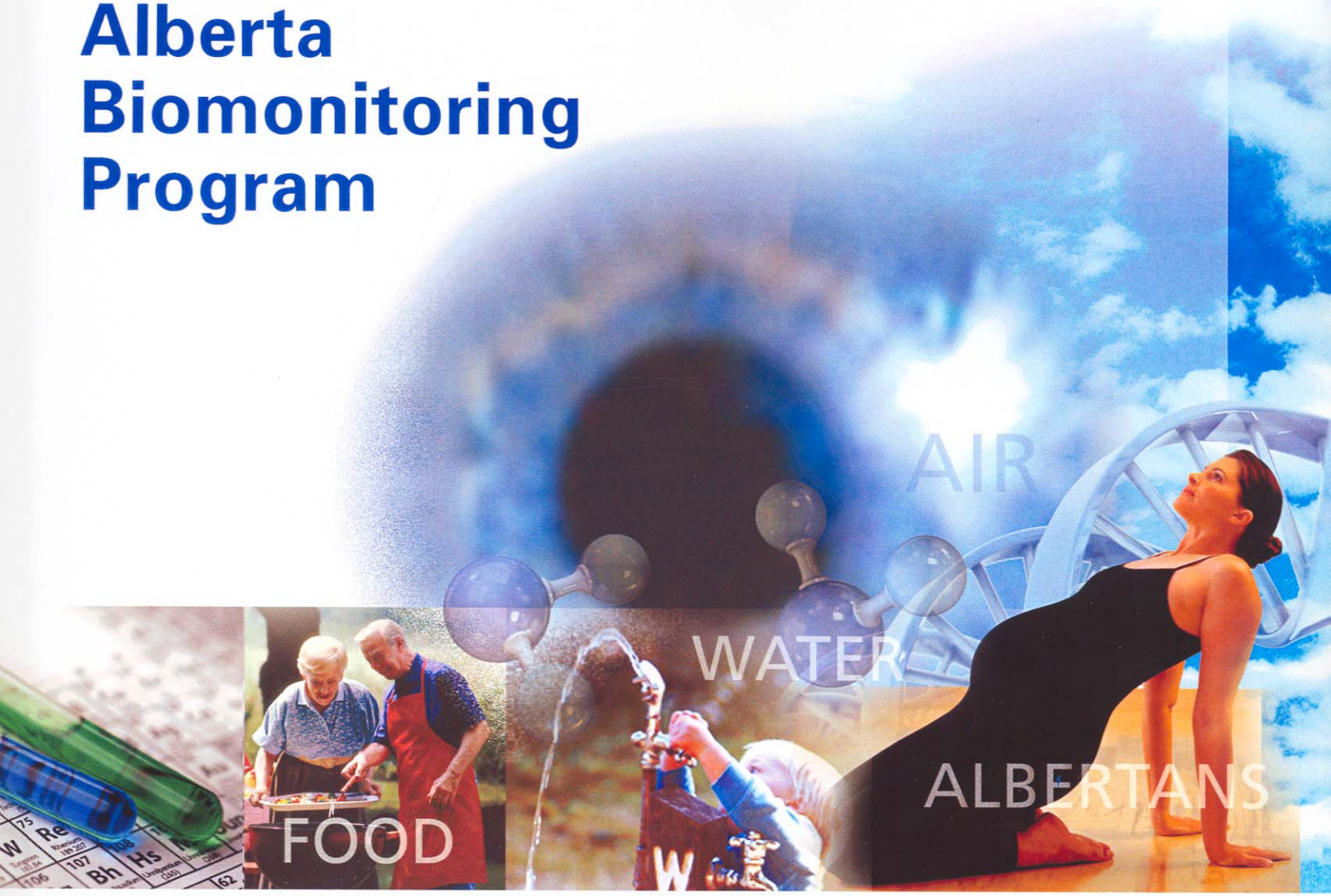
Alberta Biomonitoring Program



Chemicals in Serum of Pregnant Women in Alberta

Alberta

Alberta Health and Wellness (2008)

Alberta Biomonitoring Program: Chemicals in Serum of Pregnant Women in Alberta, Edmonton: Alberta Health and Wellness

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The

ALBERTA BIOMONITORING PROGRAM

Chemical Biomonitoring in Serum of Pregnant Women in Alberta (2005)

Influence of Age, Location and Seasonality

A Final Report
Submitted to Alberta Health and Wellness May 2008

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PREFACE

Purpose of Biomonitoring

Humans routinely come into contact with natural or synthetic chemical substances in the environment. Common routes of exposure include inhalation, ingestion, or dermal contact with environmental media such as air, water, food, and consumer products. The human health risks posed by a chemical are a function of the inherent chemical toxicity, how long we are exposed, and how much of the substance gets absorbed into our bodies; the internal dose. Any chemical has the potential to cause harm if the dose is high enough, and hence *the dose makes the poison*. The internal dose is difficult to predict because it is controlled by many environmental variables, and also because humans differ widely in physiology and behaviour. The most accurate way to assess the internal dose is to measure concentrations in relevant human samples such as blood, urine, hair, fat, or breast milk - this is known as *biomonitoring*.

Purpose of Biomonitoring Pregnant Women

In general, the developing fetus is the most sensitive human life-stage and can be exposed to chemicals that cross the placenta. 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. The purpose of this biomonitoring study was to establish the magnitude of women's exposure to environmental contaminants during pregnancy in all regions of Alberta. Neither Canada nor Alberta currently monitor chemicals in the human population in a systematic fashion, thus this report represents the first Province-wide biomonitoring results for a comprehensive set of established and emerging environmental chemicals that may be present in food, drinking water, commercial products, air, soil or household dust. The influence of age, geographic location, and seasonality (in Southern Alberta only) are also examined. The range of exposure concentrations 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 Alberta.

What Do These Data Mean for Me?

In this study individual human samples were combined and analyzed as 'pools', thus the concentrations reported in this report represent the average exposure in the population. With recent advancements in analytical methodologies and sophisticated analytical instrumentation, we are now able to measure very low concentrations of environmental chemicals in human samples. Therefore, it is important to keep in mind that the detection of an environmental chemical in human blood does not imply that this chemical is causing an adverse health effect. This is because, for most chemicals, there is a dose threshold below which no measurable health effects can be detected. This threshold is sometimes called the 'No Observed Effect Level' or the 'Safe Human Dose'. The exception is for chemicals that may cause cancer in humans. Chemical carcinogens are sometimes treated for regulatory purposes as if there is no level of exposure below

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which the risk of cancer is zero. Although the merits of this approach for carcinogens cannot be proven or disproven by scientific experiment, it is commonly used for precautionary public policy to establish acceptable limits of exposure representing negligible health risks.

In this report, results are listed by each chemical or chemical group, and some basic information about each chemical is provided. Possible exposure sources, toxicological effects in animals, human health effects, and any relevant exposure guidelines are discussed. Wherever possible, the exposure levels in the Alberta population are compared to other populations.

Selection of Chemicals for Biomonitoring

The targeted chemicals monitored in this report were selected using expert guidance and by reviewing data from similar studies in other countries. For example, the United States Centers for Disease Control and Prevention routinely publishes biomonitoring results for the general American population, the most recent of which was published in 2005 under the title: "The Third National Report on Human Exposure to Environmental Chemicals" (http://www.cdc.gov/exposurereport/). Therefore, data from some chemicals in the current report may be compared to several other populations, but the current report also includes concentrations for 'emerging contaminants', such as bisphenol A, brominated flame retardants, and perfluorinated acids for which fewer studies are available for comparison. In general, the chemicals reported in this study may be naturally occurring or man-made, current-use or phased-out, and rapidly excreted or bioaccumulative. Each chemical, or chemical family, has unique sources, behaviour, and toxicological profiles which are discussed later in this report.

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EXECUTIVE SUMMARY

This biomonitoring study of pregnant women in Alberta was conducted by a multi-disciplinary committee of academic and professional experts from Alberta Health and Wellness, the University of Alberta, the Alberta Centre for Toxicology at Calgary, the Provincial Public Health Laboratory (Edmonton), and Alberta Environment. A comprehensive list of priority environmental contaminants were monitored in blood serum of pregnant women in 2005, with the purpose of establishing the magnitude of exposure to environmental contaminants during pregnancy for women living in all regions of Alberta. The objectives were specifically to examine the influence of age, geographic location, and seasonality on the maternal serum concentrations of man-made or naturally occurring chemicals that women may absorb from food, drinking water, air, soil, household dust, or commercial products. This is the first Province-wide biomonitoring study for Alberta, and few other studies are currently available from elsewhere in Canada.

Samples of pregnant women's serum were drawn anonymously and randomly from a population of 50,599 individual serum samples that were submitted to the Provincial Public Health Laboratory (Edmonton) between January 1st, 2005, and December 31st, 2005. These samples are normally used for screening of infectious disease markers during early pregnancy, thus the current study used the small volumes that remained after routine processing. All samples that included information on maternal age and health region were eligible for pooling. Individual blood samples were not analyzed. Rather, within each geographic region and age class, 150-200 individual samples were pooled with a minimum of 8 replicate pools for each region-age class combination. The nine existing Health Regions of Alberta (in 2005) were stratified into 3 general geographic regions to represent Northern, Central, and Southern Alberta. Three age classes were further defined within each geographic region, representing ≤25 years, 26-30 years, and 31+ years of age. In Southern Alberta only, samples were also pooled by month of collection to investigate seasonal trends. We did not have enough resources to conduct an investigation of seasonality in all geographic areas.

The following classes of chemical contaminants were monitored: *List A* - tobacco smoke markers, phytoestrogens, polychlorinated biphenyls (PCBs), dioxins and furans, organochlorine pesticides, polybrominated compounds, perfluorinated compounds, methylmercury, lead and various other metals or mineral micronutrients; *List B* – polycyclic aromatic hydrocarbons (PAHs), organophosphate pesticides, various herbicides, and phenolic compounds, including Bisphenol A. *List B* contaminants were measured only in Southern Alberta because it was suspected that the current study design would not be sensitive enough to detect them in serum as they are normally measured in urine samples in similar biomonitoring studies; however some of these were detected and are reported.

The laboratories tasked with analyzing the samples included the Alberta Centre for Toxicology (Calgary, AB) (nicotine, cotinine, phytoestrogens, and most metals) and ALS Laboratory Group (Edmonton, AB) (all other chemicals). Quality control samples were also analyzed with the subject serum samples to monitor for possible contamination

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by collection vessels and routine sample handling. Both analytical laboratories were blind to the nature of the samples.

In general, the concentrations of detected contaminants were either lower or similar to concentrations previously determined in other studies in North America or around the world. Some chemicals displayed significant trends with age or geography. Seasonal variation was examined for many chemicals in Southern Alberta, but there was no apparent temporal or seasonal trend, with the exception of lead (Pb) which displayed a consistent seasonal trend. Lead concentrations were always below levels of concern, but higher concentrations were determined among all age groups in January, and in the ≤25 age group in July. The reason for this seasonal variability is unknown.

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. First, the current study, which analyzed 158 samples, was more cost effective than the alternative of analyzing thousands of individual samples. Nonetheless, the pooled study design remained an effective means of determining the distribution of chemical contaminants in the population while also enabling hypotheses to be tested on age, geography, and seasonality. Furthermore, because the distribution of concentrations in the population for any particular contaminant 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. For example, despite that Albertans had similar concentrations of mirex and hexachlorobenzene (organochlorine pesticides) in serum as recently reported for the U.S. population, the current study was able to detect these contaminants in 80% and 70% of all samples, respectively, compared to <50% of all individually analyzed U.S. samples by the same analytical method. Consequently, the median concentration of mirex and hexachlorobenzene is reported here for the Province of Alberta, whereas it has not been reported for the U.S.

Although the health implications posed by most of these contaminants at the concentrations observed are difficult to assess at this time, the clinical implications of smoking during pregnancy are comparably well understood by scientists and the general public. Therefore, serum cotinine (a nicotine metabolite and marker of cigarette smoke exposure) concentrations determined in this study are a concern. Serum cotinine concentrations measured among all pooled samples ranged from 5 to 55 ng/mL. Non-smokers are normally defined as having serum cotinine concentrations below 15 ng/mL. Therefore, the concentrations of cotinine measured here indicate that many pregnant Albertan women were smokers at the time of their blood sample collection, particularly in the youngest women examined, and in Northern Alberta (Health Regions 7, 8, and 9). The baseline concentrations of serum cotinine recorded here may serve as a benchmark by which the effectiveness of current and future smoking regulations and education campaigns may be measured. Similarly, future biomonitoring campaigns in Alberta are warranted to follow how bisphenol A, perfluorinated chemicals, and polybrominated flame retardant concentrations in human serum respond to recent national and global regulations on their use.

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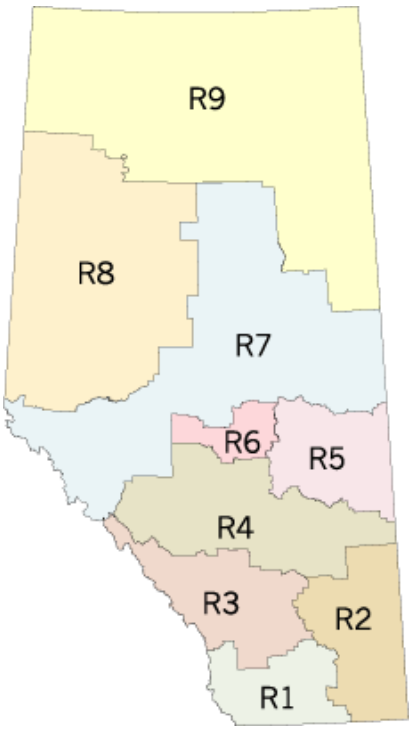
STUDY PROTOCOL

Selection of Sample Population

From January to December 2005, 50,599 serum samples from pregnant women were received at Provincial Public Health Laboratory (ProvLab) for routine infectious disease marker screening. Rather than be discarded, any left-over sample containing at least 1 mL was retained for the current study. Ethics approval was obtained for this study at the University of Alberta and the University of Calgary. Each sample was then designated as belonging to a specific health region based on the demographic information provided: patient's residence (n=41,761, 82.5%), submitter location (n=8,805, 17.4%), and submitting agency (n=33, 0.1%). Blood samples from Alberta were then stratified into three general geographic regions based on the 9 health regions (see map below) to represent Northern Alberta (health regions 7-9), Central Alberta (health regions 4-6), and Southern Alberta (health regions 1-3). The samples within each region were further stratified into three age classes (≤25 yrs, 26-30 yrs, 31+ yrs) with the samples from Southern Alberta further sub-divided by calendar month to assess seasonal and temporal trends. The following groups of samples were excluded from the current biomonitoring initiative:

1) Samples from outside the province of Alberta (n=1,999)
2) Samples that tested positive for antibodies to HIV and/or syphilis and/or Hepatitis B surface antigen (n=266)
3) Duplicate samples from the same individual (n=3,176)
4) Samples classified as from Southern Alberta but received at the Edmonton site of ProvLab (n=225), samples from Central Alberta but received at the Calgary site of ProvLab (n=280), samples from Northern Alberta but received in Calgary (n=29)
5) Samples with unknown maternal age (n=40)

Map of Alberta Health Regions (2005)



R1: Chinook Regional Health Authority
R2: Palliser Health Region
R3: Calgary Health Region
R4: David Thompson Regional Health Authority
R5: East Central Health
R6: Capital Health
R7: Aspen Regional Health Authority
R8: Peace Country Health
R9: Northern Lights Health Region

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Study Design for Pooling of Samples and Controls

A total of 28,484 samples with at least 1 mL of serum were randomly selected from the 44,584 samples stratified by geographic region, age group, and month of receipt. The pooling of serum samples was conducted at ProvLab (Edmonton) with equal volumes of blood serum (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 all the chemicals (150 mL). All pools in Central and Southern Alberta contained 200 mL of serum, whereas pools in Northern Alberta contained only 150-170 mL of serum due to fewer samples available for pooling. A total of 15 replicate pools were generated for each age class in Central Alberta and the <25 year old age class of Northern Alberta. A total of 24 replicate pools per age class were generated in Southern Alberta; 2 for each month to enable seasonal and temporal trend analysis. In the 26-30 and >30 year old age classes of Northern Alberta, only 11 and 8 replicate pools could be generated, respectively, due to fewer samples available for pooling. For quality assurance purposes, 7 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.

Only the staff of ProvLab had access to the demographic information of individual samples. Study codes were assigned to each pool before the samples were tested so that the subsequent chemical analyses were performed ‘blind’. The study codes were revealed for statistical analysis once the chemical analysis had been completed.

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LIST OF CHEMICALS

The following are the lists of specific chemical contaminants that were analyzed in this study. 'List A' chemicals were screened in all blood samples, whereas 'List B' analytes were measured only in the subset from Southern Alberta. This is because List B chemicals are normally measured in urine samples, and so there was less confidence over the sensitivity to detect these substances in blood. This was also done to keep the cost of analysis within budget. Since 'List B' chemicals were only analyzed in the 26-30 year age group of Southern Alberta, geographic trends could not be examined for these substances. When there were fewer than 25% of the pooled samples with detectable concentrations, the results were not reported.

Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
LIST A			
1. TOBACCO SMOKE			
Cotinine	5 ng/mL serum	> 25%	Reported
Nicotine	5 ng/mL serum	< 25%	Not Reported
2. PHYTOESTROGENS			
Daidzein	0.2 ng/mL serum	> 25%	Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
3. POLYCHLORINATED DIBENZO-P-DIOXINS AND POLYCHLORINATED DIBENZOFURANS			
1,2,3,6,7,8- Hexachlorodibenzo-p-dioxin (HxCDD)	0.01 pg/g serum	> 25%	Reported
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	0.01 pg/g serum	> 25%	Reported
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin (OCDD)	0.01 pg/g serum	> 25%	Reported
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	0.01 pg/g serum	> 25%	Reported
2,3,4,7,8- Pentachlorodibenzofuran (PCDF)	0.01 pg/g serum	> 25%	Reported
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	0.01 pg/g serum	> 25%	Reported
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	0.01 pg/g serum	> 25%	Reported
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	0.01 pg/g serum	> 25%	Reported
1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)	0.01 pg/g serum	> 25%	Reported
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	0.01 pg/g serum	< 25%	Not Reported
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PCDD)	0.01 pg/g serum	< 25%	Not Reported
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	0.01 pg/g serum	< 25%	Not Reported
1,2,3,7,8,9- Hexachlorodibenzo-p-dioxin (HxCDD)	0.01 pg/g serum	< 25%	Not Reported
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	0.01 pg/g serum	< 25%	Not Reported
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	0.01 pg/g serum	< 25%	Not Reported
2,3,4,6,7,8- Hexachlorodibenzofuran (HxCDF)	0.01 pg/g serum	< 25%	Not Reported
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	0.01 pg/g serum	< 25%	Not Reported
4. COPLANAR AND MONO-ORTHOSUBSTITUTED POLYCHLORINATED BIPHENYLS			
2,2′,3,4′,5,5′-Hexachlorobiphenyl (PCB 146)	5 pg/g serum	> 25%	Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
2,3,3′,4,4′,5-Hexachlorobiphenyl (PCB 156)	5 pg/g serum	> 25%	Reported
2,3,3′,4,4′,6-Hexachlorobiphenyl (PCB 158)	5 pg/g serum	> 25%	Reported
2,2′,3,3′,4,4′,5-Heptachlorobiphenyl (PCB 170)	5 pg/g serum	> 25%	Reported
2,2′,3,4,4′,5,5′-Heptachlorobiphenyl (PCB 180)	5 pg/g serum	> 25%	Reported
2,2′,3,4,4′,5′,6-Heptachlorobiphenyl (PCB 183)	5 pg/g serum	> 25%	Reported
2,2′,3,4′,5,5′,6-Heptachlorobiphenyl (PCB 187)	5 pg/g serum	> 25%	Reported
2,2′,3,3′,4,4′,5,5′-Octachlorobiphenyl (PCB 194)	5 pg/g serum	> 25%	Reported
2,2′,3,3′,4,5,5′,6′-Octachlorobiphenyl (PCB 199)	5 pg/g serum	> 25%	Reported
2,4,4′- Trichlorobiphenyl (PCB 28)	5 pg/g serum	< 25%	Not Reported
2,2,5,5 - Tetrachlorobiphenyl (PCB 43/52)	5 pg/g serum	< 25%	Not Reported
2,3′,4,4′- Tetrachlorobiphenyl (PCB 66)	5 pg/g serum	< 25%	Not Reported
2,4,4′,5- Tetrachlorobiphenyl (PCB 74)	5 pg/g serum	< 25%	Not Reported
3,3′,4,4′- Tetrachlorobiphenyl (PCB 77)	5 pg/g serum	< 25%	Not Reported
3,4,4′,5- Tetrachlorobiphenyl (PCB 81)	5 pg/g serum	< 25%	Not Reported
2,2′,3,4,5′-Pentachlorobiphenyl (PCB 87)	5 pg/g serum	< 25%	Not Reported
2,2′,4,4′,5-Pentachlorobiphenyl (PCB 99)	5 pg/g serum	< 25%	Not Reported
2,2′,4,5,5′-Pentachlorobiphenyl (PCB 101)	5 pg/g serum	< 25%	Not Reported
2,3,3′,4,4′-Pentachlorobiphenyl (PCB 105)	5 pg/g serum	< 25%	Not Reported
2,3,3′,4′,6-Pentachlorobiphenyl (PCB 110)	5 pg/g serum	< 25%	Not Reported
2,3′,4,4′,5-Pentachlorobiphenyl (PCB 118)	5 pg/g serum	< 25%	Not Reported
3,3′,4,4′,5-Pentachlorobiphenyl (PCB 126)	5 pg/g serum	< 25%	Not Reported
,2′,3,3′,4,4′-Hexachlorobiphenyl (PCB 128)	5 pg/g serum	< 25%	Not Reported
2,2′,3,4,4′,5-Hexachlorobiphenyl (PCB 138)	5 pg/g serum	< 25%	Not Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
2,2′,3,4′,5′,6-Hexachlorobiphenyl (PCB 149)	5 pg/g serum	< 25%	Not Reported
2,2′,3,5,5′,6-Hexachlorobiphenyl (PCB 151)	5 pg/g serum	< 25%	Not Reported
2,2′,4,4′,5,5′-Hexachlorobiphenyl (PCB 153	5 pg/g serum	< 25%	Not Reported
2,3,3′,4,4′,5′-Hexachlorobiphenyl (PCB 157)	5 pg/g serum	< 25%	Not Reported
2,3′,4,4′,5,5′-Hexachlorobiphenyl (PCB 167)	5 pg/g serum	< 25%	Not Reported
3,3′,4,4′,5,5′-Hexachlorobiphenyl (PCB 169)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,5,5′-Heptachlorobiphenyl (PCB 172)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,5′,6′-Heptachlorobiphenyl (PCB 177)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,5,5′,6-Heptachlorobiphenyl (PCB 178)	5 pg/g serum	< 25%	Not Reported
2,3,3′,4,4′,5,5′-Heptachlorobiphenyl (PCB 189)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,4′,5,6-Octachlorobiphenyl (PCB 195)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,4′,5,6′-Octachlorobiphenyl (PCB 196)	5 pg/g serum	< 25%	Not Reported
2,2′,3,4,4′,5,5′,6-Octachlorobiphenyl (PCB 203)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,4′,5,5′,6-Nonachlorobiphenyl (PCB 206)	5 pg/g serum	< 25%	Not Reported
2,2′,3,3′,4,4′,5,5′,6,6′-Decachlorobiphenyl (PCB 209)	5 pg/g serum	< 25%	Not Reported
5. ORGANOCHLORINE PESTICIDES			
Mirex	0.05 ng/g serum	> 25%	Reported
4,4′-DDE	0.1 ng/g serum	> 25%	Reported
Hexachlorobenzene	0.1 ng/g serum	> 25%	Reported
alpha Hexachlorocyclohexane	0.1 ng/g serum	< 25%	Not Reported
beta Hexachlorocyclohexane	0.05 ng/g serum	< 25%	Not Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
gamma Hexachlorocyclohexane	0.05 ng/g serum	< 25%	Not Reported
delta Hexachlorocyclohexane	0.1 ng/g serum	< 25%	Not Reported
4,4′-DDT	0.1 ng/g serum	< 25%	Not Reported
2,4′-DDT	0.1 ng/g serum	< 25%	Not Reported
Chlordane	0.05 ng/g serum	< 25%	Not Reported
Oxychlordane	0.05 ng/g serum	< 25%	Not Reported
***trans*-Nonachlor**	0.1 ng/g serum	< 25%	Not Reported
Heptachlor	0.1 ng/g serum	< 25%	Not Reported
Heptachlor Epoxide	0.1 ng/g serum	< 25%	Not Reported
Aldrin	0.05 ng/g serum	< 25%	Not Reported
Dieldrin	0.1 ng/g serum	< 25%	Not Reported
Endrin	0.05 ng/g serum	< 25%	Not Reported
4,4′-DDD	0.1 ng/g serum	< 25%	Not Reported
Endosulfan	0.1 ng/g serum	< 25%	Not Reported
Methoxychlor	0.1 ng/g serum	< 25%	Not Reported
Octachlorostyrene	0.1 ng/g serum	< 25%	Not Reported
6. POLYBROMINATED COMPOUNDS			
Polybrominated Biphenyls (PBBs)	5 - 25 pg/g serum	Not Detected	Not reported
Tetrabromobisphenol A (TBBP-A)	30 pg/g serum	Not Detected	Not reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
Polybrominated diphenyl ethers (PBDEs)			
BDE 28	0.07 ng/g lipid	> 25%	Reported
BDE 47	0.2 ng/g lipid	> 25%	Reported
BDE 66	0.2 ng/g lipid	> 25%	Reported
BDE 77	0.2 ng/g lipid	< 25%	Not Reported
BDE 85	0.3 ng/g lipid	> 25%	Reported
BDE 99	0.3 ng/g lipid	> 25%	Reported
BDE 100	0.2 ng/g lipid	> 25%	Reported
BDE 138	0.2 ng/g lipid	< 25%	Not Reported
BDE 153	0.2 ng/g lipid	> 25%	Reported
BDE 154	0.2 ng/g lipid	> 25%	Reported
BDE 183	0.2 ng/g lipid	< 25%	Not Reported
BDE 209	0.2 ng/g lipid	< 25%	Not Reported
7. PERFLUORINATED COMPOUNDS			
Perfluorohexane sulfonate (PFHxS)	0.5 ng/g serum	> 25%	Reported
Perfluorooctane sulfonate (PFOS)	0.1 ng/g serum	> 25%	Reported
Perfluorodecane sulfonate (PFDS)	0.15 ng/g serum	< 25%	Not Reported
Perfluorooctanoate (PFOA)	0.02 ng/g serum	> 25%	Reported
Perfluorononanoate (PFNA)	0.05 ng/g serum	> 25%	Reported
Perfluorodecanoate (PFDA)	0.1 ng/g serum	> 25%	Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
Perfluoroundecanoate (PFUA)	0.1 ng/g serum	> 25%	Reported
Perfluorododecanoate (PFDoA)	0.05 ng/g serum	> 25%	Reported
Perfluorotetradecanoate (PFTA)	0.1 ng/g serum	> 25%	Reported
8. MERCURY			
Methylmercury	0.03 ng/g serum	> 25%	Reported
9. METALS			
Aluminium	10 µg/L serum	> 25%	Reported
Antimony	0.2 µg/L serum	> 25%	Reported
Arsenic	0.5 µg/L serum	< 25%	Not Reported
Barium	0.2 µg/L serum	> 25%	Reported
Beryllium	0.2 µg/L serum	< 25%	Not Reported
Boron	2 µg/L serum	> 25%	Reported
Cadmium	0.2 µg/L serum	< 25%	Not Reported
Chromium	0.2 µg/L serum	> 25%	Reported
Cobalt	0.2 µg/L serum	> 25%	Reported
Copper	0.2 µg/L serum	> 25%	Reported
Iron	10 µg/L serum	> 25%	Reported
Lead	0.2 µg/L serum	> 25%	Reported
Zinc	5 µg/L serum	> 25%	Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
Mercury	0.2 µg/L serum	> 25%	Reported
Manganese	0.2 µg/L serum	> 25%	Reported
Molybdenum	0.2 µg/L serum	> 25%	Reported
Nickel	0.2 µg/L serum	> 25%	Reported
Selenium	0.5 µg/L serum	> 25%	Reported
Silver	0.2 µg/L serum	> 25%	Reported
Thallium	0.2 µg/L serum	< 25%	Not Reported
Vanadium	0.2 µg/L serum	> 25%	Reported
Cesium	0.2 µg/L serum	> 25%	Reported
Platinum	0.2 µg/L serum	< 25%	Not Reported
Tungsten	0.2 µg/L serum	< 25%	Not Reported
Uranium	0.2 µg/L serum	< 25%	Not Reported

LIST B

1. POLYCYCLIC AROMATIC HYDROCARBONS			
benz[a]Anthracene	3 pg/g serum	< 25%	Not Reported
benzo[c]Phenanthrene	2 pg/g serum	< 25%	Not Reported
Chrysene	5 pg/g serum	< 25%	Not Reported
Pyrene	200 pg/g serum	< 25%	Not Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
2. ORGANOPHOSPHATE PESTICIDES			
Acephate	1 pg/g serum	Not Detected	Not Reported
Azinphos methyl	1 pg/g serum	Not Detected	Not Reported
Chlorpyrifos	1 pg/g serum	Not Detected	Not Reported
Coumaphos	1 pg/g serum	Not Detected	Not Reported
Dichlorvos	0.5 pg/g serum	Not Detected	Not Reported
Diazinon	0.5 pg/g serum	Not Detected	Not Reported
Dicrotophos	0.2 pg/g serum	Not Detected	Not Reported
Dimethoate	1 pg/g serum	Not Detected	Not Reported
Disulfoton	1 pg/g serum	Not Detected	Not Reported
Ethion	1 pg/g serum	Not Detected	Not Reported
Fenitrothion	1 pg/g serum	Not Detected	Not Reported
Fenthion	1 pg/g serum	Not Detected	Not Reported
Malathion	0.5 pg/g serum	Not Detected	Not Reported
Methamidophos	1 pg/g serum	Not Detected	Not Reported
Methidathion	0.5 pg/g serum	Not Detected	Not Reported
Methyl parathion	1 pg/g serum	Not Detected	Not Reported
Oxydemeton-methyl	1 pg/g serum	Not Detected	Not Reported
Parathion	0.5 pg/g serum	Not Detected	Not Reported
Phorate	0.5 pg/g serum	Not Detected	Not Reported
Phosmet	1 pg/g serum	Not Detected	Not Reported
Pirimiphos-methyl	1 pg/g serum	Not Detected	Not Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
Sulfotepp	0.2 pg/g serum	Not Detected	Not Reported
Temephos	1 pg/g serum	Not Detected	Not Reported
Terbufos	1 pg/g serum	Not Detected	Not Reported
Tetrachlorvinphos	1 pg/g serum	Not Detected	Not Reported
3. HERBICIDES & PESTICIDES			
***cis*-DCCA**	30 pg/g serum	< 25%	Not Reported
***trans*-DCCA**	30 pg/g serum	< 25%	Not Reported
2,4-Dichlorophenoxyacetic acid	100 pg/g serum	< 25%	Not Reported
2,4,5-Trichlorophenoxyacetic acid	60 pg/g serum	< 25%	Not Reported
fluoro-3-phenoxybenzoic acid	25 pg/g serum	< 25%	Not Reported
3-phenoxybenzoic acid	50 pg/g serum	< 25%	Not Reported
4. PHENOLS			
Bisphenol A	10 pg/g serum	> 25%	Reported
Octylphenol	2000 pg/g serum	>25%	Not Reported due to contamination of quality control samples
Nonylphenol	2000 pg/g serum	> 25%	Reported
Pentachlorophenol	250 pg/g serum	< 25%	Not Reported
2,4,5-Trichlorophenol	40 pg/g serum	< 25%	Not Reported

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Chemical Name	LOD	Percent of Samples Above Detection Limits	Report Status
2,4,6- Trichlorophenol	40 pg/g serum	< 25%	Not Reported

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ANALYTICAL LABORATORIES

Select chemical analyses were done internally at the Alberta Centre for Toxicology (Calgary). These chemical classes included nicotine, cotinine, phytoestrogen and metals. The remaining chemicals were analyzed in a private laboratory by ALS Laboratory Group (Edmonton, AB Canada). 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)

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SUMMARY OF ANALYTICAL METHODS

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. 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.

Dioxins/Furans

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 C_{18} (10 g / 75 mL) cartridge at 5 mL per minute. 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).

Polychlorinated Biphenyls and Polybrominated Biphenyls

Isotopically labelled PCBs and PBBs along with an equal amount of formic acid and HPLC grade water (1:1:1) were added to an aliquot (5 g) of serum. The mixture was vortexed, sonicated and subjected to solid phase extraction using an EZ-Extract C_{18} (10g / 75 mL) cartridge at 5 mL per minute. 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).

Organochlorine Pesticides

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.

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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)

Polybrominated Diphenyl Ethers

Isotopically labelled PBDEs along with an equal amount of formic acid and HPLC grade water (1:1:1) were added to an aliquot (10 g) of serum. The mixture was vortexed, sonicated and subjected to solid-phase extraction using an EZ-Extract C18 cartridge at 5 mL per minute. 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. Analyses were performed using high resolution gas chromatography/high resolution mass spectrometry (Mass spectrometer: Finnigan MAT 95 XP (Thermo) equipped with two GC Agilent Technologies 6890, Mass spectrometrer: DFS (Thermo), equipped with Trace GC Ultra).

Tetrabromobisphenol A

Isotopically labelled TBBP-A was added to an aliquot (10 g) of serum, and the mixture was vortexed and sonicated. The mixture was serially extracted with ethyl acetate and centrifuged. The extract was concentrated just to dryness and reconstituted with acetonitrile. Hexane was added and an acetonitrile/hexane partition was performed discarding the hexane layer. The partition was repeated and the resulting acetonitrile was concentrated. Following the addition of 0.2 M acetate buffer (pH 5.2), 10000 units of β-glucuronidase were added and incubation was performed overnight. Following enzymatic hydrolysis, the mixture was centrifuged and extracted using an OASIS HLB cartridge. Following washing and drying of the cartridge, TBBP-A was eluted with dichloromethane. Further cleanup was performed on a SPE silica column. Analysis was performed by LC/MS/MS using multiple reaction monitoring (MRM).

Perfluorinated Compounds

Formic acid along with isotopically labelled PFOS, PFOA, PFNA and PFDA were added to 1 mL of serum. The mixture was vortexed, sonicated and subjected to solid phase extraction using an OASIS HLB cartridge. The perfluorinated compounds were then eluted with 1% ammonium hydroxide/acetonitrile. The extract was concentrated to 100 μL and recovery standard (fluoro-n-heptanoic acid) was added along with 200 μL of 90% 20 mM acetic acid/10% methanol. Analysis was performed by LC/MS/MS (API 3000 LC/MS/MS Sciex, Perkin-Elmer 200 Autosampler, Series 200 Micropump Perkin-Elmer) using multiple reaction monitoring (MRM).

Phenols

These included: 2,4,5-trichlorophenol, 2,4,6-trichlorophenol, bisphenol A, nonylphenol and octylphenol. A subset of samples were also analyzed for ortho-phenylphenol and 2,5-dichlorophenol. Isotopically labelled standards and an equal

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volume of formic acid and HPLC grade water (1:1:1) were added to an aliquot (5g) of serum. The mixture was extracted using a 500 mg / 6 mL Strata x SPE cartridge. Following drying with a stream of nitrogen, the SPE cartridge was eluted with acetone and dichloromethane. The resulting extract was concentrated, derivatized using acetic anhydride/pyridine and analyzed using high resolution gas chromatography/high resolution mass spectrometry (Micromass (Waters) Autospec M-Series with Agilent 5890 Series II GC and with CTC A200S Autosampler).

