (0.58–0.81)	0.89 (0.70–1.1)
2 (2009–2011)[c]	1058	91.8 (89.0–94.0)	0.28 (0.24–0.33)	0.096 (<LOD–0.12)	0.29 (0.24–0.33)	0.65 (0.50–0.80)	0.80 (0.67–0.93)
5 (2016–2017)	531	27.4[E] (18.0–39.5)	—	<LOD	<LOD	0.17 (0.13–0.21)	0.22 (0.16–0.27)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)[c]	982	89.3 (85.2–92.4)	0.24 (0.22–0.26)	<LOD	0.23 (0.21–0.25)	0.41 (0.34–0.48)	0.53 (0.40–0.66)
2 (2009–2011)[c]	1039	94.2 (89.7–96.8)	0.20 (0.17–0.23)	0.099 (<LOD–0.12)	0.20 (0.18–0.21)	0.37 (0.31–0.44)	0.46 (0.33–0.58)
5 (2016–2017)	530	41.4 (27.7–56.6)	—	<LOD	<LOD	0.13 (0.11–0.15)	0.16 (0.13–0.19)
20–39 years							
1 (2007–2009)[c]	1165	86.5 (82.0–90.0)	0.31 (0.29–0.33)	<LOD	0.30 (0.29–0.30)	0.69 (0.61–0.77)	0.83 (0.69–0.97)
2 (2009–2011)[c]	1319	92.8 (88.3–95.6)	0.27 (0.24–0.31)	0.11 (<LOD–0.14)	0.27 (0.21–0.33)	0.63 (0.53–0.73)	0.79 (0.69–0.89)
5 (2016–2017)	372	67.9 (56.4–77.6)	0.12 (0.10–0.14)	<LOD	0.12 (0.095–0.15)	0.33[E] (0.12–0.54)	0.59[E] (0.24–0.95)
40–59 years							
1 (2007–2009)[c]	1218	92.4 (90.4–94.1)	0.54 (0.51–0.57)	0.20 (<LOD–0.23)	0.51 (0.46–0.56)	1.4 (1.2–1.5)	1.9 (1.7–2.1)
2 (2009–2011)[c]	1223	94.9 (92.2–96.7)	0.47 (0.43–0.53)	0.19 (0.17–0.21)	0.45 (0.40–0.50)	1.2 (0.95–1.5)	1.8 (1.2–2.4)
5 (2016–2017)	359	87.8 (82.1–91.9)	0.23 (0.18–0.29)	<LOD	0.23 (0.18–0.28)	0.85[E] (0.49–1.2)	1.2 (0.91–1.4)
60–79 years							
1 (2007–2009)[c]	1083	96.2 (93.2–97.9)	0.70 (0.64–0.77)	0.30 (<LOD–0.31)	0.69 (0.62–0.76)	1.6 (1.5–1.7)	2.1 (1.8–2.4)
2 (2009–2011)[c]	1080	98.5 (97.2–99.1)	0.64 (0.58–0.70)	0.26 (0.20–0.31)	0.63 (0.57–0.68)	1.6 (1.4–1.7)	2.0 (1.7–2.3)
5 (2016–2017)	354	92.2 (87.8–95.1)	0.42 (0.36–0.49)	0.12[E] (<LOD–0.17)	0.44 (0.39–0.49)	1.4 (1.1–1.8)	1.8 (1.4–2.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

c Urinary cadmium results from cycles 1 and 2 are not comparable with those from cycle 5 due to a change in analytical reporting based on molybdenum interference.

E Use data with caution.

F Data are too unreliable to be published.

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8.5 CHROMIUM

Chromium (CASRN 7440-47-3) is a naturally occurring element that is found in the Earth's crust in trace amounts (0.01%) (ATSDR, 2012; Health Canada, 2016). It is a transition metal that exhibits different properties depending on its oxidation state. Chromium can exist in nine different oxidation states, with the trivalent (chromium [III]) and the hexavalent (chromium [VI]) forms found most commonly in the environment (Environment Canada and Health Canada, 1994; Health Canada, 2016). In nature, chromium is not found in its elemental form but rather in complexes with oxygen, iron or lead (Health Canada, 2016).

Chromium is released into the environment by both natural and anthropogenic processes. Natural processes include weathering and erosion of soil and rocks as well as volcanic emissions (WHO, 2003; Health Canada, 2016). More than 70% of chromium released into air, soil, and water comes from anthropogenic sources, such as smelting and refining of nonferrous base metals, the production and combustion of fossil fuels, industrial manufacturing, and processing of chromium-based products (ATSDR, 2012; Health Canada, 2016; Environment and Climate Change Canada, 2017). Chromium (VI) rarely occurs naturally. It is produced mainly during the reduction of chromite ore in the industrial production of chromium metal. This oxidation state represents one-third of the total anthropogenic chromium released into the atmosphere (ATSDR, 2012; IARC, 2012).

Chromium is primarily used in electrical applications, wood preservation, the automobile industry, and the metallurgical industry, where it is used to produce stainless steel and high-chromium cast iron alloys (ATSDR, 2012; Health Canada, 2016). It is also used in many other processes, such as the production of paint, textile dyes and mordants, catalysts, pulp and paper, as well as in leather tanning, electroplating, and clinical medicine (Health Canada, 2016; WHO, 2003).

While exposure to chromium (III) occurs mainly through food, exposure to chromium (VI) occurs through drinking water and ambient air (Health Canada, 2016; IARC, 2012). However, the majority of drinking water samples analyzed for total chromium across Canada were found to be below the detection limit (Health Canada, 2016). Inhalation of chromium occurs mainly from cigarette smoke or from living near a contaminated area or an emission source, such as an industrial facility. Dermal exposure occurs through the use of consumer products containing chromium, such as cleaning materials, textiles, and leather (ATSDR, 2012).

Chromium (III) is an essential nutrient that plays a role in human metabolism, while chromium (VI) is the oxidation state that poses the greatest health risk (ATSDR, 2012; Dayan and Paine, 2001; IOM, 2001). As such, the summary of toxicokinetics and health effects will focus on chromium (VI). Chromium (VI) can be absorbed after oral or inhalation exposure. Absorption of chromium (VI) from the gastrointestinal tract is low (~7%), and chromium (VI) is partially reduced to chromium (III) at the intragastric level, which lowers its absorption (Health Canada, 2016; IARC, 2012; WHO, 2003). Chromium (VI) is readily absorbed via inhalation, but the fraction absorbed depends on several factors, such as the properties of the inhaled particles and the degree of reduction of chromium (VI) to chromium (III). Significant dermal absorption of chromium (VI) can occur, especially in damaged skin (ATSDR, 2012). After absorption into the bloodstream, chromium (VI) is taken up into red blood cells, where it is reduced to chromium (III), bound to hemoglobin and other intracellular proteins, and slowly lost from the cell (ATSDR, 2012; Dayan and Paine, 2001; IARC, 2012). Generally speaking, chromium (VI) is unstable in the body and is reduced to chromium (III), which can lead to the formation of reactive intermediates, chromium adducts with proteins and DNA, and secondary free radicals (ATSDR, 2012). Chromium is distributed to nearly all tissues, including blood, liver, lung, spleen, and kidney, and has a half-life in blood of about 30 days (EPA, 1998; Health Canada, 2016; WHO, 2003). Chromium can be transferred to infants via the placenta and breast milk (ATSDR, 2012). Elimination of chromium (VI) absorbed by inhalation occurs mainly in urine as the trivalent form (Health Canada, 2016; WHO, 2003); whereas after oral exposure, excretion occurs mainly through feces (IARC, 2012).

Measured levels of chromium in urine, whole blood, plasma, red blood cells, and lymphocytes can be used as biomarkers of exposure (ATSDR, 2012; Devoy et al., 2016). As chromium (III) is not able to cross the red blood cell membrane, chromium measured in red blood cells is a specific marker of chromium (VI) exposure, whereas the level of total chromium in urine may reflect either chromium (III) or chromium (VI) exposure (Devoy et al., 2016).

The toxicity of chromium depends upon its form and the route of exposure (Health Canada, 2016). Acute toxicity resulting from ingestion of chromium (VI) can occur at high doses, leading to gastrointestinal, kidney, liver and respiratory disorders, hemorrhagic diathesis, convulsions, and at very high concentrations, death from cardiovascular shock (Health Canada, 2016; WHO, 2003). There is a lack of clear evidence for chronic non-cancer toxicity from oral ingestion of chromium. However, chronic inhalation exposure of workers to chromium (VI) has been associated with respiratory tract effects, including nose bleeds, irritations or atrophy of the lining of the nose, bronchitis, and pneumonia (ATSDR, 2012). Dermal disorders, such as chronic skin ulcers or acute irritative dermatitis, have been reported in workers dermally exposed to chromium-containing material (Dayan and Paine, 2001).

There is limited information on the reproductive toxicity of chromium (VI) in humans, but some studies suggest that occupational exposure in males may lead to abnormal sperm count, morphology, and motility (ATSDR, 2012). Occupational exposure studies have also demonstrated genotoxic effects of chromium (VI) and its compounds (ATSDR, 2012). Several epidemiological studies in workers employed in chromate production, chromate pigment production, or chromium electroplating have reported that inhalation of chromium (VI) is associated with lung cancer and possibly cancer of the nose and nasal sinuses (Health Canada, 2016; IARC, 2012; WHO, 2003). The International Agency for Research on Cancer (IARC) has classified chromium (VI) compounds as carcinogenic to humans (Group 1) based on sufficient evidence for carcinogenicity

(lung cancer) in humans and sufficient evidence in experimental animals (IARC, 2012).

Health Canada and Environment Canada concluded that chromium (VI) compounds may be harmful to the environment and may constitute a danger to human life or health (Environment Canada and Health Canada, 1994). Chromium (VI) and its compounds have been added to the List of Toxic Substances under Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999). The Act allows the federal government to control the importation, manufacture, distribution, and use of chromium (VI) compounds in Canada. Risk management actions, including regulations and emission guidelines, have been developed under CEPA 1999 to control the release of chromium (VI) from thermal electricity generation, wood preservation applications, electroplating, anodizing and reverse etching (Environment and Climate Change Canada, 2017). Chromium, chromic acid, and its salts are identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetics Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

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 total chromium in drinking water (Health Canada, 2016). The guideline also takes into account the ability of currently available treatment technologies to remove chromium from drinking water at or below the guideline level.

Chromium was measured in the red blood cells of Canadian Health Measures Survey (CHMS) cycle 5 (2016–2017) participants aged 3–79 years. Data are presented as µg/L red blood cells. Chromium (VI) is the only form of inorganic chromium to penetrate cells. Thus, finding a measurable amount of chromium in red blood cells is an indicator of recent exposure to chromium (VI). The presence of chromium in red blood cells does not necessarily mean that an adverse health effect will occur. In addition, total chromium was analyzed in hair from CHMS participants aged 20–59 in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Table 8.5.1

Chromium (VI)[a] — Geometric means and selected percentiles of red blood cell concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2567	43.4 (32.4–55.1)	—	<LOD	<LOD	0.24 (0.20–0.29)	0.33 (0.26–0.39)
Males, 3–79 years							
5 (2016–2017)	1282	41.0 (31.7–50.9)	—	<LOD	<LOD	0.22 (0.17–0.27)	0.32 (0.21–0.42)
Females, 3–79 years							
5 (2016–2017)	1285	45.8 (31.9–60.4)	—	<LOD	<LOD	0.26 (0.22–0.31)	0.34 (0.27–0.41)
3–5 years							
5 (2016–2017)	480	53.3 (39.0–67.0)	—	<LOD	<LOD	0.23 (0.19–0.27)	0.28 (0.27–0.29)
6–11 years							
5 (2016–2017)	520	46.1 (33.8–58.9)	—	<LOD	<LOD	0.23 (0.20–0.26)	0.27 (0.23–0.32)
12–19 years							
5 (2016–2017)	523	45.7 (33.0–59.0)	—	<LOD	<LOD	0.23 (0.19–0.28)	0.29 (0.22–0.36)
20–39 years							
5 (2016–2017)	358	35.0[E] (23.4–48.7)	—	<LOD	<LOD	0.20 (0.14–0.26)	0.27 (0.19–0.35)
40–59 years							
5 (2016–2017)	340	45.3 (30.9–60.5)	—	<LOD	<LOD	0.29 (0.19–0.39)	0.43 (0.31–0.55)
60–79 years							
5 (2016–2017)	346	49.0 (36.6–61.5)	—	<LOD	<LOD	0.25 (0.18–0.32)	0.38[E] (0.18–0.58)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.12 µg/L.

a Chromium (VI) was measured indirectly as total chromium in red blood cells.

b 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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8.6 MERCURY

Mercury (CASRN 7439-97-6) is a naturally occurring, soft, silvery white metal that is liquid at room temperature. It is present in the Earth's crust at an average concentration of approximately 0.000005% (Emsley, 2001). 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 and Climate Change Canada, 2017).

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, 2013). Metabolism of inorganic mercury by micro-organisms

in the environment creates organic mercury (e.g., 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 wastewater (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 is 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 the 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 64% of the Canadian population age six and over had one or more amalgam-restored tooth surfaces (Richardson, 2014).

Mercury exposure in the general population of Canada is primarily through the consumption of larger species of fish in which methylmercury is the predominant form (Health Canada, 2007). To a lesser extent, the general population is exposed to inorganic mercury from sources such 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). Methylmercury exposure can occur in utero via cord blood, and it can be transferred to infants via breast milk (Environment and Climate Change Canada, 2016).

Approximately 95% of methylmercury 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. Methylmercury readily passes the blood-brain barrier and enters the brain, and in pregnant women it can easily cross the placental barrier into the fetus (Environment and Climate Change Canada, 2016; 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 in blood has been reported to range between 42 and 70 days in humans (Environment and Climate Change Canada, 2016). 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; Environment and Climate Change Canada, 2016).

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 one to two 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 may also 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 a number of western countries, the World Health Organization (WHO) 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 chemical form, the route of exposure, the timing and duration of exposure, and the absorbed concentration. Chronic exposure to low levels of methylmercury through ingestion 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 affect the development of the nervous system, resulting in effects on 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). Most published 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 Mercury Assessment has concluded that there is sufficient evidence of adverse impacts from mercury to warrant international action to reduce the risks to human health and the environment (UNEP, 2013). 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, 2017). 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. It also supports a gradual phase down in the use of dental amalgam in restorative treatment.

In Canada, mercury and its compounds are listed as toxic substances on Schedule 1 of the *Canadian Environmental Protection Act, 1999* (Canada, 1999; Canada, 2012). 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; Environment Canada and Health Canada, 2013). 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 identified as being prohibited on the List of Prohibited

and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018a). The Food and Drug Regulations prohibit the 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, 1978).

Health Canada has established a methylmercury blood guidance value of 20 µg/L for the general adult population; a methylmercury concentration in blood below this value is considered within 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, 2017a). Health Canada has also established maximum levels for mercury in retail fish (Health Canada, 2018b), and provides consumption advice for consumers of certain types of fish (Health Canada, 2017b). Mercury was analyzed as part of Health Canada's ongoing Total Diet Study surveys (Dabeka et al., 2003; Health Canada, 2009). 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 (GM) 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 GM urinary inorganic mercury concentration in dental amalgam-free participants from cycle 1 of the CHMS was 0.10 µg/L compared with the GM 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 females tended to have slightly greater urinary mercury concentrations than males (Nicolae et al., 2013). The population coverage of the CHMS excludes persons living on reserves and other Aboriginal settlements in Canadian provinces. 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 comprised 13 randomly selected First Nations communities in Canada with 503 First Nations participants aged 20 years and older. In 2011, the GM 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 GM 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), cycle 4 (2014–2015) and cycle 5 (2016–2017). Methylmercury was analyzed in the whole blood of CHMS participants aged 20–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015), and 3–19 years in cycle 5 (2016–2017). Inorganic mercury was analyzed in the whole blood of CHMS participants aged 6–79 years in cycle 1 (2007–2009) and 3–19 years in cycle 5 (2016–2017). Data from these cycles are presented in blood as µg/L. Finding a measurable amount of mercury in blood is an indicator of exposure to mercury and does not necessarily mean that an adverse health effect will occur. Total mercury was also analyzed in hair from CHMS participants aged 20–59 in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Inorganic mercury was analyzed in the urine of CHMS participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015).

Table 8.6.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	88.6 (86.0–90.8)	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	71.2 (66.4–75.6)	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	61.5 (55.5–67.2)	—	<LOD	0.59 (0.47–0.72)	2.5 (1.9–3.1)	3.5 (2.9–4.2)
5 (2016–2017)	4488	82.9 (80.1–85.4)	0.64 (0.54–0.75)	<LOD	0.70 (0.57–0.82)	2.6 (1.9–3.3)	3.8 (2.9–4.8)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	88.0 (84.9–90.5)	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	69.5 (64.3–74.3)	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	60.7 (54.7–66.4)	—	<LOD	0.58 (0.45–0.71)	2.8 (2.0–3.6)	3.7 (2.6–4.8)
5 (2016–2017)	2241	83.3 (77.5–87.8)	0.63 (0.52–0.77)	<LOD	0.69 (0.55–0.82)	2.6 (2.0–3.2)	3.4 (2.8–3.9)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	89.3 (86.6–91.5)	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	73.0 (67.1–78.2)	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	62.4 (55.9–68.5)	—	<LOD	0.60 (0.47–0.74)	2.2 (1.6–2.8)	3.3 (2.7–4.0)
5 (2016–2017)	2247	82.6 (79.6–85.2)	0.65 (0.54–0.78)	<LOD	0.71 (0.57–0.85)	2.6[E] (1.5–3.7)	4.5 (3.3–5.7)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	67.4 (58.2–75.4)	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	37.3 (28.6–46.8)	—	<LOD	<LOD	1.3 (1.0–1.7)	1.7[E] (0.88–2.5)
4 (2014–2015)	479	25.7[E] (16.7–37.4)	—	<LOD	<LOD	0.85[E] (<LOD–1.3)	1.3[E] (0.54–2.1)
5 (2016–2017)	465	58.2 (47.8–67.9)	—	<LOD	0.24 (<LOD–0.31)	1.1[E] (0.60–1.5)	1.7[E] (1.0–2.3)
6–11 years							
1 (2007–2009)	910	74.3 (69.1–78.9)	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	72.9 (67.2–78.0)	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	47.0 (37.6–56.7)	—	<LOD	<LOD	1.2 (0.78–1.7)	1.9[E] (0.91–2.9)
4 (2014–2015)	925	36.7 (29.4–44.6)	—	<LOD	<LOD	1.1 (0.84–1.3)	1.5 (0.96–2.0)
5 (2016–2017)	503	53.2 (43.1–63.0)	—	<LOD	0.21[E] (<LOD–0.33)	0.99[E] (0.59–1.4)	1.5 (1.1–1.8)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	945	79.5 (73.5–84.4)	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	70.3 (60.8–78.3)	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	45.0 (35.5–54.8)	—	<LOD	<LOD	1.6[E] (0.62–2.6)	2.8[E] (1.3–4.4)
4 (2014–2015)	975	39.2 (31.8–47.1)	—	<LOD	<LOD	1.3 (0.92–1.7)	2.2[E] (1.2–3.2)
5 (2016–2017)	512	67.0 (58.4–74.7)	0.33 (0.27–0.41)	<LOD	0.35 (0.27–0.43)	1.2 (1.0–1.4)	1.5 (0.99–2.1)
20–39 years							
1 (2007–2009)	1165	90.6 (87.9–92.8)	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	88.0 (82.4–92.0)	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	72.9 (65.6–79.1)	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	56.1 (47.9–64.0)	—	<LOD	0.48 (<LOD–0.65)	2.0 (1.6–2.4)	2.9 (2.0–3.8)
5 (2016–2017)	1037	78.5 (74.6–81.9)	0.55 (0.43–0.69)	<LOD	0.60 (0.43–0.78)	2.1[E] (0.89–3.4)	3.5[E] (2.1–4.9)
40–59 years							
1 (2007–2009)	1220	96.7 (95.0–97.8)	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	96.1 (94.2–97.5)	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	80.6 (73.9–86.0)	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	73.6 (66.4–79.7)	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)
5 (2016–2017)	987	89.4 (85.7–92.3)	0.85 (0.72–1.0)	<LOD	0.98 (0.81–1.1)	3.2 (2.5–3.9)	4.7 (3.5–5.9)
60–79 years							
1 (2007–2009)	1079	95.1 (91.4–97.3)	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	95.4 (92.0–97.4)	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	80.6 (73.4–86.3)	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	74.9 (69.0–80.0)	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)
5 (2016–2017)	984	92.1 (89.3–94.2)	0.83 (0.70–0.98)	0.22 (<LOD–0.29)	0.85 (0.72–0.98)	2.9 (2.5–3.3)	3.9 (3.0–4.7)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 0.1, 0.1, 0.42, 0.42, and 0.20 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 8.6.2

Methylmercury — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 3–19 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–19 years							
5 (2016–2017)	1505	54.1 (47.0–61.0)	—	<LOD	0.22[E] (<LOD–0.32)	1.3 (1.0–1.6)	1.9 (1.5–2.3)
Males, 3–19 years							
5 (2016–2017)	754	53.0 (46.0–59.8)	—	<LOD	0.21[E] (<LOD–0.34)	1.5 (1.2–1.8)	2.2 (1.6–2.7)
Females, 3–19 years							
5 (2016–2017)	751	55.3 (47.0–63.4)	—	<LOD	0.23 (<LOD–0.31)	1.1 (0.80–1.4)	1.7[E] (1.1–2.4)
3–5 years							
5 (2016–2017)	473	49.2 (39.5–59.1)	—	<LOD	<LOD	1.1[E] (0.52–1.7)	1.8 (1.3–2.4)
6–11 years							
5 (2016–2017)	511	49.3 (40.1–58.6)	—	<LOD	<LOD	1.3[E] (0.73–1.8)	2.0[E] (1.2–2.8)
12–19 years							
5 (2016–2017)	521	59.2 (49.9–68)	—	<LOD	0.29[E] (<LOD–0.41)	1.3 (1.1–1.6)	1.9 (1.4–2.5)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.19 µg/L.

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 8.6.3

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	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–79 years							
3 (2012–2013)	1032	81.6 (75.7–86.3)	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	81.6 (77.9–84.8)	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	81.2 (71.9–88.0)	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	81.7 (76.2–86.2)	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	81.9 (72.4–88.6)	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	81.5 (74.8–86.7)	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	78.9 (68.5–86.6)	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	72.0 (63.8–78.9)	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	80.6 (71.8–87.2)	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	86.8 (79.4–91.8)	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	87.4 (79.0–92.8)	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	87.9 (81.4–92.3)	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)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3 and 4 is 0.19 µg/L.

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 are too unreliable to be published.

Table 8.6.4

Mercury (inorganic) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 6–19 years[a], Canadian Health Measures Survey cycle 1 (2007–2009) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 6–19 years							
1 (2007–2009)	425	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1032	1.8[E] (1.1–2.9)	—	<LOD	<LOD	<LOD	<LOD
Males, 6–19years							
1 (2007–2009)	227	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	514	F	—	<LOD	<LOD	<LOD	<LOD
Females, 6–19 years							
1 (2007–2009)	198	F	—	<LOD	<LOD	<LOD	X
5 (2016–2017)	518	2.1[E] (1.2–3.9)	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1 and 5 are 0.4 and 0.22 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 6–19 years were included, as participants under the age of six years were not included in cycle 1 and participants over the age of 19 years were not included in cycle 5.

b 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 are too unreliable to be published.

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

Table 8.6.5

Mercury (inorganic) — Geometric means and selected percentiles of whole blood concentrations (µg/L) for the Canadian population aged 3–19 years by age group, Canadian Health Measures Survey cycle 1 (2007–2009) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1505	1.8[E] (1.0–3.2)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
5 (2016–2017)	754	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
5 (2016–2017)	751	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
5 (2016–2017)	473	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
1 (2007–2009)	221	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	511	3.3[E] (1.8–5.9)	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
1 (2007–2009)	204	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	521	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1 and 5 are 0.4 and 0.22 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1; the complete data set for cycle 1 participants aged 6–79 years is available in the *Report on Human Biomonitoring of Environmental Chemicals in Canada* (2010).

E Use data with caution.

F Data are too unreliable to be published.

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8.7 SELENIUM

Selenium (CASRN 7782-49-2) is a naturally occurring trace mineral distributed widely in the environment and present in the Earth's crust at concentrations averaging 0.000009% (Schamberger, 1984). Selenium is present in the environment in the inorganic form as selenide, selenate, and selenite but rarely as elemental selenium. Selenium is an essential trace element required for the maintenance of good health in humans.

Selenium in its organic form is found in trace quantities in most plants and animal tissues (Schamberger, 1984). Elevated levels of selenium in the environment may occur naturally from weathering of base-metal deposits and soils (CCME, 2009). Selenium is also released into the environment as a result of anthropogenic activities, such as mining or metallurgical processes (CCME, 2009). Other sources of anthropogenic selenium emissions include incinerator stacks, burning coal and oil, and large-scale combustion processes.

Historically, the primary use of selenium was in the electronics industry in the form of arsenic triselenide, used as a photoreceptor for photocopiers (USGS, 2001). Because selenium has various electrical and conductive properties, it is also used in light meters, photoelectric and solar cells, semiconductors, and arc-light electrodes. It is also used as a colourizing and decolourizing agent for glass, and to reduce solar heat for architectural glass (USGS, 2004). Selenium is also present in stainless steel, enamels, inks, rubber, batteries, explosives, fertilizers, animal feed, pharmaceuticals, and shampoos (ATSDR, 2003).

The Canadian population is exposed to selenium compounds in food, ambient air, drinking water, soil, and natural health products. More than 99% of the total daily intake of selenium is estimated to occur through the diet for the general population and all age classes (CCME, 2009). Absorption of selenium depends on the chemical form; organic forms are absorbed

more readily (>90%) than inorganic forms (>50%) (IOM, 2000). Absorption also depends on the overall exposure level; absorption increases when selenium levels in the body are low (IOM, 2000). Once inside the body, selenium generally concentrates in the liver and kidneys regardless of the initial chemical form. It can also be found in nails and hair (IOM, 2000). Selenium elimination is triphasic, with biological half-lives of approximately one day, one week, and three months (ATSDR, 2003). Approximately 50% to 80% of absorbed selenium is eliminated in the urine (Marier and Jaworski, 1983). Selenium levels in the body following both short- and long-term exposure can be determined through blood and urine tests (IOM, 2000). Human breath can also be used as a biomarker for selenium exposure when large amounts of selenium are being excreted (IOM, 2000).

Selenium is an essential trace element and a component of several proteins and enzymes in the body (ATSDR, 2003; Health Canada, 2010). Selenium aids in the defence of oxidative stress, the regulation of thyroid hormone action, and the regulation of the redox status of vitamin C and other molecules (IOM, 2000). Selenium deficiency seldom causes overt illness in isolation; however, it may lead to biochemical changes that predispose people to illness associated with other stresses (IOM, 2000). There is some evidence that suboptimal levels of selenium may lead to sperm abnormalities and effects on sperm motility (Ahsan et al., 2014). On account of its essentiality, Health Canada has established recommended dietary allowances for selenium (Health Canada, 2010; IOM, 2000).

There is a narrow therapeutic window for selenium, and adverse health effects can occur when ingested at levels greater than the tolerable upper intake level (Health Canada, 2010; IOM, 2000). The level at which selenium toxicity occurs can be difficult to determine because it is affected by the types of protein in the diet, levels of vitamin E, and the forms of selenium to which the individual is exposed (Health Canada, 2014). Acute oral intake of excess selenium can result in nausea, vomiting, and diarrhea. Selenosis, a disease that results in hair loss, nail brittleness and neurological abnormalities, is the critical health effect associated with chronic exposure to elevated levels of selenium (i.e., 10 to 20 times more than the recommended dietary allowances) (ATSDR, 2003; IOM, 2000; WHO, 2011). The role of selenium in other chronic diseases, such as diabetes, hypertension, and cardiovascular disease, is a subject of ongoing investigation (Benstoem et al., 2015; Boosalis, 2008; Ogawa-Wong et al., 2016). The International Agency for Research on Cancer (IARC) has determined that selenium's carcinogenicity to humans is not classifiable (Group 3) (IARC, 1975).

The Government of Canada conducted a science-based screening assessment under the Chemicals Management Plan to determine whether selenium and its compounds (including 29 selenium-containing substances on the Domestic Substances List) present or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2017a). The assessment concluded that selenium and its compounds are toxic under CEPA 1999 as they are harmful to human health, based on the potential for elevated levels in certain subpopulations in Canada that have higher selenium intake, as well as being harmful to the environment. Selenium and its compounds are proposed to be added to Schedule 1, List of Toxic Substances, under CEPA 1999 (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2017a). Risk management actions for selenium and its compounds have been proposed that include measures to reduce the release of selenium into water and finalizing the revised maximum daily dose allowed for selenium in natural health products (Environment and Climate Change Canada and Health Canada, 2017b). Selenium and its compounds (except selenium sulfide) are identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist, or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018a).

In Canada, the leachable selenium content in a variety of consumer products is regulated under the *Canada Consumer Product Safety Act* (Canada, 2010a). Consumer products regulated for selenium content 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 has also set a maximum level for selenium in natural health products in Canada (Health Canada, 2018b). Health Canada has developed a Canadian

drinking water quality guideline that sets out the maximum acceptable concentration of selenium on the basis of health considerations (Health Canada, 2014). Tolerable upper intake levels for selenium, which account for its potential toxicity, have been developed by the Institute of Medicine and adopted by Health Canada (Health Canada, 2010; IOM, 2000). Selenium is also included in the list of various chemicals analyzed as part of Health Canada's ongoing Total Diet Study surveys (Health Canada, 2016). These surveys provide estimates of the levels of 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 Québec City with 500 participants aged 18–65 years, the geometric mean for selenium in whole blood was 2.8 µmol/L (221.2 µg/L) (INSPQ, 2004).

Selenium was measured in the whole blood of all Canadian Health Measures Survey (CHMS) participants aged 6–79 years in cycle 1 (2007–2009) and 3–79 years in cycle 2 (2009–2011) and cycle 5 (2016–2017). Data from these cycles are presented in blood as µg/L. Finding a measurable amount of selenium in blood or urine is an indicator of exposure to selenium and does not necessarily mean that an adverse health effect will occur. Because selenium is an essential trace element, its presence in biological fluids is expected. Selenium was also analyzed in hair from CHMS participants aged 20–59 years in cycle 5 (2016–2017); summary data from this analysis in hair can be found in Appendix D.

Selenium was also measured 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).

Table 8.7.1

Selenium — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	100	190 (190–190)	160 (150–160)	180 (180–190)	220 (210–230)	240 (230–240)
5 (2016–2017)	4517	100	170 (170–170)	130 (130–140)	160 (160–170)	200 (200–210)	210 (200–210)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2940	100	190 (190–200)	160 (160–160)	190 (180–190)	220 (210–230)	240 (230–250)
5 (2016–2017)	2257	100	170 (170–180)	140 (130–140)	170 (160–170)	200 (200–210)	210 (200–220)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	3130	100	190 (180–190)	150 (150–160)	180 (180–180)	220 (210–230)	240 (230–240)
5 (2016–2017)	2260	100	170 (170–170)	130 (130–140)	160 (160–170)	200 (190–210)	210 (210–220)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	495	100	170 (160–170)	140 (130–150)	160 (160–170)	190 (180–200)	210 (200–210)
5 (2016–2017)	473	100	150 (140–150)	120 (120–120)	140 (130–150)	170 (160–170)	170 (170–170)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	910	100	190 (180–190)	150 (150–160)	180 (180–180)	210 (210–220)	230 (220–240)
2 (2009–2011)	961	100	170 (170–180)	140 (140–150)	170 (160–170)	200 (200–210)	210 (200–220)
5 (2016–2017)	511	100	150 (150–160)	120 (120–130)	150 (150–150)	170 (160–180)	180 (170–190)
12–19 years							
1 (2007–2009)	945	100	200 (190–200)	160 (160–170)	190 (190–190)	230 (230–240)	250 (240–260)
2 (2009–2011)	997	100	190 (180–190)	160 (160–160)	180 (170–180)	210 (200–220)	230 (220–240)
5 (2016–2017)	521	100	160 (160–170)	130 (130–130)	160 (150–160)	180 (170–190)	200 (190–210)
20–39 years							
1 (2007–2009)	1165	100	200 (200–210)	160 (160–170)	200 (190–200)	240 (230–240)	250 (240–260)
2 (2009–2011)	1313	100	190 (190–200)	160 (160–160)	190 (180–190)	220 (210–230)	240 (220–260)
5 (2016–2017)	1038	100	170 (170–180)	140 (130–140)	170 (160–170)	200 (190–220)	210 (200–220)
40–59 years							
1 (2007–2009)	1220	100	200 (200–210)	170 (160–170)	200 (190–200)	240 (230–240)	250 (240–260)
2 (2009–2011)	1222	100	190 (190–200)	160 (160–160)	190 (180–200)	230 (220–240)	240 (230–250)
5 (2016–2017)	990	100	170 (170–180)	140 (140–150)	170 (160–170)	200 (200–210)	220 (200–230)
60–79 years							
1 (2007–2009)	1079	100	200 (200–210)	170 (160–170)	200 (190–200)	240 (230–250)	250 (240–270)
2 (2009–2011)	1082	100	190 (190–190)	160 (160–160)	180 (180–190)	220 (210–230)	240 (230–240)
5 (2016–2017)	984	100	170 (170–180)	140 (130–140)	170 (160–170)	200 (200–210)	210 (210–220)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 8, 20, and 32 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

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SUMMARIES AND RESULTS FOR SELF-CARE AND CONSUMER PRODUCT CHEMICALS 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 plastics have wide application in consumer products, including storage containers for foods and beverages; they were 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 updated its dietary exposure estimates for BPA after completing a number of surveys in which BPA concentrations were measured in various foods, including canned foods and beverages, liquid infant formula, and samples from the Total Diet Study (Health Canada, 2012). Dermal exposure through handling of thermal printing paper is considered an important secondary route of exposure (EFSA CEF Panel, 2015). Oral exposure can also result from leaching of BPA from dental materials; however, the contribution to total BPA exposure is likely negligible (Becher et al., 2018; SCENIHR, 2015). 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).

In humans, BPA is readily absorbed and undergoes extensive metabolism in the gut wall and the liver (WHO, 2011). 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, occurring primarily in the liver and 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 not considered to be biologically active and is rapidly excreted in urine with a half-life of less than two hours (WHO, 2011). Urinary levels of total BPA, including both conjugated and free unconjugated forms, are commonly used as biomarkers to assess recent exposures (Arbuckle et al., 2015a; Ye et al., 2005).

Characterization of the potential risk to human health from exposure to BPA includes key effects on the liver and kidneys as well as effects on reproduction, development, neurodevelopment and behaviour (EFSA CEF Panel, 2015; Environment Canada and Health Canada, 2008a; EU, 2010). In 2018, the U.S. National Toxicology Program published results of a comprehensive investigation of BPA toxicity and concluded that early-life and long-term exposures are unlikely to pose a health risk at low doses (NTP, 2018). However, 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, remains under investigation (Heindel et al., 2015; Seachrist et al., 2016).

The Government of Canada conducted a science-based screening assessment under phase one of the Chemicals Management Plan to determine whether BPA presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment Canada and Health Canada, 2008a). Based on information available at that time, the assessment concluded that BPA is toxic under CEPA 1999, as it is considered harmful to the environment and human health (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 is not expected to pose a health risk to the general population, including newborns and young children (Health Canada, 2012). However, exposure to BPA should be as low as reasonably achievable (ALARA) and efforts should continue to limit BPA exposure in infants and newborns from food packaging applications — specifically pre-packaged infant formula products as a sole-source food. As part of the ALARA approach, Health Canada committed to supporting industry to reduce levels of BPA in infant formula can linings (Health Canada, 2014). Health Canada's findings confirm that alternative packaging materials for liquid infant formula products manufactured without BPA have been adopted by industry (Health Canada, 2014). 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). The removal of bisphenol A in polycarbonate baby bottles and liquid infant formula can linings has led Health Canada to conclude that there has been significant progress toward meeting the human health objective for BPA set out in 2008 (Health Canada, 2018a). BPA is also identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018b). 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., 2015b). 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 Nations 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 (CHMS) participants aged 6–79 years in cycle 1 (2007–2009), and 3–79 years in cycle 2 (2009–2011), cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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 by age group, Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	93.8 (91.2–95.7)	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	91.7 (90.1–93.1)	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	91.9 (88.5–94.4)	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)
5 (2016–2017)	2647	81.5 (74.7–86.7)	0.81 (0.71–0.93)	<LOD	0.85 (0.75–0.96)	2.9 (2.6–3.2)	4.2 (3.1–5.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1281	93.3 (89.1–96.0)	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	93.0 (90.9–94.6)	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	94.6 (91.3–96.7)	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)
5 (2016–2017)	1315	80.7 (72.1–87.0)	0.84 (0.69–1.0)	<LOD	0.85 (0.69–1.0)	2.9 (2.5–3.4)	5.6 (3.7–7.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	94.3 (91.8–96.1)	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	90.5 (88.1–92.5)	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	89.3 (82.8–93.5)	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)
5 (2016–2017)	1332	82.3 (74.7–88.0)	0.78 (0.69–0.89)	<LOD	0.85 (0.72–0.99)	2.6 (2.2–3.0)	3.3 (2.6–4.0)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	524	94.1 (89.3–96.8)	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	92.6 (82.9–97.0)	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	91.3 (84.2–95.4)	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)
5 (2016–2017)	547	86.2 (77.2–92.0)	0.94 (0.72–1.2)	<LOD	0.99 (0.78–1.2)	3.0[E] (1.9–4.1)	4.4[E] (2.4–6.3)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1031	93.5 (89.1–96.2)	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	93.4 (88.9–96.2)	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	95.9 (94.6–96.9)	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	94.4 (89.0–97.2)	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)
5 (2016–2017)	516	88.6 (83.8–92.1)	0.97 (0.83–1.1)	<LOD	0.94 (0.75–1.1)	2.9[E] (1.8–4.0)	5.5[E] (3.1–7.8)
12–19 years							
1 (2007–2009)	980	93.7 (90.2–96.0)	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	94.4 (88.9–97.2)	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	92.3 (86.2–95.8)	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	93.7 (88.7–96.6)	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)
5 (2016–2017)	524	86.8 (79.7–91.7)	0.96 (0.80–1.2)	<LOD	0.96 (0.83–1.1)	3.2 (2.6–3.8)	4.1 (2.9–5.3)
20–39 years							
1 (2007–2009)	1165	92.1 (87.0–95.4)	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	96.1 (89.8–98.6)	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	91.1 (85.0–94.9)	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	90.2 (82.7–94.7)	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)
5 (2016–2017)	362	75.2 (56.4–87.7)	0.84[E] (0.57–1.2)	<LOD	1.0 (0.74–1.4)	2.9 (1.9–4.0)	5.4[E] (1.9–8.8)
40–59 years							
1 (2007–2009)	1219	87.5 (82.5–91.2)	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	92.7 (86.4–96.2)	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	93.1 (91.2–94.7)	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	92.5 (85.9–96.1)	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)
5 (2016–2017)	348	82.4 (74.1–88.5)	0.73 (0.59–0.89)	<LOD	0.79 (0.61–0.96)	2.4 (1.9–3.0)	3.1 (2.6–3.7)
60–79 years							
1 (2007–2009)	1081	88.1 (83.3–91.6)	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	91.9 (86.5–95.2)	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	88.4 (83.9–91.7)	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	92.0 (87.5–95.0)	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)
5 (2016–2017)	350	83.3 (77.1–88.1)	0.77 (0.66–0.90)	<LOD	0.79 (0.66–0.92)	2.7 (1.9–3.4)	3.7[E] (2.3–5.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 0.2, 0.2, 0.23, 0.23, and 0.32 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 9.1.2

Bisphenol A (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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	93.8 (91.2–95.7)	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	91.7 (90.1–93.1)	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	91.9 (88.5–94.4)	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)
5 (2016–2017)	2620	81.5 (74.7–86.7)	0.79 (0.71–0.87)	<LOD	0.76 (0.65–0.86)	2.4 (1.8–2.9)	3.3 (2.8–3.8)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	93.3 (89.1–96.0)	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	93.0 (90.9–94.6)	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	94.6 (91.3–96.7)	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)
5 (2016–2017)	1305	80.7 (72.1–87.0)	0.73 (0.64–0.83)	<LOD	0.70 (0.53–0.86)	2.4 (1.7–3.2)	3.2 (2.5–3.9)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	94.3 (91.8–96.1)	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	90.5 (88.1–92.5)	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	89.3 (82.8–93.5)	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)
5 (2016–2017)	1315	82.3 (74.7–88.0)	0.85 (0.77–0.95)	<LOD	0.80 (0.70–0.90)	2.2 (1.5–2.9)	3.4 (2.3–4.4)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	94.1 (89.3–96.8)	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	92.6 (82.9–97.0)	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	91.3 (84.2–95.4)	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)
5 (2016–2017)	538	86.2 (77.2–92.0)	1.6 (1.4–1.9)	<LOD	1.5 (1.2–1.8)	4.8 (3.7–5.9)	F
6–11 years							
1 (2007–2009)	1028	93.5 (89.1–96.2)	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	93.4 (88.9–96.2)	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	95.9 (94.6–96.9)	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	94.4 (89.0–97.2)	1.2 (1.0–1.5)	0.41 (<LOD–0.54)	1.1 (0.94–1.3)	3.2 (2.6–3.8)	F
5 (2016–2017)	507	88.6 (83.8–92.1)	1.1 (0.99–1.3)	<LOD	1.0 (0.90–1.1)	3.1 (2.3–3.8)	5.0[E] (2.9–7.0)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	978	93.7 (90.2–96.0)	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	94.4 (88.9–97.2)	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	92.3 (86.2–95.8)	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	93.7 (88.7–96.6)	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)
5 (2016–2017)	520	86.8 (79.7–91.7)	0.74 (0.58–0.94)	<LOD	0.66 (0.52–0.80)	2.0[E] (0.79–3.3)	3.2[E] (2.1–4.4)
20–39 years							
1 (2007–2009)	1161	92.1 (87.0–95.4)	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	96.1 (89.8–98.6)	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	91.1 (85.0–94.9)	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	90.2 (82.7–94.7)	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)
5 (2016–2017)	359	75.2 (56.4–87.7)	0.75 (0.60–0.94)	<LOD	0.84 (0.62–1.1)	2.4E (1.5–3.3)	3.0 (2.0–4.1)
40–59 years							
1 (2007–2009)	1214	87.5 (82.5–91.2)	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	92.7 (86.4–96.2)	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	93.1 (91.2–94.7)	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	92.5 (85.9–96.1)	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)
5 (2016–2017)	347	82.4 (74.1–88.5)	0.66 (0.56–0.79)	<LOD	0.61 (0.53–0.69)	1.9E (1.2–2.6)	2.8 (2.1–3.5)
60–79 years							
1 (2007–2009)	1081	88.1 (83.3–91.6)	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	91.9 (86.5–95.2)	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	88.4 (83.9–91.7)	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	92.0 (87.5–95.0)	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)
5 (2016–2017)	349	83.3 (77.1–88.1)	0.89 (0.80–0.99)	<LOD	0.84 (0.71–0.97)	2.2[E] (0.85–3.5)	4.7[E] (2.7–6.7)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

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9.2 PARABENS

Parabens are a group of para-hydroxybenzoic (p-hydroxybenzoic) acid esters, four of which were measured in cycle 3, cycle 4, and cycle 5 of the Canadian Health Measures Survey (CHMS): methyl, ethyl, propyl, and butyl paraben.

Table 9.2.1

Parabens measured in the Canadian Health Measures Survey cycle 5 (2016–2017).

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 personal care products owing to their antibacterial and antifungal properties (Health Canada, 2019). These products include makeup, moisturizers, sunscreens, hair-care products, facial and skin cleansers, and shaving products. Methyl, propyl, butyl, and ethyl paraben are the most common ones used in cosmetic products (FDA, 2018). Typical concentrations of parabens in cosmetic products are generally 0.5% or less (Health Canada, 2019). Methyl paraben and propyl paraben are permitted for use as food additives (preservatives) in foods sold in Canada. Parabens are also used in pharmaceutical drugs (Ye et al., 2008). Propyl paraben and butyl paraben are classified as active ingredients in natural health products as they are used medicinally as a source of p-hydroxybenzoic acid, a major metabolite of parabens (Health Canada, 2018).

Although parabens in commercial use are synthetically produced, some parabens may also occur naturally in certain fruits and vegetables, such as blueberries and carrots (Health Canada, 2019). 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 that contain parabens, such as moisturizers and cosmetics. 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 through 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. One study of premature infants who received intramuscular injections of paraben-containing gentamicin observed that excretion of methyl paraben, primarily in the conjugated form, was variable (approximately 10% to 90%), possibly due to factors such as variation in intramuscular absorption and gestational and postpartum ages (Hindmarsh et al., 1983). A study of parabens in the 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 have not been observed as a result of exposures to parabens at concentrations found in cosmetics, with acute, subchronic and chronic experimental animal studies demonstrating a low order of toxicity of parabens (Cosmetic Ingredient Review Expert Panel, 2008). It should be noted that most of the available toxicity data are from single paraben exposure studies, and that the additive and cumulative risks of exposures to multiple parabens are not well studied (Karpuzoglu et al., 2013). 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 vitro, but well-conducted animal studies do not demonstrate estrogenic effects (Sivaraman et al., 2018); human data do not support a link to estrogenic effects because exposure to parabens has not been observed to affect hormone levels or sperm quality (Adoamnei et al., 2018; Meeker et al., 2011). Parabens have not been found to be carcinogenic in chronic animal studies. The International Agency for Research on Cancer (IARC) has not evaluated parabens with respect to human carcinogenicity.

Methyl, ethyl, propyl, and butyl paraben have been identified as priorities for risk assessment based on human health concerns under the Chemicals Management Plan Identification of Risk Assessment Priorities (IRAP) initiative (Environment and Climate Change Canada and Health Canada, 2015). A screening-level risk assessment is currently under way to determine whether parabens present or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (Canada, 1999; Environment and Climate Change Canada, 2019). As part of this assessment, Health Canada will present currently available evidence on the use of these parabens in each of the various products regulated by Health Canada as well as the results of its risk assessment. Health Canada continues to monitor and review any new scientific data on parabens (Health Canada, 2019).

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 CHMS participants aged 3–79 years in cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). Data are presented as µg/L and µg/g creatinine. Finding a measurable quantity of parabens in urine is an indicator of exposure to parabens and does not necessarily mean that an adverse health effect will occur.

Table 9.2.2

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2339	91.0 (87.4–93.7)	21 (17–25)	<LOD[E] (<LOD–1.8)	19 (16–23)	320[E] (200–450)	470[E] (210–730)
4 (2014–2015)	2564	89.6 (85.4–92.7)	17 (13–22)	<LOD	15 (9.8–20)	270 (190–340)	490 (340–640)
5 (2016–2017)	2720	87.9 (84.2–90.8)	14 (11–18)	<LOD	11 (7.3–15)	230[E] (100–350)	550[E] (260–830)
Males, 3–79 years							
3 (2012–2013)	1171	85.1 (78.3–90.0)	9.6 (7.2–13)	<LOD	5.9[E] (3.5–8.3)	F	F
4 (2014–2015)	1275	85.5 (78.2–90.7)	9.4 (6.9–13)	<LOD	6.8[E] (4.2–9.4)	F	F
5 (2016–2017)	1356	84.4 (79.8–88.2)	7.2 (5.8–8.9)	<LOD	5.0 (3.9–6.1)	110 (73–140)	190[E] (78–290)
Females, 3–79 years							
3 (2012–2013)	1168	97.1 (95.2–98.3)	45 (33–63)	3.7 (2.4–4.9)	53[E] (21–85)	410 (330–480)	480[E] (220–740)
4 (2014–2015)	1289	93.7 (91.4–95.4)	30 (21–43)	1.8 (1.3–2.4)	F	310[E] (170–440)	510[E] (170–850)
5 (2016–2017)	1364	91.4 (87.7–94.0)	28 (19–39)	1.6[E] (<LOD–2.3)	26[E] (8.4–43)	480[E] (210–750)	860[E] (510–1200)
3–5 years							
3 (2012–2013)	463	91.7 (86.8–94.8)	20[E] (14–28)	1.5[E] (<LOD–2.2)	16 (11–21)	270[E] (85–450)	660[E] (340–980)
4 (2014–2015)	511	94.3 (91.4–96.3)	12 (9.3–15)	1.9[E] (<LOD–2.7)	8.0 (5.9–10)	110[E] (50–170)	330[E] (110–560)
5 (2016–2017)	552	88.9 (81.9–93.4)	9.9[E] (6.8–14)	<LOD	6.9 (4.4–9.4)	F	F
6–11 years							
3 (2012–2013)	481	87.9 (82.5–91.8)	7.7 (5.7–10)	<LOD	6.0 (4.4–7.7)	80[E] (30–130)	150[E] (55–240)
4 (2014–2015)	514	91.9 (89.5–93.8)	7.6 (6.4–9.1)	1.4 (<LOD–1.8)	6.1 (4.0–8.2)	43 (30–57)	F
5 (2016–2017)	540	88.4 (84.2–91.6)	7.5 (5.6–9.9)	<LOD	4.9 (3.8–6.0)	140[E] (62–230)	F
12–19 years							
3 (2012–2013)	469	93.7 (89.3–96.4)	15[E] (10–22)	1.5[E] (<LOD–2.3)	10[E] (3.1–18)	F	F
4 (2014–2015)	505	89.4 (84.3–92.9)	14[E] (9.1–21)	<LOD	9.7 (6.4–13)	300[E] (130–470)	520[E] (250–780)
5 (2016–2017)	538	87.5 (82.6–91.2)	9.5 (6.7–13)	<LOD	F	130 (100–160)	280[E] (150–400)
20–39 years							
3 (2012–2013)	328	91.3 (77.9–96.9)	21[E] (13–34)	F	21[E] (6.4–36)	F	F
4 (2014–2015)	362	90.8 (82.9–95.3)	16[E] (9.3–28)	1.3[E] (<LOD–1.9)	F	300[E] (170–430)	390[E] (180–610)
5 (2016–2017)	376	85.6 (75.8–91.9)	15[E] (9.8–22)	<LOD	16[E] (7.0–24)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	284	90.3 (79.6–95.7)	25[E] (14–43)	1.3[E] (<LOD–2.1)	26[E] (8.0–44)	400[E] (180–620)	430[E] (190–670)
4 (2014–2015)	312	86.3 (77.7–91.9)	21[E] (11–38)	<LOD	F	270[E] (93–440)	550[E] (250–860)
5 (2016–2017)	360	89.8 (83.1–94.0)	14[E] (9.6–21)	<LOD	12[E] (5.0–18)	F	F
60–79 years							
3 (2012–2013)	314	91.6 (84.7–95.5)	25[E] (16–37)	1.7[E] (<LOD–2.7)	30[E] (8.1–51)	360 (230–480)	460 (330–600)
4 (2014–2015)	360	91.6 (87.3–94.5)	20 (16–26)	1.4[E] (<LOD–2.0)	22[E] (8.2–36)	F	680[E] (210–1200)
5 (2016–2017)	354	88.4 (79.3–93.8)	20 (15–28)	<LOD	17[E] (9.9–24)	430[E] (190–660)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 1.3 µg/L.

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 are too unreliable to be published.

Table 9.2.3

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2338	91.0 (87.4–93.7)	21 (18–26)	<LOD	23 (15–31)	320[E] (190–450)	620 (410–840)
4 (2014–2015)	2563	89.6 (85.4–92.7)	15 (11–21)	<LOD	13[E] (6.5–19)	230 (180–290)	340 (230–440)
5 (2016–2017)	2688	87.9 (84.2–90.8)	14 (11–18)	<LOD	9.6 (6.5–13)	250[E] (150–360)	500[E] (300–710)
Males, 3–79 years							
3 (2012–2013)	1171	85.1 (78.3–90.0)	8.1 (6.2–11)	<LOD	5.9 (3.9–7.9)	F	F
4 (2014–2015)	1274	85.5 (78.2–90.7)	7.4 (5.4–10)	<LOD	5.3[E] (3.3–7.2)	99[E] (<LOD–150)	230[E] (130–340)
5 (2016–2017)	1341	84.4 (79.8–88.2)	6.2 (5.1–7.6)	<LOD	4.0 (2.9–5.1)	110 (81–130)	200 (140–250)
Females, 3–79 years							
3 (2012–2013)	1167	97.1 (95.2–98.3)	58 (43–79)	4.5[E] (2.8–6.2)	60[E] (28–93)	460[E] (200–710)	760 (630–890)
4 (2014–2015)	1289	93.7 (91.4–95.4)	31[E] (21–46)	2.1 (1.5–2.7)	37[E] (17–56)	290[E] (180–400)	480[E] (250–700)
5 (2016–2017)	1347	91.4 (87.7–94.0)	30 (21–43)	2.0 (<LOD–2.6)	33[E] (15–51)	470[E] (290–640)	780[E] (470–1100)
3–5 years							
3 (2012–2013)	462	91.7 (86.8–94.8)	38[E] (25–58)	3.9[E] (<LOD–6.2)	27[E] (16–38)	540[E] (180–910)	F
4 (2014–2015)	511	94.3 (91.4–96.3)	21 (16–27)	3.7 (<LOD–4.6)	13[E] (8.2–19)	210[E] (72–360)	430[E] (200–660)
5 (2016–2017)	542	88.9 (81.9–93.4)	17[E] (11–26)	<LOD	13[E] (7.2–18)	260[E] (86–430)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
3 (2012–2013)	481	87.9 (82.5–91.8)	9.8 (6.9–14)	<LOD	7.5 (4.9–10)	F	250[E] (98–390)
4 (2014–2015)	513	91.9 (89.5–93.8)	8.4 (7.1–9.8)	1.8 (<LOD–2.2)	7.1 (5.3–8.8)	41 (30–52)	F
5 (2016–2017)	531	88.4 (84.2–91.6)	8.7 (6.6–11)	<LOD	5.3 (4.0–6.6)	160[E] (49–280)	F
12–19 years							
3 (2012–2013)	469	93.7 (89.3–96.4)	11 (8.0–16)	0.97 (<LOD–1.2)	9.7[E] (5.7–14)	F	F
4 (2014–2015)	505	89.4 (84.3–92.9)	9.9[E] (6.7–15)	<LOD	7.2 (4.9–9.5)	180[E] (66–290)	370[E] (100–640)
5 (2016–2017)	531	87.5 (82.6–91.2)	7.2 (5.4–9.6)	<LOD	4.9[E] (2.4–7.4)	110[E] (50–180)	190[E] (120–270)
20–39 years							
3 (2012–2013)	328	91.3 (77.9–96.9)	17[E] (12–25)	<LOD	18[E] (6.0–30)	320[E] (<LOD–530)	630[E] (340–920)
4 (2014–2015)	362	90.8 (82.9–95.3)	13[E] (6.9–25)	0.90[E] (<LOD–1.4)	F	230 (150–310)	280[E] (94–460)
5 (2016–2017)	372	85.6 (75.8–91.9)	13[E] (8.7–20)	<LOD	F	F	F
40–59 years							
3 (2012–2013)	284	90.3 (79.6–95.7)	29[E] (17–49)	F	34[E] (14–55)	390[E] (140–630)	610[E] (230–990)
4 (2014–2015)	312	86.3 (77.7–91.9)	19[E] (10–35)	<LOD	F	250[E] (140–370)	310[E] (130–490)
5 (2016–2017)	359	89.8 (83.1–94.0)	13[E] (8.7–20)	<LOD	F	230[E] (<LOD–370)	470[E] (<LOD–690)
60–79 years							
3 (2012–2013)	314	91.6 (84.7–95.5)	28[E] (19–41)	1.6[E] (<LOD–2.5)	36[E] (13–59)	340[E] (98–590)	710[E] (310–1100)
4 (2014–2015)	360	91.6 (87.3–94.5)	20 (16–23)	1.2[E] (<LOD–1.7)	22[E] (13–31)	320[E] (<LOD–510)	620[E] (340–890)
5 (2016–2017)	353	88.4 (79.3–93.8)	23 (17–31)	<LOD	20[E] (13–28)	F	790[E] (<LOD–1100)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 9.2.4

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2339	41.9 (38.8–45.0)	—	<LOD	<LOD	F	F
4 (2014–2015)	2564	42.1 (36.7–47.6)	—	<LOD	<LOD	27[E] (14–39)	73[E] (33–110)
5 (2016–2017)	2720	39.6 (34.6–44.9)	—	<LOD	<LOD	17 (11–23)	65[E] (38–93)
Males, 3–79 years							
3 (2012–2013)	1171	30.8 (24.3–38.0)	—	<LOD	<LOD	6.9[E] (2.9–11)	14[E] (6.2–22)
4 (2014–2015)	1275	32.5 (28.4–36.9)	—	<LOD	<LOD	11 (6.9–14)	F
5 (2016–2017)	1356	29.8 (22.7–38.1)	—	<LOD	<LOD	6.5[E] (2.2–11)	F
Females, 3–79 years							
3 (2012–2013)	1168	53.3 (48.3–58.2)	—	<LOD	<LOD	49[E] (16–83)	120[E] (53–190)
4 (2014–2015)	1289	51.7 (43.9–59.4)	—	<LOD	<LOD	39[E] (14–64)	F
5 (2016–2017)	1364	49.3 (43.3–55.4)	—	<LOD	<LOD	F	F
3–5 years							
3 (2012–2013)	463	30.6 (21.3–41.8)	—	<LOD	<LOD	F	F
4 (2014–2015)	511	32.3 (24.8–40.9)	—	<LOD	<LOD	F	F
5 (2016–2017)	552	35.8 (27.3–45.4)	—	<LOD	<LOD	F	F
6–11 years							
3 (2012–2013)	481	20.5[E] (13.2–30.5)	—	<LOD	<LOD	F	6.8[E] (2.2–11)
4 (2014–2015)	514	21.2[E] (14.2–30.6)	—	<LOD	<LOD	2.0[E] (1.1–2.9)	3.4[E] (1.3–5.5)
5 (2016–2017)	540	26.3 (20.2–33.5)	—	<LOD	<LOD	F	F
12–19 years							
3 (2012–2013)	469	29.8 (21.8–39.2)	—	<LOD	<LOD	11[E] (3.8–18)	20[E] (8.3–32)
4 (2014–2015)	505	29.6 (22.2–38.2)	—	<LOD	<LOD	F	28[E] (11–45)
5 (2016–2017)	538	28.2 (22.6–34.5)	—	<LOD	<LOD	F	F
20–39 years							
3 (2012–2013)	328	44.6 (35.6–54.0)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	44.5 (35.3–54.1)	—	<LOD	<LOD	F	F
5 (2016–2017)	376	43.4 (33.4–53.9)	—	<LOD	<LOD	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	284	46.4 (38.8–54.2)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	49.6 (38.2–61.1)	—	<LOD	<LOD	F	98[E] (44–150)
5 (2016–2017)	360	34.6 (29.8–39.8)	—	<LOD	<LOD	F	F
60–79 years							
3 (2012–2013)	314	46.5 (38.2–55.1)	—	<LOD	<LOD	F	73[E] (33–110)
4 (2014–2015)	360	41.7 (36.7–47.0)	—	<LOD	<LOD	38[E] (22–55)	78[E] (44–110)
5 (2016–2017)	354	51.7 (43.7–59.7)	—	<LOD	F	57[E] (16–98)	160[E] (46–270)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 0.90 µg/L.

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 are too unreliable to be published.

Table 9.2.5

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2338	41.9 (38.8–45.0)	—	<LOD	<LOD	F	72[E] (23–120)
4 (2014–2015)	2563	42.1 (36.7–47.6)	—	<LOD	<LOD	25[E] (8.9–42)	59[E] (23–95)
5 (2016–2017)	2688	39.6 (34.6–44.9)	—	<LOD	<LOD	18[E] (5.0–31)	54[E] (17–91)
Males, 3–79 years							
3 (2012–2013)	1171	30.8 (24.3–38.0)	—	<LOD	<LOD	5.1[E] (3.0–7.2)	8.7[E] (3.5–14)
4 (2014–2015)	1274	32.5 (28.4–36.9)	—	<LOD	<LOD	6.5 (4.4–8.6)	F
5 (2016–2017)	1341	29.8 (22.7–38.1)	—	<LOD	<LOD	5.9[E] (3.0–8.7)	F
Females, 3–79 years							
3 (2012–2013)	1167	53.3 (48.3–58.2)	—	<LOD	<LOD	F	130[E] (68–180)
4 (2014–2015)	1289	51.7 (43.9–59.4)	—	<LOD	<LOD	54[E] (22–86)	120[E] (54–190)
5 (2016–2017)	1347	49.3 (43.3–55.4)	—	<LOD	<LOD	36[E] (11–61)	140[E] (80–200)
3–5 years							
3 (2012–2013)	462	30.6 (21.3–41.8)	—	<LOD	<LOD	F	F
4 (2014–2015)	511	32.3 (24.8–40.9)	—	<LOD	<LOD	F	F
5 (2016–2017)	542	35.8 (27.3–45.4)	—	<LOD	<LOD	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
3 (2012–2013)	481	20.5[E] (13.2–30.5)	—	<LOD	<LOD	3.3[E] (<LOD–5.0)	6.3[E] (3.2–9.3)
4 (2014–2015)	513	21.2[E] (14.2–30.6)	—	<LOD	<LOD	2.0[E] (1.2–2.8)	4.6[E] (2.2–7.1)
5 (2016–2017)	531	26.3 (20.2–33.5)	—	<LOD	<LOD	F	F
12–19 years							
3 (2012–2013)	469	29.8 (21.8–39.2)	—	<LOD	<LOD	F	F
4 (2014–2015)	505	29.6 (22.2–38.2)	—	<LOD	<LOD	F	F
5 (2016–2017)	531	28.2 (22.6–34.5)	—	<LOD	<LOD	F	27[E] (<LOD–46)
20–39 years							
3 (2012–2013)	328	44.6 (35.6–54.0)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	44.5 (35.3–54.1)	—	<LOD	<LOD	F	F
5 (2016–2017)	372	43.4 (33.4–53.9)	—	<LOD	<LOD	F	32[E] (<LOD–48)
40–59 years							
3 (2012–2013)	284	46.4 (38.8–54.2)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	49.6 (38.2–61.1)	—	<LOD	<LOD	41[E] (<LOD–70)	F
5 (2016–2017)	359	34.6 (29.8–39.8)	—	<LOD	<LOD	F	F
60–79 years							
3 (2012–2013)	314	46.5 (38.2–55.1)	—	<LOD	<LOD	39[E] (<LOD–63)	80 (52–110)
4 (2014–2015)	360	41.7 (36.7–47.0)	—	<LOD	<LOD	44[E] (26–62)	70[E] (29–110)
5 (2016–2017)	353	51.7 (43.7–59.7)	—	<LOD	1.6 (<LOD–2.0)	78[E] (36–120)	180[E] (69–290)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 9.2.6

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2339	79.2 (74.9–83.0)	2.9 (2.4–3.6)	<LOD	2.4 (1.8–3.0)	78[E] (47–110)	110[E] (38–190)
4 (2014–2015)	2564	78.7 (72.9–83.5)	2.5 (1.8–3.5)	<LOD	2.0[E] (1.2–2.7)	59[E] (34–85)	130[E] (67–180)
5 (2016–2017)	2720	73.7 (67.4–79.1)	1.9 (1.4–2.4)	<LOD	1.1 (0.74–1.4)	65[E] (39–91)	140 (92–180)
Males, 3–79 years							
3 (2012–2013)	1171	68.4 (60.5–75.4)	1.3 (0.94–1.8)	<LOD	0.84 (0.55–1.1)	F	F
4 (2014–2015)	1275	71.7 (65.2–77.4)	1.3 (0.96–1.8)	<LOD	0.77 (0.55–0.99)	F	F
5 (2016–2017)	1356	64.2 (55.5–72.0)	0.78 (0.63–0.97)	<LOD	0.46[E] (<LOD–0.64)	F	32[E] (15–49)
Females, 3–79 years							
3 (2012–2013)	1168	90.3 (85.9–93.5)	6.7[E] (4.2–10)	<LOD	F	100 (71–130)	150[E] (47–250)
4 (2014–2015)	1289	85.7 (79.0–90.5)	4.9[E] (3.2–7.6)	<LOD	5.6[E] (1.9–9.4)	83[E] (38–130)	170[E] (58–280)
5 (2016–2017)	1364	83.1 (76.2–88.4)	4.4 (3.1–6.2)	<LOD	4.4[E] (2.8–6.0)	110[E] (65–150)	160 (110–210)
3–5 years							
3 (2012–2013)	463	76.3 (67.5–83.3)	1.7[E] (1.1–2.6)	<LOD	1.3[E] (0.64–2.0)	28[E] (10–47)	F
4 (2014–2015)	511	81.9 (76.4–86.3)	1.5 (1.1–2.0)	<LOD	1.2[E] (0.67–1.7)	16[E] (7.3–24)	F
5 (2016–2017)	552	70.7 (59.7–79.8)	1.2[E] (0.77–1.8)	<LOD	0.87[E] (0.47–1.3)	F	34[E] (10–58)
6–11 years							
3 (2012–2013)	481	71.7 (62.9–79.2)	0.99 (0.70–1.4)	<LOD	0.71[E] (<LOD–1.2)	9.1[E] (2.9–15)	F
4 (2014–2015)	514	81.1 (76.5–84.9)	1.2 (0.99–1.6)	<LOD	0.95[E] (0.58–1.3)	11 (7.8–14)	F
5 (2016–2017)	540	70.3 (62.1–77.4)	0.96 (0.69–1.3)	<LOD	0.69 (0.48–0.90)	14[E] (6.7–21)	F
12–19 years							
3 (2012–2013)	469	82.3 (71.3–89.7)	2.5[E] (1.4–4.4)	<LOD	F	F	250[E] (84–420)
4 (2014–2015)	505	81.3 (73.7–87.1)	2.3[E] (1.6–3.3)	<LOD	1.8[E] (1.1–2.4)	55[E] (17–92)	110[E] (56–170)
5 (2016–2017)	538	70.1 (63.1–76.2)	1.4 (1.1–1.7)	<LOD	0.97 (0.67–1.3)	F	89[E] (36–140)
20–39 years							
3 (2012–2013)	328	84.9 (78.9–89.5)	3.9 (2.7–5.6)	<LOD	2.7[E] (1.0–4.4)	F	F
4 (2014–2015)	362	78.7 (65.3–88.0)	F	<LOD	F	F	F
5 (2016–2017)	376	75.5 (67.5–82.0)	2.1[E] (1.4–3.0)	<LOD	F	F	140[E] (54–230)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	284	76.6 (65.9–84.7)	2.8 (2.1–3.9)	<LOD	2.5[E] (1.4–3.7)	75[E] (23–130)	100 (73–140)
4 (2014–2015)	312	79.6 (66.9–88.3)	F	<LOD	F	F	F
5 (2016–2017)	360	77.0 (65.3–85.6)	2.0[E] (1.3–2.9)	<LOD	0.98[E] (<LOD–1.7)	71[E] (31–110)	150[E] (63–240)
60–79 years							
3 (2012–2013)	314	78.5 (70.5–84.8)	3.7[E] (2.5–5.6)	<LOD	F	79[E] (42–120)	110 (74–140)
4 (2014–2015)	360	74.5 (64.3–82.6)	3.0[E] (2.0–4.6)	<LOD	F	F	F
5 (2016–2017)	354	69.5 (59.3–78.1)	2.3 (1.6–3.1)	<LOD	1.4[E] (0.55–2.2)	100[E] (60–150)	150 (120–180)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 0.30 µg/L.

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 are too unreliable to be published.

Table 9.2.7

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2338	79.2 (74.9–83.0)	3.0 (2.5–3.7)	<LOD	2.1 (1.4–2.7)	85[E] (53–120)	130 (96–160)
4 (2014–2015)	2563	78.7 (72.9–83.5)	2.3[E] (1.6–3.3)	<LOD	1.5[E] (0.85–2.1)	63[E] (30–96)	110 (73–140)
5 (2016–2017)	2688	73.7 (67.4–79.1)	1.8 (1.4–2.3)	<LOD	0.97 (0.70–1.2)	66 (49–82)	120 (88–150)
Males, 3–79 years							
3 (2012–2013)	1171	68.4 (60.5–75.4)	1.1 (0.81–1.5)	<LOD	0.75 (0.49–1.0)	30[E] (<LOD–52)	F
4 (2014–2015)	1274	71.7 (65.2–77.4)	1.0 (0.74–1.4)	<LOD	0.70 (0.46–0.93)	F	F
5 (2016–2017)	1341	64.2 (55.5–72.0)	0.68 (0.56–0.82)	<LOD	0.46 (<LOD–0.55)	F	42[E] (18–66)
Females, 3–79 years							
3 (2012–2013)	1167	90.3 (85.9–93.5)	8.6[E] (5.6–13)	<LOD	F	120 (86–150)	190[E] (94–280)
4 (2014–2015)	1289	85.7 (79.0–90.5)	5.1[E] (3.0–8.5)	<LOD	F	87[E] (51–120)	160[E] (91–230)
5 (2016–2017)	1347	83.1 (76.2–88.4)	4.8 (3.4–6.7)	<LOD	4.5[E] (1.6–7.4)	110 (75–140)	150 (120–190)
3–5 years							
3 (2012–2013)	462	76.3 (67.5–83.3)	3.3[E] (2.1–5.2)	<LOD	2.2[E] (1.3–3.1)	F	F
4 (2014–2015)	511	81.9 (76.4–86.3)	2.6 (2.0–3.4)	<LOD	1.8[E] (1.0–2.6)	30[E] (17–43)	68[E] (20–120)
5 (2016–2017)	542	70.7 (59.7–79.8)	2.0[E] (1.3–3.0)	<LOD	1.3[E] (0.59–2.1)	24[E] (<LOD–39)	53[E] (15–92)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
3 (2012–2013)	481	71.7 (62.9–79.2)	1.3[E] (0.85–1.8)	<LOD	0.87[E] (<LOD–1.3)	F	F
4 (2014–2015)	513	81.1 (76.5–84.9)	1.4 (1.1–1.7)	<LOD	1.1 (0.74–1.4)	9.1 (6.4–12)	F
5 (2016–2017)	531	70.3 (62.1–77.4)	1.1 (0.82–1.5)	<LOD	0.85 (0.54–1.2)	14[E] (4.9–23)	F
12–19 years							
3 (2012–2013)	469	82.3 (71.3–89.7)	1.9[E] (1.1–3.2)	<LOD	1.1[E] (0.51–1.7)	F	140[E] (87–200)
4 (2014–2015)	505	81.3 (73.7–87.1)	1.7 (1.2–2.4)	<LOD	1.1[E] (0.64–1.6)	F	85[E] (42–130)
5 (2016–2017)	531	70.1 (63.1–76.2)	1.0 (0.81–1.3)	<LOD	0.64 (0.44–0.83)	F	F
20–39 years							
3 (2012–2013)	328	84.9 (78.9–89.5)	3.1 (2.3–4.1)	<LOD	1.8[E] (0.59–3.0)	F	F
4 (2014–2015)	362	78.7 (65.3–88.0)	F	<LOD	F	F	F
5 (2016–2017)	372	75.5 (67.5–82.0)	1.9 (1.3–2.7)	<LOD	F	F	130[E] (60–190)
40–59 years							
3 (2012–2013)	284	76.6 (65.9–84.7)	3.3 (2.4–4.5)	<LOD	2.6[E] (1.2–3.9)	94[E] (45–140)	120 (83–160)
4 (2014–2015)	312	79.6 (66.9–88.3)	F	<LOD	F	F	96[E] (29–160)
5 (2016–2017)	359	77.0 (65.3–85.6)	1.8[E] (1.2–2.7)	<LOD	0.95[E] (<LOD–1.4)	75 (50–100)	120 (83–150)
60–79 years							
3 (2012–2013)	314	78.5 (70.5–84.8)	4.3[E] (2.7–6.8)	<LOD	F	94[E] (51–140)	130[E] (54–210)
4 (2014–2015)	360	74.5 (64.3–82.6)	2.9[E] (2.0–4.2)	<LOD	2.6[E] (0.95–4.2)	F	190[E] (110–280)
5 (2016–2017)	353	69.5 (59.3–78.1)	2.6 (2.0–3.3)	<LOD	1.7[E] (0.85–2.5)	98[E] (58–140)	190[E] (85–300)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 9.2.8

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2339	24.6 (20.2–29.7)	—	<LOD	<LOD	F	F
4 (2014–2015)	2564	19.4 (15.7–23.8)	—	<LOD	<LOD	F	4.3[E] (2.0–6.6)
5 (2016–2017)	2720	13.1 (10.6–16.0)	—	<LOD	<LOD	0.70[E] (<LOD–1.1)	F
Males, 3–79 years							
3 (2012–2013)	1171	12.2[E] (7.8–18.7)	—	<LOD	<LOD	F	F
4 (2014–2015)	1275	11.4[E] (7.4–17.0)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	1356	5.1[E] (3.1–8.3)	—	<LOD	<LOD	<LOD	F
Females, 3–79 years							
3 (2012–2013)	1168	37.4 (31.3–44.0)	—	<LOD	<LOD	F	F
4 (2014–2015)	1289	27.5 (22.2–33.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	1364	20.9 (16.8–25.8)	—	<LOD	<LOD	1.4[E] (0.63–2.2)	F
3–5 years							
3 (2012–2013)	463	17.0[E] (10.9–25.6)	—	<LOD	<LOD	F	F
4 (2014–2015)	511	15.7[E] (10.7–22.3)	—	<LOD	<LOD	F	F
5 (2016–2017)	552	8.4[E] (4.7–14.8)	—	<LOD	<LOD	<LOD	F
6–11 years							
3 (2012–2013)	481	11.8[E] (6.6–20.4)	—	<LOD	<LOD	F	0.68[E] (0.33–1.0)
4 (2014–2015)	514	10.8[E] (7.1–16.2)	—	<LOD	<LOD	<LOD	1.1[E] (0.30–1.8)
5 (2016–2017)	540	7.2[E] (3.8–13.1)	—	<LOD	<LOD	<LOD	F
12–19 years							
3 (2012–2013)	469	23.7[E] (16.0–33.5)	—	<LOD	<LOD	2.5[E] (0.85–4.2)	F
4 (2014–2015)	505	19.7 (14.3–26.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	538	9.8 (7.3–13.0)	—	<LOD	<LOD	<LOD	F
20–39 years							
3 (2012–2013)	328	28.3 (21.3–36.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	17.5[E] (11.2–26.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	376	13.0[E] (6.9–23.2)	—	<LOD	<LOD	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	284	26.4 (18.7–35.9)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	21.2[E] (12.5–33.6)	—	<LOD	<LOD	F	F
5 (2016–2017)	360	15.5 (11.0–21.4)	—	<LOD	<LOD	1.3[E] (0.53–2.1)	F
60–79 years							
3 (2012–2013)	314	23.7 (16.5–32.7)	—	<LOD	<LOD	F	F
4 (2014–2015)	360	23.1 (17.3–30.1)	—	<LOD	<LOD	F	6.8 (4.4–9.1)
5 (2016–2017)	354	13.8 (10.4–18.0)	—	<LOD	<LOD	0.65[E] (<LOD–1.1)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 0.30 µg/L.

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 are too unreliable to be published.

Table 9.2.9

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 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2338	24.6 (20.2–29.7)	—	<LOD	<LOD	F	16[E] (4.6–27)
4 (2014–2015)	2563	19.4 (15.7–23.8)	—	<LOD	<LOD	F	4.2[E] (1.5–6.8)
5 (2016–2017)	2688	13.1 (10.6–16.0)	—	<LOD	<LOD	0.87 (<LOD–1.2)	2.2[E] (0.88–3.6)
Males, 3–79 years							
3 (2012–2013)	1171	12.2[E] (7.8–18.7)	—	<LOD	<LOD	0.52[E] (<LOD–0.84)	F
4 (2014–2015)	1274	11.4[E] (7.4–17.0)	—	<LOD	<LOD	<LOD	0.79[E] (<LOD–1.2)
5 (2016–2017)	1341	5.1[E] (3.1–8.3)	—	<LOD	<LOD	<LOD	0.79[E] (<LOD–1.3)
Females, 3–79 years							
3 (2012–2013)	1167	37.4 (31.3–44.0)	—	<LOD	<LOD	F	29[E] (<LOD–49)
4 (2014–2015)	1289	27.5 (22.2–33.4)	—	<LOD	<LOD	F	9.2[E] (<LOD–15)
5 (2016–2017)	1347	20.9 (16.8–25.8)	—	<LOD	<LOD	1.6[E] (0.90–2.4)	F
3–5 years							
3 (2012–2013)	462	17.0[E] (10.9–25.6)	—	<LOD	<LOD	F	F
4 (2014–2015)	511	15.7[E] (10.7–22.3)	—	<LOD	<LOD	F	3.1[E] (<LOD–5.1)
5 (2016–2017)	542	8.4[E] (4.7–14.8)	—	<LOD	<LOD	<LOD	1.3[E] (<LOD–1.8)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
3 (2012–2013)	481	11.8[E] (6.6–20.4)	—	<LOD	<LOD	0.73[E] (<LOD–1.0)	0.99 (0.74–1.2)
4 (2014–2015)	513	10.8[E] (7.1–16.2)	—	<LOD	<LOD	<LOD	0.81[E] (0.30–1.3)
5 (2016–2017)	531	7.2[E] (3.8–13.1)	—	<LOD	<LOD	<LOD	F
12–19 years							
3 (2012–2013)	469	23.7[E] (16.0–33.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	505	19.7 (14.3–26.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	531	9.8 (7.3–13.0)	—	<LOD	<LOD	<LOD	F
20–39 years							
3 (2012–2013)	328	28.3 (21.3–36.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	362	17.5[E] (11.2–26.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	372	13.0[E] (6.9–23.2)	—	<LOD	<LOD	F	F
40–59 years							
3 (2012–2013)	284	26.4 (18.7–35.9)	—	<LOD	<LOD	F	F
4 (2014–2015)	312	21.2[E] (12.5–33.6)	—	<LOD	<LOD	F	F
5 (2016–2017)	359	15.5 (11.0–21.4)	—	<LOD	<LOD	1.6[E] (0.62–2.5)	F
60–79 years							
3 (2012–2013)	314	23.7 (16.5–32.7)	—	<LOD	<LOD	F	F
4 (2014–2015)	360	23.1 (17.3–30.1)	—	<LOD	<LOD	4.2[E] (<LOD–6.5)	6.7[E] (2.1–11)
5 (2016–2017)	353	13.8 (10.4–18.0)	—	<LOD	<LOD	0.87 (<LOD–1.0)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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SUMMARY AND RESULTS FOR NICOTINE 10

10.1 NICOTINE

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 delivery products, such as nicotine gum, patches, lozenges, inhalers, buccal sprays, and vaping products (Etter et al, 2011).

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

Inhalation is the most effective intake route; on average, 60% to 80% of nicotine is absorbed through the lungs (Iwase et al., 1991). Nicotine absorption through the mouth varies with the pH of the smoke or nicotine delivery product, increasing as alkalinity rises (Benowitz et al., 2009). Nicotine can also be absorbed through the skin and gastrointestinal tract, but at a much lower efficiency compared with inhalation (Karaconji, 2005). Once inside the body, approximately 70% to 80% of nicotine is metabolized into cotinine, primarily by a liver cytochrome P-450 enzyme. It has a half-life of 10 to 20 hours and can remain in the body at detectable levels for up to seven 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). It has also been shown to be a biomarker of exposure to nicotine via other types of nicotine delivery products, such as e-cigarettes (Schick et al., 2017; Vélez de Mendizábal et al., 2015). It should be noted that there are no validated biomarkers that can differentiate among the use of various combustible products (e.g., cigars, cigarillos, water pipes, and cigarettes), and there are no validated biomarkers that are specific to nicotine-containing or nicotine-free vaping products (Schick et al., 2017).

Nicotine reaches the brain rapidly following inhalation and can cause several reactions in the body, such as: increased heart rate and blood pressure, muscle relaxation, altered brain activity, and constriction of blood vessels leading to a drop in temperature of the hands and feet (Health Canada, 2013). Other effects may include nausea, weakness, stomach cramps, and headache, with symptoms lessening as nicotine tolerance is developed. Nicotine mimics the effects of acetylcholine in the nervous system. Through the release of dopamine and effects on other neurotransmitters, it can activate areas of the brain that are associated with feelings of alertness, calmness, and pleasure (Pandey et al., 2018). As the body builds tolerance to nicotine, the delivery product must continue to be used for the effects to last; use over time may lead to dependence and addiction (Health Canada, 2013). The use of nicotine-containing products is associated with exposure to other chemicals that have their own effects. For example, tobacco smoke contains more than 4,000 chemicals, including at least 70 that cause, initiate, or promote cancer, and others that contribute to adverse

health effects, such as emphysema, heart disease, and increased risk of asthma (CDC, 2004; Health Canada, 2011; IARC, 2004). Levels of cotinine in the blood and urine of non-smokers have been correlated with some adverse health effects related to environmental tobacco smoke exposure. 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 sales of tobacco products and electronic nicotine delivery systems 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). In 2018, Health Canada enacted the *Tobacco and Vaping Products Act*, which amends the *Canada Consumer Product Safety Act* to allow the effective regulation of vaping products as well as the ability to establish plain and standardized appearance requirements for tobacco product packages (Health Canada, 2018). This legislation aims to protect young people and non-smokers from inducements to nicotine addiction and tobacco use, and to enhance public awareness of the health and safety hazards posed by tobacco and vaping products.

