Northern Saskatchewan Prenatal Biomonitoring Study Technical Report

A Final Report

Submitted to Saskatchewan Ministry of Health

March 2019

Saskatchewan Health Authority

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Saskatchewan Ministry of Health and
Alberta Health and Wellness (2019)

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EXECUTIVE SUMMARY

The current biomonitoring study was conducted with a multi-disciplinary committee of academic and professional experts from the Saskatchewan Ministry of Health, Northern Saskatchewan Population Health Unit, Alberta Health, the University of Alberta, the Alberta Centre for Toxicology, and the Saskatchewan Disease Control Laboratory (SDCL). Provincial stakeholders include the northern Saskatchewan health regions, Northern Inter-Tribal Health Authority (NITHA), and First Nations and Inuit Health. The Ministries of Environment and Energy and Resources are aware of the study. Other relevant ministries, agencies and industry representatives have been informed as appropriate.

A comprehensive list of priority environmental chemicals and heavy metals was monitored in blood serum of pregnant women living in northern Saskatchewan between 2011 and 2013. The purpose was to establish the magnitude of typical human exposure to environmental chemicals during pregnancy for women living in northern Saskatchewan via the maternal blood serum concentrations of synthetic or naturally occurring chemicals that women may absorb from food, drinking water, air, soil, household dust, or commercial products. This data will serve as a baseline to monitor temporal trends and act as a comparison for the biomonitoring study conducted in Alberta (Alberta Health and Wellness, 2008), currently available Canadian or North American data, and potential future biomonitoring surveillance in northern Saskatchewan following further industrialization. This was the first biomonitoring study for northern Saskatchewan residents, and few other studies are currently available from elsewhere in Canada.

From a pool of 1233 blood samples from pregnant women in northern Saskatchewan during the selected time frame received at SDCL, 841 total participants were included in the study. Samples were retrieved from 'leftover' blood samples that had been collected during routine prenatal infectious disease screening. Two hundred fifty-five samples did not have enough 'leftover' blood to be included in the study, 6 women declined participation and 118 were not contactable for retrospective consent. Across each geographic region, far North, Northwest, and Northeast, six pools were created, with approximately 140 persons per pool. Individual blood samples were not analyzed. The samples were pooled at the SDCL before transfer to ALS Laboratory Group in Alberta.

The following classes of chemicals were monitored: tobacco smoke markers, phenols, phytoestrogens, polychlorinated biphenyls, dioxins and furans, organochlorine pesticides, polybrominated compounds, perfluorinated compounds, parabens, phthalates, methylmercury, lead and various other trace metals or mineral micronutrients. Some of these chemical classes are typically measured in urine samples in similar biomonitoring studies, and it was suspected that the current study design using serum sampling would not be as sensitive at estimating total body burden.

The laboratories tasked with analyzing the samples included the Alberta Centre for Toxicology and ALS Laboratory Group. Quality control samples were also analyzed with the participant serum samples to monitor for possible contamination by collection vessels and routine sample handling. Both analytical laboratories were blind to the nature of the samples.

The use of pooled human samples for biomonitoring in the current work was demonstrated to have advantages over the more common practice of analyzing thousands of individual samples. The pooled study design remained an effective means of determining the distribution of chemical in the selected population while also enabling hypotheses to be tested on geography. The current study, which analyzed approximately 283 chemicals, was also more cost effective than the alternative of analyzing hundreds of individual samples. Furthermore, because the distribution of concentrations in the population for any particular chemical is considered to be log-normally distributed, the pooling of samples had the added benefit of effectively increasing the analytical sensitivity for detecting the population median concentration.

While the health implications posed by background concentrations of these environmental chemicals are difficult to assess at this time, the clinical implications of tobacco smoking are relatively well understood. Upwards of 70% of nicotine in the body is converted into a metabolite called cotinine which can be measured via biomonitoring and can be used as a proxy to measure exposure to nicotine. Non-smokers are normally defined as having serum cotinine concentrations below 10 ng/mL. Serum concentrations measured in Saskatchewan exceeded the level typically found in non-smokers which suggests that pregnant women in northern Saskatchewan are smoking or being exposed to second-hand smoke at the time of their blood sample collection. In general, the concentrations of detected chemicals of pregnant northern Saskatchewan women sampled in this study were either lower or similar to concentrations previously determined in other studies across North America. Note that the presence of an environmental chemical in the body does not necessarily indicate harm – the detection of chemicals has advanced more rapidly than the interpretation of potential health consequences. Care should also be taken when extrapolating the results of this study to the entirety of the population of pregnant women in northern Sasaktchewan due to sampling methods employed in this study; however, biomonitoring does provide an indication of possible exposure and in the case of tobacco exposure may lead to the development of programs and guidelines in the future to further decrease fetal exposures to nicotine metabolites. Another weakness in the interpretation of the data from this study, is that some chemical concentrations within the current study cannot be compared to other reports due to differences in biological matrix; these instances are noted where relevant.

INTRODUCTION TO BIOMONITORING

People come into contact with a variety of natural and man-made chemicals every day. Contact can occur via ingestion, inhalation or dermal contact with consumer products, water, soil, food or air. Health risks resulting from everyday contact will be a complex function of the substances' pharmacokinetics or what the body does to a chemical; and pharmacodynamics or what a drug does to the body. Pharmacokinetics refers to the movement of a chemical into, through and out of the body and includes: chemicals characteristics, route of exposure, duration of exposure, rate and volume of absorption metabolism and excretion. These complex functions determines the internal dose of the chemical. The internal dose is difficult to predict as it is influenced by numerous environmental variables and because humans differ widely in physiology and behaviour. If the internal dose of any substance if high enough, it may result in adverse effects. Health effects are also influenced by pharmacodynamics involving things like receptor binding, post receptor effects and chemical interactions.

Biomonitoring is the measurement of chemicals or their metabolites in people via a biological matrix (i.e.: blood, urine, hair, breast milk). It is the most accurate way to assess internal dose of natural or synthetic chemicals from environmental exposures. The measurements indicate how much of the chemical or an element is present in the person at that point in time; biomonitoring does not provide information on source, duration or route of exposure. Periodic biomonitoring of populations provides measurable levels of exposure to environmental chemicals and enables assessment of potential associated health risks, if any. It has become an essential tool in identifying and monitoring peoples' exposure to environmental chemicals. Canadian biomonitoring initiatives, in addition to the Alberta program, include the Canadian Health Measures Survey (CHMS), the Maternal-Infant Research on Environmental Chemicals (MIREC), the First Nations Biomonitoring Initiative, and the Northern Contaminants Program (in Nunavut, Northwest Territories and the Yukon). None of these projects have included people from northern Saskatchewan; in fact only the more recent cycles of the CHMS (with one urban center) and the First Nations Biomonitoring Initiative (one southern community) included people from Saskatchewan). The First Nations' Food, Nutrition and Environment Study which includes several First Nation communities in northern Saskatchewan l provides some mercury in hair biomonitoring information. International biomonitoring initiatives include the United States' biennial National Health and Nutrition Examination Survey (NHANES), and the European Union-wide Consortium to Perform Human Biomonitoring on a European Scale (COPHES).

Biomonitoring is a tool that allows for the determination of environmental chemicals in the population. It helps support evidence-based policy making decisions and promotes comprehensive health impact assessments of policy options. The current challenge lies with proper communication in what biomonitoring results mean, and what they don't mean, in order to avoid public misconceptions. In order to be able to effectively communicate results of biomonitoring studies to

the public, it is essential to provide open and consistent information. Biomonitoring is not indicative of an individual's likelihood of becoming ill, nor is it an accurate indicator of an individual's state of health.

BACKGROUND AND RATIONALE

The purpose of the northern Saskatchewan biomonitoring program, in partnership with Alberta, is to establish baseline magnitudes of typical human exposure to environmental chemicals during pregnancy for women living in northern Saskatchewan. Notably, this population has not yet been exposed to extensive industrial or agricultural development, but will likely experience further industrial development in the future. Saskatchewan currently does not monitor background chemical concentrations of its population in a systematic manner. The range of exposure concentrations measured from this study may serve as a starting point to assess health risks; as a benchmark to track future exposure; as an indicator of exposure source; and to prioritize future research in Saskatchewan. This health surveillance approach, in a relevant Saskatchewan population, will represent the first measurements for several emerging environmental chemicals, which could potentially affect human health, and could help with the development of future public health strategies to reduce risk.

Pregnant women were selected as the sample population because the fetus is one of the most sensitive human life stages. The fetus can be exposed to chemicals that cross the placenta via the mother's daily environmental exposures. Relatively high maternal body burdens of some chemicals have been well documented to have adverse effects on fetal development, whereas the effects of new and emerging chemicals are less well defined. An advantage of this study is that is does not require additional blood tests as blood samples already taken for routine prenatal blood screening were utilized.

Advances in the precision and accuracy of analytical instruments and scientific procedures improve with each reporting period. Scientific advances have allowed for the measurement of very low concentrations of environmental chemicals. However, the detection of an environmental chemical in the body does not equate to an adverse health effect. Further, detection in one study and non-detection in another may be a result of the limit of detection of the analytical instrument and may not reflect actual differences in exposures. For most chemicals or metals, there is a threshold at which quantifiable health effects are known to occur that is dependent on the effective or internal dose of the substance. Carcinogens (chemicals that may cause cancer in humans) and some chemicals like lead should be considered as if there is no level of exposure below which health risk is zero.

Laboratory analysis of Saskatchewan samples was provided by Alberta Health. The data obtained is shared with Alberta and provides a comparable population for Alberta's Biomonitoring Program (phase 1 was complete in 2005 with a report in 2008; phase 2 was completed in 2006 with a report in 2010; phase 3 is underway). Alberta's biomonitoring program was established after the

development of the oil sands in Northern Alberta; therefore, a baseline reading on the level of chemicals of potential concern was not obtained. Northern Saskatchewan residents provide a suitably comparable population from which to draw a comparison. The data from this project may also assist in mitigating chemical risks at the population health level and provide a mechanism to respond to community health risks in the future. The baseline information obtained from this surveillance study will be invaluable for Saskatchewan in the future. It may allow for quantification of the actual environmental health impact of development on the residents of Northern Saskatchewan.

In this report, results are listed by each chemical or chemical group, and some basic information about each chemical is provided. Possible exposure sources, potential adverse human health effects, and any relevant exposure guidelines are discussed. Where possible, the exposure levels in the northern Saskatchewan population are compared to other sample populations.

SURVEY DESIGN

ETHICAL CONSIDERATIONS

Ethics approval was submitted to the University of Saskatchewan's Research Ethics Board on May 9, 2011 (Bio-REB 11-109). University of Saskatchewan's Research Ethics Board (in a letter from the Biomedical Ethics Chair, dated May 26, 2011) deemed the project to be surveillance (versus research) in its intent. As such, the project was deemed exempt from the requirement of research ethics review; however, the Chair of the Board provided guidance on the consent process as if it was considered as research. Approval was also received from the four northern health authorities of Athabasca Health Authority, Keewatin Yatthé, Mamawetan Churchill River and Kelsey Trail Health Regions; and the Northern Intertribal Health Authority representing Prince Albert Grand Council, Meadow Lake Tribal Council, Lac La Ronge Indian Band and the Peter Ballantyne Cree Nation.

Plans were also discussed with the Northern Saskatchewan Environmental Quality Committees, the Prince Albert Grand Council Chiefs and the Meadow Lake Tribal Council Health and Social Services group. Following ethical approval, further information was provided to northern health professionals involved with the care of prenatal women including public health nurses and physicians. Community awareness was enhanced through the use of radio messaging in Cree, Dene and English as well as pamphlets available at all health centers and through prenatal education, and posters used at health centers and other community centers or bulletin boards.

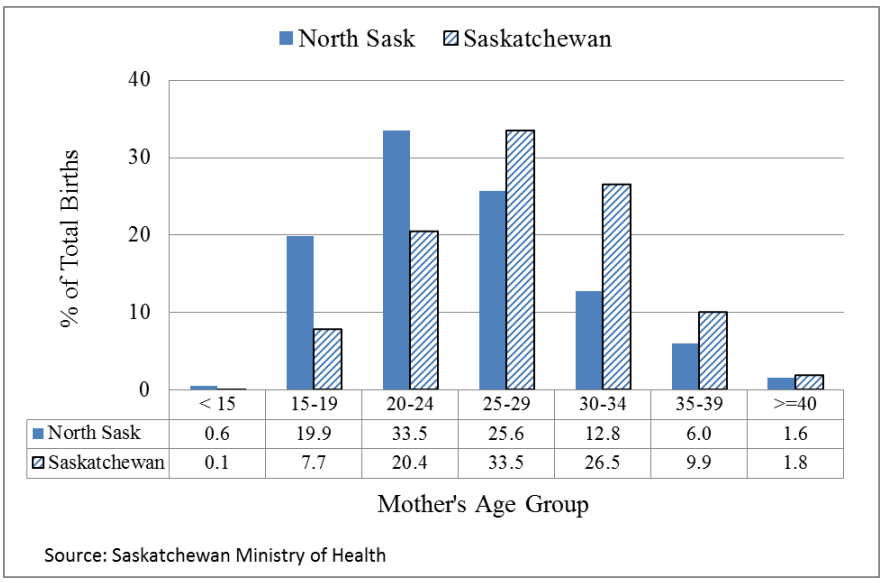
THE GEOGRAPHY AND POPULATION OF NORTHERN SASKATCHEWAN

The population of northern Saskatchewan, defined as those living in the Northern Administrative District of Saskatchewan, roughly equivalent to Census Division 18, includes those

living in the area of Athabasca Health Authority (AHA), and Keewatin Yatthé (KYHR) and Mamawetan Churchill River (MCRHR) Health Regions, as well as the Northern Village of Cumberland House and the Cumberland House Cree Nation (both within the Kelsey Trail Health Region or KTHR). The Northern Administrative District includes three ecologic regions across 270,000 square kilometers: the taiga shield, the boreal shield and the boreal plains, including coniferous and broadleaf tree forests, lakes and rivers along with muskegs, and rock in the central and northern aspects of the District.

The total population of the area in 2010 living in about 70 communities was 37,138 including 1001 infants under the age of 1 year (as an estimate of the number of newborns in a year) (Saskatchewan Ministry of Health, Covered Population 2010). By 2013, the population of the Northern Administrative District had increased to 38,999. In this area nearly 87% of people self-identify as Aboriginal (67% First Nations, 20% Metis and 13% non-Aboriginal) (Statistics Canada National Household Survey, 2011).

The Alberta Biomonitoring Program report on the prenatal study for women in 2005 showed some variation of blood levels of some chemicals by age of the mother. In Alberta, the average age for women giving birth at the time of the study (AHW, 2008) was 29.1 years of age (and in 2010 was 29.5 years) (Alberta Reproductive Health Report Working Group, 2011). The northern Saskatchewan average age for women giving birth during the study period was 24.7 years (about 4 ½ years younger than Alberta women). The age distribution for women giving birth during the study period for those living in northern Saskatchewan compared to those living in Saskatchewan as a whole shows larger percentages of births in northern Saskatchewan at younger ages than in Saskatchewan.



	< 15	15-19	20-24	25-29	30-34	35-39	>=40
North Sask	0.6	19.9	33.5	25.6	12.8	6.0	1.6
Saskatchewan	0.1	7.7	20.4	33.5	26.5	9.9	1.8

Figure 1: Percentage distribution of births by mother's age group – northern Saskatchewan compared to overall Saskatchewan: August 2011 to April 2013

Potential participants were notified of the biomonitoring study during routine prenatal check-ups. An opt-out process was used to obtain consent for individuals who decided not to participate. Study information was provided by the physician to obtain prospective consent during blood draw, allowing the use of any leftover blood that remained after routine blood testing had been conducted. Retrospective consent was obtained from participants whose 'leftover' blood samples were selected to be used after blood draw had already occurred. Remaining blood serum samples following completion of laboratory testing for the biomonitoring study are currently frozen and stored at the Alberta Center for Toxicology.

STUDY PROTOCOL

Participation and Sample Collection

In Saskatchewan, serum specimens are routinely collected from pregnant women during their initial prenatal assessment and tested for routine prenatal infectious disease screening. Following the testing, any residual serum remaining is stored for several months prior to being discarded.

For this study, eligibility for the study included:

- any women with a residential postal code within the Northern Administrative District who had prenatal blood work sent to the Saskatchewan Disease Control Laboratory between April 2011 and April 2013;
- when the specimen had at least 1 ml of residual serum remaining after the routine prenatal testing; and
- when either opt-in consent was provided for samples collected prior to August 1, 2011 or opt-out consent was implemented following a broad awareness strategy.

There were two phases of the study when it was initiated the beginning of August 2011. For those women who were identified with northern postal codes having specimens remaining at the Saskatchewan Disease Control Laboratory (SDCL) from April 1 to July 31, 2011, they were invited to participate in the study by letters sent through their health care provider who had done the initial assessment. Written consent was obtained for inclusion in the study of those specimens collected prior to August 1, 2011. During July 2011 and continuing until the completion of the study, there was broad public education through posters at health centers, phlebotomy sites and public places; pamphlets provided by public health and community health nurses, prenatal educators, and family physicians. For the second phase of the project, women were notified that if they wished not to participate in the study, they would just need to identify this to their health care provider. This 'opt-out' phase of the study started August 1, 2011 and continued until April 8, 2013.

From April 1, 2011 to April 8, 2013, 1,233 serum samples from pregnant women were received at Saskatchewan Disease Control Laboratory in Regina (SDCL) for routine infectious disease marker screening – this included all women who had received a prenatal checkup whose blood testing was normally done at SDCL and thus could potentially be included in the study. Rather than be discarded, any left-over sample containing at least 1 mL was retained for consideration in the study. There were 189 specimens available at the SDCL samples submitted from April 1, 2011 to July 31, 2011 and 1044 were submitted from August 1, 2011 to April, 2013. For those specimens submitted from April 1, 2011 to July 31, 2011, letters were mailed to the women's health care providers for samples. Seventy-one women were contacted and responded (71/189) for a response rate of 38%. Of those who responded to the mailed letters, 52 consented to have their specimens included, 6 declined, 13 consent forms were incomplete. Following the initiation of the community and clinic awareness program of the biomonitoring initiative, there were no refusals received asking to be excluded from the study.

Of the 1096 specimens received at the SDCL with written or opt-out consent from April 1, 2011 to April 8, 2013, 841 were found to have a minimum of 1 ml of residual blood to allow for inclusion in the study. (See Figure 2) About 59% of the total number of specimens from pregnant women in northern Saskatchewan were included in the pool which is relatively comparable to the Alberta sampling at 64% (28,484 samples drawn from 44,584 specimens collected).

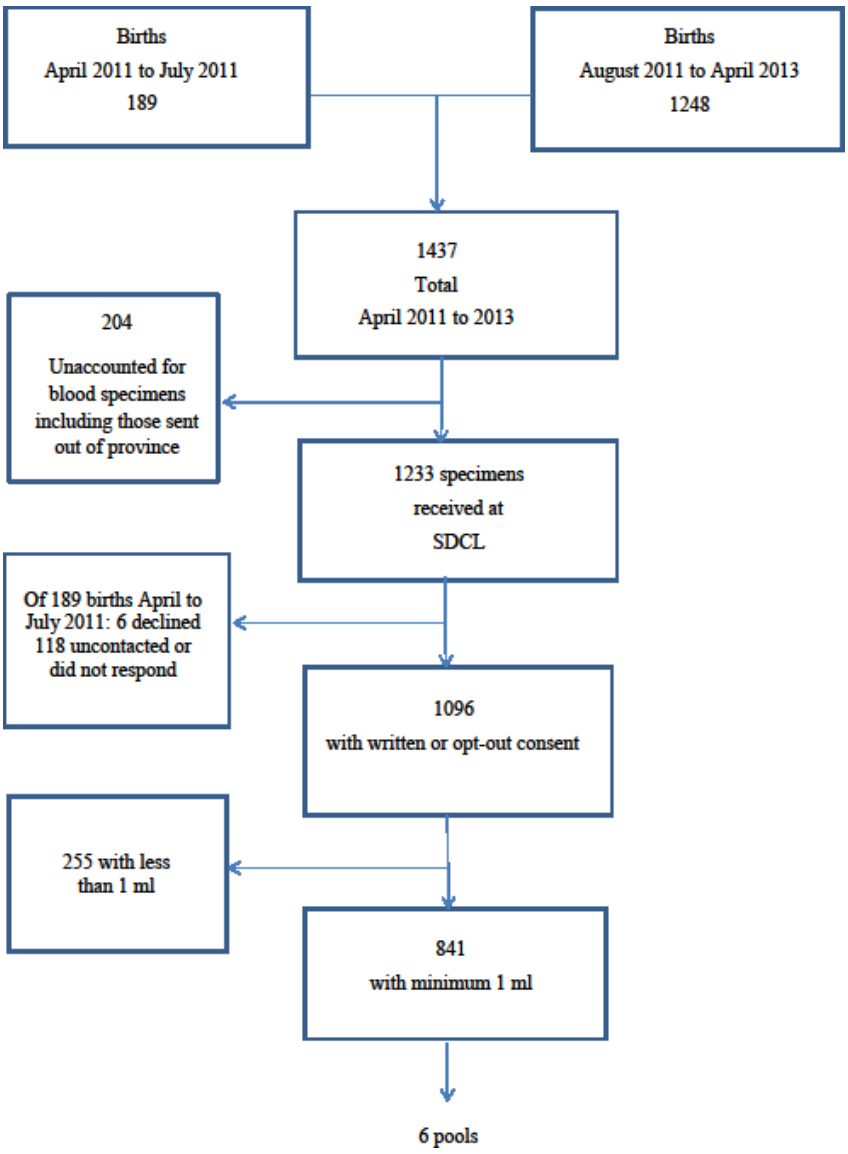


Figure 2: Sampling of perinatal blood tests from prenatal women April 2011 – April 2013

Further personal information other than postal code was not collected from study participants and, as such, data on birth number (multiparous vs. primiparous), age of the mother, length of gestation, trimester, and height/weight information is unavailable.

Pooled samples

The pooling of serum samples was performed at the Saskatchewan Disease Control Laboratory (SDCL) in Regina, SK with equal volumes of blood serum (estimated 1 mL) from each individual sample. The minimum number of individuals in a pool was determined by the minimum

volume of serum required for the analysis of the chemicals (approximately 100 mL). Due to the limited population in northern Saskatchewan, only six replicate pools could be generated. For quality assurance purposes, three control pools consisting of bovine serum were prepared in the same manner. The purpose of the controls was to monitor chemical contamination introduced by the routine handling of the blood samples or during the pooling process. The 6 pools were not analyzed in duplicate, nor were control samples sent along with the samples with the six pools to the labs.

Shipments were packaged according to the International Air Transport Association (IATA) and Transport of Dangerous Goods (TDG) Regulations. Codes were assigned to each pool (designating age class, geographic zone, and pool number) to ensure that all subsequent analysis was performed in a "blind" manner. The study codes were revealed for statistical analysis once the chemical analysis had been completed; thus, no subsequent data linkage to individual subject records will be possible.

Blood samples from northern Saskatchewan were stratified into pools of at least 120 samples (range 123-160). Six pools were created based on the geographic location of the postal code: one in the far north, two in the northeast and three in the northwest. Not all of northern Saskatchewan was sampled to the same extent. Individuals who lived in communities within the geographic areas served by the Flin Flon Hospital and had their blood specimens processed through the Flin Flon Hospital and subsequently the Cadham Provincial Laboratory in Manitoba were not included in the study though there were initial attempts to have the specimens referred on from Cadham Provincial Laboratory. This would include women living in Pelican Narrows, Deschambault, Denare Beach and Creighton. As well, due to some differences in the identification of prenatal blood specimens at the LaRonge Health Center, some of the initial specimens from the referral area were not included during the first year of the project. The results for the Pools 1, 2, 3, 4 and 6 will be fairly representative samples; however, Pool 5 will be less reflective for those communities whose prenatal blood work was done in Manitoba. This will introduce some bias for the overall northern mean levels as there will be over representation of the Pools 1, 2, 3, and 6.

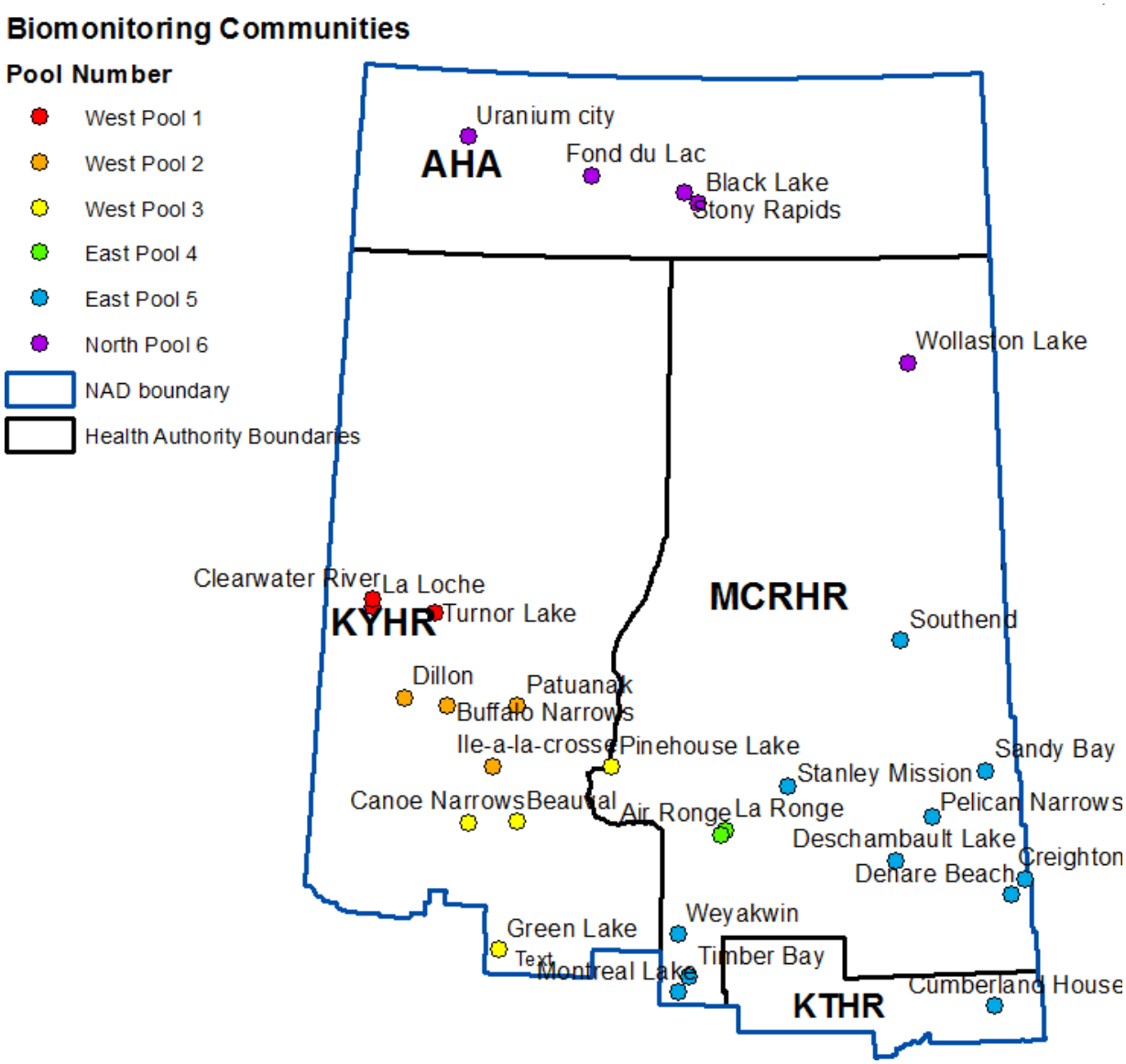


Figure 3: Sample pool locations

Table 1: Description of Pools

Pool	1	2	3	4	5	6
Number of individual samples per pool	162	138	120	130	150	141
Geographic Area	NW	NW	NW	NE	NE	Far North (FarN)
Postal code areas[a] included in this pool	Clearwater River La Loche Turnor Lake	Buffalo Narrows Dillon Ile-à-la-crosse Patuanak	Beauval Canoe Narrows Green Lake Pinehouse Lake	Air Ronge La Ronge	Creighton[b] Cumberland House Denare Beach[b] Deschambault Lake[b] Montreal Lake Pelican Narrows[b] Sandy Bay Southend Stanley Mission Timber Bay Weyakwin	Black Lake Fond du Lac Stony Rapids Uranium City Wollaston Lake

a = Specimens were sorted by postal code area – these postal code areas includes other communities that utilize this postal code area such as some smaller communities or some First Nations which use the same postal code to a neighbouring community.
b = limited sampling as most blood work done out of province

Selection of Chemicals for Biomonitoring

The chemicals monitored in this report were selected using expert guidance and by reviewing data from similar studies. The chemicals under study include industrial/agricultural by-products and chemicals used in the manufacture of consumer goods. The chemicals selected also include those labelled as contaminants of potential concern as outlined in The Stockholm Convention or other Federal regulations. Similar sets of chemicals are being tested in Alberta as part of past and ongoing phases of the Alberta Biomonitoring Study to provide comparisons.

Chemicals in this report, and other North American biomonitoring reports, include those that are known environmental contaminants but also includes concentrations for 'emerging contaminants', such as bisphenol A, parabens and phthalates for which fewer studies are available for comparison. In general, the chemicals reported in this study may be naturally occurring or synthetic, current-use or phased-out, and rapidly excreted or bioaccumulative. Each chemical, or chemical family, has unique sources, behaviour, and toxicological profiles that are discussed later in this report.

In following with the Canadian Health Measures Survey (CHMS Cycle 2, 2013), the selection of priority chemicals was chosen following one or more of the criteria described below:

- "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 respondents;
- availability and efficiency of laboratory analytical methods; costs of performing the test; and,
- parity of selected chemicals with other national and international surveys and studies."

Selection of Biomonitoring Matrix

Typically, multiple types of biological matrices can be sampled when collecting data on the internal concentration of a chemical in the body; however, each matrix presents a unique set of disadvantages and advantages. The validity of a biomarker is affected by the choice of matrix, and when designing a biomonitoring study additional criteria warrant consideration (e.g. using less invasive techniques could affect voluntary participation rates; the biological sample may reflect acute or long term exposures) (Hays et al., 2010; Arbuckle, 2010). A major impediment to the widespread use of biomonitoring in epidemiology studies is the cost of laboratory analysis chemicals in biological samples (Arbuckle, 2010). As such, in large biomonitoring studies where many chemical classes are analyzed, cost may become a factor when deciding whether or not to analyze chemicals in more than

one type of biological medium. For the sake of convenience and consistency within a study, and for potential cost reasons it may be desirable to choose only one matrix for analysis.

Blood is typically considered to provide a good reflection of internal body burden and is often used to compare and validate analysis of exposure measured using other biomarkers (Esteban and Castano, 2009). As well, many consider blood to be an ideal biomonitoring matrix for analyzing most chemicals due to its contact with all biological tissues in an organism, as well as being in a state of equilibrium with organs and tissues where chemicals may be stored (Esteban and Castano, 2009). However, where in the blood serum chemicals partition depends upon their chemical properties. Lipophilic chemicals, such as persistent organic pollutants, are primarily found within the lipid portion of the plasma or serum and as such their concentrations in the blood serum are typically made in reference to the lipid weight of the serum sample. This is done by dividing the total concentration of a lipophilic chemical in serum by the percent lipid content of blood. In this report, for chemicals that are considered lipophilic, both the measured serum chemical concentration and the calculated lipid level concentration, will be provided for reference. It is also worth noting that for certain metals, such as mercury, hexavalent chromium and cadmium, a large proportion of the compound absorbed into the blood will be found within in the blood cells, and as such serum measures will underestimate exposure and whole blood samples more accurately represent body burden (Kershaw et al., 1980, Health Canada, 1986, ATSDR, 2012). Care therefore must be taken when comparing the results of various studies as concentrations of the same chemical in different biological mediums (blood serum vs. whole blood sample vs. lipid adjusted blood serum) cannot be directly done. This is because certain biological sampling mediums will underestimate the presence of certain chemicals as compared to other biological sampling mediums, and therefore comparison between different biological sampling mediums may suggest differences in concentrations greater than what actually exists.

LABORATORY ANALYSIS

LABORATORY SELECTION

Select chemical analyses were done internally at the Alberta Centre for Toxicology (Calgary, AB). These chemical classes included cotinine, phytoestrogens and metals. PBDEs were analyzed by ALS in Prague, Czech Republic while methylmercury was analyzed by ALS in Sweden. The remaining chemicals were analyzed by ALS Laboratory Group (Edmonton, AB). This laboratory was selected in a competitive bid process based on the following evaluation criteria:

a. Evaluated fee as a fixed amount,
b. Timeline, processes and procedures,
c. Experience as a blood analysis firm (years, number and type of projects).

Furthermore, the same laboratories were selected for Alberta's Biomonitoring Program.

SUMMARY OF ANALYTICAL METHODS

The limit of detection (LOD) is the lowest concentration at which an analyte can be distinguished from the background (Armbruster et al., 1994). The LOD is defined by meeting pre-determined acceptance criteria (e.g., ion ratios within 20%, precision less than 20%, etc.) specific to a certain analytical method. The limit of quantitation (LOQ) is often set at a higher value and is the concentration at which concentrations of the analyte can be reported with confidence. The LOQ can also be determined by meeting pre-determined acceptance criteria related to LOD determination.

Concentrations are provided for chemicals measured above the LOD of the analytical instrument; however how to handle data that falls below the limit of detection represents a unique problem. While it is ideal to have the lab provide their best estimate for that value as opposed to having statisticians generate data for the sake of statistical analysis, it is not always possible for labs to provide these estimates (Arbuckle, 2010). A commonly used ad hoc method is to substitute the non-detected values with one half the value of the limit of detection (LOD/2) or by the limit of detection divided by the square root of 2 (LOD/√2) (Arbuckle, 2010; Baccarelli et al., 2005; Zeghnoun et al., 2007). While there is no theoretical basis behind this substitution method, it is commonly employed technique and studies have found that when the proportion of nondetect data is low, that is below 30%, the method by which nondetect data makes little difference in the results and does not significantly bias the data (Zeghnoun et al., 2007). Likewise, the EPA Guidance for Data Quality Assessment states that a substitution of half the detection limit may be used to estimate left censored data when the percentage of non-detects is low (Baccarelli et al., 2005; US EPA, 2000). Therefore, as the substitution method is not likely to bias the results, this study utilizes the substitution method for the estimation of concentrations below limits of detection or quantification in order to be consistent with the LOD/2 substitution method employed in Alberta Biomonitoring Program: Chemicals in serum of pregnant women in Alberta (AHW, 2008). A value of LOD/2 or LOQ/2 is substituted for concentrations designated as non-detects that fell below the LOD orthe LOQ (for the metals, minerals, and cotinine). This rule of substitution was applied to all chemical classes except PBDEs. Congeners with concentrations provided as <LOQ were substituted with the value of the ½ LOQ and values <LOD, marked as a non-detect, were substituted with the value of the ½ LOD (although none of the congeners tested required this particular substitution). This provides the upper bound estimate for the concentrations of PBDEs. This was done to be consistent with the previous Alberta study.

Cotinine

Serum samples (200 µL) were prepared for analysis via extraction on Bond Elute Certify solid phase extraction cartridges. Analytes were eluted with 78:20:2 dichloromethane: isopranol: ammonium hydroxide, dried under nitrogen, and reconstituted in mobile phase.

The extracts were analyzed using liquid chromatography-tandem mass spectrometry (LC/MS/MS) using a Zorbax Eclipse Plus Phenyl-Hexyl column (4.6 x 100 mm, 5 μm). The mobile phases are 20 mM ammonium formate and 0.1% formic acid in water and 20 mM ammonium formate and 0.1% formic acid in methanol. Analysis was performed in multiple reaction monitoring (MRM) mode with cotinine-d3 acting as an internal standard. Identification was based on retention time and MRM ratio. Quantification is based on area ratio and a 5 point calibration curve (5, 10, 25, 50, and 100 ng/mL) for concentrations above 5 ng/mL and a 6 point calibration curve (0.05, 0.1, 0.5, 1, 2, and 5 ng/mL) for concentrations below 5 ng/mL. The limit of detection (LOD) for this method is the concentration with at least a signal to noise ratio of 3, an ion ratio within 20% of that in the standard, and is reproducible (15% or less CV over three days). LOQ is the concentration with at least a signal to noise ratio of 10, an ion ratio within 20% of that in the standard, and is reproducible (15% or less CV over three days).

Analysis was performed on six human serum pooled samples from Saskatchewan, a set of high calibrators, a negative bovine serum control, a negative human serum control and four quality control (QC) samples. Two QC samples were pooled samples from the previous Alberta Biomonitoring Program study with known concentrations of cotinine, and the two other QC samples were verified blank bovine serum and verified blank human serum spiked with 5 ng/mL of cotinine standard from a differed source than the calibrators. Precision estimates were derived from running 8 replicates of two different cotinine concentrations over three days (Table 2).

Table 2: The analytical precision of the isotope dilution LC/MS/MS method used to analyze cotinine. Eight replicates were run at two concentration levels over a three day period.

Cotinine Concentration (ng/mL)	Interday Precision (n=8) (%)
5.00	3.70
100	2.80

Dioxins/Furans

Dioxins and furans were analyzed according to the U.S. EPA Method 1613, *Tetra- through Octa-Chlorinated Dioxins and Furans by Isotope Dilution HRGC/HRMS* (U.S. EPA, 1994). Isotopically labelled dioxins and furans, and an equal amount of formic acid and HPLC grade water (1:1:1) were added to an aliquot (25 g) of serum. The mixture was vortexed, sonicated and subjected to solid-phase extraction using an EZ-Extract C18 (10 g / 75 mL) cartridge. The cartridge was dried (via vacuum for 1h) and the analytes were eluted with hexane (50 mL). The extract was concentrated and cleaned up using a multi silica column. Further cleanup was performed using basic alumina and Florisil.

Analyses were performed using high resolution gas chromatography/high resolution mass spectrometry (GC: Hewlett Packard 5890 Series II, HRMS: Kratos Concept 1S HRMS W/ SUN Sparc computer running Mach 3 Data system, Autosampler: LEAP Technologies CTC A200SE).

A computer averaged concentration (X) of four 1 L aliquots of reagent water spiked with the diluted labeled compound spiking solution was calculated along with the standard deviation (s) of the concentrations in ng/mL for each compound. The average concentration and standard deviation was compred to corresponding limits for initial precision (Table 3). For more information refer to EPA method 1613 (U.S. EPA, 1994).

Table 3: Acceptance criteria for precision performance tests of dioxins and furans analyzed with Isotope Dilution HRGC/HRMS using EPA method 1613.[1]

Congener	Test Concentration	$IPR_{2,3}$	
	(ng/mL)	s (ng/mL)	X (ng/mL)
2,3,7,8-TCDD	10	2.8	8.3-13
2,3,7,8-TCDF	10	2.0	8.7-14
1,2,3,7,8-PeCDD	50	7.5	38-66
1,2,3,7,8-PeCDF	50	7.5	43-62
2,3,4,7,8-PeCDF	50	8.6	36-75
1,2,3,4,7,8-HxCDD	50	9.4	39-76
1,2,3,6,7,8-HxCDD	50	7.7	42-62
1,2,3,7,8,9-HxCDD	50	11	37-71
1,2,3,4,7,8-HxCDF	50	8.7	41-59
1,2,3,6,7,8-HxCDF	50	6.7	46-60
1,2,3,7,8,9-HxCDF	50	6.4	42-61
2,3,4,6,7,8-HxCDF	50	7.4	37-74
1,2,3,4,6,7,8-HpCDD	50	7.7	38-65
1,2,3,4,6,7,8-HpCDF	50	6.3	45-56
1,2,3,4,7,8,9-HpCDF	50	8.1	43-63
OCDD	100	19	$89\text{-}1.3 \times 10^2$
OCDF	100	27	$74\text{-}1.5 \times 10^2$

1 All specifications are given as concentration in the final extract, assuming a 20 µL volume.

2 s = standard deviation of the concentration.

3 X = average concentration.

Metals

Serum samples were diluted in a basic solution containing ammonium hydroxide, butanol, EDTA, Triton X-100, gold and internal standard. An aliquot of serum sample (400uL) was mixed with 1.6mL of deionized water. 2mL of basic solution with IS was added to the diluted sample, which results in 10-fold dilution. The samples were analyzed by ICP-MS-MS (Agilent 8800) directly after 10

minutes of sonication. Boron and silver serum samples were diluted in an acidic solution containing 1% nitric acid, 0.5% hydrochloric acid, gold and internal standard (IS). An aliquot of serum sample (500uL) was mixed with 2mL of acidic solution containing 1% nitric acid, 0.5% hydrochloric acid and gold. 2.5mL of acidic solution containing 1% nitric acid, 0.5% hydrochloric acid, gold with IS was added to the diluted sample, which results in 10-fold dilution. The samples were analyzed by ICP-MS-MS directly after 10 minutes of sonication. The LOD and LOQ for metals and minerals were determined from the calibration curve. The LOD was defined as the concentration yielding at least 3 times of the absolute abundance of the blank diluent. The LOQ used in the report is a "reporting limit" that is 10 times the value of the interim LOQ because the 10x takes into account a 10x dilution in the sample preparation.

In each batch of samples, calibrators and two sources of CRMs (Certified Reference Materials) were run prior to sample injections. The CRMs used in this analysis are Seronorm Trace Metals Serum Control Level 1 and 2 and Clinchek Trace Metals Serum Control Level 1 and 2. The CRMs were re-injected after every 10 samples and the results were accepted within 20% range of the target values.

Two aliquots of each sample were analyzed. The difference in percentage (%diff) was calculated and the results were accepted if the %diff is less than 15%. If the % diff was higher than 15%, the sample was repeated in a different run. In order to investigate the precision, accuracy, and recovery of the method, three runs were set up on different days. Precision measurements are shown in Table 4 and Table 5. Calibrators and two sources of CRMs were run prior to sample injections. Blank serum was spiked at two different levels. On each day, the blank serum, low spiked serum sample and high spiked samples were injected 10 times. Chemical analysis of metals provides a measurement of total metals in the sample, that is, both inorganic and organic metals as well as metals of various speciations.

Table 4: The analytical precision estimates of the ICP-MS-MS method used to analyze metals and minerals. The precision of the method was determined by calculating the percent difference (% coefficient of variation) between three runs on three different days. If the values were below LOD, the result is shown as <LOD.

Analyte	Ave Conc. from 3 runs(ug/L)	Run #1 (%CV)	Run #2 (%CV)	Run #3 (%CV)	Between runs (%CV)
Be	<LOD	101.9	109.7	<LOD	<LOD
B	3.9	6.9	1.7	1.0	3.3
Mg	1840.2	0.9	2.1	1.1	6.5
Al	2.6	4.4	12.5	4	2.5
Ti	0.12	9.7	16.8	34.4	25.5
V	<LOD	<LOD	<LOD	88.4	<LOD
Cr	0.32	2.2	1.2	4.3	4.7

Mn	0.07	12	4.7	3.6	8.8
Fe	61.9	3.6	1.1	0.4	2.5
Co	0.027	2.4	4.2	2.8	11.3
Ni	0.07	6.4	8.9	6.9	3.7
Cu	98.8	1.4	1.3	1.1	1.1
Zn	82.3	1.4	1.5	1.2	2.4
As	0.022	2.8	3.4	1.8	4
Se	10.1	3.1	3.6	4.3	7.8
Sr	2.6	3.2	2.1	1.6	2.8
Mo	0.013	16.2	26.5	53.7	76.5
Ag	0.001	32.1	12.8	36.9	21.7
Cd	0.005	11.2	9.5	13.3	35.3
Sb	0.094	2	1.6	2.5	5.7
Cs	0.039	6.5	6.6	2.9	10.3
Ba	0.30	5.7	10.4	9	4.5
W	0.01	25.6	111	16.5	107.9
Pt	0.003	59.7	106.2	20.3	71.8
Hg	0.014	5.4	2.6	3.6	12.2
Tl	0.001	42	5.6	5.9	54.9
Pb	0.004	40.5	17.3	22	30.3
U	0.002	57.6	60.7	106.5	136.8

Table 5: The analytical precision estimates of the ICP-MS-MS method used to analyze boron and silver. The precision of the method was determined by calculating the percent difference (% coefficient of variation) between three runs on three different days. On each day, Seronorm Trace Metals Serum Control Level 1 (Ref 201405, Lot# 1309438) and Clinchek Trace Metals Serum Control Level 1 (Ref 8880, Lot# 347) were injected 10 times.

Analyte	Run #1 (%CV)	Run #2 (%CV)	Run #3 (%CV)	Inter-day (%CV)
B	2.9	2.9	6.3	4
Ag	3.3	2.9	5.9	4

Methylmercury

Isotopically labelled methylmercury was added to 2.5 mL of blood. The enriched sample was subjected to alkaline digestion followed by extraction of methylmercury into dichloromethane and back extraction into water (Baxter et al. 2011; Baxter et al., 2007). Methylmercury was converted to the volatile ethyl derivative, purged and trapped on a solid-phase collection medium, and then introduced into the gas chromatography – inductively coupled plasma mass spectrometry (GC-ICPMS) system. The GC-ICPMS system consisted of a Fisons Instruments (now Thermo Electron) 8000 Series gas chromatograph equipped with a 15- m capillary column (0.53 mm i.d., 1.5 µm BP-1,

Supelco) and coupled to an ICP – sector field mass spectrometry instrument (Element2, Thermo Scientific, Bremen, Germany) operated in low resolution mode and with guard electrode in order to maximize sensitivity. Performance characteristics of the method were tested at several concentrations of methylmercury added to human serum (Baxter at al., 2007). The LOQ was estimated at 10 times the standard deviation of concentrations measured in preparation blanks. Within and between-run relative standard deviations (RSDs) are consistently below 10% using this method. Precision estimates of this method developed by Baxter et al. (2011) are given in Table 6.

Table 6: The analytical recovery and imprecision of the isotope dilution GC-ICPMS method developed by Baxter et al. (2011).

Added methylmercury* (ug/L)	Within run (n=7) RSD (%)	Between run (n=13) RSD (%)
0.14	2.6	3.2
0.35	4.9	5.6
2.8	9.3	9.7

*Concentration in the unspiked, commercial, human serum was (0.138 ± 0.018) g L−1; uncertainty term is 95% confidence interval.

Organochlorine Pesticides

Organic pesticides were analyzed according to U.S. EPA Method 8081B for Organic Pesticides by Gas Chromatography (U.S. EPA, 2007). Surrogates (tetrachloro-m-xylene and decachlorobiphenyl) were added to an aliquot (4 g) of serum. Following denaturation with 4 mL methanol, the sample was extracted with 8 mL hexane/diethyl ether (1:1) via vortex and sonication. Following removal of the solvent, the extraction was repeated twice more with hexane/diethyl ether.

The resulting extracts were combined, dried over sodium sulphate and concentrated to 1 mL. Cleanup was performed using Florisil and the extract was concentrated to 75 µL. After addition of internal standard (25 µL pentachloronitrobenzene) the final extract was analyzed by gas chromatography/dual column electron capture detection (Agilent Model: 6890N, Towers/ Injectors: 7683B). For organochlorine pesticides, the LOD differs by pool depending on number of samples in the pool and the signal-to-noise ratio of the analytical instrument

The method used in this study utilized SPE to extract the pesticides, this method was used for groundwater and waste water extraction in EPA method 8081B (U.S. EPA, 2007). As much precision estimates for this ground water and waste water from the EPA method are provided in Table 7. Precision estimates were determined from seven replicates of each sample type. Two spiking levels were used. "Low" samples were spiked at 5-10 µg/L for each analyte, while high samples were spiked at 250 - 500 µg/L.

Table 7: Precision estimates taken from EPA method 8081B for the extraction of OC pesticides using gas chromatography.

Compound	Precision (%)			
	Ground water (low)	Ground water (high)	Waste water (low)	Waste water (high)
Aldrin	24	5.5	6.7	3.4
β-BHC	6.5	2.5	1.6	4.2
δ-BHC	5.6	2.4	2.5	4.2
cis-Chlordane	13	2.7	4.7	2.4
trans-Chlordane	16	2.7	4.6	2.9
Dieldrin	7.1	2.3	3.8	3.6
Endosulfan I	11	2.3	4.1	3.8
Endosulfan II	5.8	2.8	4.2	4.1
Endrin	6.2	2.3	3.1	2.9
Endrin aldehyde	6.0	4.0	3.3	5.9
Heptachlor	19	3.9	5.0	2.8
Heptachlor epoxide	12	2.4	2.9	3.3
Lindane	11	3.2	2.4	3.1
4,4'-DDE	8.3	2.5	4.4	2.4
4,4’-DDT	4.4	2.7	4.3	4.7
4,4’-TDE (DDD)	4.8	2.4	4.6	2.9

Perfluorinated Compounds

The sample is prepared for extraction by vortexing and sonicating a mixture of 0.1M formic acid along with isotopically labelled standards and 1 mL of serum. A solid phase extraction (SPE) is performed on the sample using OASIS HLB SPE cartridges. The samples were eluted with 1% ammonium hydroxide/acetonitrile and the resulting extract was concentrated to 100 µL under nitrogen and 25 mL of recovery standard (fluoro-n-heptanoic acid) was added along with 175 µL of 90% 20 mM acetic acid/10% methanol.

The extracted sampled was analysed using liquid chromatography tandem mass spectrometry (LC/MS/MS) (API 3000 LC/MS/MS Sciex, Perkin-Elmer 200 Autosampler, Series 200 Micropump Perkin- Elmer) using multiple reaction monitoring (MRM). Precision estimates for this method are provided in Table 8.

Table 8: The analytical precision estimates of the LC/MS/MS method used to analyze PFCs. The precision of the method was determined by calculating the percent difference between two duplicate runs. If the values were below LOD, the result is shown as <LOD.

Compound	% Difference
PFHX	12.6
PFOS	9.7
PFDS	<LOD
PFOA	7
PFNA	<LOD
PFDA	<LOD
PFDoA	<LOD
PFUA	<LOD

Phenols

20 µL of labelled internal standard and 20 µL of 4-methylumbelliferone surrogate mix are added to 100 µL of serum. 50 µL of β-glucuronidase is added and the vials are capped, vortexed, and incubated at 37ºC for two hours. After incubation, the vials are removed from the oven, uncapped, and the incubation is stopped by the addition of 80 µL of 1M formic acid and HPLC water to make up a 1 mL final volume. The vials are then recapped and transferred to the autosampler tray, which is set at 4˚C. Samples are further cleaned and pre-concentrated using on-line solid phase extraction (SPE), where the sample is injected into the Transcend Multiplexing system in TX mode and loaded onto a Cyclone-P SPE HTLC column. Following extraction the samples are separated by reverse-phase HPLC and detected using atmospheric pressure chemical ionization (APCI)-MS/MS. Precision estimates for this method are provided in Table 9.

Table 9: The analytical precision estimates of the On-line SPE-HPLC-Isotope Dilution-MS/MS method used to analyze phenols. The precision of the method was determined by calculating the percent difference between two duplicate runs. If the values were below LOD, the result is shown as <LOD.

Compound	% Difference
Octylphenol	15.3
	16.3
Nonylphenol	<LOD
	<LOD
BPA	<LOD
	<LOD
Pentachlorophenol	<LOD
Trichlorophenol	<LOD

Phytoestrogens

0.5 mL of serum per sample was used for analysis of daidzein and genistein. An internal standard was added for quantification and compensation for any sample loss during extraction. This method involved enzymatic hydrolysis using a purified extract of Helix pomatia containing β-glucuronidase and sulphatase from Helix pomatia, followed by protein precipitation. Samples are then centrifuged and filtered before injecting on LC/MS/MS. 10 µL of extracted sample is injected onto LC/MS/MS (Agilent 1100 HPLC/Sciex API 4000). The LC column is a Zorbax SB-C18 rapid resolution column (4.6 x 50mm, 3.5µm). The mobile phases are 5 mM ammonium formate in D.I. water and 5 mM ammonium formate in methanol. Daidzein and genistein are analyzed in MRM (multiple reaction monitoring) mode. Identification is based on retention time and MRM ratio. Quantification is based on area ratio and a six point calibration curve (0, 0.5, 1, 2, 5, 10, 20ng/mL). The LOD for this method was defined as the concentration at which the S/N (signal/noise) of the analyte >3, and ion ratio within the range of +/- 20% of that in the standard. The LOQ was defined as the concentration at which the S/N of the analyte >10, and ion ratio within the range of +/- 20% of that in the standard.

Analysis was performed on six human serum samples from Saskatchewan, a set of calibrators, a negative control and one quality control. The calibrators, negative control and QC were prepared by spiking daidzein and genistein standard solution into verified blank bovine serum. The quality control had to be within ±20% of the target values. The recovery for sample spike must be between 70 to 130%. The calibration curve is acceptable if its correlation coefficient is ≥ 0.997. Percent coefficient of variation for the replicate analysis of 6 samples of 5 ng/mL of daidzein and genistein, respectively is given in Table 10.

Table 10: The analytical precision estimates of the isotope dilution LC/MS/MS method used to analyze daidzein and genistein. The precision of the method was determined by running 6 samples, each spiked with 5 ng/mL of daidzein and genistein.

Compound	Coefficient of Variation (%)
Daidzein	5.3
Genistein	3.9

Polybrominated Diphenyl Ethers

Isotopically labelled standards were added to 50 g of serum, in addition to ammonium sulphate, methanol, and hexane/diethylether (2/1). The mixture is sonicated for ten minutes and then shaken for another 10 minutes before transferring the hexane layer to another flask. The extraction is repeated with a fresh portion of hexane/diethylether. The combined hexane extracts are dried with anhydrous sodium sulfate and concentrated using a Kuderna-Danish apparatus. Residual solvent is evaporated in an oven. Fat content is determined gravimetrically, diluted in 5 mL

of hexane and transferred into a separatory funnel. An equal amount of dimethylsulfoxide is added and the mixture is shaken intensively. The DMSO layer is removed and the extraction with DMSO is repeated three times. The DMSO portions are diluted with an equal amount of water and reverse extractions with 3 x 5mL of n-hexane are performed and the final extract is concentrated. Analyses were performed using high resolution gas chromatography/high resolution mass spectrometry (Mass spectrometer: Termo Electron Corp, DFS operated in MID mode, reference gas PFTBA equipped with Trace GC Ultra with autosampler Thermo Electron Corp). The percent relative difference between duplicates should be less than or equal to 50% for nona- through deca-brominated analytes and 35% for all other levels of bromination, but only where the response is greater than the low calibration standard.

The Limit of Detection for this analytical method is defined for an individual congener as the concentration of an analyte in the extract of a sample which produces an instrumental response at two different ions with S/N (Signal/Noise) ratio of 3:1 for the less sensitive signal. The Limit of Quantification is defined as the double of the detection limit (LOD) in the case of an individual congener which is not detected in the on-going blank (negative blank). Limit of Quantification for an individual congener which is detected in the blank (positive blank) is defined as the double of the maximal concentration in the on-going blank.

Polychlorinated Biphenyls

PCBs were analyzed according to protocol in U.S. EPA's Method 1668c for chlorinated biphenyl congeners in water, soil, sediment, biosolids and tissue by HRGC/HRMS (U.S. EPA, 2010).

Isotopically labelled PCBs along with an equal amount of formic acid and HPLC grade water were added to an aliquot (5 g) of serum. The mixture was vortexed, sonicated and subjected to solid phase extraction using an EZ-Extract C18 (10 g / 75 mL) cartridge. The cartridge was dried (via vacuum for 1h) and the analytes were eluted with hexane. The extract was concentrated and cleaned up using a multi silica column, followed by basic alumina and Florisil. Analyses were performed using high resolution gas chromatography/high resolution mass spectrometry (GC: Hewlett Packard 5890 Series II, HRMS: Kratos Concept 1S HRMS W/ SUN Sparc computer running Mach 3 Data system, Autosampler: LEAP Technologies CTC A200SE).

For each PCB and labeled compound, the Relative Standard deviation (RSD or coefficient of variation) and the computed average percent recovery of results of the set of four analyses of spiked samples (X) were compared with the corresponding limits for initial precision and recovery. If RSD and X for all compounds meet the acceptance criteria, system performance is acceptable and analysis of blanks and samples may begin. If, however, any individual RSD exceeds the precision limit or any individual X falls outside the range for recovery, system performance is unacceptable for that

compound. The Initial Precision and Recovery limits used in this method was a RSD value of 25% or 50% depending on the congener tested. For more information refer to EPA method (U.S. EPA, 2010).

Phthalates

Serum samples are subjected to enzymatic hydrolysis by adding 500 µL of serum sample and 50 µL of β-glucuronidase from H. Pomatia H1 in pH 5 ammonium acetate buffer to a 5 mL deep well plate. Following the addition of phthalate internal standard mix and 4-methylumbelliferone gluronide, the plate is incubated at 37˚C ±1°C for 17 hours. The incubation is stopped by the addition of 0.1 M formic acid.

Serum samples are added to preconditioned SPE. Following washes with water, and elution with methanol, the culture tubes are placed in a Genevac evaporator for 3 hours. Following evaporation, the tubes are reconstituted with acetonitrile in water, along with d4-MEHP recovery standard. The samples are sonicated, vortexed and transferred to autosampler vial inserts and then vortexed again for 10 seconds. Samples are injected onto a Synergi Max-RP column (4.6 × 250 mm, 4 µm) and analyzed by APCI-MS/MS operating in negative mode (Applied Biosystems MDS Sciex API 3000). Precision estimates for this method are included in Table 11.

Table 11: The analytical precision estimates of the isotope dilution LC/MS/MS method used to analyze phthalate congeners. The precision of the method was determined by calculating the percent difference between two duplicate runs. If the values were below LOD, the result is shown as <LOD

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Compound	% Difference
Monomethyl Phthalate	<LOD
	<LOD
Monoethyl Phthalate	25.3
	14.5
Monoisobutyl Phthalate	16.7
	2.2
Monocyclohexyl Phthalate	<LOD
	<LOD
Monobenzyl Phthalate	5.8
	15.2
Mono-(2-ethylhexyl) Phthalate	11.2
	20.4
Mono-n-octyl Phthalate	<LOD
	<LOD
Monoisononyl Phthalate	<LOD
	<LOD
Mono-(2-ethyl-5-hydroxyhexyl) phthalate	<LOD
	<LOD

Parabens

20 µL of paraben surrogate and 4-methylumbelliferone surrogate mix was added to 100 4-methylumbelliferone surrogate mix of the serum sample. The serum samples are hydrolyzed with 4-methylumbelliferone glucuronide and β-glucuronidase in ammonia acetate buffer and are incubated at 37˚C±1°C for 90 minutes. Incubation is stopped by the acidification of the sample by 1.0 M formic acid. Samples are filtered, capped, and transferred to the autosampler tray. A 300 µL aliquot of the sample is injected into the Transcend Multiplexing system in TX mode and loaded onto a Cyclone-P SPE column (0.5 x 50 mm). The samples are then injected onto a Hypersil Gold column (50 x 2.1mm) and analyzed by atmospheric pressure chemical ionization (APCI)-MS/MS (Thermo TSQ Vantage mass spectrometer). Precision estimates for this method are provided in Table 12.

Table 12: The analytical precision estimates of the On-line SPE-HPLC-Isotope Dilution-MS/MS method used to analyze parabens. The precision of the method was determined by calculating the percent difference between two duplicate runs. If the values were below LOD, the result is shown as <LOD.

Compound	% Difference
Methyl Paraben	27.5
	17.5
Ethyl Paraben	23.1
	<LOD
Propyl Paraben	10.5
	0
Butyl Paraben	<LOD
	<LOD
Benzyl Paraben	<LOD

DATA ANALYSIS

There are two limitations to data analysis related to pooled sampling. The first is the fact that underlying statistical assumptions limit interpretation of pooled samples, and the second is the sampling method used in this study. The interplay between these realities limits extrapolation of findings and limits higher analysis of the pooled samples. In a study where the sampling method is random, measures of central tendency and variance can be determined, these values can then be used to extrapolate to the general population. However, in this study a random sampling approach was not applied as individual women were not selected at random to participate in this study, and instead a form of convenience sampling was utilised with varying degrees of coverage in each geographic area. As well, there is no information available on the demographics of women who

chose not to participiate, therefore self-selection bias may affect the degree to which these results may be applied to the larger population. Therefore, means calculated in this study from the six

Saskatchewan pools are to be interpreted as means of the pregnant women who were tested in these regions as opposed to means representative of the entire northern population of pregnant women in Saskatchewan.

Descriptive statistics in this report were only calculated for chemicals with at least five pools with concentrations above the limit of detection or limit of quantification. As previously discussed, substituting a value of the half the limit of detection (LOD/2) is an appropriate method of estimation when the proportion of data that is below the limit of detection, and thereby the proportion of data that is given a substituted value is low (Zeghnoun et al., 2007). For chemicals with five pools with concentrations that were above the LOD or LOQ, the value of LOD/2 is substituted as the concentration for the pool with a concentration lower than the LOD or LOQ. This allows for a minimal proportion of estimated or substituted data for each chemical, that is 1 out of 6 pools (~17% censored data). This method of substitution was only used for 9 of the chemicals included in this report (PCB 13, PCB 29, PCB 63/76, PCB 151, PCB 132, PCB 141, PCB 158/129, hexachlorobenzene and, PBDE 85), as all the other chemicals reported in this study were detected in all 6 pools. For phytoestrogens, metals, and minerals the LOQ/2 was used to substitute pool values below the LOQ. PBDEs with values of <LOQ were substituted with the value of the ½ LOQ and values that were lower than the LOD, marked as a non-detect, were substituted with ½ LOD (although none of the PBDE congeners in this study actually required this substitution) in order to be consistent with methodology used in the Alberta Biomonitoring study.

Analyses were conducted using Microsoft Excel (2003) and SAS 9.4, and graphs were generated using SigmaPlot (version 12.5). The estimated concentrations were analyzed using descriptive statistics: weighted mean, and 95% confidence interval. To obtain a representative overall northern Saskatchewan mean concentration from the six pools arising from the three regions that could be comparable to the results of the Alberta Biomonitory Program (AHW, 2008), weighting was necessary as all pools differed in number of samples. Due to the pooled nature of the samples analyzed in this study, arithmetic means were used to calculate descriptive statistics (Heffernan et al., 2014). Calculations of weighted arithemetic means and weighted standard deviations were calculated using SAS 9.4 software. The detected concentration in a pool was multiplied by the number of its samples. The sum of these products was divided by the sum of the number of samples (total participants), resulting in a weighted arithemetic mean of the concentration for all the pools.

Calculation of standard deviation and standard error was adjusted to account for the pooled nature of the data using the square root of either the average sample size per pool (140.2) or the total size of the sample (841), as appropriate. If the average number of samples per pool is used, the variability can be multiplied across the means by the square root of the average sample size which gives us an estimate of the standard deviation that would have occurred within the sample as a whole. The estimated standard deviation is then divided by the square root of the total number of

samples for an estimated standard error. The estimated standard error of the sample mean is an estimate of the standard deviation of the imaginary normal distribution of all possible sample means.

The confidence limits presented uses the estimated standard error derived from the sample data (as outlined above) to calculate the 95% confidence intervals. That is to say, in a normal distribution, only 5% of the values are more the ±1.96 standard deviations away from the expected value, so only 5% of the sample mean values are more than ±1.96 SÊ (x) distant from the mean. Tests of statistical significance were not performed as the percentage of values requiring imputation is too great and would create a substantial bias in the data. ANOVA or weighted regression analysis for effects by region could not be performed with the relatively small number of pools in the study.

Only chemicals that met the inclusion criteria for statistical analysis are included and discussed in depth in the results section of this report, with the exceptions of methylmercury, chromium, cadmium, arsenic, boron, uranium and bisphenol-A as these are compounds of interest.

INTERPRETATION CONSIDERATIONS - IMPACT OF SAMPLE POOLING

Advantages and disadvantages of pooled serum samples over using individual data should be considered:

<u>Improved detection at the expense of inter-individual information</u>

Pooled data may increase the sensitivity for detecting the chemicals in a population, but will limit the ability to determine inter-individual variations within then the sample population. Pooling of samples may mitigate some of the variance between individual samples. The distribution of chemicals within individual samples is generally assumed to be log-normally distributed; however, estimates of pooled samples which are comprised of many individual samples are normally distributed according to the Central Limit Theorem (Alberta Health and Wellness, 2008; Caudill, 2010; Heffernan et al., 2014,). As pooling may average concentrations around outliers, information about regional variability may be lost. Further, pooling can bias the signal-to-noise ratio if either high or low outliers are present resulting in a high or low biased signal. As well, in the presence of extreme outliers, the pooled concentration which represents an arithmetic mean of the samples making up the pool may be skewed to the right and may not as accurately reflect the true central tendencies of the data, therefore care must be taken to not interpret or apply pooled data concentrations at the individual level.

<u>Increased detection and increased feasibility at the expense of not being able to connect outcome to exposure at the individual level</u>

Evidence of a detected chemical within a pooled sample, while technically representative of the mean concentration of all individual samples making up that particular pool, does not imply that

every individual sample within the pool actually contained that chemical. It is possible that a few individual samples within the pool had higher concentrations of the detected chemical thereby

skewing the mean. Since samples were pooled for analysis, results will not correspond to individuals but to the population; aside from the inability to provide individual level results this also limits the ability to quantify risk by exposure patterns/behaviour of the individual. It will also limit the ability to have statistically definitive relationships between the regions and detected concentrations. As such, it is impossible to infer exposure on an individual level from pooled samples and the concentrations presented in this report should be interpreted with care. Likewise, due to statistical limitations stemming from limitations in the study design, the overall means calculated from Saskatchewan's six pools should not be used to make conclusions about the entire population of women in Saskatchewan's north. This is due to the fact that random sampling or sampling the entirety of a population are necessary in order to make assertions of population level values from the means of pooled samples, and this study did neither (Caudill, 2010; Heffernan et al., 2014). Therefore, a geometric mean that is representative of the mean concentration of the entire northern population of pregnant women in Saskatchewan cannot be calculated from the six pools, and instead means in this study should be interpreted as "the means of tested, pregnant women in Saskatchewan's north".

Increased feasibility at the expense of reduced options of comparison and analysis

Statistically significant comparisons between the current study and another biomonitoring study where the samples are more stratified (by age or region) are not possible (age of the mother was not collected). Likewise, determining if there is variation overall by region across northern Saskatchewan is a challenge because of the number of pools. With the smaller Saskatchewan dataset, further statistical tests for region effects (ANOVA) are not possible.

Use of modern instrumentation at the expense of comparison and interpretation of results

Improvements to analytical instrumentation in the time periods between various biomonitoring studies will alter the reporting limits of the data i.e.: limit of detection or limit of quantification. Alterations in reporting limits can limit the comparisons that can be made between studies. The absence of a measurement does not necessarily mean a person has not been exposed. It may mean the instrument cannot detect such a small amount or that exposure did not occur close enough to the time of sample collection. Likewise, detection of an extremely small concentration of a compound due to highly sensitive analytical methods does not mean that the individual (or group of individuals in the case of pooled samples) are at risk. Many environmental chemicals, such as metals and minerals, are naturally present in the environment and therefore commonly found in the body. Breakdown products of certain chemicals can be the result of natural degradation by photolysis or microbial processes in various environmental compartments.

Feasible biological matrix selection at the expense of limiting comparison to existing information

Differences in biological matrix, reporting units, and propagation of measurement errors can account for apparent differences between results of different biomonitoring studies. The use of creatinine adjusted urine concentrations is not the same as lipid adjusted blood serum concentrations, and blood serum concentrations are not the same as whole blood concentrations due to pharmacokinetic differences in how chemicals move, are stored, metabolized and excreted. Therefore care must be taken to consider the biological matrix used in sampling when comparing results of one study to another.

Sampling proportion and comparability to other studies

The proportion of samples with a detectable concentration may be useful when comparing different populations. With respect to the Saskatchewan study design, the proportion of samples detecting a chemical could not be directly compared to the data from NHANES or CHMS owing to the pooled nature of the data - 80% detection in northern Saskatchewan pooled samples is not the same percent detected from hundreds of individual samples. While the First Nations Biomonitoring Initiative (Assembly of First Nations, 2013) study and the CHMS Cycle 1 and 2 (Health Canada, 2010a; Health Canada 2013) used 40% of samples above detection limits as a cut-off, and Alberta (AHW, 2008) used 25% detection as a cut-off for further data analysis, this method could not be applied to the northern Saskatchewan data due to the smaller number of samples. In preliminary simulations using log normal data, less than 1% of pooled samples of 100 individuals would be non-detects, even when up to 54% of individual samples would have been non-detects. With pooled samples of 10 individuals, fewer than 1% of pooled samples would be non-detects even when up to 27% of individual samples would have been non-detects. In short, the sensitivity of detecting a chemical in a population is greater with pooled samples.

Further, the CHMS was conducted in a geographic area with a population of at least 10,000 and a maximum respondent travel distance of 100 kilometres, with total participants ranging from 5,000 to 6,400 persons. The Alberta province-wide study recruited approximately 28,400 total eligible participants for a total of 151 pools, vastly improving statistical power in analysis. Data analysis was stratified into nine sub-groups by region and age with an average of 7 pools per sub-group (AHW, 2008). The statistical precision may be reduced in Saskatchewan compared to Alberta due to these numbers. The NHANES reports also have a sample population that is one or two magnitudes greater than the northern Saskatchewan study. The current survey was not designed to cover the entire Saskatchewan population, nor was it designed to permit further breakdown by community. Therefore care must be taken when attempting to directly compare the results of this study with the results of other similar studies.

COMPARISON TO ALBERTA DATA: CONSIDERATIONS

Alberta data presented in this report is represented in the form in which it had previously been published in "Alberta Biomonitoring Program: Chemicals in Serum of Pregnant Women in Alberta (2005): Influence of Age, Location and Seasonality" (AHW, 2008). 28,484 serum samples for this study were drawn from 44,584 samples collected for the purpose of infectious disease screening in pregnant women from January 1st to December 31st 2005. These samples were stratified by geographic region, age, and month of receipt and were sent to Provincial Public Health Laboratory in Edmonton (AHW, 2008). 1 ml of serum was taken from individual samples and these aliquots were physically combined into replicate pools based on age and geographic region. 150 to 200 individual samples were used per pool, based on the minimum volume required for chemical analysis. A minimum of 8 replicate pools were analyzed per age group/geographic region combination. Samples were organized into three general geographic regions of Alberta- northern, central and southern Alberta. As well, samples were stratified into 3 age groups (18 to 25, 26 to 30, and 31 years of age and older). The final dataset consisted of data from chemical analyses performed on a total of 156 pools. All methods used in chemical analysis are described in the original program report (AHW, 2008).

The number of chemicals analyzed in the reported Saskatchewan study is greater than the number analyzed in the 2008 Alberta study; therefore, comparative data from Alberta is not available for all of the chemicals included in this report. For example, parabens and phthalates were not analyzed in the previous Alberta study, and therefore only the Saskatchewan data is presented graphically. Additionally, in the Alberta report only chemicals with more than 25% of pools having concentrations above the limit of detection were reported graphically. Inclusion criteria of data in this report follows similar standards. Furthermore, comparative Alberta data included in this report in graph formation represents only significant stratifications in the data. For example, chemicals graphed to show the data stratified by age and not geographical regions would have significant differences in concentrations between the 3 age groups, but not between geographical regions. Likewise chemicals graphically stratified by geographical region but not age, are those for which concentrations differed significantly by geographical region and not by age. Chemicals that differed significantly by both age and geographical region are graphed such that the data are stratified by both age and geographical region. Conversely, for chemicals in which no significant differences in concentrations arose along either stratification, the concentration of the chemical is provided as a single mean of all the pools.

RESULTS BY CHEMICAL CLASS

ORGANIC CHEMICALS

COTININE

GENERAL INFORMATION

Sources

Tobacco is considered to be the most preventable cause of death, killing more than malaria, tuberculosis, and HIV/AIDs combined, with over 37 000 Canadians dying from tobacco usage annually (WHO, 2008 and Rehm et al., 2006). Tobacco exposure can come through both inhalable and non-inhalable sources with an estimated 1 billion people worldwide smoking tobacco in the form of cigarettes (Alwan, 2010). Cigarettes are known to contain over 4,000 types of chemicals, 50 of which are classified as carcinogenic, and as such, cigarette smoke presents a serious health risk not only to smokers but to others in the form of second hand smoke (Alwan, 2010). Nicotine is a naturally occurring component in tobacco products and Canadian cigarettes contain on average 12.5 (SEM 0.33) mg of nicotine per cigarette and approximately 1 mg of nicotine is absorbed into the body of the smoker for each cigarette smoked with the remaining 75% of the nicotine emitted to the air (Benowitz and Jacob, 1984; U.S. EPA, 1992; CDC, 2013a; Kozlowski et al., 1998). Nicotine in environmental tobacco smoke is absorbed in the lungs and is distributed throughout the body making nicotine and its metabolites a good biomarker of tobacco exposure; however nicotine and its metabolites are eliminated from both the urine and blood plasma within 24 hours so these biomarkers are indicative of short term exposures (Sorenson et al., 2007).

Cotinine is a primary nicotine metabolite, with upwards of 70% of nicotine being converted into cotinine (Eskenazi et al., 1995). The concentrations of cotinine in body fluids are proportional to the extent of exposure to tobacco smoke. Cotinine remains in the body for a longer period of time (16 hr) than nicotine making it the preferred biomarker of environmental tobacco smoke. Additionally cotinine is able to readily cross the placenta making it a good biomarker for intrauterine exposure to tobacco smoke (Ivorra et al., 2014; Benowitz, 1983; Etzel, 1990, Benowitz and Jacob, 1994; Scherer et al., 1988; Tuomi et al., 1999).

Tobacco smoke is associated with increased blood level concentrations of certain heavy metals, such as cadmium, arsenic and lead. Concentrations of serum cotinine, a nicotine metabolite and biomarker of tobacco smoke exposure, are a concern in this study.

Smoking rates and regulations in Saskatchewan

Current tobacco use among Saskatchewan residents is approximately 18.5% of the adult (15+) population, above the national average of 16.1% (approximately 4.6 million), but not significantly. In youth aged 15-19, one in four (24.3%) reported ever having smoked a whole cigarette. Current smoking prevalence among youth aged 15-19 in Saskatchewan (2012) was 20.2%, the highest in the country, significantly above the national average of 10.9%. Although smoking rates fluctuated, there was a net decrease in prevalence from 1999 to 2015 within all age groups in Saskatchewan; however, the percentage of smokers in Saskatchewan 15 years and older stands at 16.9%, above the Canadian average of 13% (Reid et al., 2017).

Information from an in-hospital birth questionnaire administered to women postpartum in Saskatchewan revealed that between November 2007 and March 2010, approximately 54.2% of the northern population indicated that they smoked while pregnant compared to 24.2% of the general Saskatchewan population (Saskatchewan Ministry of Education Early Childhood Development and Integrated Services, 2011). The Athabasca Health Authority, Keewatin Yatthé and Mamawetan Churchill River health authorities reported 73%, 54.5%, and 49.5%, respectively, of women who smoked while pregnant (Irvine et al., 2011). The percentage of off-reserve females aged 12 and over in northern Saskatchewan who reported daily or occasional smoking was 42.4% from 2009-2010. For the general Saskatchewan population in the same category, it was 20.2% (Irvine et al., 2011). In 2013/14, the prevalence of current smokers aged 12 years and older in the combined three Northern Health Regions (41%) was almost twice as high as in Saskatchewan (22%). This statistically significant difference was magnified for women with 20% of female current smokers in SK compared to 49% in the North (Source: Statistics Canada, Canadian Community Health Survey (CCHS) CANSIM table 105-0502.)

The Saskatchewan Ministry of Health is responsible for developing and amending *The Tobacco Control Act* and *The Tobacco Control Regulations*. The goal of this legislation is to reduce youth access to tobacco products and protect Saskatchewan residents from the harms associated with environmental tobacco smoke. The Act was put into place in 2002 and has since been amended, most significantly in 2005 and 2010. Laws within *The Tobacco Control Act* include a ban on smoking in enclosed public places, in cars with children under the age of 16 present, around doorways, windows and air intakes of public buildings, a ban on tobacco use on school grounds, and a number of restrictions on the sale and advertising of tobacco products. As of October 1, 2010, amendments to *The Tobacco Control Act* further reduce youth access to tobacco products and continue to protect Saskatchewan people from the harms of environmental tobacco smoke. On April 1, 2011, the provision of *The Tobacco Control Act* banning the sale of tobacco and tobacco-related products in pharmacies came into effect.

Furthermore the Federal government has instituted Tobacco Reporting Regulations which ensures that tobacco manufacturers provide annual reports to Health Canada with their product ingredients, sale numbers, promotional activities and toxic constituents (Health Canada, 2011).

Possible health effects

Direct and second-hand tobacco smoke causes adverse effects related to diseases of the cardiovascular system, diseases of the respiratory tract, and cancers; particularly lung, larynx, and mouth (Hecht, 2008; Veglia et al., 2007). In addition to increasing the risk of noncommunicable diseases such as heart disease, various forms of cancer, and chronic respiratory diseases, tobacco usage is also known to cause adverse health effects during pregnancy (Alwan, 2010; Ejaz and Lim, 2005). Tobacco consumption during pregnancy has been linked with disruption of development, pre-term birth, adverse birthing outcomes and sudden infant death syndrome (Ejaz and Lim, 2005; Duskova et al., 2014). A Spanish study investigated the relationship between maternal and infant serum cotinine concentrations and found that infants born to moderate or heavily smoking mothers were significantly smaller than those infants born to non-smoking mothers (Ivorra et al., 2014). Another study suggests that infant birth weight decreases by approximately 1 g for each ng/mL unit increase in cotinine concentration in fetal serum (Eskenazi et al 1995). It is suggested that third semester cotinine exposure is the most detrimental to infant growth due to rapid development that typically occurs during the third semester (Eskenazi et al. 1995; Richard et al 1988).

Possible effects on biomonitoring results of other chemicals

Various biomonitoring studies have shown that smokers have higher levels of various metals in their serum such as cadmium, lead, and arsenic. In an update to the Fourth National Exposure Report (February 2015) (Fourth National Exposure Report – Updated Tables (2015) from the NHANES 2011-2012 survey period, there was a comparison of urinary concentrations of metals and arsenic species, urinary perchlorate, nitrate, and thiocyanate; metabolites of several polycyclic aromatic hydrocarbons (PAHs): and metabolites of several volatile organic compounds. Of the 66 different chemicals assessed, 29 were found to be significantly higher in smokers as compared to non-smokers – some as much as almost 10 times higher levels.

Table 13: Chemicals Compared and found to be statistically significantly different for smokers and non-smokers from the National Health and Nutritional Examination Survey (2011-12) for males and females combined 20 years of age and over*

Chemical	Geometric Mean Concentrations (urine) (95% Confidence Intervals)		Fold Difference
	Smokers	Non-Smokers	
Metals and Arsenic Species			
Cadmium (ug/g)	0.366 (0.295 – 0.383)	0.199 (0.163-0.216)	1.8
Lead (ug/g)	0.518 (0.465-0.576)	0.417 (0.386-0.451)	1.24
Molybdenum	35.6 (33.7 – 37.7)	40.4 (38.7 – 42.2)	0.88
Uranium	0.008 (0.007 - 0.009)	0.006 (0.005 – 0.007)	1.4
Thiocyanate			
Thiocyanate (mg/g)	4.53 (4.02-5.10)	0.933 (0.881-0.988)	4.86
Metabolites of Polyaromatic hydrocarbons (PAHs)			
2-Hydroxyfluorene (ng/g)	1260 (1140-1400)	190 (176-203)	6.63
3-Hydroxyfluorene (ng/g)	662 (595-738)	66.9 (62.0-72.1)	9.9
9-Hydroxyfluorene (ng/g)	666 (592-751)	240 (220-261)	2.8
1-Hydroxyphenanthrene	216 (200 – 234)	134 (123 – 145)	1.6
2-Hydroxyphenanthrene	123 (114 – 133)	62.4 (57.9 – 67.3)	2.0
3-Hydroxyphenanthrene	153 (144 – 163)	56.6 (52.3 – 61.3)	2.7
4-Hydroxyphenanthrene	41.6 (37.5 – 46.1)	20.4 (19.1 – 21.9)	2.0
1-Hydroxypyrene	266 (246 - 269)	96.8 (90.8 – 103)	2.7
1-Hydroxynapthalene	10.5 (9.06 – 12.1)	1.37 (1.25 – 1.51)	7.7
2-Hydroxynapthalene	13.5 (12.3 – 14.8)	3.69 (3.46 – 3.93)	3.7
Metabolites of Volatile Organic Compounds (VOCs)			
N-Acetyl-S-(2-carbamoyl-2-hydroxyethyl)-L-cysteine	31.1 (28.0 – 34.6)	15.0 (14.1 – 15.9)	2.1
N-Acetyl-S-(2-carbamoylethyl)-L-cysteine	121 (110 – 134)	42.4 (39.9 – 44. 9)	2.9
N-Acetyl-S-(2-carboxyethyl)-L-cysteine	250 (224 – 278)	93.6 (87.6 – 100)	2.7
N-Acetyl-S-(3-hydroxypropyl)-L-cysteine	1090 (968 – 1230)	224 (208 – 241)	4.9
N-Acetyl-S-(2-cyanoethyl)-L-cysteine	134 (118 – 151)	1.75 (1.57 – 1.94)	76.6
N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine	455 (390 – 531)	126 (119 – 134)	3.6
N-Acetyl-S-(3,4-dihydroxybutyl)-L-cysteine	365 (340 – 392)	269 (257 – 281)	1.4
N-Acetyl-S-(4-hydroxy-2-butenyl)-L-cysteine	63.1 (55.1 – 72.2)	8.12 (7.41 – 8.89)	7.8

N-Acetyl-S-(2-hydroxypropyl)-L-cysteine	115 (99.4 – 134)	61.0 (54.8 – 67.9)	1.9
N-Acetyl-S-(3-hydroxypropyl-1-methyl)-L-cysteine	1969 (1720 – 2240)	388 (362 – 416)	5.1
t,t-Muconic acid	132 (117 – 150)	73.8 (67.1 – 81.1)	1.8
Mandelic acid	311 (280 – 345)	150 (141 – 160)	2.1
2-Methylhippuric acid	109 (96.5 – 123)	30.1 (27.0 – 33.6)	3.6
3-and 4-Methylhippuric acid	732 (647 -828)	201 (187 – 215)	3.6
Phenylglyoxylic acid	338 (306 – 374)	186 (172 – 200)	1.8

* = where applicable, creatinine corrected levels provided

This information should be kept in mind, when assessing the serum levels in northern Saskatchewan where the percentage of women smoking in pregnancy is significant.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Serum cotinine concentrations measured among pregnant women in all pooled samples ranged from 46.8 ng/mL to 66.4 ng/mL (weighted arithmetic mean ± 95% confidence interval = 58.0 ng/mL ± 5.63 ng/mL). Non-smokers exposed to typical levels of secondhand tobacco smoke have serum cotinine levels of less than 1 ng/mL, though active smokers almost always have levels higher than 10 ng/ml and sometimes higher than 500 ng/ml. Non-smokers are normally defined as having serum cotinine concentrations below 10 ng/mL (CDC, 2013a). Therefore, the concentrations of cotinine measured here indicate that some of the pregnant northern Saskatchewan women included in this study were smoking or exposed to second-hand smoke or were otherwise exposed to nicotine at the time of their blood sample collection.

The First Nations Biomonitoring Initiative (AFN, 2013) reported cotinine geometric concentrations of 71.97 (46.93 – 110.37) µg/L creatinine adjusted urine in adults aged 20 years and older. However, concentrations in urine cannot be directly compared to concentrations in serum. The National Health and Nutrition Examination Survey (NHANES), Fourth Report (CDC, updated 2014) reported geometric mean concentrations among females in 2009-2010 to be 0.037 (0.033 – 0.042) ng/mL serum in the non-smoking population. The arithmetic mean concentrations of cotinine in pooled serum samples in phase one (2008) of Alberta's biomonitoring program ranged from 5.13 ng/mL to 55.0 ng/mL. Values of cotinine in Alberta across age groups and regions are all lower in Alberta than in Saskatchewan. The highest concentration in AB is 46.0 ± 2.72 (mean ± 95% confidence interval) ng/mL in North, age 18-25.

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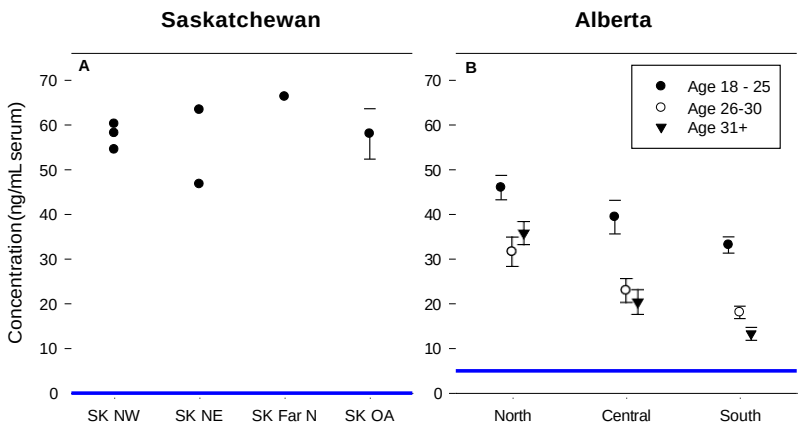

Figure 4: Concentrations of cotinine in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data presents the concentrations of the 6 pooled samples that were analyzed, as well as an overall (OA) mean concentration of the 6 pools. Means concentrations of each age group are given for the 3 regions of Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

PHYTOESTROGENS

GENERAL INFORMATION

Sources

Phytoestrogens are naturally occurring estrogen-like phenolic compounds found in plant material (Foster et al., 2002; (Bandera et al., 2011). There are three major groups of phytoestrogensL isoflavones (e.g. genistein, daidzein and glycitein), lignans and coumestans. .Daidzein and genistein are types of phytoestrogens known as isoflavones whichh are structurally similar to mammalian estrogen (Atkinson et al., 2005). The popularity of soybeans and other plants containing phytoestrogens has grown due to the suggestion that phytoestrogens may have protective effects against breast cancer, heart disease and osteoporosis without proper attention given to the possible negative side effects of phytoestrogen consumption (Schmitt et al., 2003).

The main route of exposure to phytoestrogens is diet and as was previously alluded to, isoflavones are naturally found in plants such as soy, and as such cultural implications such as diet may impact an individual's exposure to phytoestrogens. Daily isoflavone ingestion in women in Ontario is estimated to be 230 µg/day, that is, 143 µg/day of genistein and 64 µg/day daidzein (Cotterchio et al., 2008). Other studies estimate a daily intake of <2mg of isoflavones in people who live in Western nations as compared to the estimated 39-47.2 mg/day measured in Japanese people, 25.4 mg/day measured in Chinese and 20.9 mg/day measured in Koreans (Vergne et al., 2009). There appears to be a lot of inter-individual differences in the ability of a person to absorb and metabolize phytoestrogens that may be depend on differences in human pharmacokinetics or gut microflora, as

well as diet and ethnicity (CDC, 2013q; Vergne et al., 2009). Due in part to urinary excretion, run off

from agricultural waste of animals fed soy, and discharges from wood pulp mills, phytoestrogens can be found in waste water effluent thereby representing another route of potential exposure (Liu et al., 2010; Kang and Price, 2009). Exposure to infants can come from soy-based formula and human breast milk are the major sources of phytoestrogens to infants (Franke and Custer, 1996; Knight et al., 1998).

Regulations in Canada

The Natural Health Products Directorate and Health Canada provides scientific guidance on the regulation of soy isoflavone products and labeling of products containing isoflavones (Health Canada, 2009a). For example, the guidelines provide scientific rationale for recommended uses of soy protein and isoflavone products that provide 30-100 mg aglycone isoflavone equivalents (AIE) that include at least 15 mg AIE from genistein when taken for the purpose of reducing menopausal symptoms by menopausal and postmenopausal women when taken for at least 2 weeks. Another example is the recommended use of products containing 75-125 AIE for at least 6 months to reduce post-menopausal bone density when taken in conjunction with calcium and vitamin D. Examples of cautionary statements include warnings about taking more than 30 mg AIE per day without consulting a health professional if they plan on taking it for more than a year, are starting hormone replacement therapy, if they have or have previously had breast cancer or a familial predisposition to breast cancer, if they have a history of gynecological disease, or if they are on blood thinners. Likewise people using products daily that contain more than 10 mg AIE per day are warned to consult a health care provider prior to use if they have liver disorders or are on thyroid hormone replacement therapy. According to guidelines Health Canada does not require labelling on soy proteins and isoflavone products providing doses that are less than 10 mg AIE per day, nor is labelling required that is targeted towards people on blood pressure medication. As well Health Canada does not require labelling targeted to women and vegetarians who consume soy regularly.

Possible health effects

Phytoestrogens do not accumulate in the human body and after being absorbed in the gastrointestinal tract and entering the bloodstream, they are rapidly excreted in urine (Harrisoon and Hester, 1999). Some research suggests that phytoestrogens may have various health benefits, such as protection against breast and colorectal cancer, cardiovascular disease, osteoporosis and menopausal symptoms (Cotterchio et al., 2006; Duffy et al., 2007).

Due to the similar structure to estrogen, much research has been done with varying outcomes to clarify the ability of phytoestrogens to cause endocrine disruption (Atkinson et al., 2005). While the estrogenic properties of phytoestrogens and their metabolites are much less potent than endogenous estrogenic compounds, they may be present in concentrations considerably higher than that of endogenous compounds. Maternal exposure to phytoestrogens via diet in animals supports the hypothesis that high doses of phytoestrogens may alter the hormonal environment of the fetus

(CDC; 2013j). Unfortunately, the effects of phytoestrogens on human development is still inconclusive, and definitive conclusions have not been reached as to the possible adverse health effects occurring from phytoestrogen exposure due to contradictory study results. In addition to endocrine disrupting properties, phytoestrogens have been linked to adverse health effects on the immune system and thyroid functioning, in addition to genotoxic effects (CDC, 2013q, Schmitt et al., 2003).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Two isoflavones (daidzein and genistein) were measured in blood serum samples of pregnant northern Saskatchewan women. Concentrations of daidzein ranged from 0.940 ng/mL to 2.03 ng/mL (weighted arithmetic mean ± 95% confidence interval: 1.41 ± 0.326 ng/mL), while genistein ranged from 3.00 ng/mL to 5.30 ng/mL (weighted arithmetic mean ± 95% confidence interval: 4.30 ng/mL ± 0.770 ng/mL). Pool 6 (far N) had the lowest blood serum concentration of both daidzein and genistein whereas pool 2 (NW) had the highest. Concentrations of genistein are higher than daidzein in the blood serum of pregnant northern Saskatchewan women included in this study. Similarly, a study of Japanese women whose blood was collected at the time of giving birth via caeasarn section found that concentrations of genistein in the maternal blood samples was higher than concentrations of daidzein with median (range) of 4.9 (nondetect-59.2) ng/mL and 1.1 (nondetect-16.2) ng/mL, respectively.