Polycyclic Aromatic Hydrocarbons

Isotopically labelled standards were added to an aliquot (10 g) of serum. The sample was extracted with dichloromethane and subjected to silica gel cleanup. Following the addition of a recovery standard the sample was analyzed using high resolution gas chromatography/high resolution mass spectrometry (Micromass (Waters) Autospec M-Series with Agilent 5890 Series II GC and with CTC A200S Autosampler).

Organophosphate Pesticides

Isotopically labelled pesticides (diethoxyterbufos, diethylchlorpyrifos and diethylparathion) were added to an aliquot (10 g) of serum. The sample was extracted with dichloromethane, concentrated and analyzed using gas chromatography/selected ion-monitoring mass spectrometry (Micromass (Waters) Autospec M-Series with Agilent 5890 Series II GC and with CTC A200S Autosampler).

Herbicides

Isotopically labelled pesticides (2,4-D, Atrazine, Alachlor and Metolachlor) were added to an aliquot (10 g) of serum. Following the addition of trichloroacetic acid the mixture was extracted with methyl-t-butyl ether. The resulting extract was concentrated, methylated with diazomethane and analyzed using gas chromatography/selected ion-monitoring mass spectrometry (Micromass (Waters) Autospec M-Series with Agilent 5890 Series II GC and with CTC A200S Autosampler).

Pyrethroids

Isotopically labelled permethrin was added to an aliquot (10 g) of serum, and was then extracted with methyl-t-butyl ether. The resulting extract was concentrated, exchanged into toluene and analyzed using gas chromatography/mass spectrometry (Micromass (Waters) Autospec M-Series with Agilent 5890 Series II GC and with CTC A200S Autosampler).

Metals

8.9 mL of 1%HNO_3/0.5%HCl solution was placed in a 13 mL pre-rinse tube. 1mL of serum sample was added to it, followed by 20µL of 10µg/mL internal standard mix. The supernatant was analyzed following 10 minutes centrifugation of the sample. The final dilution factor should be 10-fold and the final concentration of internal standards was 20µg/L. Due to high iron, copper and zinc concentrations in the sample, the sample was also analyzed with 100-fold dilution.

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The determination of trace metals in serum was performed on an Agilent 7500c Inductively Coupled Plasma Mass Spectrometer (ICP-MS) with Octopole Reaction System (ORS). The diluted sample was directly introduced into the ICP-MS through a Babington nebulizer. The ICP-MS employed plasma as the ionization source. When ions from the sample entered the ORS, they interacted with the reaction gas (either hydrogen or helium), resulting in the reduction of the molecular interference. Then the ions were focused into a quadrupole mass analyzer, which separates the ions based on their mass/charge ratio.

Phytoestrogens

1 mL of serum per sample was used. An internal standard was added for quantification and to compensate for any sample loss during extraction. This method involved enzymatic hydrolysis using a purified extract of Helix pomatia containing β-glucuronidase and sulphatase (Type H-5). The sample was then undergone a solid phase extraction on C_{18} cartridge. The extract was evaporated to dryness under a gentle stream of nitrogen gas. Daidzein was reconstituted in 200µL of water-acetonitrile mixture (80:20) before injecting onto the LC/MS/MS.

10 µL of extract was injected onto a MDS Sciex API4000 LC/MS/MS. The LC column was a Zorbax SB-C C_{18} rapid resolution column. The mobile phases were 0.01% formic acid in Type I water and 0.01% formic acid in acetonitrile. The flow rate was 800 µL/min and the run time was 5 minutes.

Daidzein was identified based on retention time in multiple reactions monitoring (MRM) mode. It was quantified based on area ratios and a six point calibration curve (0, 0.2, 0.5, 1, 2 and 10ng/mL). The mass to charge ratios (m/z) monitored for daidzein were m/z 253 and 91, and for the internal standard (daidzein-d_3) were m/z 256.2 and 226.1.

Nicotine & Cotinine

1mL of serum per sample was used for analysis of nicotine and cotinine. An internal standard was added for quantification and to compensate for any sample loss during extraction. The serum was treated with 0.1 M acetic acid and then was undergone a solid phase extraction on Clean Screen EZ-Extract Extraction columns. The columns were conditioned with methanol, then acetic acid. The samples were loaded on the columns, afterwards the columns were rinsed with acetic acid and methanol and the columns were dried briefly to eliminate water. Nicotine and cotinine were eluted from the columns with 2% ammonium hydroxide in ethyl acetate. The extract was concentrated under nitrogen at 40ºC to a final volume of 150 µL.

2µL of extract was injected in splitless mode onto a Gas Chromatograph (HP 6890) coupled to a Mass Spectrometer (HP5973) detector. The GC column was a J&W Scientific HP-5MS. The GC temperature program was ramped from 80ºC to 290ºC. The run time was 11 minutes.

Nicotine was identified based on retention time and ion ratios in selected ion monitoring mode, and quantified based on area ratios and a six point calibration curve (0,

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5, 10, 25, 50, and 100 ng/mL). The ions monitored for nicotine were *m/z* 133, 161, and 84, and for the internal standard (nicotine-d3) the ions were *m/z* 87, 136, and 164.

Cotinine was also identified based on retention time and ion ratios in selected ion monitoring mode, and quantified based on area ratios and a six point calibration curve (0, 5, 10, 50, 100, and 500ng/mL). The ions monitored for cotinine were *m/z* 98, 176, and 119, and for the internal standard (cotinine-d3) the ions were *m/z* 101, and 179.

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DATA ANALYSIS

The estimated concentrations can be analyzed by regression methods in which age and geographic region are considered as independent variables. There are two complications to this analysis related to pooled sampling. First, the distribution of chemical contaminants within individual samples is generally assumed to be log-normally distributed. According to the Central Limit Theorem, however, estimates derived from a sample created by pooling a large number of individual samples will be normally distributed. Consequently, no transformation of data is required prior to a regression analysis, and standard statistical significance tests which assume normal distributions remain appropriate. The second complication is that the parameters of the distribution of pooled estimates depend upon the number of individual samples that make up each pooled sample. Because pooled samples were constructed with differing numbers of samples (especially in Southern Alberta), data must be differentially weighted for accurate regression analyses according to the number of individual samples used to create each pooled sample. These choices were verified to be accurate through Monte Carlo simulation.

Weighted regression analyses were conducted using SPSS (version 15). Graphs were generated using Sigmaplot (version 10). In what follows, graphs of estimated concentrations are presented and descriptions in the text provided only for effects that were discovered to be statistically significant (at the level of $\alpha < 0.05$). The 95% confidence intervals presented on the graphs were separately derived for each estimate.

In Southern Alberta, temporal variation was examined graphically, and statistical analysis followed when a trend was evident. However, there were very few indications of temporal (i.e. upward or downward trends with time) or seasonal (consistently higher or lower in some months compared to others) relationships within the data, and only for lead are details presented describing the seasonal trend.

For cases in which there were non-detects reported, a value of LOD/2 was substituted. In any situation in which this occurs, this biases the analyses towards reporting significant findings by underestimating variability. When there were fewer than 25% of the pooled samples with detectable concentrations, no analyses were conducted.

It should be noted that the use of pooled sampling allows relatively precise detection of contaminant concentrations even where a substantial portion of individual samples would have had non-detectable concentrations. In preliminary simulations using log-normal data, fewer 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.

Ott (1995) has presented a detailed argument to support the contention that the distribution of contaminants in blood will be log-normal [1]. Under this assumption, it is possible to use data from the current study to estimate the parameters of a log-normal distribution, and in particular, the median. It is possible that this method will estimate median contaminant levels at or below the LOD since the median is below the mean in skewed distributions, and the mean relies strongly on the largest individual values.

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ORGANIC CONTAMINANTS

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Cotinine

GENERAL INFORMATION

Sources

Tobacco smoking can lead to disability, disease, and death, and maternal smoking during pregnancy is one of the most crucial risk factors for pregnancy complications and is harmful to the fetus [2]. There is a growing interest about the assessment of prenatal tobacco smoke exposure and outcomes of pregnancy [3,4]. There are thousands of chemical components in tobacco smoke, so rather than attempting to determine our exposure to all of these, nicotine and its metabolites are often used as markers of cigarette smoke exposure.

Nicotine is a naturally occurring component in all tobacco products. One cigarette contains approximately 6-12 mg of nicotine [5]. With each cigarette smoked, about 1 mg of nicotine is absorbed into the body of the smoker [6]. The remaining 75%, or more, of the nicotine from a cigarette is emitted to the air, and thus nicotine and its metabolites are also a marker of environmental tobacco smoke (ETS) [7,8].

Assessment of exposure to tobacco smoke can be done by measuring nicotine and its metabolites in different body fluids such as serum [9], saliva [10], urine [11] and cord blood [12]. However, one of the main human metabolites of nicotine is cotinine. The concentrations of cotinine in body fluids are proportional to the extent of exposure to tobacco smoke. Thus, cotinine has been widely used as a biomarker for tobacco exposure, including second hand smoke [13,14], because it has a longer half-life in blood (16 hr), and has fewer analytical interferences [15,16,17].

Smoking Rates and Regulations in Alberta

In Alberta, approximately one-fifth (22.8%) of the population smokes. Among smokers, 78% are daily smokers, and 22% are considered occasional smokers [18]. Unfortunately, the rate of smoking among women increases during childbearing years (between 15 and 44 years of age). In Alberta, approximately 25-32% of current or former female smokers (ages 15 to 55) smoke, or smoked, regularly during their most recent pregnancy that had occurred within the past 5 years. This smoking rate among pregnant women in Alberta is higher than the national average of 19 to 22% [19,20].

In March 2002, the Alberta Government introduced the Alberta Tobacco Reduction Strategy (ATRS) to co-ordinate tobacco reduction efforts in Alberta. The ATRS aims to reduce smoking rates in pregnant women from 32% in 2000/2001 to 12% in 2010/2011.

On November 14, 2007, Alberta declared a new Tobacco Reduction Act and approved a province-wide smoking ban in all public places and workplaces. This took effect on January 1, 2008. Smoking from windows and near doorways of public places is also restricted to protect indoor air quality.

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Possible Health Effects

Tobacco smoke, both direct and second-hand smoke, has significant adverse effects on health. The main health risks from tobacco smoke are related to diseases of the cardiovascular system, diseases of the respiratory tract, and cancers; particularly lung, larynx, and mouth [21,22]. In addition to adverse effects on the mother, smoking during pregnancy has negative effects on the fetus, newborn infant, and young child [2,4]. Smoking during pregnancy significantly increases risks of stillbirth, spontaneous abortion, premature delivery, neonatal death and subnormal birth weight [23,24]. It is also linked to higher rates of sudden infant death and increased incidence of childhood respiratory illness [24,25].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean cotinine concentrations in blood serum of pregnant Albertan women ranged from 5.1 ng/mL to 55 ng/mL. Concentrations depended on age and geographic region as shown in Figure 1. The concentrations in the north were greater than those in the central region, and in turn those in the central region were greater than those in the south. In general, the concentrations in the ≤25 year age group were greater than those in the 26-30 year age group which were in turn greater than those in the 31+ age group. In the north, however, the concentrations for the age 26-30 year age group were lower than those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

The concentrations of cotinine measured here indicate that many pregnant Albertan women were smokers at the time of their blood sample collection. Non-smokers are often defined as having serum cotinine concentrations below 15 ng/mL [26]. For example, nonsmokers (20 years and older) examined in the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third Report, CDC) had a geometric mean of 0.05 ng/mL serum cotinine [27]. In contrast, in another U.S. NHANES study (1988-1991), serum cotinine concentrations among smokers (males and females, >17 years age) ranged from 22 to 113 ng/mL [28].

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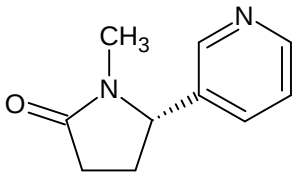

Cotinine

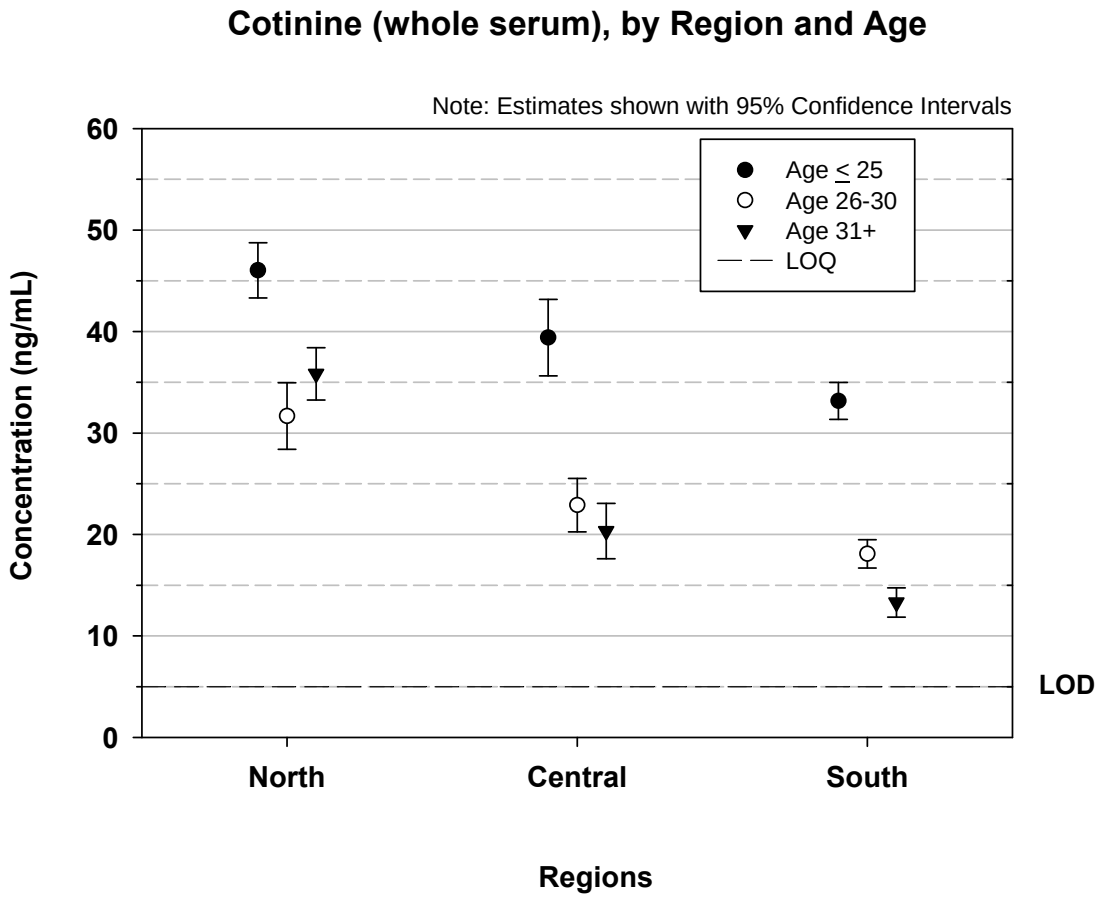

Figure 1

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Phytoestrogens

GENERAL INFORMATION

Sources

Phytoestrogens are naturally occurring chemicals in plants, and have non-steroidal estrogen-like activities [29,30]. These compounds consist of three major groups: isoflavones (e.g. genistein, daidzein and glycitein), lignans and coumestans [31,32]. Dietary intake is the major source of phytoestrogens for humans, and the main sources include isoflavones in soy bean, soy bean products (e.g. tofu), and legumes; while lignans are found in flaxseed, citrus fruit, wheat, fennel, celery, and nuts [33].

Normally, the Asian and Latin American diets are relatively high in phytoestrogens because of the common use of soy products and legumes. Western diets (i.e. U.S. and Canada) generally contain a low or moderate amount of phytoestrogens due to lesser intake of soy products and legumes [34,35,36]. For example, 20-80 mg/day of isoflavones are present in the Japanese diet, whereas the typical Western diet contains less than 1 mg of isoflavones per day [37]. However, due to the health benefits of phytoestrogens, such as protection against various cancers, the use of soy products in foodstuffs, and the use of phytoestrogens as dietary supplements are gradually increasing in Canada [37]. Soy-based formula and human breast milk are the major sources of phytoestrogens to infants, and exposure through breast milk is related to the level of maternal dietary intake of soy products [38,39].

Possible Health Effects

Phytoestrogens do not accumulate in the human body – after being readily absorbed in the gut, they circulate in blood and are rapidly excreted in the urine [40]. Several studies have suggested that phytoestrogens have various health benefits, including protection against breast and colorectal cancer, cardiovascular disease, osteoporosis and menopausal symptoms [41,42,43]. Most of these studies have focused on the beneficial effects of phytoestrogens, but recently a few studies were conducted to investigate their potential adverse health effects. Since phytoestrogens have estrogen-like activities, high consumption of these substances may result in effects similar to those associated with excess estrogen. However, no definitive conclusions have been reached, as some results are contradictory. For example, one report has shown that a high soy content vegetarian diet during pregnancy could be associated with an increased incidence of hypospadias [44], while other studies reported no observed hormonal effects in long-term soy-based formula fed infants [45]. More clinical and epidemiological studies are required in this area.

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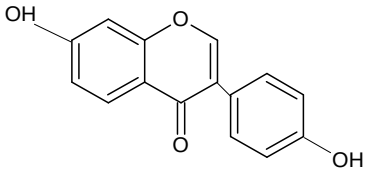
BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

In the present study, only one isoflavone (Daidzein) was measured in blood serum samples of pregnant women. Overall, the mean concentration for daidzein ranged from 0.7 – 6.1 ng/mL. Daidzein concentrations in blood serum were dependent on age and geographic region (Figure 2). The concentrations in the north were lower than those in the south region and in turn those in the south region were lower than those in the central. In general, the concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. In the north, however, the concentrations for the age 26-30 year age group were not different from those in the age group 31+. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. The estimated 50th centile among individuals was 0.3 ng/mL. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), daidzein was detectable in urine samples of the U.S. population [27], but those concentrations cannot be compared with the present blood serum results for Alberta. In a study in the U.S. population, a subset of adults (208 samples, >20 years age, 61% female) from NHANES (1988-1994), the mean daidzein concentration in serum was 3.9 ng/mL [46].