The First Nations Biomonitoring Initiative (FNBI) was a nationally representative biomonitoring study of adult First Nations peoples living on reserves south of the 60° parallel (AFN, 2013). It comprised 13 randomly selected First Nations 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 is exposed to secondhand smoke. The study found detectable cotinine levels (≥1.1 ng/ml) in non-smokers, indicating secondhand smoke exposure, and reported that children and adolescent subpopulations had higher levels compared with adults (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 one-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; greater concentrations 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. This indicates 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), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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; a non-smoker is defined as someone who does not currently smoke, and has either never smoked or 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 10.1.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	14.3 (11.5–17.7)	—	<LOD	<LOD	2.6[E] (<LOD–4.4)	F
3 (2012–2013)	4978	9.3 (6.9–12.5)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	4907	11.3 (9.2–13.8)	—	<LOD	<LOD	F	F
5 (2016–2017)	4928	9.8 (7.2–13.2)	—	<LOD	<LOD	<LOD	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2594	17.9 (14.3–22.2)	—	<LOD	<LOD	F	F
3 (2012–2013)	2444	11.0 (7.6–15.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	2446	12.0 (9.6–14.8)	—	<LOD	<LOD	F	F
5 (2016–2017)	2440	12.6[E] (8.1–19.0)	—	<LOD	<LOD	F	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2874	10.9 (8.6–13.7)	—	<LOD	<LOD	1.5[E] (<LOD–2.5)	F
3 (2012–2013)	2534	7.9 (5.7–10.8)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	2461	10.6 (7.6–14.7)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	2488	7.2 (5.6–9.4)	—	<LOD	<LOD	<LOD	1.7[E] (<LOD–2.7)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	573	17.3[E] (9.2–30.1)	—	<LOD	<LOD	F	F
3 (2012–2013)	522	10.5[E] (6.7–16.0)	—	<LOD	<LOD	F	F
4 (2014–2015)	512	16.7[E] (11.2–24.2)	—	<LOD	<LOD	2.3 (1.7–3.0)	F
5 (2016–2017)	543	9.6[E] (5.7–15.6)	—	<LOD	<LOD	<LOD	F
6–11 years							
1 (2007–2009)	1045	15.9 (12.6–19.8)	—	<LOD	<LOD	3.9[E] (1.9–5.8)	10[E] (5.7–14)
2 (2009–2011)	1061	16.9 (12.4–22.8)	—	<LOD	<LOD	4.9[E] (1.9–7.9)	12[E] (6.3–18)
3 (2012–2013)	1007	10.5 (7.3–14.9)	—	<LOD	<LOD	F	7.1[E] (2.7–11)
4 (2014–2015)	1008	9.6[E] (6.3–14.5)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	991	9.2[E] (5.8–14.2)	—	<LOD	<LOD	<LOD	3.3[E] (1.1–5.5)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	882	22.4 (15.3–31.7)	—	<LOD	<LOD	8.3[E] (3.8–13)	19[E] (8.3–30)
2 (2009–2011)	928	21.5 (16.6–27.4)	—	<LOD	<LOD	F	F
3 (2012–2013)	889	16.6 (12.0–22.6)	—	<LOD	<LOD	F	13[E] (7.6–19)
4 (2014–2015)	901	14.1 (10.7–18.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	903	16.5 (12.1–22.0)	—	<LOD	<LOD	F	F
20–39 years							
1 (2007–2009)	874	14.8 (11.0–19.7)	—	<LOD	<LOD	F	F
2 (2009–2011)	1009	20.5[E] (14.0–29.0)	—	<LOD	<LOD	F	F
3 (2012–2013)	792	7.3[E] (4.2–12.3)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	785	13.2 (9.1–18.7)	—	<LOD	<LOD	F	F
5 (2016–2017)	809	11.0[E] (6.2–18.8)	—	<LOD	<LOD	F	F
40–59 years							
1 (2007–2009)	947	11.4 (9.0–14.2)	—	<LOD	<LOD	F	F
2 (2009–2011)	972	8.1[E] (5.6–11.7)	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	851	10.1[E] (6.2–16.0)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	827	12.2[E] (7.9–18.3)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	814	7.1[E] (4.1–12.1)	—	<LOD	<LOD	<LOD	F
60–79 years							
1 (2007–2009)	956	8.8 (6.3–12.2)	—	<LOD	<LOD	<LOD	F
2 (2009–2011)	925	8.9 (6.5–12.1)	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	917	6.5[E] (4.2–10.0)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	874	5.9 (4.5–7.7)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	868	9.0 (6.4–12.4)	—	<LOD	<LOD	<LOD	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 1, 1, 1.1, 1.1, and 1.1 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 10.1.2

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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	14.3 (11.5–17.7)	—	<LOD	<LOD	3.3 (<LOD–4.4)	F
3 (2012–2013)	4976	9.3 (6.9–12.5)	—	<LOD	<LOD	<LOD	6.1[E] (<LOD–10)
4 (2014–2015)	4906	11.3 (9.2–13.8)	—	<LOD	<LOD	2.6 (<LOD–3.5)	F
5 (2016–2017)	4914	9.8 (7.2–13.2)	—	<LOD	<LOD	<LOD	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2588	17.9 (14.3–22.2)	—	<LOD	<LOD	3.9[E] (<LOD–5.9)	F
3 (2012–2013)	2444	11.0 (7.6–15.5)	—	<LOD	<LOD	2.4[E] (<LOD–3.3)	F
4 (2014–2015)	2445	12.0 (9.6–14.8)	—	<LOD	<LOD	2.6[E] (<LOD–4.3)	F
5 (2016–2017)	2435	12.6[E] (8.1–19.0)	—	<LOD	<LOD	F	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	2867	10.9 (8.6–13.7)	—	<LOD	<LOD	3.0 (<LOD–3.9)	F
3 (2012–2013)	2532	7.9 (5.7–10.8)	—	<LOD	<LOD	<LOD	5.2[E] (<LOD–7.8)
4 (2014–2015)	2461	10.6 (7.6–14.7)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	2479	7.2 (5.6–9.4)	—	<LOD	<LOD	<LOD	4.1[E] (<LOD–5.9)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	572	17.3[E] (9.2–30.1)	—	<LOD	<LOD	F	F
3 (2012–2013)	521	10.5[E] (6.7–16.0)	—	<LOD	<LOD	5.6[E] (<LOD–7.7)	F
4 (2014–2015)	512	16.7[E] (11.2–24.2)	—	<LOD	<LOD	3.7[E] (2.2–5.2)	F
5 (2016–2017)	541	9.6[E] (5.7–15.6)	—	<LOD	<LOD	<LOD	F
6–11 years							
1 (2007–2009)	1042	15.9 (12.6–19.8)	—	<LOD	<LOD	6.2[E] (1.9–10)	F
2 (2009–2011)	1059	16.9 (12.4–22.8)	—	<LOD	<LOD	5.2[E] (1.9–8.5)	12[E] (5.4–18)
3 (2012–2013)	1007	10.5 (7.3–14.9)	—	<LOD	<LOD	3.5[E] (<LOD–5.8)	7.7[E] (2.6–13)
4 (2014–2015)	1007	9.6[E] (6.3–14.5)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	990	9.2[E] (5.8–14.2)	—	<LOD	<LOD	<LOD	5.7 (3.9–7.6)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	881	22.4 (15.3–31.7)	—	<LOD	<LOD	7.9[E] (4.6–11)	F
2 (2009–2011)	926	21.5 (16.6–27.4)	—	<LOD	<LOD	F	F
3 (2012–2013)	889	16.6 (12.0–22.6)	—	<LOD	<LOD	3.2[E] (<LOD–5.5)	F
4 (2014–2015)	901	14.1 (10.7–18.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	900	16.5 (12.1–22.0)	—	<LOD	<LOD	F	F
20–39 years							
1 (2007–2009)	871	14.8 (11.0–19.7)	—	<LOD	<LOD	4.5[E] (<LOD–7.4)	F
2 (2009–2011)	1007	20.5[E] (14.0–29.0)	—	<LOD	<LOD	F	F
3 (2012–2013)	792	7.3[E] (4.2–12.3)	—	<LOD	<LOD	<LOD	3.3[E] (<LOD–5.2)
4 (2014–2015)	785	13.2 (9.1–18.7)	—	<LOD	<LOD	F	F
5 (2016–2017)	807	11.0[E] (6.2–18.8)	—	<LOD	<LOD	F	F
40–59 years							
1 (2007–2009)	944	11.4 (9.0–14.2)	—	<LOD	<LOD	4.6[E] (<LOD–6.4)	F
2 (2009–2011)	970	8.1[E] (5.6–11.7)	—	<LOD	<LOD	<LOD	4.7[E] (<LOD–7.8)
3 (2012–2013)	850	10.1[E] (6.2–16.0)	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	827	12.2[E] (7.9–18.3)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	810	7.1[E] (4.1–12.1)	—	<LOD	<LOD	<LOD	F
60–79 years							
1 (2007–2009)	956	8.8 (6.3–12.2)	—	<LOD	<LOD	<LOD	F
2 (2009–2011)	921	8.9 (6.5–12.1)	—	<LOD	<LOD	<LOD	F
3 (2012–2013)	917	6.5[E] (4.2–10.0)	—	<LOD	<LOD	<LOD	4.1[E] (<LOD–6.8)
4 (2014–2015)	874	5.9 (4.5–7.7)	—	<LOD	<LOD	<LOD	F
5 (2016–2017)	866	9.0 (6.4–12.4)	—	<LOD	<LOD	<LOD	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 10.1.3

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
1 (2007–2009)	805	96.8 (94.1–98.3)	590 (420–820)	F	1000 (810–1200)	2200 (2000–2400)	2600 (2300–2900)
2 (2009–2011)	819	94.5 (91.0–96.7)	490 (340–700)	F	1000 (810–1200)	2200 (1900–2500)	2600 (2100–3100)
3 (2012–2013)	701	95.0 (91.0–97.3)	490 (410–590)	F	990 (900–1100)	2000 (1600–2300)	2300 (2000–2600)
4 (2014–2015)	667	95.5 (91.2–97.7)	550 (420–710)	F	1000 (830–1200)	2300 (1900–2700)	2800 (2400–3200)
5 (2016–2017)	571	96.8 (93.5–98.5)	580 (460–730)	F	910 (790–1000)	1900 (1700–2100)	2300 (2000–2600)
Males, 12–79 years							
1 (2007–2009)	406	96.0 (91.2–98.3)	660[E] (400–1100)	F	1200 (920–1500)	2300 (2000–2600)	2800 (2400–3300)
2 (2009–2011)	425	94.6 (89.3–97.4)	470[E] (280–770)	F	1000 (780–1200)	2300 (1900–2700)	2900 (2300–3500)
3 (2012–2013)	387	94.4 (86.6–97.8)	460 (340–630)	F	990 (820–1100)	2100 (1700–2500)	2400 (2100–2600)
4 (2014–2015)	359	97.4 (89.3–99.4)	610 (470–800)	F	980 (830–1100)	2200 (1800–2500)	2600 (1800–3400)
5 (2016–2017)	312	97.8 (96.3–98.7)	660 (520–830)	F	940 (740–1100)	1900 (1600–2200)	2300 (1700–2800)
Females, 12–79 years							
1 (2007–2009)	399	97.6 (95.4–98.8)	520 (390–700)	F	860 (640–1100)	2100 (1900–2300)	2500 (2300–2700)
2 (2009–2011)	394	94.4 (87.2–97.6)	510[E] (320–810)	F	1000 (720–1300)	2100 (1800–2400)	2400 (1900–2900)
3 (2012–2013)	314	95.9 (89.8–98.4)	550 (380–790)	F	990 (760–1200)	1700 (1200–2300)	2100 (1700–2500)
4 (2014–2015)	308	92.8 (82.8–97.2)	470[E] (250–870)	F	1100 (820–1400)	2500 (1900–3100)	2800 (2500–3100)
5 (2016–2017)	259	95.1 (84.7–98.5)	460[E] (280–760)	F	850 (640–1100)	1800 (1500–2100)	2300 (1700–3000)
12–19 years							
1 (2007–2009)	102	90.7 (81.1–95.7)	160[E] (78–330)	<LOD	F	1600 (1400–1900)	X
2 (2009–2011)	102	82.4 (59.2–93.8)	F	<LOD	F	1700 (1200–2300)	X
3 (2012–2013)	98	84.1 (68.9–92.6)	F	X	F	X	X
4 (2014–2015)	73	82.2 (53.7–94.8)	F	X	430[E] (260–610)	X	X
5 (2016–2017)	57	95.2 (83.4–98.7)	240[E] (120–470)	X	430[E] (200–660)	X	X
20–39 years							
1 (2007–2009)	300	96.2 (88.8–98.8)	500[E] (300–850)	F	930 (620–1200)	2000 (1800–2200)	2500 (2100–2900)
2 (2009–2011)	311	92.1 (85.0–95.9)	400[E] (260–630)	F	850 (570–1100)	2200 (1600–2900)	2900 (2200–3600)
3 (2012–2013)	254	93.5 (76.4–98.4)	310[E] (190–520)	F	700[E] (350–1100)	1600 (1300–1900)	2000 (1600–2400)
4 (2014–2015)	271	93.0 (81.2–97.6)	360[E] (220–600)	F	970 (620–1300)	2400 (1600–3200)	2900 (2200–3500)
5 (2016–2017)	220	95.4 (90.2–97.9)	520[E] (340–780)	F	1000 (730–1300)	1900 (1700–2000)	2100 (1900–2200)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	275	98.4 (96.1–99.3)	830 (610–1100)	F	1200 (910–1500)	2500 (2200–2800)	2800 (2400–3100)
2 (2009–2011)	253	99.2 (96.6–99.8)	800[E] (480–1300)	F	1400 (1000–1700)	2200 (1900–2600)	2600 (2000–3300)
3 (2012–2013)	228	96.9 (89.7–99.1)	770 (550–1100)	340[E] (150–530)	1000 (890–1200)	2100 (1700–2600)	2300 (2000–2700)
4 (2014–2015)	208	98.7 (95.0–99.7)	880 (770–1000)	360[E] (190–540)	1100 (870–1400)	2600 (1900–3200)	2900 (2400–3300)
5 (2016–2017)	182	97.6 (86.6–99.6)	630[E] (430–920)	F	910 (730–1100)	2000 (1600–2500)	X
60–79 years							
1 (2007–2009)	128	96.7 (86.1–99.3)	650[E] (430–980)	F	860 (600–1100)	2200 (1900–2400)	X
2 (2009–2011)	153	94.1 (75.3–98.8)	F	F	980 (720–1200)	1800 (1500–2000)	X
3 (2012–2013)	121	99.5 (96.5–99.9)	940 (800–1100)	390[E] (240–540)	990 (830–1200)	2100 (1400–2700)	X
4 (2014–2015)	115	99.0 (95.1–99.8)	920 (720–1200)	440[E] (250–630)	990[E] (620–1400)	1900 (1500–2200)	X
5 (2016–2017)	112	99.4 (91.7–100)	850 (640–1100)	400[E] (160–640)	910 (710–1100)	1900[E] (970–2800)	X

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, 3, 4, and 5 are 1, 1, 1.1, 1.1, and 1.1 µg/L, respectively.

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 are too unreliable to be published.

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

Table 10.1.4

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
1 (2007–2009)	803	96.8 (94.1–98.3)	650 (480–890)	F	1000 (830–1200)	3000 (2500–3500)	4400 (3500–5300)
2 (2009–2011)	816	94.5 (91.0–96.7)	430[E] (290–630)	F	840 (620–1100)	2700 (1800–3700)	3800[E] (2300–5300)
3 (2012–2013)	701	95.0 (91.0–97.3)	440 (340–570)	F	750 (590–900)	2600[E] (1600–3700)	3900[E] (2100–5800)
4 (2014–2015)	666	95.5 (91.2–97.7)	480 (360–630)	F	780 (650–910)	2500 (1700–3300)	3300 (2900–3700)
5 (2016–2017)	571	96.8 (93.5–98.5)	590 (470–730)	130[E] (36–230)	830 (700–970)	2700 (2200–3100)	3400 (2700–4100)
Males, 12–79 years							
1 (2007–2009)	405	96.0 (91.2–98.3)	560[E] (360–880)	F	930 (680–1200)	2300 (1900–2700)	3200 (2300–4200)
2 (2009–2011)	425	94.6 (89.3–97.4)	370[E] (210–620)	F	730 (480–980)	2700[E] (1600–3700)	3700[E] (2300–5100)
3 (2012–2013)	387	94.4 (86.6–97.8)	360[E] (250–520)	F	710 (500–920)	2300 (1500–3100)	3000[E] (1900–4100)
4 (2014–2015)	358	97.4 (89.3–99.4)	500 (410–610)	F	770 (630–900)	2900[E] (1600–4200)	3300 (2500–4200)
5 (2016–2017)	312	97.8 (96.3–98.7)	600 (470–760)	F	830 (650–1000)	2400 (1600–3200)	3300 (2300–4300)
Females, 12–79 years							
1 (2007–2009)	398	97.6 (95.4–98.8)	780 (590–1000)	F	1100 (900–1400)	3700 (2900–4500)	5500 (4300–6600)
2 (2009–2011)	391	94.4 (87.2–97.6)	520[E] (300–890)	F	1000 (650–1400)	F	4800[E] (2300–7400)
3 (2012–2013)	314	95.9 (89.8–98.4)	600 (420–850)	F	860[E] (510–1200)	3200[E] (1000–5300)	4900 (3300–6400)
4 (2014–2015)	308	92.8 (82.8–97.2)	450[E] (240–850)	F	830[E] (440–1200)	2500 (1800–3100)	F
5 (2016–2017)	259	95.1 (84.7–98.5)	570[E] (360–890)	F	850[E] (510–1200)	2800[E] (1600–4000)	3800 (2900–4700)
12–19 years							
1 (2007–2009)	102	90.7 (81.1–95.7)	120[E] (58–250)	<LOD	290[E] (<LOD–470)	1400[E] (600–2200)	X
2 (2009–2011)	102	82.4 (59.2–93.8)	F	<LOD	F	1300 (990–1500)	X
3 (2012–2013)	98	84.1 (68.9–92.6)	F	X	F	X	X
4 (2014–2015)	72	82.2 (53.7–94.8)	F	X	F	X	X
5 (2016–2017)	57	95.2 (83.4–98.7)	F	X	F	X	X
20–39 years							
1 (2007–2009)	299	96.2 (88.8–98.8)	510[E] (310–840)	F	850 (560–1100)	2200 (1900–2600)	2500 (1900–3000)
2 (2009–2011)	311	92.1 (85.0–95.9)	330[E] (200–530)	F	710 (470–940)	2300 (1500–3000)	3200[E] (1700–4700)
3 (2012–2013)	254	93.5 (76.4–98.4)	230[E] (120–410)	F	520[E] (310–720)	1500[E] (830–2200)	2100[E] (1300–2900)
4 (2014–2015)	271	93.0 (81.2–97.6)	300[E] (170–520)	F	600 (390–800)	2300[E] (1200–3400)	3200 (2300–4200)
5 (2016–2017)	220	95.4 (90.2–97.9)	420[E] (270–640)	F	640 (480–810)	1800[E] (960–2700)	1900 (1400–2400)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	275	98.4 (96.1–99.3)	1000 (810–1300)	F	1300 (920–1600)	4100 (2900–5400)	5500 (4400–6600)
2 (2009–2011)	251	99.2 (96.6–99.8)	710[E] (400–1200)	F	990[E] (560–1400)	3400[E] (1400–5400)	4900[E] (2800–7000)
3 (2012–2013)	228	96.9 (89.7–99.1)	840[E] (520–1300)	390[E] (190–580)	940[E] (570–1300)	3500[E] (1500–5500)	5200[E] (2500–7800)
4 (2014–2015)	208	98.7 (95.0–99.7)	780 (610–1000)	210[E] (120–300)	1000 (740–1300)	3000 (2200–3700)	3300 (2700–4000)
5 (2016–2017)	182	97.6 (86.6–99.6)	850[E] (560–1300)	F	1200 (840–1600)	3500 (2700–4300)	X
60–79 years							
1 (2007–2009)	127	96.7 (86.1–99.3)	840[E] (530–1300)	F	1300 (1000–1500)	3200 (2100–4300)	X
2 (2009–2011)	152	94.1 (75.3–98.8)	F	F	1000 (700–1400)	3000[E] (1700–4300)	X
3 (2012–2013)	121	99.5 (96.5–99.9)	960 (730–1200)	390 (270–500)	960[E] (530–1400)	3100[E] (1600–4700)	X
4 (2014–2015)	115	99.0 (95.1–99.8)	980 (780–1200)	400[E] (250–560)	1100 (820–1400)	2100 (1700–2500)	X
5 (2016–2017)	112	99.4 (91.7–100)	970 (720–1300)	330[E] (160–510)	1100 (980–1200)	2700 (1900–3400)	X

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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

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SUMMARY AND RESULTS FOR ACRYLAMIDE 11

11.1 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, polyacrylamides are used as coagulants and flocculants 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 in certain foods as a reaction between naturally present components when foods are processed or cooked at high temperatures (Health Canada, 2009a). It is formed mainly in carbohydrate-rich, plant-based foods, such as potatoes and grains. The highest concentrations have been detected in potato chips and french fries, acrylamide has been found in other foods as well (Health Canada, 2012).

Acrylamide may enter the environment during production and industrial use (ATSDR, 2012). The main source of acrylamide in drinking water is through the release of residual monomers from polyacrylamides used as clarifiers in drinking water treatment processes (ATSDR, 2012). Acrylamide is also 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 (ATSDR, 2012; Environment Canada and Health Canada, 2009b). Inhalation of tobacco smoke, including second-hand smoke, is also a major source of inhalation exposure for the general population; tobacco smoke may be the main source of acrylamide exposure for some smokers (ATSDR, 2012; Environment Canada and Health Canada, 2009b; EFSA CONTAM Panel, 2015). Compared with food and cigarettes, exposure from other sources (e.g., drinking water, air, consumer product use) is very low (Environment Canada and Health Canada, 2009b). 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 also undergo 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 has been reported to cause neurotoxicity in humans. 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 have shown that acrylamide is genotoxic and carcinogenic (Environment Canada and Health Canada, 2009b; FAO/WHO, 2006). Reviews of existing epidemiological studies have found 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 animal studies, the International Agency for Research on Cancer (IARC) has classified acrylamide as a Group 2A probable carcinogen (IARC, 1994). Further, on the basis of available evidence from animal studies, the Joint Food and Agriculture organization of the United Nations (FAO) and World Health Organization (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). Similarly, an assessment by the European Food Safety Authority (ESFA) concluded that acrylamide in food potentially increases the risk of developing cancer for consumers in all age groups (EFSA CONTAM Panel, 2015).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether acrylamide may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment Canada and Health Canada, 2009b). The assessment concluded, on the basis of carcinogenic potential, that acrylamide is toxic under CEPA 1999, as it is considered harmful to human health (Environment Canada and Health Canada, 2009b). Acrylamide is 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 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 efforts to reduce acrylamide in processed foods (Health Canada, 2012). Data on the occurrence of acrylamide in foods available for sale in Canada do not demonstrate a decreasing trend in acrylamide concentrations in the food types that can significantly contribute to dietary acrylamide exposure; therefore, continued mitigation efforts are supported (Health Canada, 2017). 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, 2013).

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 treated drinking water (NSF International, 2015; NSF International, 2016). 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, 2018b). Acrylamide is identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018a).

In a study carried out in Montreal 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 globin and 45.6 pmol/g globin, respectively (Brisson et al., 2014).

Acrylamide and its metabolite glycidamide were analyzed as haemoglobin adducts in the whole blood of CHMS participants aged 3–79 years in cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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 11.1.1

Acrylamide — Geometric means and selected percentiles of haemoglobin adduct concentrations (pmol/g Hb) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2492	100	73 (65–82)	35 (30–40)	64 (57–70)	190 (160–230)	240 (190–290)
4 (2014–2015)	2529	100 (99.5–100)	67 (62–73)	38 (35–41)	60 (55–66)	150 (130–180)	200 (180–230)
5 (2016–2017)	2573	100	73 (68–78)	39 (33–44)	65 (61–69)	160 (130–180)	220 (200–250)
Males, 3–79 years							
3 (2012–2013)	1225	100	79 (69–90)	36 (31–40)	68 (61–75)	200 (150–260)	270[E] (160–380)
4 (2014–2015)	1267	99.9 (98.9–100)	70 (62–79)	37 (33–42)	64 (57–71)	170[E] (110–230)	220 (180–250)
5 (2016–2017)	1284	100	81 (74–89)	39 (33–44)	72 (65–79)	200 (160–230)	260[E] (140–380)
Females, 3–79 years							
3 (2012–2013)	1267	100	68 (59–78)	35 (29–41)	60 (51–69)	180 (130–230)	210 (180–250)
4 (2014–2015)	1262	100	65 (58–72)	38 (36–41)	58 (53–62)	140 (100–180)	180 (140–220)
5 (2016–2017)	1289	100	66 (61–71)	38 (32–45)	62 (58–65)	120 (96–140)	160 (120–210)
3–5 years							
3 (2012–2013)	471	100	59 (55–64)	39 (35–43)	59 (55–63)	87 (73–100)	100 (82–120)
4 (2014–2015)	484	100	60 (56–65)	37 (32–43)	61 (55–66)	96 (84–110)	100 (83–120)
5 (2016–2017)	479	100	69 (63–75)	44 (39–48)	69 (61–76)	100 (91–110)	120 (100–130)
6–11 years							
3 (2012–2013)	505	100	61 (57–65)	37 (34–41)	62 (58–67)	100 (88–110)	110 (98–120)
4 (2014–2015)	507	100	62 (59–66)	42 (39–45)	62 (58–66)	90 (83–96)	100 (94–110)
5 (2016–2017)	507	100	71 (67–74)	47 (43–50)	70 (65–74)	100 (94–110)	130 (110–150)
12–19 years							
3 (2012–2013)	507	100	63 (59–67)	37 (31–42)	57 (53–61)	110 (87–130)	170[E] (96–240)
4 (2014–2015)	505	100	63 (55–72)	37 (33–42)	60 (51–70)	100 (83–120)	120 (91–160)
5 (2016–2017)	530	100	68 (61–76)	42 (35–49)	64 (59–70)	100 (82–120)	140 (110–180)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
3 (2012–2013)	348	100	80 (65–97)	34 (24–43)	74 (59–89)	190 (130–260)	260 (190–340)
4 (2014–2015)	363	100	70 (60–80)	37 (33–41)	61 (53–70)	170 (120–220)	210 (170–250)
5 (2016–2017)	363	100	83 (72–97)	37 (27–47)	74 (63–85)	220 (170–280)	400[E] (170–640)
40–59 years							
3 (2012–2013)	311	100	83 (67–100)	35 (24–47)	66 (49–82)	230 (180–290)	330 (210–450)
4 (2014–2015)	312	99.9 (98.3–100)	71 (62–80)	38 (34–42)	60 (50–70)	180 (130–230)	250 (170–330)
5 (2016–2017)	345	100	69 (62–78)	39 (35–43)	58 (48–68)	170 (130–210)	220 (200–240)
60–79 years							
3 (2012–2013)	350	100	63 (59–68)	34 (29–40)	62 (59–65)	130 (100–150)	160 (130–190)
4 (2014–2015)	358	100	63 (56–71)	34 (26–43)	59 (53–65)	150 (110–190)	190 (170–210)
5 (2016–2017)	349	100	69 (65–73)	38 (32–44)	65 (61–69)	130 (91–170)	170 (140–200)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 11 pmol/g Hb.

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 11.1.2

Glycidamide — Geometric means and selected percentiles of haemoglobin adduct concentrations (pmol/g Hb) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2492	97.8 (94.9–99.1)	68 (62–75)	36 (34–38)	65 (59–70)	150 (120–180)	190 (150–220)
4 (2014–2015)	2529	97.4 (93.7–98.9)	60 (54–67)	34 (30–37)	57 (52–62)	120 (100–140)	170 (150–200)
5 (2016–2017)	2573	99.2 (97.3–99.8)	74 (69–80)	39 (34–43)	72 (67–77)	130 (110–160)	180 (140–210)
Males, 3–79 years							
3 (2012–2013)	1225	97.3 (92.6–99.1)	69 (62–77)	37 (35–38)	66 (58–74)	170 (120–210)	210 (160–260)
4 (2014–2015)	1267	97.0 (93.6–98.6)	61 (53–70)	33 (27–39)	58 (50–66)	130 (100–160)	170 (130–200)
5 (2016–2017)	1284	98.5 (94.8–99.6)	76 (68–85)	37 (30–44)	74 (66–82)	150 (130–170)	210 (160–270)
Females, 3–79 years							
3 (2012–2013)	1267	98.2 (90.5–99.7)	67 (60–74)	36 (32–40)	64 (57–71)	130 (100–160)	160 (120–200)
4 (2014–2015)	1262	97.8 (92.1–99.4)	59 (53–67)	34 (31–37)	56 (51–62)	110 (81–140)	170 (110–240)
5 (2016–2017)	1289	100 (99.7–100)	72 (68–78)	42 (38–46)	71 (66–75)	120 (100–130)	150 (110–200)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
3 (2012–2013)	471	100	80 (75–85)	51 (43–59)	78 (74–81)	120 (110–130)	140 (120–150)
4 (2014–2015)	484	99.9 (99.5–100)	76 (69–84)	49 (44–53)	73 (65–82)	120 (100–130)	140 (110–180)
5 (2016–2017)	479	100	93 (85–100)	59 (48–69)	92 (83–100)	140 (120–160)	170 (150–190)
6–11 years							
3 (2012–2013)	505	100	73 (70–77)	47 (45–48)	74 (68–81)	110 (97–120)	130 (110–150)
4 (2014–2015)	507	99.7 (96.0–100)	70 (65–74)	44 (41–48)	66 (60–73)	100 (95–110)	120 (110–130)
5 (2016–2017)	507	99.9 (99.1–100)	88 (81–95)	52 (47–58)	86 (80–92)	140 (110–160)	170 (120–230)
12–19 years							
3 (2012–2013)	507	99.0 (96.8–99.7)	62 (59–65)	35 (32–37)	60 (57–62)	110 (95–130)	160 (120–200)
4 (2014–2015)	505	98.0 (93.8–99.4)	58 (51–67)	34 (27–41)	55 (49–62)	99 (83–120)	120[E] (58–180)
5 (2016–2017)	530	99.9 (98.6–100)	71 (64–78)	42 (34–49)	70 (63–78)	110 (96–130)	140 (120–160)
20–39 years							
3 (2012–2013)	348	96.6 (80.0–99.5)	72 (60–86)	38 (30–46)	74 (62–86)	160 (130–190)	210 (160–260)
4 (2014–2015)	363	97.0 (91.3–99.0)	62 (52–74)	34 (29–39)	57 (49–66)	170 (110–230)	190 (170–220)
5 (2016–2017)	363	99.7 (90.3–100)	82 (74–91)	45 (35–55)	74 (64–83)	170 (130–210)	220[E] (82–360)
40–59 years							
3 (2012–2013)	311	97.4 (89.6–99.4)	71 (58–86)	36 (31–42)	62 (50–74)	180 (140–220)	230 (170–290)
4 (2014–2015)	312	98.3 (94.2–99.5)	63 (55–71)	35 (30–39)	58 (50–65)	130 (97–160)	160[E] (57–260)
5 (2016–2017)	345	99.4 (96.4–99.9)	71 (65–79)	38 (33–42)	72 (64–80)	140 (100–170)	160 (110–210)
60–79 years							
3 (2012–2013)	350	98.2 (95.9–99.2)	60 (53–67)	34 (29–39)	60 (50–70)	100 (90–110)	120 (110–130)
4 (2014–2015)	358	94.8 (86.4–98.1)	50 (44–57)	25 (<LOD–33)	50 (44–56)	98 (87–110)	120 (93–150)
5 (2016–2017)	349	97.6 (90.3–99.5)	63 (58–70)	35 (31–39)	63 (56–69)	110 (88–140)	150[E] (85–210)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3, 4, and 5 is 23 pmol/g Hb.

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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SUMMARY AND RESULTS FOR PERFLUOROALKYL AND POLYFLUOROALKYL SUBSTANCES 12

12.1 PERFLUOROALKYL AND POLYFLUOROALKYL SUBSTANCES

Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are structurally related persistent organic compounds that have a fluorinated alkyl (carbon) chain structure. Perfluoroalkyl substances are characterized by the presence of a fully fluorinated alkyl chain that is typically four to 14 carbons in length. In contrast, polyfluoroalkyl substances are not fully fluorinated and have a hydrogen or oxygen attached to at least one carbon in the alkyl chain. Nine perfluoroalkyl substances were measured in cycle 5 of the Canadian Health Measures Survey (CHMS) (Table 12.1.1).

Table 12.1.1
Perfluoroalkyl substances measured in the Canadian Health Measures Survey cycle 5 (2016–2017)

Perfluoroalkyl substance	CASRN
Perfluorobutanoic acid (PFBA)	375-22-4
Perfluorohexanoic acid (PFHxA)	307-24-4
Perfluorooctanoic acid (PFOA)	335-67-1
Perfluorononanoic acid (PFNA)	375-95-1
Perfluorodecanoic acid (PFDA)	335-76-2
Perfluoroundecanoic acid (PFUnDA)	2058-94-8
Perfluorobutane sulfonate (PFBS)	375-73-5
Perfluorohexane sulfonate (PFHxS)	355-46-4
Perfluorooctane sulfonate (PFOS)	1763-23-1

PFAS are synthetic chemicals with high chemical and thermal stability and the ability to repel both water and oils (Kissa, 2001). These characteristics make them ideal for use in a number of industrial and commercial applications (Kissa, 2001). PFAS are used as stain-repellent, water-repellent, and oil-repellent fabric protectors, in water-repellent and oil-repellent paper coatings, wiper blades, bike-chain lubricant, wire and cable insulation, pharmaceutical packaging, and food packaging (Kissa, 2001). They are also used in engine-oil additives, nail polish, hair curling and straightening products, metal plating and cleaning products, fire retardant foams, inks, varnishes, polyurethane production, and vinyl polymerization (Kissa, 2001). Fluoropolymers manufactured using salts of PFAS are used in many industrial and consumer products, including surface coatings on textiles and carpets, personal care products, and non-stick coatings on cookware (INAC, 2009; Kissa, 2001; Prevedouros et al., 2005).

PFOS and PFOA are the most extensively studied and measured PFAS in humans (Dallaire et al., 2009; Hölzer et al., 2008; Kato et al., 2011). PFHxS is another perfluorinated compound that has been measured in humans, but it has not been examined as extensively as PFOS and PFOA. Other PFAS, such as PFBA, PFHxA, PFNA, PFDA, PFUnDA, and PFBS, have been measured less frequently in the human population.

Worldwide use of PFOS and PFOS-related products has decreased significantly since 2002, when the world's largest producer at the time completed its voluntary phase-out of production (ITRC, 2017). PFHxS, a

known by-product in the production of PFOS, was also phased out as a result. In 2008, replacements for PFOA were introduced, resulting in the subsequent phase-out of PFOA in the production of fluoropolymers (ITRC, 2017). Potential replacements for PFOS-based substances include new PFBS-based compounds that are rapidly eliminated from the body with relatively low bioaccumulation potential and toxicity; however, it is not yet clear if long-chain PFAS alternatives can achieve the same performance effectiveness of their predecessors (Chang et al., 2008; Newsted et al., 2008; ITRC, 2017).

PFAS do not occur naturally in the environment. Entry into the environment occurs through releases during manufacturing and transport, use of consumer products, and the disposal and breakdown of larger PFAS. As a result, PFAS have been detected in a wide array of environmental media (Houde et al., 2006).

For the general public, exposure to PFAS is widespread through food, drinking water, consumer products, dust, soil, and air (Fromme et al., 2009; Fromme et al., 2007; Hölzer et al., 2008; Kubwabo et al., 2005). PFAS have been analyzed as part of Health Canada's ongoing Total Diet Study surveys; levels in foods that are commercially sold in Canada are low, similar to levels that have been reported in other countries (Health Canada, 2014; Health Canada, 2016a; Tittlemier et al., 2006; Tittlemier et al., 2007). The contribution of individual pathways and sources of exposure appears to depend on age, dose, and substance. Generally, ingestion of food, drinking water, and house dust are expected to be the main routes of exposure for adults in the general population, whereas oral hand-to-mouth contact with consumer products, such as carpets, clothing, and upholstery, is a significant contributor for infants, toddlers, and children (Trudel et al., 2008).

Longer-chain PFAS are well absorbed in the body, poorly excreted, and not extensively metabolized (Harada et al., 2005; INAC, 2009; Johnson et al., 1984). Average half-lives of PFOS, PFOA, and PFHxS in humans range from 3–9 years (Olsen et al., 2007). Shorter-chain PFAS are eliminated much more quickly; for example, the elimination half-life for PFBA is 72 to 81 hours (ATSDR, 2015). In humans, PFOS and PFOA are found in serum, plasma, kidneys, and the liver (Butenhoff et al., 2006; Fromme et al., 2009; Kärrman et al., 2010). PFAS have also been measured in breast milk and umbilical cord blood (Kärrman et al., 2010; Monroy et al., 2008). PFAS have a strong affinity for the protein fraction in blood and do not typically accumulate in lipids (Kärrman et al., 2010; Martin et al., 2004). Serum levels of PFAS, in particular PFOA and PFOS, can reflect cumulative exposure over several years (CDC, 2009). Although both PFOA and PFOS are biomarkers of exposures to themselves, animal studies have indicated that their presence in serum may also result from exposure to and subsequent metabolism of other PFAS (ATSDR, 2015). Absorbed PFOA and PFOS are ultimately excreted in urine (ATSDR, 2015).

The primary concern with PFAS is their persistence in both the environment and the human body (Olsen et al., 2007). Possible linkages between exposure to PFAS and adverse human health effects have been examined in occupational studies and studies of populations exposed to contaminated drinking water (ATSDR, 2015). Although no definitive links have been established, reports in children and neonates suggest associations between serum PFAS and thyroid effects (Lopez-Espinosa et al., 2012; Wang et al., 2014). A recent review by Ballesteros et al. (2017) also reported a positive association between maternal or teenage male exposure to certain PFAS and thyroid-stimulating hormone levels, despite heterogeneity across studies. In several animal species, the liver has been identified as the primary target organ of toxicity for PFAS regardless of the route of exposure (ATSDR, 2015; EPA, 2002; Health Canada, 2006). PFOA has been associated with increased incidence of tumours in rodent bioassays; following the identification of PFOA and other PFAS as priority agents for International Agency for Research on Cancer (IARC) monographs, PFOA was classified as possibly carcinogenic to humans (Group 2B) based on limited evidence in humans for a positive association with cancers of the testis and kidney (IARC, 2017).

In 2006, Environment Canada and Health Canada concluded that PFOS was not a concern for human health at current levels of exposure (Health Canada, 2006). However, PFOS and its salts were declared toxic to the environment and its biological diversity, and PFOS was added to Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment Canada, 2006). In 2009, PFOS and its salts were added to the Virtual Elimination List under CEPA 1999 (Canada, 2009). In 2016, PFOS was added to the Prohibition of Certain Toxic Substances Regulations, prohibiting most uses aside from exemptions for specific uses (Health Canada, 2016c). Canada is also working through the Convention

on Long-Range Transboundary Air Pollution and the Stockholm Convention on Persistent Organic Pollutants under the United Nations to reduce the global production of PFOS (Health Canada, 2016c).

In 2012, Environment Canada and Health Canada published screening assessments for PFOA and long-chain perfluorocarboxylic acids (including PFNA, PFDA, and PFUnDA), along with their salts and their precursors (Environment Canada, 2012; Environment Canada and Health Canada, 2012b). The assessments concluded that the substances are an ecological concern, but that PFOA and its salts and precursors are not a concern for human health (Environment Canada, 2012; Environment Canada and Health Canada, 2012b). Long-chain perfluorocarboxylic acids and their salts and precursors were not determined to be a high priority for assessment of potential risks to human health; as such, no human health assessment was conducted. Based on the assessments, both PFOA and long-chain perfluorocarboxylic acids and their salts and precursors have been added to the List of Toxic Substances in Schedule 1 of CEPA 1999 (Canada, 2012a).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has also developed guidelines for Canadian drinking water quality that establish maximum acceptable concentrations for PFOS and PFOA in drinking water (Health Canada, 2018a; Health Canada, 2018b). Both guidelines were developed based on liver effects in laboratory animals and are considered to be protective of both non-cancer and cancer effects. Health Canada has also developed drinking water screening values for several additional PFAS, including PFBA, PFHxA, PFNA, PFBS, and PFHxS (Health Canada, 2018c).

A number of risk management measures for perfluorocarboxylic acids and their precursors have been implemented by the Government of Canada. These measures include regulations prohibiting the manufacture, use, sale, offer for sale, and import of four fluorotelomer-based substances found to be precursors to long-chain perfluorinated carboxylic acids, unless present in certain manufactured items (Canada, 2010; Environment Canada and Health Canada, 2012a). A five-year Environmental Performance Agreement that commenced in 2010 resulted in participating companies successfully meeting their commitments to eliminate residual PFOA, long-chain perfluorocarboxylic acids, and their precursors in products (Health Canada, 2016b). Long-chain perfluorocarboxylic acids, PFOA, and PFOS, along with their salts and precursors, are now regulated under the Prohibition of Certain Toxic Substances Regulations, 2012 (Canada, 2012b).

Globally, there is an initiative to reduce PFOA emissions and product content. In 2006, the United States Environmental Protection Agency (EPA) and eight major companies in the industry launched the 2010/15 PFOA Stewardship Program. Under this voluntary effort, several companies exited the PFAS industry altogether, while others stopped the manufacture and import of long-chain PFAS and transitioned to alternative chemicals (EPA, 2018a; EPA, 2018c).The EPA recently released draft toxicity assessments for GenX chemicals and PFBS, members of a larger group of PFAS (EPA, 2018b). Canada's 2010 Environmental Performance Agreement was consistent with the targets and commitments by industry in the United States (Environment Canada, 2010). The European Union and the Australian government have initiated similar policies where PFAS are either prohibited or subject to further toxicity testing for evaluation.

Several human biomonitoring studies in Canada have measured PFAS in serum and plasma (Alberta Health and Wellness, 2008; Fisher et al., 2016; Hamm et al., 2010; Kubwabo et al., 2004; Monroy et al., 2008; Tittlemier et al., 2004; Turgeon O'Brien et al., 2012). In 2002, serum samples from 56 individuals in Ottawa, Ontario, and Gatineau, Québec were analyzed for PFOA and PFOS. Mean concentrations of PFOA and PFOS were 3.4 µg/L and 28.8 µg/L, respectively (Kubwabo et al., 2004). In 2004, PFOS was measured in plasma samples from 883 Nunavik Inuit living in the Canadian Arctic with a geometric mean concentration of 18.68 µg/L (Dallaire et al., 2009). The concentrations of PFAS were measured in 86 Inuit children, 11 months to 4.5 years of age, attending childcare centres in Nunavik between 2006 and 2008 (Turgeon O'Brien et al., 2012). The geometric mean concentrations in plasma for PFOA, PFHxS, and PFOS were 1.62 µg/L, 0.33 µg/L, and 3.37 µg/L, respectively. In the Maternal–Infant Research on Environmental Chemicals (MIREC) study of 1,940 participants, the geometric means (95th percentile) for PFOA, PFHxS, and PFOS in plasma were 1.65 µg/L (4.1 µg/L), 1.03 µg/L (4.3 µg/L), and 4.56 µg/L (11 µg/L), respectively (Fisher et al., 2016). MIREC is a national-level prospective biomonitoring study carried out in pregnant women aged 18 years and older recruited from 10 sites across Canada between 2008 and 2011 (Arbuckle et al., 2013).

PFOS, PFOA, and PFHxS were measured in the plasma of CHMS participants aged 20–79 years in cycle 1 (2007–2009), 12–79 years in cycle 2 (2009–2011) and 3–79 years in cycle 5 (2016–2017). PFBA, PFHxA, PFBS, PFNA, PFDA, and PFUnDA were measured in the plasma of CHMS participants aged 12–79 years in cycle 2 (2009–2011) and 3–79 years in cycle 5 (2016–2017). Data for the PFAS are presented as µg/L in plasma (Tables 12.1.2 to 12.1.19). Finding a measurable amount of PFAS in plasma is an indicator of exposure to PFAS and does not necessarily mean that an adverse health effect will occur.

Table 12.1.2

Perfluorobutanoic acid (PFBA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1524	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1583	F	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
2 (2009–2011)	765	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	788	F	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
2 (2009–2011)	759	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	795	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.5 and 0.075 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 12–79 years were included as participants under the age of 12 years were not included in cycle 2.

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

F Data are too unreliable to be published.

Table 12.1.3

Perfluorobutanoic acid (PFBA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2590	4.2[E] (2.3–7.7)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1292	5.4[E] (2.7–10.3)	—	<LOD	<LOD	<LOD	0.082 (<LOD–0.092)
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1298	3.1[E] (1.7–5.8)	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	489	12.9[E] (7.6–21.2)	—	<LOD	<LOD	0.081 (<LOD–0.10)	0.099 (<LOD–0.13)
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	518	5.9[E] (3.8–9.1)	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	507	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	527	2.0[E] (1.0–4.2)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	362	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	362	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	334	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	345	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	321	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	349	7.5[E] (3.7–14.5)	—	<LOD	<LOD	<LOD	0.096[E] (<LOD–0.14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.5 and 0.075 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.4

Perfluorohexanoic acid (PFHxA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1524	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1583	9.2[E] (4.9–16.4)	—	<LOD	<LOD	<LOD	0.13[E] (<LOD–0.18)
Males, 12–79 years							
2 (2009–2011)	765	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	788	11[E] (6.0–19.3)	—	<LOD	<LOD	0.095[E] (<LOD–0.14)	0.15[E] (0.094–0.21)
Females, 12–79 years							
2 (2009–2011)	759	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	795	7.4[E] (3.6–14.5)	—	<LOD	<LOD	<LOD	0.11[E] (<LOD–0.16)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.1 and 0.084 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 12–79 years were included as participants under the age of 12 years were not included in cycle 2.

b 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 are too unreliable to be published.

Table 12.1.5

Perfluorohexanoic acid (PFHxA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2593	9.2[E] (5.0–16.2)	—	<LOD	<LOD	<LOD	0.13[E] (<LOD–0.18)
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1294	10.9[E] (6.0–18.9)	—	<LOD	<LOD	0.094[E] (<LOD–0.13)	0.15[E] (0.094–0.21)
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1299	7.5[E] (3.7–14.4)	—	<LOD	<LOD	<LOD	0.11[E] (<LOD–0.16)
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	490	F	—	<LOD	<LOD	<LOD	0.12[E] (<LOD–0.18)
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	520	10.2[E] (5.6–17.7)	—	<LOD	<LOD	<LOD	0.14[E] (<LOD–0.21)
12–19 years							
2 (2009–2011)	507	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	527	F	—	<LOD	<LOD	<LOD	0.11[E] (<LOD–0.16)
20–39 years							
2 (2009–2011)	362	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	362	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	334	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	346	15.2[E] (7.6–28.1)	—	<LOD	<LOD	0.12[E] (<LOD–0.17)	0.19[E] (0.091–0.30)
60–79 years							
2 (2009–2011)	321	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	348	F	—	<LOD	<LOD	<LOD	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.1 and 0.084 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.6

Perfluorooctanoic acid (PFOA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 20–79 years[a], Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–79 years							
1 (2007–2009)	2880	99.0 (97.7–99.6)	2.5 (2.4–2.7)	1.3 (1.1–1.4)	2.6 (2.4–2.8)	4.6 (4.3–5.0)	5.5 (5.1–5.8)
2 (2009–2011)	1017	100	2.3 (2.1–2.5)	1.1 (0.91–1.2)	2.4 (2.1–2.6)	4.3 (3.9–4.7)	5.3 (3.9–6.7)
5 (2016–2017)	1055	100	1.3 (1.2–1.5)	0.63 (0.57–0.68)	1.3 (1.1–1.4)	2.7 (2.2–3.2)	3.2 (2.5–3.8)
Males, 20–79 years							
1 (2007–2009)	1376	99.4 (98.6–99.8)	2.9 (2.7–3.2)	1.6 (1.4–1.7)	3.1 (2.8–3.3)	5.0 (4.5–5.5)	5.9 (5.4–6.4)
2 (2009–2011)	511	100	2.6 (2.4–2.9)	1.3 (0.99–1.6)	2.7 (2.5–2.9)	4.5 (3.2–5.8)	6.0 (4.3–7.7)
5 (2016–2017)	525	100	1.5 (1.3–1.7)	0.89 (0.80–0.98)	1.4 (1.1–1.6)	2.8 (2.1–3.6)	3.5 (2.6–4.3)
Females, 20–79 years							
1 (2007–2009)	1504	98.6 (96.3–99.5)	2.2 (2.0–2.4)	1.0 (0.92–1.2)	2.2 (2.1–2.4)	4.1 (3.7–4.5)	5.0 (4.4–5.5)
2 (2009–2011)	506	100	2.0 (1.8–2.2)	0.92 (0.73–1.1)	2.0 (1.7–2.3)	3.9 (3.6–4.3)	4.4 (3.8–5.1)
5 (2016–2017)	530	100	1.1 (1.0–1.3)	0.54 (0.47–0.60)	1.0 (0.90–1.2)	2.5 (2.0–3.0)	3.0 (2.7–3.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.1, and 0.066 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 20–79 years were included, as participants under the age of 20 years were not included in cycle 1.

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

Table 12.1.7

Perfluorooctanoic acid (PFOA) — Geometric means and selected percentiles of plasma 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)[c]	—	—	—	—	—	—	—
5 (2016–2017)	2593	100	1.3 (1.2–1.4)	0.64 (0.58–0.71)	1.2 (1.1–1.3)	2.6 (2.2–3.0)	3.1 (2.6–3.6)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1294	100	1.5 (1.3–1.6)	0.87 (0.79–0.95)	1.3 (1.2–1.5)	2.6 (2.1–3.2)	3.4 (2.5–4.3)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1299	100	1.1 (1.0–1.3)	0.56 (0.51–0.60)	1.1 (0.95–1.2)	2.4 (2.0–2.8)	2.9 (2.7–3.2)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	491	100	1.5 (1.3–1.6)	0.81 (0.72–0.90)	1.3 (1.1–1.5)	2.7 (2.1–3.2)	3.6 (2.4–4.7)
6–11 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	520	100	1.3 (1.2–1.4)	0.81 (0.74–0.88)	1.2 (1.1–1.3)	2.1 (1.7–2.4)	2.4 (2.0–2.9)
12–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	507	100	2.1 (1.9–2.3)	1.2 (1.0–1.4)	2.1 (1.9–2.3)	3.4 (3.0–3.7)	4.1 (3.6–4.5)
5 (2016–2017)	527	100	1.1 (0.95–1.2)	0.63 (0.57–0.70)	1.0 (0.90–1.1)	1.6 (1.5–1.8)	1.9 (1.4–2.4)
20–39 years							
1 (2007–2009)	979	99.1 (96.5–99.8)	2.4 (2.2–2.7)	1.1 (0.95–1.3)	2.5 (2.3–2.8)	4.5 (4.0–5.1)	5.4 (4.8–5.9)
2 (2009–2011)	362	100	2.2 (1.9–2.5)	0.88 (0.64–1.1)	2.3 (1.9–2.8)	4.4 (3.2–5.7)	5.8 (3.9–7.6)
5 (2016–2017)	362	100	1.1 (1.0–1.2)	0.56 (0.49–0.62)	1.1 (0.94–1.2)	2.1 (1.8–2.4)	2.5 (2.2–2.9)
40–59 years							
1 (2007–2009)	983	99.3 (97.9–99.8)	2.5 (2.3–2.7)	1.3 (1.2–1.4)	2.5 (2.3–2.8)	4.5 (4.0–4.9)	5.4 (4.6–6.1)
2 (2009–2011)	334	100	2.2 (2.0–2.4)	1.1 (0.87–1.3)	2.1 (1.7–2.5)	3.9 (3.6–4.1)	4.4 (3.9–5.0)
5 (2016–2017)	345	100	1.4 (1.2–1.6)	0.68 (0.58–0.77)	1.3 (1.1–1.4)	3.1[E] (1.8–4.3)	3.8[E] (2.3–5.3)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	918	98.3 (95.3–99.4)	2.8 (2.5–3.0)	1.5 (1.3–1.7)	2.8 (2.6–3.0)	5.2 (4.7–5.7)	6.3 (5.4–7.1)
2 (2009–2011)	321	100	2.8 (2.4–3.2)	1.5 (1.0–2.0)	2.7 (2.1–3.2)	4.6 (3.1–6.0)	6.4 (4.6–8.1)
5 (2016–2017)	348	100	1.6 (1.4–1.8)	0.86 (0.71–1.0)	1.6 (1.3–1.9)	2.9 (2.8–3.1)	3.4 (2.7–4.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.1, and 0.066 µg/L, respectively.

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

b Data are not available, as participants under the age of 20 years were not included in cycle 1.

c Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

Table 12.1.8

Perfluorononanoic acid (PFNA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1524	99.4 (98.6–99.8)	0.82 (0.75–0.90)	0.39 (0.33–0.44)	0.80 (0.70–0.90)	1.5 (1.3–1.8)	1.9[E] (1.1–2.7)
5 (2016–2017)	1497	98.8 (96.9–99.6)	0.51 (0.45–0.58)	0.24 (0.21–0.27)	0.50 (0.46–0.54)	1.1 (0.80–1.4)	1.5 (1.2–1.8)
Males, 12–79 years							
2 (2009–2011)	765	99.2 (97.5–99.8)	0.84 (0.75–0.94)	0.43 (0.37–0.48)	0.80 (0.69–0.91)	1.6 (1.4–1.8)	1.9 (1.5–2.2)
5 (2016–2017)	755	99.4 (97.9–99.8)	0.54 (0.47–0.62)	0.27 (0.24–0.31)	0.51 (0.46–0.56)	1.1 (0.72–1.4)	1.4 (1.0–1.9)
Females, 12–79 years							
2 (2009–2011)	759	99.6 (99.1–99.8)	0.81 (0.73–0.89)	0.35 (0.30–0.40)	0.79 (0.69–0.90)	1.5 (1.1–2.0)	2.3[E] (1.2–3.4)
5 (2016–2017)	742	98.2 (94.8–99.4)	0.49 (0.43–0.55)	0.21 (0.19–0.23)	0.48 (0.44–0.53)	1.1 (0.77–1.5)	1.7[E] (0.79–2.5)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.2 and 0.13 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 12–79 years were included, as participants under the age of 12 years were not included in cycle 2.

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

E Use data with caution.

Table 12.1.9

Perfluorononanoic acid (PFNA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2442	98.8 (97.1–99.5)	0.51 (0.45–0.57)	0.24 (0.21–0.26)	0.49 (0.45–0.53)	1.1 (0.81–1.3)	1.5 (1.2–1.8)
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1236	99.3 (98.1–99.8)	0.53 (0.46–0.61)	0.27 (0.23–0.30)	0.51 (0.46–0.56)	1.0 (0.73–1.4)	1.4 (1.0–1.8)
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1206	98.3 (95.2–99.4)	0.48 (0.43–0.54)	0.21 (0.19–0.23)	0.47 (0.42–0.52)	1.1 (0.76–1.4)	1.6[E] (0.79–2.5)
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	453	99.3 (97.7–99.8)	0.45 (0.40–0.51)	0.21 (0.19–0.24)	0.39 (0.34–0.44)	0.95 (0.81–1.1)	1.3[E] (0.76–1.8)
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	492	98.7 (95.8–99.6)	0.45 (0.37–0.53)	0.23 (0.19–0.28)	0.40 (0.35–0.44)	1.0 (0.66–1.4)	1.5[E] (0.45–2.6)
12–19 years							
2 (2009–2011)	507	99.1 (97.8–99.6)	0.71 (0.62–0.81)	0.33 (0.27–0.38)	0.69 (0.63–0.75)	1.4 (1.0–1.7)	1.7[E] (0.47–2.9)
5 (2016–2017)	494	99.4 (97.2–99.9)	0.41 (0.33–0.51)	0.21 (0.18–0.24)	0.37 (0.33–0.41)	1.0[E] (0.51–1.5)	F
20–39 years							
2 (2009–2011)	362	99.0 (96.9–99.7)	0.79 (0.72–0.86)	0.38 (0.30–0.46)	0.77 (0.62–0.92)	1.5 (1.3–1.7)	F
5 (2016–2017)	336	98.4 (95.6–99.4)	0.41 (0.36–0.47)	0.21 (0.14–0.28)	0.44 (0.37–0.50)	0.77 (0.61–0.92)	0.91 (0.71–1.1)
40–59 years							
2 (2009–2011)	334	99.7 (97.6–100)	0.79 (0.69–0.90)	0.41 (0.32–0.50)	0.78 (0.65–0.91)	1.3 (0.99–1.6)	1.7 (1.1–2.2)
5 (2016–2017)	332	98.7 (90.2–99.8)	0.60 (0.48–0.74)	0.27 (0.22–0.33)	0.56 (0.47–0.64)	1.4 (0.94–2.0)	1.7[E] (0.77–2.6)
60–79 years							
2 (2009–2011)	321	100	1.1 (0.87–1.3)	0.45[E] (0.25–0.65)	1.0 (0.86–1.1)	2.0[E] (1.2–2.8)	2.7[E] (1.5–3.8)
5 (2016–2017)	335	99.3 (98.2–99.7)	0.62 (0.55–0.69)	0.31[E] (0.19–0.43)	0.61 (0.56–0.66)	1.2 (0.99–1.4)	1.5 (1.2–1.8)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.2 and 0.13 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.10

Perfluorodecanoic acid (PFDA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1524	79.3 (72.6–84.7)	0.20 (0.17–0.22)	<LOD	0.17 (0.15–0.19)	0.46 (0.31–0.62)	0.66 (0.45–0.87)
5 (2016–2017)	1450	91.4 (85.9–94.9)	0.18 (0.16–0.21)	<LOD	0.17 (0.15–0.18)	0.48 (0.34–0.62)	0.65 (0.45–0.84)
Males, 12–79 years							
2 (2009–2011)	765	83.1 (75.2–88.9)	0.20 (0.18–0.23)	<LOD	0.18 (0.15–0.20)	0.38 (0.26–0.51)	0.55 (0.41–0.70)
5 (2016–2017)	715	94.1 (80.3–98.4)	0.18 (0.16–0.22)	0.10 (<LOD–0.13)	0.17 (0.14–0.19)	0.44[E] (0.28–0.60)	0.55 (0.35–0.74)
Females, 12–79 years							
2 (2009–2011)	759	75.6 (66.9–82.5)	0.19 (0.16–0.23)	<LOD	0.17 (0.14–0.19)	0.50 (0.32–0.68)	F
5 (2016–2017)	735	88.8 (82.3–93.0)	0.18 (0.16–0.21)	<LOD	0.17 (0.15–0.18)	0.54 (0.35–0.73)	0.76[E] (0.32–1.2)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.1 and 0.092 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 20–79 years were included, as participants under the age of 20 years were not included in cycle 2.

b 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 are too unreliable to be published.

Table 12.1.11

Perfluorodecanoic acid (PFDA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2360	91.4 (86.0–94.8)	0.18 (0.16–0.20)	0.094 (<LOD–0.12)	0.16 (0.15–0.18)	0.44 (0.31–0.56)	0.64 (0.47–0.81)
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1175	94.0 (81.9–98.2)	0.18 (0.15–0.21)	0.10 (<LOD–0.13)	0.16 (0.14–0.18)	0.40E (0.25–0.56)	0.52 (0.34–0.71)
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1185	89.0 (82.8–93.1)	0.18 (0.15–0.20)	<LOD	0.16 (0.14–0.18)	0.48[E] (0.29–0.67)	0.74[E] (0.37–1.1)
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	443	91.6 (83.7–95.9)	0.14 (0.13–0.16)	0.095 (<LOD–0.13)	0.14 (0.13–0.15)	0.25 (0.20–0.30)	0.32 (0.25–0.38)
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	467	91.7 (85.9–95.2)	0.14 (0.13–0.15)	0.093 (<LOD–0.10)	0.14 (0.13–0.15)	0.24 (0.22–0.26)	0.28 (0.24–0.31)
12–19 years							
2 (2009–2011)	507	72.1 (62.0–80.3)	0.15 (0.13–0.18)	<LOD	0.14 (0.12–0.16)	0.31 (0.24–0.37)	0.39[E] (0.22–0.55)
5 (2016–2017)	474	86.7 (78.9–91.9)	0.13 (0.11–0.15)	<LOD	0.13 (0.11–0.14)	0.22 (0.19–0.26)	0.34[E] (0.11–0.57)
20–39 years							
2 (2009–2011)	362	84.7 (76.1–90.6)	0.22 (0.20–0.23)	<LOD	0.17 (0.16–0.19)	0.39[E] (0.21–0.56)	F
5 (2016–2017)	331	88.9 (71.0–96.3)	0.16 (0.13–0.20)	<LOD	0.15 (0.12–0.18)	0.32 (0.23–0.41)	0.47[E] (0.23–0.71)
40–59 years							
2 (2009–2011)	334	73.6 (62.3–82.5)	0.17 (0.14–0.21)	<LOD	0.16 (0.13–0.19)	0.34[E] (0.17–0.52)	0.51 (0.35–0.66)
5 (2016–2017)	322	91.7 (82.6–96.3)	0.21 (0.17–0.26)	0.099 (<LOD–0.12)	0.18 (0.15–0.21)	0.64[E] (0.36–0.93)	0.89[E] (0.40–1.4)
60–79 years							
2 (2009–2011)	321	83.7 (70.9–91.5)	0.25 (0.17–0.35)	<LOD	0.23 (0.17–0.29)	0.65 (0.43–0.87)	F
5 (2016–2017)	323	96.6 (90.7–98.8)	0.21 (0.19–0.24)	0.10 (<LOD–0.13)	0.20 (0.17–0.22)	0.47 (0.36–0.58)	0.62 (0.44–0.79)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.1 and 0.092 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.12

Perfluoroundecanoic acid (PFUnDA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1522	59.3 (47.5–70.0)	0.12 (0.098–0.14)	<LOD	0.095 (<LOD–0.10)	0.37 (0.28–0.45)	0.56[E] (0.30–0.82)
5 (2016–2017)	1576	38.5 (29.1–48.9)	—	<LOD	<LOD	0.35 (0.23–0.47)	0.50 (0.34–0.67)
Males, 12–79 years							
2 (2009–2011)	765	55.5 (43.1–67.3)	—	<LOD	0.094 (<LOD–0.11)	0.34 (0.26–0.42)	0.47[E] (0.27–0.67)
5 (2016–2017)	783	35.7 (24.7–48.5)	—	<LOD	<LOD	0.37[E] (0.21–0.52)	0.42[E] (0.25–0.58)
Females, 12–79 years							
2 (2009–2011)	757	63.0 (50.8–73.7)	0.12 (0.10–0.15)	<LOD	0.096 (<LOD–0.11)	0.39 (0.26–0.52)	0.63[E] (0.24–1.0)
5 (2016–2017)	793	41.2 (32.2–50.9)	—	<LOD	<LOD	0.33[E] (0.19–0.47)	0.55[E] (0.30–0.79)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.09 and 0.12 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 12–79 years were included, as participants under the age of 12 years were not included in cycle 2.

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

E Use data with caution.

Table 12.1.13

Perfluoroundecanoic acid (PFUnDA) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2583	35.8 (26.9–45.8)	—	<LOD	<LOD	0.32 (0.21–0.43)	0.46 (0.30–0.63)
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1289	33.3 (23.2–45.2)	—	<LOD	<LOD	0.34[E] (0.19–0.49)	0.42 (0.27–0.57)
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1294	38.3 (29.8–47.7)	—	<LOD	<LOD	0.31 (0.21–0.41)	0.54[E] (0.32–0.76)
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	487	10.0[E] (6.3–15.4)	—	<LOD	<LOD	<LOD	0.14 (<LOD–0.17)
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	520	13.6[E] (7.6–23.1)	—	<LOD	<LOD	0.13 (<LOD–0.16)	0.20[E] (<LOD–0.28)
12–19 years							
2 (2009–2011)	506	36.8 (25.1–50.2)	—	<LOD	<LOD	0.19 (0.13–0.24)	0.30 (0.21–0.38)
5 (2016–2017)	525	16.4[E] (10.5–24.7)	—	<LOD	<LOD	0.15 (<LOD–0.19)	0.19 (0.14–0.23)
20–39 years							
2 (2009–2011)	362	58.9 (45.7–71.0)	0.13 (0.10–0.16)	<LOD	0.098 (<LOD–0.12)	0.36[E] (0.21–0.51)	0.64[E] (0.22–1.1)
5 (2016–2017)	358	33.2[E] (20.9–48.3)	—	<LOD	<LOD	0.27[E] (0.15–0.40)	0.36[E] (0.16–0.56)
40–59 years							
2 (2009–2011)	334	66.0 (51.3–78.1)	0.11 (0.095–0.14)	<LOD	0.095 (<LOD–0.10)	0.35[E] (0.22–0.49)	0.43 (0.28–0.58)
5 (2016–2017)	346	43.2 (29.7–57.7)	—	<LOD	<LOD	0.43[E] (0.19–0.67)	0.64[E] (0.36–0.91)
60–79 years							
2 (2009–2011)	320	62.2 (38.9–81.0)	0.14[E] (0.090–0.23)	<LOD	0.11[E] (<LOD–0.17)	0.54[E] (0.17–0.90)	0.84[E] (0.42–1.3)
5 (2016–2017)	347	49.5 (38.7–60.4)	—	<LOD	<LOD	0.36 (0.27–0.46)	0.49 (0.37–0.62)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.09 and 0.12 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

Table 12.1.14

Perfluorobutane sulfonate (PFBS) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 12–79 years[a], Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
2 (2009–2011)	1524	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1577	F	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
2 (2009–2011)	765	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	784	F	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
2 (2009–2011)	759	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	793	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.4 and 0.066 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 12–79 years were included, as participants under the age of 12 years were not included in cycle 2.

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

F Data are too unreliable to be published.

Table 12.1.15

Perfluorobutane sulfonate (PFBS) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 3–79 years by age group, Canadian Health Measures Survey cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	2584	0.10[E] (0.10–0.30)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1289	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	1295	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	490	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
2 (2009–2011)[b]	—	—	—	—	—	—	—
5 (2016–2017)	517	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
2 (2009–2011)	507	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	526	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
2 (2009–2011)	362	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	361	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
2 (2009–2011)	334	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	343	0	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
2 (2009–2011)	321	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	347	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.4 and 0.066 µg/L, respectively.

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

b Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.16

Perfluorohexane sulfonate (PFHxS) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 20–79 years[a], Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–79 years							
1 (2007–2009)	2880	97.8 (96.2–98.8)	2.3 (2.0–2.6)	0.70 (0.50–0.89)	2.2 (1.8–2.5)	7.3 (6.6–8.1)	12 (9.2–15)
2 (2009–2011)	1015	98.4 (96.4–99.3)	1.7 (1.6–2.0)	0.55 (0.44–0.65)	1.7 (1.5–1.9)	5.9 (4.0–7.9)	8.9[E] (4.6–13)
5 (2016–2017)	1057	99.6 (98.6–99.9)	0.98 (0.85–1.1)	0.28 (0.21–0.34)	0.99 (0.88–1.1)	3.1 (2.2–4.0)	F
Males, 20–79 years							
1 (2007–2009)	1376	99.8 (99.6–99.9)	3.2 (2.8–3.7)	1.3 (1.1–1.6)	2.8 (2.4–3.2)	9.3 (7.6–11)	16 (11–20)
2 (2009–2011)	510	99.6 (98.4–99.9)	2.4 (2.0–2.7)	0.94 (0.76–1.1)	2.1 (1.9–2.4)	6.1 (4.5–7.7)	9.4[E] (4.9–14)
5 (2016–2017)	525	99.6 (97.7–99.9)	1.5 (1.3–1.8)	0.56 (0.40–0.73)	1.3 (1.0–1.5)	F	F
Females, 20–79 years							
1 (2007–2009)	1504	95.9 (92.8–97.7)	1.6 (1.4–1.9)	0.50 (0.38–0.62)	1.5 (1.2–1.7)	5.3 (3.9–6.7)	8.5 (6.6–10)
2 (2009–2011)	505	97.2 (93.9–98.8)	1.3 (1.1–1.5)	0.40 (0.34–0.45)	1.2 (1.0–1.3)	F	8.2[E] (3.4–13)
5 (2016–2017)	532	99.6 (97.8–99.9)	0.65 (0.57–0.74)	0.20 (0.15–0.25)	0.62 (0.50–0.74)	1.9[E] (0.96–2.8)	3.8 (2.4–5.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.2, and 0.063 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 20–79 years were included, as participants under the age of 20 years were not included in cycle 1.

b 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 are too unreliable to be published.

Table 12.1.17

Perfluorohexane sulfonate (PFHxS) — Geometric means and selected percentiles of plasma 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)[c]	—	—	—	—	—	—	—
5 (2016–2017)	2595	99.7 (98.9–99.9)	0.90 (0.78–1.0)	0.27 (0.21–0.33)	0.90 (0.76–1.0)	3.0 (2.4–3.7)	5.3[E] (1.8–8.7)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1294	99.7 (98.2–99.9)	1.3 (1.1–1.5)	0.43 (0.35–0.50)	1.1 (0.96–1.3)	3.6[E] (1.2–6.0)	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1301	99.7 (98.2–99.9)	0.64 (0.55–0.73)	0.20 (0.16–0.25)	0.58 (0.48–0.68)	1.9 (1.2–2.6)	3.5 (2.2–4.8)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	491	100	0.61 (0.46–0.81)	0.24 (0.19–0.30)	0.54 (0.37–0.72)	1.8[E] (1.1–2.5)	3.1[E] (1.0–5.1)
6–11 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	520	100	0.59 (0.45–0.77)	0.24 (0.16–0.31)	0.49 (0.41–0.58)	1.7 (1.1–2.3)	F
12–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	506	99.2 (97.5–99.7)	1.9 (1.6–2.3)	0.60 (0.50–0.70)	1.6 (1.3–1.9)	7.8 (5.0–11)	11[E] (5.7–16)
5 (2016–2017)	527	100	0.69 (0.59–0.80)	0.25 (0.17–0.32)	0.58 (0.48–0.67)	2.1 (1.6–2.6)	3.6 (3.0–4.3)
20–39 years							
1 (2007–2009)	979	96.0 (93.2–97.6)	2.1 (1.8–2.4)	0.61 (0.49–0.73)	1.9 (1.5–2.2)	7.9 (5.4–10)	16[E] (10–23)
2 (2009–2011)	361	97.1 (92.1–99.0)	1.5 (1.3–1.8)	0.41 (0.28–0.54)	1.6 (1.1–2.1)	4.7 (3.1–6.3)	6.0[E] (2.1–9.9)
5 (2016–2017)	362	99.5 (96.9–99.9)	0.84 (0.73–0.97)	0.20[E] (0.096–0.30)	0.69 (0.46–0.92)	F	F
40–59 years							
1 (2007–2009)	983	98.8 (96.7–99.6)	2.2 (1.9–2.5)	0.79 (0.54–1.0)	2.2 (1.8–2.5)	6.9 (6.2–7.5)	9.2 (7.4–11)
2 (2009–2011)	333	99.3 (97.8–99.8)	1.8 (1.4–2.3)	0.58[E] (0.33–0.83)	1.7 (1.3–2.0)	F	12[E] (3.5–21)
5 (2016–2017)	346	100	0.93 (0.72–1.2)	0.28 (0.20–0.36)	0.91 (0.68–1.1)	2.6 (1.8–3.4)	4.2[E] (2.1–6.3)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	918	99.3 (98.4–99.7)	2.8 (2.4–3.3)	1.1 (0.90–1.3)	2.6 (2.1–3.0)	8.4 (6.3–11)	13 (9.0–16)
2 (2009–2011)	321	99.4 (94.1–99.9)	2.2 (1.8–2.7)	0.86 (0.64–1.1)	2.0 (1.6–2.4)	6.9[E] (3.5–10)	9.8 (6.7–13)
5 (2016–2017)	349	99.1 (95.0–99.9)	1.3 (1.0–1.7)	0.58 (0.38–0.79)	1.1 (0.89–1.4)	3.4[E] (1.4–5.3)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.2, and 0.063 µg/L, respectively.

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

b Data are not available, as participants under the age of 20 years were not included in cycle 1.

c Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

Table 12.1.18

Perfluorooctane sulfonate (PFOS) — Geometric means and selected percentiles of plasma concentrations (µg/L) for the Canadian population aged 20–79 years[a], Canadian Health Measures Survey cycle 1 (2007–2009), cycle 2 (2009–2011) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[b] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–79 years							
1 (2007–2009)	2880	99.9 (99.9–100)	8.9 (8.0–9.8)	3.6 (3.1–4.1)	9.1 (8.1–10)	19 (16–22)	27 (22–32)
2 (2009–2011)	1017	99.8 (99.1–99.9)	6.9 (6.2–7.6)	2.6 (1.9–3.2)	6.8 (6.0–7.6)	16 (13–18)	19 (13–25)
5 (2016–2017)	1057	99.9 (99.8–100)	3.4 (3.0–3.9)	1.3 (1.2–1.5)	3.3 (2.9–3.7)	8.5 (7.0–9.9)	13 (8.0–17)
Males, 20–79 years							
1 (2007–2009)	1376	100 (98.4–100)	11 (10–12)	5.1 (4.3–6.0)	11 (9.5–12)	23 (18–29)	31 (23–39)
2 (2009–2011)	511	99.7 (98.3–99.9)	8.3 (7.4–9.3)	4.7 (3.6–5.8)	8.2 (6.6–9.8)	16 (14–18)	19 (14–25)
5 (2016–2017)	525	99.9 (99.4–100)	4.3 (3.7–5.1)	1.9 (1.3–2.5)	3.9 (3.1–4.7)	9.1[E] (5.7–13)	13[E] (7.8–19)
Females, 20–79 years							
1 (2007–2009)	1504	99.9 (99.7–99.9)	7.1 (6.3–7.9)	3.0 (2.6–3.4)	7.4 (6.4–8.4)	15 (14–17)	20 (15–24)
2 (2009–2011)	506	99.9 (99.1–100)	5.7 (4.9–6.5)	2.0 (1.5–2.4)	6.0 (5.1–6.9)	15 (11–19)	19[E] (7.8–30)
5 (2016–2017)	532	99.9 (99.6–100)	2.7 (2.4–3.1)	0.99 (0.83–1.2)	2.4 (1.9–2.8)	7.6 (6.2–9.0)	10[E] (5.6–14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.3, and 0.43 µg/L, respectively.

a For the purpose of total population comparisons, only values from participants aged 20–79 years were included, as participants under the age of 20 years were not included in cycle 1.

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

E Use data with caution.

Table 12.1.19

Perfluorooctane sulfonate (PFOS) — Geometric means and selected percentiles of plasma 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)[c]	—	—	—	—	—	—	—
5 (2016–2017)	2594	99.9 (99.8–99.9)	3.0 (2.7–3.4)	1.1 (1.0–1.3)	2.9 (2.5–3.3)	8.1 (7.0–9.3)	11 (7.1–15)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1294	99.9 (99.8–99.9)	3.6 (3.2–4.1)	1.4 (1.3–1.6)	3.5 (3.1–3.9)	8.6 (6.6–11)	13[E] (7.7–17)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	1300	99.9 (99.8–100)	2.5 (2.2–2.8)	0.99 (0.91–1.1)	2.3 (2.0–2.5)	6.9 (5.8–8.1)	8.7[E] (5.1–12)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	491	99.8 (99.2–100)	1.7 (1.5–2.1)	0.89 (0.76–1.0)	1.6 (1.1–2.0)	3.7 (2.7–4.6)	5.5[E] (3.2–7.8)
6–11 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)[c]	—	—	—	—	—	—	—
5 (2016–2017)	520	99.3 (98.0–99.8)	1.7 (1.5–2.0)	0.96 (0.85–1.1)	1.6 (1.3–1.8)	3.4 (3.0–3.9)	4.2 (3.8–4.7)
12–19 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	507	99.8 (97.9–100)	4.6 (4.0–5.2)	2.1 (1.9–2.4)	4.6 (3.9–5.3)	9.0 (7.7–10)	11 (9.2–13)
5 (2016–2017)	526	100	1.9 (1.7–2.0)	1.0 (0.90–1.1)	1.8 (1.6–2.0)	3.3 (3.0–3.5)	3.9 (3.7–4.2)
20–39 years							
1 (2007–2009)	979	99.9 (97.6–100)	8.2 (7.2–9.3)	3.5 (2.8–4.1)	8.6 (7.3–9.9)	17 (15–18)	21 (19–24)
2 (2009–2011)	362	99.8 (99.2–100)	6.2 (5.4–7.1)	2.1[E] (0.99–3.2)	6.7 (5.8–7.6)	15[E] (9.7–21)	19[E] (9.6–29)
5 (2016–2017)	362	99.9 (99.5–100)	2.5 (2.3–2.8)	1.2 (0.95–1.5)	2.6 (2.2–2.9)	5.1 (4.1–6.1)	6.4[E] (4.0–8.9)
40–59 years							
1 (2007–2009)	983	99.9 (98.7–100)	8.6 (7.7–9.5)	3.4 (2.8–4.0)	8.8 (7.9–9.7)	19 (13–24)	28 (19–37)
2 (2009–2011)	334	99.6 (97.7–99.9)	6.4 (5.7–7.2)	2.3 (1.6–3.0)	6.7 (5.7–7.7)	13 (9.8–17)	16 (13–19)
5 (2016–2017)	346	100	3.8 (3.1–4.7)	1.4 (1.1–1.6)	3.4 (2.9–4.0)	F	19[E] (5.2–33)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	918	100	11 (9.6–13)	4.4 (3.3–5.5)	11 (9.6–13)	24 (21–28)	30 (24–35)
2 (2009–2011)	321	100	9.4 (8.3–11)	4.6 (3.9–5.3)	9.8 (8.1–11)	19 (16–21)	21[E] (7.5–35)
5 (2016–2017)	349	99.8 (98.9–99.9)	4.5 (3.7–5.6)	1.8[E] (0.81–2.9)	5.0 (4.0–6.0)	9.9 (7.9–12)	12 (10–14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.3, 0.3, and 0.43 µg/L, respectively.

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

b Data are not available, as participants under the age of 20 years were not included in cycle 1.

c Data are not available, as participants under the age of 12 years were not included in cycle 2.

E Use data with caution.

F Data are too unreliable to be published.

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SUMMARIES AND RESULTS FOR PESTICIDES 13

13.1 ORGANOPHOSPHATE PESTICIDES

Organophosphates are a group of closely related chemicals that are extensively used in Canada as pesticides in agriculture, in and around the home, and in veterinary practice (Health Canada, 2013a; Health Canada, 2018a; Health Canada, 2019). This class of pesticides gained popularity in use when organochlorine pesticides were banned in the 1970s. Organophosphate pesticides are less persistent in the environment and less susceptible to pest resistance compared with organochlorine pesticides (Wessels et al., 2003). Eighteen organophosphate pesticides were registered for use in Canada during the Canadian Health Measures Survey (CHMS) cycle 2 sampling period (2009–2011) — the last cycle to report dialkyl phosphate metabolites — while 14 were registered for use during the cycle 5 sampling period (2016–2017). The latter are listed in Table 13.1.1 (Health Canada, 2019).

Organophosphate pesticides have been linked to naturally occurring compounds produced by algae and bacteria; however, their presence in the environment is almost exclusively due to their anthropogenic use as pesticides (Neumann and Peter, 1987). Despite their rapid degradation in the environment, small amounts can be detected in food and drinking water (Hao et al., 2010; Health Canada, 2003; Health Canada, 2004).

Major uses of organophosphates include as an insecticide on food and feed crops, livestock, and ornamental plants; for seed treatment and insect control in food storage areas, greenhouses, and forestry structures; for control of pet parasites; and for mosquito control (Health Canada, 2013a; Health Canada, 2019). Although the majority of organophosphates are used as insecticides, bensulide is used as a selective herbicide to control weeds in turf and cucumbers (Health Canada, 2013a). Dichlorvos and trichlorfon have veterinary drug uses for the control of parasites in livestock (Health Canada, 2018a).

The primary route of exposure for the general public is through ingestion of food previously treated with organophosphate pesticides and drinking water contaminated with agricultural runoff (ATSDR, 1997a; ATSDR, 1997b; ATSDR, 2003). Other routes of exposure include dermal contact and inhalation during the use of products containing organophosphates or during activity in areas previously treated with organophosphates.