The U.S. National Health and Nutrition Examination Survey (NHANES, 2003-2004) (CDC, 2009) reports urinary creatinine corrected daidzein and genistein concentrations. The results cannot be compared due to differences in biological matrix. Alberta did not test for genistein in their first phase of biomonitoring (AHW, 2008). However, mean concentrations of daidzein measured in Saskatchewan overlaps with the 18-25 year old age group in Northern and Southern AB (1.59 ± 0.333 and 1.41 ± 0.197 ng/mL, respectively). Other regions and age groups in Alberta were above the range found in Saskatchewan When looking at concentrations of daidzein between northern Saskatchewan and Alberta, the results are comparable.

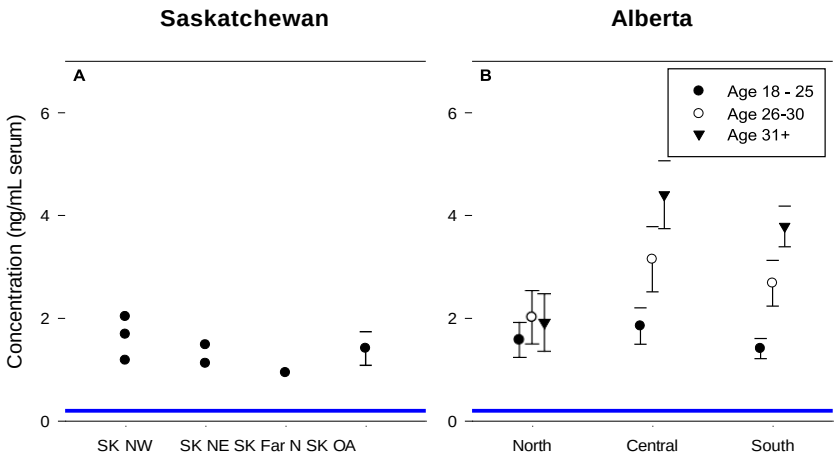

Figure 5: Concentrations of daidzein in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data presents the concentrations of the 6 pooled samples that were analyzed, as well as an overall (OA) mean concentration of the 6 pools. Mean concentrations of each age group are given for the 3 regions of Alberta. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

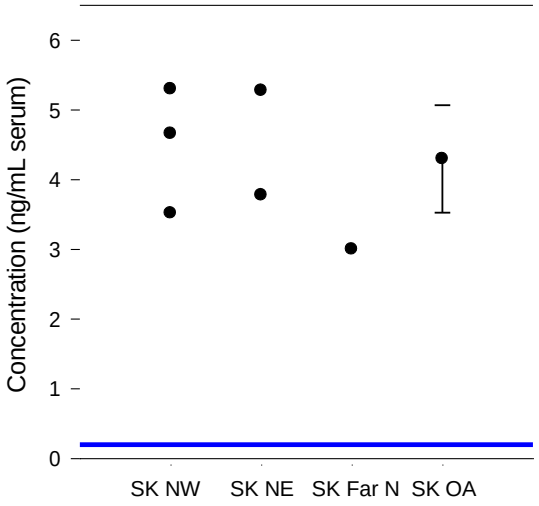

Figure 6: Concentrations of genistein in the blood serum of pregnant women in Saskatchewan. Data points represent the concentrations of the 6 pooled samples that were analyzed, along with an overall (OA) mean concentration of the 6 pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

DIOXINS AND FURANS

GENERAL INFORMATION

Sources

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), more commonly known as dioxins and furans are chemicals that have no real commercial uses and are produced unintentionally during a variety of industrial activities such as combustion, metal recycling, pulp and paper bleaching, and the manufacturing of certain chemicals such as pesticides (Environment Canada, 2013g; Vanden Heuval and Lucier, 1993). Additionally, dioxins have been found in automobile exhaust, as well as in smoke from tobacco, wood and coal burning; whereas furans are also found in commercial mixtures of PCBs. There are 75 PCDD isomers and 135 PCDF isomers, which are distinguished by the number and pattern of chlorine atoms on these molecules, resulting in widely varying toxic potency (Van den Berg et al. 1998; Van den Berg et al. 2006). Dioxins and furans can also be produced naturally, such as during forest fires and volcanic eruptions. Compounds emitted from combustion sources exist in the atmosphere primarily bound to particulates (Lippmann, 1992). After their release to the environment, dioxin and furan particulates can migrate long distances prior to deposition, resulting in a large number of exposure sources across a wide geographic area. Due to their hydrophobicity, dioxins and furans bind strongly to organic material in the environment and is not readily soluble in water (Vanden Heuval and Lucier, 1993). Dioxins and furans found in the soil either from deposition of atmospheric sources or from the application of pesticides, tend to persist for many years and adsorb strongly to soil particles and thus are not easily transported into the groundwater unless solubilized by the addition of other chemicals to the soil which may facilitate mobilization of dioxins (ATSDR, 1998; ATSDR, 1994). Both furans and dioxins may be taken up into plants via the roots and concentrated in animals such as cattle.

The predominant source of dioxin and furan exposure in the general human population is through diet or occupational exposures. The number of chlorine atoms and their positions on the molecule determine the chemical properties of dioxins and furans. This will also dictate which congeners are retained by humans and animals within fatty tissues. Combined with their lipophilic nature (literally ‘fat-loving’), they accumulate in fatty tissues of fish and animals after deposition in soil or water. Food intake, particularly food sources of animal origins, represents the most likely sources of human exposure to these compounds, with food accounting for as much as 90% of the daily intake (Vanden Heuval and Lucier, 1993; ATSDR 1998; ATSDR 1994). Another source of potential exposure is via contamination of food packaging materials. Concentrations in vegetation are generally very low and result from particulate deposition rather than soil uptake.

Due to their lipophilicity, dioxins and furans accumulate in the fatty tissues of our bodies and can be excreted in breast milk (Furest et al., 1989; Schecter et al., 1990). Infants may be exposed to higher levels due to their higher consumption of breast milk, cow’s milk and infant formula, all of

which have been found to contain measurable levels of dioxins and furans; as well, dioxins are able to cross the placenta suggesting that infants are also exposed in utero (ATSDR, 1998). However, breastfeeding is encouraged due to the many associated health-benefits that currently outweigh known risks (American Academy of Pediatrics, 1997, AHW, 2008).

Regulations in Canada

Dioxins and furans are designated to be virtually eliminated in Canada under the *Canadian Environmental Protection Act,* 1999, the federal Toxic Substances Management Policy, and the CCME Policy for Management of Toxic Substances (CCME, 1999). Since July 1st, 1992, the releases of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and 2,3,7,8- tetrachlorodibenzofuran (TCDF) have been prohibited in pulp and paper mill effluent in Canada resulting in non-detectable levels of this congeners by 1995 (Government of Canada, 2015; CDC, 2005). Furthermore, Polychlorinated dibenzofurans and polychlorinated dibenzodioxins are included on Priority Substance List 1 under Priority Substances Assessment Program (Environment Canada, 1989). Canada has also joined international campaigns to regulate dioxin and furan use, and have signed the United Nations Economic Commission for Europe's (UNECE) protocol (Dec 1998) and the Stockholm Convention on persistent organic pollutants (POPs) (May 2001), respectively, to protect human health and the environment from chemicals such as dioxins and furans (Government of Canada, 2015). Emissions and releases, except for residential wood combustion, are covered under the CCME Canada-wide Standards (CCME, 1999).

Possible Health Effects

Dioxin and furan exposure can cause adverse health effects in humans, and as with any chemical exposure the magnitude of the effects will depend on dose, route of exposure, duration of exposure, and timing of exposure (AHW, 2008). The evidence of a link between cancer incidence or mortality and exposure of human populations to dioxins and furans is equivocal with some classified as Group 3 (not classifiable as carcinogens in humans) through IARC though 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is classified as Group 1 (carcinogen in humans) (International Agency for Research on Cancer. http://monographs.iarc.fr/ENG/Classification/ - accessed online April 16, 2015). It is difficult to assess the effects that dioxins and furans may have on human health because of concomitant exposures to other chemicals and imprecise exposure information (Health Canada, 2006). Acute, higher dose exposures to 2,3,7,8-TCDD has resulted in chloracne in humans, which are acne-like skin lesions, as well as skin discoloration and rashes, excessive body hair and changes to the metabolic functioning of the liver (ATSDR; 1998). Similar health effects have been observed in accidental poisonings with furans, with the addition of vomiting, diarrhea, anemia, numbness and other nervous system effects (ATSDR, 1994).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

The following polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were measured in the blood serum samples of pregnant women:

1. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
2. 1,2,3,7,8-pentachlorodibenzo-p-dioxin (PCDD)
3. 1,2,3,4,7,8- hexachlorodibenzo-p-dioxin (HxCDD)
4. 1,2.3,6,7,8- hexachlorodibenzo-p-dioxin (HxCDD)
5. 1,2,3,7,8,9- hexachlorodibenzo-p-dioxin (HxCDD)
6. 1,2,3,4,6,7,8- heptachlorodibenzo-p-dioxin (HpCDD)
7. 1,2,3,4,6,7,8,9- octachlorodibenzo-p-dioxin (OCDD)
8. 2,3,7,8-tetrachlorodibenzofuran (TCDF)
9. 1,2,3,7,8-pentachlorodibenzofuran (PCDF)
10. 2,3,4,7,8-pentachlorodibenzofuran (PCDF)
11. 1,2,3,4,7,8- hexachlorodibenzofuran (HxCDF)
12. 1,2,3,6,7,8- hexachlorodibenzofuran (HxCDF)
13. 1,2,3,7,8,9- hexachlorodibenzofuran (HxCDF)
14. 2,3,4,6,7,8- hexachlorodibenzofuran (HxCDF)
15. 1,2,3,4,6,7,8- heptachlorodibenzofuran (HpCDF)
16. 1,2,3,4,7,8,9- heptachlorodibenzofuran (HpCDF)
17. 1,2,3,4,6,7,8,9- octachlorodibenzofuran (OCDF)

Dioxins and furans are lipophilic chemicals and therefore are found primarily within the lipid portion of the plasma or serum. As such their concentrations in the blood serum are typically made in reference to the lipid weight of the serum sample. This is done by dividing the total concentration of a lipophilic chemical in serum by the percent lipid content of blood. Comparison of results of chemical concentrations measured in difference matrixes (that is, lipid adjusted blood serum vs blood serum) cannot be directly done. In this report, both the measured serum chemical concentration and the calculated lipid level concentration, will be provided for reference and ease of comparison to other biomonitoring studies. The following congeners were detected, and concentrations are shown based on both serum weight and lipid weight:

Table 14: Concentrations detected by pool for dioxins and furans analyzed in pregnant women in northern Saskatchewan.

PCDDs and PCDFs	pg/g	Pool 1	Pool 2	Pool 3	Pool 4	Pool 5	Pool 6
1,2,3,6,7,8 - HxCDD	Serum	0.030	<LOD	0.050	0.040	<LOD	<LOD
	lipid	5.9	<LOD	8.9	7.5	<LOD	<LOD
1,2,3,4,6,7,8 - HpCDD	Serum	0.060	0.080	0.050	0.070	0.060	0.070
	lipid	12	16	8.9	13	12	13
OCDD	Serum	0.46	0.54	0.59	0.56	0.54	0.54
	lipid	90	1.1×10^2	1.1×10^2	1.1×10^2	1.1×10^2	1.0×10^2
1,2,3,4,7,8 - HxCDF	Serum	<LOD	<LOD	<LOD	0.020	<LOD	<LOD
	lipid	<LOD	<LOD	<LOD	3.8	<LOD	<LOD
1,2,3,6,7,8- HxCDF	Serum	<LOD	<LOD	<LOD	0.020	<LOD	<LOD
	lipid	<LOD	<LOD	<LOD	3.8	<LOD	<LOD
1,2,3,4,6,7,8 - HpCDF	Serum	<LOD	0.020	<LOD	0.030	0.040	0.030
	lipid	<LOD	4.1	<LOD	5.7	8.2	5.6

All of the detected congers were detected at levels above the LOD in pool 4 (SK NE); concentrations of 1,2,3,4,7,8-HxCDF and 1,2,3,4,6,7,8-HpCDF were not detected in pools 1-2 (NW) and 5-6 (NE, far N). While only three congeners were detected in pool 2, the detected concentrations were the highest among the pools for two of the three congeners (OCDD and 1,2,3,4,6,7,8-HpCDD).

While 6 dioxin and furan congeners were detected to some degree in the 6 pools, only two compounds met the inclusion criteria of reporting for this study: OCDD and 1,2,3,4,6,7,8-HpCDD, as they had ≤ 1 pools below the LOD. OCDD had a lipid adjusted, weighted mean arithmetic concentration (± 95% confidence interval) of 1.0×10^2 pg/g lipid ± 6.4 pg/g lipid and 1,2,3,4,6,7,8 – HpCDD had a lipid adjusted, and weighted aritmetic mean concentration of 13 pg/g lipid ± 1.9 pg/g lipid. Compared to the Alberta phase one (AHW, 2008) results, the concentrations detected in pregnant women from northern Saskatchewan are slightly lower. The overall mean lipid concentration of OCDD in Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.0×10^2 ± 6.4 pg/g) is comparable to 18 to 25 year old women in northern (mean ± 95% confidence interval: 1.2×10^2 ± 24 pg/g lipid) and central Alberta (mean ± 95% confidence interval: 1.0×10^2 ± 23 pg/g lipid), as well as 31+ year old women in northern Alberta (mean ± 95% confidence interval: 1.4×10^2 ± 44 pg/g). The other regions and age groups in Alberta had mean serum lipid concentrations of OCCD higher than that found in Saskatchewan. The overall mean lipid concentration in Saskatchewan of 1,2,3,4,6,7,8-HpCDD (weighted arithmetic mean ± 95% confidence interval: 13 ± 1.9 pg/g lipid) is comparable to women aged 18-25 years in northern AB (mean ± 95% confidence

interval: 17 ± 4.1 pg/g lipid) and central AB (mean ± 95% confidence interval: 16 ± 3.2 pg/g lipid). The other regions and age groups have mean concentrations higher than what was detected in this study in pregnant women from northern Saskatchewan.

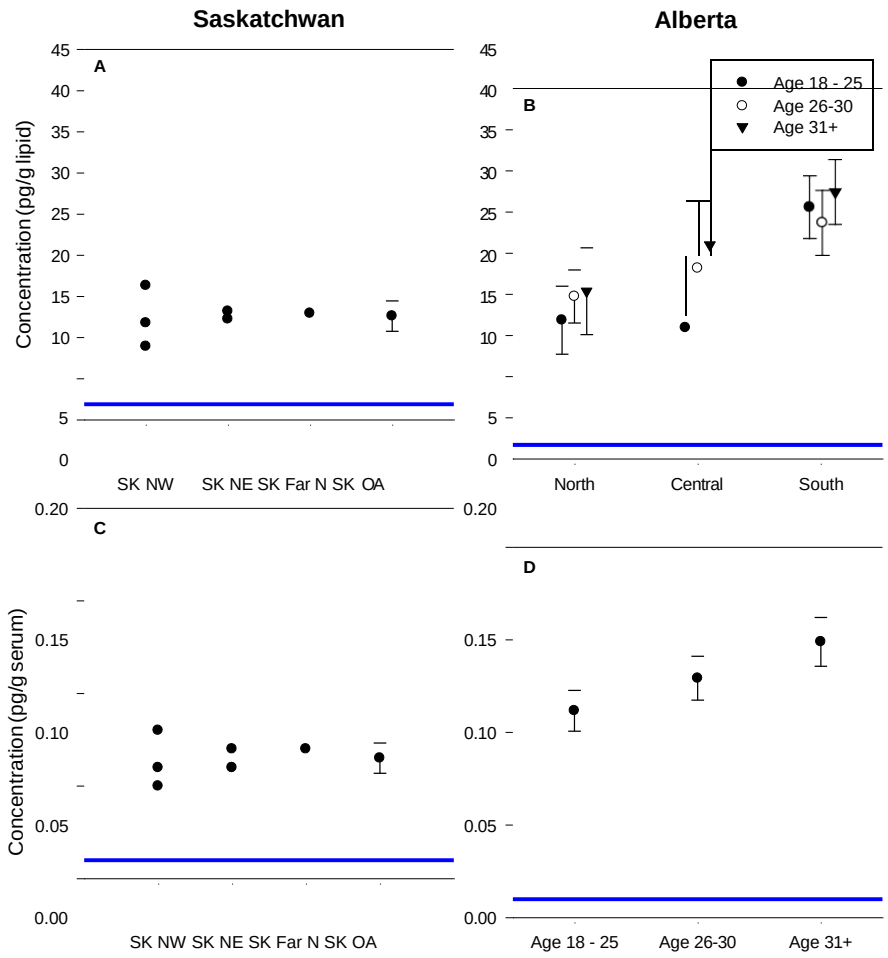

Figure 7: Concentrations of 1,2,3,4,6,7,8-HpCDD in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). The Saskatchewan data presents the concentrations of the 6 pooled samples that were analyzed, in addition to an overall (OA) mean concentration of the 6 pools. The lipid Alberta data is presented as mean concentrations of the three regions of Alberta, whereas serum concentrations are presented as mean concentrations of the three age groups. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

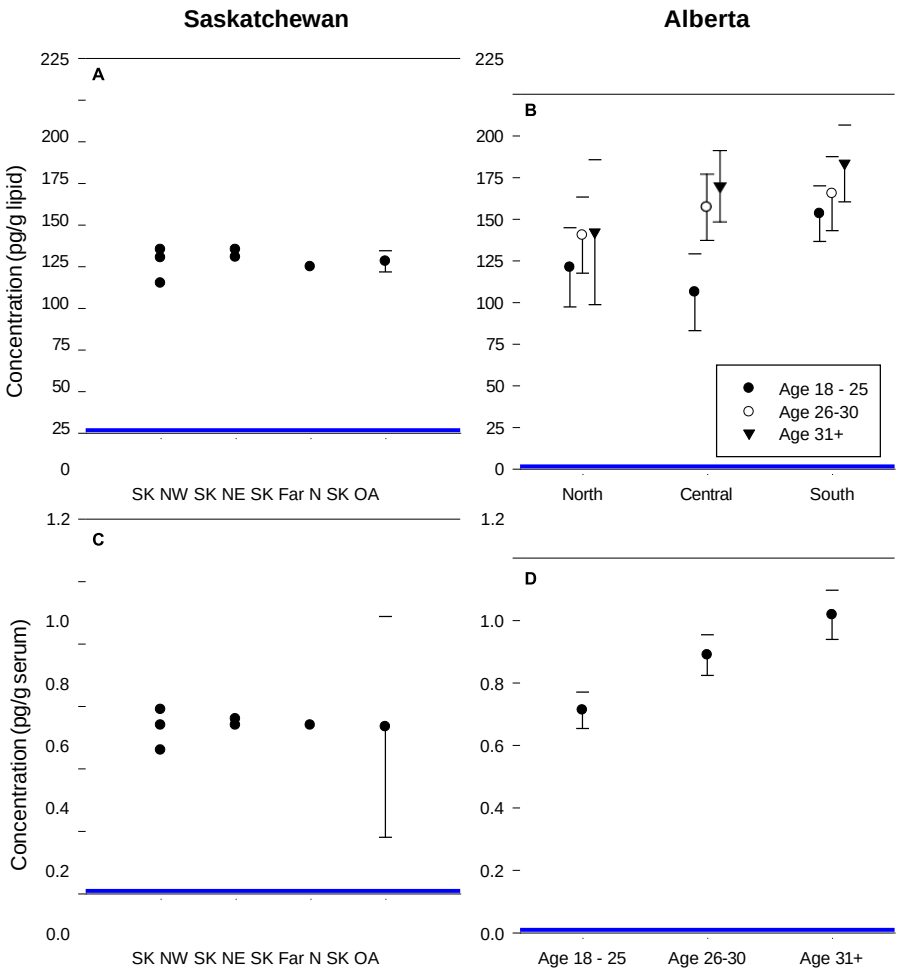

Figure 8: Concentrations of OCDD in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data presents the concentrations of the 6 pooled samples that were analyzed, in addition to an overall (OA) mean concentration of the 6 pools. The lipid Alberta data is presented as mean concentrations of the three regions of Alberta, whereas serum concentrations were presented as mean concentrations of the three age groups. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

POLYCHLORINATED BIPHENYLS (PCBS)

GENERAL INFORMATION

Sources

Polychlorinated biphenyls (PCBs) is the name given to refer to a group of 208 congeners that were first manufactured in 1929 (Environment Canada, 2013g; U.S EPA Feb 2014; Health Canada, 2005). Commercial mixtures of PCBs are also known under several trade names, including Aroclor, Phenolclor, Pyranol and others (ATSDR, 2000a). They were used for many decades as dielectric fluids in transformers and capacitors, in heat-exchange systems, as lubricants, plasticizers, and adhesives, as well as an additive in sealants, plastics, paint, fire retardants, hydraulic oil, pesticide extenders (Environment Canada, 2013g; U.S EPA, 2014; Health Canada, 2005). PCBs are released into the environment as an unintentional emission from combustion, and are extremely persistent compounds as they are resistant to breakdown. They are predominately sorbed onto the soil or accumulated in biota while surface waterways are a major environmental reservoir (Lippmann, 1992). PCBs can undergo long-range global transport via air and water, and so are detectable at trace levels all over the world, including remote regions (Health Canada, 2005). Chlorination of a PCB compound may affect its environmental fate as processes such as partitioning into soil and sediment, photolysis, volatiziation, chemical and biological transformation (biodedgradation) and preferential accumulation are all affected by degree of chlorination (ATSDR, 2000a).

Due to their lipophilic nature, PCBs are able to bioaccumulate in biological organisms and biomagnify up the food chain; however, PCBs congeners that are more highly chlorinated and are substituted in the meta- and para- positions are more prone to accumulation in bodily tissues (ATSDR, 2000a; Health Canada, 2005). Humans are most commonly exposed to PCBs via ingestion of contaminated food, and those individuals who ingest more fish and other marine life tend to have higher exposures. Since PCBs are environmentally persistent at low concentrations in oceans, freshwater bodies, and most pasture and agricultural soils around the world, its main dietary sources include animal fats from fish, meat and dairy products (ATSDR, 2000a). PCBs are lipophilic and accumulate in fatty tissues of animals, and in human breast milk (Jorissen, 2007), with human milk representing a major source of PCBs to infants, in addition to PCB exposure in utero due to the ability of PCBs to cross the placenta (Jensen and Slovach, 1991; Jorissen, 2007). However, breastfeeding is encouraged due to the many associated health-benefits (American Academy of Pediatrics, 1997) that currently outweigh known risks. PCB exposure may also occur when people come into contact with PCB containing materials such as in older buildings that were constructed with PCB laden materials, or where old electrical devices and PCB containing transformers may be found.

Regulations in Canada

The presence of PCBs was detected in the Great Lakes for the first time in 1966 and by 1977, a North American ban on the manufacture and import of PCBs was in place (Health Canada, 2005). While PCBs were never manufactured in Canada, they were widely used here. PCBs are managed under Track I of the Government of Canada's Toxic Substances Management Policy (Canada Gazette, 2006a) to minimize exposure and environmental releases of PCBs. They are on the Export Control List in Part 2, Schedule 3 of the *Canadian Environmental Protection Act,* 1999 as "substances subject to notification and consent", and Environment Canada maintains an inventory of PCB use and stored PCB waste within Canada (Environment Canada, 2008). The International Agency for Research on Cancer classified PCBs as a Group 2A probable human carcinogen (ATSDR, 2000a).

In November 2006, the Canadian Federal Government set a specific deadline of December 2009 for ending the use and storage of equipment and other materials containing PCBs in concentrations at or above 50 mg/kg (Canada Gazette, 2006a). The continued use of certain equipment containing PCBs is still allowed in Canada, but according to the Stockholm Convention on Persistent Organic Pollutants, Canada is required to phase out the remaining uses of PCBs by 2025, and to dispose of these PCBs properly by 2028 (Canada Gazette, 2006a).

Possible Health Effects

Similar to other chemical exposures, the human health effects of PCBs depend on many factors including dose, duration and timing of exposure (AHW, 2008). Like dioxins and furans, human populations are exposed to very low concentrations of PCBs via the diet and environment. The retention of PCBs within the body depends on the species being exposed, the organ, the degree of chlorination and the chemical substitution pattern (ATSDR, 2000a). Likewise metabolism of a PCB congener may be affected by both the degree of chlorination and the pattern of substitution on the phenyl rings. Chronic exposure to PCBs could lead to health effects such as skin conditions, immune deficiencies, as well as disruption to the reproductive and nervous systems (U.S EPA, 2014a). Skin conditions as well as irritations of the respiratory system are more common in those individuals who are occupationally exposed (ATSDR, Nov 2000). There is also evidence to suggest that PCB exposure interferes with endocrinological regulation of thyroid hormones, as well being shown to have estrogenic, anti-estrogenic, androgenic and anti-androgenic activity (ATSDR, 2000a; Ulbrich and Stahlman, 2004). IARC has classified working group PCBs, as well as dioxin-like PCBs as group 1 carcinogens, that is to say that they are human carcinogens (Lauby-Secretan et al., 2013). In cases of high doses health effects include chloracne, pigmentation of the skin and nails, swelling of limbs, jaundice, and neurological effects.

Human studies show evidence that intrauterine exposure to PCBs may lead to decreased birth weight in infants as well as behavioural abnormalities in the form of decreased short term memory and difficulties with motor skills (ATSDR, 2000a; Carpenter, 2006; Jacobson and Jacobson, 2001;

Jacobson and Jacobson, 1996). The most commonly detected PCBs in human tissues are PCB 138, 153 and 180 (ATSDR, 2000a). A study conducted by Dallaire et al. (2014) found that chronic PCB exposure during childhood, particularly PCB 153, is negatively associated with skeletal growth and weight.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

The following polychlorinated biphenyls (PCBs) were selected for measurement in the blood serum samples of pregnant women in northern Saskatchewan:

PCB 2	PCB 48/49	PCB 85	PCB 136	PCB 175/182
PCB 1	PCB 55	PCB 88/121	PCB 144	PCB 176
PCB 3	PCB 60	PCB 92	PCB 148	PCB 177
PCB 4/10	PCB 61	PCB 95	PCB 151	PCB 178
PCB 15	PCB 73	PCB 96	PCB 152	PCB 179
PCB 6	PCB 58/67	PCB 103	PCB 153/168	PCB 183
PCB 8	PCB 78	PCB 105	PCB 159	PCB 190
PCB 9	PCB 81	PCB 106	PCB 161	PCB 191
PCB 11	PCB 41	PCB 113	PCB 167	PCB 181
PCB 14	PCB 45	PCB 120	PCB 128/162	PCB 184
PCB 7	PCB 50	PCB 122	PCB 132	PCB 186
PCB 5	PCB 57	PCB 127	PCB 137	PCB 187
PCB 12	PCB 63/76	PCB 94	PCB 139/143	PCB 192
PCB 13	PCB 66	PCB 99	PCB 145	PCB 174
PCB 16	PCB 72	PCB 100	PCB 150	PCB 193
PCB 19	PCB 79	PCB 108/86/125	PCB 156	PCB 180
PCB 37	PCB 46	PCB 111/117	PCB 158/129	PCB 188
PCB 26	PCB 59/42	PCB 114	PCB 160/163	PCB 189
PCB 27	PCB 80	PCB 118	PCB 165	PCB 173
PCB 30	PCB 64	PCB 84/89	PCB 141	PCB 185
PCB 31	PCB 69	PCB 93	PCB 146	PCB 194
PCB 32	PCB 43/52	PCB 112	PCB 147/149	PCB 195
PCB 34	PCB 44	PCB 116	PCB 154	PCB 200
PCB 35	PCB 54	PCB 102	PCB 138	PCB 201/204
PCB 36	PCB 56	PCB 97	PCB 155	PCB 197
PCB 38	PCB 77	PCB 124	PCB 169	PCB 199
PCB 22	PCB 70	PCB 87	PCB 131/142/133	PCB 203/196
PCB 23	PCB 51	PCB 98	PCB 134	PCB 202
PCB 24	PCB 53	PCB 104	PCB 157	PCB 205
PCB 28	PCB 71	PCB 110	PCB 140	PCB 198
PCB 29	PCB 74	PCB 123/107/109	PCB 164	PCB 206

PCB 18	PCB 75/65/62	PCB 90/101	PCB 166	PCB 207
PCB 29	PCB 47	PCB 91	PCB 135	PCB 208
PCB 18	PCB 40/68	PCB 115	PCB 170	PCB 209
PCB 21/20/33	PCB 82	PCB 126	PCB 171	
PCB 25	PCB 83/119	PCB 130	PCB 172	

Because PCBs are lipophilic chemicals, they are found primarily within the lipid portion of the plasma or serum and as such their concentrations in the blood serum are typically made in reference to the lipid weight of the serum sample. This is done by dividing the total concentration of a lipophilic chemical in serum by the percent lipid content of blood. Comparison of results of chemical concentrations measured in difference matrixes (that is, lipid adjusted blood serum vs blood serum) cannot be directly done. In this report, both the measured serum chemical concentration and the calculated lipid level concentration, will be provided for reference and ease of comparison to other biomonitoring studies. The following congeners were detected, and ranges of concentrations found in the 6 pools are shown below based on both wet weight (pg/g serum) and lipid adjusted weight (ng/g lipid):

Table 15: Ranges of serum concentrations and lipid adjusted concentrations of PCB congeners tested in the 6 pools of pregnant women from northern Saskatchewan.

Chemical	Wet weight (pg/g serum)	Lipid weight (ng/g lipid)
PCB 2	9.6 – 21	1.9 – 4.3
PCB 1	$2.2 \times 10^2 – 5.2 \times 10^2$	$46 – 1.0 \times 10^2$
PCB 3	40 – 87	8.1 – 17
PCB 4/10	$4.7 \times 10^2 – 1.3 \times 10^3$	$88 – 2.5 \times 10^2$
PCB 15	32 – 90	6.5 – 18
PCB 6	$70 – 2.0 \times 10^2$	14 – 39
PCB 8	$3.0 \times 10^2 – 8.4 \times 10^2$	$60 – 1.6 \times 10^2$
PCB 9	33 – 91	6.8 – 18
PCB 11	55 – 91	9.8 – 18
PCB 7	20 – 55	3.9 – 11
PCB 5	8.2 – 21	1.6 – 4.1
PCB 13	<LOD – 9.3	<LOD – 26
PCB 16	$54 – 1.3 \times 10^2$	11 – 26
PCB 19	32 – 93	6.0 – 18
PCB 37	12 – 24	1.8 – 6.0
PCB 26	12 – 29	2.4 – 5.7
PCB 27	9.0 – 21	1.7 – 4.0
PCB 31	$62 – 1.5 \times 10^2$	13 – 30
PCB 32	31 – 77	6.3 – 15
PCB 22	20 – 52	4.1 – 10
PCB 24	2.6 – 6.6	0.53 – 1.3
PCB 28	$66 – 1.8 \times 10^2$	13 – 35
PCB 17	$67 – 1.8 \times 10^2$	13 – 36
PCB 29	<LOD – 2.5	<LOD – 0.48

Chemical	Wet weight (pg/g serum)	Lipid weight (ng/g lipid)
PCB 18	$1.8 \times 10^2 – 4.9 \times 10^2$	37 – 96
PCB 21/20/33	$43 – 1.1 \times 10^2$	8.8 – 22
PCB 25	5.9 – 15	1.2 – 2.9
PCB 48/49	30 – 66	6.2 – 13
PCB 60	2.3 – 7.4	0.43 – 1.5
PCB 41	4.0 – 6.8	0.74 – 1.3
PCB 45	7.8 – 18	1.6 – 3.5
PCB 63/76	<LOD – 1.4	<LOD – 0.27
PCB 66	14 – 27	2.5 – 5.6
PCB 46	2.7 – 6.0	0.51 – 1.2
PCB 59/42	8.8 - 17	1.6 – 3.3
PCB 64	13 – 25	2.7 – 4.8
PCB 43/52	$65 – 1.2 \times 10^2$	12 – 23
PCB 44	32 – 58	5.9 – 11
PCB 56	3.5 – 58	0.65 – 2.1
PCB 77	0.89 – 2.5	0.17 – 0.50
PCB 70	14 – 54	2.7 – 11
PCB 51	3.9 – 5.5	0.74 – 1.1
PCB 53	8.4 – 16	1.6 – 3.1
PCB 71	6.4 – 10	1.2 – 2.0
PCB 74	8.2 – 21	1.5 – 4.3
PCB 47	13 – 19	2.6 – 3.6
PCB 40/68	1.4 – 3.0	0.25 – 0.59
PCB 85	1.4 – 5.5	0.26 – 1.1
PCB 92	3.7 – 7.9	0.68 – 1.6
PCB 95	23 – 46	4.2 – 8.9
PCB 105	3.4 – 5.6	0.63 – 1.1
PCB 99	10 – 19	1.8 – 3.7
PCB 111/117	<LOD – 1.3	<LOD – 0.25
PCB 118	13 - 22	2.4 – 4.5
PCB 84/89	6.5 – 11	0.20 – 2.2
PCB 97	4.5 – 9.4	0.82 – 1.9
PCB 87	5.9 – 16	1.1 – 3.2
PCB 110	13 – 32	2.3 – 6.6
PCB 123/107	<LOD – 1.9	<LOD – 0.35
PCB 90/101	18 – 47	3.4 – 9.2
PCB 91	3.6 – 7.4	0.67 – 1.4
PCB 115	<LOD – 1.4	<LOD – 0.29
PCB 130	<LOD – 0.72	<LOD – 0.13
PCB 136	0.93 – 4.2	0.17 – 0.82
PCB 144	<LOD – 1.5	<LOD – 0.28
PCB 151	<LOD – 4.7	<LOD – 0.89
PCB 153/168	16 – 36	2.9 – 6.8
PCB 167	<LOD – 1.5	<LOD – 0.28

Chemical	Wet weight (pg/g serum)	Lipid weight (ng/g lipid)
PCB 128/162	<LOD – 1.0	<LOD – 0.21
PCB 132	<LOD – 4.0	<LOD – 0.81
PCB 137	<LOD – 2.2	<LOD – 0.42
PCB 156	1.3 – 4.1	0.22 – 0.78
PCB 158/129	<LOD – 2.7	<LOD – 0.51
PCB 160/163	2.9 – 7.6	0.52 – 1.4
PCB 141	<LOD – 2.7	<LOD – 0.55
PCB 146	1.3 – 5.7	0.23 – 1.1
PCB 147/149	6.7 – 16	1.2 – 3.2
PCB 138	13 – 26	2.3 – 4.9
PCB 157	<LOD – 1.2	<LOD – 0.22
PCB 135	1.7 – 3.1	0.31 – 0.63
PCB 170	3.2 – 8.5	0.66 – 1.6
PCB 172	<LOD – 2.4	<LOD – 0.48
PCB 177	<LOD – 2.6	<LOD – 0.48
PCB 183	2.2 – 3.8	0.39 – 0.73
PCB 190	<LOD – 1.4	<LOD – 0.26
PCB 187	4.5 – 9.9	0.81 – 2.0
PCB 180	10 – 27	1.8 – 5.1
PCB 194	<LOD - 5.5	<LOD – 1.0
PCB 199	3.1 – 8.7	0.63 – 1.6
PCB 203/196	3.2 -5.5	0.57 – 1.0
PCB 202	1.7 – 5.1	0.39 – 1.0
PCB 206	6.0 – 17	1.2 – 3.2
PCB 207	<LOD – 1.7	<LOD – 0.32
PCB 208	5.2 – 8.2	0.98 – 1.5
PCB 209	12 – 39	2.5 – 3.3

Table 16: Serum and lipid adjusted weighted mean concentrations of all pools from Northern Saskatchewan for PCB congeners with 5 or more pools above the value of the analytical limit of detection.

Congener	Mean ± 95% Confidence Interval	
	Serum adjusted (pg/g)	Lipid adjusted (ng/g lipid)
PCB 2	15 ± 3.6	2.9 ± 0.75
PCB 1	$3.9 \times 10^2 \pm 91$	76 ± 18
PCB 3	65 ± 15	12 ± 3.1
PCB 4/10	$8.5 \times 10^2 \pm 2.6 \times 10^2$	$1.6 \times 10^2 \pm 51$
PCB 15	55 ± 16	11 ± 3.3
PCB 6	$1.3 \times 10^2 \pm 41$	25 ± 7.9
PCB 8	$5.3 \times 10^2 \pm 1.7 \times 10^2$	$1.0 \times 10^2 \pm 33$
PCB 9	60 ± 18	12 ± 3.6
PCB 11	76 ± 11	15 ± 2.6
PCB 7	35 ± 11	6.7 ± 2.1
PCB 5	13 ± 4.0	2.6 ± 0.76

PCB 13	5.3 ± 2.3	1.0 ± 0.46
PCB 16	86 ± 27	17 ± 5.1
PCB 19	60 ± 20	12 ± 3.8
PCB 37	18 ± 7.3	3.4 ± 1.5
PCB 26	19 ± 5.1	3.7 ± 0.99
PCB 27	14 ± 4.1	2.6 ± 0.78
PCB 31	$1.0 \times 10^2 \pm 26$	20 ± 5.0
PCB 32	50 ± 15	9.7 ± 2.9
PCB 22	35 ± 8.6	6.7 ± 1.7
PCB 24	4.6 ± 1.3	0.89 ± 0.25
PCB 28	$1.2 \times 10^2 \pm 31$	23 ± 6.1
PCB 17	$1.2 \times 10^2 \pm 40$	22 ± 7.6
PCB 29	1.5 ± 0.62	0.29 ± 0.12
PCB 18	$3.2 \times 10^2 \pm 1.1 \times 10^2$	61 ± 20
PCB 21/20/33	71 ± 19	14 ± 3.7
PCB 25	9.5 ± 2.5	1.8 ± 0.49
PCB 48/49	43 ± 10	8.4 ± 2.0
PCB 60	4.9 ± 1.5	0.95 ± 0.31
PCB 41	4.9 ± 0.88	0.95 ± 0.18
PCB 45	12 ± 3.3	2.2 ± 0.62
PCB 63/76	0.98 ± 0.34	0.19 ± 0.066
PCB 66	19 ± 6.0	3.6 ± 1.2
PCB 46	3.6 ± 1.1	0.70 ± 0.21
PCB 59/42	11 ± 2.6	2.2 ± 0.52
PCB 64	17 ± 3.8	3.4 ± 0.78
PCB 43/52	81 ± 17	16 ± 3.5
PCB 44	41 ± 7.7	8.0 ± 1.6
PCB 56	7.6 ± 2.0	1.5 ± 0.42
PCB 77	1.5 ± 0.42	0.29 ± 0.091
PCB 70	36 ± 12	7.0 ± 2.4
PCB 51	4.5 ± 0.55	0.87 ± 0.10
PCB 53	11 ± 2.6	2.2 ± 0.49
PCB 71	7.9 ± 1.3	1.5 ± 0.28
PCB 74	15 ± 4.5	3.0 ± 0.93
PCB 47	15 ± 2.0	2.9 ± 0.39
PCB 40/68	1.9 ± 0.53	0.37 ± 0.11
PCB 85	3.5 ± 1.1	0.69 ± 0.24
PCB 92	6.5 ± 1.3	1.3 ± 0.29
PCB 95	34 ± 6.3	6.6 ± 1.4
PCB 105	4.7 ± 0.66	0.91 ± 0.15
PCB 99	15 ± 2.8	2.9 ± 0.62
PCB 118	18 ± 3.2	3.5 ± 0.71
PCB 84/89	9.5 ± 1.3	1.9 ± 0.32
PCB 97	7.5 ± 1.6	1.5 ± 0.34
PCB 87	13 ± 5.4	2.5 ± 0.67
PCB 110	23 ± 5.4	4.6 ± 1.2
PCB 90/101	37 ± 8.7	7.2 ± 1.9

PCB 91	5.5 ± 1.2	1.1 ± 0.26
PCB 136	3.1 ± 0.93	0.60 ± 0.19
PCB 151	3.0 ± 1.2	0.59 ± 0.24
PCB 153/168	27 ± 7.3	5.2 ± 1.4
PCB 132	3.0 ± 1.0	0.59 ± 0.20
PCB 156	2.7 ± 0.81	0.53 ± 0.16
PCB 158/129	1.3 ± 0.67	0.25 ±0.13
PCB 160/163	4.6 ± 1.4	0.89 ± 0.27
PCB 141	1.7 ± 0.74	0.33 ± 0.15
PCB 146	3.6 ± 1.2	0.69 ± 0.24
PCB 147/149	12 ± 2.9	2.3 ± 0.61
PCB 138	18 ± 4.0	3.4 ± 0.78
PCB 135	2.2 ± 0.40	0.43 ± 0.090
PCB 170	5.3 ± 1.6	1.0 ± 0.31
PCB 183	3.0 ± 0.45	0.59 ± 0.10
PCB 187	7.3 ± 2.0	1.4 ± 0.40
PCB 180	18 ± 6.1	3.4 ± 1.2
PCB 199	5.5 ± 1.9	1.1 ± 0.34
PCB 203/196	4.3 ± 0.79	0.83 ± 0.15
PCB 202	2.9 ± 0.95	0.57 ± 0.20
PCB 206	8.5 ± 3.5	1.6 ± 0.62
PCB 208	5.8 ± 0.94	1.1 ± 0.17
PCB 209	19 ± 7.8	3.7 ± 1.4

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Out of the 178 PCB congeners included for chemical analysis, 81 met the inclusion criteria for reporting and had ≤ 1 pool below the analytical limit of detection. Overall mean concentrations of all tested PCB congeners ranged from <LOD to 2.5 x 10^2 ng/g lipid. Mean serum concentrations and lipid adjusted serum concentrations and standard errors for all the PCB congeners with 5 or more pools detected above the limit of detection are provided in Table 16. Of the 8 PCB congeners that were analyzed for and detected in both Saskatchewan and Alberta (PCB 156, 158/129, 146, 170, 183, 187, 180, 199), 7 had overlapping confidence intervals suggesting comparable exposures between the provinces. Only concentrations of PCB 158/129 were found to be lower in pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 0.25 ± 0.13 ng/g lipid) than in pregnant women sampled in Alberta (mean ± 95% confidence interval: 0.62 ± 0.13 ng/g lipid).

Three major population studies in North America have also investigated lipid adjusted concentrations in the blood of women: CDC's Fourth National Report on Human Exposure to Environmental Chemicals (CDC, 2009), the First Nations Biomonitoring Initiative (AFN, 2013) and the Canadian Health Measures Survey (Health Canada, 2010a). Detailed comparisons of concentrations

of congeners that were included for analysis are available in Appendix E Table 1. CHMS and FNBI measure concentrations of PCBs in μg/kg lipid, whereas this study and the Fourth Report use ng/g lipid. While these units are different, they are all equivalent to parts per billion (ppb).

Lipid adjusted concentrations of PCB congers 170, 187, and 180 in Saskatchewan were lower than levels measured in the Canadian Health Measures Act cycle 1 (Health Canada, 2010a), First Nations Biomonitoring Initiative (AFN, 2013), and the CDC's Fourth National Report (CDC, 2009) (exact values are available in Table 1 of Appendix E). Concentrations of PCB 146 measured in pregnant women in Saskatchewan were found to be lower than both the concentrations measured by the CDC and CHMS. Whereas lipid adjusted concentrations of PCB congeners 28 and 66 measured in the serum of pregnant women included in this study were higher than levels measured in the First Nations Biomonitoring Initiative (AFN, 2013), and the CDC's Fourth National Report (CDC, 2013) and the Canadian Health Measures Survey (Health Canada, 2010a). While there are differences between concentrations of individual congeners in the various studies, concentrations of congeners that were detected in this study and these 3 major population studies are largely comparable. For example, while PCB 66 was detected at a higher in pregnant woman sampled from northern Saskatchewan than in the other studies, the differences in concentration are not large enough to warrant concern. The concentration measured in pregnant women from northern Saskatchewan was 3.6 ± 1.2 ng/g lipid, whereas the concentration reported in the CDC was 1.50 ng/g lipid (95% CI: 1.42-1.58 ng/g lipid). Concentrations in both the Canadian Health Measures Survey and First Nations Biomonitoring Inititiave were found to be below the LOD (0.03 μg/L for both studies).

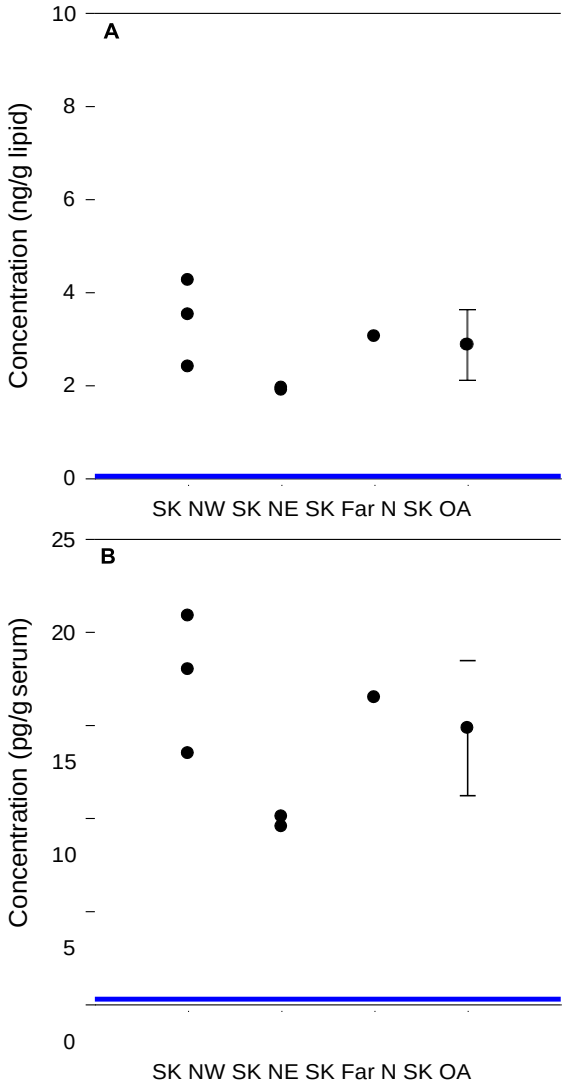

Figure 9: Concentrations of PCB congener 2 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

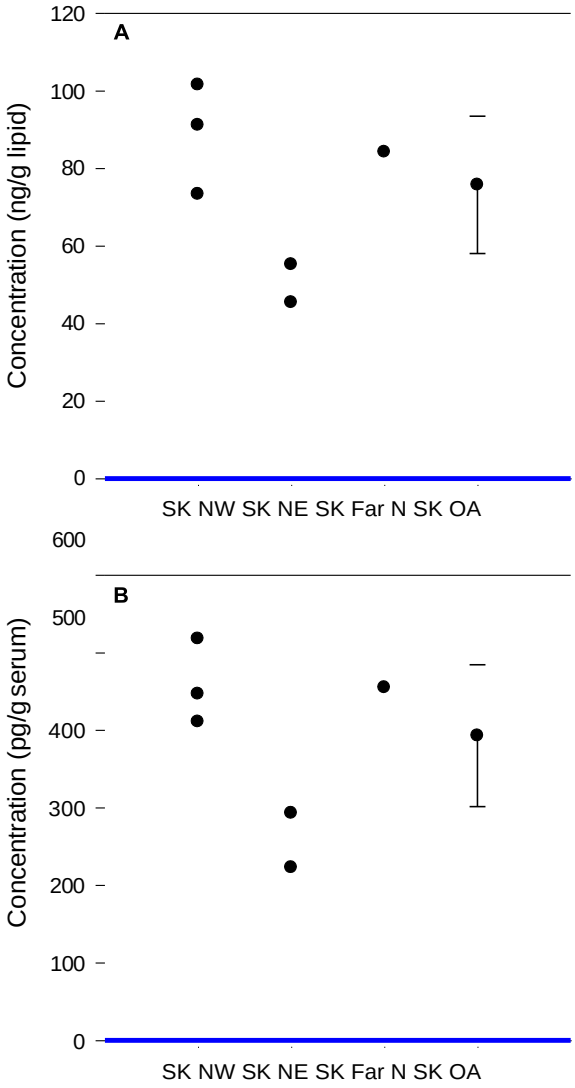

Figure 10: Concentrations of PCB congener 1 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

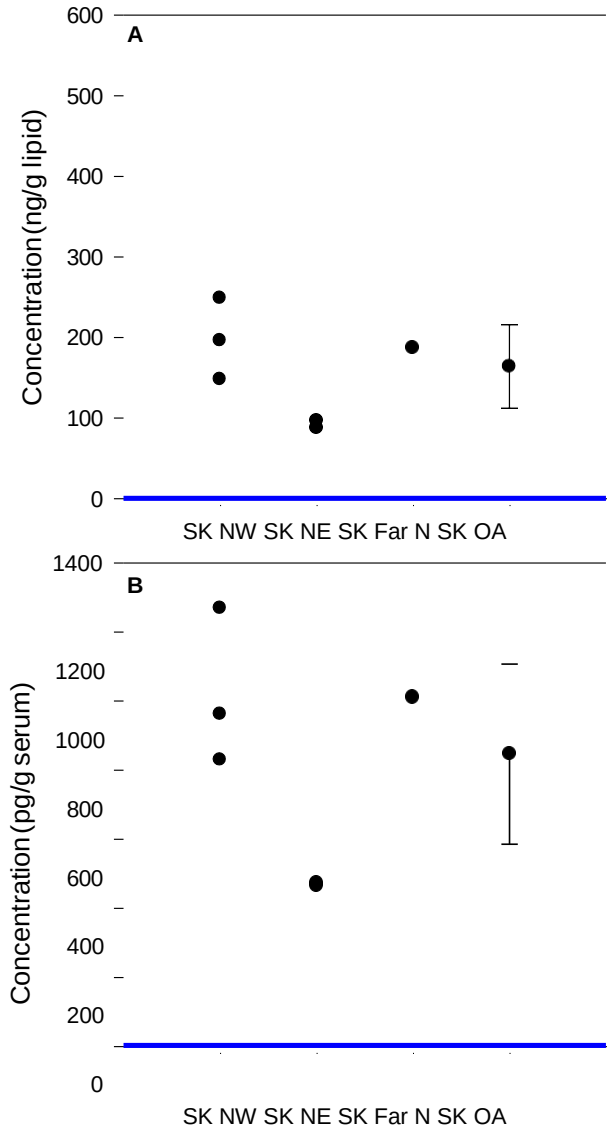

Figure 11: Concentrations of PCB congener 4/10 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

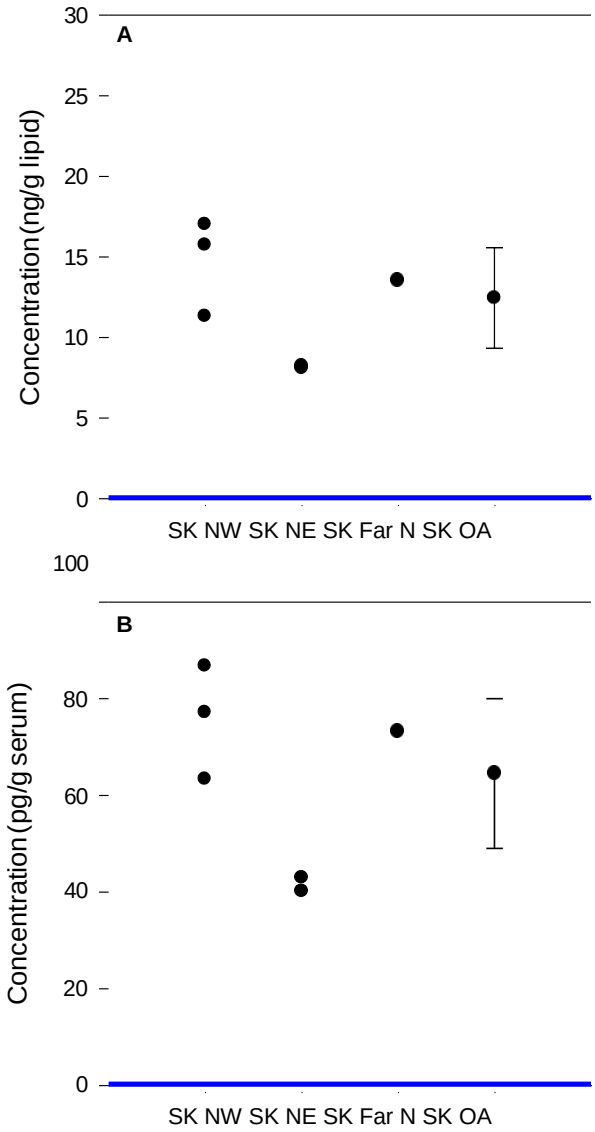

Figure 12: Concentrations of PCB congener 3 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

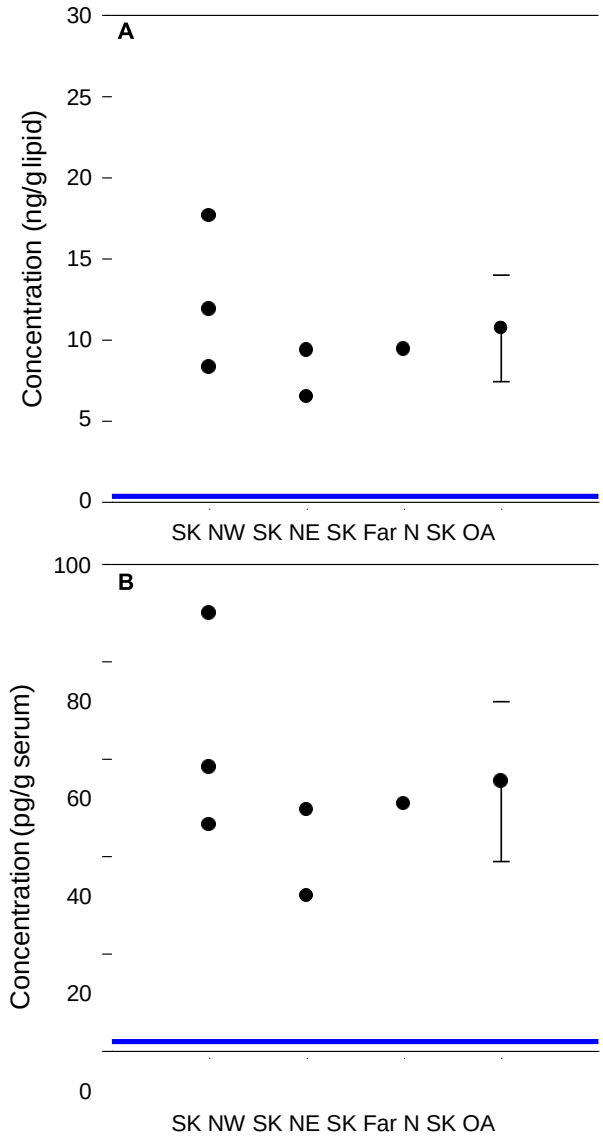

Figure 13: Concentrations of PCB congener 15 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

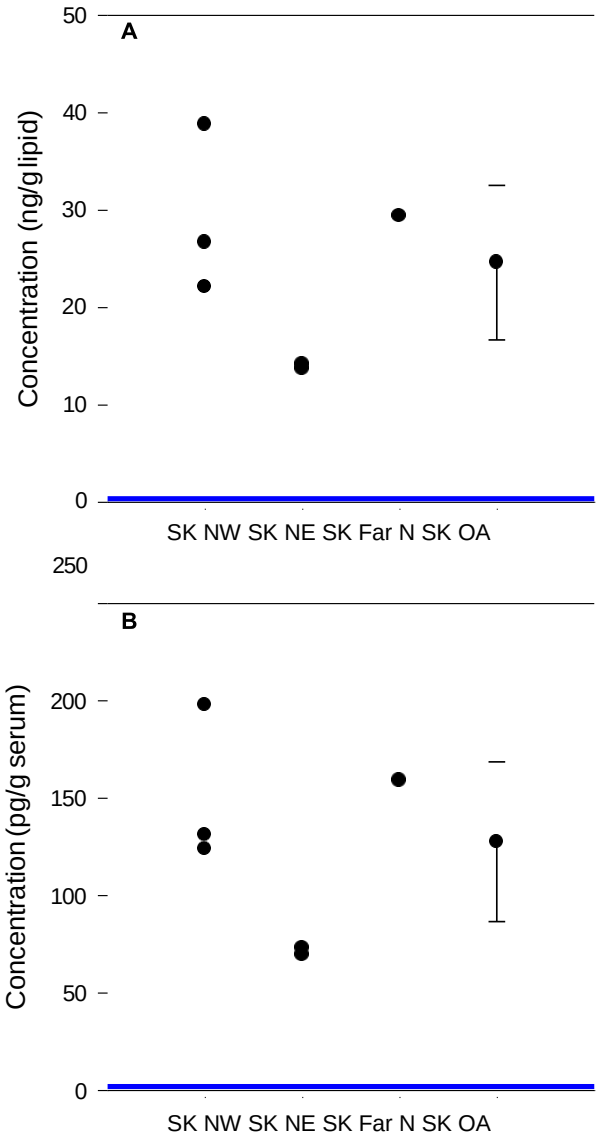

Figure 14: Concentrations of PCB congener 6 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

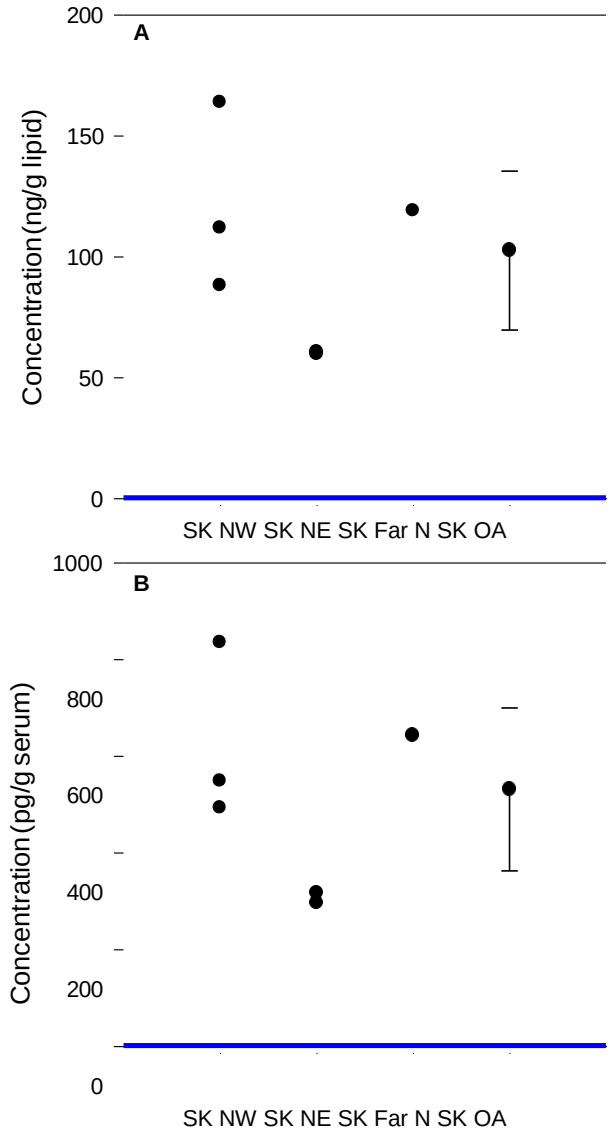

Figure 15: Concentrations of PCB congener 8 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

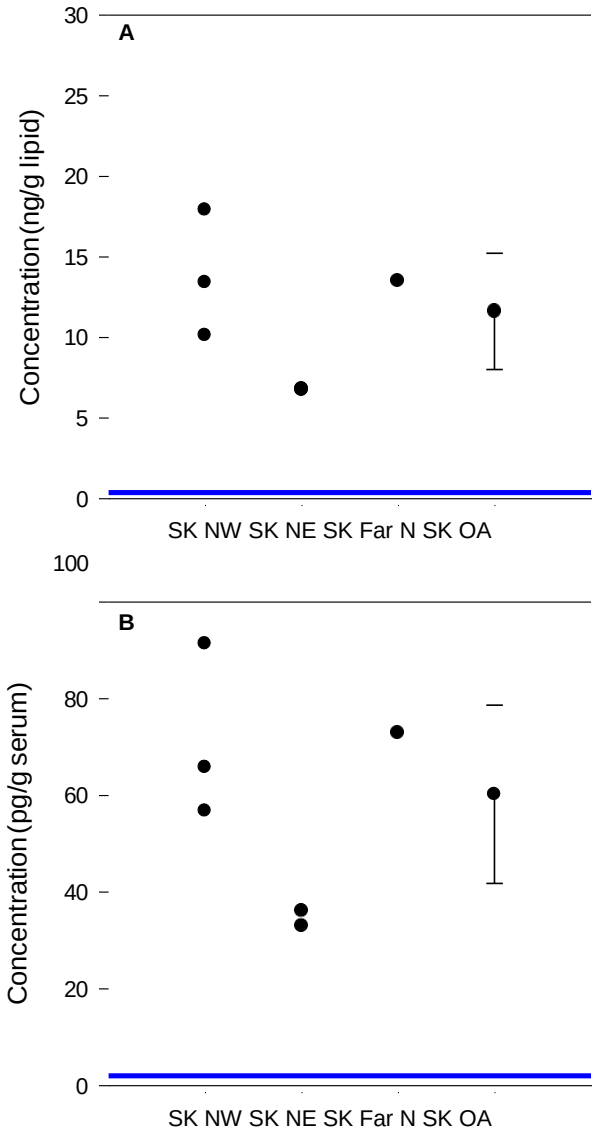

Figure 16: Concentrations of PCB congener 9 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

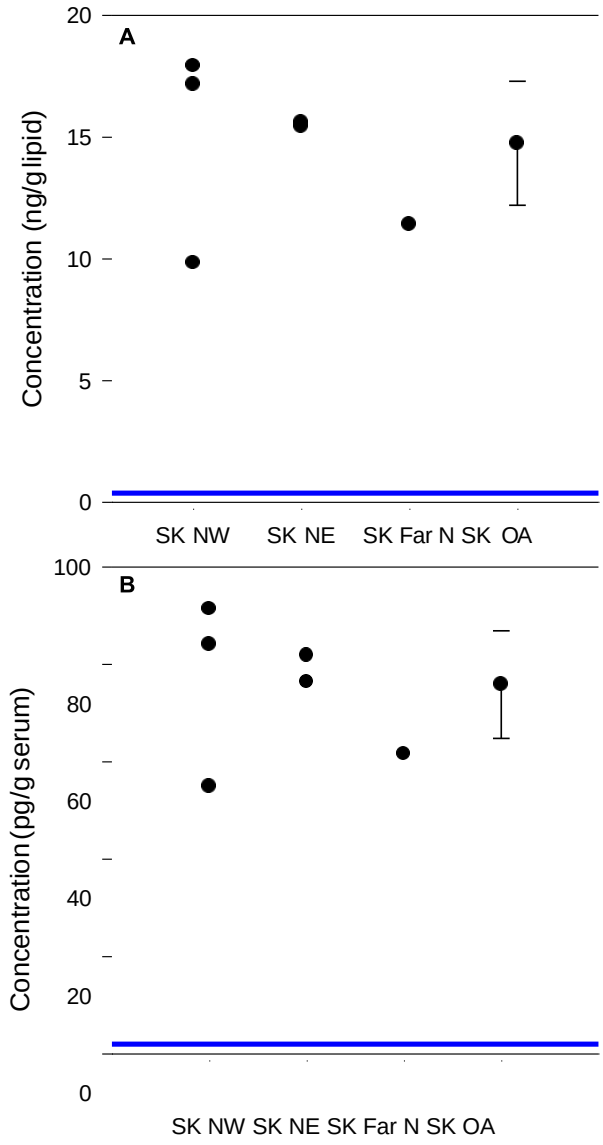

Figure 17: Concentrations of PCB congener 11 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

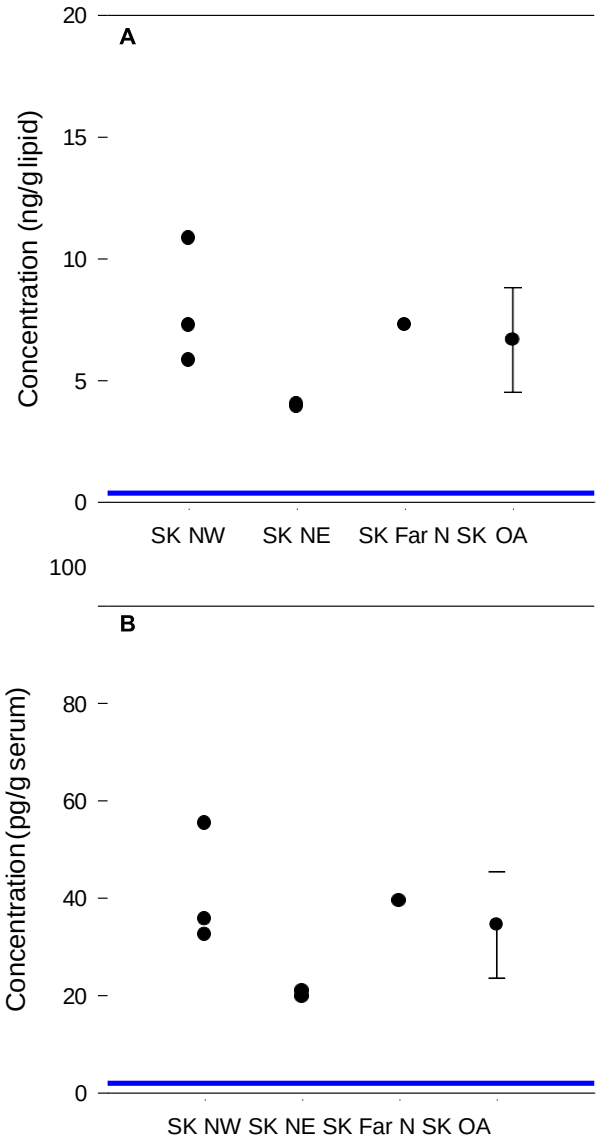

Figure 18: Concentrations of PCB congener 7 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

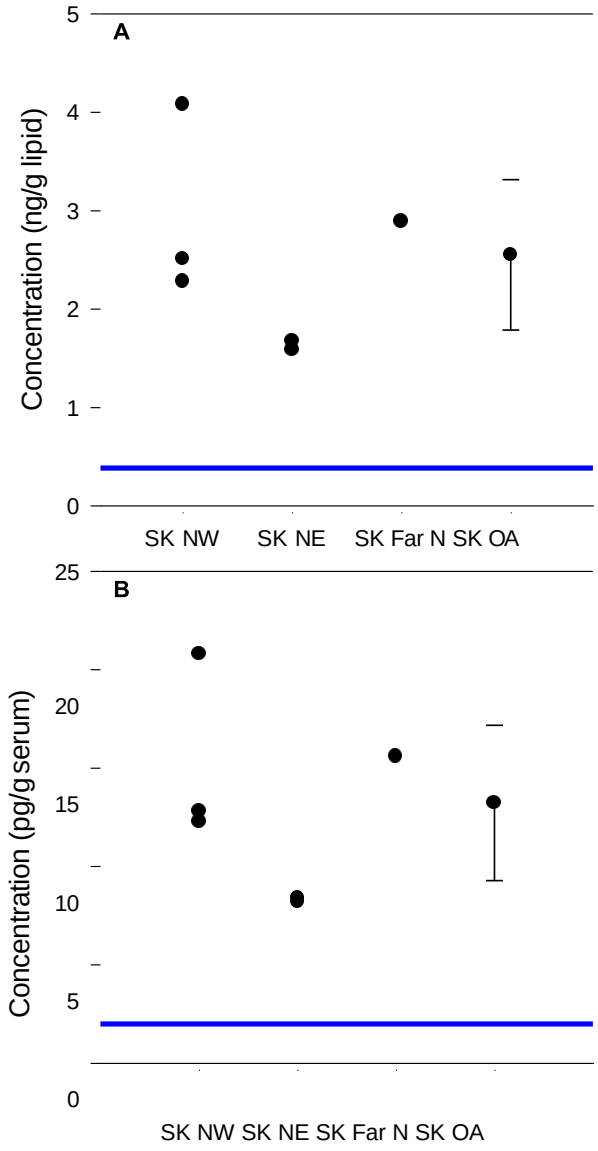

Figure 19: Concentrations of PCB congener 5 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

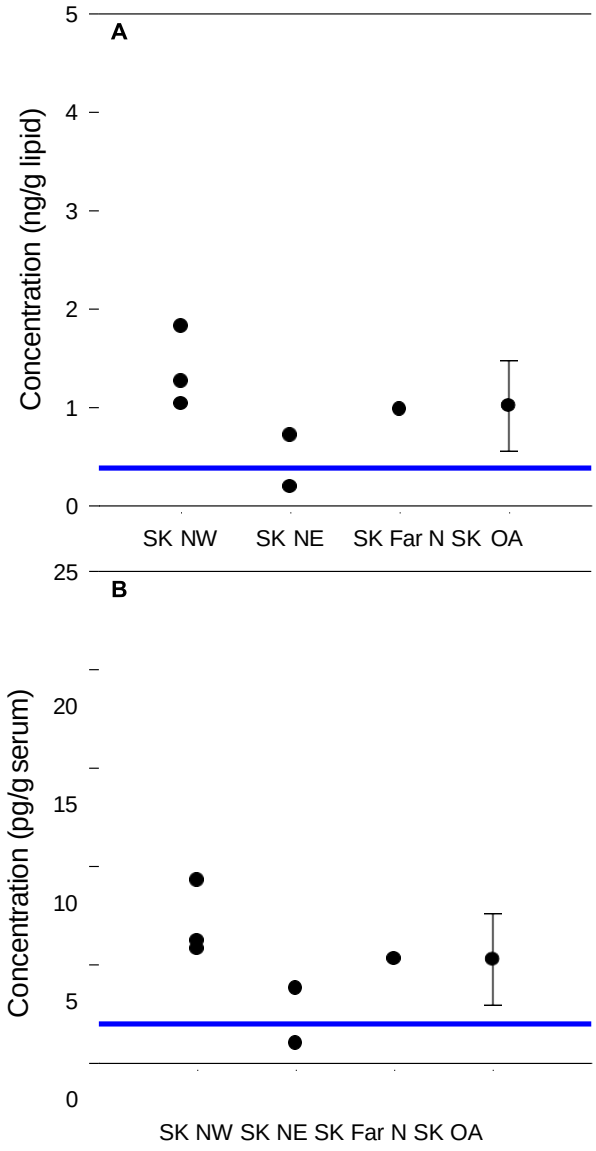

Figure 20: Concentrations of PCB congener 13 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

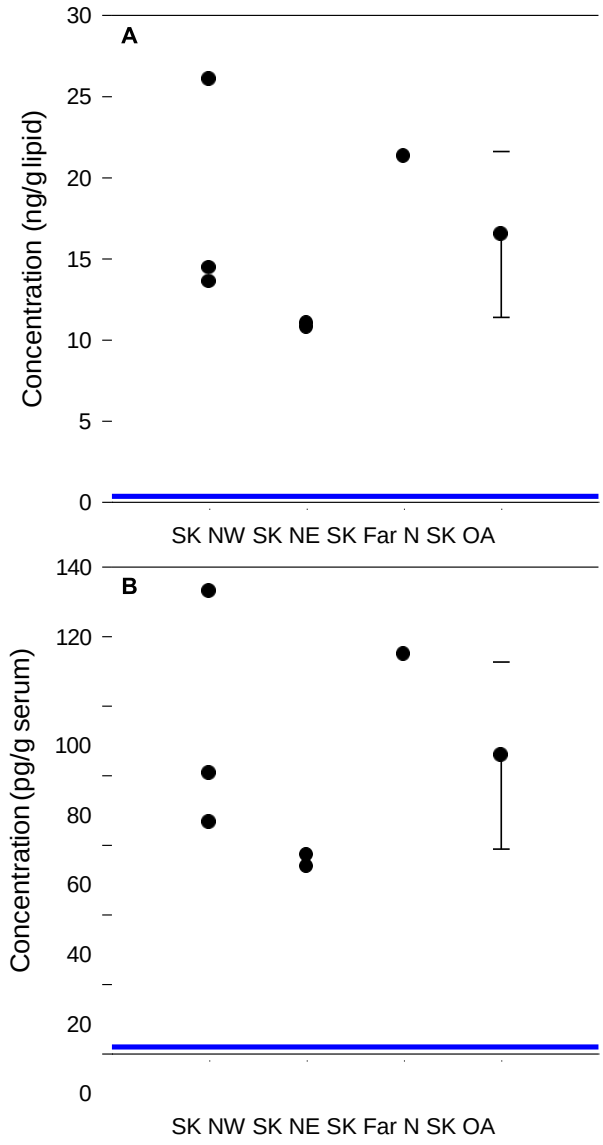

Figure 21: Concentrations of PCB congener 16 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

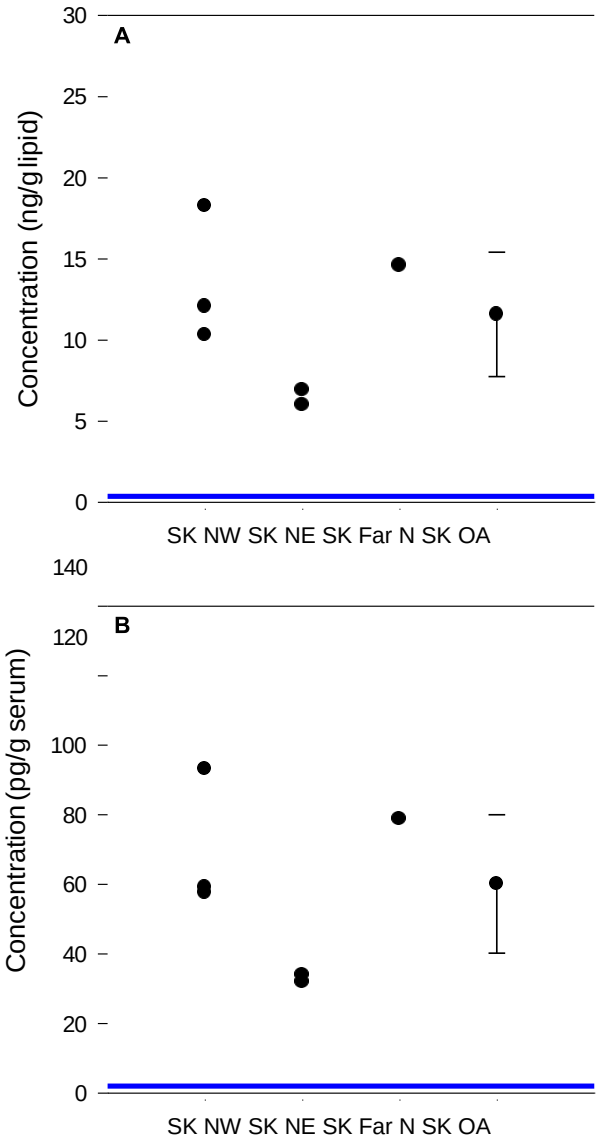

Figure 22: Concentrations of PCB congener 19 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

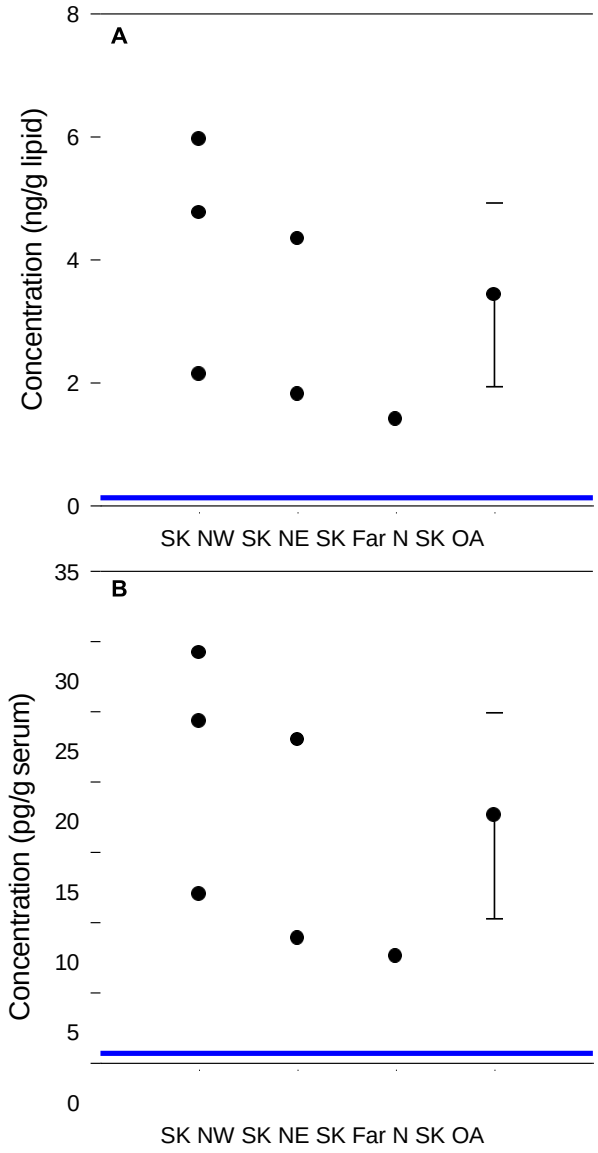

Figure 23: Concentrations of PCB congener 37 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

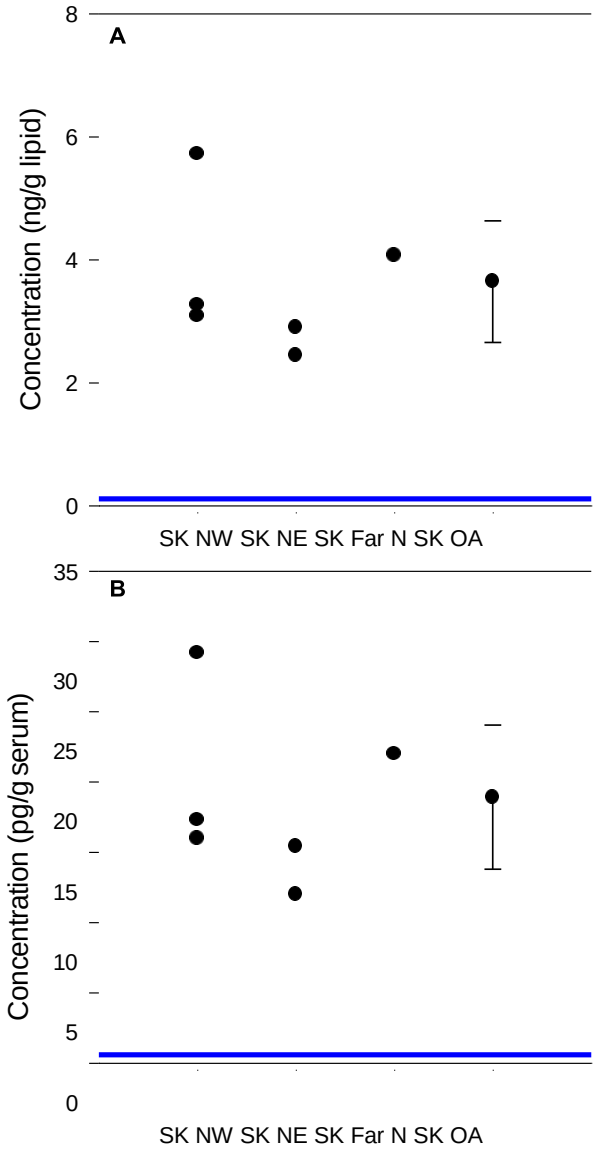

Figure 24: Concentrations of PCB congener 26 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

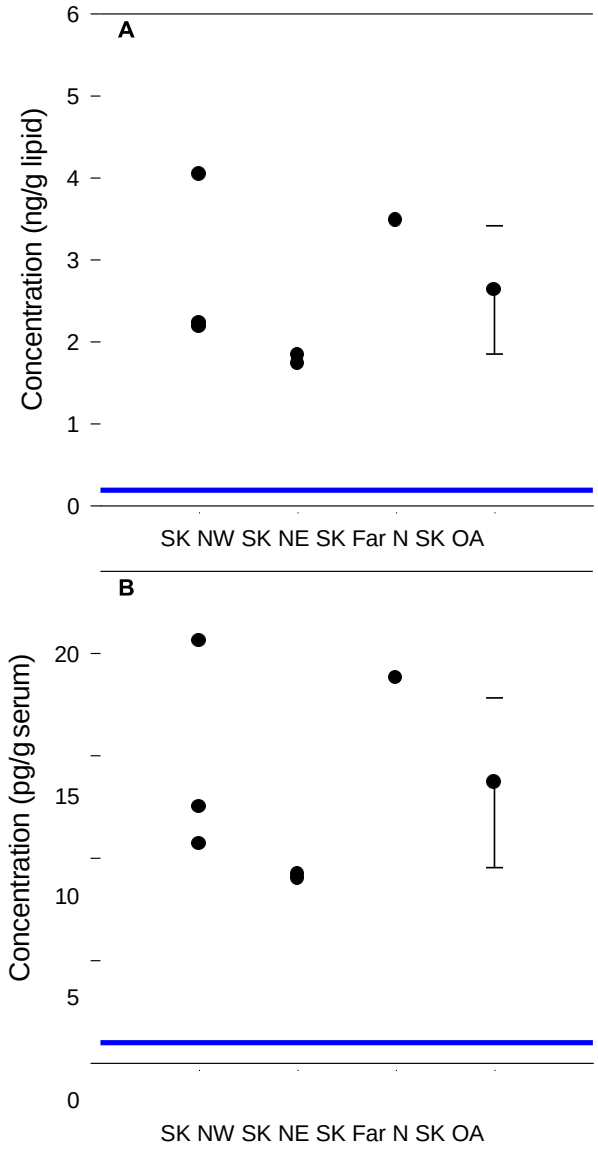

Figure 25: Concentrations of PCB congener 27 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

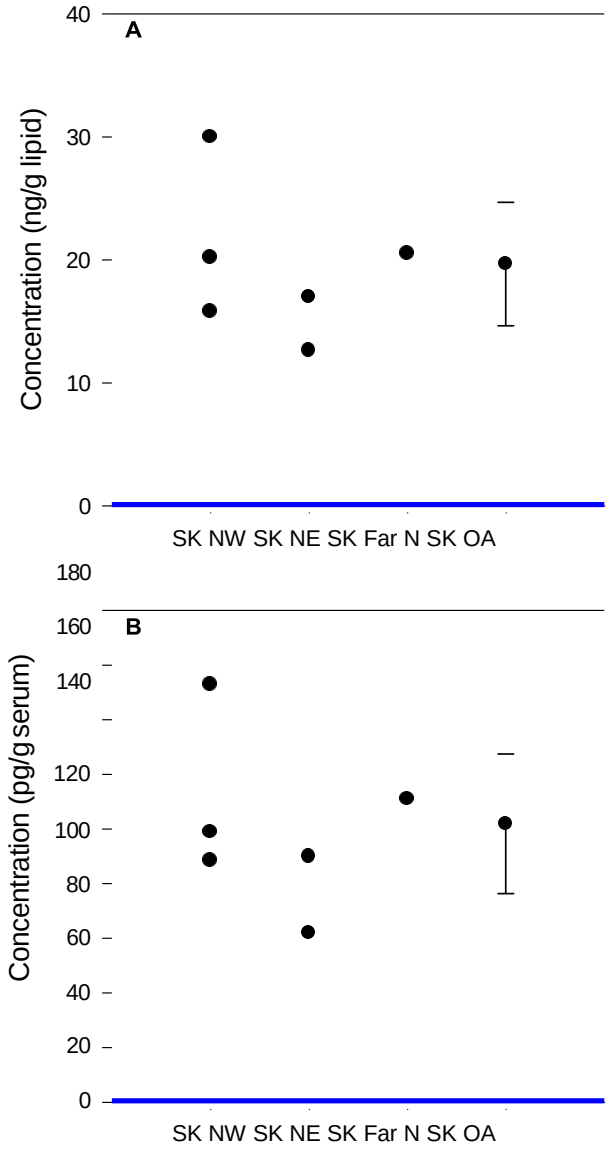

Figure 26: Concentrations of PCB congener 31 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

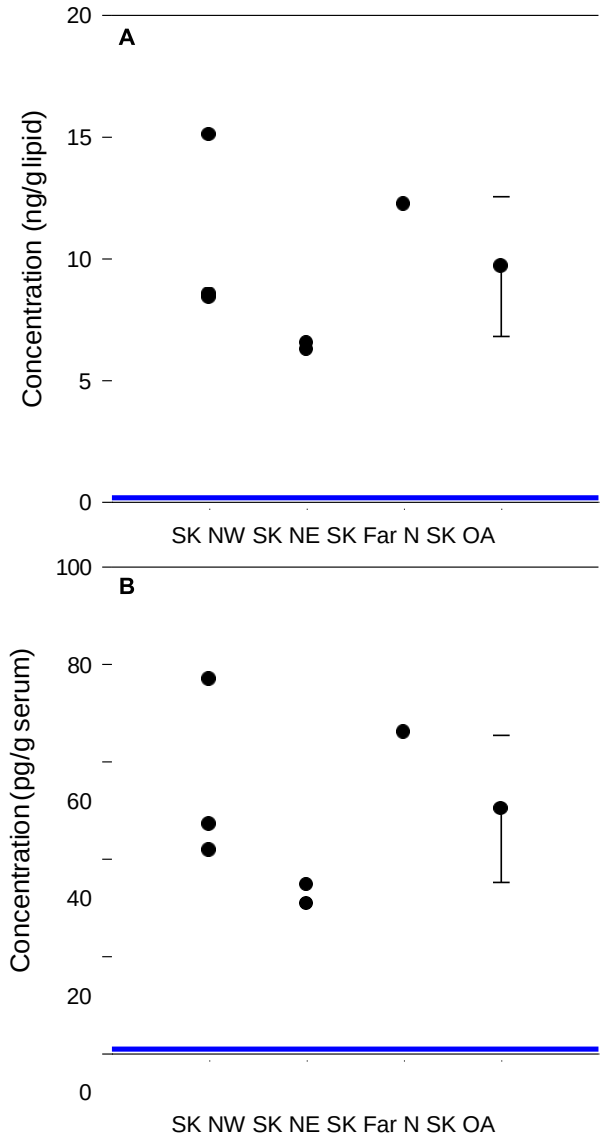

Figure 27: Concentrations of PCB congener 32 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

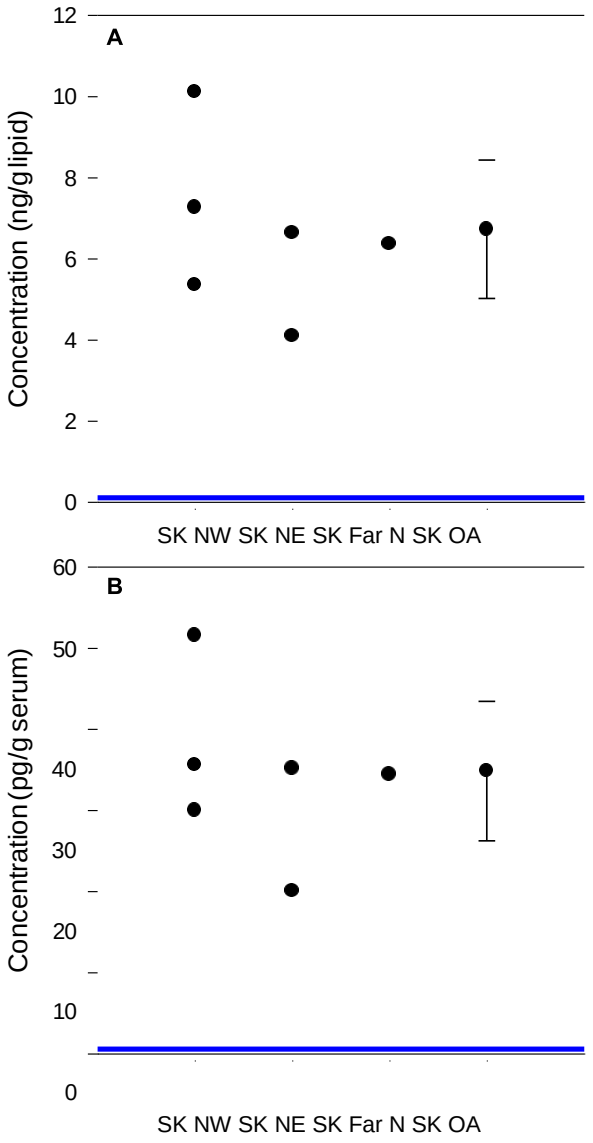

Figure 28: Concentrations of PCB congener 22 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

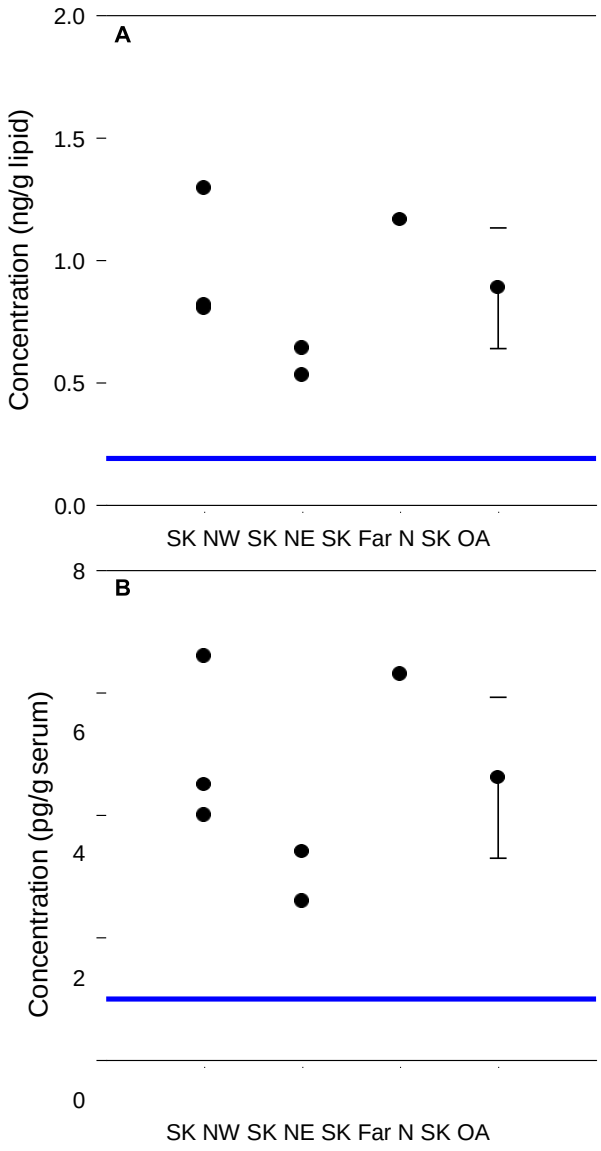

Figure 29: Concentrations of PCB congener 24 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