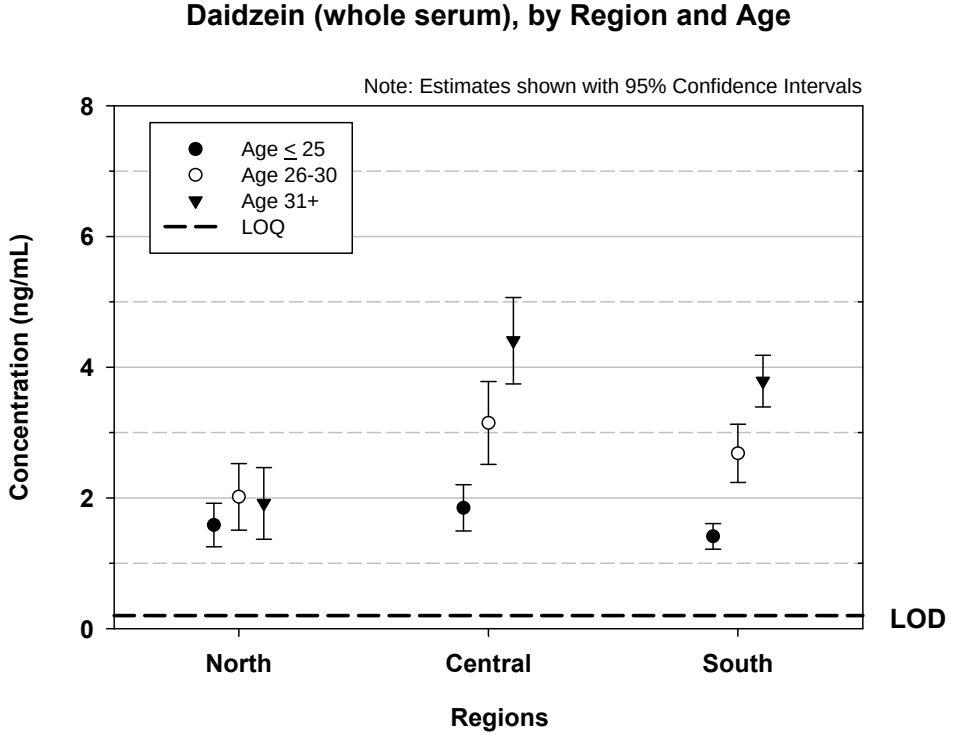


Figure 2

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Dioxins and Furans

GENERAL INFORMATION

Sources

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), more commonly known as dioxins and furans, are relatively toxic and persistent environmental contaminants. Depending on the pattern of chlorine atoms on these molecules, dioxin and furan molecules can vary widely in their toxic potency [47,48]. They are not directly produced for any purpose, but are by-products of several industrial processes (e.g. incineration, pulp bleaching, pesticide production), burning of municipal and medical waste, backyard burning of household waste, wood burning and electrical power generation. Dioxins and furans can also be produced naturally, such as during forest fires and volcanic eruptions. After their release to the environment, they can travel long distances including to remote regions far from their sources.

The predominant source of dioxin and furan exposure in the general human population is through the diet. Due to their lipophilic (literally 'fat-loving') nature, they accumulate in fatty tissues of fish and animals after being deposited to soil or water. Dietary intake through food products of animal origin is estimated to account for 90% of total human exposure [49,50,51]. These compounds can also accumulate in our bodies and take a long time to be excreted. Some of the maternal body burden can be transferred to the fetus or infant during pregnancy or lactation, respectively [52,53]. However, breastfeeding is encouraged due to the many associated health-benefits [54] that currently outweigh known risks.

Regulations in Canada

Dioxins and furans are designated to be virtually eliminated in Canada [55]. Since July 1st, 1992, the releases of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and 2,3,7,8-tetrachlorodibenzofuran (TCDF), which are the most toxic forms of dioxin and furan, have been prohibited in pulp and paper mill effluent in Canada [55]. By 1995, the emissions from this source reached non-detectable levels [55]. Canada 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 [55].

Possible Health Effects

Dioxins and furans can cause several adverse health effects in humans. These depend on the dose, the length of exposure, and the timing of exposure. Due to their ubiquity in the environment and our food, all people have a certain concentration of dioxins and furans in their body. Such background concentrations of dioxins and furans usually do not affect human health. At higher doses, such as which may occur through

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occupational exposures (e.g. past workers in the pulp and paper industry, in incineration plants, and at hazardous waste sites) or by accidental consumption of highly contaminated food, dioxins and furans may cause skin disorders (e.g. chloracne), liver problems, impairment of the immune system and the endocrine system, including diabetes, reproductive dysfunctions, and they have also been associated with certain types of cancers [56,57,58,59].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

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

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)

However, only the following congeners were detected, and ranges of mean concentrations are shown based on wet weight and lipid weight:

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PCDDs and PCDFs	Wet weight (pg/g serum)	Lipid weight (pg/g lipid)
1,2,3,6,7,8- hexachlorodibenzo-p-dioxin (HxCDD)	0.02 to 0.10	2.8 to 23
1,2,3,4,6,7,8- heptachlorodibenzo-p-dioxin (HpCDD)	0.03 to 0.27	5.5 to 55
1,2,3,4,6,7,8,9- octachlorodibenzo-p-dioxin (OCDD)	0.05 to 1.6	5.3 to 280
1,2,3,7,8-pentachlorodibenzofuran (PCDF)	0.01 to 0.08	2.4 to 15
2,3,4,7,8-pentachlorodibenzofuran (PCDF)	0.01 to 0.03	1.8 to 16
1,2,3,4,7,8- hexachlorodibenzofuran (HxCDF)	0.01 to 0.09	1.8 to 12
1,2,3,6,7,8- hexachlorodibenzofuran (HxCDF)	0.01 to 0.03	1.4 to 16
1,2,3,4,6,7,8- heptachlorodibenzofuran (HpCDF)	0.02 to 0.08	2.7 to 24
1,2,3,4,6,7,8,9- octachlorodibenzofuran (OCDF)	0.01 to 0.08	3.2 to 17

Concentrations (lipid adjusted and whole serum) depended on age and geographic regions as shown in Figures 3 – 20. For 1,2,3,6,7,8-HxCDD (in both lipid adjusted and whole serum, Figures 3 and 4), in general the concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For 1,2,3,4,6,7,8- HpCDD (only for lipid adjusted, Figure 5), the concentrations in the north were equal to those in the central region, and those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were lower than those in the 31+ age group, but neither differed from those in 26-30 year age group. For OCDD (only for lipid adjusted, Figure 7), the concentrations in the north were equal to those in the central region, and in turn those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn equal to those in 31+ age group. For 1,2,3,7,8-PCDF and 2,3,4,7,8-PCDF (both for lipid adjusted and whole serum, Figures 9 to 12), the concentrations in the north were equal to those in the central region, and in turn those in the central region were lower than those in the south. For 1,2,3,4,7,8-HxCDF and 1,2,3,6,7,8-HxCDF (only for lipid adjusted, Figures 13 and 15, respectively), the concentrations in the north were equal to those in the central region, and in turn those in the central region were lower than those in the south. For 1,2,3,4,6,7,8-HpCDF and OCDF (in both lipid adjusted and whole serum Figures 17 to 20), there were no differences in concentrations apparent between regions or across age groups. The seasonal variation of dioxin and furan concentrations was examined in the south, but there was no apparent temporal or seasonal trend observed.

In the long-term follow-up health assessment program (1997-2002) at Swan Hills Waste Treatment Centre, the mean concentrations of 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, OCDD, 2,3,4,7,8-PCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDF and OCDF in blood were 743, 390, 3371, 34, 33, 46, 142 and 48 pg/g lipid, respectively, in 2001 [60]. In the U.S. National Health and Nutrition Survey (NHANES

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2001-2002) (Third Report, CDC), the geometric mean of 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,6,7,8,9–OCDD and 1,2,3,4,6,7,8-HpCDF concentrations (in both lipid adjusted and whole serum) in females were higher than in the present study (as shown in the Figures); and for 1,2,3,7,8-PCDF, 2,3,4,7,8-PCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF and 1,2,3,4,6,7,8,9-OCDF, the geometric mean of their serum concentrations were not calculated because most samples were below the limits of detection of 5.8, 5.5, 6.5, 6.1 and 21 pg/g of lipid, respectively [27]. The 50th centiles of 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,6,7,8,9–OCDD (lipid adjusted) in females were higher than in the present study, but for the rest of the analytes the 50th centiles were similar to those in the present study.

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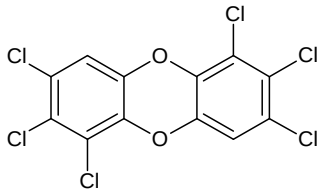

1,2,3,6,7,8 HxCDD

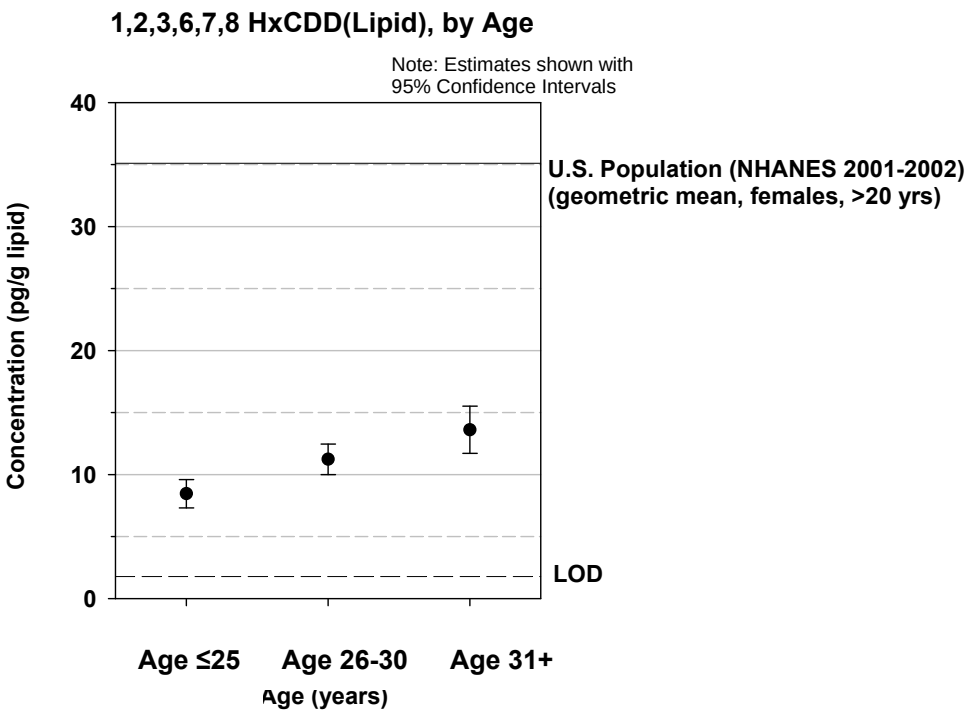

Figure 3

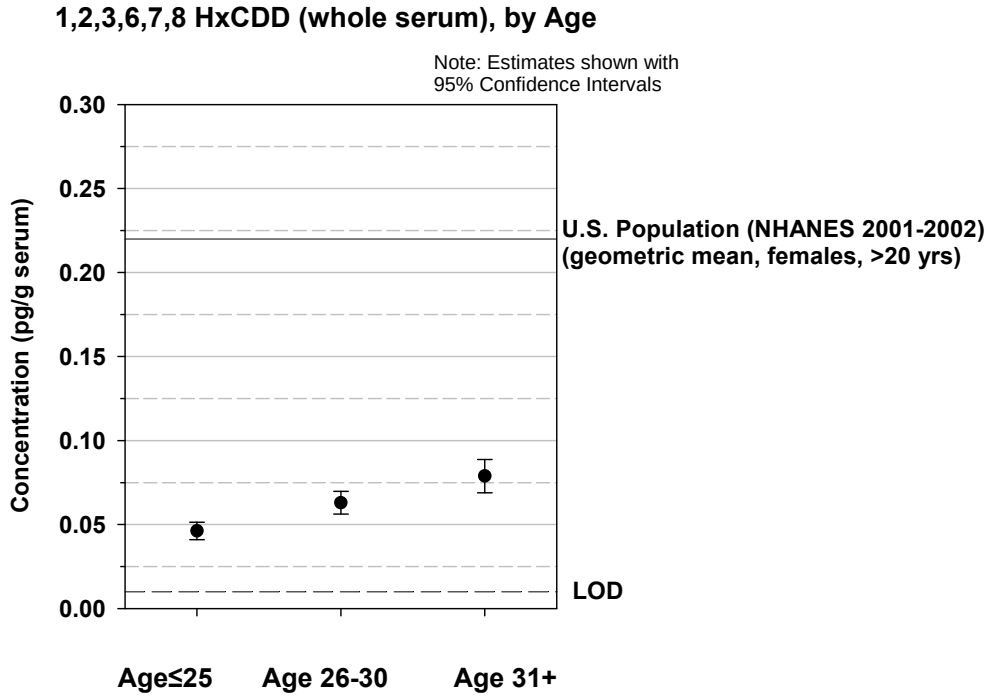

Figure 4

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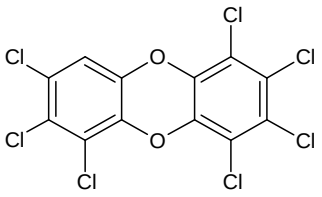

1,2,3,4,6,7,8 HpCDD

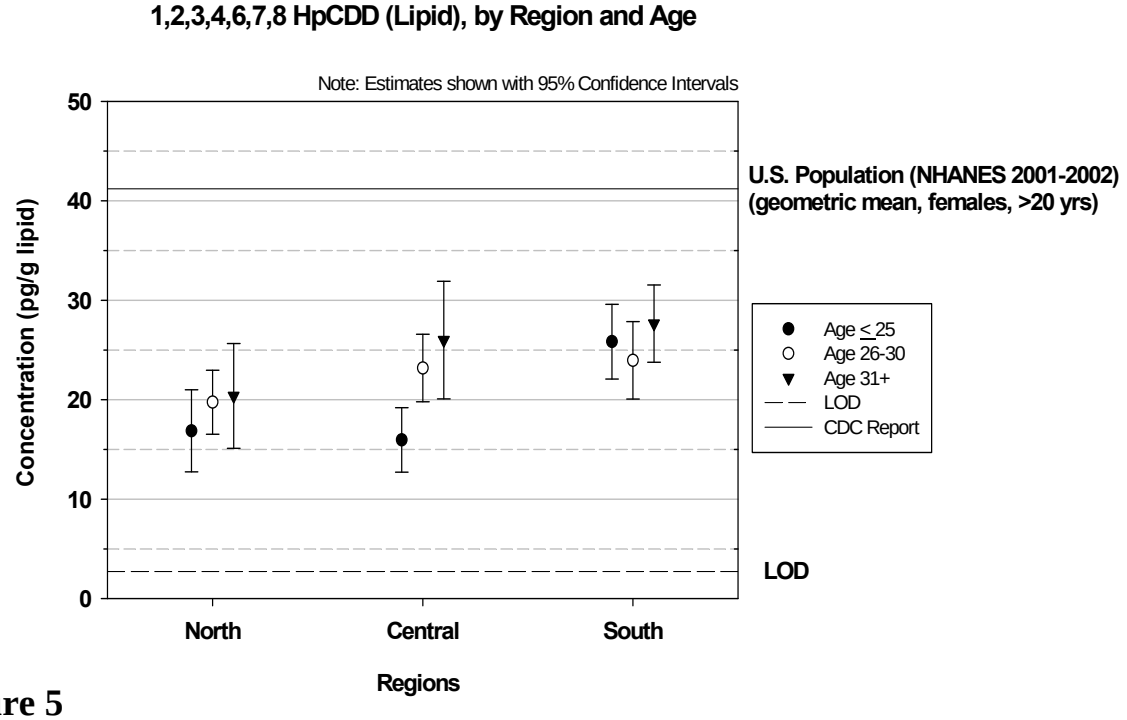

Figure 5

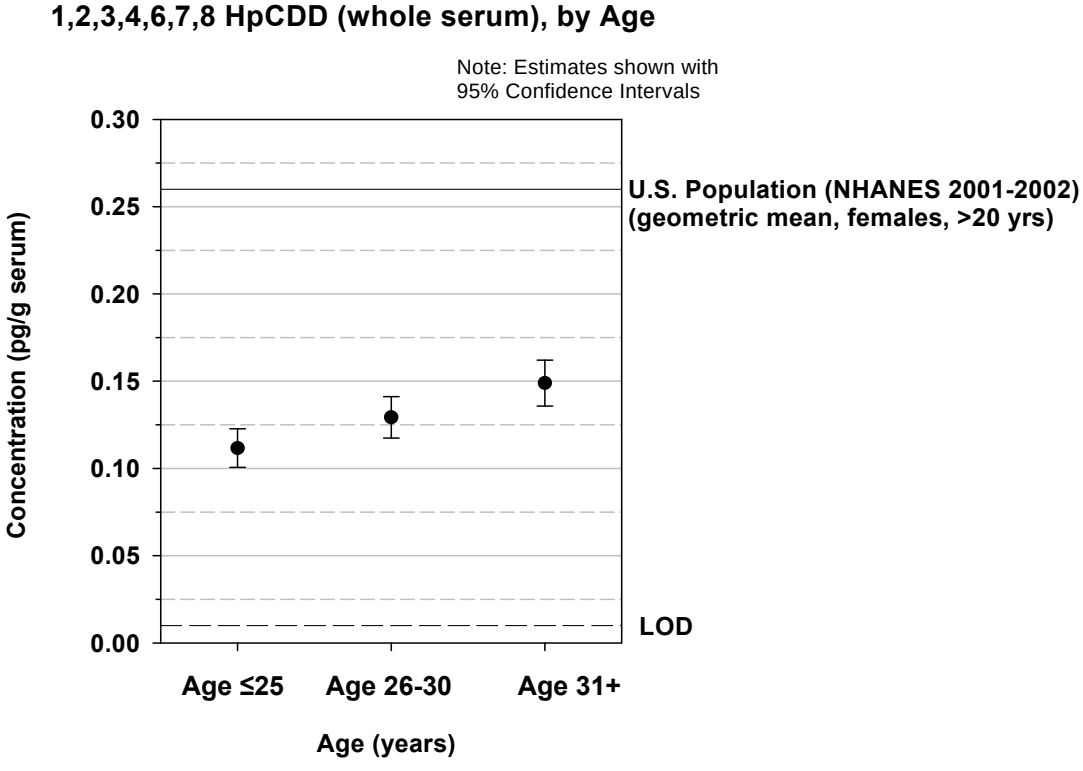

Figure 6

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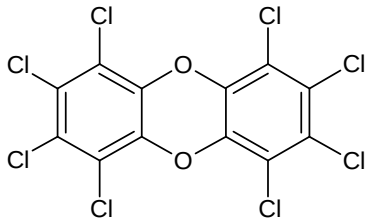