Organophosphates are efficiently absorbed through inhalation and ingestion. Absorption following dermal penetration can vary with the specific substance (EPA, 2013). After entry into the body, organophosphate pesticides are metabolized rapidly, primarily in the liver, and excreted in urine (Barr and Needham, 2002). Hydrolysis of the parent compound yields various dialkyl phosphate metabolites. Each metabolite is associated with several different organophosphate pesticides, and many organophosphates can form more than one of these metabolites (Table 13.1.1). These metabolites also occur in the environment following degradation of the parent compound. Dialkyl phosphate metabolites are not considered toxic, but are considered to be biomarkers of exposure to the parent pesticides

or their metabolites in the environment (CDC, 2009; EPA, 2013). In addition to the dialkyl phosphate metabolites, organophosphate parent compounds and other breakdown products can be measured in blood and urine; detection generally reflects exposures over the previous few days (CDC, 2009; EPA, 2013). Examples of organophosphate metabolites other than dialkyl phosphates include 3,5,6-trichloro-2-pyridinol (TCPy), which is formed by the metabolism of chlorpyrifos or chlorpyrifos-methyl, and malathion dicarboxylic acid (DCA), formed by the metabolism of malathion (although metabolism of the parent organophosphates also results in the formation of dialkyl phosphate metabolites). Some organophosphate pesticides, namely acephate and methamidophos, do not breakdown into dialkyl phosphate metabolites (Barr and Needham, 2002; Wessels et al., 2003).

The following table outlines the dialkyl phosphate metabolites that were measured in urine collected from CHMS participants in cycle 5, along with their corresponding organophosphate pesticide parent compounds. There are six dialkyl phosphate metabolites: dimethylphosphate (DMP), dimethylthiophosphate (DMTP), dimethyldithiophosphate (DMDTP), diethylphosphate (DEP), diethylthiophosphate (DETP), and diethyldithiophosphate (DEDTP). The table includes two other organophosphate metabolites that were measured in urine collected from CHMS participants in cycle 3 (TCPy and DCA), along with their corresponding parent compounds.

Table 13.1.1

Parent organophosphate pesticides registered for use in Canada during the cycle 5 sampling period along with dialkyl phosphate metabolites measured in cycle 5 (2016–2017) and other metabolites measured in cycle 3 (2012–2013) of the Canadian Health Measures Survey

Organophosphate pesticide	Dialkyl phosphate metabolites (CASRN)						Other organophosphate metabolites (CASRN)[a]	
	DMP (813-79-5)	DMTP 1112-38-5)	DMDTP (765-80-9)	DEP (598-02-7)	DETP (2465-65-8)	DEDTP (298-06-6)	TCPy (6515-38-4)	DCA (1190-28-9)
Acephate	—	—	—	—	—	—	—	—
Bensulide	—	—	—	—	—	—	—	—
Chlorpyrifos	—	—	—	Yes	Yes	—	Yes	—
Coumaphos	—	—	—	Yes	Yes	—	—	—
Diazinon	—	—	—	Yes	Yes	—	—	—
Dichlorvos	Yes	—	—	—	—	—	—	—
Dimethoate	Yes	Yes	Yes	—	—	—	—	—
Malathion	Yes	Yes	Yes	—	—	—	—	Yes
Naled	Yes	—	—	—	—	—	—	—
Phorate	—	—	—	Yes	Yes	Yes	—	—
Phosmet	Yes	Yes	Yes	—	—	—	—	—
Propetamphos	—	—	—	—	—	—	—	—
Tetrachlorvinphos	Yes	—	—	—	—	—	—	—
Trichlorfon	Yes	—	—	—	—	—	—	—

(Bravo et al., 2004; CDC, 2005; Wessels et al., 2003)

a These chemicals were measured in cycle 3 (2012–2013); the data are available for release in the cycle 5 report.

Organophosphates are cholinesterase-inhibiting pesticides that act to overstimulate the nervous systems of insects and mammals by interrupting the transmission of nerve impulses (EPA, 2013). 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 paralysis, seizures, loss of consciousness, or even death (ATSDR, 1997a; ATSDR, 1997b; ATSDR, 2003; EPA, 2013). However, typical exposure to organophosphate pesticides through food ingestion is generally low. Nevertheless, there is potential for toxic effects resulting from chronic low-dose exposure (Ray and Richards, 2001). Prenatal exposure to organophosphates has been associated with shortened gestation, reduced birth weight, and impaired neurodevelopment in young children (Bouchard et al., 2011; EPA, 2016; Eskenazi et al., 2007; González-Alzaga et al. 2014; Muñoz-Quezada et al., 2013; Rauch et al., 2012). Several organophosphate pesticides registered for use in Canada have been classified by the International Agency for Research on Cancer (IARC). Malathion and diazinon are classified as probably carcinogenic to humans (Group 2A); tetrachlorvinphos and dichlorvos are classified as possibly carcinogenic to humans (Group 2B); trichlorfon's carcinogenicity to humans is not classifiable (Group 3) (IARC, 1983; IARC, 1991; IARC, 2017).

The sale and use of organophosphate pesticides is regulated in Canada by the Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002). 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 maximum residue limits of pesticides in food, including registered organophosphate pesticides (Health Canada, 2012a). Registered pesticides are re-evaluated by PMRA on a cyclical basis. A re-evaluation of malathion found that most uses do not pose unacceptable risks to human health, including commercial products applied in agricultural, non-agricultural, and residential settings, while most uses of diazinon have been phased out due to health and environmental risk concerns, except for limited applications (Health Canada, 2012b; Health Canada, 2013b). Health Canada has recently proposed that products containing dichlorvos are acceptable for continued registration for sale and use in Canada, provided that risk mitigation measures are in place (Health Canada, 2017). Most recently, PMRA has announced a workplan for the prioritization and re-evaluation of pesticides, including the following organophosphates: acephate, bensulide, chlorpyrifos, coumaphos, dichlorvos, naled, phorate, phosmet, propetamphos, and tetrachlorvinphos (Health Canada, 2018b).

Health Canada has established Canadian drinking water quality guidelines that set out the maximum acceptable concentrations of chlorpyrifos, diazinon, dimethoate, malathion, and phorate (Health Canada, 1989a; Health Canada, 1989b; Health Canada, 1989c; Health Canada, 1990; Health Canada, 1991). Several organophosphate pesticides have also been analyzed as part of Health Canada's Total Diet Study surveys (Health Canada, 2016). These surveys provide estimate levels of chemicals that Canadians in different age-sex groups are exposed to through the food supply.

Six dialkyl phosphate metabolites were measured in morning urine voids from 89 children aged 3–7 years in a biomonitoring study in the province of Québec in 2003. The geometric mean and 95th percentile concentrations were 20 µg/g creatinine and 97 µg/g creatinine, respectively, for DMP; 18.8 µg/g creatinine and 210.9 µg/g creatinine, respectively, for DMTP; 2.8 µg/g creatinine and 45.9 µg/g creatinine, respectively, for DMDTP; 4.8 µg/g creatinine and 29 µg/g creatinine, respectively, for DEP; 0.7 µg/g creatinine and 8 µg/g creatinine, respectively, for DETP; and 0.4 µg/g creatinine and 0.4 µg/g creatinine, respectively, for DEDTP (Valcke et al., 2006).

Six dialkyl phosphate metabolites (see Table 13.1.1) were measured in the urine of CHMS participants aged 6–79 years in cycle 1 (2007–2009) and aged 3–79 years in cycle 2 (2009–2011) and cycle 5 (2016–2017). 3,5,6-Trichloro-2-pyridinol and malathion dicarboxylic acid were analyzed in the urine of CHMS participants aged 3–79 years in cycle 3 (2012–2013) and cycle 4 (2014–2015). In addition to the data for organophosphate metabolites discussed above, data are now available and presented below for two organophosphate pesticides, acephate and methamidophos, that were measured in the urine of CHMS participants aged 3–79 years in cycle 3 (2012–2013). Data from these cycles are presented as both µg/L and µg/g creatinine (Tables 13.1.2 to 13.1.21). Finding a measurable amount of organophosphate pesticides or their metabolites in urine is an indicator of exposure to organophosphate pesticides and/or their metabolites and does not necessarily mean that an adverse health effect will occur.

Table 13.1.2

Dimethylphosphate (DMP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2556	83.0 (78.3–86.8)	3.3 (2.9–3.7)	<LOD	3.5 (3.0–4.0)	17 (15–20)	26 (22–29)
5 (2016–2017)	2633	80.9 (75.1–85.6)	1.7 (1.4–2.1)	<LOD	1.6 (1.3–1.9)	8.6 (6.2–11)	14 (10–18)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1280	82.5 (76.3–87.4)	3.3 (2.8–3.8)	<LOD	3.4 (2.8–4.0)	17 (13–21)	26 (21–31)
5 (2016–2017)	1308	77.6 (69.2–84.3)	1.6 (1.3–2.1)	<LOD	1.5 (0.97–2.0)	8.4 (6.5–10)	13 (9.4–18)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	83.4 (77.9–87.8)	3.4 (2.9–3.9)	<LOD	3.6 (2.8–4.5)	17 (14–20)	24 (17–31)
5 (2016–2017)	1325	84.1 (79.6–87.8)	1.8 (1.6–2.2)	<LOD	1.6 (1.4–1.8)	9.9[E] (6.1–14)	16 (10–21)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	93.9 (90.7–96.0)	6.7 (5.6–8.1)	1.4 (1.0–1.8)	6.8 (4.9–8.6)	32[E] (19–46)	F
5 (2016–2017)	545	93.9 (90.2–96.3)	3.2 (2.6–3.8)	0.78 (0.59–0.97)	3.0[E] (1.8–4.1)	15 (12–19)	22 (16–28)
6–11 years							
1 (2007–2009)	1028	80.7 (74.6–85.6)	3.8 (3.3–4.5)	<LOD	4.3 (3.9–4.8)	21 (18–23)	29 (23–36)
2 (2009–2011)	516	92.2 (89.2–94.4)	6.1 (5.2–7.2)	<LOD	5.9 (4.6–7.3)	24[E] (14–35)	F
5 (2016–2017)	515	90.4 (81.4–95.3)	2.9[E] (2.0–4.2)	F	2.7 (1.8–3.6)	17[E] (8.5–26)	28[E] (13–42)
12–19 years							
1 (2007–2009)	980	82.8 (75.0–88.5)	3.9 (3.2–4.7)	<LOD	4.1 (3.3–4.9)	21 (18–24)	28 (23–32)
2 (2009–2011)	512	87.5 (82.1–91.5)	3.8 (3.2–4.5)	<LOD	4.0 (3.2–4.8)	18 (12–24)	30 (19–41)
5 (2016–2017)	519	84.3 (73.6–91.2)	2.1[E] (1.4–3.1)	<LOD	2.0[E] (1.1–2.8)	11[E] (4.9–18)	19[E] (10–27)
20–39 years							
1 (2007–2009)	1162	76.0 (69.3–81.6)	2.7 (2.2–3.3)	<LOD	2.9 (2.2–3.7)	13 (9.8–17)	23[E] (10–36)
2 (2009–2011)	356	81.0 (69.2–89.0)	3.1 (2.4–4.0)	<LOD	3.5 (2.6–4.5)	17 (11–23)	29 (20–39)
5 (2016–2017)	358	76.3 (62.8–86.0)	1.5 (1.2–1.9)	<LOD	1.5 (1.2–1.8)	6.8 (4.4–9.2)	12[E] (6.3–19)
40–59 years							
1 (2007–2009)	1221	74.3 (66.0–81.1)	2.6 (2.1–3.3)	<LOD	2.9[E] (1.4–4.4)	15 (11–18)	24 (18–31)
2 (2009–2011)	360	80.7 (71.4–87.5)	2.8 (2.2–3.7)	<LOD	2.8 (2.1–3.5)	13[E] (5.2–20)	20[E] (12–27)
5 (2016–2017)	347	79.5 (71.1–85.9)	1.6 (1.3–1.9)	<LOD	1.4 (1.2–1.7)	6.8[E] (2.5–11)	11[E] (4.1–18)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
1 (2007–2009)	1076	80.7 (76.1–84.6)	3.1 (2.6–3.6)	<LOD	3.3 (2.7–3.9)	15 (12–17)	20 (15–26)
2 (2009–2011)	290	81.9 (73.2–88.2)	3.1 (2.5–3.7)	<LOD	3.4 (2.7–4.2)	14[E] (7.3–20)	19[E] (9.9–28)
5 (2016–2017)	349	82.5 (73.4–89.0)	1.7 (1.3–2.4)	<LOD	1.6[E] (0.90–2.3)	7.7[E] (4.5–11)	13[E] (6.5–19)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.8, 1, and 0.58 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.3

Dimethylphosphate (DMP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2546	83.0 (78.3–86.8)	3.2 (2.9–3.6)	<LOD	3.0 (2.7–3.3)	15 (11–18)	24 (19–30)
5 (2016–2017)	2606	80.9 (75.1–85.6)	1.7 (1.4–2.0)	<LOD	1.5 (1.3–1.8)	7.2 (5.3–9.0)	12 (8.7–15)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	82.5 (76.3–87.4)	2.8 (2.5–3.1)	<LOD	2.5 (2.1–2.9)	13 (9.6–16)	21 (17–25)
5 (2016–2017)	1298	77.6 (69.2–84.3)	1.4 (1.1–1.8)	<LOD	1.3 (1.0–1.6)	5.7 (4.5–6.9)	9.4 (6.3–13)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1270	83.4 (77.9–87.8)	3.8 (3.2–4.6)	<LOD	3.4 (2.6–4.2)	16 (11–21)	28 (20–36)
5 (2016–2017)	1308	84.1 (79.6–87.8)	2.0 (1.7–2.4)	<LOD	1.8 (1.5–2.0)	8.8 (6.2–11)	13[E] (7.7–18)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	93.9 (90.7–96.0)	12 (9.8–14)	2.6 (1.9–3.3)	12 (8.6–15)	51 (33–68)	100[E] (41–160)
5 (2016–2017)	536	93.9 (90.2–96.3)	5.4 (4.2–6.9)	1.1[E] (0.61–1.6)	5.4 (3.8–7.0)	22 (15–28)	33 (23–43)
6–11 years							
1 (2007–2009)	1025	80.7 (74.6–85.6)	5.9 (5.2–6.7)	<LOD	6.3 (5.0–7.6)	26 (23–30)	40 (36–45)
2 (2009–2011)	514	92.2 (89.2–94.4)	6.9 (6.0–7.9)	<LOD	7.2 (6.0–8.4)	32[E] (18–46)	52[E] (22–83)
5 (2016–2017)	506	90.4 (81.4–95.3)	3.5[E] (2.4–5.1)	<LOD	3.1[E] (1.8–4.3)	19[E] (9.1–29)	26 (17–36)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
1 (2007–2009)	978	82.8 (75.0–88.5)	3.4 (2.9–4.0)	<LOD	3.7 (3.0–4.5)	14 (11–16)	20 (15–24)
2 (2009–2011)	510	87.5 (82.1–91.5)	2.9 (2.5–3.4)	<LOD	2.9 (2.4–3.3)	12 (8.0–15)	18[E] (9.3–27)
5 (2016–2017)	515	84.3 (73.6–91.2)	1.6 (1.2–2.2)	<LOD	1.5 (1.1–1.9)	8.2[E] (5.1–11)	10 (7.8–12)
20–39 years							
1 (2007–2009)	1158	76.0 (69.3–81.6)	3.0 (2.6–3.6)	<LOD	3.0 (2.6–3.3)	12 (9.0–16)	21[E] (13–30)
2 (2009–2011)	354	81.0 (69.2–89.0)	2.6 (2.1–3.4)	<LOD	2.4 (1.8–3.1)	16[E] (8.0–25)	23 (16–31)
5 (2016–2017)	355	76.3 (62.8–86.0)	1.4 (1.1–1.6)	<LOD	1.4 (1.2–1.7)	5.2 (3.7–6.7)	7.0 (5.3–8.8)
40–59 years							
1 (2007–2009)	1216	74.3 (66.0–81.1)	3.4 (2.8–4.2)	<LOD	3.4 (2.6–4.2)	14 (9.9–18)	24 (16–32)
2 (2009–2011)	358	80.7 (71.4–87.5)	2.9 (2.4–3.5)	<LOD	2.8 (2.4–3.2)	9.6 (7.9–11)	16[E] (7.6–25)
5 (2016–2017)	346	79.5 (71.1–85.9)	1.4 (1.1–1.8)	<LOD	1.3 (1.1–1.4)	5.7[E] (2.5–9.0)	9.5[E] (3.9–15)
60–79 years							
1 (2007–2009)	1076	80.7 (76.1–84.6)	4.3 (3.7–5.1)	<LOD	4.3 (3.4–5.1)	16 (14–17)	23 (18–27)
2 (2009–2011)	289	81.9 (73.2–88.2)	3.5 (2.9–4.3)	<LOD	3.7 (2.8–4.7)	13 (10–17)	19 (14–24)
5 (2016–2017)	348	82.5 (73.4–89.0)	2.0 (1.5–2.7)	<LOD	1.9 (1.3–2.5)	7.5[E] (4.0–11)	12[E] (5.5–18)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

Table 13.1.4

Dimethylthiophosphate (DMTP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2559	80.0 (75.1–84.0)	2.7 (2.3–3.2)	<LOD	2.8 (2.2–3.5)	23 (17–28)	37 (27–47)
5 (2016–2017)	2645	70.6 (64.8–75.8)	1.3 (1.1–1.5)	<LOD	1.1 (0.89–1.3)	10 (8.8–12)	20 (15–25)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1281	80.2 (73.8–85.4)	2.5 (2.1–3.0)	<LOD	2.4 (1.8–3.0)	22[E] (13–32)	37[E] (17–57)
5 (2016–2017)	1315	70.6 (60.7–78.9)	1.3 (1.0–1.6)	<LOD	1.1[E] (0.65–1.5)	9.9 (7.3–12)	16[E] (8.3–24)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	79.7 (74.4–84.2)	2.9 (2.4–3.6)	<LOD	3.2 (2.4–4.1)	23 (17–30)	37 (29–45)
5 (2016–2017)	1330	70.6 (64.3–76.2)	1.3 (1.1–1.6)	<LOD	1.1 (0.82–1.4)	11 (8.4–13)	21 (16–25)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	524	92.6 (89.2–95.1)	6.3 (5.1–7.8)	0.72 (<LOD–0.97)	6.4 (4.5–8.3)	49 (33–66)	89 (60–120)
5 (2016–2017)	547	86.6 (81.3–90.6)	2.6 (2.1–3.3)	<LOD	2.1[E] (0.94–3.2)	23[E] (13–33)	39[E] (22–56)
6–11 years							
1 (2007–2009)	1029	67.3 (58.5–75.1)	2.5 (1.9–3.2)	<LOD	2.5[E] (1.4–3.5)	36 (29–42)	54 (45–64)
2 (2009–2011)	516	91.6 (89.4–93.3)	5.0 (4.2–6.0)	<LOD	5.3 (3.7–6.9)	32 (21–43)	66[E] (31–100)
5 (2016–2017)	516	84.6 (80.1–88.3)	2.3 (1.8–3.0)	<LOD	2.0 (1.5–2.4)	24[E] (10–37)	55[E] (24–86)
12–19 years							
1 (2007–2009)	980	68.7 (60.7–75.6)	2.3 (1.8–2.8)	<LOD	2.1 (1.4–2.8)	26 (19–32)	44 (30–58)
2 (2009–2011)	512	80.0 (73.0–85.6)	2.6 (2.1–3.3)	<LOD	2.7 (2.0–3.3)	19[E] (12–26)	36[E] (22–50)
5 (2016–2017)	524	76.5 (68.8–82.8)	1.5 (1.2–2.0)	<LOD	1.4[E] (0.85–1.9)	10[E] (5.6–14)	19[E] (5.5–33)
20–39 years							
1 (2007–2009)	1163	66.0 (57.1–73.9)	1.8 (1.3–2.4)	<LOD	1.6[E] (<LOD–2.6)	17[E] (10–24)	36[E] (19–53)
2 (2009–2011)	356	78.1 (65.6–86.9)	2.4 (1.8–3.2)	<LOD	2.7 (1.8–3.7)	20[E] (10–29)	29[E] (17–41)
5 (2016–2017)	361	60.8 (52.0–68.9)	1.0 (0.80–1.3)	<LOD	0.71[E] (0.44–0.99)	F	20[E] (5.3–35)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1223	63.9 (58.0–69.4)	1.8 (1.5–2.2)	<LOD	1.4[E] (<LOD–2.3)	20 (15–25)	38 (27–49)
2 (2009–2011)	360	79.2 (70.6–85.8)	2.4 (1.8–3.2)	<LOD	2.2[E] (1.2–3.1)	20[E] (7.8–33)	F
5 (2016–2017)	347	70.1 (56.6–80.8)	1.2 (0.89–1.6)	<LOD	1.0 (0.68–1.4)	9.9[E] (5.3–15)	12[E] (6.6–17)
60–79 years							
1 (2007–2009)	1079	73.6 (67.0–79.3)	2.6 (2.2–3.2)	<LOD	3.0 (2.1–3.8)	26 (21–31)	40 (35–45)
2 (2009–2011)	291	77.4 (67.0–85.3)	2.8 (2.1–3.8)	<LOD	3.3[E] (2.1–4.6)	23[E] (12–35)	44[E] (20–68)
5 (2016–2017)	350	75.2 (69.7–80.1)	1.4 (1.1–1.8)	<LOD	1.4 (1.1–1.7)	11[E] (3.6–18)	21[E] (11–32)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.6, 0.6, and 0.44 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.5

Dimethylthiophosphate (DMTP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2549	80.0 (75.1–84.0)	2.7 (2.3–3.1)	<LOD	2.5 (1.8–3.1)	21 (17–25)	35 (31–39)
5 (2016–2017)	2618	70.6 (64.8–75.8)	1.3 (1.1–1.5)	<LOD	1.0 (0.88–1.1)	10 (7.0–13)	19[E] (12–27)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	80.2 (73.8–85.4)	2.1 (1.8–2.5)	<LOD	1.9 (1.5–2.4)	16 (11–22)	28 (18–38)
5 (2016–2017)	1305	70.6 (60.7–78.9)	1.1 (0.91–1.4)	<LOD	0.88 (0.62–1.1)	7.3[E] (3.7–11)	19[E] (7.3–30)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1272	79.7 (74.4–84.2)	3.3 (2.6–4.2)	<LOD	3.3 (2.3–4.4)	27 (20–35)	37 (25–50)
5 (2016–2017)	1313	70.6 (64.3–76.2)	1.4 (1.2–1.7)	<LOD	1.1 (0.89–1.3)	12 (8.2–16)	21[E] (10–33)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	92.6 (89.2–95.1)	11 (9.1–13)	1.3[E] (<LOD–2.1)	11 (8.0–13)	79 (61–98)	110 (87–140)
5 (2016–2017)	538	86.6 (81.3–90.6)	4.5 (3.3–6.1)	<LOD	4.2[E] (2.6–5.8)	39[E] (22–56)	61[E] (37–84)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1026	67.3 (58.5–75.1)	3.8 (3.1–4.8)	<LOD	3.8 (2.7–4.9)	45 (36–54)	70 (52–88)
2 (2009–2011)	514	91.6 (89.4–93.3)	5.7 (4.6–7.0)	<LOD	5.9[E] (3.4–8.4)	40[E] (19–60)	90[E] (31–150)
5 (2016–2017)	507	84.6 (80.1–88.3)	2.8 (2.1–3.6)	<LOD	2.7 (2.0–3.4)	29[E] (9.5–49)	43[E] (18–67)
12–19 years							
1 (2007–2009)	978	68.7 (60.7–75.6)	2.0 (1.6–2.4)	<LOD	2.0 (1.8–2.1)	19 (13–24)	30 (23–36)
2 (2009–2011)	510	80.0 (73.0–85.6)	2.0 (1.6–2.5)	<LOD	1.9 (1.3–2.5)	13[E] (7.6–19)	25[E] (12–38)
5 (2016–2017)	520	76.5 (68.8–82.8)	1.2 (0.95–1.5)	<LOD	1.1 (0.89–1.3)	8.4[E] (4.3–13)	F
20–39 years							
1 (2007–2009)	1159	66.0 (57.1–73.9)	2.0 (1.6–2.6)	<LOD	1.9 (<LOD–2.5)	17[E] (9.0–25)	34[E] (18–51)
2 (2009–2011)	354	78.1 (65.6–86.9)	2.0 (1.6–2.6)	<LOD	2.0 (1.5–2.5)	14[E] (6.8–21)	33[E] (17–50)
5 (2016–2017)	358	60.8 (52.0–68.9)	0.91 (0.67–1.2)	<LOD	0.69 (0.46–0.92)	F	F
40–59 years							
1 (2007–2009)	1218	63.9 (58.0–69.4)	2.3 (1.9–2.7)	<LOD	1.9 (<LOD–2.4)	19 (15–22)	45 (37–54)
2 (2009–2011)	358	79.2 (70.6–85.8)	2.4 (1.9–3.1)	<LOD	2.4[E] (1.4–3.4)	15[E] (7.2–22)	29 (19–40)
5 (2016–2017)	346	70.1 (56.6–80.8)	1.1 (0.90–1.4)	<LOD	0.92 (0.76–1.1)	7.7[E] (2.1–13)	13[E] (6.3–19)
60–79 years							
1 (2007–2009)	1079	73.6 (67.0–79.3)	3.7 (3.1–4.4)	<LOD	3.9 (2.9–5.0)	30 (22–38)	53 (40–67)
2 (2009–2011)	290	77.4 (67.0–85.3)	3.2 (2.4–4.3)	<LOD	3.5[E] (2.1–4.8)	25 (16–34)	F
5 (2016–2017)	349	75.2 (69.7–80.1)	1.6 (1.2–2.1)	<LOD	1.5[E] (0.95–2.1)	F	21[E] (5.7–37)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.6

Dimethyldithiophosphate (DMDTP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2537	49.1 (44.2–53.9)	—	<LOD	<LOD	2.9 (2.4–3.5)	6.5 (5.2–7.8)
5 (2016–2017)	2618	51.8 (46.9–56.6)	—	<LOD	0.097 (<LOD–0.12)	1.4 (0.94–1.9)	4.1[E] (2.6–5.6)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1271	42.2 (37.4–47.2)	—	<LOD	<LOD	2.6[E] (1.5–3.7)	5.7 (3.8–7.6)
5 (2016–2017)	1296	49.5 (43.2–55.8)	—	<LOD	<LOD	1.2[E] (0.64–1.7)	4.1[E] (1.2–7.0)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1266	55.8 (49.5–61.8)	—	<LOD	0.33 (<LOD–0.42)	3.4 (2.5–4.2)	7.8 (5.5–10)
5 (2016–2017)	1322	54.0 (46.4–61.4)	0.17 (0.14–0.21)	<LOD	0.099[E] (<LOD–0.14)	1.5[E] (0.48–2.5)	3.8[E] (1.9–5.7)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	63.0 (55.1–70.2)	0.85 (0.68–1.1)	<LOD	0.57[E] (0.32–0.83)	8.8[E] (4.4–13)	18[E] (9.6–26)
5 (2016–2017)	542	64.0 (53.6–73.2)	0.29[E] (0.20–0.44)	<LOD	0.25[E] (<LOD–0.42)	3.9[E] (2.1–5.7)	F
6–11 years							
1 (2007–2009)	1029	40.7 (32.2–49.7)	—	<LOD	<LOD	3.6 (2.5–4.6)	7.2 (4.8–9.5)
2 (2009–2011)	512	62.0 (55.2–68.3)	—	<LOD	0.49[E] (<LOD–0.75)	4.5[E] (1.7–7.4)	9.3[E] (5.6–13)
5 (2016–2017)	515	68.2 (60.6–75.0)	0.27 (0.20–0.36)	<LOD	0.19 (0.14–0.23)	F	8.0[E] (4.0–12)
12–19 years							
1 (2007–2009)	980	35.1 (28.0–43.0)	—	<LOD	<LOD	2.3[E] (1.4–3.2)	7.0 (4.9–9.1)
2 (2009–2011)	512	43.7 (37.3–50.3)	—	<LOD	<LOD	2.0[E] (0.84–3.1)	F
5 (2016–2017)	521	54.9 (45.2–64.1)	—	<LOD	0.12 (<LOD–0.16)	1.4[E] (0.82–2.0)	4.9[E] (1.9–7.9)
20–39 years							
1 (2007–2009)	1163	34.6 (25.0–45.6)	—	<LOD	<LOD	1.9[E] (1.1–2.7)	4.6[E] (2.4–6.7)
2 (2009–2011)	357	43.9 (34.8–53.5)	—	<LOD	<LOD	2.5[E] (0.79–4.3)	4.4[E] (2.3–6.4)
5 (2016–2017)	360	46.1 (37.6–54.9)	—	<LOD	<LOD	0.73[E] (0.45–1.0)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1223	33.9 (26.9–41.7)	—	<LOD	<LOD	2.4[E] (0.74–4.0)	5.8 (4.3–7.4)
2 (2009–2011)	353	48.1 (41.0–55.3)	—	<LOD	<LOD	2.8[E] (1.7–3.9)	6.1[E] (2.7–9.5)
5 (2016–2017)	346	51.4 (43.0–59.6)	—	<LOD	0.099[E] (<LOD–0.14)	F	F
60–79 years							
1 (2007–2009)	1080	44.5 (40.0–49.2)	—	<LOD	<LOD	3.9[E] (2.4–5.4)	7.5 (5.0–9.9)
2 (2009–2011)	280	55.3 (45.7–64.6)	—	<LOD	F	F	9.5[E] (3.7–15)
5 (2016–2017)	334	51.5 (42.8–60.0)	—	<LOD	0.098[E] (<LOD–0.15)	2.0[E] (0.75–3.2)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.09, 0.3, and 0.093 µg/L, respectively.

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

b Data are not available, as participants under the age of six were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.7

Dimethyldithiophosphate (DMDTP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2527	49.1 (44.2–53.9)	—	<LOD	<LOD	3.3 (2.4–4.2)	7.2 (5.2–9.3)
5 (2016–2017)	2591	51.8 (46.9–56.6)	—	<LOD	0.11 (<LOD–0.13)	1.4 (1.1–1.8)	3.9[E] (1.4–6.4)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1267	42.2 (37.4–47.2)	—	<LOD	<LOD	2.0 (1.5–2.5)	4.4 (3.0–5.8)
5 (2016–2017)	1286	49.5 (43.2–55.8)	—	<LOD	<LOD	1.2[E] (0.61–1.8)	F
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1260	55.8 (49.5–61.8)	—	<LOD	0.40[E] (<LOD–0.55)	4.6 (3.2–6.0)	9.4 (7.5–11)
5 (2016–2017)	1305	54.0 (46.4–61.4)	0.19 (0.14–0.24)	<LOD	0.13[E] (<LOD–0.19)	F	4.9[E] (1.5–8.4)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	63.0 (55.1–70.2)	1.3 (1.0–1.6)	<LOD	0.97 (0.71–1.2)	17[E] (8.4–25)	27[E] (16–38)
5 (2016–2017)	533	64.0 (53.6–73.2)	0.50[E] (0.31–0.81)	<LOD	0.37[E] (<LOD–0.62)	5.5[E] (2.5–8.4)	12[E] (5.4–18)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1026	40.7 (32.2–49.7)	—	<LOD	<LOD	5.5 (3.6–7.4)	11 (8.5–13)
2 (2009–2011)	510	62.0 (55.2–68.3)	—	<LOD	0.50[E] (<LOD–0.70)	6.8[E] (3.1–10)	9.8[E] (5.3–14)
5 (2016–2017)	506	68.2 (60.6–75.0)	0.32 (0.23–0.43)	<LOD	0.22[E] (0.12–0.31)	4.6[E] (1.5–7.7)	7.1[E] (2.6–12)
12–19 years							
1 (2007–2009)	978	35.1 (28.0–43.0)	—	<LOD	<LOD	2.1[E] (1.3–3.0)	5.3[E] (3.2–7.4)
2 (2009–2011)	510	43.7 (37.3–50.3)	—	<LOD	<LOD	1.5[E] (0.84–2.1)	F
5 (2016–2017)	517	54.9 (45.2–64.1)	—	<LOD	0.098 (<LOD–0.13)	1.1[E] (0.60–1.5)	F
20–39 years							
1 (2007–2009)	1159	34.6 (25.0–45.6)	—	<LOD	<LOD	2.0[E] (0.95–3.0)	4.8[E] (2.6–6.9)
2 (2009–2011)	355	43.9 (34.8–53.5)	—	<LOD	<LOD	1.9[E] (1.2–2.6)	4.2[E] (1.3–7.0)
5 (2016–2017)	357	46.1 (37.6–54.9)	—	<LOD	<LOD	1.2[E] (0.48–2.0)	2.0[E] (<LOD–2.7)
40–59 years							
1 (2007–2009)	1218	33.9 (26.9–41.7)	—	<LOD	<LOD	3.2 (2.3–4.1)	8.7 (6.1–11)
2 (2009–2011)	351	48.1 (41.0–55.3)	—	<LOD	<LOD	2.9[E] (0.92–4.9)	6.7[E] (3.0–10)
5 (2016–2017)	345	51.4 (43.0–59.6)	—	<LOD	0.11 (<LOD–0.15)	F	F
60–79 years							
1 (2007–2009)	1080	44.5 (40.0–49.2)	—	<LOD	<LOD	4.6 (2.9–6.2)	9.3[E] (3.8–15)
2 (2009–2011)	279	55.3 (45.7–64.6)	—	<LOD	0.50[E] (<LOD–0.72)	5.3[E] (1.7–8.9)	10[E] (6.0–14)
5 (2016–2017)	333	51.5 (42.8–60.0)	—	<LOD	0.14[E] (<LOD–0.19)	F	7.6[E] (2.9–12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.8

Diethylphosphate (DEP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2556	83.3 (79.1–86.7)	2.8 (2.6–3.1)	<LOD	2.8 (2.5–3.1)	11 (8.4–13)	19 (16–21)
5 (2016–2017)	2646	97.8 (95.9–98.8)	2.2 (2.0–2.5)	0.52 (0.44–0.60)	2.1 (1.8–2.4)	9.9 (8.4–11)	14 (10–17)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	85.3 (81.7–88.3)	2.9 (2.6–3.3)	<LOD	2.9 (2.5–3.4)	10 (8.4–12)	18[E] (11–26)
5 (2016–2017)	1315	97.9 (95.9–98.9)	2.2 (1.9–2.6)	0.53 (0.44–0.61)	2.1 (1.6–2.6)	9.8 (7.9–12)	15 (11–20)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	81.2 (75.4–85.9)	2.7 (2.4–3.1)	<LOD	2.6 (2.2–2.9)	12[E] (7.5–17)	19 (15–23)
5 (2016–2017)	1331	97.7 (93.6–99.2)	2.2 (1.9–2.6)	0.51 (0.37–0.64)	2.1 (1.7–2.5)	9.7 (7.6–12)	12 (8.3–16)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	92.9 (89.5–95.3)	4.9 (4.1–5.9)	1.2 (<LOD–1.6)	5.1 (4.1–6.1)	19[E] (10–27)	29[E] (9.9–48)
5 (2016–2017)	547	98.7 (96.6–99.5)	4.2 (3.4–5.0)	0.99 (0.77–1.2)	4.1 (2.8–5.4)	16 (13–19)	22[E] (11–33)
6–11 years							
1 (2007–2009)	1029	80.9 (68.2–89.3)	2.8 (2.2–3.6)	<LOD	3.0 (2.3–3.6)	11 (9.3–14)	17 (14–20)
2 (2009–2011)	515	92.0 (89.5–94.0)	4.1 (3.7–4.7)	<LOD	4.0 (3.5–4.5)	16 (12–19)	23[E] (12–33)
5 (2016–2017)	516	98.2 (96.5–99.1)	3.2 (2.8–3.6)	0.80 (0.52–1.1)	2.9 (2.2–3.6)	12[E] (7.3–17)	19[E] (11–27)
12–19 years							
1 (2007–2009)	980	82.2 (72.1–89.2)	2.9 (2.4–3.6)	<LOD	3.1 (2.4–3.7)	12 (9.3–15)	18 (14–22)
2 (2009–2011)	512	88.1 (84.3–91.0)	3.4 (3.0–3.9)	<LOD	3.1 (2.6–3.7)	16 (11–22)	23[E] (14–31)
5 (2016–2017)	524	98.6 (97.3–99.3)	2.6 (2.1–3.1)	0.59[E] (0.33–0.85)	2.7 (2.0–3.4)	9.9 (7.4–12)	17 (12–22)
20–39 years							
1 (2007–2009)	1163	77.7 (65.9–86.3)	2.1 (1.7–2.6)	<LOD	2.1 (1.8–2.5)	7.8 (6.0–9.6)	12 (8.6–14)
2 (2009–2011)	357	83.1 (75.7–88.6)	2.7 (2.3–3.2)	<LOD	2.6 (2.2–3.0)	9.5[E] (5.4–14)	20[E] (7.9–32)
5 (2016–2017)	361	95.7 (89.9–98.2)	2.0 (1.6–2.7)	0.47 (0.31–0.62)	1.8[E] (1.1–2.5)	10 (7.6–13)	15[E] (8.9–22)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1223	75.6 (66.0–83.1)	2.1 (1.8–2.4)	<LOD	2.1 (1.8–2.4)	7.6 (5.9–9.2)	11 (8.2–13)
2 (2009–2011)	360	80.3 (71.6–86.9)	2.5 (2.0–3.1)	<LOD	2.5 (1.9–3.1)	9.5[E] (5.3–14)	16[E] (8.6–23)
5 (2016–2017)	348	98.5 (96.0–99.5)	2.0 (1.7–2.3)	0.58 (0.42–0.73)	2.0 (1.6–2.3)	6.8 (5.5–8.0)	8.2 (6.6–9.9)
60–79 years							
1 (2007–2009)	1080	82.4 (74.0–88.5)	2.4 (2.1–2.6)	<LOD	2.3 (2.0–2.7)	8.8 (7.8–9.9)	12 (9.8–13)
2 (2009–2011)	289	80.6 (73.0–86.5)	2.6 (2.0–3.2)	<LOD	2.6 (1.9–3.4)	10[E] (4.7–16)	16 (12–21)
5 (2016–2017)	350	99.0 (96.8–99.7)	2.1 (1.7–2.5)	0.42[E] (<LOD–0.57)	1.9 (1.4–2.4)	9.7 (6.5–13)	13 (10–17)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.5, 1, and 0.29 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

Table 13.1.9

Diethylphosphate (DEP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2546	83.3 (79.1–86.7)	2.7 (2.5–2.9)	<LOD	2.6 (2.3–2.9)	9.5 (8.3–11)	14 (11–17)
5 (2016–2017)	2619	97.8 (95.9–98.8)	2.1 (2.0–2.3)	0.66 (0.52–0.80)	2.1 (1.8–2.3)	7.1 (6.7–7.5)	10 (8.8–11)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1275	85.3 (81.7–88.3)	2.4 (2.1–2.7)	<LOD	2.2 (1.8–2.5)	9.1 (6.9–11)	14 (9.5–18)
5 (2016–2017)	1305	97.9 (95.9–98.9)	1.9 (1.6–2.2)	0.55 (0.40–0.71)	1.8 (1.4–2.1)	7.1 (6.1–8.1)	9.5 (8.1–11)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1271	81.2 (75.4–85.9)	3.1 (2.7–3.5)	<LOD	2.9 (2.5–3.3)	9.9 (7.5–12)	14 (9.9–19)
5 (2016–2017)	1314	97.7 (93.6–99.2)	2.4 (2.1–2.7)	0.80 (0.58–1.0)	2.4 (2.0–2.7)	7.1 (6.5–7.7)	11 (9.1–12)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	92.9 (89.5–95.3)	8.5 (7.3–9.9)	2.6 (<LOD–3.4)	8.6 (7.2–10)	31 (22–41)	44 (33–55)
5 (2016–2017)	538	98.7 (96.6–99.5)	7.1 (6.3–8.0)	1.9 (1.3–2.5)	7.9 (6.7–9.2)	20 (17–24)	32 (21–43)
6–11 years							
1 (2007–2009)	1026	80.9 (68.2–89.3)	4.4 (3.7–5.2)	<LOD	4.2 (3.5–4.9)	16 (13–20)	24 (19–29)
2 (2009–2011)	513	92.0 (89.5–94.0)	4.8 (4.3–5.3)	<LOD	4.5 (3.9–5.2)	18 (13–22)	24[E] (11–38)
5 (2016–2017)	507	98.2 (96.5–99.1)	3.7 (3.1–4.4)	1.1 (0.82–1.3)	3.5 (2.8–4.2)	11 (7.4–14)	16[E] (9.2–24)
12–19 years							
1 (2007–2009)	978	82.2 (72.1–89.2)	2.6 (2.2–3.1)	<LOD	2.6 (2.1–3.1)	8.7 (7.9–9.5)	12 (10–13)
2 (2009–2011)	510	88.1 (84.3–91.0)	2.6 (2.2–3.0)	<LOD	2.5 (2.0–3.0)	9.9 (7.8–12)	15[E] (9.6–21)
5 (2016–2017)	520	98.6 (97.3–99.3)	2.0 (1.7–2.3)	0.55 (0.38–0.72)	1.9 (1.6–2.3)	7.5 (5.5–9.4)	9.2 (7.1–11)
20–39 years							
1 (2007–2009)	1159	77.7 (65.9–86.3)	2.4 (2.0–2.8)	<LOD	2.2 (1.8–2.7)	7.4 (6.6–8.2)	9.9 (8.4–11)
2 (2009–2011)	355	83.1 (75.7–88.6)	2.2 (1.9–2.6)	<LOD	2.0 (1.4–2.6)	7.2[E] (3.5–11)	F
5 (2016–2017)	358	95.7 (89.9–98.2)	1.8 (1.6–2.1)	0.50[E] (0.30–0.70)	1.8 (1.4–2.2)	6.3 (5.2–7.4)	7.2[E] (4.4–10)
40–59 years							
1 (2007–2009)	1218	75.6 (66.0–83.1)	2.7 (2.4–3.0)	<LOD	2.9 (2.6–3.2)	8.9 (7.2–11)	11 (10–13)
2 (2009–2011)	358	80.3 (71.6–86.9)	2.5 (2.2–2.8)	<LOD	2.4 (1.9–2.8)	7.9 (5.5–10)	11 (7.5–15)
5 (2016–2017)	347	98.5 (96.0–99.5)	1.8 (1.6–2.1)	0.72 (0.48–0.95)	1.8 (1.5–2.1)	4.5 (3.6–5.4)	6.4 (4.3–8.4)
60–79 years							
1 (2007–2009)	1080	82.4 (74.0–88.5)	3.4 (3.0–3.7)	<LOD	3.5 (3.0–4.0)	9.9 (8.9–11)	13 (11–16)
2 (2009–2011)	288	80.6 (73.0–86.5)	3.0 (2.5–3.6)	<LOD	2.9 (2.2–3.6)	9.0[E] (5.6–12)	13 (9.9–17)
5 (2016–2017)	349	99.0 (96.8–99.7)	2.4 (2.1–2.8)	0.76 (<LOD–0.99)	2.2 (2.0–2.5)	7.3 (6.0–8.6)	9.5 (6.8–12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.10

Diethylthiophosphate (DETP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2511	72.7 (68.1–76.8)	0.66 (0.60–0.72)	<LOD	0.60 (0.51–0.70)	2.7 (1.9–3.4)	5.3[E] (3.2–7.4)
5 (2016–2017)	2610	75.5 (70.3–80.1)	0.37 (0.33–0.42)	<LOD	0.32 (0.27–0.38)	2.6 (2.1–3.2)	4.4 (3.6–5.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1261	73.1 (67.9–77.7)	0.63 (0.57–0.71)	<LOD	0.58 (0.49–0.67)	2.5 (1.9–3.1)	3.5[E] (1.6–5.5)
5 (2016–2017)	1294	75.7 (66.1–83.3)	0.37 (0.29–0.48)	<LOD	0.33 (0.22–0.45)	2.4[E] (1.1–3.7)	4.5 (2.9–6.2)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1250	72.2 (65.9–77.8)	0.68 (0.59–0.79)	<LOD	0.61 (0.46–0.76)	F	5.6[E] (1.6–9.5)
5 (2016–2017)	1316	75.3 (70.9–79.3)	0.37 (0.29–0.47)	<LOD	0.32 (0.24–0.40)	2.7[E] (1.4–4.1)	4.3[E] (1.5–7.1)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	512	88.2 (83.8–91.5)	1.0 (0.92–1.2)	<LOD	1.0 (0.91–1.1)	4.3[E] (2.7–6.0)	6.7[E] (3.4–10)
5 (2016–2017)	539	88.3 (83.0–92.1)	0.68 (0.51–0.91)	<LOD	0.80[E] (0.47–1.1)	5.2[E] (2.6–7.8)	7.4[E] (4.2–11)
6–11 years							
1 (2007–2009)	1029	41.8 (31.7–52.5)	—	<LOD	<LOD	2.9 (2.1–3.7)	4.8 (3.9–5.7)
2 (2009–2011)	508	80.8 (74.7–85.7)	0.85 (0.74–0.98)	<LOD	0.78 (0.68–0.88)	3.4[E] (2.1–4.6)	F
5 (2016–2017)	511	84.1 (80.2–87.4)	0.54 (0.45–0.65)	<LOD	0.49 (0.32–0.67)	3.2 (2.2–4.2)	5.4[E] (2.9–7.8)
12–19 years							
1 (2007–2009)	979	44.6 (34.2–55.4)	—	<LOD	<LOD	2.6 (1.8–3.3)	4.1 (3.1–5.1)
2 (2009–2011)	504	74.6 (66.2–81.4)	0.67 (0.57–0.78)	<LOD	0.59 (0.47–0.71)	2.6 (2.0–3.2)	4.1[E] (2.5–5.7)
5 (2016–2017)	520	77.1 (66.5–85.2)	0.41 (0.31–0.55)	<LOD	0.35[E] (0.21–0.49)	3.4[E] (1.7–5.1)	5.8[E] (2.9–8.8)
20–39 years							
1 (2007–2009)	1163	36.1 (27.5–45.7)	—	<LOD	<LOD	1.9 (1.5–2.3)	2.9 (1.9–3.8)
2 (2009–2011)	349	67.8 (58.7–75.7)	0.57 (0.48–0.69)	<LOD	0.47[E] (<LOD–0.64)	2.6[E] (1.2–3.9)	5.4[E] (1.6–9.1)
5 (2016–2017)	354	73.3 (60.9–82.9)	0.37 (0.28–0.48)	<LOD	0.31[E] (0.19–0.43)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1223	34.1 (27.0–42.0)	—	<LOD	<LOD	2.2 (1.6–2.8)	4.6[E] (2.8–6.5)
2 (2009–2011)	352	73.6 (64.4–81.2)	0.66 (0.53–0.82)	<LOD	0.65 (0.46–0.84)	F	F
5 (2016–2017)	342	74.5 (67.4–80.6)	0.31 (0.25–0.38)	<LOD	0.29 (0.21–0.37)	2.0[E] (0.63–3.4)	3.9[E] (2.3–5.5)
60–79 years							
1 (2007–2009)	1080	40.0 (32.9–47.5)	—	<LOD	<LOD	2.6 (2.2–3.0)	4.1 (3.5–4.7)
2 (2009–2011)	286	71.6 (63.7–78.3)	0.67 (0.55–0.82)	<LOD	0.59 (0.41–0.76)	2.7[E] (0.76–4.6)	F
5 (2016–2017)	344	74.4 (66.2–81.2)	0.37 (0.28–0.48)	<LOD	0.36[E] (0.20–0.52)	2.1 (1.5–2.8)	3.1[E] (1.1–5.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.08, 0.3, and 0.13 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.11

Diethylthiophosphate (DETP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2501	72.7 (68.1–76.8)	0.60 (0.54–0.66)	<LOD	0.59 (0.50–0.68)	2.8 (2.1–3.4)	4.1 (3.5–4.7)
5 (2016–2017)	2583	75.5 (70.3–80.1)	0.36 (0.32–0.40)	<LOD	0.34 (0.29–0.39)	2.3 (1.7–2.8)	3.8 (2.8–4.7)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1257	73.1 (67.9–77.7)	0.50 (0.44–0.57)	<LOD	0.44 (0.32–0.55)	2.1 (1.5–2.7)	3.3 (2.2–4.4)
5 (2016–2017)	1284	75.7 (66.1–83.3)	0.32 (0.26–0.40)	<LOD	0.29 (0.19–0.39)	1.9 (1.3–2.4)	3.0[E] (1.5–4.6)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1244	72.2 (65.9–77.8)	0.72 (0.60–0.87)	<LOD	0.69 (0.55–0.82)	3.6 (2.8–4.5)	5.2[E] (2.9–7.5)
5 (2016–2017)	1299	75.3 (70.9–79.3)	0.40 (0.32–0.49)	<LOD	0.36 (0.28–0.44)	2.9[E] (1.6–4.1)	3.9[E] (1.9–6.0)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	511	88.2 (83.8–91.5)	1.7 (1.5–2.0)	<LOD	1.6 (1.3–2.0)	6.7[E] (4.2–9.2)	9.6[E] (5.5–14)
5 (2016–2017)	530	88.3 (83.0–92.1)	1.1[E] (0.79–1.7)	<LOD	1.0[E] (0.61–1.5)	6.5[E] (3.7–9.3)	12[E] (3.9–19)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1026	41.8 (31.7–52.5)	—	<LOD	<LOD	4.2 (3.1–5.3)	6.5 (4.5–8.6)
2 (2009–2011)	506	80.8 (74.7–85.7)	0.92 (0.77–1.1)	<LOD	0.89 (0.77–1.0)	3.7 (2.5–4.9)	F
5 (2016–2017)	502	84.1 (80.2–87.4)	0.64 (0.53–0.77)	<LOD	0.64 (0.45–0.83)	3.5 (2.4–4.6)	5.2 (3.6–6.8)
12–19 years							
1 (2007–2009)	977	44.6 (34.2–55.4)	—	<LOD	<LOD	1.9 (1.8–2.1)	3.2 (2.2–4.3)
2 (2009–2011)	502	74.6 (66.2–81.4)	0.47 (0.40–0.55)	<LOD	0.48 (0.37–0.59)	1.7 (1.5–2.0)	2.7[E] (1.4–4.1)
5 (2016–2017)	516	77.1 (66.5–85.2)	0.31 (0.25–0.40)	<LOD	0.27 (0.20–0.34)	1.9[E] (1.0–2.7)	F
20–39 years							
1 (2007–2009)	1159	36.1 (27.5–45.7)	—	<LOD	<LOD	2.0 (1.5–2.5)	3.7 (2.7–4.7)
2 (2009–2011)	347	67.8 (58.7–75.7)	0.45 (0.39–0.52)	<LOD	0.39 (<LOD–0.49)	2.3[E] (1.3–3.3)	3.7[E] (2.3–5.1)
5 (2016–2017)	351	73.3 (60.9–82.9)	0.32 (0.25–0.42)	<LOD	0.28 (0.20–0.36)	2.8[E] (0.85–4.7)	4.5[E] (1.6–7.3)
40–59 years							
1 (2007–2009)	1218	34.1 (27.0–42.0)	—	<LOD	<LOD	3.0 (2.5–3.5)	5.4 (3.5–7.4)
2 (2009–2011)	350	73.6 (64.4–81.2)	0.62 (0.48–0.80)	<LOD	0.60 (0.43–0.76)	2.8[E] (<LOD–4.0)	4.0[E] (<LOD–6.2)
5 (2016–2017)	341	74.5 (67.4–80.6)	0.28 (0.23–0.35)	<LOD	0.31 (0.21–0.40)	1.4[E] (0.58–2.2)	2.4 (1.6–3.2)
60–79 years							
1 (2007–2009)	1080	40.0 (32.9–47.5)	—	<LOD	<LOD	2.9 (2.5–3.4)	4.1 (2.7–5.4)
2 (2009–2011)	285	71.6 (63.7–78.3)	0.71 (0.58–0.88)	<LOD	0.69 (0.54–0.85)	3.5[E] (1.9–5.1)	F
5 (2016–2017)	343	74.4 (66.2–81.2)	0.42 (0.34–0.53)	<LOD	0.38 (0.27–0.49)	2.5[E] (1.5–3.5)	3.4 (2.3–4.4)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.12

Diethyldithiophosphate (DEDTP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2557	3.4[E] (1.8–6.4)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2643	5.8[E] (3.7–9.0)	—	<LOD	<LOD	<LOD	0.072 (<LOD–0.091)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1312	5.9[E] (3.0–11.4)	—	<LOD	<LOD	<LOD	0.074[E] (<LOD–0.10)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1331	5.7 (3.9–8.1)	—	<LOD	<LOD	<LOD	0.071 (<LOD–0.081)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	524	1.0[E] (0.5–1.9)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	547	F	—	<LOD	<LOD	<LOD	0.071 (<LOD–0.093)
6–11 years							
1 (2007–2009)	1029	3.3[E] (2.1–5.2)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	516	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	514	7.1[E] (4.8–10.4)	—	<LOD	<LOD	<LOD	0.079 (<LOD–0.095)
12–19 years							
1 (2007–2009)	980	4.0[E] (2.8–5.8)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	511	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	524	9.7[E] (5.2–17.3)	—	<LOD	<LOD	<LOD	0.10[E] (<LOD–0.17)
20–39 years							
1 (2007–2009)	1163	2.4 (1.8–3.3)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	356	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	361	5.6[E] (2.8–11.0)	—	<LOD	<LOD	<LOD	0.083 (<LOD–0.11)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1223	2.3[E] (1.1–4.4)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	360	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	347	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)	1080	2.4[E] (1.4–4.0)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	290	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	350	6.1[E] (3.4–10.6)	—	<LOD	<LOD	<LOD	0.071 (<LOD–0.092)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.06, 0.3, and 0.067 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.13

Diethyldithiophosphate (DEDTP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2547	3.4[E] (1.8–6.4)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2616	5.8[E] (3.7–9.0)	—	<LOD	<LOD	<LOD	0.13 (<LOD–0.16)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1275	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1302	5.9[E] (3.0–11.4)	—	<LOD	<LOD	<LOD	0.12 (<LOD–0.15)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1272	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1314	5.7 (3.9–8.1)	—	<LOD	<LOD	<LOD	0.15 (<LOD–0.17)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	1.0[E] (0.5–1.9)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	538	F	—	<LOD	<LOD	<LOD	0.20 (<LOD–0.25)
6–11 years							
1 (2007–2009)	1026	3.3[E] (2.1–5.2)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	514	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	505	7.1[E] (4.8–10.4)	—	<LOD	<LOD	<LOD	0.13[E] (<LOD–0.18)
12–19 years							
1 (2007–2009)	978	4.0[E] (2.8–5.8)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	509	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	520	9.7[E] (5.2–17.3)	—	<LOD	<LOD	<LOD	0.13 (<LOD–0.17)
20–39 years							
1 (2007–2009)	1159	2.4 (1.8–3.3)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	354	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	358	5.6[E] (2.8–11.0)	—	<LOD	<LOD	<LOD	0.14[E] (<LOD–0.19)
40–59 years							
1 (2007–2009)	1218	2.3[E] (1.1–4.4)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	358	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	346	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)	1080	2.4[E] (1.4–4.0)	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	289	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	349	6.1[E] (3.4–10.6)	—	<LOD	<LOD	<LOD	0.15 (<LOD–0.18)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.1.14

3,5,6-Trichloro-2-pyridinol (TCPy) — 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 3 (2012–2013) and cycle 4 (2014–2015)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2439	96.8 (94.6–98.1)	1.0 (0.87–1.2)	0.29 (0.23–0.35)	0.99 (0.84–1.1)	3.3 (2.8–3.7)	4.3 (2.7–5.9)
4 (2014–2015)	2422	98.4 (97.0–99.1)	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							
3 (2012–2013)	1215	96.7 (93.1–98.4)	1.0 (0.86–1.3)	0.32 (0.22–0.43)	1.0 (0.85–1.2)	3.3 (2.2–4.4)	F
4 (2014–2015)	1209	98.3 (96.5–99.1)	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							
3 (2012–2013)	1224	96.9 (94.0–98.5)	0.97 (0.85–1.1)	0.25[E] (0.16–0.34)	0.96 (0.82–1.1)	3.2 (2.2–4.2)	5.6 (4.1–7.2)
4 (2014–2015)	1213	98.5 (96.0–99.4)	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							
3 (2012–2013)	470	97.2 (93.9–98.7)	1.0 (0.90–1.2)	0.30[E] (0.15–0.45)	0.99 (0.87–1.1)	3.3 (2.6–4.0)	4.8 (3.4–6.2)
4 (2014–2015)	479	99.0 (97.5–99.6)	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							
3 (2012–2013)	483	97.2 (92.4–99.0)	1.0 (0.84–1.3)	0.27[E] (0.13–0.41)	1.1 (0.93–1.2)	3.6 (2.3–4.9)	4.9 (3.5–6.3)
4 (2014–2015)	489	99.7 (98.4–99.9)	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							
3 (2012–2013)	498	97.7 (92.5–99.3)	1.0 (0.82–1.2)	0.31[E] (<LOD–0.50)	0.98 (0.76–1.2)	3.1 (2.6–3.6)	4.3 (3.0–5.6)
4 (2014–2015)	478	100	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							
3 (2012–2013)	344	98.1 (92.3–99.6)	1.0 (0.77–1.4)	0.27[E] (0.15–0.39)	0.95 (0.70–1.2)	F	F
4 (2014–2015)	336	99.2 (95.5–99.9)	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							
3 (2012–2013)	303	95.9 (89.9–98.4)	0.98 (0.80–1.2)	0.29[E] (0.17–0.41)	0.92 (0.70–1.1)	3.5 (2.5–4.4)	5.9[E] (2.9–9.0)
4 (2014–2015)	299	96.9 (90.6–99.0)	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							
3 (2012–2013)	341	96.1 (87.5–98.9)	1.0 (0.86–1.2)	0.32[E] (0.19–0.46)	1.1 (0.91–1.3)	2.9 (2.3–3.5)	4.4 (2.8–6.0)
4 (2014–2015)	341	98.0 (95.9–99.0)	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)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3 and 4 is 0.13 µg/L.

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 are too unreliable to be published.

Table 13.1.15

3,5,6-Trichloro-2-pyridinol (TCPy) (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 3 (2012–2013) and cycle 4 (2014–2015)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2438	96.8 (94.6–98.1)	1.0 (0.90–1.2)	0.37 (0.32–0.42)	0.96 (0.87–1.1)	3.1 (2.6–3.7)	4.3 (2.7–5.9)
4 (2014–2015)	2421	98.4 (97.0–99.1)	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							
3 (2012–2013)	1215	96.7 (93.1–98.4)	0.87 (0.73–1.0)	0.34 (0.27–0.42)	0.83 (0.71–0.95)	2.6 (1.8–3.4)	F
4 (2014–2015)	1208	98.3 (96.5–99.1)	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							
3 (2012–2013)	1223	96.9 (94.0–98.5)	1.2 (1.1–1.3)	0.47 (0.40–0.55)	1.1 (0.99–1.2)	3.5 (2.9–4.1)	4.9 (3.3–6.6)
4 (2014–2015)	1213	98.5 (96.0–99.4)	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							
3 (2012–2013)	469	97.2 (93.9–98.7)	2.0 (1.8–2.2)	0.81 (0.71–0.90)	1.8 (1.5–2.1)	5.1 (4.2–6.0)	7.4 (5.1–9.7)
4 (2014–2015)	479	99.0 (97.5–99.6)	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							
3 (2012–2013)	483	97.2 (92.4–99.0)	1.3 (1.1–1.6)	0.51 (0.43–0.59)	1.1 (0.89–1.3)	3.8[E] (2.3–5.2)	6.0 (3.8–8.2)
4 (2014–2015)	488	99.7 (98.4–99.9)	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							
3 (2012–2013)	498	97.7 (92.5–99.3)	0.74 (0.64–0.85)	0.31 (<LOD–0.37)	0.70 (0.61–0.79)	1.9 (1.4–2.3)	2.9[E] (1.4–4.4)
4 (2014–2015)	478	100	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							
3 (2012–2013)	344	98.1 (92.3–99.6)	0.79 (0.62–1.0)	0.33[E] (0.21–0.46)	0.67 (0.46–0.88)	F	F
4 (2014–2015)	336	99.2 (95.5–99.9)	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							
3 (2012–2013)	303	95.9 (89.9–98.4)	1.1 (0.96–1.4)	0.39[E] (0.22–0.56)	1.0 (0.91–1.2)	3.5 (2.4–4.5)	4.5[E] (1.8–7.2)
4 (2014–2015)	299	96.9 (90.6–99.0)	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							
3 (2012–2013)	341	96.1 (87.5–98.9)	1.1 (0.95–1.3)	0.47 (0.36–0.58)	1.1 (0.88–1.3)	3.4 (2.5–4.3)	4.1 (3.5–4.6)
4 (2014–2015)	341	98.0 (95.9–99.0)	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)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 13.1.16

Malathion dicarboxylic acid (DCA) — 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 3 (2012–2013) and cycle 4 (2014–2015)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2475	18.8 (14.5–23.9)	—	<LOD	<LOD	0.56[E] (0.21–0.90)	1.2[E] (0.70–1.6)
4 (2014–2015)	2519	16.4 (12.5–21.2)	—	<LOD	<LOD	0.46[E] (0.23–0.69)	0.95[E] (0.46–1.4)
Males, 3–79 years							
3 (2012–2013)	1227	15.8[E] (10.3–23.5)	—	<LOD	<LOD	F	1.3[E] (0.63–2.0)
4 (2014–2015)	1257	15.2 (12.4–18.6)	—	<LOD	<LOD	0.31[E] (<LOD–0.47)	F
Females, 3–79 years							
3 (2012–2013)	1248	21.7 (15.7–29.2)	—	<LOD	<LOD	F	1.1[E] (0.37–1.9)
4 (2014–2015)	1262	17.6[E] (11.8–25.5)	—	<LOD	<LOD	0.68[E] (0.25–1.1)	1.3[E] (0.58–2.0)
3–5 years							
3 (2012–2013)	490	15.1[E] (10.0–22.4)	—	<LOD	<LOD	0.46[E] (<LOD–0.77)	F
4 (2014–2015)	499	19.6 (14.5–26.0)	—	<LOD	<LOD	0.70 (0.44–0.95)	1.8[E] (0.62–3.0)
6–11 years							
3 (2012–2013)	497	17.9[E] (11.9–25.9)	—	<LOD	<LOD	0.68[E] (0.26–1.1)	1.4[E] (0.58–2.3)
4 (2014–2015)	510	26.8 (20.5–34.2)	—	<LOD	<LOD	F	F
12–19 years							
3 (2012–2013)	499	15.6[E] (9.0–25.9)	—	<LOD	<LOD	0.34[E] (<LOD–0.54)	0.68[E] (<LOD–1.2)
4 (2014–2015)	501	13.9 (9.8–19.4)	—	<LOD	<LOD	0.31[E] (<LOD–0.53)	0.89 (0.62–1.2)
20–39 years							
3 (2012–2013)	345	15.5[E] (8.8–25.9)	—	<LOD	<LOD	F	F
4 (2014–2015)	357	12.7[E] (8.0–19.5)	—	<LOD	<LOD	F	0.78[E] (0.23–1.3)
40–59 years							
3 (2012–2013)	304	26.3[E] (16.8–38.6)	—	<LOD	<LOD	F	1.0[E] (0.33–1.7)
4 (2014–2015)	302	18.3[E] (11.1–28.7)	—	<LOD	<LOD	F	F
60–79 years							
3 (2012–2013)	340	12.6[E] (7.8–19.6)	—	<LOD	<LOD	0.33[E] (<LOD–0.51)	F
4 (2014–2015)	350	16.2 (11.6–22.2)	—	<LOD	<LOD	F	1.4[E] (0.52–2.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycles 3 and 4 is 0.19 µg/L.

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 are too unreliable to be published.

Table 13.1.17

Malathion dicarboxylic acid (DCA) (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 3 (2012–2013) and cycle 4 (2014–2015)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2474	18.8 (14.5–23.9)	—	<LOD	<LOD	0.64 (0.46–0.82)	0.99 (0.78–1.2)
4 (2014–2015)	2518	16.4 (12.5–21.2)	—	<LOD	<LOD	0.42 (0.37–0.48)	0.95[E] (0.40–1.5)
Males, 3–79 years							
3 (2012–2013)	1227	15.8[E] (10.3–23.5)	—	<LOD	<LOD	0.43 (<LOD–0.57)	0.80[E] (0.30–1.3)
4 (2014–2015)	1256	15.2 (12.4–18.6)	—	<LOD	<LOD	0.35 (<LOD–0.45)	F
Females, 3–79 years							
3 (2012–2013)	1247	21.7 (15.7–29.2)	—	<LOD	<LOD	0.86 (<LOD–1.1)	1.0 (0.67–1.4)
4 (2014–2015)	1262	17.6[E] (11.8–25.5)	—	<LOD	<LOD	0.46 (0.31–0.61)	F
3–5 years							
3 (2012–2013)	489	15.1[E] (10.0–22.4)	—	<LOD	<LOD	0.98 (<LOD–1.3)	F
4 (2014–2015)	499	19.6 (14.5–26.0)	—	<LOD	<LOD	1.1[E] (0.68–1.5)	F
6–11 years							
3 (2012–2013)	497	17.9[E] (11.9–25.9)	—	<LOD	<LOD	0.88[E] (0.48–1.3)	1.5[E] (0.81–2.2)
4 (2014–2015)	509	26.8 (20.5–34.2)	—	<LOD	<LOD	F	2.1[E] (0.87–3.2)
12–19 years							
3 (2012–2013)	499	15.6[E] (9.0–25.9)	—	<LOD	<LOD	0.34[E] (<LOD–0.51)	F
4 (2014–2015)	501	13.9 (9.8–19.4)	—	<LOD	<LOD	0.32 (<LOD–0.42)	0.48 (0.31–0.65)
20–39 years							
3 (2012–2013)	345	15.5[E] (8.8–25.9)	—	<LOD	<LOD	0.60[E] (0.19–1.0)	F
4 (2014–2015)	357	12.7[E] (8.0–19.5)	—	<LOD	<LOD	0.32 (<LOD–0.42)	0.50[E] (0.19–0.80)
40–59 years							
3 (2012–2013)	304	26.3[E] (16.8–38.6)	—	<LOD	<LOD	0.73[E] (<LOD–1.1)	0.97 (0.73–1.2)
4 (2014–2015)	302	18.3[E] (11.1–28.7)	—	<LOD	<LOD	0.42[E] (<LOD–0.64)	F
60–79 years							
3 (2012–2013)	340	12.6[E] (7.8–19.6)	—	<LOD	<LOD	0.55[E] (<LOD–0.84)	0.89[E] (0.51–1.3)
4 (2014–2015)	350	16.2 (11.6–22.2)	—	<LOD	<LOD	F	1.7[E] (0.82–2.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 13.1.18

Acephate — 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 3 (2012–2013)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2384	16.7 (11.9–23.0)	—	<LOD	<LOD	0.054[E] (0.022–0.085)	0.12[E] (0.033–0.20)
Males, 3–79 years							
3 (2012–2013)	1180	16.9[E] (11.2–24.8)	—	<LOD	<LOD	F	0.22[E] (0.070–0.38)
Females, 3–79 years							
3 (2012–2013)	1204	16.5[E] (10.8–24.2)	—	<LOD	<LOD	0.041[E] (<LOD–0.067)	0.10[E] (0.060–0.14)
3–5 years							
3 (2012–2013)	465	15.8[E] (10.7–22.8)	—	<LOD	<LOD	0.036[E] (<LOD–0.060)	F
6–11 years							
3 (2012–2013)	477	18.1 (12.8–25.1)	—	<LOD	<LOD	0.051[E] (0.020–0.082)	0.12[E] (0.044–0.20)
12–19 years							
3 (2012–2013)	474	15.0 (10.4–21.3)	—	<LOD	<LOD	F	0.098[E] (0.041–0.15)
20–39 years							
3 (2012–2013)	336	17.9[E] (10.8–28.1)	—	<LOD	<LOD	F	F
40–59 years							
3 (2012–2013)	297	F	—	<LOD	<LOD	F	0.097[E] (0.032–0.16)
60–79 years							
3 (2012–2013)	335	17.7[E] (11.9–25.4)	—	<LOD	<LOD	0.052[E] (0.023–0.082)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 3 is 0.018 µg/L.

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 are too unreliable to be published.

Table 13.1.19

Acephate (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 3 (2012–2013)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2383	16.7 (11.9–23.0)	—	<LOD	<LOD	0.057[E] (0.033–0.081)	0.13[E] (0.047–0.21)
Males, 3–79 years							
3 (2012–2013)	1180	16.9[E] (11.2–24.8)	—	<LOD	<LOD	0.052[E] (<LOD–0.087)	0.16[E] (0.045–0.27)
Females, 3–79 years							
3 (2012–2013)	1203	16.5[E] (10.8–24.2)	—	<LOD	<LOD	0.061[E] (<LOD–0.094)	0.12[E] (0.035–0.21)
3–5 years							
3 (2012–2013)	464	15.8[E] (10.7–22.8)	—	<LOD	<LOD	0.096 (<LOD–0.13)	F
6–11 years							
3 (2012–2013)	477	18.1 (12.8–25.1)	—	<LOD	<LOD	0.067[E] (0.033–0.10)	F
12–19 years							
3 (2012–2013)	474	15.0 (10.4–21.3)	—	<LOD	<LOD	F	0.071[E] (0.035–0.11)
20–39 years							
3 (2012–2013)	336	17.9[E] (10.8–28.1)	—	<LOD	<LOD	F	F
40–59 years							
3 (2012–2013)	297	F	—	<LOD	<LOD	0.044[E] (<LOD–0.076)	F
60–79 years							
3 (2012–2013)	335	17.7[E] (11.9–25.4)	—	<LOD	<LOD	F	0.13[E] (<LOD–0.20)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 13.1.20

Methamidophos — 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 3 (2012–2013)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2384	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
3 (2012–2013)	1180	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
3 (2012–2013)	1204	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
3 (2012–2013)	465	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
3 (2012–2013)	477	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	474	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	336	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	297	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	335	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 3 is 0.028 µg/L.

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

F Data are too unreliable to be published.

Table 13.1.21

Methamidophos (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 3 (2012–2013)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	2383	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
3 (2012–2013)	1180	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
3 (2012–2013)	1203	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
3 (2012–2013)	464	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
3 (2012–2013)	477	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	474	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	336	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	297	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	335	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

F Data are too unreliable to be published.

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13.2 PYRETHROIDS

Pyrethrins are naturally occurring compounds found in certain chrysanthemum flowers (ATSDR, 2003). They have been used for their insecticidal properties since the early 1800s in Asia to control ticks and various insects, such as fleas and mosquitoes (ATSDR, 2003). Pyrethroids are synthetic versions of pyrethrins that have been structurally altered to improve their efficacy as pesticides by increasing their stability in the environment and their toxicity (ATSDR, 2003; EPA, 2017). Many commercial pyrethroid pesticides are currently registered for use in Canada. Table 13.2.1 lists the pesticides that are the parent compounds of the metabolites measured in cycle 5 of the Canadian Health Measures Survey (CHMS) (Health Canada, 2019). Other pyrethroid insecticides, such as tetramethrin and bifenthrin, are registered in Canada but are not included in the table, as they do not form the metabolites analyzed in the current cycle.

Pyrethroids enter the environment primarily because of their use as pesticides; however, they break down rapidly and, as a result, only trace amounts of the chemicals are typically found in air, water, soil, and food (ATSDR, 2003). Pyrethroids degrade to carboxylic and phenoxybenzoic metabolites in the environment; these metabolites have been measured in dust collected from homes and daycare centres (Starr et al., 2008). Pyrethroids bind strongly to soil particles, and thus they usually do not leach into the groundwater but rather remain in the soil (ATSDR, 2003).

Pyrethroid pesticides are used in Canada for insect control on agricultural crops and on turf; in orchards, nurseries, and greenhouses; as a general indoor and outdoor residential insecticide for controlling crawling and flying insect pests; for controlling adult mosquitoes around buildings; in cattle ear tags; for controlling mites in bee colonies; and for flea and tick control on pets (Health Canada, 2004; Health Canada, 2019). In malaria-endemic zones, pyrethroids are used to impregnate mosquito nets and clothing to prevent malaria (Health Canada, 2004). The use of pyrethrins and pyrethroids has increased during the past decade with the declining use of organophosphate pesticides, which are more acutely toxic to birds and mammals (EPA, 2017).

Permethrin is the most widely used pyrethroid pesticide in Canada, and is found in more than 350 registered pesticide products (CCME, 2006; Health Canada, 2019). It is used for a variety of agricultural, livestock, forestry, and residential insect control applications. In addition to pesticide uses, permethrin is used in medications to treat scabies (Health Canada, 2013). Cyfluthrin and beta-cyfluthrin are used as agricultural and surface insecticides to control crawling and flying insect pests (Health Canada, 2019). Cypermethrin has agricultural, forestry, livestock, and non-crop industrial uses (Health Canada, 2018c). Lambda-cyhalothrin is used for a variety of agricultural, turf, livestock, and structural purposes (Health Canada, 2017a). Deltamethrin is used in several agricultural applications, on turf, and in greenhouses; it is also used to treat sleeping areas and clothing in malaria-affected countries (Health Canada, 2004; Health Canada, 2009). D-phenothrin is used primarily in residential settings, whereas fluvalinate-tau is used to control mites in bee colonies (Health Canada, 2009).

The primary routes of exposure for the general population are through the use of products that contain pyrethroids, such as household insecticides and pet sprays, and through the ingestion of pyrethroid residues in food (EPA, 2009a).

Pyrethroid pesticides are rapidly metabolized and eliminated from the body through hydrolysis, oxidation, and conjugation. Following oral ingestion, inhalation, or dermal exposure, pyrethroids are metabolized into carboxylic and phenoxybenzoic acids and excreted in urine. Pyrethroids and metabolites can be measured in blood and urine, and are reflective of recent exposure to the parent compound or the metabolite (as an environmental degradate) in the environment (ATSDR, 2003; CDC, 2009; Kuhn et al., 1999; Starr et al., 2008). Urinary metabolites of pyrethroids can be specific to one pyrethroid or common to several pyrethroids. Table 13.2.1 outlines the pyrethroid metabolites measured as part of this survey and their corresponding parent compounds that are registered for use in Canada.

Table 13.2.1

Pyrethroid pesticide metabolites measured in the Canadian Health Measures Survey cycle 5 (2016–2017) and their parent pesticide compounds.

Pyrethroid pesticide (CASRN)	Metabolite (CASRN)
Cypermethrin (52315-07-8) Deltamethrin (52918-63-5) Permethrin (52645-53-1) *lambda*-Cyhalothrin (91465-08-6) D-Phenothrin (26046-85-5) Fluvalinate-tau (102851-06-9)	3-PBA: 3-phenoxybenzoic acid (3739-38-6)
Cyfluthrin (68359-37-5) Flumethrin (69770-45-2)	4-F-3-PBA: 4-fluoro-3-phenoxybenzoic acid (77279-89-1)
Deltamethrin (52918-63-5)	*cis*-DBCA: *cis*-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropane-1-carboxylic acid (63597-73-9)
Cyfluthrin (68359-37-5) Permethrin (52645-53-1) Cypermethrin (52315-07-8)	*cis*-DCCA: *cis*-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (55701-05-8)
Cyfluthrin (68359-37-5) Permethrin (52645-53-1) Cypermethrin (52315-07-8)	*trans*-DCCA: *trans*-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (55701-03-6)

(Barr and Needham, 2002; CDC, 2009; Fortin et al., 2008; Starr et al., 2008)

Pyrethroids, much like the naturally occurring pyrethrins, primarily affect the nervous systems of insects and mammals (Davies et al., 2007). They act on the axons in the peripheral and central nervous systems by prolonging the opening time of small conductance sodium channels, leading to membrane depolarizations and excess excitability. This action causes paralysis in target insect pests, eventually resulting in death. Pyrethroids are more than 2,000 times more toxic to insects than mammals because insects have higher sodium channel sensitivity, smaller body sizes, and lower body temperatures (Bradberry et al., 2005). Mammals are also able to quickly metabolize pyrethroids into their inactive forms and eliminate them (Health Canada, 2009).

Adverse effects can include dizziness, nausea, headaches, tremor, salivation, involuntary movements and seizures; very high exposures may result in unconsciousness (ATSDR, 2003; CDC, 2005). There is evidence for neurobehavioural effects, such as decreased motor activity, in laboratory animals following oral exposure to pyrethroid pesticides (Wolansky and Harrill, 2008). However, there remains a general lack of evidence concerning long-term exposures to low levels of pyrethroids and neurological and reproductive effects in mammals, which may be due to the rapid metabolism and elimination of these compounds from the body (ATSDR, 2003; Kolaczinski and Curtis, 2004; Saillenfait et al., 2015). Allergic reactions in humans have been reported following exposure to pyrethroids; however, the United States Environmental Protection Agency (EPA) found no clear and consistent pattern of effects reported to indicate conclusively whether there is an association between pyrethroid exposure and asthma and allergies (EPA, 2009b; Moretto, 1991; Salome et al., 2000; Vanden Driessche et al., 2010). The International Agency for Research on Cancer (IARC) has classified permethrin and deltamethrin as not classifiable as to its carcinogenicity to humans based on a lack of evidence (Group 3) (IARC, 1991).

The sale and use of pyrethroid pesticides is regulated in Canada by the Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002). PMRA evaluates toxicity and potential exposure in order to determine whether a pesticide should be registered for a specific use. As part of this registration process, PMRA specifies maximum residue limits of pesticides in food. Maximum residue limits exist for several pyrethroid pesticides in food, including cyfluthrin, cypermethrin, deltamethrin, lambda-cyhalothrin, and permethrin (Health Canada, 2012). PMRA re-evaluates registered pesticides on a cyclical basis. As part of this process, Health Canada has completed the re-evaluation of deltamethrin, cyfluthrin, cypermethrin, and d-phenothrin, and determined that most uses do not present unacceptable risks to humans or the environment when used according to product label directions. As such, these products were granted continued registration (Health Canada, 2016; Health Canada 2018a; Health Canada 2018b; Health Canada 2018c). Health Canada has recently published proposed

decisions for permethrin and lambda-cyhalothrin (Health Canada, 2017a; Health Canada, 2017b); final re-evaluation decisions for these chemicals will be published in 2019. Fluvalinate-tau is listed on PMRA's workplan for the prioritization and re-evaluation of pesticides extending to 2023 (Health Canada, 2018d).

Pyrethroid metabolites were measured in 89 children (6–12 years) and 81 adults (18–64 years) in the province of Québec in 2005 (Fortin et al., 2008). Metabolites were identified in urine collected for 12 hours from children and in urine collected for two consecutive 12-hour periods in adults. In children, the median and 95th percentile concentrations were <0.005 µg/L and 0.02 µg/L, respectively, for 4-F-3-PBA; <0.006 µg/L and 0.09 µg/L, respectively, for *cis*-DBCA; 0.10 µg/L and 0.76 µg/L, respectively, for *cis*-DCCA; 0.24 µg/L and 4.10 µg/L, respectively, for *trans*-DCCA; and 0.20 µg/L and 1.54 µg/L, respectively, for 3-PBA.

In adults, the median and 95th percentile concentrations were <0.005 µg/L and 0.03 µg/L, respectively, for 4-F-3-PBA; <0.006 µg/L and 0.14 µg/L, respectively, for *cis*-DBCA; 0.10 µg/L and 1.15 µg/L, respectively, for *cis*-DCCA; 0.25 µg/L and 3.48 µg/L, respectively, for *trans*-DCCA; and 0.17 µg/L and 4.23 µg/L, respectively, for 3-PBA (Fortin et al., 2008).

Ferland et al. (2015) reported geometric mean (GM) concentrations ranging between 0.038 and 0.605 µmol/mol creatinine for *trans*-DCCA and between 0.032 and 2.56 µmol/mol creatinine for 3-PBA over the course of a three-day sampling period in 12 workers on a corn production farm in Québec (92% male, mean age of 39 years). In a study of 26 agricultural workers in Québec exposed to cypermethrin (85% male, median age of 37 years, eight of whom provided two serial urinary measurements following different tasks), the GM concentrations of 3-PBA in urine during a three-day period ranged between 0.080 and 1.668 µmol/mol creatinine, while most of the samples for *trans*-DCCA were below the LOD (Ratelle et al., 2016).

Five pyrethroid metabolites (see Table 13.2.1) were measured in the urine of CHMS participants aged 6–79 years in cycle 1 (2007-2009) and aged 3–79 years in cycle 2 (2009-2011) and cycle 5 (2016–2017). Data from these cycles are presented as both µg/L and µg/g creatinine (Tables 13.2.2 to 13.2.11). Finding a measurable amount of pyrethroid metabolites in urine is an indicator of exposure to pyrethroid pesticides and does not necessarily mean that an adverse health effect will occur.