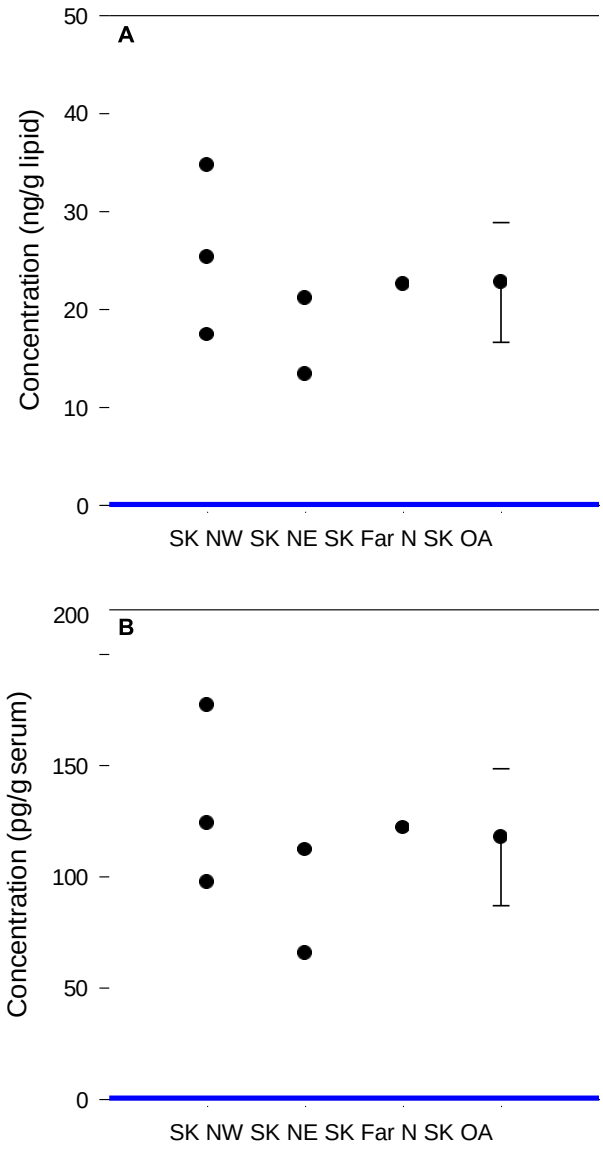

Figure 30: Concentrations of PCB congener 28 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

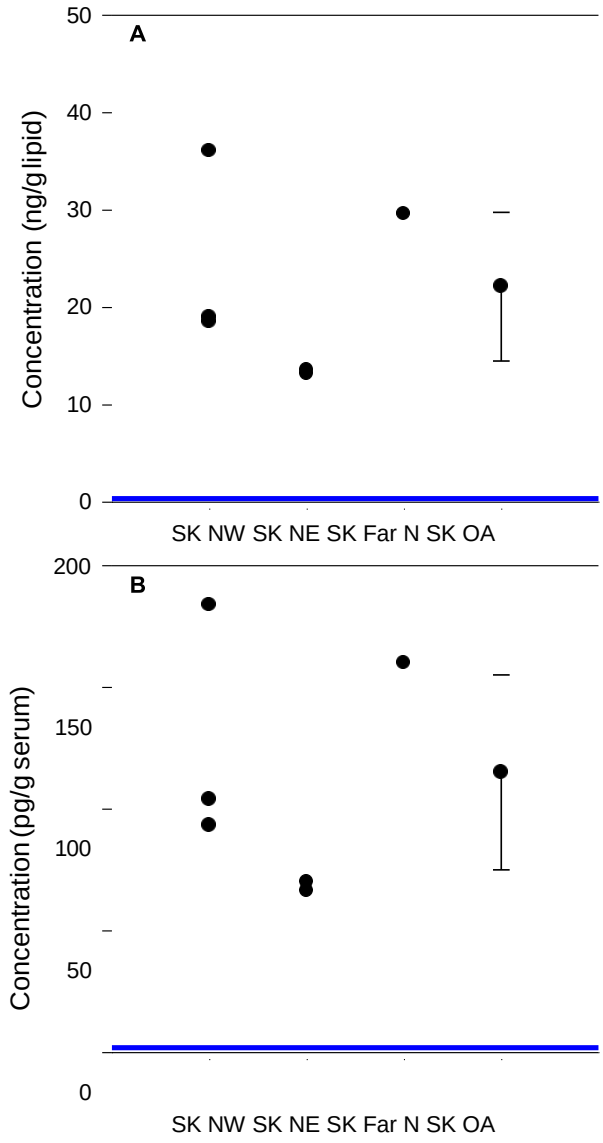

Figure 31: Concentrations of PCB congener 17 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

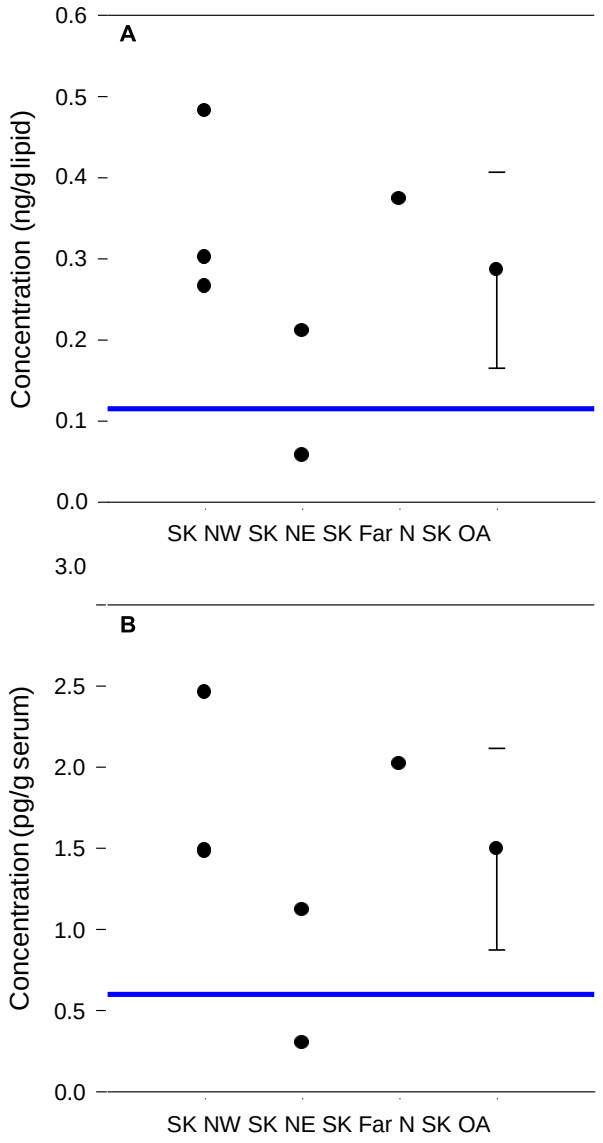

Figure 32: Concentrations of PCB congener 29 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

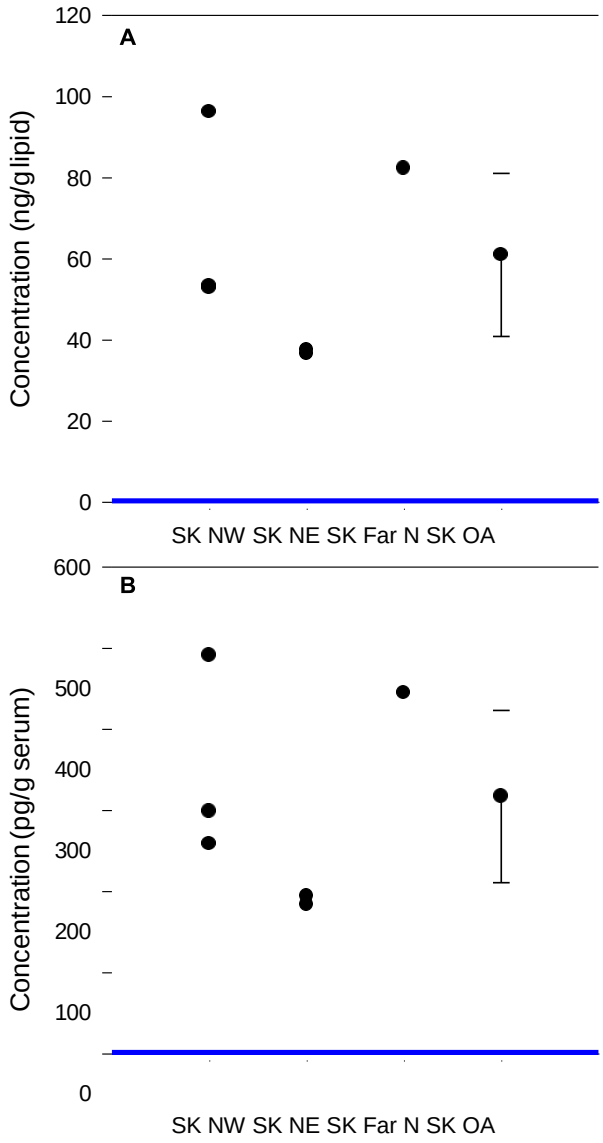

Figure 33: Concentrations of PCB congener 18 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

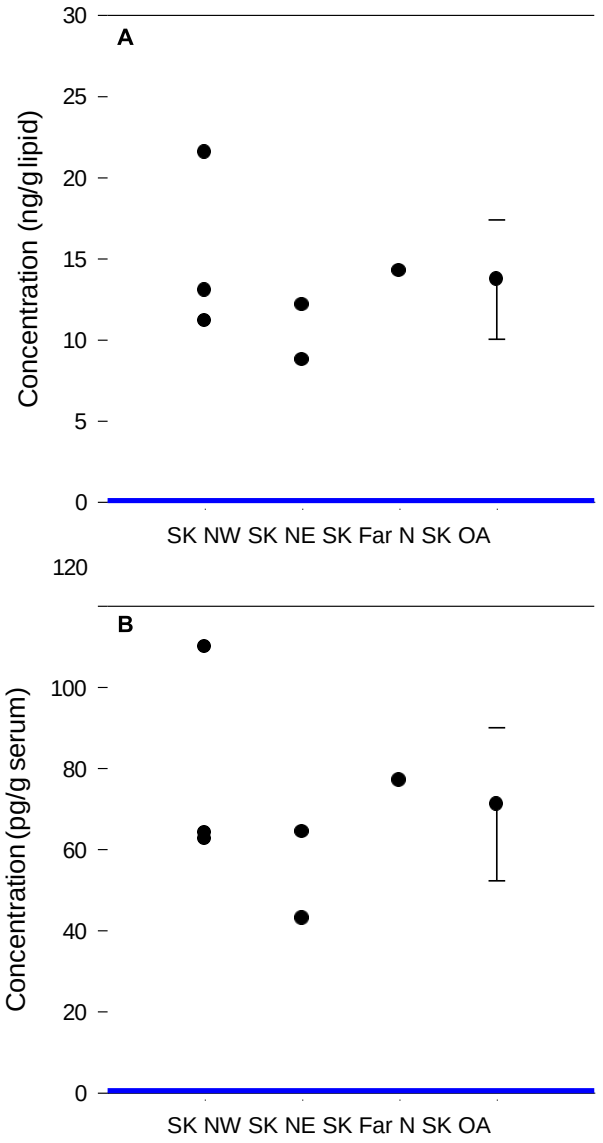

Figure 34: Concentrations of PCB congener 21/20/33 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentrations (A) and whole serum concentrations (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

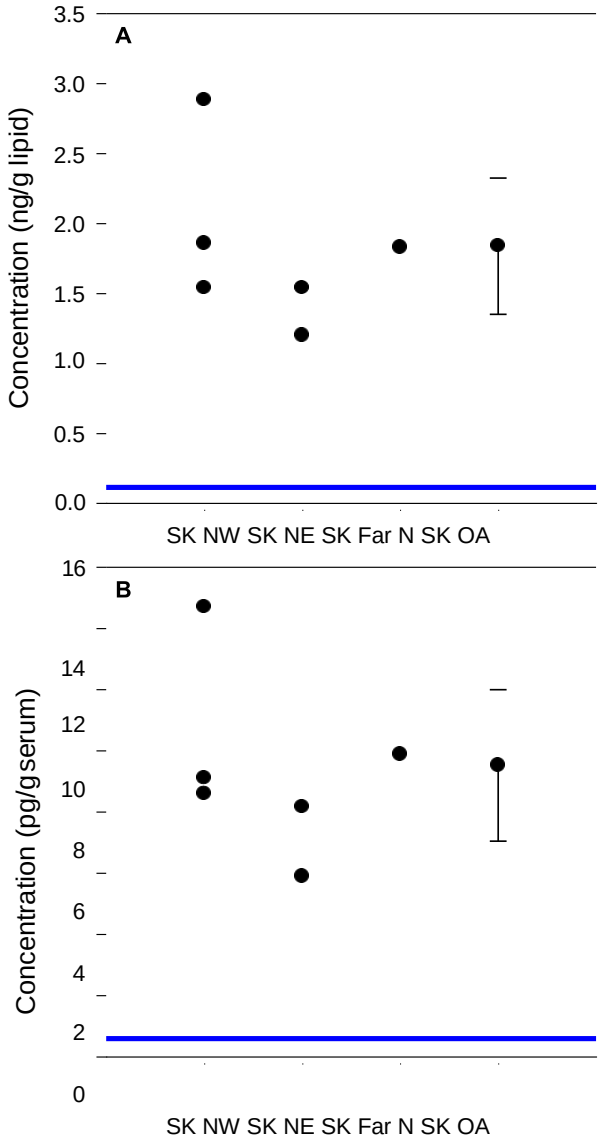

Figure 35: Concentrations of PCB congener 25 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

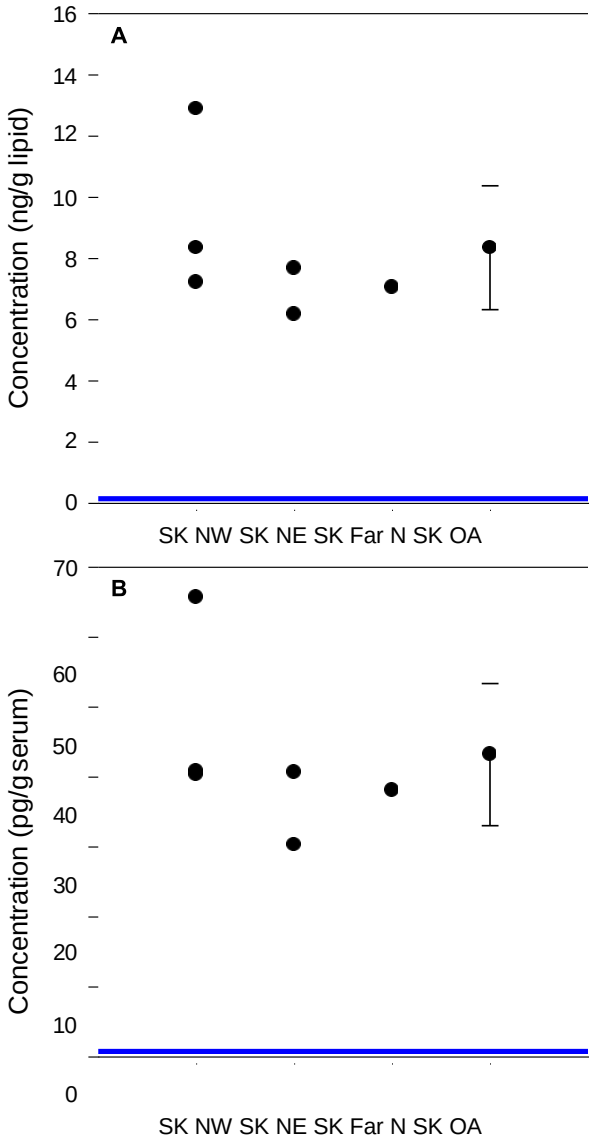

Figure 36: Concentrations of PCB congener 48/49 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

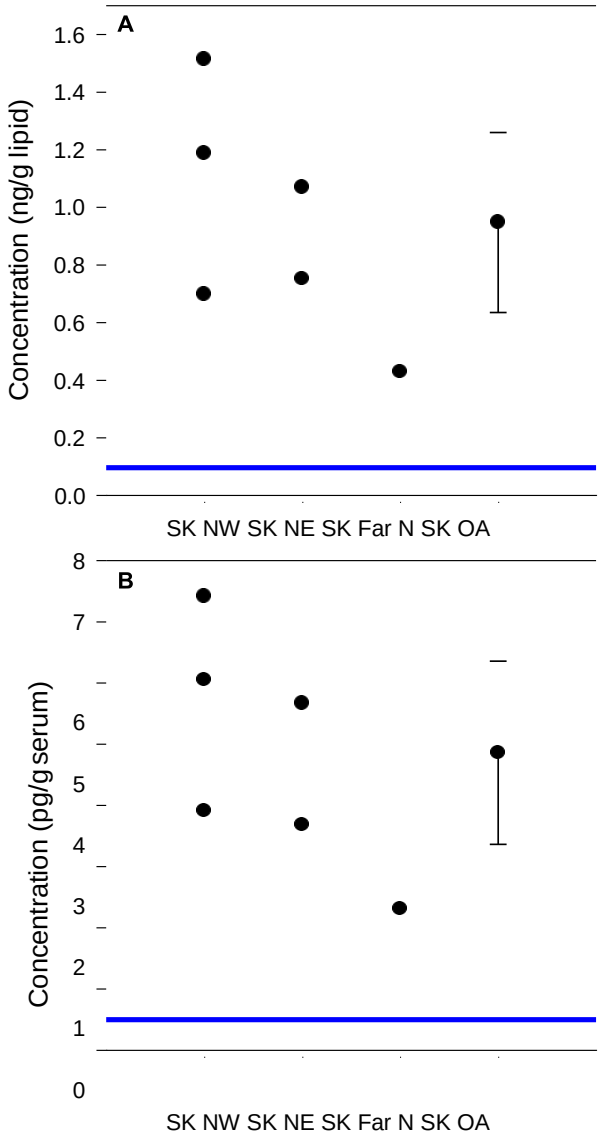

Figure 37: Concentrations of PCB congener 60 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

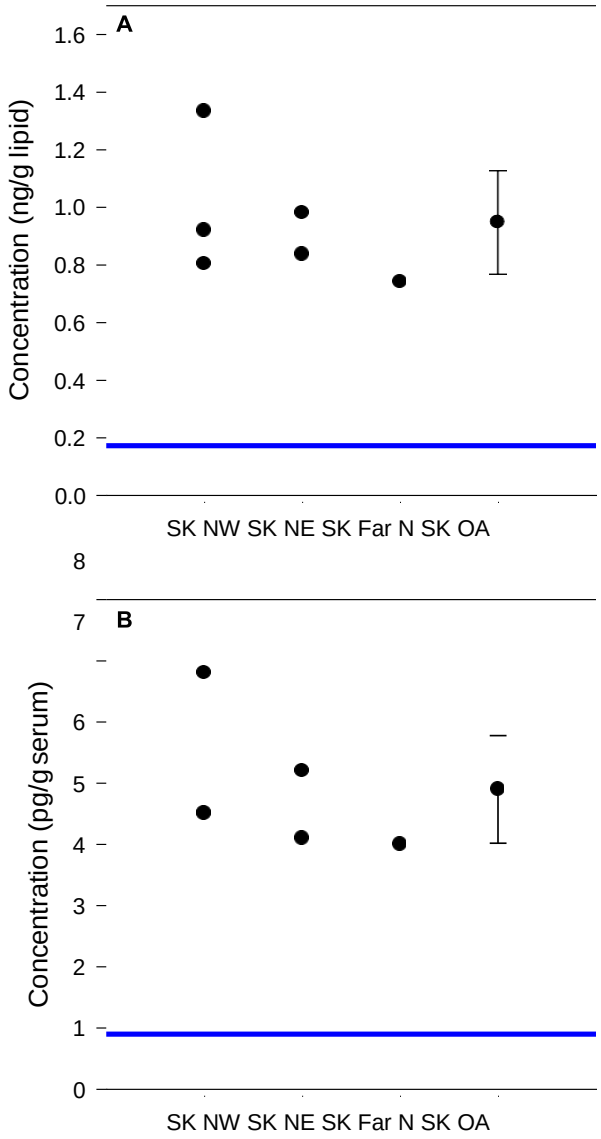

Figure 38: Concentrations of PCB congener 41 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

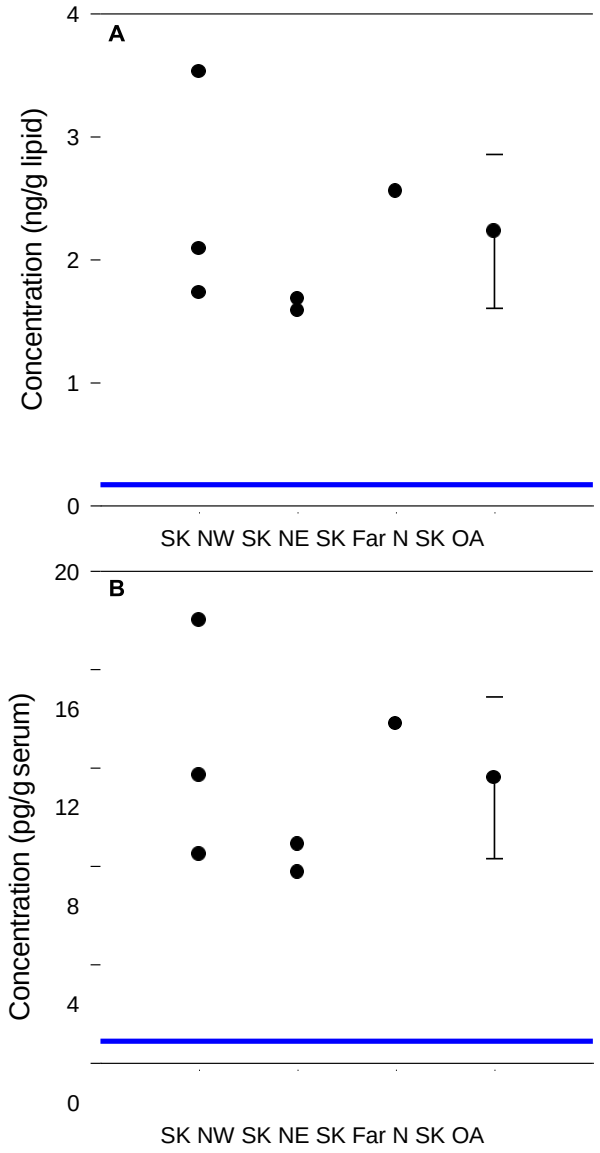

Figure 39: Concentrations of PCB congener 45 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

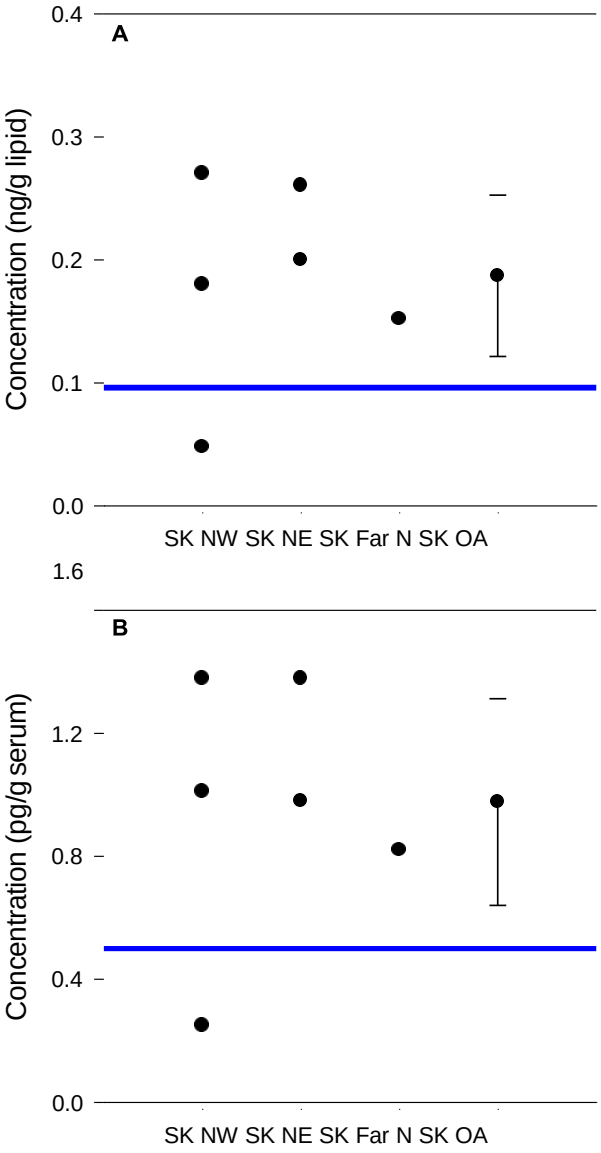

Figure 40: Concentrations of PCB congener 63/76 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

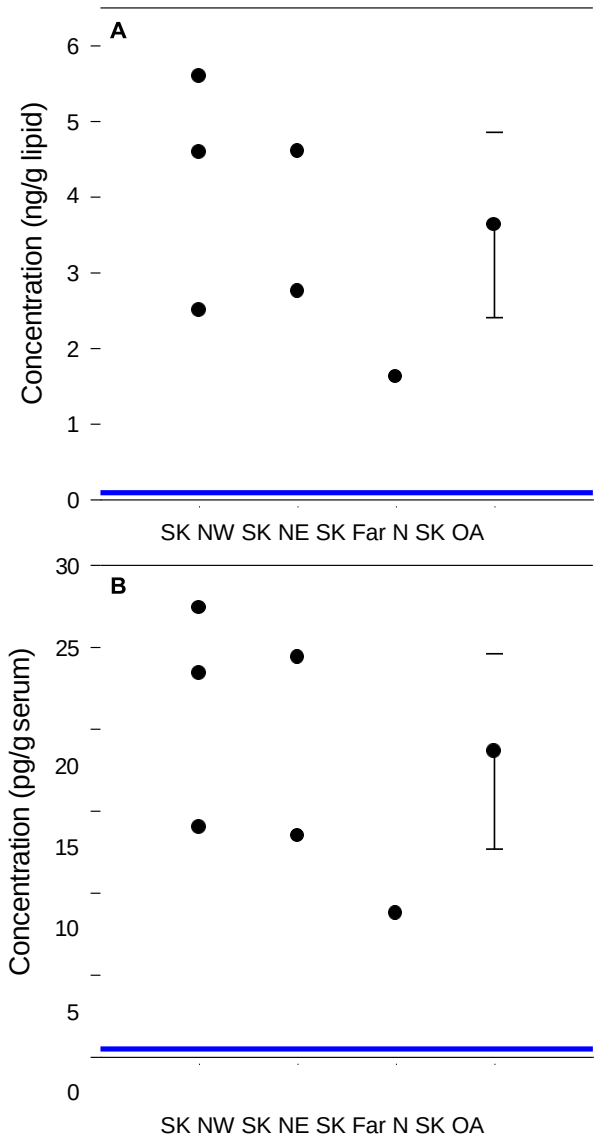

Figure 41: Concentrations of PCB congener 66 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

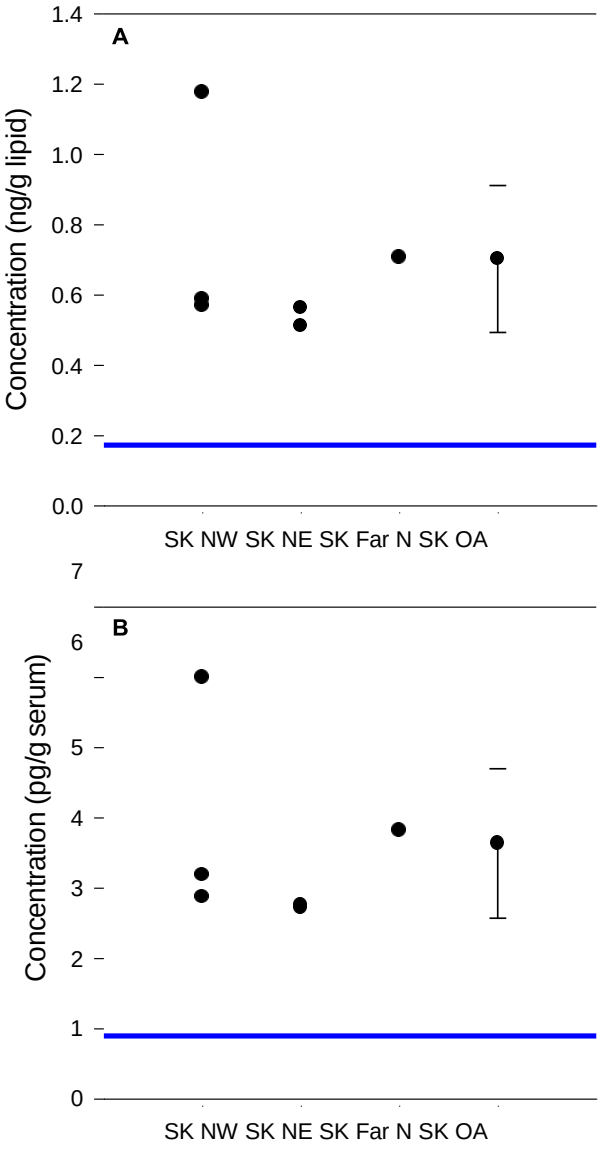

Figure 42: Concentrations of PCB congener 46 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

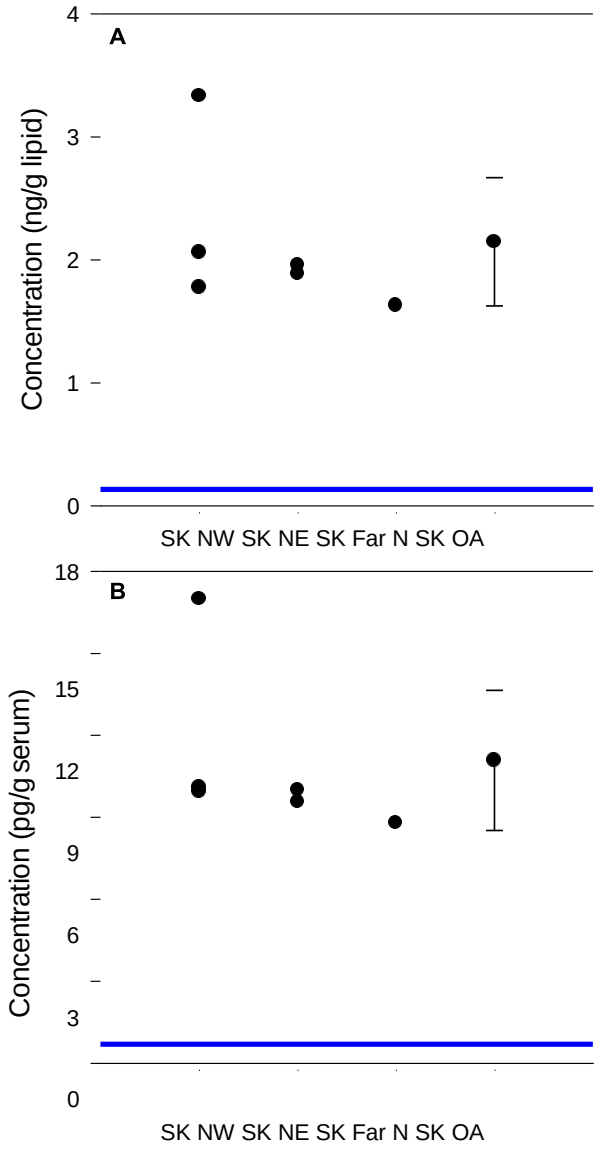

Figure 43: Concentrations of PCB congener 59/42 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

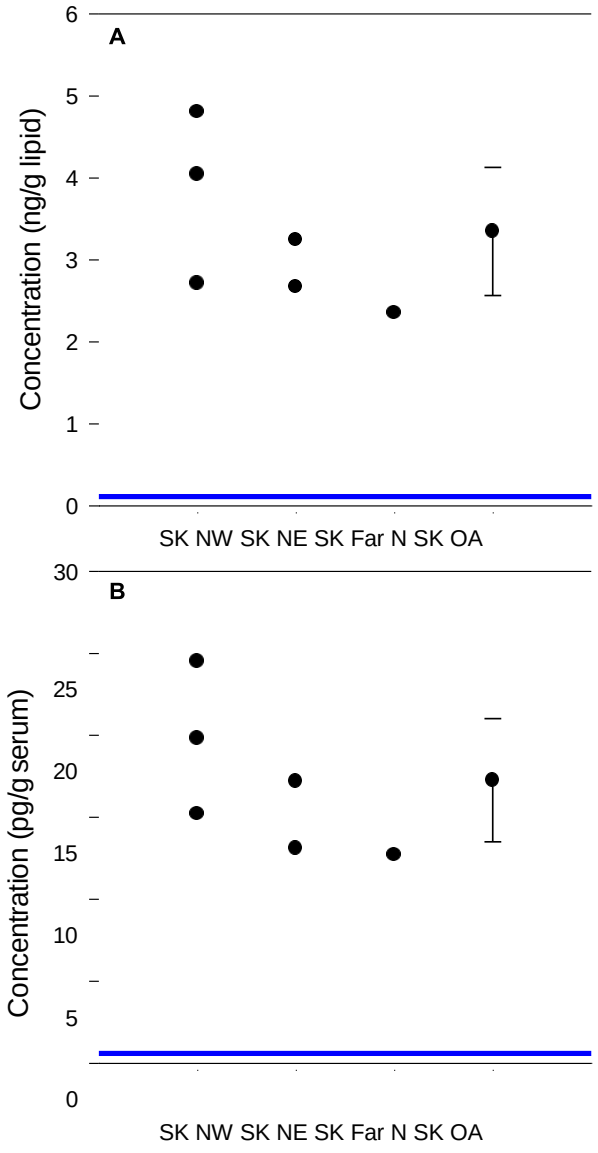

Figure 44: Concentrations of PCB congener 64 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

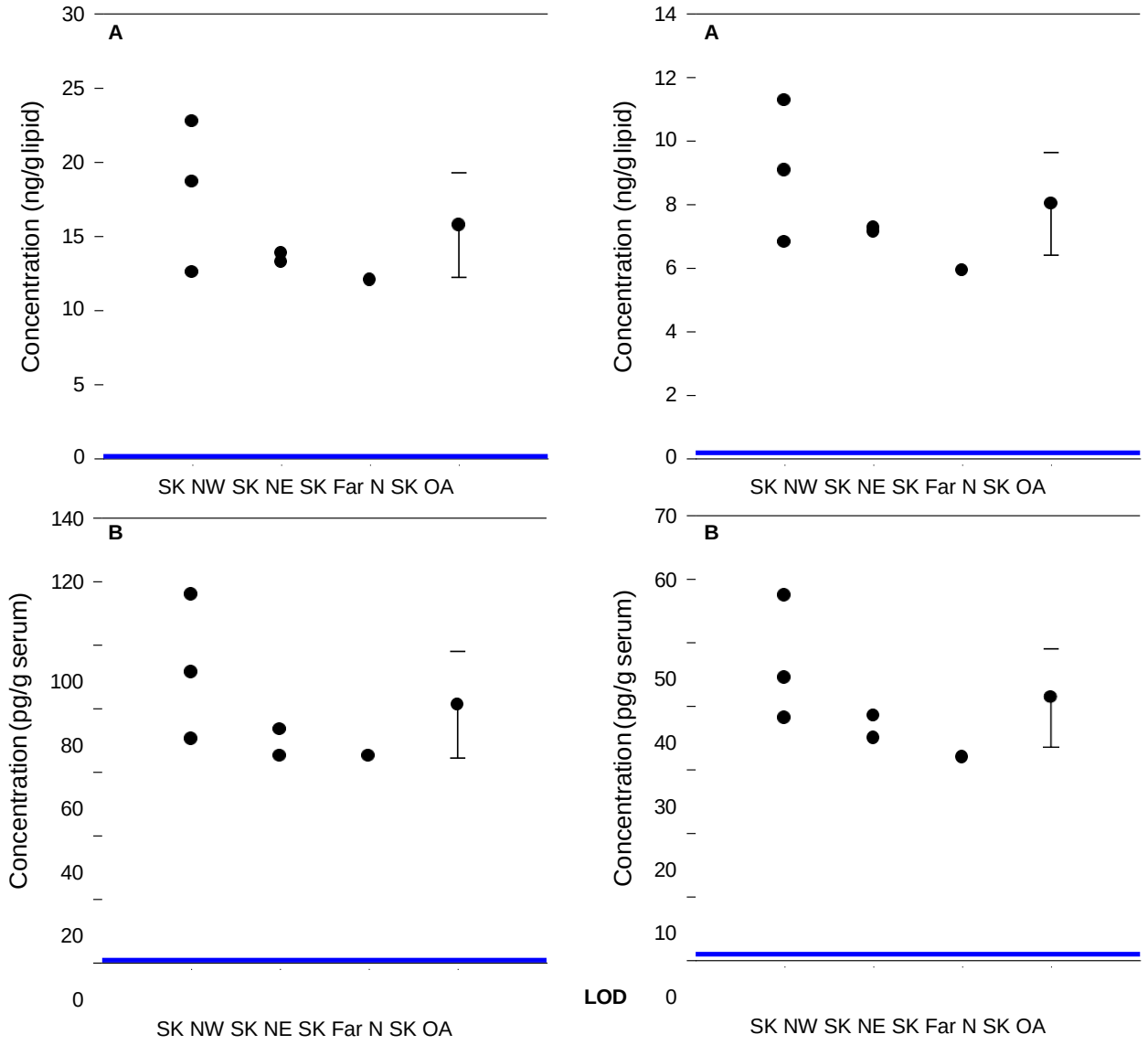

Figure 45: Concentrations of PCB congener 43/52 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

Figure 46: Concentrations of PCB congener 44 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

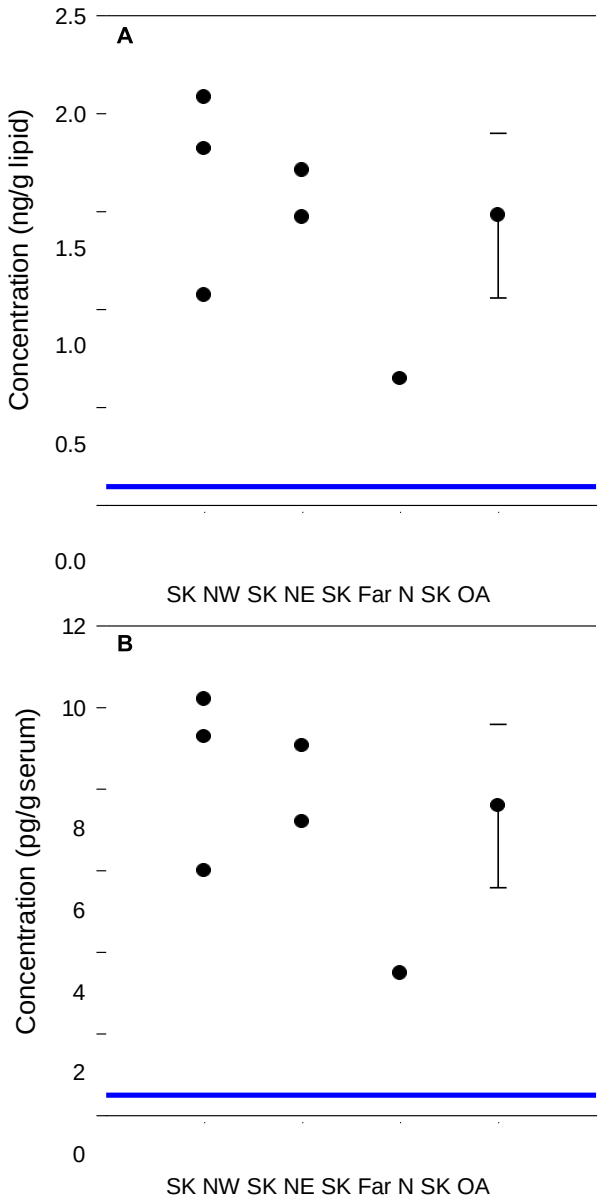

Figure 47: Concentrations of PCB congener 56 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

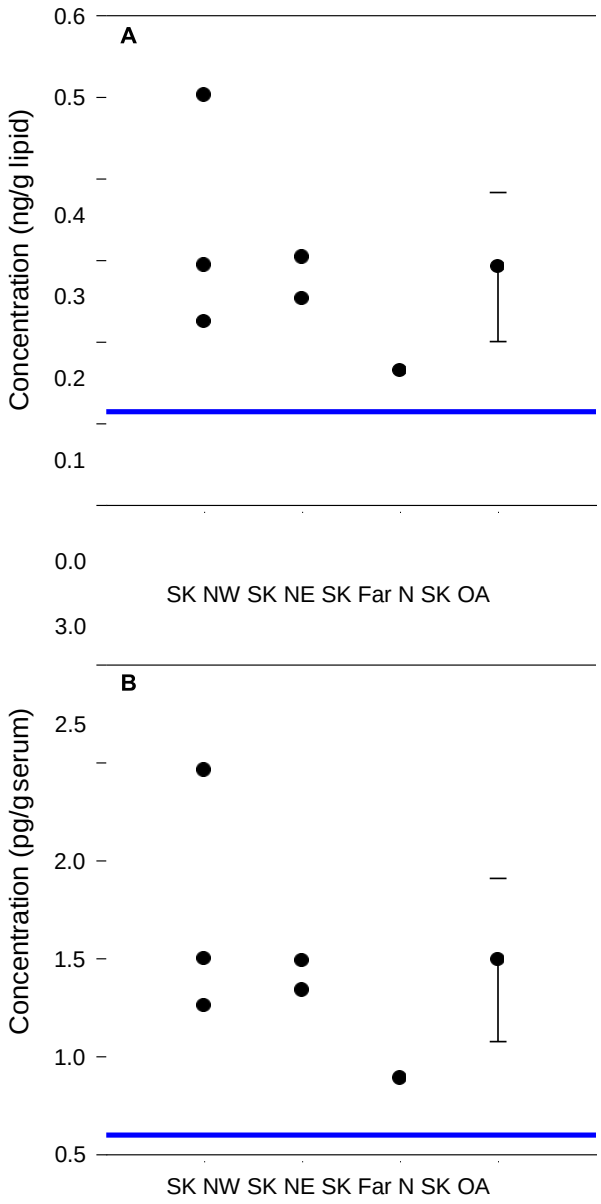

Figure 48: Concentrations of PCB congener 77 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

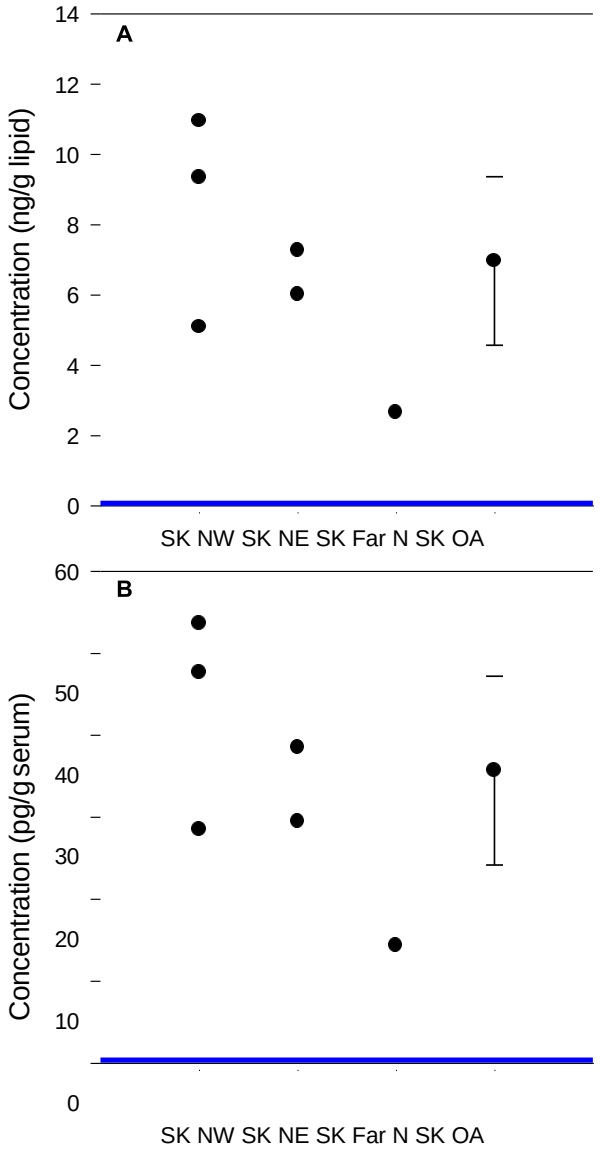

Figure 49: Concentrations of PCB congener 70 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

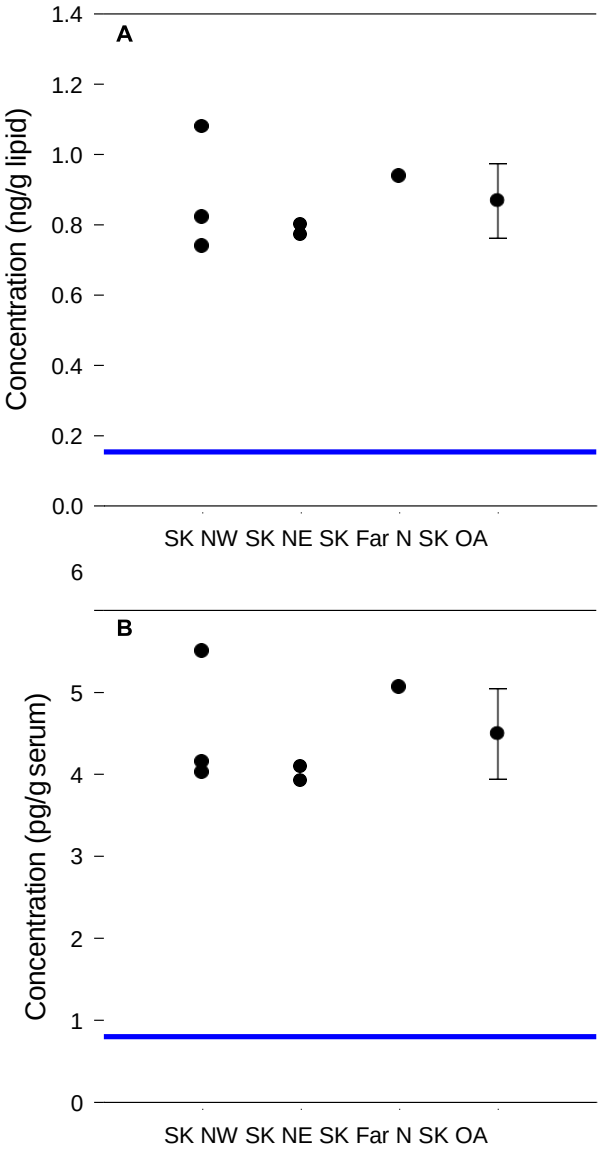

Figure 50: Concentrations of PCB congener 51 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

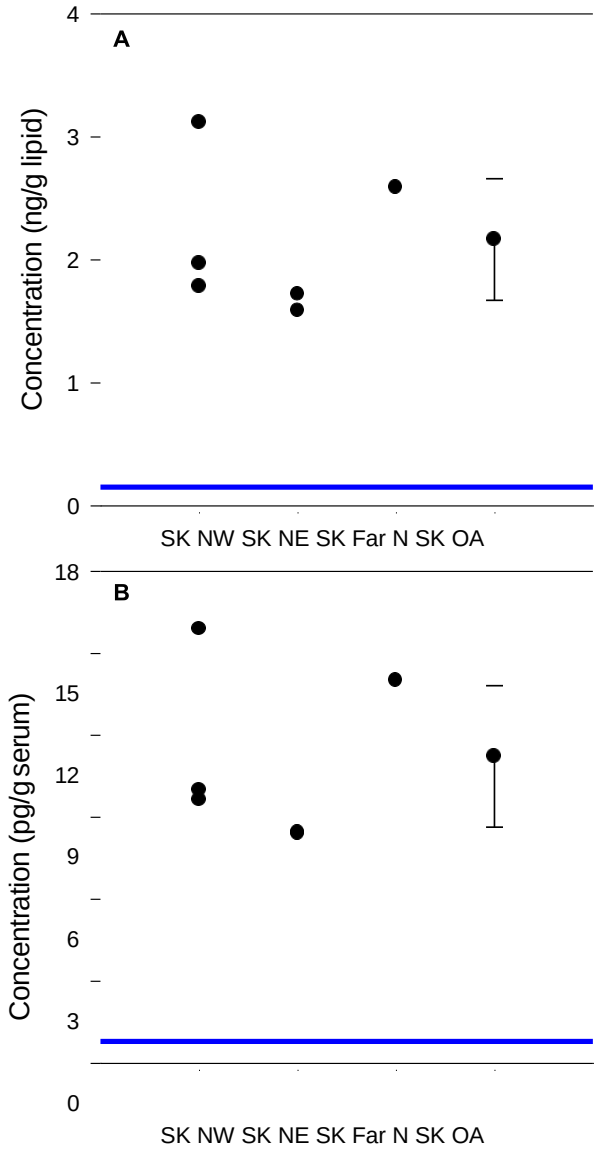

Figure 51: Concentrations of PCB congener 53 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

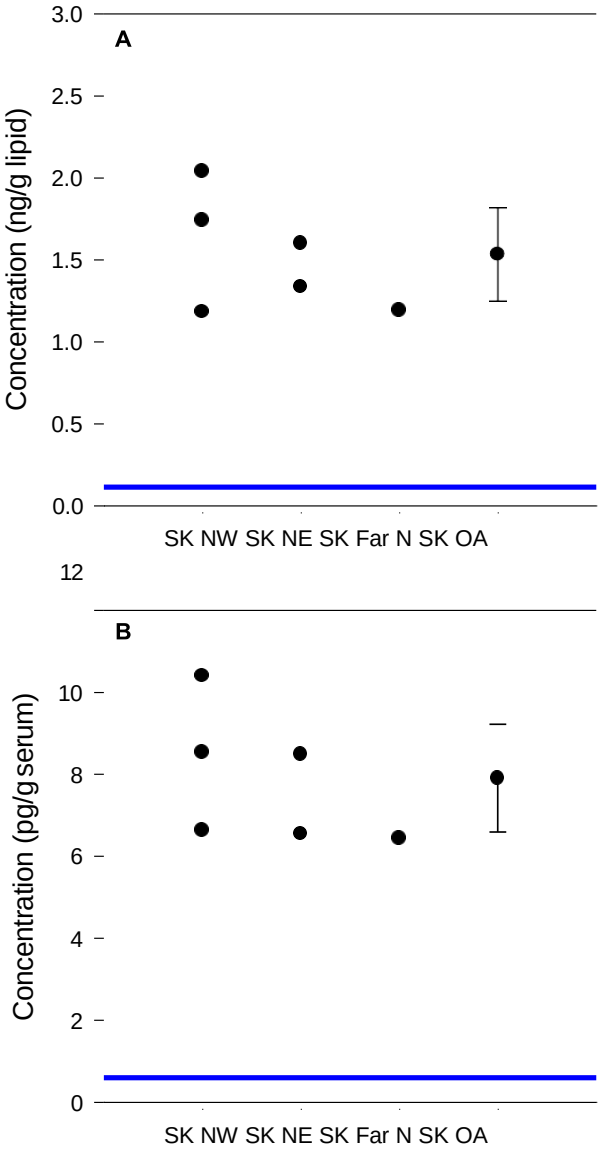

Figure 52: Concentrations of PCB congener 71 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

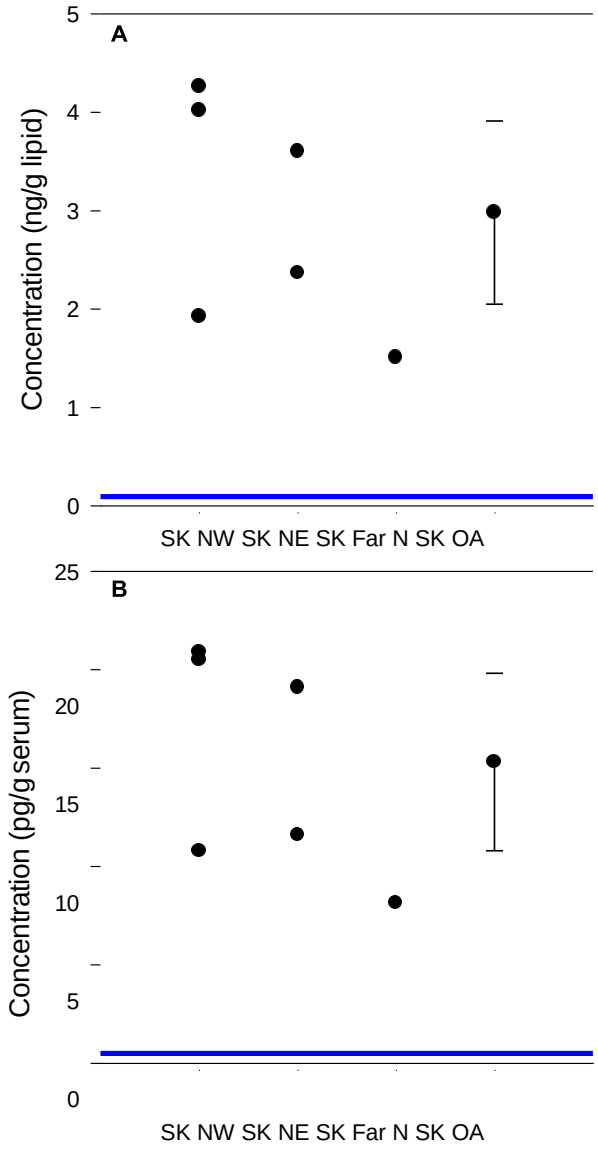

Figure 53: Concentrations of PCB congener 74 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

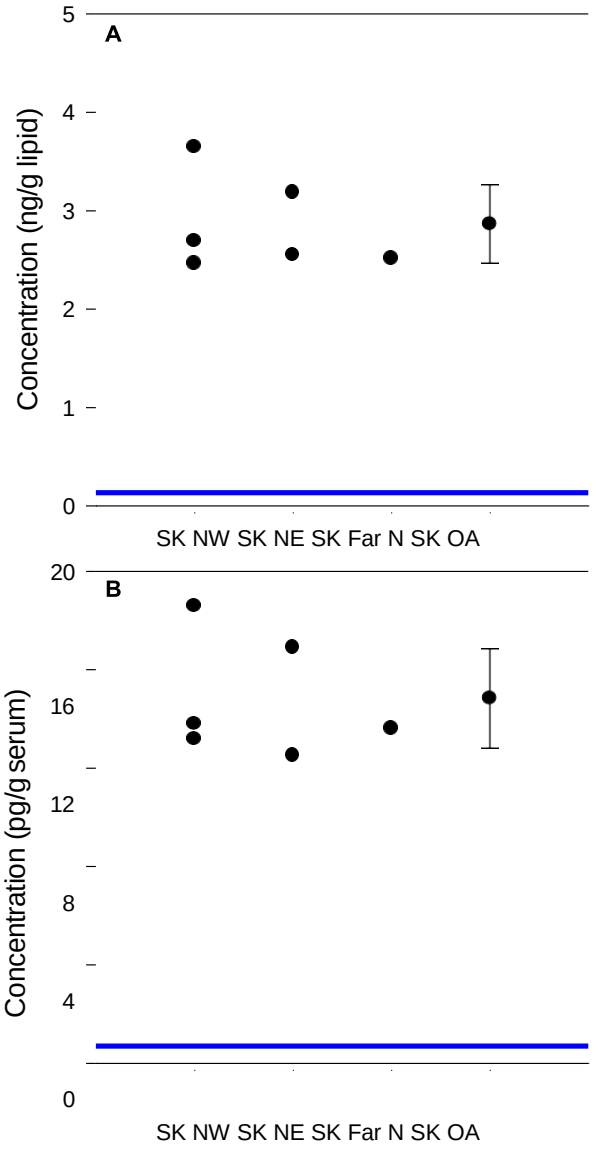

Figure 54: Concentrations of PCB congener 47 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean

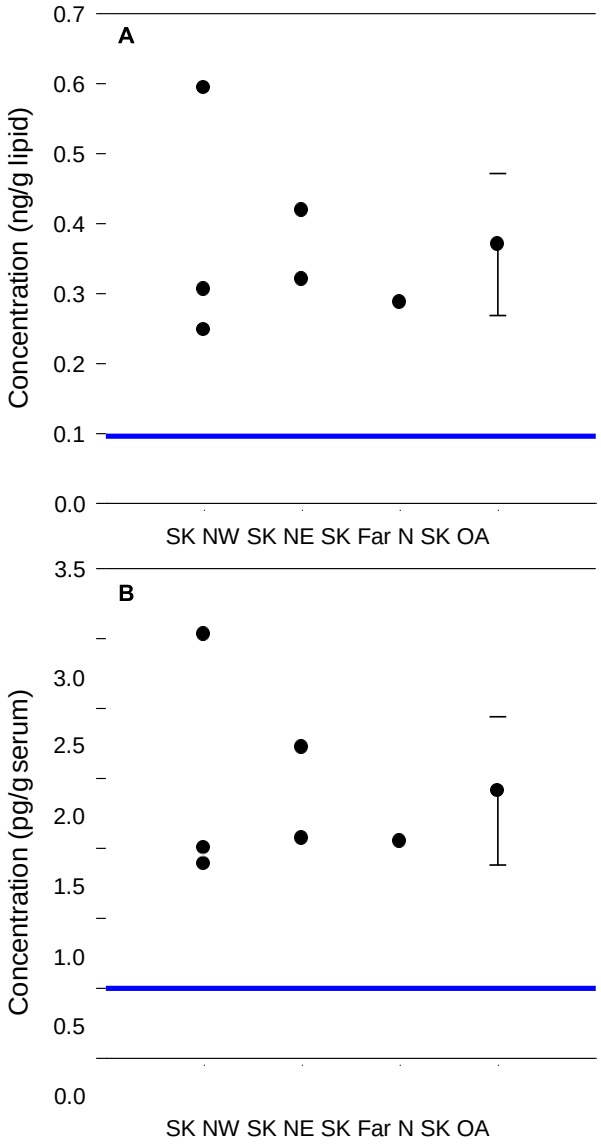

Figure 55: Concentrations of PCB congener 40/68 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

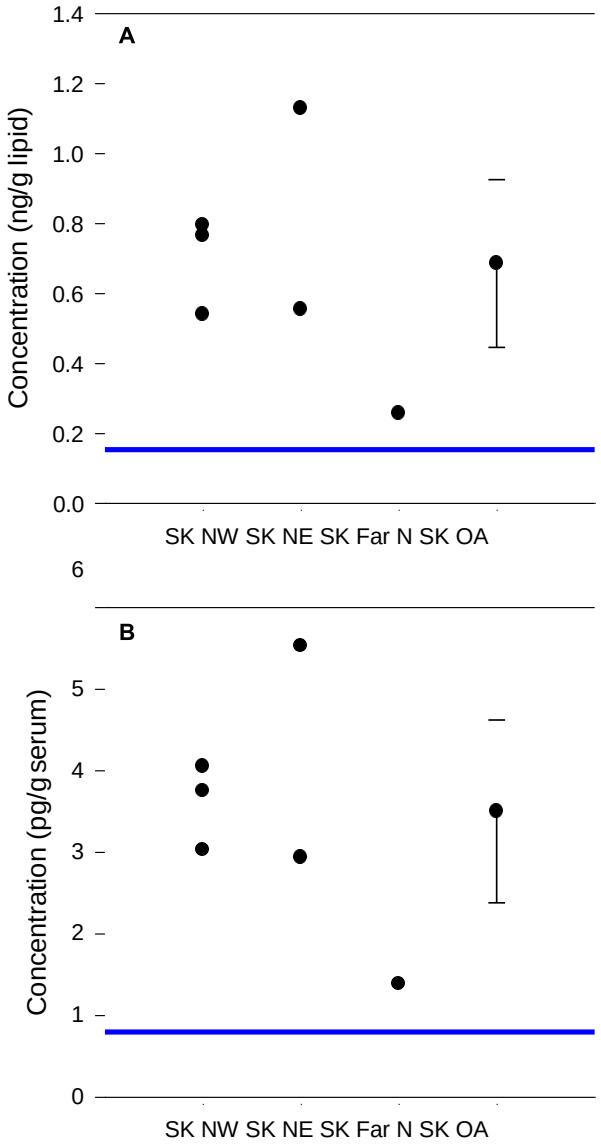

Figure 56: Concentrations of PCB congener 85 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

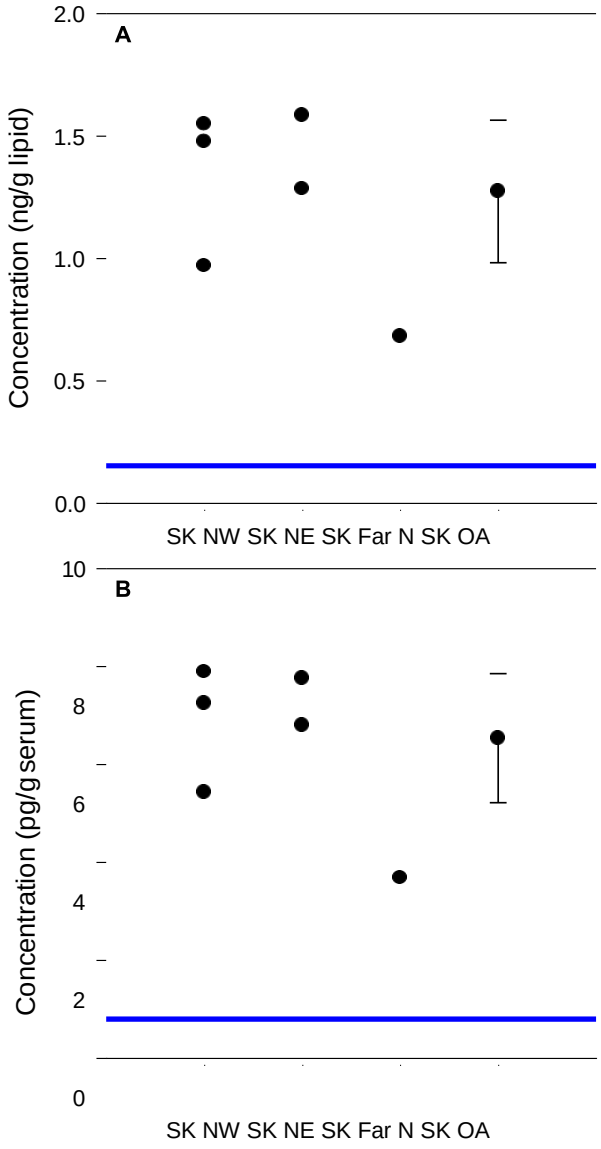

Figure 57: Concentrations of PCB congener 92 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

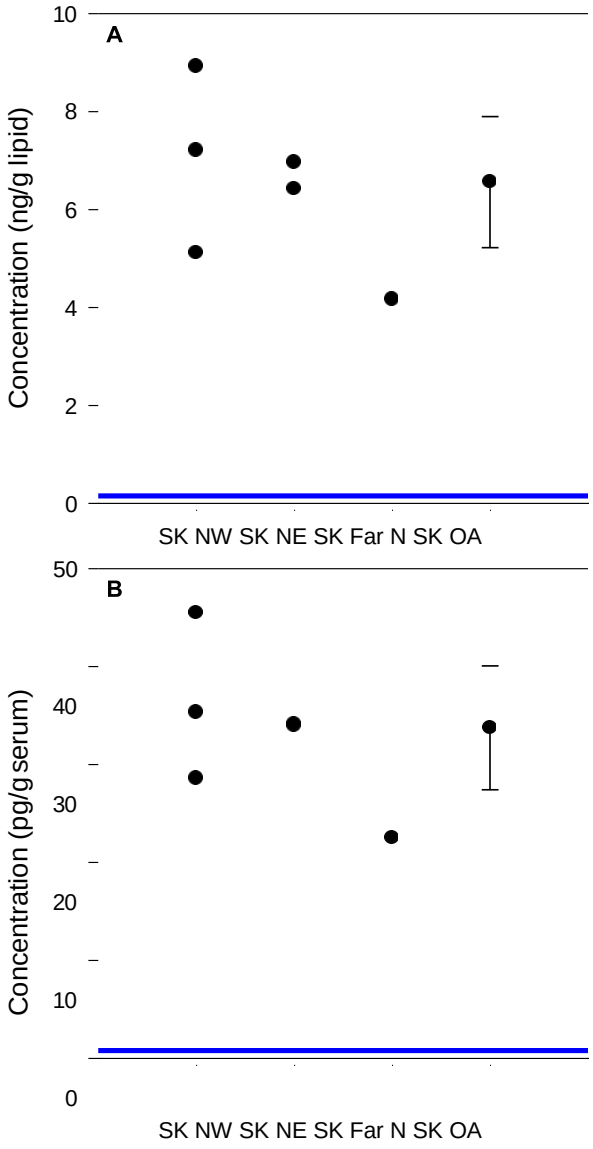

Figure 58: Concentrations of PCB congener 95 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

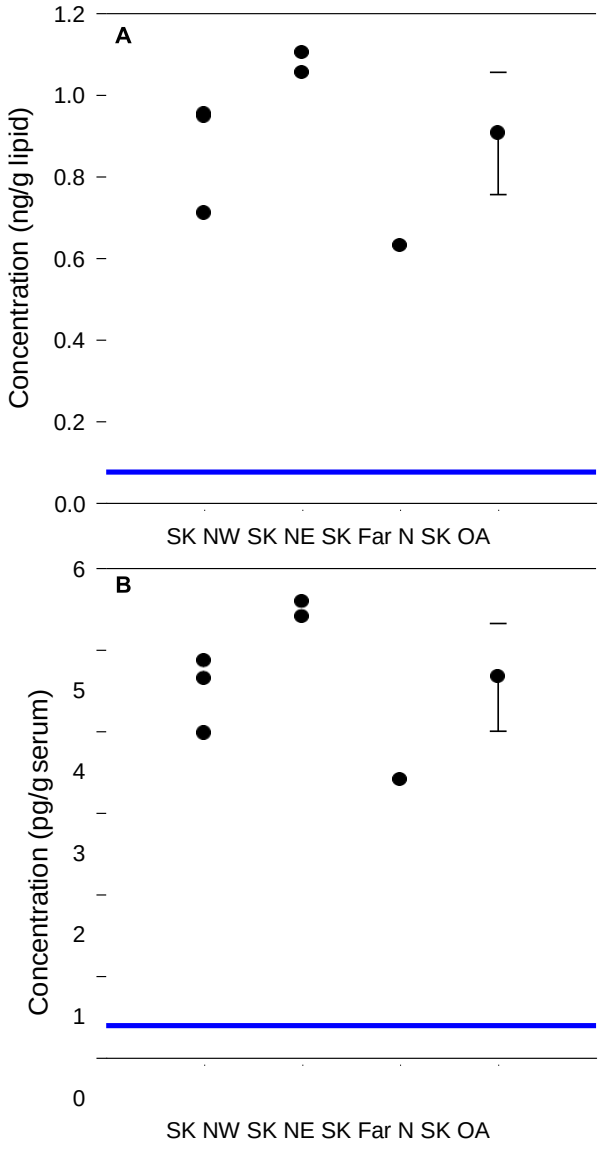

Figure 59: Concentrations of PCB congener 105 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

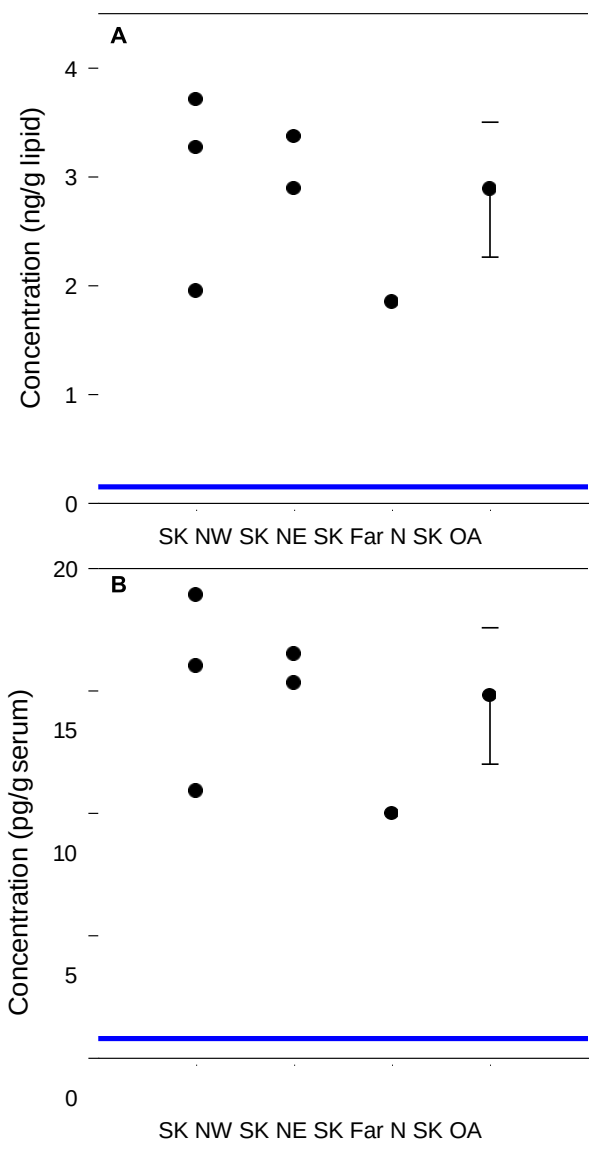

Figure 60: Concentrations of PCB congener 99 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

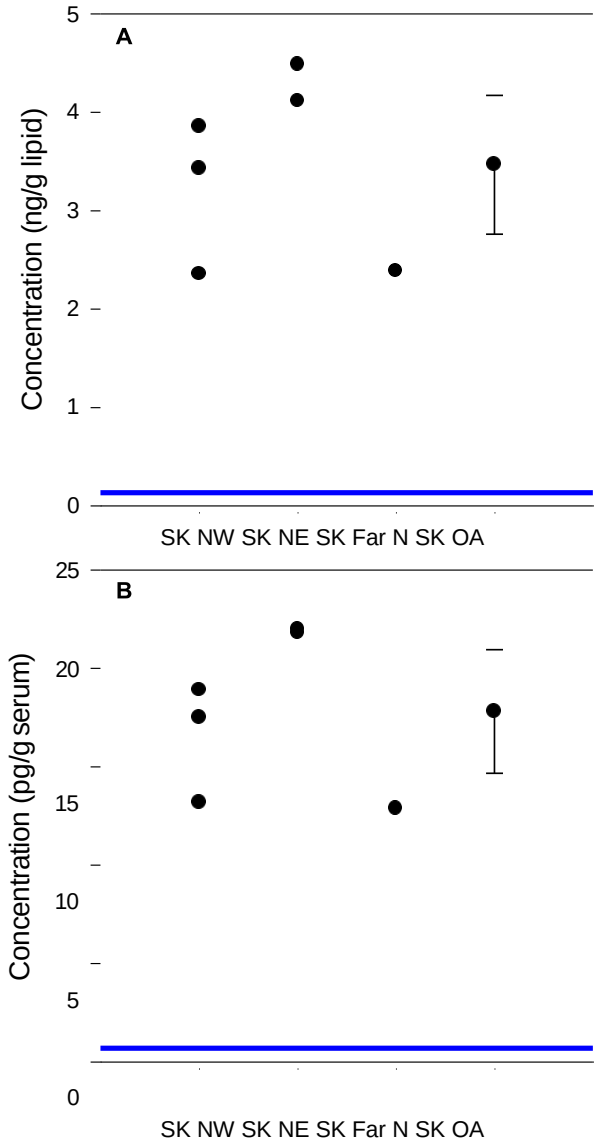

Figure 61: Concentrations of PCB congener 118 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

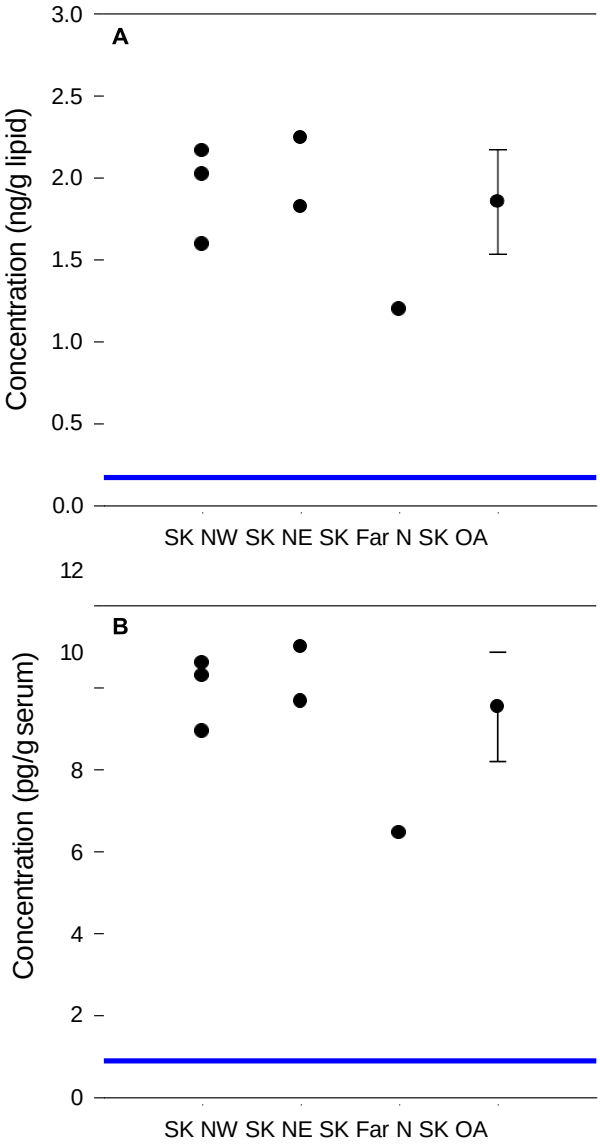

Figure 62: Concentrations of PCB congener 84/89 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

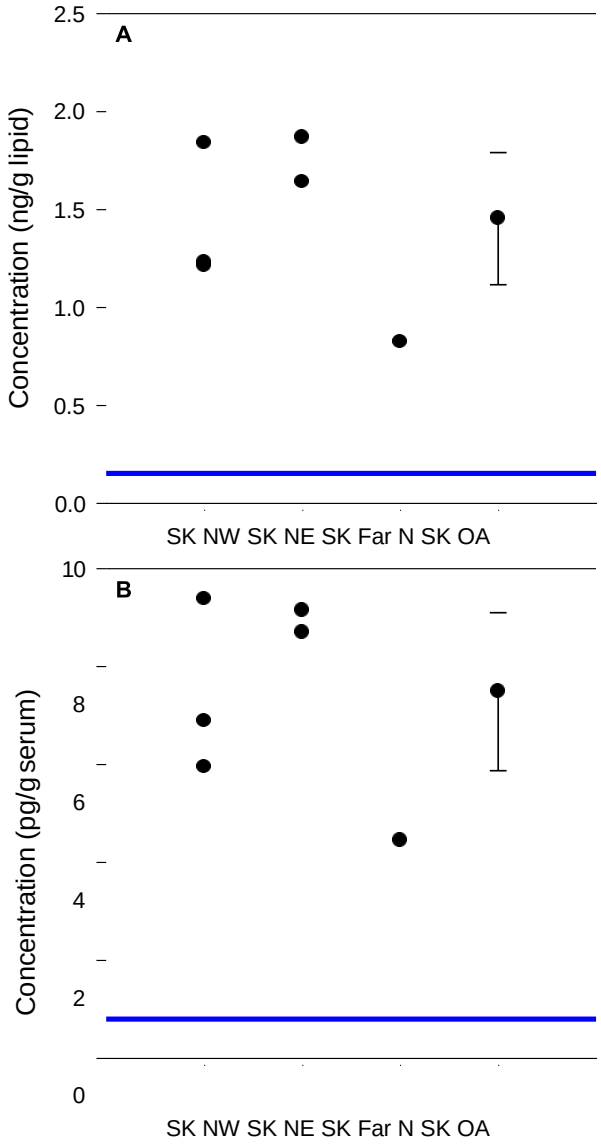

Figure 63: Concentrations of PCB congener 97 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

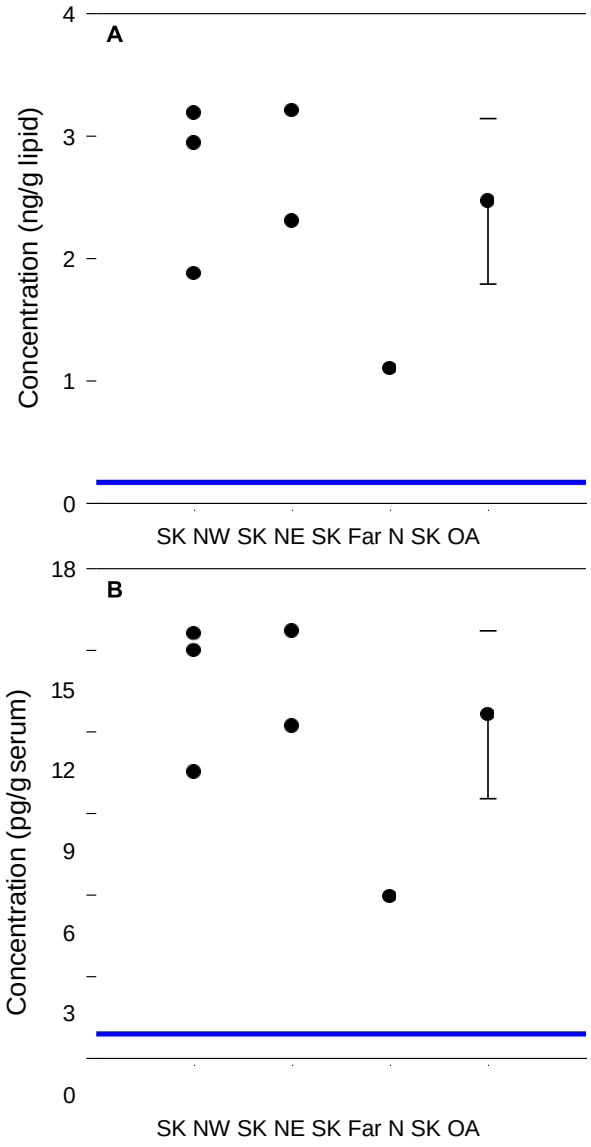

Figure 64: Concentrations of PCB congener 87 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

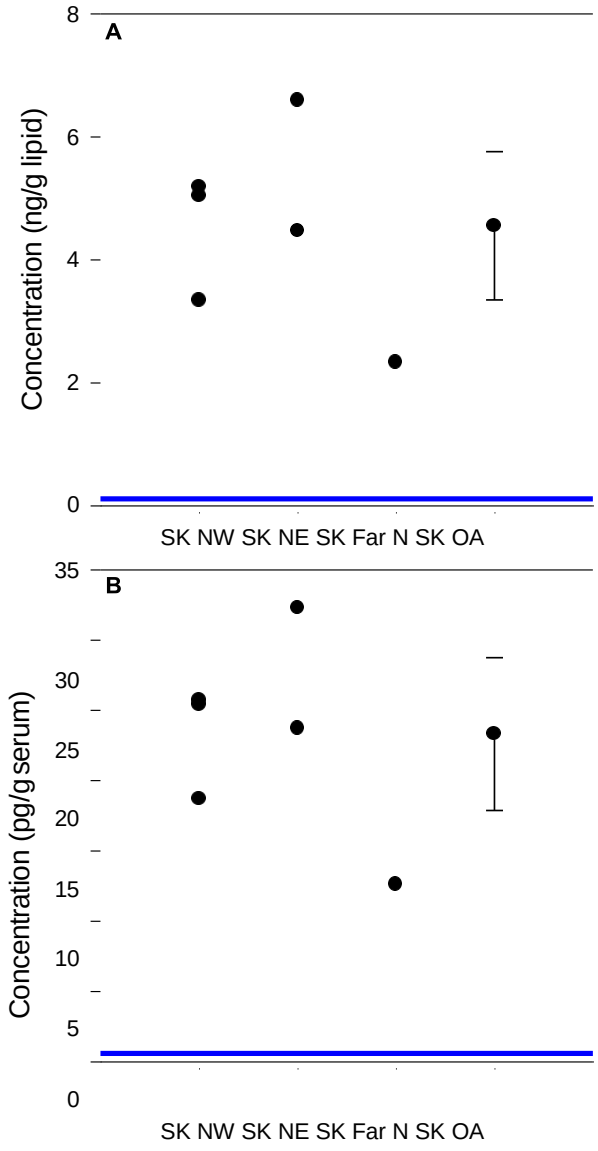

Figure 65: Concentrations of PCB congener 110 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the LOD used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

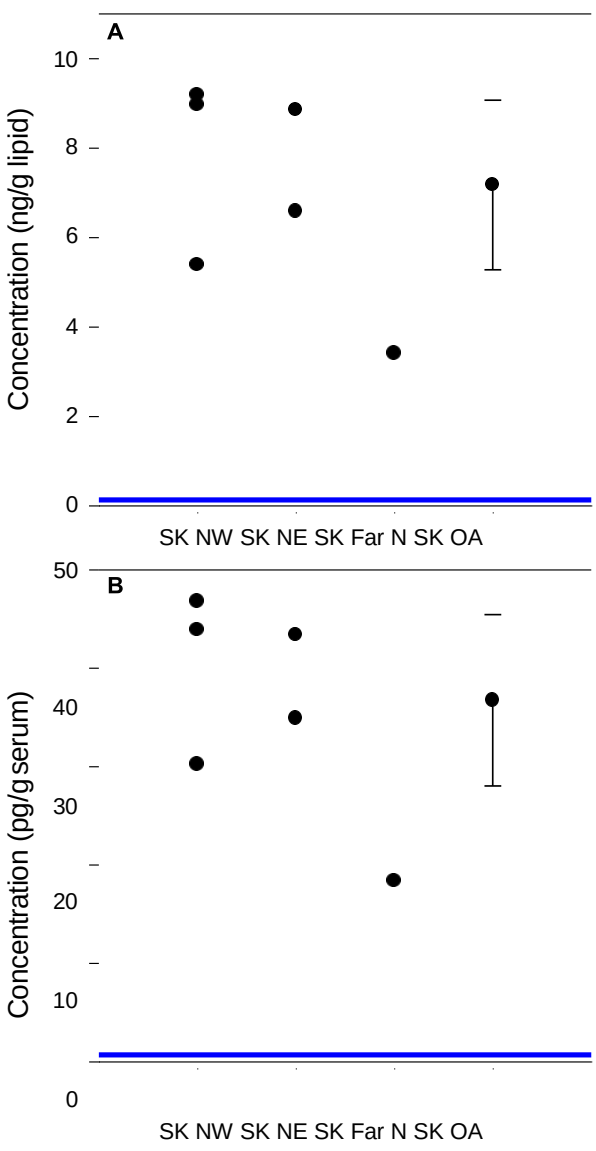

Figure 66: Concentrations of PCB congener 90/101 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

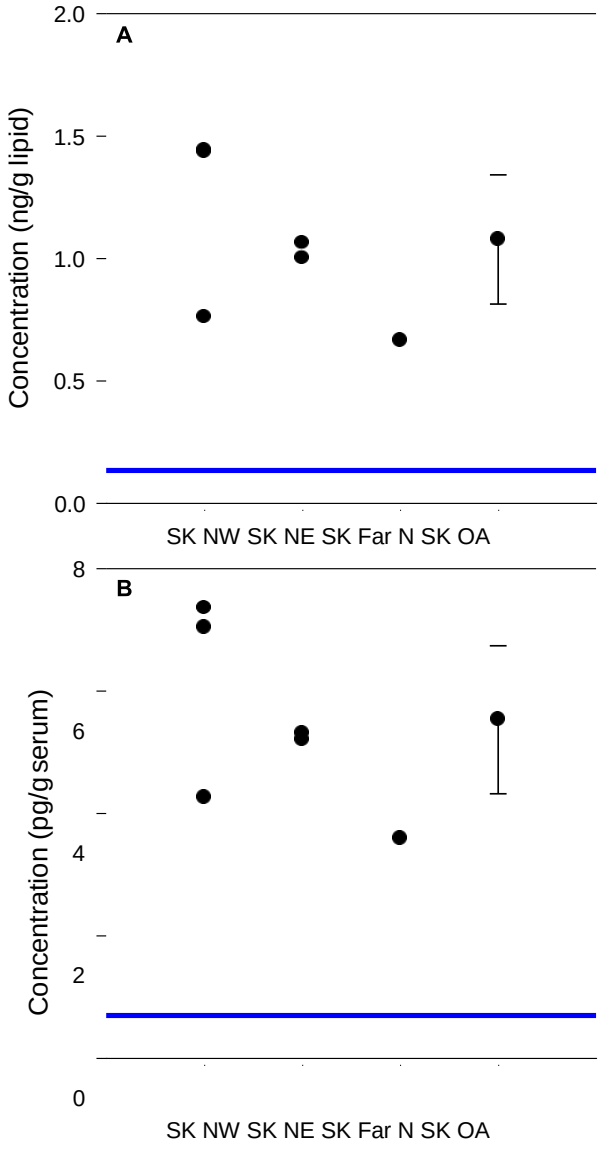

Figure 67: Concentrations of PCB congener 91 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

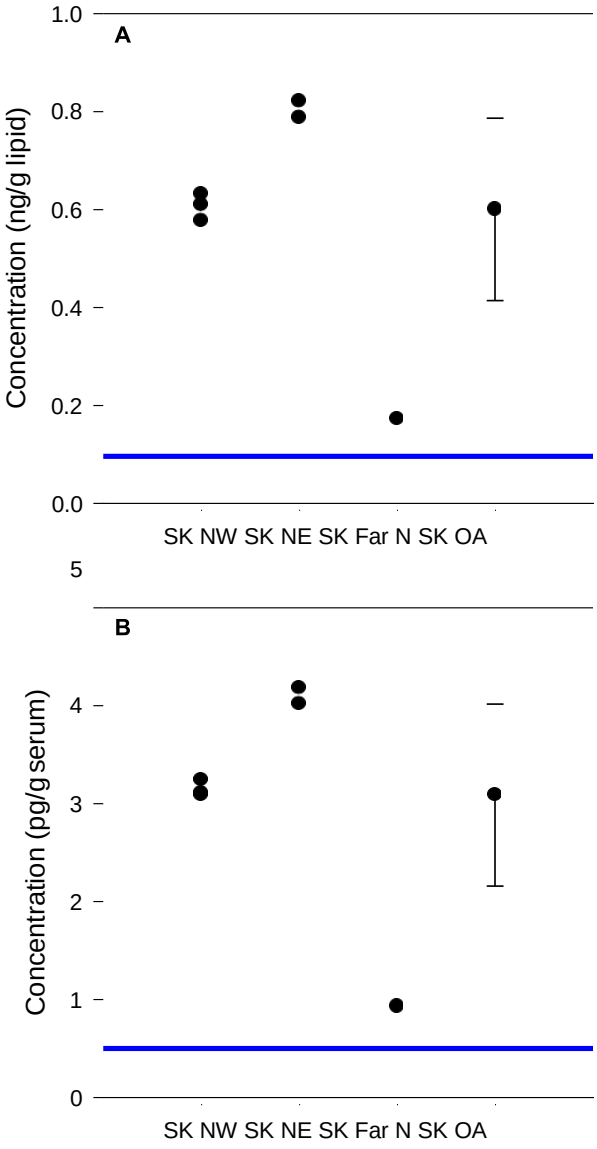

Figure 68: Concentrations of PCB congener 136 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

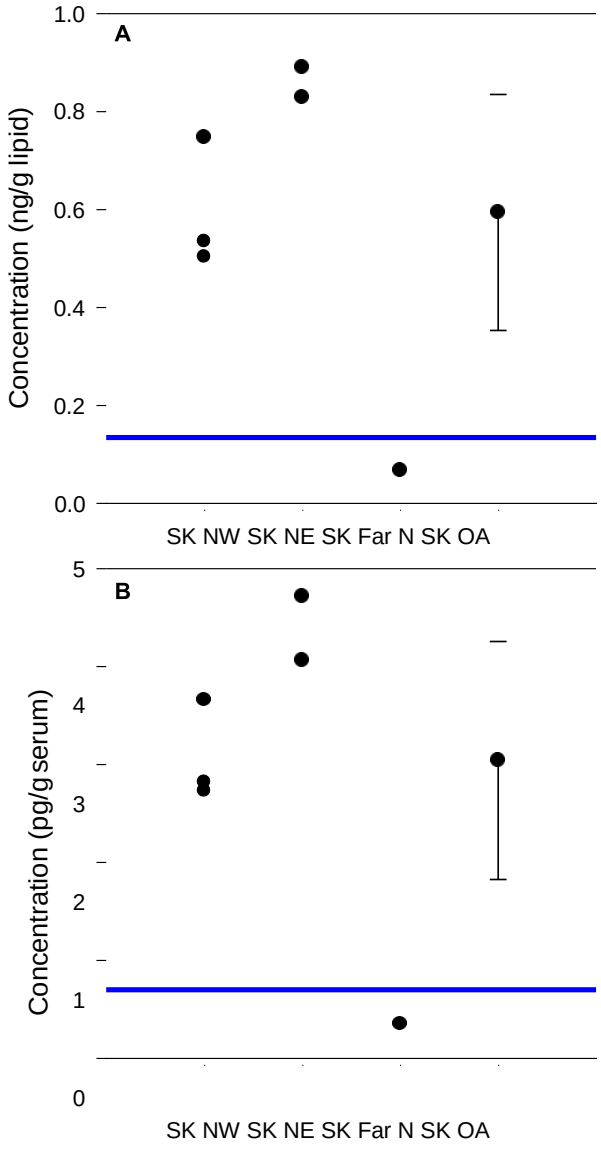

Figure 69: Concentrations of PCB congener 151 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