OCDD

OCDD (Lipid), by Region and Age

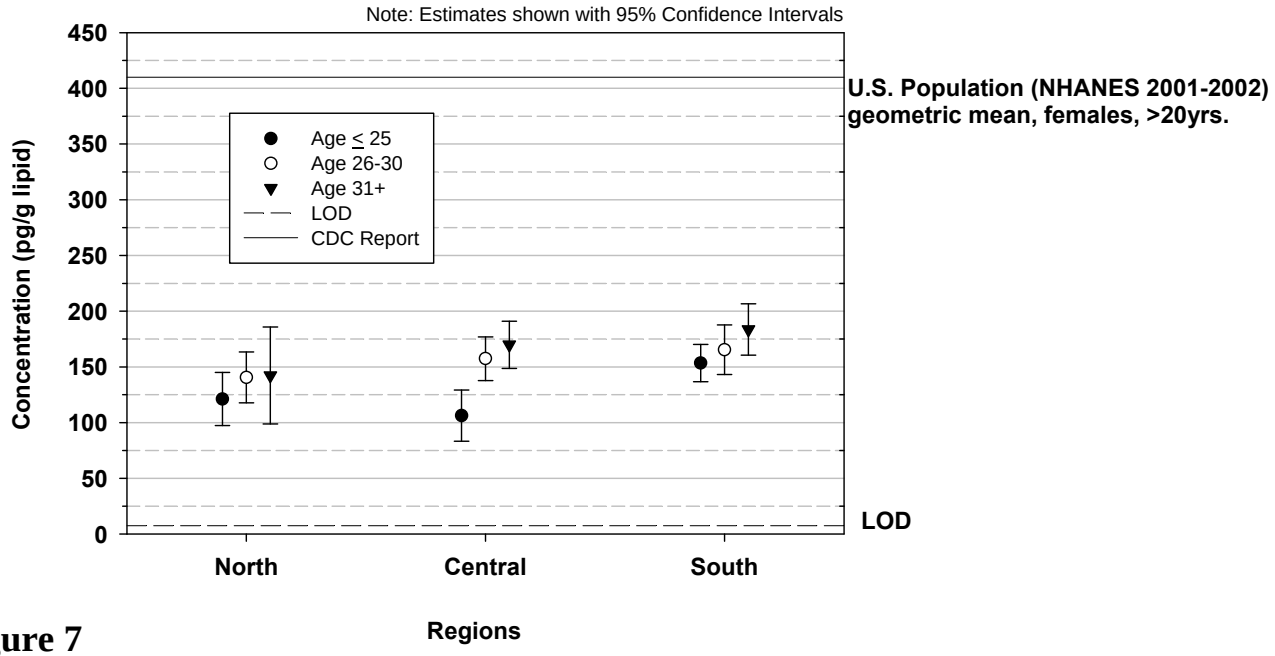

Figure 7

OCDD (whole serum), by Age

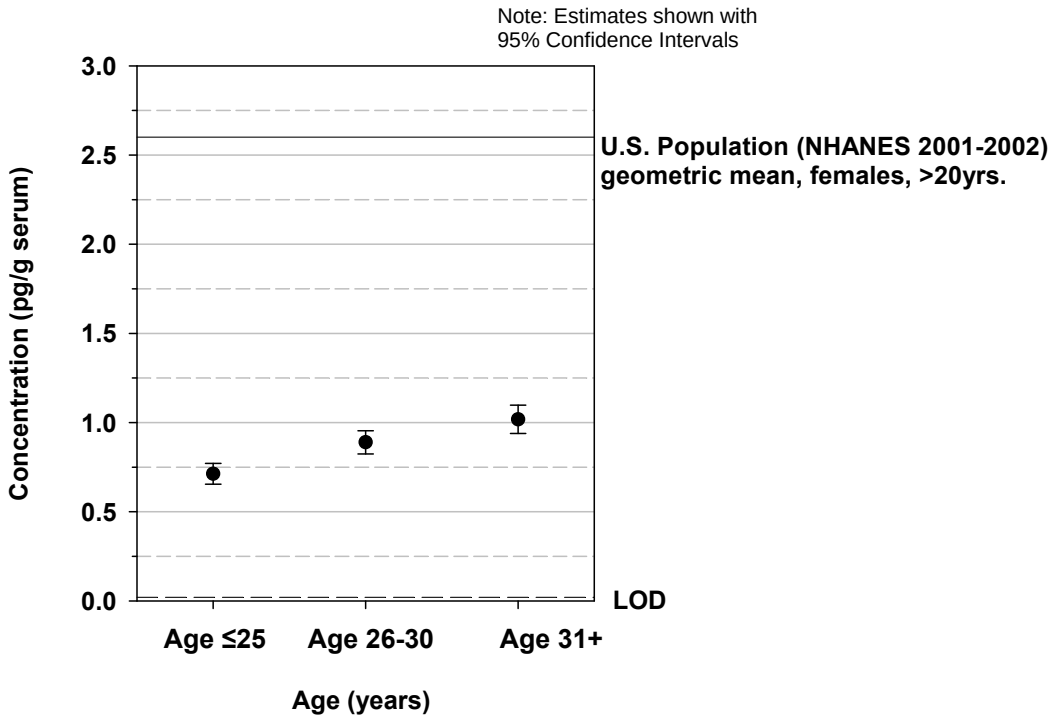

Figure 8

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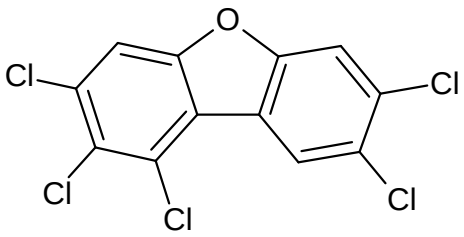

1,2,3,7,8 PeCDF

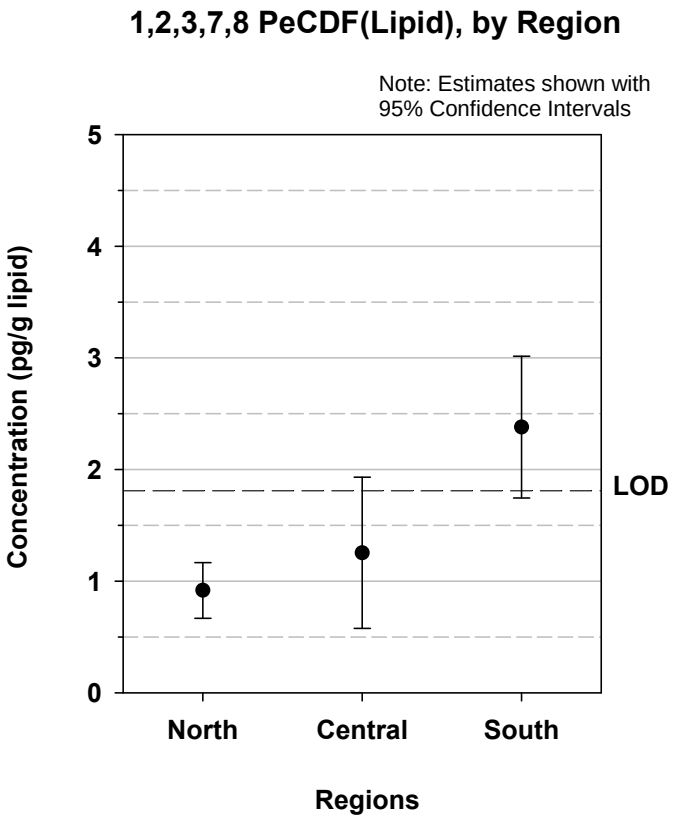

Figure 9

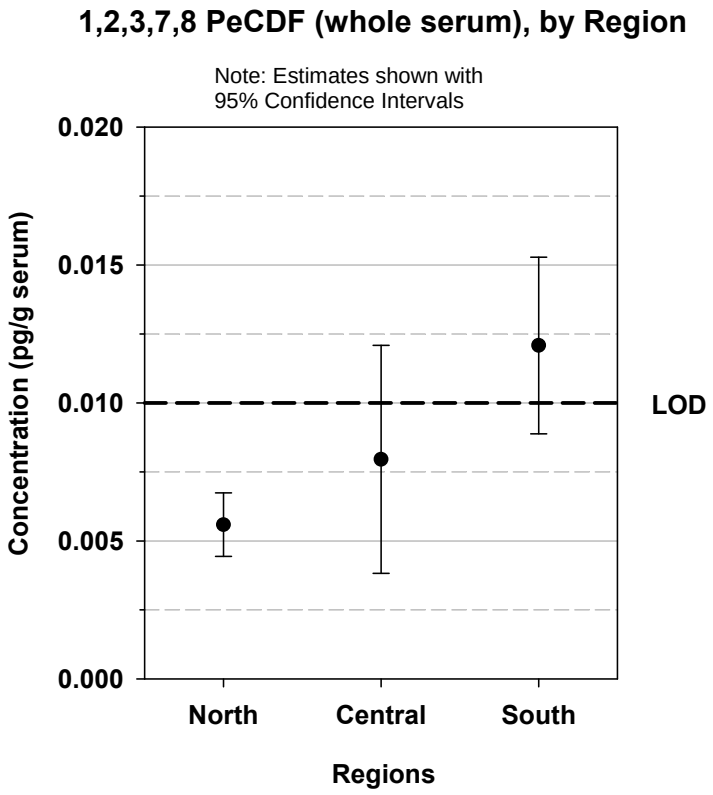

Figure 10

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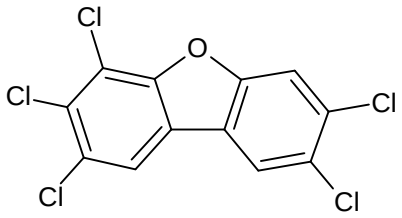
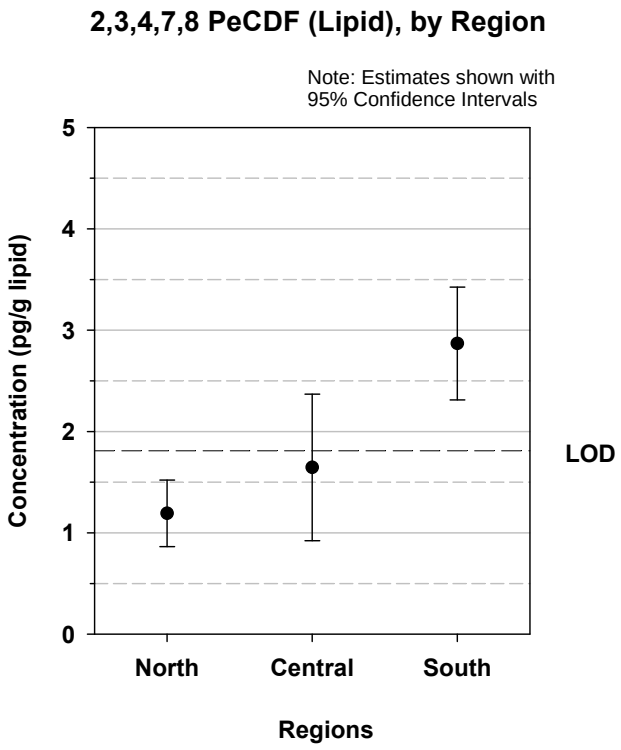


2,3,4,7,8 PeCDF



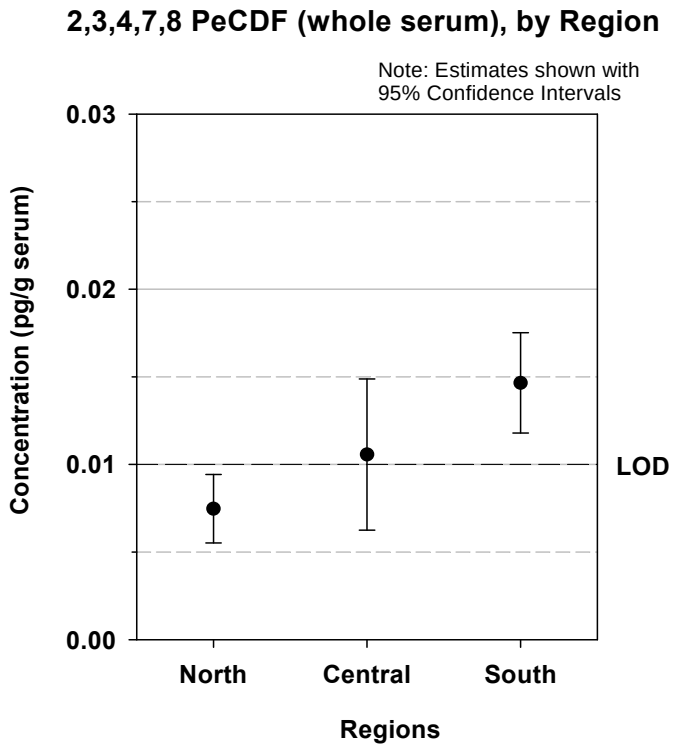


Figure 11

Figure 12

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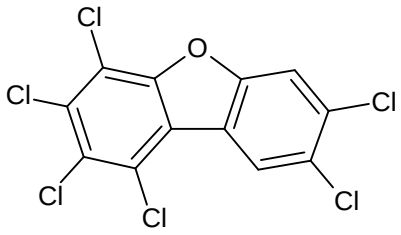

1,2,3,4,7,8 HxCDF

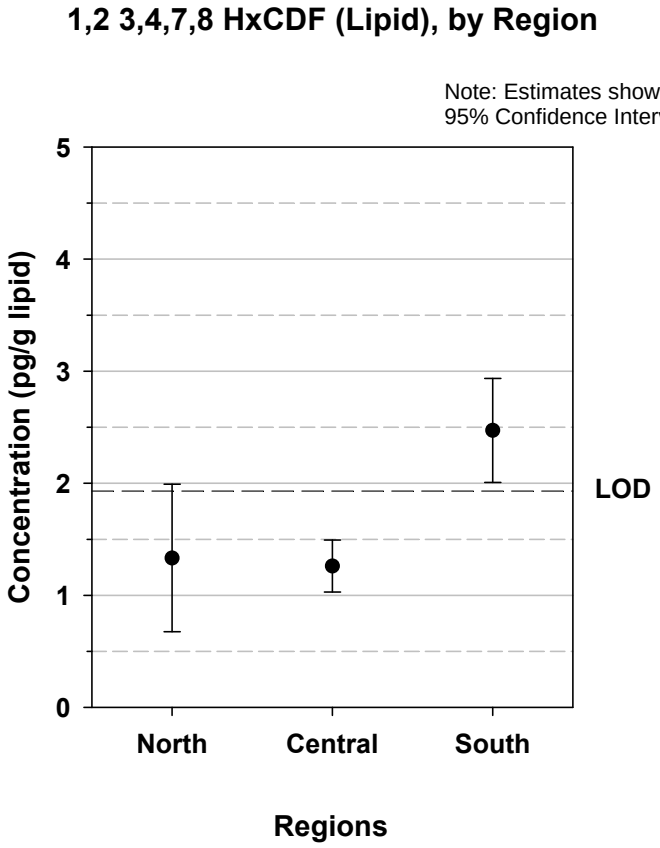

Figure 13

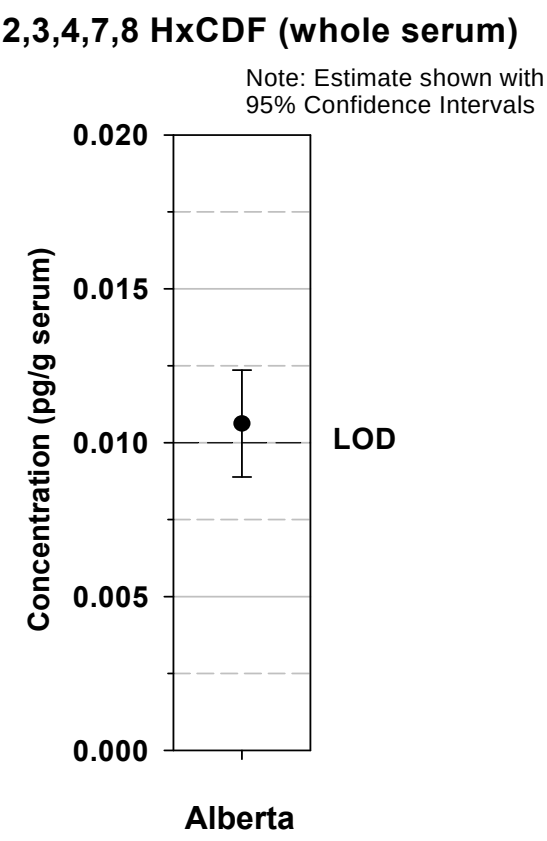

Figure 14

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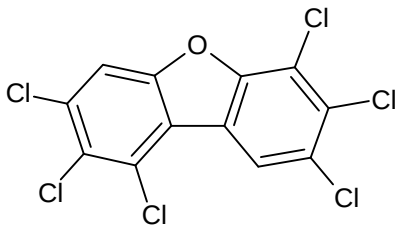
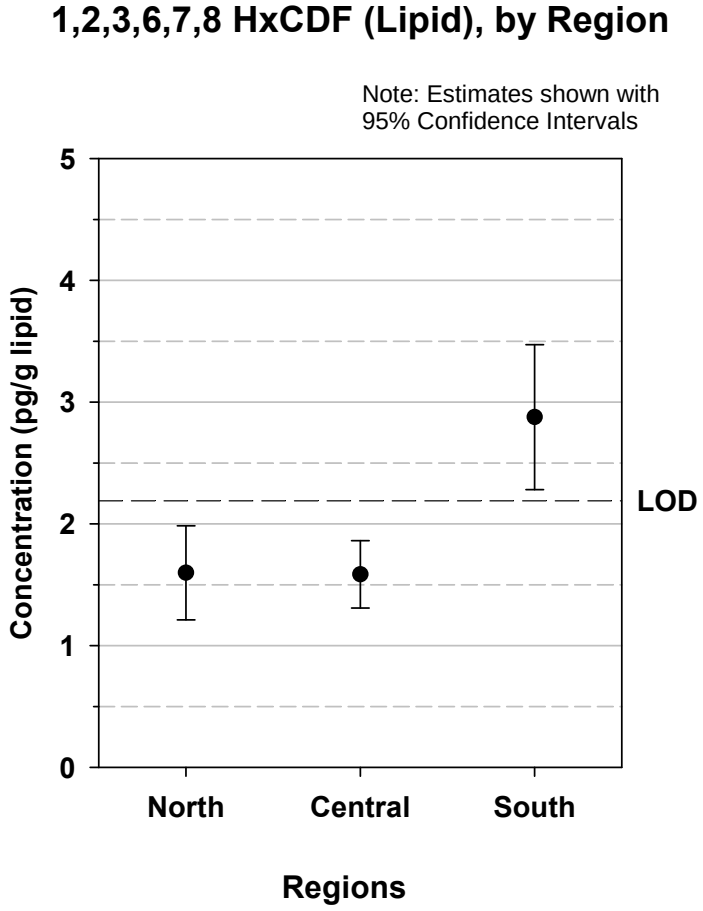