Table 13.2.2

3-Phenoxybenzoic acid (3-PBA) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2516	100	0.43 (0.35–0.53)	0.079 (0.066–0.091)	0.36 (0.29–0.43)	2.6[E] (1.6–3.6)	5.9[E] (2.2–9.5)
5 (2016–2017)	2706	100 (99.9–100)	0.53 (0.42–0.66)	0.091 (0.065–0.12)	0.46 (0.37–0.55)	3.6[E] (1.7–5.4)	9.7[E] (3.6–16)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1256	100	0.37 (0.30–0.46)	0.073 (0.053–0.092)	0.33 (0.27–0.39)	1.9[E] (0.85–3.0)	F
5 (2016–2017)	1348	100 (99.9–100)	0.46 (0.37–0.56)	0.074[E] (0.044–0.10)	0.41 (0.30–0.52)	2.6 (1.7–3.4)	4.3[E] (2.5–6.1)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1260	100	0.49 (0.37–0.64)	0.084 (0.069–0.10)	0.38 (0.27–0.48)	3.9[E] (1.6–6.2)	F
5 (2016–2017)	1358	100 (99.7–100)	0.62 (0.46–0.83)	0.096 (0.082–0.11)	0.49 (0.36–0.61)	F	15[E] (6.1–24)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	99.9 (99.4–100)	0.32 (0.23–0.45)	0.078 (0.057–0.099)	0.27 (0.21–0.33)	F	F
5 (2016–2017)	551	100	0.40 (0.30–0.54)	0.079[E] (0.047–0.11)	0.33 (0.25–0.42)	2.9[E] (0.80–5.1)	6.3[E] (3.7–9.0)
6–11 years							
1 (2007–2009)	1025	99.3 (97.9–99.8)	0.21 (0.16–0.28)	0.047 (0.034–0.060)	0.19 (0.14–0.24)	1.1 (0.76–1.4)	1.7[E] (0.51–2.9)
2 (2009–2011)	515	100	0.30 (0.25–0.35)	0.079 (0.063–0.095)	0.24 (0.19–0.30)	1.2 (0.79–1.6)	F
5 (2016–2017)	534	100	0.47 (0.39–0.57)	0.10 (0.082–0.12)	0.38 (0.30–0.45)	2.7[E] (1.4–4.0)	F
12–19 years							
1 (2007–2009)	977	99.8 (98.8–100)	0.28 (0.21–0.38)	0.059[E] (0.030–0.088)	0.25 (0.18–0.32)	2.0[E] (1.0–3.0)	3.2[E] (2.0–4.5)
2 (2009–2011)	509	100	0.36 (0.29–0.45)	0.096 (0.085–0.11)	0.27 (0.20–0.35)	2.3[E] (1.2–3.4)	5.6[E] (2.8–8.3)
5 (2016–2017)	533	100	0.45 (0.34–0.60)	0.094[E] (0.053–0.13)	0.37 (0.28–0.47)	3.0[E] (1.8–4.2)	F
20–39 years							
1 (2007–2009)	1159	99.6 (97.5–99.9)	0.25 (0.20–0.32)	0.051 (0.036–0.067)	0.21 (0.16–0.26)	1.4 (1.0–1.8)	2.5[E] (1.6–3.5)
2 (2009–2011)	345	100	0.61[E] (0.41–0.91)	0.094[E] (0.056–0.13)	0.48[E] (0.28–0.67)	F	F
5 (2016–2017)	375	100 (99.9–100)	0.61[E] (0.34–1.1)	0.074[E] (0.032–0.12)	0.48[E] (0.29–0.67)	F	F
40–59 years							
1 (2007–2009)	1216	99.2 (98.4–99.6)	0.27 (0.21–0.34)	0.046 (0.032–0.060)	0.25 (0.18–0.32)	1.7[E] (0.91–2.5)	3.5[E] (2.0–5.0)
2 (2009–2011)	346	100	0.40 (0.29–0.55)	0.064[E] (0.041–0.088)	0.36 (0.24–0.48)	2.4[E] (1.2–3.5)	4.2[E] (2.0–6.4)
5 (2016–2017)	359	99.9 (99.5–100)	0.55 (0.41–0.74)	0.12[E] (0.060–0.19)	0.53 (0.34–0.71)	2.7[E] (1.5–4.0)	F
60–79 years							
1 (2007–2009)	1073	99.3 (96.8–99.8)	0.24 (0.20–0.29)	0.051 (0.041–0.062)	0.21 (0.17–0.25)	1.3 (0.88–1.7)	2.2 (1.5–2.8)
2 (2009–2011)	279	100	0.36[E] (0.24–0.54)	0.074 (0.055–0.093)	0.27[E] (0.14–0.41)	2.4[E] (0.97–3.8)	F
5 (2016–2017)	354	100	0.48[E] (0.33–0.70)	0.075[E] (0.041–0.11)	0.41 (0.27–0.54)	F	9.3[E] (4.4–14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.01, 0.01, and 0.012 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.3

3-Phenoxybenzoic acid (3-PBA) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2506	100	0.42 (0.34–0.51)	0.10 (0.093–0.11)	0.33 (0.26–0.39)	2.3[E] (1.2–3.4)	F
5 (2016–2017)	2676	100 (99.9–100)	0.52 (0.43–0.62)	0.11 (0.088–0.13)	0.39 (0.33–0.44)	3.2 (2.1–4.2)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1252	100	0.31 (0.26–0.38)	0.088 (0.072–0.10)	0.26 (0.21–0.32)	1.3[E] (0.69–1.9)	2.7[E] (0.77–4.6)
5 (2016–2017)	1333	100 (99.9–100)	0.39 (0.34–0.46)	0.099 (0.083–0.12)	0.29 (0.22–0.36)	2.3[E] (1.5–3.2)	3.7 (2.9–4.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1254	100	0.56 (0.44–0.72)	0.11 (0.093–0.13)	0.41 (0.31–0.51)	F	F
5 (2016–2017)	1343	100 (99.7–100)	0.68 (0.53–0.88)	0.15 (0.12–0.17)	0.50 (0.41–0.59)	F	18[E] (6.3–29)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	99.9 (99.4–100)	0.56 (0.40–0.78)	0.15 (0.10–0.19)	0.46 (0.35–0.57)	F	F
5 (2016–2017)	542	100	0.70 (0.58–0.85)	0.16[E] (0.081–0.25)	0.53 (0.42–0.65)	4.2[E] (1.8–6.6)	F
6–11 years							
1 (2007–2009)	1022	99.3 (97.9–99.8)	0.32 (0.26–0.40)	0.099 (0.082–0.12)	0.27 (0.22–0.33)	1.3[E] (0.72–1.9)	3.0[E] (1.4–4.6)
2 (2009–2011)	513	100	0.34 (0.29–0.41)	0.12 (0.10–0.14)	0.26 (0.19–0.33)	1.3 (0.89–1.7)	2.7[E] (1.1–4.2)
5 (2016–2017)	526	100	0.56 (0.47–0.68)	0.15 (0.13–0.18)	0.41 (0.32–0.49)	3.3[E] (1.6–5.1)	F
12–19 years							
1 (2007–2009)	975	99.8 (98.8–100)	0.25 (0.19–0.32)	0.067 (0.052–0.082)	0.19 (0.14–0.25)	1.4[E] (0.83–2.0)	2.9[E] (1.7–4.0)
2 (2009–2011)	507	100	0.27 (0.22–0.34)	0.081 (0.070–0.092)	0.21 (0.15–0.26)	1.4[E] (0.89–2.0)	2.6 (2.1–3.1)
5 (2016–2017)	526	100	0.35 (0.28–0.44)	0.087 (0.075–0.098)	0.28 (0.24–0.32)	1.9[E] (0.54–3.2)	4.9[E] (1.6–8.1)
20–39 years							
1 (2007–2009)	1155	99.6 (97.5–99.9)	0.28 (0.22–0.35)	0.073 (0.052–0.094)	0.23 (0.17–0.29)	1.3[E] (0.75–1.9)	2.4 (1.5–3.2)
2 (2009–2011)	343	100	0.52[E] (0.36–0.75)	0.10 (0.083–0.13)	0.35[E] (0.22–0.48)	F	F
5 (2016–2017)	371	100 (99.9–100)	0.54[E] (0.34–0.86)	0.099 (0.081–0.12)	0.37[E] (0.17–0.56)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1211	99.2 (98.4–99.6)	0.34 (0.28–0.43)	0.084 (0.068–0.10)	0.30 (0.24–0.35)	1.9[E] (1.1–2.6)	2.9 (2.2–3.7)
2 (2009–2011)	344	100	0.41 (0.33–0.50)	0.10 (0.088–0.12)	0.35 (0.25–0.45)	1.8[E] (1.2–2.5)	3.4[E] (1.2–5.6)
5 (2016–2017)	358	99.9 (99.5–100)	0.51 (0.43–0.60)	0.13 (0.11–0.16)	0.39 (0.29–0.49)	3.0[E] (1.9–4.2)	F
60–79 years							
1 (2007–2009)	1073	99.3 (96.8–99.8)	0.34 (0.28–0.40)	0.090 (0.079–0.10)	0.29 (0.24–0.34)	1.6[E] (0.94–2.2)	3.0 (1.9–4.1)
2 (2009–2011)	278	100	0.42[E] (0.29–0.63)	0.092 (0.075–0.11)	0.32 (0.24–0.41)	F	F
5 (2016–2017)	353	100	0.55 (0.40–0.76)	0.11[E] (0.066–0.15)	0.44 (0.31–0.58)	3.3[E] (<LOD–5.7)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.4

4-Fluoro-3-phenoxybenzoic acid (4-F-3-PBA) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2539	58.6 (53.1–63.9)	—	<LOD	0.0091 (<LOD–0.010)	0.049[E] (0.028–0.070)	0.11[E] (0.040–0.17)
5 (2016–2017)	2649	34.5 (28.3–41.4)	—	<LOD	<LOD	0.048 (0.036–0.060)	0.082 (0.055–0.11)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1268	57.4 (52.0–62.6)	—	<LOD	0.0090 (<LOD–0.012)	0.055[E] (0.032–0.079)	0.10[E] (0.044–0.16)
5 (2016–2017)	1319	34.2 (26.0–43.5)	—	<LOD	<LOD	0.049[E] (0.027–0.072)	0.082 (0.060–0.10)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1271	59.8 (52.5–66.6)	—	<LOD	0.0092 (0.0083–0.010)	0.049[E] (0.015–0.082)	F
5 (2016–2017)	1330	34.9 (28.3–42.1)	—	<LOD	<LOD	0.044 (0.031–0.057)	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	517	49.6 (41.1–58.2)	—	<LOD	<LOD	0.043 (0.031–0.056)	0.050 (0.032–0.067)
5 (2016–2017)	539	34.5[E] (20.6–51.7)	—	<LOD	<LOD	0.047[E] (0.017–0.077)	0.079[E] (0.040–0.12)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	998	41.5 (36.3–46.8)	—	<LOD	<LOD	0.023[E] (0.014–0.032)	F
2 (2009–2011)	514	56.7 (48.3–64.8)	—	<LOD	0.0087 (<LOD–0.011)	0.037 (0.026–0.048)	0.056[E] (0.028–0.085)
5 (2016–2017)	525	38.4 (29.2–48.6)	—	<LOD	<LOD	0.041[E] (0.018–0.064)	0.072[E] (0.035–0.11)
12–19 years							
1 (2007–2009)	947	50.7 (45.3–56.0)	—	<LOD	F	0.035 (0.025–0.045)	0.060[E] (0.017–0.10)
2 (2009–2011)	510	58.8 (50.5–66.7)	—	<LOD	0.0090 (<LOD–0.011)	0.032[E] (0.0098–0.054)	F
5 (2016–2017)	530	30.1 (23.5–37.7)	—	<LOD	<LOD	0.039[E] (0.013–0.064)	0.071 (0.051–0.090)
20–39 years							
1 (2007–2009)	1100	44.7 (36.9–52.8)	—	<LOD	<LOD	0.038[E] (0.015–0.062)	0.089[E] (0.030–0.15)
2 (2009–2011)	352	61.3 (49.9–71.6)	—	<LOD	0.0093 (<LOD–0.012)	F	0.11[E] (0.033–0.19)
5 (2016–2017)	367	32.8[E] (21.1–47.2)	—	<LOD	<LOD	0.057[E] (0.031–0.083)	F
40–59 years							
1 (2007–2009)	1161	39.6 (32.2–47.5)	—	<LOD	<LOD	0.037 (0.026–0.048)	0.079[E] (0.048–0.11)
2 (2009–2011)	357	62.8 (51.5–72.9)	—	<LOD	0.0094 (0.0083–0.010)	0.057[E] (0.018–0.097)	F
5 (2016–2017)	345	40.7 (28.2–54.5)	—	<LOD	<LOD	0.046[E] (0.020–0.073)	F
60–79 years							
1 (2007–2009)	1018	38.6 (32.7–44.8)	—	<LOD	<LOD	0.032[E] (0.014–0.050)	0.069[E] (0.021–0.12)
2 (2009–2011)	289	48.6 (42.7–54.5)	—	<LOD	<LOD	F	F
5 (2016–2017)	343	29.3 (20.8–39.6)	—	<LOD	<LOD	F	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.008, 0.008, and 0.0060 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.5

4-Fluoro-3-phenoxybenzoic acid (4-F-3-PBA) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2529	58.6 (53.1–63.9)	—	<LOD	0.0099 (<LOD–0.012)	0.048[E] (0.030–0.066)	F
5 (2016–2017)	2619	34.5 (28.3–41.4)	—	<LOD	<LOD	0.034 (0.026–0.043)	0.084[E] (0.045–0.12)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1264	57.4 (52.0–62.6)	—	<LOD	0.0079 (<LOD–0.010)	0.036[E] (0.019–0.054)	F
5 (2016–2017)	1304	34.2 (26.0–43.5)	—	<LOD	<LOD	0.036[E] (0.021–0.052)	0.085[E] (0.036–0.13)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1265	59.8 (52.5–66.6)	—	<LOD	0.0099 (0.0093–0.011)	0.050[E] (0.024–0.076)	F
5 (2016–2017)	1315	34.9 (28.3–42.1)	—	<LOD	<LOD	0.032 (0.021–0.044)	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	516	49.6 (41.1–58.2)	—	<LOD	<LOD	0.048[E] (0.030–0.066)	0.091[E] (0.056–0.12)
5 (2016–2017)	530	34.5[E] (20.6–51.7)	—	<LOD	<LOD	0.062[E] (0.036–0.089)	F
6–11 years							
1 (2007–2009)	995	41.5 (36.3–46.8)	—	<LOD	<LOD	0.039 (0.031–0.048)	0.071[E] (0.044–0.099)
2 (2009–2011)	512	56.7 (48.3–64.8)	—	<LOD	0.0092 (<LOD–0.0097)	0.037 (0.026–0.048)	0.065[E] (0.024–0.11)
5 (2016–2017)	517	38.4 (29.2–48.6)	—	<LOD	<LOD	0.052[E] (0.022–0.082)	0.098[E] (0.043–0.15)
12–19 years							
1 (2007–2009)	945	50.7 (45.3–56.0)	—	<LOD	<LOD	0.029 (0.024–0.035)	0.044[E] (0.015–0.074)
2 (2009–2011)	508	58.8 (50.5–66.7)	—	<LOD	0.0068 (<LOD–0.0083)	0.024[E] (0.011–0.038)	F
5 (2016–2017)	523	30.1 (23.5–37.7)	—	<LOD	<LOD	0.028 (0.024–0.031)	0.049[E] (0.025–0.073)
20–39 years							
1 (2007–2009)	1096	44.7 (36.9–52.8)	—	<LOD	<LOD	0.040[E] (0.025–0.055)	F
2 (2009–2011)	350	61.3 (49.9–71.6)	—	<LOD	0.0085 (<LOD–0.011)	0.050[E] (0.020–0.080)	F
5 (2016–2017)	363	32.8[E] (21.1–47.2)	—	<LOD	<LOD	0.029[E] (0.016–0.043)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1156	39.6 (32.2–47.5)	—	<LOD	<LOD	0.043 (0.032–0.053)	0.081[E] (0.041–0.12)
2 (2009–2011)	355	62.8 (51.5–72.9)	—	<LOD	0.0095 (0.0083–0.011)	0.051[E] (0.017–0.084)	F
5 (2016–2017)	344	40.7 (28.2–54.5)	—	<LOD	<LOD	F	F
60–79 years							
1 (2007–2009)	1018	38.6 (32.7–44.8)	—	<LOD	<LOD	0.038[E] (0.019–0.058)	0.095[E] (0.026–0.16)
2 (2009–2011)	288	48.6 (42.7–54.5)	—	<LOD	<LOD	F	F
5 (2016–2017)	342	29.3 (20.8–39.6)	—	<LOD	<LOD	F	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.6

cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2535	59.6 (52.5–66.3)	0.012 (0.010–0.014)	<LOD	0.0094 (0.0084–0.010)	0.066 (0.045–0.087)	0.15[E] (0.076–0.23)
5 (2016–2017)	2633	79.5 (73.6–84.3)	0.019 (0.016–0.023)	<LOD	0.019 (0.016–0.021)	0.11 (0.083–0.13)	0.18 (0.13–0.22)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1272	61.7 (54.5–68.4)	0.012 (0.010–0.015)	<LOD	0.0096 (0.0068–0.012)	0.070[E] (0.044–0.095)	0.14[E] (0.048–0.23)
5 (2016–2017)	1305	78.7 (71.6–84.4)	0.019 (0.015–0.023)	<LOD	0.018 (0.015–0.020)	0.12 (0.077–0.15)	0.20 (0.13–0.26)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1263	57.5 (48.6–66.0)	0.011 (0.0092–0.013)	<LOD	0.0092 (0.0069–0.011)	F	F
5 (2016–2017)	1328	80.2 (73.3–85.8)	0.020 (0.016–0.024)	<LOD	0.019 (0.016–0.022)	0.10 (0.082–0.12)	0.17 (0.12–0.22)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	65.9 (55.6–74.8)	0.014 (0.010–0.018)	<LOD	F	0.092[E] (0.054–0.13)	F
5 (2016–2017)	544	87.7 (83.9–90.7)	0.030 (0.022–0.041)	<LOD	0.025 (0.017–0.033)	0.23[E] (0.11–0.35)	F
6–11 years							
1 (2007–2009)	974	48.0 (39.8–56.4)	—	<LOD	<LOD	0.045 (0.033–0.056)	0.097 (0.064–0.13)
2 (2009–2011)	513	70.6 (57.8–80.8)	0.015 (0.012–0.020)	<LOD	F	0.098[E] (0.030–0.17)	F
5 (2016–2017)	526	93.3 (91.1–95.1)	0.035 (0.029–0.042)	0.0077 (0.0059–0.0095)	0.026 (0.020–0.032)	0.23[E] (0.091–0.37)	0.39[E] (0.19–0.59)
12–19 years							
1 (2007–2009)	927	56.3 (44.2–67.6)	—	<LOD	0.0072[E] (<LOD–0.011)	0.048 (0.035–0.060)	0.085 (0.069–0.10)
2 (2009–2011)	507	65.2 (55.6–73.7)	0.014 (0.012–0.017)	<LOD	F	0.092 (0.062–0.12)	0.19 (0.14–0.24)
5 (2016–2017)	521	85.4 (75.2–91.8)	0.026 (0.020–0.032)	<LOD	0.024 (0.018–0.030)	0.13 (0.10–0.15)	0.18[E] (0.075–0.29)
20–39 years							
1 (2007–2009)	1055	47.1 (38.1–56.4)	—	<LOD	<LOD	0.037 (0.025–0.050)	0.085[E] (0.051–0.12)
2 (2009–2011)	355	58.8 (47.4–69.3)	0.012 (0.0086–0.015)	<LOD	F	F	F
5 (2016–2017)	363	79.6 (67.5–88.0)	0.019 (0.014–0.025)	<LOD	0.019 (0.014–0.023)	0.10[E] (0.056–0.14)	0.15[E] (0.097–0.21)
40–59 years							
1 (2007–2009)	1109	45.4 (37.4–53.6)	—	<LOD	<LOD	0.045 (0.035–0.055)	0.067 (0.056–0.077)
2 (2009–2011)	352	58.2 (47.8–68.0)	—	<LOD	0.0093 (0.0060–0.013)	F	F
5 (2016–2017)	342	75.4 (59.7–86.4)	0.015 (0.011–0.021)	<LOD	0.015 (0.011–0.019)	0.085[E] (0.036–0.13)	0.14[E] (0.082–0.19)
60–79 years							
1 (2007–2009)	957	46.1 (38.4–54.0)	—	<LOD	<LOD	0.042 (0.033–0.052)	0.071 (0.052–0.091)
2 (2009–2011)	286	54.5 (46.5–62.4)	—	<LOD	0.0089[E] (<LOD–0.013)	F	F
5 (2016–2017)	337	76.4 (66.9–83.8)	0.018 (0.013–0.025)	<LOD	0.018 (0.013–0.023)	F	0.22[E] (0.10–0.35)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.006, 0.006, and 0.0059 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.7

cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2525	59.6 (52.5–66.3)	0.011 (0.0097–0.013)	<LOD	0.0099 (0.0091–0.011)	0.060[E] (0.035–0.085)	0.12[E] (0.069–0.17)
5 (2016–2017)	2603	79.5 (73.6–84.3)	0.019 (0.016–0.022)	<LOD	0.017 (0.014–0.020)	0.097 (0.076–0.12)	0.16 (0.13–0.19)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1268	61.7 (54.5–68.4)	0.010 (0.0085–0.012)	<LOD	0.0098 (0.0082–0.011)	0.059[E] (0.036–0.081)	0.11[E] (0.043–0.18)
5 (2016–2017)	1290	78.7 (71.6–84.4)	0.016 (0.014–0.019)	<LOD	0.015 (0.012–0.018)	0.079 (0.051–0.11)	0.15 (0.098–0.20)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1257	57.5 (48.6–66.0)	0.013 (0.011–0.015)	<LOD	0.010[E] (0.0062–0.014)	0.066[E] (0.027–0.10)	0.14[E] (0.071–0.21)
5 (2016–2017)	1313	80.2 (73.3–85.8)	0.021 (0.018–0.026)	<LOD	0.021 (0.017–0.025)	0.098 (0.083–0.11)	0.16 (0.11–0.22)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	65.9 (55.6–74.8)	0.023 (0.017–0.031)	<LOD	0.020 (<LOD–0.026)	0.13[E] (0.065–0.19)	F
5 (2016–2017)	535	87.7 (83.9–90.7)	0.052[E] (0.035–0.078)	<LOD	0.043[E] (0.026–0.059)	0.26 (0.17–0.36)	F
6–11 years							
1 (2007–2009)	971	48.0 (39.8–56.4)	—	<LOD	<LOD	0.065 (0.047–0.082)	0.13[E] (0.074–0.19)
2 (2009–2011)	511	70.6 (57.8–80.8)	0.018 (0.013–0.024)	<LOD	0.016[E] (<LOD–0.024)	0.11[E] (0.033–0.19)	F
5 (2016–2017)	518	93.3 (91.1–95.1)	0.040 (0.033–0.049)	0.011 (0.0090–0.013)	0.032 (0.027–0.036)	0.21[E] (0.094–0.32)	F
12–19 years							
1 (2007–2009)	925	56.3 (44.2–67.6)	—	<LOD	0.0077 (<LOD–0.0091)	0.039 (0.034–0.044)	0.071 (0.050–0.092)
2 (2009–2011)	505	65.2 (55.6–73.7)	0.011 (0.0090–0.012)	<LOD	0.0099 (<LOD–0.011)	0.063[E] (0.033–0.093)	0.12 (0.090–0.16)
5 (2016–2017)	514	85.4 (75.2–91.8)	0.020 (0.016–0.024)	<LOD	0.017 (0.014–0.021)	0.095 (0.066–0.13)	0.15 (0.11–0.19)
20–39 years							
1 (2007–2009)	1051	47.1 (38.1–56.4)	—	<LOD	<LOD	0.040 (0.028–0.052)	0.083[E] (0.047–0.12)
2 (2009–2011)	353	58.8 (47.4–69.3)	0.0097 (0.0071–0.013)	<LOD	0.0092 (<LOD–0.012)	0.045[E] (0.014–0.076)	F
5 (2016–2017)	359	79.6 (67.5–88.0)	0.017 (0.014–0.021)	<LOD	0.015 (0.012–0.017)	0.073[E] (0.040–0.11)	0.11 (0.080–0.14)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1104	45.4 (37.4–53.6)	—	<LOD	<LOD	0.047 (0.037–0.058)	0.088 (0.059–0.12)
2 (2009–2011)	350	58.2 (47.8–68.0)	—	<LOD	0.0099 (0.0082–0.012)	0.056[E] (0.018–0.095)	0.10[E] (0.039–0.17)
5 (2016–2017)	341	75.4 (59.7–86.4)	0.014 (0.010–0.019)	<LOD	0.015[E] (0.0093–0.021)	0.063 (0.043–0.083)	0.11[E] (0.051–0.16)
60–79 years							
1 (2007–2009)	957	46.1 (38.4–54.0)	—	<LOD	<LOD	0.057 (0.046–0.069)	0.081 (0.061–0.10)
2 (2009–2011)	285	54.5 (46.5–62.4)	—	<LOD	0.0099[E] (<LOD–0.014)	0.073[E] (0.024–0.12)	0.18[E] (0.079–0.27)
5 (2016–2017)	336	76.4 (66.9–83.8)	0.021 (0.016–0.028)	<LOD	0.019[E] (0.010–0.027)	0.11[E] (0.050–0.16)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.8

cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2553	99.4 (98.0–99.8)	0.12 (0.10–0.15)	0.024 (0.021–0.028)	0.093 (0.076–0.11)	0.85[E] (0.47–1.2)	2.2[E] (0.78–3.6)
5 (2016–2017)	2715	100 (99.9–100)	0.18 (0.13–0.24)	0.029[E] (0.016–0.042)	0.15 (0.11–0.19)	1.1[E] (0.43–1.8)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	99.2 (96.8–99.8)	0.10 (0.087–0.13)	0.024 (0.018–0.029)	0.088 (0.068–0.11)	0.55 (0.43–0.68)	1.2[E] (0.39–2.1)
5 (2016–2017)	1355	100 (99.9–100)	0.16 (0.12–0.22)	0.027[E] (0.011–0.042)	0.13 (0.089–0.18)	1.1[E] (0.61–1.5)	2.5[E] (0.96–4.0)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	99.6 (97.9–99.9)	0.15 (0.11–0.20)	0.025 (0.020–0.030)	0.099 (0.077–0.12)	F	F
5 (2016–2017)	1360	99.9 (99.9–100)	0.19[E] (0.13–0.29)	0.034[E] (0.021–0.048)	0.17 (0.12–0.22)	F	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	520	98.2 (93.1–99.6)	0.067 (0.049–0.090)	0.016 (0.011–0.022)	0.065 (0.047–0.082)	F	F
5 (2016–2017)	553	99.9 (98.7–100)	0.10 (0.077–0.14)	0.023[E] (0.014–0.031)	0.084 (0.061–0.11)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1026	97.2 (95.0–98.5)	0.054 (0.043–0.067)	0.014 (0.0099–0.018)	0.049 (0.038–0.060)	0.22 (0.15–0.28)	0.38[E] (0.18–0.57)
2 (2009–2011)	514	99.3 (97.5–99.8)	0.069 (0.059–0.082)	0.018 (0.014–0.022)	0.056 (0.046–0.065)	0.35[E] (0.22–0.48)	F
5 (2016–2017)	536	99.9 (99.2–100)	0.12 (0.10–0.14)	0.030 (0.024–0.036)	0.10 (0.090–0.11)	0.77[E] (0.32–1.2)	1.6[E] (0.55–2.6)
12–19 years							
1 (2007–2009)	970	98.8 (95.5–99.7)	0.090 (0.067–0.12)	0.019 (0.013–0.025)	0.077 (0.055–0.099)	0.52[E] (0.20–0.84)	1.0[E] (0.44–1.6)
2 (2009–2011)	510	99.8 (99.4–99.9)	0.10 (0.083–0.13)	0.026 (0.022–0.030)	0.080 (0.065–0.095)	0.65[E] (0.36–0.94)	1.7[E] (0.90–2.4)
5 (2016–2017)	538	99.8 (99.2–100)	0.15 (0.11–0.20)	0.030[E] (0.018–0.043)	0.11 (0.084–0.14)	0.89[E] (0.55–1.2)	F
20–39 years							
1 (2007–2009)	1151	98.7 (96.0–99.6)	0.086 (0.070–0.11)	0.020 (0.015–0.024)	0.076 (0.057–0.094)	0.45 (0.30–0.60)	0.75[E] (0.35–1.1)
2 (2009–2011)	359	99.2 (93.7–99.9)	0.18[E] (0.12–0.28)	0.027 (0.019–0.035)	0.13 (0.092–0.17)	F	F
5 (2016–2017)	376	100 (99.9–100)	0.21[E] (0.11–0.40)	F	0.18[E] (0.11–0.25)	F	F
40–59 years							
1 (2007–2009)	1208	98.3 (96.4–99.2)	0.092 (0.073–0.12)	0.018 (0.012–0.024)	0.077 (0.054–0.099)	0.60 (0.42–0.78)	1.2 (0.91–1.5)
2 (2009–2011)	359	99.7 (98.7–99.9)	0.12 (0.089–0.16)	0.024 (0.016–0.031)	0.10[E] (0.054–0.15)	0.64[E] (0.32–0.96)	1.6[E] (0.46–2.7)
5 (2016–2017)	360	100	0.19[E] (0.13–0.27)	F	0.16[E] (0.061–0.26)	F	F
60–79 years							
1 (2007–2009)	1076	99.1 (94.0–99.9)	0.083 (0.066–0.10)	0.019 (0.015–0.024)	0.067 (0.050–0.083)	0.42[E] (0.25–0.58)	0.75 (0.47–1.0)
2 (2009–2011)	291	98.9 (90.0–99.9)	0.11[E] (0.072–0.16)	0.021 (0.014–0.029)	0.086 (0.064–0.11)	0.92[E] (0.37–1.5)	F
5 (2016–2017)	352	100	0.17[E] (0.11–0.26)	F	0.15 (0.11–0.20)	1.5[E] (0.69–2.3)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.007, 0.007, and 0.0045 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.9

cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2543	99.4 (98.0–99.8)	0.12 (0.10–0.15)	0.028 (0.025–0.031)	0.087 (0.072–0.10)	0.83[E] (0.42–1.2)	F
5 (2016–2017)	2685	100 (99.9–100)	0.17 (0.13–0.23)	0.036 (0.026–0.045)	0.14 (0.10–0.17)	1.0 (0.71–1.4)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	99.2 (96.8–99.8)	0.088 (0.075–0.10)	0.026 (0.023–0.029)	0.068 (0.053–0.083)	0.41[E] (0.23–0.59)	0.96[E] (0.46–1.5)
5 (2016–2017)	1340	100 (99.9–100)	0.14 (0.10–0.18)	0.029 (0.020–0.038)	0.10 (0.065–0.13)	0.92[E] (0.55–1.3)	1.8[E] (1.1–2.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1270	99.6 (97.9–99.9)	0.17 (0.13–0.22)	0.034 (0.029–0.039)	0.11 (0.077–0.14)	F	F
5 (2016–2017)	1345	99.9 (99.9–100)	0.21 (0.15–0.30)	0.051 (0.039–0.062)	0.17 (0.13–0.21)	F	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	519	98.2 (93.1–99.6)	0.12 (0.085–0.16)	0.031 (0.022–0.040)	0.091 (0.068–0.11)	0.63[E] (0.19–1.1)	F
5 (2016–2017)	544	99.9 (98.7–100)	0.18 (0.15–0.22)	0.048 (0.033–0.062)	0.15 (0.12–0.18)	F	F
6–11 years							
1 (2007–2009)	1023	97.2 (95.0–98.5)	0.083 (0.070–0.098)	0.028 (0.023–0.033)	0.071 (0.063–0.079)	0.30[E] (0.19–0.41)	0.58[E] (0.21–0.95)
2 (2009–2011)	512	99.3 (97.5–99.8)	0.080 (0.069–0.094)	0.026 (0.022–0.031)	0.059 (0.052–0.066)	0.39[E] (0.24–0.54)	0.70[E] (<LOD–1.2)
5 (2016–2017)	528	99.9 (99.2–100)	0.14 (0.12–0.16)	0.045 (0.035–0.056)	0.10 (0.094–0.11)	0.69[E] (0.29–1.1)	1.7[E] (0.82–2.5)
12–19 years							
1 (2007–2009)	968	98.8 (95.5–99.7)	0.079 (0.062–0.10)	0.022 (0.018–0.025)	0.061 (0.042–0.080)	0.43[E] (0.23–0.63)	0.98[E] (0.55–1.4)
2 (2009–2011)	508	99.8 (99.4–99.9)	0.079 (0.063–0.099)	0.024 (0.020–0.027)	0.060 (0.046–0.074)	0.45[E] (0.24–0.66)	0.88[E] (0.54–1.2)
5 (2016–2017)	531	99.8 (99.2–100)	0.11 (0.088–0.14)	0.030 (0.026–0.034)	0.085 (0.062–0.11)	0.60[E] (0.23–0.98)	1.8[E] (0.56–3.1)
20–39 years							
1 (2007–2009)	1147	98.7 (96.0–99.6)	0.096 (0.080–0.12)	0.026 (0.021–0.031)	0.084 (0.069–0.099)	0.38[E] (0.23–0.53)	0.82[E] (0.40–1.2)
2 (2009–2011)	357	99.2 (93.7–99.9)	0.16[E] (0.10–0.23)	0.028 (0.024–0.033)	0.098 (0.070–0.13)	F	F
5 (2016–2017)	372	100 (99.9–100)	0.18[E] (0.10–0.32)	0.035 (0.028–0.043)	0.15[E] (0.073–0.22)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1203	98.3 (96.4–99.2)	0.12 (0.097–0.14)	0.029 (0.023–0.035)	0.097 (0.080–0.11)	0.64 (0.42–0.86)	1.2 (0.97–1.5)
2 (2009–2011)	357	99.7 (98.7–99.9)	0.12 (0.10–0.15)	0.032 (0.023–0.041)	0.084[E] (0.050–0.12)	0.62[E] (0.28–0.96)	1.3[E] (0.51–2.0)
5 (2016–2017)	359	100	0.17 (0.12–0.23)	0.034[E] (0.019–0.049)	0.15 (0.10–0.19)	1.0[E] (0.49–1.5)	F
60–79 years							
1 (2007–2009)	1076	99.1 (94.0–99.9)	0.12 (0.093–0.14)	0.034 (0.031–0.037)	0.093 (0.073–0.11)	0.48[E] (0.18–0.79)	1.2[E] (0.61–1.7)
2 (2009–2011)	290	98.9 (90.0–99.9)	0.12[E] (0.084–0.19)	0.030 (0.019–0.041)	0.095 (0.071–0.12)	F	F
5 (2016–2017)	351	100	0.19 (0.14–0.28)	0.038[E] (<LOD–0.062)	0.15[E] (0.093–0.20)	0.99 (0.67–1.3)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.10

trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2558	99.4 (97.8–99.9)	0.29 (0.23–0.36)	0.051 (0.043–0.059)	0.22 (0.17–0.26)	2.0[E] (0.90–3.2)	6.8[E] (2.1–11)
5 (2016–2017)	2719	99.6 (98.7–99.9)	0.27 (0.20–0.37)	0.038[E] (0.023–0.052)	0.23 (0.18–0.28)	2.2[E] (0.95–3.4)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	99.3 (96.4–99.9)	0.25 (0.20–0.31)	0.048 (0.036–0.060)	0.21 (0.17–0.25)	1.3[E] (0.82–1.8)	F
5 (2016–2017)	1355	99.7 (99.1–99.9)	0.25 (0.19–0.33)	0.036[E] (0.0098–0.062)	0.21 (0.16–0.26)	2.1[E] (1.3–3.0)	4.5[E] (1.6–7.3)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	99.6 (97.7–99.9)	0.34 (0.25–0.46)	0.052 (0.040–0.064)	0.22 (0.16–0.28)	F	F
5 (2016–2017)	1364	99.5 (96.8–99.9)	0.29[E] (0.19–0.43)	0.039 (0.029–0.050)	0.24 (0.17–0.31)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	99.4 (95.1–99.9)	0.22 (0.16–0.31)	0.055 (0.038–0.071)	0.19 (0.13–0.25)	F	F
5 (2016–2017)	553	100 (99.9–100)	0.22 (0.16–0.30)	0.047[E] (0.029–0.064)	0.18 (0.13–0.23)	F	F
6–11 years							
1 (2007–2009)	1027	99.9 (99.6–100)	0.17 (0.15–0.21)	0.041[E] (0.025–0.057)	0.15 (0.12–0.18)	0.82 (0.57–1.1)	1.4 (1.1–1.8)
2 (2009–2011)	516	99.7 (98.1–100)	0.21 (0.18–0.25)	0.048 (0.037–0.059)	0.17 (0.15–0.19)	1.1 (0.80–1.4)	F
5 (2016–2017)	538	99.4 (95.5–99.9)	0.23 (0.20–0.26)	0.053 (0.039–0.066)	0.20 (0.16–0.24)	1.3[E] (0.60–2.0)	2.9[E] (1.2–4.6)
12–19 years							
1 (2007–2009)	978	99.9 (99.5–100)	0.24 (0.18–0.33)	0.048[E] (0.030–0.066)	0.20 (0.16–0.24)	1.5[E] (0.58–2.4)	3.8[E] (2.0–5.6)
2 (2009–2011)	511	100	0.27 (0.21–0.34)	0.057 (0.048–0.067)	0.20 (0.16–0.25)	1.8[E] (1.0–2.5)	4.8[E] (2.1–7.5)
5 (2016–2017)	538	99.4 (98.3–99.8)	0.26 (0.19–0.35)	0.049[E] (0.020–0.078)	0.21 (0.16–0.26)	1.7[E] (0.80–2.6)	4.6[E] (1.5–7.7)
20–39 years							
1 (2007–2009)	1158	99.3 (97.3–99.8)	0.20 (0.16–0.24)	0.042 (0.031–0.053)	0.17 (0.14–0.21)	1.0 (0.71–1.4)	2.0[E] (1.1–2.8)
2 (2009–2011)	359	100	0.41[E] (0.26–0.66)	0.061 (0.040–0.082)	0.28[E] (0.12–0.43)	F	F
5 (2016–2017)	376	99.3 (93.5–99.9)	0.33[E] (0.17–0.64)	0.036[E] (0.012–0.061)	0.24[E] (0.11–0.37)	F	F
40–59 years							
1 (2007–2009)	1216	99.6 (98.8–99.9)	0.21 (0.17–0.26)	0.037 (0.029–0.044)	0.18 (0.13–0.22)	1.6[E] (0.86–2.3)	3.2[E] (1.9–4.5)
2 (2009–2011)	360	98.9 (94.4–99.8)	0.27 (0.20–0.35)	0.041[E] (0.022–0.060)	0.22 (0.15–0.30)	1.8[E] (1.1–2.5)	F
5 (2016–2017)	360	99.9 (99.3–100)	0.26[E] (0.17–0.39)	0.042[E] (0.012–0.072)	0.25[E] (0.14–0.35)	F	F
60–79 years							
1 (2007–2009)	1078	99.6 (98.0–99.9)	0.18 (0.15–0.22)	0.040 (0.032–0.047)	0.15 (0.12–0.18)	1.1 (0.79–1.3)	1.9[E] (1.1–2.6)
2 (2009–2011)	291	99.0 (93.3–99.9)	0.23[E] (0.14–0.39)	0.041[E] (0.026–0.056)	0.17[E] (0.082–0.25)	F	F
5 (2016–2017)	354	99.6 (97.7–99.9)	0.23[E] (0.16–0.34)	0.029[E] (0.011–0.048)	0.18 (0.11–0.24)	2.5[E] (1.3–3.7)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.01, 0.01, and 0.0094 µg/L, respectively.

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 13.2.11

trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2548	99.4 (97.8–99.9)	0.28 (0.23–0.35)	0.062 (0.054–0.070)	0.19 (0.15–0.24)	1.9[E] (0.72–3.1)	F
5 (2016–2017)	2689	99.6 (98.7–99.9)	0.26 (0.20–0.34)	0.050 (0.038–0.062)	0.21 (0.15–0.26)	2.0[E] (1.1–3.0)	F
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1275	99.3 (96.4–99.9)	0.21 (0.18–0.25)	0.055 (0.045–0.064)	0.17 (0.13–0.20)	1.0[E] (0.53–1.5)	F
5 (2016–2017)	1340	99.7 (99.1–99.9)	0.21 (0.17–0.28)	0.045 (0.030–0.060)	0.17[E] (0.11–0.23)	1.5[E] (0.60–2.3)	3.5 (2.3–4.8)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	99.6 (97.7–99.9)	0.38 (0.29–0.51)	0.070 (0.058–0.083)	0.24 (0.18–0.30)	F	F
5 (2016–2017)	1349	99.5 (96.8–99.9)	0.32 (0.22–0.45)	0.058 (0.045–0.070)	0.25 (0.20–0.31)	F	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	520	99.4 (95.1–99.9)	0.39 (0.28–0.54)	0.094 (0.065–0.12)	0.32 (0.22–0.41)	F	F
5 (2016–2017)	544	100 (99.9–100)	0.38 (0.31–0.46)	0.087[E] (0.053–0.12)	0.33 (0.29–0.37)	1.9[E] (<LOD–3.2)	F
6–11 years							
1 (2007–2009)	1024	99.9 (99.6–100)	0.27 (0.24–0.31)	0.086 (0.078–0.095)	0.21 (0.19–0.24)	1.0[E] (0.63–1.4)	2.4[E] (1.3–3.6)
2 (2009–2011)	514	99.7 (98.1–100)	0.24 (0.21–0.29)	0.077 (0.067–0.087)	0.18 (0.16–0.20)	1.1[E] (0.64–1.5)	F
5 (2016–2017)	530	99.4 (95.5–99.9)	0.27 (0.24–0.30)	0.085 (0.071–0.099)	0.20 (0.17–0.23)	1.6[E] (0.68–2.5)	3.4[E] (1.5–5.4)
12–19 years							
1 (2007–2009)	976	99.9 (99.5–100)	0.21 (0.17–0.27)	0.056 (0.047–0.065)	0.15 (0.11–0.20)	1.2[E] (0.62–1.8)	2.4[E] (1.2–3.5)
2 (2009–2011)	509	100	0.21 (0.16–0.26)	0.057 (0.049–0.065)	0.15 (0.11–0.19)	1.2[E] (0.50–1.9)	2.4[E] (1.2–3.5)
5 (2016–2017)	531	99.4 (98.3–99.8)	0.20 (0.16–0.25)	0.047 (0.040–0.054)	0.14 (0.096–0.18)	F	3.5[E] (1.2–5.9)
20–39 years							
1 (2007–2009)	1154	99.3 (97.3–99.8)	0.22 (0.19–0.26)	0.058 (0.050–0.067)	0.17 (0.14–0.21)	1.0 (0.75–1.3)	2.3[E] (1.4–3.3)
2 (2009–2011)	357	100	0.35[E] (0.23–0.53)	0.059 (0.040–0.078)	0.22[E] (0.13–0.30)	F	F
5 (2016–2017)	372	99.3 (93.5–99.9)	0.29[E] (0.16–0.52)	0.048 (0.033–0.063)	0.22[E] (0.11–0.33)	F	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)	1216	99.6 (98.8–99.9)	0.27 (0.23–0.32)	0.062 (0.057–0.068)	0.20 (0.17–0.23)	1.7 (1.1–2.3)	3.6 (2.9–4.4)
2 (2009–2011)	358	98.9 (94.4–99.8)	0.27 (0.23–0.32)	0.065 (0.051–0.079)	0.19[E] (0.12–0.27)	1.6[E] (0.89–2.2)	F
5 (2016–2017)	359	99.9 (99.3–100)	0.24 (0.17–0.32)	0.049[E] (0.030–0.068)	0.23[E] (0.14–0.31)	1.5[E] (<LOD–2.5)	F
60–79 years							
1 (2007–2009)	1078	99.6 (98.0–99.9)	0.25 (0.21–0.31)	0.066 (0.060–0.073)	0.20 (0.16–0.24)	1.3[E] (0.68–1.9)	2.9[E] (1.7–4.2)
2 (2009–2011)	290	99.0 (93.3–99.9)	0.27[E] (0.16–0.44)	0.052 (0.035–0.070)	0.18[E] (0.10–0.27)	F	F
5 (2016–2017)	353	99.6 (97.7–99.9)	0.27 (0.19–0.37)	0.047[E] (0.029–0.065)	0.20 (0.13–0.27)	2.1[E] (1.0–3.2)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

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13.3 ETHYLENE BISDITHIO-CARBAMATES

Ethylene bisdithiocarbamates (EBDCs) are a group of pesticides used primarily as broad-spectrum organometallic fungicides. Three EBDCs were registered for use in Canada during the Canadian Health Measures Survey (CHMS) cycle 5 sampling period (2016–2017), namely mancozeb, metiram, and nabam (Health Canada, 2019). Ethylene thiourea (ETU) (CASRN 96-45-7), also known as 2-imidazolidinethione, is a metabolite, an environmental degradation product and synthesis contaminant of EBDCs. ETU can also be produced commercially and is used primarily in plastic and rubber production (CDC, 2016; Environment and Climate Change Canada and Health Canada, 2017; EPA, 2016; IARC, 2001; NTP, 2016).

EBDCs enter the environment as a result of their use as fungicides. They break down rapidly to ETU and other metabolites. As a result, they are not expected to be present in drinking water (Health Canada, 2018c; Health Canada, 2018d). Conversely, ETU is moderately persistent and more mobile than the parent fungicides; therefore, it may be present in the water column (NTP, 2016). In soil, ETU is highly mobile, but biodegrades rapidly, while in air it is photochemically degraded. ETU may also be released to the environment during plastic and rubber production. While the curing of rubber converts ETU to other compounds, residual amounts of ETU may be present (IARC, 1974). Therefore, ETU can potentially migrate from rubber surfaces.

EBDCs are used in a range of applications. Nabam is a broad-spectrum biocide registered for use in Canada to control slime-forming microorganisms in process fluids for a number of industries. As a slime-control agent, it is used in air washers, cooling towers, evaporative condensers, pulp and paper mills, drilling fluids for oil field operations, and secondary and tertiary petroleum recovery (Health Canada, 2012). Mancozeb and metiram are protectant contact fungicides with a multi-site mode of action and have historically been used to control a broad spectrum of plant diseases in a variety of food and feed crops. Following a 2018 re-evaluation decision, Health Canada cancelled all uses of metiram with the exception of foliar application in potatoes (Health Canada, 2018d). Health Canada has also recently proposed to phase out all uses of mancozeb, with the exception of use on greenhouse tobacco (Health Canada, 2018c).

Exposure of the general population may occur from the ingestion of food treated with EBDCs. Other routes of exposure include inhalation during activity in areas adjacent to fields treated with EBDCs. ETU exposure results from its presence as a contaminant in the applied fungicide, the degradation of the parent fungicide, or as a product of heating food contaminated with an EBDC (IARC, 2001). As a result of EBDC use, ETU may also be present as a contaminant in food or drinking water. Cigarette smoke may also be an important source of EBDCs and ETU exposure owing to the use of fungicides on tobacco crops (Houeto et al., 1995; IARC, 2001). Exposure to EBDCs and ETU can also occur through dermal contact with pesticide products; direct ETU exposure may result from dermal contact with rubber that contains ETU (Environment and Climate Change Canada and Health Canada, 2017; EPA, 2016; Health Canada, 2012; Health Canada, 2018c; Health Canada, 2018d; HSDB, 2010).

EBDCs are primarily absorbed by ingestion, less so by inhalation or dermally, and are metabolized rapidly in the body to produce ETU and other substances (CDC, 2016; Houeto, 1995). ETU itself is readily absorbed following oral exposure and excreted in urine as unchanged ETU and other oxidative metabolites (CDC, 2016; Houeto et al., 1995). Once absorbed, ETU distributes throughout the body, with predominant distribution to the thyroid gland (IARC, 2001). ETU can cross the placental barrier and has been measured in the milk of lactating laboratory animals (CDC, 2016; HSDB, 2010). ETU elimination following EBDC exposure has a reported half-life in humans ranging from 32 to 100 hours (Kurttio and Savolainen, 1990). Animal and human studies report that ETU is rapidly eliminated, mainly through urine, with a small amount excreted in feces (CDC, 2016, Houeto et al., 1995). ETU measured in urine reflects recent exposure to EBDCs or ETU (CDC, 2016).

Potential human health risks from exposure to EBDCs and their metabolite ETU include key effects on the thyroid. Of the EBDC fungicides, nabam has the greatest toxicity, possibly due to its higher water solubility and absorbability (Frakes and Hicks, 1993). Overall, the toxicity of parent EBDC compounds is relatively low; most is attributed to the metabolites, particularly ETU (Frakes and Hicks, 1993). Acute oral exposure to ETU has been reported to result in thyroid gland hyperplasia and reduced thyroid hormone levels, while short-term inhalation exposure may irritate the respiratory tract (EPA, 2016; Houeto et al., 1995). Acute exposure to higher levels of ETU can result in symptoms ranging from nausea and sweating to pulmonary edema leading to death. Animal studies show that the target organ for chronic ETU toxicity is the thyroid gland; consequently, several symptoms associated with reduced thyroid hormone may be observed, such as myxedema and goiter. Damage to liver, kidneys, or pituitary gland may also occur (HSDB, 2010).

Experimental animal studies have also shown that ETU is a potential endocrine disruptor, as it interferes with the synthesis of thyroid hormones; and that it is teratogenic, with effects such as musculoskeletal and central nervous system abnormalities reported in laboratory animals (CDC, 2016; EPA, 1991; Hurley, 1998). Evidence suggests that ETU may be weakly genotoxic. Exposure in laboratory animals has resulted in liver tumours and benign pituitary tumours by a less understood mode of action (Health Canada, 2018c; IARC, 2001; NTP, 2016). Studies suggest that ETU is carcinogenic, as exposed animals have been shown to develop thyroid tumours with a clear non-genotoxic mode of action. The International Agency for Research on Cancer (IARC) has classified ETU as not classifiable as to its carcinogenicity to humans (Group 3) (IARC, 2001). To date, IARC has not assessed the EBDC fungicides registered for use in Canada for their carcinogenic potential.

The sale and use of EBDC fungicides are regulated in Canada by the Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002). PMRA evaluates toxicity and potential exposure in order to determine whether a pesticide should be registered for a specific use. As part of this registration process, PMRA specifies maximum residue limits (MRLs) of pesticides in food. All established maximum residue limits for EBDCs are currently proposed for revocation, and an MRL on potatoes for metiram has been proposed (Health Canada, 2018b). PMRA re-evaluates registered pesticides on a cyclical basis. As part of this process, Health Canada has completed the re-evaluation of nabam and metiram, and recently published a proposed re-evaluation decision for mancozeb (Health Canada, 2014; Health Canada 2018c; Health Canada, 2018d).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether ETU presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2017). The assessment proposes to conclude that ETU does not meet any of the criteria for being considered toxic under CEPA 1999 (Environment and Climate Change Canada and Health Canada, 2017). Although exposure is not of concern at current levels, according to the draft screening assessment, ETU is associated with human health effects and there may be concern if exposure were to increase; follow-up activities to track changes in exposure or commercial use patterns are under consideration (Canada, 2017). ETU is included in the List of Contaminants and Other Adulterating Substances in Foods found in Division 15 of the Food and Drug Regulations, which prohibits the presence of contaminants in certain food and includes Maximum

Levels (MLs) for other ones (Canada, 1978; Health Canada, 2018a). The list stipulates that no amount of ETU is acceptable in food except for fruits, vegetables, and cereals for which an ML has to be respected (Health Canada, 2018a).

ETU was analyzed in the urine of CHMS participants aged 3–79 years in cycle 5 (2016–2017). Data from this cycle are presented as both µg/L and µg/g creatinine. Finding a measurable amount of ETU in urine is an indicator of direct exposure to ETU or an EBDC fungicide and does not necessarily mean that an adverse health effect will occur.

Table 13.3.1

Ethylene thiourea (ETU) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2704	97.0 (93.5–98.6)	0.42 (0.35–0.51)	0.074[E] (0.043–0.11)	0.44 (0.36–0.51)	2.0 (1.6–2.5)	3.5[E] (2.0–4.9)
Males, 3–79 years							
5 (2016–2017)	1354	96.8 (91.7–98.8)	0.43 (0.33–0.54)	0.074[E] (<LOD–0.12)	0.46 (0.38–0.53)	2.1[E] (1.1–3.0)	3.9[E] (1.9–5.9)
Females, 3–79 years							
5 (2016–2017)	1350	97.2 (93.6–98.8)	0.41 (0.34–0.49)	0.075[E] (0.047–0.10)	0.41 (0.34–0.49)	1.9 (1.4–2.4)	2.7 (1.7–3.6)
3–5 years							
5 (2016–2017)	553	99.0 (87.7–99.9)	0.54 (0.42–0.70)	0.11[E] (0.050–0.18)	0.47[E] (0.29–0.66)	2.7 (2.0–3.4)	3.7 (3.0–4.4)
6–11 years							
5 (2016–2017)	534	96.9 (85.9–99.4)	0.50 (0.40–0.61)	0.094 (0.067–0.12)	0.53 (0.36–0.70)	2.1 (1.7–2.5)	3.7 (2.5–4.9)
12–19 years							
5 (2016–2017)	537	96.8 (88.9–99.1)	0.49 (0.36–0.68)	0.075[E] (0.034–0.12)	0.53 (0.34–0.72)	2.4 (1.5–3.3)	3.7 (2.8–4.7)
20–39 years							
5 (2016–2017)	375	97.9 (89.3–99.6)	0.41 (0.32–0.53)	0.076[E] (0.040–0.11)	0.41 (0.31–0.51)	2.1[E] (0.99–3.1)	F
40–59 years							
5 (2016–2017)	355	95.3 (85.0–98.6)	0.38 (0.29–0.49)	0.072[E] (<LOD–0.11)	0.42 (0.27–0.57)	1.5 (0.95–2.0)	F
60–79 years							
5 (2016–2017)	350	97.9 (93.7–99.3)	0.42 (0.31–0.56)	0.076[E] (<LOD–0.12)	0.43 (0.32–0.55)	2.1[E] (1.2–3.0)	3.8[E] (1.8–5.8)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.033 µg/L.

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 are too unreliable to be published.

Table 13.3.2

Ethylene thiourea (ETU) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2677	97.0 (93.5–98.6)	0.41 (0.34–0.50)	0.075 (0.054–0.096)	0.46 (0.37–0.55)	2.0 (1.5–2.5)	3.4 (2.6–4.1)
Males, 3–79 years							
5 (2016–2017)	1340	96.8 (91.7–98.8)	0.37 (0.29–0.48)	0.073[E] (<LOD–0.10)	0.40 (0.28–0.52)	2.0[E] (1.2–2.7)	3.5 (2.6–4.4)
Females, 3–79 years							
5 (2016–2017)	1337	97.2 (93.6–98.8)	0.45 (0.37–0.54)	0.086[E] (0.050–0.12)	0.50 (0.43–0.57)	2.0 (1.4–2.5)	3.3 (2.4–4.3)
3–5 years							
5 (2016–2017)	543	99.0 (87.7–99.9)	0.91 (0.65–1.3)	0.20[E] (0.11–0.29)	0.81[E] (0.47–1.2)	4.2 (3.4–5.0)	5.7 (4.1–7.3)
6–11 years							
5 (2016–2017)	529	96.9 (85.9–99.4)	0.59 (0.46–0.74)	0.13[E] (0.079–0.19)	0.58 (0.39–0.78)	2.5 (2.0–3.1)	3.3 (2.2–4.3)
12–19 years							
5 (2016–2017)	531	96.8 (88.9–99.1)	0.38 (0.29–0.49)	0.073[E] (0.028–0.12)	0.42 (0.29–0.56)	1.7 (1.4–2.0)	2.1 (1.8–2.3)
20–39 years							
5 (2016–2017)	371	97.9 (89.3–99.6)	0.37 (0.28–0.49)	0.083[E] (0.051–0.12)	0.37[E] (0.23–0.51)	1.8[E] (0.67–3.0)	F
40–59 years							
5 (2016–2017)	354	95.3 (85.0–98.6)	0.34 (0.26–0.46)	0.055[E] (<LOD–0.094)	0.47 (0.31–0.63)	1.3[E] (0.70–1.9)	2.9[E] (1.1–4.7)
60–79 years							
5 (2016–2017)	349	97,9 (93,7–99,3)	0,48 (0,36–0,63)	0,090 (<LD–0,12)	0,51 (0,34–0,68)	2,9[E] (1,4–4,3)	3,8 (2,9–4,6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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13.4 *ortho*-PHENYLPHENOL

ortho-Phenylphenol (OPP) (CASRN 90-43-7), also known as 2-phenylphenol or biphenyl-2-ol, is a synthetic phenol compound that has the appearance of white- to pink-coloured flaky crystals under ambient conditions (ANSES, 2014; IARC, 1999). OPP can be synthesized by a number of chemical processes. For example, it can be produced from cyclohexanone using a catalyst (ANSES, 2014, Dow, 2008). OPP and its salts are used as fungicides, germicides, and bacteriostats for commercial/industrial, agricultural, and residential purposes (Appel, 2000; Brusick, 2005). In Canada, they are registered to control fungal and bacterial growth on pears (Health Canada, 2008b). OPP and its salts are also used domestically and commercially as hard-surface disinfectants, wood preservatives, and material preservatives in items such as ceramic glazes, felt gaskets, paper dyes, laundry starches, concrete additives, adhesives, paints, leathers, textiles, metalworking fluids, fire extinguisher solutions, floor-wax emulsions, chemical toilets, construction materials, and polyvinyl alcohol (Appel, 2000; Brusick, 2005; CDC, 2016; EPA, 2006; Health Canada, 2008b; Health Canada, 2014).

OPP does not occur naturally. It is released into the environment from anthropogenic sources. Entry into the environment may occur when emissions from manufacturing and processing facilities are released into air or water (Dow, 2008; IARC, 1999). OPP may also potentially be released into the environment from the use of commercial or residential products.

Exposure of the general population to OPP can occur through environmental media, such as outdoor and indoor air, and through consumer products, such as disinfectants or items treated with OPP (CDC, 2016). Children may be exposed via ingestion after touching floors or textiles treated with OPP (Health Canada, 2008a). Exposure can also occur through ingestion of treated food (Appel, 2000; CDC, 2016).

While no human studies on absorption via oral exposure or inhalation were identified, occupational studies have shown that OPP is readily absorbed by dermal contact (Bomhard et al., 2002; INRS, 2016; IARC, 1999; European Commission, 2015). Experimental animal studies have demonstrated absorption following oral and inhalation exposures (ANSES, 2014; INRS, 2016). OPP is rapidly distributed throughout the body following absorption (ANSES, 2014; INRS, 2016). Some evidence from human and animal studies suggests that OPP does not accumulate in the body; this is supported by its short elimination half-life (0.8 hours) (ANSES, 2014; Bomhard et al., 2002; CDC, 2016; European Commission, 2002). In vivo and in vitro studies demonstrate that OPP is metabolized extensively by cytochrome p450. The main metabolic pathways are conjugation of OPP with glucuronide or sulphate (European Commission, 2002). The two major metabolites, OPP-glucuronide and OPP-sulfate, are inactive compounds; in vitro studies also demonstrate that multiple metabolic pathways can produce a variety of OPP metabolites, including active compounds such as phenylhydroquinone and phenylbenzoquinone (ANSES, 2014; Brusick, 2005; INRS, 2016). Elimination is rapid and occurs mainly through urine (90%) as well as feces (5%). Experimental animal studies have shown that 99% of OPP is eliminated as metabolites after 48 hours. Urinary levels of OPP reflect recent exposure (CDC, 2016).

Experimental animal studies have reported minimal toxicity following acute oral and inhalation exposure to OPP; however, OPP is a strong skin irritant and moderate eye irritant (Bomhard et al., 2002; CDC, 2016; Stouten, 1998). No human data evaluating chronic toxicity were identified, but animal studies have reported systemic toxicity following chronic oral exposure to OPP, including anemia, weight loss, and increased weight of several organs (ANSES, 2014;

CDC, 2016). Long-term exposure via skin contact showed only local toxicity (skin lesions) but no systemic effects (Stouten, 2018). High doses of OPP in animal studies have been associated with bladder and kidney tumours. This finding is supported by in vitro studies reporting mutagenic effects for OPP (Bomhard et al., 2002; CDC, 2016, IARC 1999). According to the International Agency for Research on Cancer (IARC), OPP is not classifiable as to its carcinogenicity to humans (Group 3) based on inadequate evidence in humans and limited evidence in experimental animals; however, sodium OPP is possibly carcinogenic to humans (Group 2B) based on inadequate evidence in humans and sufficient evidence in experimental animals (IARC, 1999).

The sale and use of OPP as a pesticide is regulated in Canada by Health Canada's Pest Management Regulatory Agency (PMRA) under the *Pest Control Product Act* (Canada, 2006). The PMRA registration process also recognizes the maximum residue limits (MRLs) for OPP in food, established under the *Food and Drugs Act*; the Act prohibits the sale of foods containing pesticides that exceed the established MRLs.

OPP is currently registered in Canada as a post-harvest treatment for pears and as a material preservative in a wide range of products (Health Canada, 2008b). In 2008, the PMRA re-evaluated OPP and determined that these uses do not present unacceptable risks to humans or the environment when used according to product label directions; it granted them continued registration.

Hard-surface disinfectants are regulated as drugs and subject to the requirements of the *Food and Drug Act* and its Regulations; in 2014, Health Canada issued guidelines on labelling and the use of products intended for use as hard-surface disinfectants, including OPP (Health Canada, 2014).

Two metabolites of OPP (*ortho*-phenylphenol-glucuronide and *ortho*-phenylphenol-sulfate) were analyzed in the urine of Canadian Health Measure Survey participants aged 3–79 years in cycle 5 (2016–2017). Data from this cycle are presented as both µg/L and µg/g creatinine. Finding a measurable amount of OPP in urine can be an indicator of exposure to OPP and does not necessarily mean that an adverse health effect will occur.

Table 13.4.1

ortho-Phenylphenol-glucuronide — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2620	12.4 (9.3–16.3)	—	<LOD	<LOD	0.18[E] (<LOD–0.27)	0.38[E] (0.18–0.58)
Males, 3–79 years							
5 (2016–2017)	1303	15.9 (11.1–22.3)	—	<LOD	<LOD	0.25[E] (<LOD–0.36)	0.43[E] (0.18–0.67)
Females, 3–79 years							
5 (2016–2017)	1317	8.8[E] (5.8–13.1)	—	<LOD	<LOD	<LOD	F
3–5 years							
5 (2016–2017)	531	14.3[E] (8.0–24.3)	—	<LOD	<LOD	F	F
6–11 years							
5 (2016–2017)	521	12.3[E] (7.4–19.7)	—	<LOD	<LOD	F	0.44[E] (0.17–0.71)
12–19 years							
5 (2016–2017)	507	12.6 (8.8–17.8)	—	<LOD	<LOD	0.22 (0.15–0.28)	0.37 (0.28–0.46)
20–39 years							
5 (2016–2017)	361	F	—	<LOD	<LOD	F	F
40–59 years							
5 (2016–2017)	351	17.5[E] (9.7–29.6)	—	<LOD	<LOD	0.25[E] (<LOD–0.40)	0.44[E] (<LOD–0.74)
60–79 years							
5 (2016–2017)	349	7.6[E] (4.3–13.0)	—	<LOD	<LOD	<LOD	0.21[E] (<LOD–0.30)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.15 µg/L.

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 are too unreliable to be published.

Table 13.4.2

ortho-Phenylphenol-glucuronide (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2599	12.4 (9.3–16.3)	—	<LOD	<LOD	0.29 (<LOD–0.34)	0.39 (0.31–0.47)
Males, 3–79 years							
5 (2016–2017)	1293	15.9 (11.1–22.3)	—	<LOD	<LOD	0.28 (<LOD–0.34)	0.36 (0.31–0.42)
Females, 3–79 years							
5 (2016–2017)	1306	8.8[E] (5.8–13.1)	—	<LOD	<LOD	<LOD	0.42[E] (<LOD–0.65)
3–5 years							
5 (2016–2017)	524	14.3[E] (8.0–24.3)	—	<LOD	<LOD	0.46 (<LOD–0.58)	F
6–11 years							
5 (2016–2017)	516	12.3[E] (7.4–19.7)	—	<LOD	<LOD	0.27 (<LOD–0.31)	0.43 (0.30–0.56)
12–19 years							
5 (2016–2017)	503	12.6 (8.8–17.8)	—	<LOD	<LOD	0.23 (0.17–0.29)	0.33 (0.26–0.41)
20–39 years							
5 (2016–2017)	358	F	—	<LOD	<LOD	<LOD	F
40–59 years							
5 (2016–2017)	350	17.5[E] (9.7–29.6)	—	<LOD	<LOD	0.30 (<LOD–0.40)	F
60–79 years							
5 (2016–2017)	348	7.6[E] (4.3–13.0)	—	<LOD	<LOD	<LOD	0.35 (<LOD–0.41)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 13.4.3

ortho-Phenylphenol-sulfate — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2694	99.8 (99.1–100)	1.7 (1.5–2.0)	0.42 (0.31–0.53)	1.6 (1.4–1.8)	7.0 (4.8–9.1)	13[E] (7.8–18)
Males, 3–79 years							
5 (2016–2017)	1345	99.7 (98.1–99.9)	2.0 (1.6–2.4)	0.51[E] (0.31–0.71)	1.7 (1.3–2.1)	8.9[E] (5.1–13)	14[E] (8.6–19)
Females, 3–79 years							
5 (2016–2017)	1349	100 (99.8–100)	1.6 (1.3–1.8)	0.39 (0.31–0.47)	1.5 (1.2–1.8)	6.2 (4.3–8.2)	F
3–5 years							
5 (2016–2017)	550	100	1.7 (1.3–2.3)	0.48 (0.30–0.65)	1.5 (1.2–1.8)	F	F
6–11 years							
5 (2016–2017)	535	100	1.7 (1.6–1.9)	0.58 (0.50–0.67)	1.6 (1.3–1.9)	5.5 (4.1–6.8)	9.3[E] (5.3–13)
12–19 years							
5 (2016–2017)	532	99.8 (99.2–100)	1.9 (1.7–2.1)	0.67 (0.52–0.82)	1.7 (1.5–1.9)	5.2 (4.5–6.0)	8.7 (5.7–12)
20–39 years							
5 (2016–2017)	371	100 (99.7–100)	1.7 (1.2–2.4)	F	1.6 (1.1–2.2)	F	F
40–59 years							
5 (2016–2017)	352	99.6 (95.5–100)	2.0 (1.4–2.7)	0.42[E] (0.23–0.61)	1.7 (1.1–2.2)	9.7[E] (5.1–14)	15[E] (7.3–23)
60–79 years							
5 (2016–2017)	354	99.8 (98.4–100)	1.5 (1.3–1.7)	0.36 (0.24–0.47)	1.5 (1.3–1.6)	5.5[E] (3.2–7.8)	8.7 (6.9–10)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.092 µg/L.

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 are too unreliable to be published.

Table 13.4.4

ortho-Phenylphenol-sulfate (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2673	99.8 (99.1–100)	1.7 (1.5–1.9)	0.62 (0.53–0.71)	1.4 (1.2–1.6)	5.4 (4.3–6.5)	8.3[E] (5.0–12)
Males, 3–79 years							
5 (2016–2017)	1335	99.7 (98.1–99.9)	1.7 (1.5–1.9)	0.59 (0.48–0.69)	1.5 (1.3–1.8)	5.4 (4.1–6.7)	7.2[E] (4.1–10)
Females, 3–79 years							
5 (2016–2017)	1338	100 (99.8–100)	1.7 (1.5–1.9)	0.65 (0.53–0.77)	1.4 (1.2–1.5)	5.3 (3.6–6.9)	F
3–5 years							
5 (2016–2017)	543	100	2.9 (2.4–3.6)	1.1 (0.77–1.4)	2.4 (1.9–2.9)	9.5 (7.2–12)	F
6–11 years							
5 (2016–2017)	530	100	2.0 (1.8–2.2)	0.85 (0.76–0.95)	1.8 (1.5–2.0)	5.3 (3.7–6.9)	7.5 (5.2–9.9)
12–19 years							
5 (2016–2017)	528	99.8 (99.2–100)	1.4 (1.2–1.6)	0.56 (0.45–0.68)	1.3 (1.1–1.5)	3.9 (2.8–5.1)	5.8 (4.8–6.7)
20–39 years							
5 (2016–2017)	368	100 (99.7–100)	1.5 (1.2–1.9)	0.63 (<LOD–0.79)	1.3 (1.0–1.6)	F	F
40–59 years							
5 (2016–2017)	351	99.6 (95.5–100)	1.8 (1.4–2.3)	0.60 (0.46–0.75)	1.4 (1.0–1.7)	5.9 (4.0–7.8)	F
60–79 years							
5 (2016–2017)	353	99.8 (98.4–100)	1.7 (1.5–2.0)	0.58 (0.48–0.69)	1.4 (1.2–1.7)	5.8 (4.5–7.1)	8.5[E] (5.2–12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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SUMMARIES AND RESULTS FOR PLASTICIZERS 14

14.1 PHTHALATES

Diesters of phthalic acid, commonly called phthalates, are a class of high-production volume industrial chemicals that are used in the manufacture of a variety of consumer products. Table 14.1.1 lists phthalates commonly found in commerce and their major metabolites measured in cycle 5 of the Canadian Health Measures Survey (CHMS).

Table 14.1.1
Phthalate metabolites measured in the Canadian Health Measures Survey cycle 5 (2016–2017) and their parent phthalate compounds

Phthalate	CASRN	Metabolite	CASRN
Dimethyl phthalate (DMP)	131-11-3	Monomethyl phthalate (MMP)	4376-18-5
Diethyl phthalate (DEP)	84-66-2	Monoethyl phthalate (MEP)	2306-33-4
Di-*n*-butyl phthalate (D*n*BP)	84-74-2	Mono-*n*-butyl phthalate (M*n*BP)	131-70-4
		Mono-3-hydroxy-n-butyl phthalate (3OH-MBP)	—
Diisobutyl phthalate (DiBP)	84-69-5	Monoisobutyl phthalate (MiBP)	30833-53-5
Dicyclohexyl phthalate (DCHP)	84-61-7	Monocyclohexyl phthalate (MCHP)	7517-36-4
Benzyl butyl phthalate (BBP)	85-68-7	Monobenzyl phthalate (MBzP) (some M*n*BP)	2528-16-7
Di-2-ethylhexyl phthalate (DEHP)	117-81-7	Mono[2-(carboxymethyl)hexyl] phthalate (MCMHP)	82975-93-7
		Mono(2-ethylhexyl) phthalate (MEHP)	4376-20-9
		Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP)	40809-41-4
		Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP)	40321-98-0
		Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)	40321-99-1
Di-*n*-octyl phthalate (DOP)	117-84-0	Mono(3-carboxypropyl) phthalate (MCPP)	66851-46-5
		Mono-carboxy-*n*-heptyl phthalate (MCHpP)	—
		Mono-*n*-octyl phthalate (MOP)	5393-19-1

Phthalate	CASRN	Metabolite	CASRN
Diisononyl phthalate (DiNP)	28553-12-0, 68515-48-0	Mono(carboxyisooctyl) phthalate (MCiOP)	898544-09-7
		Monoisononyl phthalate (MiNP)	519056-28-1
		Monooxoisononyl phthalate (MOiNP)	—
		Monohydroxyisononyl phthalate (MHiNP)	—
Di-isodecyl phthalate (DiDP)	26761-40-0	Monocarboxyisononyl phthalate (MCiNP)	—
		Monoisodecyl phthalate (MiDP)	—
		Monooxoisodecyl phthalate (MOiDP)	—
		Monohydroxyisodecyl phthalate (MHiDP)	—

Phthalates are primarily used as plasticizers to impart flexibility and resilience to plastics (Frederiksen et al., 2007; Graham, 1973; Health Canada, 2019). They are also used as solvents in household products, including self-care products, construction and renovation products, and textiles (Health Canada, 2019). In particular, BBP, D*n*BP, DMP, and DEP are found in self-care products, such as cosmetics (e.g., hair-care products and fragrance) and non-prescription drugs (Cosmetic Ingredient Review Expert Panel, 2005; Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada, 2000; Environment Canada and Health Canada, 2015e). DiNP, DiBP, DCHP, D*n*BP, BBP, and DEHP are used in construction and renovation products, such as lubricants and greases, adhesives and sealants, paints and coatings, and building materials (Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada, 2000; Environment Canada and Health Canada, 2015b; Environment Canada and Health Canada, 2015d; Health Canada, 2019). DOP, BBP, DMP, D*n*BP, DiDP, DiBP, and DiNP are used in electrical items and electronics (Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada, 2015b; Environment Canada and Health Canada, 2015c; Environment Canada and Health Canada, 2015d; Environment Canada and Health Canada, 2015e; Health Canada, 2019). Phthalates can also be used in the automotive sector (e.g., BBP, DEHP, DiBP, and DCHP), in printing ink (e.g., DiNP and BBP), and in the formulation of pesticides (e.g., BBP) (Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada, 2015d; Health Canada, 2019). Finally, certain phthalates, including BBP, DEHP, DiDP, and DMP, can be found in food-packaging materials (Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada, 2015c; Environment Canada and Health Canada, 2015e; Health Canada, 2019). Prior to restrictions enacted in 2010 (see below), phthalates, namely BBP, D*n*BP, DiNP, and DEHP, were used in Canada as plasticizers in soft vinyl toys and child-care articles.

There are no known major natural sources of phthalates. Releases to the environment are associated with anthropogenic activities (Environment and Climate Change Canada and Health Canada, 2017). Releases may occur during the manufacture and processing of phthalates, including transportation and storage, as well as during the production, use, and disposal of products that contain phthalates (Environment and Climate Change Canada and Health Canada, 2017). Although release into air may occur, water is expected to be the primary receiving medium for phthalates, and occurs through wastewater effluents from industrial sources and disperse releases from consumer products (Environment and Climate Change Canada and Health Canada, 2017; Environment Canada and Health Canada 2015d).

Phthalates have been detected in food, water, air, soil and dust (Clark, 2003; Environment and Climate Change Canada and Health Canada, 2017). The general population can be exposed to phthalates through the inhalation of indoor air; through the ingestion of water, food, beverages, soil, and dust; and through the use of consumer products (Health Canada, 2019). Other potential sources of exposure are breast milk and the mouthing of children's toys and articles

(Environment and Climate Change Canada and Health Canada, 2017).

In laboratory animals, phthalates have been observed to undergo rapid absorption following oral exposure and generally slow absorption following dermal exposure (ATSDR, 1995; ATSDR, 1997; ATSDR, 2001; ATSDR, 2002). Phthalate diesters are converted to their corresponding monoesters in the gastrointestinal tract or saliva prior to absorption (ATSDR, 1995; ATSDR, 1997; ATSDR, 2001; ATSDR, 2002; NRC, 2008). Phthalates are rapidly metabolized in the body to form hydrolytic and oxidative monoesters, which can either be excreted in the urine and feces unchanged, or can undergo phase II biotransformation to produce glucuronide conjugates with increased water solubility and, therefore, increased urinary excretion (Hauser and Calafat, 2005; Samandar et al., 2009). Although the metabolism and excretion of monoester phthalates varies based on a number of factors, they are generally characterized by rapid metabolism and short biological half-lives (ATSDR, 1995; ATSDR, 1997; ATSDR, 2001; ATSDR, 2002; Hauser and Calafat, 2005). Phthalates do not bioaccumulate in humans (CDC, 2009). Measurement of phthalate metabolites in urine has become the most common approach to assessing phthalate exposure in humans, and reflects relatively recent exposure (Blount et al., 2000; Calafat and McKee, 2006).

In laboratory animals, exposure to some phthalates adversely affects the male reproductive system. In particular, prenatal exposure to phthalates, such as D*n*BP, BBP, DEHP, DCHP, and DiNP, has been shown to disrupt the androgen-mediated development of the male reproductive tract (David, 2006; Environment Canada and Health Canada, 2015b; Environment Canada and Health Canada, 2015d; Foster, 2005; Gray et al., 2000; Howdeshell et al., 2007; Main et al., 2006; Mariana et al., 2016; Wine et al., 1997). This response is termed rat phthalate syndrome and is characterized by malformations of the epididymis, vas deferens, seminal vesicles, prostate and external genitalia, among other effects (Lioy et al., 2015). Adverse effects on the testes have also been observed in mature laboratory animals, although these effects occurred at higher doses (David, 2006; Foster, 2005). There is also evidence from animal studies that phthalates exert adverse effects on the ovaries (Environment Canada and Health Canada, 2015b; Hannon and Flaws, 2015; Mariana et al., 2016). Other target organs identified in animal studies include the liver and kidneys, where phthalate exposure may lead to increased organ weights and peroxisome proliferation in the liver (David and Gans, 2003; Environment Canada and Health Canada, 2015b; Environment Canada and Health Canada, 2015c; Environment Canada and Health Canada, 2015d; Environment Canada and Health Canada, 2015e; Howdeshell et al., 2007; Main et al., 2006; Wine et al., 1997).

Numerous studies demonstrate exposure to phthalates in the human population, including prenatal exposure (Becker et al., 2009; Blount et al., 2000; Health Canada, 2016; Lioy et al., 2015; Marsee et al., 2006; NTP-CERHR, 2003a; NTP-CERHR, 2003b; NTP-CERHR, 2003c; NTP-CERHR, 2003d; NTP-CERHR, 2003e; NTP-CERHR, 2003f; NTP-CERHR, 2006; Praveena et al., 2018; Silva et al., 2003; Wittassek et al., 2011). An evaluation by Health Canada of 95 epidemiologic studies of phthalates and health outcomes — including endocrine system effects, reproductive parameters, and growth and development — determined that no health outcome had sufficient evidence of an association with any assessed phthalate (or its metabolite) based on the evaluation approach (Health Canada, 2016). There was only limited evidence of an association between exposure to DiNP/DEHP and sex hormone levels and semen parameters, and between DEHP exposure and birth measures. For effects detected in animal studies related to male reproductive development, the human data on relevant health outcomes were very limited and the quality of the studies was variable (Health Canada, 2016). The International Agency for Research on Cancer (IARC) has classified DEHP as possibly carcinogenic to humans (Group 2B) and BBP as not classifiable as to its carcinogenicity to humans (Group 3) (IARC, 1999; IARC, 2013).

Environment Canada and Health Canada assessed four phthalates (DBP, BBP, DEHP, and DOP) on an individual basis for the First or Second Priority Substances Lists (PSL1 and PSL2) (Environment Canada and Health Canada 1993, 1994a,b, 2000). DBP and BBP were determined not to present a risk to the environment or to human health. DOP was found not to present a risk to the environment; however, at the time of the assessment, the available information was not sufficient to allow a conclusion in terms of human health. A subsequent report concluded that DOP did not pose a risk to human health (Environment Canada and Health Canada, 2003). DEHP was determined to

present a risk to human health in Canada; however, there was insufficient information to conclude on the potential for risk to the environment. State of the Science reports were published in 2015 for the short-chain phthalate DMP, medium-chain phthalates (including DCHP and DiBP), long-chain phthalates (including DiDP), and DiNP (Environment Canada and Health Canada, 2015b; Environment Canada and Health Canada, 2015c; Environment Canada and Health Canada, 2015d; Environment Canada and Health Canada, 2015e). In 2017, a draft screening assessment was released that proposed to conclude that 13 of the 14 substances in the Phthalate Substance Grouping (including DMP, DiBP, DiNP, and DiDP) are not harmful to the environment or to human health as set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), but that the remaining substance in the Grouping (B79P), along with DEHP, is harmful to the environment as set out in section 64(a) of CEPA 1999 (Environment and Climate Change Canada and Health Canada, 2017). In addition, a cumulative risk assessment using a conservative, lower-tiered hazard index approach indicated no concern for potential cumulative risk of medium-chain phthalates for the general Canadian population, specifically the more sensitive subpopulations (pregnant women and women of childbearing age, infants, and children) at current exposure levels (Environment Canada and Health Canada, 2015a; Environment and Climate Change Canada and Health Canada, 2017).

DEHP is identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018). Health Canada has developed and implemented the Phthalates Regulations concerning the use of six phthalates (DEHP, D*n*BP, BBP, DiNP, DiDP, and DOP) in soft vinyl children's toys and child-care articles (Canada, 2010). Phthalates used in children's toys and child-care articles are also regulated in the United States and the European Union.

In the Maternal–Infant Research on Environmental Chemicals (MIREC) study of approximately 2,000 women from 10 sites across Canada, the following maximum-likelihood estimated geometric mean (GM) concentration (µg/L) of phthalate metabolites from first-trimester urine samples were reported (standardized for specific gravity): M*n*BP, 13.69; MEP, 37.73; MBzP, 6.14; MCPP, 1.02; MEHP, 2.63; MEOHP, 7.54; and MEHHP, 10.81, with non-detects reported for MMP, MCHP, MiNP, and MOP (Arbuckle et al., 2014).

Eleven monoester phthalate metabolites (M*n*BP, MEP, MBzP, MCHP, MEHP, MOP, MiNP, MMP, MCPP, MEHHP, and MEOHP) were measured in the urine of CHMS participants aged 6–49 years in cycle 1 (2007–2009), and aged 3–79 years in cycle 2 (2009–2011) and cycle 5 (2016–2017). MiBP was measured in the urine of CHMS cycle 2 (2009–2011) and cycle 5 (2016–2017) participants aged 3–79 years. MCMHP, MECPP, MCiNP, MHiDP, MiDP, MOiDP, MCiOP, MHiNP, MOiNP, 3OH-MBP, and MCHpP were measured in the urine of cycle 5 (2016–2017) participants aged 3–79 years. Data from these monoester phthalate metabolites are presented as both µg/L and µg/g creatinine (Tables 14.1.2 to 14.1.47). Finding a measurable amount of monoester phthalate metabolites in urine is an indicator of exposure to diester phthalates and does not necessarily mean that an adverse health effect will occur.