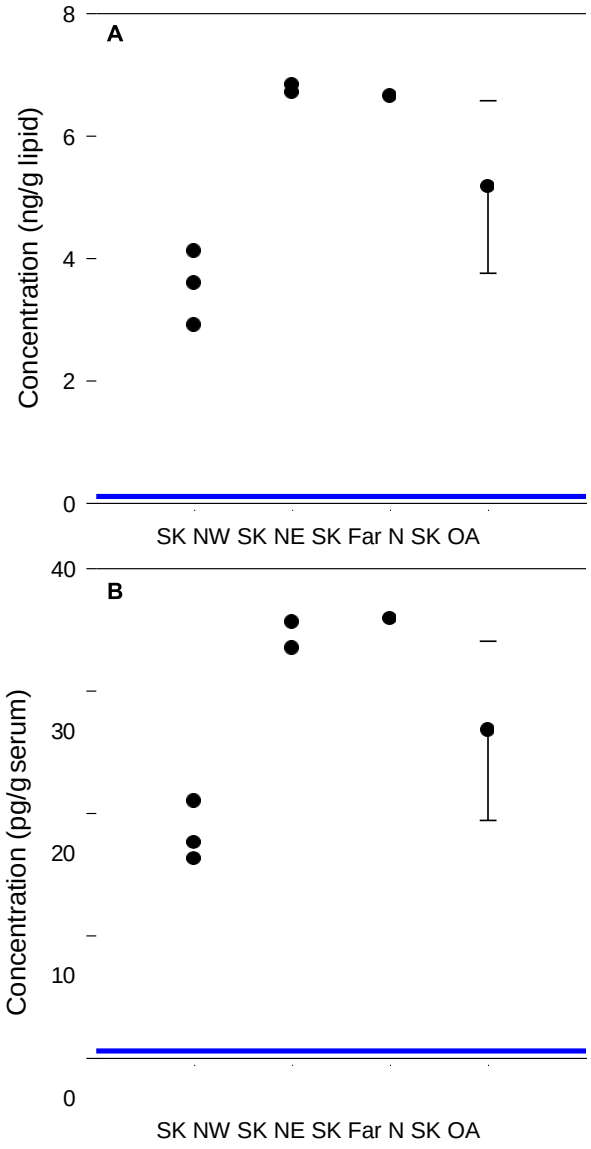

Figure 70: Concentrations of PCB congener 153/168 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

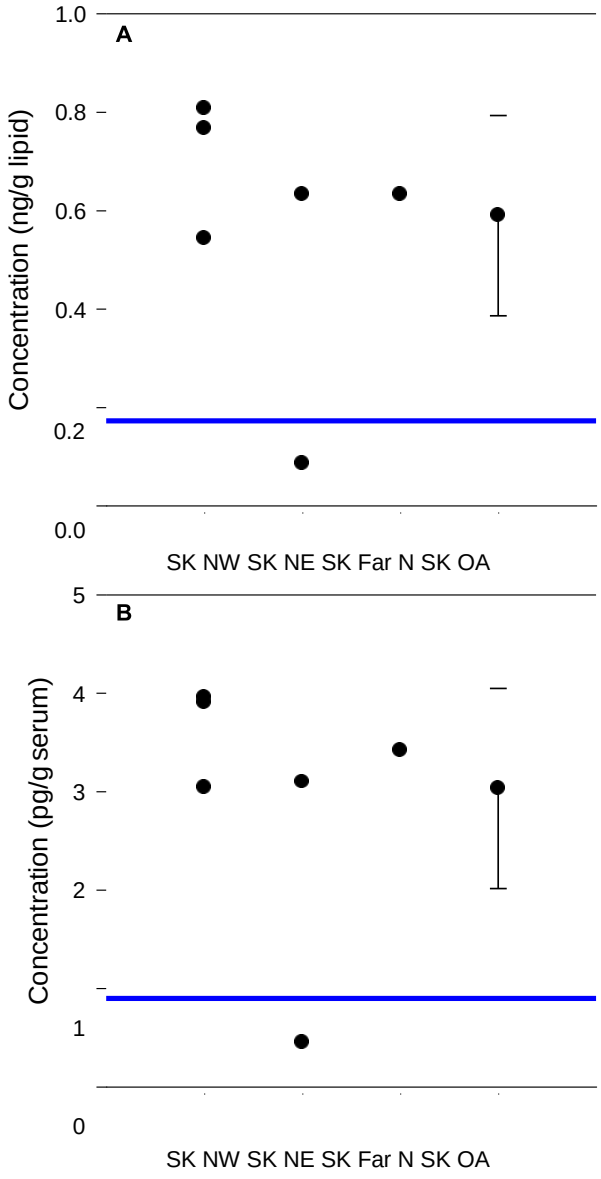

Figure 71: Concentrations of PCB congener 132 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

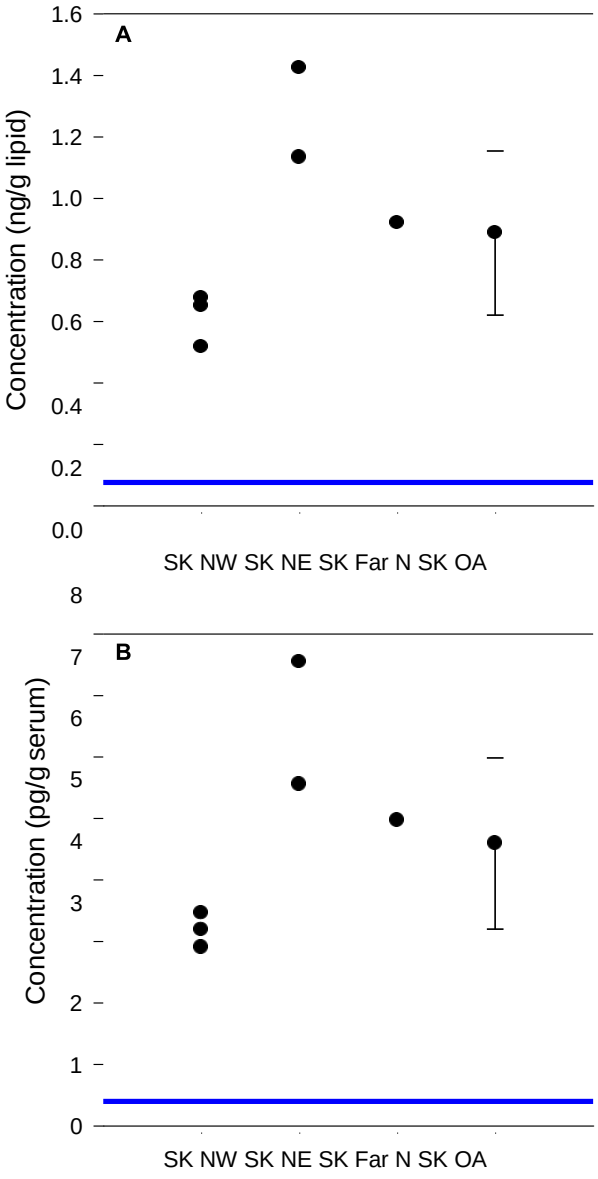

Figure 72: Concentrations of PCB congener 160/163 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

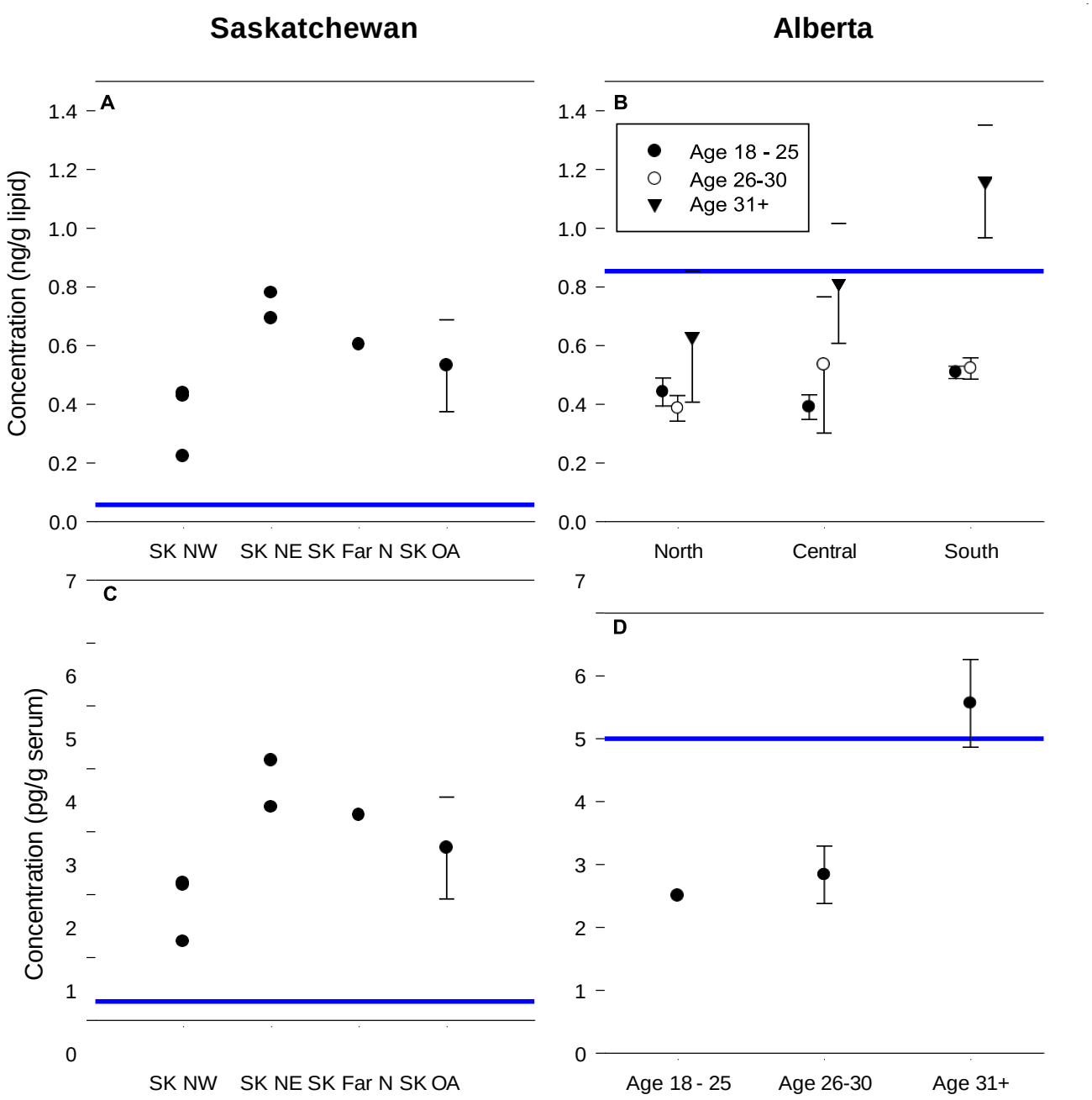

Figure 73: Concentrations of PCB congener 156 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Means are given by region and by age and region for lipid adjusted concentrations and by age group for serum adjusted concentrations in Alberta. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

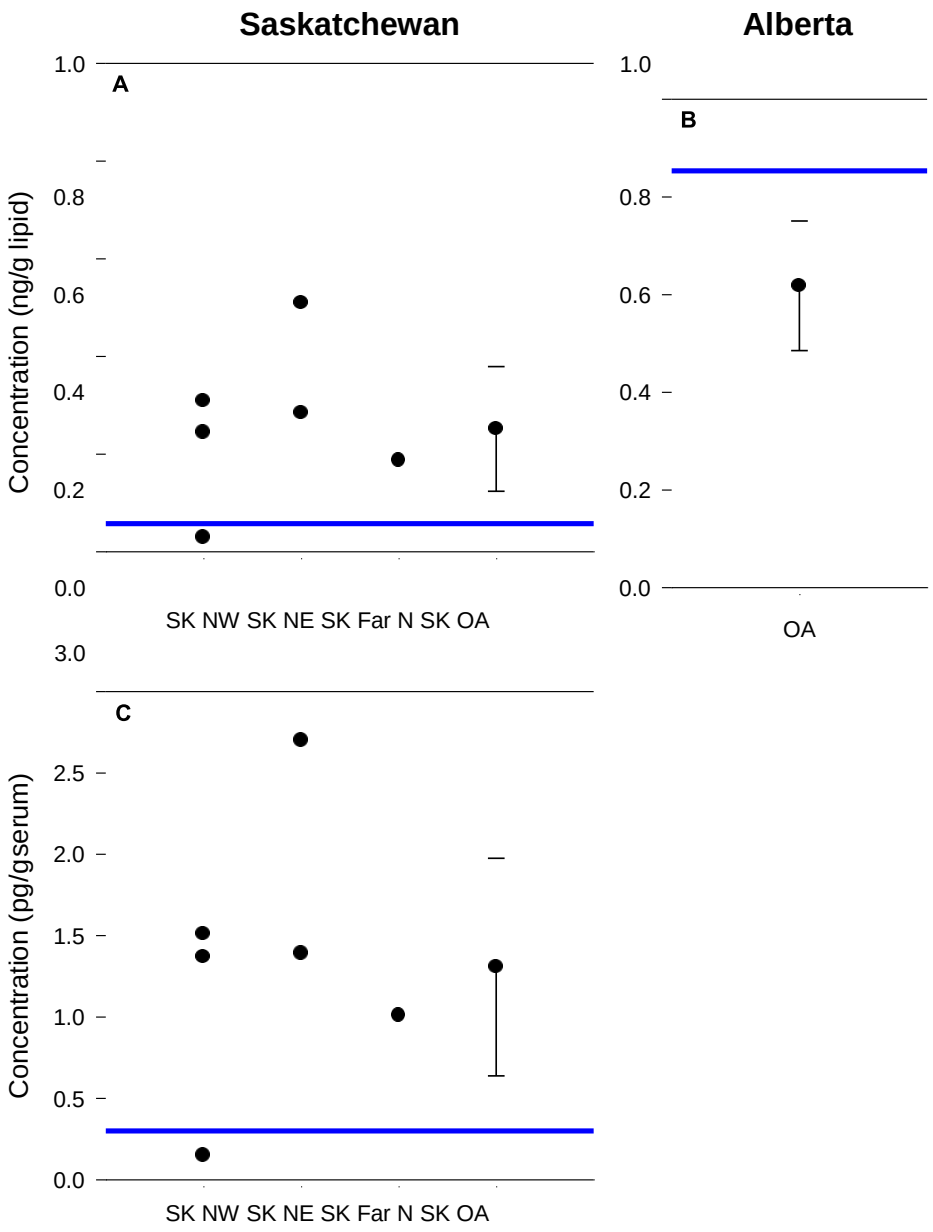

Figure 74: Concentrations of PCB congener 158/129 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. An overall (OA) concentration is provided for lipid adjusted data in Alberta. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

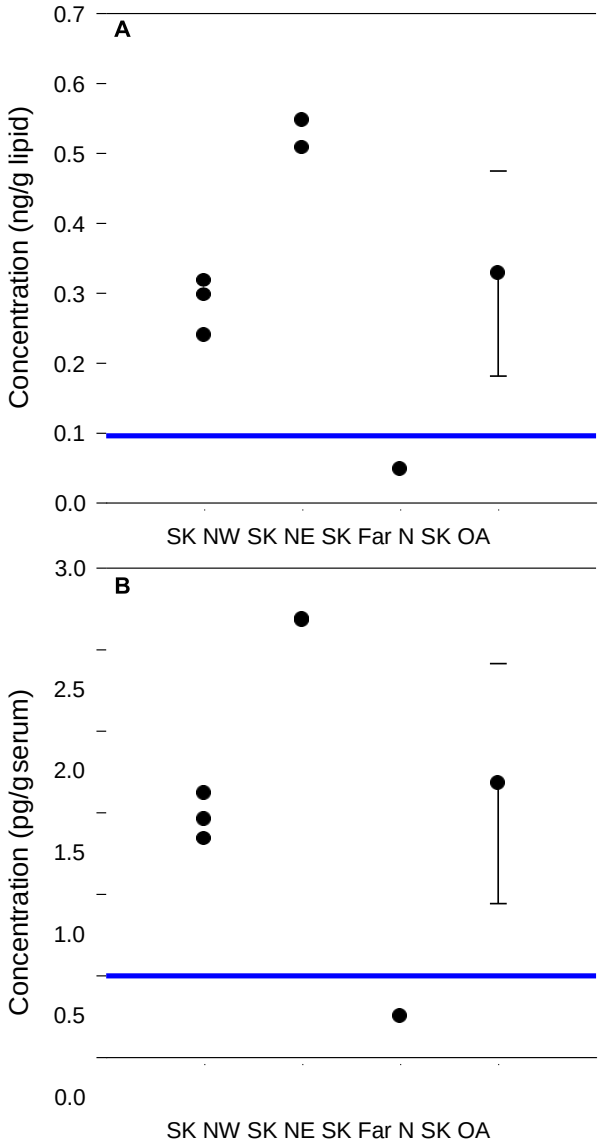

Figure 75: Concentrations of PCB congener 141 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

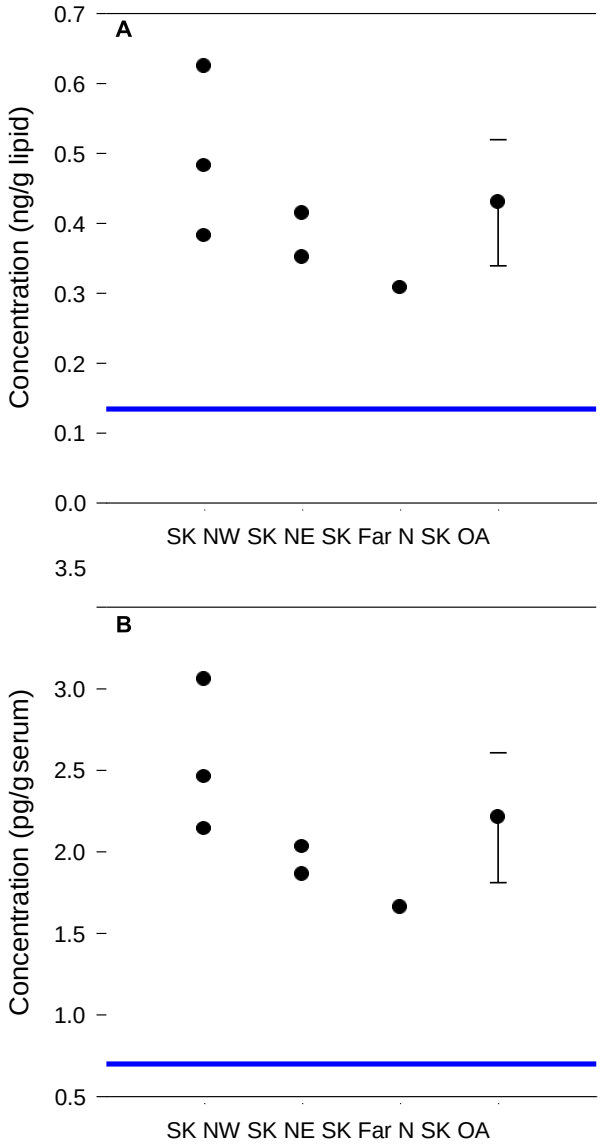

Figure 76: Concentrations of PCB congener 135 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

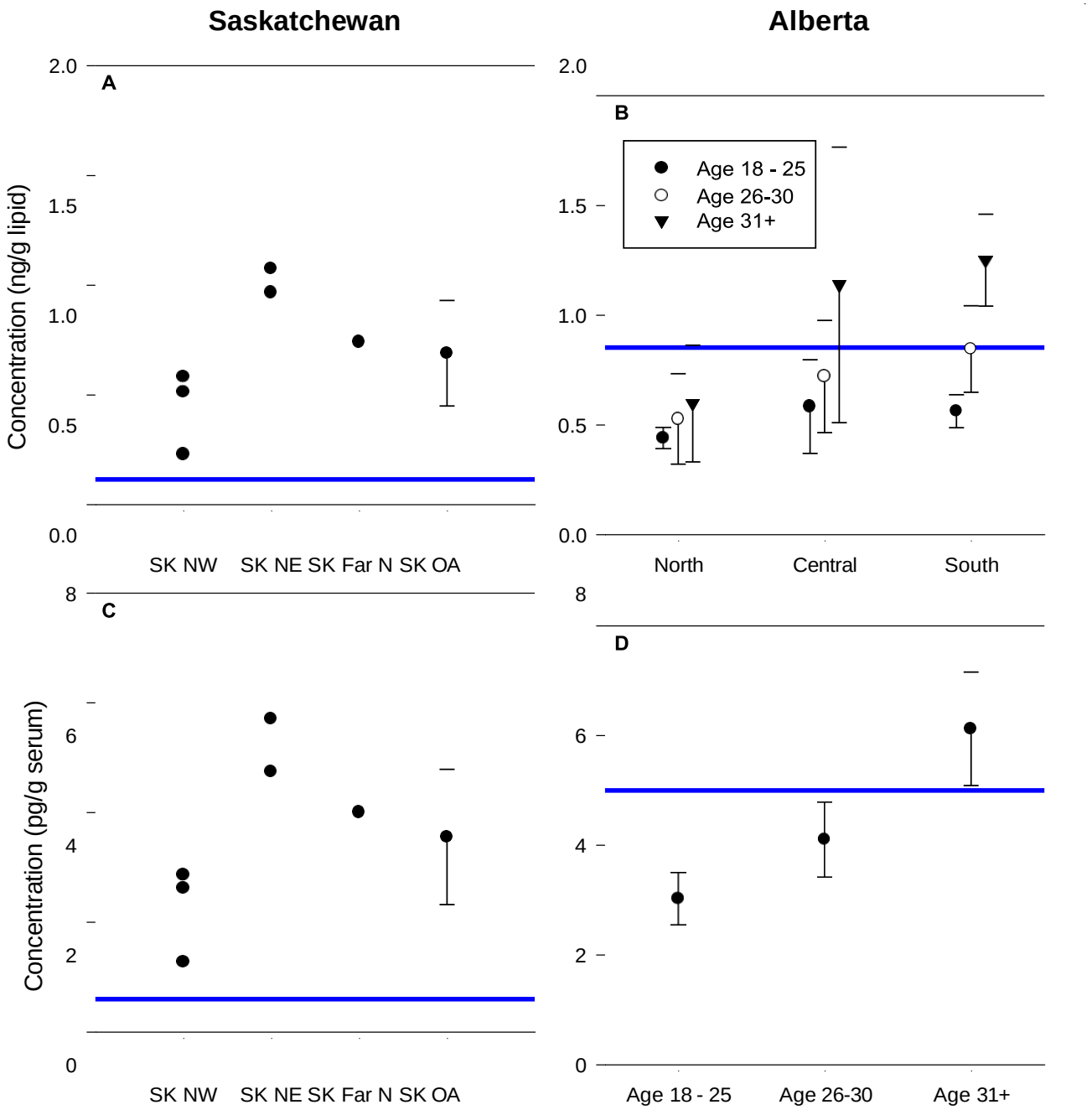

Figure 77: Concentrations of PCB congener 146 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Means are provided by age and region for lipid adjusted data, and by age for serum concentrations for the Alberta data. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

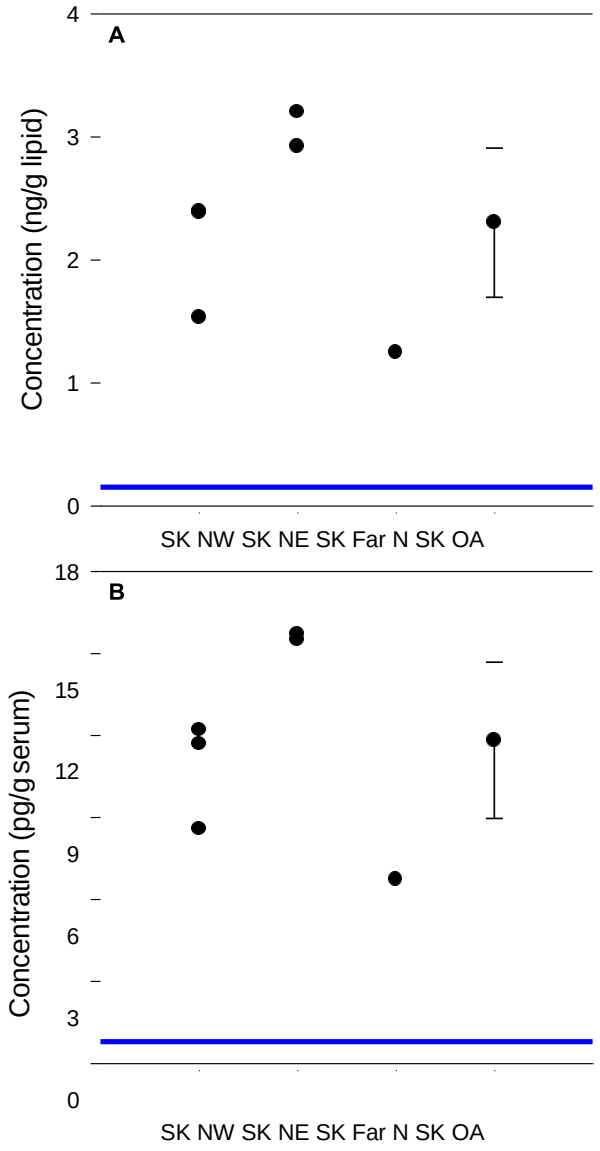

Figure 78: Concentrations of PCB congener 147/149 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

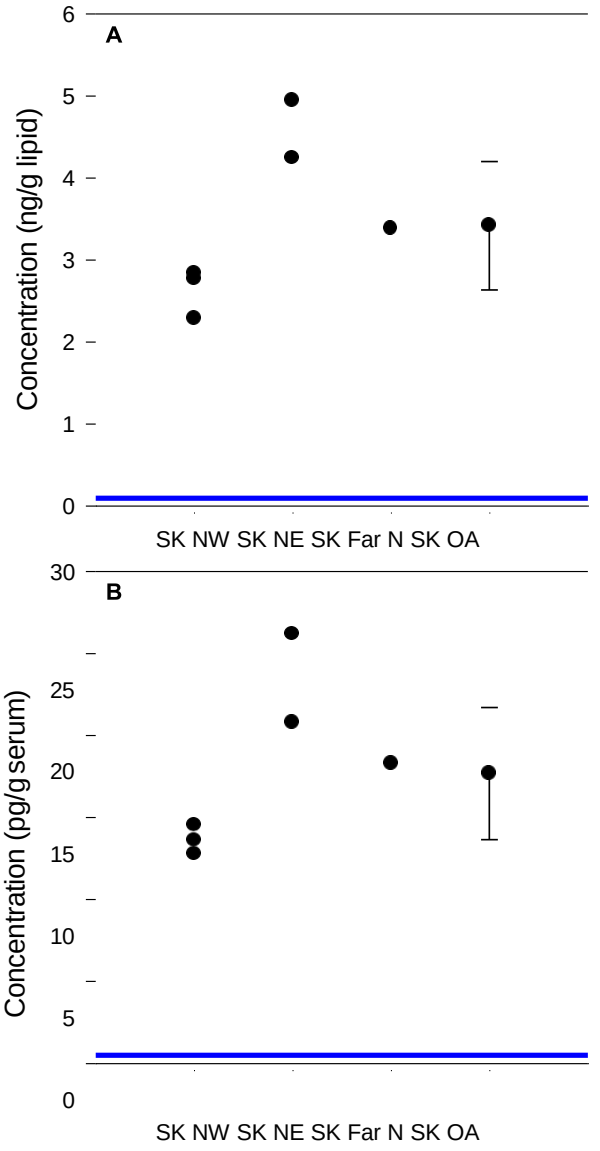

Figure 79: Concentrations of PCB congener 138 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

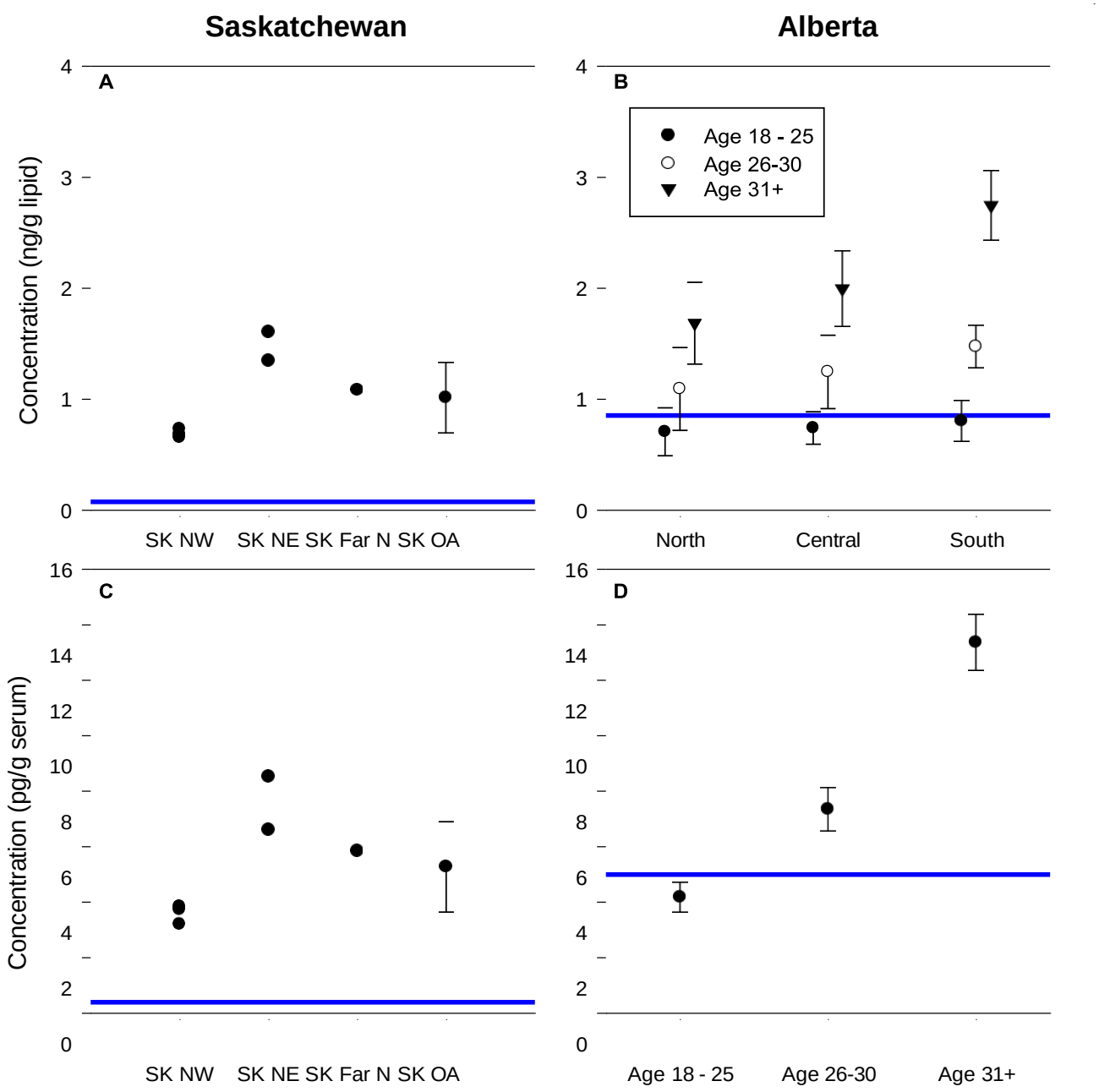

Figure 80: Concentrations of PCB congener 170 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The Alberta data is presented as means by age and region for lipid adjusted concentrations and by age for serum adjusted concentrations. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

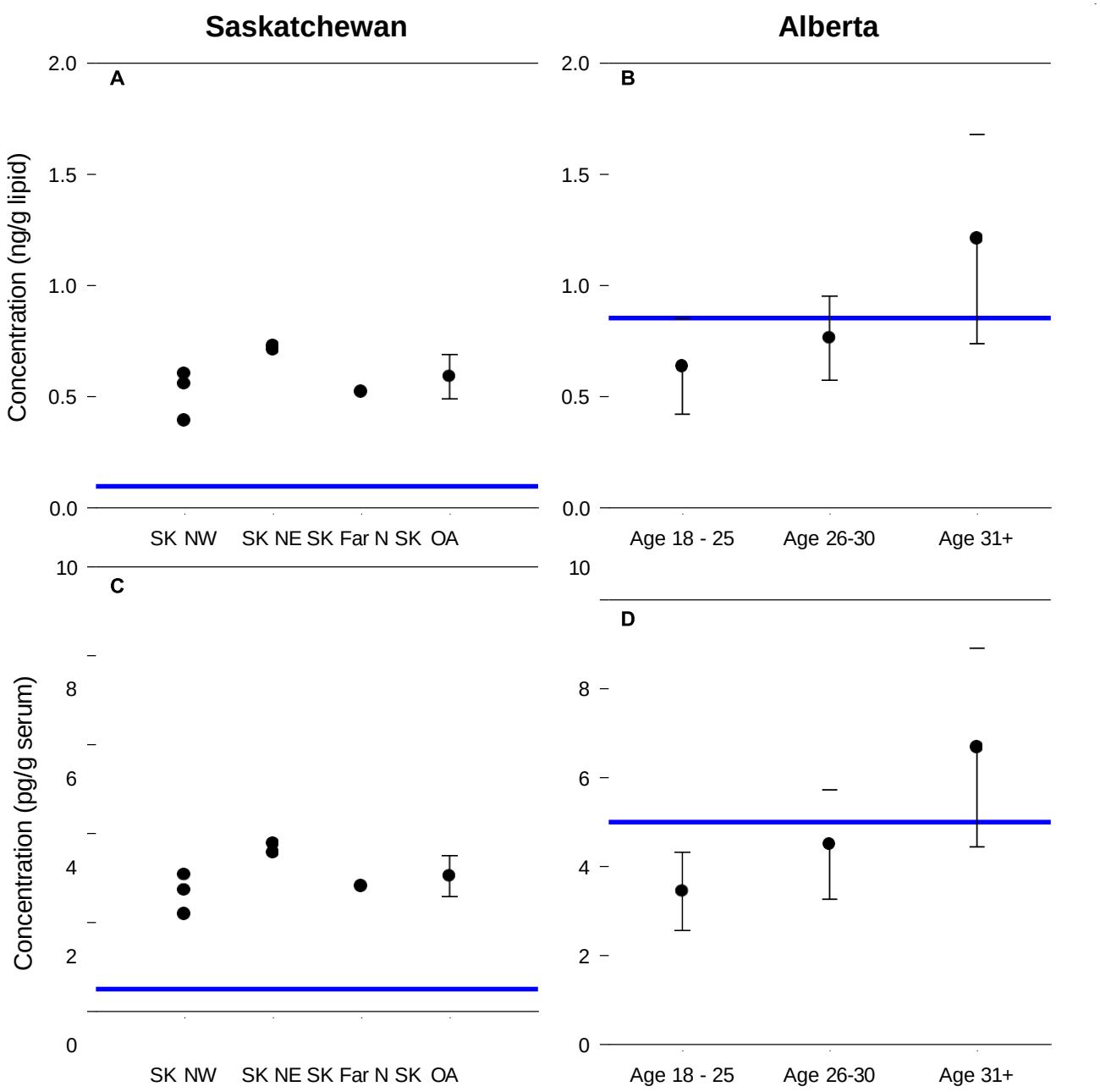

Figure 81: Concentrations of PCB congener 183 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The Alberta data is presented as means for each age group for both lipid and serum adjusted concentrations. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

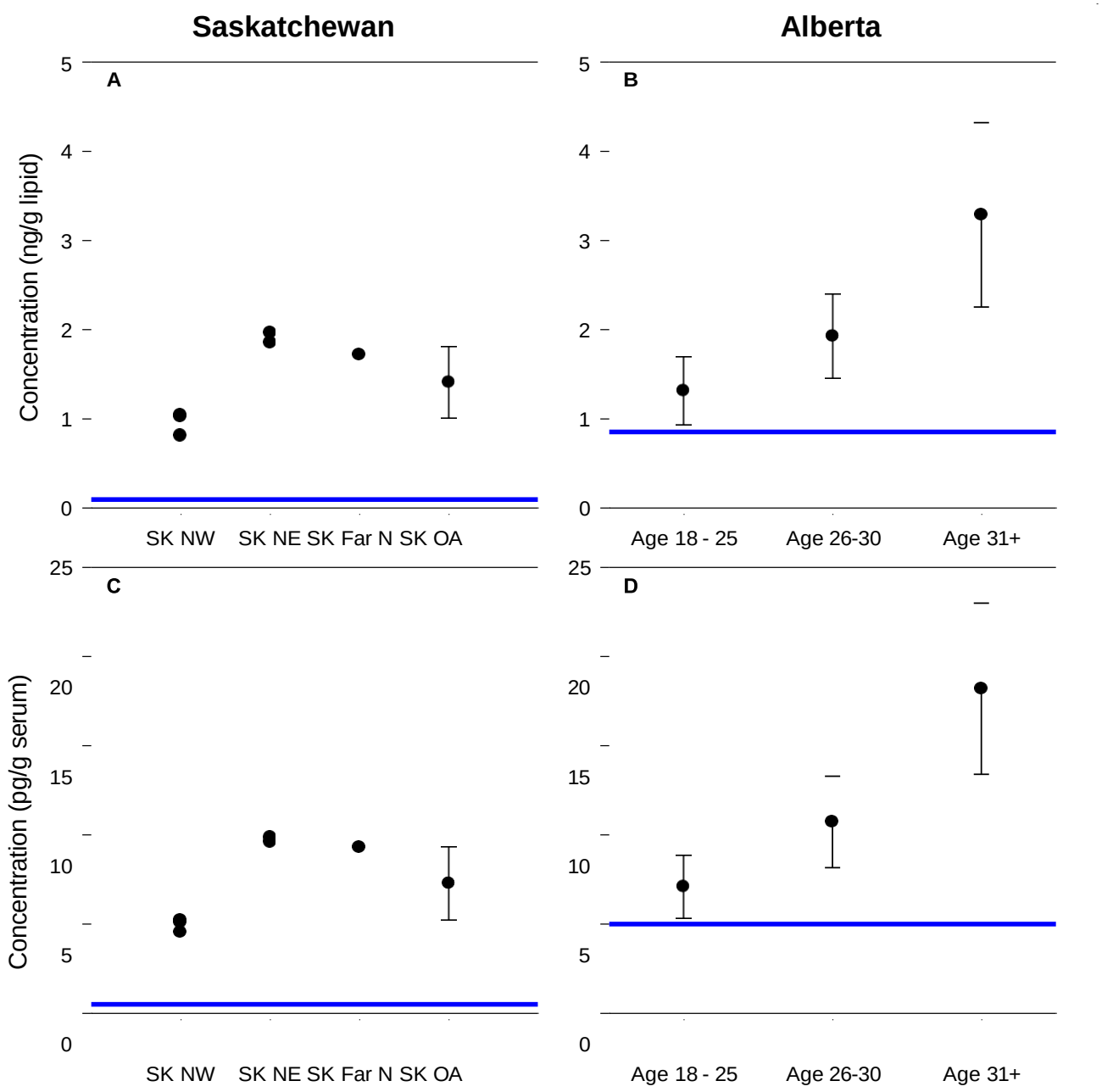

Figure 82: Concentrations of PCB congener 187 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is provided as means by age in both lipid and serum adjusted concentrations. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

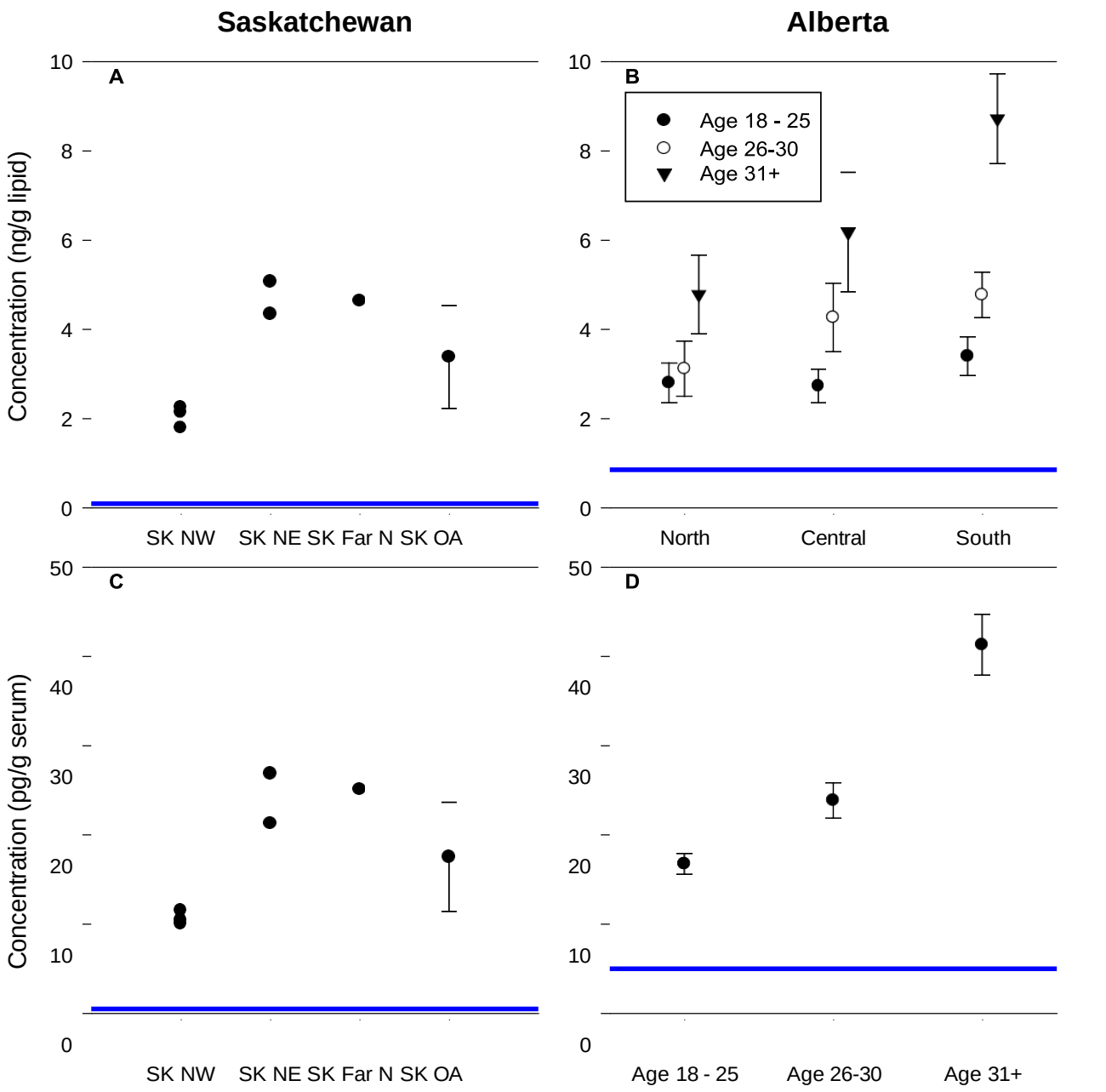

Figure 83: Concentrations of PCB congener 180 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is represented by means by age and region for lipid adjusted data, and by age for serum adjusted data. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

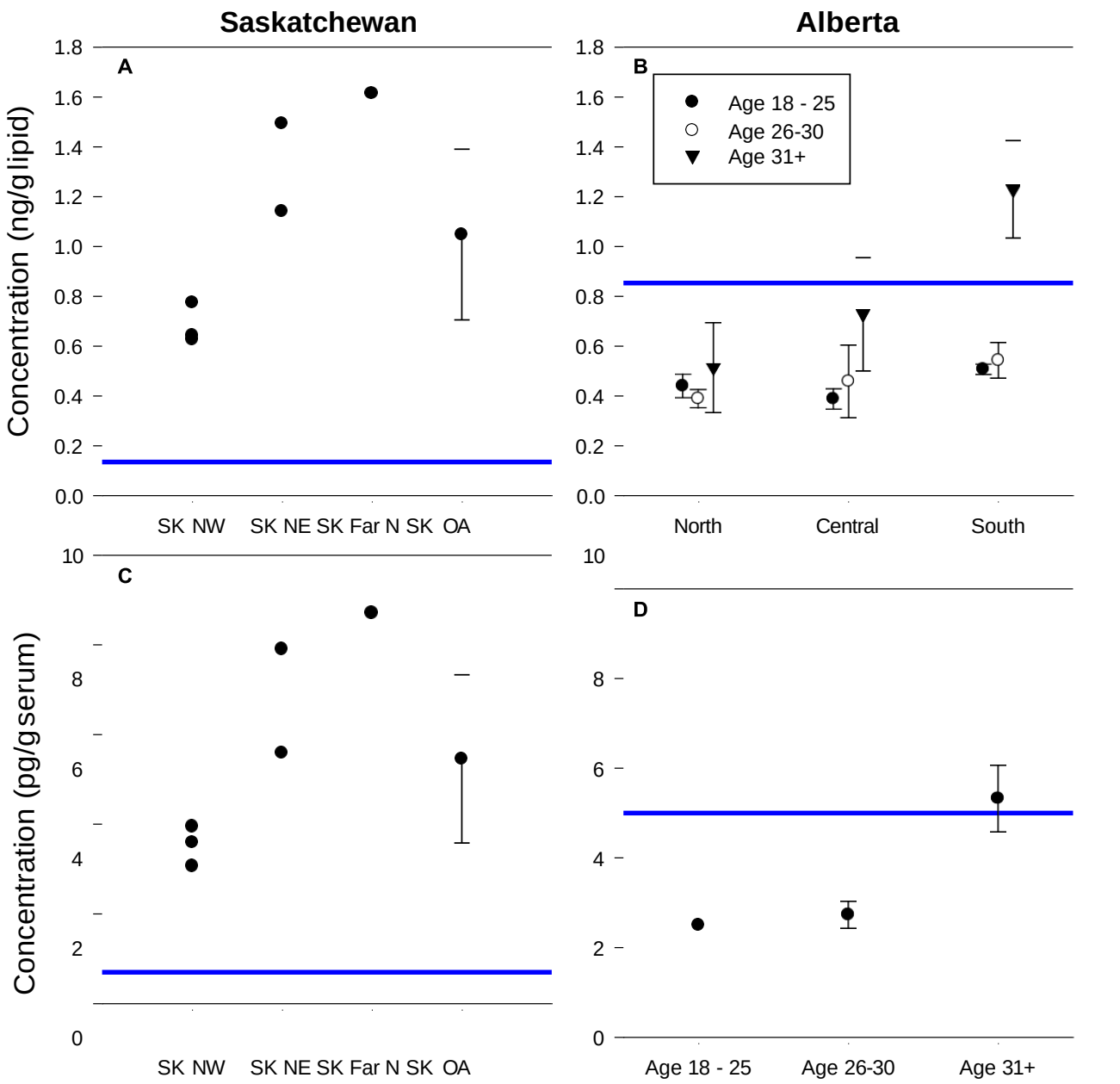

Figure 84: Concentrations of PCB congener 199 in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is represented by means by age and region for lipid adjusted data, and by age for serum adjusted data. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

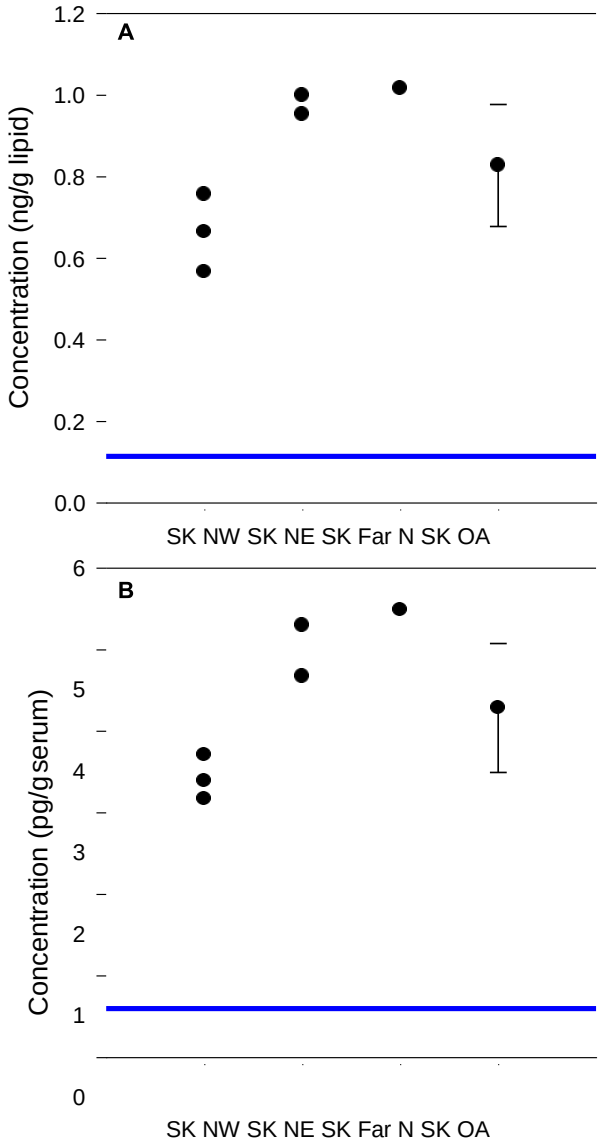

Figure 85: Concentrations of PCB congener 203/196 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

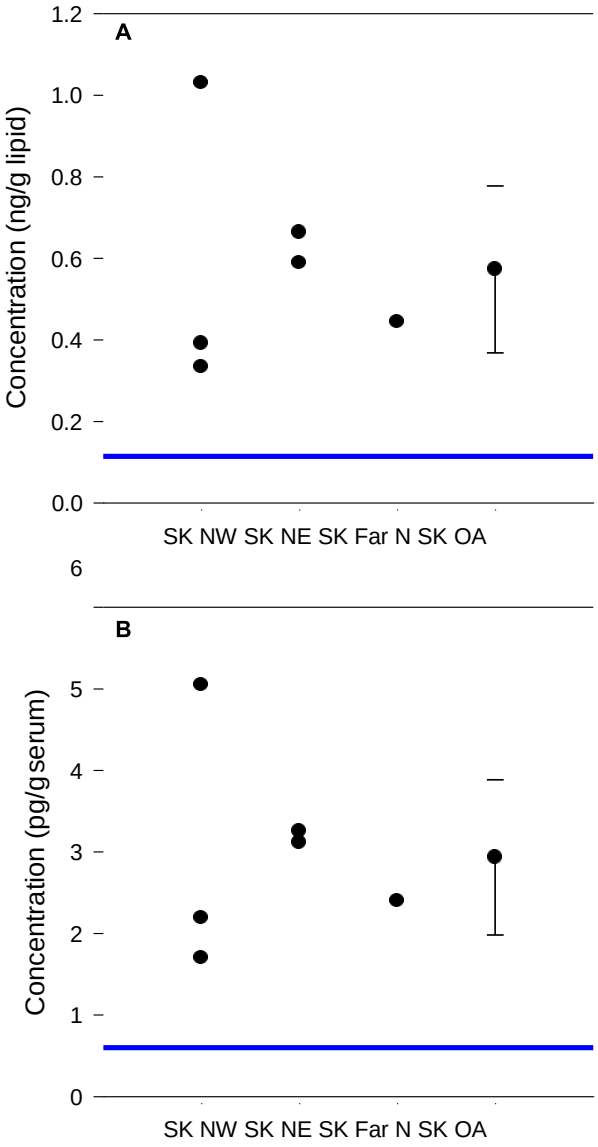

Figure 86: Concentrations of PCB congener 202 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

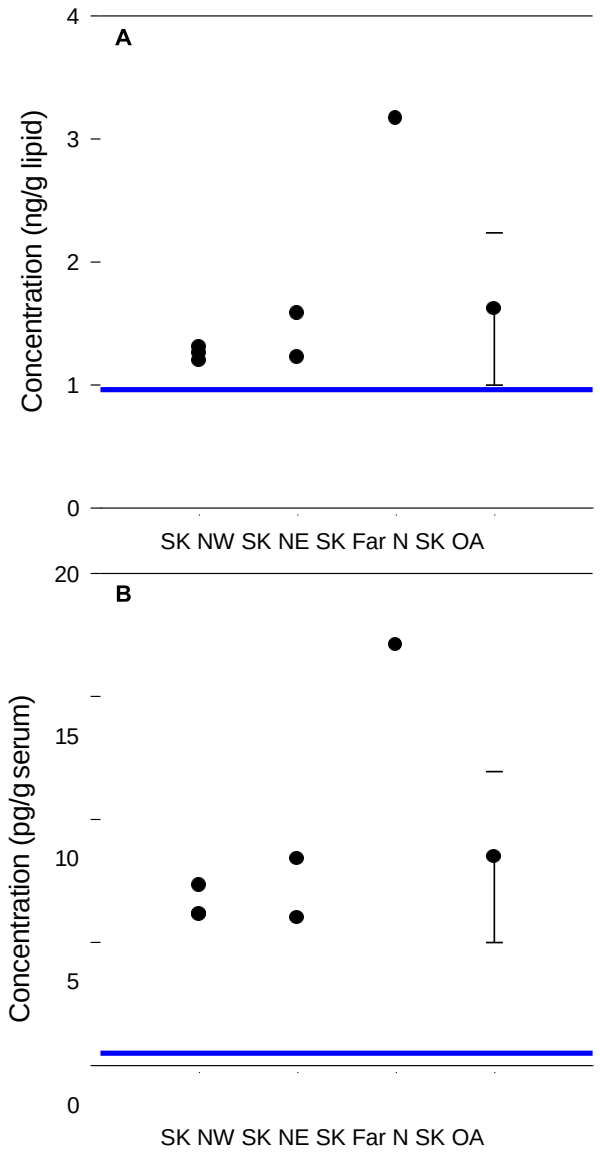

Figure 87: Concentrations of PCB congener 206 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

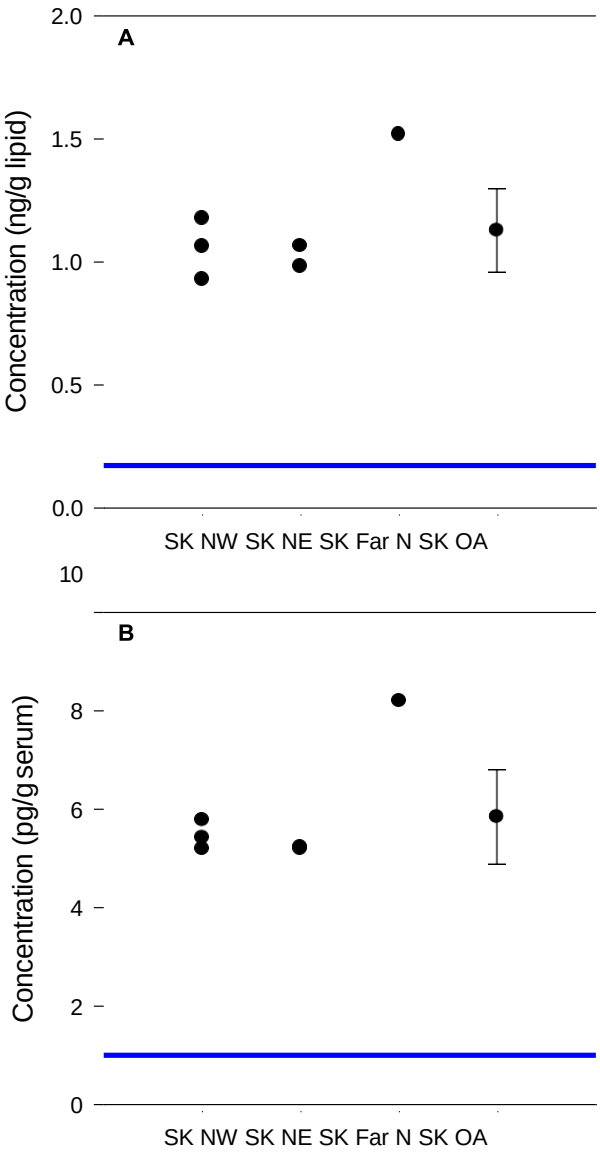

Figure 88: Concentrations of PCB congener 208 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

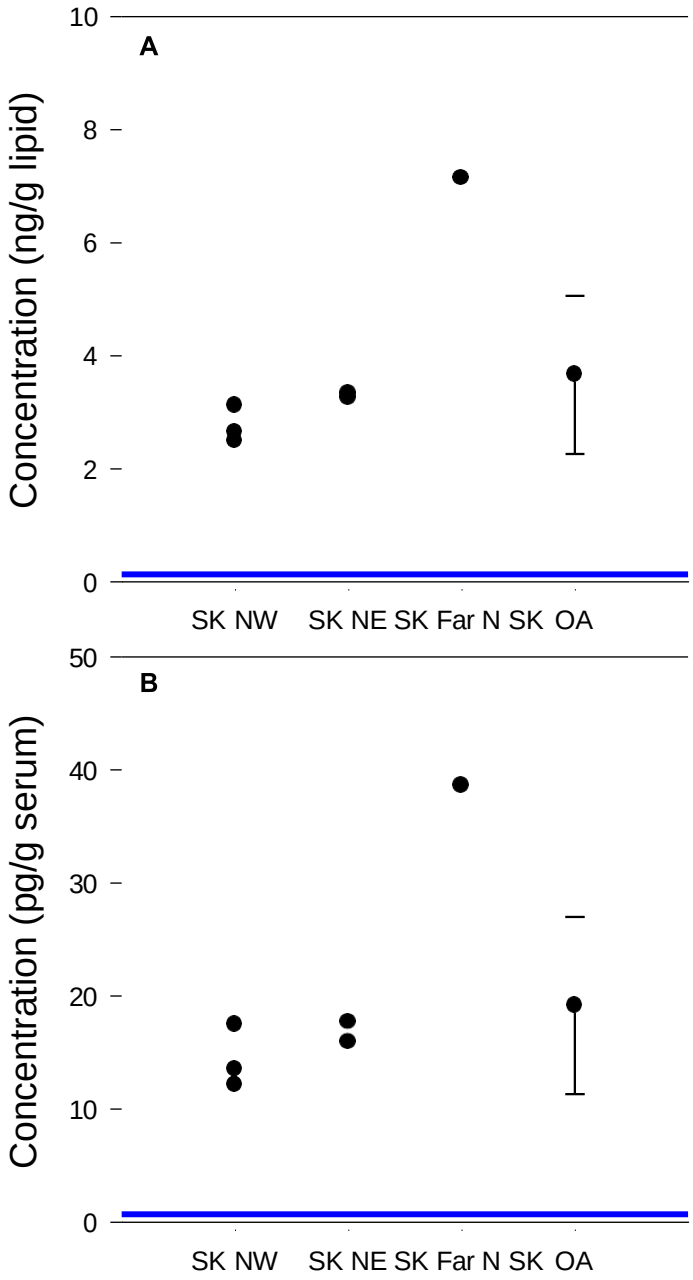

Figure 89: Concentrations of PCB congener 209 in the blood serum of pregnant women in Saskatchewan as represented by lipid adjusted concentration (A) and whole serum concentration (B). Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

ORGANOCHLORINE PESTICIDES

Organochlorine pesticides are a class of insecticides that were used to target a variety of insects starting in the 1940s and have since been restricted in terms of their use due to the fact that they are extremely persistent in the environment and have the potential to bio-accumulate (CDC, 2013b, 2013c & 2013r). While these chemicals were generally quite effective for their intended purposes, awareness of environmental, ecological and human health related issues arose during the 1960s which leading to the introduction of chemical regulations to minimize future environmental and human exposure (AWH, 2008). Many of these chemicals are no longer widely used in North America and are included in international agreements on persistent organic pollutants (POPs). However, some other countries continue to use these pesticides, as well they persist in the global environment due to their resistance to degradation. Organochlorine pesticides were released into the local environment (air, water and soil) after pesticide applications, during their manufacture, and disposal. Some organochlorines are volatile which means that they can partition into the atmosphere and travel to remote locations on wind currents.

Diet is the major route of exposure in the general population, particularly through the consumption of fish and dairy products (CDC, 2013b, 2013c & 2013r). They easily enter the foodchain due to their presence in soil and adherence to soil particles, and in aquatic ecosystems organochlorine pesticides adsorb to sediments and then bioaccumulate in fish and marine mammals. Organochlorine pesticides are lipophilic and may accumulate in the fatty tissues of animals such as livestock or in plants (AHW, 2008). People may also be exposed to organochlorine pesticides by drinking contaminated water or through inhalation of air. Infants may be exposed through breast milk and in utero as organochlorine pesticides can easily cross the placenta (Mueller et al., 2008; Lopez-Espinosa et al., 2007; Klopov, 1998). However, breastfeeding is encouraged due to the many associated health-benefits (American Academy of Pediatrics, 1997) that currently outweigh known risks.

In the present study, the following OC pesticides were measured in blood serum samples of pregnant women in northern Saskatchewan:

alpha-BHC	Oxychlordane	Endosulfan II	Hexachlorobenzene
beta-BHC	Aldrin	4,4’-DDD	Trans-nonachlor
delta-BHC	Heptachlor Epoxide	4,4’-DDT	Mirex
gamma-BHC (Lindane)	Dieldrin	Methoxychlor	2,4’-DDT
Octachlorostyrene	4,4’-DDE	alpha-Chlordane	
Heptachlor	Endrin	gamma-Chlordane	

Only endrin, 4,4-DDE, 4,4'-DDT, beta-BHC and hexachlorobenzene were detected in blood serum samples and not in all pools, and only 4,4-DDE was detected above detection limit in 6 pools and met the statistical inclusion criteria of this report. The concentrations of endrin that were detected above the LOD in 3 pools can be found in Table 1 of Appendix E. Likewise, both beta-BHC and 4,4-DDT were only detected in one pool above the detection limit and those results can be found in Table 1 of Appendix E.

Organochlorine pesticides are lipophilic chemicals and are found primarily within the lipid portion of the plasma or serum. Therefore their concentrations in the blood serum are typically made in reference to the lipid weight of the serum sample. This is done by dividing the total concentration of a lipophilic chemical in serum by the percent lipid content of blood. Comparison of results of chemical concentrations measured in difference matrixes cannot be directly done. In this report, both the measured serum chemical concentration and the calculated lipid level concentration, will be provided for reference and ease of comparison to other biomonitoring studies. Detailed information about the detected OCs can be found in the following sections. It is important to note that due to the number of samples in the pool and the signal-to-noise ratio of the analytical instrument the LOD differs by pool that for organochlorine pesticides. All of the organochlorine pesticides have the same LODs by pool, except for Methyoxychlor, as shown in Table 17:

Table 17: Limits of detection by pool for the organochlorine pesticides.

Compound		units	Pool 1	Pool 2	Pool 3	Pool 4	Pool 5	Pool 6
Methoxychlor	serum adjusted	ng/g serum	1.2	1.4	0.77	1.0	1.0	1.6
	lipid adjusted	ng/g lipid	2.3×10^2	2.7×10^2	1.5×10^2	2.0×10^2	2.0×10^2	3.0×10^2
All other tested OC pesticides	serum adjusted	ng/g serum	0.060	0.069	0.038	0.047	0.052	0.078
	lipid adjusted	ng/g lipid	12	13	7.4	9.0	10	15

DDT AND RELATED COMPOUNDS

GENERAL INFORMATION

Sources

In the 1940s, dichloro-diphenyl-trichloroethane (4,4'-DDT) was widely applied as a broad-spectrum insecticide and in controlling vectors of insect-borne human disease, mainly mosquitoes (malaria), midge, and lice (typhus). It was applied directly to soil or food crops, eventually entering surface water bodies via runoff. Technical grade mixtures of DDT may also contain DDD, a pesticide in its own right, and DDE (ATSDR, 2002a). As well, DDT may be broken down into DDE and DDD both in the environment and after being taken up by the body (ATSDR, 2002a). The 4,4'-DDE breakdown

product is the most stable, but has no applicable use (ATSDR, 2002a). DDT remains in limited use in some countries, primarily in Africa, to fight malaria. DDT and its breakdown products are stable under ambient environmental conditions with low solubility in water and high solubility in lipids. They preferentially accumulate on soil or aquatic sediments prior to tissue uptake by aquatic organisms (CCME, 1999). DDT is also subject to long range atmospheric transport leading to contamination of remote areas, such as Canada's far north, where DDT was never actually used (Aboriginal Affairs and Northern Development, 2010).

Possible routes of exposure to DDT and its derivative chemicals include through inhalation, ingestion of water or through contaminated food. Food is the most common route of exposure as DDT and its derivatives are extremely bio-accumulative and can be taken up and stored in aquatic animals from the water, as well as in fauna from their respective food sources (WHO, 2004f; CDC 2013k). It has been estimated that up to 90% of stored DDT found in humans was derived from food exposures. DDT and DDE are also measurable in human breast milk and can cross the placenta and thus exposures in utero and following birth are likely to be high (WHO 2004d, Mueller et al., 2008; Lopez-Espinosa et al., 2007; ATSDR 2002).

Regulations in Canada

In Canada, DDT was first registered for use in 1946 as an insecticide, but was never manufactured in the country. Import as an insecticide continued until the mid-1970s. As of 1985, these pesticides are no longer registered for use in Canada and have been identified as persistent organic pollutants to be targeted for elimination by the Stockholm Convention (Environment Canada 2013b). DDT is included on the *Toxic Substances List* in Schedule 1 of CEPA 1999 (Environment Canada, 2013b) and is a substance subject to notification or consent according to *Schedule 3* of Environment Canada's *Export Control List (*Environment Canada, 2013b). DDT is listed as a contaminant of health or environmental concern according to List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern of the Pest Control Products Act thereby suggesting that DDT meets the Toxic Substances Management Policy criteria as a track 1 substance which should be targeted for environmental elimination (Government of Canada, 2005). All storage of DDT was supposed to be sold or disposed of by December 31, 1990, and any uses of DDT after that time would be considered violations under this *Act*

Possible Health Effects

DDT is considered moderately toxic to humans and may affect both the hepatic and the nervous system (Aboriginal Affairs and Northern Development, 2010). The effects of DDT on children and the developing fetus are largely unknown (ATSDR, 2002a). Furthermore, DDE persists for a longer period of time in the body than DDT, and as such DDE may be a better indicator of historic exposure (CDC, 2013b). Background concentrations of DDE in humans usually are not known to cause any adverse health effects; however, at high doses, DDE may cause adverse health effects

including respiratory problems, impairment of the immune system and neurotoxicity (Chedrese and Feyles, 2001; Noakes et al., 2006; Charlier and Foidart, 2005; Eskenazi et al., 2006; Sunyer et al., 2005). The effects of DDT on children and developing fetuses are largely unknown (ATSDR, 2002a). However, animal studies suggest that DDT may affect growth, as well as development of the reproductive and nervous systems. The U.S. EPA has classified DDT, DDE and DDD as probable human carcinogens (ATSDR, 2002a).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

All 6 pools were detected above their respective LODs and 4,4'-DDE concentrations in blood serum of pregnant northern Saskatchewan women ranged from 0.11 ng/g to 0.68 ng/g (weighted arithmetic mean ± 95% confidence interval: 0.28 ng/g ± 0.17 ng/g) or, expressed on a serum lipid basis, from 19 ng/g to 1.4 x 10^2 ng/g lipid (weighted arithmetic mean ± 95% confidence interval: 54 ± 1.7 ng/g lipid). Maximum pooled concentrations were found in pool 2 (NW) and 4 (NE), at 1.4 x 10^2 ng/g lipid and 68 ng/g lipid, respectively.

The results from the Alberta biomonitoring program study conducted during 2005 reported concentrations from 0.11 ng/g to 1.5 ng/g of serum or, expressed on a serum lipid basis, from 12 to 2.1 x 10^2 ng/g lipid. The overall mean serum concentration in Saskatchewan is comparable to women of all ages in Northern Alberta and 18 to 25 year olds and 26 to 30 year olds in Central Alberta, and lower than serum concentrations found in other age groups and Southern Alberta (AHW, 2008). The overall mean lipid concentration in Saskatchewan is comparable to women above the age of 31 in Northern Alberta (mean ± 95% confidence interval: 56 ± 19 ng/g lipid). Younger women in Northern Alberta have mean serum concentrations lower than that of Saskatchewan and women in Central and Southern Alberta have serum blood concentrations of 4'4 DDE higher than that of women in Saskatchewan. In the U.S. National Health and Nutrition Examination Survey (NHANES, 2003-2004 Fourth Report) (CDC, 2009), the geometric mean of serum DDE concentrations in females were 241 ng/g of lipid and 1.5 ng/g of serum, and the 50^{th} percentile was reported as 207 ng/g lipid.

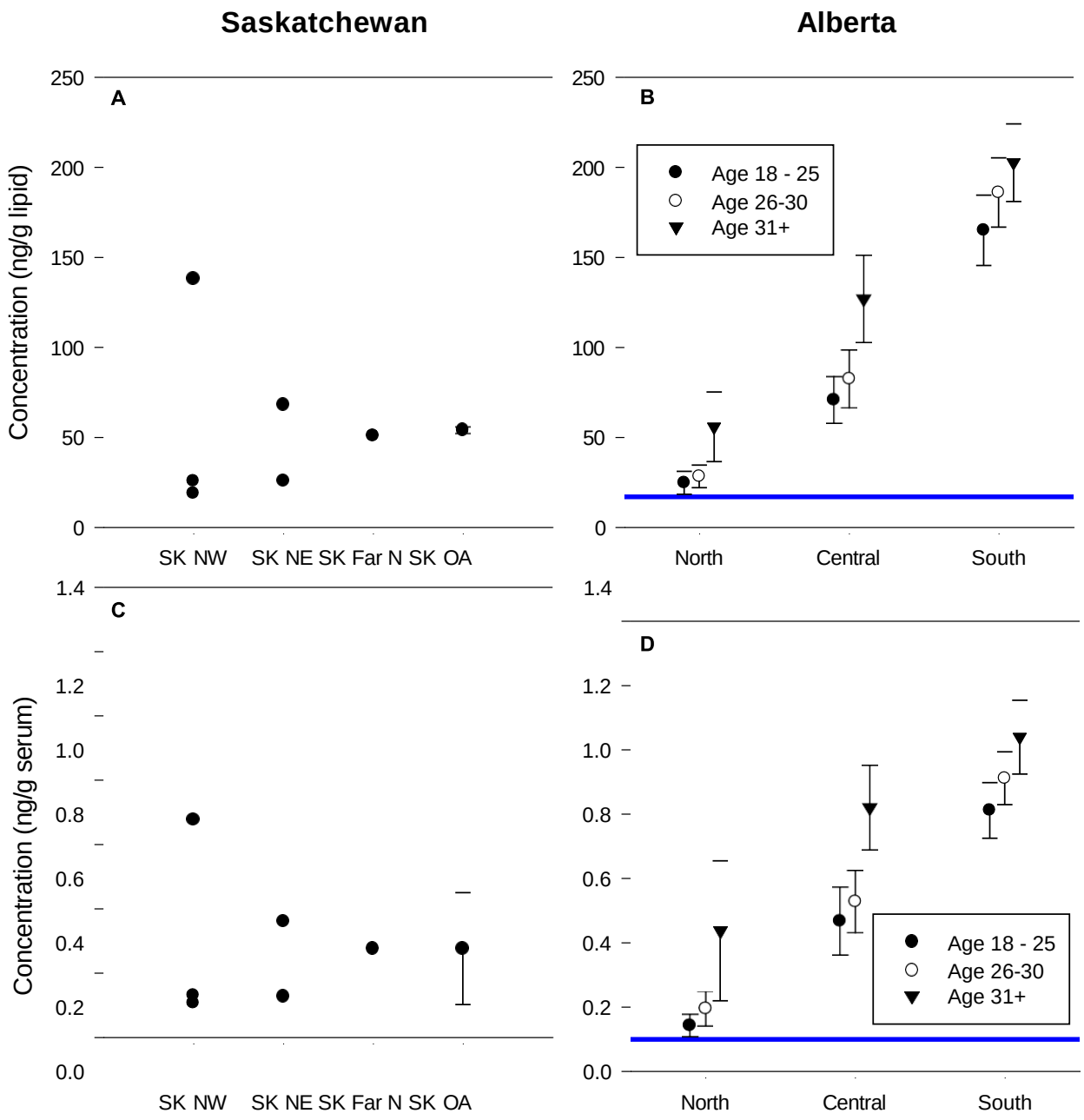

Figure 90: Concentrations of 4,4'-DDE in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is represented by mean concentrations by both region and age. The blue line represents the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean. For the Saskatchewan study, the LOD varied by pool. LODs by pool are given in Table 17. All of the Saskatchewan pools were detected above their respective limits of detection.

HEXACHLOROBENZENE

GENERAL INFORMATION

Sources

Hexachlorobenzene (HCB) is a lipophilic, synthetic organochlorine pesticide that was traditionally used as a seed treatment for plants such as wheat, rye, and barley to prevent fungus (Aylward et al 2010; Environment Canada, 2013g; WHO, 2004c). Its use as a fungicide in Canada began in the 1940s and continued through the 1970s; however, a result over concerns about its effects on human and environmental health, its agricultural uses began declining in many countries starting in the 1970s. (ATSDR, 2002b). While it is no longer produced intentionally in Canada and the United States, HCB can also be produced unintentionally as a by-product of manufacturing during combustion activities (Environment Canada, 2013g; Aylward et al., 2010). It was also previously used in fireworks, ammunition, synthetic rubber, wood preservative, and dielectric fluids (ATSDR, 2002b; Environment Canada, 2013a). HCB previously entered the environment through application to crops and disposal of industrial and commercial waste, and is still released as a by-product of manufacturing and use of chlorinated solvents and pesticides, emission from incinerators (incomplete combustion processes), and through long-range transport in air and water from other countries (ATSDR, 2002b; Jacoff et al., 1986; Dellinger et al., 1991). Hexachlorobenzene is resistant to degradation and can persist in the environment for long periods of time (WHO, 2004c). While it strongly adsorbs onto soil particles, it may leach over time providing constant input to the environment, despite the fact that it has not been applied to the environment for a long period of time. HCB can be found in groundwater, surface water and drinking water typically in the ppb to ppt range (ATSDR, 2013a). While in the water, it tends to adsorb onto particulate matter and sediment, and is capable of bioaccumulating in aquatic organisms. HCB can be carried long distances in the air, and has a half-life of 80 days in the troposphere as it is subject to slow photolysis (WHO, 2004c). Concentrations of HCB in the air are similar between urban, rural and remote locations which reflects its persistence and ability to be transported great distances.

Human exposure is generally a result of ingestion of food grown in contaminated soil or the ingestion of an animal product that has bioaccumulated HCB. It is poorly absorbed through the lungs and moderately absorbed from the gastrointestinal tract. Diet has been estimated to be the most important route of exposure, accounting for 92% of hexachlorobenzene exposure, as compared to drinking water and air at 1% and 7% respectively (WHO, 2004e). Hexachlorobenzene has been detected in food products, fish and breast milk (ATSDR, 2013a; Lopez-Espinosa et al., 2007; ATSDR, 2002b). Due to its ability to cross the placenta, human fetuses and infants are exposed through the placenta and breastfeeding.

Regulations in Canada

Hexachlorobenzene had its registration for use as a pesticide discontinued in 1976 in Canada and have been identified as a persistent organic pollutant to be targeted for elimination by the Stockholm Convention in 2004, as well as being prohibited by CEPA 1999 (Environment Canada 2013g). Hexachlorobenzene is included on Priority Substance List 1 under Priority Substances Assessment Program (Environment Canada, 1989). Hexachlorobenzene is listed as a contaminant of health or environmental concern according to List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern of the Pest Control Products Act (Government of Canada, 2005). Furthermore, in 1994 HCB was added to the List of Toxic Substances in Schedule 1 of CEPA, and it has been targeted for a virtual elimination from Canada (Canada Gazette, 2003). Regulations have been in place for HCB since 2003. With the publication of the Prohibition of Certain Toxic Substances Regulations, 2012, Canada has strengthened its controls for HCB in order to ensure continued compliance with international obligations (Canada Gazette, 2003).

Possible Health Effects

Background concentrations of HCB in humans are not known to cause any adverse health effects. Effects of HCB exposure include toxicity of the endocrine system, immunological system and nervous system causing as tremors and convulsions, as well as liver disease, a decrease in the body's ability to produce heme in the blood which is part of the oxygen carrying protein in hemoglobin and skin discoloration (ATSDR, June 2013; ATSDR, 2002b; Gocmen et al., 1989; Euriquez de Salamanca et al., 1990). Human and animal studies suggest that HCB exposure during development and early life may lead to neurological impairment and reduces viability and growth of newborns (ASTDR, June 2013). Due to evidence from animal studies, the U.S. EPA has classified HCB as a probable human carcinogen and IARC has labelled it as possibly carcinogenic to humans (ATSDR, June 2013).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

With the exception of pool 5 (NE), HCB was detected in all sample pools from pregnant women in northern Saskatchewan with concentrations in serum ranging from 0.042 ng/g to 0.35 ng/g (weighted arithmetic mean ± 95% confidence interval: 0.13 ng/g ± 0.094 ng/g) or, expressed as lipid serum weight, 7.5 ng/g to 71 ng/g lipid (weighted arithmetic mean ± 95% confidence interval: 25 ± 19 ng/g lipid). Concentrations of HCB in pool 2, comprised of samples from the northwest, are elevated in comparison to other pools taken from the northwest and the pools from other geographical regions. In comparison, concentrations in blood serum of pregnant Albertan women (AHW, 2008) ranged from 0.050 to 0.39 ng/g of serum or, expressed on a serum lipid basis, from 10 to 65 ng/g lipid (mean ± 95% confidence interval: 0.16 ± 0.013 ng/g; 28 ± 2.4 ng/g lipid).

The U.S. National Health and Nutrition Examination Survey (NHANES, 2003-2004 Fourth Report) (CDC, 2009) reported a geometric mean for females as 15.8 ng/g in lipid, slightly lower than pregnant northern Saskatchewan women sampled in this study; however this is comparing a geometric mean with an arithmetic mean.

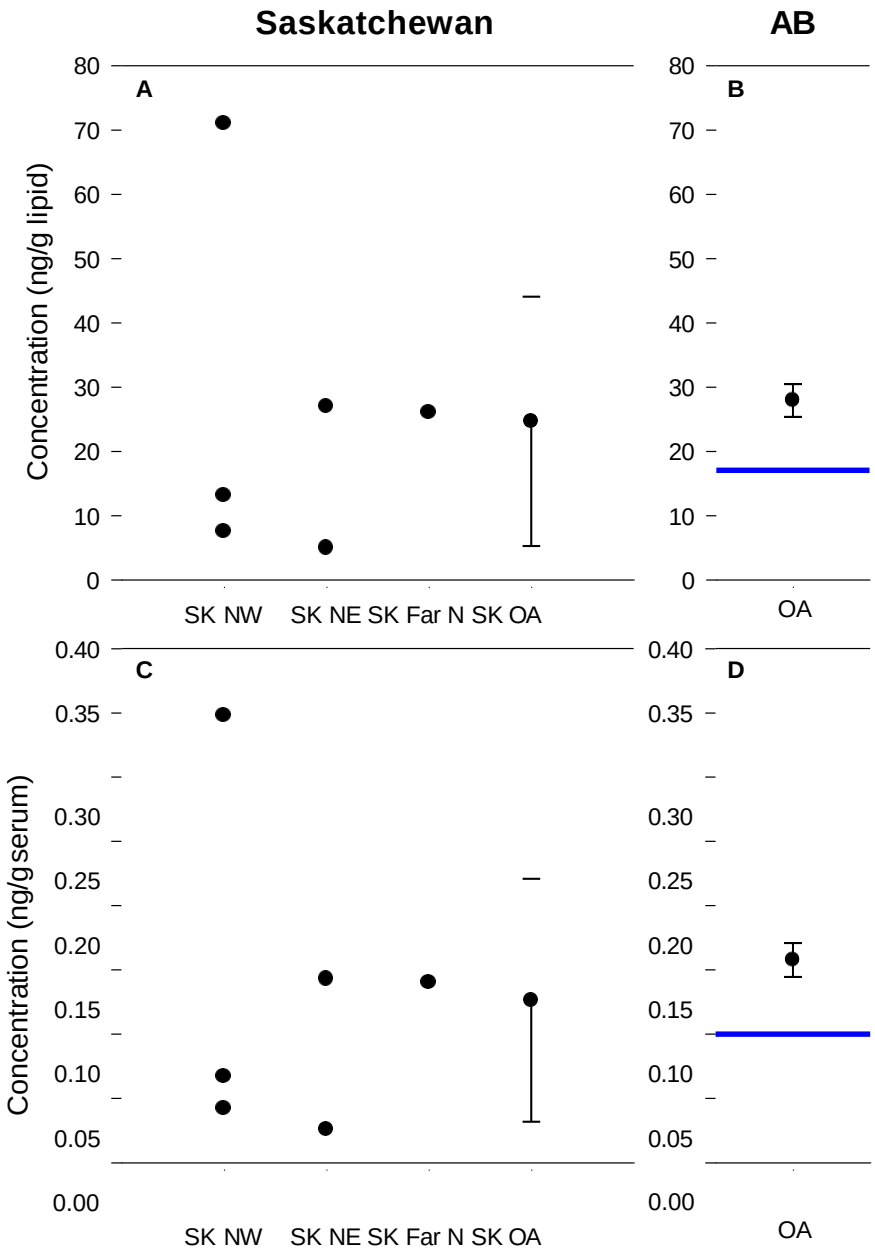

Figure 91: Concentrations of hexachlorobenzene in blood serum of pregnant women in Saskatchewan and Alberta as determined by lipid weight (A, B) and by total concentration in serum (C and D). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented by an overall average for all of the regions and age groups included. The blue lines represent the limit of detection used in laboratory analysis in the Alberta data. The limit of detection varied by pool in the Saskatchewan

data and are given in Table 17. The only pool below the LOD was pool 5 from Saskatchewan's northeast region. Estimates provided represent a 95% confidence interval around the mean.

POLYBROMINATED DIPHENYL ETHERS

GENERAL INFORMATION

Sources

Polybrominated diphenyl ethers (PBDEs or BDEs) are a class of brominated compounds commonly used as a flame retardant in a variety of consumer products not limited to electronics, carpets, furniture etc., that were first commercially produced in the 1970s (Chen, 2013; Krishnan et al., 2011; CDC, 20130; ATSDR, 2004a; Swedish National Chemicals Inspectorate, 1999; WHO, 1994). There are 209 possible BDE congeners with 1 – 10 bromine atoms. PBDEs are manufactured as commercial mixtures, and three main types have been used historically: penta-BDE, octa-BDE and deca-BDE (named according to their average bromine content) (AHW, 2008). Penta-BDE was used mainly in household furniture and other products requiring foam for stuffing, whereas octa-BDE has typically been used in high-impact plastic products, and deca-BDE has been used primarily in plastics for electric components (such as wire and cable insulation) (WHO, 1994; Alaee et al., 2003). These chemicals may be released during their manufacture and from degradation of BDE containing products, and can ultimately accumulate in the air, water or soil (CDC, 2013o). PBDEs are environmentally persistent and can travel long distances, such that they are detectable all over the world, including in remote regions far from their source. They have low vapour pressures, very low water solubility, and high octanol/water partition coefficient so it tends to bind to the organic fraction of particulate matter. Due to their high affinity for soil particles are unlikely to move into groundwater (ATSDR, 2004a).

Exposure to BDEs can occur in the household and workplace via dust due to BDE use as an ingredient in commercial products (Harrad and Diamond, 2006; Jones-Otazo et al., 2005; Webster et al., 2006). Exposure to BDEs can also occur through the ingestion of food material such as meat, dairy, fish and eggs (Schecter et al., 2006; De Wit, 2002). Because PBDEs are lipophilic (literally 'fat-loving') compounds, they build-up in the fatty tissues of our bodies over time and can concentrate in human breast milk. PBDEs can also cross the placenta (Gomara et al., 2007). In these ways, PBDEs can be passed to the fetus or to infants during pregnancy and lactation, respectively (Hooper and McDonald, 2000).

Due to measurements of PBDE in ambient air, water, food material and breast milk, it has been estimated that the upper estimate of exposure of the general Canadian population is 0.2–2.6 µg/kg bw per day with food representing the largest source of exposure particularly food with a high fat content such as fish (Health Canada, 2006; ATSDR, 2004a). Breast fed infants 0–6 months in age are estimated to be the highest exposed demographic with breast milk accounting for up to 92% of exposure. Concentrations of PBDEs in breast milk are thought to vary largely based on the individual, and based on limited data collection are increasing over time (Health Canada, 2006). However,

breastfeeding is encouraged due to the many associated health-benefits (American Academy of Pediatrics, 1997) that currently outweigh known risks. Growing environmental and human heath-related concerns have caused many countries to begin regulating these chemicals in an effort to minimize future environmental and human exposure.

Regulations in Canada

PBDEs are not manufactured in Canada but imported in the form of chemical formulations (Canada Gazette, 2006b). In 2006, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209 have all been included on the *Toxic Substances List* in Schedule 1 of CEPA 1999 (Environment Canada, 2013e). *Polybrominated Diphenyl Ether Regulations* as part of subsection 93 of *CEPA, 1999* were released with the intention of minimizing the release of PBDEs into the environment by preventing their manufacture and restricting their use in Canada, as well as prevent the sale, import and offer of sale of those listed on the *Virtual Elimination List* under CEPA, 1999 (Environment Canada, 2008b). Also by the end of 2006, the Canadian Government announced regulations to ban the manufacturing, use, sale, and import of commercial PBDE mixtures of penta-BDE and octa-BDE (Canada Gazette, 2006b).

Possible health effects

Human health effects due to low environmental exposures of PBDEs are relatively unknown but interest in these compounds has arisen due to increasing environmental concentrations of various congeners (ATSDR, 2004a). Animal studies suggest PBDE exposure may result in some effects on the liver, thyroid and brain development, as well as neurobehavioural development leading to issues in locomotory activities (CDC, 2013o; Health Canada, 2006). There are 209 PBDE congeners, varying in the number and relative position of bromine atom substitution which affect their toxicokinetic properties (Darnerud et al., 2001). For example, smaller PBDE congeners (with 1-5 bromine atoms) are better absorbed and eliminated more slowly from our bodies, and are thought to be more toxic than larger BDEs (McDonald, 2002; Sjödin, 2000). The U.S. EPA has classified decabromodiphenyl ether (BDE 209) as a possible human carcinogen and those congeners with fewer bromine atoms than BDE 208 are listed as not being classifiable in terms of their human carcinogenicity due to lack of evidence (ATSDR, 2004a). PBDE congeners with lower levels of bromination are more prone to bio-accumulation (ATSDR, 2004a).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

The following PBDEs were measured in the blood serum samples of pregnant women in northern Saskatchewan:

1. 2,4,4'-tribromodiphenyl ether (BDE 28)
2. 2,2’,4,4’-tetrabromodiphenyl ether (BDE 47)
3. 2,3',4,4'-tetrabromodiphenyl ether (BDE 66)
4. 3,3',4,4'-tetrabromodiphenyl ether (BDE 77)
5. 2,2’,3,4,4’-pentabromodiphenyl ether (BDE 85)
6. 2,2’,4,4’,5-pentabromodiphenyl ether (BDE 99)
7. 2,2’,4,4’,6-pentabromodiphenyl ether (BDE 100)
8. 2,3,3’,4,4’,5’- hexabromodiphenyl ether (BDE 138)
9. 2,2’,4,4’,5,5’-hexabromodiphenyl ether (BDE 153)
10. 2,2’,4,4’,5,6’-hexabromodiphenyl ether (BDE 154)
11. 2,2’,3,4,4’,5’,6-heptabromodiphenyl ether (BDE 183)
12. Decabromodiphenyl ether (BDE 209)

Table 18: Limits of detection and limits of quanitifaction of the PBDE congeners included in this study by pool.

Congener		Units	Pool 1	Pool 2	Pool 3	Pool 4	Pool 5	Pool 6
BDE 28	LOD	ng/g lipid	0.30	0.64	0.38	0.42	0.28	0.43
	LOQ	ng/g lipid	0.71	1.3	0.76	1.1	0.55	0.87
BDE 47	LOD	ng/g lipid	0.12	0.027	0.097	0.20	0.042	0.063
	LOQ	ng/g lipid	1.4	1.9	2.3	2.2	1.5	1.6
BDE 99	LOD	ng/g lipid	0.16	0.10	0.17	0.18	0.097	0.099
	LOQ	ng/g lipid	1.3	1.7	2.1	2.0	1.4	1.4
BDE 100	LOD	ng/g lipid	0.18	0.14	0.18	0.19	0.12	0.11
	LOQ	ng/g lipid	0.37	0.50	0.62	0.59	0.39	0.42
BDE 153	LOD	ng/g lipid	0.012	0.080	0.056	0.057	0.044	0.066
	LOQ	ng/g lipid	0.71	0.96	1.2	1.1	0.76	0.81
BDE 154	LOD	ng/g lipid	0.012	0.076	0.054	0.055	0.042	0.063
	LOQ	ng/g lipid	0.46	0.63	0.79	0.75	0.50	0.13
BDE 183	LOD	ng/g lipid	0.18	0.32	1.3	0.22	0.42	0.84
	LOQ	ng/g lipid	0.36	0.63	2.6	0.44	0.83	1.7
BDE 209	LOD	ng/g lipid	13	3.4	3.1	2.5	5.8	8.0
	LOQ	ng/g lipid	25	6.8	6.1	5.0	12	16
BDE 66	LOD	ng/g lipid	0.24	0.053	0.19	0.38	0.083	0.12
	LOQ	ng/g lipid	0.47	0.11	0.38	0.77	0.17	0.25
BDE 77	LOD	ng/g lipid	0.16	0.037	0.13	0.27	0.058	0.086
	LOQ	ng/g lipid	0.33	0.073	0.26	0.53	0.12	0.17
BDE 85	LOD	ng/g lipid	0.18	0.11	0.18	0.20	0.11	0.11
	LOQ	ng/g lipid	0.36	0.23	0.37	0.39	0.21	0.22
BDE 138	LOD	ng/g lipid	0.016	0.11	0.074	0.076	0.058	0.088
	LOQ	ng/g lipid	0.032	0.21	0.15	0.15	0.12	0.18

PBDEs are lipophilic and are found primarily within the lipid portion of the plasma or serum and as such their concentrations in the blood serum are typically made in reference to the lipid weight of the serum sample. The ranges of mean concentrations of detected congeners are shown based on lipid weights in blood serum samples:

Table 19: Ranges of concentrations of BDE detected in the 6 pools of pregnant women from northern Saskatchewan.

Chemical	Lipid weight (ng/g)
BDE 28	nd – 1.3
BDE 47	7.3 – 29
BDE 99	2.6 – 12
BDE 100	2.2 – 9.7
BDE 153	6.7 – 14
BDE 154	0.36 – 1.0
BDE 183	nd – 1.7
BDE 209	nd
BDE 66	nd – 0.38
BDE 77	nd
BDE 85	0.11 – 0.76
BDE 138	nd – 0.28

where nd = non-detect

Concentrations of PBDE congeners in pregnant northern Saskatchewan women sampled in this study ranged from 0.016 ng/g to 29 ng/g lipid (non-detects excluded) (Table 19). While there is no definitive geographical trend for PBDE congeners, pools 2-4 (pools 2 and 3 comprised of samples from the northwest and pool 4 from north eastern Saskatchewan) generally had the greatest numbers of detects or the highest concentrations. Lipid adjusted concentrations in Alberta ranged from 0.20 ng/g to 4.7 x 10^2 ng/g lipid (AHW, 2008). Pregnant women in northern Saskatchewan sampled tend to have a lower range of concentrations compared to similar populations in Alberta as the overall mean concentrations of women included in the Alberta study exceed the overall mean serum concentrations calculated from pregnant women included in this Saskatchewan study. This is true for congeners that had 1 or fewer pooled concentrations below the LOD, that is BDE 99 (SK mean ± 95% CI = 6.5 ± 2.9 ng/g lipid; AB mean ± 95% CI = 20 ± 8.0 ng/g lipid), BDE 47 (SK mean ± 95% CI = 16 ± 7.3 ng/g lipid; AB mean ± 95% CI = 43 ± 5.7 ng/g lipid), BDE 100 (SK mean ± 95% CI = 5.2 ± 2.4 ng/g lipid; AB mean ± 95% CI = 11 ± 1.7 ng/g lipid), BDE 85 (SK mean ± 95% CI = 0.50 ± 0.22 ng/g lipid; AB mean ± 95% CI = 0.99 ± 0.41 ng/g lipid), and BDE 153 (SK mean ± 95% CI = 9.7 ± 2.7 ng/g lipid; AB mean ± 95% CI = 14 ± 1.1 ng/g lipid). In particular, BDE 99 and BDE 47 concentrations were twice as high in Alberta compared to northern Saskatchewan.