1,2,3,6,7,8 HxCDF



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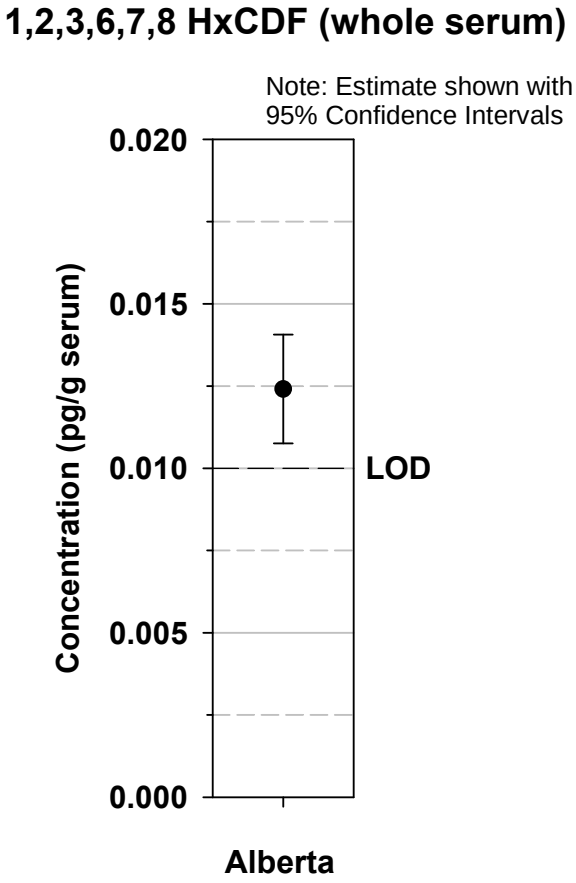

Figure 16

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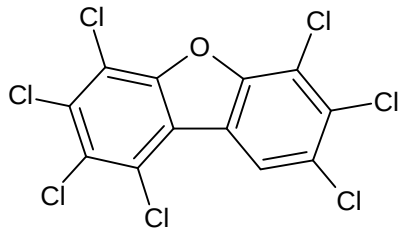

1,2,3,4,6,7,8 HpCDF

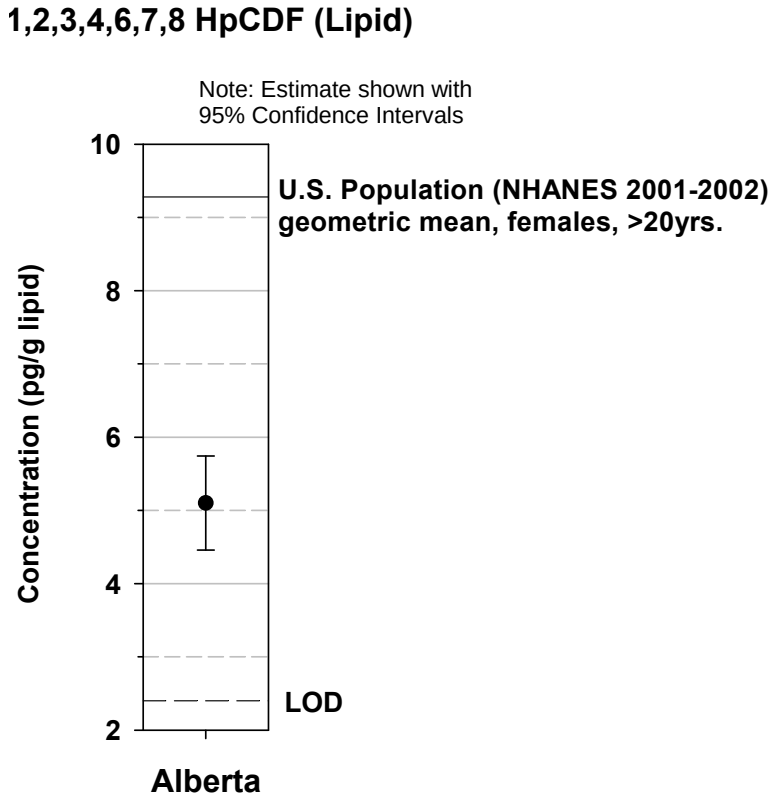

Figure 17

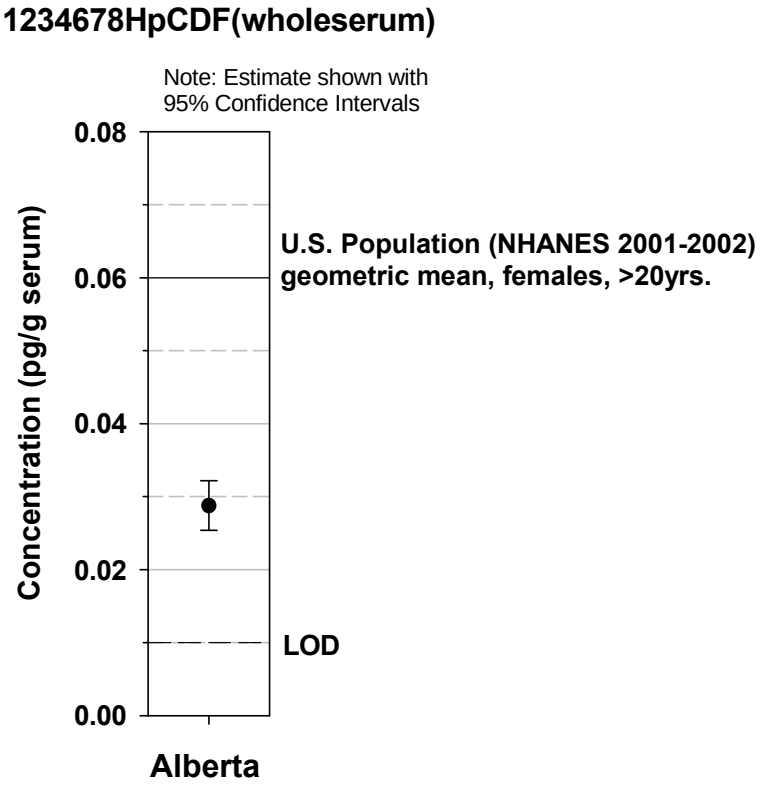

Figure 18

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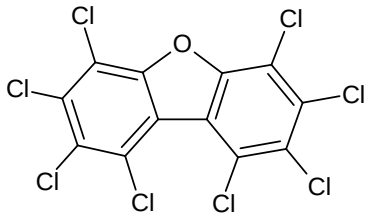

OCDF

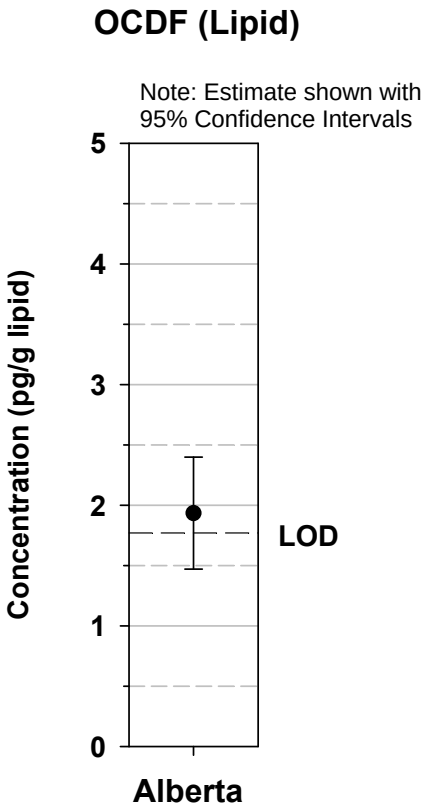

Figure 19

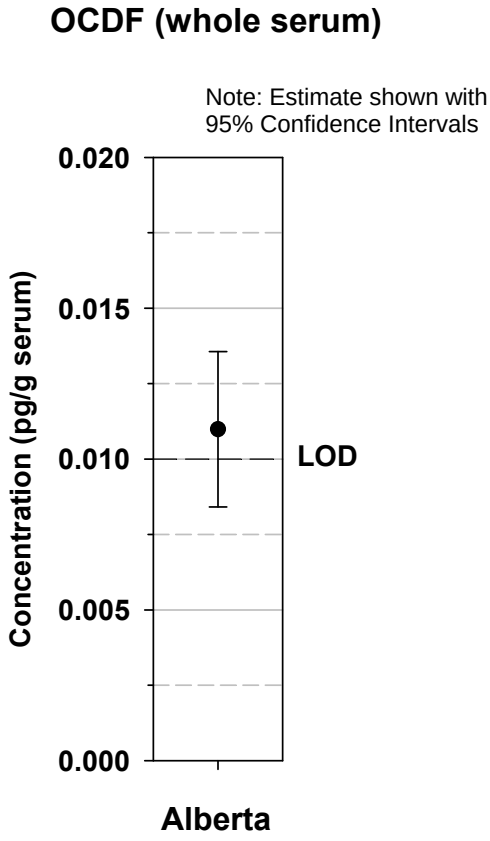

Figure 20

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PCBs

GENERAL INFORMATION

Sources

Polychlorinated biphenyls (PCBs) do not occur naturally and were historically produced commercially in large quantities as mixtures of 209 possible chlorinated congeners. They were used for many decades in a wide variety of industrial and commercial materials such as coolants and lubricants in transformers, capacitors and other electrical equipment, in sealing and caulking compounds, cutting oils, inks and paint additives [61,62]. PCBs were released to the environment (air, water and soil) during their manufacture, use, and disposal. Although PCB manufacturing no longer occurs, PCBs can still be detected in the environment because they are resistant to degradation and can persist in the environment for long periods of time. PCBs can also travel long distances in air and water, and so are detectable all over the world, including in remote regions far from their points of use.

People may be exposed to PCBs in old homes or buildings constructed with PCB-laden materials, or where old electrical devices and PCB containing transformers may be found. However, the most important source of PCB exposure in the general population is through the diet. The main dietary sources of PCBs include animal fats from fish, meat and dairy products [63]. This is because PCBs are present at low concentrations in the oceans, freshwater, and most pasture and agricultural soils around the world. Due to their lipophilic nature, PCBs can accumulate in fatty tissues of animals, and also in human breast milk [64]. In fact, human milk is a major source of PCBs to infants [64,65]. PCBs can also cross the placenta, therefore some level of PCB exposure to the fetus and infants during pregnancy and lactation, respectively, is to be expected [64]. However, breastfeeding is encouraged due to the many associated health-benefits [54] that currently outweigh known risks.

Regulations in Canada

Since 1977, the Canadian Government has adopted several regulations to minimize exposure and environmental releases of PCBs [66]. PCBs were never manufactured in Canada, but they were widely used here. In 1977, the production, processing, import and sale of PCBs, and equipment containing a liquid with a PCB concentration greater than 50 mg/kg, were restricted in Canada. The storage, export and import of equipment and other products containing a PCB concentration of 50 mg/kg or more were also controlled by several other regulations as they were adopted in 1988, 1996 and 2005, respectively [66]. 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 [66]. The continued use of certain equipment containing PCBs is still allowed in Canada, but according to the Stockholm Convention on Persistent Organic Pollutants (POPs), Canada is required to phase out the remaining uses of PCBs by 2025, and to dispose of these PCBs properly by 2028 [66].

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Possible Health Effects

The human health effects of PCBs depend on the dose, the length and timing of exposure, and other factors. It has long been recognized that background human populations are exposed to very low concentrations of PCBs in foods and the environment. At these low levels, PCBs are not known to cause any adverse health effect. At high doses, such as in the case of accidental releases, unusual occupational exposures, or accidental consumption of highly contaminated food, PCBs may cause adverse health effects such as chloracne (a severe form of acne) and skin rashes, fatigue, headache, cough, unusual skin sores and problems related to nervous systems [63,67,68,69,70]. The health effects of PCBs in children of mothers who were exposed to relatively high levels of PCBs through fish [71,72,73], were examined in a number of studies. These studies concluded that the fetus/infants of pregnant women eating large amounts of PCB-contaminated fish were at higher risk of some health effects. These effects may include low birth-weight, immune system abnormalities, depressed motor skills, and a decrease in short-term memory [68,71,72,73].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

The following polychlorinated biphenyls (PCBs) were measured in the blood serum samples of pregnant women in Alberta:

1. 2,4,4′- Trichlorobiphenyl (PCB 28)
2. 2,2,5,5 - Tetrachlorobiphenyl (PCB 43/52)
3. 2,3′,4,4′- Tetrachlorobiphenyl (PCB 66)
4. 2,4,4′,5- Tetrachlorobiphenyl (PCB 74)
5. 3,3′,4,4′- Tetrachlorobiphenyl (PCB 77)
6. 3,4,4′,5- Tetrachlorobiphenyl (PCB 81)
7. 2,2′,3,4,5′-Pentachlorobiphenyl (PCB 87)
8. 2,2′,4,4′,5-Pentachlorobiphenyl (PCB 99)
9. 2,2′,4,5,5′-Pentachlorobiphenyl (PCB 101)
10. 2,3,3′,4,4′-Pentachlorobiphenyl (PCB 105)
11. 2,3,3′,4′,6-Pentachlorobiphenyl (PCB 110)
12. 2,3′,4,4′,5-Pentachlorobiphenyl (PCB 118)
13. 3,3′,4,4′,5-Pentachlorobiphenyl (PCB 126)
14. 2,2′,3,3′,4,4′-Hexachlorobiphenyl (PCB 128)
15. 2,2′,3,4,4′,5-Hexachlorobiphenyl (PCB 138)
16. 2,2′,3,4′,5,5′-Hexachlorobiphenyl (PCB 146)
17. 2,2′,3,4′,5′,6-Hexachlorobiphenyl (PCB 149)
18. 2,2′,3,5,5′,6-Hexachlorobiphenyl (PCB 151)
19. 2,2′,4,4′,5,5′-Hexachlorobiphenyl (PCB 153)
20. 2,3,3′,4,4′,5-Hexachlorobiphenyl (PCB 156)
21. 2,3,3′,4,4′,5′-Hexachlorobiphenyl (PCB 157)
22. 2,3,3′,4,4′,6-Hexachlorobiphenyl (PCB 158)
23. 2,3′,4,4′,5,5′-Hexachlorobiphenyl (PCB 167)

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24. 3,3′,4,4′,5,5′-Hexachlorobiphenyl (PCB 169)
25. 2,2′,3,3′,4,4′,5-Heptachlorobiphenyl (PCB 170)
26. 2,2′,3,3′,4,5,5′-Heptachlorobiphenyl (PCB 172)
27. 2,2′,3,3′,4,5′,6′-Heptachlorobiphenyl (PCB 177)
28. 2,2′,3,3′,5,5′,6-Heptachlorobiphenyl (PCB 178)
29. 2,2′,3,4,4′,5,5′-Heptachlorobiphenyl (PCB 180)
30. 2,2′,3,4,4′,5′,6-Heptachlorobiphenyl (PCB 183)
31. 2,2′,3,4′,5,5′,6-Heptachlorobiphenyl (PCB 187)
32. 2,3,3′,4,4′,5,5′-Heptachlorobiphenyl (PCB 189)
33. 2,2′,3,3′,4,4′,5,5′-Octachlorobiphenyl (PCB 194)
34. 2,2′,3,3′,4,4′,5,6-Octachlorobiphenyl (PCB 195)
35. 2,2′,3,3′,4,4′,5,6′-Octachlorobiphenyl (PCB 196)
36. 2,2′,3,3′,4,5,5′,6′-Octachlorobiphenyl (PCB 199)
37. 2,2′,3,4,4′,5,5′,6-Octachlorobiphenyl (PCB 203)
38. 2,2′,3,3′,4,4′,5,5′,6-Nonachlorobiphenyl (PCB 206)
39. 2,2′,3,3′,4,4′,5,5′,6,6′-Decachlorobiphenyl (PCB 209)

However, only the following congeners were detected, and ranges of mean concentrations are shown based on wet weight and lipid weight:

PCBs	Whole weight (pg/g serum)	Lipid weight (ng/g lipid)
2,2′,3,4′,5,5′-Hexachlorobiphenyl (PCB 146)	5.1 to 13	1.0 to 2.0
2,3,3′,4,4′,5-Hexachlorobiphenyl (PCB 156)	5.1 to 13	1.0 to 2.2
2,3,3′,4,4′,6-Hexachlorobiphenyl (PCB 158)	5.4 to 55	0.9to 9.1
2,2′,3,3′,4,4′,5-Heptachlorobiphenyl (PCB 170)	5.1 to 21	1.0 to 4.2
2,2′,3,4,4′,5,5′-Heptachlorobiphenyl (PCB 180)	6.3 to 67	1.3 to 13
2,2′,3,4,4′,5′,6-Heptachlorobiphenyl (PCB 183)	5.1 to 44	5.6 to 8.8
2,2′,3,4′,5,5′,6-Heptachlorobiphenyl (PCB 187)	5.1 to 62	1.0 to 22
2,2′,3,3′,4,4′,5,5′-Octachlorobiphenyl (PCB 194)	5.3 to 11	1.0 to 2.2
2,2′,3,3′,4,5,5′,6′-Octachlorobiphenyl (PCB 199)	5.1 to 11	0.9 to 2.2

Concentrations (whole serum and lipid adjusted) depended on age and geographic regions as shown in Figures 21-35. For PCB 146 (lipid adjusted, Figure 21), the concentrations in the north were lower than those in the central region and in turn those in the central region were equal to those in the south. The concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For PCB 156 (lipid adjusted, Figure 23), the concentrations in the north were equal to those in the central region and in turn those in the central region

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were lower than those in the south. In general, the concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group. However, the difference between the ages was most pronounced in the south. For PCB 170 (lipid adjusted, Figure 25), the concentrations in the north were equal to those in the central region and in turn those in the central region were lower than those in the south. In general, the concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. However, the regional gradient was most apparent at the oldest age. For PCB 180 (lipid adjusted, Figure 27), the concentrations in the north were lower than those in the central region and in turn those in the central region were lower than those in the south. In general, the concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. However, the differences between the ages increased from north to central to south. For PCB 183 and PCB 187 (lipid adjusted, Figures 29 and 31, respectively), the concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For PCB 194 (lipid adjusted, Figure 33), the concentrations in the north were equal to those in the central region and in turn those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For PCB 199 (lipid adjusted, Figure 34), the concentrations in the north were equal to those in the central region and in turn those in the central region were lower than those in the south. In general, the concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group (not detected in ages ≤25, and in most samples ages 26-30). However, the differences between the ages increased in the south. The seasonal variation of PCB concentrations was examined in the south, but there was no apparent temporal or seasonal trend observed.