Table 14.1.2

Monomethyl phthalate (MMP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2559	27.8 (24.8–31.1)	—	<LOD	<LOD	8.4 (7.9–8.8)	15[E] (6.4–23)
5 (2016–2017)	2677	95.9 (93.0–97.6)	2.0 (1.8–2.3)	0.56 (0.38–0.74)	2.2 (1.8–2.5)	6.8 (5.4–8.1)	9.9 (7.6–12)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1280	35.3 (29.7–41.4)	—	<LOD	<LOD	8.7 (6.0–11)	F
5 (2016–2017)	1335	96.2 (91.1–98.5)	2.1 (1.8–2.5)	0.64[E] (0.32–0.95)	2.2 (1.7–2.7)	7.1 (5.2–8.9)	9.9 (7.5–12)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	20.4 (17.3–23.8)	—	<LOD	<LOD	7.6 (6.2–9.0)	F
5 (2016–2017)	1342	95.6 (92.1–97.6)	1.9 (1.7–2.2)	0.53 (0.34–0.73)	2.1 (1.7–2.5)	6.2 (4.4–8.0)	9.9[E] (5.3–15)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	58.4 (51.9–64.7)	—	<LOD	<LOD (<LOD–5.9)	20[E] (11–28)	F
5 (2016–2017)	549	100 (99.9–100)	3.6 (3.1–4.1)	1.1[E] (0.66–1.6)	3.7 (3.2–4.2)	10 (9.1–11)	13 (9.9–16)
6–11 years							
1 (2007–2009)	1037	34.0 (26.7–42.2)	—	<LOD	<LOD	14[E] (5.2–22)	25[E] (15–34)
2 (2009–2011)	515	64.1 (54.9–72.3)	—	<LOD	<LOD (<LOD–6.5)	23 (17–29)	34[E] (19–49)
5 (2016–2017)	533	99.7 (99.0–99.9)	3.7 (3.3–4.3)	1.1 (0.86–1.4)	3.7 (3.1–4.4)	12 (9.2–15)	16 (13–19)
12–19 years							
1 (2007–2009)	991	32.5 (25.4–40.5)	—	<LOD	<LOD	9.3 (8.7–9.9)	11 (6.8–14)
2 (2009–2011)	512	46.0 (38.4–53.8)	—	<LOD	<LOD	8.6 (7.6–9.7)	17[E] (9.1–25)
5 (2016–2017)	533	99.3 (97.4–99.8)	3.1 (2.8–3.6)	0.85 (0.61–1.1)	3.1 (2.8–3.4)	9.0 (7.1–11)	13[E] (4.4–22)
20–39 years							
1 (2007–2009)	730	17.0[E] (10.8–25.7)	—	<LOD	<LOD	6.9[E] (<LOD–9.4)	9.4 (8.1–11)
2 (2009–2011)	359	26.4 (20.3–33.4)	—	<LOD	<LOD	7.7 (6.2–9.1)	F
5 (2016–2017)	370	97.1 (93.7–98.7)	2.1 (1.7–2.7)	F	2.3 (1.7–2.9)	6.5[E] (4.1–9.0)	9.9[E] (6.1–14)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	17.7 (12.4–24.6)	—	<LOD	<LOD	8.1[E] (<LOD–13)	F
5 (2016–2017)	350	94.2 (85.9–97.7)	1.6 (1.3–2.1)	0.45[E] (<LOD–0.78)	1.7 (1.3–2.2)	4.8 (3.3–6.3)	7.1[E] (2.8–11)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	17.8 (13.6–23.0)	—	<LOD	<LOD	6.8 (5.3–8.3)	8.5 (7.5–9.4)
5 (2016–2017)	342	93.1 (89.4–95.6)	1.5 (1.4–1.7)	0.50[E] (0.31–0.69)	1.5 (1.2–1.7)	4.9 (3.6–6.1)	7.8[E] (4.1–12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 5, 5, and 0.21 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.3

Monomethyl phthalate (MMP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2549	27.8 (24.8–31.1)	—	<LOD	<LOD	10 (9.6–10)	19 (17–22)
5 (2016–2017)	2645	95.9 (93.0–97.6)	2.0 (1.8–2.1)	0.66 (0.54–0.78)	1.9 (1.7–2.0)	6.2 (5.5–7.0)	9.1 (7.6–11)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	35.3 (29.7–41.4)	—	<LOD	<LOD	9.7 (7.6–12)	19[E] (<LOD–28)
5 (2016–2017)	1320	96.2 (91.1–98.5)	1.8 (1.6–2.2)	0.65[E] (0.37–0.94)	1.7 (1.5–1.9)	5.6 (4.0–7.1)	9.5[E] (5.9–13)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	20.4 (17.3–23.8)	—	<LOD	<LOD	10 (8.4–12)	19 (<LOD–22)
5 (2016–2017)	1325	95.6 (92.1–97.6)	2.1 (1.9–2.3)	0.68[E] (0.43–0.93)	2.0 (1.8–2.2)	6.8 (5.8–7.9)	8.8 (7.4–10)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	58.4 (51.9–64.7)	—	<LOD	<LOD	30 (23–37)	49[E] (21–77)
5 (2016–2017)	538	100 (99.9–100)	6.2 (5.8–6.6)	2.5 (2.1–3.0)	6.1 (5.6–6.5)	14 (11–17)	22[E] (14–30)
6–11 years							
1 (2007–2009)	1034	34.0 (26.7–42.2)	—	<LOD	<LOD	23 (16–30)	38 (25–50)
2 (2009–2011)	513	64.1 (54.9–72.3)	—	<LOD	<LOD	26[E] (16–36)	32[E] (13–51)
5 (2016–2017)	525	99.7 (99.0–99.9)	4.3 (3.9–4.8)	1.9 (1.6–2.1)	3.8 (3.2–4.4)	11 (8.4–13)	18 (12–24)
12–19 years							
1 (2007–2009)	989	32.5 (25.4–40.5)	—	<LOD	<LOD	9.2 (8.8–9.7)	10[E] (5.7–14)
2 (2009–2011)	510	46.0 (38.4–53.8)	—	<LOD	<LOD	9.0 (7.0–11)	12 (9.0–15)
5 (2016–2017)	526	99.3 (97.4–99.8)	2.4 (2.2–2.6)	1.0 (0.88–1.1)	2.2 (1.9–2.5)	6.1 (4.9–7.4)	8.2[E] (4.1–12)
20–39 years							
1 (2007–2009)	728	17.0[E] (10.8–25.7)	—	<LOD	<LOD	9.1 (<LOD–9.8)	19[E] (8.6–29)
2 (2009–2011)	357	26.4 (20.3–33.4)	—	<LOD	<LOD	7.4[E] (4.4–10)	F
5 (2016–2017)	366	97.1 (93.7–98.7)	1.9 (1.6–2.3)	0.77[E] (<LOD–1.1)	1.8 (1.6–2.1)	5.7 (3.7–7.8)	8.3[E] (4.8–12)
40–59 years							
1 (2007–2009)	—	—	—	—	—	—	—
2 (2009–2011)	358	17.7 (12.4–24.6)	—	<LOD	<LOD	9.6 (<LOD–13)	18[E] (<LOD–31)
5 (2016–2017)	349	94.2 (85.9–97.7)	1.5 (1.2–1.8)	0.46 (<LOD–0.60)	1.5 (1.2–1.8)	3.9 (2.7–5.2)	7.0[E] (4.0–10)
60–79 years							
1 (2007–2009)	—	—	—	—	—	—	—
2 (2009–2011)	289	17.8 (13.6–23.0)	—	<LOD	<LOD	9.6 (6.6–13)	18[E] (8.6–28)
5 (2016–2017)	341	93.1 (89.4–95.6)	1.7 (1.5–1.9)	0.64 (0.49–0.80)	1.7 (1.5–2.0)	5.5 (4.9–6.1)	7.5 (5.8–9.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.4

Monoethyl phthalate (MEP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2561	100	44 (36–54)	7.6 (6.0–9.2)	42 (35–50)	250[E] (150–340)	F
5 (2016–2017)	2712	99.2 (95.9–99.8)	22 (19–25)	3.8 (2.9–4.7)	19 (16–22)	150 (98–210)	280[E] (100–450)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1282	100	45 (35–59)	8.8 (7.3–10)	38 (25–50)	270[E] (130–400)	F
5 (2016–2017)	1350	99.5 (98.6–99.9)	21 (17–26)	3.7[E] (2.1–5.3)	19 (15–22)	110[E] (40–190)	240[E] (100–370)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	100	43 (36–53)	6.9 (5.0–8.7)	45 (35–54)	230[E] (98–350)	F
5 (2016–2017)	1362	98.8 (92.5–99.8)	23 (19–27)	3.9 (3.0–4.9)	19 (15–24)	180[E] (120–250)	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	100	21 (18–24)	6.8 (5.4–8.2)	19 (16–23)	80 (58–100)	120 (92–140)
5 (2016–2017)	553	100	13 (10–17)	3.5[E] (2.1–4.9)	12 (11–13)	50 (37–63)	F
6–11 years							
1 (2007–2009)	1037	100 (96.5–100)	26 (21–32)	6.3 (4.5–8.0)	23 (19–28)	120 (80–160)	200[E] (120–290)
2 (2009–2011)	516	100	29 (23–37)	6.6 (4.4–8.8)	25[E] (14–36)	120[E] (60–180)	240[E] (110–380)
5 (2016–2017)	536	99.5 (98.8–99.8)	18 (14–23)	4.0 (3.2–4.8)	15 (13–18)	110[E] (34–190)	F
12–19 years							
1 (2007–2009)	991	100	65 (55–77)	14 (9.7–18)	60 (47–73)	340 (300–390)	550[E] (320–780)
2 (2009–2011)	512	100	51 (43–61)	10 (7.1–14)	47 (38–57)	230 (150–310)	490[E] (270–710)
5 (2016–2017)	537	99.0 (95.3–99.8)	25 (19–32)	5.4 (4.0–6.8)	21 (15–28)	130[E] (61–200)	F
20–39 years							
1 (2007–2009)	730	100	62 (51–75)	11 (7.3–14)	51 (35–68)	440[E] (130–740)	F
2 (2009–2011)	359	100	48[E] (31–73)	7.6 (4.8–10)	45[E] (25–65)	320[E] (120–520)	F
5 (2016–2017)	374	97.9 (83.7–99.8)	20[E] (13–32)	F	17[E] (11–24)	190[E] (77–300)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	100	44[E] (29–69)	6.9[E] (3.1–11)	43[E] (27–60)	F	F
5 (2016–2017)	359	99.9 (99.4–100)	23[E] (15–33)	3.6[E] (2.1–5.0)	20 (15–26)	F	F
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	291	100	49 (38–62)	9.1 (6.8–11)	44 (33–56)	240[E] (82–390)	F
5 (2016–2017)	353	99.8 (98.7–100)	25 (20–31)	5.6 (3.9–7.3)	20 (14–26)	110[E] (31–180)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.5, 0.3, and 0.98 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.5

Monoethyl phthalate (MEP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2551	100	44 (38–52)	10 (8.5–12)	37 (30–44)	220[E] (120–320)	410[E] (<LOD–620)
5 (2016–2017)	2680	99.2 (95.9–99.8)	21 (19–23)	5.3 (4.6–6.0)	16 (14–18)	110 (88–140)	200 (140–270)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	100	39 (31–49)	9.4 (8.0–11)	30 (23–38)	220[E] (110–330)	390[E] (<LOD–600)
5 (2016–2017)	1335	99.5 (98.6–99.9)	18 (15–21)	4.9 (3.8–6.0)	14 (12–16)	86[E] (45–130)	140[E] (67–220)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	100	50 (41–60)	14 (11–17)	43 (38–49)	F	F
5 (2016–2017)	1345	98.8 (92.5–99.8)	25 (22–29)	5.6 (4.8–6.4)	20 (17–24)	130[E] (79–170)	230[E] (<LOD–320)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	100	36 (32–41)	14 (12–16)	30 (25–35)	110 (82–140)	180 (120–230)
5 (2016–2017)	542	100	23 (19–28)	7.6 (5.7–9.6)	18 (13–22)	72[E] (28–120)	170 (110–230)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1034	100 (96.5–100)	40 (33–48)	14 (12–17)	33 (27–38)	130[E] (80–190)	210[E] (97–320)
2 (2009–2011)	514	100	34 (27–42)	10 (8.2–12)	27 (20–35)	130[E] (60–200)	230[E] (130–330)
5 (2016–2017)	528	99.5 (98.8–99.8)	21 (17–27)	6.9 (6.2–7.6)	15 (12–18)	F	F
12–19 years							
1 (2007–2009)	989	100	55 (49–62)	14 (13–16)	49 (41–57)	250 (200–300)	420 (350–480)
2 (2009–2011)	510	100	39 (34–45)	10 (8.7–12)	33 (26–39)	160[E] (100–220)	300[E] (190–410)
5 (2016–2017)	530	99.0 (95.3–99.8)	19 (15–23)	6.1 (5.2–7.0)	13 (11–16)	85[E] (38–130)	F
20–39 years							
1 (2007–2009)	728	100	65 (55–75)	14 (13–16)	54 (45–64)	340[E] (190–490)	F
2 (2009–2011)	357	100	42 (30–59)	9.9 (7.2–13)	34[E] (16–52)	F	F
5 (2016–2017)	370	97.9 (83.7–99.8)	18 (14–24)	3.8[E] (<LOD–5.4)	15 (11–19)	100[E] (58–140)	150[E] (<LOD–240)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	100	45 (32–64)	9.7[E] (5.4–14)	39 (27–50)	F	F
5 (2016–2017)	358	99.9 (99.4–100)	21 (16–27)	5.4 (4.3–6.4)	15 (10–20)	120[E] (62–190)	200[E] (<LOD–340)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	100	57 (47–70)	14 (9.7–18)	47 (33–62)	F	570[E] (220–910)
5 (2016–2017)	352	99.8 (98.7–100)	28 (24–34)	7.4 (5.4–9.4)	23 (18–27)	130[E] (65–190)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.6

Mono(3-carboxypropyl) phthalate (MCPP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2543	98.1 (95.7–99.2)	1.9 (1.8–2.1)	0.45 (0.36–0.54)	2.0 (1.7–2.2)	7.0 (6.1–7.8)	11 (9.0–12)
5 (2016–2017)	2670	90.5 (86.8–93.3)	0.73 (0.61–0.86)	0.15[E] (<LOD–0.20)	0.75 (0.63–0.88)	2.8 (2.2–3.4)	4.5 (3.4–5.5)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	98.6 (95.2–99.6)	2.1 (1.8–2.5)	0.53[E] (0.33–0.72)	2.0 (1.6–2.4)	7.8 (5.1–10)	12 (9.0–15)
5 (2016–2017)	1331	92.2 (88.4–94.9)	0.72 (0.60–0.85)	0.16 (<LOD–0.20)	0.75 (0.58–0.92)	2.7 (1.8–3.6)	4.1 (2.9–5.4)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1270	97.7 (92.9–99.3)	1.7 (1.5–2.0)	0.39 (0.26–0.53)	1.9 (1.6–2.2)	6.7 (5.2–8.2)	8.6 (6.4–11)
5 (2016–2017)	1339	88.8 (83.2–92.8)	0.74 (0.58–0.95)	<LOD	0.75 (0.58–0.92)	3.2 (2.3–4.2)	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	517	99.8 (99.4–100)	3.2 (2.8–3.7)	0.94 (0.63–1.2)	3.1 (2.6–3.6)	9.3 (6.5–12)	14[E] (8.5–19)
5 (2016–2017)	550	97.2 (91.8–99.1)	1.3[E] (0.92–1.9)	0.37[E] (0.22–0.51)	1.3[E] (0.66–2.0)	4.2[E] (1.2–7.2)	6.4[E] (3.1–9.8)
6–11 years							
1 (2007–2009)	1037	97.3 (94.9–98.6)	2.7 (2.2–3.2)	0.70[E] (0.41–0.99)	3.1 (2.5–3.7)	8.8 (7.5–10)	12 (9.8–15)
2 (2009–2011)	515	99.6 (98.3–99.9)	3.3 (2.8–4.0)	1.0 (0.80–1.2)	3.4 (2.9–3.9)	11[E] (7.0–15)	15 (11–19)
5 (2016–2017)	531	97.4 (94.7–98.8)	1.3 (1.1–1.6)	0.40 (0.26–0.54)	1.3 (1.1–1.6)	4.0 (3.3–4.7)	6.3[E] (3.4–9.2)
12–19 years							
1 (2007–2009)	991	96.2 (94.7–97.2)	2.2 (1.9–2.6)	0.40[E] (<LOD–0.60)	2.6 (2.3–2.9)	8.0 (6.4–9.6)	11[E] (6.5–15)
2 (2009–2011)	509	99.7 (98.8–99.9)	2.6 (2.2–3.1)	0.65[E] (0.33–0.98)	2.5 (2.2–2.8)	9.6 (6.3–13)	16[E] (8.0–24)
5 (2016–2017)	532	92.0 (88.3–94.6)	0.88 (0.73–1.0)	0.17[E] (<LOD–0.26)	0.94 (0.84–1.0)	3.0 (2.4–3.5)	F
20–39 years							
1 (2007–2009)	730	90.5 (86.3–93.5)	1.3 (1.1–1.6)	<LOD	1.5 (1.2–1.8)	5.6 (4.1–7.2)	8.4 (5.8–11)
2 (2009–2011)	359	96.8 (91.4–98.9)	1.9 (1.5–2.5)	0.43[E] (0.19–0.66)	2.0 (1.3–2.7)	7.1 (5.2–9.0)	10 (6.9–13)
5 (2016–2017)	365	92.0 (80.4–97.0)	0.70 (0.50–0.97)	<LOD	0.71 (0.49–0.93)	2.3[E] (0.89–3.8)	4.3[E] (2.4–6.2)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	359	98.2 (95.1–99.4)	1.6 (1.4–1.9)	0.42[E] (0.20–0.64)	1.8 (1.4–2.1)	5.3[E] (2.9–7.7)	8.7[E] (3.4–14)
5 (2016–2017)	348	86.8 (76.3–93.1)	0.66 (0.48–0.90)	<LOD	0.69[E] (0.41–0.97)	2.7 (2.1–3.3)	F
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	284	98.3 (94.4–99.5)	1.5 (1.2–1.7)	0.39[E] (0.24–0.54)	1.5 (1.3–1.7)	5.1[E] (2.7–7.5)	8.7[E] (5.5–12)
5 (2016–2017)	344	89.5 (83.7–93.4)	0.61 (0.50–0.73)	<LOD	0.64 (0.52–0.76)	2.6 (2.0–3.3)	4.4 (2.9–5.9)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.06, and 0.14 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.7

Mono(3-carboxypropyl) phthalate (MCPP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2533	98.1 (95.7–99.2)	1.9 (1.7–2.0)	0.66 (0.58–0.74)	1.7 (1.6–1.9)	5.9 (4.8–7.0)	9.1 (7.3–11)
5 (2016–2017)	2639	90.5 (86.8–93.3)	0.70 (0.62–0.78)	0.22 (<LOD–0.25)	0.62 (0.54–0.70)	2.3 (1.6–3.0)	4.1 (3.1–5.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1269	98.6 (95.2–99.6)	1.8 (1.6–2.0)	0.60 (0.48–0.71)	1.6 (1.3–1.9)	6.4 (4.7–8.2)	9.9 (6.6–13)
5 (2016–2017)	1317	92.2 (88.4–94.9)	0.61 (0.54–0.70)	0.20 (<LOD–0.24)	0.58 (0.51–0.65)	1.9 (1.6–2.3)	3.2[E] (1.9–4.5)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1264	97.7 (92.9–99.3)	1.9 (1.7–2.2)	0.69 (0.57–0.82)	1.9 (1.7–2.2)	5.5 (4.4–6.6)	8.4 (7.0–9.9)
5 (2016–2017)	1322	88.8 (83.2–92.8)	0.79 (0.65–0.96)	<LOD	0.71 (0.56–0.87)	2.8[E] (1.2–4.5)	5.8[E] (3.0–8.7)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	516	99.8 (99.4–100)	5.6 (4.8–6.4)	2.4 (2.0–2.8)	5.5 (4.6–6.4)	13 (10–17)	21[E] (12–30)
5 (2016–2017)	539	97.2 (91.8–99.1)	2.3 (1.8–3.0)	0.94 (0.70–1.2)	2.2 (1.7–2.8)	5.6[E] (3.2–8.0)	8.7[E] (4.0–13)
6–11 years							
1 (2007–2009)	1034	97.3 (94.9–98.6)	4.1 (3.6–4.6)	1.4 (1.1–1.7)	3.9 (3.4–4.3)	12 (8.6–15)	16 (11–20)
2 (2009–2011)	513	99.6 (98.3–99.9)	3.8 (3.4–4.3)	1.5 (1.1–1.8)	3.7 (3.3–4.1)	10[E] (6.3–14)	15 (11–20)
5 (2016–2017)	523	97.4 (94.7–98.8)	1.5 (1.3–1.9)	0.67 (0.57–0.76)	1.4 (1.2–1.7)	3.7 (2.6–4.8)	6.0[E] (3.6–8.4)
12–19 years							
1 (2007–2009)	989	96.2 (94.7–97.2)	1.9 (1.7–2.1)	0.69 (<LOD–0.81)	1.7 (1.6–1.9)	5.5 (4.3–6.7)	8.3 (5.3–11)
2 (2009–2011)	507	99.7 (98.8–99.9)	2.0 (1.8–2.3)	0.79 (0.59–0.99)	1.7 (1.5–1.9)	6.1[E] (3.5–8.7)	11[E] (4.0–18)
5 (2016–2017)	526	92.0 (88.3–94.6)	0.66 (0.54–0.82)	0.22 (<LOD–0.28)	0.58 (0.41–0.76)	1.8 (1.3–2.3)	F
20–39 years							
1 (2007–2009)	728	90.5 (86.3–93.5)	1.4 (1.2–1.6)	<LOD	1.3 (1.1–1.5)	4.2 (2.9–5.4)	5.5[E] (3.5–7.5)
2 (2009–2011)	357	96.8 (91.4–98.9)	1.6 (1.4–1.9)	0.58 (0.43–0.73)	1.4 (1.1–1.8)	5.3[E] (2.9–7.7)	7.8[E] (3.4–12)
5 (2016–2017)	361	92.0 (80.4–97.0)	0.63 (0.54–0.73)	<LOD	0.62 (0.44–0.79)	1.8[E] (1.1–2.4)	2.5[E] (0.89–4.1)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	357	98.2 (95.1–99.4)	1.6 (1.5–1.8)	0.60 (0.47–0.73)	1.7 (1.4–2.0)	4.4 (3.8–5.0)	7.0[E] (3.7–10)
5 (2016–2017)	347	86.8 (76.3–93.1)	0.59 (0.47–0.74)	<LOD	0.54 (0.45–0.64)	F	4.1[E] (<LOD–6.0)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	283	98.3 (94.4–99.5)	1.7 (1.5–1.9)	0.68[E] (0.37–0.99)	1.6 (1.3–1.9)	5.0 (3.9–6.2)	6.1[E] (3.2–9.0)
5 (2016–2017)	343	89.5 (83.7–93.4)	0.68 (0.58–0.81)	<LOD	0.58 (0.47–0.70)	2.0 (1.5–2.5)	4.6[E] (1.3–7.9)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.8

Mono-*n*-butyl phthalate (M*n*BP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2555	100	20 (18–22)	5.7 (4.3–7.1)	20 (18–22)	67 (57–77)	87 (74–100)
5 (2016–2017)	2711	99.9 (97.9–100)	12 (11–14)	3.7 (2.7–4.6)	12 (10–14)	38 (31–44)	54 (36–71)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	100	21 (18–24)	6.4 (4.7–8.1)	21 (18–24)	69 (49–90)	96 (70–120)
5 (2016–2017)	1350	99.7 (95.9–100)	12 (10–15)	3.6 (2.3–4.8)	12 (9.1–14)	39 (27–51)	59[E] (26–92)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	100	19 (17–22)	5.2[E] (3.3–7.2)	19 (16–22)	64 (50–78)	86 (70–100)
5 (2016–2017)	1361	100	13 (11–14)	3.7 (2.9–4.6)	13 (11–15)	36 (32–41)	54 (47–60)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	100	32 (28–37)	11 (8.0–14)	30 (26–34)	110 (75–150)	130 (110–150)
5 (2016–2017)	555	100	20 (16–25)	5.9 (4.4–7.4)	19 (14–25)	66[E] (36–95)	85 (67–100)
6–11 years							
1 (2007–2009)	1037	100	33 (29–38)	8.6 (6.4–11)	32 (27–38)	110 (89–130)	160 (130–200)
2 (2009–2011)	515	100	36 (30–43)	9.7 (7.7–12)	32 (26–37)	F	F
5 (2016–2017)	536	100	20 (18–23)	6.9 (5.7–8.1)	19 (16–22)	65 (47–84)	84 (71–98)
12–19 years							
1 (2007–2009)	991	100	32 (29–35)	9.1 (7.4–11)	33 (29–36)	98 (81–120)	140 (130–140)
2 (2009–2011)	512	100	28 (25–33)	9.1 (7.0–11)	28 (23–33)	77 (67–88)	110 (81–130)
5 (2016–2017)	537	100	16 (14–18)	4.6 (3.5–5.7)	18 (16–20)	39 (33–46)	58 (37–79)
20–39 years							
1 (2007–2009)	730	99.9 (99.6–100)	22 (20–25)	6.0 (4.2–7.8)	22 (18–27)	69 (48–90)	100[E] (34–170)
2 (2009–2011)	358	100	20 (16–25)	6.3[E] (3.9–8.7)	21 (16–26)	54 (47–61)	77 (56–99)
5 (2016–2017)	372	99.6 (93.2–100)	12 (9.4–15)	3.3[E] (1.1–5.5)	11 (7.7–14)	44 (29–58)	58[E] (17–99)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	357	100	17 (14–21)	4.0[E] (1.8–6.2)	17 (15–20)	61[E] (31–91)	83 (57–110)
5 (2016–2017)	358	100	11 (8.8–14)	3.3[E] (1.9–4.7)	12 (8.5–15)	30 (23–37)	39[E] (15–64)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	291	100	17 (14–21)	5.3 (3.6–7.0)	16 (12–19)	63 (48–79)	81[E] (34–130)
5 (2016–2017)	353	100	11 (9.8–12)	3.5 (3.0–4.1)	10 (9.5–11)	28 (22–35)	41 (29–54)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.2, and 0.60 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.9

Mono-*n*-butyl phthalate (M*n*BP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2545	100	20 (18–21)	8.7 (7.7–9.8)	17 (15–19)	48 (42–55)	78 (65–91)
5 (2016–2017)	2679	99.9 (97.9–100)	12 (11–13)	5.1 (4.4–5.9)	11 (9.8–12)	31 (26–35)	41 (35–47)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1275	100	18 (16–20)	7.6 (6.9–8.3)	15 (13–17)	46 (38–54)	73 (60–86)
5 (2016–2017)	1335	99.7 (95.9–100)	11 (9.3–12)	4.4 (3.5–5.3)	9.9 (8.9–11)	27 (21–34)	39 (29–49)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1270	100	22 (19–24)	9.9 (8.8–11)	19 (16–22)	52 (41–63)	83 (64–100)
5 (2016–2017)	1344	100	14 (13–15)	6.5 (5.8–7.3)	12 (10–14)	33 (28–39)	45 (40–51)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	100	56 (49–63)	26 (23–30)	52 (45–59)	120 (88–150)	170 (110–220)
5 (2016–2017)	544	100	35 (31–40)	16 (15–17)	33 (27–40)	80 (65–95)	110[E] (58–160)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1034	100	51 (46–56)	22 (19–25)	45 (40–51)	120 (110–140)	210 (140–270)
2 (2009–2011)	513	100	42 (36–48)	16 (14–19)	35 (30–39)	F	F
5 (2016–2017)	528	100	23 (22–25)	11 (9.4–13)	23 (21–25)	50 (41–60)	63 (55–72)
12–19 years							
1 (2007–2009)	989	100	27 (25–30)	12 (11–13)	25 (23–27)	68[E] (42–93)	99 (89–110)
2 (2009–2011)	510	100	22 (19–25)	10 (8.8–11)	19 (16–22)	48 (38–58)	62 (49–75)
5 (2016–2017)	530	100	12 (11–14)	5.6 (4.8–6.4)	11 (8.5–13)	26 (17–35)	33[E] (20–46)
20–39 years							
1 (2007–2009)	728	99.9 (99.6–100)	23 (21–26)	9.9 (8.5–11)	21 (19–22)	56 (41–70)	F
2 (2009–2011)	356	100	17 (15–20)	8.5 (7.2–9.8)	14 (11–17)	36 (29–43)	47[E] (16–77)
5 (2016–2017)	368	99.6 (93.2–100)	11 (9.7–12)	4.7 (3.8–5.5)	10 (9.1–11)	24 (16–32)	32[E] (20–44)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	355	100	17 (15–19)	7.3 (5.9–8.7)	16 (13–18)	36 (26–46)	55[E] (18–92)
5 (2016–2017)	357	100	10 (8.6–12)	4.9 (3.6–6.2)	9.9 (8.6–11)	21 (14–28)	33[E] (18–48)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	100	20 (17–23)	9.3 (7.6–11)	18 (13–22)	51 (33–68)	71 (61–81)
5 (2016–2017)	352	100	12 (11–14)	5.9 (4.8–6.9)	11 (10–13)	27 (21–34)	36 (26–46)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.10

Monoisobutyl phthalate (MiBP) — 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) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2547	99.9 (99.6–100)	14 (12–16)	3.5 (2.7–4.4)	14 (12–16)	43 (33–54)	64 (50–79)
5 (2016–2017)	2715	99.7 (97.4–100)	10 (8.6–12)	2.7 (2.2–3.2)	10 (9.1–11)	35 (25–45)	51 (37–64)
Males, 3–79 years							
2 (2009–2011)	1275	100	14 (12–17)	3.7[E] (2.2–5.1)	15 (12–17)	49 (32–67)	67 (43–90)
5 (2016–2017)	1353	99.5 (92.4–100)	10 (8.6–12)	2.8 (2.0–3.6)	10 (8.8–11)	31 (22–41)	46[E] (29–64)
Females, 3–79 years							
2 (2009–2011)	1272	99.9 (99.3–100)	13 (12–15)	3.5 (2.5–4.5)	13 (11–16)	39 (33–46)	58 (42–73)
5 (2016–2017)	1362	99.9 (99.7–100)	10 (8.2–12)	2.5 (1.9–3.0)	11 (8.6–12)	39 (26–52)	51 (36–66)
3–5 years							
2 (2009–2011)	517	100	22 (19–25)	6.9 (5.0–8.9)	22 (18–26)	63[E] (38–88)	96 (68–120)
5 (2016–2017)	555	100	16 (13–19)	4.2[E] (2.4–6.1)	16 (13–19)	54 (36–72)	77 (49–110)
6–11 years							
2 (2009–2011)	515	100	22 (18–27)	6.6 (5.0–8.3)	22 (18–26)	67[E] (40–93)	120[E] (67–160)
5 (2016–2017)	536	99.9 (99.2–100)	15 (13–17)	5.3 (3.9–6.8)	14 (12–17)	48 (39–57)	74 (50–97)
12–19 years							
2 (2009–2011)	508	100	18 (16–21)	5.6 (4.0–7.2)	18 (16–20)	49 (34–64)	83[E] (38–130)
5 (2016–2017)	538	99.9 (99.5–100)	13 (11–15)	3.7 (2.6–4.8)	13 (11–15)	37 (29–44)	51[E] (32–70)
20–39 years							
2 (2009–2011)	359	99.8 (98.8–100)	15 (13–18)	3.2[E] (1.7–4.7)	18 (15–20)	51 (40–63)	65 (49–81)
5 (2016–2017)	374	99.1 (90.4–99.9)	10 (7.5–14)	F	10 (7.0–14)	41[E] (15–66)	57[E] (28–86)
40–59 years							
2 (2009–2011)	359	100	12 (9.7–15)	3.0[E] (1.4–4.7)	12 (8.3–15)	36[E] (20–51)	47[E] (18–75)
5 (2016–2017)	359	100	9.2 (7.3–12)	2.5 (1.9–3.0)	10 (6.9–14)	31[E] (17–46)	45 (29–62)
60–79 years							
2 (2009–2011)	289	100	9.7 (7.6–12)	2.4[E] (1.4–3.4)	9.3 (7.5–11)	35 (23–47)	42 (29–55)
5 (2016–2017)	353	99.9 (99.1–100)	8.0 (7.3–8.9)	2.4 (2.0–2.8)	7.7 (6.3–9.1)	23 (18–28)	33 (23–44)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 2 and 5 are 0.1 and 0.57 µg/L, respectively.

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 are too unreliable to be published.

Table 14.1.11

Monoisobutyl phthalate (MiBP) (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) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	2537	99.9 (99.6–100)	13 (12–15)	5.4 (4.7–6.1)	13 (12–14)	34 (28–40)	47 (37–58)
5 (2016–2017)	2683	99.7 (97.4–100)	9.8 (8.8–11)	4.0 (3.7–4.3)	8.8 (7.6–9.9)	25 (19–31)	38 (26–50)
Males, 3–79 years							
2 (2009–2011)	1271	100	12 (11–14)	4.9 (3.8–6.0)	11 (9.7–12)	34 (26–42)	46 (34–58)
5 (2016–2017)	1338	99.5 (92.4–100)	8.7 (8.0–9.5)	3.8 (3.3–4.3)	7.5 (7.0–8.1)	23 (19–27)	33 (27–40)
Females, 3–79 years							
2 (2009–2011)	1266	99.9 (99.3–100)	15 (13–17)	6.2 (5.1–7.2)	14 (12–16)	34 (27–42)	49 (33–65)
5 (2016–2017)	1345	99.9 (99.7–100)	11 (9.6–13)	4.5 (3.8–5.1)	10 (9.1–11)	29 (19–40)	49[E] (26–72)
3–5 years							
2 (2009–2011)	516	100	37 (33–42)	16 (13–18)	34 (29–38)	87 (65–110)	120 (86–150)
5 (2016–2017)	544	100	27 (23–32)	11 (8.4–13)	24 (20–28)	79 (62–95)	98[E] (51–150)
6–11 years							
2 (2009–2011)	513	100	26 (22–30)	11 (8.8–13)	23 (19–27)	63 (45–80)	94[E] (40–150)
5 (2016–2017)	528	99.9 (99.2–100)	17 (16–19)	8.1 (7.1–9.1)	15 (13–17)	39 (31–47)	59 (40–77)
12–19 years							
2 (2009–2011)	506	100	14 (12–16)	7.1 (6.1–8.1)	12 (11–14)	30 (23–38)	41[E] (18–64)
5 (2016–2017)	531	99.9 (99.5–100)	9.8 (8.3–11)	4.7 (3.8–5.6)	8.8 (7.2–10)	21 (17–25)	29[E] (18–41)
20–39 years							
2 (2009–2011)	357	99.8 (98.8–100)	13 (12–14)	5.4 (4.3–6.5)	13 (11–14)	33 (23–42)	44[E] (26–62)
5 (2016–2017)	370	99.1 (90.4–99.9)	9.4 (7.7–11)	3.9 (<LOD–4.2)	8.7 (7.4–9.9)	24[E] (8.4–40)	46[E] (14–79)
40–59 years							
2 (2009–2011)	357	100	12 (11–14)	5.2 (4.5–6.0)	12 (9.8–13)	24[E] (15–33)	32 (22–41)
5 (2016–2017)	358	100	8.4 (7.3–9.8)	3.9 (3.3–4.4)	7.6 (6.2–9.0)	21 (17–25)	26 (19–33)
60–79 years							
2 (2009–2011)	288	100	11 (9.0–14)	4.5 (3.0–6.1)	11 (7.8–13)	30 (23–37)	37 (27–46)
5 (2016–2017)	352	99.9 (99.1–100)	9.2 (8.4–10)	3.9 (3.1–4.6)	8.2 (7.5–9.0)	23 (17–30)	39[E] (22–55)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 14.1.12

Mono-3-hydroxy-*n*-butyl phthalate (3OH-MBP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2644	98.9 (97.3–99.5)	1.7 (1.4–1.9)	0.42 (0.32–0.52)	1.7 (1.4–2.0)	5.9 (5.0–6.8)	9.2 (7.0–11)
Males, 3–79 years							
5 (2016–2017)	1309	99.0 (96.9–99.7)	1.6 (1.3–1.9)	0.37[E] (0.23–0.52)	1.6 (1.3–1.9)	6.1 (4.7–7.6)	10[E] (5.8–14)
Females, 3–79 years							
5 (2016–2017)	1335	98.8 (96.0–99.6)	1.7 (1.5–1.9)	0.49 (0.41–0.57)	1.8 (1.4–2.2)	5.5 (4.6–6.5)	8.6 (7.2–10)
3–5 years							
5 (2016–2017)	538	99.8 (97.9–100)	3.8 (3.2–4.5)	1.0 (0.69–1.3)	3.9 (3.1–4.7)	12 (8.5–15)	15 (11–20)
6–11 years							
5 (2016–2017)	525	99.9 (99.2–100)	3.4 (3.0–4.0)	1.1 (0.73–1.5)	3.3 (2.7–3.9)	10 (7.0–13)	15[E] (6.9–22)
12–19 years							
5 (2016–2017)	525	99.7 (98.8–99.9)	2.4 (2.0–2.7)	0.57[E] (0.30–0.84)	2.5 (2.3–2.8)	7.5 (5.9–9.1)	10 (7.2–14)
20–39 years							
5 (2016–2017)	363	97.4 (92.5–99.1)	1.5 (1.2–2.1)	0.37[E] (0.12–0.61)	1.8[E] (0.92–2.7)	6.1 (4.1–8.1)	9.0[E] (3.7–14)
40–59 years							
5 (2016–2017)	351	99.5 (98.1–99.9)	1.4 (1.1–1.8)	0.37 (0.26–0.47)	1.4 (1.1–1.7)	5.1 (3.6–6.6)	6.3[E] (2.9–9.7)
60–79 years							
5 (2016–2017)	342	99.2 (97.9–99.7)	1.3 (1.2–1.5)	0.45 (0.35–0.56)	1.3 (1.1–1.5)	3.5 (2.4–4.6)	5.2[E] (2.8–7.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.079 µg/L.

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 14.1.13

Mono-3-hydroxy-*n*-butyl phthalate (3OH-MBP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2613	98.9 (97.3–99.5)	1.6 (1.5–1.7)	0.60 (0.53–0.66)	1.5 (1.4–1.6)	4.7 (4.2–5.1)	6.8 (6.0–7.5)
Males, 3–79 years							
5 (2016–2017)	1294	99.0 (96.9–99.7)	1.3 (1.2–1.5)	0.54 (0.42–0.66)	1.2 (1.1–1.4)	4.0 (3.0–5.0)	6.5 (4.8–8.1)
Females, 3–79 years							
5 (2016–2017)	1319	98.8 (96.0–99.6)	1.9 (1.7–2.0)	0.72 (0.57–0.88)	1.7 (1.5–1.9)	4.9 (4.4–5.4)	7.1 (6.2–8.0)
3–5 years							
5 (2016–2017)	527	99.8 (97.9–100)	6.5 (5.7–7.4)	2.4 (1.9–3.0)	6.7 (5.9–7.6)	15 (11–19)	25[E] (14–37)
6–11 years							
5 (2016–2017)	517	99.9 (99.2–100)	4.0 (3.5–4.5)	1.5 (1.1–1.8)	3.9 (3.4–4.4)	8.1 (7.1–9.0)	10 (7.2–14)
12–19 years							
5 (2016–2017)	519	99.7 (98.8–99.9)	1.8 (1.5–2.0)	0.77 (0.68–0.87)	1.7 (1.5–1.9)	3.9 (2.9–4.9)	5.0[E] (3.1–6.9)
20–39 years							
5 (2016–2017)	359	97.4 (92.5–99.1)	1.4 (1.2–1.7)	0.54[E] (0.27–0.81)	1.3 (1.1–1.6)	3.5[E] (2.2–4.8)	4.8 (3.7–5.9)
40–59 years							
5 (2016–2017)	350	99.5 (98.1–99.9)	1.3 (1.1–1.5)	0.56 (0.48–0.64)	1.2 (1.0–1.4)	2.9 (2.1–3.6)	3.7[E] (2.1–5.4)
60–79 years							
5 (2016–2017)	341	99.2 (97.9–99.7)	1.5 (1.3–1.7)	0.61 (0.48–0.74)	1.5 (1.2–1.7)	3.6 (2.7–4.5)	4.8 (3.8–5.8)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 14.1.14

Monocyclohexyl phthalate (MCHP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2551	26.0 (19.8–33.3)	—	<LOD	<LOD	0.24[E] (0.15–0.32)	0.47[E] (0.28–0.67)
5 (2016–2017)	2706	3.4[E] (1.9–6.0)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	26.6 (19.2–35.5)	—	<LOD	<LOD	0.25[E] (0.15–0.34)	0.57[E] (0.29–0.84)
5 (2016–2017)	1348	3.9[E] (1.9–7.7)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	25.3[E] (17.3–35.5)	—	<LOD	<LOD	0.22[E] (0.11–0.34)	0.44 (0.30–0.59)
5 (2016–2017)	1358	2.9[E] (1.5–5.4)	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	30.9 (23.2–39.8)	—	<LOD	<LOD	0.74[E] (0.21–1.3)	F
5 (2016–2017)	555	4.6[E] (2.8–7.5)	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
1 (2007–2009)	1037	15.1 (11.0–20.5)	—	<LOD	<LOD	0.43[E] (<LOD–0.73)	1.1[E] (0.48–1.7)
2 (2009–2011)	516	33.8 (25.6–43)	—	<LOD	<LOD	0.49[E] (0.28–0.71)	1.3[E] (0.46–2.0)
5 (2016–2017)	535	F	—	<LOD	<LOD	<LOD	0.33[E] (<LOD–0.46)
12–19 years							
1 (2007–2009)	991	13.3 (9.5–18.3)	—	<LOD	<LOD	0.28[E] (<LOD–0.43)	1.1[E] (0.58–1.6)
2 (2009–2011)	507	25.5 (19.4–32.8)	—	<LOD	<LOD	0.30 (0.20–0.40)	F
5 (2016–2017)	538	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
1 (2007–2009)	730	11.8 (8.2–16.7)	—	<LOD	<LOD	F	0.86[E] (0.45–1.3)
2 (2009–2011)	359	25.4 (17.7–35.0)	—	<LOD	<LOD	0.18[E] (<LOD–0.29)	0.33[E] (0.19–0.47)
5 (2016–2017)	372	F	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	23.0[E] (15.3–33.0)	—	<LOD	<LOD	F	F
5 (2016–2017)	356	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	289	28.6 (20.3–38.5)	—	<LOD	<LOD	0.23 (0.16–0.30)	F
5 (2016–2017)	350	4.0[E] (2.1–7.2)	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.09, and 0.25 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.15

Monocyclohexyl phthalate (MCHP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2541	26.0 (19.8–33.3)	—	<LOD	<LOD	0.29 (0.24–0.35)	0.56[E] (0.33–0.80)
5 (2016–2017)	2674	3.4[E] (1.9–6.0)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1274	26.6 (19.2–35.5)	—	<LOD	<LOD	0.29 (0.20–0.38)	0.59 (0.38–0.79)
5 (2016–2017)	1333	3.9[E] (1.9–7.7)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1267	25.3[E] (17.3–35.5)	—	<LOD	<LOD	0.30 (0.20–0.39)	F
5 (2016–2017)	1341	2.9[E] (1.5–5.4)	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	30.9 (23.2–39.8)	—	<LOD	<LOD	F	F
5 (2016–2017)	544	4.6[E] (2.8–7.5)	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
1 (2007–2009)	1034	15.1 (11.0–20.5)	—	<LOD	<LOD	0.90[E] (<LOD–1.4)	2.0[E] (1.1–2.9)
2 (2009–2011)	514	33.8 (25.6–43)	—	<LOD	<LOD	0.51[E] (0.23–0.79)	F
5 (2016–2017)	527	F	—	<LOD	<LOD	<LOD	0.48 (<LOD–0.61)
12–19 years							
1 (2007–2009)	989	13.3 (9.5–18.3)	—	<LOD	<LOD	0.40[E] (<LOD–0.55)	0.82[E] (0.40–1.2)
2 (2009–2011)	505	25.5 (19.4–32.8)	—	<LOD	<LOD	0.28[E] (0.15–0.40)	F
5 (2016–2017)	531	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
1 (2007–2009)	728	11.8 (8.2–16.7)	—	<LOD	<LOD	0.54 (<LOD–0.72)	0.89 (0.68–1.1)
2 (2009–2011)	357	25.4 (17.7–35.0)	—	<LOD	<LOD	0.20[E] (<LOD–0.31)	F
5 (2016–2017)	368	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	356	23.0[E] (15.3–33.0)	—	<LOD	<LOD	F	F
5 (2016–2017)	355	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	288	28.6 (20.3–38.5)	—	<LOD	<LOD	0.29 (0.21–0.37)	0.42[E] (0.19–0.65)
5 (2016–2017)	349	4.0[E] (2.1–7.2)	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.16

Monobenzyl phthalate (MBzP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2559	100	7.5 (6.6–8.6)	1.7 (1.3–2.2)	7.1 (6.1–8.1)	32 (25–38)	57 (48–65)
5 (2016–2017)	2714	96.3 (92.6–98.2)	3.9 (3.1–4.7)	0.82 (0.61–1.0)	3.6 (3.0–4.3)	21 (17–24)	32 (23–40)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1281	100	8.0 (6.9–9.2)	2.1 (1.5–2.7)	7.5 (6.2–8.9)	33 (26–40)	54 (42–65)
5 (2016–2017)	1353	95.9 (89.6–98.4)	3.8 (2.9–5.0)	0.74 (0.48–1.0)	3.5 (2.6–4.3)	21 (17–26)	29 (23–35)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	100	7.1 (5.7–8.7)	1.5[E] (0.89–2.0)	6.7 (4.8–8.6)	30 (20–40)	58 (41–75)
5 (2016–2017)	1361	96.8 (94.0–98.3)	3.9 (3.2–4.7)	0.91 (0.66–1.2)	3.8 (3.2–4.4)	19 (13–26)	36[E] (22–49)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	100	17 (14–20)	4.1[E] (2.6–5.7)	16 (13–18)	59[E] (25–92)	120 (86–150)
5 (2016–2017)	555	97.0 (90.5–99.1)	7.7[E] (4.3–14)	1.3[E] (0.38–2.2)	7.8[E] (4.9–11)	F	F
6–11 years							
1 (2007–2009)	1037	100	21 (17–25)	4.8 (3.2–6.3)	21 (17–25)	91 (74–110)	120 (98–150)
2 (2009–2011)	516	100	19 (15–23)	4.9 (3.5–6.4)	20 (15–24)	76[E] (45–110)	100 (72–140)
5 (2016–2017)	537	99.4 (98.4–99.8)	10 (8.3–12)	2.2[E] (1.3–3.2)	9.6 (7.0–12)	42 (34–50)	58 (37–79)
12–19 years							
1 (2007–2009)	991	100	19 (16–22)	4.4 (3.1–5.7)	20 (16–24)	74 (56–93)	99 (86–110)
2 (2009–2011)	512	100	12 (10–15)	3.3 (2.2–4.4)	12 (8.9–15)	42 (33–50)	59 (43–75)
5 (2016–2017)	538	99.1 (98.1–99.6)	5.3 (4.2–6.7)	1.0 (0.69–1.3)	5.2[E] (3.3–7.2)	24 (15–32)	41 (28–54)
20–39 years							
1 (2007–2009)	730	100	10 (8.1–13)	2.0 (1.5–2.5)	9.9 (7.0–13)	51 (38–64)	77 (50–100)
2 (2009–2011)	359	100	7.3 (5.5–9.7)	1.8[E] (0.78–2.7)	7.0 (5.2–8.7)	30[E] (12–48)	60 (39–80)
5 (2016–2017)	374	93.8 (80.2–98.2)	3.7[E] (2.3–6.0)	0.83[E] (<LOD–1.4)	4.1[E] (2.5–5.6)	21[E] (8.9–32)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	100	6.0 (4.8–7.5)	1.6[E] (0.95–2.2)	5.6[E] (2.9–8.2)	20[E] (12–28)	F
5 (2016–2017)	358	97.3 (92.6–99.0)	3.3 (2.4–4.6)	0.72[E] (0.41–1.0)	3.1 (2.1–4.1)	18[E] (11–25)	23[E] (9.3–37)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	100	5.2 (4.3–6.4)	1.0[E] (0.55–1.5)	4.7 (3.7–5.8)	23 (16–30)	36[E] (15–57)
5 (2016–2017)	352	96.2 (92.9–98.0)	2.8 (2.4–3.3)	0.65 (0.47–0.82)	2.7 (2.1–3.2)	15[E] (7.3–23)	21 (15–27)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.05, and 0.37 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.17

Monobenzyl phthalate (MBzP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2549	100	7.4 (6.4–8.5)	2.1 (1.8–2.5)	6.8 (5.6–8.0)	28 (22–34)	44 (37–51)
5 (2016–2017)	2682	96.3 (92.6–98.2)	3.7 (3.1–4.4)	0.96 (0.83–1.1)	3.4 (2.7–4.1)	16 (13–19)	25 (18–32)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1277	100	6.8 (6.0–7.7)	2.1 (1.8–2.4)	5.9 (5.0–6.8)	25 (20–30)	39 (27–50)
5 (2016–2017)	1338	95.9 (89.6–98.4)	3.3 (2.6–4.1)	0.86 (0.67–1.1)	2.9 (2.2–3.6)	16 (9.9–21)	22 (16–28)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1272	100	8.1 (6.6–9.9)	2.2 (1.5–3.0)	7.7 (5.7–9.7)	32 (24–41)	46 (40–52)
5 (2016–2017)	1344	96.8 (94.0–98.3)	4.2 (3.6–5.0)	1.1 (0.88–1.3)	3.9 (3.1–4.8)	16 (13–19)	29 (19–39)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	100	29 (24–35)	9.4 (8.1–11)	26 (20–32)	100 (70–130)	150 (110–200)
5 (2016–2017)	544	97.0 (90.5–99.1)	13[E] (8.6–20)	3.2[E] (1.9–4.5)	12[E] (7.0–17)	F	120[E] (<LOD–190)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1034	100	32 (27–39)	10 (8.5–12)	31 (25–37)	100 (86–110)	140 (110–170)
2 (2009–2011)	514	100	22 (18–26)	6.2 (4.3–8.2)	21 (17–25)	73 (58–88)	98 (78–120)
5 (2016–2017)	529	99.4 (98.4–99.8)	11 (9.7–14)	2.9 (2.0–3.8)	10 (7.4–13)	43 (32–53)	58[E] (33–84)
12–19 years							
1 (2007–2009)	989	100	16 (14–19)	5.6 (4.1–7.1)	15 (13–17)	49[E] (30–69)	70 (57–83)
2 (2009–2011)	510	100	9.4 (7.7–11)	3.1 (2.4–3.9)	9.3 (7.7–11)	28 (21–36)	44 (34–54)
5 (2016–2017)	531	99.1 (98.1–99.6)	4.0 (3.0–5.4)	1.0 (0.70–1.3)	3.4[E] (2.0–4.8)	16 (10–21)	22[E] (8.6–35)
20–39 years							
1 (2007–2009)	728	100	11 (8.9–13)	3.0[E] (1.9–4.0)	10 (8.1–12)	36 (27–45)	54 (42–65)
2 (2009–2011)	357	100	6.3 (4.8–8.3)	2.0[E] (1.1–2.9)	5.6 (4.0–7.2)	22[E] (11–34)	36 (26–46)
5 (2016–2017)	370	93.8 (80.2–98.2)	3.4 (2.5–4.6)	0.81[E] (<LOD–1.2)	3.3[E] (2.0–4.5)	12[E] (6.4–18)	21[E] (9.6–33)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	100	6.1 (5.1–7.2)	2.0 (1.7–2.4)	5.4[E] (3.4–7.4)	17 (13–20)	28[E] (16–39)
5 (2016–2017)	357	97.3 (92.6–99.0)	3.0 (2.5–3.6)	0.93 (0.80–1.1)	2.6 (1.9–3.4)	11[E] (6.6–16)	15[E] (9.6–21)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	289	100	6.0 (5.2–7.0)	1.9 (1.6–2.3)	5.8 (4.5–7.1)	24 (17–31)	27[E] (9.5–44)
5 (2016–2017)	351	96.2 (92.9–98.0)	3.2 (2.6–3.9)	0.93 (0.74–1.1)	2.9 (2.4–3.5)	13 (9.0–18)	18 (16–21)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.18

Mono[2-(carboxymethyl)hexyl]phthalate (MCMHP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2705	96.5 (93.9–98.0)	1.8 (1.5–2.1)	0.47 (0.34–0.61)	1.8 (1.5–2.2)	6.1 (4.4–7.7)	8.4 (6.5–10)
Males, 3–79 years							
5 (2016–2017)	1348	97.2 (93.8–98.7)	1.8 (1.5–2.2)	0.50[E] (0.27–0.72)	1.8 (1.5–2.2)	6.3 (4.1–8.6)	8.4 (5.8–11)
Females, 3–79 years							
5 (2016–2017)	1357	95.8 (92.0–97.8)	1.7 (1.4–2.2)	0.46[E] (0.28–0.65)	1.9 (1.5–2.3)	5.9 (4.4–7.3)	8.0 (6.2–9.8)
3–5 years							
5 (2016–2017)	553	99.5 (97.8–99.9)	3.3 (2.5–4.4)	0.97 (0.75–1.2)	3.3[E] (2.1–4.5)	10 (8.4–12)	14 (11–17)
6–11 years							
5 (2016–2017)	537	98.8 (96.1–99.6)	3.1 (2.7–3.6)	0.99 (0.76–1.2)	3.1 (2.5–3.7)	8.8 (6.2–11)	13[E] (5.9–19)
12–19 years							
5 (2016–2017)	537	97.4 (95.4–98.5)	1.9 (1.7–2.2)	0.60 (0.40–0.80)	2.2 (2.0–2.3)	5.1 (4.3–5.9)	7.3 (5.5–9.2)
20–39 years							
5 (2016–2017)	369	95.8 (88.0–98.6)	1.5 (1.1–2.0)	0.29[E] (<LOD–0.49)	1.6 (1.1–2.1)	5.5[E] (3.1–8.0)	8.5[E] (4.7–12)
40–59 years							
5 (2016–2017)	358	96.4 (90.4–98.7)	1.7 (1.3–2.1)	0.50 (0.33–0.66)	1.7 (1.3–2.2)	5.2[E] (2.8–7.5)	7.4 (4.8–10)
60–79 years							
5 (2016–2017)	351	95.8 (92.5–97.7)	1.7 (1.5–2.0)	0.56 (0.42–0.70)	1.8 (1.4–2.1)	5.1 (3.3–6.9)	7.2 (6.1–8.2)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.27 µg/L.

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 14.1.19

Mono[2-(carboxymethyl)hexyl] phthalate (MCMHP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2673	96.5 (93.9–98.0)	1.7 (1.5–1.9)	0.71 (0.61–0.81)	1.6 (1.4–1.8)	4.4 (3.8–5.0)	6.5 (4.8–8.2)
Males, 3–79 years							
5 (2016–2017)	1333	97.2 (93.8–98.7)	1.5 (1.4–1.7)	0.68 (0.59–0.77)	1.4 (1.2–1.5)	3.9 (3.5–4.3)	5.9 (4.5–7.3)
Females, 3–79 years							
5 (2016–2017)	1340	95.8 (92.0–97.8)	1.9 (1.6–2.3)	0.79 (0.56–1.0)	1.8 (1.5–2.2)	4.8 (4.0–5.6)	6.6 (4.4–8.9)
3–5 years							
5 (2016–2017)	542	99.5 (97.8–99.9)	5.8 (4.9–6.9)	2.2[E] (1.3–3.1)	5.6 (4.4–6.9)	13 (12–15)	19 (12–25)
6–11 years							
5 (2016–2017)	529	98.8 (96.1–99.6)	3.6 (3.1–4.2)	1.5 (1.3–1.8)	3.3 (2.9–3.7)	9.4 (6.3–13)	12[E] (7.5–17)
12–19 years							
5 (2016–2017)	530	97.4 (95.4–98.5)	1.5 (1.3–1.7)	0.76 (0.67–0.85)	1.4 (1.2–1.6)	2.9 (2.4–3.4)	3.6[E] (2.0–5.1)
20–39 years							
5 (2016–2017)	365	95.8 (88.0–98.6)	1.4 (1.2–1.5)	0.65 (<LOD–0.74)	1.1 (0.89–1.3)	3.7 (2.7–4.6)	4.3[E] (2.2–6.4)
40–59 years							
5 (2016–2017)	357	96.4 (90.4–98.7)	1.5 (1.3–1.8)	0.59[E] (0.30–0.88)	1.5 (1.2–1.8)	3.8 (2.9–4.7)	4.6 (3.8–5.5)
60–79 years							
5 (2016–2017)	350	95.8 (92.5–97.7)	2.0 (1.7–2.3)	0.85 (0.75–0.95)	1.8 (1.6–2.1)	4.6 (3.4–5.8)	5.6 (3.6–7.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 14.1.20

Mono(2-ethylhexyl) phthalate (MEHP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2498	99.2 (98.0–99.7)	1.9 (1.7–2.1)	0.55 (0.44–0.66)	1.9 (1.6–2.1)	6.5 (5.4–7.6)	9.0 (7.8–10)
5 (2016–2017)	2691	98.5 (96.5–99.4)	1.0 (0.86–1.2)	0.26 (0.20–0.31)	0.96 (0.83–1.1)	3.9 (2.8–4.9)	5.8[E] (3.6–8.0)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1253	99.3 (97.5–99.8)	2.1 (1.8–2.5)	0.58 (0.39–0.76)	2.2 (1.8–2.5)	7.2 (5.2–9.2)	11 (7.6–14)
5 (2016–2017)	1342	98.4 (94.5–99.6)	1.0 (0.83–1.2)	0.27 (0.19–0.36)	0.99 (0.78–1.2)	3.8 (2.7–4.9)	5.4 (4.1–6.6)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1245	99.1 (97.5–99.7)	1.7 (1.5–1.9)	0.55 (0.41–0.69)	1.7 (1.3–2.1)	5.3 (4.2–6.3)	7.9 (6.5–9.3)
5 (2016–2017)	1349	98.6 (96.5–99.4)	1.0 (0.80–1.3)	0.25 (0.17–0.33)	0.90 (0.74–1.1)	4.0[E] (2.2–5.8)	7.4[E] (4.3–11)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	512	99.9 (99.4–100)	2.7 (2.4–3.2)	0.94 (0.77–1.1)	2.7 (2.3–3.1)	7.5 (5.8–9.3)	F
5 (2016–2017)	553	99.8 (98.6–100)	1.5 (1.3–1.8)	0.36[E] (0.21–0.52)	1.4 (1.0–1.7)	5.8 (4.7–7.0)	8.2 (6.9–9.5)
6–11 years							
1 (2007–2009)	1037	100	3.3 (2.9–3.8)	0.86 (0.70–1.0)	3.3 (2.7–3.8)	12 (10–13)	18 (14–21)
2 (2009–2011)	508	100	2.7 (2.3–3.1)	0.85[E] (0.53–1.2)	2.5 (2.1–2.9)	8.1[E] (5.2–11)	11 (8.3–14)
5 (2016–2017)	534	99.9 (98.8–100)	1.4 (1.2–1.6)	0.35 (0.27–0.43)	1.4 (1.1–1.7)	5.1 (3.7–6.4)	5.8 (4.8–6.8)
12–19 years							
1 (2007–2009)	991	99.4 (98.6–99.7)	3.5 (2.8–4.3)	0.79 (0.58–1.0)	3.2 (2.5–3.9)	14 (9.9–19)	23[E] (6.6–40)
2 (2009–2011)	501	99.2 (96.9–99.8)	2.4 (2.0–2.8)	0.64 (0.52–0.76)	2.4 (2.0–2.8)	6.8 (5.0–8.6)	13[E] (7.7–18)
5 (2016–2017)	530	97.8 (93.8–99.2)	1.1 (0.96–1.2)	0.32 (0.24–0.41)	1.0 (0.89–1.2)	3.8 (3.0–4.5)	5.4 (4.6–6.2)
20–39 years							
1 (2007–2009)	730	99.9 (99.3–100)	4.0 (3.5–4.5)	0.95 (0.72–1.2)	3.9 (3.2–4.6)	15 (11–19)	23[E] (12–34)
2 (2009–2011)	349	99.5 (97.2–99.9)	1.9 (1.6–2.2)	0.44[E] (0.19–0.70)	1.9 (1.5–2.4)	7.1 (5.3–8.8)	8.8[E] (4.9–13)
5 (2016–2017)	371	99.6 (98.5–99.9)	1.2 (0.95–1.6)	F	1.0 (0.67–1.4)	5.0[E] (1.3–8.7)	11[E] (5.5–17)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	349	98.9 (96.3–99.7)	1.9 (1.5–2.2)	0.63 (0.55–0.72)	1.9 (1.4–2.3)	5.4 (3.7–7.1)	9.0[E] (4.8–13)
5 (2016–2017)	356	96.9 (89.5–99.1)	0.87 (0.66–1.1)	0.20 (0.13–0.27)	0.90 (0.72–1.1)	2.8[E] (1.0–4.7)	5.2[E] (1.7–8.7)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	279	98.8 (94.7–99.7)	1.3 (1.1–1.5)	0.44 (0.29–0.59)	1.2 (0.96–1.5)	4.7[E] (2.8–6.5)	7.1[E] (4.2–9.9)
5 (2016–2017)	347	98.8 (97.2–99.5)	0.78 (0.71–0.86)	0.25 (0.22–0.29)	0.73 (0.61–0.85)	2.3 (1.6–3.0)	3.7 (2.8–4.6)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.08, and 0.11 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.21

Mono(2-ethylhexyl) phthalate (MEHP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2489	99.2 (98.0–99.7)	1.8 (1.7–2.0)	0.64 (0.52–0.76)	1.7 (1.6–1.9)	5.5 (4.8–6.1)	8.7 (7.3–10)
5 (2016–2017)	2660	98.5 (96.5–99.4)	0.98 (0.86–1.1)	0.29 (0.24–0.34)	0.97 (0.85–1.1)	3.3 (2.6–4.1)	4.6 (3.0–6.2)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1249	99.3 (97.5–99.8)	1.7 (1.5–2.0)	0.58 (0.45–0.70)	1.6 (1.3–1.8)	5.7 (4.7–6.7)	9.3 (6.6–12)
5 (2016–2017)	1328	98.4 (94.5–99.6)	0.87 (0.77–0.99)	0.28 (0.22–0.34)	0.83 (0.62–1.0)	2.8 (2.1–3.5)	3.9 (3.2–4.7)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1240	99.1 (97.5–99.7)	1.9 (1.7–2.2)	0.75 (0.59–0.91)	1.8 (1.6–2.1)	5.2 (4.3–6.0)	7.9 (6.3–9.6)
5 (2016–2017)	1332	98.6 (96.5–99.4)	1.1 (0.91–1.3)	0.30 (0.21–0.39)	1.0 (0.86–1.2)	3.7 (2.6–4.8)	6.0[E] (3.3–8.6)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	511	99.9 (99.4–100)	4.7 (4.1–5.4)	1.8 (1.4–2.2)	4.4 (3.9–4.9)	12 (7.6–16)	19[E] (12–26)
5 (2016–2017)	542	99.8 (98.6–100)	2.7 (2.2–3.2)	1.0 (0.91–1.1)	2.5 (2.0–2.9)	8.3 (7.0–9.6)	10[E] (6.2–14)
6–11 years							
1 (2007–2009)	1034	100	5.1 (4.5–5.7)	1.9 (1.6–2.1)	4.8 (4.3–5.3)	16 (14–18)	22 (18–25)
2 (2009–2011)	506	100	3.1 (2.7–3.5)	1.1 (0.83–1.3)	2.8 (2.4–3.2)	8.8 (7.0–11)	11 (8.5–13)
5 (2016–2017)	526	99.9 (98.8–100)	1.6 (1.4–1.9)	0.59 (0.43–0.75)	1.4 (1.2–1.7)	4.6 (3.6–5.5)	5.7 (4.3–7.0)
12–19 years							
1 (2007–2009)	989	99.4 (98.6–99.7)	3.0 (2.3–3.8)	0.82 (0.63–1.0)	2.8 (2.3–3.2)	10 (6.6–14)	F
2 (2009–2011)	499	99.2 (96.9–99.8)	1.8 (1.6–2.0)	0.64 (0.56–0.72)	1.8 (1.6–2.0)	4.8 (3.4–6.2)	6.4[E] (3.2–9.6)
5 (2016–2017)	524	97.8 (93.8–99.2)	0.82 (0.68–1.0)	0.24 (0.20–0.29)	0.82 (0.64–1.0)	2.2 (1.5–2.9)	3.1 (2.0–4.3)
20–39 years							
1 (2007–2009)	728	99.9 (99.3–100)	4.2 (3.7–4.8)	1.2[E] (0.75–1.7)	3.7 (3.1–4.3)	14 (11–16)	21[E] (13–30)
2 (2009–2011)	347	99.5 (97.2–99.9)	1.6 (1.3–1.9)	0.65[E] (0.41–0.90)	1.4 (1.1–1.7)	4.8 (3.3–6.4)	6.6[E] (3.4–9.9)
5 (2016–2017)	367	99.6 (98.5–99.9)	1.1 (0.88–1.4)	0.29 (<LOD–0.37)	0.96 (0.71–1.2)	F	F
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	348	98.9 (96.3–99.7)	1.8 (1.6–2.1)	0.59[E] (0.35–0.83)	1.8 (1.5–2.1)	4.8 (4.1–5.5)	7.6[E] (4.0–11)
5 (2016–2017)	355	96.9 (89.5–99.1)	0.79 (0.66–0.96)	0.22[E] (0.088–0.35)	0.81 (0.55–1.1)	2.5 (1.7–3.3)	3.5 (2.5–4.4)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	278	98.8 (94.7–99.7)	1.5 (1.3–1.7)	0.50 (0.33–0.68)	1.4 (1.1–1.6)	4.6 (3.3–5.8)	7.2[E] (4.4–10)
5 (2016–2017)	346	98.8 (97.2–99.5)	0.89 (0.80–1.0)	0.30 (0.26–0.34)	0.88 (0.76–1.0)	2.6 (2.0–3.3)	4.2 (2.9–5.5)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.22

Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2705	99.5 (97.0–99.9)	6.1 (5.2–7.3)	1.8 (1.3–2.3)	6.3 (5.1–7.4)	21 (16–26)	30 (23–36)
Males, 3–79 years							
5 (2016–2017)	1346	99.1 (93.6–99.9)	6.0 (4.9–7.3)	1.7[E] (1.0–2.4)	6.2 (5.1–7.3)	21 (15–27)	29[E] (15–44)
Females, 3–79 years							
5 (2016–2017)	1359	99.9 (99.5–100)	6.3 (5.0–8.0)	1.9 (1.3–2.5)	6.4 (4.6–8.2)	21 (16–27)	30 (23–37)
3–5 years							
5 (2016–2017)	555	100	15 (12–19)	4.7[E] (2.9–6.5)	15 (11–19)	49 (40–57)	57 (49–64)
6–11 years							
5 (2016–2017)	535	100	13 (11–15)	4.2 (2.7–5.7)	13 (11–14)	38 (26–49)	52[E] (29–75)
12–19 years							
5 (2016–2017)	536	99.9 (99.5–100)	6.9 (6.1–7.7)	2.0 (1.6–2.3)	7.8 (7.0–8.6)	19 (15–22)	23 (18–29)
20–39 years							
5 (2016–2017)	373	100 (99.9–100)	5.6 (4.1–7.5)	1.5[E] (0.56–2.4)	5.3[E] (3.1–7.5)	22 (15–29)	F
40–59 years							
5 (2016–2017)	355	98.6 (89.5–99.8)	5.3 (4.1–6.8)	1.6 (1.0–2.1)	5.6 (4.4–6.8)	15[E] (7.1–24)	24[E] (14–35)
60–79 years							
5 (2016–2017)	351	99.8 (98.6–100)	5.7 (5.1–6.3)	2.1 (1.8–2.4)	5.4 (4.4–6.5)	15 (12–18)	21 (13–28)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.28 µg/L.

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 are too unreliable to be published.

Table 14.1.23

Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2673	99.5 (97.0–99.9)	5.9 (5.2–6.7)	2.4 (2.0–2.8)	5.4 (4.5–6.3)	17 (13–21)	26 (21–31)
Males, 3–79 years							
5 (2016–2017)	1331	99.1 (93.6–99.9)	5.2 (4.6–5.7)	2.1 (1.9–2.4)	4.6 (4.0–5.3)	14 (11–16)	22 (18–26)
Females, 3–79 years							
5 (2016–2017)	1342	99.9 (99.5–100)	6.8 (5.7–8.3)	2.9 (2.5–3.3)	6.6 (5.3–7.9)	20 (15–26)	28 (21–35)
3–5 years							
5 (2016–2017)	544	100	26 (23–30)	10 (8.5–12)	25 (21–29)	61 (52–71)	76 (56–95)
6–11 years							
5 (2016–2017)	527	100	14 (12–17)	6.2 (4.9–7.6)	14 (12–15)	34[E] (19–49)	54 (36–72)
12–19 years							
5 (2016–2017)	529	99.9 (99.5–100)	5.2 (4.5–6.1)	2.1 (1.6–2.6)	5.4 (4.6–6.2)	11 (9.0–14)	15 (12–18)
20–39 years							
5 (2016–2017)	369	100 (99.9–100)	5.0 (4.2–6.0)	2.2 (1.7–2.7)	3.7 (3.1–4.3)	18[E] (7.6–28)	25[E] (16–35)
40–59 years							
5 (2016–2017)	354	98.6 (89.5–99.8)	4.8 (4.0–5.7)	2.1 (1.5–2.7)	4.8 (4.0–5.5)	10 (9.1–11)	11 (8.1–14)
60–79 years							
5 (2016–2017)	350	99.8 (98.6–100)	6.5 (5.8–7.4)	2.7 (2.2–3.2)	6.5 (5.3–7.7)	13 (10–16)	18 (13–23)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 14.1.24

Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2561	100	7.4 (6.9–8.0)	2.3 (2.1–2.5)	7.4 (6.7–8.1)	23 (20–26)	34 (30–39)
5 (2016–2017)	2716	99.3 (97.1–99.8)	3.5 (3.0–4.0)	0.99 (0.83–1.2)	3.5 (2.9–4.1)	12 (9.3–14)	17 (14–21)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1282	100	7.9 (6.8–9.1)	2.4 (2.0–2.8)	7.8 (7.0–8.7)	25 (20–31)	37[E] (22–52)
5 (2016–2017)	1353	99.2 (93.7–99.9)	3.4 (2.8–4.1)	0.98 (0.70–1.3)	3.4 (2.8–4.1)	12 (8.5–16)	17[E] (8.6–26)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	100	7.0 (6.5–7.6)	2.3 (2.0–2.6)	6.7 (5.7–7.8)	22 (18–25)	29 (23–36)
5 (2016–2017)	1363	99.5 (95.1–99.9)	3.5 (2.9–4.3)	1.0 (0.86–1.1)	3.7 (2.8–4.5)	11 (8.5–14)	17 (13–22)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	100	17 (15–19)	6.0 (4.7–7.2)	17 (15–20)	46 (35–57)	67[E] (38–95)
5 (2016–2017)	555	100	8.5 (6.9–10)	2.6 (1.8–3.5)	8.7 (6.6–11)	27 (23–30)	34 (26–42)
6–11 years							
1 (2007–2009)	1037	100	20 (18–22)	5.5 (4.7–6.4)	20 (17–22)	69 (59–78)	100 (84–120)
2 (2009–2011)	516	100	15 (13–18)	4.7 (3.4–6.1)	16 (12–20)	44 (28–60)	57 (50–65)
5 (2016–2017)	537	100	7.0 (6.0–8.2)	2.1 (1.5–2.8)	7.5 (6.2–8.8)	20[E] (12–28)	31[E] (19–43)
12–19 years							
1 (2007–2009)	991	100	18 (15–21)	4.5 (3.6–5.5)	17 (15–20)	61 (51–72)	99[E] (43–150)
2 (2009–2011)	512	100	10 (8.6–12)	3.2[E] (1.7–4.7)	9.9 (8.6–11)	30 (22–38)	44 (30–59)
5 (2016–2017)	538	99.9 (99.5–100)	4.0 (3.6–4.6)	1.2[E] (0.73–1.6)	4.5 (4.1–4.9)	11 (9.3–13)	14 (9.6–19)
20–39 years							
1 (2007–2009)	730	100	13 (11–15)	3.5 (2.6–4.4)	13 (10–15)	48 (34–62)	75[E] (32–120)
2 (2009–2011)	359	100	6.6 (5.6–7.8)	2.4 (1.9–2.9)	6.7 (5.4–8.0)	19 (16–21)	24[E] (14–35)
5 (2016–2017)	374	99.1 (91.7–99.9)	3.1 (2.4–4.1)	0.86[E] (0.33–1.4)	3.0[E] (1.9–4.2)	12 (8.2–15)	17[E] (9.7–24)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	100	6.6 (5.5–7.8)	2.3 (1.7–2.8)	6.2 (5.1–7.2)	19 (15–23)	26[E] (16–36)
5 (2016–2017)	359	98.6 (89.7–99.8)	3.0 (2.3–3.8)	0.98 (0.62–1.3)	2.9 (2.2–3.5)	8.4[E] (3.3–13)	13[E] (7.1–20)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	291	100	6.0 (5.1–7.0)	2.0 (1.5–2.5)	6.0 (4.7–7.4)	17 (12–22)	F
5 (2016–2017)	353	100	3.2 (2.8–3.6)	0.99 (0.83–1.2)	3.2 (2.8–3.6)	8.7 (7.7–9.8)	14[E] (7.7–19)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.2, 0.1, and 0.17 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.25

Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2551	100	7.3 (6.9–7.7)	2.8 (2.5–3.1)	6.9 (6.3–7.5)	19 (17–21)	31 (27–34)
5 (2016–2017)	2684	99.3 (97.1–99.8)	3.4 (3.0–3.7)	1.3 (1.1–1.5)	3.1 (2.7–3.6)	9.5 (8.3–11)	15 (12–19)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	100	6.7 (6.0–7.4)	2.6 (2.1–3.1)	6.1 (5.3–7.0)	20 (14–26)	32 (26–37)
5 (2016–2017)	1338	99.2 (93.7–99.9)	3.0 (2.7–3.3)	1.1 (1.0–1.3)	2.5 (2.2–2.8)	8.1 (6.9–9.4)	13 (9.7–16)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	100	8.0 (7.3–8.8)	3.2 (2.5–3.8)	7.8 (6.8–8.7)	19 (17–21)	26 (20–32)
5 (2016–2017)	1346	99.5 (95.1–99.9)	3.8 (3.3–4.5)	1.4 (1.3–1.6)	3.7 (2.9–4.5)	10 (7.0–13)	17 (12–22)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	100	30 (27–33)	13 (11–15)	28 (25–31)	71 (58–85)	90 (58–120)
5 (2016–2017)	544	100	15 (13–17)	6.2 (4.4–8.0)	14 (12–15)	35 (31–39)	43 (28–58)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1034	100	30 (27–34)	12 (9.7–14)	28 (24–31)	84 (74–94)	120 (95–140)
2 (2009–2011)	514	100	17 (16–19)	7.7 (6.5–8.8)	17 (15–18)	45 (35–56)	52 (40–65)
5 (2016–2017)	529	100	8.2 (7.0–9.6)	3.4 (2.8–4.0)	8.0 (7.1–9.0)	19 (12–26)	31 (20–41)
12–19 years							
1 (2007–2009)	989	100	15 (13–18)	5.5 (4.5–6.4)	13 (11–15)	45 (31–60)	F
2 (2009–2011)	510	100	7.8 (6.9–8.9)	3.2 (2.8–3.7)	7.6 (6.5–8.8)	17 (13–21)	25[E] (14–35)
5 (2016–2017)	531	99.9 (99.5–100)	3.1 (2.6–3.7)	1.3 (1.1–1.5)	3.1 (2.3–3.8)	6.7 (4.9–8.4)	9.4 (7.7–11)
20–39 years							
1 (2007–2009)	728	100	14 (12–16)	5.2 (4.3–6.1)	12 (10–13)	46 (33–59)	84 (56–110)
2 (2009–2011)	357	100	5.6 (4.8–6.6)	2.2 (1.6–2.7)	4.9 (3.9–5.9)	13[E] (7.4–19)	19 (14–25)
5 (2016–2017)	370	99.1 (91.7–99.9)	2.8 (2.4–3.3)	1.1 (0.80–1.4)	2.4 (2.1–2.6)	8.5[E] (2.9–14)	14[E] (6.5–22)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	100	6.6 (5.9–7.4)	2.8 (2.3–3.3)	6.6 (5.8–7.5)	14 (12–17)	21[E] (13–29)
5 (2016–2017)	358	98.6 (89.7–99.8)	2.7 (2.4–3.2)	1.1 (0.78–1.5)	2.7 (2.4–3.0)	5.8 (4.4–7.3)	7.6 (5.0–10)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	100	6.9 (6.3–7.6)	3.1 (2.5–3.7)	7.0 (6.3–7.7)	14 (11–16)	18 (11–24)
5 (2016–2017)	352	100	3.6 (3.3–4.1)	1.6 (1.3–1.8)	3.6 (3.1–4.1)	7.5 (6.2–8.7)	10 (6.6–14)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.26

Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2561	99.5 (97.5–99.9)	13 (12–14)	3.9 (3.4–4.4)	12 (12–13)	39 (34–44)	59 (48–70)
5 (2016–2017)	2716	99.5 (97.0–99.9)	5.2 (4.4–6.1)	1.5 (1.1–1.8)	5.3 (4.3–6.3)	17 (12–21)	26 (20–31)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1282	99.2 (94.3–99.9)	14 (12–16)	4.2 (3.1–5.2)	13 (12–15)	43 (31–54)	69 (53–84)
5 (2016–2017)	1353	99.2 (93.7–99.9)	5.1 (4.3–6.2)	1.4 (0.91–1.8)	5.3 (4.2–6.3)	17 (12–21)	26 (20–32)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1279	99.9 (98.6–100)	12 (11–12)	3.8 (3.2–4.4)	11 (9.4–13)	35 (31–40)	47 (39–56)
5 (2016–2017)	1363	99.9 (99.4–100)	5.2 (4.3–6.4)	1.5 (1.1–2.0)	5.5 (3.9–7.0)	17 (11–22)	24 (18–31)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	100	26 (23–30)	8.6 (6.4–11)	25 (21–30)	69 (53–86)	99[E] (59–140)
5 (2016–2017)	555	100	12 (9.5–14)	3.6 (2.7–4.5)	11 (8.5–14)	36 (33–39)	44 (33–55)
6–11 years							
1 (2007–2009)	1037	100	31 (28–35)	8.8 (7.6–10)	31 (27–35)	100 (90–120)	180 (130–230)
2 (2009–2011)	516	100	24 (20–27)	7.0 (5.4–8.7)	24 (19–29)	71 (52–90)	97 (73–120)
5 (2016–2017)	537	100	9.7 (8.3–11)	3.1[E] (1.9–4.4)	9.9 (8.2–12)	29 (19–39)	44[E] (25–63)
12–19 years							
1 (2007–2009)	991	100	29 (24–34)	7.5 (5.8–9.3)	29 (25–32)	99 (78–120)	160[E] (64–260)
2 (2009–2011)	512	100	16 (14–20)	4.6[E] (2.4–6.8)	16 (13–20)	47 (36–58)	68 (49–87)
5 (2016–2017)	538	100	5.9 (5.3–6.6)	2.1 (1.5–2.8)	6.6 (5.8–7.5)	15 (10–20)	23 (17–29)
20–39 years							
1 (2007–2009)	730	100	22 (19–26)	5.7 (4.5–6.9)	21 (16–26)	90 (67–110)	150[E] (83–220)
2 (2009–2011)	359	98.6 (90.7–99.8)	11 (9.1–13)	3.8 (3.0–4.6)	12 (10–13)	30 (23–37)	40[E] (20–60)
5 (2016–2017)	374	100	4.7 (3.5–6.3)	1.2[E] (0.50–2.0)	4.8[E] (2.6–7.0)	16[E] (6.4–25)	25 (16–34)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	99.8 (97.8–100)	12 (9.7–14)	4.1[E] (2.6–5.5)	11 (8.7–13)	35 (28–41)	44[E] (21–67)
5 (2016–2017)	359	98.6 (89.7–99.8)	4.6 (3.6–5.8)	1.5[E] (0.91–2.1)	4.7 (3.5–5.9)	14[E] (8.7–19)	19 (14–25)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	291	100	10 (8.8–12)	3.4 (2.5–4.3)	10 (7.4–13)	28[E] (17–38)	44[E] (23–66)
5 (2016–2017)	353	99.8 (98.6–100)	4.7 (4.2–5.4)	1.5 (1.2–1.8)	4.8 (4.0–5.5)	12 (9.7–15)	18[E] (7.9–29)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.4, 0.4, and 0.22 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

Table 14.1.27

Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2551	99.5 (97.5–99.9)	12 (12–13)	5.0 (4.5–5.4)	12 (11–12)	32 (28–35)	51 (44–59)
5 (2016–2017)	2684	99.5 (97.0–99.9)	5.0 (4.5–5.6)	2.0 (1.7–2.4)	4.6 (4.1–5.2)	13 (11–15)	20 (16–23)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	99.2 (94.3–99.9)	12 (10–13)	4.3 (3.4–5.2)	10 (9.3–11)	33 (24–43)	58 (45–72)
5 (2016–2017)	1338	99.2 (93.7–99.9)	4.4 (4.0–4.9)	1.8 (1.5–2.1)	3.9 (3.4–4.4)	12 (10–13)	19 (14–25)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1273	99.9 (98.6–100)	13 (12–14)	5.4 (4.8–5.9)	13 (11–14)	30 (26–35)	43 (34–51)
5 (2016–2017)	1346	99.9 (99.4–100)	5.7 (4.9–6.7)	2.4 (2.0–2.7)	5.5 (4.8–6.2)	15 (10–19)	20[E] (12–28)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	100	46 (41–51)	20 (17–23)	42 (37–46)	110 (89–120)	130[E] (67–190)
5 (2016–2017)	544	100	20 (18–23)	8.5 (6.2–11)	19 (16–22)	49 (44–54)	62 (43–81)
6–11 years							
1 (2007–2009)	1034	100	48 (44–53)	19 (16–22)	44 (39–48)	130 (110–150)	190 (150–240)
2 (2009–2011)	514	100	27 (24–30)	11 (9.6–13)	24 (22–27)	70 (56–83)	90 (68–110)
5 (2016–2017)	529	100	11 (9.5–13)	4.8 (3.5–6.0)	10 (8.9–12)	29 (19–40)	43 (30–56)
12–19 years							
1 (2007–2009)	989	100	25 (21–29)	9.0 (7.9–10)	22 (18–25)	76[E] (44–110)	F
2 (2009–2011)	510	100	12 (11–14)	5.0 (4.2–5.9)	12 (10–14)	27 (21–34)	37[E] (15–60)
5 (2016–2017)	531	100	4.5 (3.9–5.3)	2.0 (1.6–2.4)	4.5 (3.6–5.5)	9.7 (7.2–12)	13 (8.1–17)
20–39 years							
1 (2007–2009)	728	100	23 (21–26)	8.5 (6.7–10)	20 (18–22)	80 (53–110)	140 (93–190)
2 (2009–2011)	357	98.6 (90.7–99.8)	9.4 (7.9–11)	3.9 (2.6–5.2)	8.5 (6.2–11)	23 (18–27)	29[E] (16–42)
5 (2016–2017)	370	100	4.3 (3.6–5.0)	1.8 (1.4–2.2)	3.9 (3.2–4.6)	12[E] (4.1–19)	19[E] (10–27)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	99.8 (97.8–100)	12 (11–13)	5.1 (4.3–5.9)	11 (10–13)	27 (22–33)	36[E] (18–54)
5 (2016–2017)	358	98.6 (89.7–99.8)	4.2 (3.6–4.9)	1.8 (1.3–2.3)	4.1 (3.8–4.5)	8.8 (6.5–11)	13 (8.7–17)
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	100	12 (11–13)	5.2 (4.2–6.1)	12 (11–14)	24 (21–27)	35 (27–42)
5 (2016–2017)	352	99.8 (98.6–100)	5.4 (4.9–6.1)	2.5 (2.1–2.8)	5.3 (4.7–5.8)	11 (9.0–13)	14[E] (8.1–20)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.28

Mono-carboxy-*n*-heptyl phthalate (MCHpP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2213	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1106	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1107	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	441	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	425	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	433	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	308	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	303	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	303	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.083 µg/L.