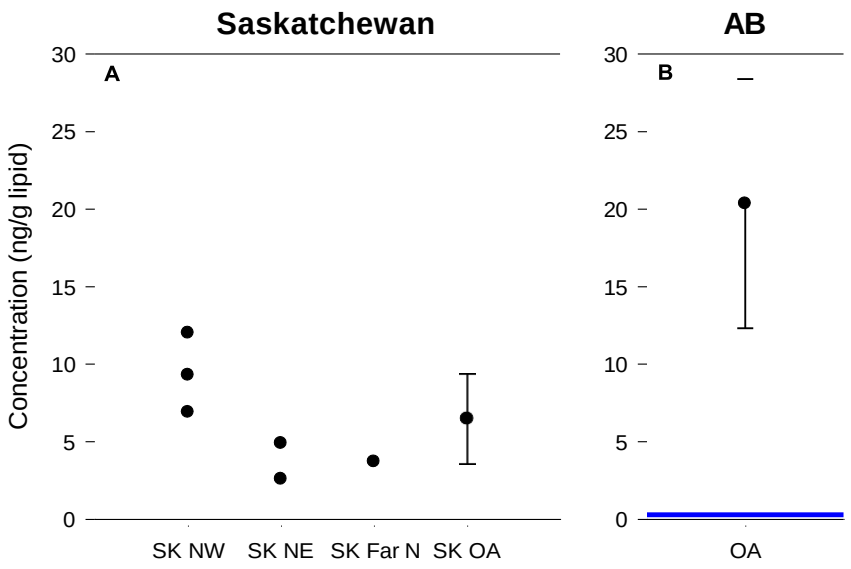

Figure 92: Concentrations of BDE 99 in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented by a mean concentration of all pools. The blue line represents the limit of detection used in laboratory analysis in the Alberta study. LODS and LOQs used in the Saskatchewan study are provided in Table 18. All 6 Saskatchewan pools were detected at levels above the LOQ and the LOD. Estimates provided represent a 95% confidence interval around the mean.

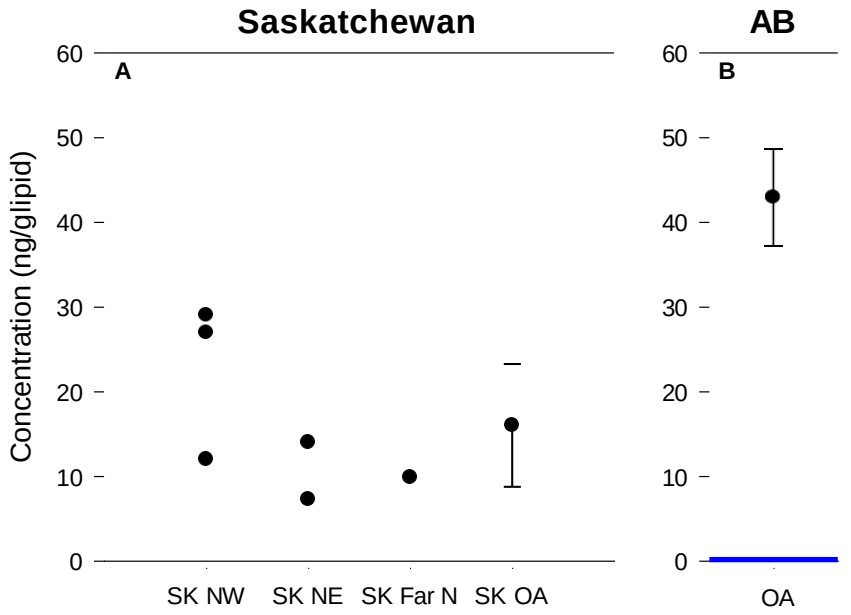

Figure 93: Concentrations of BDE 47 in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented as an overall mean of all pools. The blue line represents the limit of detection used in laboratory analysis in the Alberta study. LODS and LOQs used in the Saskatchewan study are provided in Table 18. All 6 Saskatchewan pools were detected at levels above the LOQ and the LOD. Estimates provided represent a 95% confidence interval around the mean.

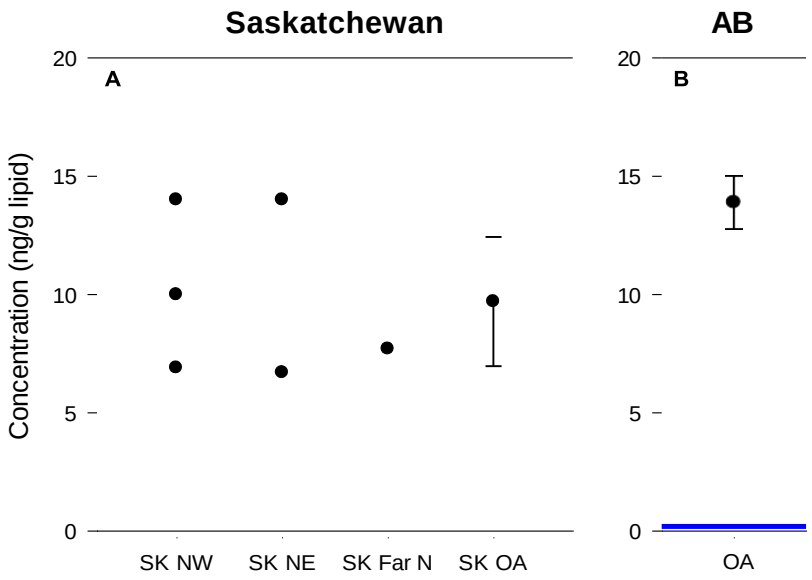

Figure 94: Concentrations of BDE 153 in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented as an overall mean of all pools. The blue line represents the limit of detection used in laboratory analysis in the Alberta study. LODS and LOQs used in the Saskatchewan study are provided in Table 18. All 6 Saskatchewan pools were detected at levels above the LOQ and the LOD. Estimates provided represent a 95% confidence interval around the mean.

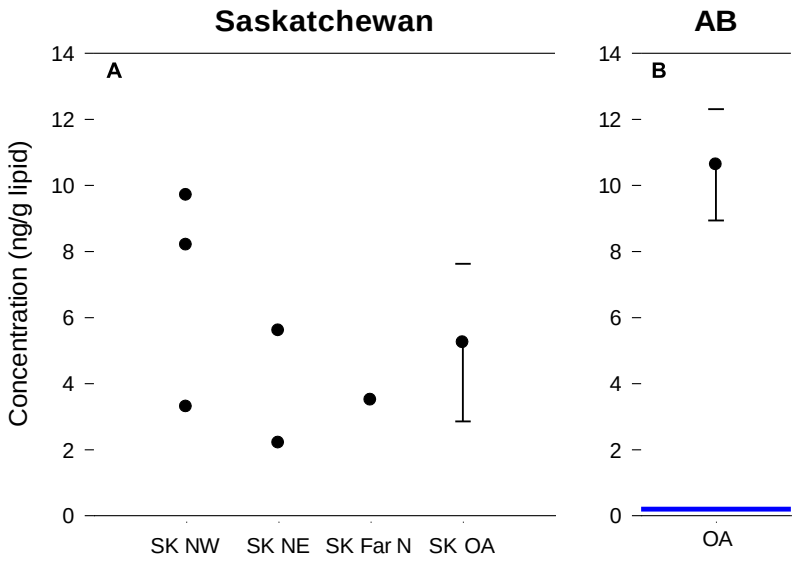

Figure 95: Concentrations of BDE 100 in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented as an overall mean of all pools. The blue line represents the limit of detection used in laboratory analysis in the Alberta study. LODS and LOQs used in the Saskatchewan study are provided in Table 18. All 6 Saskatchewan pools were detected at levels above the LOQ and the LOD. Estimates provided represent a 95% confidence interval around the mean.

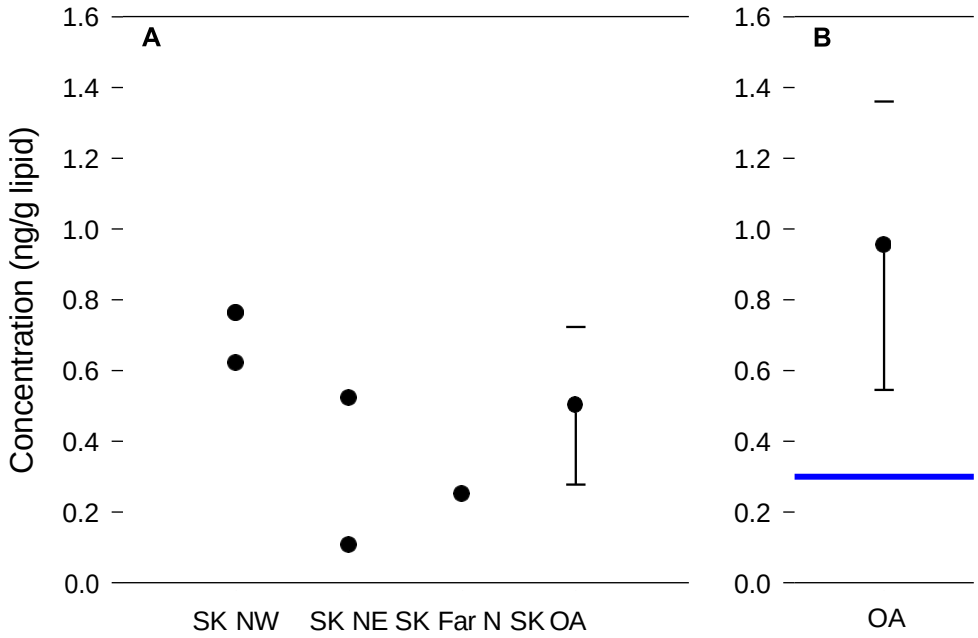

Figure 96: Concentrations of BDE 85 in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented by an overall mean of all pools. The blue line represents the limit of detection used in laboratory analysis in the Alberta study. LODS and LOQs used in the Saskatchewan study are provided in Table 18. All of the Saskatchewan pools were detected at levels above the LOQ and the LOD except for pool 5 of the northeastern region which was below the LOQ, but above the LOD. Estimates provided represent a 95% confidence interval around the mean.

PERFLUOROCHEMICALS

GENERAL INFORMATION

Sources

Perfluoroalkyls, also referred to perfluorinated chemicals, are a group of synthetic compounds that have been commonly used in industry and in the manufacturing of cleaning products, cosmetics, adhesives, fire-fighting foams, as well as water, oil and stain repellents for fabrics and paper (Health Canada, 2009d). PFCs are highly fluorinated molecules that are heat stable and repel oil, grease and water. Fluoropolymers manufactured using salts of PFCs are used in many industrial and consumer products, including surface coatings on textiles and carpets, in personal care products, and in non-stick coating on cookware. PFCs may be released into the environment near sites that manufacture them, as a result of use of products that contain them, or via breakdown of other compounds (ATSDR, 2009). PFCs are extremely resistant and are not known to break down in water or in soil, and may be carried through soil by the movement of groundwater. PFCs do degrade in air, albeit slowly and can stay suspended for days to weeks. PFCs can travel long-distances, such

that they are detectable all over the world far from their point source. Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are the two most commonly detected isomers and can be detected in a wide variety of environmental compartments, and in human and animal populations.

The perfluorochemicals (PFCs) used in Canada were imported from other countries, and not manufactured in Canada (Canada Gazette, 2006c). PFCs have been detected in water, soil, air, dust, sewage, sediment and food, and as such humans may become exposed through a variety of routes (Health Canada, 2009d). The use of PFCs in grease and water repellent coatings for food packaging contributes to PFC exposure via ingestion (Begley et al., 2005; Tittlemier et al., 2006). Despite the fact that levels found in food are lower than what would be expected to cause adverse health effects, Health Canada considers food to be a major route of exposure. Perfluorinated chemicals are commonly biomonitored using serum, plasma or whole blood sampling (Fromme et al., 2009). PFCs can cross the placental barrier and have also been detected in breast milk, which may be sampled to better predict potential exposures in infants (ATSDR, 2009; Inoue et al., 2004; Apelberg et al., 2007).

Regulations in Canada

In 2002, the predominant global manufacturer voluntarily phased out the production of PFOS and other related chemicals. As a result, PFOS use and its by-products decreased significantly in Canada after 2002 (Canada Gazette, 2006c). In 2009, Environment Canada adding PFOS and its salts to the *Virtual Elimination List* compiled under subsection 65(2) of CEPA 1999 (Environment Canada, 2009). Following the release of a *Notice of Action Plan for the Assessment and Management of Perfluorinated Carboxylic Acids (PFCAs) and their Precursors* in 2006 indicating the Government of Canada's intention to reduce long chain PFCA's, a voluntary agreement, *Environmental Performance Agreement Respecting Perfluorinated Carboxylic Acids (PFCAs) and their Precursors in Perfluorochemical Products Sold in Canada* was signed by 5 industrial companies with the goal of eliminating residual precursors, long chain PFCAs and PFOA contained in perfluorochemical products sold in Canada by 2015 (Environment Canada, 2013c; Environment Canada, 2013d). Additionally, the sale, manufacturing, offer for sale and use of PFOS or other products containing PFOS are prohibited through *Perfluorooctane Sulfonate and its Salts and Certain Other Compounds Regulations (PFOS Regulations)* except for certain exemptions such as aqueous film-forming foams and photographic films, papers and plates (Environment Canada, 2013c; Environment Canada, 2013d; Environment Canada, 2009). PFOS and its salts, as well as perfluorooctanoic acid (PFOA), which has the molecular formula $C_7F_{15}CO_2H$, and its salts have been included on the *Toxic Substances List* in Schedule 1 of CEPA 1999 (Environment Canada, 2013e). The Canadian Government has added fluorotelomers, which are PFCs that can degrade to PFOA in the atmosphere and in organisms to the List of Toxic Substances under Schedule 1 of CEPA 1999 (Canada Gazette, 2006d).

Possible health effects

Human studies relating to possible health effects of PFC exposure are very limited. There is comparably more data from animal studies at higher concentrations, and adverse health effects in animals include developmental and reproductive effects, as well as general systemic toxicity and effects on the liver (Lau et al., 2004; Seacat et al., 2003; Butenhoff et al., 2004). No definitive links between exposure to these substances and human health effects have been established based on occupational studies or studies of populations exposed to contaminated drinking water; however, adverse effects have been observed in animals (ATSDR, 2009).

A study conducted in Germany found low levels of PFCs in cord sera and an increase in concentrations through the first months of infant life (Fromme et al., 2010). Although the concentrations in breast milk were low, the intake led to a body burden at age six months similar to (PFOS) or higher than (PFOA) than that found in adults. The maternal serum lipid adjusted concentrations of organohalogen compounds averaged 1.7 times those of cord serum, 2.8 times those of cord tissue and placenta, and 0.7 those of milk (Needham L et al., 2011). A few studies suggested negative associations between PFOS or PFOA concentrations in pregnant women or cord blood and infant birth weight or size (Apelberg et al., 2007; Fei et al., 2007).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

The following PFCs were measured in the blood serum of pregnant women in northern Saskatchewan:

Perfluoroalkyl sulfonates

1. Perfluorohexane sulfonate (PFHxS)
2. Perfluorooctane sulfonate (PFOS)
3. Perfluorodecane sulfonate (PFDS)

Perfluoroalkyl carboxylates

1. Perfluorooctanoate (PFOA)
2. Perfluorononanoate (PFNA)
3. Perfluorodecanoate (PFDA)
4. Perfluoroundecanoate (PFUA)
5. Perfluorododecanoate (PFDoA)

All PFCs, except PFOS and PFOA, had concentrations where two or more sample pools were below the limit of detection (<0.50 ng/mL). The following table lists the range of serum adjusted concentrations for PFCs.

Table 20: The ranges of various perfluorinated compounds measured in 6 pools of pregnant women from northern Saskatchewan.

Chemical	Wet weight (ng/mL)
PFOS	2.55 – 3.33
PFOA	0.633 – 0.912
PFNA	<LOD – 3.45
PFDA	<LOD – 0.738
PFUA	<LOD – 0.507
PHFxS	<LOD
PFDS	<LOD
PFDoA	<LOD

While there was no definitive geographical trend, pool 6 (far N) contained five out of eight of the maximum concentrations among the results. Concentrations of pregnant women from northern Saskatchewan sampled here are lower than those of pregnant woman sampled from Alberta where age and geographic trends were present; however, due to stratification of the Alberta data, comparisons should be made with care. The maximum concentrations in pregnant women from northern Saskatchewan women are lower than the maximum concentrations detected in Alberta samples. While only two out of eight isomers were detected in northern Saskatchewan, in Alberta, eight out of nine isomers were detected above LOD.

Weighted arithmetic mean concentrations (± 95% confidence interval) in serum was 0.738 ± 0.0874 ng/mL and 3.00 ng/mL ± 0.232 ng/mL for PFOA and PFOS, respectively. The ranges of concentrations detected in all 6 pools for all of the PFCs included in this study are presented in Table 20. In pooled samples from the CHMS Cycle 2, the geometric mean (95% confidence interval) of female participants aged 20 – 79 years was 2.0 (1.8 to 2.2) µg/L plasma for PFOA and 5.7 (4.9 to 6.6) µg/L plasma for PFOS. Reported female PFC geometric mean concentrations from the U.S. National Health and Nutrition Examination Survey (NHANES, 2003-2004 Fourth Report) (CDC, 2009) ranged from 0.139 µg/L (PFUA) to 5.11 µg/L serum (PFOS).

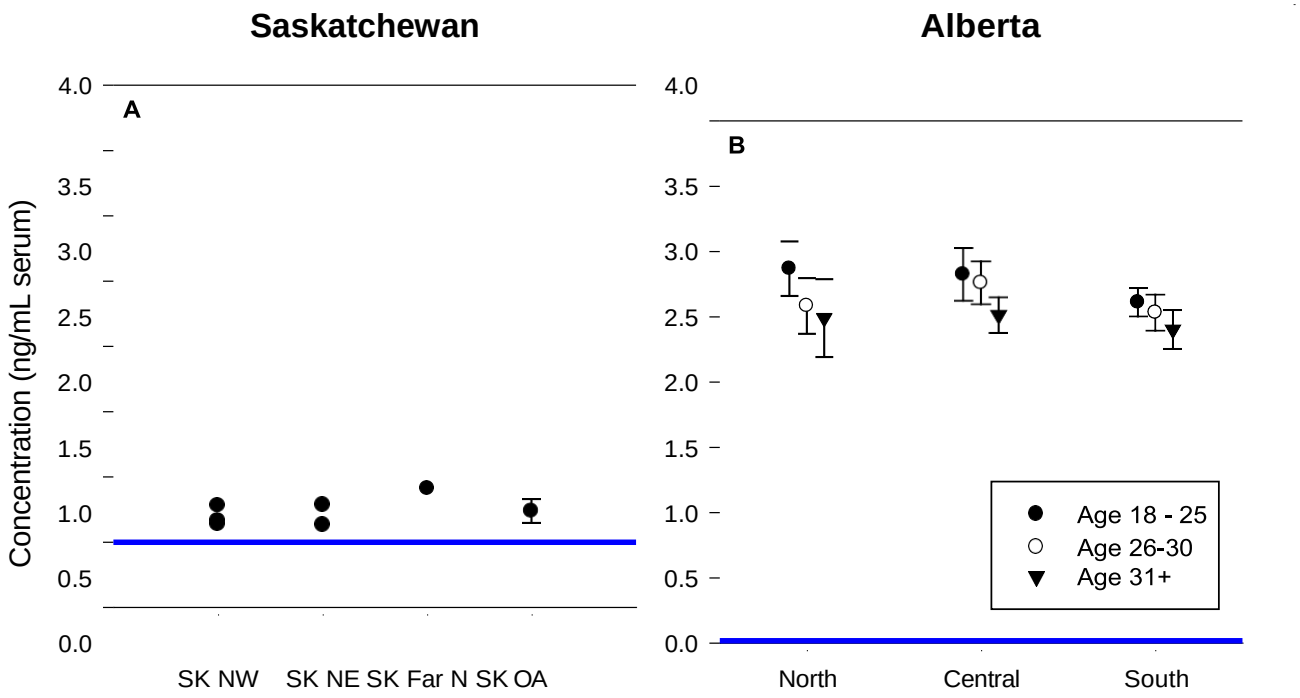

Figure 97: Concentrations of PFOA in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented by mean concentrations stratified by both age and region. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

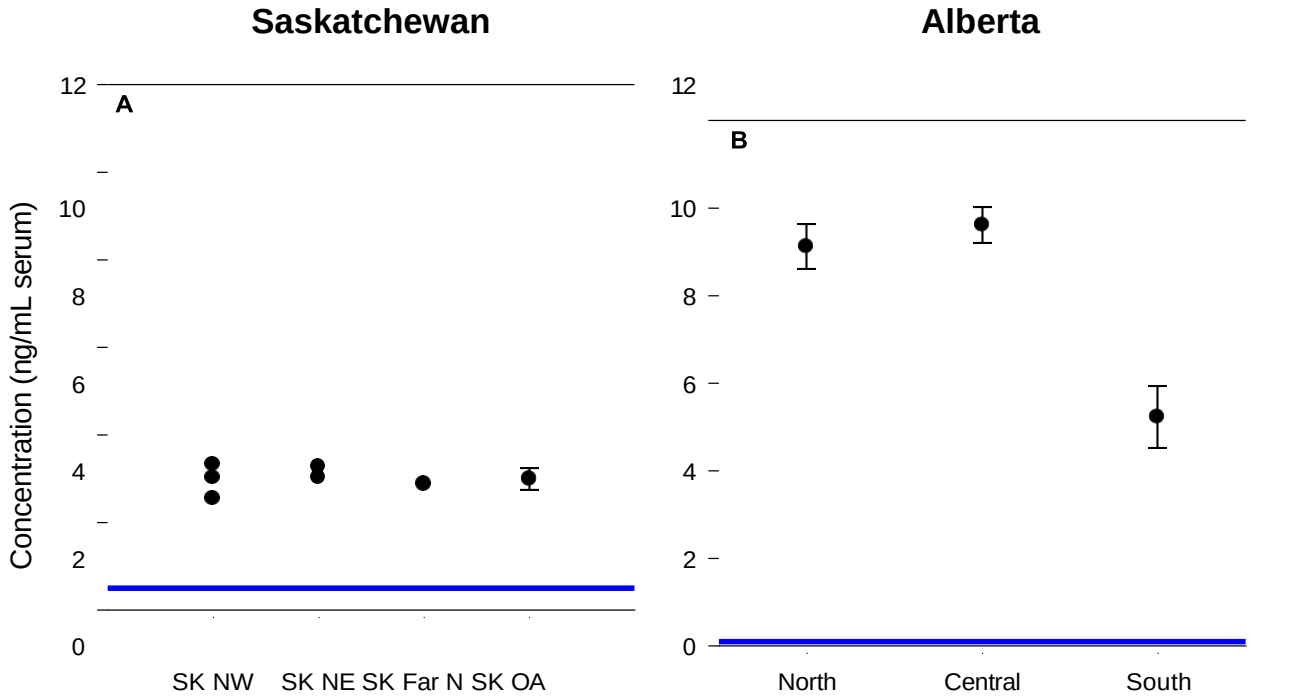

Figure 98: Concentrations of PFOS in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented by region with mean of all

the pools that were analyzed from each region. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

BISPHENOL-A

GENERAL INFORMATION

Sources

Phenols are a class of aromatic alcohols with the chemical formula C_6H_6O (Environment Canada, 2000). Bisphenol A is used as an epoxy resin and used to manufacture polycarbonate plastics. BPA is found in a variety of consumer products including food packaging, toys, baby bottles, automobile parts, eye glasses lenses, medical equipment, water pipes etc. (CDC, 2013p; Vandenberg et al., 2007, Brotons et al., 1995; ENDS, 1995; Le et al., 2008). The extensive use of BPA in the manufacture of consumer products has led to widespread exposure and the predominant source of BPA exposure to the general population is the use of everyday plastic products, and consumption of contaminated canned and bottled foods and beverages (Burridge, 2003; Kang et al., 2006). BPA has also been shown to leach from municipal waste landfills into the surrounding ecosystem (Kawagoshi et al., 2003; Coors et al., 2003).

Infants have historically been at risk of exposure to BPA through leaching of BPA from infant formula cans into baby food, and in baby bottles, (Burridge, 2003; Vandenberg et al., 2007; Maragou et al., 2008). In addition to exposure from food containers, infants can also be exposed to BPA in utero as it is able to cross the placenta, and after birth in breastmilk (Sun et al., 2004); Schonfelder et al., 2002). Despite the risk for exposure for BPA in breaskmilk, breastfeeding is encouraged due to the many associated health-benefits that currently outweigh known risks (American Academy of Pediatrics, 1997).

Regulations in Canada

In 2008, the Canadian Federal Government, as a part of the Chemicals Management Plan, completed a detailed safety review to assess the potential human and environmental effects of BPA and an update was released in September 2012. Based on the Assessment Report, the Canadian Federal Government proposed to add BPA to the List of Toxic Substances in Schedule 1 of the *Canadian Environmental Protection Act,* 1999. Health Canada has determined BPA to be of concern to human health and the environment as per the criteria set out under the *Canadian Environmental Protection Act*, 1999 (Canada Gazette, 2010b). A prohibition of polycarbonate baby bottles that contain BPA came into effect on March 11, 2010 and published in the Canada Gazette on March 31, 2010. Part I of Schedule I to the *Hazardous Products Act* was amended to include this item, thus prohibiting the advertisement, sale and importation in Canada of these products (Canada Gazette, 2010c).

In 2013 the Government of Canada signed a voluntary agreement with 13 paper recycling mills entitled *Environmental Performance Agreement Respecting Bisphenol A in Paper Recycling Mill Effluents* with the goal of reducing the environment impact of mill effluent (Environment Canada, 2013f). The objective of this agreement was to achieve and maintain a concentration of BPA in the effluence of less than 1.75 µg/L in the final effluent, or less than 0.75 µg/L in the receiving environment 100 m downstream from the environmental depositing.

Possible health effects

Due to the previously widespread use BPA, there is an increasing interest in investigating the effects of BPA exposure on human health. Bisphenol A is not considered mutagenic and while it is unlikely a carcinogen, IARC and NTP have not yet classified Bisphenol A as to its human carcinogenicity (CDC, 2013p). Bisphenol A is weakly estrogenic, and while not considered a teratogen has been shown to effect both development and reproduction in animal, in addition to possible neurotoxicity, ovarian dysfunction and recurrent miscarriages (Le et al., 2008; Newbold et al., 2007; Takeuchi et al., 2004; Sugiura-Ogasawara et al., 2005). Levels of phenol exposure can be sampled using blood or urine samples to test for the parent compounds (ATSDR, 2008b). However, it is important to note that phenols and phenol metabolites may result from exposure to other chemicals.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

The measured concentrations for bisphenol-A in pregnant women sampled from northern Saskatchewan were all below the limit of detection (0.20 ng/mL). The limit of detection used in the Alberta study was 0.01 ng/g and while these units of measurement are different than those used in Saskatchewan the density of human serum is ~1.024 g/ml (Sniegoski and Moody, 1979), therefore ng/g and g/mL of human serum are equivalent and can be used interchangeably for the purposes of comparison. LOD for the Alberta study was considerably (~20x) lower which indicates a much higher degree of sensitivity in the ability to detect BPA in the samples. This difference in LODs is due to differences in analytical methodology as the AB samples were analyzed using high resolution GC-MS while the Saskatchewan samples were run using LC-MSMS. This may partially explain why all six of the Saskatchewan pools were lower than the 0.20 ng/mL limit of detection used, while more than 25% of the Alberta pools were found to have concentrations above the 0.01 ng/g limit of detection (or 10 pg/g). Also, it is important to note that the Alberta samples were collected before the prohibition of BPA in baby bottles and similar plastic products, while the Saskatchewan samples were collected after this change in regulation. Therefore it is possible that women in Saskatchewan were exposed to a smaller amount of BPA due to regulatory changes.

The U.S. National Health and Nutrition Examination Survey (NHANES, 2003-2004 Fourth Report) (CDC, 2009) measured BPA concentrations in urine and reported a female creatinine adjusted geometric mean of 2.78 µg/g. The CHMS Cycle 2 reported female (aged 6-79 years) creatinine adjusted geometric mean concentration of 1.3 µg/g which is slightly lower than the concentrations found in the United States (Health Canada, 2013). These results cannot be compared directly to the northern Saskatchewan serum results due to differences in analyte medium. However, in phase two of Alberta's biomonitoring program (2010), BPA was measured in women aged 26-30 years in southern Alberta where overall mean concentrations ranged from 1.7 x 10^2 to 3.5 x 10^2 pg/g serum. Seasonal variation was observed in southern Alberta without any apparent temporal trend. Similar trends cannot be uncovered in the population of women from northern Saskatchewan due to limitations within the study design and sampling timeframe.

A study measuring levels of BPA in the serum of pregnant women from eastern townships of Canada, found a mean (SD) serum concentration of 1.36 (1.18) ng/mL (Aris, 2014). This is higher than the concentrations found in northern Saskatchewan as all 6 pools in Saskatchewan had concentrations below 0.2 ng/mL (Aris, 2014). Nonpregnant women from the same study were found to have even higher concentrations of BPA in their blood with a mean (SD) serum concentration of 3.83 ±1.98 ng/mL (Aris, 2014). Another study of women which measured serum levels of bisphenol A in women receiving screening mammograms in Wisconsin found median serum concentration among those subjects with detectable levels of 0.55 ng/mL serum, which is higher than the levels measured in northern Saskatchewan, but lower than those measured in both pregnant and nonpregnant women from eastern Canada (Sprague et al., 2013).

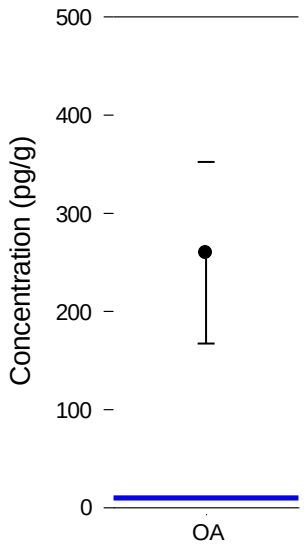

Figure 99: Mean serum concentration of bisphenol A in pregnant women in southern Alberta between 26 and 30 years of age. Data is presented as an overall (OA) arithmetic mean of all the pools included in this study. Estimate represents a 95% confidence around the mean, and the limit of detection is represented by the blue line. Note: 10 pg/g = 0.01 ng/g and 0.01 g/ml

OCTYLPHENOL

GENERAL INFORMATION

Sources

Octylphenol (OP), an alkylphenol, refers to a group of chemicals used in the manufacture anionic surfactants for detergents, industrial cleaners, and emulsifers (alkylphenol ethoxylates). OPs are mainly used in the manufacture of octylphenol ethoxylates (OPEs) which may be added to a number of commercial products such as paints, adhesives, plastics or rubbers. In its solid form, it does not mix well with water and is corrosive. It enters the environment through human use of octylphenol-containing products, through sewage, and through manufacturing waste streams. Human exposure to octylphenols may occur through ingestion of contaminated foods and drinking water, and from contact with some personal care products and detergents. Major Canadian industrial users of octylphenol are textile mills and the pulp and paper industry.

These chemicals enter the aquatic environment through discharge from urban, municipal, and industrial wastewater and by direct discharge in such activities as pesticide application (Ying et al., 2002). Absorption, ingestion and inhalation of water and air containing trace concentrations of these are sources of minor exposure to general population (Monteiro-Riviere et al., 2000). However, due to their lipophilic nature, they accumulate in fatty tissues of wildlife and enter the human food chain, thus dietary intake is another potential source of exposure (Guenther et al., 2002). OP can also accumulate in breast milk (Guenther et al., 2002) and can cross the placenta (Environment Canada, 2000). In these ways, OP can be passed to the fetus or to infants during pregnancy and lactation, respectively. However, breastfeeding is encouraged due to the many associated health-benefits (American Academy of Physicians, 1997) that currently outweigh known risks.

Regulations in Canada

Canadian regulations of octylphenol are lacking; however, during the 1980s and 1990s, several European nations banned the use of alkylphenol ethoxylates which degrade into alkylphenols such as octylphenol and nonylphenol, in domestic detergents and other uses (CDC, 2013n).

Possible Health Effects

Human health effects from octylphenol from low environmental exposures are unknown. Based on preliminary review of data by Health Canada, the estrogenicity of OP and OPEs may be greater than that of NP and NPEs. Exposure concentrations are dependent on a variety of factors including but not limited to race/ethnicity, age, sex, and socio-economic status. Chronic exposure to

the material may cause or increase risk of impaired fertility due to potential estrogenic effects, which may also be a secondary non-specific consequence of other toxic effects.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Octylphenol was detected in all sample pools with concentrations ranging from 13.7 ng/mL to 19.0 ng/mL (weighted arithmetic mean ± 95% confidence interval: 17.3 ng/mL ± 1.79 ng/mL). Octylphenol was evaluated in phase two (AHW, 2010) of Alberta's biomonitoring program but was not reported due to contamination of quality control samples. It was not reported in other biomonitoring studies across Canada. A study of women receiving screening mammograms in Wisconsin measured a median serum concentration among those subjects with detectable levels of serum octylphenol of 1.78 ng/mL (Sprague et al. 2013). This is lower the levels of octyphenol measured in the 6 pools of women from northern Saskatchewan. Alternatively, a Chinese study investigating the relationship between environmental toxins in paired maternal and fetal samples from participants in the Yangtze River Delta measured a median (IQR) maternal serum concentration of 470 (280-660) ng/mL octylphenol which is considerably larger than the concentrations found in the blood serum analyzed from women in northern Saskatchewan (Li et al., 2013).

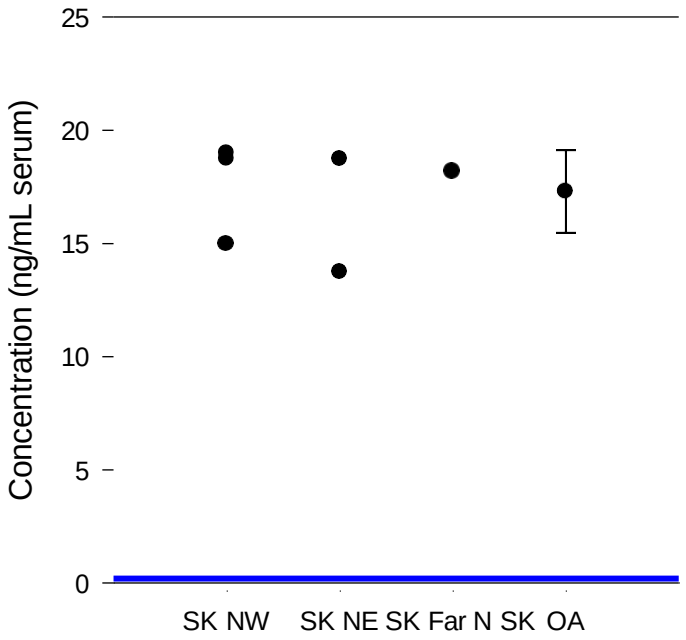

Figure 100: Concentrations of octylphenol in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

METHYLMERCURY (CH_3Hg)

GENERAL INFORMATION

Sources

Mercury is a naturally occurring chemical element that is widely distributed around the earth in its elemental, inorganic and organic forms (CDC 2013l; ATSDR 1999a; ATSDR, 1999b, ATSDR, 2013c; WHO 2005; Environment Canada, 2013h). Mercury is released to air and water from the combustion of fossil fuels (mainly coal), mining, smelting, and other industrial processes as well as through natural processes such as release from soil and rocks through weathering, from volcanoes and forest fires, and deforestation leading to soil erosion (Health Canada, 2007). Mercury is not commonly found in water as it generally binds to soil and sediment; however it may enter the water system from spills or industrial effluent, irrigation run off or drainage from areas in which agricultural pesticides are in use (Health Canada, 2012). Mercury can be changed between forms in the environment by natural processes and by microorganisms. One of the most important processes is the methylation of inorganic mercury in aquatic environments creating the organic compound Methylmercury which is readily absorbed and can bioaccumulate in living organisms (WHO 2005; ATSDR 1999a &1999b). Methylmercury concentrations increase at each stage in this food chain through the process of biomagnification in which larger prey animals accumulate the methylmercury found in their food, resulting in higher concentrations of methylmercury in our food sources (ATSDR, 1999a; Mahaffey, 1999). Mercury is found throughout the global environment, including remote Arctic regions, due to its persistence, mobility and tendency to bioaccumulate in colder climates through atmospheric circulation.

Food is the main source of mercury exposure in populations that are not exposed to mercury occupationally (WHO, 2005; WHO, 1990). Methylmercury is absorbed extremely well through the gastrointestinal tract with up to 95% of the total external dose absorbed. As previously noted, methylmercury concentrations increase up the food chain, therefore large predatory fish contain higher concentrations of methylmercury, as such fish such as shark, large tuna, swordfish, marlin, and king mackerel may contain 10-20 times higher concentrations than fish such as herring, cod, pollack, and shellfish such as shrimp or scallops (Mahaffey, 2004). Health Canada updated (March 28, 2007) the guidelines on fish consumption to limit mercury exposure and to consume a variety of fish/seafood in the diet. Canadians are advised to limit their consumption of higher mercury fish such as fresh and frozen tuna, shark, swordfish, escolar, and marlin to a maximum of 150 grams per week. For pregnant women, breastfeeding mothers and women who may become pregnant, the guidelines include a limit of 150 grams per month of these fish (AHW, 2008). The suggested maximum amount of these fish for children aged five to eleven is 125 grams per month, and for children aged one to four is 75 grams per month. Saskatchewan has additional consumption guidelines available for sports fish based on the lake, the type and size of the fish recommendations

and whether is for the general population or for women of child bearing age and for children.. (Government of Saskatchewan, 2013; Government of Saskatchewan, 2017). Health Canada recommends that all Canadians, including pregnant women and children, eat at least 150 grams of fish per week to benefit from the nutrients found in fish and seafood, but to limit the consumption of fish known to have higher mercury levels (Health Canada, 2009e). Canned light tuna is a good alternative for limiting mercury intake while maintaining fish consumption, as it generally has lower methylmercury concentrations than canned albacore (white) tuna or fresh tuna steaks (Health Canada, 2007; AHW, 2008).

Behaviour in the Body

Methylmercury enters the bloodstream after absorption from the gastrointestinal tract. Blood-borne methylmercury is present primarily in red blood cells where it has the potential to breach the blood-brain barrier, depositing methylmercury in the brain where it may accumulate while dealkylating back into inorganic mercury (Vahter et al., 1994). Studies indicate that it takes approximately 70 days to remove half of the body's methylmercury stores (WHO, 1990; Gosselin et al., 2006). It is removed slowly through urine, feces, and breast milk.

Blood and urine samples are both commonly used as biological matrices in which to measure mercury exposure, as well hair has been used as an indicator of Methylmercury exposure. It is important to note that only a small portion of Methylmercury in the blood is found within the serum (~5%), as blood-borne Methylmercury is typically found within red blood cells (Kershaw et al., 1980). Therefore methylmercury exposure is more accurately measured using whole blood sampling as opposed to serum samples, and care must be taken not to directly compare results from studies using whole blood concentrations and serum concentrations.

Possible health effects

As with any chemical exposure, the human health effects arising from exposure to mercury are diverse and can depend on the dose, mercury speciation, length of exposure, and timing of the exposure, however, areas of the body such as the brain and the kidneys are particularly sensitive to the effects of mercury (ATSDR, March 1999, AHW, 2008; CDC, 2013l; NRC, 2000). At moderate to high doses, such as in the case of accidental poisonings, methylmercury is well documented to be a human neurotoxin, and may cause adverse effects to the motor and sensory systems such as hearing impairment, parasthesias, ataxia and dysarthria (Tsubaki and Irukyama, 1977; Bakor et al., 1973). Metallic mercury vapours or organic mercury may affect different areas of the brain and their associated functions, resulting in a variety of symptoms (ATSDR, 1999a). High exposures to methyl mercury can result in a variety of central nervous system symptoms. High exposures during prenatal development can lead to developmental issues such as limb deformities, cognitive issues, and altered physical growth. IARC has classified methylmercury as being a possible human carcinogen and inorganic mercury as being unclassifiable (CDC, 2013y; Bakor et al., 1973). Health Canada's guidelines for methylmercury in whole blood are as follows: (i) concentrations from 20 to 100 ng/g

are considered a “level of concern” or “increasing risk”, and (ii) concentrations greater than 100 ng/g are considered “at risk” or at a “level of action” (Health Canada, 1999).

While it is important to limit the consumption of mercury, there are health concerns that people may limit their overall consumption of fish because of concerns for mercury. There are significant benefits of fish consumption. Fish is an excellent source of high quality protein and is one of the best food sources of the long-chain omega-3 fatty acides, docosahexaeonoic (DHA) and eicosapentaenoic (EPA) acid (Health Canada, 2007). These omega-3fatty acids are required in the diet and are considered important to cardiovascular health, and brain and eye development of infants and children. Fish are also excellent sources of minerals (selenium, iodine, magnesium, iron and copper) and vitamins (the most significant source of naturally-occurring vitamin D). Messages regarding mercury concerns in fish need to be framed in a way that result in lower mercury exposure without increasing unintended public health risks from lower fish consumption (Cohen et al., 2005).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Methylmercury was detected in 4 out of 6 sample pools comprised of pregnant women from northern Saskatchewan, ranging in concentration from 0.1 ng/g to 0.3 ng/g. Pool 6 (far N) had the highest concentration. Methylmercury concentrations in blood serum of pregnant Albertan women ranged from 0.04 ng/g to 0.2 ng/g. Concentrations depended on age and geographic region.

Although Health Canada has set guidelines for methylmercury in whole blood, it is difficult to interpret the present results in this context because serum is known to contain only a small fraction (5%) of total methylmercury. The high pool 6 (far N) concentration may be attributed to the consumption of larger, predator fish.

The recent First Nations Food, Nutrition and Environment Survey in Saskatchewan reported on mercury exposures as measured in hair samples and calculated through dietary estimates, to be low and not a health concern for Saskatchewan First Nations generally; however, there were some exceedances of Health Canada guidelines among women of childbearing age living in the Boreal Shield ecozone of the province (about 5%) where higher fish consumption was also documented. (Chan et al., 2018)

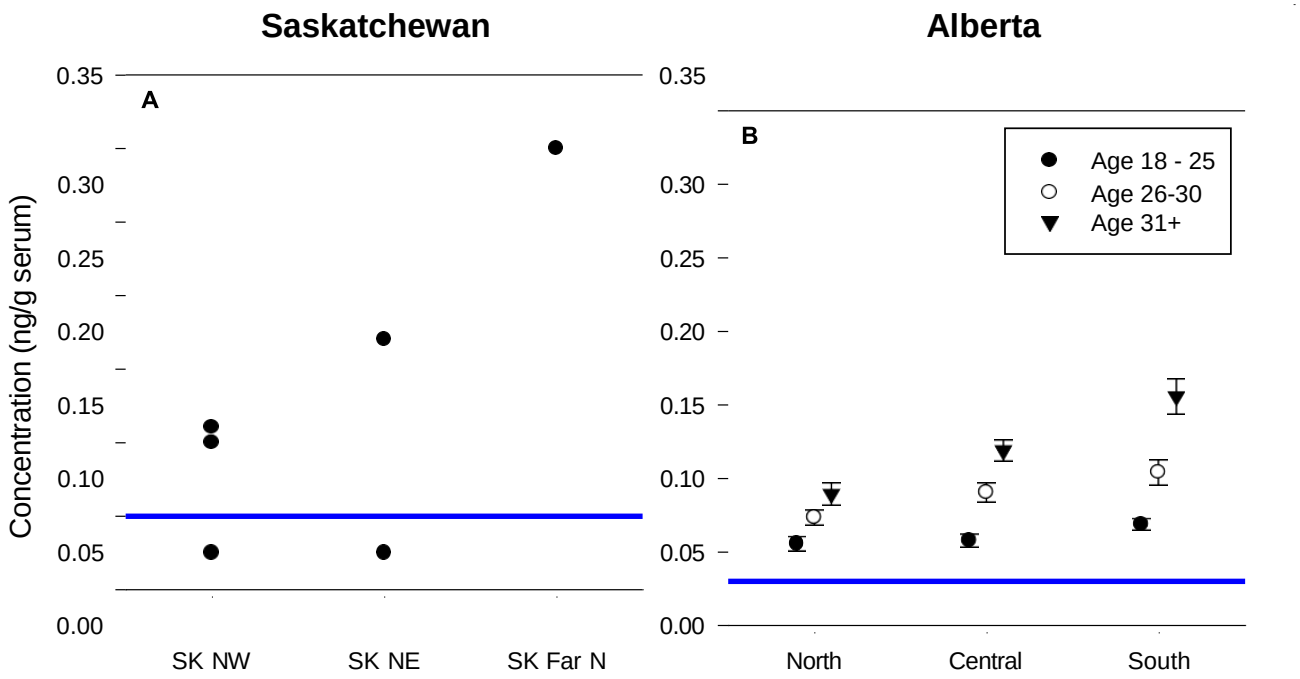

Figure 101: Concentrations of methylmercury in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples. Alberta data is organized by region and age and represented with mean concentrations. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

PHTHALATES

GENERAL INFORMATION

Sources

Diesters of phthalic acid, or phthlates, are a class of industrial chemicals that are primarily used as plasticizers to impart flexibility and resilience to plastics (U.S. EPA, 2007). They are dialkyl or aryl esters of 1,2-benzenedicarboxylic acid. Some phthalates occur naturally in oil and coal, but the vast majority are man-made. Phthlates have a wide range of applications and can be found in PVC flooring, printing inks, personal care products, medial equipment and insect repellents, among other products.

Phthalates can be released to the environment through air emissions during their manufacture and use, via waste waters from various industries, in municipal sewage, from the incomplete combustion of plastics, and from the use and disposal of consumer products. Phthalates have been detected in food, water, air and dust. Due to the sheer volume of phthalates produced and used each year – over 18 billion pounds – and since phthalates are not chemically bound to plastics used in consumer products, potential leaching could occur during the use of the products (Clark et al., 2003; Crinnion, 2010). Infants may have greater exposure from ingesting household dust or breast milk (Calafat et al., 2004)

For the general public, contaminated foods and the use of consumer products made out of PVC plastics are the primary sources of exposure to phthalates. Hand to mouth behaviours in children may increase the risk of phthalate exposures. Phthalates are not bio–accumulative compounds and following exposure are metabolized and excreted in the urine and feces. As such human biomonitoring of phthalate exposure is most commonly approached through the analysis of urinary metabolites (Hauser, 2008).

Regulations in Canada

Several phthalates have been assessed as priority substances by Environment Canada and Health Canada. Di-2-ethylhexyl phthalate (DEHP), which metabolizes to mono-2-ethylhexyl phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), and mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), was declared toxic under Schedule 1 of CEPA 1999 because it was considered to be a potential danger to human health based on available data. DEHP has recently been included on Health Canada's list of prohibited and restricted cosmetic ingredients (also known as the Cosmetic Ingredient Hotlist) under the *Food and Drugs Act* (Environment Canada). Health Canada's *Phthalates Regulations*, 2010 of the *Consumer Products Safety Act* restricts the six phthalates in soft vinyl children's toys and child-care products (Government of Canada, 2010). These regulations follow similar regulations set out in the United States and the European Union.

The Government of Canada will be evaluating 14 substances that are part of the phthalate substance grouping. Fourteen additional substance are under consideration to be included in the grouping as well. The anticipated date for release of the draft screening assessment is winter 2014/2015 (Health Canada, 2014).

Possible Health Effects

In humans, phthalates are rapidly metabolized, do not bioaccumulate and have short biological half-lives. Phthalate diesters are converted to their corresponding monoesters in the gastrointestinal tract or saliva prior to absorption. Measurement of phthalate metabolites in urine has become the most common approach to assess phthalate exposure in humans and reflects relatively recent exposure.

Human health effects data is limited; however, there are multiple studies demonstrating human exposure to phthalates in the human population, including prenatal exposure. Phthalates are often classified as endocrine disruptors or hormonally-active agents because of their ability to interfere with the endocrine system in the body (US NTP, 2007). Although no causal relationship has been established, several studies suggest an association between urinary phthalate metabolite concentrations and adverse effects on development and reproduction, particularly the male reproductive system with phthalate exposure resulting in increased incidence of undescended testes, decreased testes weight, decreased anogenital distance (distance between the anus and the base of

the penis), and other effects (US EPA, 2007b; ATSDR. 2002a; ATSDR, 2002c; ATSDR, 1997a; ATSDR, 1997b, ATSDR, 1995). Exposure to phthalates has been reported to result in increased incidence of developmental abnormalities such as cleft palate and skeletal malformations, and increased fetal death in experimental animal studies (US EPA, 2007b; ATSDR. 2002a; ATSDR, 2002c; ATSDR, 1997a; ATSDR, 1997b, ATSDR, 1995).

The monoester metabolites are thought to mediate toxic effects for some of the phthalates, but there are known species-related differences in the hydrolysis of diester phthalates, efficiency of absorption, and extent of metabolite conjugation to gluronide (Albro and Lavenhar, 1989; Kessler et al., 2004).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

The following phthalate metabolites were evaluated in the current study:

1. Monomethyl phthalate
2. Monoethyl phthalate
3. Monoisobutyl phthalate
4. Monocyclohexyl phthalate
5. Monobenzyl phthalate
6. Mono-(2-ethylhexyl) phthalate
7. Mono-n-octyl phthalate
8. Mono-(2-ethyl-5-oxohexyl) phthalate
9. Monoisononyl phthalate
10. Mono-(2-ethyl-5-hydroxyhexyl) phthalate

However, only monoethyl-, monoisobutyl-, monobenzyl-, and mono-(2-ethylhexyl) phthalates were detected above the LOD of 0.25 ng/mL. These ranged in concentration from 0.920 ng/mL to 241 ng/mL serum.

Table 21: The ranges of concentrations detected from the 6 pools of pregnant women from northern Saskatchewan, and the corresponding mean serum concentration and 95% confidence interval.

Chemical	Serum concentration (ng/mL serum)	Mean (95% CI) (ng/mL serum)
monoethyl phthalate	2.5 – 6.1	4.7 (1.1)
monoisobutyl phthalate	12.8 – 16.9	14.2 (1.12)
monobenzyl phthalate	0.920 – 2.05	1.48 (0.321)

There were no apparent geographical trends between regions for the detected phthalates. However, monoethyl phthalate appears to be lower in the far north than in other regions. Phthalates were not tested in Alberta's biomonitoring phase one (AHW, 2008). Ranges of concentrations detected in individual pools, and mean concentrations and 95% confidence intervals from the 3 pthalates that met the inclusion criteria of this study are presented in Table 21.

Concentrations of mono-(2-ethylhexyl) phthalate (MEHP) detected in this study ranging from 131 to 242 ng/mL serum (weighted arithmetic mean ± 95% confidence interval: 175 ± 38.4 ng/mL serum) were significantly higher (between 6 to 30 times higher) than concentrations detected in other similar studies. Upon review of the analytical methodology, it was discovered that sample containers used for storage and analysis were plastic. Previous studies have shown that storage of blood and serum in plastic containers containing di(2-ethylhexyl) phthalate (DEHP) can lead to increased concentrations of both DEHP and its metabolite MEHP in the biological samples as enzymes present in blood serum are capable of metabolizing DEHP in to MEHP (Inoue et al. 2005).

Two other metabolites of DEHP (MEOHP and MEHHP) measured during analysis were found to be below the detection limit for the analytical method. MEOHP and MEHHP are not produced by the serum enzymes that are able to metabolize leached DEHP into MEHP, and as such their levels are considered to be more accurate representations of environmental DEHP exposure than MEHP which can be influenced by storage of serum in plastic containers (Kato et al. 2004). While it is not known whether the concentrations detected in the current analysis are accurate reflections of population levels of MEHP, it is likely that the relatively high levels of MEHP measured in the Saskatchewan samples are due to contamination. Therefore, despite the fact that concentrations of MEHP were detected above the level of detection in all of the Saskatchewan pools, it has been removed from analysis and will not be reported.

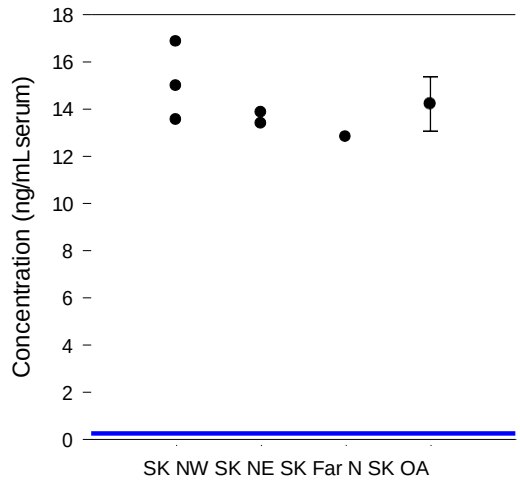

Figure 102: Concentrations of monoisobutyl phthalate in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

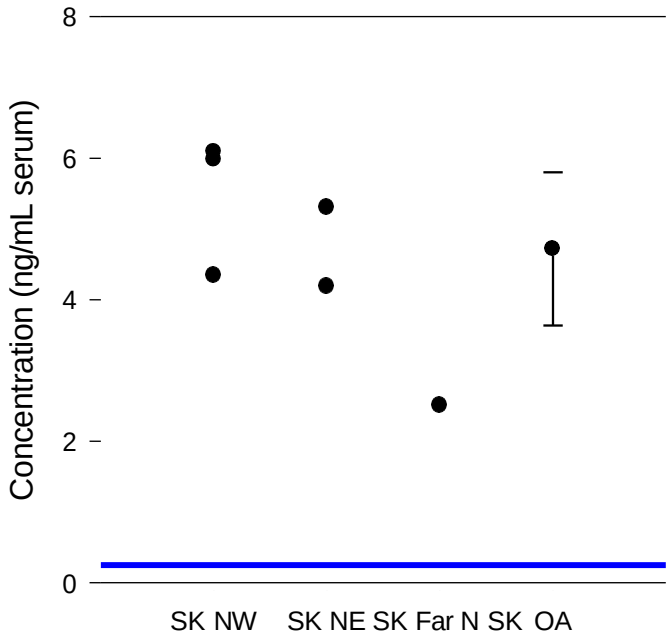

Figure 103: Concentrations of monoethyl phthalate in the blood serum of pregnant women in Saskatchewan. Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

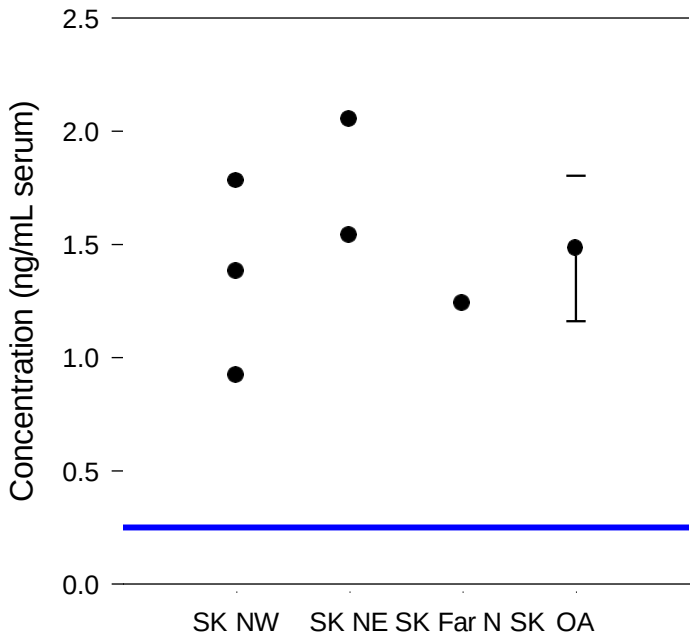

Figure 104: Concentrations of monobenzyl phthalate in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

PARABENS

GENERAL INFORMATION

Sources

Parabens are short alkyl chain esters of para-hydroxybenzoic acid. These chemicals are widely used as preservatives in cosmetics and in personal care products such as shampoos, hair and shaving products, facial and skin cleansers, and lotions. These products typically include less than 0.3% parabens, either as a mixture or a single paraben (CDC, 2013d; CDC, 2013e; Health Canada, 2014). The most frequently used parabens are generally added as antimicrobials in packaging to prevent food spoilage. All commercially used parabens are synthetically produced although some can be found naturally in certain fruits (blueberries and carrots) (CDC, 2013d; 2013e). They do not persist in the environment and break down by photolysis in the air and biodegradation in water.

The general population is exposed with use of paraben-containing personal care products, consumer foods or pharmaceuticals containing parabens. Dermal application of lotions and cleansers may result in small amounts being absorbed through the skin and into the bloodstream. However, the concentration that reaches the bloodstream is low as enzymes in the skin rapidly metabolize parabens to para-hydroxybenzoic acid. Production and usage of products containing parabens can result in the release into the environment through various waste streams.

Regulations in Canada

Parabens fall under regulation as per the *Cosmetic Regulations* under the *Food and Drugs Act.* Health Canada currently states that there is not enough evidence to suggest a causal link between paraben exposure and breast cancer (Health Canada, 2014). While they will continue to monitor and review scientific evidence produced surrounding potential effects of paraben exposure, they are currently in agreement with the U.S. Food and Drug Administration's stance on paraben use and human exposure in that there is no reason for consumers to be concerned about paraben exposure from cosmetics (Health Canada, 2014; U.S. FDA, 2014).

Possible Health Effects

Human health effects from environmental exposure to low levels of parabens are unknown. In animal studies, parabens have been found to weakly mimic estrogen but there have not been confirmed causal links in humans between cancer and parabens. In the U.S., parabens were re-examined in 2012 by the Cosmetic Ingredient Review Expert Panel and reaffirmed the safety of parabens as preservatives in the present practises of use and concentration in cosmetics (U.S. FDA, 2014).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Methyl-, ethyl-, propyl-, butyl- , benzyl butyl-, and benzyl paraben isomers were analyzed; only methyl-, ethyl- and propyl paraben were detected in pregnant women in northern Saskatchewan, ranging in concentration from 0.66 ng/mL to 14 ng/mL. Methyl paraben had the highest concentrations with a weighted arithmetic mean (± 95% confidence interval) of 9.4 ng/mL (± 2.7 ng/mL). Propyl-paraben had an average concentration of 1.6 ± 0.39 ng/mL. Parabens were not evaluated in the first phase of Alberta's biomonitoring study.

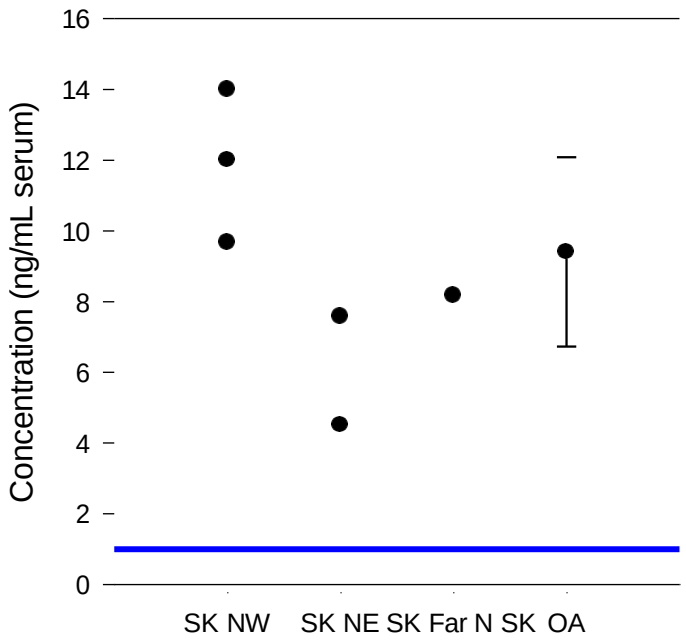

Figure 105: Concentrations of methyl paraben in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue line represents the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

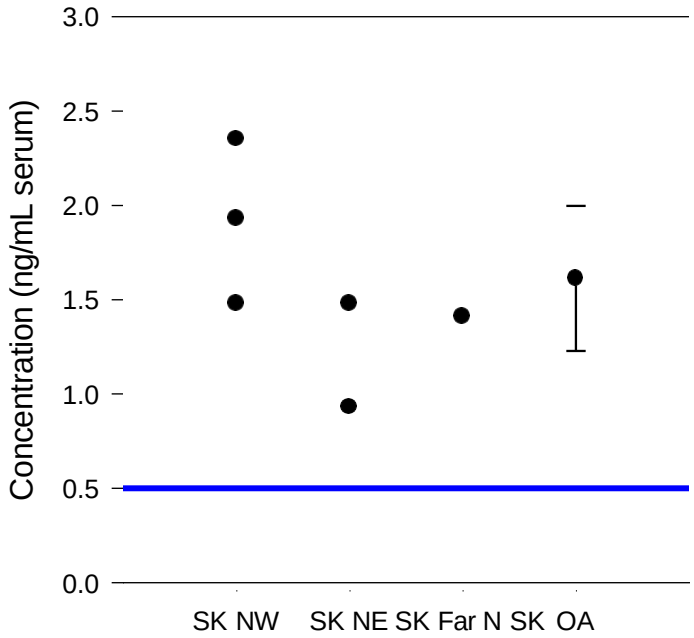

Figure 106: Concentrations of propyl paraben in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue line represents the limit of detection used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

TRACE METALS AND MINERALS

In the present study, the following metals were measured in blood serum samples of pregnant women in northern Saskatchewan:

Mineral micronutrients	Non-micronutrients
Boron	Aluminum
Cobalt	Antimony
Copper	Arsenic
Iron	Barium
Manganese	Beryllium
Magnesium	Cadmium
Molybdenum	Cesium
Nickel	Chromium
Selenium	Lead
Zinc	Mercury
	Platinum
	Silver
	Strontium
	Thallium

Titanium
Tungsten
Uranium
Vanadium

Uranium, thallium, tungsten, cadmium, arsenic, chromium, vanadium, titanium, beryllium and boron concentrations in pregnant northern Saskatchewan women had more than 1 pool below detection limits. As such, no aggregate statistics were performed on these trace metals. The limits of quantification of the analytical instruments were lower for Sb, Ba, B, Cd, Cr, Cu, Fe, Zn, Mn and V. During phase one (AHW, 2008) testing of Alberta's biomonitoring program, three separate methods optimized for different groups of metals were used. This differs from the analytical method used in Saskatchewan (single test method), thus, the quantity of non-detects between studies may be attributed to level of instrument accuracy at the time and not lack of exposure. Detailed analytical results for all detected metals are found in the subsequent sections, with an emphasis on discussion of exposure sources and possible health effects of the 'non-micronutrient' metals. Chemical analysis of metals in this study provides a measurement of total metals in the sample, that is, both inorganic and organic metals as well as metals of various speciations.

TRACE METALS (NON-MICRONUTRIENTS)

At least 37 of the elements in the periodic table have been found in the human body, 26 of which are metals present in trace amounts. Some of these metals and minerals are essential in maintaining proper function of the human body, with incorporation into cells, enzymatic processes, internal organs, and other physiological functions. Most elements found in the tissues and body fluids are also present in the blood. Non-micronutrients consist of trace metals, occasionally heavy metals, found in the human body at very low levels. They are not required in the basic function of our bodily systems. The health effects of the presence of non-micronutrients in the human body are not yet fully understood, but some of the heavy metals can have severe toxicity even at low concentrations.

ALUMINUM (Al)

GENERAL INFORMATION

Sources

Aluminum (Al) is the third most abundant chemical element and is widely distributed in mineral rocks such as silicates, hydroxides, and oxides. As such aluminum is found ubiquitously in soil, water and air in compound form (Riihimaki and Aitio, 2012; Health Canada, 2008, ATSDR, 2008). As a result of its ideal chemical and physical properties, aluminum and its alloys are used in a variety

of products including automobiles, wiring, electrical devices, paints, antiperspirants, explosives, cooking accessories, as well as an additive in pharmaceuticals and food (Riihimaki and Aitio, 2012; Health Canada, 1998; Health Canada 2008). Aluminum sulphate is also widely used as a flocculent in the treatment of drinking water (Lippmann, 1992). Despite naturally occurring in the environment, aluminum may also be released to the environment through industrial processes such as the mining and processing of aluminum ores, as well as release from the burning of coal (ATSDR, 2008).

Aluminum can be released into our homes and into the environment (air, water and soil) through use or disposal of Al-containing products and various industrial processes. Aluminum exposure occurs commonly through the ingestion of contaminated food, and to a lesser ingestion of drinking water and inhalation, and dermal contact with consumer products such as antiperspirant (Health Canada, 1998; ATSDR Sept 2008). Pharmaceuticals and occupational activities where aluminum-containing products are made represent alternative sources of aluminum exposure (ATSDR, 2008; Sjogren and Elinder, 1994; Sjogren et al., 1985; Alberta Health, 2008).

Absorption of aluminum is dependent upon exposure route, for example, aluminum is better absorbed from ingestion of drinking water than food; however, the concentration of aluminum in drinking water is such that a larger proportion still is absorbed from food. Absorption of aluminum through the gastrointestinal tract is dependent upon the composition of the food, the age of the person, the health status of the individual and the type of aluminum compound (Health Canada, 2008). Most aluminum that is ingested will be released unabsorbed in the feces, and the small amount of aluminum that is absorbed into the blood stream is excreted in the urine (ATSDR, Sept 2008).

Possible Health Effects

As with any other chemical, the human health effects of aluminum depend on the dose, the form of aluminum present in the environment, route of exposure, length of exposure, and other physiological factors (AHW, 2008); however, background concentrations of aluminum in humans are not known to cause any adverse health effects (ATSDR, 2008). Most aluminum that is ingested will be released unabsorbed in the feces, and the small amount of aluminum that is absorbed into the blood stream is excreted in the urine (ATSDR, 2008).

Exposure to aluminum in patients with kidney disease may result in the development of a type of dementia known as dialysis encephalopathy; as well there are associations with aluminum exposure and other nervous system diseases such as Parkinson's, Alzheimer's and Lou Gehrig's disease, although the significance of these associations is unknown (Health Canada, 1998). At higher doses, such as in the case of accidental releases and unusual occupational exposures, aluminum is well documented to be a human neurotoxin, and may also cause respiratory problems, kidney disease, vomiting and skin rash (ATSDR, 2008; Polizzi et al., 2002).

Aluminum is commonly sampled using urine, whole blood, bone, feces, plasma or serum samples, with plasma and serum samples giving identical results (Riihamki and Aitio, 2012, ATSDR, 2008). However, due to the fact that anticoagulants such as heparin may contain aluminum serum is preferred over plasma samples (Riihimaki and Aitio, 2012). There are mixed results concerning the elongated retention time of aluminum in erythrocytes compared to retention in serum that may make whole blood a preferable sampling medium. The current study utilizes serum concentrations of aluminum and as such direct comparison of this study to other studies which utilize whole blood concentrations of aluminum may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of aluminum ranged from 6.42 µg/L to 15.0 µg/L (weighted arithmetic mean ± 95% confidence interval: 9.01 µg/L ± 2.66 µg/L) in the 6 pools of pregnant women sampled from northern Saskatchewan. While pool 1 (NW) presented the highest concentration, there were no apparent regional differences in mean concentrations. Compared to Alberta, which has an overall serum concentration (± 95% confidence interval) 22 ± 0.88 µg/L, the blood serum concentrations of pregnant women in northern Saskatchewan are lower.

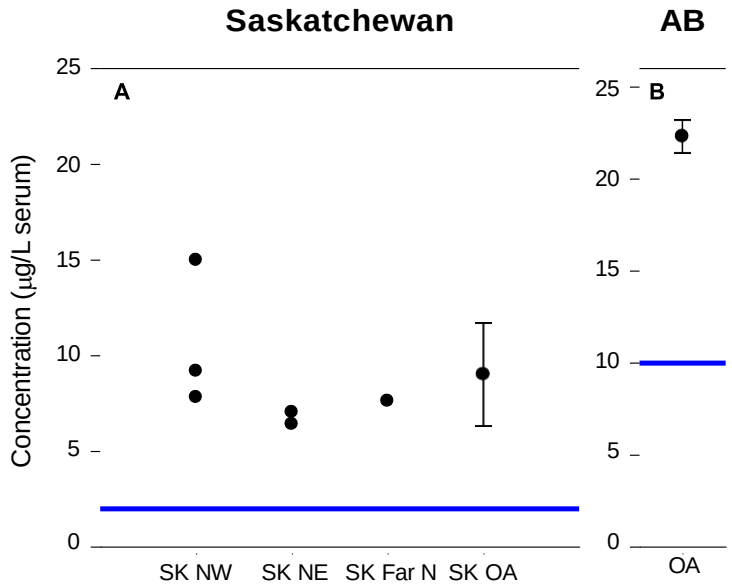

Figure 107: Concentrations of aluminum in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Alberta data is presented with an overall mean concentration of all pools. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

ANTIMONY (Sb)

GENERAL INFORMATION

Sources

Antimony (Sb) is a naturally occurring chemical element that can enter the environment through natural processes such as weathering of rocks and minerals and run off from soil, in addition to release via anthropogenic activities such as industrial effluence and leaching from materials used in plumbing (Food Inspection Agency of Canada, 2011; Health Canada, 2017; ATSDR, Sept 1992). Antimony exists in multiple forms and compounds in the environment such as oxides, fluorides and hydrides. Such antimony compounds are used in a wide variety of products including semiconductors, batteries, paints, ceramics and fireworks (ATSDR, 1992). Antimony is used in the manufacture of ceramics, glass, pigments, flame retardants, batteries, as well as in polyethylene terephthalate (PET) plastic in the form of antimony trioxide (FIAC, 2011). Antimony can enter the water system naturally through erosion and runoff from soil, as well as from industrial effluent and from leaching of plumbing materials (Health Canada, 2012; ATSDR, 1992).

Exposure to antimony may occur through ingestion of food, and to a lesser extent from inhalation, dermal contact with soil or materials containing antimony and drinking water (CDC, 2013f; ATSDR, 1992). Dietary exposure may occur through ingestion of food and drinks that may have been contaminated with antimony due to contact with food contact materials such as utensils or food storage containers containing PET. People may be exposed to higher antimony concentrations in occupational settings where antimony-containing products are manufactured or used (ATSDR, 1992). The current study utilizes serum concentrations of antimony and as such direct comparison of this study to other studies which utilize whole blood or urinary concentrations of aluminum may not be accurate.

Possible Health Effects

As with other chemical exposures, health effects arising from exposure to antimony depend on the dose, the form of antimony present in the environment, the length and timing of exposure, and other physiological factors (AHW, 2008). Antimony is not metabolized by the body, so antimony itself is used as a biomarker of exposure and elevated exposures to antimony can be measured using blood, urine, hair and feces (ATSDR, 1992). Background concentrations of antimony in humans are not known to cause any adverse health effects (ATSDR, 1992); however acute exposures to high doses may cause diarrhea and vomiting as well as cause irritation to mucous membranes following inhalation and irritation to the skin and eyes, whereas chronic exposure may lead to increased blood cholesterol and hypoglycemia (CDC 2013f; FIAC, 2011). Animal studies have shown health effects such as degeneration of the lung, liver, heart muscle, and kidney following high levels of exposure

(CDC, 2013f). The International Agency for Research on Cancer has determined that antimony trioxide is possibly carcinogenic to humans.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of antimony ranged from 3.3 µg/L to 3.8 µg/L (weighted arithmetic mean ± 95% confidence interval: 3.5 µg/L ± 0.16 µg/L) in the 6 pools of pregnant women sampled from northern Saskatchewan. There was no apparent difference between regions. Mean concentrations between northern Saskatchewan and Alberta are similar, with individual pools in Alberta exhibiting slightly higher mean values. The overall mean serum concentration in Saskatchewan overlaps with the mean ± 95% confidence interval in southern Alberta (3.50 ± 0.319 µg/L), and is lower than the mean of northern (4.09 ± 0.0941 µg/L) and central Alberta (3.95 ± 0.0844 µg/L).

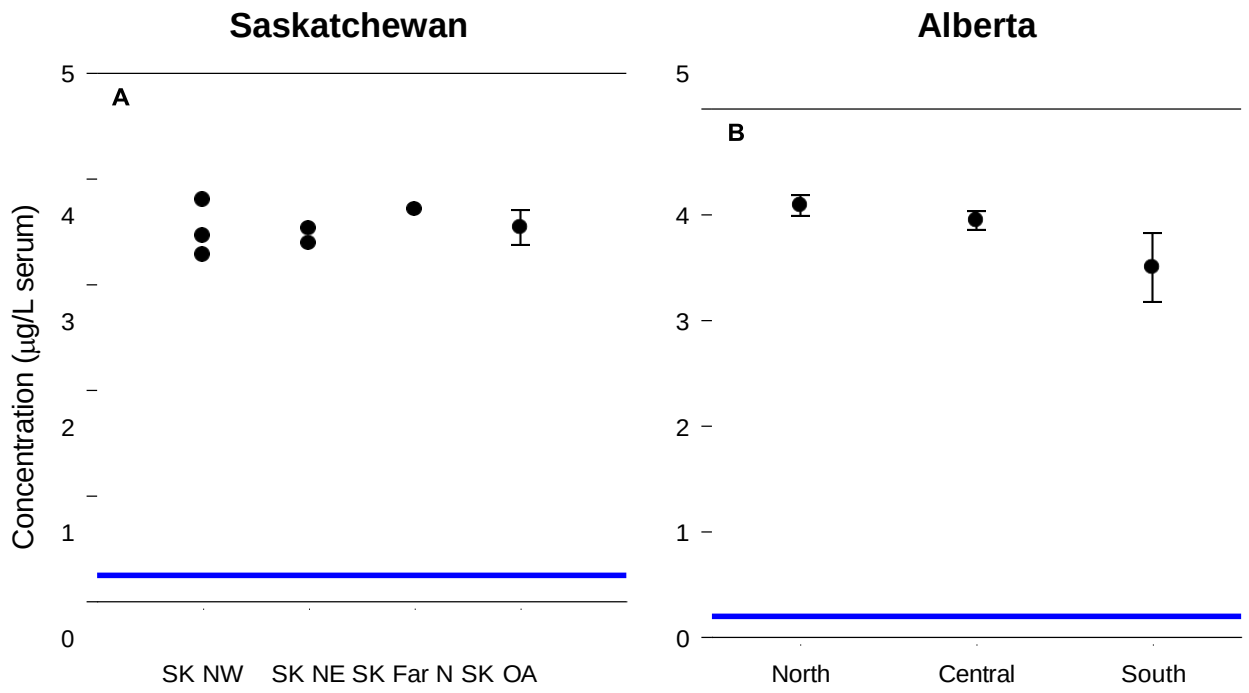

Figure 108: Concentrations of antimony in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The Alberta data presented are the mean concentrations of the pools from each region. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

ARSENIC (As)

GENERAL INFORMATION

Sources

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic can combine with other elements to form inorganic arsenic compounds or it can combine with carbon and hydrogen to form organic arsenic compounds (ATSDR, 2004b). Inorganic arsenic is mainly used to preserve wood. Copper chromated arsenic is used to make pressure-treated lumber but is no longer used for residential purposes; it is still used for industrial applications. Organic arsenic compounds can be used as a pesticide, primarily on cotton plants. It is released into the environment from several industrial processes and is predominately released during the generation of power from coal-fired furnaces. Arsenic compounds are also widely used in agricultural and silvicultural products, and small quantities are utilized as a feed additive to boost immune systems and assure rapid disease-free growth (Lippmann, 1992).

There are numerous ways in which a person may become exposed to low levels of arsenic. A person normally takes in small amounts of arsenic via the air, water and food. Food is the major source of intake with total arsenic concentrations being highest in seafood. Several organic arsenicals, generally felt to be essentially nontoxic, accumulate in fish and seafood (and sometimes referred to as 'fish arsenic') (ATSDR, 2004b; Health Canada, 2013). In the Canadian Total Diet Survey, marine fish, fresh water fish, and canned fish contribute substantially to the total arsenic intake; however, this is contributing mostly organic, and essentially nontoxic forms of arsenic to the diet. (Health Canada, 2016). Certain geographical areas may naturally contain higher levels of arsenic in the rock resulting in higher concentrations in soil or water. Exposure may occur through the occupational environment or during home renovations where arsenic-treated wood sawdust may be released (CDC, 2013g).

Possible Health Effects

Inorganic arsenic has been associated with human toxicity (ATSDR, 2004b). Ingestion may result in gastrointestinal irritation and decreased production of red and white blood cells. Inhalation may result in a sore throat and irritated lungs. A characteristic effect of long-term arsenic exposure is a pattern of skin changes such as patches of darkened skin and the appearance of small corns or warts on the palms, soles and torso (CDC, 2013g). Arsenic toxicity symptoms also include death, hyperkeratosis, Blackfoot disease, myocardial ischemia, liver dysfunction, epithelioma and hypertension. The current study utilizes serum concentrations of arsenic and as such direct comparison of this study to other studies which utilize whole blood concentrations of arsenic may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of total arsenic ranged from 0.0703 µg/L to 0.145 µg/L in pools 1, 4 and 6 from the northwest (Pool 1), northeast (Pool 4) and the far north (pool 6). These total arsenic values do not distinquish the various forms of inorganic versus organic arsenic which have different degrees of toxicity. As 3 pools were below the limit of quantification, arsenic did not meet the inclusion criteria for analysis of summary statistics. Concentrations for arsenic in pregnant Albertan women were not reported. Results from Cycle 1 and Cycle 2 of the CHMS (Health Canada 2010a; Health Canada, 2013) for women aged 6 – 79 years were 15.78 µg/g and 9.2 µg/g creatinine in urine, respectively. These results cannot be directly compared to those of northern Saskatchewan due to differences in chosen biological matrix.

Higher levels in the Far N pool may be a result of the essentially non-toxic organic form common in fish as significantly higher fish consumption rates have been documented in the more northernly parts of Saskatchewan (with higher levels in the Boreal Shield compared to Boreal Plains and the Prairies) (Chan, et al., 2018).

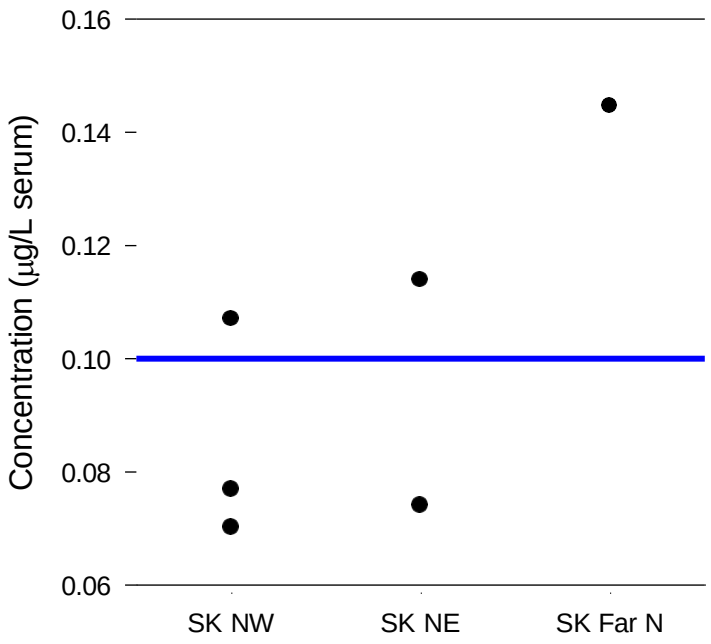

Figure 109: Serum concentration of arsenic in pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue line represents the analytical limit of quantification.

BARIUM (Ba)

GENERAL INFORMATION

Sources

Barium is a naturally occurring trace element found in sedimentary and igneous rocks, and is found predominantly in compounds rather than in a free elemental state (Health Canada, 1990; CCME, 2013). Alberta, Prince Edward Island and Saskatchewan are the only Canadian provinces in which barite deposits have not yet been found (CCME, 2013). In the environment, barium exists as various forms of barium salts such as sulphate, carbonate, nitrate and chlorate. Globally, 85% of barite is used as a drilling fluid additive, 10% is used in the chemical sector, and 5% as fillers (CCME, 2013). Barium compounds have many industrial uses and are used in the textile, rubber, oil and gas, glass, ceramic and rubber industries, as well as uses as an additive in pharmaceuticals and cosmetics (Health Canada, 1990; CCME, 2013). Barium can be released into the environment through use or disposal of barium containing products, and as well as through mining, burning of coal and fossil fuels, and various other industrial processes. The solubility of barium compounds in water is dependent upon the salt that it is bound to and the solubility of barium compounds in aquatic ecosystems increases with decreasing pH (CCME, 2013). Acetate, nitrate and halide salt soluble in water, and oxalate, phosphate and sulphates salts being insoluble (Health Canada, 1990). Barium is found ubiquitously in the soil, with higher concentrations occurring nearer to natural deposits of barium; certain plants can accumulate barium in their tissues when grown in soil contaminated with barium (Health Canada, 1990; CCME 2013).

Exposure routes include inhalation, and ingestion of drinking water and food; however, people may be exposed to higher concentrations of barium in occupational settings where barium-containing products are made (ATSDR, 2007a; Health Canada, 1990). Foods that have been found to contain high amounts of barium include milk, flour, potatoes, as well as some cereal products and nuts. However, most foods contain less than 0.002 mg/g of barium (Health Canada, 1990). Based on an average consumption of 1.5 L of water/day and the median concentration of barium in distributed water, an average person ingests approximately 0.03 mg/day of barium. The amount of barium inhaled is negligible compared to the amount ingested from all sources. Barium has also been measured in human breast milk which represents an exposure route to infants (CCME, 2013). The current study utilizes serum concentrations of barium and as such direct comparison of this study to other studies which utilize whole blood concentrations of barium may not be accurate.

Possible Health Effects

Health effects of barium depend on the dose, the form of barium present in the environment, the length and timing of exposure, and other physiological factors. Barium is not considered essential to human to health (Health Canada, 1990). The absorption of barium into the body via the gastrointestinal tract and lungs is dependent upon the solubility of the barium salt, in addition to diet, age and contents of the gastrointestinal tract (Health Canada, 1990; ATSDR, 2007a). Barium salts, such as barium sulphate, that are less water soluble have considerably fewer negative health effects because they are not absorbed as well into the body (ATSDR, 2007a). However, soluble barium salts are extremely toxic at high doses causing vasoconstriction of the arteries, as well as convulsions and paralysis (Health Canada, 1990). Other symptoms of acute barium toxicity include vomiting, diarrhea, abdominal pain and death (CCME, 2013; ATSDR, 2007a).

Absorbed barium is distributed in the blood plasma where it primarily travels to and is stored in the bone and connective tissue, with smaller amounts measureable in the skin and fat. Approximately 7% of absorbed barium is excreted in the urine as the main excretory route of barium is fecal (Health Canada, 1990). The majority of barium that is absorbed into the body is eliminated within 2 weeks (ATSDR, 2007a). While barium can be measured in the feces, urine, blood and bone, there is no data that links internal concentrations of barium with exposure levels (ATSDR, 2007a).

Background concentrations of barium in humans are not known to cause adverse health effects; however, long-term exposures of high exposures may lead to adverse health effects such as effects to the nervous, cardiac, respiratory and digestive systems, as well as general weakness, vomiting and muscular paralysis (ATSDR, 2007a). The US EPA recommends that barium in drinking water should not exceed 2.0 mg/L. The Occupational Safety and Health Administration (OSHA) has an enforceable exposure limit of 0.5 mg of soluble barium per cubic meter of air averaged over an 8 hour work day, and NIOSH considers exposure to barium chloride of levels above 50 mg/m^3 as being dangerous to life or health (ATSDR, 2007a).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of barium ranged from 2.63 µg/L to 3.53 µg/L (weighted arithmetic mean ± 95% confidence interval: 3.17 µg/L ± 0.263 µg/L) in the 6 pools of pregnant women sampled from northern Saskatchewan. Concentrations from northern Saskatchewan are lower than those detected in Alberta (AHW, 2008), where concentrations among the pools ranged from 5.11 µg/L to 14.7 µg/L. The overall mean serum concentration in Saskatchewan is lower than the means of pregnant women in Alberta regardless of age group or region. Means in Alberta stratified by age and region ranged from 6.63 ± 0.322 µg/L to 11.3 ± 0.753 µg/L.

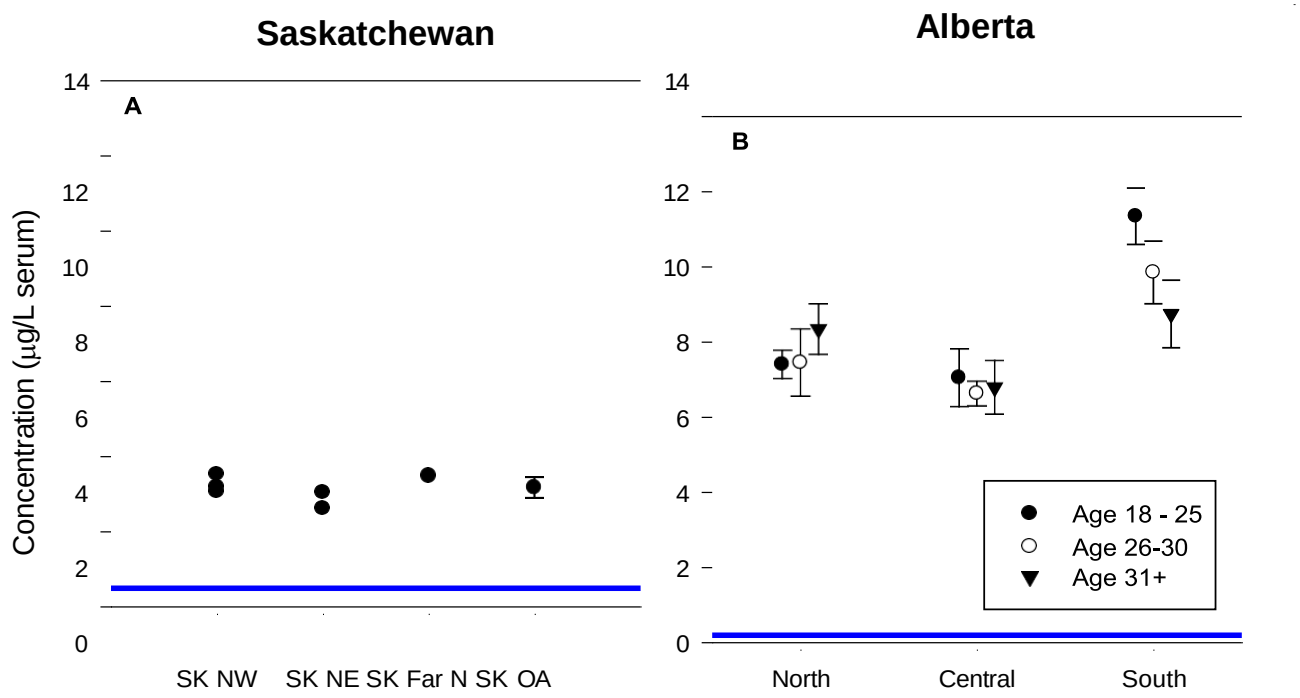

Figure 110: Concentrations of barium in the blood serum of pregnant women in Saskatchewan (A) and Alberta by geography and age (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by both age and region are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

CADMIUM (Cd)

GENERAL INFORMATION

Sources

Cadium is a soft, silver-white metal that is generally extracted as a by-product during the refinement of other metals such as zinc, copper or lead (Health Canada, 1986). Cadmium is used in electroplating metals to inhibit corrosion and in pigments and heat stabilizers for plastics production. Cadmium alloys are commonly used in soldering and brazing as well as in Ni-Cd batteries (Lippmann, 1992). Cadmium chloride and cadmium sulfate are soluble in water. Cadmium is also used in pigments, coatings and platings, and as stabilizers for plastics, among other uses (Health Canada, 1986).

Cadmium is naturally found in the soil; however, its mobility is dependent upon environmental conditions. It is typically strongly bound to organic constituents and will be taken up by the roots of plants thereby entering the food chain (ATSDR, 2012a), primarily cereal grains, vegetables and tobacco (Charania et al., 2014). Cadmium is also likely to enter the groundwater from

industrial and municipal wastes, as well as through leaching from soldering, black or galvanized

piping (Health Canada, 2017; Health Canada, 1986). The solubility of cadmium in water is influenced by the acidity of the environment and increases in acidity may lead to dissolution of cadmium from suspended or particle bound compounds (WHO, 2011). High concentrations of cadmium can also be found in the air of industrialized areas near smelters (Health Canada, 1986).

Cadmium is released to the environment as a result of natural processes including forest fires, vocanic emissions, and weathering of soil and bedrock. (Morrow, 2000). Cadmium is emitted to soil, water and air by non-ferrous metal mining and refining, manufacture and application of phosphate fertilizers, fossil fuel combustion, and waste incineration and disposal. It can accumulate in aquatic organisms and agricultural crops. In smokers, inhalation of cigarette smoke is the major source of cadmium exposure (Health Canada, 2013). The cadmium content of cigarettes typically ranges from 1 to 2 µg per cigarette. Tobacco leaves accumulate high levels of cadmium from the soil and as such, direct measurement of cadmium levels in body tissues has revealed that smoking roughly doubles the cadmium body burden in comparison to not smoking (CDC, 2013i). For non-smokers, food is the primary source of cadmium exposure with ingestion of contaminated water secondary (WHO, 2011; Health Canada, 1986; ATSDR, 2012a). The primary food sources of cadmium exposure is ingestion of leafy green vegetables, potatoes, grains and peanuts. The occupational guideline for blood cadmium is 5 µg/L (INSPQ, 2008).

Cadmium is taken up by lichens and other plants (including willow), thereby accumulating in the organs of herbivorous animals that are then consumed by Aboriginal populations (Charania et al., 2014, Gamberg M et al., 2005). The First Nations Food, Nutrition and Environment Survey (FNFNES) for Alberta suggested that smokers who consumed large amounts of organ meats are at greater risk of cadmium toxicity.(Chan, et al., 2016) The FNFNES for Saskatchewan found that for both the average and high traditional food consumers, the risk of harm from cadmium consumption through traditional foods is negligible. (Chan, et al., 2018) The A study in northern Saskatchewan moose revealed cadmium levels in moose liver in northern Saskatchewan (Thomas et al., 2005, Population Health Unit, 2005) were lower than levels found in southern Saskatchewan, Ontario (Glooschenko et al., 1988), Yukon (Gamberg et al., 2005), northern British Columbia (Jin et al., 2004), Manitoba (Crichton et al., 2000) and Alaska (Arnold et al., 2006).