In a recent study, the geometric mean of total PCB concentrations in the serum of pregnant women from Southern Alberta (Calgary Health Region, 2001-2003) was reported as 62 ng/g lipid [74]. In another study of a long-term follow-up health assessment program (1997-2002) at Swan Hills Waste Treatment Centre, the mean concentrations of PCB 156, 170, 180, 183, 187 and 194 were 0.03, 0.05, 0.13, 0.01, 0.04 and 0.02 ng/g serum, respectively, in 2001 [60]. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), the geometric mean serum concentration of PCB 180 in females was 17.5 ng/g lipid (as shown in Figure 27), and the geometric means of serum concentrations (both for lipid adjusted and whole weight) of PCB 146, PCB 156, PCB 158, PCB 170, PCB 183, PCB 187, PCB 194 and PCB 199 in the U.S. population were not calculated because most of the samples were below the limit of detection of 10.5 ng/g lipid [27]. The 50th centiles of PCB congener concentrations were either higher or similar in concentration to the present study.

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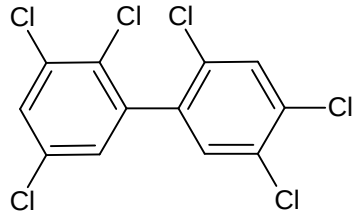

PCB 146

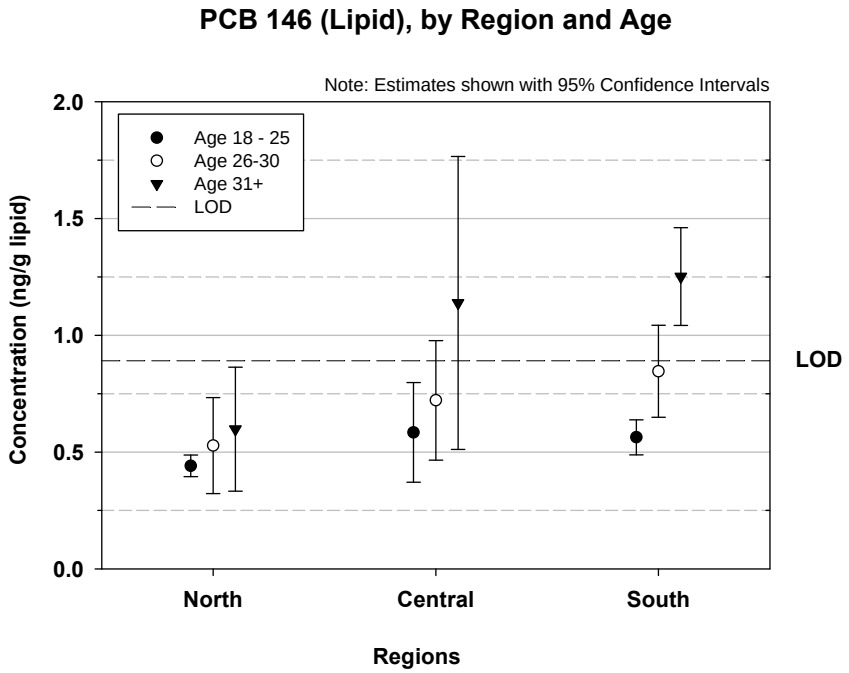

Figure 21

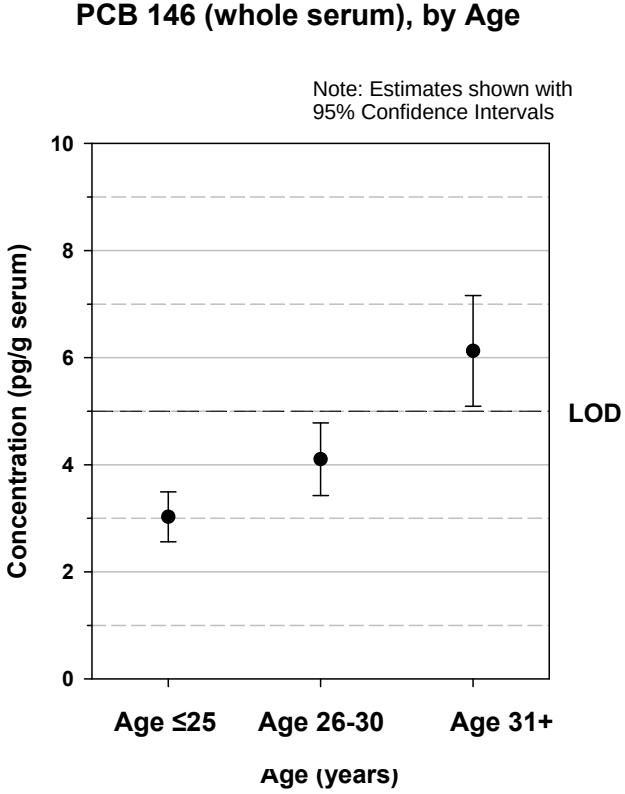

Figure 22

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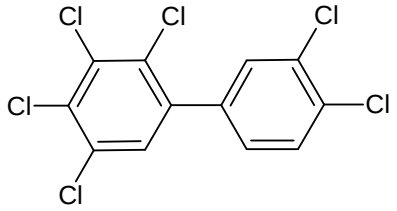

PCB 156

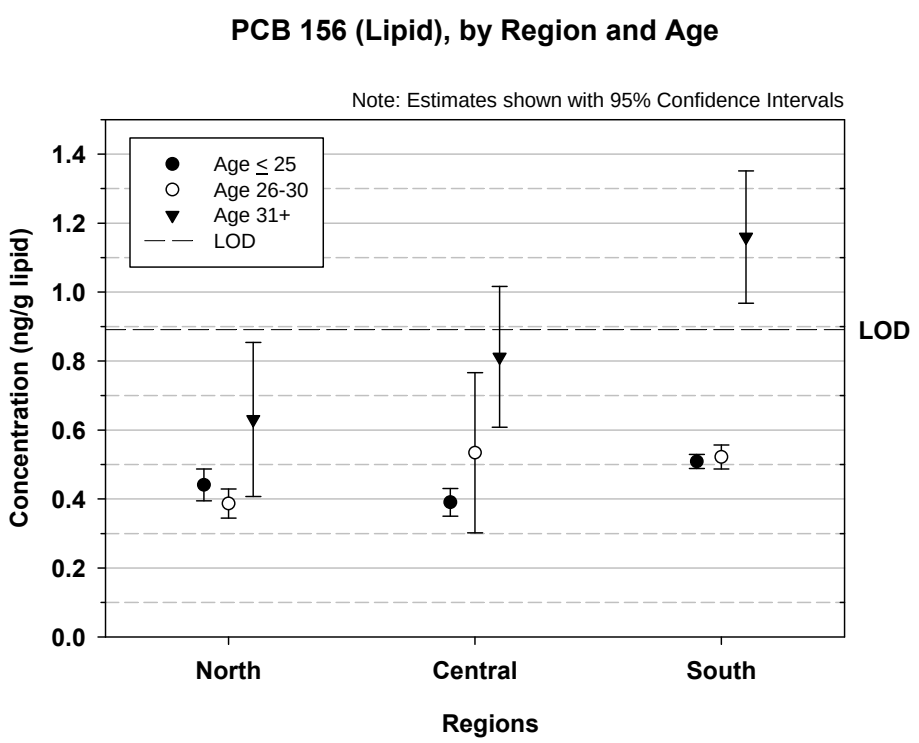

Figure 23

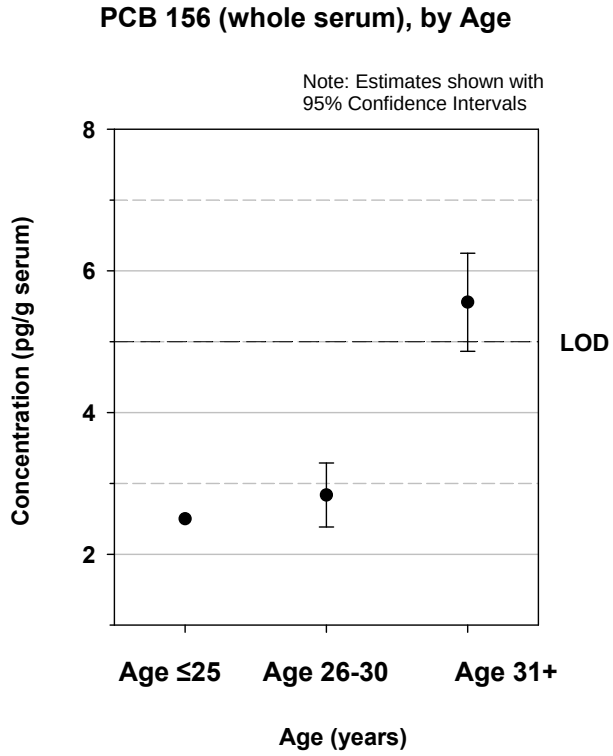

Figure 24

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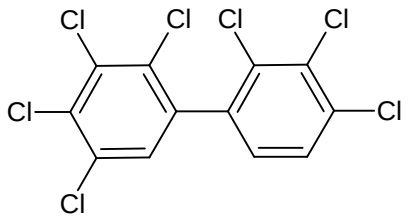

PCB 170

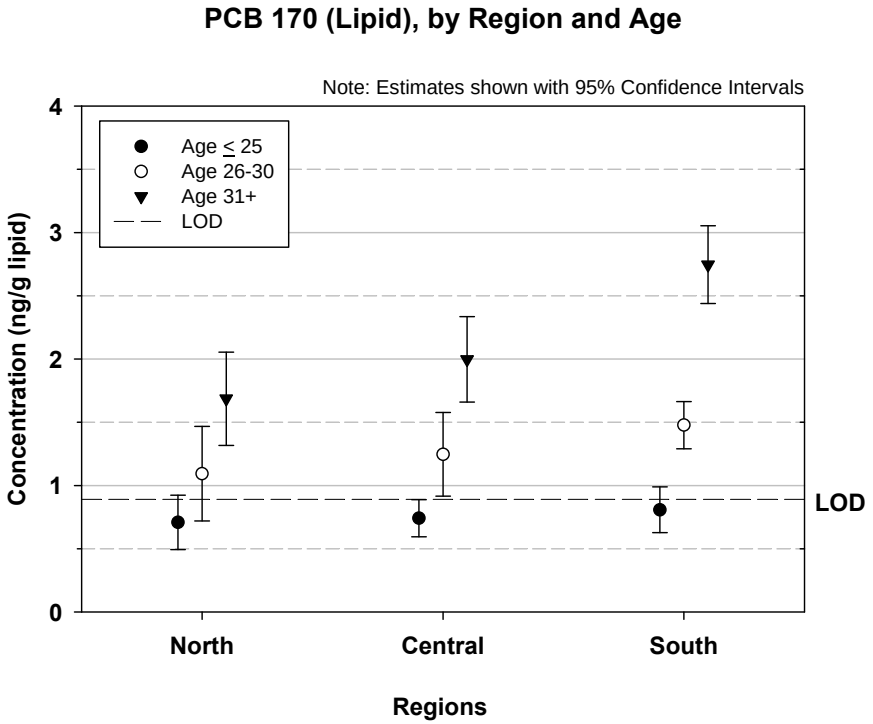

Figure 25

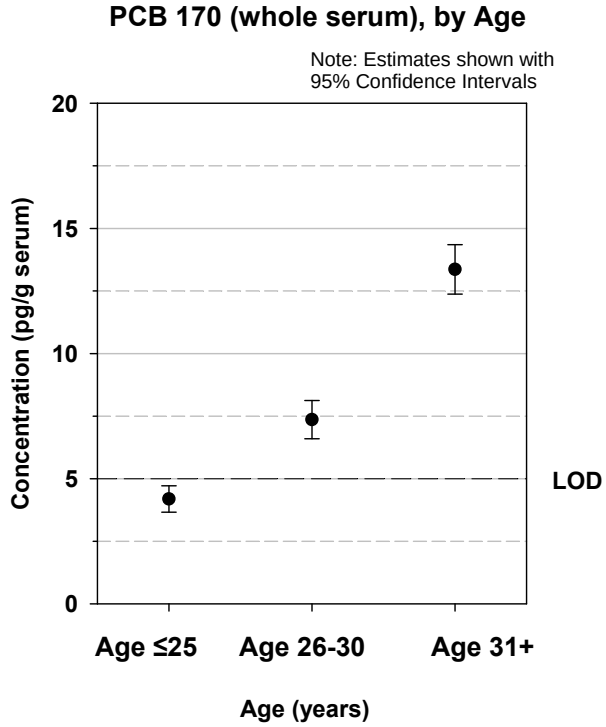

Figure 26

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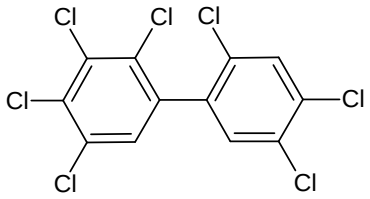

PCB 180

PCB 180 (Lipid), by Region and Age

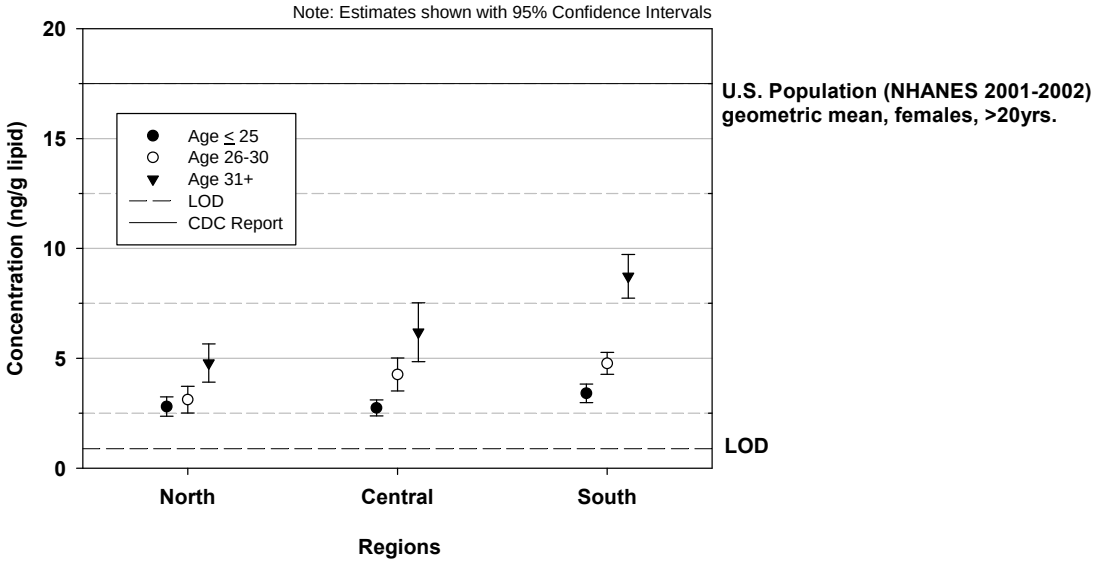

Figure 27

PCB 180 (whole serum), by Age

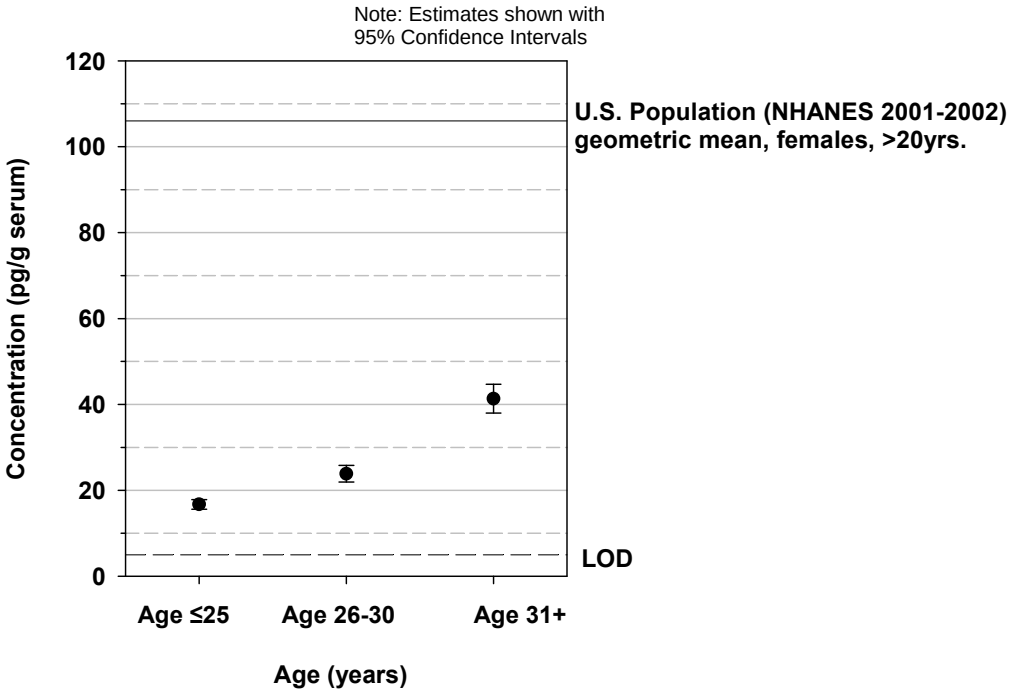

Figure 28

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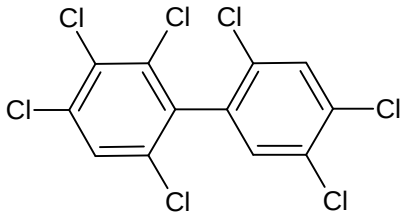