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

F Data are too unreliable to be published.

Table 14.1.29

Mono-carboxy-*n*-heptyl phthalate (MCHpP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2184	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1092	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1092	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	433	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	417	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	426	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	304	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	302	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	302	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

F Data are too unreliable to be published.

Table 14.1.30

Mono-*n*-octyl phthalate (MOP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2558	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2715	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1280	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1353	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1278	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1362	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	523	2.3[E] (1.4–3.6)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	554	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
1 (2007–2009)	1037	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	516	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	537	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
1 (2007–2009)	991	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	511	1.4[E] (0.7–2.9)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	538	1.3[E] (0.8–2.0)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
1 (2007–2009)	730	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	358	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	374	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	359	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	353	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.7, 0.3, and 0.16 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.31

Mono-*n*-octyl phthalate (MOP) (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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2548	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2683	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1338	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1272	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1345	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	2.3[E] (1.4–3.6)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	543	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
1 (2007–2009)	1034	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	514	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	529	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
1 (2007–2009)	989	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	509	1.4[E] (0.7–2.9)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	531	1.3[E] (0.8–2.0)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
1 (2007–2009)	728	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	356	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	370	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	358	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	289	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	352	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.32

Mono(carboxyisooctyl) phthalate (MCiOP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2713	84.2 (79.2–88.2)	0.97 (0.85–1.1)	<LOD	0.98 (0.84–1.1)	4.2 (3.1–5.3)	7.2 (4.8–9.7)
Males, 3–79 years							
5 (2016–2017)	1350	83.2 (75.8–88.7)	0.96 (0.79–1.2)	<LOD	0.99 (0.83–1.2)	3.9 (2.8–5.1)	6.8 (5.8–7.8)
Females, 3–79 years							
5 (2016–2017)	1363	85.1 (80.3–88.9)	0.99 (0.86–1.2)	<LOD	0.96 (0.81–1.1)	4.6[E] (2.9–6.4)	F
3–5 years							
5 (2016–2017)	555	91.8 (84.4–95.9)	1.3 (1.0–1.7)	0.33[E] (<LOD–0.48)	1.3[E] (0.81–1.8)	5.6[E] (2.2–8.9)	9.9[E] (4.2–15)
6–11 years							
5 (2016–2017)	537	91.9 (86.3–95.3)	1.3 (1.1–1.6)	0.36[E] (<LOD–0.51)	1.4 (1.0–1.7)	4.6[E] (2.4–6.7)	9.3[E] (4.3–14)
12–19 years							
5 (2016–2017)	537	85.8 (81.2–89.5)	1.2 (0.97–1.4)	<LOD	1.2 (0.85–1.5)	5.1 (3.6–6.6)	F
20–39 years							
5 (2016–2017)	373	82.8 (69.7–91.0)	0.95 (0.67–1.4)	<LOD	1.0[E] (0.55–1.5)	3.7[E] (2.1–5.4)	F
40–59 years							
5 (2016–2017)	359	82.8 (76.5–87.7)	0.92 (0.77–1.1)	<LOD	0.95 (0.66–1.2)	5.3[E] (2.9–7.6)	F
60–79 years							
5 (2016–2017)	352	83.5 (75.7–89.1)	0.86 (0.72–1.0)	<LOD	0.83 (0.70–0.96)	4.1[E] (1.8–6.3)	6.6 (5.1–8.0)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.30 µg/L.

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 are too unreliable to be published.

Table 14.1.33

Mono(carboxyisooctyl) phthalate (MCiOP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2681	84.2 (79.2–88.2)	0.94 (0.84–1.1)	<LOD	0.82 (0.73–0.91)	3.7 (3.1–4.3)	6.4 (5.5–7.4)
Males, 3–79 years							
5 (2016–2017)	1335	83.2 (75.8–88.7)	0.82 (0.70–0.96)	<LOD	0.77 (0.63–0.91)	3.3 (2.4–4.3)	6.0 (4.7–7.4)
Females, 3–79 years							
5 (2016–2017)	1346	85.1 (80.3–88.9)	1.1 (0.95–1.2)	<LOD	0.92 (0.79–1.0)	4.0[E] (2.2–5.8)	7.3[E] (<LOD–12)
3–5 years							
5 (2016–2017)	544	91.8 (84.4–95.9)	2.4 (2.0–2.8)	0.73 (<LOD–0.96)	2.2 (1.8–2.6)	8.1[E] (4.3–12)	13[E] (6.1–19)
6–11 years							
5 (2016–2017)	529	91.9 (86.3–95.3)	1.6 (1.3–1.9)	0.51 (<LOD–0.63)	1.4 (1.2–1.6)	5.1 (3.5–6.7)	7.3[E] (3.5–11)
12–19 years							
5 (2016–2017)	530	85.8 (81.2–89.5)	0.90 (0.75–1.1)	<LOD	0.77 (0.66–0.89)	3.5[E] (1.5–5.4)	F
20–39 years							
5 (2016–2017)	369	82.8 (69.7–91.0)	0.85 (0.68–1.1)	<LOD	0.77 (0.61–0.94)	2.6[E] (1.5–3.7)	F
40–59 years							
5 (2016–2017)	358	82.8 (76.5–87.7)	0.84 (0.67–1.1)	<LOD	0.75 (0.63–0.88)	F	6.5 (5.2–7.7)
60–79 years							
5 (2016–2017)	351	83.5 (75.7–89.1)	0.98 (0.83–1.2)	<LOD	0.83 (0.70–0.95)	4.0 (2.9–5.1)	5.2 (3.4–7.0)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.34

Monoisononyl phthalate (MiNP) — 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), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2556	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2716	41.9 (33.2–51.1)	—	<LOD	<LOD	1.4[E] (0.89–2.0)	3.5[E] (1.5–5.4)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1280	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1353	44.8 (35.8–54.1)	—	<LOD	<LOD	1.1 (0.81–1.3)	2.2[E] (1.4–3.0)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1363	38.9 (29.8–48.9)	—	<LOD	<LOD	F	4.0[E] (1.2–6.8)
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	522	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	555	49.1 (32.5–65.9)	—	<LOD	<LOD	1.5 (1.0–2.0)	F
6–11 years							
1 (2007–2009)	1036	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	514	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	537	49.5 (38.9–60.2)	—	<LOD	<LOD	1.2 (0.75–1.6)	2.0 (1.5–2.6)
12–19 years							
1 (2007–2009)	991	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	511	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	538	49.1 (38.1–60.2)	—	<LOD	<LOD	1.5[E] (0.70–2.3)	F
20–39 years							
1 (2007–2009)	730	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	358	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	374	50.1 (33.9–66.3)	—	<LOD	<LOD	F	4.9[E] (2.1–7.7)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	360	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	359	37.2 (29.2–45.9)	—	<LOD	<LOD	1.2[E] (0.71–1.8)	F
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	291	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	353	29.9 (20.8–40.9)	—	<LOD	<LOD	0.94 (0.63–1.3)	1.5[E] (0.75–2.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 1, 2, and 5 are 0.4, 0.3, and 0.37 µg/L, respectively.

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.35

Monoisononyl phthalate (MiNP) (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) and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (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)	2546	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2684	41.9 (33.2–51.1)	—	<LOD	<LOD	1.3[E] (0.73–1.8)	3.3[E] (1.4–5.1)
Males, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1276	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1338	44.8 (35.8–54.1)	—	<LOD	<LOD	1.0 (0.89–1.2)	1.8[E] (0.67–3.0)
Females, 3–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	1270	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1346	38.9 (29.8–48.9)	—	<LOD	<LOD	F	F
3–5 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	521	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	544	49.1 (32.5–65.9)	—	<LOD	<LOD	2.4[E] (1.1–3.8)	F

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
6–11 years							
1 (2007–2009)	1033	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	512	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	529	49.5 (38.9–60.2)	—	<LOD	<LOD	1.3[E] (0.76–1.8)	3.1 (2.0–4.2)
12–19 years							
1 (2007–2009)	989	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	509	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	531	49.1 (38.1–60.2)	—	<LOD	<LOD	1.1[E] (0.63–1.6)	F
20–39 years							
1 (2007–2009)	728	F	—	<LOD	<LOD	<LOD	<LOD
2 (2009–2011)	356	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	370	50.1 (33.9–66.3)	—	<LOD	<LOD	F	F
40–59 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	358	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	358	37.2 (29.2–45.9)	—	<LOD	<LOD	F	F
60–79 years							
1 (2007–2009)[b]	—	—	—	—	—	—	—
2 (2009–2011)	290	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	352	29.9 (20.8–40.9)	—	<LOD	<LOD	1.1[E] (0.61–1.5)	1.7[E] (0.99–2.4)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

b Data are not available, as participants under the age of six years and over 49 years were not included in cycle 1.

E Use data with caution.

F Data are too unreliable to be published.

Table 14.1.36

Monocarboxyisononyl phthalate (MCiNP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2551	95.9 (92.2–97.9)	0.68 (0.57–0.80)	0.16 (0.12–0.21)	0.71 (0.55–0.88)	2.5 (1.9–3.1)	4.0 (3.0–5.0)
Males, 3–79 years							
5 (2016–2017)	1272	94.9 (90.7–97.2)	0.68 (0.57–0.83)	0.17[E] (0.081–0.25)	0.73 (0.59–0.88)	2.4 (1.8–2.9)	4.2 (3.1–5.4)
Females, 3–79 years							
5 (2016–2017)	1279	96.9 (91.4–98.9)	0.67 (0.54–0.82)	0.16 (0.11–0.21)	0.67 (0.44–0.90)	2.5 (1.8–3.2)	3.8 (2.6–5.0)
3–5 years							
5 (2016–2017)	520	99.9 (99.5–100)	0.96[E] (0.62–1.5)	0.27[E] (0.16–0.38)	0.87[E] (0.52–1.2)	3.8[E] (1.2–6.3)	F
6–11 years							
5 (2016–2017)	517	99.5 (99.0–99.8)	1.1 (0.87–1.3)	0.33 (0.24–0.41)	1.0 (0.77–1.2)	3.3 (2.6–4.0)	4.5[E] (2.1–6.8)
12–19 years							
5 (2016–2017)	498	97.2 (93.9–98.7)	0.79 (0.68–0.92)	0.22[E] (0.098–0.34)	0.85 (0.70–0.99)	2.3 (1.8–2.9)	3.8 (2.6–5.0)
20–39 years							
5 (2016–2017)	356	94.3 (80.2–98.5)	0.58 (0.41–0.84)	F	0.56[E] (0.28–0.83)	2.3[E] (1.4–3.3)	3.5[E] (2.0–5.0)
40–59 years							
5 (2016–2017)	325	95.0 (89.5–97.7)	0.69 (0.53–0.90)	0.15[E] (<LOD–0.23)	0.77 (0.58–0.96)	2.8[E] (1.8–3.8)	4.6[E] (2.6–6.5)
60–79 years							
5 (2016–2017)	335	97.0 (92.1–98.9)	0.60 (0.51–0.70)	0.16 (0.12–0.19)	0.61 (0.47–0.76)	2.1 (1.7–2.6)	3.4 (2.4–4.5)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.077 µg/L.

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 are too unreliable to be published.

Table 14.1.37

Monocarboxyisononyl phthalate (MCiNP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2522	95.9 (92.2–97.9)	0.64 (0.56–0.73)	0.22 (0.18–0.26)	0.58 (0.50–0.66)	2.0 (1.6–2.3)	2.7 (2.2–3.2)
Males, 3–79 years							
5 (2016–2017)	1257	94.9 (90.7–97.2)	0.57 (0.51–0.64)	0.20 (0.15–0.24)	0.52 (0.48–0.56)	1.9 (1.5–2.3)	2.7 (2.1–3.3)
Females, 3–79 years							
5 (2016–2017)	1265	96.9 (91.4–98.9)	0.71 (0.60–0.85)	0.28 (0.23–0.33)	0.64 (0.50–0.78)	2.0 (1.6–2.4)	2.7 (2.0–3.4)
3–5 years							
5 (2016–2017)	510	99.9 (99.5–100)	1.7 (1.2–2.3)	0.63 (0.41–0.85)	1.5 (1.2–1.7)	F	F
6–11 years							
5 (2016–2017)	509	99.5 (99.0–99.8)	1.2 (1.0–1.5)	0.44 (0.40–0.49)	1.1 (0.90–1.2)	2.9[E] (1.6–4.2)	F
12–19 years							
5 (2016–2017)	493	97.2 (93.9–98.7)	0.58 (0.49–0.68)	0.22 (0.17–0.27)	0.53 (0.41–0.65)	1.8 (1.4–2.2)	2.4 (1.5–3.2)
20–39 years							
5 (2016–2017)	352	94.3 (80.2–98.5)	0.51 (0.42–0.62)	0.18 (<LOD–0.21)	0.47 (0.38–0.56)	1.3[E] (0.55–2.1)	2.0[E] (0.99–3.0)
40–59 years							
5 (2016–2017)	324	95.0 (89.5–97.7)	0.61 (0.51–0.74)	0.21[E] (<LOD–0.31)	0.57 (0.44–0.70)	2.0 (1.4–2.6)	2.6 (2.0–3.2)
60–79 years							
5 (2016–2017)	334	97.0 (92.1–98.9)	0.67 (0.58–0.78)	0.26 (0.23–0.30)	0.64 (0.55–0.73)	1.8 (1.4–2.2)	2.5 (1.8–3.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.38

Monooxoisononyl phthalate (MOiNP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2640	90.2 (86.5–92.9)	0.67 (0.60–0.75)	F	0.63 (0.57–0.69)	3.2 (2.8–3.6)	5.4 (3.7–7.2)
Males, 3–79 years							
5 (2016–2017)	1318	90.2 (84.4–93.9)	0.65 (0.54–0.78)	F	0.61 (0.52–0.70)	2.5 (1.8–3.2)	4.3[E] (2.1–6.4)
Females, 3–79 years							
5 (2016–2017)	1322	90.2 (86.1–93.2)	0.69 (0.59–0.82)	F	0.66 (0.56–0.77)	3.5 (2.6–4.5)	F
3–5 years							
5 (2016–2017)	548	97.4 (91.5–99.3)	1.1 (0.88–1.4)	0.26[E] (<LOD–0.42)	1.1 (0.89–1.2)	4.4 (2.8–6.0)	6.7[E] (3.4–10)
6–11 years							
5 (2016–2017)	524	97.5 (94.9–98.8)	1.0 (0.87–1.3)	0.31 (0.27–0.36)	1.0 (0.80–1.3)	3.6 (2.6–4.6)	5.0[E] (1.6–8.4)
12–19 years							
5 (2016–2017)	525	94.4 (90.7–96.6)	0.93 (0.79–1.1)	0.20 (<LOD–0.26)	0.90 (0.73–1.1)	3.8[E] (2.0–5.5)	F
20–39 years							
5 (2016–2017)	358	88.4 (76.3–94.8)	0.67 (0.50–0.90)	<LOD	0.59 (0.41–0.76)	3.3 (2.2–4.3)	5.8[E] (1.7–9.9)
40–59 years							
5 (2016–2017)	343	89.1 (75.9–95.5)	0.59 (0.46–0.75)	<LOD	0.56 (0.45–0.67)	2.9 (1.9–4.0)	F
60–79 years							
5 (2016–2017)	342	88.7 (84.1–92.1)	0.55 (0.46–0.66)	<LOD	0.54 (0.40–0.68)	2.6[E] (1.4–3.7)	3.7[E] (2.1–5.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.15 µg/L.

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 are too unreliable to be published.

Table 14.1.39

Monooxoisononyl phthalate (MOiNP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2609	90.2 (86.5–92.9)	0.65 (0.59–0.71)	<LOD	0.55 (0.50–0.60)	2.8 (2.3–3.2)	5.2 (3.7–6.7)
Males, 3–79 years							
5 (2016–2017)	1304	90.2 (84.4–93.9)	0.55 (0.48–0.64)	<LOD	0.48 (0.37–0.58)	2.1 (1.7–2.5)	4.3 (2.9–5.8)
Females, 3–79 years							
5 (2016–2017)	1305	90.2 (86.1–93.2)	0.76 (0.68–0.85)	<LOD	0.63 (0.51–0.76)	3.4 (2.2–4.7)	5.9[E] (<LOD–9.2)
3–5 years							
5 (2016–2017)	537	97.4 (91.5–99.3)	1.9 (1.6–2.3)	0.65 (<LOD–0.82)	1.8 (1.5–2.1)	6.2 (4.3–8.1)	10[E] (5.2–16)
6–11 years							
5 (2016–2017)	517	97.5 (94.9–98.8)	1.2 (1.0–1.5)	0.44 (0.37–0.51)	1.1 (0.92–1.2)	3.9 (2.6–5.2)	5.9[E] (2.7–9.0)
12–19 years							
5 (2016–2017)	518	94.4 (90.7–96.6)	0.71 (0.60–0.82)	0.24 (<LOD–0.29)	0.59 (0.50–0.68)	2.3[E] (1.4–3.2)	5.7[E] (1.9–9.5)
20–39 years							
5 (2016–2017)	354	88.4 (76.3–94.8)	0.60 (0.51–0.71)	<LOD	0.46 (0.35–0.57)	F	5.8[E] (3.2–8.5)
40–59 years							
5 (2016–2017)	342	89.1 (75.9–95.5)	0.53 (0.44–0.64)	<LOD	0.50 (0.36–0.64)	2.2[E] (1.0–3.3)	F
60–79 years							
5 (2016–2017)	341	88.7 (84.1–92.1)	0.62 (0.53–0.73)	<LOD	0.55 (0.43–0.67)	2.1 (1.5–2.7)	4.3[E] (2.3–6.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.40

Monohydroxyisononyl phthalate (MHiNP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2712	98.2 (95.9–99.3)	0.70 (0.62–0.79)	0.20 (0.16–0.24)	0.71 (0.57–0.84)	2.5 (2.0–3.0)	4.0 (3.0–4.9)
Males, 3–79 years							
5 (2016–2017)	1351	97.4 (95.1–98.6)	0.70 (0.61–0.80)	0.21 (0.14–0.28)	0.71 (0.58–0.84)	2.3 (1.9–2.8)	4.3 (3.2–5.4)
Females, 3–79 years							
5 (2016–2017)	1361	99.1 (91.1–99.9)	0.70 (0.58–0.85)	0.19 (0.14–0.25)	0.70 (0.49–0.91)	2.7 (2.2–3.3)	3.8 (2.7–4.9)
3–5 years							
5 (2016–2017)	555	100	0.96[E] (0.62–1.5)	0.27[E] (0.16–0.38)	0.87[E] (0.52–1.2)	F	F
6–11 years							
5 (2016–2017)	537	99.6 (98.2–99.9)	1.1 (0.86–1.3)	0.33 (0.24–0.42)	1.0 (0.76–1.2)	3.3 (2.6–4.0)	4.5[E] (2.1–6.9)
12–19 years							
5 (2016–2017)	535	98.2 (94.7–99.4)	0.79 (0.67–0.94)	0.27[E] (0.15–0.40)	0.84 (0.68–1.0)	2.4 (1.7–3.1)	3.8 (2.7–4.8)
20–39 years							
5 (2016–2017)	374	97.1 (78.6–99.7)	0.60 (0.43–0.83)	0.20[E] (<LOD–0.33)	0.54[E] (0.30–0.77)	2.3[E] (1.4–3.2)	3.7[E] (2.1–5.3)
40–59 years							
5 (2016–2017)	358	97.6 (91.2–99.4)	0.72 (0.56–0.92)	0.18[E] (0.11–0.26)	0.77 (0.57–0.97)	2.8 (1.9–3.6)	4.2[E] (2.4–6.1)
60–79 years							
5 (2016–2017)	353	100	0.66 (0.58–0.75)	0.18 (0.15–0.21)	0.65 (0.50–0.81)	2.2 (1.5–3.0)	3.5 (2.5–4.6)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.065 µg/L.

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 are too unreliable to be published.

Table 14.1.41

Monohydroxyisononyl phthalate (MHiNP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2680	98.2 (95.9–99.3)	0.68 (0.61–0.75)	0.25 (0.22–0.29)	0.60 (0.53–0.66)	2.0 (1.7–2.3)	2.8 (2.2–3.3)
Males, 3–79 years							
5 (2016–2017)	1336	97.4 (95.1–98.6)	0.60 (0.57–0.64)	0.21 (0.18–0.24)	0.53 (0.50–0.57)	1.9 (1.5–2.2)	2.7 (2.1–3.4)
Females, 3–79 years							
5 (2016–2017)	1344	99.1 (91.1–99.9)	0.76 (0.64–0.91)	0.30 (0.26–0.33)	0.66 (0.52–0.79)	2.1 (1.8–2.5)	2.9 (2.0–3.8)
3–5 years							
5 (2016–2017)	544	100	1.7 (1.2–2.3)	0.63 (0.40–0.86)	1.5 (1.2–1.8)	5.0[E] (1.9–8.0)	F
6–11 years							
5 (2016–2017)	529	99.6 (98.2–99.9)	1.2 (1.0–1.5)	0.44 (0.39–0.49)	1.1 (0.89–1.2)	2.9[E] (1.6–4.2)	F
12–19 years							
5 (2016–2017)	528	98.2 (94.7–99.4)	0.60 (0.51–0.71)	0.23 (0.19–0.28)	0.56 (0.44–0.67)	1.8 (1.4–2.2)	2.4[E] (1.5–3.3)
20–39 years							
5 (2016–2017)	370	97.1 (78.6–99.7)	0.54 (0.44–0.65)	0.19 (<LOD–0.25)	0.51 (0.43–0.60)	1.3[E] (0.61–2.0)	F
40–59 years							
5 (2016–2017)	357	97.6 (91.2–99.4)	0.66 (0.57–0.75)	0.26 (0.20–0.33)	0.58 (0.47–0.69)	2.1 (1.4–2.8)	2.6 (2.0–3.2)
60–79 years							
5 (2016–2017)	352	100	0.75 (0.65–0.87)	0.29 (0.26–0.32)	0.68 (0.59–0.77)	2.0 (1.5–2.5)	3.5[E] (1.9–5.1)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.42

Monoisodecyl phthalate (MiDP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2710	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1348	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1362	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	554	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	535	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	537	1.9[E] (1.1–3.4)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	373	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	358	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	353	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.16 µg/L.

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 are too unreliable to be published.

Table 14.1.43

Monoisodecyl phthalate (MiDP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2678	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1333	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1345	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	543	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	527	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	530	1.9[E] (1.1–3.4)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	369	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	357	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	352	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.44

Monooxoisodecyl phthalate (MOiDP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2688	73.4 (68.6–77.6)	0.27 (0.23–0.31)	<LOD	0.24 (0.19–0.29)	1.6 (1.1–2.1)	3.4[E] (1.4–5.4)
Males, 3–79 years							
5 (2016–2017)	1341	72.7 (65.5–78.8)	0.27 (0.21–0.34)	<LOD	0.26 (0.18–0.34)	1.6[E] (0.81–2.3)	3.8[E] (1.6–6.0)
Females, 3–79 years							
5 (2016–2017)	1347	74.0 (67.9–79.4)	0.26 (0.21–0.34)	<LOD	0.22 (0.16–0.28)	1.7[E] (0.97–2.3)	F
3–5 years							
5 (2016–2017)	547	79.8 (72.0–85.8)	0.33 (0.27–0.41)	<LOD	0.31[E] (0.17–0.45)	2.0 (1.5–2.5)	3.2[E] (1.6–4.8)
6–11 years							
5 (2016–2017)	534	85.2 (79.6–89.6)	0.38 (0.30–0.49)	<LOD	0.35 (0.25–0.45)	1.9[E] (0.81–2.9)	4.8[E] (2.2–7.3)
12–19 years							
5 (2016–2017)	534	80.3 (73.7–85.5)	0.36 (0.27–0.49)	<LOD	0.37 (0.24–0.50)	F	4.3[E] (2.4–6.3)
20–39 years							
5 (2016–2017)	371	74.4 (63.9–82.7)	0.25 (0.19–0.34)	<LOD	0.21[E] (0.13–0.30)	1.6[E] (0.64–2.5)	F
40–59 years							
5 (2016–2017)	354	69.0 (59.9–76.7)	0.26[E] (0.18–0.37)	<LOD	0.24[E] (0.13–0.36)	1.6[E] (0.49–2.7)	F
60–79 years							
5 (2016–2017)	348	69.9 (63.8–75.4)	0.22 (0.17–0.29)	<LOD	0.18 (0.14–0.23)	F	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.097 µg/L.

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 are too unreliable to be published.

Table 14.1.45

Monooxoisodecyl phthalate (MOiDP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2656	73.4 (68.6–77.6)	0.26 (0.21–0.32)	<LOD	0.21 (0.17–0.25)	1.5 (0.97–2.0)	3.0[E] (1.0–5.0)
Males, 3–79 years							
5 (2016–2017)	1326	72.7 (65.5–78.8)	0.23 (0.18–0.30)	<LOD	0.19 (0.16–0.23)	1.3[E] (0.39–2.2)	3.4[E] (1.1–5.8)
Females, 3–79 years							
5 (2016–2017)	1330	74.0 (67.9–79.4)	0.29 (0.22–0.38)	<LOD	0.24 (0.19–0.29)	1.6 (1.0–2.1)	F
3–5 years							
5 (2016–2017)	536	79.8 (72.0–85.8)	0.57 (0.47–0.70)	<LOD	0.53 (0.43–0.64)	2.5 (1.8–3.2)	4.5[E] (1.5–7.5)
6–11 years							
5 (2016–2017)	526	85.2 (79.6–89.6)	0.45 (0.35–0.59)	<LOD	0.38 (0.27–0.48)	2.0[E] (0.90–3.0)	4.6[E] (1.9–7.3)
12–19 years							
5 (2016–2017)	527	80.3 (73.7–85.5)	0.28 (0.21–0.38)	<LOD	0.22[E] (0.12–0.31)	1.4[E] (0.58–2.3)	3.0[E] (1.0–5.0)
20–39 years							
5 (2016–2017)	367	74.4 (63.9–82.7)	0.23 (0.17–0.31)	<LOD	0.21 (0.16–0.26)	1.3[E] (0.70–2.0)	F
40–59 years							
5 (2016–2017)	353	69.0 (59.9–76.7)	0.23[E] (0.16–0.34)	<LOD	0.18 (0.12–0.24)	2.0[E] (0.76–3.3)	F
60–79 years							
5 (2016–2017)	347	69.9 (63.8–75.4)	0.26 (0.20–0.33)	<LOD	0.20 (0.15–0.25)	1.5[E] (0.68–2.2)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.1.46

Monohydroxyisodecyl phthalate (MHiDP) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2700	71.4 (62.2–79.0)	0.21 (0.17–0.26)	<LOD	0.19 (0.14–0.24)	1.2 (0.88–1.6)	3.0[E] (1.7–4.4)
Males, 3–79 years							
5 (2016–2017)	1347	73.3 (63.0–81.5)	0.22 (0.17–0.28)	<LOD	0.22 (0.14–0.30)	1.2[E] (0.51–1.8)	2.6[E] (1.3–3.8)
Females, 3–79 years							
5 (2016–2017)	1353	69.5 (60.0–77.5)	0.20 (0.15–0.26)	<LOD	0.18 (0.13–0.22)	1.3 (0.82–1.7)	3.7[E] (1.5–6.0)
3–5 years							
5 (2016–2017)	552	79.6 (69.9–86.8)	0.28 (0.23–0.35)	<LOD	0.29[E] (0.17–0.41)	1.9 (1.5–2.4)	2.5 (1.9–3.0)
6–11 years							
5 (2016–2017)	532	78.7 (70.3–85.2)	0.29 (0.23–0.38)	<LOD	0.31 (0.21–0.41)	1.8[E] (1.0–2.5)	4.2[E] (2.4–5.9)
12–19 years							
5 (2016–2017)	536	76.1 (62.2–86.1)	0.27[E] (0.18–0.39)	<LOD	0.27 (0.18–0.37)	1.6[E] (0.50–2.7)	3.8[E] (1.6–6.0)
20–39 years							
5 (2016–2017)	371	71.5 (60.6–80.3)	0.19 (0.13–0.28)	<LOD	0.18[E] (0.11–0.26)	F	F
40–59 years							
5 (2016–2017)	359	68.6 (53.0–80.8)	0.21[E] (0.14–0.32)	<LOD	0.18[E] (0.071–0.28)	1.3[E] (0.41–2.2)	F
60–79 years							
5 (2016–2017)	350	69.3 (57.7–78.9)	0.18 (0.13–0.24)	<LOD	0.15 (0.11–0.19)	F	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.067 µg/L.

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 are too unreliable to be published.

Table 14.1.47

Monohydroxyisodecyl phthalate (MHiDP) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2669	71.4 (62.2–79.0)	0.20 (0.15–0.27)	<LOD	0.19 (0.15–0.23)	1.3 (0.88–1.7)	2.4[E] (0.84–4.0)
Males, 3–79 years							
5 (2016–2017)	1332	73.3 (63.0–81.5)	0.19 (0.14–0.25)	<LOD	0.19 (0.16–0.22)	1.1[E] (0.50–1.7)	2.9[E] (0.90–4.9)
Females, 3–79 years							
5 (2016–2017)	1337	69.5 (60.0–77.5)	0.22 (0.16–0.30)	<LOD	0.19 (0.14–0.24)	1.4 (0.89–1.9)	F
3–5 years							
5 (2016–2017)	541	79.6 (69.9–86.8)	0.49 (0.39–0.62)	<LOD	0.48 (0.37–0.60)	2.4 (1.9–3.0)	4.0 (2.8–5.2)
6–11 years							
5 (2016–2017)	525	78.7 (70.3–85.2)	0.34 (0.26–0.46)	<LOD	0.34 (0.26–0.41)	1.6[E] (0.97–2.2)	3.8[E] (1.3–6.4)
12–19 years							
5 (2016–2017)	529	76.1 (62.2–86.1)	0.20 (0.14–0.29)	<LOD	0.16[E] (0.092–0.24)	1.1[E] (0.32–2.0)	2.3[E] (0.91–3.6)
20–39 years							
5 (2016–2017)	367	71.5 (60.6–80.3)	0.17[E] (0.11–0.27)	<LOD	0.18 (0.12–0.23)	0.99[E] (0.41–1.6)	F
40–59 years							
5 (2016–2017)	358	68.6 (53.0–80.8)	0.19[E] (0.12–0.29)	<LOD	0.19[E] (0.11–0.27)	1.5[E] (0.52–2.4)	F
60–79 years							
5 (2016–2017)	349	69.3 (57.7–78.9)	0.20 (0.15–0.27)	<LOD	0.16[E] (0.099–0.21)	1.1[E] (0.69–1.6)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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14.2 DI(ISONONYL)-CYCLOHEXANE-1,2-DICARBOXYLATE (DINCH)

Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH) (CASRN 166412-78-8) is an aliphatic ester compound with the appearance of a colourless liquid at room temperature. This substance may also be referred to as diisononyl hexahydrophthalate, among other synonyms. Produced commercially, DINCH is commonly synthesized by catalytic hydrogenation of the aromatic ring of diisononyl phthalate (DiNP), and consists mostly of *cis*-isomers (~90%) with a much smaller proportion of *trans*-isomers (Bhat et al., 2014; Koch et al., 2013; SCENIHR, 2016). DINCH is used as a substitute for high molecular-weight medium-chain phthalate plasticizers such as DiNP and di(2-ethylhexyl) phthalate (DEHP), primarily in polyvinyl chloride (PVC) materials used in beverage and food contact applications (Bhat et al., 2014; NICNAS, 2012). DINCH is also used as a plasticizer and impact modifier in polystyrene for sensitive applications such as toys, food contact materials, and medical devices (Bhat et al., 2014; Koch et al., 2013; NICNAS, 2012).

DINCH does not occur naturally. It is only released to the environment from anthropogenic sources. Release of this substance into the environment can occur during PVC product manufacture and from degradation of consumer products. DINCH has very low volatility and water solubility; therefore, it is expected to occur minimally in air and water (NICNAS, 2012). Given that DINCH can leach out of the polymer matrix when used as a plasticizer, the general population may be exposed to it dermally from contact with consumer products such as plastic toys and car upholstery, orally from materials that contact food or beverages, orally or intravenously through medical applications, or via the inhalation or ingestion of house dust (Bhat et al., 2014; NICNAS, 2012; SCENIHR, 2016). However, due to the limited use of DINCH in products available to consumers and the low leaching of the compound out of the polymer matrix when used as a plasticizer, exposure via use of consumer products is expected to be low (NICNAS, 2012; SCENIHR, 2016). Given the very low vapour pressure of DINCH, exposure via inhalation is of minimal concern (NICNAS, 2012).

The toxicokinetics of DINCH in humans are not well studied. Experimental animal studies have reported rapid absorption of DINCH following ingestion, while no data were identified concerning dermal absorption (Bhat et al., 2014; SCENIHR, 2016). The oral bioavailability of DINCH is inversely proportional to the dose due to saturation of gastrointestinal absorption, with one study demonstrating that a higher oral dose led to a lower absorbed fraction in laboratory animals. Studies of orally exposed animals indicate that DINCH is distributed throughout the body following absorption (Bhat et al., 2014; ECHA, 2016; SCENIHR, 2016). In animals exposed by ingestion, DINCH was found to undergo hydrolysis to cyclohexane-1,2-dicarboxylic mono isononyl ester (MINCH) before being further hydrolyzed to cyclohexane-1,2-dicarboxylic acid (CHDA), the main urinary metabolite; unmetabolized DINCH was the main compound identified in feces (Bhat et al., 2014; Koch et al., 2013). In studies with laboratory animals, total excretion of radiolabelled DINCH and its metabolites represented ~90% of the administered dose. DINCH is mainly excreted in feces within 48 hours, whereas a smaller fraction is eliminated via urinary metabolites over the same period (Bhat et al., 2014).

Acute dermal and ocular exposure to DINCH was found to be non-irritating in laboratory animals, and no skin sensitization was observed. Experimental animal studies have reported that DINCH has low toxicity following acute, short-term, or sub-chronic ingestion exposure. However, chronic ingestion of high doses of this substance was found to result in increased liver, kidney, and thyroid weights (Bhat et al., 2014; SCENIHR, 2016). One study of laboratory animals exposed *in utero* to DINCH from gestational day 14 until parturition reported a long-term effect on Leydig cells of the testis, indicated by reduced circulating testosterone levels and altered testicular morphology

(Campioli et al., 2017). However, this finding has not been repeated in other studies. In a two-generation animal study, DINCH had no adverse reproductive or developmental effects (Bhat et al., 2014). DINCH is not considered genotoxic or carcinogenic (Bhat et al., 2014; ECHA, 2016; SCENIHR, 2016).

Six metabolites of DINCH (*trans*-cyclohexane-1,2-dicarboxylic mono isononyl ester [*trans*-MINCH]; cyclohexane-1,2-dicarboxylic mono oxoisononyl ester [oxo-MINCH]; cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester [OH-MINCH]; *cis*-cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester [*cis*-cx-MINCH]; *trans*-cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester [*trans*-cx-MINCH]; and CHDA) were analyzed in the urine of Canadian Health Measures Survey participants aged 3–79 years in cycle 5 (2016–2017). Data from the metabolites are presented both as µg/L and µg/g creatinine. Finding a measurable amount of these metabolites in urine can be an indicator of recent exposure to DINCH and does not necessarily mean that an adverse effect will occur.

Table 14.2.1

trans-Cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2680	7.7[E] (5.0–11.8)	—	<LOD	<LOD	<LOD	0.024 (<LOD–0.032)
Males, 3–79 years							
5 (2016–2017)	1340	8.7[E] (5.0–14.5)	—	<LOD	<LOD	<LOD	0.025 (<LOD–0.034)
Females, 3–79 years							
5 (2016–2017)	1340	6.8[E] (4.5–10.2)	—	<LOD	<LOD	<LOD	0.023 (<LOD–0.030)
3–5 years							
5 (2016–2017)	540	32.3 (26.0–39.3)	—	<LOD	<LOD	0.045 (0.032–0.059)	0.076[E] (0.047–0.10)
6–11 years							
5 (2016–2017)	532	19.8 (14.6–26.2)	—	<LOD	<LOD	0.029 (0.023–0.036)	0.035[E] (0.022–0.049)
12–19 years							
5 (2016–2017)	535	F	—	<LOD	<LOD	<LOD	0.024[E] (<LOD–0.034)
20–39 years							
5 (2016–2017)	368	F	—	<LOD	<LOD	<LOD	0.019[E] (<LOD–0.030)
40–59 years							
5 (2016–2017)	357	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	348	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.017 µg/L.

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 are too unreliable to be published.

Table 14.2.2

trans-Cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2650	7.7[E] (5.0–11.8)	—	<LOD	<LOD	<LOD	0.041 (<LOD–0.048)
Males, 3–79 years							
5 (2016–2017)	1325	8.7[E] (5.0–14.5)	—	<LOD	<LOD	<LOD	0.034 (<LOD–0.045)
Females, 3–79 years							
5 (2016–2017)	1325	6.8[E] (4.5–10.2)	—	<LOD	<LOD	<LOD	0.042 (<LOD–0.046)
3–5 years							
5 (2016–2017)	530	32.3 (26.0–39.3)	—	<LOD	<LOD	0.075 (0.049–0.10)	0.13[E] (0.058–0.21)
6–11 years							
5 (2016–2017)	525	19.8 (14.6–26.2)	—	<LOD	<LOD	0.034 (0.028–0.040)	0.046[E] (0.029–0.063)
12–19 years							
5 (2016–2017)	528	F	—	<LOD	<LOD	<LOD	0.031 (<LOD–0.042)
20–39 years							
5 (2016–2017)	364	F	—	<LOD	<LOD	<LOD	0.041[E] (<LOD–0.057)
40–59 years							
5 (2016–2017)	356	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	347	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.2.3

Cyclohexane-1,2-dicarboxylic mono oxoisononyl ester (oxo-MINCH) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2655	36.8 (32.2–41.6)	—	<LOD	<LOD	0.19 (0.15–0.22)	0.35 (0.27–0.43)
Males, 3–79 years							
5 (2016–2017)	1328	37.0 (29.5–45.1)	—	<LOD	<LOD	0.19 (0.12–0.25)	0.34 (0.22–0.46)
Females, 3–79 years							
5 (2016–2017)	1327	36.6 (30.4–43.2)	—	<LOD	<LOD	0.19 (0.15–0.23)	0.36 (0.25–0.48)
3–5 years							
5 (2016–2017)	541	79.1 (71.2–85.3)	0.16 (0.13–0.20)	<LOD	0.19 (0.15–0.23)	0.81[E] (0.47–1.2)	1.1 (0.85–1.4)
6–11 years							
5 (2016–2017)	527	68.3 (62.5–73.6)	0.097 (0.085–0.11)	<LOD	0.096 (0.077–0.11)	0.49 (0.37–0.61)	0.68 (0.58–0.79)
12–19 years							
5 (2016–2017)	525	41.8 (36.3–47.5)	—	<LOD	<LOD	0.19 (0.14–0.25)	0.34[E] (0.11–0.58)
20–39 years							
5 (2016–2017)	363	36.8 (29.6–44.6)	—	<LOD	<LOD	0.18[E] (0.089–0.26)	0.28[E] (0.17–0.38)
40–59 years							
5 (2016–2017)	353	29.4 (22.0–38.1)	—	<LOD	<LOD	F	F
60–79 years							
5 (2016–2017)	346	27.6 (19.1–38.1)	—	<LOD	<LOD	0.11[E] (0.069–0.15)	0.14 (0.093–0.19)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.047 µg/L.

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 are too unreliable to be published.

Table 14.2.4

Cyclohexane-1,2-dicarboxylic mono oxoisononyl ester (oxo-MINCH) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2626	36.8 (32.2–41.6)	—	<LOD	<LOD	0.15 (0.14–0.17)	0.28 (0.21–0.34)
Males, 3–79 years							
5 (2016–2017)	1313	37.0 (29.5–45.1)	—	<LOD	<LOD	0.15 (0.12–0.18)	0.31[E] (0.19–0.42)
Females, 3–79 years							
5 (2016–2017)	1313	36.6 (30.4–43.2)	—	<LOD	<LOD	0.16 (0.15–0.17)	0.25[E] (0.14–0.36)
3–5 years							
5 (2016–2017)	531	79.1 (71.2–85.3)	0.28 (0.23–0.35)	<LOD	0.28 (0.21–0.35)	1.4[E] (0.53–2.2)	1.8[E] (0.97–2.7)
6–11 years							
5 (2016–2017)	520	68.3 (62.5–73.6)	0.11 (0.093–0.13)	<LOD	0.11 (0.082–0.13)	0.46[E] (0.28–0.63)	0.69 (0.46–0.93)
12–19 years							
5 (2016–2017)	519	41.8 (36.3–47.5)	—	<LOD	<LOD	0.13 (0.091–0.17)	0.20[E] (0.074–0.33)
20–39 years							
5 (2016–2017)	359	36.8 (29.6–44.6)	—	<LOD	<LOD	0.13 (0.092–0.16)	0.16 (0.11–0.21)
40–59 years							
5 (2016–2017)	352	29.4 (22.0–38.1)	—	<LOD	<LOD	0.098[E] (<LOD–0.14)	F
60–79 years							
5 (2016–2017)	345	27.6 (19.1–38.1)	—	<LOD	<LOD	0.10 (0.070–0.13)	0.14[E] (0.084–0.19)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.2.5

Cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (OH-MINCH) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2634	42.4 (36.3–48.8)	—	<LOD	<LOD	0.41 (0.31–0.51)	0.78 (0.57–0.99)
Males, 3–79 years							
5 (2016–2017)	1312	41.1 (31.8–51.1)	—	<LOD	<LOD	0.42 (0.29–0.55)	0.71[E] (0.42–0.99)
Females, 3–79 years							
5 (2016–2017)	1322	43.8 (37.0–50.8)	—	<LOD	<LOD	0.38 (0.26–0.50)	0.80 (0.51–1.1)
3–5 years							
5 (2016–2017)	533	80.2 (73.6–85.5)	0.30 (0.24–0.38)	<LOD	0.33 (0.22–0.44)	1.5[E] (0.94–2.1)	2.4[E] (1.5–3.3)
6–11 years							
5 (2016–2017)	519	66.6 (58.1–74.1)	0.17 (0.14–0.21)	<LOD	0.19 (0.14–0.24)	0.91 (0.73–1.1)	1.2 (0.81–1.7)
12–19 years							
5 (2016–2017)	517	45.3 (37.7–53.2)	—	<LOD	<LOD	0.39 (0.26–0.52)	0.88[E] (0.27–1.5)
20–39 years							
5 (2016–2017)	361	44.3 (32.0–57.3)	—	<LOD	<LOD	F	0.58[E] (0.31–0.85)
40–59 years							
5 (2016–2017)	355	34.9 (25.7–45.3)	—	<LOD	<LOD	F	F
60–79 years							
5 (2016–2017)	349	35.7 (26.5–46.0)	—	<LOD	<LOD	0.24[E] (0.091–0.38)	0.38[E] (0.18–0.58)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.078 µg/L.

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 are too unreliable to be published.

Table 14.2.6

Cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (OH-MINCH) 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2605	42.4 (36.3–48.8)	—	<LOD	<LOD	0.35 (0.27–0.43)	0.59 (0.46–0.71)
Males, 3–79 years							
5 (2016–2017)	1297	41.1 (31.8–51.1)	—	<LOD	<LOD	0.32 (0.22–0.42)	0.65 (0.46–0.85)
Females, 3–79 years							
5 (2016–2017)	1308	43.8 (37.0–50.8)	—	<LOD	<LOD	0.36 (0.27–0.44)	0.54[E] (0.33–0.75)
3–5 years							
5 (2016–2017)	523	80.2 (73.6–85.5)	0.53 (0.43–0.66)	<LOD	0.58 (0.40–0.75)	2.8[E] (1.2–4.4)	3.4 (2.4–4.5)
6–11 years							
5 (2016–2017)	512	66.6 (58.1–74.1)	0.20 (0.16–0.26)	<LOD	0.21 (0.16–0.27)	0.86[E] (0.48–1.2)	1.7[E] (0.73–2.7)
12–19 years							
5 (2016–2017)	511	45.3 (37.7–53.2)	—	<LOD	<LOD	0.23[E] (0.12–0.33)	0.54[E] (0.23–0.85)
20–39 years							
5 (2016–2017)	357	44.3 (32.0–57.3)	—	<LOD	<LOD	0.24[E] (<LOD–0.37)	0.42[E] (0.24–0.60)
40–59 years							
5 (2016–2017)	354	34.9 (25.7–45.3)	—	<LOD	<LOD	0.20[E] (<LOD–0.31)	F
60–79 years							
5 (2016–2017)	348	35.7 (26.5–46.0)	—	<LOD	<LOD	0.24 (0.18–0.30)	0.29 (0.21–0.36)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.2.7

cis-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2681	25.7 (21.7–30.1)	—	<LOD	<LOD	0.12 (0.099–0.14)	0.24 (0.18–0.31)
Males, 3–79 years							
5 (2016–2017)	1335	25.3 (19.3–32.5)	—	<LOD	<LOD	0.12 (0.084–0.15)	0.24[E] (0.15–0.33)
Females, 3–79 years							
5 (2016–2017)	1346	26.0 (20.9–31.8)	—	<LOD	<LOD	0.12 (0.091–0.15)	0.25[E] (0.15–0.34)
3–5 years							
5 (2016–2017)	545	74.8 (66.9–81.3)	0.12 (0.10–0.15)	<LOD	0.11 (0.074–0.15)	0.54 (0.39–0.68)	0.94[E] (0.51–1.4)
6–11 years							
5 (2016–2017)	530	53.8 (48.3–59.2)	—	<LOD	0.066 (<LOD–0.080)	0.28 (0.22–0.34)	0.49[E] (0.30–0.68)
12–19 years							
5 (2016–2017)	534	29.4 (23.2–36.5)	—	<LOD	<LOD	0.14 (0.095–0.18)	F
20–39 years							
5 (2016–2017)	368	27.1 (20.2–35.4)	—	<LOD	<LOD	0.11 (0.077–0.14)	0.14 (0.099–0.18)
40–59 years							
5 (2016–2017)	356	17.0[E] (11.5–24.4)	—	<LOD	<LOD	0.099[E] (<LOD–0.17)	0.27[E] (0.11–0.43)
60–79 years							
5 (2016–2017)	348	17.2[E] (10.0–27.9)	—	<LOD	<LOD	0.082[E] (<LOD–0.12)	0.11 (0.075–0.15)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.059 µg/L.

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 are too unreliable to be published.

Table 14.2.8

cis-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2652	25.7 (21.7–30.1)	—	<LOD	<LOD	0.13 (0.11–0.14)	0.21 (0.17–0.25)
Males, 3–79 years							
5 (2016–2017)	1321	25.3 (19.3–32.5)	—	<LOD	<LOD	0.12 (0.091–0.14)	0.20 (0.14–0.27)
Females, 3–79 years							
5 (2016–2017)	1331	26.0 (20.9–31.8)	—	<LOD	<LOD	0.13 (0.12–0.15)	0.22 (0.15–0.29)
3–5 years							
5 (2016–2017)	536	74.8 (66.9–81.3)	0.22 (0.18–0.25)	<LOD	0.19 (0.15–0.24)	0.84[E] (0.33–1.3)	F
6–11 years							
5 (2016–2017)	523	53.8 (48.3–59.2)	—	<LOD	0.080 (<LOD–0.092)	0.28 (0.20–0.36)	0.43 (0.31–0.55)
12–19 years							
5 (2016–2017)	527	29.4 (23.2–36.5)	—	<LOD	<LOD	0.11 (0.073–0.14)	F
20–39 years							
5 (2016–2017)	364	27.1 (20.2–35.4)	—	<LOD	<LOD	0.094 (0.064–0.13)	0.14 (0.099–0.18)
40–59 years							
5 (2016–2017)	355	17.0[E] (11.5–24.4)	—	<LOD	<LOD	0.11 (<LOD–0.13)	F
60–79 years							
5 (2016–2017)	347	17.2[E] (10.0–27.9)	—	<LOD	<LOD	0.11 (<LOD–0.13)	0.14 (0.12–0.17)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.2.9

trans-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2700	2.6 (1.9–3.6)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1345	2.2[E] (1.4–3.6)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1355	3.0[E] (1.9–4.8)	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	549	20.1 (14.1–27.7)	—	<LOD	<LOD	0.56[E] (<LOD–0.80)	0.79 (0.58–1.0)
6–11 years							
5 (2016–2017)	535	6.3[E] (3.9–9.9)	—	<LOD	<LOD	<LOD	0.37[E] (<LOD–0.53)
12–19 years							
5 (2016–2017)	537	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	372	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	357	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	350	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.33 µg/L.

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 are too unreliable to be published.

Table 14.2.10

trans-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2670	2.6 (1.9–3.6)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1330	2.2[E] (1.4–3.6)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1340	3.0[E] (1.9–4.8)	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	539	20.1 (14.1–27.7)	—	<LOD	<LOD	0.92 (<LOD–1.1)	1.3[E] (0.53–2.1)
6–11 years							
5 (2016–2017)	528	6.3[E] (3.9–9.9)	—	<LOD	<LOD	<LOD	0.65 (<LOD–0.81)
12–19 years							
5 (2016–2017)	530	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	368	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	356	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	349	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.2.11

Cyclohexane-1,2-dicarboxylic acid (CHDA) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2553	24.6 (21.0–28.7)	—	<LOD	<LOD	0.64 (0.51–0.76)	1.3[E] (0.79–1.8)
Males, 3–79 years							
5 (2016–2017)	1288	23.4 (18.4–29.3)	—	<LOD	<LOD	0.61 (0.49–0.73)	1.1[E] (0.53–1.7)
Females, 3–79 years							
5 (2016–2017)	1265	25.8 (21.8–30.2)	—	<LOD	<LOD	0.80[E] (0.49–1.1)	1.4[E] (0.53–2.2)
3–5 years							
5 (2016–2017)	485	68.4 (58.3–77.1)	0.66 (0.50–0.86)	<LOD	0.69 (0.48–0.89)	3.3[E] (1.6–5.0)	5.0 (3.4–6.7)
6–11 years							
5 (2016–2017)	505	54.7 (46.4–62.8)	—	<LOD	<LOD	1.6 (1.2–2.0)	2.4 (1.7–3.0)
12–19 years							
5 (2016–2017)	511	30.4 (22.3–40.0)	—	<LOD	<LOD	0.83 (0.59–1.1)	1.4[E] (0.38–2.3)
20–39 years							
5 (2016–2017)	363	22.4 (16.9–29.0)	—	<LOD	<LOD	0.47 (0.30–0.64)	0.62 (0.41–0.84)
40–59 years							
5 (2016–2017)	347	18.9[E] (12.4–27.7)	—	<LOD	<LOD	0.56[E] (<LOD–0.93)	F
60–79 years							
5 (2016–2017)	342	16.9[E] (10.9–25.3)	—	<LOD	<LOD	0.43[E] (<LOD–0.61)	0.61 (0.46–0.76)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.30 µg/L.

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 are too unreliable to be published.

Table 14.2.12

Cyclohexane-1,2-dicarboxylic acid (CHDA) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2526	24.6 (21.0–28.7)	—	<LOD	<LOD	0.77 (0.71–0.83)	1.2 (0.98–1.5)
Males, 3–79 years							
5 (2016–2017)	1273	23.4 (18.4–29.3)	—	<LOD	<LOD	0.63 (0.46–0.80)	1.2 (0.90–1.5)
Females, 3–79 years							
5 (2016–2017)	1253	25.8 (21.8–30.2)	—	<LOD	<LOD	0.82 (0.67–0.98)	1.3 (0.87–1.6)
3–5 years							
5 (2016–2017)	477	68.4 (58.3–77.1)	1.1 (0.87–1.5)	<LOD	1.1 (0.70–1.5)	5.0[E] (2.8–7.2)	7.1 (5.8–8.5)
6–11 years							
5 (2016–2017)	499	54.7 (46.4–62.8)	—	<LOD	<LOD	1.7 (1.1–2.3)	F
12–19 years							
5 (2016–2017)	504	30.4 (22.3–40.0)	—	<LOD	<LOD	0.62 (0.47–0.77)	0.80[E] (0.39–1.2)
20–39 years							
5 (2016–2017)	359	22.4 (16.9–29.0)	—	<LOD	<LOD	0.48[E] (0.16–0.81)	0.80 (0.63–0.97)
40–59 years							
5 (2016–2017)	346	18.9[E] (12.4–27.7)	—	<LOD	<LOD	0.59[E] (<LOD–0.82)	0.97[E] (0.58–1.4)
60–79 years							
5 (2016–2017)	341	16.9[E] (10.9–25.3)	—	<LOD	<LOD	0.59 (<LOD–0.70)	0.77 (0.67–0.87)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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14.3 2,2,4-TRIMETHYL-1,3-PENTANEDIOL DIISOBUTYRATE (TXIB)

2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB) (CASRN 6846-50-0) is an ester compound with the appearance of a clear liquid at room temperature. This substance may also be referred to as propanoic acid, 2-methyl-, 2,2-dimethyl-1-(1-methylethyl)-1,3-propanediyl ester, among other synonyms. It is produced commercially, and is commonly synthesized by the esterification of isobutyraldehyde with 2,2,4-trimethyl-1,3-pentanediol (Törmäkangas and Koskinen, 2001). TXIB is a secondary plasticizer, used in combination with other plasticizers, and is used in products like weather stripping, furniture, wallpaper, vinyl flooring, sporting goods, traffic cones, vinyl gloves, inks, water-based paints, and toys (CIR, 2017).

TXIB does not occur naturally. It is only released to the environment from anthropogenic sources. TXIB has moderate volatility and water solubility; therefore, it can be expected to occur in air and water, although it will likely volatilize from water surfaces (CIR, 2017). Given that TXIB can leach out of the polymer matrix when used as a plasticizer, the general population may be exposed to it dermally from the use of products available to consumers such as cosmetics. Exposure may also occur through the inhalation of indoor air or dust.

The toxicokinetics of TXIB in humans are not well studied. Experimental animal studies have reported a high level of absorption of TXIB following ingestion; however, no dermal absorption data were identified. Animal studies indicate that TXIB is not significantly distributed throughout the body following absorption. In animals exposed by ingestion, TXIB was found to undergo metabolic hydrolysis to the monoisobutyrate of 2,2,4-trimethyl-1,3-pentanediol (TMPD). Total excretion of TXIB following metabolism represented 95% to 99% of the administered dose. It is mainly excreted in urine within 72 hours; a smaller fraction is eliminated in feces over approximately one week (CIR, 2017). Animal studies report that TXIB is predominantly excreted as the *O*-glucuronide of TMPD in urine, and to a much lesser extent as 2,2,4-trimethyl-3-hydroxyvaleric acid (HTMV) and its glucuronides, 2-methylmalonic acid, and unchanged TXIB (CIR, 2017). In feces, TXIB is excreted unchanged and as

the metabolite TMPD and its monoester (CIR, 2017; ECHA, 2018).

Experimental animal studies have reported that TXIB has low toxicity following acute ingestion exposure (producing moderate weakness and some vasodilation), while chronic ingestion of this substance has been associated with increased liver and kidney weights (CIR, 2017; OECD, 1995). Histopathological investigation of chronically exposed animals revealed necrosis of the proximal tubules, dilatation of distal tubules, fibrosis in the kidneys, and centrilobular swelling of hepatocytes in the liver (CIR, 2017; OECD, 1995). TXIB has been associated with reproductive toxicity in animal studies on the basis of a reduction in the number of implantation sites in females (ECHA, 2001). TXIB is not considered genotoxic and is not expected to be carcinogenic (OECD, 1995).

A Chemicals Management Plan screening-level risk assessment is currently underway to determine whether TXIB presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (Canada, 1999; Environment and Climate Change Canada, 2019). TXIB is also found in cosmetic products notified under the Cosmetic Regulations of the *Food and Drugs Act* (Canada, 1985).

Two metabolites of TXIB (TMPD and HTMV) were analyzed in the urine of Canadian Health Measures Survey (CHMS) participants aged 3–79 years in cycle 5 (2016–2017). Data from the metabolites are presented as µg/L and µg/g creatinine. Finding a measurable amount of these metabolites in urine can be an indicator of recent exposure to TXIB and does not necessarily mean that an adverse effect will occur.

Table 14.3.1

2,2,4-Trimethyl-1,3-pentanediol (TMPD) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2589	98.5 (96.7–99.3)	17 (15–19)	4.4 (4.0–4.8)	16 (14–18)	69 (48–89)	150[E] (84–220)
Males, 3–79 years							
5 (2016–2017)	1295	98.0 (94.4–99.3)	18 (15–22)	4.4 (3.8–5.0)	17 (14–20)	78[E] (47–110)	F
Females, 3–79 years							
5 (2016–2017)	1294	99.0 (97.9–99.5)	15 (14–17)	4.4 (3.5–5.2)	15 (12–17)	57 (40–75)	100[E] (32–170)
3–5 years							
5 (2016–2017)	514	100	25[E] (17–36)	7.3 (5.4–9.2)	22[E] (12–32)	87 (59–120)	140 (100–180)
6–11 years							
5 (2016–2017)	518	99.6 (98.8–99.8)	23 (19–29)	6.4 (5.0–7.8)	23 (17–28)	74 (57–91)	F
12–19 years							
5 (2016–2017)	513	99.9 (99.4–100)	21 (18–25)	6.8 (4.9–8.8)	18 (14–22)	91[E] (54–130)	130[E] (78–180)
20–39 years							
5 (2016–2017)	365	99.0 (94.2–99.8)	18 (14–23)	4.3[E] (1.8–6.8)	17 (13–20)	F	F
40–59 years							
5 (2016–2017)	345	97.1 (88.0–99.3)	15 (11–19)	4.4 (3.2–5.6)	14 (10–18)	54[E] (34–75)	F
60–79 years							
5 (2016–2017)	334	98.6 (94.1–99.7)	14 (11–18)	3.7 (2.8–4.6)	14 (11–16)	52[E] (27–78)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 1.7 µg/L.

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 are too unreliable to be published.

Table 14.3.2

2,2,4-Trimethyl-1,3-pentanediol (TMPD) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2562	98.5 (96.7–99.3)	17 (15–19)	5.0 (4.1–6.0)	15 (12–17)	62 (49–75)	92 (65–120)
Males, 3–79 years							
5 (2016–2017)	1281	98.0 (94.4–99.3)	16 (14–19)	5.0 (3.8–6.2)	13 (10–16)	66 (42–90)	F
Females, 3–79 years							
5 (2016–2017)	1281	99.0 (97.9–99.5)	17 (15–20)	5.1 (3.9–6.2)	16 (13–18)	58 (45–71)	86 (69–100)
3–5 years							
5 (2016–2017)	506	100	42 (32–55)	14 (10–18)	37[E] (23–52)	140[E] (81–190)	240[E] (150–330)
6–11 years							
5 (2016–2017)	512	99.6 (98.8–99.8)	28 (23–33)	9.4 (8.0–11)	26 (21–31)	74 (56–92)	F
12–19 years							
5 (2016–2017)	506	99.9 (99.4–100)	16 (13–20)	5.4 (3.9–6.8)	14 (11–18)	53 (40–66)	77 (50–100)
20–39 years							
5 (2016–2017)	361	99.0 (94.2–99.8)	17 (14–20)	5.3 (4.1–6.6)	14 (11–17)	54[E] (28–81)	F
40–59 years							
5 (2016–2017)	344	97.1 (88.0–99.3)	14 (12–16)	4.4 (2.9–6.0)	12 (10–14)	51 (35–67)	73 (51–96)
60–79 years							
5 (2016–2017)	333	98.6 (94.1–99.7)	17 (13–20)	4.2 (3.1–5.2)	15 (12–18)	59[E] (29–88)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.3.3

2,2,4-Trimethyl-3-hydroxy valeric acid (HTMV) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2587	96.7 (94.2–98.2)	3.3 (2.8–3.8)	0.80 (0.62–0.98)	3.2 (2.9–3.6)	13 (8.8–17)	28 (18–38)
Males, 3–79 years							
5 (2016–2017)	1297	95.7 (91.8–97.8)	3.3 (2.6–4.2)	0.74 (0.48–1.0)	3.1 (2.3–4.0)	17[E] (9.1–25)	32[E] (11–52)
Females, 3–79 years							
5 (2016–2017)	1290	97.7 (95.4–98.8)	3.2 (2.9–3.6)	0.93 (0.75–1.1)	3.3 (3.0–3.7)	11 (8.9–14)	22[E] (9.6–35)
3–5 years							
5 (2016–2017)	529	99.9 (99.1–100)	5.4 (3.8–7.7)	1.6 (1.1–2.0)	4.8 (3.1–6.4)	23 (15–32)	30 (21–40)
6–11 years							
5 (2016–2017)	518	99.4 (98.2–99.8)	5.0 (4.0–6.4)	1.5 (1.0–2.0)	4.6 (3.0–6.2)	17 (14–20)	F
12–19 years							
5 (2016–2017)	509	99.4 (97.8–99.8)	4.2 (3.6–4.9)	1.1 (0.97–1.3)	4.1 (3.3–5.0)	16[E] (5.0–27)	27[E] (11–43)
20–39 years							
5 (2016–2017)	356	97.3 (90.3–99.3)	3.5 (2.6–4.6)	0.81[E] (<LOD–1.2)	3.3 (2.3–4.3)	F	F
40–59 years							
5 (2016–2017)	341	95.3 (87.2–98.4)	2.8 (2.2–3.6)	0.64[E] (<LOD–1.0)	2.7 (2.2–3.3)	11[E] (5.9–16)	20[E] (7.7–32)
60–79 years							
5 (2016–2017)	334	95.1 (89.5–97.8)	2.7 (2.2–3.3)	0.64 (0.50–0.78)	2.6 (2.1–3.2)	11 (6.9–14)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.42 µg/L.

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 are too unreliable to be published.

Table 14.3.4

2,2,4-Trimethyl-3-hydroxy valeric acid (HTMV) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2563	96.7 (94.2–98.2)	3.3 (2.8–3.8)	0.98 (0.81–1.2)	2.9 (2.3–3.5)	11 (8.5–14)	19 (15–23)
Males, 3–79 years							
5 (2016–2017)	1284	95.7 (91.8–97.8)	3.0 (2.4–3.6)	0.86 (0.56–1.2)	2.4 (1.7–3.1)	11 (8.9–14)	22[E] (14–31)
Females, 3–79 years							
5 (2016–2017)	1279	97.7 (95.4–98.8)	3.6 (3.2–4.1)	1.2 (0.96–1.4)	3.4 (3.0–3.7)	11[E] (5.1–17)	19 (15–22)
3–5 years							
5 (2016–2017)	520	99.9 (99.1–100)	9.4 (7.4–12)	3.0 (2.0–4.0)	8.3 (5.4–11)	31[E] (19–44)	43[E] (25–62)
6–11 years							
5 (2016–2017)	512	99.4 (98.2–99.8)	5.9 (4.7–7.3)	2.0 (1.5–2.4)	5.7 (4.3–7.0)	17 (13–22)	29[E] (<LOD–49)
12–19 years							
5 (2016–2017)	505	99.4 (97.8–99.8)	3.2 (2.6–4.0)	1.1 (0.73–1.4)	2.9 (2.1–3.7)	9.7 (7.7–12)	14[E] (4.0–23)
20–39 years							
5 (2016–2017)	353	97.3 (90.3–99.3)	3.2 (2.7–3.9)	1.2 (<LOD–1.4)	2.7 (1.9–3.5)	11 (7.1–15)	F
40–59 years							
5 (2016–2017)	340	95.3 (87.2–98.4)	2.6 (2.2–3.2)	0.84 (<LOD–1.0)	2.2 (1.6–2.9)	8.4[E] (4.9–12)	14[E] (6.5–22)
60–79 years							
5 (2016–2017)	333	95.1 (89.5–97.8)	3.2 (2.7–3.7)	0.79[E] (0.46–1.1)	3.1 (2.5–3.8)	12[E] (7.1–18)	21 (<LOD–28)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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14.4 TRI-(2-ETHYLHEXYL) TRIMELLITATE (TEHT)

Tri-(2-ethylhexyl) trimellitate (TEHT) (CASRN 3319-31-1) is an ester compound with the appearance of a yellow oily liquid at room temperature. This substance may also be referred to as trioctyl trimellitate (TOTM) or triethylhexyl trimellitate, among other synonyms. It is most commonly manufactured through the esterification of trimellitic anhydride with 2-ethylhexyl alcohol (CIR, 2015). TEHT is primarily used as a plasticizer in floor coverings, building and construction materials, plastic and rubber materials, medical devices, and in cosmetics as an emollient and skin-conditioning agent. It may also be used as a fuel additive, in adhesives and sealants used in the transportation sector, and as a lubricant and lubricant additive (CIR, 2015; Environment and Climate Change Canada and Health Canada, 2019).

TEHT does not occur naturally and is only released to the environment from anthropogenic sources (Environment and Climate Change Canada and Health Canada, 2019). TEHT has very low volatility and water solubility, and is therefore expected to minimally occur in air and water (Environment and Climate Change Canada and Health Canada, 2019). The general population may be exposed to TEHT dermally from the use of products available to consumers, including cosmetics, and through the ingestion of dust (CIR, 2015; Environment and Climate Change Canada and Health Canada, 2019). Since only low levels leach out of the polymer matrix when TEHT is used as a plasticizer, exposure via the use of consumer products is expected to be low (SCENIHR, 2016). The mouthing of plastic toys is not expected to result in exposure to TEHT (Environment and Climate Change Canada and Health Canada, 2019). Given the very low volatility of TEHT, exposure via inhalation is of minimal concern (Environment and Climate Change Canada and Health Canada, 2019).

The toxicokinetics of TEHT in humans are not well studied. Experimental animal studies have shown that the absorption of TEHT following ingestion or dermal exposure is low (<1%). TEHT was mainly distributed to the liver, lungs, and spleen in animals following an intravenous dose. In animals exposed through ingestion, TEHT was found to undergo hydrolysis, and its elimination was biphasic, with half-lives of 3.1 and 42 hours in urine, and 4.3 and 31 hours in expired

CO_2 (CIR, 2015). TEHT is mostly excreted in feces; a small fraction is excreted in urine. An animal study reported that orally administered TEHT was excreted in feces predominantly in an unchanged form and to a much lesser extent as mono-(2-ethylhexyl) trimellitate, di-(2-ethylhexyl) trimellitate, and unidentified polar metabolites; in urine, the metabolites were identified as mono-(2-ethylhexyl) trimellitate, 2-ethylhexanol, 2-ethylhexanoic acid, and 2-heptanone (CIR, 2015).

Experimental animal studies have reported that TEHT has low toxicity following acute ingestion exposure; chronic ingestion was found to result in enlarged liver and spleen (CIR, 2015; OECD, 2002). Animals acutely exposed to high concentrations of TEHT via inhalation showed lung irritation, but no other signs of toxicity (CIR, 2015; OECD, 2002). TEHT has been associated with male reproductive toxicity based on reduced testes weight and decreased numbers of spermatocytes and spermatids in laboratory animals (Environment and Climate Change Canada and Health Canada, 2019; OECD, 2002). TEHT is not considered genotoxic and is not expected to be carcinogenic (Environment and Climate Change Canada and Health Canada, 2019).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether TEHT presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2019). The assessment concluded that TEHT does not meet any of the criteria for being considered toxic under CEPA 1999. TEHT is found in cosmetic products notified under the Cosmetic Regulations of the *Food and Drugs Act* (Canada, 1985).

Three metabolites of TEHT — 1-mono*(*2-ethylhexyl) trimellitate (1-MEHTM), 2-mono*(*2-ethylhexyl) trimellitate (2-MEHTM), and 4-mono*(*2-ethylhexyl) trimellitate (4-MEHTM) — were analyzed in the urine of Canadian Health Measures Survey participants aged 3–79 years in cycle 5 (2016–2017). Data for the metabolites are presented as both µg/L and µg/g creatinine. Finding a measurable amount of these metabolites in urine can be an indicator of recent exposure to TEHT and does not necessarily mean that an adverse effect will occur.

Table 14.4.1

1-Mono(2-ethylhexyl) trimellitate (1-MEHTM) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2701	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1347	0	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1354	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	551	0	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	535	0	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	537	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	371	0	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	357	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	350	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.22 µg/L.

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

F Data are too unreliable to be published.

Table 14.4.2

1-Mono(2-ethylhexyl) trimellitate (1-MEHTM) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2671	F	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1332	0	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1339	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	541	0	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	528	0	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	530	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	367	0	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	356	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	349	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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

F Data are too unreliable to be published.

Table 14.4.3

2-Mono(2-ethylhexyl) trimellitate (2-MEHTM) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2637	7.1[E] (4.6–11.0)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1314	6.3[E] (3.6–10.9)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1323	7.9[E] (4.7–12.9)	—	<LOD	<LOD	<LOD	0.25[E] (<LOD–0.42)
3–5 years							
5 (2016–2017)	538	3.9[E] (2.0–7.3)	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	525	6.0[E] (3.0–11.5)	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	526	5.2[E] (2.9–9.0)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	364	F	—	<LOD	<LOD	<LOD	0.21 (<LOD–0.28)
40–59 years							
5 (2016–2017)	347	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	337	9.9[E] (5.3–17.9)	—	<LOD	<LOD	<LOD	0.53[E] (0.31–0.75)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.16 µg/L.

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 are too unreliable to be published.

Table 14.4.4

2-Mono(2-ethylhexyl) trimellitate (2-MEHTM) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2607	7.1[E] (4.6–11.0)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1299	6.3[E] (3.6–10.9)	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1308	7.9[E] (4.7–12.9)	—	<LOD	<LOD	<LOD	0.46 (<LOD–0.61)
3–5 years							
5 (2016–2017)	528	3.9[E] (2.0–7.3)	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	518	6.0[E] (3.0–11.5)	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	519	5.2[E] (2.9–9.0)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	360	F	—	<LOD	<LOD	<LOD	0.39 (<LOD–0.48)
40–59 years							
5 (2016–2017)	346	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	336	9.9[E] (5.3–17.9)	—	<LOD	<LOD	<LOD	0.49 (0.34–0.64)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

Table 14.4.5

4-Mono(2-ethylhexyl) trimellitate (4-MEHTM) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2309	2.0[E] (1.0–4.1)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1144	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1165	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	475	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	417	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	450	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	337	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	308	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	322	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.098 µg/L.

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 are too unreliable to be published.

Table 14.4.6

4-Mono(2-ethylhexyl) trimellitate (4-MEHTM) (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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	2279	2.0[E] (1.0–4.1)	—	<LOD	<LOD	<LOD	<LOD
Males, 3–79 years							
5 (2016–2017)	1129	F	—	<LOD	<LOD	<LOD	<LOD
Females, 3–79 years							
5 (2016–2017)	1150	F	—	<LOD	<LOD	<LOD	<LOD
3–5 years							
5 (2016–2017)	465	F	—	<LOD	<LOD	<LOD	<LOD
6–11 years							
5 (2016–2017)	410	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	443	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	333	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	307	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	321	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

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 are too unreliable to be published.

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SUMMARIES AND RESULTS FOR VOLATILE ORGANIC COMPOUNDS 15

15.1 BENZENE

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. Today, it 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 most common route of exposure to benzene for the general population is inhalation; exposure is attributed predominantly to indoor air because indoor levels of benzene generally exceed those outdoors (Health Canada, 2010a; Health Canada 2010b; Health Canada, 2012; Health Canada, 2013a), and because people typically spend more time indoors than outdoors (Health Canada, 2013b). Exposure to benzene in 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; Mallach et al., 2016). Various marketplace products containing benzene can also contribute to its presence in indoor air including stored combustion equipment, air fresheners, incense, candles, mothballs, building materials and cleaning products (Won et al., 2013; Won et al. 2014; Won et al., 2015; Won and Yang, 2012; Environment Canada and Health Canada, 1993). Outdoor benzene exposure sources include motor vehicle exhaust, gasoline service stations, and gasoline storage facilities (ATSDR, 2007). Foods, beverages, and tap water are not major sources of benzene exposure for the general population (ATSDR, 2007; Health Canada, 2009).

Following inhalation, benzene is readily absorbed into the blood and distributed throughout the

body, concentrating primarily in adipose tissue and bone marrow (EPA, 2002; ATSDR 2007). Benzene metabolism occurs mainly in the liver, but also in other tissues such as bone marrow. It is metabolized into several reactive metabolites, including benzene oxide (Environment Canada and Health Canada, 1993; EPA, 2002; McHale et al., 2012). After initial formation of benzene oxide, metabolism can branch into several alternative pathways: spontaneous rearrangement 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 exposure. Exposure to benzene can be hematotoxic in humans and laboratory animals, with bone marrow being 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). Epidemiological 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). Benzene is listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 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 Canada, regulations have been put in place to limit the concentration of benzene in gasoline fuel (Canada, 1997; Environment Canada, 1998) and reduce emissions from on-road (Canada, 2003; Canada, 2015) and off-road (Canada, 2013; Canada, 2017) engines and vehicles. The Gasoline and Gasoline Blend Dispensing Flow Regulations, implemented in 2001, also limit emissions of benzene and other VOCs into the environment during the refuelling of on-road vehicles (Canada, 2000). In 2000–2001, the Canadian Council of Ministers of the Environment (CCME) endorsed the Canada-wide standard for benzene, requiring a reduction in total benzene emissions from industrial facilities and the 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 identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

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 proposed and implemented 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 end points and 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, 2013b). On the basis that there may be a low but non-negligible cancer risk at indoor exposure levels measured in Canadian homes in Health Canada studies, it is recommended that individuals take actions to reduce their exposure to benzene indoors as much as possible. In particular, exposure reduction strategies have been recommended that target 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), cycle 4 (2014–2105), and cycle 5 (2016–2017) participants aged 12–79 years. Data are presented as µg/L blood for benzene. Finding a measurable amount of benzene in blood can be an indicator of exposure to benzene and does not necessarily mean that an adverse health effect will occur.

Benzene metabolites, *t,t*-MA and *S*-PMA, were analyzed in the urine of CHMS cycle 2 (2009–2011), cycle 3 (2012–2013), and cycle 4 (2014–2015) participants aged 3–79 years (Health Canada, 2017).

Benzene was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Statistics Canada, 2013; Wheeler et al., 2013; Zhu et al., 2013), cycle 3 (2012–2013) (Statistics Canada, 2015), 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@canada.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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2488	88.4 (76.6–94.7)	0.035 (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	94.6 (89.1–97.4)	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)
5 (2016–2017)	2436	74.5 (58.9–85.6)	0.037 (0.028–0.047)	<LOD	0.035 (0.027–0.043)	0.15 (0.099–0.19)	0.20 (0.15–0.25)
Males, 12–79 years							
3 (2012–2013)	1245	89.0 (78.1–94.8)	0.036 (0.025–0.052)	<LOD	0.040 (0.030–0.049)	0.15 (0.13–0.18)	0.24 (0.18–0.30)
4 (2014–2015)	1164	95.2 (89.8–97.9)	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)
5 (2016–2017)	1216	74.8 (60.4–85.2)	0.041 (0.031–0.052)	<LOD	0.037 (0.026–0.047)	0.16 (0.14–0.19)	0.23 (0.16–0.31)
Females, 12–79 years							
3 (2012–2013)	1243	87.8 (73.5–94.9)	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	93.9 (87.5–97.1)	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)
5 (2016–2017)	1220	74.2 (56.5–86.5)	0.033 (0.025–0.043)	<LOD	0.034 (0.026–0.042)	0.096[E] (0.051–0.14)	0.19[E] (0.099–0.28)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
12–19 years							
3 (2012–2013)	750	86.0 (73.0–93.3)	0.028[E] (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	93.0 (83.6–97.2)	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)
5 (2016–2017)	790	74.4 (55.5–87.1)	0.032 (0.025–0.040)	<LOD	0.033 (0.025–0.041)	0.072 (0.055–0.088)	0.099 (0.085–0.11)
20–39 years							
3 (2012–2013)	548	90.2 (73.6–96.8)	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	96.5 (91.2–98.7)	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)
5 (2016–2017)	559	78.7 (61.9–89.3)	0.041 (0.032–0.053)	<LOD	0.038 (0.027–0.049)	0.17 (0.13–0.21)	0.20 (0.15–0.25)
40–59 years							
3 (2012–2013)	598	89.7 (79.1–95.2)	0.040 (0.029–0.055)	<LOD	0.039 (0.028–0.050)	0.23 (0.16–0.31)	0.40[E] (0.24–0.56)
4 (2014–2015)	575	93.9 (85.3–97.6)	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)
5 (2016–2017)	539	72.4 (55.1–84.9)	0.036 (0.026–0.051)	<LOD	0.035 (0.025–0.045)	0.15[E] (0.071–0.23)	0.23 (0.15–0.31)
60–79 years							
3 (2012–2013)	592	84.6 (69.6–93.0)	0.031[E] (0.020–0.047)	<LOD	0.038 (0.026–0.051)	0.13 (0.085–0.17)	0.20 (0.16–0.24)
4 (2014–2015)	548	93.4 (87.9–96.5)	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)
5 (2016–2017)	548	71.4 (54.4–84.0)	0.034 (0.025–0.045)	<LOD	0.032 (0.023–0.041)	0.12[E] (0.048–0.19)	0.22[E] (0.097–0.35)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.0070, 0.0070, and 0.022 µg/L, respectively.

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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15.2 CARBON TETRACHLORIDE

Carbon tetrachloride (CASRN 56-23-5), also known as tetrachloromethane, is a colourless, non-flammable, heavy liquid with a characteristic sweet, aromatic, non-irritating odour (IARC, 1999; Health Canada, 2010a). It is a haloalkane and is considered a volatile organic compound (VOC). Carbon tetrachloride is generally produced industrially either by chlorination of methane or monochloromethane, or by perchlorination or chlorinolysis (chlorination at pyrolytic temperatures) of low molecular-weight (C1–C3) hydrocarbons (e.g., methane) or other chlorinated hydrocarbons (e.g., methylene chloride) (ATSDR, 2005; Holbrook, 2000). Carbon tetrachloride is no longer manufactured in Canada, and the quantity of carbon tetrachloride imported into Canada has generally been declining, though at a much more gradual rate since 2006 (Statistics Canada, 2018).