With Aboriginal smoking rates approximately double that of the general non-Aboriginal population in Canada (Irvine et al., 2011), increased cadmium exposure poses a potential health issue. Results of a study conducted by Charania (2014) in nine First Nations communities in northern Quebec found a significant and positive partial correlation with cadmium concentrations and the number of cigarettes smoked daily. It was found that traditional food consumption was not associated with higher cadmium levels. Studies in the Northwest Territories where cadmium levels are elevated in moose organs, confirmed that smoking was the main contributor to cadmium exposure (Ratelle et al., 2018)

Possible Health Effects

Cadmium enters the body primarily through ingestion or inhalation. Absorption to the body various with the route of exposure with about 25% of inhaled cadmium being absorbed compared to between 4 to 7% for ingestion though intestinal absorption is increased with iron-deficiency. (Health Canada 1986). Cadmium is distributed to all major organs but the liver and kidneys are the main storage sites. Within minutes of exposure cadmium is measurable in blood plasma and within 24 hours is distributed to blood cells, where it is bound to the protein metallothionein. Only a small amount of cadmium is excreted, mainly through urine and feces, and a negligible amount is released in the hair, nails and sweat (Health Canada, 1986). Cadmium accumulates in the kidneys over a lifetime, but blood concentrations reflect mainly recent exposure, while urine concentrations reflect both recent and cumulative exposure (Fontaine et al., 2008). Chronic, low-level exposure to cadmium can cause a build-up in the kidneys, and, since urinary excretion is slow with a biological half-life ranging from 10-30 years, kidney damage may occur.

Exposures to high concentrations of cadmium can lead to acute toxicity symptoms such as vomiting, headaches, chills, pulmonary edema and stomach cramps (Health Canada, 1986). Long-term exposure to cadmium has been associated with skeletal deformations, lumbar pain, myalgia, and renal effects such as proteinuria in which the urine contains a larger than expected amount of proteins which suggests problems in the kidney's ability to properly filter blood (Health Canada, 1986).

The International Agency for Research on Cancer (IARC) has determined cadmium to be a human carcinogen and the US EPA has determined cadmium to be a probable human carcinogen (ATSDR, 2012a). Due to binding of cadmium to metallothien of red blood cells within 24 hours of exposure (Health Canada, 1986), direct comparisons of serum concentrations of cadmium (as is measured in this study) to other studies which use whole blood concentrations may not be accurate and may create artificial differences in the data between study populations.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

While all pools were below the limit of quantification (<0.05 µg/L), pool 6 (far N) had a concentration of 0.050 µg/L. The Canadian Health Measures Survey Cycle 1 and Cycle 2 found geometric mean concentrations in women aged 6 -79 years to be 0.38 µg/L and 0.34 µg/L in whole blood, respectively (Health Canada, 2010a; Health Canada, 2013); however, because this was whole blood versus serum, these values are not comparable to the values measured in northern Saskatchewan. The First Nations Biomonitoring Initiative (2013) also reported blood geometric mean cadmium concentrations of 1.00 (95% CI: 0.80 – 1.25) µg/L in females aged 20 years and older on reserve and crown land. Cadmium was not reported in phase one (AHW, 2008) of Alberta's

biomonitoring program as fewer than 25% of the pools had concentrations above the analytical limit of quantification. Serum concentrations of cadmium measured in pregnant women sampled from northern Saskatchewan are approximately ten times lower than those in comparable populations across Canada measured in whole blood; however, cadmium concentrations in pregnant women in northern Saskatchewan measured in blood serum cannot be directly compared to concentrations detected in populations across Canada because CHMS analyzed whole blood samples for cadmium, and partitioning of cadmium into red blood cells may affect analysis.

CESIUM (Cs)

GENERAL INFORMATION

Sources

Cesium is a naturally occurring element found in rocks, clay and soil (CDC, 2013j; ATSDR, 2004c). Natural cesium exists as a stable isotope (^{133}Cs) and as well as existing as various naturally occurring compounds including hydroxides, carbonates, iodides and bromides. Inorganic cesium compounds have use in scintillation counters, infrared lamps, vacuum tubes, and polymerization catalysts, among other uses. Radioactive forms of cesium are produced during the fission of uranium in nuclear power plants and these radioactive forms eventually decay into stable atoms (ATSDR, 2004c). Cesium can be released into the environment through the natural weathering of rocks, as well as from mining and milling of ores. Cesium is mined in southeastern Manitoba for the production of a biodegradable lubricant fluid used in oil drilling. (Government of Manitoba).

In addition, radioactive cesium, a form of cesium not included in this study, is released into the environment as a result of nuclear processes but not by uranium mining. Cesium can travel long distances in the air before settling via gravitation settling or by precipitation. Further, cesium compounds are generally very soluble in both water and moist soil; however, cesium compounds bind strongly to soil particles and thus do not migrate far in soil. Vegetables and plants do not readily take up cesium via their roots.

Exposure to cesium can come through inhalation of ambient air, and ingestion of drinking water and food (ATSDR, 2004c; CDC, 2013j). Levels of cesium in water and air are generally quite low therefore most environmental exposure to cesium comes through ingestion of food. Tea and coffee contributes to the largest consumption source of cesium by the average Canadian adult though yeast, herbs and spices have high concentrations. (Health Canada, 2016). Lichens are also high in cesium, which is a major dietary source for caribou. Studies in lichen and caribou in the Northwest Territories (Larter et al., 2016) and Saskatchewan (Personal communication J. Irvine) show realtively high levels of stable cesium. People who work in occupational settings where cesium containing products are made may be exposed to higher concentrations of cesium (CDC, 2013j). Cesium

chloride is sold as an oral alternative cancer therapy though there is no evidence of effectiveness and doses recommended can cause significant exposure and some health risks (Health Canada, 2009f)

Soluble cesium compounds are readily dissolved in the blood and distributed through the body. Cesium is accumulated in the kidneys and is eliminated primarily in the urine, as well as in the feces. Some absorbed cesium will persist in the body for weeks to months, slowly being eliminated. Exposures to both radioactive and stable cesium compounds are detectable in the blood, urine, feces and body tissues

Possible Health Effects

Similar to other chemical exposures, the human health effects of cesium depend on the dose, the length and timing of exposure, and other environmental and physiological factors (AHW, 2008). Background concentrations of cesium in humans are not known to cause any adverse health effects and there is a limited amount of human studies investigating possible health effects from long term exposure to cesium (ATSDR, 2004c). Ingestion of extremely large doses of cesium chloride, such as from alternative medications, has been known to result in vomiting, diarrhea, and cardiac arrhythmia (CDC, 2013j). Cesium in general is considered to have low toxicity when used in animal studies and little is actually known about the health effects of low, environmental exposures to humans. Stable cesium is thought to be of low toxicological concern for humans (ATSDR, 2004c).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Cesium analyzed in this study represents cesium 133 (stable cesium or non-radioactive cesium) and does not represent concentrations of total cesium. Concentrations of cesium ranged from 0.29 µg/L to 3.5 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 0.85 µg/L ± 1.0 µg/L). Due to an extremely large confidence interval, there is overlap between the overall mean serum concentration in pregnant women sampled from northern Saskatchewan and in pregnant women from all regions and age groups of Alberta (AHW, 2008). In general, results from northern Saskatchewan are similar to those of Alberta with the exception of the far north (pool 6), which had the highest mean concentration of cesium. Alberta trended towards lower concentrations to the north and higher concentrations to the south, and a range of concentrations of 0.370 to 0.750 µg/Lwere detected in the pools sampled in Alberta.

The elevated concentrations of cesium in pool 6, which are a magnitude higher than concentrations detected in northern Alberta, may be attributed to differences geological crustal features and the higher levels of consumption of caribou, which consume lichen in abundance. Differences in traditional activities such as subsistence hunting and consumption of country foods

may play a role; however, biomonitoring data represents body burden from all sources and does not allow us to determine exposure sources, therefore these are only speculations.

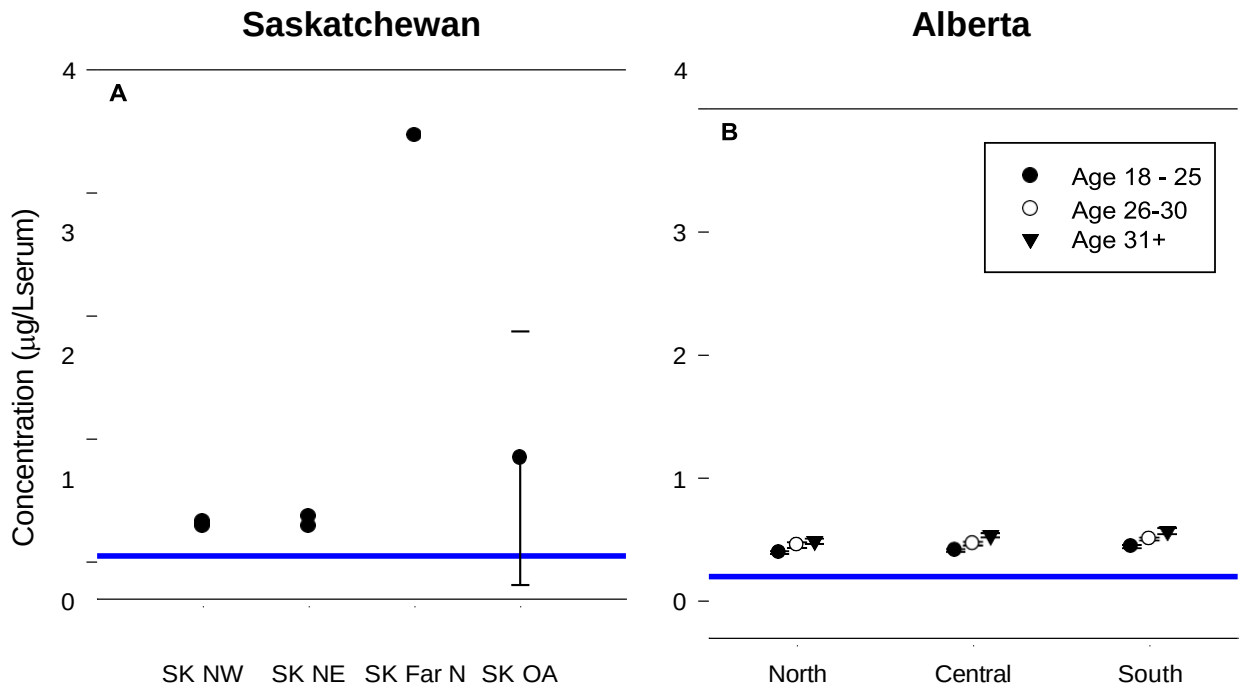

Figure 111: Concentrations of cesium in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by both age and region are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

CHROMIUM (Cr)

GENERAL INFORMATION

Sources

Chromium is a grey metal that can be found in 9 different oxidation states ranging from –II to +VI and is widely distributed throughout the earth's crust (CCME, 1999c; WHO, 2003; ATSDR, 2012b). The type of chromium species found in an environment depends on the characteristics of the environment itself. For example, hexavalent chromium is the predominant chromium species found in surface waters and aerobic soil environments, while trivalent chromium dominates in reducing environments such as sediments and wetlands. Hexavalent chromium salts are more soluble than trivalent chromium compounds, making them the more mobile species (WHO, 2003). Chromium compounds such as chromium oxide, chromium sulphate, and chromium chloride are used as wood preservatives, in metal plating, leather tanning, as a catalyst, and as an ingredient in pigments, paints

and fertilizers. Natural sources of chromium release into the atmosphere include emissions from volcanoes, forest fires, and marine aerosols (CCME, 1999c). Chromium is released into aquatic environments in effluent from tanneries, pulp and paper mills, cement and fertilizer plants, and cool tannings among other industrial sources. Chromium has not been found to bio-accumulate in the bodies of organisms living in contaminated aquatic environments (CCME, 1999c). Chromium released to the air is typically found in aerosol form and can be deposited through both wet and dry deposition (WHO, 2003).

The general population is most likely to be exposed to trace levels of chromium in food. Low levels of the non-toxic form of chromium (chromium III) is found naturally in a variety of foods, such as fruits, vegetables, nuts, and meats. However, the general population is exposed to the more toxic forms of chromium most often by ingestion of contaminated foods, but also through tobacco smoking and contact with older forms of pressure treated lumber (with chromated copper arsensate). (ATSDR 2012b.) Ingestion of contaminated food is the major source of chromium in the general population; however, in areas where drinking water contains more than 25 µg/L of chromium, ingestion of drinking water contributes significantly to exposure. People may also be exposed to higher chromium concentrations in occupational settings where chromium containing products are manufactured or used (ATSDR, 2000b).

Possible Health Effects

As with any other chemical exposure, the human health effects of chromium depend on the dose, chromium speciation in the environment, the length and timing of exposure, and other physiological factors (AHW, 2008). As an essential element, chromium (III), is considered an essential element in human metabolism and thus is expected to be found in the blood and urine (Health Canada, 2012; ATSDR, 2012b). Hexavalent chromium is able to cross cell membranes, while trivalent chromium cannot; it is reduced to trivalent chromium and forms adducts with macromolecules (WHO 2003; ATSDR 2012b). Chromium (VI) may accumulate in tissues such as the lymph nodes, liver, spleen, kidneys and lungs. Fecal elimination if the main route of excretion for trivalent chromium, which is poorly absorbed, whereas urine represents the major route of excretion for absorbed chromium (WHO, 2003).

The International Agency for Research on Cancer has classified hexavalent chromium as being a human carcinogen, while trivalent chromium was determined to be unclassifiable in regards to its potential as a carcinogen (WHO, 2003). The most common health ailment found in workers occupationally exposed is irritation of the respiratory tract and breathing problems (ATSDR, Sept 2012b). As well animal studies have shown effects on the male reproductive system, irritation and ulceration of the gastro-intestinal tract, as well as miscarriage and low birth weights in infants. The absorption and toxicokinetics of chromium in the human body appear to be dependent upon the species of chromium compound with hexavalent chromium being absorbed to a greater extent in the gastrointestinal tract than trivalent chromium (WHO, 2003). Therefore following exposure, trivalent

chromium would only be detectable in the blood plasma, whereas hexavalent chromium is detectable both in blood plasma and in the erythrocytes.

In addition to differences in health effects stemming from the different species of chromium compounds, there are differences in regards to measuring exposure of each of the species. For example, hexavalent chromium is able to cross cell membranes such as the cell membranes of red blood cells where it is reduced to trivalent chromium and forms adducts with macromolecules (WHO 2003; ATSDR 2012b). Trivalent chromium cannot cross cell membraines and therefore following exposure, trivalent chromium would only be detectable in the blood plasma, whereas hexavalent chromium is detectable both in blood plasma and in the erythrocytes. Studies have found that serum and whole blood sampling cannot be used interchangeably when evaluating concentrations of chromium ions within the body (Ziaee et al., 2007). Therefore care must be taken when comparing the results of the current study which are presented as serum concentrations of chromium to other studies as direct comparisons between serum and whole blood concentrations cannot be made because they account for different species of chromium compounds.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

All pools of pregnant women sampled from northern Saskatchewan were below the limit of quantification (0.5 µg/L). Blood serum concentrations in pregnant women from Alberta had pool concentrations ranging from 0.850 µg/L to 4.62 µg/L and a mean of 1.51 ± 0.741 µg/L.

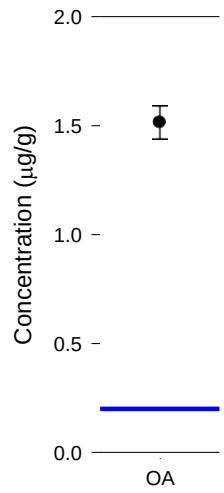

Figure 112: Overall mean concentration of chromium in the blood serum of pregnant women in Alberta (AHW, 2008). Data presented is an overall (OA) arithmetic mean of all of the pools included in analysis. Estimate represents 95% concentration around the mean, and the analaytical limit of quantification is represented by the blue line.

LEAD (Pb)

GENERAL INFORMATION

Sources

Lead is the most common of the heavy metals and accounts for 13 mg/kg of the earth's crust (WHO, 2011b). In the environment, it predominantly exists as a stable ion that is able to readily form alloys with other metals, as well as being able to form organic and inorganic compounds (CCME, 1999d; CDC 2013k). While lead can be introduced into the environment from natural sources such as forest fires, emissions from volcanoes and sea salt, the majority of high levels of lead found in the environment is due to release from human activies (CCME, 1999d; ATSDR, 2007b). Lead has many uses in commercial manufacturing including being used as an ingredient in batteries, plastics, leaded glass, ceramic glaze, ammunition, and radiation shielding (CDC, 2013k; WHO, 2011b). As well lead has historically been used as an ingredient in house paint, in the soldering of food cans, plumbing and in gasoline. Lead can be released into homes and the environment through use or disposal of lead containing products, and also through mining, and various industrial processes (Reis et al., 2007). Levels of lead in the soil have been found to be elevated due to deposition from atmospheric lead as far as 35 km away from ore smelters and emission stacks (CCME, 1999d). Depending on the type of lead compound, soil compound, and pH, lead may be mobilized in the soil and contaminate both ground and surface water. As well, lead may enter the water system through leaching from plumbing materials (CCME, 1999d; Health Canada, 2017; ATSDR, 2007b).

Prior to the 1980's in North America, the primary source of exposure to lead was through inhalation or ingestion of lead aerosolized from the burning of leaded gasoline (CDC, 2013p; ATSDR, 2007b). Exposure to lead now is limited to occupational sources, deterioration of lead based paints that are still in use, leaching from lead plumbing or lead soldered piping, imported children's toys that contain lead, folk remedies, cosmetics, lead-glazed ceramics, pewter utensils or contact with water or soil that is contaminated by nearby industrial activities such as smelting (CDC, 2013k; WHO, 2011b). Lead exposure is also increased by the exposure to cigarette smoke (CCME, 1999d). Absorption, ingestion and inhalation of air and water containing trace concentrations of lead are common sources of minor exposure to the general population. Exposure to lead from various consumer products is regulated in Canada (Government of Canada, 2005).

Concentrations of lead in blood has been steadily decreasing over the past decades especially after the ban on lead gasoline, in paints and in pipes (CDC, 2012b). A number of studies have shown that lead ammunition is a source of lead exposure in subsistence hunting people in northern Canada (Tsuji 2008, Tsuji 2009). In 1999, the Government of Canada instituted regulations on the use of lead shot migratory birds. However, lead shotshell can still be legally purchased and used to hunt upland game birds and small mammals and lead core bullets for harvesting of large game is still allowed. A

1992 Quebec Public Health authority cross-sectional health survey in Nunavik found mean blood-lead levels 5 times higher than that observed in the United States for the same time period (1991-1994); however, subsequent studies following the ban on lead shot for migratory birds and a public awareness campaign, found the blood lead levels in newborns and in adults decreased significantly since the ban although levels remained elevated compared to other US and Canadian studies. (Couture et al., 2012). In the First Nations Food Nutrition and Environment Study for Saskatchewan (Chan et al., 2018), British Columbia (Chan et al., 2011), Ontario (Chan et al., 2014), Manitoba (Chan et al., 2012) and Alberta (Chan 2016), elevated lead levels were found in some of the game mammals and birds tested. These reports recommended the use of non-lead steel shot or ammunition when hunting and to cut away the portion of meat surrounding the bullet entry area to decrease the risk of lead exposure.

Like other metals, lead is detectable in human breast milk and may cross the placenta (Gundacker et al., 2002; Ong et al., 1993; Tsuchiya et al., 1984). In these ways, lead may be passed to the fetus and to infants during pregnancy and lactation, respectively (Gundacker et al., 2002; Ong et al, 1993; Tsuchiya et al., 1984). However, breastfeeding is encouraged due to the many associated health-benefits, and these outweigh any known risks from lead for the general population (American Academy of Pediatrics, 1997; Abadin et al., 1997). Lead is able to cross the placenta as early as 12 weeks into gestation and it has been found that levels of lead in the umbilical cord (and therefore in the fetal circulation) are 80-100% of the maternal blood lead levels (CDC, 2013k; CCME, 1999d; WHO, 2011b).

Possible Health Effects

Lead is absorbed and distributed in the body via the blood where it binds to erythrocytes following inhalation or ingestion of small lead particles, and can be slowly excreted through urine and breast milk. Due to the fact that lead binds to red blood cells within the blood, comparisons should not be directly made between serum and whole blood concentrations of lead. As with any chemical exposure, the human health effects of lead are diverse and depend on the dose, the length of exposure, and the timing of the exposure (AWH, 2008). Lead exerts its toxic effects via interference with the physiologic actions of minerals such as calcium, iron and zinc, as well as through inhibition of enzymes, and disruption of ion channels (CDC, 2013k). Low environmental exposures to lead in pregnant women have been associated with spontaneous abortion, premature delivery and neurotoxic effects in the developing fetus as lead is able to cross the placenta as early as 12 weeks into gestation (CDC, 2013k; CCME 1999d; WHO 2011b). Other possible health effects that may be related to lead exposure are anemia, miscarriage, still birth, and several adverse effects on the motor and sensory systems, reproductive and immune systems, of fetus/infants (NRC, 1993; ATSDR, 2007b; Thanapop et al, 2007; Luang-on et al., 2003; Tawichasri et al, 2000; Ahamed et al., 2007; AHW, 2008).

High concentrations of lead in the body have been associated with health effects such as anemia, impaired function of the kidneys, abdominal pain, and effects on the nervous system including seizures, paralysis and encephalopathy. Occupational exposures to lead have resulted in impaired reproductive functioning in men with effects such as reduced sperm count and decreased fertility (CDC, 2013k). Lead is considered a neurotoxin and produces effects such as hallucinations, headaches, dullness, muscle tremors, poor attention span and loss of memory (CCMEd, 1999; WHO, 2011b). The International Agency for Research on Cancer (IARC) has determined that inorganic lead compounds are a probable human carcinogen, while organic lead compounds are not yet classifiable as to their carcinogenicity (CDC, 2013k).

The current study utilizes serum concentrations of lead and as such direct comparison of this study to other studies which utilize whole blood concentrations of lead may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of lead ranged from 0.29 µg/L to 0.62 µg/L (weighted arithmetic mean ± 95% confidence interval: 0.48 µg/L ± 0.10 µg/L) in the 6 pools of pregnant women sampled from northern Saskatchewan. While lead was detected above the LOQ in all serum pools from northern Saskatchewan, lead concentration in blood serum of pregnant Albertan women were largely below quantification limits (< 0.20 µg/L), except for a few sample pools that ranged in concentration from 0.21 µg/L to 1.0 µg/L. The overall mean serum concentration in pregnant women in northern Saskatchewan (weighted mean ± 95% confidence interval: 0.48 ± 0.10 µg/L) is higher than the mean serum concentrations in pregnant women stratified by region in Alberta.

Comparison with results from the Canadian Health Measures Survey for these serum lead levels is not suitable as the CHMS measured whole blood as well as urine concentrations.

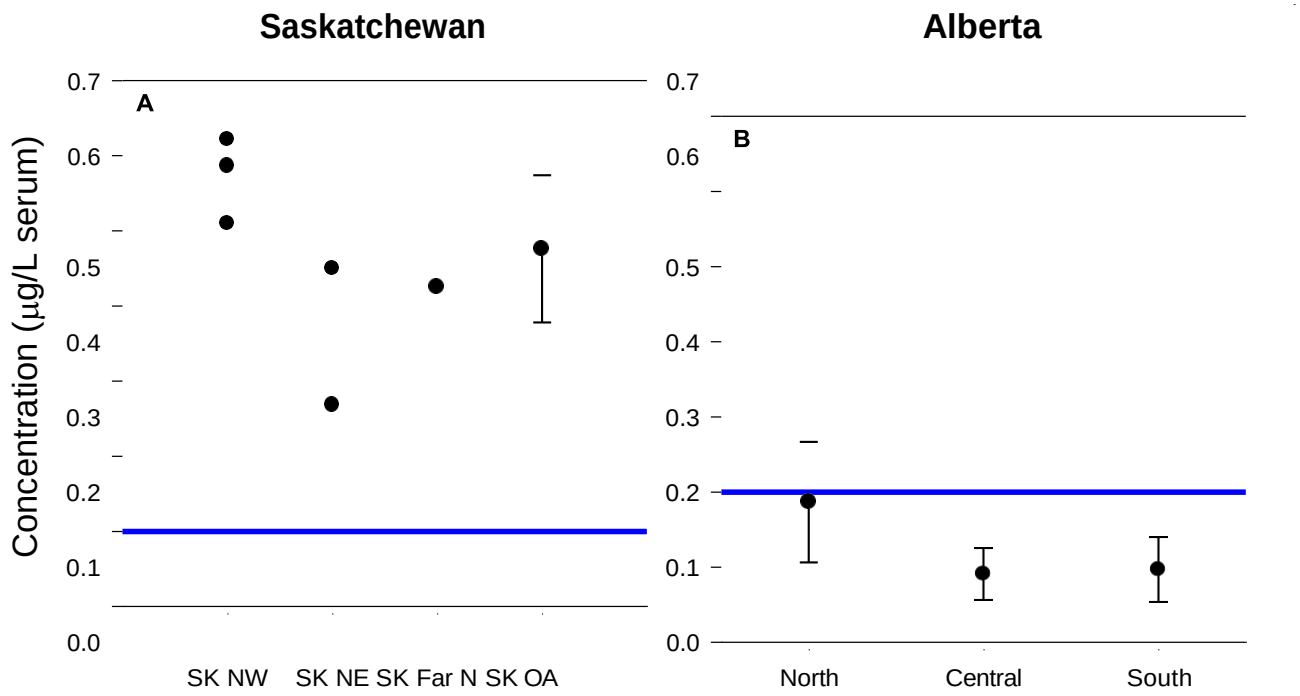

Figure 113: Concentrations of lead in the blood serum of pregnant women in Saskatchewan (A) and Alberta by geographic region (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by region are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

MERCURY (Hg)

GENERAL INFORMATION

Sources

Mercury is a naturally occurring chemical element that is widely distributed around the earth in its elemental, inorganic and organic forms (CDC 2013l; ATSDR 1999a, ATSDR 2013c; WHO 2005; Environment Canada, 2013h). It is the only metal that is liquid at room temperature. Elemental and inorganic mercury compounds are used in or found in a wide variety of industrial, commercial and medicinal products such as electrical instrument such as thermostats and switches, thermometers, batteries, antiseptics, fungicides, preservatives, and dental fillings. Its use has been greatly reduced or phased out of most products (CCME, 1999e; Environment Canada, 2010). It is still present in many lamps and light, including fluorescent lamps, mercury vapour lamps and compact fluorescent bulbs (Environment Canada, 2010).

Mercury enters the environment from natural processes such as weathering of rocks and minerals, and volcanic activity. Inorganic and elemental mercury can also be released from anthropogenic activities such as from the combustion of fossil fuels (mainly coal), mining, smelting, and other industrial processes. Mercury is not commonly found in water as it generally binds to soil and sediment; however it may enter the water system from spills or industrial effluent, irrigation run off or drainage from areas in which agricultural pesticides are in use (Health Canada, 2012). High mercury levels can be found in the Arctic regions as a result of global atmospheric circulation and long-range transboundary transport. Dependent the on form of mercury, particularly organic mercury, bioaccumulation and biomagnifications can occur.

There are many ways in which humans can be exposed to mercury such as through ingestion and inhalation of food, water, soil and air containing trace concentrations of mercury. Total blood mercury concentrations in the general population are due primarily to the dietary intake of organic mercury forms (CDC, 2013l). Food is the main source of mercury exposure in populations that are not exposed to mercury occupationally (WHO, 2005); however occupational settings where mercury containing products are manufactured or used may cause people to be exposed to higher mercury concentrations. Other routes of exposure include inhalation of mercury particles or vapor, dental fillings, and ingestion of drinking water. Approximately 80% of inhaled inorganic mercury is absorbed into blood making it the most significant route of exposure leading to internal doses of inorganic mercury (WHO, 2005; CDC 2013l). Inorganic mercury is absorbed poorly from the gastrointestinal tract with less than 15% of total exposed mercury actually absorbed (CDC, 2013l). Inorganic mercury is widely distributed throughout the body with the highest concentrations occurring in the kidneys and excretion occurs primarily in the urine.

Possible Health Effects

Health effects associated with exposure to mercury are dependent upon the length of exposure, dose and the form of mercury (CDC, 2013l); however areas of the body such as the brain and the kidneys are particularly sensitive to the effects of mercury (ATSDR, 1999a). Exposure to inhaled elemental mercury can result in health effects such as pneumonitis, as well as tremors, depression, fatigue, sleep disturbances, and neurocognitive and behavioral disturbances. Ingestion of inorganic mercury can result in irritation of the gastrointestinal tract, and once absorbed can lead to effects on the kidneys such as renal tubular necrosis. IARC has classified methylmercury as being a possible human carcinogen and inorganic mercury as being unclassifiable (CDC, 2013l). Under a high dose long-term exposure scenario elemental and inorganic mercury may cause adverse health effects such as problems with brain, kidney as well as general weakness, nausea, vomiting, skin rash and eye irritation (ATSDR, 1999a). In 2004, Health Canada established a total mercury blood guidance value of 20 µg/L for adults (Health Canada, 2004b).

The current study utilizes serum concentrations of mercury and as such direct comparison of this study to other studies which utilize whole blood concentrations of mercury may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of mercury ranged from 0.214 µg/L to 0.696 µg/L in the 6 pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 0.376 µg/L ± 0.136 µg/L). While there is no apparent trend with geography the highest concentration was detected in pool 6 (far north). The overall mean serum concentration in Saskatchewan overlaps with the mean serum concentrations in the 26 to 30 year old age group (mean ± 95% confidence interval: 0.241 ± 0.0253 µg/L), the group of Albertan women above the aveage of 31 (mean ± 95% confidence interval: 0.300 ± 0.0326 µg/L), and is higher than the concentration found in women aged 18 to 25 (mean ± 95% confidence interval: 0.161 ± 0.0214 µg/L). However, concentrations of individual pools collected in north western Saskatchewan have similar concentrations as the Albertan women aged 18 to 25. Concentrations in pregnant women in Alberta ranged from 0.204 µg/L to 0.844 µg/L with increasing concentration with age. Concentrations in northern Saskatchewan are comparable to other similar studies.

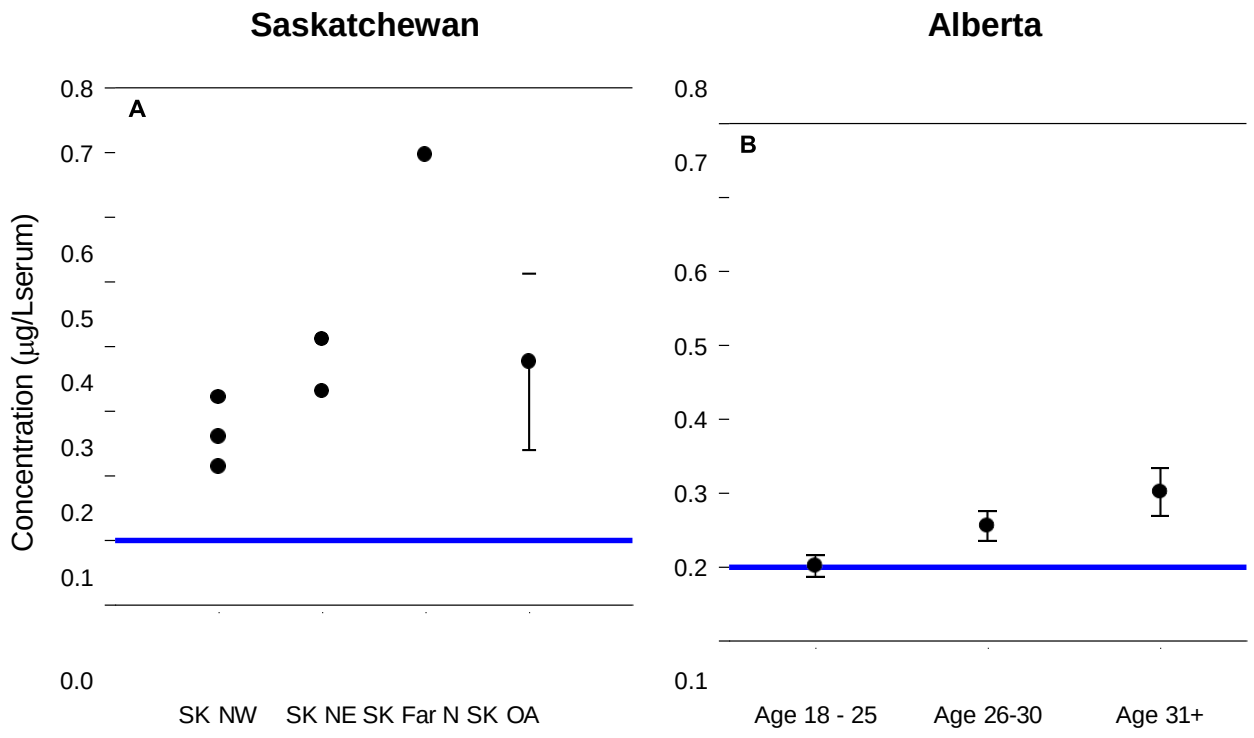

Figure 114: Concentrations of inorganic mercury in the blood serum of pregnant women in Saskatchewan (A) and Alberta by age (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by age are provided for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

A study conducted by Walker et al. (2006) in Arctic Canada (Northwest Territories and Nunavut), found the maternal geometric mean of total mercury concentrations ranged from 0.87 µg/L in whole blood samples (SD = 1.95) in the Caucasian group of participants (n = 134) to 3.51 µg/L (SD = 8.30) in the Inuit group (n = 146). The geometric mean of the Inuit group was 2.6-fold higher than that of the Dene/Métis group (1.35 µg/L, SD = 1.60, n = 92) and significantly higher than those of all other groups ($p<0.0001$). A similar trend was found in measurements of inorganic mercury in that the concentration of inorganic mercury in the Inuit group (arithmetic mean [SD]: 1.09 [0.84]) was significantly higher than the concentrations found in the other ethnic groups. Analysis was done according to ethnicity to account for the differences in traditional food species eaten by the different ethnic groups. Therefore the differences in mercury between ethnic groups are thought to be the result of differences in diet. For example, a type of inorganic mercury, mercuric selenide, makes of the majority of the mercury found in beluga whale organs. This could contribute to levels of inorganic mercury found in the Inuit group as beluga makes up an important part of Inuit diet in some areas. The results of this study cannot be directly compared to the serum concentrations analyzed in Saskatchewan and Alberta because they utilized whole blood samples.

STRONTIUM (Sr)

GENERAL INFORMATION

Sources

Pure strontium is a hard, white-coloured metal but is more commonly found in nature in the form of minerals. It turns yellow upon reacting with air. Rocks, soil, dust, coal, oil, air, plants and animals all contain varying amounts of strontium. Strontium compounds are used in making ceramics and glass products, pyrotechnics, paint pigments, and other products (ATSDR, 2004d). Strontium is available as an ingredient in over-the-counter natural health products. (Health Canada, 2005c.) Disposal of coal ash, incinerator ash, and industrial wastes may increase the concentration of strontium in soil and emissions from burning coal and oil increases stable strontium levels in air.

Strontium can also exist as four different natural isotopes and two radioactive isotopes though only stable strontium (non-radioactive) was measured in this study. Strontium-90 (radioactive form) was widely dispersed in the 1950s and 1960s in the United States as fallout from atmospheric testing of nuclear weapons. It has since been decaying to very low environmental levels. It has a half-life of 29.1 years (ATSDR, 2004d).

Possible Health Effects

Both stable and radioactive strontium enter and leave the body in the same way. Depending on the form and solubility of the strontium compound, it may reside in the lungs or bone for a long time or may be readily excreted via urine, feces or sweat (ATSDR, 2004d). Strontium behaves similar to calcium and may be incorporated into the bones mineral itself or attached to the surface of the bones, depending on age of exposure (ATSDR, 2004d). The binding of strontium to human plasma proteins is low (25%) and strontium has a high affinity for bone tissue. The effective half-life of strontium is about 60 hours.

There are no harmful effects of stable strontium in humans at the levels typically found in the environment. The Health Canada maximum acceptable concentration in Canadian drinking water for ^{90}Sr is 5 Bq/L (Health Canada, 2009c). Radioactive strontium (^{89}Sr, ^{90}Sr) over time can damage bones and the surrounding soft tissue by radiation release over time. Lowered blood cell counts have also been seen in ingestion or inhalation of radioactive strontium.

The current study utilizes serum concentrations of strontium and as such direct comparison of this study to other studies which utilize whole blood concentrations of strontium may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

Concentrations of strontium 88, a stable or non-radioactive form of strontrium, ranged from 20.5 µg/L to 39.1 µg/L in the 6 pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 26.9 µg/L ± 5.54 µg/L). The radioactive isotope of strontium, strontium 90, was not monitored in the metals method. Concentrations of strontium are not reported in other similar biomonitoring studies in Canada. The creatinine adjusted geometric mean (95% confidence interval) of strontium measured in the urine of female participants measured in the NHANES study was 105 (96.6-114) ug/L urine (CDC, 2015). A study of 280 healthy Brazilians (47% women) from the ages of 18 to 60 from 3 different Brazilian states were measured in regards to their hair, blood and plasma concentrations of various trace metals (Rodrigues et al., 2008). The mean concentration (SD) of strontium measured in plasma samples was found to be 15.4 (4.2) µg/L which is slightly lower than the concentrations measured in populations of women from nothern Saskatchewan. A study of 369 non-pregnant, non-smoking women recruited from a Reproductive Medicine Center in Xiamen China measured a median serum strontium concentration (25^{th}-75^{th} percentiles) of 57.59 (51.33-68.14) µg/L (Zheng et al., 2015). This is considerably higher than what was measured in both the Brazilian population as well as the population of pregnant women sampled in northern Saskatchewan. These differences may be due to the differences in environmentally existing strontium in these 3 areas.

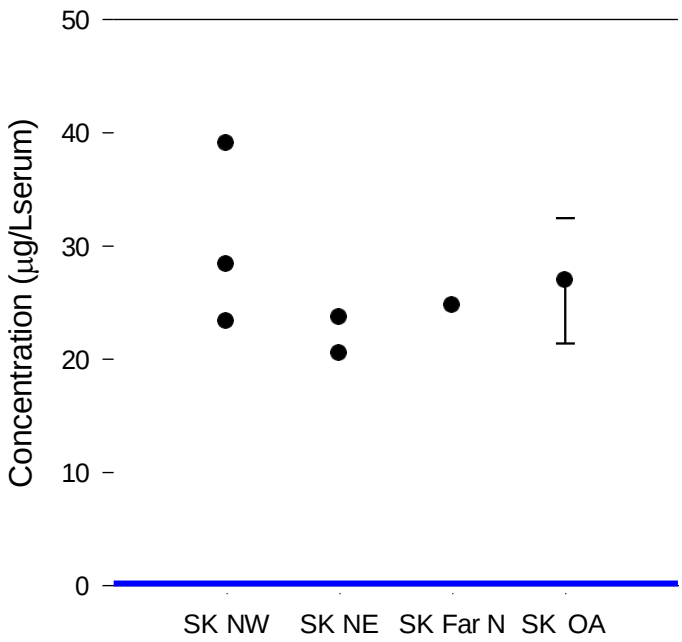

Figure 115: Concentrations of strontium in the blood serum of pregnant women in Saskatchewan. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

URANIUM (U)

GENERAL INFORMATION

Sources

Uranium is a naturally occurring element found at low levels in virtually all rock, soil and water. It is a silver-white metal that is extremely dense and weakly radioactive. Significant concentrations of uranium occur in some substances such as phosphate rock deposits. It usually occurs as an oxide and is extracted from ores containing less than 1% natural uranium. Distribution in the environment is based on climatic and geologic processes. It has a long half-life, thus the total amount on earth remains nearly the same. Natural uranium is a mixture of three isotopes: ^{238}U, ^{235}U, and ^{234}U. All three naturally occurring isotopes are radioactive. Canada is the world's second largest producer of uranium, with 15% of global production in 2012 (Government of Canada, 2014). Northern Saskatchewan is home to some of the world's most abundant and high grade uranium deposits, with several active mine/mill sites.

Uranium, when depleted as U-238, is used by the United States military as shielding to protect army tanks, and also in parts of armor-piercing bullets and missiles (ATSDR, 2013b). The main use of uranium is to fuel commercial nuclear power plants, where fuel is typically enriched in U-235 to 2-3%. Variable concentrations of uranium occur naturally in drinking water sources. The primary exposure sources for non-occupationally exposed persons are dietary and drinking water

Regulations in Canada

Uranium mining is heavily regulated in Canada. Most forms of mining in Canada are regulated provincially as well as federally under the Metal and Diamond Mining Effluent Regulations established under the Fisheries Act. Uranium mining is further regulated under an independent federal governmental organization known as the Canadian Nuclear Safety Commission (CNSC).

Under the *Nuclear Safety and Control Act* (NSCA), uranium mines and mills are highly regulated under an extensive array of safety control areas, with those most relevant to the public being, radiation protection and environmental protection. These facilities are required to have extensive environmental monitoring programs for nuclear and hazardous substances with results reported to the CNSC. The CNSC presents an annual Regulatory Oversight Report (ROR) on the performance of these facilities to the independent Commission at public meetings (web cast). These RORs as well as other relevant environmental and safety performance documents are publicly available on the CNSC web site. https://www.cnsc-ccsn.gc.ca/eng/

The current study utilizes serum concentrations of uranium and as such direct comparison of this study to other studies which utilize whole blood concentrations of uranium may not be accurate.

Possible Health Effects

Exposure occurs through inhalation of dust in air or ingestion of contaminated food or water. The average daily intake of uranium from food ranges from 0.07 to 1.1 µg per day (ATSDR. 2013b). Soluble forms of uranium salts are poorly absorbed in the gastrointestinal tract. About 99% of the ingested uranium will leave a person's body in the feces, with the remainder entering the blood, most of which will be removed by the kidneys and excreted in the urine within a few days. A small amount of the uranium in the bloodstream will deposit in a person's bones, where it will remain for years (ATSDR, 2013b). Intakes exceeding federal and provincial standards can lead to increased cancer risk, liver damage or both.

Human health effect from uranium at low environmental doses or background concentrations are unknown. Radiation risks from exposure to natural uranium are very low. Health effects from uranium exposure result from chemical toxicity to the kidney, which can occur in instances of high occupational exposure (Kurttio et al., 2006). The Health Canada maximum acceptable concentration in Canadian drinking water for total uranium is 0.02 mg/L (Health Canada, 2009).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and trends

All mean concentrations were below the limit of quantification (<0.05 µg/L) for the 6 pools of pregnant women sampled from northern Saskatchewan. Uranium was not reported in phase one of Alberta's biomonitoring program (AHW, 2008).

MINERAL MICRONUTRIENTS

Mineral micronutrients are naturally occurring substances that are needed in small quantities to sustain life. Humans ingest mineral micronutrients in their diet, from fruits and vegetables to water to animal products. As they are naturally occurring, the ultimate source is soil and water and human serum concentrations will reflect regional differences in local soil and drinking water chemistry.

All micronutrients examined in the current study were detected in blood serum above the LOQ except boron. As micronutrients, Health Canada sets allowable daily intakes for these substances; however the focus is to ensure that the population is receiving an adequate intake. Possible exposure sources, nutritional information and potential health effects (deficiency/excess) are also included in this report. The mean ranges of concentrations for the evaluated mineral micronutrients are outlined in the subsequent sections.

BORON (B)

GENERAL INFORMATION

Boron occurs naturally in soil, water and food. The average daily intake of boron from natural sources by Canadian adults is estimated to be approximately 0.86 mg from water and 2.5 mg from food for a total of 3.4 mg/day (Health Canada, 2007b). The Tolerable Upper Intake Levels for pregnant women aged 19-50 years is 20 mg/day (Health Canada, 2005b).

Boron is an essential nutrient for the normal growth and development in plants. The World Health Organization has concluded that boron is "probably essential" in humans, that it has not been proven conclusively since no specific biochemical function has been identified for boron in higher animals or humans. Studies in animals and humans have shown that boron interacts with magnesium, copper, vitamin D and estorgen to affect calcium metabolism, which suggests implications for reducing the risk of osteoporosis (Health Canada, 1991). However, beneficial effects ave only been seen in animals and humans deficient in these nutrients or any combination thereof. Artificial boron deficiency also adversely affects embryonic development, brain function and

cognitive performance, but natural boron deficiency is rare anywhere in the world and unknown in North America.

The effect of boron on major mineral metabolism and its potential role as in inhibitor of osteoporosis in humans have been investigated (Health Canada, 1991). Boron compounds are rapidly and completely absorbed from the gastrointestinal tract, through mucous membranes and through damaged or abraded skin. It is eliminated mainly from the kidney, with minor amounts being excreted in feces, sweat and saliva. Boron does not accumulate in normal tissues but may concentrate in malignant brain tumours (Health Canada, 1991).

Calculations for the reference dose or tolerable daily intake values are mostly calculated based on the No Observed Adverse Effect Level of 9.6 mg boron/kg/bw/day for fetal effects in a study using Sprague-Dawley rats, reduced by uncertainty factors (UF) varying from 22 to 1000. The Natural Health Products Directorate (NHPD) of Health Canada has set a chronic reference dose for boron as 0.7 mg/day based on a 70 kg adult and a UF of 1000. As a safe dosage maximum for therapeutic prducts, the NHPD has set a maximum Acceptable Daily Intake (ADI) value for all sources of boron exposure as 6.72 mg/day (Health Canada, 2010b). The maximum permissible dose is 3.36 mg/day for boron in oral therapeutic natural health products. Boron supplementations in people who are not deficient will not necessarily provide any benefits to the structures and functions affected by deprivation (Health Canada, 2007b).

The current study utilizes serum concentrations of boron and as such direct comparison of this study to other studies which utilize whole blood concentrations of boron may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Boron concentration in blood serum of pregnant northern Saskatchewan women ranged from 13 µg/L to 24 µg/L (weighted arithmetic mean ± 95% confidence interval: 17 ± 3.1 µg/L). The pool with the highest concentration was found in the northwest region; however, there were no differences in concentrations across the three regions. Blood serum concentrations of boron in pregnant women in Alberta had mean concentrations ranging from 13.2 µg/L to 34.4 µg/L, with no trends between regions or across age groups. The overall mean concentration of pregnant women from Saskatchewan is largely comparable to the mean concentrations stratified by age in region in Alberta. Mean concentrations measured in all age groups from northern Alberta (≤25 mean ± 95 % CI: 17.5 ± 2.52 µg/L; 26-30 mean ± 95 % CI: 19.6 ± 1.15 µg/L; 30+ mean ± 95 % CI: 19.7 ± 2.03 µg/L) and women 25 years and younger (mean ± 95 % CI: 18.6 ± 1.83 µg/L) fell within the confidence interal of the overall mean measured from the six Saskatchewan pools. Mean concentrations measured in women 26-30, and 30 years and older from Central Alberta (26-30 mean ± 95 % CI: 23.6 ± 2.76 µg/L; 30+ mean ± 95 % CI:21.6 ± 1.20 µg/L) and women of all age groups from southern

Alberta (≤25 mean ± 95 % CI: 20.4 ± 1.04 µg/L; 26-30 mean ± 95 % CI: 23.9 ± 1.39 µg/L; 30+ mean ± 95 % CI:25.3 ± 1.13 µg/L) exceeded the 95% confidence interval of the overall mean calculated from the six Saskatchewan pools.

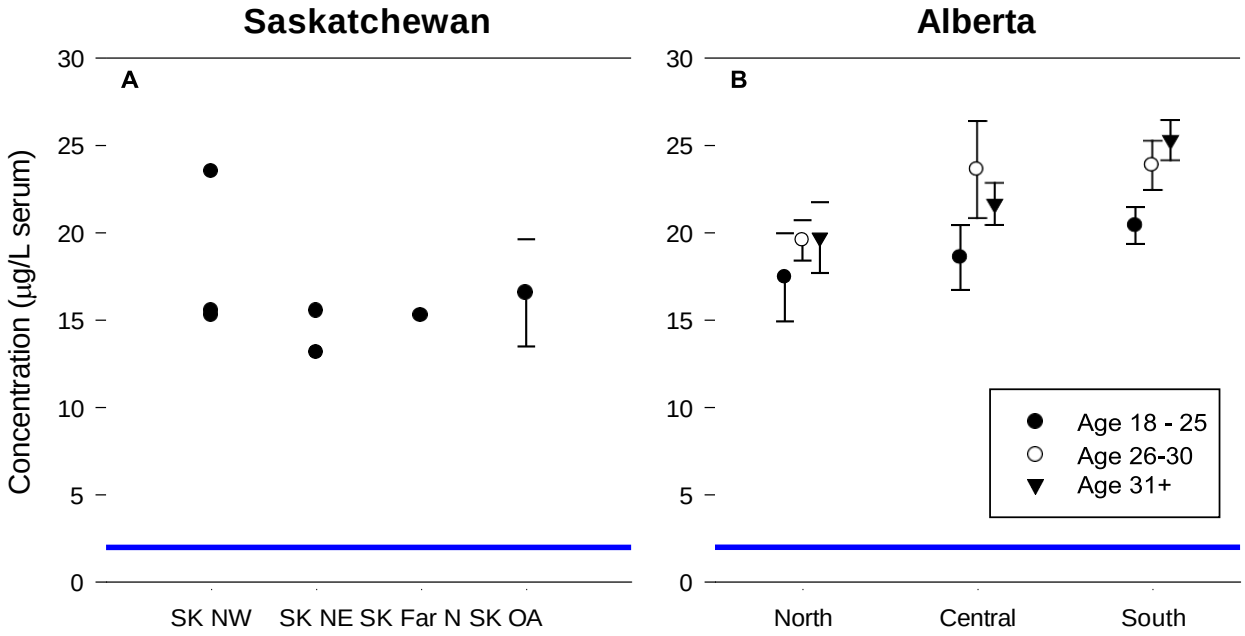

Figure 116: Concentrations of boron in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B) by geographic area. Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by both age and region are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

COBALT (Co)

GENERAL INFORMATION

A small amount of cobalt is naturally found in most rocks, water, soil, plants and animals, but is usually found in the environment combined with other elements such as oxygen, sulphur and arsenic. Cobalt is naturally released into the environment through leaching from soil, airborne dust, sea spray, volcanic eruptions and forest fires. Anthropogenic sources to the environment include burning of fossil fuels, sewage sludge, phosphate fertilizers, mining and smelting of cobalt-containing ores and industrial processes that use cobalt compounds. Two important radioactive isotopes include cobalt-60 and cobalt-57 (ATSDR, 2004e), but for this biomonitoring study, radioactive isotopes were not measured as only the non-radioactive cobalt-59 was analyzed in this study.

Cobalt is an essential trace element required for the maintenance of good health in humans (Health Canada 2013). It is a micronutrient required in the formation of vitamin B12 and for its function in enzymatic processes. For most people, the vast majority of cobalt intake is from food including coffee and through vitamin B12 found in meat and dairy products (ATSDR). Caribou meat in Norway was found to have five and twelve times the concentration of vitamin B12 compared to beef and chicken (Hassan, Sandanger, and Brustad, 2012). Vitamin B12 exposure can also be increased with the use of various supplements including common energy drinks (Higgins et al., 2010). The average person consumes about 11 µg/day of cobalt, including vitamin B12. The recommended daily intake of vitamin B12 is 6 µg/day. Cobalt has been used as a treatment for anemia, including in pregnant women, by increasing production of red blood cells. Cobalt may be transferred from the pregnant mother to the fetus or from the mother to the infant via breast milk (ATSDR, 2004e). Children living near waste sites containing cobalt are likely to be exposed to higer environmental levels of cobalt through breathing, touching soil, and eating contaminated soil (from hand-to-mouth activity). Cobalt levels can also be elevated in people who have had a hip prosthesis (metal-on-metal) with levels ranging from 0.3 to 7.5 µg/L in serum (Jantzen et al., 2013).

Low levels of vitamin B-12 can lead to anemia and neurological troubles. Exposure to cobalt levels normally found in the environment is not harmful to humans. High exposures can cause neurological, cardiovascular and endocrine deficits but health effects are unlikely to occur at blood cobalt concentrations under 300 ug/L (Leyssens et al.,2017)

The current study utilizes serum concentrations of cobalt and as such direct comparison of this study to other studies which utilize whole blood concentrations of cobalt may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of cobalt ranged from 0.40 µg/L to 0.48 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 0.45 µg/L ± 0.027 µg/L). There were no differences in concentrations across the three regions. The overall mean concentration of pregnant women from Saskatchewan is higher than the overall mean serum concentration measured in pregnant women from Alberta (mean ± 95% confidence interval: 0.329 ± 0.0458 µg/L) though the range of levels included higher concentrations in some areas in Alberta. Blood serum concentrations of cobalt in pregnant women in Alberta had mean concentrations ranging from 0.193 µg/L to 3.62 µg/L, with no trends between regions or across age groups.

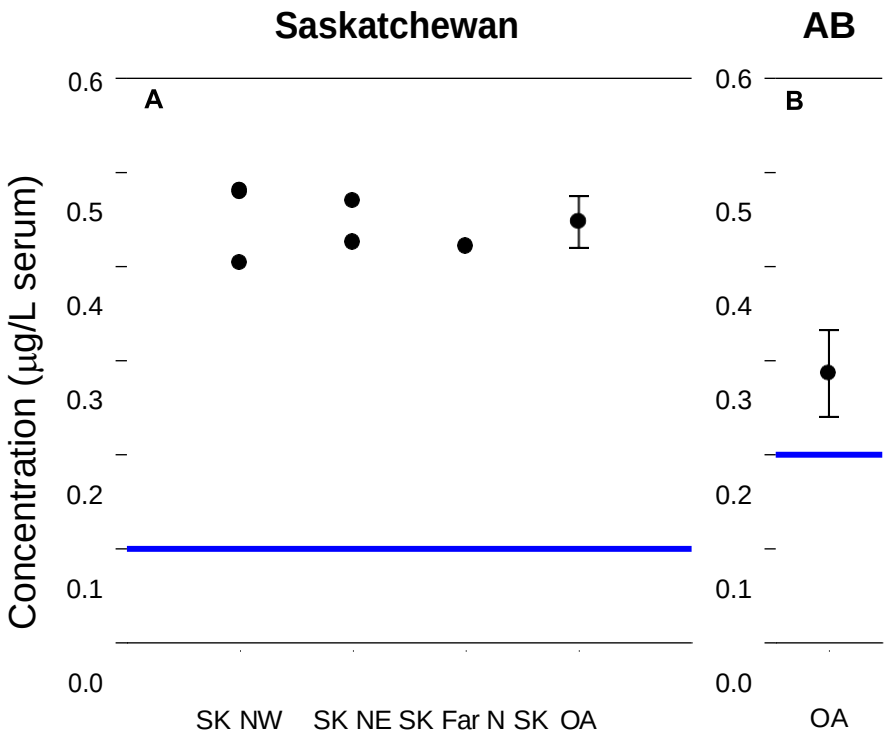

Figure 117: Concentrations of cobalt in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. An overall mean concentration (OA) is provided for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

COPPER (Cu)

GENERAL INFORMATION

Copper has been known, mined and used by humans for more than 5,000 years. It is second only to iron in its usefulness to humans. It is used in plumbing, electroplating, the production and manufacture of alloys, and as a fungicide and antimicrobial agent (Health Canada, 1992).

Copper is an essential element in mammalian nutrition and is required in many enzymatic reactions. It is essential for the normal utilization of iron particularly iron transport (ceruloplasmin). Copper deficiency (less than 2 mg/day) is accompanied by anemia. Other copper-containing enzyme systems include monoamine oxidase enzymes, required for pigmentation and control of neurotransmitters and neuropeptides; lysyl oxidase, essential for the maintenance of connective tissue in lungs, bones and elastin in the cardiovascular system; cytochrome c oxidase, involved in oxidative metabolism, brain functioning, heme synthesis and phospholipids synthesis; and superoxide dismutase, required for the destruction of superoxide radicals (Health Canada, 1992).

For the general population, most exposure to copper originates from ingestion of food.

Additional exposure may result from inhalation of dust particles and ingestion of drinking water (CCME, 1999h). Most copper is absorbed through the gastrointestinal tract. Maximum blood copper

levels were observed within 1 to 3 hours following oral administration, and about 50% of ingested copper was absorbed. Copper absorbed from the gastrointestinal tract is transported rapidly to blood serum and deposited in the liver bound to metallothionein, from which it is released and incorporated into ceruloplasmin, a specific copper-transporting protein. It travels to the liver followed by redistribution from the liver to other tissues (ATSDR, 2004f). Copper absorption is proposed to occur through two mechanisms, one energy-dependent and the other enzymatic.

The World Health Organization has recommended a daily intake of 30 µg/kg body weight per day (or 2.1 mg/day) for an adult male and 80 µg/kg body weight per day for infants. The Recommended Dietary Allowance (RDA) for pregnant women is 1,000 µg/day (Health Canada, 2005b).

Copper is generally considered to be non-toxic except in high doses, in excess of 15 mg/day (Health Canada, 1992). Bile is the major excretory route for copper; up to 70% of orally ingested copper may be excreted in the feces (ATSDR, 2004f). Normally, 0.5% to 3.0% of daily copper intake is excreted in the urine (ATSDR, 2004f). Concentrations in serum have been observed to decrease rapidly after exposure, indicating that they may only reflect recent exposures (ATSDR, 2004f). Overt copper deficiency is relatively rare. Health Canada has established an aesthetic objective for copper in drinking water which is deemed protective of adverse health effects but a health-based value has not been established in Canada.

The current study utilizes serum concentrations of copper and as such direct comparison of this study to other studies which utilize whole blood concentrations of copper may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of copper ranged from 1.81 x 10^3 µg/L to 2.13 x 10^3 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.96 x 10^3 ± 1.11 x 10^2 µg/L). In comparison to Alberta, pregnant women in northern Saskatchewan had similar blood serum concentrations with no apparent regional trend. The overall mean concentration in Saskatchewan was not significantly different than mean concentrations of pregnant women of all ages in both northern and central Alberta. However, the mean concentrations of copper in the serum of women in southern AB (18-25 years: 1.8 x 10^3 ± 31 µg/L; 26-30 years 1.7 x 10^3 ± 27 µg/L; 31+ years 1.8 x 10^3 ± 33 µg/L) were lower than the overall mean concentration found in Saskatchewan.

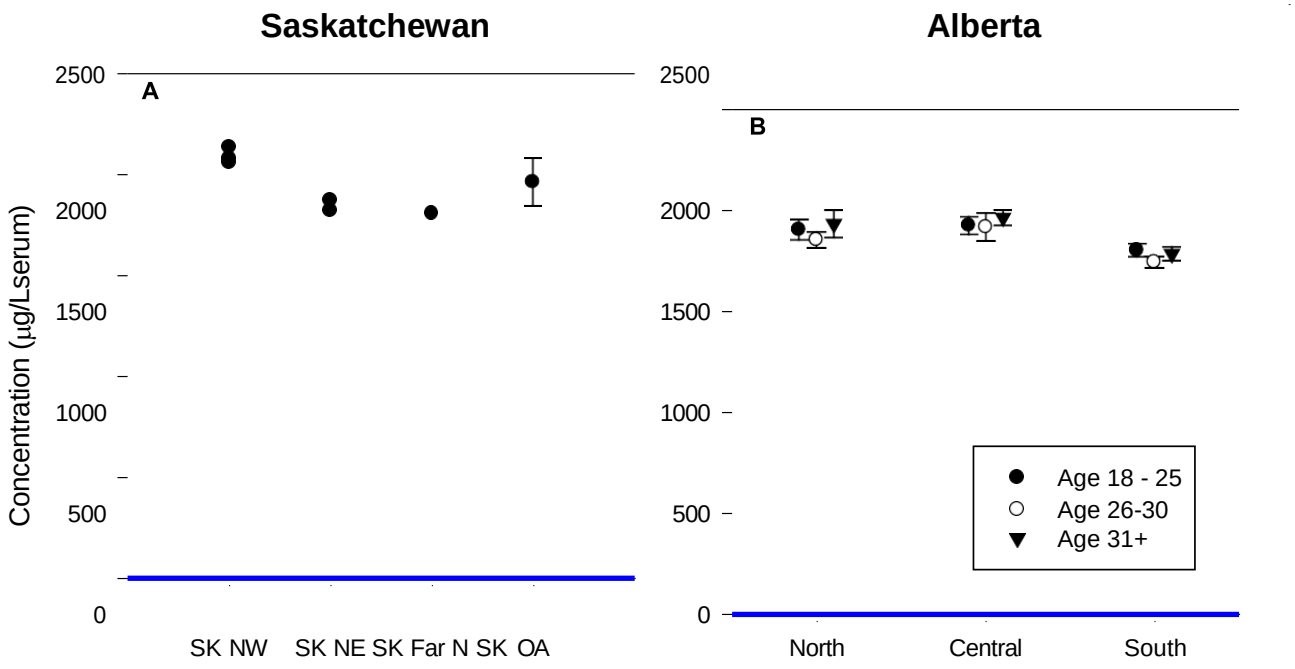

Figure 118: Concentrations of copper in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B) by geographic area. Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations stratified by both age and region are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

Iron (FE)

GENERAL INFORMATION

Iron is a critical component of proteins such as enzymes and hemoglobin. Almost two-thirds of the iron in the body is present in hemoglobin circulating in red blood cells. Hemoglobin moves oxygen to the tissues for metabolism. During pregnancy, women need more iron to support the increased maternal red blood cell mass. This supplies the growing fetus and placenta, and supports normal brain development in the fetus. In the third trimester of pregnancy, the fetus builds iron stores for the first six months of life (Health Canda, 2009b). As pregnancy progresses, more iron is needed.

The Recommended Dietary Allowance (RDA) for iron during pregnancy is 27 mg/day. The Tolerable Upper Intake Level during pregnancy is 45 mg/day (Health Canada, 2005b). Not enough iron during pregnancy can cause fatigue, reduced work capacity, cardiovascular stress, lower resistance to infection. Iron deficiency which can also lead to maternal anemia, premature delivery, low birth weight and an increased risk of perinatal infant mortality (Health Canada, 2009b). Iron

deficiency is a concern because it can delay normal infant motor function or mental function,

increase risk or preterm babies, and can cause fatigue that impairs the ability to do physical work in adults. The RDA for males aged 19 years or older is 8 mg/day. The RDA for females aged 19-50 years is 18 mg/day (Health Canada, 2005b).

In the general adult population, only 18% of the iron from food is absorbed by the body. For vegetarian diets, it is approximately 10%. Iron absorption is influenced by what is eaten at the same time. The three main inhibitors of non-heme iron absorption in the diet include polyphenols, phytate, and calcium levels greater than 300 mg. Vitamin C strongly enhances iron absorption as it releases non-heme iron bound to inhibitors (Health Canada, 2009b).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of iron ranged from 967 µg/L to 1.23 x 10^3 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.07 x 10^3 ± 82.4 µg/L). There were no apparent differences between regions. The mean concentration in pregnant women serum in Alberta (mean ± 95% confidence interval: 1.2 x 10^3 ± 25 µg/L) is higher than in Saskatchewan.

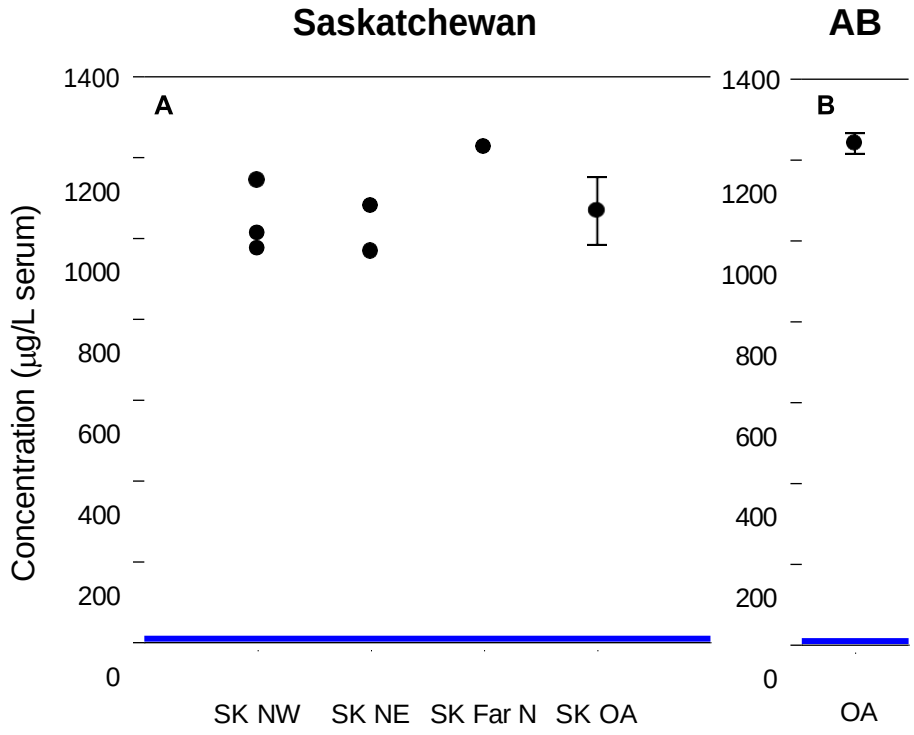

Figure 119: Concentrations of iron in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration of all of the pools included in analysis is presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

MANGANESE (Mn)

GENERAL INFORMATION

Manganese is naturally occurring and most often found in rocks and soils; however, it does not occur in the environment as a pure metal. It is usually combined with oxygen, sulphur or chlorine. It is principally used in steel production to improve hardness, stiffness, and strength. It can also be used in fireworks, dry cell batteries, paints, as a medical imaging agent, and in cosmetics (ATSDR, 2012c).

The general population is exposed through food, water, air and consumer products containing manganese. Manganese is an essential nutrient required as a cofactor for a variety of enzymes (ATSDR, 2012c). The highest concentrations are found in grains, nuts, legumes and fruit. The extent of absorption is a function of particle size, which determines where manganese will be deposited. The amount of manganese absorbed across the gastrointestinal tract is variable but typically averages 3-5%. Adults maintain stable tissue levels of manganese through the regulation of gastrointestinal absorption and hepatobiliary excretion. Absorbed manganese is widely distributed throughout the body, with higher levels found in the liver, pancreas, and kidney. The primary route of excretion is through the feces.

Health effects will depend on the dose, duration, and the route of exposure. As an essential nutrient, it is involved in the formation of bone, in cellular protection from free radical damage, and in amino acid, cholesterol, and carbohydrate metabolism (ATSDR, 2012c). Manganese deficiency is rare, but excessive exposure can cause neurological effects. Inhaled manganese can be transported directly to the brain and can result in a permanent neurological disorder known as manganism with symptoms that include tremors, difficulty walking, and facial muscle spasms. Exposure to high levels of manganese, such as those in accidental or occupational exposures, can result in lung inflammation and impaired lung function (ATSDR, 2012c). These effects occur at high or very high levels of manganese.

The Tolerable Upper Level Intake (UL) for pregnant women is 11 mg/day with an Adequate Intake (AI) of 2 mg/day. The AI for males aged 14 years and older is 2.3 mg/day with a UL of 11mg/day. The AI for females aged 19 years and older is 1.8 mg/day with a UL of 11 mg/day (Health Canada, 2005b). Note that the UL values provided only account for intake from pharmacological sources and does not include intake from dietary sources.

The current study utilizes serum concentrations of manganese and as such direct comparison of this study to other studies which utilize whole blood concentrations of manganese may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of manganese ranged from 2.6 µg/L to 4.2 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 3.5 µg/L ± 0.42 µg/L). There were no differences between geographical regions in Saskatchewan. When compared to the data obtained from the first phase of Alberta's biomonitoring program (mean ± 95% confidence interval: 2.87 ± 0.258 µg/L) (AHW, 2008), pregnant women in northern Saskatchewan have higher levels of blood serum manganese, but it is not significant. Saskatchewan has a 95% confidence interval of 3.0 to 3.9 µg/L; while AB has an overall 95% confidence interval of 2.62 to 3.13 µg/L. The serum levels for Alberta pools ranged from 1.90 ug/L to 21.1 ug/L.

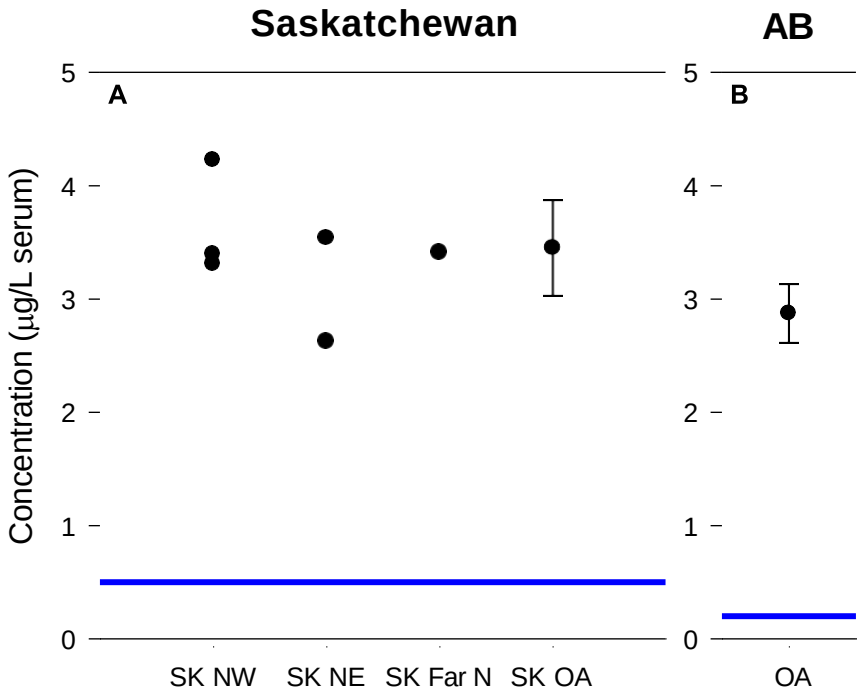

Figure 120: Concentrations of manganese in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration of all of the pools is provided for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

MAGNESIUM (Mg)

GENERAL INFORMATION

Magnesium is present in many foods, added to food products, and is present in some medicines. Green leafy vegetables, unpolished grains and nuts are rich in magnesium. Magnesium plays a major role in bone and mineral homeostasis and can directly affect bone cell function (IOM, 1997). It is a required cofactor in more than 300 enzyme systems that regulate diverse biochemical reactions in the body, including protein synthesis, muscle and nerve function, blood glucose control, and blood pressure regulation (IOM, 1997). Magnesium is required for energy production, oxidative phsophorylation, and glycoloysis. It plays an important role in the active transport of calcium and potassium ions across cell membranes, a process important in nerve impulse conduction, muscle contraction, and normal heart rhythm (IOM, 1997).

Less than 1% of total magnesium is in blood serum. Total body magnesium is about 25 g, of which 50-60% resides in the bone or cells (IOM, 1997). Normal serum magnesium concentration is 1.8 to 2.3 mg/dL (IOM, 1997). Magnesium homeostasis is largely controlled by the kidney, which typically excretes about 120 mg magnesium into the urine each day. It excretes less when Mg concentrations and intake are low. Magnesium deficiency in otherwise healthy people is rare because of kidney regulation. Early signs of deficiency include loss of appetite, nausea, vomiting, fatigue, and weakness. As deficiency worsens, numbnessm tingling, muscle contractions and cramps can occur.

The RDA for pregnant women under 18 years is 400 mg/day; 19-30 years is 350 mg/day; and 31-50 years is 360 mg/day. The RDA during lactation is slightly lower (Health Canada, 2005b). The RDA for females aged 19 and older ranges from 310 mg/day to 320 mg/day. In males aged 31 years and older, the RDA is 420 mg/day. In males aged 19-30 years, the RDA is 400 mg/day (Health Canada, 2005b).

The current study utilizes serum concentrations of magnesium and as such direct comparison of this study to other studies which utilize whole blood concentrations of magnesium may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of magnesium ranged from 1.70×10^4 to 1.97×10^4 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.86×10^4 µg/L ± 685 µg/L). Concentrations of magnesium are not reported in

other similar studies in North America. Although reference values may differ between laboratories, the magnesium reference value for Mayo Clinic Laboratories is between 1.7 and 2.3 X 10^4 ug/L.(Mayo Clinic Laboratories).

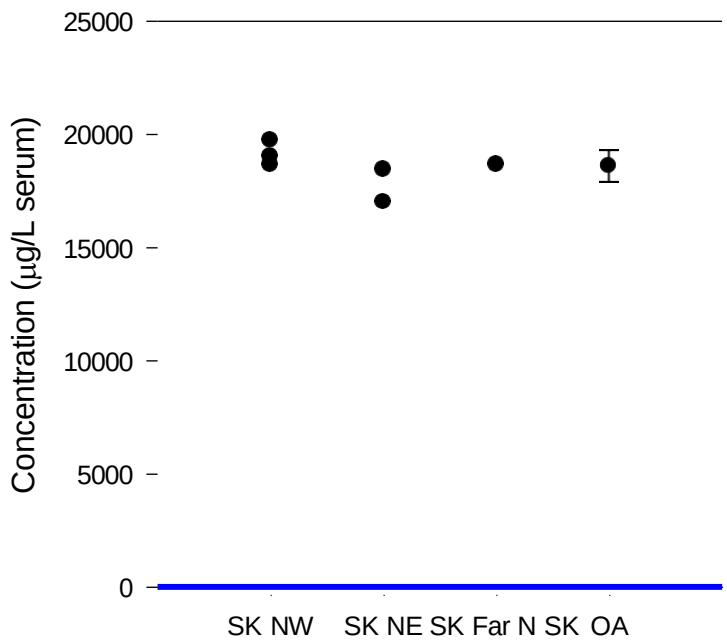

Figure 121: Concentrations of magnesium in the blood serum of pregnant women in Saskatchewan. The blue lines represent the limit of quantification used in laboratory analysis. Data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Estimates provided represent a 95% confidence interval around the mean.

MOLYBDENUM (Mo)

GENERAL INFORMATION

Molybdenum exists throughout the Earth's crust, usually in combination with other elements and does not occur as a free metal in nature. It is found naturally in rocks, soil, sediment, surface water, groundwater, plants, animals and humans. It may be released to the environment through natural weathering processes (CCME, 1999g) or through anthropogenic sources such as combustion of coal, municipal sewage sludge, and mining operations (CCME, 1999g). The use of fertilizers is an important source of molybdenum to aquatic systems. It is also used in the steel industry and as a pigment in inks and paints (CDC, 2009).

Molybdenum is a cofactor for three enzyme classes: sulphite oxidase, aldehyde dehydrogenase, and xanthine oxidase (CDC, 2013m). It also aids in protein metabolism. Absorption of dietary molybdenum from the gastrointestinal tract depends on the chemical form and ranges from 30-70% (WHO, 2011c). Following gastrointestinal absorption, molybdenum appears rapidly in

the blood and most organs. Highest concentrations are found in the liver, kidneys and bones (WHO, 2011c). Molybdenum is primarily excreted in the urine via the kidneys.

Human health effects from molybdenum at low environmental doses are unknown. Molybdenum is generally considered of low human toxicity (CDC, 2013m). However, chronic exposure to high levels of molybdenum (10-15 mg/day) has been associated with gout-like symptoms (CDC, 2013l). An RDA of 50 µg/day during pregnancy and 45 µg/day in males and females aged 19 years and older has been set by Health Canada (Health Canada, 2005b).

The current study utilizes serum concentrations of molybdenum and as such direct comparison of this study to other studies which utilize whole blood concentrations of molybdenum may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of molybdenum (Mo) ranged from 1.1 µg/L to 1.3 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.2 µg/L ± 0.060 µg/L). There were no differences between the regions in northern Saskatchewan. Compared to the range of blood serum concentrations in pregnant women in Alberta (1.06 µg/L to 4.29 µg/L), northern Saskatchewan presented with slightly lower overall concentrations. The overall mean serum concentration in Saskatchewan (1.2 ± 0.060 µg/L) is comparable to the mean concentration in women ≤25 years old (1.27 ± 0.0569 µg/L), and is lower than the mean concentrations found in women 26-30 years and ≥31 years of age (1.49 ± 0.161 µg/L; 1.40 ± 0.100 µg/L).

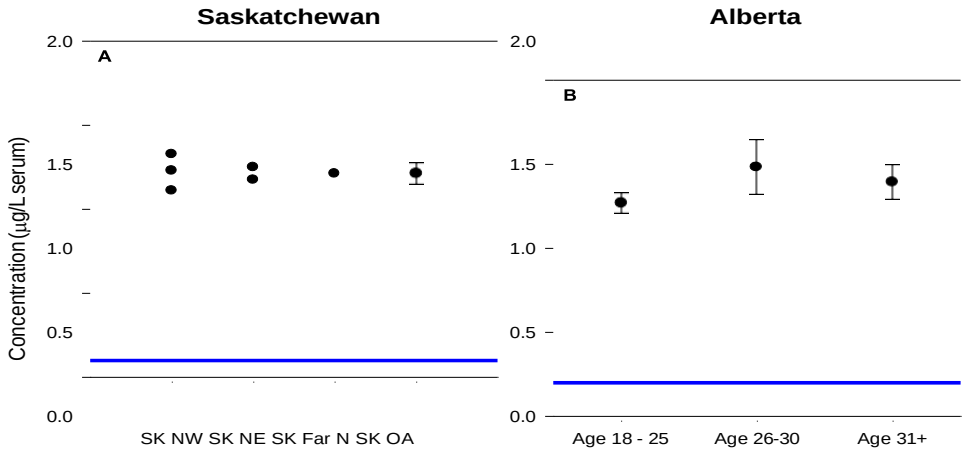

Figure 122: Concentrations of molybdenum in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. Mean concentrations organized by age group

are presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

NICKEL (Ni)

GENERAL INFORMATION

Nickel is a hard, silvery-white metal that is commonly combined with other metals to form alloys. It is commonly used to make stainless steel, in nickel plating, batteries, and substances known as catalysts. Nickel alloys are used in metal coins and jewelry. It occurs naturally in rocks and is released into the environment via weathering of geological deposits. Nickel is anthropogenically released into the environment during nickel mining, industries that use nickel and nickel compounds, by oil-burning power plants, coal-burning power plants, and trash incinerators (ATSDR, 2005b). Nickel tends to partition to soil or sediment in the environment, particularly to fractions containing iron or manganese.

Food contains nickel and is the major source of nickel exposure for the general population. Foods naturally high in nickel include chocolate, soybeans, nuts and oatmeal (ATSDR, 2005b). Approximately 1-10% of ingested nickel is absorbed compared to 20-35% of inhaled nickel is absorbed into the blood from the respiractory tract (ATSDR, 2005b). About 20-30% of the nickel contained in cigarettes is released in mainstream smoke. Wearing nickel plated jewelry or using consumer products containing nickel can also add to exposure. Nickel is distributed widely in the body, but most of it will go to the kidneys. Nickel that enters the bloodstream is excreted in the urine. Nickel that is eaten is excreted in the feces.

The most common health effect of nickel is an allergic reaction. Approximately 10-20% of the population (ATSDR, 2005b) is sensitive to nickel. The common type of reaction is a rash at the site of contact. In some sensitized people, dermatitis may develop in an area of the skin that is away from the site of contact. In occupational scenarios, such as nickel refineries or processing plants, inhalation of nickel containing dust may cause chronic bronchitis, reduced lung function, and cancer of the lung and nasal sinus (ATSDR, 2005b). Health Canada (2005b) has set a Tolerable Upper Intake Level (UL) for nickel of 1.0 mg/day for pregnant women, lactating women, and females and males aged 14 years and older.

The current study utilizes serum concentrations of nickel and as such direct comparison of this study to other studies which utilize whole blood concentrations of nickel may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Serum nickel concentrations ranged from 0.375 µg/L to 2.08 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval:

0.755 µg/L ± 0.571 µg/L). There were no apparent differences in concentration between regions in northern Saskatchewan. Concentrations of pregnant women from Alberta (AWH, 2008) ranged from 0.386 µg/L to 5.58 µg/L in blood serum and the overall mean of Saskatchewan 0.755 ± 0.571 µg/L overlaps with the overall mean found in Alberta of 0.881 ± 0.0836 µg/L.

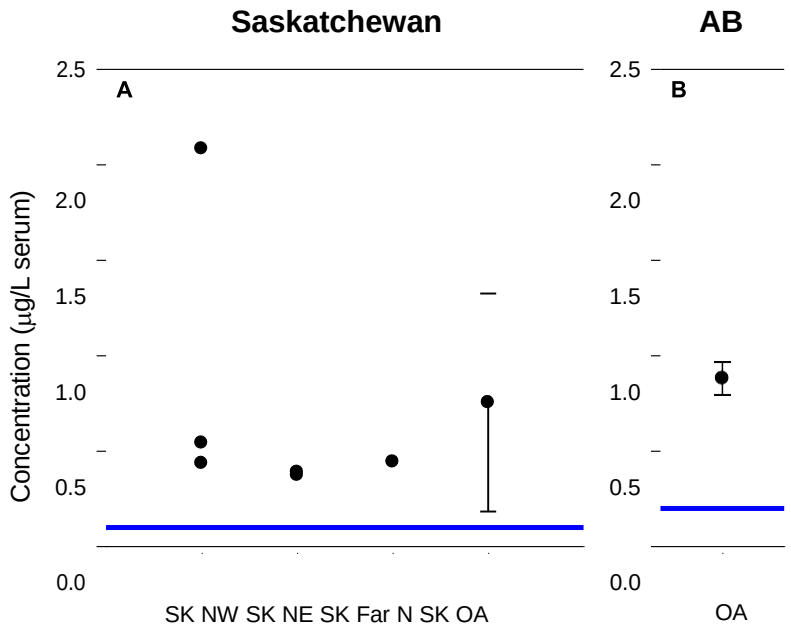

Figure 123: Concentrations of nickel in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration of all of the pools analyzed in Alberta is presented. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

SELENIUM (Se)

GENERAL INFORMATION

Selenium is a naturally occurring substance found widely but unevenly distributed in the Earth's crust (ATSDR, 2003). It can be elevated in areas with soil that originate from marine sedimentary deposits. Weathering of rocks and soils may result in low levels of selenium in water, which may be taken up by plants. It is present as the inorganic forms selenide, selenate, and selenite. The forms and fate of selenium will depend largely on the acidity of the surroundings and its interaction with oxygen. Selenium can be released during the burning of fossil fuels, combine with oxygen and react with water to produce soluble selenium compounds. It was traditionally used in the electronics industry in the form of arsenic triselenide, used as as photoreceptor for photociopiers (ATSDR, 2003). In its organic form, it is found in trace quanities in most plants and animal tissues.

People are exposed to low levels of selenium daily, the majority as a result of food and water intake. Selenium is also a constituent of multivitamin supplements including some prenatal vitamins. Higher-than-normal levels of selenium exposure can occur near hazardous waste sites. Organic selenium compounds are more easily absorbed by the human body (>90%) compared to its inorganic forms (>50%) (IOM, 2000). Inorganic forms in drinking water are also easily absorbed from the digestive tract. They are then converted into forms that the human body can use. The RDA for pregnant women 60 µg/day; 55 µg/day for females aged 14 years and older and males 14 years and older (Health Canada, 2005b). The major dietary source of selenium is plant foods (CDC, 2008). Selenium functions through selenoproteins, several of which are oxidant-defense enxymes. Other selenium associated proteins regulate the action of thyroid hormones and the oxidation-reduction status of vitamin C and other molecules (IOM, 2000).

Selenium is necessary to human functioning as antioxidant enzymes, enzymes that protect the body from tissue damage, require it. It is also required for normal growth and metabolism. Most of the selenium that enters the body leaves the body within 24 hours. Selenium leaves mainly in the urine, but also in feces and breath. Effects of exceeding the RDA will depend on how much is consumed and how often. If midly excessive amounts of selenium are eaten over long periods of time, brittle hair and deformed nails can develop. A deficiency of selenium can cause heart problems and muscle pain. Selenium is used by the body in antioxidant enzymes that protect against damage to tissues done by oxygen, and in an enzyme that affects growth and metabolism. Preterm babies may be more sensitive to a selenium deficiency, and this may contribute to lung effects (ATSDR, 2003). Insufficient selenium is not common in North America compared to China where soil levels of selenium are very low. Blood concentrations greater than 1,000 µmol/L can cause selenosis (CDC, 2008). Selenosis is characterized by symptoms such as hair loss, skin lesions, tooth decay, and abnormalities of the nervous system

Selenium deficiency does not cause illness on its own, but can make the body more susceptible to illnesses caused by other nutritional, biochemical or infectious stresses (CDC, 2008). Few studies have examined how selenium can affect the health of children. It is not known if selenium exposure could result in birth defects. Minimize hand-to-mouth contact in children to reduce the incidence of ingesting contaminated soil.

The current study utilizes serum concentrations of selenium and as such direct comparison of this study to other studies which utilize whole blood concentrations of selenium may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Serum concentrations of selenium ranged from 108 µg/L to 124 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 118 ± 4.77 µg/L). There are no apparent trends with geography in northern Saskatchewan. Blood serum concentrations of selenium in pregnant northern Saskatchewan women are slightly lower than those of Alberta where overall mean concentrations ranged from 130 µg/L to 180 µg/L. The Saskatchewan overall mean serum concentration is lower than the overall mean serum concentration in Alberta of 154 ± 2.84 µg/L.

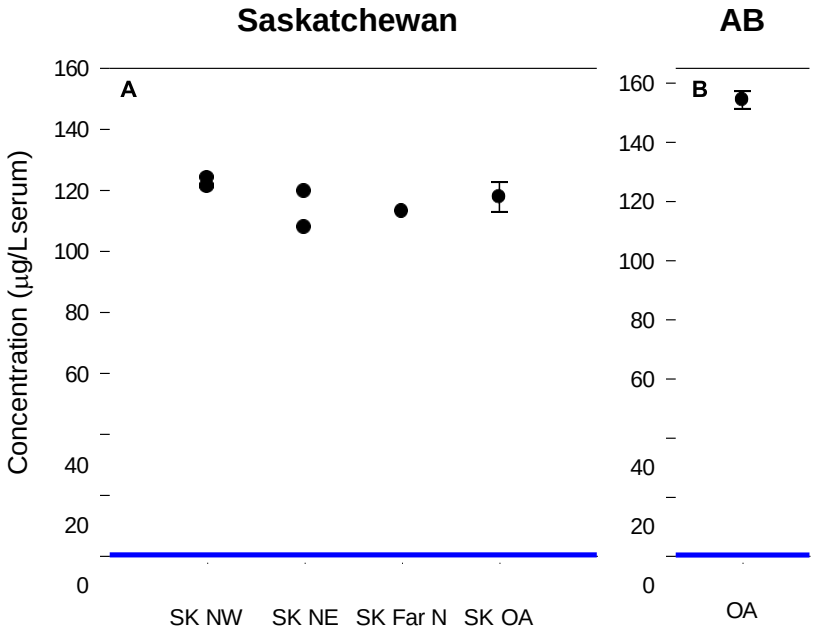

Figure 124: Concentrations of selenium in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B). Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration of all pools is presented for Alberta. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

SILVER (AG)

GENERAL INFORMATION

Silver is a naturally occurring element found within the earth's crust and is often found combined with other elements such as sulfide, nitrates and chloride (ATSDR, 1990; Health Canada, 1986b). Silver in river water can be dissolved by complexation with chloride and humic matter (WHO, 2003b). Silver has high electrical and thermal conductivity compared to other metals making

it useful in electrical equipment and batteries (WHO, 2003b). Other uses of silver include mirror, catalysts, table silver, jewellery, coins, an ingredient in lozenges and gum to help people stop smoking, as an external antiseptic agent, a disinfectant agent in drinking water and as a salt, oxide and halide in photographic materials (WHO, 2003b; ATSDR, 1990). Silver can be released into the air or water from the natural erosion of rocks and from human activities such as processing ores, burning fossil fuels and manufacturing cement.

Exposures to silver may come through inhalation of low levels of silver found in the air, ingestion of contaminated food and water, using anti-smoking lozenges or taking other medications containing silver, and by carrying out activities such as photography, soldering and jewelry making (ATSDR, 1990; WHO, 2003b; Health Canada, 1986b). Drinking water is the major contributor to daily oral exposure of silver in areas that use silver salts as bacteriostatic agents. For example, only 0.1% of Canadian tapwater surveyed contained 1-5 ng/L of silver whereas drinking water treated with silver tend to contain 50 µg/L or more (WHO, 2003b). Most food contains traces of silver ranging from 10 to 100 µg/kg and the median daily intake of silver from 84 self selected diets which factored in exposure from drinking water was 7.1 µg; however, older estimates range from 20 to 80 µg (WHO, 2003b).

The estimated acute lethal dose of silver nitrate is at least 10 g (WHO, 2003b). At high concentrations, silver exposure may lead to blue-gray discoloration of the skin called argyria (ATSDR, 1990). As well, lower levels of exposure can lead to deposition of silver in the skin and organs which isn't known to be harmful. High levels of exposure to silver in the air can lead to irritation of the airways, and skin contact with silver can lead to allergic reactions such as rashes; however, less silver is absorbed into the body through dermal contact as compared to absorption in the gastrointestinal tract and lungs (ATSDR, 1990). There have been a lack of studies investigating the developmental and reproductive effects of silver exposure, and the EPA has determined that the carcinogencity of silver cannot be classified. Silver can be measured in urine, feces, blood and in skin samples.

Due to the fact that food represents the main route of exposure to silver and levels of silver in drinking water in Canada and the United States is largely neglible and below what would cause adverse health effects in humans, a maximum acceptable concentration for silver in drinking water has not been set in Canada (Health Canada, 1986b).

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of silver ranged from 0.177 µg/L to 0.272 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 0.216 ± 0.0263 µg/L). There were no apparent differences between regions whereas mean concentrations in Alberta were dependent on both age and region. Results from northern

Saskatchewan are comparable to mean concentrations found in Alberta which ranged from 0.100 µg/L to 0.540 µg/L. The mean concentration in Saskatchewan (0.216 ± 0.0263 µg/L) overlaps with the overall mean concentrations and 95% confidence intervals of pregnant women aged 25 years or younger in northern (0.217 ± 0.328 µg/L), central (0.161 ± 0.273 µg/L) and southern (0.149 ± 0.218 µg/L) Alberta, pregnant women 26 to 30 years old in northern (0.189 ± 0.351µg/L), central (0.181 ± 0.306 µg/L) and southern (0.160 ± 0.240 µg/L) Alberta, and pregnant women 31 years and older in central (0.219 ± 0.357 µg/L) and southern (0.238 ± 0.293µg/L) Alberta. The mean concentration of pregnant women aged 31 years and older from northern Alberta (0.310 ± 0.444 µg/L) was higher than the mean concentrations found in Saskatchewan.