Currently, carbon tetrachloride is only permitted to be imported into Canada for limited use as a feedstock/intermediate in chemical synthesis (see below). In the past, it was mainly used as a feedstock in the production of chlorofluorocarbons (e.g., for refrigerant use), but was also used in the 20th century in industrial and domestic degreasers, fire extinguishers, dry-cleaning agents, and grain fumigants. Other past uses include as a solvent for oils, fats, lacquers, varnishes, rubber waxes, and resins, and as an ingredient in pharmaceutical products (ATSDR, 2005; Health Canada, 2010a).

Carbon tetrachloride is not known to occur naturally. Most atmospheric carbon tetrachloride is a result of direct releases to the atmosphere; however, it can also form in the troposphere from photochemical reactions with chlorinated alkenes (ATSDR, 2005; Health Canada, 2010a). Carbon tetrachloride disperses rapidly in air due to its very high volatility and persists in air due to its very slow atmospheric degradation rate, with an estimated lifetime of 30 to 100 years (NTP, 2016). Indoor air may contain higher concentrations of carbon tetrachloride as a result of volatilization from contaminated drinking water and/or from the use of discontinued household products containing carbon tetrachloride (ATSDR, 2005). Carbon tetrachloride concentrations measured in Canadian homes were found to be similar in indoor and outdoor air in studies conducted by Health Canada over the last decade (Health Canada, 2013; Health Canada 2012; Health Canada, 2010b; Health Canada, 2010c). Limited evidence suggests that the use of chlorine bleach may contribute to the presence of carbon tetrachloride in indoor air (NTP, 2016; Odabasi, 2008). In some areas where historical contamination has occurred, drinking water may be contaminated with carbon tetrachloride; concentrations of carbon tetrachloride in groundwater sources are expected to be higher than in surface water due to the limited potential for volatilization and biodegradation in groundwater systems (Health Canada, 2010a).

Canadians may be exposed to carbon tetrachloride from its continued presence in the environment from historical releases, from permitted industrial processes, or from the use of old or discontinued household products (ATSDR, 2005; Health Canada, 2010a). While inhalation of ambient and indoor air is the primary route of exposure for the general population, oral exposure may also occur from ingestion of contaminated drinking water, and dermal exposure may occur during bathing or showering (ATSDR, 2005; Health Canada, 2010a). Exposure of Canadians to carbon tetrachloride from foods is not expected for a number of reasons: it is no longer used for grain fumigation in Canada (and its use as such in other countries is limited); the Canadian Pest Management Regulatory Agency has prohibited its use as a formulant in pest control products in Canada; and it is not frequently detected in foods (ATSDR, 2005; FDA, 2006; Health Canada, 2006; Health Canada, 2010a).

Carbon tetrachloride is rapidly absorbed orally and by inhalation, and to a lesser extent dermally. Based on animal studies, a small amount of carbon tetrachloride is expired in breath directly following absorption, with the remainder entering systemic circulation and being distributed to all major organs. The highest concentrations are found in fat and in organs or tissues with high fat content, such as liver, kidney, brain, and bone marrow, as well as in the lungs and adrenals (ATSDR, 2005; Health Canada, 2010a). Carbon tetrachloride can transiently accumulate in fatty/adipose tissues, where it is slowly released back into the blood stream (CDC, 2009; OECD, 2011). It may undergo hepatic metabolism via cytochrome P450 oxygenases. Carbon tetrachloride is excreted primarily in exhaled air and in the feces, and in lower amounts in the urine; it is eliminated in exhaled breath primarily as the unchanged parent compound and to a lesser extent as CO2 or chloroform. It is eliminated in feces and urine as urea and other metabolites (ATSDR, 2005).

Acute inhalation or oral exposures to carbon tetrachloride at high dose levels in humans have been associated with neurological effects involving central nervous system depression with symptoms such as headaches, dizziness, and weakness, and in severe cases tremor, blurred vision, drowsiness, seizures, loss of consciousness, and mortality from suppression of respiratory centres (ATSDR 2005). A number of other adverse health effects in humans have been associated with acute inhalation or oral exposures, such as decreased serum iron, gastrointestinal irritation, nausea, proteinuria, increased hepatic bilirubin, and liver necrosis (Health Canada, 2010a). Hepatotoxicity is the major chronic effect of exposure to carbon tetrachloride by any route in humans, because of the higher sensitivity of the liver due to the abundance of CYP2E1 enzymes and other cytochromes that have been shown to activate carbon tetrachloride into reactive metabolites (ATSDR 2005). Symptoms of liver injury in humans include jaundice and swollen or tender liver. Based on animal studies, chronic liver effects may include steatosis, fibrosis, cirrhosis, and necrosis (ATSDR 2005). The kidney is another sensitive target organ; in humans, kidney injury is often observed at the same exposure levels as liver injury. A principal symptom in severe cases is reduced urinary output, which can lead to azotemia, hypertension, and pulmonary edema. Chronic animal studies have demonstrated kidney effects, including nephropathy and reduction in kidney enzyme activity.

Occupational studies have reported associations between exposure to halogenated solvents and reduced fertility and spontaneous abortions, although the extent of involvement of carbon tetrachloride in these effects is unclear (CDC, 2009). Animal studies have not consistently demonstrated reproductive toxicity in the absence of maternal toxicity (CDC, 2009). Carbon tetrachloride is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 1999).

Under the *Montreal Protocol on Substances that Deplete the Ozone Layer*, an international agreement reached in 1987, the production of chlorofluorocarbons was mandated to be phased out by 2030 (UNEP, 2019). To meet these commitments, the manufacture, import and export of carbon tetrachloride has been prohibited in Canada since 1995, except for its import as a feedstock in the synthesis of chlorofluorocarbons (CFCs), hydrochlorofluorocarbons (HCFCs), and hydrofluorocarbons (HFCs) (Environment and Climate Change Canada and Health Canada, 2016; Health Canada, 2010a).

Carbon tetrachloride is listed on Schedule 1, List of Toxic Substances, under the *Canadian Environmental Protection Act, 1999*, and is a risk-managed substance that entails a full life cycle management approach to prevent or minimize its release into the environment (Canada, 1999). Canada has developed regulations to manage the risks associated with carbon tetrachloride that control the export, import, manufacture, sale, and use of ozone-depleting substances as well as products containing or designed to contain them (Canada, 2003; Canada, 2016). Carbon tetrachloride is also identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

Carbon tetrachloride is also part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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 2017, Health Canada published an Indoor Air Reference Level (IARL) for carbon tetrachloride (Health Canada, 2017b).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has also developed a guideline for Canadian drinking water quality that establishes a maximum acceptable concentration (MAC) for carbon tetrachloride that is protective of human health and takes into consideration all exposures from drinking water (including ingestion as well as inhalation and dermal absorption during showering and bathing) (Health Canada, 2010a; Health Canada, 2017a).The sale and use of pesticides is regulated in Canada by PMRA under the *Pest Control Products Act (Canada, 2002).* PMRA has prohibited the use of carbon tetrachloride as a formulant in pest control products in Canada due to it being an ozone-depleting chemical (Health Canada, 2006).

Carbon tetrachloride was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of carbon tetrachloride in blood can be an indicator of recent exposure to carbon tetrachloride and does not necessarily mean that an adverse health effect will occur.

Carbon tetrachloride was also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013).

Table 15.2.1

Carbon tetrachloride — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2574	36.3 (28.7–44.6)	—	<LOD	<LOD	0.0071 (0.0060–0.0083)	0.0086 (0.0071–0.010)
Males, 12–79 years							
5 (2016–2017)	1281	40.3 (31.9–49.3)	—	<LOD	<LOD	0.0072 (0.0057–0.0086)	0.0089 (0.0063–0.012)
Females, 12–79 years							
5 (2016–2017)	1293	32.2 (24.2–41.5)	—	<LOD	<LOD	0.0071 (0.0059–0.0083)	0.0083 (0.0071–0.0095)
12–19 years							
5 (2016–2017)	834	34.7 (24.9–45.9)	—	<LOD	<LOD	0.0072 (0.0057–0.0087)	0.0088 (0.0065–0.011)
20–39 years							
5 (2016–2017)	591	38.4 (26.9–51.4)	—	<LOD	<LOD	0.0071 (0.0062–0.0080)	0.0080 (0.0070–0.0090)
40–59 years							
5 (2016–2017)	568	36.7 (25.9–49.1)	—	<LOD	<LOD	0.0079 (0.0053–0.010)	0.0093[E] (0.0052–0.013)
60–79 years							
5 (2016–2017)	581	33.2 (27.7–39.2)	—	<LOD	<LOD	0.0067 (0.0060–0.0075)	0.0082 (0.0073–0.0092)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.005 µg/L.

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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15.3 1,4-DICHLOROBENZENE

1,4-Dichlorobenzene (CASRN 106-46-7), also known as para-dichlorobenzene, is a solid ranging from colourless to white that sublimates at room temperature, producing a characteristic penetrating odour that smells like moth balls (ATSDR, 2006; IARC, 1999;). It is a halogenated aromatic hydrocarbon and is considered a volatile organic compound (VOC). It is a high-production volume industrial chemical typically produced by reacting liquid benzene with gaseous chlorine in the presence of a catalyst, followed by crystallization and distillation (ATSDR, 2006; Beck and Löser, 2012; EPA, 2018; IARC, 1999; OECD, 2018). 1,4-Dichlorobenzene has been manufactured in Canada and is also imported into the country, although the quantity of dichlorobenzenes (ortho, meta, and para isomers) imported into Canada has been decreasing since 1995 (Environment Canada and Health Canada, 1993; 2003; Statistics Canada, 2018).

Major uses of 1,4-dichlorobenzene include moth-control products (as an active ingredient in registered pesticides in Canada) and urinal and toilet rim blocks (i.e., room deodorants). It is also used as an intermediate in the production of polyphenylene sulfide resin and 1,2,4-trichlorobenzene (ATSDR, 2006; CAREX Canada, 2018; Environment Canada and Health Canada, 1993). It also may be used as a disinfectant, as an intermediate in the production of pigments and dyes, and as an ingredient in certain pharmaceuticals and resin-bonded abrasives (CAREX Canada, 2018).

Chlorinated benzenes are not known to occur naturally (ATSDR, 2006; IARC, 1999). Concentrations of 1,4-dichlorobenzene in indoor air may be significantly higher than in ambient air (ATSDR, 2006; Health Canada, 2010c; Health Canada 2010d; Health Canada, 2012; Health Canada, 2013; NTP, 2016). Sources of indoor air exposure include air fresheners, candles, furniture, and various building materials (Won et al., 2013; Won et al., 2014; Won and Lustyk, 2011; Won and Yang, 2012). Major sources of 1,4-dichlorobenzene in ambient air are volatilization during its consumer or commercial use, and emissions from municipal and industrial waste sites and incinerator facilities (ATSDR, 2006; IARC, 1999).

While inhalation of ambient and indoor air is the primary route of exposure for the general population, exposure may also occur from ingestion of foods and

drinking water. 1,4-Dichlorobenzene has been found in a variety of foods sampled in Canada, including soft drinks, butter, margarine, peanut butter, flour, pastry mixes, and cow's milk (IARC, 1999), as well as in the breast milk of Canadian women (Mes et al., 1986). The U.S. Food and Drug Administration Total Diet Study reported that 1,4-dichlorobenzene was identified in 33 different food items, but concluded that concentrations were generally low and exposures were less than from air (FDA, 2006; NTP, 2016). 1,4-Dichlorobenzene is the main dichlorobenzene isomer found in drinking water, probably resulting largely from its use in urinal/toilet blocks and releases or spills from industrial effluents (Health Canada, 2017a; IARC, 1999). 1,4-Dichlorobenzene has been detected at low concentrations in treated drinking water, including in Canadian samples, but this is thought to be a minor pathway for human exposure (ATSDR, 2006; Health Canada, 1987; Oliver and Nicol, 1982; Otson et al., 1982).

1,4-Dichlorobenzene is rapidly and almost completely absorbed orally and by inhalation, but not appreciably through skin contact (ATSDR, 2006; HSDB, 2008). Quantitative data on absorption kinetics by inhalation in animals and humans are not available; however, numerous human and animal studies detecting 1,4-dichlorobenzene or its metabolites in blood, urine, adipose tissue, and other peripheral tissues, as well as in breast milk, provide evidence of its absorption and distribution (ATSDR, 2006). Animal studies indicate that 1,4-dichlorobenzene temporarily accumulates in adipose tissue before being rapidly distributed to the rest of the body, with the highest levels found in fat, liver, and kidney (ATSDR, 2006). Animal studies also demonstrate that 1,4-dichlorobenzene primarily undergoes oxidative hepatic metabolism by epoxidation and hydrolysis followed by phase II metabolism to form sulphate and glucuronic conjugates of 2,5-dichlorophenol, which are excreted almost exclusively in urine, with small amounts eliminated through biliary excretion and negligible elimination in expired breath. These studies also indicate that elimination occurs in the form of metabolites, rather than as the parent compound, over a period of several days post-exposure (ATSDR, 2006; HSDB, 2008).

In humans, 1,4-dichlorobenzene can cause ocular, nasal, and respiratory irritation (OECD, 2003). Acute inhalation exposure in humans has been associated with symptoms of nausea, headache, and vomiting (ATSDR, 2006). Based on clinical information, central nervous system depression can result following inhalation of very high concentrations, while in severe cases, dizziness, headache, facial twitching, vomiting, weight loss, and cirrhosis can occur (HSDB, 2008). Chronic exposure in humans may result in hepatotoxicity with symptoms of jaundice, cirrhosis, and possible death. Chronic inhalation studies in animals have reported mortality as well as liver, kidney, and respiratory effects (HSDB, 2008; IARC, 1999; OECD, 2003). 1,4-Dichlorobenzene is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 1999).

The Government of Canada conducted a Priority Substances List (PSL) scientific assessment on the impact of 1,4-dichlorobenzene exposure on humans and the environment and concluded that it was not entering the environment in quantities or under conditions that would constitute a danger to the environment on which human life depends, or to human life or health (Environment Canada and Health Canada, 1993). Based upon available data at the time, there was insufficient information to conclude whether it was entering the environment in quantities or under conditions that may be harmful to the environment. A follow-up PSL report concluded that 1,4-dichlorobenzene was not entering the environment in a quantity or at a concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity; therefore, it is not considered toxic as defined in paragraph 64(a) of the *Canadian Environmental Protection Act, 1999* (Environment Canada and Health Canada, 2003).

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has also developed a maximum acceptable concentration (MAC) for 1,4-dichlorobenzene in Canadian drinking water that is protective of human health, as well as an aesthetic objective based on its odour threshold (Health Canada, 1987; Health Canada, 2017a). The guideline was developed based on the development of benign liver and adrenal gland tumours in animals (Health Canada, 1987; Health Canada, 2017a; NTP, 1986). In 2017, Health Canada published an Indoor Air Reference Level (IARL) for 1,4-dichlorobenzene (Health Canada, 2017b). 1,4-Dichlorobenzene is part of a larger class of

VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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).

The sale and use of 1,4-dichlorobenzene as a pesticide is regulated in Canada by Health Canada's Pest Management Regulatory Agency (PMRA) under the *Pest Control Products Act* (Canada, 2002). 1,4-Dichlorobenzene is an active ingredient in registered pest control products in Canada, namely in insecticides used to control moths and moth larvae (Health Canada, 2010b; Health Canada, 2019). PMRA evaluates the toxicity of pesticides and potential exposure to determine whether a pesticide should be registered for a specific use, and re-evaluates registered pesticides on a cyclical basis. In its most recent re-evaluation, PMRA determined that products containing 1,4-dichlorobenzene are acceptable for continued registration for sale and use in Canada, provided they are used according to label directions and specified risk-reduction measures are implemented (Health Canada, 2010b).

1,4-Dichlorobenzene was analyzed in whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of 1,4-dichlorobenzene in blood can be an indicator of recent exposure to 1,4-dichlorobenzene and does not necessarily mean that an adverse health effect will occur.

1,4-Dichlorobenzene 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 indoor air sampling 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@canada.ca.

Table 15.3.1

1,4-Dichlorobenzene — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2544	69.8 (61.2–77.2)	0.031 (0.024–0.040)	<LOD	0.024 (0.017–0.031)	0.24[E] (0.091–0.38)	0.76[E] (0.44–1.1)
Males, 12–79 years							
5 (2016–2017)	1261	71.0 (62.8–78.1)	0.034 (0.025–0.047)	<LOD	0.027 (0.021–0.033)	F	0.84[E] (0.29–1.4)
Females, 12–79 years							
5 (2016–2017)	1283	68.5 (58.6–77.0)	0.028 (0.021–0.039)	<LOD	0.022[E] (<LOD–0.031)	0.20[E] (0.12–0.28)	F
12–19 years							
5 (2016–2017)	827	65.9 (52.6–77.1)	0.028[E] (0.019–0.040)	<LOD	0.021 (0.014–0.027)	0.26[E] (0.082–0.44)	0.72[E] (0.26–1.2)
20–39 years							
5 (2016–2017)	582	72.6 (63.7–80.0)	0.033[E] (0.023–0.049)	<LOD	0.029[E] (0.018–0.040)	F	F
40–59 years							
5 (2016–2017)	562	68.0 (56.7–77.5)	0.029 (0.021–0.041)	<LOD	0.021 (0.015–0.028)	F	F
60–79 years							
5 (2016–2017)	573	70.1 (60.0–78.5)	0.033 (0.024–0.044)	<LOD	0.024 (0.019–0.030)	0.27[E] (0.097–0.44)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.013 µg/L.

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 are too unreliable to be published.

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15.4 2,5-DIMETHYLFURAN

2,5-Dimethylfuran (CASRN 625-86-5) is an alkylfuran with the appearance of a clear yellow oily liquid that has a pungent spicy, smoky, or ethereal/solvent-like odour (Burdock, 2010; Yang et al. 2016). It is produced commercially by acid-catalyzed dehydration of *D*-fructose from food material to 5-hydroxymethylfurfural, followed by consecutive hydrogenolysis over a copper-ruthenium catalyst (Lichtenhaler, 2012; Román-Leshkov et al., 2007). Advancements have been made in the biofuels sector to develop bulk scale processes to produce 5-hydroxymethylfurfural (and, thus, 2,5-dimethylfuran) from cellulosic biomass raw materials rather than food-based sources (Binder and Raines 2009; Lichtenhaler 2012).

2,5-Dimethylfuran is released to the environment—primarily to air—from natural and anthropogenic sources. Low levels of 2,5-dimethylfuran may be found in various foods, and it generally co-occurs with furan and other alkylfurans (e.g., 2-methylfuran) (Burdock, 2010; EFSA 2017). 2,5-Dimethylfuran is a Maillard product in foods (i.e., a product of heat-driven reactions between amino acids and sugars) and is formed by the thermal degradation of glucose (Heyns et al., 1966; Yang et al., 2016). 2,5-Dimethylfuran is not permitted as a food additive in Canada. It has been reported to occur either as a natural component of tobacco, a pyrolysis product (in tobacco smoke), or a tobacco additive (NTP, 2018). It has been measured in incense smoke and tobacco smoke, in the headspace above brewed coffee, as well as in Canadian and U.S. indoor air (Charles et al., 2008; EFSA, 2017; Eggert and Hanson, 2004; Pazo et al., 2016; Yang et al., 2016; Li et al., 2019). Although 2,5-dimethylfuran is a strong candidate as a next-generation biofuel, with an energetic content similar to gasoline, its current status in Canada for use as a biofuel is unknown (Lichtenhaler, 2012; Simmie and Würmel, 2013).

The general Canadian population is expected to be exposed to 2,5-dimethylfuran primarily through smoking, inhalation of indoor air, and diet. 2,5-Dimethylfuran is considered a reliable and specific tracer of environmental tobacco smoke in indoor air; levels in breath and blood have been used as indicators of smoking status (Alonso et al., 2010; Besalú et al., 2014; Bi et al., 2005; Blount et al., 2006; Charles et al., 2008; CDC, 2016). Blood levels of 2,5-dimethylfuran provide a rough estimate of the number of cigarettes smoked per day (CDC, 2016). It should be noted that concentrations of 2,5-dimethylfuran in urine may be non-specific in regard to the parent compound, and could, for example, also result from metabolism of hexane where such exposures may occur.

Experimental animal studies have shown that following exposure, 2,5-dimethylfuran is rapidly absorbed, metabolized, and excreted in urine, as with other alkylfurans (CDC, 2016; Williams and Bend, 2006). Specific data on the distribution of 2,5-dimethylfuran are lacking. However, a study using laboratory animals administered radiolabelled 2-methylfuran, a related alkylfuran, and reported distribution as liver > kidney > lung > blood, with most of it eliminated within 24 hours (Williams and Bend, 2006). Alkylfurans are metabolized by the CYP450 pathway to hydroxylated furans, which are then excreted in urine either as phase II conjugates or as corresponding ketones (Williams and Bend, 2006). They also have the potential to form reactive intermediates during metabolism. Alkylfurans can undergo ring opening epoxidation by mixed function oxidases in the liver, in the case of 2,5-dimethylfuran forming *cis*-enedione 3(Z)-hexene-2,5-dione, which can form adducts by binding with amino acids/proteins (EFSA, 2017; Williams and Bend, 2006). Based on human occupational and animal studies, 2,5-dimethylfuran is also a known urinary metabolite of n-hexane; together with 2,5-hexanedione and 4,5-dihydroxy-2-hexanone, it is one of its main metabolites in humans (EPA, 2005).

Available information on the health effects of 2,5-dimethylfuran is very limited. Furan, 2-methylfuran and 3-methylfuran share a common metabolic pathway and the ability to bind irreversibly to proteins. 2,5-Dimethylfuran can be considered a structural analogue of substances such as furan, 2-methylfuran, and furfuryl alcohol, and may have similar health effects (Phuong et al., 2012; Williams and Bend, 2006). Furan, 2-methylfuran, and 3-methylfuran demonstrate similar potencies for hepatotoxicity in animal studies, and the European Food Safety Authority (EFSA) (2017) assumed dose additivity of these substances in its risk determination. However, it was concluded that there was insufficient information in vivo to assume additivity for 2,5-dimethylfuran in producing this effect. There is evidence that 2,5-dimethylfuran induces chromosomal damage in vitro in mammalian cells, and limited evidence of its ability to induce DNA breaks in vivo (EFSA, 2017). 2,5-Dimethylfuran may also have neurotoxic potential, as it has been shown to induce

in vitro cytotoxicity in Schwann neural cells, and may play a role in the neurotoxicity of hexane (Williams and Bend, 2006).

2,5-Dimethylfuran is also part of a larger class of volatile organic compounds (VOCs) that, as a group, pose environmental and health concerns because of their contributions 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).

2,5-Dimethylfuran was analyzed in whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of 2,5-dimethylfuran in blood can be an indicator of a recent exposure to 2,5-dimethylfuran and does not necessarily mean that an adverse health effect will occur.

2,5-Dimethylfuran was also analyzed in indoor air from households of CHMS participants in cycle 3 (2012–2013) and cycle 4 (2014–2015). Further details on indoor air sampling 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@canada.ca.

Table 15.4.1

2,5-Dimethylfuran — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2544	15.7 (12.9–18.9)	—	<LOD	<LOD	0.085[E] (0.039–0.13)	0.17 (0.12–0.21)
Males, 12–79 years							
5 (2016–2017)	1270	21.5 (17.5–26.1)	—	<LOD	<LOD	0.12 (0.082–0.16)	0.18 (0.14–0.21)
Females, 12–79 years							
5 (2016–2017)	1274	9.9[E] (6.4–15.2)	—	<LOD	<LOD	<LOD	0.14[E] (0.045–0.23)
12–19 years							
5 (2016–2017)	822	8.9[E] (5.7–13.7)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	585	22.0 (16.4–28.9)	—	<LOD	<LOD	0.12[E] (0.057–0.17)	0.16 (0.11–0.20)
40–59 years							
5 (2016–2017)	564	15.0[E] (8.5–25.0)	—	<LOD	<LOD	F	0.18 (0.12–0.24)
60–79 years							
5 (2016–2017)	573	10.6 (7.8–14.3)	—	<LOD	<LOD	F	0.19[E] (0.080–0.31)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.018 µg/L.

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 are too unreliable to be published.

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15.5 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 of up to 25%; 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 occurs through 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 attached garages, with a higher number of occupants, with recent renovations, and in which fragrances and paint remover have recently been 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 not likely to be 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, such as caulking, building materials, and automotive products (ATSDR, 2010; Environment and Climate Change Canada and Health Canada, 2016). Outdoor air, drinking water, soil, and food are not considered to constitute major sources of exposure for the general population (Health Canada, 2007).

Ethylbenzene is readily absorbed and distributed throughout the body following inhalation, oral exposure, or dermal exposure (ATSDR, 2010; IARC, 2000). The proportion of ethylbenzene absorbed following 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 less than one hour up to 25 hours (ATSDR, 2010). Following oral exposure, the proportion of absorbed ethylbenzene is approximately 72% to 92% in laboratory animals, with rapid elimination occurring predominantly via urinary excretion (ATSDR, 2010). In contrast, research suggests that 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). In general, acute inhalation exposure has been associated with reversible neurological symptoms and respiratory tract irritation. 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 orally has been associated with kidney and liver damage, some minor developmental effects (such as decreased fetal body weight), 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) 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).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether ethylbenzene presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2016). The assessment concluded that ethylbenzene does not meet any of the criteria for being considered toxic under CEPA 1999 (Environment and Climate Change Canada and Health Canada, 2016). Ethylbenzene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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), as well as from on-road (Canada, 2003; Canada, 2015) and off-road (Canada, 2013; Canada, 2017) engines and vehicles.

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 is based on non-cancer effects in the liver and pituitary gland in experimental animals, and is considered to be protective of both cancer and non-cancer health effects (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), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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, 2013; Wheeler et al., 2013; Zhu et al., 2013), cycle 3 (2012–2013) (Statistics Canada, 2015), 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@canada.ca.

Table 15.5.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2441	82.0 (74.4–87.7)	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	89.7 (83.1–93.9)	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)
5 (2016–2017)	2576	78.2 (67.4–86.1)	0.024 (0.019–0.030)	<LOD	0.023 (0.017–0.028)	0.083 (0.059–0.11)	0.12 (0.092–0.16)
Males, 12–79 years							
3 (2012–2013)	1212	83.8 (77.9–88.4)	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	89.6 (82.7–93.9)	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)
5 (2016–2017)	1281	78.2 (68.8–85.4)	0.027 (0.022–0.033)	<LOD	0.026 (0.020–0.032)	0.10 (0.077–0.12)	0.13 (0.10–0.16)
Females, 12–79 years							
3 (2012–2013)	1229	80.2 (70.1–87.5)	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	89.8 (83.1–94.1)	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)
5 (2016–2017)	1295	78.1 (63.5–88.0)	0.021 (0.016–0.028)	<LOD	0.019 (0.014–0.025)	0.064[E] (0.033–0.095)	0.11[E] (0.059–0.15)
12–19 years							
3 (2012–2013)	731	78.6 (66.8–87.0)	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	86.2 (75.4–92.7)	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)
5 (2016–2017)	835	69.7 (53.3–82.3)	0.018 (0.014–0.025)	<LOD	0.019 (0.014–0.024)	0.047 (0.032–0.062)	0.065[E] (0.032–0.097)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
20–39 years							
3 (2012–2013)	532	80.6 (73.5–86.2)	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	88.7 (81.3–93.4)	0.024 (0.019–0.032)	<LOD	0.023 (0.016–0.029)	0.062[E] (0.034–0.089)	F
5 (2016–2017)	591	77.2 (59.9–88.4)	0.024 (0.018–0.032)	<LOD	0.023 (0.015–0.031)	0.079 (0.054–0.10)	0.11 (0.081–0.14)
40–59 years							
3 (2012–2013)	591	84.7 (75.7–90.8)	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	91.5 (82.2–96.1)	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)
5 (2016–2017)	569	81.7 (71.0–89.1)	0.026 (0.020–0.033)	<LOD	0.024 (0.018–0.030)	0.097 (0.062–0.13)	0.13 (0.095–0.17)
60–79 years							
3 (2012–2013)	587	81.6 (71.0–88.9)	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	90.2 (84.6–93.9)	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)
5 (2016–2017)	581	78.3 (70.0–84.8)	0.023 (0.019–0.028)	<LOD	0.021 (0.016–0.026)	0.083 (0.056–0.11)	0.12 (0.080–0.17)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.011, 0.011, and 0.013 µg/L, respectively.

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 are too unreliable to be published.

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15.6 ISOPROPYLBENZENE

Isopropylbenzene (CASRN 98-82-8), also known as cumene, is a colourless liquid that is classified as a volatile organic compound (VOC) (IARC, 2013; WHO, 2005). It is a natural constituent of crude oil and can be found in plants and food. Commercially, isopropylbenzene can be produced via distillation of coal tar and petroleum or alkylation of benzene with propene (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013). As a component of finished fuel, isopropylbenzene can be found in gasoline blends and high-octane aviation fuel.

Isopropylbenzene is used as an intermediate for chemical production, mostly for the manufacture of acetone and phenol (European Commission, 2016). It is also used in printing, ore mining, the manufacture of plastic, rubber, pesticides and pharmaceuticals, and as a solvent for fat or resin. It is also used in several consumer products, such as automotive-related products, adhesives, lubricants, specialty cleaning products, and paints (Environment and Climate Change Canada and Health Canada, 2019; EPA, 1997; IARC, 2013; NTP, 2013; WHO, 2005).

Isopropylbenzene is released to the environment mainly from the use, manufacture, and transport of processed hydrocarbon fuel. Primary anthropogenic sources include release from petrochemical refineries, accidental petroleum spills, and evaporation and combustion of petroleum products from petrol stations and motor vehicles. Other anthropogenic sources include release from products containing isopropylbenzene, tobacco smoke, jet engine exhaust, and several industrial processes (EPA, 1997; IARC, 2013; WHO, 2005).

The primary route of human exposure to isopropylbenzene is inhalation (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013). Exposure in air accounts for an estimated 97% of total isopropylbenzene intake for Canadians (Environment and Climate Change Canada and Health Canada, 2019). In Canada, levels of isopropylbenzene

measured in indoor air are generally low, but are higher than those detected in outdoor air (Environment and Climate Change Canada and Health Canada, 2019; Health Canada, 2010a; Health Canada, 2010b; Health Canada, 2012; Health Canada, 2013). Various building materials and stored combustion equipment can contribute to the presence of isopropylbenzene in indoor air (Won et al., 2013; Won et al. 2014; Won et al., 2015). Given the volatility of isopropylbenzene, inhalation as well as dermal exposures may result from the use of products available to consumers (Environment and Climate Change Canada and Health Canada, 2019; HSDB, 2013). To a lesser extent, exposure may also occur from the ingestion of food or water. Although isopropylbenzene has been monitored in drinking water in three Canadian cities since 2000, no detectable levels have been found (Environment and Climate Change Canada and Health Canada, 2019).

Human studies show that isopropylbenzene is readily absorbed following inhalation exposure. This finding is supported by experimental animal studies demonstrating rapid absorption following exposure by inhalation, ingestion, or dermal contact (EPA, 1997; NTP, 2013). Animal studies demonstrate that isopropylbenzene is widely distributed throughout the body following absorption, with higher concentrations found in adipose tissue, bones, liver, and kidneys (EPA, 1997). Isopropylbenzene is highly lipophilic and can potentially accumulate in adipose tissue (European Commission, 2016). It is extensively metabolized in the body by hepatic and extrahepatic tissues, including the lungs, to water soluble compounds (NTP, 2013; WHO, 2005). Several metabolites of isopropylbenzene were detected in animal experiments, the main one being 2-phenyl-2-propanol, which is also detected in human studies (NTP, 2013). Different elimination half-lives have been estimated for humans, with the average being less than a day. Isopropylbenzene is rapidly eliminated from the body, principally as a conjugate of 2-phenyl-2-propanol. Experimental studies show that isopropylbenzene is excreted mainly in urine (>70%), and to a lesser extent in feces or exhaled air (NTP, 2013; WHO, 2005). The parent chemical in blood or exhaled air, as well as the urinary level of its main metabolite, 2-phenyl-2-propanol, can be used as biomarkers of exposure (European Commission, 2016).

Although the acute systemic toxicity of isopropylbenzene is regarded as generally low, this substance has been shown to cause dizziness, slight incoordination, and unconsciousness in humans following exposure to high levels through inhalation (HSDB, 2013). In laboratory animals, central nervous system depression and transient ataxia have been observed following high-level exposure (Environment and Climate Change Canada and Health Canada, 2019; HSDB, 2013; Jahnke et al., 2013). Human and laboratory animal studies both report low to moderate toxicity from oral administration of isopropylbenzene. Irritation has been observed after dermal or eye contact (European Commission, 2016; HSDB, 2013; Jahnke et al., 2013). Chronic inhalation exposure has been found to lead to increased liver, kidney, and adrenal weights in laboratory animals (European Commission, 2016). Chronic inhalation exposure is also associated with tumours of the respiratory tract, kidneys, liver, and spleen in laboratory animals; however, there is a lack of human data on the carcinogenicity of isopropylbenzene (IARC, 2013; NTP, 2013). The International Agency for Research on Cancer (IARC) has classified isopropylbenzene and one of its metabolites (α-methylstyrene) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 2013).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether isopropylbenzene presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2019). The assessment concluded that isopropylbenzene does not meet any of the criteria for being considered toxic under CEPA 1999 (Environment and Climate Change Canada and Health Canada, 2019).

In 2017, Health Canada published an Indoor Air Reference Level (IARL) for isopropylbenzene (EPA, 1997; Health Canada, 2017). Isopropylbenzene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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).

Isopropylbenzene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of isopropylbenzene in blood can be an indicator of recent exposure to isopropylbenzene and does not necessarily mean that an adverse health effect will occur.

Isopropylbenzene 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 indoor air sampling 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@canada.ca.

Table 15.6.1

Isopropylbenzene — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2571	70.1 (56.7–80.8)	0.015 (0.011–0.021)	<LOD	0.016 (0.010–0.022)	0.044[E] (0.021–0.067)	0.068[E] (0.031–0.10)
Males, 12–79 years							
5 (2016–2017)	1280	65.7 (47.9–80.0)	0.014[E] (<LOD–0.021)	<LOD	0.015[E] (<LOD–0.021)	0.044[E] (0.014–0.075)	0.071[E] (0.039–0.10)
Females, 12–79 years							
5 (2016–2017)	1291	74.5 (63.6–83.0)	0.016 (0.012–0.021)	<LOD	0.017 (0.011–0.023)	0.043[E] (0.026–0.060)	0.060[E] (0.016–0.10)
12–19 years							
5 (2016–2017)	833	68.6 (56.2–78.8)	0.014 (0.010–0.019)	<LOD	0.015 (0.012–0.019)	0.037[E] (0.012–0.063)	F
20–39 years							
5 (2016–2017)	590	64.4 (51.8–75.4)	0.013 (<LOD–0.019)	<LOD	0.014[E] (<LOD–0.020)	F	0.075[E] (0.031–0.12)
40–59 years							
5 (2016–2017)	569	72.0 (54.2–84.8)	0.016[E] (0.011–0.023)	<LOD	0.017[E] (0.010–0.025)	0.043[E] (0.025–0.061)	F
60–79 years							
5 (2016–2017)	579	76.3 (62.5–86.2)	0.017 (0.012–0.024)	<LOD	0.017[E] (0.011–0.024)	0.051[E] (0.030–0.072)	0.074[E] (0.031–0.12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/L.

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 are too unreliable to be published.

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15.7 METHYL ISOBUTYL KETONE

Methyl isobutyl ketone (MIBK) (CASRN 108-10-1), also known as 4-methyl-2-pentanone, among other synonyms, belongs to the ketone class of organic compounds and takes the form of a clear colourless liquid with an odour described as pleasant. MIBK can occur naturally in foods (e.g., fruit, olive oil, chicken, eggs, beer, coffee, and cow's milk) or as a flavouring ingredient in food items, such as baked goods, frozen dairy products, gelatins/puddings, meat products, and soft candy (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013). MIBK is also a high-production volume industrial chemical that is most commonly synthesized by aldol condensation of acetone and its derivative intermediates, diacetone alcohol and mesityl oxide (IARC, 2013). MIBK is used as an industrial organic solvent for gums, resins, paints, varnishes, lacquers, and nitrocellulose (NCBI, 2018). It may also be used as a denaturant, chemical intermediate, bulking agent in drugs, food-flavouring agent, and as a component of food-packaging materials (IARC, 2013; OECD, 2009; OEHHA, 2018).

MIBK enters the environment primarily from anthropogenic sources. It can be released into the atmosphere during its production through fugitive emissions and the incomplete removal of vapours from reaction gases (IARC, 2013; OECD, 2009). It may also leak from landfills or be released to surface waters during the discharge of spent scrubbing water from industrial production processes (IARC, 2013).

The most likely routes of exposure to MIBK are the ingestion of contaminated drinking water and inhalation or dermal exposure from the use of consumer products (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013). Concentrations of MIBK are higher in indoor air than in outdoor air in Canadian homes (Health Canada, 2010a; Health Canada 2010b; Health Canada, 2012; Health Canada, 2013). MIBK is found in and/or emitted by products such as building materials, pesticides, automotive products, and agents for wax/oil separation, leather finishing, and textile coating (EPA, 2003; IARC, 2013; Won et al., 2013; Won et al., 2014; Won et al. 2015; Won and Yang, 2012). Exposure can also occur through the ingestion of food that contains MIBK as a natural constituent, as a flavouring agent, or from its migration from food packaging (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013). MIBK has been shown to be readily biodegradable and will likely volatilize rapidly from water or soil; therefore, it is not expected to persist in the environment (OECD, 2009).

Toxicokinetic studies demonstrate that MIBK is readily absorbed into the blood following exposure by any route, and that its level in blood is related to the oral or inhalation exposure level (EPA, 2003). MIBK can be found as a volatile component of urine, and its presence in urine also serves as a biological marker of exposure (NCBI, 2018). Data from human studies show that following absorption, MIBK can be distributed throughout the body to tissues that include the liver, kidney, lung, and brain; it is rapidly eliminated from the blood following cessation of exposure (generally within two hours), with exhalation being the major route of elimination (IARC, 2013). Experimental animal data indicate that major metabolites of MIBK include diacetone alcohol, 4-hydroxymethyl isobutyl ketone, and 4-methyl-2-pentanol, and that the

metabolic pathway likely involves alcohol dehydrogenase and cytochrome P450 mono-oxygenases (IARC, 2013). MIBK has been detected in breast milk; evidence from one human study suggests that MIBK can enter the umbilical cord and cross the placenta (Environment and Climate Change Canada and Health Canada, 2019; IARC, 2013).

Acute exposure to MIBK in humans has been shown to irritate the eyes and mucous membranes, and produce effects on the central nervous system such as headache, weakness, nausea, light-headedness, lack of coordination, irritation, and narcosis (NCBI, 2018; OECD, 2009). Similar effects have been observed in occupational studies of long-term human exposure to MIBK, along with insomnia, intestinal pain, and slight enlargement of the liver (NCBI, 2018). Increased liver and kidney weights, reversible kidney damage and lethargy have been noted in chronic studies with laboratory animals following repeated inhalation or oral exposures to high concentrations of MIBK (NCBI, 2018; OECD, 2009). MIBK may produce its effects by disrupting nerve membrane integrity, which could potentially explain the transient neurological symptoms in humans and animals that occur only during or immediately following exposure (EPA, 2003). Maternal toxicity (e.g., increased liver and kidney weights) and fetotoxicity (e.g., reduced fetal body weights and delayed ossification) have been observed at high exposure concentrations in animal studies (OECD, 2009). The International Agency for Research on Cancer (IARC) has classified MIBK as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 2013).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether MIBK presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2019). The assessment proposes to conclude that MIBK is toxic under CEPA 1999, as it is considered harmful to human health (Environment and Climate Change Canada and Health Canada, 2019).

In 2017, Health Canada published an Indoor Air Reference Level (IARL) for MIBK (Health Canada, 2017). MIBK is part of a larger class of volatile organic compounds (VOCs) that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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). Environment Canada identified MIBK as a substance of concern that may be found in screen-printing and digital imaging processes. It is also part of an environmental performance agreement that involves targeted VOC emissions reductions by participating facilities (Environment Canada, 2012). MIBK is listed on Health Canada's Natural Health Products Ingredients Database as having a non-medicinal role for oral use as a flavour enhancer or topical use as a denaturant (Health Canada, 2018a). Health Canada recently implemented the International Council for Harmonisation Guidelines for Residual Solvents, which places MIBK in Class 2 (solvents to be limited) (Health Canada, 2018b).

MIBK was analyzed in the whole blood of Canadian Health Measure Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented in blood as µg/L. Finding a measurable amount of MIBK in blood is an indicator of exposure to MIBK and does not necessarily mean that an adverse health effect will occur.

MIBK 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 indoor air sampling 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@canada.ca.

Table 15.7.1

Methyl isobutyl ketone (MIBK) — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2363	73.4 (61.9–82.5)	0.040 (0.033–0.049)	<LOD	0.043 (0.035–0.051)	0.094 (0.072–0.12)	0.12 (0.084–0.15)
Males, 12–79 years							
5 (2016–2017)	1166	74.1 (63.1–82.7)	0.041 (0.033–0.051)	<LOD	0.043 (0.035–0.050)	0.11 (0.077–0.13)	0.13[E] (0.050–0.22)
Females, 12–79 years							
5 (2016–2017)	1197	72.8 (59.7–82.9)	0.039 (0.032–0.048)	<LOD	0.043 (0.034–0.052)	0.085 (0.070–0.10)	0.10 (0.080–0.13)
12–19 years							
5 (2016–2017)	767	63.8 (48.8–76.5)	0.032 (<LOD–0.039)	<LOD	0.034 (<LOD–0.042)	0.066 (0.049–0.083)	0.088 (0.066–0.11)
20–39 years							
5 (2016–2017)	533	68.1 (54.5–79.2)	0.037 (0.030–0.047)	<LOD	0.042 (0.033–0.050)	0.090 (0.058–0.12)	0.12[E] (0.043–0.20)
40–59 years							
5 (2016–2017)	519	77.6 (63.2–87.4)	0.041 (0.033–0.052)	<LOD	0.043 (0.033–0.054)	0.092 (0.067–0.12)	0.11 (0.087–0.14)
60–79 years							
5 (2016–2017)	544	79.3 (63.7–89.3)	0.047 (0.037–0.060)	<LOD	0.051 (0.040–0.062)	0.11 (0.086–0.13)	0.14 (0.10–0.18)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.029 µg/L.

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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15.8 NITROBENZENE

Nitrobenzene (CASRN 98-95-3) is a nitroaromatic substance that is a greenish-yellow to yellow oily liquid at room temperature, with an odour similar to bitter almonds or shoe polish (NTP, 2016). Nitrobenzene is a high-production volume chemical generally produced industrially in a batch-wise or continuous nitration process in which mixed acid is added to an excess of benzene under temperature-controlled conditions (Booth, 2012; EPA, 2009).

The major use of nitrobenzene is as an intermediate in the production of aniline, a chemical that is used to produce diphenylmethane and diisocyanate (MDI) for polyurethane foams (CAREX, 2018; NTP, 2016). It also has a number of minor uses, including as a solvent (e.g., in petroleum refining or in gun cleaners), an ingredient of metal polishes and soaps, and in the manufacture of rubber chemicals, herbicides, dyes, pigments, fibres, and other chemicals (CAREX, 2018; NICNAS, 2016).

Nitrobenzene is not known to occur naturally (CAREX, 2018). It may be present in groundwater, surface water, and air, but concentrations are generally low and below detection limits (WHO, 2009). Nitrobenzene in ambient air may originate from industrial processes (e.g., manufacturing facilities and petroleum refineries), abandoned hazardous waste sites, or from the atmospheric photochemical reaction of nitrous oxides and benzene from automobile exhaust; however, most nitrobenzene produced during its manufacture is retained in closed systems. Restrictions on benzenes in gasoline have reduced levels of nitrobenzene in the atmosphere in Canada (CAREX, 2018). Levels of nitrobenzene in air, ground water, soil, and plant tissues may be higher around abandoned hazardous waste sites. Data indicate that levels in groundwater are expected to be higher than in surface waters, likely due to the limited potential for volatilization and biodegradation in groundwater systems (WHO, 2009).

Exposure of the Canadian general population to nitrobenzene from air, water, and soil and from the use of consumer products is expected to be negligible (Environment and Climate Change Canada and Health Canada, 2016). Populations in the vicinity of petroleum refineries, abandoned hazardous waste sites, and certain manufacturing activities could potentially have elevated exposures to nitrobenzene.

Nitrobenzene is readily absorbed following exposure, with estimated uptake rates ranging from 73% to 87% in human volunteers following inhalation and 43% to 69% in animals following oral exposure (NICNAS, 2016). Nitrobenzene has been shown to distribute primarily to erythrocytes, spleen, liver, testes, and brain tissue following absorption (NICNAS, 2016). Based on animal studies, there are three major metabolic pathways for nitrobenzene: a two-step reduction to aniline by intestinal microflora, a six-step reduction to aniline in hepatic microsomes and erythrocytes, and oxidative metabolism by hepatic microsomes to nitrophenols (likely involving cytochrome P450s) (EPA, 2009; NICNAS, 2016). Nitrobenzene is excreted mainly in urine, and to a lesser extent in feces and expired air, in both animals and humans (EPA, 2009; NICNAS, 2016). In humans, about 70% of orally dosed nitrobenzene was found to be eliminated from the body within the course of a week, with about 58% of the dose eliminated as metabolites in urine and 9% in feces, with a small fraction in expired air (~1%). The elimination half-life of nitrobenzene in urine — based on a small number of studies in humans and different routes of exposure — was estimated to range from 20 hours to a few days (IARC, 1996). A major portion of the absorbed dose is excreted into urine as 4-nitrophenol, with a smaller fraction excreted as 4-aminophenol or 3-nitrophenol. Studies suggest that the portion of absorbed nitrobenzene not eliminated from the body may bind to hemoglobin and plasma proteins (EPA, 2009).

In humans, acute exposure to nitrobenzene can potentially lead to respiratory depression, general weakness, severe headaches and dizziness, vomiting, convulsions, and unconsciousness (NICNAS, 2016). The characteristic symptom of acute exposure in humans is methemoglobinemia, coupled with cyanosis (EPA, 2009; NICNAS, 2016). Other reported acute systemic effects in humans include the formation of Heinz bodies in erythrocytes, effects on the bone marrow and lymphoid organs, neurotoxicity, and hepatotoxicity (NICNAS, 2016). Studies in animals demonstrate a wide spectrum of non-cancer effects following sub-chronic or chronic exposure to nitrobenzene, including increased organ weights and histopathologic lesions of the spleen, liver, adrenals, kidney, and brain, methemoglobinemia with subsequent hemolytic anemia and splenic congestion, neurotoxicity, and a significant and pronounced adverse effect on the male reproductive system resulting in reduced

fertility (EPA, 2009; NICNAS, 2016). In addition, long-term inhalation exposure has been reported to result in olfactory degeneration and bronchiolization of the alveoli in laboratory animals (EPA, 2009). Increased incidences of non-neoplastic lesions have been observed in the lung, thyroid, liver, kidney, spleen, nasal turbinates, and testes of chronically exposed animals at both low and high doses, with the severity of effect increasing with dose (EPA, 2009; NICNAS, 2016). Nitrobenzene is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 1996).

The Government of Canada has conducted a rapid screening assessment under the Chemicals Management Plan to determine whether nitrobenzene presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2016). The assessment concluded that nitrobenzene does not meet any of the criteria for being considered toxic under CEPA 1999 based on current use patterns and quantities in commerce in Canada being unlikely to cause harm to the environment or human health (Environment and Climate Change Canada and Health Canada, 2016). However, nitrobenzene was recommended as a potential candidate for a significant new activity notice (SNAc) under CEPA 1999 on the basis of classifications for health effects of concern (carcinogenicity and reproductive effects) by other national or international agencies (Environment and Climate Change Canada and Health Canada, 2016).

Nitrobenzene is identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

Nitrobenzene is also part of a larger class of volatile organic compounds (VOCs) that, as a group, pose environmental and health concerns because of their contributions 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).

Nitrobenzene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of nitrobenzene in blood can be an indicator of a recent exposure to nitrobenzene and does not necessarily mean that an adverse health effect will occur.

Table 15.8.1

Nitrobenzene — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2327	F	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
5 (2016–2017)	1164	F	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
5 (2016–2017)	1163	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	753	0	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	539	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	523	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	512	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 1.1 µg/L.

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

F Data are too unreliable to be published.

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15.9 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 can contain traces of other 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 manufacture 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 (Health Canada, 2010a; Health Canada, 2010b; Health Canada, 2012; Health Canada, 2013; 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). Sources of styrene in indoor air include stored combustion equipment, various building materials, furniture, air fresheners, shampoo, incense, candles and mothballs (Won et al., 2013; Won et al., 2014; Won and Lusztyk, 2011; Won and Yang, 2012). 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 food packaged 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). In one study of laboratory animals, styrene absorption following oral exposure was rapid and complete followed by distribution to the kidney, liver, pancreas, adipose tissue, and, to a lesser extent, the stomach and small and large intestines (ATSDR, 2010). The absorbed styrene was rapidly eliminated from all tissues within one to three days. In a study with human volunteers, half-lives for the concentration of styrene in blood were estimated to range between one and 13 hours depending on the phase of elimination; in adipose tissue, an elimination half-life of two to five days was 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 orally exposed laboratory animals, styrene was rapidly excreted in urine, with 90% eliminated within 24 hours, and less than 2% eliminated in feces (ATSDR, 2010). The most reliable biomarker of recent exposure to styrene is measurement 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).

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). In 2017, Health Canada published an Indoor Air Reference Level (IARL) for styrene (Health Canada, 2017). Styrene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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). Further details on indoor air sampling 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@canada.ca.

Table 15.9.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2063	91.4 (74.1–97.5)	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	96.2 (87.1–99.0)	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)
5 (2016–2017)	2527	89.1 (84.7–92.4)	0.027 (0.024–0.030)	<LOD	0.028 (0.024–0.032)	0.067 (0.054–0.079)	0.094 (0.075–0.11)
Males, 12–79 years							
3 (2012–2013)	1036	91.7 (75.1–97.6)	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	95.0 (82.0–98.8)	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)
5 (2016–2017)	1256	88.3 (80.8–93.1)	0.028 (0.025–0.033)	<LOD	0.029 (0.026–0.033)	0.074 (0.056–0.093)	0.099 (0.068–0.13)
Females, 12–79 years							
3 (2012–2013)	1027	91.1 (72.6–97.5)	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	97.4 (91.8–99.2)	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)
5 (2016–2017)	1271	90.0 (86.3–92.7)	0.026 (0.022–0.030)	<LOD	0.026 (0.021–0.031)	0.062 (0.049–0.074)	0.087 (0.060–0.11)
12–19 years							
3 (2012–2013)	626	91.8 (73.0–97.9)	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	97.1 (86.5–99.4)	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)
5 (2016–2017)	824	90.5 (86.1–93.7)	0.025 (0.022–0.030)	<LOD	0.027 (0.022–0.031)	0.053 (0.044–0.063)	0.066 (0.048–0.085)
20–39 years							
3 (2012–2013)	435	89.8 (69.4–97.2)	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	97.0 (86.2–99.4)	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)
5 (2016–2017)	574	88.1 (79.1–93.6)	0.027 (0.023–0.032)	<LOD	0.027 (0.021–0.034)	0.072 (0.051–0.094)	0.095 (0.073–0.12)
40–59 years							
3 (2012–2013)	493	93.5 (76.8–98.4)	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	94.0 (80.9–98.3)	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)
5 (2016–2017)	555	88.6 (82.9–92.5)	0.028 (0.025–0.032)	<LOD	0.029 (0.025–0.033)	0.067 (0.053–0.080)	0.11[E] (0.067–0.15)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
60–79 years							
3 (2012–2013)	509	90.2 (70.4–97.3)	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	98.0 (94.5–99.3)	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)
5 (2016–2017)	574	90.7 (85.3–94.2)	0.026 (0.023–0.031)	0.011[E] (<LOD–0.016)	0.027 (0.022–0.033)	0.062 (0.041–0.083)	0.092 (0.069–0.12)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.012, 0.012, and 0.011 µg/L, respectively.

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 are too unreliable to be published.

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15.10 1,1,1,2-TETRACHLOROETHANE

1,1,1,2-Tetrachloroethane (CASRN 630-20-6) is a halogenated organic compound that is a colourless liquid at room temperature. It can be synthesized in a highly purified form by isomerization of 1,1,2,2-tetrachloroethane or by chlorination of 1,1-dichloroethylene, but it is also a by-product of the manufacture of other chlorinated ethanes (1,1,1-trichloroethane, 1,1,2-trichloroethane and 1,1,2,2-tetrachloroethane) (IARC, 2014). 1,1,1,2-Tetrachloroethane is not as widely studied as its isomer, 1,1,2,2-tetrachloroethane. However, this chemical summary will focus on the available information for 1,1,1,2-tetrachloroethane.

1,1,1,2-Tetrachloroethane is primarily used in the production of solvents such as trichloroethylene. It is also used as a laboratory reagent and as a solvent in the manufacture of bleaches, paint varnishes, insecticides, herbicides, and soil fumigants (IARC 2014). 1,1,1,2-Tetrachloroethane does not occur naturally. It is only released into the environment from anthropogenic sources (IARC, 2014). It can enter air through industrial air emissions and water through industrial waste streams (IARC, 2014). The general population may be exposed through inhalation of ambient air. Ingestion and skin or eye contact are other possible routes of exposure (Pohanish, 2012).

No data are available for the toxicokinetics of 1,1,1,2-tetrachloroethane in humans (IARC, 2014). In vitro pharmacokinetic data indicate that 1,1,1,2-tetrachloroethane can be absorbed by inhalation. This finding is supported by an animal study that showed substantial respiratory uptake of this substance (IARC 2014). Pharmacokinetic modelling suggests that 1,1,1,2-tetrachloroethane is likely widely distributed to tissues after systemic absorption (IARC, 2014). In experimental animal studies it was observed that the major urinary metabolite of 1,1,1,2-tetrachloroethane was trichloroethanol; other metabolites may include trichloroacetic acid, 1,1- dichloroethylene, and 1,1,2-trichloroethane. It can also be dechlorinated in the presence of oxygen (IARC, 2014). Studies in animals show that 1,1,1,2-tetrachloroethane is eliminated rapidly from the body, with most of it excreted 24 hours after absorption, and that elimination of 1,1,1,2-tetrachloroethane occurs mainly as urinary metabolites along with a small amount of exhaled

carbon dioxide; at high doses it can be eliminated unchanged in exhaled air (IARC, 2014).

Available information suggests that exposure to 1,1,1,2-tetrachloroethane may produce adverse health effects, but the effects are not well studied in humans or animals. Acute inhalation exposure in humans may cause irritation of the respiratory tract, leading to coughing, wheezing, and shortness of breath, while direct contact may irritate the eyes and skin (Pohanish, 2012). Acute exposure in humans may also lead to central nervous system depression, weakness, tremor, reduced muscle coordination, drowsiness, respiratory difficulties, headache, vomiting and coma (National Research Council, 1977; Pohanish, 2012). One study using various animal species reported that acute exposure to 1,1,1,2-tetrachloroethane was associated with hepatotoxicity (microvacuolation and/or central lobular necrosis in the liver), and that the substance passed through the placental barrier and affected the fetus (National Research Council, 1977). Chronic exposure to 1,1,1,2-tetrachloroethane in animals has been reported to result in damage to the central nervous system, skin, kidneys, and liver (IARC, 2014; Pohanish, 2012; National Research Council, 1977). 1,1,1,2-Tetrachloroethane is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence for carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 2014).

The Government of Canada conducted a rapid screening assessment under the Chemicals Management Plan to determine whether 1,1,1,2-tetrachloroethane presents or may present a risk to the environment or human health as per the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2018). Based on the information available at the time of the assessment, it was concluded that 1,1,1,2-tetrachloroethane does not meet any of the criteria for being considered toxic under CEPA 1999 on the basis of negligible exposure of the general population of Canada and low ecological concern (Environment and Climate Change Canada and Health Canada, 2018).

1,1,1,2-Tetrachloroethane was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented as μg/L blood. Finding a measurable amount of 1,1,1,2-tetrachloroethane in blood can be an indicator of recent exposure to 1,1,1,2-tetrachloroethane and does not necessarily mean that an adverse effect will occur.

1,1,1,2-Tetrachloroethane was also analyzed in indoor air from households of CHMS participants in cycle 3 (2012–2013) and cycle 4 (2014–2015). Further details on indoor air sampling 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@canada.ca.

Table 15.10.1

1,1,1,2-Tetrachloroethane — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2576	F	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
5 (2016–2017)	1281	F	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
5 (2016–2017)	1295	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
5 (2016–2017)	835	0	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	591	0	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	569	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
5 (2016–2017)	581	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.007 µg/L.

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

F Data are too unreliable to be published.

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15.11 TETRACHLORO-ETHYLENE

Tetrachloroethylene (CASRN 127-18-4), commonly known as perchloroethylene, is a colourless liquid classified as a volatile organic compound (VOC) (Canada, 2003; Canada, 2011; 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, it 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, 2011; Canada, 2003; 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 will be phased out by 2030 (IARC, 2014; UNEP, 2019). In Canada, tetrachloroethylene production ceased in 1992. 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, 2015).

Releases of tetrachloroethylene are mainly to the atmosphere by evaporative losses from anthropogenic sources (ATSDR, 2014; 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, various building materials, automotive products, and other consumer products containing tetrachloroethylene (Won et al., 2013; Won et al., 2015; Environment Canada and Health Canada, 1993). Concentrations of tetrachloroethylene are higher in indoor air than in outdoor air in Canadian homes (Health Canada, 2010a; Health Canada 2010b; Health Canada, 2012; Health Canada, 2013). 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; Health Canada, 2015). Exposure can also occur via ambient air and food (ATSDR, 2014; Environment Canada and Health Canada, 1993). Living near a dry-cleaning facility may also increase the potential for exposure (ATSDR, 2014; CDC, 2009; IARC, 2014).

Tetrachloroethylene is rapidly absorbed into the blood and distributed throughout the body, with some concentration in adipose tissue (ATSDR, 2014; 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 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 hours, 30 to 40 hours, and 55 hours, respectively (ATSDR, 2014). 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, 2014; 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, 2014). The central nervous system is a primary target for tetrachloroethylene toxicity. At very high concentrations, acute exposure to tetrachloroethylene can induce central nervous system depression and loss of consciousness, while prolonged exposure may result in neurobehavioral effects and vision changes (ATSDR, 2014; Environment Canada and Health Canada, 1993). Tetrachloroethylene exposure is associated with narcotic and anesthetic effects that increase in severity with increasing exposure (ATSDR, 2014; 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, 2014; Environment Canada

and Health Canada, 1993; IARC, 2014). Available animal data also identify the kidney, liver, reproductive system, and developing fetus as potential targets of tetrachloroethylene toxicity. Multiple cancer sites of interest have been evaluated by the International Agency for Research on Cancer (IARC) Expert Working Group; 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 of 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, 2011; Canada, 2003). 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, 2003; Environment Canada, 2013a). Tetrachloroethylene is identified as being prohibited on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may not be compliant with requirements of the *Food and Drugs Act* or the Cosmetic Regulations (Health Canada, 2018).

In 2017, Health Canada published an Indoor Air Reference Level (IARL) for tetrachloroethylene (Health Canada, 2017). Tetrachloroethylene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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, 2013b).

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, 2015). 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), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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). Further details on indoor air sampling 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@canada.ca.

Table 15.11.1

Tetrachloroethylene — 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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2453	44.1 (34.5–54.3)	—	<LOD	<LOD	0.10 (0.067–0.14)	0.17[E] (0.10–0.23)
4 (2014–2015)	2527	28.4 (22.6–34.9)	—	<LOD	<LOD	0.066[E] (0.022–0.11)	F
5 (2016–2017)	2487	73.3 (55.5–85.8)	0.035[E] (0.018–0.069)	<LOD	0.034[E] (<LOD–0.057)	F	F
Males, 12–79 years							
3 (2012–2013)	1228	47.6 (37.8–57.6)	—	<LOD	<LOD	0.13 (0.086–0.17)	0.19 (0.13–0.25)
4 (2014–2015)	1251	30.1 (23.7–37.3)	—	<LOD	<LOD	F	F
5 (2016–2017)	1238	72.7 (50.7–87.4)	F	<LOD	0.038[E] (<LOD–0.065)	F	F
Females, 12–79 years							
3 (2012–2013)	1225	40.7 (29.2–53.4)	—	<LOD	<LOD	0.096[E] (0.060–0.13)	0.13[E] (0.039–0.22)
4 (2014–2015)	1276	26.7 (20.3–34.3)	—	<LOD	<LOD	0.068[E] (<LOD–0.12)	F
5 (2016–2017)	1249	73.9 (59.1–84.8)	0.034[E] (0.018–0.064)	<LOD	0.031[E] (<LOD–0.050)	F	F
12–19 years							
3 (2012–2013)	739	37.9 (29.2–47.5)	—	<LOD	<LOD	F	F
4 (2014–2015)	713	20.0 (14.3–27.4)	—	<LOD	<LOD	0.042[E] (<LOD–0.065)	F
5 (2016–2017)	816	65.8 (45.6–81.5)	F	<LOD	F	F	F
20–39 years							
3 (2012–2013)	543	47.7 (32.2–63.5)	—	<LOD	<LOD	0.093[E] (0.052–0.13)	0.15[E] (0.080–0.23)
4 (2014–2015)	600	24.6[E] (14.5–38.4)	—	<LOD	<LOD	F	F
5 (2016–2017)	570	71.7 (51.4–85.9)	0.031[E] (0.015–0.063)	<LOD	0.033[E] (<LOD–0.056)	F	F
40–59 years							
3 (2012–2013)	587	40.3 (29.8–51.8)	—	<LOD	<LOD	0.10[E] (0.058–0.14)	0.13 (0.089–0.17)
4 (2014–2015)	625	28.4 (21.8–36.2)	—	<LOD	<LOD	0.061[E] (<LOD–0.10)	F
5 (2016–2017)	546	75.9 (58.1–87.7)	F	<LOD	F	F	F
60–79 years							
3 (2012–2013)	584	48.3 (36.6–60.2)	—	<LOD	<LOD	0.16[E] (0.062–0.25)	F
4 (2014–2015)	589	38.3 (32.5–44.5)	—	<LOD	<LOD	0.088[E] (0.028–0.15)	F
5 (2016–2017)	555	75.3 (56.5–87.8)	0.039[E] (0.020–0.075)	<LOD	0.034[E] (0.014–0.054)	F	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.020, 0.020, and 0.013 µg/L, respectively.

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 are too unreliable to be published.

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15.12 TETRAHYDROFURAN

Tetrahydrofuran (CASRN 109-99-9) is a colourless, volatile liquid with an odour similar to ether or acetone (EPA, 2012). It is a high-production volume chemical (EPA, 2018; OECD, 2018) and is typically produced industrially by the Reppe Process involving the reaction of acetylene and formaldehyde, followed by hydrogenation and acid catalysis (Müller, 2012). Based on 2011 survey information collected pursuant to Section 71 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), tetrahydrofuran was not reported to be manufactured in Canada (Environment and Climate Change Canada and Health Canada, 2018). The quantity of tetrahydrofuran imported into Canada has remained relatively steady over the last 30 years (Statistics Canada, 2018).

The single largest use of tetrahydrofuran in Canada and internationally is in the production of polytetramethylene ether glycol (PTMEG), an important component of synthetic elastic construction materials, thermoplastics and moulded elastomers, elastic Spandex fibres, and polyurethane coatings (Environment and Climate Change Canada and Health Canada, 2018; Müller, 2012; OECD, 2000). Other uses of tetrahydrofuran in Canada are in the production of adhesives such as PVC cement, varnish and paint removers, and paints and coatings. Tetrahydrofuran may also be present in nail adhesives and can be formed as an impurity during the manufacture of some resins used in food-packaging materials. It is also a formulant in pest control products currently registered in Canada (Environment and Climate Change Canada and Health Canada, 2018; Health Canada, 2018). There are some reports of its use in furniture polish and cleaners, laundry starch preparations, and stain removers, but no evidence of these uses was identified in Canada (Environment and Climate Change Canada and Health Canada, 2018). Tetrahydrofuran can be used in the production of cellophane, protective coatings, magnetic strips, and printing inks, and as an intermediate in the production of other chemicals, including acrylic acid, adipic acid, butadiene, butyrolactone, succinic acid, and 1,4-butanediol diacetate. It can also be used in the fabrication of materials for food packaging, transport, and storage, and in motor fuels, vitamins, hormones, pharmaceuticals, synthetic perfumes, organometallic compounds, and insecticides (EPA, 2012).

Tetrahydrofuran is not known to occur naturally (EPA, 2012). It may be present in ambient and indoor air from anthropogenic sources. Measured concentrations in Canadian ambient air are generally very low, but available monitoring data indicate higher levels in indoor air (Environment and Climate Change Canada and Health Canada, 2018). Tetrahydrofuran may be elevated in indoor air where polyvinyl chloride (PVC) cements or other consumer products containing tetrahydrofuran have been used (Environment and Climate Change Canada and Health Canada, 2018). Tetrahydrofuran has been identified as a volatile component of some foods (including coffee, cooked meat, honey, and blackberries), potentially formed from thermal degradation or chemical rearrangement of naturally occurring precursors during cooking or processing. Tetrahydrofuran has been identified (but not quantified) in the breast milk of mothers living in the United States; however, similar data in Canada are lacking (Environment and Climate Change Canada and Health Canada, 2018; Pellizzari et al., 1982).

Canadians are exposed to tetrahydrofuran primarily through indoor air. Given the use patterns of tetrahydrofuran and its physical-chemical properties (i.e., very high vapour pressure and low octanol-water partition coefficient), exposure of the general population from food and drinking water is expected to be much lower compared with exposure from air. Use of consumer products such as PVC cements and adhesives by the general population may result in exposure through inhalation or dermal contact (Environment and Climate Change Canada and Health Canada, 2018).

Tetrahydrofuran is readily absorbed by inhalation, with uptake ratios in humans shown to range from approximately 60% to 80% (EPA, 2012). Tetrahydrofuran has been shown to widely distribute from blood to various organs following inhalation in animal studies (EPA, 2012). Based on chronic studies of animals, the thymus and spleen are the organs with the highest levels of tetrahydrofuran following exposure, but it may also distribute to the brain, heart, kidney, liver, and lung, among other tissues. Tetrahydrofuran does not accumulate in organs. It rapidly decreases to background levels following cessation of exposure. Following absorption, tetrahydrofuran is oxidized by CYP450 enzymes in the liver—followed by paraoxonase 1 enzymatic hydrolysis and additional oxidation by cytosolic dehydrogenases to succinic acid—before undergoing further metabolic transformations to ultimately yield CO_2, the major terminal metabolite of tetrahydrofuran, which is eliminated in exhaled air (EPA, 2012).

Occupational studies have reported that acute and chronic inhalation exposure to tetrahydrofuran is associated with central nervous system depression (including symptoms of headaches, dizziness, tiredness, and a diminished sense of smell), respiratory tract irritation (cough, chest pain, rhinorrhea, dyspnea), haematological changes, decreased white blood cells, and effects on the liver (e.g., increased liver enzymes) and kidney (autoimmune glomerulonephritis) (EPA, 2012). Systemic effects have been observed in experimental animal studies following inhalation of tetrahydrofuran, including decreased body weight, effects on the liver (e.g., centrilobular cytomegaly, hepatocellular necrosis), haematological alteration, increased organ weights, respiratory tract irritation, and immunotoxicity (EPA, 2012). Fetal toxicity and developmental effects have also been observed in animals following chronic inhalation exposures, including intrauterine mortality and reduced body weight (Environment and Climate Change Canada and Health Canada, 2018; EPA, 2012; OECD, 2000). Tetrahydrofuran is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (Group 2B) based on sufficient evidence of carcinogenicity in experimental animals and inadequate evidence in humans (IARC, 2019).

The Government of Canada has conducted a science-based screening assessment under the Chemicals Management Plan to determine whether tetrahydrofuran presents or may present a risk to the environment or human health as per the criteria set out in section 64 of CEPA 1999 (Canada, 1999; Environment and Climate Change Canada and Health Canada, 2018). The assessment proposes to conclude that tetrahydrofuran is toxic under CEPA 1999 as it is considered harmful to human health (Environment and Climate Change Canada and Health Canada, 2018).

Tetrahydrofuran is also part of a larger class of volatile organic compounds (VOCs) that, as a group, pose environmental and health concerns because of their contributions 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).

Tetrahydrofuran was analyzed in whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 5 (2016–2017). Data are presented as µg/L blood. Finding a measurable amount of tetrahydrofuran in blood can be an indicator of a recent exposure to tetrahydrofuran and does not necessarily mean that an adverse health effect will occur.

Tetrahydrofuran 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 indoor air sampling 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@canada.ca.

Table 15.12.1

Tetrahydrofuran — 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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
5 (2016–2017)	2548	10.8 (8.3–13.8)	—	<LOD	<LOD	<LOD[E] (<LOD–0.019)	0.018 (0.017–0.019)
Males, 12–79 years							
5 (2016–2017)	1265	10.7[E] (6.6–17.0)	—	<LOD	<LOD	<LOD[E] (<LOD–0.019)	0.019 (<LOD–0.024)
Females, 12–79 years							
5 (2016–2017)	1283	10.8 (8.3–13.9)	—	<LOD	<LOD	<LOD	0.018 (0.017–0.019)
12–19 years							
5 (2016–2017)	827	9.8[E] (6.0–15.8)	—	<LOD	<LOD	<LOD	0.016 (<LOD–0.018)
20–39 years							
5 (2016–2017)	582	14.4[E] (9.1–22.1)	—	<LOD	<LOD	0.017 (<LOD–0.020)	0.019[E] (<LOD–0.026)
40–59 years							
5 (2016–2017)	561	7.0[E] (4.7–10.4)	—	<LOD	<LOD	<LOD	0.017 (0.015–0.019)
60–79 years							
5 (2016–2017)	578	11.3 (9.6–13.2)	—	<LOD	<LOD	0.015 (<LOD–0.020)	0.020 (0.016–0.024)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.015 µg/L.

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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15.13 TOLUENE

Toluene (CASRN 108-88-3) is a colourless liquid classified as a volatile organic compound (VOC). It is produced commercially, primarily through the conversion of petroleum to gasoline and other fuels or when recovered as a by-product in coke ovens and styrene-manufacturing industries (ATSDR, 2017; Environment Canada and Health Canada, 1992).

Toluene is used widely as an industrial solvent and as an intermediate in the production of a variety of chemicals. Major uses have included the manufacture of benzene, benzene derivatives, trinitrotoluene and toluene diisocyanate, and in the blending of gasoline fuels as octane boosters (ATSDR, 2017; 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, 2017; Environment Canada and Health Canada, 1992; Health Canada, 2018). 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 (Environment Canada and Health Canada, 1992). Primary anthropogenic sources of atmospheric toluene include the volatilization of petroleum fuels, toluene-based solvents, and thinners, motor vehicle exhaust, and the off-gassing of toluene from some building materials and consumer and automotive products (ATSDR, 2017; 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, 2017; CCME, 2004; Environment Canada and Health Canada, 1992).

The most common route of exposure to toluene for the general population is inhalation; exposure is attributed predominantly to indoor air because indoor levels generally exceed outdoor levels and because people typically spend more time indoors than out (Health Canada, 2010a; Health Canada, 2010b; Health Canada, 2011; Health Canada 2012a; Health Canada, 2012b; Health Canada, 2013). Sources of toluene in indoor air include stored combustion equipment, various building materials, automotive products, furniture, candles, and mothballs (Won et al., 2013; Won et al., 2014; Won et al., 2015; Won and Yang, 2012). Toluene is also found in tobacco smoke; regular smoking in the home is a predictor of toluene in indoor air (Health Canada, 2012b; Wheeler et al., 2013). Smokers have considerably higher exposure to toluene than non-smokers (ATSDR, 2017). 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). 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, 2017; 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, 2017). Up to 20% of absorbed toluene is exhaled unchanged; less than 1% is excreted unchanged in the urine (ATSDR, 2017; Donald et al., 1991). The elimination of toluene following inhalation has estimated half-lives ranging from less than three minutes to 12 hours in blood and from 0.5 to 3 days in human subcutaneous adipose tissues (ATSDR, 2017). The level of toluene in blood is the most accurate biomarker of exposure and is reflective of recent exposure (ATSDR, 2017; 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, 2018; Health Canada, 2011; Health Canada, 2012b; IARC, 1999). Acute inhalation exposure has generally been associated with reversible neurological symptoms; chronic exposure is associated with impaired neurological function, including cognitive and neuromuscular performance as well as negative effects on colour vision and hearing (ATSDR, 2017; CCOHS, 2018; 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, 2017; 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, 2017; Bowen and Hannigan, 2006; Donald et al., 1991; Yücel et al., 2008). The International Agency for Research on Cancer (IARC) has classified toluene as Group 3, not classifiable as to its carcinogenicity to humans (IARC, 1999).

Under the *Canadian Environmental Protection Act, 1999* Health Canada and Environment Canada concluded that toluene is not a concern for human life or health based on measured environmental concentrations (Environment Canada and Health Canada, 1992). Toluene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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), as well as from on-road (Canada, 2003; Canada, 2015) and off-road (Canada, 2013; Canada, 2017) engines and vehicles.

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 end points reported in human occupational studies.

Toluene was analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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, 2013; Wheeler et al., 2013; Zhu et al. 2013), cycle 3 (2012–2013) (Statistics Canada, 2015), 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@canada.ca.

Table 15.13.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2449	99.5 (98.9–99.8)	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	100 (99.6–100)	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)
5 (2016–2017)	2558	99.2 (94.9–99.9)	0.085 (0.070–0.10)	0.029 (0.023–0.036)	0.071 (0.057–0.084)	0.37 (0.29–0.44)	0.55 (0.46–0.64)
Males, 12–79 years							
3 (2012–2013)	1224	99.4 (98.5–99.7)	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	99.9 (99.1–100)	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)
5 (2016–2017)	1270	99.8 (98.9–100)	0.097 (0.082–0.12)	0.029 (0.024–0.035)	0.078 (0.065–0.090)	0.42 (0.29–0.55)	0.64[E] (0.40–0.87)
Females, 12–79 years							
3 (2012–2013)	1225	99.6 (99.0–99.9)	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	100	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)
5 (2016–2017)	1288	98.6 (90.8–99.8)	0.074 (0.058–0.094)	0.028 (0.020–0.036)	0.061 (0.047–0.076)	0.28[E] (0.16–0.40)	0.48 (0.37–0.60)
12–19 years							
3 (2012–2013)	732	99.6 (97.2–99.9)	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	100	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)
5 (2016–2017)	832	99.5 (95.1–99.9)	0.065 (0.053–0.080)	0.028 (0.023–0.032)	0.059 (0.046–0.072)	0.17 (0.11–0.22)	0.25 (0.17–0.34)
20–39 years							
3 (2012–2013)	533	99.2 (97.0–99.8)	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	100	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)
5 (2016–2017)	587	98.5 (89.3–99.8)	0.086 (0.066–0.11)	0.026 (0.018–0.034)	0.075[E] (0.044–0.11)	0.35 (0.25–0.44)	0.51 (0.42–0.60)
40–59 years							
3 (2012–2013)	594	99.9 (99.6–100)	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	100 (99.6–100)	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)
5 (2016–2017)	562	99.6 (96.5–100)	0.091 (0.073–0.11)	0.029 (0.019–0.040)	0.072 (0.060–0.085)	0.45 (0.29–0.60)	0.65[E] (0.36–0.93)
60–79 years							
3 (2012–2013)	590	99.1 (89.3–99.9)	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	99.8 (98.6–100)	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)
5 (2016–2017)	577	99.3 (96.3–99.9)	0.086 (0.071–0.10)	0.031 (0.025–0.036)	0.069 (0.059–0.079)	0.39 (0.28–0.49)	0.54 (0.43–0.64)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.011, 0.011, and 0.012 µg/L, respectively.

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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15.14 TRICHLORO-ETHYLENE

Trichloroethylene (CASRN 79-01-6) is a colourless liquid classified as a volatile organic compound (VOC). It has been produced commercially by chlorinating 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 has continued to be imported for use primarily as a solvent for the vapour-degreasing and cold-cleaning of metal parts. Smaller amounts are used 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). The majority of anthropogenic releases enter the atmosphere, but it can also enter the environment via wastewater during the production, use, and disposal of trichloroethylene and trichloroethylene-containing products. A small amount of trichloroethylene is produced naturally in the environment by marine algae (Abrahamsson et al., 1995).

The most common exposure route 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, ambient 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 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 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 direct measurement in blood and breath (ATSDR, 1997; IARC, 1995). Measurement of the metabolites TCA and TCOH in blood and urine is 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 (ATSDR, 1997; Health Canada, 2005; IARC, 1995). Trichloroethylene exposure is also associated with narcotic and anesthetic effects that increase 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). The International Agency for Research on Cancer (IARC) has classified trichloroethylene as carcinogenic to humans (Group 1) based on sufficient evidence for cancer of the kidney in humans and strong support from experimental animal studies (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 of the impact of trichloroethylene exposure on humans and the environment, and concluded that trichloroethylene may enter the environment in quantities or under conditions that may constitute a danger 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). Trichloroethylene is part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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, 2013d).

A guideline establishing the maximum acceptable concentration for trichloroethylene in Canadian drinking water was developed by Health Canada in collaboration with the Federal-Provincial-Territorial Committee on 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), cycle 4 (2014–2015), and cycle 5 (2016–2017). 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).

Table 15.14.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2474	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2527	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2576	F	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1240	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1281	F	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1234	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1276	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1295	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	746	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	0	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	835	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	543	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	591	F	—	<LOD	<LOD	<LOD	<LOD

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
40–59 years							
3 (2012–2013)	594	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	569	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	591	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	589	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	581	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.027, 0.027, and 0.010 µg/L, respectively.

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

F Data are too unreliable to be published.

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15.15 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 cycles 3, 4, and 5 of the Canadian Health Measures Survey (CHMS), four THMs were measured: bromodichloromethane, dibromochloromethane, bromoform, and chloroform. Each of these compounds consists of three halogen groups attached to a single carbon atom; all 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 15.15.1

Trihalomethanes measured in the Canadian Health Measures Survey cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017)

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 anesthetic 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 in 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 sources. 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 that cause waterborne 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, 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, through inhalation during showering and bathing, and by skin absorption during bathing and swimming (CDC, 2009; Environment Canada and Health Canada, 2001; 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, 2005). 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, 2005). Estimated half-lives for THMs in the body generally range from 1.5 hours to six hours; in a study of orally exposed laboratory animals, about 95% of absorbed bromodichloromethane was eliminated from the body in eight hours (ATSDR, 1989; Health Canada, 2006; WHO, 2005). Absorbed THMs are metabolized primarily to carbon dioxide and/or carbon monoxide, and 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. Acute inhalation exposure has been associated with reddening of the face (Health Canada, 2006; IPCS, 2000; WHO, 2005). Acute high-level inhalation and oral exposures to these disinfection by-products in laboratory animals induce general narcotic and anesthetic effects that increase in severity with exposure level, and are generally reversible following cessation of exposure (Health Canada, 2006; IPCS, 2000; WHO, 2005). Similar effects can be expected to occur in humans. Some animal studies indicate that exposure to high doses of bromoform or dibromochloromethane may also lead to liver and kidney injury within a short period of time (ATSDR, 2005). THMs containing bromine, such as bromodichloromethane, may be more toxic than chloroform and other chlorine-containing disinfection by-products according to experimental animal studies (Health Canada, 2006). Studies in humans and animals suggest a link between reproductive effects and exposure to high levels of trihalomethanes; however, the evidence is inconclusive (Health Canada, 2006). Chronic exposures to THMs in drinking water are weakly and inconsistently associated with cancers of the liver, kidney, colon, rectum, brain, pancreas, and bladder in human epidemiological studies (Health Canada, 2006; IPCS, 2000; WHO, 2005). 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, 2005). 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 was 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 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, 1978; 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 chloroform, bromoform, dibromochloromethane, and bromodichloromethane) 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.

Bromodichloromethane, dibromochloromethane, bromoform, and chloroform were analyzed in the whole blood of CHMS cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017) 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.

Bromodichloromethane, dibromochloromethane, bromoform, and chloroform were also analyzed in indoor air from households of CHMS participants in cycle 2 (2009–2011) (Zhu et al., 2013a, 2013b), 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@canada.ca.