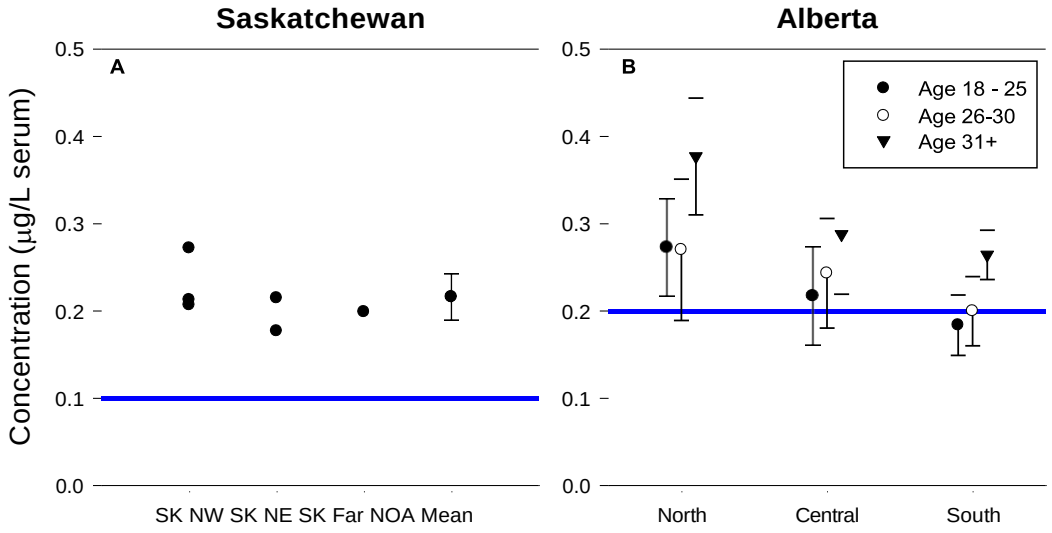

Figure 125: Concentrations of silver in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B) by geographic region. Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration for each region in Alberta is presented. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

ZINC (Zn)

GENERAL INFORMATION

Zinc is essential for microorganisms, plants, and animals. It functions as a component of numerous enzymes in the maintenance of the structural intergrity of proteins and in the regulation of gene expression (IOM, 2001). It is involved in many core areas of metabolism. The vast majority of zinc is absorbed by the small intestine (IOM, 2001). Transfer from the intestine is via the portal system with most newly absorbed zinc bound to albumin. Plasma zinc is only about 0.1% of the total body zinc; its concentration is tightly resulated at 10 to 15 µmol/L. Stress, acute trauma, and

infection cause changes in hormones and cytokines that lower plasma concentration.

In mild human zinc deficiency states, the detectable features and functional abnormalities of mild zinc deficiency are diverse. Impaired growth velocity is a primary clinincal feature of mild zinc deficiency and can be corrected with supplementation. Severe zinc deficiency in humans in rare. Pregnancy outcome and immune function is also affected by zinc supplementation. It has been suggested that maternal zinc deficiency may lead to poor birth outcomes and compromise infant development. Low plasma zinc concentrations reduce placental zinc transport and may affect the supply of zinc to the fetus. Zinc deficiency also alters circulating levels of a number of hormones associated with the onset of labour, in particular, pre-term birth (WHO, 2013).

RDA in pregnant women aged 19-50 years 11 mg/day, and 12 mg/day under 18 years. 1 mg/day higher when lactating. RDA of 8 mg/day for females aged 19 and older, 11 mg/day for males aged 14 and older (Health Canada, 2005b).

The current study utilizes serum concentrations of zinc and as such direct comparison of this study to other studies which utilize whole blood concentrations of zinc may not be accurate.

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN NORTHERN SASKATCHEWAN

Concentrations and Trends

Concentrations of zinc ranged from 1.30 x 10^3 µg/L to 1.53 x 10^3 µg/L in the six pools of pregnant women sampled from northern Saskatchewan (weighted arithmetic mean ± 95% confidence interval: 1.41 x 10^3 ± 66.4 µg/L). There were no apparent differences between regions whereas mean concentrations in Alberta were dependent on region, but not age or season. Results from northern Saskatchewan are comparable to mean concentrations found in Alberta which ranged from 1200 µg/L to 1560 µg/L. The mean concentration in Saskatchewan (1.41 x 10^3 ± 66.4 µg/L) overlaps with the overall mean concentrations in women in Northern Alberta (1.45 x 10^3 ± 21.3 µg/L), central Alberta (1.32 x 10^3 ± 23.0 µg/L) and Southern Alberta (1.41 x 10^3 ± 18.5 µg/L).

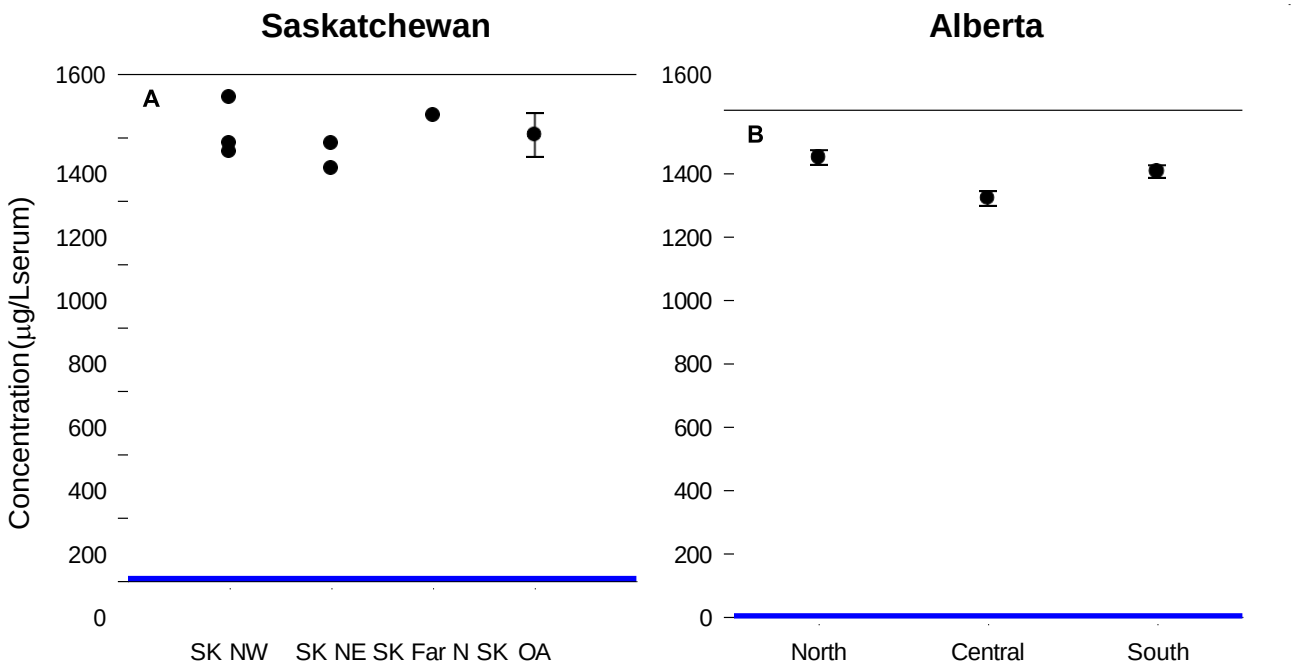

Figure 126: Concentrations of zinc in the blood serum of pregnant women in Saskatchewan (A) and Alberta (B) by geographic region. Saskatchewan data is presented for each of the six pooled samples, and for an overall (OA) weighted arithmetic mean of the six pools. A mean concentration for each region in Alberta is presented. The blue lines represent the limit of quantification used in laboratory analysis. Estimates provided represent a 95% confidence interval around the mean.

GLOSSARY

Bioaccumulation	Accumulation of substances in an organism (plant, animal or human) above what is in the environment (e.g. water, air, food).
Man-made chemicals	Chemicals that are produced by human activities, either intentionally or non-intentionally, and are not normally found in the environment. Also referred to as synthetic chemicals or anthropomorphic (human made) chemicals.
Naturally occurring chemicals	Chemicals that are present or produced naturally in the environment. Some manmade chemicals are also naturally occurring.
Serum samples	The clear yellowish liquid part of whole blood. It is obtained by clotting the whole blood, and then by separating the liquid from the solids.
Metabolite	A substance produced from another precursor substance, through metabolic transformation (by enzymes or microorganisms in our bodies).
Aliquot	A small portion of the total sample
Persistent	Resistant to degradation processes in our bodies or in the environment
Lipid	Synonym of fat or oils
Lipophilic	"Fat loving" – describes compounds that can be easily dissolved or stored in lipids
Background concentration of chemicals	A subjective term normally used to describe the baseline concentration of a chemical in humans or the environment where there has been no occupational or accidental exposure to high concentrations.
LOD	The limit of detection (LOD) is the lowest concentration at which an analyte can be distinguished from the background.

	Limit of detection of an individual congener is defined by meeting pre-determined acceptance criteria (e.g., ion ratios within 20%, precision less than 20%, etc.) specific to a certain analytical method.
LOQ	The limit of quantitation (LOQ) is set at a higher value and is the concentration at which concentrations of the analyte can be reported with confidence. The LOQ can also be determined by meeting pre-determined acceptance criteria related to LOD determination

ABBREVIATIONS AND ACRONYMS

AMAP	Arctic Monitoring and Assessment Programme
ATSDR	(U.S.) Agency for Toxic Substances and Diseases Registry
CDC	(U.S.) Centers for Disease Control and Prevention
CHMS	Canadian Health Measures Survey
FNBI	First Nations Biomonitoring Initiative
NCP	Northern Contaminants Program
NHANES	National Health and Nutrition Examination Survey

APPENDIX A - FORMS

MEDICAL CLINICS: PLEASE RETURN COMPLETED CONSENT FORM BY FAX TO 306-787-3237

Saskatchewan Ministry of Health

NORTHERN INTER-TRIBAL HEALTH AUTHORITY

POPULATION HEALTH UNIT
Northern Saskatchewan

CONSENT TO PARTICIPATE

Biomonitoring Maternal Blood in Saskatchewan

To: [put merged name here]
Patient City: [merged]
Postal Code: [merged]

Health number: [merged]
Age: [merged]

Physician/Nurse Practitioner: [merged]

√ one of the following:

☐ I agree to allow my "leftover" prenatal blood sample to be tested as described.

OR

☐ I do not agree to allow my "leftover" prenatal blood sample to be tested as described. I understand the "leftover" portion of my sample will be destroyed and not used in this project. I also understand that my not participating in this project will have no negative effect on the health care that I receive, including routine prenatal care.

Signature:_____ **Date**: _____

Medical clinics: Please fax completed forms to (306) 787-3237 Attn: Maureen Anderson

Ministry of Health

POPULATION HEALTH UNIT
Northern Saskatchewan

NORTHERN INTER-TRIBAL HEALTH AUTHORITY

DECLINE TO PARTICIPATE (OPT-OUT)

STUDY TITLE: Biomonitoring Maternal Blood for Environmental Chemicals in Northern Saskatchewan

PRINCIPAL INVESTIGATORS: Saskatchewan Ministry of Health (Dr. Moira McKinnon) and Athabasca, Keewatin Yatthé and Mamawetan Churchill River Health Authorities (Dr. James Irvine)

Important → Women who consent to participate do not require a form.

Please √ the box and complete sections below:

☐ **I do not agree to participate in the biomonitoring project.** I understand the "leftover" portion of my prenatal blood sample will be discarded and **not** used in this project. I also understand that only regular prenatal tests will be completed on my sample and that my health care services will not be impacted in any way by choosing to opt-out.

Printed Name of Patient: _____

Personal Health #: _____

Date: _____

Date of Birth: _____
Day /Month / Year

Postal Code of Patient: _____

(Please provide your Personal Health Number, postal code and date of birth so that your blood sample can be separated from those who choose to participate. This information will be destroyed, along with your blood sample, after routine testing is complete).

Medical Clinics:

Please FAX a copy of the completed opt-out form to (306)787-3237

Please maintain a copy of this opt-out form in patient chart.

APPENDIX B – LOD/LOQ TABLE

Table 1: The limits of detection/quanitification for every chemical included for analysis in this study, along with the number of pools above the limit of detection (or quantification) and whether or not individual results for each compound are included in this report.

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
Chlorophenols					
Pentachlorophenol (PCP)		ng/g	0.500	2	N
Trichlorophenols		ng/g	0.500	0	N
Dioxins and Furans					
2378 TeCDD	serum adjusted	pg/g serum	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
12378 PeCDD	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
123478 HxCDD	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
123678 HxCDD	serum adjusted	pg/g	0.010	3	N
	lipid adjusted	pg/g lipid	1.9		
123789 HxCDD	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
1234678 HpCDD	serum adjusted	pg/g	0.010	6	Y
	lipid adjusted	pg/g lipid	1.9		
OCDD	serum adjusted	pg/g	0.010	6	Y
	lipid adjusted	pg/g lipid	1.9		
2378 TeCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
12378 PeCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
23478 PeCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
123478 HxCDF	serum adjusted	pg/g	0.010	1	N
	lipid adjusted	pg/g lipid	1.9		
123678 HxCDF	serum adjusted	pg/g	0.010	1	N
	lipid adjusted	pg/g lipid	1.9		
123789 HxCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
234678 HxCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
1234678 HpCDF	serum adjusted	pg/g	0.010	4	N
	lipid adjusted	pg/g lipid	1.9		
1234789 HpCDF	serum adjusted	pg/g	0.010	0	N
	lipid adjusted	pg/g lipid	1.9		
OCDF	serum adjusted	pg/g	0.010	0	N

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	pg/g lipid	1.9		
Parabens					
Methyl Paraben		ng/mL	1.0	6	Y
Ethyl Paraben		ng/mL	1.0	3	N
Propyl Paraben		ng/mL	0.50	6	Y
Butyl Paraben		ng/mL	0.50	0	N
Benzyl Paraben		ng/mL	0.50	0	N
Polychlorinated biphenyls (PCBs)					
PCB 2	serum adjusted	pg/g	0.30	6	Y
	lipid adjusted	ng/g of lipid	0.058		
PCB 1	serum adjusted	pg/g	0.30	6	Y
	lipid adjusted	ng/g of lipid	0.058		
PCB 3	serum adjusted	pg/g	0.3	6	Y
	lipid adjusted	ng/g of lipid	0.058		
PCB 4/10	serum adjusted	pg/g	3.0	6	Y
	lipid adjusted	ng/g of lipid	0.58		
PCB 15	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 6	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 8	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 9	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 11	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 14	serum adjusted	pg/g	2.0	0	N
	lipid adjusted	ng/g of lipid	0.39		
PCB 7	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 5	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 12	serum adjusted	pg/g	2.0	0	N
	lipid adjusted	ng/g of lipid	0.39		
PCB 13	serum adjusted	pg/g	2.0	5	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 16	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 19	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 37	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 26	serum adjusted	pg/g	0.60	6	Y

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	ng/g of lipid	0.12		
PCB 27	serum adjusted	pg/g	1.0	6	Y
	lipid adjusted	ng/g of lipid	0.19		
PCB 30	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 31	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 32	serum adjusted	pg/g	1.0	6	Y
	lipid adjusted	ng/g of lipid	0.19		
PCB 34	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 35	serum adjusted	pg/g	0.60	1	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 36	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 38	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 22	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 23	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 24	serum adjusted	pg/g	1.0	6	Y
	lipid adjusted	ng/g of lipid	0.19		
PCB 28	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 39	serum adjusted	pg/g	0.60	1	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 17	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 29	serum adjusted	pg/g	0.60	5	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 18	serum adjusted	pg/g	2.0	6	Y
	lipid adjusted	ng/g of lipid	0.39		
PCB 21/20/33	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 25	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 48/49	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 55	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 60	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
PCB 61	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 73	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 58/67	serum adjusted	pg/g	0.50	2	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 78	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 81	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 41	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 45	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 50	serum adjusted	pg/g	0.80	0	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 57	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 63/76	serum adjusted	pg/g	0.50	5	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 66	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 72	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 79	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 46	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 59/42	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 80	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 64	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 69	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 43/52	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 44	serum adjusted	pg/g	1.0	6	Y
	lipid adjusted	ng/g of lipid	0.19		
PCB 54	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 56	serum adjusted	pg/g	0.50	6	Y

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	ng/g of lipid	0.096		
PCB 77	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 70	serum adjusted	pg/g	0.40	6	Y
	lipid adjusted	ng/g of lipid	0.077		
PCB 51	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 53	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 71	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 74	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 75/65/62	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 47	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 40/68	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 82	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 83/119	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 85	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 88/121	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 92	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 95	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 96	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 103	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 105	serum adjusted	pg/g	0.40	6	Y
	lipid adjusted	ng/g of lipid	0.077		
PCB 106	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 113	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 120	serum adjusted	pg/g	1.0	1	N
	lipid adjusted	ng/g of lipid	0.19		

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
PCB 122	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 127	serum adjusted	pg/g	0.30	0	N
	lipid adjusted	ng/g of lipid	0.058		
PCB 94	serum adjusted	pg/g	0.90	0	N
	lipid adjusted	ng/g of lipid	0.17		
PCB 99	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 100	serum adjusted	pg/g	0.80	0	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 108/86/125	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 111/117	serum adjusted	pg/g	0.60	2	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 114	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 118	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 84/89	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 93	serum adjusted	pg/g	0.90	0	N
	lipid adjusted	ng/g of lipid	0.17		
PCB 112	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 116	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 102	serum adjusted	pg/g	0.80	2	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 97	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 124	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 87	serum adjusted	pg/g	0.90	6	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 98	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 104	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 110	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 123/107/109	serum adjusted	pg/g	0.70	3	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 90/101	serum adjusted	pg/g	0.70	6	Y

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	ng/g of lipid	0.14		
PCB 91	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 115	serum adjusted	pg/g	0.60	1	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 126	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 130	serum adjusted	pg/g	0.40	1	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 136	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 144	serum adjusted	pg/g	0.70	2	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 148	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 151	serum adjusted	pg/g	0.70	5	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 152	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 153/168	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 159	serum adjusted	pg/g	0.30	0	N
	lipid adjusted	ng/g of lipid	0.058		
PCB 161	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 167	serum adjusted	pg/g	0.40	3	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 128/162	serum adjusted	pg/g	0.40	1	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 132	serum adjusted	pg/g	0.90	5	Y
	lipid adjusted	ng/g of lipid	0.17		
PCB 137	serum adjusted	pg/g	0.40	3	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 139/143	serum adjusted	pg/g	0.90	0	N
	lipid adjusted	ng/g of lipid	0.17		
PCB 145	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 150	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 156	serum adjusted	pg/g	0.30	6	Y
	lipid adjusted	ng/g of lipid	0.058		
PCB 158/129	serum adjusted	pg/g	0.30	5	Y
	lipid adjusted	ng/g of lipid	0.058		

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
PCB 160/163	serum adjusted	pg/g	0.40	6	Y
	lipid adjusted	ng/g of lipid	0.077		
PCB 165	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 141	serum adjusted	pg/g	0.50	5	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 146	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 147/149	serum adjusted	pg/g	0.80	6	Y
	lipid adjusted	ng/g of lipid	0.15		
PCB 154	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 138	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 155	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 169	serum adjusted	pg/g	0.30	0	N
	lipid adjusted	ng/g of lipid	0.058		
PCB 131/142/133	serum adjusted	pg/g	0.90	0	N
	lipid adjusted	ng/g of lipid	0.17		
PCB 134	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 157	serum adjusted	pg/g	0.30	4	N
	lipid adjusted	ng/g of lipid	0.058		
PCB 140	serum adjusted	pg/g	0.80	0	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 164	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 166	serum adjusted	pg/g	0.30	0	N
	lipid adjusted	ng/g of lipid	0.058		
PCB 135	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 170	serum adjusted	pg/g	0.40	6	Y
	lipid adjusted	ng/g of lipid	0.077		
PCB 171	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 172	serum adjusted	pg/g	0.80	1	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 175/182	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 176	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 177	serum adjusted	pg/g	0.60	2	N

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	ng/g of lipid	0.12		
PCB 178	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 179	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 183	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 190	serum adjusted	pg/g	0.40	1	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 191	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 181	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 184	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 186	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 187	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 192	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 174	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 193	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 180	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 188	serum adjusted	pg/g	1.0	0	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 189	serum adjusted	pg/g	0.50	0	N
	lipid adjusted	ng/g of lipid	0.096		
PCB 173	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 185	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 194	serum adjusted	pg/g	0.90	2	N
	lipid adjusted	ng/g of lipid	0.19		
PCB 195	serum adjusted	pg/g	0.70	0	N
	lipid adjusted	ng/g of lipid	0.14		
PCB 200	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 201/204	serum adjusted	pg/g	0.80	0	N
	lipid adjusted	ng/g of lipid	0.15		

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
PCB 197	serum adjusted	pg/g	0.40	0	N
	lipid adjusted	ng/g of lipid	0.077		
PCB 199	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
PCB 203/196	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 202	serum adjusted	pg/g	0.60	6	Y
	lipid adjusted	ng/g of lipid	0.12		
PCB 205	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 198	serum adjusted	pg/g	0.60	0	N
	lipid adjusted	ng/g of lipid	0.12		
PCB 206	serum adjusted	pg/g	0.50	6	Y
	lipid adjusted	ng/g of lipid	0.096		
PCB 207	serum adjusted	pg/g	0.80	1	N
	lipid adjusted	ng/g of lipid	0.15		
PCB 208	serum adjusted	pg/g	1.0	6	Y
	lipid adjusted	ng/g of lipid	0.19		
PCB 209	serum adjusted	pg/g	0.70	6	Y
	lipid adjusted	ng/g of lipid	0.14		
Perfluorochemicals (PFCs)					
PFHxS		ng/mL	0.500	0	N
PFOS		ng/mL	0.500	6	Y
PFDS		ng/mL	0.500	0	N
PFOA		ng/mL	0.500	6	Y
PFNA		ng/mL	0.500	3	N
PFDA		ng/mL	0.500	1	N
PFDoA		ng/mL	0.500	0	N
PFUA		ng/mL	0.500	1	N
Phthalates					
Monomethyl phthalate		ng/mL	0.50	0	N
Monoethyl phthalate		ng/mL	0.25	6	Y
Monoisobutyl phthalate		ng/mL	0.250	6	Y
Monocyclohexyl phthalate		ng/mL	0.25	0	N
Monobenzyl phthalate		ng/mL	0.250	6	Y
Mono-(2-ethylhexyl) phthalate		ng/mL	0.250	6	Y
Mono-n-octyl phthalate		ng/mL	0.25	0	N
Mono-(2-ethyl-5-oxohexyl) phthalate		ng/mL	0.25	0	N
Monoisononyl phthalate		ng/mL	0.25	0	N
Mono-(2-ethyl-5-hydroxyhexyl) phthalate		ng/mL	0.25	0	N
Organochlorine Pesticides*					
alpha-BHC	serum adjusted	ng/g	0.038 – 0.079*	0	N

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
	lipid adjusted	ng/g lipid	7.4 – 15*		
beta-BHC	serum adjusted	ng/g	0.038 – 0.079*	1	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
delta-BHC	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
gamma-BHC (Lindane)	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Octachlorostyrene	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Heptachlor	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Oxychlordane	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Aldrin	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Heptachlor Epoxide	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Dieldrin	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
4,4’-DDE	serum adjusted	ng/g	0.038 – 0.079*	6	Y
	lipid adjusted	ng/g lipid	7.4 – 15*		
Endrin	serum adjusted	ng/g	0.038 – 0.079*	3	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Endosulfan II	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
4,4’-DDD	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
4,4’-DDT	serum adjusted	ng/g	0.038 – 0.079*	1	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Methoxychlor	serum adjusted	ng/g	0.77 – 1.6*	0	N
	lipid adjusted	ng/g lipid	$1.5 \times 10^2 – 3.0 \times 10^2$*		
alpha-Chlordane	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
gamma-Chlordane	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Hexachloro-	serum adjusted	ng/g	0.038 – 0.079*	5	Y
benzene	lipid adjusted	ng/g lipid	7.4 – 15*		
Trans-nonachlor	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
Mirex	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		
2,4’-DDT	serum adjusted	ng/g	0.038 – 0.079*	0	N
	lipid adjusted	ng/g lipid	7.4 – 15*		

Chemical Name		Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
*LODs for individual pools given in Table 17					
Alkylphenols					
Octylphenol		ng/mL	0.200	6	Y
Nonylphenol		ng/mL	0.200	0	N
Bisphenol-A					
Bisphenol A		ng/mL	0.20	0	Y
Polybrominated diethylethers (PBDEs)					
BDE 28	LOD	ng/g lipid	0.28 – 0.64*	0 > LOD & LOQ	N
	LOQ	ng/g lipid	0.55 – 1.3*		
BDE 47	LOD	ng/g lipid	0.027 – 0.2*	6 > LOD & LOQ	Y
	LOQ	ng/g lipid	1.4 – 2.3*		
BDE 99	LOD	ng/g lipid	0.097 – 0.18*	6 > LOD & LOQ	Y
	LOQ	ng/g lipid	1.3 – 2.1*		
BDE 100	LOD	ng/g lipid	0.11 – 0.19*	6 > LOD & LOQ	Y
	LOQ	ng/g lipid	0.37 – 0.62*		
BDE 153	LOD	ng/g lipid	0.012 – 0.080*	6 > LOD & LOQ	Y
	LOQ	ng/g lipid	0.71 – 1.2*		
BDE 154	LOD	ng/g lipid	0.012 – 0.76*	3 >LOD & LOQ	N
	LOQ	ng/g lipid	0.13 – 0.79*		
BDE 183	LOD	ng/g lipid	0.18 – 1.3*	0 > LOD & LOQ	N
	LOQ	ng/g lipid	0.36 – 2.6*		
BDE 209	LOD	ng/g lipid	2.5 – 13*	0 > LOD & LOQ	N
	LOQ	ng/g lipid	6.1 – 25*		
BDE 66	LOD	ng/g lipid	0.053 – 0.38*	0 > LOD & LOQ	N
	LOQ	ng/g lipid	0.11 – 0.77*		
BDE 77	LOD	ng/g lipid	0.037 – 0.27*	0 > LOD & LOQ	N
	LOQ	ng/g lipid	0.073 – 0.53*		
BDE 85	LOD	ng/g lipid	0.11 – 0.20*	5 > LOD & LOQ	Y
	LOQ	ng/g lipid	0.21 – 0.39*		
BDE 138	LOD	ng/g lipid	0.016 – 0.11*	2 > LOD & LOQ	N
	LOQ	ng/g lipid	0.032 – 0.21*		
*LODs and LOQs by pool for PBDEs are provided in Table 18					
Phytoestrogens					
Daidzein		ng/mL	0.200	6	Y
Genistein		ng/mL	0.200	6	Y
Metals and Minerals					
Beryllium - Be		µg/L	0.10	0	N
Boron - B		µg/L	2.0	6	Y
Magnesium - Mg		µg/L	25.0	6	Y
Aluminmum - Al		µg/L	2.00	6	Y
Titanium - Ti		µg/L	5.0	0	N
Vanadium - V		µg/L	0.50	0	N

Chemical Name	Units	LOD/LOQ	No. of Pools Above LOD/LOQ	Report Status
Chromium - Cr	µg/L	0.50	0	Y
Manganese - Mn	µg/L	0.50	6	Y
Iron - Fe	µg/L	10.0	6	Y
Cobalt - Co	µg/L	0.10	6	Y
Nickel - Ni	µg/L	0.100	6	Y
Copper - Cu	µg/L	1.00	6	Y
Zinc - Zn	µg/L	10.0	6	Y
Silver – Ag	µg/L	0.100	6	Y
Arsenic - As	µg/L	0.100	3	Y
Selenium - Se	µg/L	0.500	6	Y
Stronium - Sr	µg/L	0.200	6	Y
Cadmium - Cd	µg/L	0.050	0	Y
Antimony - Sb	µg/L	0.25	6	Y
Molybdenum - Mo	µg/L	0.10	6	Y
Cesium - Cs	µg/L	0.050	6	Y
Barium - Ba	µg/L	0.500	6	Y
Tungsten - W	µg/L	0.10	0	N
Platinum - Pt	µg/L	0.050	0	N
Mercury - Hg	µg/L	0.100	6	Y
Thallium - Tl	µg/L	0.050	0	N
Lead - Pb	µg/L	0.10	6	Y
Uranium - U	µg/L	0.050	0	Y
Methylmercury				
MeHg	ng/g	0.05	4	Y
Tobacco smoke				
Cotinine	ng/mL	0.0500	6	Y

APPENDIX C – UNIT CONVERSIONS

Table 1: Unit conversions relevant to interpreting the results of the current study.

Units	Abbreviations	Values/Conversions
Litre	L	
Deciliter	dL	10^{-1} L
Milliliter	mL	10^{-3} L
Microlitre	µL	10^{-6} L
Gram	g	
Microgram	µg	10^{-6} g
Nanogram	ng	10^{-9} g
Pictogram	pg	10^{-12} g
1 µg/g	Equivalent to approximately 1 µg/mL or 1 mg/L	
1 ng/g	Equivalent to approximately 1 ng/g or 1 µg/L	
1 pg/g	Equivalent to approximately 1 pg/mL or 1 ng/L	
ng/g serum	Conversion to ng/g lipid = ng/g serum ÷ % lipid content in blood serum. This conversion is valid only for lipophilic chemicals.	

APPENDIX D – BIOLOGICAL EQUIVALENTS (BEs)

Table 1: Biological equivalents (where available) for compounds included for analysis in this study.

Chemical Name	Biomonitoring Equivalent (BE)		Reference
	Underlying Exposure Guidance	**BE (biological matrix)**	
Dioxins and Furans			
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) (values can be applied to other dioxin like compounds)	ATSDR MRL monkey data: 1 pg/kg/d	15 ng TEQ/kg lipid (Serum equivalent)	Aylward L.L., Lakind J.S., Hays S.M. Derivation of Biomonitoring Equivalent (BE) Values for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds: A Screening Tool for Interpretation of Biomonitoring Data in a Risk Assessment Context, Journal of Toxicology and Environmental Health, 2008 Part A;71:1499-1508
	WHO PTMI (JECFA) Rat toxicology data: 2.3 pg/kg/d	40-70 ng TEQ/kg lipid (Serum equivalent)	
	EFSA TWI (ECSCF) Rat toxicology data: 2 pg/kg/d)	42-74 ng TEQ/kg lipid (serum equivalent)	
	EFSA TDI (UKCOT) Rat toxicology data: 2 pg/kg/d	31-55 ng TEQ/kg lipid)Serum equivalent)	
Phthalates			
Mono-ethyl phthalate (MEP) (from diethyl phthalate)	USEPA RfD 8 x 10^{-1} mg/kg/day	23 µg/g creatinine; 18 µg/L urine	Aylward L.L., Hays S.M., Gagne M., Krishnan K. Derivation of Biomonitoring Equivalents for di-n-butyl phthalate (DBP), benzylbutyl phthalate (BzBP), and diethyl phthalate (DEP), Regulatory Toxicology and Pharmacology, 2009;55(3):259-267
Mono-benzyl phthalate (MBzP) [from benzyl-butyl phthalate)	Health Canada TDI oral 1.3 mg/kg/day	40 µg/g creatinine; 31 µg/L urine	Aylward L.L., Hays S.M., Gagne M., Krishnan K. Derivation of Biomonitoring Equivalents for di-n-butyl phthalate (DBP), benzylbutyl phthalate (BzBP), and diethyl phthalate (DEP), Regulatory Toxicology and Pharmacology, 2009;55(3):259-267
	USEPA RfD 2 X 10E- 1 mg/kg/day	4.9µg/g creatinine; 3.8 µg/L	
	EFSA TDI 5 X10E- 1 mg/kg/day	15 µg/g creatinine; 12 µg/L	
Di(2-ethylhexyl) phthalate as Mono (2-ethylhexyl) phthalate (MEHP) + Mono (2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) + Mono (2-ethyl-5-oxohexyl) phthalate (MEOHP)	Health Canada TDI, oral 4.4 X 10E-2 mg/kg/day	780 µg/g creatinine; 610 µg/L urine (vol adj)	Aylward L.L., Hays S.M., Gagne M., Krishnan K. Derivation of Biomonitoring Equivalents for di(2-ethylhexyl)phthalate (CAS No. 117-81-7), Regulatory Toxicology and Pharmacology, 2009;55:249-258
	USEPA Chronic RfD 2 x 10E- 2 mg/kg/day	340 µg/g creatinine; 260 µg/L urine (vol adj)	
	ATSDR Chronic MRL 6 X 10E- 2 mg/kg/day	1000 µg/g creatinine; 800 µg/L urine (vol adj)	
	ATSDR Intermediate MRL 1 X10E- 1 mg/kg/day	2500 µg/g creatinine; 1900 µg/L urine (vol adj)	

Chemical Name	Biomonitoring Equivalent (BE)		Reference
	EFSA TDI oral 5 X 10E-2 mg/kg/day	850 µg/g creatinine; 660 µg/L (vol adj)	
Mono-isononyl phthalate (MiNP)	Health Canada pTDI 0.29 mg/kg/day	220 µg/g creatinine	Hays S.M., Aylward L.L., Kirman C.R., Krishnan K., Nong A. Biomonitoring Equivalents for di-isononyl phthalate (DINP), Regulatory Toxicology and Pharmacology, 2011;60:181-188
		170 µg/L urine	
	USEPA ADI US CPSC 0.12 mg/kg/day	92 µg/g creatinine	
		72 µg/L urine	
	EFSA TDI 0.15 mg/kg/day	110 µg/g creatinine	
		89 µg/L urine	
Organochlorine Pesticides			
Hexachlorobenzene	Health Canada TDI, oral 5x 10E-5 mg/kg/day	25 ng/g lipid (serum lipid adjusted)	Aylward L.L., Hays S.M., Gagne M., Nong A., Krishnan K. Biomonitoring equivalents for hexachlorobenzene, Regulatory Toxicology and Pharmacology, 2010;58:25-32
	Health Canada CRSD 10-4 of 1.20E-04 mg/kg/day	1500 ng/g lipid (serum lipid adjusted)	
	Health Canada CRSD 10-5 of 1.20E-05 mg/kg/day	150 ng/g lipid (serum lipid adjusted)	
	Health Canada CRSD 10-6 of 1.20E0-6 mg/kg/day	15 ng/g lipid (serum lipid adjusted)	
	US EPA RfD (8E-04 mg/kg/day)	340 ng/g lipid	
	US EPA CRSD 10-4 of 6.25E-05 mg/kg/day	800 ng/g lipid in Serum	
	US EPA CRSD (10-5) of 6.25E-06 mg/kg/day	80 ng/g lipid in Serum	
	US EPA CRSD 10-6 of 6.26E-07 mg/kg/day	8 ng/g lipid in Serum	
	ATSDR: MRL, oral (5E-04 mg/kg/day)	47 ng/g lipid in serum	
	WHO TDI, oral	82 ng/g lipid (1.7E-04 mg/kg/day)	
	WHO: TDI neoplastic	43 ng/g lipid (1.6E-04 mg/kg/day)	
4,4-DDT	USEPA Rfd 0.0005 mg/kg-d	4000 ng/g lipid blood	Kirman C.R., Aylward L.L., Hays S.M., Krishnan K., Nong A. Biomonitoring Equivalents for DDT/DDE, Regulatory Toxicology
	USEPA CRSD 10E-4	3000 ng/g lipid blood	

Chemical Name	Biomonitoring Equivalent (BE)		Reference
	0.0000029 mg/kg-day		and Pharmacology, 2011;60:172-180
	USEPA CRSD 10E-6 0.00029 mg/kg/day	30 ng/g lipid blood	
	ATSDR: MRL intermediate 0.0005 mg/kg-d	4000 ng/g lipid blood	
	WHO pTDI (adopted by Health Canada) 0.01 mg/kg-d	30,000 ng/g lipid blood	
	EFSA RIVM 0.0005 mg/kg-d	4000 ng/g lipid blood	
DDT + DDE + DDD (4,4' for each)	USEPA Rfd 0.0005 mg/kg-d	5000 ng/g lipid blood	Kirman C.R., Aylward L.L., Hays S.M., Krishnan K., Nong A. Biomonitoring Equivalents for DDT/DDE, Regulatory Toxicology and Pharmacology, 2011;60:172-180
	USEPA CRSD 10E-4 0.0000029 mg/kg-day	5000 ng/g lipid blood	
	USEPA CRSD 10E-6 0.00029 mg/kg/day	50 ng/g lipid blood	
	ATSDR: MRL intermediate 0.0005 mg/kg-d	5000 ng/g lipid blood	
	WHO pTDI (adopted by Health Canada) 0.01 mg/kg-d	40,000 ng/g lipid blood	
	EFSA RIVM 0.0005 mg/kg-d	5000 ng/g lipid blood	
Phenols			
Bisphenol A (BPA)	Health Canada's pTDI of 25 µg/kg-d	1 mg/L urinary,	Krishnan K., Gagane M., Nong A., Aylward L.L., Hays S.M. Biomonitoring Equivalents for bisphenol A (BPA), Regulatory Toxicology and Pharmacology, 2010;58:18-24
	Health Canada's pTDI of 25 µg/kg-d	1.3 mg/L (urine creatinine)	
	RfD, USA EPA 1993 value of 0.05 mg/kg/day	2 000 µg/L urine (volume)	
	RfD, USA EPA 1993 value	2.6 mg/L urine (creatinine)	
	TDIc, (EFSA-EU, 2006) of 50 µg/kg -day	2.6 mg/g urine (creatinine) and 2 mg/L urine (volume)	
Polybrominated diphenyl ethers			
BDE 99	USEPA RfD 0.1 µg/kg/day	520 ng/g serum/plasma/blood	Krishnan K., Adamou T., Aylward L.L., Hays S.M., Kirman C.R., Nong

Chemical Name	Biomonitoring Equivalent (BE)		Reference
			A. Biomonitoring Equivalents for 2,20,4,40,5-pentabromodiphenylether (PBDE-99), Regulatory Toxicology and Pharmacology, 2011;60:165-171
Metals and Minerals			
Arsenic (inorganic iAs+MMA+DMA)	Health Canada MAC (maximum acceptable concentration) 10-4 of 5.6E-05 mg/kg/day	1.7 µg/g urine creatinine	Hays S.M., Aylward L.L., Gagne M., Nong A., Krishnan K. Biomonitoring Equivalents for inorganic arsenic, Regulatory Toxicology and Pharmacology, 2010;5:1–9
	Health Canada MAC (maximum acceptable concentration) 10-6 of 5.6E-07 mg/kg/day	1.7E-02 µg/g creatinine	
	Health Canada Food Division CRSD 10-4 = 3.3 x 10E-3 mg/kg/day	100 µg/g creatinine	
	Health Canada Food Division CRSD 10-6 = 3.3 x 10E-5 mg/kg/day	1 µg/g creatinine	
	Health Canada PMRA CRSD 10-4 = 2.7 X 10E- 5 mg/kg/day	0.84 µg/g creatinine	
Arsenic (inorganic iAs+MMA+DMA)	Health Canada PMRA CRSD 10-6 = 2.7 X 10E- 7 mg/kg/day	8.4E-03 µg/g creatinine	Hays S.M., Aylward L.L., Gagne M., Nong A., Krishnan K. Biomonitoring Equivalents for inorganic arsenic, Regulatory Toxicology and Pharmacology, 2010;5:1–9
	US EPA RfD 3 X 10 E-04 mg/kg-d US EPA CRSD 10-4 = 2.7 X 10E- 5 mg/kg/day	8.3 µg/g creatinine (6.4 µg/L urine) 0.84 µg/g creatinine	
	US EPA CRSD 10-6 = 2.7 X 10E- 7 mg/kg/day	8.4E-03 µg/g creatinine	
	ASTDR: acute MRL 5 X10E- 3 mg/kg-d	155.6 µg/g creatinine (120.9 µg/L urine)	
	ASTDR: chronic MRL 3 X10E-3 mg/kg-d	8.3 µg/g creatinine (6.4 µg/L urine)	
Cadmium	US EPA Chronic RfD (0.0005 mg/kg/day in water; 0.001 mg/kg/day in food)	1.7 µg/L blood	Hays S.M., Nordberg M., Yager J.W., Aylward L.L. Biomonitoring equivalents (BE) dossier for cadmium (Cd) (CAS No. 7440-43-9), Regulatory Toxicology and Pharmacology, 2008;51: S49-S56
	US EPA Chronic RfD	2..0 µg/g creatinine;	

Chemical Name	Biomonitoring Equivalent (BE)		Reference
	(0.0005 mg/kg/day in water; 0.001 mg/kg/day in food)	1.5 µg/L urine	
	ASTDR Chronic MRL, oral (0.0002 mg/kg/day)	1.7 µg/L blood	
	ASTDR Chronic MRL, oral (0.0002 mg/kg/day)	2.0 µg/g creatinine; 1.5 µg/L urine	

APPENDIX E – RESULTS COMPARISON TABLE

Comparison of Saskatchewan results to Alberta, CHMS, CDC Fourth Report, and FNBI

Color codes and abbreviations:
Black- Female only population
Red – General population
Blue – Unit conversion
Green – percentile comparison (lowest available percentile is usually taken. If there is a 10th percentile, but it is <LOD, the next percentile is taken)
GM – Geometric mean

Note: the concentrations provided for the Saskatchewan pools include the raw analytical instrument values where applicable (<LOD).

Urine sample: Not comparable to SK
Not reported

Table 1: Comparison of pooled results for the current Saskatchewan study with the Alberta Biomonitoring Program: Chemicals in Serum of Children in Southern Alberta (2004–2006) – Influence of Age and Comparison to Pregnant Women (Alberta Health, 2008), Report on human biomonitoring of environmental chemicals in Canada: Results of the Canadian Health Measures Survey Cycle 1 and Cycle 2 (Health Canada, 2010a; 2013), Center for Disease Control's Fourth Report on Human Exposure to Environmental Chemicals (CDC, 2009) and the Assembly of First Nation's First Nations Biomonitoring Initiative (AFN, 2013). Values given in black text represent concentrations from female only populations, red represents the general population, blue represents unit conversions, and green text provides percentile comparison (lowest available percentile is usually taken, if the lowest percentile is <LOD, the next percentile was used).

Chemical	**Saskatchewan**						**Alberta** (2005)	**CHMS**		**CDC 4th report** (Aug 2014)	**FNBI**
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Alkylphenol in Serum by LCMS											
Octylphenol	19.0 ng/mL	15.0 ng/mL	18.7 ng/mL	13.7 ng/mL	18.7 ng/mL	18.2 ng/mL	*Not reported*			*< LOD Urine creatinine*	
Nonylphenol	<0.200 ng/mL	<0.200 ng/mL	<0.200 ng/mL	<0.200 ng/mL	<0.200 ng/mL	<0.200 ng/mL	12 – 80 ng/mL Blood serum concentrations in pregnant women age 26-30				

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Bisphenol A in serum by LC/MS/MS											
Bisphenol A	<0.20 ng/mL	<0.20 ng/mL	<0.20 ng/mL	<0.20 ng/mL	<0.20 ng/mL	<0.20 ng/mL	0.071 to 0.98 ng/mL Blood serum concentrations in pregnant women age 26-30	GM: 1.4 µg/g creatinine (urine concentrations for 6-79 years) GM: 1.5 µg/g creatinine (females, 6-79 years)	GM: 1.4 µg/g creatinine (urine concentrations for 6-79 years) GM: 1.3 µg/g creatinine (females, 6-79 years)	GM: 2.58 µg/g creatinine GM: 2.78 µg/g creatinine	GM: 1.74 µg/g creatinine GM: 2.02 µg/g creatinine
Phthalate mono-esters											
Monomethyl phthalate	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL		<LOD Creatinine	< LOD creatinine	<LOD Creatinine	< LOD creatinine
Monoethyl phthalate	6.0 ng/mL	4.3 ng/mL	6.1 ng/mL	5.3 ng/mL	4.2 ng/mL	2.5 ng/mL		GM: 62 µg/g creatinine (urine concentrations for 6-49 years) GM: 70 µg/g creatinine (females, 6-49 years)	GM: 40 µg/g creatinine (urine concentrations for 6-49 years) GM: 44 µg/g creatinine (females, 6-49 years)	GM: 181 µg /g creatinine GM: 211 µg/g creatinine	GM: 24.48 µg/g creatinine GM: 31.60 µg/g creatinine
Monoisobutyl phthalate	15.0 ng/mL	13.6 ng/mL	16.9 ng/mL	13.4 ng/mL	13.9 ng/mL	12.8 ng/mL			GM: 13 µg/g creatinine (urine concentrations for 3-79 years) GM: 15µg/g creatinine (females, 3-	GM: 3.57 µg /g creatinine GM: 3.96 µg/g creatinine	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
									79 years)		
Monocyclohexyl phthalate	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL		<LOD Creatinine	< LOD creatinine	<LOD Creatinine	< LOD creatinine
Monobenzyl phthalate	1.38 ng/mL	0.920 ng/mL	1.78 ng/mL	1.54 ng/mL	2.05 ng/mL	1.24 ng/mL		GM: 13 µg/g creatinine (urine concentrations for 6-79 years) GM: 11 µg/g creatinine (females, 6-49 years)	GM: 7.7 µg/g creatinine (urine concentrations for 6-79 years) GM: 8.4 µg/g creatinine (females, 6-49 years)	GM: 12.9 µg /g creatinine GM: 2.78 µg/g creatinine	GM: 18.58 µg/g creatinine GM: 18.81 µg/g creatinine
Mono-(2-ethylhexyl) phthalate	242 ng/mL	157 ng/mL	131 ng/mL	152 ng/mL	134 ng/mL	221 ng/mL		GM: 4 µg/g creatinine (urine concentrations for 6-49 years) GM: 4.2 µg/g creatinine (females, 6-49 years)	GM: 1.8 µg/g creatinine (urine concentrations for 6-49 years) GM: 1.8 µg/g creatinine (females, 6-49 years)	GM: 2.20 µg /g creatinine GM: 2.40 µg/g creatinine	GM: 2.27 µg/g creatinine GM: 2.17 µg/g creatinine
Mono-n-octyl phthalate	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL		<LOD Creatinine	< LOD creatinine	<LOD Creatinine	< LOD creatinine
Mono-(2-ethyl-5-oxohexyl) phthalate	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL		GM: 16 µg/g creatinine (urine concentrations for 6-49 years) GM: 18 µg/g creatinine (females, 6-49 years)	GM: 6.9 µg/g creatinine (urine concentrations for 6-49 years) GM: 7.3 µg/g creatinine (females, 6-49 years)	GM: 13.6 µg /g creatinine GM: 13.4 µg/g creatinine	GM: 7.29 µg/g creatinine GM: 7.69 µg/g creatinine

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Monoisononyl phthalate	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL		<LOD Creatinine	< LOD creatinine	<LOD Creatinine	< LOD creatinine
Mono-(2-ethyl-5-hydroxyhexyl) phthalate	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL	<0.250 ng/mL		GM: 26 µg/g creatinine (urine concentrations for 6-49 years) GM: 28 µg/g creatinine (females, 6-49 years)	GM: 24 µg/g creatinine (urine concentrations for 6-49 years) GM: 12 µg/g creatinine (females, 6-49 years)	GM: 20.4 µg /g creatinine GM: 21.9 µg/g creatinine	GM: 12.26 µg/g creatinine GM: 12,39 µg/g creatinine
Perfluorinated Compounds by LC/MS/MS											
PFHxS	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	0.645 to 9.75 ng/mL Blood serum concentrations in pregnant women	GM: 2.3 µg/L (plasma concentrations for 20-79 years) 2.3 ng/mL 10th percentile: 0.70 µg/L 0.70 ng/mL GM: 1.6 µg/L (females, 20-79 years) 1.6 ng/mL 10th percentile 1.6 µg/L 1.6 ng/mL	GM: 1.7 µg/L (plasma concentrations for 20-79 years) 1.7 ng/mL 10th percentile: 0.55 µg/L 0.55 ng/mL GM: 1.3 µg/L (females, 20-79 years) 1.3 ng/mL 10th percentile: 1.3 µg/L 1.3 ng/mL	GM: 1.93 µg/L serum concentration 1.93 ng/mL 50th percentile = 1.90 µg/L GM: 1.72 µg/L serum concentration 1.72 ng/mL 50th percentile = 1.60 µg/L	GM: 0.86 µg/L (plasma concentrations for 20+ years) 0.86 ng/mL GM: 0.56 µg/L (plasma concentrations for 20+ years) 0.56 ng/mL
PFOS	2.55 ng/mL	3.03 ng/mL	3.33 ng/mL	3.03 ng/mL	3.27 ng/mL	2.88 ng/mL	0.321 to 14.3 ng/mL Blood serum	GM: 8.9 µg/L (plasma concentratio	GM: 6.9 µg/L (plasma concentratio	GM: 20.7 µg/L serum concentration	GM: 3.12 µg/L (plasma concentrations

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
							concentrations in pregnant women	ns for 20-79 years) 8.9 ng/mL 10th percentile: 3.60 µg/L 3.60 ng/mL GM: 7.1µg/L (females, 20-79 years) 7.1 ng/mL 10th percentile 3.0 µg/L 3.0 ng/mL	ns for 20-79 years) 6.9 ng/mL 10th percentile: 2.60 µg/L 2.60 ng/mL GM: 5.7 µg/L (females, 20-79 years) 5.7 ng/mL 10th percentile 2.0 µg/L 2.0 ng/	20.7 ng/mL 50th percentile = 21.2 µg/L GM: 18.4 µg/L serum concentration 18.4 ng/mL 50th percentile = 18.2 µg/L	for 20+ years) GM: 2.14 µg/L (plasma concentrations for 20+ years)
PFDS	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	*Not reported*				
PFOA	0.642 ng/mL	0.780 ng/mL	0.669 ng/mL	0.633 ng/mL	0.786 ng/mL	0.912 ng/mL	1.7 to 3.42 ng/mL Blood serum concentrations in pregnant women	GM: 2.5 µg/L (plasma concentrations for 20-79 years) 2.5 ng/mL 10th percentile: 1.30 µg/L 1.30 ng/mL GM: 2.2 µg/L (females, 20-79 years) 2.2 ng/mL 10th percentile: 1.0 µg/L 1.0 ng/mL	GM: 2.3 µg/L (plasma concentrations for 20-79 years) 2.3 ng/mL 10th percentile: 1.10 µg/L 1.10 ng/mL GM: 2.0 µg/L (females, 20-79 years) 2.0 ng/mL 10th percentile: 0.92 µg/L 0.92 ng/mL	GM: 3.95 µg/L serum concentration 3.95 ng/mL 50th percentile = 4.10 µg/L GM: 3.50 µg/L serum concentration 3.50 ng/mL 50th percentile = 3.60 µg/L	GM: 1.39 µg/L (plasma concentrations for 20+ years) GM: 1.05 µg/L (plasma concentrations for 20+ years)

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
PFNA	0.627 ng/mL	<0.500 ng/mL	0.363 ng/mL	0.585 ng/mL	<0.500 ng/mL	3.45 ng/mL	0.298 to 0.696 ng/mL Blood serum concentrations in pregnant women			GM: 0.966 µg/L serum concentration 0.966 ng/mL 50th percentile = 1.00 µg/L GM: 0.861 µg/L serum concentration 0.861 ng/mL 50th percentile = 0.900 µg/L	GM: 0.72 µg/L (plasma concentrations for 20+ years) GM: 0.63 µg/L (plasma concentrations for 20+ years)
PFDA	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	0.738 ng/mL	0.0248 to 0.348 ng/mL Blood serum concentrations in pregnant women				GM: 0.16 µg/L (plasma concentrations for 20+ years) GM: not calculated due to survey estimates could not be calculated
PFDoA	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	0.0687 to 1.11 ng/mL Blood serum concentrations in pregnant women			< LOD Serum concentration	
PFUA	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	<0.500 ng/mL	0.507 ng/mL	0.0241 to 0.766 ng/mL Blood serum concentrations in pregnant women			< LOD Serum concentration	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
PCB's by High Res/Mass Spectrometry (lipid adjusted) (ng/g lipid)								**Lipid Adjusted**		**Lipid Adjusted**	**Lipid Adjusted**
PCB 2	3.5	4.3	2.4	1.9	2.0	3.1					
PCB 1	1.0×10^2	91	73	55	46	84					
PCB 3	17	16	11	8.1	8.2	14					
PCB 4/10	2.5×10^2	2.0×10^2	1.5×10^2	88	97	1.9×10^2					
PCB 15	18	12	8.3	9.4	6.5	9.4					
PCB 6	39	27	22	14	14	29					
PCB 8	1.7×10^2	1.1×10^2	88	60	61	1.2×10^2					
PCB 9	18	13	10	6.8	6.8	14	*Not Reported*				
PCB 11	18	17	9.8	15	16	11					
PCB 14	<0.38	<0.38	<0.38	<0.38	<0.38	<0.38					
PCB 7	11	7.3	5.8	3.9	4.0	7.3					
PCB 5	4.1	2.5	2.3	1.6	1.7	2.9					
PCB 12	<0.38	<0.38	<0.38	<0.38	<0.38	<0.38					
PCB 13	1.8	1.3	1.0	0.72	<0.38	0.98					
PCB 16	26	14	14	11	11	21					
PCB 19	18	12	10	6.0	6.9	15					
PCB 37	4.8	6.0	2.1	4.3	1.8	1.4					
PCB 26	5.7	3.3	3.1	2.9	2.4	4.1					
PCB 27	4.0	2.2	2.2	1.7	1.8	3.5					
PCB 30	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 31	30	20	16	17	13	21					
PCB 32	15	8.5	8.4	6.5	6.3	12					
PCB 34	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 35	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 36	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 38	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 22	10	7.3	5.4	6.6	4.1	6.4					
PCB 23	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 24	1.3	0.82	0.80	0.64	0.53	1.2					
PCB 28	35	25	17	21	13	23	*Not reported*			GM: 4.90 ng/g lipid 50th percentile: 4.96 ng/g lipid	< LOD Serum concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										GM: 4.99 ng/g lipid 50th percentile: 5.07 ng/g lipid	
PCB 39	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 17	36	19	19	13	14	30					
PCB 29	0.48	0.30	0.27	0.21	<0.12	0.37					
PCB 18	96	53	53	37	38	82					
PCB 21/20/33	22	13	11	12	8.8	14					
PCB 25	2.9	1.9	1.5	1.5	1.2	1.8					
PCB 48/49	13	8.3	7.2	7.7	6.2	7.0					
PCB 55	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 60	1.2	1.5	0.70	1.1	0.75	0.43					
PCB 61	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 73	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 58/67	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 78	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 81	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096	*Not reported*			< LOD Serum concentration	
PCB 41	1.3	0.92	0.80	0.98	0.84	0.74					
PCB 45	3.5	1.7	2.1	1.7	1.6	2.6					
PCB 50	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15					
PCB 57	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 63/76	0.27	<0.096	0.18	0.26	0.20	0.15					
PCB 66	4.6	5.6	2.5	4.6	2.8	1.6	*Not reported*	< LOD Serum concentration		GM: 1.39 ng/g lipid 50th percentile: 1.37 ng/g lipid GM: 1.50 ng/g lipid 50th percentile: 1.41 ng/g lipid	< LOD Serum concentration
PCB 72	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 79	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
PCB 46	1.2	0.59	0.57	0.51	0.56	0.71					
PCB 59/42	3.3	2.1	1.8	1.9	2.0	1.6					
PCB 80	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 64	4.8	4.0	2.7	3.2	2.7	2.4					
PCB 69	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 43/52	23	19	13	14	13	12	*Not reported*				
PCB 44	11	9.1	6.8	7.3	7.1	5.9				GM: 2.06 ng/g lipid 50th percentile: 2.05 ng/g lipid GM: 1.99 ng/g lipid 50th percentile: 1.98 ng/g lipid	
PCB 54	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 56	1.8	2.1	1.1	1.7	1.5	0.65					
PCB 77	0.29	0.50	0.23	0.25	0.30	0.16	*Not reported*				
PCB 70	9.4	11	5.1	7.3	6.0	2.7					
PCB 51	1.1	0.82	0.74	0.77	0.80	0.94					
PCB 53	3.1	2.0	1.8	1.6	1.7	2.6					
PCB 71	2.0	1.7	1.2	1.6	1.3	1.2					
PCB 74	4.0	4.3	1.9	3.6	2.4	1.5	*Not reported*	< LOD plasma concentration		GM: 4.81 ng/g lipid 50th percentile: 4.36 ng/g lipid GM: 5.67 ng/g lipid 50th percentile: 5.38 ng/g lipid	< LOD plasma concentration
PCB 75/65/62	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 47	3.6	2.7	2.5	3.2	2.6	2.5					
PCB 40/68	0.59	0.31	0.25	0.42	0.32	0.29					
PCB 82	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 83/119	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
PCB 85	0.79	0.77	0.54	0.55	1.1	0.26					
PCB 88/121	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 92	1.5	1.5	0.97	1.3	1.6	0.68					
PCB 95	8.9	7.2	5.1	6.4	7.0	4.2					
PCB 96	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 103	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 105	0.95	0.95	0.71	1.1	1.1	0.63	*Not reported*	< LOD plasma concentration		GM: 1.20 ng/g lipid 50th percentile: 1.09 ng/g lipid GM: 1.40 ng/g lipid 50th percentile: 1.20 ng/g lipid	< LOD plasma concentration
PCB 106	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 113	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 120	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 122	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 127	<0.058	<0.058	<0.058	<0.058	<0.058	<0.058					
PCB 94	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17					
PCB 99	3.7	3.3	1.9	2.9	3.4	1.8	*Not reported*	< LOD plasma concentration		GM: 4.16 ng/g lipid 50th percentile: 3.79 ng/g lipid GM: 4.35 ng/g lipid 50th percentile: 3.90 ng/g lipid	< LOD plasma concentration
PCB 100	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15					
PCB 108/86/125	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 111/117	0.25	0.17	<0.12	<0.12	<0.12	<0.12					
PCB 114	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 118	3.4	3.9	2.4	4.1	4.5	2.4	*Not reported*	GM: 4.43		GM: 6.00 ng/g	GM: 2.84 µg/kg

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								µg/kg lipid (plasma concentrations 20-79 years) 4.43 ng/g lipid 25th percentile: 2.49 µg/kg lipid 2.49 ng/g lipid GM: 5.13 µg/kg lipid (plasma concentrations 20-79 years) 5.13 ng/g lipid 25th percentile: 3.05 µg/kg lipid 3.05 ng/g lipid		lipid 50th percentile: 5.19 ng/g lipid GM: 6.99 ng/g lipid 50th percentile: 6.02 ng/g lipid	lipid (plasma concentrations 20+ years) 2.84 ng/g lipid 50th percentile: 2.26 µg/kg lipid 2.26 ng/g lipid GM: 3.15 µg/kg lipid (plasma concentrations 20-79 years) 3.15 ng/g lipid 50th percentile: 2.16 µg/kg lipid 2.16 ng/g lipid
PCB 84/89	2.0	2.1	1.6	1.8	2.2	1.2					
PCB 93	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17					
PCB 112	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 116	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 102	<0.15	<0.15	<0.15	0.24	<0.15	0.17					
PCB 97	1.8	1.2	1.2	1.6	1.9	0.82					
PCB 124	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 87	2.9	3.2	1.9	2.3	3.2	1.1	*Not reported*			GM: 0.656 ng/g	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										lipid 50th percentile: 0.900 ng/g lipid GM: 0.648 ng/g lipid 50th percentile: 0.870 ng/g lipid	
PCB 98	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 104	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 110	5.0	5.2	3.3	4.5	6.6	2.3	*Not reported*			GM: 1.22 ng/g lipid 50th percentile: 1.20 ng/g lipid GM: 1.17 ng/g lipid 50th percentile: 1.17 ng/g lipid	
PCB 123/107/109	<0.13	0.30	<0.13	0.35	<0.13	0.14					
PCB 90/101	9.2	9.0	5.4	6.6	8.9	3.4					
PCB 91	1.4	1.4	0.76	1.0	1.1	0.66					
PCB 115	<0.12	2.9×10^2	<0.12	<0.12	<0.12	<0.12					
PCB 126	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077	*Not reported*			GM: 16.3 pg/g lipid 0.0163 ng/g lipid 50th percentile: 14.7 pg/g lipid 0.0147 ng/g lipid GM: 17.8 pg/g 0.0178 ng/g lipid 50th percentile: 15.7 pg/g lipid	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										0.0157 ng/g lipid	
PCB 130	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 136	0.61	0.63	0.58	0.79	0.82	0.17					
PCB 144	<0.15	<0.15	<0.15	0.28	<0.15	<0.15					
PCB 148	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 151	0.54	0.75	0.50	0.89	0.83	<0.13	*Not reported*			< LOD serum concentration	
PCB 152	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 153/168	4.1	3.6	2.9	6.7	6.8	6.6					
PCB 159	<0.058	<0.058	<0.058	<0.058	<0.058	<0.058					
PCB 161	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 167	<0.077	0.26	0.22	0.28	<0.077	<0.077	*Not reported*	< LOD plasma concentration		GM: 0.494 ng/g lipid 50th percentile: 0.700 ng/g lipid GM: 0.573 ng/g lipid 50th percentile: 0.880 ng/g	< LOD plasma concentration
PCB 128/162	<0.077	0.21	<0.077	<0.077	<0.077	<0.077					
PCB 132	0.77	0.81	0.54	<0.17	0.63	0.63					
PCB 137	<0.077	0.21	0.15	0.42	<0.077	<0.077					
PCB 139/143	<1.7	<1.7	<1.7	<1.7	<1.7	<1.7					
PCB 145	<1.3	<1.3	<1.3	<1.3	<1.3	<1.3					
PCB 150	<1.3	<1.3	<1.3	<1.3	<1.3	<1.3					
PCB 156	0.43	0.44	0.22	0.78	0.69	0.60	5.1 to 13 pg/g serum; 1.0-2.2 ng/g lipid	GM: 2.64 µg/kg lipid (plasma concentrations 20-79 years) 2.64 ng/g lipid 50th		GM: 2.54 ng/g lipid 50th percentile: 3.29 ng/g lipid GM: 2.51 ng/g lipid 50th percentile: 3.42 ng/g lipid	< LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								percentile: 2.78 ng/g lipid GM: 2.71 µg/kg lipid (plasma concentrations 20-79 years) 2.71 ng/g lipid 50th percentile: 2.96 ng/g			
PCB 158/129	<0.077	0.38	0.24	0.51	0.28	0.19	0.90 to 9.1 ng/g lipid ; 5.4 to 55 pg/g serum				
PCB 160/163	0.68	0.65	0.52	1.4	1.1	0.92					
PCB 165	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 141	0.32	0.30	0.24	0.51	0.55	<0.096					
PCB 146	0.52	0.58	0.23	1.1	0.97	0.74	5.1 to 13 pg/g serum; 1.0-2.0 ng/g lipid	GM: 2.02 µg/kg lipid (plasma concentrations 20-79 years) 2.02 ng/g lipid 50th percentile: 2.02 ng/g lipid GM: 2.05		GM: 2.17 ng/g lipid 50th percentile: 2.21 ng/g lipid GM: 2.17 ng/g lipid 50th percentile: 2.35 ng/g lipid	< LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								µg/kg lipid (plasma concentrations 20-79 years) 2.05 ng/g lipid 50th percentile: 2.03 ng/g lipid			
PCB 147/149	2.4	2.4	1.5	2.9	3.2	1.2					
PCB 154	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 138	2.8	2.8	2.3	4.9	4.2	3.4	*Not reported*	GM: 10.13 µg/kg lipid (plasma concentrations 20-79 years) 10.13 ng/g lipid 10th percentile: 3.16 ng/g GM: 10.53 µg/kg lipid (plasma concentrations 20-79 years) 10.53 ng/g lipid 10th percentile:			GM: 6.42 µg/kg lipid (plasma concentrations 20+ years) 6.42 ng/g lipid 25th percentile: 2.20 ng/g lipid GM: 5.83 µg/kg lipid (plasma concentrations 20+ years) 5.83 ng/g lipid 25th percentile: 1.90 ng/g lipid

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								3.16 ng/g lipid			
PCB 155	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 169	<0.058	<0.058	<0.058	<0.058	<0.058	<0.058	*Not reported*			< LOD serum concentration	
PCB 131/142/133	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17					
PCB 134	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 157	0.22	<0.058	<0.19	0.21	0.25	0.18	*Not reported*			GM: 0.605 ng/g lipid 50th percentile: 0.800 ng/g GM: 0.615 ng/g 50th percentile: 0.890 ng/g	
PCB 140	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15					
PCB 164	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 166	<0.058	<0.058	<0.058	<0.058	<0.058	<0.058					
PCB 135	0.48	0.62	0.38	0.35	0.41	0.31					
PCB 170	0.73	0.66	0.69	1.6	1.3	1.1	1.0 to 4.2 ng/g lipid 5.1 to 21 pg/g serum	GM: 4.60 µg/kg lipid (plasma concentrations 20-79 years) 4.60 ng/g lipid 25th percentile: 2.21 ng/g lipid GM: 4.46 µg/kg lipid (plasma concentratio		GM: 5.46 ng/g lipid 50th percentile: 6.30 ng/g lipid GM: 5.14 ng/g lipid 50th percentile: 6.28 ng/g lipid	GM: 3.98 µg/kg lipid (plasma concentrations 20+ years) 3.98 ng/g lipid 50th percentile: 3.74 ng/g lipid GM: 3.52 µg/kg lipid (plasma concentrations 20+ years) 3.52 ng/g lipid 50th percentile: 2.99 ng/g lipid

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								ns 20-79 years) 4.46 ng/g lipid 25th percentile: 2.11 ng/g lipid			
PCB 171	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 172	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	*Not reported*			GM: 0.647 ng/g lipid 50th percentile: 0.900 ng/g lipid GM: 0.627 ng/g lipid 50th percentile: 0.900 ng/g lipid	
PCB 175/182	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 176	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 177	<0.12	<0.12	<0.12	0.48	<0.12	0.34	*Not reported*			GM: 1.13 ng/g lipid 50th percentile: 1.30 ng/g lipid GM: 1.15 ng/g lipid 50th percentile: 1.30 ng/g lipid	
PCB 178	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12	*Not reported*	< LOD plasma concentration		GM: 0.933 ng/g lipid 50th percentile: 1.20 ng/g lipid GM: 0.881 ng/g lipid 50th percentile:	< LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										1.20 ng/g	
PCB 179	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 183	0.60	0.56	0.39	0.71	0.73	0.52	5.6 to 8.8 ng/g lipid 5.1 to 44 pg/g serum	< LOD plasma concentration		GM: 1.45 ng/g lipid 50th percentile: 1.60 ng/g lipid GM: 1.44 ng/g lipid 50th percentile: 1.69 ng/g lipid	< LOD plasma concentration
PCB 190	<0.077	<0.077	<0.077	0.26	<0.077	<0.077					
PCB 191	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 181	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13					
PCB 184	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 186	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 187	1.0	1.0	0.81	1.9	2.0	1.7	1.0 to 22 ng/g lipid 5.1 to 62 pg/g serum	GM: 3.72 µg/kg lipid (plasma concentrations 20-79 years) 3.72 ng/g lipid 25th percentile: 1.79 ng/g lipid GM: 3.66 µg/kg lipid (plasma concentrations 20-79 years) 3.66 ng/g lipid 25th		GM: 4.23 ng/g lipid 50th percentile: 4.60 ng/g lipid GM: 4.12 ng/g lipid 50th percentile: 4.67 ng/g lipid	GM: 3.93 µg/kg lipid (plasma concentrations 20+ years) 3.93 ng/g lipid 50th percentile: 3.83 ng/g lipid GM: 3.50 µg/kg lipid (plasma concentrations 20+ years) 3.50 ng/g lipid 50th percentile: 3.07 ng/g lipid

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								percentile: 1.56 ng/g lipid			
PCB 192	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12	*Not Reported*				
PCB 174	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 193	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096					
PCB 180	2.3	2.1	1.8	5.1	4.3	4.6	1.3 to 13 ng/g lipid 6.3 to 67 pg/g serum	GM: 15.21 µg/kg lipid (plasma concentrations 20-79 years) 15.21 ng/g lipid 10th percentile: 3.71 ng/g lipid GM: 14.6 µg/kg lipid (plasma concentrations 20-79 years) 14.6 ng/g lipid 10th percentile: 3.34 ng/g lipid		GM: 15.1 ng/g lipid 50th percentile: 18.0 ng/g lipid GM: 14.2 ng/g lipid 50th percentile: 17.8 ng/g lipid	GM: 10.45 µg/kg lipid (plasma concentrations 20+ years) 10.45 ng/g lipid 25th percentile: 3.11 ng/g lipid GM: 8.78 µg/kg lipid (plasma concentrations 20+ years) 8.78 ng/g lipid 25th percentile: 2.56 ng/g lipid
PCB 188	<0.19	<0.19	<0.19	<0.19	<0.19	<0.19					
PCB 189	<0.096	<0.096	<0.096	<0.096	<0.096	<0.096	*Not reported*			< LOD serum concentration	
PCB 173	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 185	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 194	<0.17	<0.17	<0.17	1.0	<0.17	0.83	1.0 to 2.2 ng/g	GM: 2.91		GM: 2.69 ng/g	GM: 2.81 µg/kg

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
							lipid 5.3 to 11 pg/g serum	µg/kg lipid (plasma concentrations 20-79 years) 2.91 ng/g lipid 50th percentile: 3.03 ng/g lipid GM: 2.78 µg/kg lipid (plasma concentrations 20-79 years) 2.78 ng/g lipid 50th percentile: 2.93 ng/g lipid		lipid 50th percentile: 4.19 ng/g lipid GM: 2.45 ng/g lipid 50th percentile: 4.00 ng/g lipid	lipid (plasma concentrations 20-79 years) 2.81 ng/g lipid 50th percentile: 2.09 ng/g lipid GM: < LOD
PCB 195	<0.13	<0.13	<0.13	<0.13	<0.13	<0.13	*Not reported*			GM: < LOD 50th percentile: 0.900 ng/g lipid GM: <LOD 50th percentile: 0.970 ng/g lipid	
PCB 200	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 201/204	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15					
PCB 197	<0.077	<0.077	<0.077	<0.077	<0.077	<0.077					
PCB 199	0.77	0.63	0.64	1.5	1.1	1.6	0.87 to 2.2 ng/g lipid			GM: 2.81 ng/g lipid	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
							5.1 to 11 pg/g serum			50th percentile: 3.80 ng/g lipid GM: 2.63 ng/g lipid 50th percentile: 3.70 ng/g	
PCB 203/196	0.66	0.76	0.57	1.0	0.95	1.0				GM: 2.61 ng/g lipid 50th percentile: 3.40 ng/g lipid GM: 2.46 ng/g lipid 50th percentile: 3.32 ng/g lipid	
PCB 202	0.33	1.0	0.39	0.59	0.66	0.44					
PCB 205	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 198	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12					
PCB 206	1.2	1.3	1.3	1.6	1.2	3.2	*Not reported*	< LOD plasma concentration		GM: 2.13 ng/g lipid 50th percentile: 2.34 ng/g lipid GM: 2.05 ng/g lipid 50th percentile: 2.34 ng/g lipid	< LOD plasma concentration
PCB 207	0.32	<0.15	<0.15	<0.15	<0.15	<0.15					
PCB 208	1.1	1.2	0.93	0.98	1.1	1.5					
PCB 209	2.6	2.5	3.1	3.3	3.3	7.1	*Not reported*			GM: 1.40 ng/g lipid 50th percentile: 1.18 ng/g lipid GM: 1.42 ng/g lipid	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										50th percentile: 1.20 ng/g lipid	
Total Mono-TriCB	4.8 ng/g	3.4 ng/g	3.1 ng/g	2.1 ng/g	1.9 ng/g	3.9 ng/g					
Total TetraCB	0.47 ng/g	0.36 ng/g	0.28 ng/g	0.32 ng/g	0.25 ng/g	0.25 ng/g					
Total PentaCB	0.20 ng/g	0.18 ng/g	0.12 ng/g	0.14 ng/g	0.17 ng/g	0.074 ng/g					
Total HexaCB	0.053 ng/g	0.060 ng/g	0.052 ng/g	0.096 ng/g	0.071 ng/g	0.058 ng/g					
Total HeptaCB	0.017 ng/g	0.011 pg/g	<0.005 ng/g	0.037 ng/g	0.038 ng/g	0.034 ng/g					
Total OctaCB	<0.005 ng/g	0.0088 ng/g	<0.005 ng/g	0.019 ng/g	0.0088 ng/g	0.010 ng/g					
Total Nona/DecaCB	0.020 ng/g	0.024 ng/g	0.030 ng/g	0.026 ng/g	0.006 ng/g	0.064 ng/g					
Total PCB	5.6 ng/g	4.0 ng/g	3.6 ng/g	2.8 ng/g	2.4 ng/g	4.4 ng/g					
Note: 13C12 PCB 206 & 209 responded slightly out of limits. Data is considered reliable.											
Dioxins and Furans (lipid adjusted)										**Lipid Adjusted**	
2378 TeCDD	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			<LOD serum concentration	
12378 PeCDD	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			<LOD serum concentration	
123478 HxCDD	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			<LOD serum concentration	
123678 HxCDD	5.9 pg/g lipid	<1.9 pg/g lipid	8.9 pg/g lipid	7.6 pg/g lipid	0 pg/g lipid	0 pg/g lipid	2.8 to 23 pg/g lipid			GM: 17.2 pg/g lipid 50th percentile: 20.0 pg/g lipid GM: 16.9 pg/g lipid 50th percentile: 20.5 pg/g lipid	
123789 HxCDD	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			<LOD serum concentration	
1234678 HpCDD	12 pg/g lipid	16 pg/g lipid	8.9 pg/g lipid	13 pg/g lipid	12 pg/g lipid	13 pg/g lipid	5.5 to 55 pg/g lipid			GM: 25.3 pg/g lipid 50th percentile: 24.9 pg/g lipid	

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										GM: 26.3 pg/g lipid 50th percentile: 26.8 pg/g lipid	
OCDD	90 pg/g lipid	1.1 x 10^2 pg/g lipid	1.1 x 10^2 pg/g lipid	1.1 x 10^2 pg/g lipid	1.1 x 10^2 pg/g lipid	1.0 x 10^2 pg/g lipid	5.3 to 2.8 x 10^2 pg/g lipid				
Total TCDDs	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total PeCDD	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total HxCDD	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	0.040 pg/g	<0.010 pg/g	<0.010 pg/g					
Total HpCDD	0.100 pg/g	0.080 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total PCDDs	0.56 pg/g	0.62 pg/g	0.59 pg/g	0.60 pg/g	0.54 pg/g	0.54 pg/g					
2378 TeCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			< LOD serum concentration	
12378 PeCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			< LOD serum concentration	
23478 PeCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*1.8 to 16 pg/g lipid*			< LOD serum concentration	
123478 HxCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	3.8 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*1.8 to 12 pg/g lipid*			< LOD serum concentration	
123678 HxCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	3.8 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*1.4 to 16 pg/g lipid*			< LOD serum concentration	
123789 HxCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			< LOD serum concentration	
234678 HxCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			< LOD serum concentration	
1234678 HpCDF	<1.9 pg/g lipid	4.1 pg/g lipid	0 pg/g lipid	5.7 pg/g lipid	8.2 pg/g lipid	5.6 pg/g lipid	*2.7 to 24 pg/g lipid*			< LOD serum concentration	
1234789 HpCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	*Not reported*			< LOD serum concentration	
OCDF	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	<1.9 pg/g lipid	3.2 to 17 pg/g lipid				
Total TCDF	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Total PeCDF	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total HxCDF	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total HpCDF	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Total PCDFs	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g	<0.010 pg/g					
Parabens by LC/MS/MS											
Methyl Paraben	12 ng/mL	14 ng/mL	9.7 ng/mL	4.5 ng/mL	7.6 ng/mL	8.2 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Ethyl Paraben	2.6 ng/mL	0.86 ng/mL	0.42 ng/mL	0.66 ng/mL	1.6 ng/mL	1.7 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Propyl Paraben	1.9 ng/mL	2.4 ng/mL	1.5 ng/mL	0.93 ng/mL	1.9 ng/mL	1.4 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Butyl Paraben	0.015 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Benzyl Paraben	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	<0.50 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Methyl Mercury (MeHg)											
Me-Hg	0.1 ng/g	0.1 ng/g	<0.05 ng/g	0.2 ng/g	<0.05 ng/g	0.3 ng/g	0.04 ng/g to 0.2 ng/g blood serum	Only Hg and inorganic Hg measured	Only Hg and inorganic Hg measured	Only Hg and inorganic Hg measured	Only Hg and inorganic Hg measured
Phytoestrogen in serum by LC-MS-MS											
Daidzein	1.69 ng/mL	2.03 ng/mL	1.18 ng/mL	1.48 ng/mL	1.12 ng/mL	0.940 ng/mL	0.704 ng/mL to 6.11 ng/mL blood serum	*Not Reported*	*Not Reported*	GM: 62.5 µg/g urine creatinine GM: 67.4 µg/g urine creatinine, females	*Not Reported*
Genistein	4.66 ng/mL	5.30 ng/mL	3.52 ng/mL	3.78 ng/mL	5.28 ng/mL	3.00 ng/mL	*Not Reported*	*Not Reported*	*Not Reported*	GM: 29.1 µg/g urine creatinine GM: 31.9 µg/g urine	*Not Reported*

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
											creatinine, females	
Chlorophenol by LCMS in Serum												
Pentachlorophenol (PCP)		0.65 ng/g	0.54 ng/g	<0.50 ng/g	<0.50 ng/g	<0.50 ng/g	<0.50 ng/g	*Not Reported*	*Not Reported*	<LOD Creatinine	< LOD creatinine	*Not Reported*
Trichlorophenols		< 0.50 ng/g	< 0.50 ng/g	<0.50 ng/g	<0.50 ng/g	<0.50 ng/g	<0.50 ng/g	*Not Reported*	*Not Reported*	<LOD Creatinine	< LOD creatinine	*Not Reported*
	LOQ	**Metals**										
Beryllium (Be)	0.10 ug/L	<0.10 ug/L	<0.10 ug/L	<0.10 ug/L	<0.10 ug/L	<0.10 ug/L	<0.10 ug/L	*Not Reported*	*Not Reported*	*Not Reported*	< LOD creatinine	*Not Reported*
Boron (B)	2.0 ug/L	24 ug/L	16 ug/L	15 ug/L	16 ug/L	13 ug/L	15 ug/L	13.3 µg/L to 34.4 µg/L Blood serum, pregnant women	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Magnesium (Mg)	25.0 ug/L	1.90 x 10^4 ug/L	1.87 x 10^4 ug/L	1.97 x 10^4 ug/L	1.70 x 10^4 ug/L	1.84 x 10^4 ug/L	1.87 x 10^4 ug/L	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Aluminum (Al)	2.00 ug/L	15.0 ug/L	7.82 ug/L	9.19 ug/L	6.42 ug/L	7.04 ug/L	7.62 ug/L	12 µg/L to 56 µg/L Blood serum, pregnant women	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Titanium (Ti)	5.0 ug/L	< 5.0 ug/L	< 5.0 ug/L	< 5.0 ug/L	< 5.0 ug/L	< 5.0 ug/L	< 5.0 ug/L	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Vanadium (V)	0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	0.261 µg/L to 0.420 µg/L Blood serum, pregnant women	< LOD creatinine	< LOD creatinine	*Not Reported*	< LOD creatinine
Chromium (Cr)	0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	< 0.50 ug/L	0.652 µg/L to 4.62 µg/L Blood serum, pregnant women	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Manganese (Mn)	0.50 ug/L	4.2 ug/L	3.4 ug/L	3.3 ug/L	2.6 ug/L	3.5 ug/L	3.4 ug/L	1.90 µg/L to 21.1 µg/L Blood serum, pregnant women	GM: 9.2 (9.0-9.5) µg/L whole blood concentrations for 6-79 years GM: 9.7 (9.4-9.9) µg/L whole blood concentrations for 6-79 years females	GM: 9.8 (9.5-10) µg/L whole blood concentrations for 6-79 years GM: 10 (9.8-11) µg/L whole blood concentrations for 6-79 years females	*Not Reported*	GM: 12.22 (11.76-12.69) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 13.50 (13.04-13.98) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Iron (Fe)	10.0 ug/L	974 ug/L	1.14 x 10^3 ug/L	1.01 x 10^3 ug/L	967 ug/L	1.08 x 10^3 ug/L	1.23 x 10^3 ug/L	9.8 x 10^2 µg/L to 2.3 x 10^3 µg/L Blood serum, pregnant women	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Cobalt (Co)	0.10 ug/L	0.48 ug/L	0.40 ug/L	0.48 ug/L	0.43 ug/L	0.47 ug/L	0.42 ug/L	0.193 µg/L to 3.62 µg/L Blood serum, pregnant women	*Not Reported*	GM: 0.23 (0.21-0.24) µg/L whole blood concentrations for 3-79 years GM: 0.24 (0.22-0.26) µg/L whole blood concentrations for 3-79	GM: 0.314 (0.303-0.325) µg/g urine creatinine GM: 0.393 (0.378-0.409) µg/g urine creatinine, females	*Not Reported*

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										years females		
Nickel (Ni)	0.100 ug/L	2.08 ug/L	0.544 ug/L	0.437 ug/L	0.375 ug/L	0.391 ug/L	0.444 ug/L	0.386 µg/L to 5.58 µg/L Blood serum, pregnant women	GM: 0.63 (0.57-0.70) µg/L whole blood concentrations for 6-79 years GM: 0.64 (0.58-0.71) µg/L whole blood concentrations for 6-79 years females	GM: 0.48 (0.45-0.51) µg/L whole blood concentrations for 6-79 years GM: 0.47 (0.44-0.50) µg/L whole blood concentrations for 6-79 years females	*Not Reported*	GM: 0.44 (0.37-0.52) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 0.45 (0.38-0.54) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Copper (Cu)	1.00 ug/L	2.08 x 10^3 ug/L	2.13 x 10^3 ug/L	2.06 x 10^3 ug/L	1.87 x 10^3 ug/L	1.82 x 10^3 ug/L	1.81 x 10^3 ug/L	1.7 x 10^3 µg/L to 2.3 x 10^3 µg/L Blood serum, pregnant women	GM: 910 (900-930) µg/L whole blood concentrations for 6-79 years GM: 980 (970-1000) µg/L whole blood concentrations for 6-79 years females	GM: 900 (890-910) µg/L whole blood concentrations for 6-79 years GM: 970 (960-980) µg/L whole blood concentrations for 6-79 years females	*Not Reported*	G.M.: 932.09 (903.50-961.57) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 997.50 (961.07-1035.31) µg/L blood concentration (female, 20 yrs and older, on reserve and

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
												crown land)
Zinc (Zn)	10.0 ug/L	1.53 x 10^3 ug/L	1.38 x 10^3 ug/L	1.36 x 10^3 ug/L	1.30 x 10^3 ug/L	1.38 x 10^3 ug/L	1.47 x 10^3 ug/L	1.20 x 10^3 µg/L to 1.56 x 10^3 µg/L Blood serum, pregnant women	GM: 6.4 (6.3-6.5) mg/L whole blood concentrations for 6-79 years 6400 µg/L GM: 6.1 (6.0-6.2) mg/L whole blood concentrations for 6-79 years females 6100 µg/L	GM: 6.0 (5.9-6.1) mg/L whole blood concentrations for 6-79 years 6000 µg/L GM: 5.7 (5.7-5.8) mg/L whole blood concentrations for 6-79 years females 5700 µg/L	*Not Reported*	GM: 5.75 (5.61-5.88) mg/L blood concentration (20 yrs and older, on reserve and crown land) 5750 µg/L GM: 5.53 (5.39-5.67) mg/L blood concentration (female, 20 yrs and older, on reserve and crown land) 5530 µg/L
Arsenic (As)	0.100 ug/L	0.107 ug/L	< 0.100 ug/L	< 0.100 ug/L	0.114 ug/L	< 0.100 ug/L	0.145 ug/L	*Not Reported*	GM: 14.24 (CI: 11.44-17.72) µg/g creatinine (urine concentrations for 6-79 years) GM: 15.78 (12.61-19.75) µg/g creatinine (females, 6-79 years)	GM: 8.6 (CI: 7.2-10) µg/g creatinine (urine concentrations for 6-79 years) GM: 9.2 (7.6-11) µg/g creatinine (females, 6-79 years)	GM: 8.24 (7.07-9.59) µg/g urine creatinine GM: 8.47 (7.12-10.1) µg/g urine creatinine, females	GM: 0.49 (0.39-0.62) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 0.51 (0.39-0.66) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Selenium (Se)	0.500 ug/L	121 ug/L	121 ug/L	124 ug/L	108 ug/L	120 ug/L	113 ug/L	130 µg/L to 180 µg/L Blood serum, pregnant women	GM: 200 (200-210) µg/L whole blood concentrations for 6-79 years GM: 200 (190-200) µg/L whole blood concentrations for 6-79 years females	GM: 190 (190-190) µg/L whole blood concentrations for 6-79 years GM: 190 (180-190) µg/L whole blood concentrations for 6-79 years females	*Not Reported*	GM: 189.16 (182.18-196.41) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 187.09 (179.93-194.54) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Strontium (Sr)	0.200 ug/L	39.1 ug/L	23.3 ug/L	28.3 ug/L	20.5 ug/L	23.7 ug/L	24.7 ug/L	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Cadmium (Cd)	0.050 ug/L	< 0.050 ug/L	< 0.050 ug/L	< 0.050 ug/L	< 0.050 ug/L	< 0.050 ug/L	< 0.050 ug/L	*Not Reported*	GM: 0.34 (0.31-0.37) µg/L whole blood concentrations for 6-79 years GM: 0.38 (0.35-0.41) µg/L whole blood concentrations for 6-79 years	GM: 0.31 (0.28-0.34) µg/L whole blood concentrations for 6-79 years GM: 0.34 (0.29-0.38) µg/L whole blood concentrations for 6-79 years	GM: 0.304 (.289-.320) µg/L blood concentration GM: 0.326 (0.309-0.344) µg/L blood concentration, females	GM: 0.96 (0.84-1.10) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 1.00 (0.80-1.25) µg/L blood concentration (female, 20 yrs and older, on

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
									females	females		reserve and crown land)
Antimony (Sb)	0.25 ug/L	3.8 ug/L	3.3 ug/L	3.5 ug/L	3.4 ug/L	3.5 ug/L	3.7 ug/L	2.83 µg/L to 14.8 µg/L Blood serum, pregnant women	GM: 0.053 (0.051-0.056) µg/g creatinine (urine concentrations for 6-79 years) GM: 0.055 (0.052-0.059) µg/g creatinine (females, 6-79 years)	GM: 0.045 (0.042-0.047) µg/g creatinine (urine concentrations for 6-79 years) GM: 0.046 (0.043-0.050) µg/g creatinine (females, 6-79 years)	< LOD creatinine	GM: 0.05 (0.04-0.06) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 0.04 (0.04-0.05) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Molybdenum (Mo)	0.10 ug/L	1.1 ug/L	1.3 ug/L	1.2 ug/L	1.3 ug/L	1.2 ug/L	1.2 ug/L	1.06 µg/L to 4.29 µg/L Blood serum, pregnant women	GM: 0.67 (0.66-0.69) µg/L whole blood concentrations for 6-79 years GM: 0.68 (0.66-0.71) µg/L whole blood concentrations for 6-79 years females	GM: 0.65 (0.63-0.67) µg/L whole blood concentrations for 6-79 years GM: 0.67 (0.64-0.70) µg/L whole blood concentrations for 6-79 years females	GM: 39.4 (37.6-41.3) µg/g urine creatinine GM: 40.5 (38.1-43.0) µg/g urine creatinine, females	GM: 0.67 (0.61-0.73) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: 0.68 (0.64-0.71) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Cesiu	0.050	0.33	0.29	0.30	0.37 ug/L	0.30	3.5 ug/L	0.370 µg/L to	*Not Reported*	GM: 4.4 (4.2-	GM: 4.64	*Not Reported*

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
m (Cs)	ug/L	ug/L	ug/L	ug/L		ug/L		0.750 µg/L Blood serum, pregnant women		4.6) µg/g creatinine (urine concentrations for 3-79 years) GM: 4.9 (4.6-5.2) µg/g creatinine (females, 3-79 years)	(4.42-4.87) µg/g urine creatinine GM: 5.05 (4.77-5.35) µg/g urine creatinine, females	
Barium (Ba)	0.500 ug/L	3.53 ug/L	3.19 ug/L	3.07 ug/L	2.63 ug/L	3.04 ug/L	3.48 ug/L	5.11 µg/L to 14.7 µg/L Blood serum, pregnant women	*Not Reported*	*Not Reported*	GM: 1.48 (1.37-1.60) µg/g urine creatinine GM: 1.60 (1.45-1.77) µg/g urine creatinine, females	*Not Reported*
Tungsten (W)	0.1 ug/L	<0.1 ug/L	<0.1 ug/L	<0.1 ug/L	<0.1 ug/L	<0.1 ug/L	<0.1 ug/L	*Not Reported*	*Not Reported*	GM: <LOD creatinine (urine concentrations for 3-79 years) GM: <LOD creatinine (females, 3-79 years)	GM: 0.070 (0.063-0.078) µg/g urine creatinine GM: 0.072 (0.065-0.079) µg/g urine creatinine, females	*Not Reported*
Platinum (Pt)	0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	*Not Reported*	*Not Reported*	*Not Reported*	< LOD creatinine	*Not Reported*
Mercu	0.100	0.322	0.260	0.214	0.330 ug/L	0.411	0.696 ug/L	0.204 µg/L to	GM: 0.69	GM: 0.72	GM: 0.797	GM: 0.95 (0.51-

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
ry (Hg)	ug/L	ug/L	ug/L	ug/L		ug/L		0.844 µg/L Blood serum, pregnant women	(0.55-0.86) µg/L whole blood concentrations for 6-79 years GM: 0.70 (0.56-0.88) µg/L whole blood concentrations for 6-79 years females	(0.57-0.90) µg/L whole blood concentrations for 6-79 years GM: 0.69 (0.55-0.86) µg/L whole blood concentrations for 6-79 years females	(0.703-0.903) µg/L blood concentration GM: 0.781 (0.689-0.886) µg/L blood concentration, females	1.77) µg/L blood concentration (20 yrs and older, on reserve and crown land) GM: survey estimates unreliable for females only population
Thallium (Tl)	0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	*Not Reported*	*Not Reported*	GM: 0.22 (0.20-0.23) µg/g creatinine (urine concentrations for 3-79 years) GM: 0.24 (0.22-0.25) µg/g creatinine (females, 3-79 years)	GM: 0.154 (0.149-0.158) µg/g urine creatinine GM: 0.167 (0.162-0.173) µg/g urine creatinine, females	*Not Reported*
Lead (Pb)	0.10 ug/L	0.51 ug/L	0.59 ug/L	0.62 ug/L	0.27 ug/L	0.45 ug/L	0.43 ug/L	Mostly <0.20 µg/L Few 0.20 µ/L to 1.0 µg/L Blood serum, pregnant women	GM: 1.3 (1.2-1.4) µg/dL whole blood concentrations for 6-79 years 13 µg/L	GM: 1.2 (1.1-1.3) µg/dL whole blood concentrations for 6-79 years 12 µg/L	GM: 1.43 (1.36-1.50) µg/dL blood concentrations 14.3 µg/L GM: 1.22 (1.14-	GM: 1.17 (1.05-1.31) µg/L blood concentration (20 yrs and older, on reserve and

Chemical		Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
		Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
									GM: 1.2 (1.1-1.3) µg/dL whole blood concentrations for 6-79 years females 12 µg/L	GM: 1.1 (1.0-1.1) µg/dL whole blood concentrations for 6-79 years females 11 µg/L	1.31) µg/dL blood concentrations, females 12.2 µg/L	crown land) GM: 0.98 (0.86-1.12) µg/L blood concentration (female, 20 yrs and older, on reserve and crown land)
Uranium (U)	0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	<0.05 ug/L	*Not Reported*	GM: <LOD µg/L whole blood concentrations for 6-79 years GM: <LOD µg/L whole blood concentrations for 6-79 years females	GM: <LOD µg/L whole blood concentrations for 6-79 years GM: <LOD µg/L whole blood concentrations for 6-79 years females	GM: 0.008 (0.007-0.008) µg/g urine creatinine GM: Not calculated: proportion of results below limit of detection was too high to provide a valid result.	< LOD plasma concentration
Silver (Ag)	0.100 µg/L	0.272 µg/L	0.213 µg/L	0.207 µg/L	0.177 µg/L	0.215 µg/L	0.199 µg/L	0.200 µg/L serum to 0.540 µg/L serum, pregnant women	*Not Reported*	GM (95% CI): 0.081 (0.065-0.10) µg/L whole blood in women 20-39 years old	*Not Reported*	*Not Reported*
Cotinine												
Cotinine		58.2 ng/mL	54.6 ng/mL	60.2 ng/mL	63.4 ng/mL	46.8 ng/mL	66.4 ng/mL	5.13 ng/mL to 55.0 ng/mL Blood serum, pregnant women	< LOD Creatinine For non-smokers	< LOD Creatinine for non-smokers	Non-smokers: 0.071 (0.057-0.089) ng/mL serum concentrations	71.97 (46.93-110.37) creatinine urine aged 20 or older

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								GM: 590 (420-820) µg/L creatinine for smokers aged 12-79	GM: 490 (340-700) µg/L creatinine for smokers aged 12-79	GM: 0.060 (0.047-0.077) ng/mL blood concentrations, females	
OC Pesticides (POP Organo-Chlorine Screen) (lipid adjusted)											
2,4'-DDT	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: <LOD µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g serum concentration GM: <LOD ng/g serum concentration, females	< LOD plasma concentration
4,4'-DDD	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
4,4'-DDE	26 ng/g lipid	1.4 x 10^2 ng/g lipid	19 ng/g lipid	68 ng/g lipid	26 ng/g lipid	51 ng/g lipid	0.11 to 1.5 ng/g blood serum, 12 to 2.1 x 10^2 ng/g lipid pregnant women	GM: 94.68 (77.00-116.43) µg/kg lipid (plasma concentrations, age 20-79) GM: 102.15 (74.69-139.71) µg/kg lipid	*Not Reported*	GM: 238 (195-292) ng/g lipid adjusted serum concentration GM: 270 (226-322) ng/g lipid adjusted serum concentration, females	*Not Reported*

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								(plasma concentrations for 20-79 years females)			
4,4'-DDT	13 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: <LOD µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g serum concentration GM: <LOD ng/g serum concentration, females	< LOD plasma concentration
Aldrin	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: <LOD µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g lip adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	< LOD plasma concentration
alpha-BHC	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
alpha-	<12	<13 ng/g	<7.4	<9.0 ng/g	<10 ng/g	<15 ng/g	*Not Reported*	GM: <LOD	*Not Reported*	*Not Reported*	< LOD plasma

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
Chlordane	ng/g lipid	lipid	ng/g lipid	lipid	lipid	lipid		µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females			concentration
beta-BHC	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	14.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: 0.04 (0.03-0.05) µg/L whole blood concentrations for 20-79 years GM: 0.04 (0.03-0.06) µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g lipid adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	GM: 1.82 (1.70-1.95) µg/kg lipid concentration (20 yrs and older, on reserve and crown land) GM: 1.93 (1.69-2.20) µg/kg lipid concentration (female, 20 yrs and older, on reserve and crown land)
delta-BHC	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Dieldrin	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	GM: <LOD ng/g lip adjusted serum concentration	*Not Reported*

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										GM: <LOD ng/g lipid adjusted serum concentration, females	
Endosulfan II	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Endrin	28 ng/g lipid	1.5 x 10^2 ng/g lipid	<7.4 ng/g lipid	17 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	GM: <LOD ng/g lip adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	*Not Reported*
gamma-BHC (Lindane)	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: <LOD µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g lip adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	< LOD plasma concentration
gamma-Chlordane	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: <LOD µg/L whole blood concentratio	*Not Reported*	*Not Reported*	< LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								ns for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females			
Heptachlor	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*	*Not Reported*
Heptachlor Epoxide	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	*Not Reported*	*Not Reported*	GM: <LOD ng/g lipid adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	*Not Reported*
Hexachlorobenzene	13 ng/g lipid	71 ng/g lipid	7.5 ng/g lipid	27 ng/g lipid	<10 ng/g lipid	26 ng/g lipid	0.05 to 0.39 ng/g blood serum (10 to 66 ng/g lipid) pregnant wome	GM: 0.05 (0.05—0.06) µg/L whole blood concentrations for 20-79 years GM: 0.06 (0.05-0.07) µg/L whole blood concentrations for 20-79	*Not Reported*	GM: 15.2 (14.5-15.9) ng/g lipid adjusted serum concentration GM: 15.8 (15.0-16.6) ng/g lipid adjusted serum concentration, females	*Not Reported*

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								years females			
Methoxychlor	<2.3 x 10^2 ng/g lipid	2.7 x 10^2 ng/g lipid	1.5 x 10^2 ng/g lipid	2.0 x10^2 ng/g lipid	2.0 x 10^2 ng/g lipid	3.0 x 10^2 ng/g lipid	*Not Reported*	*Not Reported*		*Not Reported*	*Not Reported*
Mirex	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	0.086 to 0.83 ng/g blood serum (17 to 1.7 x 10^2 ng/g lipid) pregnant women	GM: <LOD µg/L whole blood concentrations for 20-79 years GM: <LOD µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: <LOD ng/g lipid adjusted serum concentration GM: <LOD ng/g lipid adjusted serum concentration, females	< LOD plasma concentration
Octachlorostyrene	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*		*Not Reported*	*Not Reported*	*Not Reported*
Oxychlordane	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: 0.03 (0.02—0.03) µg/L whole blood concentrations for 20-79 years GM: 0.03 (0.02-0.03) µg/L whole blood concentrations for 20-79 years	*Not Reported*	GM: 9.37 (8.69-10.1) ng/g lipid adjusted serum concentration GM: 9.63 (8.89-10.4) ng/g lipid adjusted serum concentration, females	GM: 2.45 (2.21-2.73) µg/kg lipid (20 yrs and older, on reserve and crown land) GM: 2.63 (2.32-2.98) µg/kg (female, 20 yrs and older, on reserve and crown land)

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
								females			
Trans-nonachlor	<12 ng/g lipid	<13 ng/g lipid	<7.4 ng/g lipid	<9.0 ng/g lipid	<10 ng/g lipid	<15 ng/g lipid	*Not Reported*	GM: 0.04 (0.03—0.04) µg/L whole blood concentrations for 20-79 years GM: 0.04 (0.03-0.04) µg/L whole blood concentrations for 20-79 years females	*Not Reported*	GM: 14.7 (13.1-16.5) ng/g lipid adjusted serum concentration GM: 14.5 (13.1-16.1) ng/g lipid adjusted serum concentration, females	GM: 4.13 (3.75-4.54) µg/kg lipid (20 yrs and older, on reserve and crown land) GM: 3.74 (3.40-4.11) µg/kg lipid (female, 20 yrs and older, on reserve and crown land)
Polybrominated diethyl ethers (PBDEs) (lipid adjusted)											**Lipid adjusted**
BDE 28	<0.71 ng/g lipid (<LOQ)	<1.3 ng/g lipid (<LOQ)	<0.76 ng/g lipid (<LOQ)	<1.1 ng/g lipid (<LOQ)	<0.28 ng/g lipid (<LOD)	<0.87 ng/g lipid (<LOD)	2.1 – 98 ng/g, lipid adjusted blood serum pregnant women			GM: 1.19 (1.03 – 1.37) ng/g lipid adjusted serum concentrations (20 years and older) GM: 1.17 (0.990 – 1.38) ng/g lipid adjusted serum concentrations, females	*Not reported*
BDE 47	12 ng/g lipid	29 ng/g lipid	27 ng/g lipid	14 ng/g lipid	7.3 ng/g lipid	9.9 ng/g lipid	11 – 3.4 x 10^2 ng/g, lipid adjusted blood serum pregnant			GM: 19.5 (16.5 – 23.1) ng/g lipid adjusted serum concentrations	<LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
							women			(20 years and older) GM: 19.6 (16.4 – 23.5) ng/g lipid adjusted serum concentrations, females	
BDE 99	6.9 ng/g lipid	12 ng/g lipid	9.3 ng/g lipid	4.9 ng/g lipid	2.6 ng/g lipid	3.7 ng/g lipid	2.5 – 4.7 x 10^2 ng/g, lipid adjusted blood serum pregnant women			<LOD serum concentrations	*Not reported*
BDE 100	3.3 ng/g lipid	9.7 ng/g lipid	8.2 ng/g lipid	5.6 ng/g lipid	2.2 ng/g lipid	3.5 ng/g lipid	2.1 – 98 ng/g, lipid adjusted blood serum pregnant women			GM: 3.77 (3.24 – 4.38) ng/g lipid adjusted serum concentrations (20 years and older) GM: 3.72 (3.15 – 4.40) ng/g lipid adjusted serum concentrations, females	*Not reported*
BDE 153	6.9 ng/g lipid	14 ng/g lipid	10 ng/g lipid	14 ng/g lipid	6.7 ng/g lipid	7.7 ng/g lipid	4.5 – 53 ng/g, lipid adjusted blood serum pregnant women			GM: 5.41 (4.83 – 6.05) ng/g lipid adjusted serum concentrations (20 years and older)	< LOD plasma concentration

Chemical	Saskatchewan						Alberta (2005)	CHMS		CDC 4th report (Aug 2014)	FNBI
	Pool 1	*Pool 2*	*Pool 3*	*Pool 4*	*Pool 5*	*Pool 6*		*Cycle 1*	*Cycle 2*		
										GM: 4.78 (4.20 – 5.43) ng/g lipid adjusted serum concentrations, females	
BDE 154	<0.46 ng/g lipid (<LOQ)	0.70 ng/g lipid	1.0 ng/g lipid	<0.75 ng/g lipid (<LOQ)	<0.50 ng/g lipid (<LOQ)	0.36 ng/g lipid	0.35 – 30 ng/g, lipid adjusted blood serum pregnant women			<LOD serum concentrations	<LOD plasma concentration
BDE 183	<0.18 ng/g lipid (<LOD)	<0.32 ng/g lipid (<LOD)	<1.3 ng/g lipid (<LOD)	<0.22 ng/g lipid (<LOD)	<0.83 ng/g lipid (<LOQ)	<1.7 ng/g lipid (<LOQ)	*Not reported*			<LOD serum concentrations	<LOD plasma concentration
BDE 209	<13 ng/g lipid (<LOD)	<3.4 ng/g lipid (<LOD)	<3.1 ng/g lipid (<LOD)	<2.5 ng/g lipid (<LOD)	<5.8 ng/g lipid (<LOD)	<8.0 ng/g lipid (<LOD)	*Not reported*			<LOD serum concentrations	<LOD plasma concentration
BDE 66	<0.24 ng/g lipid (<LOD)	<0.11 ng/g lipid (<LOQ)	<0.38 ng/g lipid (<LOQ)	<0.38 ng/g lipid (<LOD)	<0.083 ng/g lipid (<LOD)	<0.25 ng/g lipid (<LOQ)	0.24 – 4.5 ng/g, lipid adjusted blood serum pregnant women			<LOD serum concentrations	<LOD plasma concentration
BDE 77	<0.16 ng/g lipid (<LOD)	0.037 ng/g lipid (<LOD)	0.13 ng/g lipid (<LOD)	0.27 ng/g lipid (<LOD)	<0.058 ng/g lipid (<LOD)	0.086 ng/g lipid (<LOD)	*Not Reported*			*Not reported*	<LOD plasma concentration
BDE 85	0.76 ng/g lipid	0.76 ng/g lipid	0.62 ng/g lipid	0.52 ng/g lipid	<0.21 ng/g lipid (<LOQ)	0.25 ng/g lipid	0.40 – 24 ng/g, lipid adjusted blood serum pregnant women			<LOD serum concentrations	<LOD plasma concentration
BDE 138	0.032 ng/g lipid	0.28 ng/g lipid	<0.15 ng/g lipid	0.24 pg/g lipid	<0.058 ng/g lipid (<LOD)	<0.088 ng/g lipid (<LOD)	*Not reported*			*Not reported*	<LOD plasma concentration

REFERENCES:

3M Company. Fluorochemical use, distribution and release overview; EPA Docket # OPPT-2002-0043;1999.