Table 15.15.2

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2499	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2527	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2576	11.3[E] (5.9–20.4)	—	<LOD	<LOD	<LOD[E] (<LOD–0.0078)	0.0076[E] (<LOD–0.011)
Males, 12–79 years							
3 (2012–2013)	1245	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1281	10.2[E] (5.4–18.5)	—	<LOD	<LOD	F	0.0074[E] (<LOD–0.011)
Females, 12–79 years							
3 (2012–2013)	1254	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1276	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1295	12.3[E] (6.0–23.3)	—	<LOD	<LOD	0.0055[E] (<LOD–0.0080)	0.0079[E] (<LOD–0.011)
12–19 years							
3 (2012–2013)	744	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	835	12.7[E] (7.1–21.6)	—	<LOD	<LOD	<LOD	0.0087 (0.0059–0.012)
20–39 years							
3 (2012–2013)	556	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	591	12.3[E] (6.5–21.9)	—	<LOD	<LOD	0.0050[E] (<LOD–0.0083)	F
40–59 years							
3 (2012–2013)	595	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	569	F	—	<LOD	<LOD	<LOD	0.0068[E] (<LOD–0.010)
60–79 years							
3 (2012–2013)	604	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	589	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	581	12.3[E] (5.9–23.9)	—	<LOD	<LOD	0.0051[E] (<LOD–0.0079)	0.0076[E] (<LOD–0.011)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.012, 0.012, and 0.005 µg/L, respectively.

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 are too unreliable to be published.

Table 15.15.3

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2527	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	2499	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2576	5.8[E] (3.2–10.5)	—	<LOD	<LOD	<LOD	<LOD[E] (<LOD–0.0061)
Males, 12–79 years							
3 (2012–2013)	1263	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1233	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1281	7.8[E] (4.1–14.4)	—	<LOD	<LOD	<LOD	0.0051[E] (<LOD–0.0070)
Females, 12–79 years							
3 (2012–2013)	1264	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1266	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1295	3.9[E] (2.0–7.6)	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	757	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	704	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	835	7.5[E] (4.5–12.3)	—	<LOD	<LOD	<LOD	0.0059 (<LOD–0.0076)
20–39 years							
3 (2012–2013)	557	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	596	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	591	F	—	<LOD	<LOD	<LOD	0.0052[E] (<LOD–0.0083)
40–59 years							
3 (2012–2013)	604	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	617	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	569	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	609	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	582	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	581	F	—	<LOD	<LOD	<LOD	<LOD[E] (<LOD–0.0058)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.0070, 0.0070, and 0.005 µg/L, respectively.

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 are too unreliable to be published.

Table 15.15.4

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2496	6.4[E] (3.4–11.8)	—	<LOD	<LOD	<LOD	0.010[E] (<LOD–0.015)
4 (2014–2015)	2527	2.6[E] (1.6–4.3)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	2576	3.4[E] (2.0–5.8)	—	<LOD	<LOD	<LOD	<LOD
Males, 12–79 years							
3 (2012–2013)	1244	6.5[E] (3.4–11.8)	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	1251	3.5[E] (1.7–7.1)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1281	3.7[E] (2.1–6.3)	—	<LOD	<LOD	<LOD	<LOD
Females, 12–79 years							
3 (2012–2013)	1252	6.4[E] (3.0–13.1)	—	<LOD	<LOD	<LOD	<LOD[E] (<LOD–0.013)
4 (2014–2015)	1276	1.7[E] (1.0–2.8)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	1295	F	—	<LOD	<LOD	<LOD	<LOD
12–19 years							
3 (2012–2013)	744	4.7[E] (2.3–9.3)	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	713	3.9[E] (1.9–7.9)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	835	2.6[E] (1.4–4.9)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
3 (2012–2013)	554	F	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	600	F	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	591	2.4[E] (1.6–3.5)	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
3 (2012–2013)	595	7.8[E] (4.3–14.0)	—	<LOD	<LOD	<LOD	<LOD
4 (2014–2015)	625	1.5[E] (0.7–3.2)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	569	F	—	<LOD	<LOD	<LOD	<LOD
60–79 years							
3 (2012–2013)	603	F	—	<LOD	<LOD	<LOD	F
4 (2014–2015)	589	2.1[E] (1.1–3.7)	—	<LOD	<LOD	<LOD	<LOD
5 (2016–2017)	581	3.1[E] (1.6–5.9)	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.010, 0.010, and 0.013 µg/L, respectively.

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 are too unreliable to be published.

Table 15.15.5

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2527	19.6[E] (13.2–28.2)	—	<LOD	<LOD	0.021 (0.016–0.026)	0.029 (0.019–0.038)
4 (2014–2015)	2527	26.3[E] (16.1–39.8)	—	<LOD	<LOD	0.028[E] (<LOD–0.043)	0.043[E] (0.022–0.064)
5 (2016–2017)	2510	69.6 (55.9–80.5)	0.011 (0.0077–0.015)	<LOD	0.0098[E] (<LOD–0.014)	0.042[E] (0.023–0.061)	0.067[E] (0.035–0.10)
Males, 12–79 years							
3 (2012–2013)	1263	18.1 (12.5–25.5)	—	<LOD	<LOD	0.021 (0.015–0.027)	0.035[E] (0.018–0.052)
4 (2014–2015)	1251	25.0[E] (15.0–38.6)	—	<LOD	<LOD	F	0.046[E] (0.022–0.069)
5 (2016–2017)	1245	70.5 (55.6–82.0)	0.011 (0.0079–0.014)	<LOD	0.0099[E] (<LOD–0.014)	0.041[E] (0.026–0.056)	0.061[E] (0.036–0.086)
Females, 12–79 years							
3 (2012–2013)	1264	21.2[E] (13.3–32.0)	—	<LOD	<LOD	0.021 (0.016–0.027)	0.028 (0.019–0.037)
4 (2014–2015)	1276	27.6[E] (16.4–42.6)	—	<LOD	<LOD	0.030[E] (0.016–0.045)	0.039[E] (0.016–0.062)
5 (2016–2017)	1265	68.7 (54.1–80.3)	0.011 (0.0073–0.015)	<LOD	0.0097[E] (<LOD–0.015)	0.042[E] (0.017–0.067)	0.075[E] (0.036–0.11)
12–19 years							
3 (2012–2013)	757	18.1[E] (11.9–26.6)	—	<LOD	<LOD	0.020[E] (<LOD–0.028)	0.031[E] (<LOD–0.049)
4 (2014–2015)	713	27.9[E] (17.2–41.8)	—	<LOD	<LOD	0.028[E] (0.017–0.038)	0.040[E] (0.015–0.066)
5 (2016–2017)	810	70.0 (54.4–82.0)	0.010 (0.0074–0.014)	<LOD	0.0096[E] (<LOD–0.014)	0.039[E] (0.020–0.058)	0.062 (0.046–0.079)
20–39 years							
3 (2012–2013)	557	22.9[E] (12.9–37.4)	—	<LOD	<LOD	0.023 (0.016–0.029)	0.036[E] (0.015–0.058)
4 (2014–2015)	600	30.2[E] (17.7–46.5)	—	<LOD	<LOD	0.030[E] (0.016–0.045)	F
5 (2016–2017)	577	68.7 (51.6–81.9)	0.011[E] (0.0074–0.016)	<LOD	0.011[E] (<LOD–0.017)	0.043[E] (0.023–0.062)	0.087[E] (0.026–0.15)
40–59 years							
3 (2012–2013)	604	17.2[E] (9.8–28.3)	—	<LOD	<LOD	0.019 (<LOD–0.025)	0.027 (0.019–0.036)
4 (2014–2015)	625	21.9[E] (11.8–36.9)	—	<LOD	<LOD	F	0.046[E] (0.024–0.067)
5 (2016–2017)	556	71.5 (59.1–81.3)	0.010 (0.0074–0.014)	<LOD	0.0091 (0.0061–0.012)	0.041[E] (0.016–0.066)	0.067[E] (0.021–0.11)
60–79 years							
3 (2012–2013)	609	19.5[E] (12.4–29.2)	—	<LOD	<LOD	0.020[E] (<LOD–0.027)	0.028[E] (<LOD–0.041)
4 (2014–2015)	589	26.7[E] (15.2–42.5)	—	<LOD	<LOD	0.027[E] (<LOD–0.040)	0.037[E] (0.019–0.056)
5 (2016–2017)	567	67.9 (51.8–80.6)	0.011 (0.0078–0.015)	<LOD	0.011[E] (<LOD–0.016)	0.043[E] (0.024–0.062)	0.060[E] (0.036–0.085)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.014, 0.014, and 0.006 µg/L, respectively.

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 are too unreliable to be published.

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15.16 XYLENES

Xylenes (CASRN 1330-20-7) are classified as volatile organic compounds (VOCs) (ATSDR, 2007; CCOHS, 2018; 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 6% to 15% ethylbenzene (CCOHS, 2018). Xylenes are primarily produced either directly or as by-products of olefin manufacturing or petroleum and coal refining (ATSDR, 2007; Environment Canada and Health Canada, 1993).

Xylene has 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). Xylene 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 xylene include volatilization of petroleum fuels and xylene-based solvents and thinners, gasoline use and motor vehicle exhaust, off-gassing from certain building materials, and consumer and automotive products (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 implemented, along with changes in industrial and consumer use patterns and increases in fuel efficiency, releases of VOCs, including xylenes, are expected to continue to decline.

The most common route of exposure to xylenes in the general population is inhalation; exposure is attributed predominantly to indoor air because indoor air levels generally exceed outdoor levels, and because people typically spend more time indoors than out (Environment Canada and Health Canada, 1993; Health Canada, 2010a; Health Canada, 2010b; Health Canada, 2012; Health Canada, 2013). Cigarette smoking may significantly increase indoor air levels. Cigarette smoke is thought to be a major contributor to the total source of xylene exposure in smokers (ATSDR, 2007). Xylene levels in air have been shown to be higher in homes that have a garage on the property, have a higher number of occupants, have had recent renovations, and where fragrances or paint remover have been recently used (Wheeler et al., 2013). Sources of xylenes in indoor air include stored combustion equipment, various building materials, and consumer products (Won et al., 2013; Won et al., 2014; Won et al., 2015; Won and Yang, 2012). Additional exposure may result 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). Since xylenes are present as a mixture in gasoline and commercial products, the general population is expected to be exposed to xylenes primarily as a mixture rather than to individual isomers (ATSDR, 2007).

Xylene is rapidly absorbed by all routes of exposure and distributed throughout the body, primarily into adipose tissues and tissues with higher lipid content, such as

the liver and brain (ATSDR, 2007; EPA, 2003; Health Canada, 2014). Elimination of xylene from blood and most tissue compartments following inhalation is generally rapid; in humans, it has an estimated half-life in the range of 1–20 hours (ATSDR, 2007). Xylene is metabolized in humans primarily by microsomal enzymes in the liver (ATSDR, 2007). The major route of excretion of absorbed xylene in the blood and body is through 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 skin contact. In humans, xylene 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, 2018). Acute inhalation exposure has been associated with reversible neurological symptoms; 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 containing xylene 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 with solvent abuse (ATSDR, 2007; EPA, 2003; IPCS, 1997). Due to inadequate data, xylene is not classifiable as to its carcinogenicity in humans according to Environment Canada and Health Canada (Group IV) and the International Agency for Research on Cancer (IARC; Group 3) (Environment Canada and Health Canada, 1993; IARC, 1999).

Under the *Canadian Environmental Protection Act, 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 are part of a larger class of VOCs that, as a group, pose environmental and health concerns because of their contributions to the formation of smog. The Government of Canada has proposed and implemented 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), as well as from on-road (Canada, 2003; Canada, 2015) and off-road (Canada, 2013; Canada, 2017) engines and vehicles. In 2017, Health Canada published an Indoor Air Reference Level (IARL) for xylenes (Health Canada, 2017).

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 total xylenes that is protective of human health, as well as an aesthetic objective for total xylenes based on its odour threshold (Health Canada 2014). The guideline was developed based on adverse neurological effects reported in experimental animals.

Xylenes were analyzed in the whole blood of Canadian Health Measures Survey (CHMS) participants aged 12–79 years in cycle 3 (2012–2013), cycle 4 (2014–2015), and cycle 5 (2016–2017). Data are presented as µg/L blood for *o*-xylene and the sum of *m*-xylene and *p*-xylene. Finding a measurable quantity 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, 2013; Wheeler et al., 2013; Zhu et al., 2013), cycle 3 (2012–2013) (Statistics Canada, 2015), 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@canada.ca.

Table 15.16.1

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2326	84.8 (78.4–89.5)	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	90.2 (83.4–94.4)	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)
5 (2016–2017)	2576	98.2 (94.0–99.5)	0.069 (0.054–0.088)	0.026 (0.019–0.033)	0.065 (0.051–0.079)	0.23 (0.16–0.30)	0.39[E] (0.22–0.56)
Males, 12–79 years							
3 (2012–2013)	1172	85.6 (78.6–90.6)	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	89.3 (82.4–93.7)	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)
5 (2016–2017)	1281	98.0 (88.7–99.7)	0.078 (0.061–0.099)	0.029 (0.021–0.038)	0.075 (0.059–0.090)	0.26[E] (0.15–0.37)	0.44[E] (0.28–0.61)
Females, 12–79 years							
3 (2012–2013)	1154	84.0 (76.9–89.2)	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	91.0 (83.5–95.3)	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)
5 (2016–2017)	1295	98.4 (94.4–99.6)	0.061 (0.045–0.083)	0.024[E] (0.014–0.034)	0.057 (0.041–0.072)	0.17[E] (0.11–0.24)	0.28[E] (0.13–0.44)
12–19 years							
3 (2012–2013)	701	80.6 (68.1–89.0)	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	91.1 (80.1–96.3)	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)
5 (2016–2017)	835	96.4 (86.0–99.2)	0.055 (0.040–0.076)	0.025 (0.016–0.034)	0.058 (0.045–0.070)	0.14 (0.11–0.18)	0.17 (0.12–0.21)
20–39 years							
3 (2012–2013)	500	85.2 (79.6–89.5)	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	88.8 (78.0–94.6)	0.059 (0.046–0.076)	<LOD	0.055 (0.037–0.073)	0.16 (0.12–0.19)	F
5 (2016–2017)	591	96.6 (86.8–99.2)	0.064 (0.045–0.092)	0.020[E] (0.0074–0.032)	0.063[E] (0.040–0.087)	0.24 (0.17–0.31)	0.37[E] (0.15–0.58)
40–59 years							
3 (2012–2013)	559	87.2 (80.3–91.8)	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	90.1 (81.6–95.0)	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)
5 (2016–2017)	569	99.6 (97.4–99.9)	0.078 (0.064–0.095)	0.031 (0.022–0.039)	0.071 (0.062–0.080)	0.26[E] (0.14–0.39)	0.43[E] (0.24–0.62)
60–79 years							
3 (2012–2013)	566	82.4 (72.0–89.6)	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	91.9 (86.7–95.2)	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)
5 (2016–2017)	581	99.2 (88.6–99.9)	0.071 (0.055–0.092)	0.029 (0.024–0.033)	0.060 (0.045–0.075)	0.22[E] (0.13–0.30)	0.41[E] (0.20–0.61)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.023, 0.023, and 0.005 µg/L, respectively.

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 are too unreliable to be published.

Table 15.16.2

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), cycle 4 (2014–2015), and cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 12–79 years							
3 (2012–2013)	2336	60.0 (40.0–77.2)	—	<LOD	0.022[E] (0.010–0.034)	0.087 (0.061–0.11)	0.11 (0.083–0.14)
4 (2014–2015)	2428	67.1 (54.7–77.6)	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)
5 (2016–2017)	2556	91.5 (85.7–95.1)	0.021 (0.018–0.024)	<LOD	0.020 (0.017–0.022)	0.068 (0.048–0.088)	0.10[E] (0.056–0.15)
Males, 12–79 years							
3 (2012–2013)	1164	61.8 (42.3–78.2)	—	<LOD	0.022[E] (0.0097–0.033)	0.088 (0.061–0.11)	0.12 (0.075–0.16)
4 (2014–2015)	1198	69.6 (55.0–81.1)	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)
5 (2016–2017)	1274	92.7 (88.8–95.3)	0.024 (0.020–0.029)	0.0072 (<LOD–0.0094)	0.021 (0.018–0.025)	0.083 (0.054–0.11)	0.14[E] (0.069–0.21)
Females, 12–79 years							
3 (2012–2013)	1172	58.2 (37.0–76.7)	—	<LOD	0.022[E] (0.011–0.034)	0.081 (0.052–0.11)	0.11 (0.082–0.14)
4 (2014–2015)	1230	64.6 (52.3–75.3)	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)
5 (2016–2017)	1282	90.4 (79.8–95.7)	0.018 (0.015–0.023)	<LOD	0.018 (0.016–0.021)	0.051 (0.035–0.068)	0.082[E] (0.037–0.13)
12–19 years							
3 (2012–2013)	692	51.2[E] (32.4–69.7)	—	<LOD	F	0.057 (0.041–0.072)	0.075 (0.053–0.098)
4 (2014–2015)	687	67.6 (49.3–81.8)	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)
5 (2016–2017)	829	89.7 (83.6–93.7)	0.018 (0.015–0.021)	<LOD	0.018 (0.014–0.022)	0.045 (0.033–0.058)	0.066 (0.042–0.089)
20–39 years							
3 (2012–2013)	515	58.8 (37.2–77.5)	—	<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	56.4 (38.8–72.4)	0.012 (0.0090–0.017)	<LOD	0.012[E] (<LOD–0.018)	0.046 (0.036–0.057)	F
5 (2016–2017)	584	88.7 (74.5–95.4)	0.020 (0.015–0.025)	<LOD	0.019 (0.015–0.023)	0.069 (0.047–0.090)	0.097[E] (0.048–0.15)
40–59 years							
3 (2012–2013)	565	66.6 (43.5–83.8)	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	73.1 (62.2–81.7)	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)
5 (2016–2017)	565	94.7 (88.5–97.6)	0.023 (0.019–0.026)	0.0075 (<LOD–0.010)	0.020 (0.015–0.024)	0.066[E] (0.039–0.093)	0.13[E] (0.048–0.21)
60–79 years							
3 (2012–2013)	564	55.1 (35.8–72.9)	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	74.0 (66.2–80.5)	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)
5 (2016–2017)	578	92.0 (87.6–94.9)	0.022 (0.018–0.027)	0.0069 (<LOD–0.0094)	0.021 (0.018–0.023)	0.080 (0.054–0.11)	0.13[E] (0.063–0.20)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LODs for cycles 3, 4, and 5 are 0.0090, 0.0090, and 0.006 µg/L, respectively.

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 are 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).

Table A-1
Limits of detection

Chemical	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Metals and trace elements in blood					
Lead	0.02 µg/dL	0.1 µg/dL	0.16 µg/dL	0.16 µg/dL	0.17 µg/dL
Cadmium	0.04 µg/L	0.04 µg/L	0.080 µg/L	0.080 µg/L	0.097 µg/L
Chromium (VI)[a]	—	—	—	—	0.12 µg/L
Selenium	8 µg/L	20 µg/L	—	—	32 µg/L
Mercury					
Mercury (total)	0.1 µg/L	0.1 µg/L	0.42 µg/L	0.42 µg/L	0.20 µg/L
Methylmercury	—	—	0.19 µg/L	0.19 µg/L	0.19 µg/L
Mercury (inorganic)	0.4 µg/L	—	—	—	0.22 µg/L
Metals and trace elements in urine					
Boron	—	—	—	—	160 µg/L
Cadmium	0.09 µg/L	0.07 µg/L	—	—	0.066 µg/L
Arsenic (speciated)					
Arsenate	—	0.8 µg As/L	0.75 µg As/L	0.75 µg As/L	0.14 µg As/L
Arsenite	—	0.8 µg As/L	0.75 µg As/L	0.75 µg As/L	0.25 µg As/L
Arsenocholine and arsenobetaine	—	0.8 µg As/L	0.75 µg As/L	0.75 µg As/L	0.10 µg As/L
Dimethylarsinic acid (DMA)	—	0.8 µg As/L	0.75 µg As/L	0.75 µg As/L	0.14 µg As/L
Monomethylarsonic acid (MMA)	—	0.8 µg As/L	0.75 µg As/L	0.75 µg As/L	0.13 µg As/L

Chemical	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Metals and trace elements in hair					
Aluminum	—	—	—	—	0.10 µg/g
Antimony	—	—	—	—	0.0020 µg/g
Arsenic	—	—	—	—	0.0050 µg/g
Barium	—	—	—	—	0.030 µg/g
Beryllium	—	—	—	—	0.030 µg/g
Bismuth	—	—	—	—	0.010 µg/g
Cadmium	—	—	—	—	0.010 µg/g
Chromium	—	—	—	—	0.020 µg/g
Cobalt	—	—	—	—	0.0010 µg/g
Copper	—	—	—	—	0.10 µg/g
Lead	—	—	—	—	0.0050 µg/g
Lithium	—	—	—	—	0.010 µg/g
Manganese	—	—	—	—	0.020 µg/g
Mercury (total)	—	—	—	—	0.0050 µg/g
Molybdenum	—	—	—	—	0.0020 µg/g
Nickel	—	—	—	—	0.020 µg/g
Platinum	—	—	—	—	0.0010 µg/g
Selenium	—	—	—	—	0.010 µg/g
Silver	—	—	—	—	0.010 µg/g
Tellurium	—	—	—	—	0.0020 µg/g
Thallium	—	—	—	—	0.00020 µg/g
Thorium	—	—	—	—	0.10 µg/g
Uranium	—	—	—	—	0.0015 µg/g
Vanadium	—	—	—	—	0.0010 µg/g
Zinc	—	—	—	—	5.0 µg/g
Self-care and consumer product chemicals					
Bisphenol A (BPA)	0.2 µg/L	0.2 µg/L	0.23 µg/L	0.23 µg/L	0.32 µg/L
Parabens					
Methyl paraben	—	—	1.3 µg/L	1.3 µg/L	1.3 µg/L
Ethyl paraben	—	—	0.90 µg/L	0.90 µg/L	0.90 µg/L
Propyl paraben	—	—	0.30 µg/L	0.30 µg/L	0.30 µg/L
Butyl paraben	—	—	0.30 µg/L	0.30 µg/L	0.30 µg/L
Nicotine					
Cotinine	1 µg/L	1 µg/L	1.1 µg/L	1.1 µg/L	1.1 µg/L
Acrylamide					
Acrylamide haemoglobin adduct	—	—	11 pmol/g Hb	11 pmol/g Hb	11 pmol/g Hb
Glycidamide haemoglobin adduct	—	—	23 pmol/g Hb	23 pmol/g Hb	23 pmol/g Hb

Chemical	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Per- and polyfluoroalkyl substances					
Perfluorobutanoic acid (PFBA)	—	0.5 µg/L	—	—	0.075 µg/L
Perfluorobutane sulfonate (PFBS)	—	0.4 µg/L	—	—	0.066 µg/L
Perfluorohexanoic acid (PFHxA)	—	0.1 µg/L	—	—	0.084 µg/L
Perfluorohexane sulfonate (PFHxS)	0.3 µg/L	0.2 µg/L	—	—	0.063 µg/L
Perfluorooctanoic acid (PFOA)	0.3 µg/L	0.1 µg/L	—	—	0.066 µg/L
Perfluorooctane sulfonate (PFOS)	0.3 µg/L	0.3 µg/L	—	—	0.43 µg/L
Perfluorononanoic acid (PFNA)	—	0.2 µg/L	—	—	0.13 µg/L
Perfluorodecanoic acid (PFDA)	—	0.1 µg/L	—	—	0.092 µg/L
Perfluoroundecanoic acid (PFUnDA)	—	0.09 µg/L	—	—	0.12 µg/L
Pesticides					
Ethylene thiourea (ETU)	—	—	—	—	0.033 µg/L
Organophosphate pesticides					
Dimethylphosphate (DMP)	0.8 µg/L	1 µg/L	—	—	0.58 µg/L
Dimethylthiophosphate (DMTP)	0.6 µg/L	0.6 µg/L	—	—	0.44 µg/L
Dimethyldithiophosphate (DMDTP)	0.09 µg/L	0.3 µg/L	—	—	0.093 µg/L
Diethylphosphate (DEP)	0.5 µg/L	1 µg/L	—	—	0.29 µg/L
Diethylthiophosphate (DETP)	0.08 µg/L	0.3 µg/L	—	—	0.13 µg/L
Diethyldithiophosphate (DEDTP)	0.06 µg/L	0.3 µg/L	—	—	0.067 µg/L
3,5,6-Trichloro-2-pyridinol (TCPy)	—	—	0.13 µg/L	0.13 µg/L	—
Malathion dicarboxylic acid (DCA)	—	—	0.19 µg/L	0.19 µg/L	—
Acephate	—	—	0.018 µg/L	—	—
Methamidophos	—	—	0.028 µg/L	—	—
ortho-Phenylphenol (OPP)					
OPP-glucuronide	—	—	—	—	0.15 µg/L
OPP-sulfate	—	—	—	—	0.092 µg/L
Pyrethroids					
3-Phenoxybenzoic acid (3-PBA)	0.01 µg/L	0.01 µg/L	—	—	0.012 µg/L
4-Fluoro-3-phenoxybenzoic acid (4-F-3-PBA)	0.008 µg/L	0.008 µg/L	—	—	0.0060 µg/L
cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA)	0.006 µg/L	0.006 µg/L	—	—	0.0059 µg/L
cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA)	0.007 µg/L	0.007 µg/L	—	—	0.0045 µg/L
trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA)	0.01 µg/L	0.01 µg/L	—	—	0.0094 µg/L
Plasticizers					
Monomethyl phthalate (MMP)	5 µg/L	5 µg/L	—	—	0.21 µg/L
Monoethyl phthalate (MEP)	0.5 µg/L	0.3 µg/L	—	—	0.98 µg/L
Mono(3-carboxypropyl) phthalate (MCPP)	0.2 µg/L	0.06 µg/L	—	—	0.14 µg/L
Mono-*n*-butyl phthalate (M*n*BP)	0.2 µg/L	0.2 µg/L	—	—	0.60 µg/L
Monoisobutyl phthalate (MiBP)	—	0.1 µg/L	—	—	0.57 µg/L
Mono-3-hydroxy-*n*-butyl phthalate (3OH-MBP)	—	—	—	—	0.079 µg/L

Chemical	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Monocyclohexyl phthalate (MCHP)	0.2 µg/L	0.09 µg/L	—	—	0.25 µg/L
Monobenzyl phthalate (MBzP)	0.2 µg/L	0.05 µg/L	—	—	0.37 µg/L
Mono[2-(carboxymethyl)hexyl] phthalate (MCMHP)	—	—	—	—	0.27 µg/L
Mono(2-ethylhexyl) phthalate (MEHP)	0.2 µg/L	0.08 µg/L	—	—	0.11 µg/L
Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP)	—	—	—	—	0.28 µg/L
Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP)	0.2 µg/L	0.1 µg/L	—	—	0.17 µg/L
Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)	0.4 µg/L	0.4 µg/L	—	—	0.22 µg/L
Mono-carboxy-*n*-heptyl phthalate (MCHpP)	—	—	—	—	0.083 µg/L
Mono-*n*-octyl phthalate (MOP)	0.7 µg/L	0.3 µg/L	—	—	0.16 µg/L
Mono(carboxyisooctyl) phthalate (MCiOP)	—	—	—	—	0.30 µg/L
Monoisononyl phthalate (MiNP)	0.4 µg/L	0.3 µg/L	—	—	0.37 µg/L
Monocarboxyisononyl phthalate (MCiNP)	—	—	—	—	0.077 µg/L
Monooxoisononyl phthalate (MOiNP)	—	—	—	—	0.15 µg/L
Monohydroxyisononyl phthalate (MHiNP)	—	—	—	—	0.065 µg/L
Monoisodecyl phthalate (MiDP)	—	—	—	—	0.16 µg/L
Monooxoisodecyl phthalate (MOiDP)	—	—	—	—	0.097 µg/L
Monohydroxyisodecyl phthalate (MHiDP)	—	—	—	—	0.067 µg/L
Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH)					
trans-Cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH)	—	—	—	—	0.017 µg/L
Cyclohexane-1,2-dicarboxylic mono oxoisononyl ester (oxo-MINCH)	—	—	—	—	0.047 µg/L
Cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (OH-MINCH)	—	—	—	—	0.078 µg/L
cis-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH)	—	—	—	—	0.059 µg/L
trans-Cyclohexane-1,2-diarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH)	—	—	—	—	0.33 µg/L
Cyclohexane-1,2-dicarboxylic acid (CHDA)	—	—	—	—	0.30 µg/L
2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB)					
2,2,4-Trimethyl-1,3-pentanediol (TMPD)	—	—	—	—	1.7 µg/L
2,2,4-Trimethyl-3-hydroxy valeric acid (HTMV)	—	—	—	—	0.42 µg/L
Tri-(2-ethylhexyl) trimellitate (TEHT)					
1-Mono(2-ethylhexyl)trimellitate (1-MEHTM)	—	—	—	—	0.22 µg/L
2-Mono(2-ethylhexyl)trimellitate (2-MEHTM)	—	—	—	—	0.16 µg/L
4-Mono(2-ethylhexyl)trimellitate (4-MEHTM)	—	—	—	—	0.098 µg/L
Volatile organic compounds					
Benzene	—	—	0.0070 µg/L	0.0070 µg/L	0.022 µg/L
Carbon tetrachloride	—	—	—	—	0.005 µg/L
1,4-Dichlorobenzene	—	—	—	—	0.013 µg/L
2,5-Dimethylfuran	—	—	—	—	0.018 µg/L
Ethylbenzene	—	—	0.011 µg/L	0.011 µg/L	0.013 µg/L

Chemical	Cycle 1 (2007–2009)	Cycle 2 (2009–2011)	Cycle 3 (2012–2013)	Cycle 4 (2014–2015)	Cycle 5 (2016–2017)
Isopropylbenzene	—	—	—	—	0.010 µg/L
Methyl isobutyl ketone	—	—	—	—	0.029 µg/L
Nitrobenzene	—	—	—	—	1.1 µg/L
Styrene	—	—	0.012 µg/L	0.012 µg/L	0.011 µg/L
1,1,1,2-Tetrachloroethane	—	—	—	—	0.007 µg/L
Tetrachloroethylene	—	—	0.020 µg/L	0.020 µg/L	0.013 µg/L
Tetrahydrofuran	—	—	—	—	0.015 µg/L
Toluene	—	—	0.011 µg/L	0.011 µg/L	0.012 µg/L
Trichloroethylene	—	—	0.027 µg/L	0.027 µg/L	0.010 µg/L
Trihalomethanes					
Bromodichloromethane	—	—	0.012 µg/L	0.012 µg/L	0.005 µg/L
Dibromochloromethane	—	—	0.0070 µg/L	0.0070 µg/L	0.005 µg/L
Tribromomethane (bromoform)	—	—	0.010 µg/L	0.010 µg/L	0.013 µg/L
Trichloromethane (chloroform)	—	—	0.014 µg/L	0.014 µg/L	0.006 µg/L
Xylenes					
m-Xylene and *p*-xylene	—	—	0.023 µg/L	0.023 µg/L	0.005 µg/L
o-Xylene	—	—	0.0090 µg/L	0.0090 µg/L	0.006 µg/L
Adjustment factor					
Creatinine	3 mg/dL	4 mg/dL	5.0 mg/dL	5.0 mg/dL	5.0 mg/dL

a Chromium (VI) was measured indirectly as total chromium in red blood cells.

REFERENCES

EPA (U.S. Environmental Protection Agency) (2015). Definition and procedure for the determination of the method detection limit — Revision 1.11, Federal Regulation 40 CFR 136 Appendix B. U.S. Environmental Protection Agency, Washington, DC.

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.

Table B-1
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

For concentrations of environmental chemicals in blood and urine, data can be converted from µg/L to µmol/L using the molecular weight (MW) of the chemical and the formula:

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

For concentrations of environmental chemicals in hair, data can be converted from µg/g to µmol/g using the MW of the chemical and the formula:

Y µmol/g = X µg/g × CF, where the CF is equivalent to 1/MW.

Table B-2
Conversion factors for concentrations of environmental chemicals in blood and urine

Chemical	MW (g/mol)	CF (µg/L → µmol/L)
Metals and trace elements		
Lead	207.20	0.04826[a]
Boron	10.81	0.09251
Cadmium	112.41	0.00890
Chromium (VI)	52.00	0.01923
Selenium	78.97	0.01266
Arsenic (speciated)		
Arsenate	—	0.01335[b]
Arsenite	—	0.01335[b]
Arsenocholine and arsenobetaine	—	0.01335[b]
Dimethylarsinic acid (DMA)	—	0.01335[b]
Monomethylarsonic acid (MMA)	—	0.01335[b]
Mercury		
Mercury (total)	200.59	0.00499
Methylmercury	215.63	0.00464
Mercury (inorganic)	200.59	0.00499
Self-care and consumer product chemicals		
Bisphenol A (BPA)	228.29	0.00438
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
Nicotine		
Cotinine	176.22	0.00567
Acrylamide		
Acrylamide haemoglobin adduct	—	—[c]
Glycidamide haemoglobin adduct	—	—[c]
Per- and polyfluoroalkyl substances		
Perfluorobutanoic acid (PFBA)	214.04	0.00467
Perfluorobutane sulfonate (PFBS)	300.10	0.00333
Perfluorohexanoic acid (PFHxA)	314.05	0.00318
Perfluorohexane sulfonate (PFHxS)	400.11	0.00250
Perfluorooctanoic acid (PFOA)	414.07	0.00242
Perfluorooctane sulfonate (PFOS)	500.13	0.00200
Perfluorononanoic acid (PFNA)	464.08	0.00215
Perfluorodecanoic acid (PFDA)	514.08	0.00195
Perfluoroundecanoic acid (PFUnDA)	564.09	0.00177
Pesticides		
Ethylene thiourea (ETU)	102.16	0.00979

Chemical	MW (g/mol)	CF (µg/L → µmol/L)
Organophosphate pesticides		
Dimethylphosphate (DMP)	126.05	0.00793
Dimethylthiophosphate (DMTP)	141.10	0.00709
Dimethyldithiophosphate (DMDTP)	158.17	0.00632
Diethylphosphate (DEP)	154.10	0.00649
Diethylthiophosphate (DETP)	170.16	0.00588
Diethyldithiophosphate (DEDTP)	186.22	0.00537
3,5,6-Trichloro-2-pyridinol (TCPy)	198.43	0.00504
Malathion dicarboxylic acid (DCA)	274.24	0.00365
Acephate	183.16	0.00546
Methamidophos	141.13	0.00709
ortho-Phenylphenol (OPP)		
OPP-glucuronide	346.34	0.00289
OPP-sulfate	249.27	0.00401
Pyrethroids		
3-Phenoxybenzoic acid (3-PBA)	214.22	0.00467
4-Fluoro-3-phenoxybenzoic acid (4-F-3-PBA)	232.21	0.00431
cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DBCA)	297.97	0.00336
cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*cis*-DCCA)	209.07	0.00478
trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid (*trans*-DCCA)	209.07	0.00478
Plasticizers		
Monomethyl phthalate (MMP)	180.16	0.00555
Monoethyl phthalate (MEP)	194.18	0.00515
Mono(3-carboxypropyl) phthalate (MCPP)	252.22	0.00396
Mono-*n*-butyl phthalate (M*n*BP)	222.24	0.00450
Monoisobutyl phthalate (MiBP)	222.24	0.00450
Mono-3-hydroxy-*n*-butyl phthalate (3OH-MBP)	238.24	0.00420
Monocyclohexyl phthalate (MCHP)	248.27	0.00403
Monobenzyl phthalate (MBzP)	256.25	0.00390
Mono[2-(carboxymethyl)hexyl] phthalate (MCMHP)	308.33	0.00324
Mono(2-ethylhexyl) phthalate (MEHP)	278.34	0.00359
Mono(2-ethyl-5-carboxypentyl) phthalate (MECPP)	308.33	0.00324
Mono(2-ethyl-5-oxohexyl) phthalate (MEOHP)	292.33	0.00342
Mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)	294.34	0.00340
Mono-carboxy-*n*-heptyl phthalate (MCHpP)	308.13	0.00325
Mono-*n*-octyl phthalate (MOP)	278.34	0.00359
Mono(carboxyisooctyl) phthalate (MCiOP)	322.35	0.00310
Monoisononyl phthalate (MiNP)	292.37	0.00342
Monocarboxyisononyl phthalate (MCiNP)	336.38	0.00297
Monooxoisononyl phthalate (MOiNP)	306.35	0.00326
Monohydroxyisononyl phthalate (MHiNP)	308.37	0.00324
Monoisodecyl phthalate (MiDP)	306.18	0.00327
Monooxoisodecyl phthalate (MOiDP)	320.38	0.00312
Monohydroxyisodecyl phthalate (MHiDP)	322.40	0.00310

Chemical	MW (g/mol)	CF (µg/L → µmol/L)
Di(isononyl)cyclohexane-1,2-dicarboxylate (DINCH)		
trans-Cyclohexane-1,2-dicarboxylic mono isononyl ester (*trans*-MINCH)	298.42	0.00335
Cyclohexane-1,2-dicarboxylic mono oxoisononyl ester (oxo-MINCH)	312.40	0.00320
Cyclohexane-1,2-dicarboxylic mono hydroxyisononyl ester (OH-MINCH)	314.42	0.00318
cis-Cyclohexane-1,2-dicarboxylic mono carboxyisononyl ester (*cis*-cx-MINCH)	326.40	0.00306
trans-Cyclohexane-1,2-diarboxylic mono carboxyisononyl ester (*trans*-cx-MINCH)	326.40	0.00306
Cyclohexane-1,2-dicarboxylic acid (CHDA)	172.18	0.00581
2,2,4-Trimethyl-1,3-pentanediol diisobutyrate (TXIB)		
2,2,4-Trimethyl-1,3-pentanediol (TMPD)	146.23	0.00684
2,2,4-Trimethyl-3-hydroxy valeric acid (HTMV)	160.21	0.00624
Tri-(2-ethylhexyl) trimellitate (TEHT)		
1-Mono(2-ethylhexyl)trimellitate (1-MEHTM)	322.36	0.00310
2-Mono(2-ethylhexyl)trimellitate (2-MEHTM)	322.36	0.00310
4-Mono(2-ethylhexyl)trimellitate (4-MEHTM)	322.36	0.00310
Volatile organic compounds		
Benzene	78.11	0.01280
Carbon tetrachloride	153.81	0.00650
1,4-Dichlorobenzene	147.00	0.00680
2,5-Dimethylfuran	96.13	0.01040
Ethylbenzene	106.17	0.00942
Isopropylbenzene	120.20	0.00832
Methyl isobutyl ketone	100.16	0.00998
Nitrobenzene	123.11	0.00812
Styrene	104.15	0.00960
1,1,1,2-Tetrachloroethane	167.84	0.00596
Tetrachloroethylene	165.83	0.00603
Tetrahydrofuran	72.11	0.01387
Toluene	92.14	0.01085
Trichloroethylene	131.39	0.00761
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[d]

a For converting lead concentration from µg/dL to µmol/L.

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

c Not applicable.

d For converting creatinine concentration from mg/dL to µmol/L.

Table B-3

Conversion factors for concentrations of environmental chemicals in hair

Chemical	MW (g/mol)	CF (µg/g → µmol/g)
Metals and trace elements		
Aluminum	26.98	0.03706
Antimony	95.95	0.01042
Arsenic	121.76	0.00821
Barium	58.69	0.01704
Beryllium	74.92	0.01335
Bismuth	78.97	0.01266
Cadmium	137.33	0.00728
Chromium	107.87	0.00927
Cobalt	9.01	0.11099
Copper	200.59	0.00499
Lead	208.98	0.00479
Lithium	127.60	0.00784
Manganese	112.41	0.00890
Mercury (total)	63.55	0.01574
Molybdenum	204.38	0.00489
Nickel	52.00	0.01923
Platinum	232.04	0.00431
Selenium	58.93	0.01697
Silver	195.08	0.00513
Tellurium	238.03	0.00420
Thallium	207.20	0.00483
Thorium	50.94	0.01963
Uranium	6.94	0.14409
Vanadium	65.38	0.01530
Zinc	54.94	0.01820

APPENDIX C

CREATININE

Table C-1

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	Detection Frequency (95% CI)	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 6–79 years							
1 (2007–2009)	5515	99.9 (99.7–99.9)	83 (78–89)	27 (23–30)	93 (86–99)	210 (200–220)	250 (240–260)
Males, 6–79 years							
1 (2007–2009)	2663	99.8 (99.4–99.9)	100 (97–110)	36 (28–43)	110 (100–110)	230 (220–240)	270 (250–280)
Females, 6–79 years							
1 (2007–2009)	2852	99.9 (99.8–100)	68 (62–74)	22 (18–25)	75 (66–84)	180 (160–190)	210 (200–230)
6–11 years							
1 (2007–2009)	1042	99.9 (99.8–99.9)	66 (60–72)	24 (18–29)	74 (67–81)	140 (130–150)	170 (160–180)
12–19 years							
1 (2007–2009)	992	99.9 (98.8–100)	120 (110–130)	39 (30–47)	130 (120–140)	250 (230–280)	300 (260–330)
20–39 years							
1 (2007–2009)	1172	99.9 (99.4–100)	90 (81–100)	29 (22–36)	99 (91–110)	230 (210–240)	280 (250–300)
40–59 years							
1 (2007–2009)	1221	99.8 (98.9–100)	78 (73–84)	24 (19–28)	86 (76–96)	210 (190–230)	240 (230–250)
60–79 years							
1 (2007–2009)	1088	99.9 (99.4–100)	72 (68–75)	26 (22–31)	81 (77–85)	150 (140–160)	190 (170–220)

CI: confidence interval; GM: geometric mean

Note: The limit of detection for cycle 1 is 3 mg/dL.

Table C-2

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	Detection Frequency (95% CI)	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
2 (2009–2011)	6299	99.8 (98.1–100)	100 (100–110)	35 (33–38)	110 (110–120)	240 (230–260)	280 (270–300)
Males, 3–79 years							
2 (2009–2011)	3031	99.6 (96.5–99.9)	120 (120–130)	47 (42–53)	130 (120–150)	260 (240–280)	310 (280–340)
Females, 3–79 years							
2 (2009–2011)	3268	100 (99.6–100)	89 (85–94)	30 (27–32)	100 (96–100)	200 (180–230)	250 (240–270)
3–5 years							
2 (2009–2011)	572	100	59 (55–63)	26 (24–29)	61 (55–67)	110 (110–120)	140 (110–160)
6–11 years							
2 (2009–2011)	1059	99.9 (99.6–100)	88 (83–94)	37 (33–42)	98 (94–100)	170 (160–170)	190 (170–210)
12–19 years							
2 (2009–2011)	1042	100 (98.9–100)	130 (120–150)	52 (36–68)	150 (140–160)	270 (260–280)	300 (270–340)
20–39 years							
2 (2009–2011)	1322	99.3 (94.4–99.9)	120 (110–130)	37 (25–48)	140 (130–160)	260 (250–280)	330 (270–380)
40–59 years							
2 (2009–2011)	1223	100 (99.4–100)	100 (96–110)	33 (27–40)	110 (100–120)	240 (220–260)	280 (260–310)
60–79 years							
2 (2009–2011)	1081	100	85 (80–89)	32 (26–37)	96 (90–100)	180 (170–200)	230 (210–260)

CI: confidence interval; GM: geometric mean

Note: The limit of detection for cycle 2 is 4 mg/dL.

Table C-3

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	Detection Frequency (95% CI)	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
3 (2012–2013)	5704	100	97 (93–100)	33 (29–37)	100 (100–110)	240 (220–250)	280 (250–300)
Males, 3–79 years							
3 (2012–2013)	2847	100	110 (110–120)	40 (35–46)	120 (110–130)	260 (230–280)	300 (260–340)
Females, 3–79 years							
3 (2012–2013)	2857	100	83 (76–90)	26 (21–30)	93 (81–110)	210 (190–240)	250 (220–270)
3–5 years							
3 (2012–2013)	521	99.9 (99.4–100)	51 (45–58)	19 (14–24)	58 (51–65)	110 (99–110)	120 (110–120)
6–11 years							
3 (2012–2013)	1013	100	84 (77–92)	35 (28–42)	93 (82–100)	160 (150–180)	200 (170–230)
12–19 years							
3 (2012–2013)	998	100	130 (120–150)	52 (37–66)	150 (140–160)	280 (260–300)	320 (290–360)
20–39 years							
3 (2012–2013)	1048	100	110 (98–120)	36 (26–45)	110 (97–130)	270 (220–320)	330 (290–380)
40–59 years							
3 (2012–2013)	1080	100	95 (86–110)	34 (24–44)	110 (98–110)	220 (200–250)	250 (230–280)
60–79 years							
3 (2012–2013)	1044	100	84 (76–91)	26 (19–32)	96 (89–100)	190 (170–210)	230 (210–240)

CI: confidence interval; GM: geometric mean

Note: The limit of detection for cycle 3 is 5.0 mg/dL.

Table C-4

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

CI: confidence interval; GM: geometric mean

Note: The limit of detection for cycle 4 is 5.0 mg/dL.

Table C-5

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 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 3–79 years							
5 (2016–2017)	5618	99.8 (98.5–100)	98 (92–100)	32 (29–36)	100 (97–110)	230 (220–240)	260 (240–280)
Males, 3–79 years							
5 (2016–2017)	2803	100 (99.8–100)	110 (110–120)	43 (35–50)	120 (110–130)	240 (230–250)	280 (250–320)
Females, 3–79 years							
5 (2016–2017)	2815	99.7 (97.2–100)	84 (79–90)	28 (24–32)	92 (84–99)	210 (190–230)	240 (230–260)
3–5 years							
5 (2016–2017)	546	99.8 (97.9–100)	59 (52–66)	26 (22–30)	68 (60–76)	110 (98–120)	130 (110–140)
6–11 years							
5 (2016–2017)	1006	99.9 (99.4–100)	86 (81–92)	37 (32–43)	92 (85–99)	160 (150–170)	190 (160–220)
12–19 years							
5 (2016–2017)	1005	99.9 (99.5–100)	140 (120–150)	58 (47–69)	150 (130–170)	290 (250–320)	340 (300–380)
20–39 years							
5 (2016–2017)	1057	100	110 (93–120)	33 (22–44)	120 (100–140)	250 (230–270)	290 (250–330)
40–59 years							
5 (2016–2017)	1007	99.5 (95.3–100)	94 (85–100)	29 (20–37)	98 (90–110)	230 (210–250)	250 (230–270)
60–79 years							
5 (2016–2017)	997	99.9 (99.2–100)	88 (83–93)	32 (27–36)	97 (93–100)	190 (180–210)	220 (210–240)

CI: confidence interval; GM: geometric mean

Note: The limit of detection for cycle 5 is 5.0 mg/dL.

APPENDIX D

BIOMONITORING OF METALS AND TRACE ELEMENTS IN HAIR

OVERVIEW

The first nationally representative data for 25 metals and trace elements in hair of Canadians are presented here. These data were collected between January 2016 and December 2017 as part of cycle 5 of the Canadian Health Measures Survey (CHMS) from approximately 2,000 Canadians aged 20–59 years at 16 sites across Canada.

The metals and trace elements measured in hair of individual respondents in cycle 5 of the CHMS are listed in Table D-1.

Table D-1

Metals and trace elements measured in hair of Canadian Health Measures Survey cycle 5 (2016–2017) participants aged 20–59 years

Chemical	Symbol	CASRN
Aluminum	Al	7429-90-5
Antimony	Sb	7440-36-0
Arsenic	As	7440-38-2
Barium	Ba	7440-39-3
Beryllium	Be	7440-41-7
Bismuth	Bi	7440-69-9
Cadmium	Cd	7440-43-9
Chromium	Cr	7440-47-3
Cobalt	Co	7440-48-4
Copper	Cu	7440-50-8
Lead	Pb	7439-92-1
Lithium	Li	7439-93-2
Manganese	Mn	7439-96-5
Mercury (total)	Hg	7439-97-6
Molybdenum	Mo	7439-98-7
Nickel	Ni	7440-02-0
Platinum	Pt	7440-06-4
Selenium	Se	7782-49-2
Silver	Ag	7440-22-4
Tellurium	Te	13494-80-9
Thallium	Tl	7440-28-0
Thorium	Th	7440-29-1
Uranium	U	7440-61-1
Vanadium	V	7440-62-2
Zinc	Zn	7440-66-6

Metals and trace elements can accumulate in hair as it grows, making it a useful matrix to measure both recent and long-term exposures. Chemicals can enter hair through internal and external exposure. Internal exposures (via inhalation, ingestion, etc.) are incorporated into growing hair through the blood supply following absorption of a chemical into the body. External exposures result in deposition onto hair from sources such as air, water, and product use. The amount of a chemical measured in hair indicates the total amount resulting from both internal and external exposures. As such, measurements in hair do not provide information about the source or route of the exposure.

LABORATORY ANALYSES

Metal and trace element analyses in hair were performed at the Centre de toxicologie du Québec (CTQ), Institut National de Santé Publique du Québec (INSPQ), Québec, Canada (INSPQ, 2018). Hair samples were collected from the occipital region of the head by cutting strands close to the scalp, clearly identifying the proximal (scalp) end of the samples. A minimum natural hair length of 2 cm was required and approximately 100 strands of full-length hair were collected. Segments of hair 2 cm to 3 cm in length were cut from the proximal end of the sample and digested overnight in pressurized Teflon bombs at 120°C under acidic conditions (concentrated nitric acid). The digest was then diluted in a solution of L-cysteine and gold, and analyzed for metals and trace elements using inductively coupled plasma mass spectrometry (ICP-MS). The ICP-MS method employed a PerkinElmer NexION 300S with a ESI-SC-2 autosampler. Results are expressed on a wet-weight basis. The analytical method was fully validated for clinical and monitoring purposes following ISO 17025 guidelines. Internal quality control was ensured by analyzing certified hair reference materials from the Japanese National Institute for Environmental Studies and the European Commission's Institute for Reference Materials and Measurements as well as non-certified materials from the Québec Multi-element External Quality Assessment Scheme (QMEQAS). External quality and accuracy of the analytical method was assessed by participating in interlaboratory comparison programs, including QMEQAS.

BIOMONITORING IN CANADA

Of the 25 metals and trace elements measured in hair as part of the CHMS, existing Canadian hair biomonitoring data were identified for 15 of them. These metals and trace elements include aluminum, antimony, arsenic, cadmium, chromium, cobalt, copper, lead, manganese, mercury, nickel, selenium, uranium, vanadium, and zinc. The majority of the studies reporting metals and trace elements in hair predate the 1990s; most were focused on either children or occupational and industrial exposures (Chattopadhyay and Jervis, 1974; Gibson and Dewolfe, 1979; Gibson et al., 1985; Gibson et al., 1989; Jervis et al., 1977; Moon et al., 1986; O'Toole et al., 1972; Randall and Gibson, 1989; Vanderkooy and Gibson, 1987). The remaining 10 metals and trace elements (barium, beryllium, bismuth, lithium, molybdenum, platinum, silver, tellurium, thallium, and thorium) were not identified in hair biomonitoring literature in Canada for any time period. This summary focuses on recent data (1990 onward) available for a subset of metals and trace elements, namely arsenic, manganese, mercury, selenium, uranium, and zinc.

Of the six metals and trace elements for which there are recent Canadian biomonitoring data in hair, mercury is the most studied. Concentration of mercury in hair and blood indicates methylmercury exposure, while urine concentrations generally indicate inorganic mercury exposure. The normal level of mercury in hair is 1 µg/g to 2 µg/g, but people who consume fish one or more times per day may have mercury levels in hair exceeding 10 µg/g (UNEP/WHO, 2008). In Canada, a comprehensive overview of studies measuring mercury in hair is presented in the Canadian Mercury Science Assessment Report (CMSAR) (Environment and Climate Change Canada and Health Canada, 2016). The geometric mean hair mercury levels presented in the CMSAR ranged from 0.23 µg/g to 2.8 µg/g for various groups in the Canadian adult population. The highest concentrations were reported for James Bay sport fishermen (Bélanger et al., 2008). In Northern Canada, a biomonitoring project was carried out among Dene/Métis communities of the Dehcho region of the Northwest Territories from 2016 to 2018 (Ratelle et al., 2018). The geometric mean concentration for mercury in hair was 0.39 µg/g based upon measures from 279 participants (aged six years and older).

Health Canada has established a methylmercury hair guidance value of 6 µg/g for the general adult population; levels below this value are considered within the normal acceptable range (Health Canada, 2004). For individuals under 18 years of age, pregnant women, and women of childbearing age (under 50 years of age), Health Canada has proposed a provisional methylmercury blood guidance value of 8 µg/L for the protection of the developing nervous system, which is equivalent to a methylmercury hair guidance value of 2 µg/g (Legrand et al., 2010).

Studies measuring levels of arsenic, manganese, selenium, uranium, or zinc in hair have been conducted in various locations and subpopulations in Canada since 1990. Studies examining arsenic in hair reported average, geometric mean, or median levels ranging from 0.018 to 0.15 µg/g in adults (Spallholz et al., 2005; Nieboer et al., 2017; Normandin et al., 2014). Studies measuring levels of manganese in hair have focused on children (6 to 13 years of age) and reported geometric mean levels ranging from 0.29 to 0.70 µg/g (Bouchard et al., 2011; Dion et al., 2018; Ntihabose et al., 2018). Selenium in hair was reported to have a mean concentration of 0.546 µg/g in adults (Spallholz et al., 2005). Levels of uranium measured in hair from a small sample of active and retired Canadian Forces personnel were reported to range from 0.0018 µg/g to 0.354 µg/g (Ough et al., 2002). A potential exposure source in this study may be depleted uranium munitions resulting from active duty overseas. Lastly, a mean level of zinc in hair of 115 µg/g was reported in children two to six years old (Vaghri et al., 2008).

DATA ANALYSIS

Tables D-2 to D-26 present each metal and trace element measured in hair in cycle 5. The data tables include the sample size (n), detection frequency, geometric mean (GM), and the 10th, 50th, 90th, and 95th percentiles, with associated 95% confidence intervals (CIs). For each chemical, results are presented for the total population as well as by sex and age group. While the numbers of male and female respondents were similar, the minimum requirement of 2 cm of natural hair length resulted in a greatly reduced final male sample size. 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. LOD values for each chemical are provided alongside their respective data tables and in Appendix A. Conversion factors to assist in the comparison of data from other studies that report different units are provided in Appendix B. Finding a measurable amount of a chemical in hair is an indicator of exposure to that chemical and does not necessarily mean that an adverse health effect will occur.

Table D-2

Aluminum — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1207	100	5.3 (4.5–6.2)	2.2 (1.7–2.7)	5.2 (4.2–6.2)	12 (8.2–16)	18[E] (11–26)
Males, 20–59 years							
5 (2016–2017)	291	100	6.9 (5.4–8.9)	2.7 (1.8–3.7)	6.5 (4.3–8.8)	23[E] (13–32)	29 (20–37)
Females, 20–59 years							
5 (2016–2017)	916	100	4.8 (4.0–5.8)	2.1 (1.6–2.7)	4.6 (3.6–5.6)	11 (8.7–13)	16 (11–22)
20–39 years							
5 (2016–2017)	608	100	5.2 (4.5–6.1)	2.3 (1.8–2.9)	5.0 (4.1–5.9)	11 (8.7–14)	15 (11–19)
40–59 years							
5 (2016–2017)	599	100	5.3 (4.3–6.6)	2.0 (1.6–2.5)	5.3 (3.9–6.7)	15[E] (7.6–23)	24[E] (14–33)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.10 µg/g.

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 D-3

Antimony — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1204	100	0.041 (0.029–0.058)	0.011[E] (0.0058–0.016)	0.039[E] (0.024–0.054)	0.13[E] (0.052–0.20)	0.24[E] (0.089–0.39)
Males, 20–59 years							
5 (2016–2017)	290	100	0.060 (0.042–0.085)	0.020[E] (0.011–0.029)	0.052[E] (0.030–0.073)	0.18[E] (0.080–0.28)	F
Females, 20–59 years							
5 (2016–2017)	914	100	0.037[E] (0.025–0.053)	0.010[E] (0.0061–0.014)	0.035[E] (0.020–0.050)	F	F
20–39 years							
5 (2016–2017)	606	100	0.039 (0.027–0.056)	0.011[E] (0.0059–0.016)	0.040[E] (0.021–0.060)	0.10 (0.070–0.13)	0.15[E] (0.047–0.26)
40–59 years							
5 (2016–2017)	598	100	0.043 (0.030–0.062)	0.011[E] (0.0054–0.017)	0.038[E] (0.024–0.053)	F	0.30[E] (0.087–0.51)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0020 µg/g.

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 are too unreliable to be published.

Table D-4

Arsenic — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1206	89.9 (81.8–94.6)	0.019 (0.016–0.023)	<LOD	0.020 (0.018–0.023)	0.053 (0.044–0.062)	0.069 (0.056–0.082)
Males, 20–59 years							
5 (2016–2017)	291	98.6 (89.1–99.8)	0.037 (0.028–0.047)	0.014[E] (0.0082–0.019)	0.033 (0.026–0.040)	0.097[E] (0.060–0.13)	0.15[E] (0.060–0.24)
Females, 20–59 years							
5 (2016–2017)	915	87.1 (77.6–92.9)	0.016 (0.014–0.018)	<LOD	0.018 (0.017–0.020)	0.038 (0.033–0.044)	0.052 (0.045–0.059)
20–39 years							
5 (2016–2017)	608	95.5 (92.4–97.4)	0.023 (0.020–0.026)	0.0090 (0.0064–0.012)	0.023 (0.019–0.027)	0.053 (0.045–0.060)	0.063 (0.052–0.073)
40–59 years							
5 (2016–2017)	598	84.4 (70.3–92.5)	0.017 (0.012–0.022)	<LOD	0.018 (0.015–0.021)	0.058[E] (0.037–0.079)	0.072[E] (0.036–0.11)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0050 µg/g.

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 D-5

Barium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	98.9 (91.7–99.9)	0.53 (0.41–0.69)	0.12[E] (0.052–0.19)	0.56 (0.42–0.69)	1.9[E] (1.1–2.7)	3.5[E] (1.7–5.4)
Males, 20–59 years							
5 (2016–2017)	291	98.9 (94.3–99.8)	0.39 (0.30–0.51)	0.10[E] (0.045–0.16)	0.39 (0.30–0.49)	1.3[E] (0.80–1.9)	1.6[E] (0.97–2.2)
Females, 20–59 years							
5 (2016–2017)	918	98.9 (89.5–99.9)	0.59 (0.44–0.79)	0.14[E] (0.061–0.22)	0.60 (0.45–0.74)	2.2[E] (1.0–3.4)	4.0[E] (1.4–6.5)
20–39 years							
5 (2016–2017)	609	98.2 (82.6–99.8)	0.47 (0.33–0.67)	0.12[E] (0.043–0.19)	0.47[E] (0.27–0.67)	1.6[E] (0.47–2.8)	F
40–59 years							
5 (2016–2017)	600	99.5 (97.4–99.9)	0.60 (0.48–0.76)	0.14[E] (0.064–0.21)	0.58 (0.47–0.70)	2.2 (1.4–2.9)	4.1[E] (2.4–5.7)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.030 µg/g.

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 are too unreliable to be published.

Table D-6

Beryllium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	F	—	<LOD	<LOD	<LOD	<LOD
Males, 20–59 years							
5 (2016–2017)	291	F	—	<LOD	<LOD	<LOD	<LOD
Females, 20–59 years							
5 (2016–2017)	918	F	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	609	F	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	600	0	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.030 µg/g.

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

F Data are too unreliable to be published.

Table D-7

Bismuth — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	44.8 (36.3–53.6)	—	<LOD	<LOD	0.12[E] (0.038–0.20)	0.26[E] (0.14–0.38)
Males, 20–59 years							
5 (2016–2017)	291	36.8[E] (22.5–53.8)	—	<LOD	<LOD	0.031[E] (0.016–0.045)	0.046[E] (0.018–0.074)
Females, 20–59 years							
5 (2016–2017)	918	47.4 (39.3–55.6)	—	<LOD	<LOD	0.17[E] (0.072–0.27)	0.33[E] (0.16–0.51)
20–39 years							
5 (2016–2017)	609	42.5 (29.9–56.1)	—	<LOD	<LOD	0.11[E] (0.040–0.19)	F
40–59 years							
5 (2016–2017)	600	47.0 (35.2–59.1)	—	<LOD	<LOD	F	0.27[E] (0.081–0.45)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/g.

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 are too unreliable to be published.

Table D-8

Cadmium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	26.0 (18.6–35.0)	—	<LOD	<LOD	0.028[E] (0.016–0.040)	F
Males, 20–59 years							
5 (2016–2017)	291	54.9 (39.1–69.7)	—	<LOD	0.010[E] (<LOD–0.017)	F	0.24[E] (0.085–0.39)
Females, 20–59 years							
5 (2016–2017)	918	16.7 (11.8–23.2)	—	<LOD	<LOD	0.017 (0.013–0.021)	0.024 (0.018–0.030)
20–39 years							
5 (2016–2017)	609	25.0[E] (16.3–36.3)	—	<LOD	<LOD	0.028[E] (0.014–0.043)	F
40–59 years							
5 (2016–2017)	600	26.9[E] (17.5–39.0)	—	<LOD	<LOD	0.027[E] (<LOD–0.045)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/g.

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 are too unreliable to be published.

Table D-9

Chromium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1207	99.6 (97.8–99.9)	0.12 (0.098–0.15)	0.047 (0.036–0.058)	0.10 (0.080–0.13)	0.34 (0.25–0.43)	0.54 (0.40–0.68)
Males, 20–59 years							
5 (2016–2017)	291	100	0.16 (0.12–0.22)	0.055[E] (0.035–0.076)	0.14[E] (0.083–0.20)	0.42[E] (0.12–0.71)	0.80[E] (0.22–1.4)
Females, 20–59 years							
5 (2016–2017)	916	99.4 (97.0–99.9)	0.11 (0.090–0.14)	0.046 (0.036–0.056)	0.10 (0.080–0.12)	0.30 (0.21–0.40)	0.52 (0.35–0.70)
20–39 years							

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
5 (2016–2017)	608	99.9 (97.7–100)	0.11 (0.094–0.14)	0.045 (0.039–0.052)	0.10 (0.078–0.13)	0.30 (0.23–0.37)	0.40 (0.32–0.47)
40–59 years							
5 (2016–2017)	599	99.2 (96.4–99.8)	0.13 (0.10–0.17)	0.053 (0.040–0.066)	0.11 (0.077–0.14)	0.44[E] (0.27–0.61)	0.65[E] (0.39–0.90)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.020 µg/g.

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 D-10

Cobalt — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	95.6 (75.6–99.3)	0.014[E] (0.0094–0.022)	F	0.014[E] (0.0082–0.019)	0.070[E] (0.038–0.10)	0.11[E] (0.066–0.16)
Males, 20–59 years							
5 (2016–2017)	291	97.4 (85.1–99.6)	0.014 (0.0095–0.019)	0.0041[E] (0.0020–0.0063)	0.014[E] (0.0071–0.020)	F	0.071[E] (0.033–0.11)
Females, 20–59 years							
5 (2016–2017)	918	95.0 (73.1–99.3)	0.015[E] (0.0089–0.024)	F	0.014[E] (0.0076–0.020)	0.088[E] (0.049–0.13)	0.14[E] (0.076–0.21)
20–39 years							
5 (2016–2017)	609	95.6 (64.6–99.6)	0.013[E] (0.0070–0.023)	F	0.010[E] (0.0035–0.017)	0.060[E] (0.030–0.090)	0.11[E] (0.033–0.20)
40–59 years							
5 (2016–2017)	600	95.5 (79.9–99.1)	0.016 (0.012–0.023)	0.0036[E] (0.0012–0.0059)	0.016 (0.011–0.021)	0.090[E] (0.055–0.13)	0.11[E] (0.064–0.17)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0010 µg/g.

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 are too unreliable to be published.

Table D-11

Copper — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1208	100	19 (16–22)	9.9 (9.6–10)	16 (14–18)	42 (32–53)	69[E] (38–100)
Males, 20–59 years							
5 (2016–2017)	290	100	19 (17–22)	9.6 (8.7–10)	16 (14–19)	40 (32–47)	64[E] (31–98)
Females, 20–59 years							

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
5 (2016–2017)	918	100	19 (16–22)	10 (9.5–10)	16 (13–18)	43[E] (25–61)	70[E] (27–110)
20–39 years							
5 (2016–2017)	608	100	18 (15–21)	9.8 (9.5–10)	15 (12–18)	40 (27–52)	67[E] (29–100)
40–59 years							
5 (2016–2017)	600	100	20 (17–23)	10 (9.4–11)	16 (14–18)	43[E] (26–60)	70[E] (27–110)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.10 µg/g.

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 D-12

Lead — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	96.6 (83.5–99.4)	0.15[E] (0.099–0.23)	F	0.16 (0.10–0.21)	0.75 (0.54–0.95)	1.3[E] (0.46–2.2)
Males, 20–59 years							
5 (2016–2017)	291	100	0.44 (0.31–0.63)	0.094 (0.061–0.13)	0.35[E] (0.19–0.51)	2.8[E] (1.1–4.4)	F
Females, 20–59 years							
5 (2016–2017)	918	95.5 (79.2–99.2)	0.11[E] (0.071–0.16)	F	0.12 (0.092–0.15)	0.44 (0.34–0.55)	0.70[E] (0.43–0.96)
20–39 years							
5 (2016–2017)	609	95.7 (76.9–99.3)	0.14[E] (0.080–0.24)	F	0.13[E] (0.070–0.19)	0.64[E] (0.29–0.98)	F
40–59 years							
5 (2016–2017)	600	97.5 (89.2–99.5)	0.17[E] (0.11–0.24)	F	0.17 (0.12–0.22)	0.79 (0.51–1.1)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0050 µg/g.

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 are too unreliable to be published.

Table D-13

Lithium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	61.7 (51.4–71.0)	—	<LOD	0.012 (0.010–0.015)	0.055 (0.036–0.073)	0.082[E] (0.048–0.12)
Males, 20–59 years							

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
5 (2016–2017)	291	61.1 (42.1–77.2)	—	<LOD	0.013[E] (<LOD–0.018)	0.059[E] (0.032–0.086)	F
Females, 20–59 years							
5 (2016–2017)	918	61.9 (52.2–70.8)	—	<LOD	0.012 (0.010–0.015)	0.053[E] (0.033–0.072)	0.081[E] (0.047–0.11)
20–39 years							
5 (2016–2017)	609	62.4 (48.6–74.5)	—	<LOD	0.012 (<LOD–0.015)	0.053 (0.035–0.070)	F
40–59 years							
5 (2016–2017)	600	61.0 (49.9–71.1)	—	<LOD	0.013 (<LOD–0.016)	0.059[E] (0.032–0.086)	0.083 (0.062–0.11)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/g.

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 are too unreliable to be published.

Table D-14

Manganese — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	100	0.18[E] (0.12–0.30)	0.063 (0.040–0.085)	0.15 (0.094–0.20)	F	1.4[E] (0.38–2.4)
Males, 20–59 years							
5 (2016–2017)	291	100	0.27[E] (0.15–0.47)	0.076 (0.060–0.092)	F	1.5[E] (0.54–2.5)	1.9[E] (0.81–2.9)
Females, 20–59 years							
5 (2016–2017)	918	100	0.16[E] (0.10–0.26)	0.058[E] (0.032–0.083)	0.13 (0.088–0.18)	F	F
20–39 years							
5 (2016–2017)	609	100	0.18[E] (0.11–0.29)	0.060[E] (0.034–0.086)	0.14 (0.087–0.19)	F	F
40–59 years							
5 (2016–2017)	600	100	0.19[E] (0.12–0.31)	0.063 (0.041–0.086)	0.16[E] (0.097–0.23)	0.80[E] (0.27–1.3)	1.3[E] (0.46–2.2)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.020 µg/g.

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 are too unreliable to be published.

Table D-15

Mercury (total) — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
5 (2016–2017)	1209	97.5 (94.8–98.8)	0.19 (0.14–0.25)	0.028[E] (0.014–0.043)	0.23 (0.18–0.28)	0.97 (0.65–1.3)	1.4[E] (0.81–1.9)
Males, 20–59 years							
5 (2016–2017)	291	98.1 (91.6–99.6)	0.19[E] (0.12–0.29)	F	0.21[E] (0.12–0.30)	0.97[E] (0.59–1.3)	1.4[E] (0.78–1.9)
Females, 20–59 years							
5 (2016–2017)	918	97.2 (93.0–98.9)	0.19 (0.14–0.26)	0.029[E] (0.0080–0.051)	0.24 (0.17–0.30)	0.96[E] (0.60–1.3)	1.3[E] (0.76–1.9)
20–39 years							
5 (2016–2017)	609	98.3 (92.8–99.6)	0.15 (0.12–0.19)	F	0.17 (0.11–0.23)	0.79[E] (0.30–1.3)	1.1[E] (0.64–1.6)
40–59 years							
5 (2016–2017)	600	96.7 (91.7–98.7)	0.23 (0.16–0.33)	F	0.28 (0.19–0.38)	1.0 (0.69–1.3)	1.6[E] (0.86–2.3)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0050 µg/g.

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 are too unreliable to be published.

Table D-16

Molybdenum — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1208	100 (99.8–100)	0.034 (0.029–0.040)	0.019 (0.015–0.023)	0.032 (0.026–0.037)	0.055 (0.044–0.065)	0.076[E] (0.039–0.11)
Males, 20–59 years							
5 (2016–2017)	291	100	0.041 (0.032–0.052)	0.022 (0.016–0.027)	0.035 (0.027–0.043)	F	F
Females, 20–59 years							
5 (2016–2017)	917	100 (99.8–100)	0.032 (0.028–0.037)	0.018 (0.014–0.022)	0.030 (0.025–0.036)	0.050 (0.041–0.059)	0.069 (0.044–0.094)
20–39 years							
5 (2016–2017)	608	100	0.035 (0.031–0.040)	0.024 (0.021–0.026)	0.033 (0.028–0.038)	0.052 (0.045–0.059)	0.068 (0.046–0.090)
40–59 years							
5 (2016–2017)	600	100 (99.7–100)	0.033 (0.027–0.041)	0.017 (0.014–0.019)	0.030 (0.023–0.037)	0.061 (0.041–0.080)	F

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0020 µg/g.

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 are too unreliable to be published.

Table D-17

Nickel — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1207	100	0.31 (0.25–0.39)	0.11 (0.083–0.14)	0.31 (0.24–0.38)	0.86 (0.67–1.1)	1.2 (0.80–1.5)
Males, 20–59 years							
5 (2016–2017)	290	100	0.34 (0.27–0.43)	0.11[E] (0.059–0.16)	0.36 (0.24–0.47)	0.88 (0.71–1.0)	1.3[E] (0.79–1.8)
Females, 20–59 years							
5 (2016–2017)	917	100	0.30 (0.24–0.38)	0.11 (0.084–0.13)	0.30 (0.22–0.38)	0.84 (0.60–1.1)	1.2[E] (0.52–1.8)
20–39 years							
5 (2016–2017)	607	100	0.27 (0.21–0.35)	0.10 (0.068–0.13)	0.24 (0.16–0.31)	0.73[E] (0.46–1.0)	1.1[E] (0.39–1.9)
40–59 years							
5 (2016–2017)	600	100	0.36 (0.30–0.44)	0.12 (0.081–0.15)	0.37 (0.27–0.46)	0.91 (0.76–1.1)	1.2 (0.91–1.5)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.020 µg/g.

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 D-18

Platinum — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	32.6 (23.0–44.0)	—	<LOD	<LOD	0.0024 (0.0019–0.0029)	0.0040[E] (0.0021–0.0060)
Males, 20–59 years							
5 (2016–2017)	291	32.8[E] (19.5–49.5)	—	<LOD	<LOD	0.0021[E] (0.0011–0.0030)	F
Females, 20–59 years							
5 (2016–2017)	918	32.6 (23.1–43.8)	—	<LOD	<LOD	0.0026 (0.0021–0.0031)	0.0040[E] (0.0021–0.0060)
20–39 years							
5 (2016–2017)	609	34.8[E] (21.5–51.0)	—	<LOD	<LOD	0.0027 (0.0019–0.0036)	F
40–59 years							
5 (2016–2017)	600	30.6 (22.2–40.5)	—	<LOD	<LOD	0.0022 (0.0018–0.0026)	0.0031[E] (0.0019–0.0043)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0010 µg/g.

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 are too unreliable to be published.

Table D-19

Selenium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	100	0.64 (0.61–0.68)	0.49 (0.46–0.52)	0.63 (0.61–0.65)	0.77 (0.72–0.83)	0.83 (0.76–0.90)
Males, 20–59 years							
5 (2016–2017)	291	100	0.66 (0.64–0.69)	0.55 (0.53–0.58)	0.65 (0.62–0.68)	0.78 (0.73–0.84)	0.81 (0.76–0.86)
Females, 20–59 years							
5 (2016–2017)	918	100	0.63 (0.59–0.68)	0.48 (0.44–0.51)	0.63 (0.61–0.65)	0.77 (0.71–0.82)	0.84 (0.75–0.92)
20–39 years							
5 (2016–2017)	609	100	0.69 (0.62–0.75)	0.53 (0.49–0.57)	0.65 (0.62–0.67)	0.82 (0.74–0.90)	0.87 (0.78–0.96)
40–59 years							
5 (2016–2017)	600	100	0.60 (0.58–0.62)	0.46 (0.42–0.50)	0.61 (0.59–0.64)	0.73 (0.68–0.78)	0.78 (0.74–0.83)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/g.

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

Table D-20

Silver — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1208	92.3 (86.0–95.9)	0.035 (0.029–0.042)	0.011[E] (<LOD–0.015)	0.032 (0.025–0.039)	0.13 (0.099–0.16)	0.23 (0.14–0.31)
Males, 20–59 years							
5 (2016–2017)	291	90.8 (77.2–96.6)	0.037 (0.026–0.053)	F	0.033 (0.023–0.043)	0.17[E] (0.086–0.26)	F
Females, 20–59 years							
5 (2016–2017)	917	92.8 (86.2–96.3)	0.034 (0.028–0.042)	0.011 (<LOD–0.015)	0.029 (0.022–0.037)	0.12 (0.088–0.15)	0.20[E] (0.097–0.30)
20–39 years							
5 (2016–2017)	608	90.4 (80.1–95.7)	0.030 (0.023–0.039)	<LOD	0.025 (0.019–0.032)	0.10 (0.070–0.14)	0.17[E] (0.065–0.28)
40–59 years							
5 (2016–2017)	600	94.0 (87.2–97.3)	0.041 (0.033–0.050)	0.012 (<LOD–0.015)	0.036 (0.026–0.045)	0.16[E] (0.099–0.22)	0.28 (0.20–0.36)

CI: onfidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.010 µg/g.

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 are too unreliable to be published.

Table D-21

Tellurium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	13.3[E] (7.4–22.9)	—	<LOD	<LOD	0.0023[E] (<LOD–0.0032)	0.0032[E] (0.0020–0.0045)
Males, 20–59 years							
5 (2016–2017)	291	13.9[E] (6.5–27.1)	—	<LOD	<LOD	0.0025[E] (<LOD–0.0037)	0.0038[E] (<LOD–0.0061)
Females, 20–59 years							
5 (2016–2017)	918	13.2[E] (6.8–23.9)	—	<LOD	<LOD	0.0023[E] (<LOD–0.0032)	0.0030[E] (<LOD–0.0041)
20–39 years							
5 (2016–2017)	609	13.1[E] (6.5–24.5)	—	<LOD	<LOD	0.0022[E] (<LOD–0.0032)	0.0029[E] (<LOD–0.0045)
40–59 years							
5 (2016–2017)	600	13.6[E] (6.9–25.1)	—	<LOD	<LOD	0.0024[E] (<LOD–0.0033)	0.0034[E] (0.0021–0.0047)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0020 µg/g.

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 D-22

Thallium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	74.2 (58.6–85.4)	0.00043[E] (0.00028–0.00067)	<LOD	0.00040[E] (0.00024–0.00056)	F	0.0041[E] (0.0016–0.0067)
Males, 20–59 years							
5 (2016–2017)	291	79.5 (64.5–89.3)	0.00052[E] (0.00034–0.00078)	<LOD	0.00056 (0.00036–0.00076)	F	0.0040[E] (0.0018–0.0061)
Females, 20–59 years							
5 (2016–2017)	918	72.5 (55.7–84.6)	0.00041[E] (0.00025–0.00065)	<LOD	0.00039 (0.00025–0.00054)	F	0.0041[E] (0.0014–0.0069)
20–39 years							
5 (2016–2017)	609	82.2 (69.5–90.4)	0.00050 (0.00035–0.00070)	<LOD	0.00043[E] (0.00026–0.00059)	F	0.0043[E] (0.0017–0.0069)
40–59 years							
5 (2016–2017)	600	66.5 (46.3–82.1)	0.00038[E] (0.00020–0.00070)	<LOD	0.00039[E] (<LOD–0.00062)	F	0.0039[E] (0.0012–0.0065)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.00020 µg/g.

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 are too unreliable to be published.

Table D-23

Thorium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1208	3.7[E] (2.0–6.5)	—	<LOD	<LOD	<LOD	<LOD
Males, 20–59 years							
5 (2016–2017)	291	F	—	<LOD	<LOD	<LOD	<LOD
Females, 20–59 years							
5 (2016–2017)	917	3.3[E] (1.9–5.8)	—	<LOD	<LOD	<LOD	<LOD
20–39 years							
5 (2016–2017)	608	5.2[E] (2.7–9.9)	—	<LOD	<LOD	<LOD	<LOD
40–59 years							
5 (2016–2017)	600	F	—	<LOD	<LOD	<LOD	<LOD

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.10 µg/g.

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 are too unreliable to be published.

Table D-24

Uranium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1207	95.0 (83.9–98.6)	0.020[E] (0.012–0.034)	F	0.024[E] (0.014–0.034)	0.088 (0.063–0.11)	0.14 (0.10–0.18)
Males, 20–59 years							
5 (2016–2017)	290	98.3 (92.4–99.7)	0.024[E] (0.014–0.040)	F	0.028[E] (0.012–0.044)	F	0.15[E] (0.094–0.21)
Females, 20–59 years							
5 (2016–2017)	917	94.0 (81.3–98.2)	0.019[E] (0.011–0.033)	F	0.024[E] (0.014–0.033)	0.079 (0.056–0.10)	0.13 (0.10–0.15)
20–39 years							
5 (2016–2017)	608	96.4 (82.3–99.3)	0.023[E] (0.013–0.039)	F	0.027[E] (0.015–0.039)	0.093[E] (0.058–0.13)	0.14 (0.10–0.18)
40–59 years							
5 (2016–2017)	599	93.7 (82.6–97.9)	0.018[E] (0.010–0.032)	F	0.022[E] (0.012–0.032)	0.081 (0.052–0.11)	0.15[E] (0.082–0.21)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0015 µg/g.

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 are too unreliable to be published.

Table D-25

Vanadium — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1209	97.2 (86.2–99.5)	0.0092 (0.0066–0.013)	0.0038[E] (0.0013–0.0062)	0.0092 (0.0069–0.012)	0.026 (0.017–0.035)	0.038[E] (0.023–0.052)
Males, 20–59 years							
5 (2016–2017)	291	99.0 (89.9–99.9)	0.012 (0.0083–0.017)	0.0048[E] (0.0024–0.0072)	0.010[E] (0.0058–0.014)	0.033[E] (0.019–0.048)	0.048[E] (0.026–0.071)
Females, 20–59 years							
5 (2016–2017)	918	96.6 (84.7–99.3)	0.0085 (0.0060–0.012)	F	0.0085 (0.0062–0.011)	0.023[E] (0.014–0.031)	0.031[E] (0.017–0.045)
20–39 years							
5 (2016–2017)	609	98.5 (56.5–100)	0.0099 (0.0069–0.014)	0.0043[E] (0.0021–0.0066)	0.0099 (0.0070–0.013)	0.025[E] (0.015–0.035)	0.033[E] (0.020–0.047)
40–59 years							
5 (2016–2017)	600	95.9 (87.2–98.8)	0.0087[E] (0.0060–0.013)	F	0.0086 (0.0063–0.011)	0.028 (0.018–0.038)	0.047[E] (0.023–0.071)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 0.0010 µg/g.

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 are too unreliable to be published.

Table D-26

Zinc — Geometric means and selected percentiles of hair concentrations (µg/g) for the Canadian population aged 20–59 years by age group, Canadian Health Measures Survey cycle 5 (2016–2017)

Cycle	n	Detection Frequency (95% CI)	GM[a] (95% CI)	10th (95% CI)	50th (95% CI)	90th (95% CI)	95th (95% CI)
Total, 20–59 years							
5 (2016–2017)	1208	100	210 (200–230)	150 (140–150)	190 (180–210)	320 (290–340)	390 (350–440)
Males, 20–59 years							
5 (2016–2017)	290	100	210 (190–230)	140 (130–160)	190 (170–210)	330 (270–380)	440 (290–600)
Females, 20–59 years							
5 (2016–2017)	918	100	210 (190–230)	150 (140–160)	190 (180–210)	310 (270–340)	390 (350–440)
20–39 years							
5 (2016–2017)	608	100	210 (200–230)	150 (140–160)	190 (170–210)	330 (280–380)	430 (370–500)
40–59 years							
5 (2016–2017)	600	100	210 (190–220)	140 (130–150)	190 (180–210)	310 (280–340)	380 (340–420)

CI: confidence interval; GM: geometric mean; LOD: limit of detection

Note: The LOD for cycle 5 is 5.0 µg/g.

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

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