Abadin H.G., Hibbs B.F. and Pohl H.R. Brest-feeding exposure of infants to cadmium, lead and mercury: a public health viewpoint. Toxicol. Indus. Health, 1997;134:495-517

Aboriginal Affairs and Northern Development. 2010. Persistent Organic Pollutants (POPs) Fact Sheet Series: Dichlorodiphenyltrichloroethane (DDT). Government of Canada. Accessed at: https://www.aadnc-aandc.gc.ca/eng/1316102914633/1316103004743

Ademollo N., Ferrara F., Delise M., et al. Nonylphenol and octylphenol in human breast milk. Environment International 2008;34(7): 984-987.

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Silver. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp146.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Antimony. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=332&tid=58

Agency for Toxic Substances & Diseases Registry (ATSDR). 1994. Toxicological Profile of Chlorodibenzofurans. Atlanta, GA: U.S. Department of Health and Human Services. Accessed at: http://www.atsdr.cdc.gov/ToxProfiles/tp32.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological Profile for Diethyl Phthalate. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp73.html

Agency for Toxic Substances & Diseases Registry (ATSDR). 1996. Toxicological Profile of Endrin. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=617&tid=

Agency for Toxic Substances and Disease Registry (ATSDR). 1997a. Toxicological Profile for Di-n-Butyl Phthalate. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp135.html

Agency for Toxic Substances and Disease Registry (ATSDR). 1997b. Toxicological Profile for Di-n-Octylphthalate (DNOP). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp95.html.

Agency for Toxic Substances & Diseases Registry (ATSDR). 1998. Toxicological Profile of Chlorinated dibenzo-p-dioxins. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp104.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 1999a. Toxicological profile for mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=115&tid=24

Agency for Toxic Substances and Disease Registry (ATSDR). 1999b. Toxicological profile for Mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2000a. Toxicological profile polychlorinated biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp17.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2000b. Toxicological Profile for Chromium. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp7.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2002a. Toxicological profile for DDT/DDE/DDD (update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp35.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2002b. Toxicological profile for Hexachlorobenzene (update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp90.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2002c. Toxicological Profile for Di(2-Ethylhexyl)phthalate (DEHP). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=684&tid=65

Agency for Toxic Substances and Disease Registry (ATSDR). 2002d. Toxicological Profile for Beryllium. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp4.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological Profile for Selenium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp92.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004a. Toxicological Profile for Polybrominated Diphenyl Ethers . Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp68.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004b. Toxicological Profile for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp2.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004c. Toxicological Profile for Cesium. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp157.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004d. Toxicological profile for Strontium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp159.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004e. Toxicological Profile: Cobalt. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp33.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2004f. Toxicological Profile for Copper. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp132.pdf

Agency for Toxic Substances and Disease Registration (ATSDR). 2005a. Toxicological Profile for alpha, beta-, gamma- and delta-Hexachlorocyclohexane. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp43.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2005b. Toxicological Profile for Nickel. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/ToxProfiles/tp15.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2007a. Toxicological Profile for Barium and Compounds. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=327&tid=57

Agency for Toxic Substances and Disease Registry (ATSDR). 2007b. Toxicological profile for lead (Update). Atlanta, GA:U.S. Department of Public Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological Profile for Aluminum. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp22.pdf

Agency for Toxic Substances and Disease Registration. 2008b. Toxicological Profile for Phenol. Atlanta, GA: U.S. Department of Health and Human Services. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp115.pdf

Agency for Toxic Substances & Diseases Registry (ATSDR). 2009. Draft Toxicological Profile of Perfluoroalkyls. U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/ToxProfiles/tp200.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2012a. Toxicological Profile for Cadmium. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp5.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2012b. Toxicological Profile for Chromium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp7.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). Sept 2012c. Toxicological Profile for Manganese. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp151.pdf

Agency for Toxic Substances & Diseases Registry (ATSDR). 2013a. Toxicological Profile for Hexachlorobenzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp90.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2013b. Toxicological profile for Uranium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/tp150.pdf

Agency for Toxic Substances and Disease Registry (ATSDR). 2013c. Addendum to the Toxicological profile for Mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Accessed at: http://www.atsdr.cdc.gov/toxprofiles/mercury_organic_addendum.pdf

Ahamed M., Kaleem M. and Siddiqui, J. Environmental lead toxicity and nutritional factors. Clin. Nutr., 2007;26:400-408.

Alaee M., Arias P., Sjödin A., et.al. An overview of commercially used brominated flame retardants, their applications, their use patterns in different countries/regions and possible modes of release. Environ. International, 2003;29:683-689.

Alberta Health and Wellness (AHW). 2008. Alberta Biomonitoring Program: Chemicals in serum of pregnant women in Alberta. Edmonton: Alberta Health and Wellness. Accessed at: https://open.alberta.ca/publications/9780778566953

Alberta Health and Wellness (AHW). 2010. Chemicals in serum of children in Southern Alberta (2004-2006) – Influence of age and comparison to pregnant women. Edmonton: Alberta Health and Wellness

Alberta Reproductive Health Report Working Group. 2011. *Alberta Reproductive Health: Pregnancies and Births Table Update 2011.* Edmonton, AB: Alberta Health and Wellness.

Albro P.W., and Lavenhar S.R. Metabolism of di(2-ethylhexyl) phthalate. Drug Metab. Rev., 1989;21:13-34.

Alwan A. Global status report on noncommunicable diseases 2010. World Health Organization, 2011.

Arnold SM, Zarnke RL, Lynn TV, Circumpolar Chimonas M-AR, Frank A. Public health evaluation of cadmium concentrations in liver and kidney of moose (*Alces alces*) from four areas of Alaska. Sci Total Envir 2006;357:103-111

Artic Monitoring and Assessment Programme (AMAP). 2004. AMAP Assessment 2002: Persistent Organic Pollutants in the Arctic. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway. xvi +310 pp.

American Academy of Pediatrics. Breastfeeding and the use of human milk. Pediatrics, 1997;100:1035-1039.

Apelberg B.J., Goldman L.R., Calafat A.M., et al., Determinants of fetal exposure to polyfluoroalkyl compounds in Baltimore, Maryland. Environ. Sci. Technol., 2007; 41:3891-3897.

Apelberg B.J., Witer F.R., Herbstman J.B., et al. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. Environ. Health Persp., 2007;115:1670-1676.

Armbruster D.A., Tillman M.D. and Hubbs L.M. Limit of Detection (LOD)/Limit of Quantitation (LOQ): Comparison of the Empirical and Statistical Methods Exemplified with GC-MS Assays of Abused Drugs. Clin Chem. 1994;40(7):1233-1238

Arbuckle T.E. Maternal-Infant Biomonitoring of Environmental Chemicals: The Epidemiologic Challenges. Birth Defects Research (Part A), 2010;88:931–937

Aris, A. Estimation of bisphenol A (BPA) concentrations in pregnant women, fetuses and nonpregnant women in Eastern Townships of Canada. Reproductive Toxicology, 2014;45:8-13.

Assembly of First Nations (AFN). 2013. First Nations Biomonitoring Initiative: National Results (2011). Accessed at: http://www.afn.ca/uploads/files/afn_fnbi_en_-_2013-06-26.pdf

Atkinson C., Frankenfeld C.L. and Lampe J.W. Gut bacterial metabolism of the soy isoflavone daidzein: exploring the relevance to human health. Experimental Biology and Medicine, 2005;230(3):155-170.

Aylward L.L., Hays S.M., Gagne M., Nong A., Krishnan K. Biomonitoring equivalents for hexachlorobenzene. Regulatory Toxicology and Pharmacology, 2010;58:25-32

Baccarelli A., Pfeiffer R., Consonni D., et al. Handling of dioxin measurement data in the presence of non-detectable values: overview of available methods and their application in the Seveso chloracne study. Chemosphere 2005;60(7): 898-906.

Baker E.L., Landrigan P.J., Glueck C.J., et.al. Metabolic consequences of exposure to polychlorinated biphenyls (PCBs) in sewage sludge. Am. J. Epidemiol., 1980;112: 553- 563.

Bakor F., Damluji S.F., and Amin-Zaki L. Methylmercury poisoning in Iraq. Science, 1973;180:230-241.

Bandera E.V., King M., Chandran U., et al. Phystoestrogen consumption from foods and supplements and epithelial ovarian cancer: a population-based case control study. BMC Women's Health. 2011;11(40).

Baxter D.C., Faarinen M., Österlund H., et al. Serum/plasma methylmercury determination by isotope dilution gas chromatography- inductively coupled plasma mass spectrometry. Analytica chimica acta, 2011;701(2):134-138.

Baxter D.C., Rodushkin I., Engström E., et al. Methylmercury measurement in whole blood by isotope-dilution GC-ICPMS with 2 sample preparation methods. Clinical chemistry, 2007;53(1):111-116.

Bechi N., Ietta F., Romagnoli R., et al. Estrogen-like response to p-nonylphenol in human first trimester placenta and BeWo choriocarcinoma cells, Toxicol. Sci., 2006;93:75-81.

Begley T.H., White K., Honigfort P. Perfluorochemicals: Potential sources of and migration from food packaging. Food Addit. Contam., 2005;22:1023-1031.

Bellanger T.M., Caesar E.M. and Trachtman L. Blood mercury levels and fish consumption in Louisiana. J. La State Med. Soc., 2000;152:64-73.

Benowitz N.L. and Jacob P. III. Daily intake of nicotine during cigarette smoking. Clin. Pharmacol. Ther., 1984;35:499-504.

Benowitz N.L. The use of biologic fluid samples in assessing smoke consumption. In: Grabowski J, Bell CS, eds. Measurement in the analysis and treatment of smoking behavior. NIDA research monograph no. 48. Washington, DC: US GPO, 1983:6-26. (DHHS publication no. (ADM) 83-1285).

Benowitz N.L., Hall S.M., Herning R.I., et al. Smokers of low-yield cigarettes do not consume less nicotine. N. Engl. J. Med., 1983;309:139-142.

Bergdahl I.A., Schutz A., Ahlqwist M., et al. Methylmercury and inorganic Mercury in Serum – correlation to fish consumption and dental amalgam in a cohort of women born in1922, Environ. Res., 1998;77:20-24.

Biles J.E., McNeal T.P. and Begley T.H. Determination of bisphenol A migrating from epoxy can coatings to infant formula liquid concentrates. J. Agricul. Food Chem., 1997;45:4697-4700

Botella B., Crespo J., Ana Rivas A., et al. Exposure of women to organochlorine pesticides in Southern Spain. Environ. Res., 2004;96:34-40.

Brotons J.A., Olea-Serrano M.F., Villalobos M., et al. Xenoestrogens released from lacquer coatings in food cans. Environ. Health Persp., 1995;103:608-612.

Burridge E. Bisphenol A product profile. Eur. Chem. News, 2003;14-20.

Butenhoff J.L., Kennedy G.L., Frame S.R., et al. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. Toxicol., 2004;196:95-116

Calafat A.M., Kuklenyik Z., Caudill S.P., et al. Perfluorochemicals in pooled serum samples from United States residents in 2001 and 2002. Environ. Sci. Technol., 2006;40:2128-2134.

Calafat A.M., Ye X., Wong, L.-Y., et al. Exposure of the U.S. population to Bisphenol A and 4-tertiary-octylphenol 2003-2004. Environ. Health Perspect., 2008;116:39-44.

Calafat, A.M., et al. Exposure to di-(2-ethylhexyl) phthalate among premature neonates in a neonatal intensive care unit. 2004. Pediatrics 113(5):e429-e434.

Canada Gazette, Vol. 137, No. 8, April 9, 2003

Canada Gazette. 2006a. PCB Regulations, November 4, 2006.

Canada Gazette. 2006b. Polybrominated Diphenyl Ethers Regulations, in Canada Gazette. December, 2006.

Canada Gazette. 2006c. Perfluorooctane sulfonate and its salts and certain other compounds regulations, Vol. 140, No. 50, December 16, 2006.

Canada Gazette. 2006d. Order adding toxic substances to Schedule 1 to the Canadian Environment Protection Act, 1999. Vol. 140, No. 24, June 17, 2006.

Canada Gazette. 2010. Vol. 144, No. 37 — September 11, 2010. Accessed at: http://www.gazette.gc.ca/rp-pr/p1/2010/2010-09-11/pdf/g1-14437.pdf

Canada Gazette. 2010b. Order Adding a Toxic Substance to Schedule 1 to the Canadian Environmental Protection Act, 1999. Vol. 144, No. 21 — October 13, 2010. Accessed at: http://www.gazette.gc.ca/rp-pr/p1/2010/2010-05-22/pdf/g1-14421.pdf

Canada Gazette. 2010c. Order Amending Schedule 1 to the Hazardous Products Act (bisphenol A). Vol. 144, No. 7 — March 31, 2010. Accessed at: http://www.gazette.gc.ca/rp-pr/p1/2010/2010-02-13/pdf/g1-14407.pdf

Canadian Council of Ministers of the Environment (CCME). 1999. Canadian sediment quality guidelines for the protection of aquatic life: Chlordane. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg

Canadian Council of Ministers of the Environment (CCME). 1999b. Canadian sediment quality guidelines for the protection of aquatic life: Endrin. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg

Canadian Council of Ministers of the Environment (CCME). 1999c. Canadian water quality guidelines for the protection of aquatic life: Chromium — Hexavalent chromium and trivalent chromium. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Canadian Council of Ministers of the Environment (CCME). 1999d. Canadian soil quality guidelines for the protection of environmental and human health: Lead (1999). In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Canadian Council of Ministers of the Environment (CCME). 1999e. Canadian Soil Quality Guideline for the Protection of Environnemental and Human Health: Mercury (inorgranic). Accessed at: http://ceqg-rcqe.ccme.ca/download/en/270/

Canadian Council of Ministers of the Environment (CCME). 1999f. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Copper. Winnipeg, MB. Accessed at: http://ceqg-rcqe.ccme.ca/download/en/263

Canadian Council of Ministers of the Environment (CCME). 1999g. Canadian water quality guidelines for the protection of aquatic life: Molybdenum. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Canadian Council of Ministers of the Environment (CCME). 1999h. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health – Nickel. Accessed at: https://www.ccme.ca/files/ceqg/en/backup/272.pdf

Canadian Council of Ministers of the Environment (CCME). 2010. Canadian water quality guidelines for the protection of aquatic life: Endosulfan. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Canadian Council of Ministers of the Environment (CCME). 2013. Canadian soil quality guidelines for the protection of environmental and human health: Barium. In: Canadian environmental quality guidelines, Canadian Council of Ministers of the Environment, Winnipeg.

Carpenter D.O. Polychlorinated biphenyls (PCBs): routes of exposure and effects on human health. Rev. Environ. Health, 2006;21:1-23.

Castles A., Adams E.K., Melvin C.L., et al. Effects of smoking during pregnancy: Five meta-analyses. Am. J. Prev. Med., 1999;16: 208-215.

Caudill, S. P. Characterizing populations of individuals using pooled samples. Journal of Exposure Science and EnvironmentalEpidemiology, 2010;20(1):29-37.

Centers for Disease Control and Prevention (CDC). Iron Deficiency – United States, 1999-2000. MMWR 2002;51:897-899.

Centers for Disease Control and Prevention (CDC). 2003. Second National Report of Human Exposure to Environmental Chemicals. Atlanta, GA. National Center for Environmental Health. NCEH Pub No 05-0570.

Centers for Disease Control and Prevention (CDC). 2008. National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population 1999–2002. Department of Health and Human Services. Accessed at: http://www.cdc.gov/nutritionreport/99-02/pdf/nutrition_report.pdf

Centers for Disease Control and Prevention (CDC). 2009. Fourth Report on Human Exposure to Environmental Chemicals, 2009. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Accessed at: http://www.cdc.gov/exposurereport/

Centers for Disease Control and Prevention (CDC). 2012. National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population 1999-2000. Accessed at: http://www.cdc.gov/nutritionreport/99-02/pdf/nr_ch4b.pdf

Center for Disease Control and US Department of Health and Human Services, 2012b. Lead in Drinking Water and Human Blood Lead Levels in the United States. MMWR 2012;61:1-9.

Centers for Disease Control and Prevention (CDC). 2013a. National Biomonitoring Program. Biomonitoring Summary: Cotinine. Accessed at: http://www.cdc.gov/biomonitoring/Cotinine_BiomonitoringSummary.html

Center for Disease Control and Prevention (CDC). 2013b. Biomonitoring Summary: DDT. Accessed at: http://www.cdc.gov/biomonitoring/DDT_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013c. Biomonitoring Summaries: Hexachlorocyclohexane Accessed at: http://www.cdc.gov/biomonitoring/Hexachlorocyclohexane_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013d. National Biomonitoring Program: Biomonitoring Summary Parabens. Accessed at: http://www.cdc.gov/biomonitoring/Parabens_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013e. National Biomonitoring Program: Paraben Factsheet. Accessed at: http://www.cdc.gov/biomonitoring/Parabens_FactSheet.html

Centers for Disease Control and Prevention (CDC). 2013f. Biomonitoring Summaries: Antimony. Accessed at: http://www.cdc.gov/biomonitoring/Antimony_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013g. Biomonitoring Summary: Arsenic. Accessed at: http://www.cdc.gov/biomonitoring/Arsenic_FactSheet.html

Centers for Disease Control and Prevention (CDC). 2013h. Biomonitoring Summary: Beryllium. Accessed at: http://www.cdc.gov/biomonitoring/Beryllium_BiomonitoringSummary.html

Center for Disease Control and Prevention (CDC). 2013i. Biomonitoring Summary: Cadmium. Accessed at: http://www.cdc.gov/biomonitoring/Cadmium_FactSheet.html

Centers for Disease Control and Prevention (CDC). 2013j. Biomonitoring Summary: Cesium. Accessed at: http://www.cdc.gov/biomonitoring/Cesium_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013k. Biomonitoring Summaries: Lead. Accessed at: http://www.cdc.gov/biomonitoring/Lead_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013l. Biomonitoring Summaries: Mercury. Accessed at: http://www.cdc.gov/biomonitoring/Mercury_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013m. National Biomonitoring Program. Biomonitoring Summary: Molybdenum. Accessed at http://www.cdc.gov/biomonitoring/Molybdenum_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013n. National Biomonitoring Program. Biomonitoring Summary: 4-*tert*-Octylphenol. Accessed at: http://www.cdc.gov/biomonitoring/Octylphenol_BiomonitoringSummary.html

Center for Disease Control and Prevention (CDC) 2013o. Chemical factsheet: Polybrominated Diphenyl Ethers (PBDEs) and Polybrominated Biphenyls (PBBs). Accessed at: http://www.cdc.gov/biomonitoring/PBDEs_FactSheet.html

Center for Disease Control and Prevention (CDC). 2013p. Biomonitoring Summary: Bisphenol A. Accessed at:http://www.cdc.gov/biomonitoring/BisphenolA_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2013q. Biomonitoring Summary: Phytestrogen. Accessed at: http://www.cdc.gov/biomonitoring/Phytoestrogens_BiomonitoringSummary.html

Centers for Disease Control and Prevention (2013r) Biomonitoring Summary: Mirex Accesed at: http://www.cdc.gov/biomonitoring/Mirex_BiomonitoringSummary.html

Centers for Disease Control and Prevention (CDC). 2014. Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Accessed at: http://www.cdc.gov/exposurereport/

Centers for Disease Control and Prevention (CDC). 2015. Fourth National Exposure Report – Updated Tables (2015) http://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf

Chan L, Receveur O, Sharp D, et al. First Nations Food, Nutrition and Environment Study (FNFNES): results form British Columbia (2008/2009). Prince George: University of Northern British Columbia, 2011

Chan L, Receveur O, Sharp D, Schwartz H, Ing A, Fediuk K, Black A, Constantine T. First Nations Food, Nutrition and Environment Study (FNFNES): results from Manitoba (2010). Prince George: University of Northern British Columbia. 2012.

Chan L, Receveur O, Batal M, et al. First Nations Food, Nutrition and Environment Study [FNFNES]: results form Ontario (2011/2012). Ottawa: University of Ottawa, 2014.

Chan L, Receveur O, Batal M, et al. First Nations Food, Nutrition and Environment Study [FNFNES]: results from Alberta 2013. Ottawa: University of Ottawa, 2016.

Chan L, Receveur O, Batal M, et al. First Nation Food, Nutrition and Environment Study (FNFNES): Results from Saskatchewan (2015). Ottawa: University of Ottawa, 2018

Charania N.A., Tsuji L.J.S., Martin J.D., et al. An examination of traditional foods and cigarette smoking as cadmium sources among the nine First Nations of Eeyou Istchee, northern Quebec, Canada. Environ. Sci. Processes Impacts, 2014;16:1422-1433.

Charlier C.J. and Foidart J.M. Comparative study of dichlorodiphenyldichloroethylene in blood and semen of two young male populations: lack of relationship to infertility, but evidence of high exposures of the mothers, Reprod. Toxicol., 2005;20:215-220.

Chedrese P.J. and Feyles F. The diverse mechanism of action of dichlorodiphenyldichloroethylene (DDE) and methoxychlor in ovarian cells in vitro. Reprod. Toxicol., 2001;15:693-698.

Chen M.L., Chang C.C., Shen Y.J., et al. Quantification of prenatal exposure and maternal-fetal transfer of nonylphenol. Chemosphere, 2008;*73*(1):S239-S245.

Chen A., Park J.-S., Linderholm L., et al. Hydroxylated polybrominated diphenyl ethers in paired maternal and cord sera. Environmental Science & Technology, 2013;*47*(8): 3902–8. doi:10.1021/es3046839

Clark K., Cousins I.T., and Mackay D. Assessment of critical exposure pathways. In Staples CA (ed), The Handbook of Environmental Chemistry, Vol.3, Part Q: Phthalate Esters. 2003;New York, Springer: 227-262.

Cohen J.T., Bellinger D.C., Connor W.E., et al. A quantitative risk-benefit analysis of changes in population fish consumption. Am J Prev Med 2005;29(4):325-334

Coors A., Jones P.D., Giesy J.P., et al. Removal of estrogenic activity from municipal waste landfill leachate assessed with a bioassay based on reporter gene expression. Environ. Sci. Technol., 2003;37:3430-3434.

Cornwell T., Cohick W. and Raskin I. Dietary phytoestrogens and health. Phytochem., 2004;65:995-1016.

Cotterchio M., Boucher B.A., Manno M., et al. Dietary phytoestrogen intake is associated with reduced colorectal cancer risk. J. Nutr., 2006;136:3046-3053

Cotterchio M., Boucher B.A., Kreiger N. et al. Dietary phytoestrogen intake –lignans and isoflavones and breast cancer risk (Canada). Cancer Causes & Control. 2008; 19(3):259-272

Couture A., Levesque B., Dewailly E., et al. Lead exposure in Nunavik: from research to action. Int. J. Circumpolar Health, 2012;71:185-191.

Crichton V, Paquet P. Cadmium in Manitoba's wildlife. *Alces*. 2000;36:205-216.

Crinnion W.J. Toxic effects of the easily avoidable phthalates and parabens. Alternative medicine review: a journal of clinical therapeutic, 2010;15(3):190-196.

Dallaire R., Dewailly E., Ayotte P. et al. Growth in Inuit children exposed to polychlorinated biphenyls and lead during fetal development and childhood. Environmental Research, 2014;134:17-23.

Daniel J., Ziaee H., Pynsent P.B., and McMinn D.J.W. The validity of serum levels as a surrogate measure of systemic exposure to metal ions in hip replacement. Journal of Bone & Joint Surgery, British Volume, 2007;89(6):736-741.

Darnerud P., Eriksen G., Johannesson T. et al. Polybrominated diphenyl ethers (PBDEs): occurrence, dietary exposure and toxicology. Environ. Health Persp., 2001;109(suppl.)10:49-68.

Darnerud P.O. Toxic effects of brominated flame retardants in man and in wildlife. Environ. International, 2003;29:841-853.

Davies K. Concentrations and dietary intake of selected organochlorines, including PCBs, PCDDs and PCDFs in fresh food composites grown in Ontario, Canada. Chemosph., 1988;17: 263-276.

De Wit, C. An overview of brominated flame retardants in the environment. Chemosph., 2002;46:583-624.

Duffy C., Perez K., and Partridge A. Implications of phytoestrogen intake for breast cancer. CA- A Cancer Journal for Clinicians, 2007;57:260-277.

Dušková M., Hruskovicova H., Simunkova K., Parizek A. The effects of smoking on steroid metabolism and fetal programming. The Journal of steroid biochemistry and molecular biology, 2014;139:138-143.

Dellinger B., Taylor P.H., Tirey D.A. 1991. Minimization and Control of Harzardous Combustion By-products. Risk Reduction Laboratory, Cincinnati, U.S. Environmental Protection Agency (EPA/600152-90/039).

Department of Health and Human Services, Food and Drug Administration [Docket No.FDA-2012-N-0143] Harmful and potentially harmful constituents in tobacco products and tobacco smoke; established list. March 23, 2012; Accessed at: https://www.fda.gov/TobaccoProducts/Labeling/RulesRegulationsGuidance/ucm297786.htm

Ejaz S., and Lim C.W. Toxicological overview of cigarette smoking on angiogenesis. Environmental Toxicology and Pharmacology 2005;20:335–344

ENDS. Public Exposed to oestrogen risks from food cans. ENDS Report, 1995;246:p.3

Environment Canada. 1989. *Priority Substances Assessment Program (PSAP): First Priority Substances List (PSL1)*. Accessed at: http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=95D719C5-1

Environment Canada. Priority Substances List Report No. 1: Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans. 1990.

Environment Canada and Health Canada. 2000. Canadian Environmental Protection Act, priority Substances List Assessment Report: Nonylphenols and its Ethoxylates. 134 p.

Environment Canada. 2002. Canadian Environmental Quality Guidelines for nonylphenols and its ethoxylates (water, sediment and soil). Scientific supporting document. Ecosystem Health: Science based solutions Report No. 1-3. National Guidelines and Standards office, Environmental Quality Branch, Environment Canada, Ottawa.

Environment Canada. 2008. *PCB Regulations (SOR/2008-273) Canadian Environmental Protection Act, 1999*. Accessed at: http://laws-lois.justice.gc.ca/eng/regulations/SOR-2008-273/FullText.html

Environment Canada. 2008b. Polybrominated Diphenyl Ethers Regulations (SOR/2008-218) Canadian Environmental Protection Act, 1999**.** Accessed at: http://laws-lois.justice.gc.ca/eng/regulations/SOR-2008-218/FullText.html

Environment Canada. 2009. *Regulations Adding Perfluorooctane Sulfonate and Its Salts to the Virtual Elimination List (SOR/2009-15)* Accessed at: http://www.ec.gc.ca/lcpe-cepa/eng/regulations/detailReg.cfm?intReg=164

Environment Canada and Health Canada. 2010. Risk Management Strategy for Mercury. Accessed at : http://www.ec.gc.ca/doc/mercure-mercury/1241/index_e.htm

Environment Canada. 2013a. Archived- Part I: Canada's National Implementation Plan (NIP) under the Stockholm Convention on Persistent Organic Pollutants http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=3EEAC8B8-1&offset=1&toc=show (last updated 2013-04-19)

Environment Canada. 2013b. *CEPA Environmental Registry: Substances Lists: Toxic Substances List.* Accessed at: http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=0DA2924D-1

Environment Canada. 2013c. *Environmental Performance Agreement ("Agreement") Respecting Perfluorinated Carboxylic Acids (PFCAs) and their Precursors in Perfluorochemical Products Sold in Canada.* Minister of the Environment, Ottawa, ON. Accessed at: http://www.ec.gc.ca/epe-epa/default.asp?lang=En&n=81AE80CE-1

Environment Canada. 2013d. *Perfluorooctane Sulfonate and its Salts and Certain Other Compounds Regulations* (PFOS Regulations). Accessed at: http://www.ec.gc.ca/toxiques-toxics/default.asp?lang=En&n=4284EC2C-1

Environment Canada. 2013e. *CEPA Environmental Registry: Substances Lists: Toxic Substances List.* Accessed at: http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=0DA2924D-1

Environment Canada. 2013f. *Environmental Performance Agreement Respecting Bisphenol A in Paper Recycling Mill Effluents.* Minister of the Environment, Ottawa, ON. Accessed at: http://www.ec.gc.ca/epe-epa/default.asp?lang=En&n=EFFC880A-1

Environment Canada. 2013g. Archived- Part I: Canada's National Implementation Plan (NIP) under the Stockholm Convention on Persistent Organic Pollutants. Accessed at: http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=3EEAC8B8-1&offset=1&toc=show

Environment Canada. 2013h. Pollution and Waste - Mercury and the Environment. Accessed at: http://www.ec.gc.ca/mercure-mercury/

Ericson I., marti-Cid R., Nadal M., et al. Human exposure to perfluorinated chemicals through the diet: intake of perfluorinated compounds in foods from the Catalan (Spain) market. J. Agric. Food Chem., 2008;56:1787-1794.

Esteban M. and Castano A. Non-invasive matrices in human biomonitoring: a review.Environment International, 2009;35:438–449

Eskenazi B., Prehn AW., and Christianson R.E. Passive and active maternal smoking as measured by serum cotinine: the effect on birthweight. American Journal of Public Health, 1995;85(3):395-398

Eskenazi B., Marks A.R., Bradman A., et al. In utero exposure to dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethylene (DDE) and neurodevelopment among young Mexican American children. Pediatrics, 2006;118:233- 241.

Etzel R.A. A review of the use of saliva cotinine as a marker of tobacco smoke exposure. Prev. Med., 1990;19:190-197.

Euriquez de Salamanca R., Lopez-Miras A., Munoz J.J., et al. Is hexachlorobenzene human overload related to porphyria cutanea tarda? A speculative hypothesis. Med. Hypotheses, 1990;33:69-71.

Fei C., McLaughlin J.K., Tarone R.E., et al. Perfluorinated chemicals and fetal growth: a study within the Danish national birth cohort, Environ. Health Persp., 2007;115:1677-1682.

Fontaine J., Dewailly E., Beneditti J-L., et al. Re-evaluation of blood mercury, lead and cadmium concentrations in the Inuit populations of Nunavik (Quebec): a cross-sectional study. Environment Health, 2008;7(25).

Food Inspection Agency of Canada (FIAC). 2011. Antimony in Juice and Bottled Water. Food Safety Action Plan REPORT 2010-2011 Targeted Surveys Chemistry.

Foster W.G., Chan S., Platt L., and Hughes C.LF. Detection of phytoestrogens in samples of second trimester human amniotic fluid. Toxicology letters, 2002;129(3):199-205.

Franke A.A. and Custer L.J. Concentrations of daidzein and genistein in human milk after soy consumption. Clin. Chem., 1996;42:955-964.

Fries G.F. A review of the significance of animal food products as potential pathways of human exposures to dioxins. J. Anim. Sci., 1995;73:1639-1650.

Fromme H., Mosch C., Morovitz, M., et al. Pre-and postnatal exposure to perfluorinated compounds (PFCs). Environmental Science & Technology, 2010;44(18):7123-7129.

Furest P., Kruger C., Meemken H.A., et al. PCDD and PCDF levels in human milk: dependence on the period of lactation. Chemosphere, 1989;18:439-444.

Giampietro P.G., Bruno G., Furcolo G., et al. Soy protein formulas in children: no hormonal effects in long-term feeding. J. Pediatr. Endocrinol. Metab., 2004;17:191-196.

Giesy J.P. and Kannan K. Global distribution of perfluorooctane sulfonate in wildlife. Environ. Sci. Technol., 2001;35:1339-1342.

Glooschenko V, Downes C, Frank R, et al. Cadmium levels in Ontario moose and deer in relation to soil sensitivity to acid precipitation. Sci Total Envir 1988;71:173-86

Gocmen A., H.A. Peters, D.J. Cripps, et al. Hexachlorobenzene episode in Turkey. Biomed. Environ. Sci., 1989;2: 36-43.

Gomara B., Herrero L., Ramos J.J., et al. Distribution of polybrominated diphenyl ethers in human umbilical cord serum, paternal serum, maternal serum, placenta and breast milk from Madrid population, Spain. Environ. Sci. Technol., 2007;41:6961-6968.

Gosselin N.H., Brunet R.C., Carrier G., et al. Reconstruction of methylmercury intakes in indigenous populations from biomarker data, J. Expo. Sci. Environ. Epi., 2006;16:19-29.

Government of Canada. 2000. *Nuclear Safety and Control Act. Uranium Mines and Mills Regulations SOR/2000-206.*

Government of Canada. 1997. *Nuclear Safety and Control Act S.C. 1997, c. 9.* Accessed at: http://laws-lois.justice.gc.ca/PDF/N-28.3.pdf

Government of Canada. 2005. Pest Control Products Act: List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern (SI/2005-114). Accessed at: http://laws-lois.justice.gc.ca/eng/regulations/SI-2005-114/FullText.html

Government of Canada. 2010. Consumer Product Safety Act: Phthalates Regulations (SOR/2010-298). Accessed at: http://laws.justice.gc.ca/eng/regulations/SOR-2010-298/FullText.html

Government of Canada. Canada Gazette, Vol. 139, No. 11, June 1, 2005.

Government of Canada. 2014. Natural Resources Canada. Accessed at: http://www.nrcan.gc.ca/energy/uranium-nuclear/7695

Government of Canada. 2015. Pulp and Paper Mill Effluent Chlorinated Dioxins and Furans Regulations SOR/92-267 Canadian Environmental Protection Act, 1999. Accessed at: http://laws.justice.gc.ca/eng/regulations/SOR-92-267/FullText.html

Government of Manitoba, Department of Growth, Enterprise and Trade. Industrial Minerals: Commodity Summaries: Pollucite (cesium). Accessed online: https://www.gov.mb.ca/iem/geo/industrial/pollucite.html

Government of Saskatchewan. 2015. Mercury in Saskatchewan fish: guidelines for consumption. Updated to 2015. Accessed at: http://publications.gov.sk.ca/documents/66/76439-Mercury%20in%20SK%20Fish%20-%20Guidelines%20for%20Consumption%20-%202015.pdf

Government of Saskatchewan. 2017. Health benefits of eating fish and minimizing mercury concerns. May 2017. Accessed at: http://www.saskatchewan.ca/residents/environment-public-health-and-safety/food-safety#food-safety-program

.

Guenther K., Heinke V., Thiele B., et al. Endocrine disrupting nonylphenols are ubiquitous in food. Environ. Sci. Technol., 2002;36:1676-1680.

Gulkowska A., Jiang Q., So M.K., et al. Persistent perfluorinated acids in seafood collected from two cities of China. Environ. Sci. Technol., 2006;40:3736-3741.

Gundacker C., Pietschnig B., Wittmann K.J., et al. Lead and mercury in breast milk. Pediatrics, 2002;110:873-878

Hackshaw A., Rodeck C., and Boniface S. Maternal smoking in pregnancy and birth defects: a systematic review based on 173 687 malformed cases and 11.7 million controls. Human Reproduction Update. 2011;0:1-16.

Hagmar L., Bjork J., Sjodin A., et al. Plasma levels of persistent organohalogens and hormone levels in adult male humans. Arch. Environ. Health, 2001;56:138-143.

Hanrahan J.R., Tager I.B., Segal M.R., et al. The effect of maternal smoking during pregnancy on early infant lung function. Am. Rev. Resp. Dis., 1992;145:1129-1135.

Hansen K.J., Clemen L.A., Ellefson M.E., et al. Compound-specific, quantitative characterization of organic: fluorochemicals in biological matrices. Environ. Sci. Technol., 2001;35:766-770.

Harrad S. and Diamond M. New directions: exposure to polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs): current and future scenarios, Atmos. Environ., 2006;40:1187-1188.

Harrisoon M. and Hester R.E. Endocrine Disrupting Chemicals. The Royal Society of Chemistry. 1999, p. 14.

Hassan A.A., Sandanger T.M., and Brustad M. Selected vitamins and essential elements in meat from semi-domesticated reindeer (*Rangifer tarandus tarandus* L.) in mid- and northern Norwary: geographical variations and effect of animal population density. Nutrients, 2012;4:724-739.

Hauser R. Urinary phthalate metabolites and semen quality: a review of a potential biomarker of susceptibility. International Journal of Andrology, 2008;31(2):112-117

Hays S.M., Aylward L.L., Gagne M., et al. Biomonitoring Equivalents for inorganic arsenic. Regulatory Toxicology and Pharmacology 2010;5:1–9

Health Canada. 1986. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document - Cadmium. Accessed Feb 2019 from: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-cadmium.html

Health Canada. 1986b. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document - Silver. Accessed Feb 2019 at https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-silver.html

Health Canada. 1990. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Barium. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-barium.html

Health Canada. 1991. Guideline for Canadian Drinking Water Quality: Guideline Technical Document – Boron.. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-boron.html

Health Canada. 1992. Guidelines for Canadian Drinking Water Quality: Guideline technical document - Copper. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-copper.html

Health Canada. 1995. Health and environment: a handbook for health professionals. Health Canada, Ottawa

Health Canada. 1998. Guideline for Canadian Drinking Water Quality: Guideline Technical Document – Aluminum. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-aluminum.html

Health Canada. 1999. Methylmercury in Canada: Exposure of First Nations and Inuit to methylmercury in the Canadian Environment. 1999. Medical Services Branch, Health Canada, Ottawa.

Health Canada. 2004. Screening assessment report – health: polybrominated diphenyl ethers (PBDEs) [tetra-, penta-, hexa-, hepta-, octa-, nona- and deca-congeners]. Draft Health Screening Assessment. Ottawa, ON, February 25, 2004.

Health Canada. 2004b. Mercury – Your Health and the Environment. A Resource Tool. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications/environmental-contaminants/mercury-your-health-environment-resource-tool.html

Health Canada. 2005. It's Your Health: PCBS. Health Canada's Management of Toxic Substances Division. Accessed Feb 2019 at: https://www.canada.ca/en/health-canada/services/healthy-living/your-health/environment/pcbs.html

Health Canada. 2005b. Reference Values for Elements – Dietary Refences Intakes Tables. Accessed at: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/dri_tables-eng.pdf

Health Canada. 2005c. Summary Safety Review – strontium. Drugs and Health Products. Accessed online: http://hc-sc.gc.ca/dhp-mps/medeff/reviews-examens/strontium-eng.php

Health Canada. 2006. State of the Science Report for a Screening Health Assessment: Polybrominated Diphenyl Ethers (PBDEs) [Tetra-, Penta-, Hexa-, Hepta-, Octa-, Nona-, and Deca- Congeners]. Her Majesty the Queen in Right of Canada, represented by the Minister of Health Canada. ISBNL 0-662-43970-8. Accessed at: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/contaminants/pbde/pbde-eng.pdf

Health Canada. 2007. Bureau of Chemical Safety, Food Directorate, Health Products and Food Branch. Human health risk assessment of mercury in fish and health benefits of fish consumption. March 2007. https://www.canada.ca/en/health-canada/services/food-nutrition/reports-publications/human-health-risk-assessment-mercury-fish-health-benefits-fish-consumption.html

Health Canada. 2007b. Natural Health Products Directorate. Boron as a Medicinal Ingredient in Oral Natural Health Products. Accessed at: https://www.canada.ca/en/health-canada/services/drugs-health-products/reports-publications/natural-health-products/boron-medicinal-ingredient-oral-natural-health-products-natural-health-products-directorate-heath-canada-2007.html

Health Canada. 2008. Food and Nutrition: Archived –Health Canada Review of Dietary Exposure to Aluminum. Accessed at: http://www.hc-sc.gc.ca/fn-an/securit/addit/aluminum-eng.php

Health Canada (2009a) Technical report to summarize the scientific rationale for the Natural Health Products Directorate's new guidance on the regulation of soy isoflavone products. Natural Health Products Directorate (NHPD). Accessed at: https://www.canada.ca/en/health-canada/services/drugs-health-products/natural-non-prescription/legislation-guidelines/guidance-documents/technical-report-summarize-scientific-rationale-regulation-isoflavone.html

Health Canada. 2009b. Prenatal Nutrition Guidelines for Health Professionals – Iron. Accessed at: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/pubs/iron-fer-eng.pdf

Health Canada. May 2009c. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document - Radiological Parameters. Ottawa, ON. Accesed at: https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-radiological-parameters.html

Health Canada. 2009d. Food and Nutrition: Questions and answers on perfluorinated chemicals in food. Accessed at: http://www.hc-sc.gc.ca/fn-an/securit/chem-chim/environ/pcf-cpa/qr-pcf-qa-eng.php

Health Canada, 2009e. Prenatal Nutrition Guideliens for Health Professionals: Fish and Omega-3 Fatty Acids.. Accessed online: https://www.canada.ca/en/health-canada/services/publications/food-nutrition/prenatal-nutrition-guidelines-health-professionals-fish-omega-3-fatty-acids-2009.html

Health Canada, 2009f. Archived: Health Canada warns Canadians of cardiac risks associated with cesium chloride. http://www.healthycanadians.gc.ca/recall-alert-rappel-avis/hc-sc/2009/13329a-eng.php

Health Canada. 2010a. Report on human biomonitoring of environmental chemicals in Canada: Results of the Canadian Health Measures Survey Cycle 1 (2007–2009). Minister of Health, Ottawa, ON., Accessed at: https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications/environmental-contaminants/report-human-biomonitoring-environmental-chemicals-canada-health-canada-2010.html

Health Canada. 2010b. Reference Values for Elements – Dietary References Intakes Tables. Accessed at: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/dri_tables-eng.pdf

Health Canada. 2011. Health Concerns: Tobacco Legislation. Accessed at: http://www.hc-sc.gc.ca/hc-ps/tobac-tabac/legislation/reg/index-eng.php[last updated: 2011-11-30]

Health Canada. 2012. Dioxins and furans. Its's your health. 2012. Accessed at: https://www.canada.ca/en/health-canada/services/healthy-living/your-health/environment/dioxins-furans.html

Health Canada. 2013. Second Report on Human Biomonitoring of Environmental Chemicals in Canada. Results of the Canadian Health Measures Survey Cycle 2 (2009-2011). (CHMS Report) Accessed at: https://www.canada.ca/content/dam/hc-sc/migration/hc-sc/ewh-semt/alt_formats/pdf/pubs/contaminants/chms-ecms-cycle2/chms-ecms-cycle2-eng.pdf

Health Canada. 2014. *Consumer Product Safety: Cosmetic Ingredient Hotlist.* Accessed at: http://www.hc-sc.gc.ca/cps-spc/cosmet-person/hot-list-critique/hotlist-liste-eng.php#

Health Canada. 2014b. Consumer Product Safety: Safety of Cosmetic Ingredients. Accessed at: http://www.hc-sc.gc.ca/cps-spc/cosmet-person/labelling-etiquetage/ingredients-eng.php

Health Canada. 2016. Canadian Total Dietary Study. Accessed at: https://www.canada.ca/en/health-canada/services/food-nutrition/food-nutrition-surveillance/canadian-total-diet-study/concentration-contaminants-other-chemicals-food-composites.html#a10

Health Canada. 2017. Guidelines for Canadian Drinking Water Quality—Summary Table. Water and Air Quality Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.. Accessed at: https://www.canada.ca/content/dam/hc-sc/migration/hc-sc/ewh-semt/alt_formats/pdf/pubs/water-eau/sum_guide-res_recom/sum_guide-res_recom-eng.pdf

Hecht S.S. Progress and challenges in selected areas of tobacco carcinogenesis, Chem. Res. Toxicol. 2008;21:160-171.

Heffernan, A. L., Aylward, L. L., Toms, L. M. L., et al. Pooled biological specimens for human biomonitoring of environmental chemicals: opportunities and limitations. Journal of Exposure Science and Environmental Epidemiology, 2014;24(3):225-232.

Higgins JP, Tuttle TD, Higgins CL. Energy beverages: content and safety. Mayo Clin Proc. 2010;85(11):1033-1041.

Hooper K. and McDonald T.A. The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs. Environ. Health Persp., 2000;108:387-392.

Horn-Ross P.L., Barnes S., Lee M., et al. Assessing phytoestrogen exposure in epidemiologic studies: development of a database (United States). Canc. Causes Contr., 2000;11:289-298.

Inoue K., Kawaguchi M., Yamanaka R., Higuchi T., et al. Evaluation and analysis of exposure levels of di(2-ethylhexyl) phthalate from blood bags. Clinica. Chimica. Acta., 2005;358(1): 159-166.

Institute of Medicine (IOM).1997. Food and Nutrition Board. Dietary Reference Intakes: Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride. Washington, DC: National Academy Press, 1997. Accessed at: http://www.nap.edu/openbook.php?record_id=5776&page=190

Institute of Medicine (IOM). 2000. Food and Nutrition Board. Dietary Reference Intakes: Vitamin C, Vitamin E, Selenium and Carotenoids. Washington, DC: National Academy Press, 2000. Accessed at: http://books.nap.edu/openbook.php?record_id=9810

Institute of Medicine (IOM). 2001. Food and Nutrition Board. Dietary Reference Intakes: Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum,

Nickel, Silicon, Vanadium, and Zinc. Washington, DC: National Academy Press, 2001. Accessed at: http://www.nap.edu/openbook.php?isbn=0309072794 Accessed March 25, 2019.

Institut national de santé publique du Québec (INSPQ). 2008. Toxicologie Clinique, Guide des métaux. Accessed at: http://portails.inspq.qc.ca/toxicologieclinique/cadmium.aspx. Accessed March 25, 2019.

Inoue K., Okada F., Ito R., et al. Perfluorooctane sulfonate (PFOS) and related perfluorinated compounds in human maternal and cord blood samples: assessment of PFOS exposure in a susceptible population during pregnancy. Environ. Health Perspect., 2004;112:1204-1207.

International Agency for Research on Cancer. Accessed at: http://monographs.iarc.fr/ENG/Classification/ (accessed online April 16, 2015).

International Agency for Research on Cancer. Accessed at: http://monographs.iarc.fr/ENG/Classification/ (accessed online April 16, 2015).

International Agency for Research on Cancer. Accessed at: http://monographs.iarc.fr/ENG/Classification/ (accessed online April 16, 2015).

IPCS (International Program on Chemical Safety). 1994. Environment Health Criteria No. 162. Brominated diphenyl ethers. WHO. Geneva; 1994a.

Irvine J., Quinn B., and Stockdale D. Northern Saskatchewan Health Indicators Reports 2011. Athabasca Health Authority, Keewatin Yatthe and Mamawetan Churchill River Regional Health Authorities. Population Health Unit, La Ronge. https://populationhealthunit.ca/mrws/filedriver/Northern_Saskatchewan_Health_Indicators_Report_2011.pdf

Ivorra C., Garcia-Vicent C., Ponce F., et al. High cotinine levels are persistent during the first days of life in newborn second hand smokers. Drug and alcohol dependence. 2014;134:275-279.

Jacobson J.L. and Jacobson S.W. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N. Engl. J. Med., 1996;335: 783-789.

Jacobson J.L., and Jacobson S.W. Postnatal exposure to PCBs and childhood development. The Lancet 2001;358(9293): 1568-1569.

Jantzen C., Jorgensen H.L., Duus B.R., et al. Chromium and cobalt ion concentrations in blood and serum following various types of metal-on-metal hip arthroplasties. Acta Orthopaedica, 2013;84(3):229-236

Jarrell J., Chan S., Hauser R., et al. Longitudinal assessment of PCBs and chlorinated pesticides in pregnant women from western Canada. Environmental Health: A Global Access Science Source. June 2005.

Jensen A.A. and Slovach S. Chemical Contaminants in human milk, Boca Raton, Florida: CRC; 1991.

Jin A, Joseph-Quinn KM. Consumption guideline for cadmium in moose meat in northern British Columbia, Canada. Int J Health 2004;63(Supp2):169-73

Jonkers N., Knepper T.P., de Voogt P. Aerobic biodegradation studies of nonylphenol ethoxylates in river water using liquid chromatography-electrospray tandem mass spectrometry. Environ. Sci. Technol., 2001;35:335-340.

Jorissen J. Outcomes associated with postnatal exposure to polychlorinated biphenyls (PCBs) via breast milk, Adv. Neonatal care, 2007;7:230-237.

Kang J.H., Kondo F. and Katayama Y. Human exposure to bisphenol A, Toxicol., 2006;226:79-89.

Kang J., and Price W.E. Occurrence of phytoestrogens in municipal wastewater and surface waters. Journal of Environmental Monitoring 2009;11(8): 1477-1483.

Kannan K., Corsolini S., Falandysz J., et al. Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. Environ. Sci. Technol., 2004;38:4489-4495.

Kato K., Silva M. J., Reidy J. A., et al. Mono (2-ethyl-5-hydroxyhexyl) phthalate and mono-(2-ethyl-5-oxohexyl) phthalate as biomarkers for human exposure assessment to di-(2-ethylhexyl) phthalate. Environmental Health Perspectives, 2004;112(3): 327.

Kawagoshi Y., Fujita Y., Ikuko K., et al. Estrogenic chemicals and estrogenic activity in leachate from municipal waste landfill determined by yeast two-hybrid assay, J. Environ. Monit., 2003; 5:269-274.

Kershaw T.G., Dhahir P.H. and Clarkson T.W. The relationship between blood levels and dose of methylmercury in man, Arch. Environ Health, 1980;35:28-36.

Kessler, W., Numtip W., Grote K., et al. Blood burden of di(2-ethylhexyl) phthalate and its primary metabolite mono(2-ethylhexyl) phthalate in pregnant and nonpregnant rats and marmosets. Toxicol. Appl. Pharmacol. 2004;195(2):142-153.

Kissa E. Fluorinated Surfactants and Repellents, 2nd ed., Marcel Dekker, Inc., New York, 2001.

Klopov V.P. Persistent organic compounds in women residing in the Russian Arctic. Int. J. Circumpolar Health. 1998;34:135-138.

Knight D.C., Eden J.A., Huang J.L., et al. Isoflavone content of infant foods and formulas. J. Pediatr. Child Health, 1998;34:135-138.

Kosatsky T., Przybysz R., Shatenstein B.,Weber J.P., Armstrong B., Fish Consumption and Contaminant Exposure among Montreal-Area Sportfishers: Pilot Study, Environmental Research, 1999;80(2):S150-S158.

Kozlowski L.T., Mehta N.Y., Sweeney C.T., Schwartz S.S., Vogler G.P., Jarvis M.J., West R.J. Filter ventilation and nicotine content of tobacco in cigarettes from Canada, the United Kingdom, and the United States. Tobacco control 1998;7(4):369-375

Krishnan A.V., Stathis P., Permuth S.F., et al. Bisphenol A: an estrogenic substance is released from polycarbonate flasks during autoclaving. Endocrin., 1993; 132:2279- 2286

Krishnan K., Adamou T., Aylward L.L., et al. Biomonitoring Equivalents for 2,20,4,40,5-pentabromodiphenylether (PBDE-99). Regulatory Toxicology and Pharmacology, 2011;60:165-171

Kubwabo C., Stewart B., Zhu J., et al. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. J. Environ. Monit., 2005;7:1074-1078.

Kuroda N., Kinoshita Y., Sun Y., et al. Measurement of bisphenol A levels in human blood serum and ascetic fluid by HPLC using a fluorescent labelling reagent, J. Pharmaceu. Biomed. Anal., 2003;30:1743-1749.

Kurttio P, Harmionen A, Saha H, Salonen L, Karpas Z, Komulainen H, Auvinen A. Kidney toxicity of ingested uranium from drinking water. Am J Kidney Dis 2006;47(6):972-982.

Kwack S.J., Kwon O., Kim H.S., et al. Comparative evaluation of alkylphenolic compounds on estrogen activity in vitro and in vivo. J. Toxicol. Environ. Health A, 2002;65:419-431.

La Guardia M.J., Hale R.C. and Harvey E. Evidence of debromination of decabromodiphenyl ether (BDE-209) in biota from a wastewater receiving stream. Environ. Sci. Technol., 2007;41:6663-6670.

Larter NC, Macdonald CR, Elkin BT et al. Cadmium and other elements in tissues from four ungulate species from the Mackenzie Mountain region of the Northwest Territories, Canada. Ecotox Environ Safety 2016;132:9-17.

Lau C., Butenhoff J.L. and Rogers J.M. The developmental toxicity of perfluoroalkyl acids and their derivatives. Toxicol. Appl. Pharmacol., 2004;198:231-241

Lauby-Secretan B, Loomis D, Grosse Y, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, Guha N, Baan R, Mattock H, Straif K. Carcinogenicity of the polychlorinated biphenyls and polybrominated biphenyls. The Lancet Oncology, 2013;14(4):287-288

Le H.H., Carlson E.M., Chua J.P., et al. Bisphenol A is released from polycarbonate drinking bottles and mimics the neurotoxic actions of estrogen in developing cerebellar neurons, Toxicol. Letters, 2008;176:149-156.

LeBlanc G.A., Mu X. and Rider C.V. Embryotoxicity of the alkylphenol degradation product 4 nonylphenol to the crustacean Daphnia magna. Environ. Health Perspect., 2000;108:1133-1138.

Lee N. Phytoestrogens as bioactive ingredients in functional foods: Canadian regulatory update. J. AOAC Int., 2006;89:1135-1137.

Leyssens L, Vinck B, Van Der Straeten C, Wuyts F, Maes L. Cobalt toxicity in humans. A review of the potential sources and systemic health effects. Toxicology 2017; S0300-483X(17)30155-5. doi: 10.1016/j.tox.2017.05.015. [Epub ahead of print].

Lewis M.A. Chronic and sublethal toxicities of surfactants to aquatic organisms: a review and risk assessment. Water Res., 1991; 25:101-113

Li L.X., Chen L., Meng X.Z., Chen B.H., Chen S.Q., Zhao Y., Zhao L.F., Liang Y., Zhang Y.H. Exposure levels of environmental endocrine disruptors in mother-newborn pairs in China and their placental transfer characteristics. PloS one (2103) 8.5: e62526.

Liem A.K., Furst P. and Rappe C. Exposure of populations to dioxins and related compounds. Food Addit. Contam., 2000;17:241-259.

Lippmann M. Environmental Toxicants: Human Exposures and Their Health Effects. 1992. Van Nostrand Reinhold, NY.

Liu Z.H., Kanjo Y., and Mizuanti S. A review of phytoestrogens: their occurrence and fate in the environment. Water Research, 2010;567-577

Lonky E., Reihman J., Darvill T., et al. Neonatal behavioral assessment scale performance in humans influenced by maternal consumption of environmentally contaminated Lake Ontario fish. J. Great Lakes Res., 1996;22:198-212.

Lopez-Espinosa M.-J., Granada A., Carreno J., et al. Organochlorine pesticides in placentas from Southern Spain and some related factors, Placenta, 2007;28:631-638.

Luang-on W., Siriarayaporn P., Rochanachirapha P., et al. Situation analysis of occupational lead poisoning in Thailand 1992-2001. J. Health Sci., 2003;12:279-284.

Mahaffey K.R. Fish and shellfish as dietary sources of methylmercury and the omega-3 fatty acids, eicosahexaenoic acid and docosahexaenoic acid: risks and benefits. Environ. Res., 2004;95:414-428.

Mahaffey K.R. Methylmercury: A new look at the risks. Public Health Repts 1999;114:397-413.

Mahaffey K.R., Clickner R.P. and Bodurow C.C. Blood organic mercury and dietary mercury intake: National Health and Nutrition examination Survey, 1999 and 2000.

Mahaffey K.R. Mercury exposure: medical and public health issues, Trans. Am. Clin. Clim. Assoc., 2005;116:127-154.

Maragou N.C., Makri A., Eugenia L., et al. Migration of bisphenol A from polycarbonate baby bottles under real use conditions. Food Addit.Contam., 2008;25:373- 383

Martin J.W., Whittle D.M., Muir D.C.G., et al. Identification of long chain perfluorinated acids in biota from the Canadian Arctic. Environ. Sci. Technol., 2004a;38:373-380.

Mayo Clinic Laboratories. Test ID: Serum magnesium. Accessed online: https://www.mayomedicallaboratories.com/test-catalog/Clinical+and+Interpretive/8448.

McDonald T.A. A perspective on the potential health risks of PBDEs. Chemosph., 2002,46:745-755.

Messina M. and Barnes S.J. The role of soy products in reducing risk of cancer. J. Natl. Cancer Inst., 1991;83:541-546.

Monteiro-Riviere N.A., Van Miller J.P., Simon G., et al. Comparative in vitro percutaneous absorption of nonylphenol and nonylphenol ethoxylates through human, porcine and rat skin. Toxicol. Ind. Health, 2000;16:49-57.

Morrow H. 2000. Cadmium and cadmium alloys. Kirk-Othmer Encyclopedia of Chemical Technology. John Wiley & Sons, Inc, Mississauga, ON

Mueller J.F., Harden F., Toms L.M., et al. Persistent organochlorine pesticides in human milk samples from Australia, Chemosphere, 2008;70:712-720.

National Research Council (NRC): Measuring lead exposure in infants, children, and other sensitive populations. Washington, DC: National Academy Press: 1993.

National Research Council. Toxicologic effects of methylmercury. Washington (DC): National Academy of Sciences; 2000.

Newbold R.R., Jefferson W.N. and Pandilla-Banks E. Long-term adverse effects of neonatal exposure to bisphenol A on the murine female reproductive tract, Reprod. Toxicol., 2007;24:253-258.

National Toxicology Program. 2011. Report on Carcinogens, Twelfth Edition: Lindane, Hexachlorocyclohexane (Technical Grade), and Other Hexachlorocyclohexane Isomers.

National Toxicology Program, Department of Health and Human Services. Accessed at: http://ntp.niehs.nih.gov/ntp/roc/twelfth/profiles/lindane.pdf

National Institute for Environmental Health Sciences (NIEHS). 2001. Toxicological Summary for Selected Polybrominated Diphenyl Ethers. Submitted by Bonnie Carson, Integrated Laboratory Systems, Research Triangle Park, North Carolina, March, 2001.

Needham L.L., Grandjean P., Heinzow B., et al. Partition of environmental chemicals between maternal and fetal blood and tissues. Environmental Science and Technology, 2011;45:1121–1126

Noakes P.S., Taylor P., Wilkinson S., et al. The relationship between persistent organic pollutants in maternal and neonatal tissues and immune responses to allergens: a novel exploratory study. Chemosph., 2006;63:1304-1311.

Okada H., Tokunaga T., Liu X. et al., Direct evidence revealing structural elements essential for the high-binding ability of bisphenol A to human estrogen-related receptor gamma, Environ. Health Persp. 2008; 116:32-38.

Olsen G.W., Burris J.M., Ehresman D.J, et al. Half-life of serum elimination of perfluorooctane sulfonate, perfluorohexanesulfonate and perfluorooctane (PFOA) in retired flouorochemical production workers. Environ. Health Perspect., 2007;115:1298- 1305.

Ong C.N., Chia S.E., Foo S.C., et al. Concentrations of heavy metals in maternal and umbilical cord blood. Biometals, 1993;6:61-66.

Orloff K.G., Mistry K., Charp P., Metcalf S., Marino R., Shelly T., et al. Human exposure to uranium in groundwater. Environ Res 2004;94:319-326.

Parzfall, W. Risk assessment of dioxin contamination in human food. Food Chem. Toxicol., 2002;40:1185-1189.

Persistent Organic Pollutants Review Committee (POPRC). 2007. Draft Risk Profile for Beta-Hexachlorocyclohexane. Stockholm Convention of Persistent Organic Pollutants.

Peters RJB. Man-made chemicals in human blood, TNO Report R2005/129, March 2005.

Polybrominated Diphenyl Ethers Regulations, in Canada Gazette. December, 2006.

Polizzi S., Pira E., Ferrara M., et al. Neurotoxic effects of aluminium among foundry workers and Alzheimer's disease. Neuro Toxicol., 2002;23:761-774.

Population Health Unit. Moose: a safe choice in traditional food. Special supplement of Opportunity North, Spring 2005

Ratelle M, Li X, Liard BD. Cadmium exposure in First Nations communities of the Northwest Territories, Canada: smoking is a greator contributer than consumption of cadmium-accumulating organ meats. Environ Sci Process Impacts 2018;20(10):1441-1453.

Rehm J, Baliunas D, Brochu S, Fischer B, Gnam, W, Patra J, Popova S, Sarnocinska-Hart A, & Taylor B. 2006. *The Costs of Substance Abuse in Canada 2002*. Ottawa: Canadian Centre on Substance Abuse.

Reid J.L., Hammond D., Rynard V.L., Madill C.L., Burkhalter R. *Tobacco Use in Canada: Patterns and Trends, 2017 Edition*. Waterloo, ON: Propel Centre for Population Health Impact, University of Waterloo.

Reis M.F., Sampaio C., Brantes A., et al. Human exposure to heavy metals in the vicinity of Portugese solid waste incinerators – Part 3: biomonitoring of Pb in blood of children under the age of 6 years. Int. J. Hyg. Environ.-Health, 2007;210:455-459.

Renner R. European bans on surfactant trigger translantic debate. Environ. Sci. Technol., 1997;31:316A-320A.

Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: Office of Health and Environmental Assessment, Office of Research and Development, US Environmental Protection Agency.

Richard H.J., Fox N.L., and Sexton M. Dose-response of birth weight to various measures of maternal smoking during pregnancy. Journal of clinical epidemiology. 1988;41(5):483-489.

Riihimäki V., and Aitio A. Occupational exposure to aluminum and its biomonitoring in perspective. Critical reviews in toxicology. 2012;42(10): 827-853

Rodrigues, J. L., Batista, B. L., Nunes, J. A. et al. Evaluation of the use of human hair for biomonitoring the deficiency of essential and exposure to toxic elements. Science of the Total Environment, 2008;405(1):370-376.

Ryan, Jake. 2004. Polybrominated Diphenyl Ethers (PBDEs) in Human Milk: Occurrence Worldwide. Health Canada, Health Products and Food Branch, Ottawa, Ontario, Canada.

Saskatchewan Ministry of Health, Covered Population 2010.

Saskatchewan Ministry of Education Early Childhood Development and Integrated Services, 2011

Seacat A.M., Thomford P.J., Hansen K.J., et al. Subchronic dietary toxicity of potassium perfluorooctane sulfonate in rats. Toxicol., 2003;183:117-131.

Schecter A., Papke O. and Ball M., Evidence of transplacental transfer of dioxins from mother to fetus: chlorinated dioxin and dibenzofuran levels in the liver of stillborn infants. Chemosphere, 1990;21:1017-1022.

Schecter A., Papke O., Tung K.C., et al. Polybrominated diphenyl ether flame retardants in the U.S. population: current levels, temporal trends and comparison with dioxins, dibenzofurans and polychlorinated biphenyls, JOEM, 2005;47:199-211.

Schecter A., Papke O., Harris T.R., et al. Polybrominated diphenyl ether (PBDE) levels in an expanded market basket survey in U.S. food and estimated PBDE dietary intake by age and sex. Environ. Health Persp., 2006;114:1515-1520.

Scherer G., Jarczyk L., Heller W.D., et al. Pharmacokinetics of nicotine, cotinine and 3-hydroxycotinine in cigarette smokers. Klin. Wochenschr. 1988;66(Suppl. XI):5-11.

Schonfelder G., Wittfoht W., Hopp H., et al. Parent bisphenol A accumulation in the human maternal-fetal-placental unit. Environ. Health Persp., 2002;110:A703-707.

Schutz A., Bergdahl I.A, Ekholm A., et al. ICP-MS determination of lead in plasma and whole blood in lead workers and referents. Occup. Environ. Med., 1996;53:736-740.

Schmitt E,. Genotoxic activity of four metabolites of the soy isoflavone daidzein. Mutation Research/Genetic Toxicology and Environmental Mutagenesis, 2003;542(1):43-48.

Scott M.J. and Jones M.N. The biodegradation of surfactants in the environment. Biochim. Biophys. Acta, 2000;1508:235-251.

Sjödin A. 2000. Occupational and dietary exposure to organohalogen substances with special emphasis on polybrominated diphenyl ethers, ISBN 91-7265-052-4. PhD Thesis, Stockholm University, Stockholm, Sweden.

Sjogren B. and Elinder C.G. Aluminum and its compounds. In: Zenz C., Dickerson O.B. and Hovarth E.P., editors. Occupational Medicine, Vol. III. St. Louis: Mosby; 1994:458-465.

Sjogren B., Lidums V., Hakansson M., et al. Exposure and urinary excretion of aluminum during welding. Scand. J. Work Environ. Health, 1985;11:39-43.

Sniegoski, L.T., and Moody, J.R. Determination of serum and blood densities. Analytical chemistry, 1979; 51(9):1577-1578.

Sprague B.L., Trentham-Dietz A., Hedman C.J., et al. Circulating serum xenoestrogens and mammographic breast density. Health, 2013;4: 6.

Sørensen M., Bisgaard H., Stage M, and Loft S. Biomarkers of exposure to environmental tobacco smoke in infants. Biomarkers, 2007;12(1): 38-46.

Soto A.M., Justicia H., Wray J.W., et al. p-Nonylphenol: an estrogenic xenobiotic released from modified polystyrene. Environ. Health Perspect., 1991;92:167-173.

Statistics Canada. 2011. National Household Survey. Accessed at: https://www12.statcan.gc.ca/nhs-enm/index-eng.cfm

Steenland K., Piacitello L., Deddens J., et al. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetracholorodibenzo-p-dioxin. J. Natl. Cancer Inst., 1999;91:779-786

Sugiura-Ogasawara M., Ozaki Y., Sonta SI., et al. Exposure to bisphenol A is associated with recurrent miscarriage. Hum. Reprod., 2005;20:2325-2329.

Sun Y., Irie M., Kishikawa N., et al. Determination of bisphenol A in human breast milk by HPLC with column-switching and fluorescence detection. Biomed. Chromatogr., 2004;18:501-507

Sunyer J., Torrent M., Munoz-Ortiz L., et al. Prenatal dichlorodiphenyldichloroethylene (DDE) and asthma in children. Environ. Health Persp., 2005;113:1787-1790.

The Swedish National Chemicals Inspectorate, 1999. Phase-out of PBDEs and PBBs: Report on a Governmental Commission. Report No. 2/99. Solna, Sweden:Keml [The Swedish National Chemicals Inspectorate], 1999.

Sweeney T., Nicol L., Roche J.F., et al. Maternal exposure to octylphenol suppresses ovine fetal follicle stimulating hormone secretion, testis size and sertoli number. Endocrinol., 2000;141:2667-2673.

Takeuchi T., Tsutsumi O., Ikezuki Y., et al. Positive relationship between androgen and the endocrine disruptor, bisphenol A, in normal women and women with ovarian dysfunction. Endocr. J., 2004;51:165-169.

Tawichasri C., Patumanond J. and Winichakoon C. Change of health behaviour and blood lead levels in lead-exposed workers. J. Health Sci., 2000;9:558-565.

Tham D.M., Gardner C.D. and Haskell W.L. Potential health benefits of dietary phytoestrogens: a review of the clinical, epidemiological, and mechanistic evidence. J. Clin. Endocrinol. Metab., 1998;83:2223-2235.

Thanapop C., Geater A.F., Robson M.G., et al. Exposure to lead of boatyard workers in southern Thailand. J. Occup. Health, 2007;49:345-352.

The Swedish National Chemicals Inspectorate. 1999. Phase-out of PBDEs and PBBs: Report on a Governmental Commission. Report No. 2/99. Solna, Sweden:Keml [The Swedish National Chemicals Inspectorate], 1999.

Thomas P, Irvine J, Lyster J, Beaulieu R. Radionuclides and trace metals in Canadian moose near uranium mines: comparison of radiation doses and food chain transfer with cattle and caribou. Health Physics 2005, 88(5):423-438.

Thompson L.U., Boucher B.A., Liu Z., et al. Phytoestrogen content of foods consumed in Canada, including isoflavones, lignans and coumestan. Nutr. Cancer, 2006;54:184-201

Tittlemier S.A., Pepper K., Edwards L., et al. Concentrations of perfluorooctanesulfonamides in Canadian total diet study composite food samples collected between 1992 and 2004. J. Agric. Food Chem., 2006;54:8385-8389

Tittlemier S.A., Pepper K., Seymour C., et al. Dietary exposure of Canadian to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. J. Agric. Food Chem., 2007;55:3203-3210.

Toxicology Environmental Consulting Ltd. 2002. Alternatives to nonylphenol ethoxylates: review of toxicity, biodegradation and technical-economic aspects. Prepared for Environment Canada.

Tsubaki T., Irukyama K., (Eds) Minamata Disease. Amsterdam: Elsevier; 1977.

Tsuchiya H., Mitani K., Kodama K., et al. Placental transfer of heavy metals in normal pregnant Japanese women. Arch. Environ. Health, 1984;39:11-17.

Tsuji L.J.S., Wainman B.C., Martin I.D., et al. Elevated blood-lead levels in First Nation people of Northern Ontario Canada: policy implications. Bull. Environ. Contam. Toxicol., 2008;80:14-18.

Tsuji L.J.S., Wainman B.C., Martin I.D., et al. Lead shot contribution to blood lead of First Nations people: The use of lead isotopes to identify the source. Sci. Tot. Environ., 2008;80:180-185.

Tuomi T. Johnsson T. and Reijula K. Analysis of nicotine, 3-hydroxycotinine, cotinine, and caffeine in urine of passive smokers by HPLC-tandem mass spectrometry. Clin. Chem. 1999;45:2164-2172.

Ulbrich B., and Stahlman R. Developmental toxicity of polychlorinated biphenyls (PCBs): a systematic review of experimental data. Arch Toxicol, 2004;78:252-268

U.S. National Toxicology Program, Center for the Evaluation of Risks to Human Reproduction. 2007. NTP-CERHR Reports and Monographs. Available at: http://cerhr.niehs.nih.gov/reports/index.html.

U.S. Environmental Protection Agency. 1992. Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: Office of Health and Environmental Assessment, Office of Research and Development, US Environmental Protection Agency.

U.S. Environmental Protection Agency. 1994. Method 1613 Tetra- through Octa-Chlorinated Dioxins and Furans by Isotope Dilution HRGC/HRMS. U.S. Environmental Protection Agency, Office of Water Engineering and Analysis Division (4303). 401 M Street S.W. Washington, D.C. 20460

U.S. Protection Agency. 2000. Guidance for data quality assessment. Practical methods for data analysis. Office of Environmental Information, EPA QA/G-9, QA00 Version Washington, DC. Available at: https://www.epa.gov/sites/production/files/2015-06/documents/g9-final.pdf

U.S. Environmental Protection Agency. 2007. Method 8081B Organochlorine Pesticides by Gas Chromatography (Revision 2). February 2007. Environmental Protection Agency. Office of Water, Office of Science and Technology Engineering and Analysis Division (4303T). 1200 Pennsylvania Avenue, NW Washington, DC 20460 EPA-820-R-10-005

U.S. Environmental Protection Agency (EPA). 2007b. Chemical Summary: Phthalates. Accessed at: http://www.epa.gov/teach/chem_summ/phthalates_summary.pdf

U.S. Environmental Protection Agency. 2010. Method 1668C Chlorinated Biphenyl Congeners in Water, Soil, Sediment, Biosolids, and Tissue by HRGC/HRMS. April 2010 U.S. Environmental Protection Agency. Office of Water, Office of Science and Technology Engineering and Analysis Division (4303T). 1200 Pennsylvania Avenue, NW Washington, DC 20460 EPA-820-R-10-005

U.S. Environmental Protection Agency (EPA). 2010b. Nonylphenol (NP) and Nonylphenol Ethoxylates (NPEs) Action Plan [RIN 2070-ZA09]. Accessed at: http://www.epa.gov/oppt/existingchemicals/pubs/actionplans/RIN2070-ZA09_NP-NPEs%20Action%20Plan_Final_2010-08-09.pdf

U.S. Environmental Protection Agency. 2014. Basic Information about Polychlorinated Biphenyls (PCBs) in Drinking Water. Accessed at: http://water.epa.gov/drink/contaminants/basicinformation/polychlorinated-biphenyls.cfm

U.S. Food and Drug Administration. 2018. Parabens in Cosmetics. Accessed at: https://www.fda.gov/Cosmetics/ProductsIngredients/Ingredients/ucm128042.htm#What_are_parabens.

Vahter M., Mottet N.K., Friberg L., et al. Speciation of mercury in the primate blood and brain following long-term exposure to methyl mercury. Tox. Appl. Pharmacol., 1994;124:221-229.

Valentin-Blasini L., Blount B.C., Caudill S.P., et al. Urinary and serum concentrations of seven phytoestrogens in a human population subset, J. Expo. Anal. Environ. Epi., 2003;13:276-282.

Van den Berg M., Birnbaum L.S., Denison M., et. al. The 2005 World Health Organization re-evaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol. Sci., 2006;93:223-241.

Vanden Heuval J.P., and Lucier G. Environmental toxicology of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans. Environmental Health Perspectives. 1993;100:189-200.

Vandenberg L.N., Hauser R., Marcus M., et al. Human exposure to bisphenol A (BPA), Reprod. Toxicol., 2007;24:139-177.

Veglia F., Vineis P., Overvad K., et al. Occupational exposures, environmental tobacco smoke, and lung cancer, Epidemiology, 2007;18:769-775.

Vergne S., Sauvant P., Lamothe V., et al. Influence of ethnic origin (Asian vs Causasian) and background diet on the bioavailability of dietary isoflavones. British Journal of Nutrition, 2009;102:1642-1653

Vitali M., Ensabella F., Stella D., et al. Nonylphenols in freshwaters of the hydrologic system of an Italian district: association with human activities and evaluation of human exposure. Chemosph., 2004;57:1637-1647.

Walker J.B., Houseman J., Seddon L., et al. Maternal and umbilical cord blood levels of mercury, lead, cadmium and essential trace elements in Artic Canada, Environ. Res., 2006;100:295-318.

Webb A.L. and McCullough M.L. Dietary Lignans: potential role in cancer prevention. Nutr. Cancer, 2005;51:117-131.

Webster T., Vieira V. and Schecter A. Estimating human exposure to PBDE-47 via air, food and dust using Monte Carlo methods. Organohalo. Comp., 2005;67:505-508.

White R., Jobling S., Hoare S.A., et al. Environmentally persistent alkylphenolic compounds are estrogenic. Endocrinol., 1994;135:175-182.

Wong K.O., Leo L.W. and Seah H.L. Dietary exposure assessment of infants to bisphenol A from the use of polycarbonate baby milk bottles. Food Addit. Contam., 2005;22:280-288.

Wong P.S. and Matsumura F. Promotion of breast cancer by β-Hexachlorocyclohexane in MCF10AT1 cells and MMTV-neu mice. BMC Cancer. 2007;7(140):1-8.

World Health Organization (WHO).1990. Methylmercury, Vol. 101. World Health Organization, International Programme on Chemical Safety, Geneva, Switzerland, 1990.

World Health Organization (WHO). 1994. Brominated Diphenyl Ethers. IPCS Environmental Health Criteria 162. Geneva: World Health Organization, 1994.

World Health Organization (WHO). 2003. Chromium in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/chromium.pdf

World Health Organization (WHO). 2004. Endrin in drinking water: background document for the development of WHO *Guidelines for Drinking Water Quality*. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/endrin.pdf

World Health Organization (WHO). 2004b. Lindane in drinking water: background document for the development of WHO *Guidelines for Drinking Water Quality*. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/lindane.pdf

World Health Organization (WHO). 2004c. Hexachlorobenzene in drinking water: background document for the development of WHO *Guidelines for Drinking Water Quality*. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/hexachlorobenzene.pdf

World Health Organization. 2004d. DDT and its Derivatives in drinking water: background document for the development of WHO *Guidelines for Drinking Water Quality*. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/ddt.pdf

World Health Organization (WHO). 2005. Mercury in Drinking Water. Background document for development of WHO Guidelines for Drinking-water Quality. Accessed at: https://www.who.int/water_sanitation_health/dwq/chemicals/mercuryfinal.pdf

World Health Organization (WHO). 2011. Background document for development of WHO Guidelines for Drinking-water Quality. Accessed July 2014 from: http://www.who.int/water_sanitation_health/dwq/chemicals/cadmium.pdf

World Health Organization (WHO). 2011b. Lead in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/lead.pdf

World Health Organization (WHO). 2011c. Molybdenum in Drinking Water: Background document for development of WHO Guidelines for Drinking Water Quality. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/molybdenum.pdf

World Health Organization (WHO). 2013. Zinc supplementation during pregnancy: biological, behavioural and contextual rationale. Accessed at: http://www.who.int/elena/bbc/zinc_pregnancy/en/

World Health Organization (WHO). 2003b. Silver in Drinking Water. Accessed at: http://www.who.int/water_sanitation_health/dwq/chemicals/silver.pdf

Ying G.G., Williams B. and Kookana R., Environmental fate of alkylphenols and alkylphenol ethoxylates. Environ. Int., 2002;28:215-2

Zeghnoun A., Pascal M., Fréry N., et al.Dealing with the non-detected and non-quantified data. The example of the serum dioxin data in the French dioxin and incinerators study. Organohalogen Compounds 2007;69: 2288-2291

Zheng, G., Wang, L., Guo, Z. et al. Association of Serum Heavy Metals and Trace Element Concentrations with Reproductive Hormone Levels and Polycystic Ovary Syndrome in a Chinese Population. Biological trace element research, 2015;167:1-10.

Ziaee H., Daniel J., Datta, A.K., Blunt S., & McMinn D.J.W. Transplacental transfer of cobalt and chromium in patients with metal-on-metal hip arthroplasty A CONTROLLED STUDY. Journal of Bone & Joint Surgery, British Volume, 2007;89(3):301-305.

Zou E., and Matsumura F. Long-term exposure to β-hexachlorocyclohexane (β-HCH) promotes transformation and invasiveness of MCF-7 human breast cancer cells. Biochemical pharmacology, 2003;66(5):831-840