PCB 183

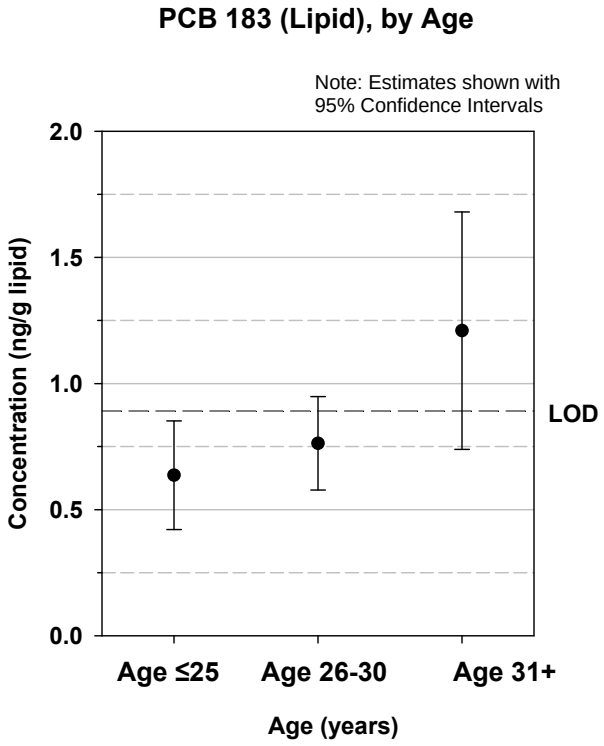

Figure 29

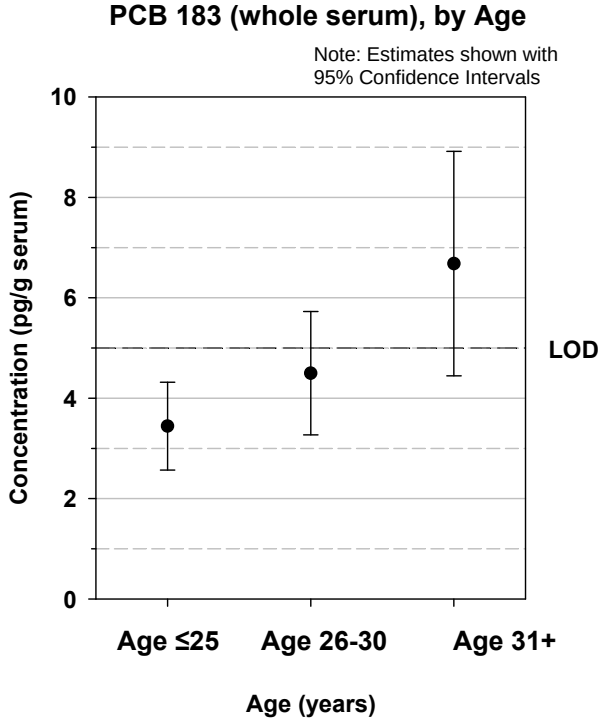

Figure 30

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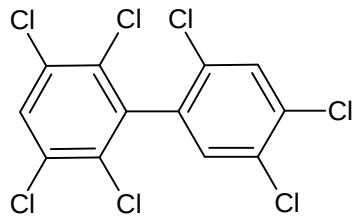

PCB 187

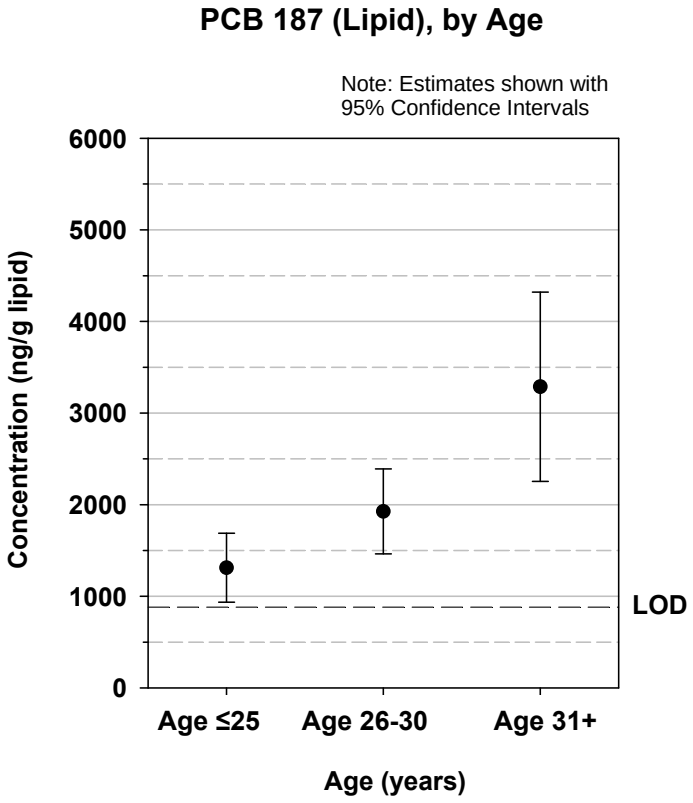

Figure 31

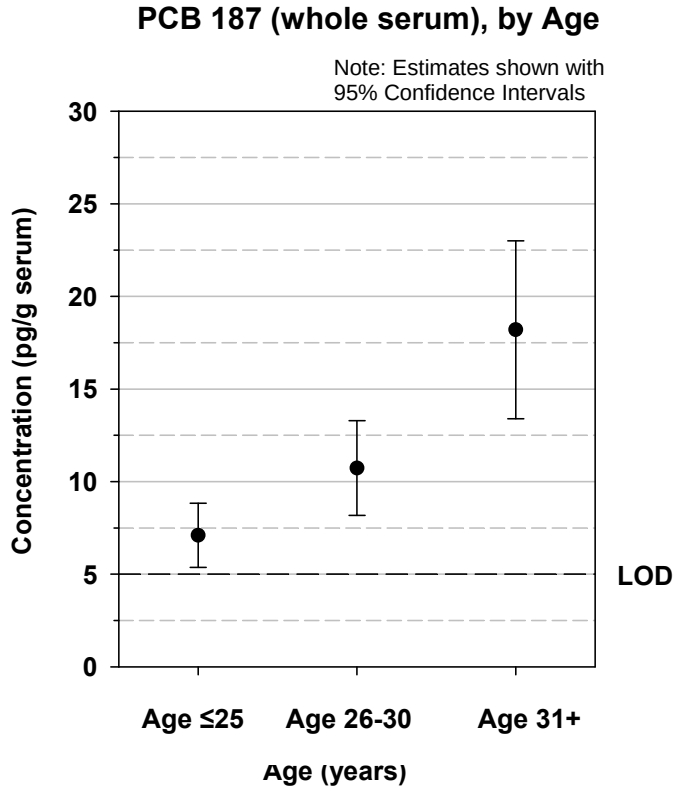

Figure32

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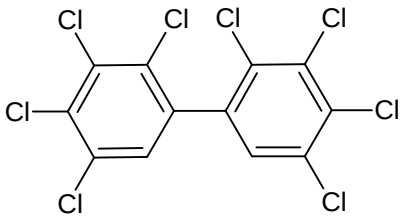

PCB 194

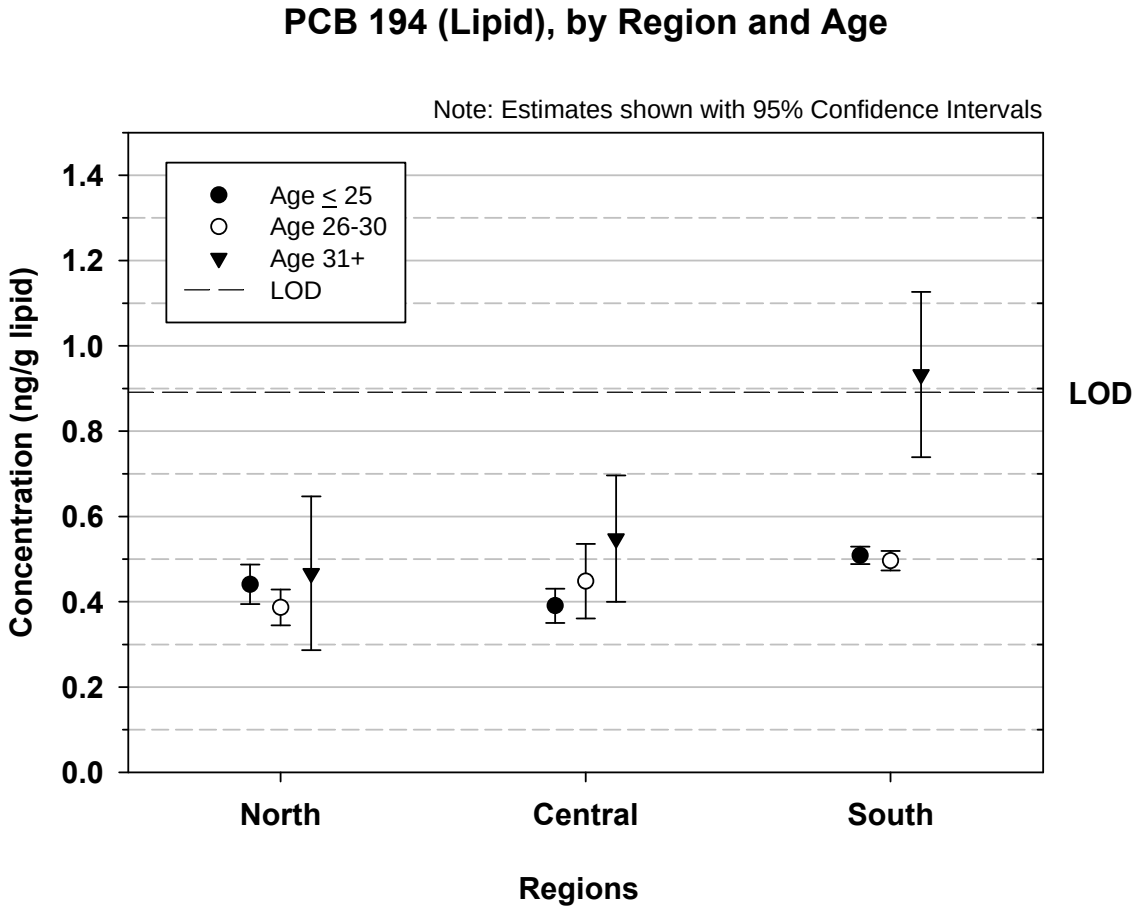

Figure 33

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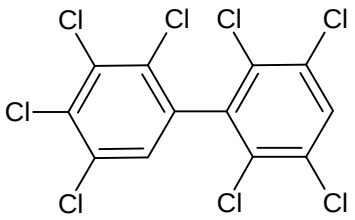

PCB 199

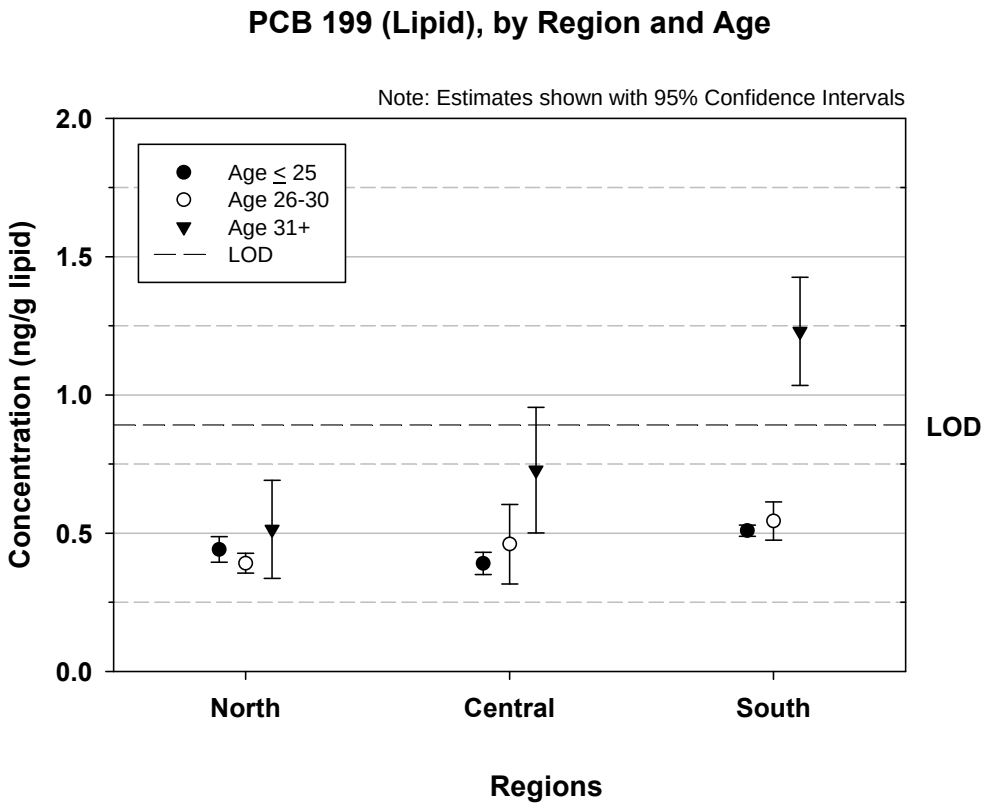

Figure 34

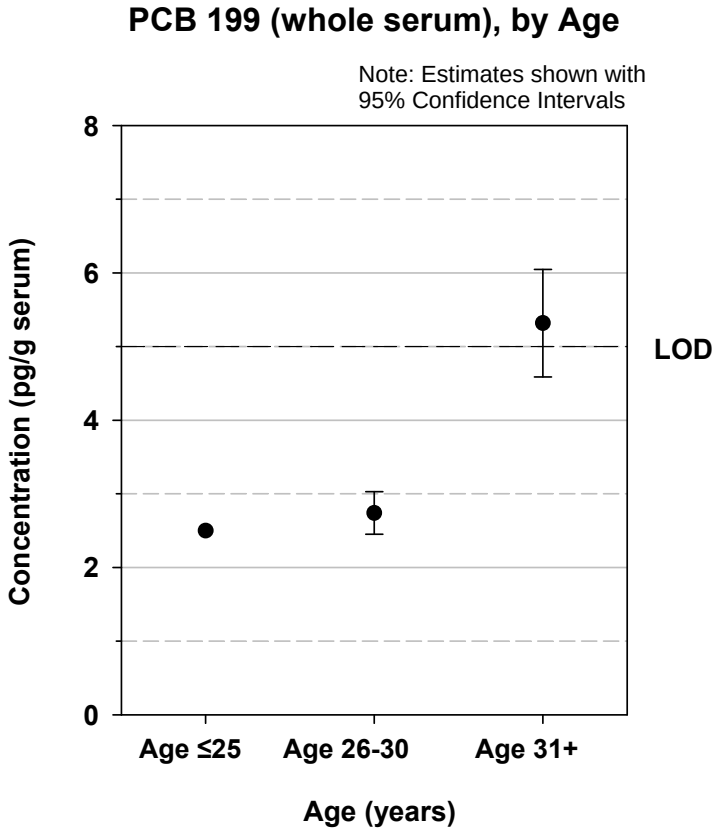

Figure 35

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Organochlorine Pesticides

GENERAL INFORMATION

Organochlorine (OC) pesticides are synthetic chlorinated hydrocarbon compounds that were once widely used as insecticides and fungicides before their extreme environmental persistence, long-range transport, and high bioaccumulation potential were recognized. While these chemicals were generally quite effective for their intended purpose, eventual awareness of these chemicals at high concentration in humans and wildlife, and eventual recognition of their potential to cause adverse health effects, led to much public awareness in the 1960s. The growing environmental and human heath-related concerns caused many countries to regulate these chemicals in an effort to minimize future environmental and human exposure. While most of these chemicals are also tightly controlled by the international agreement on persistent organic pollutants (POPs, The Stockholm Convention), they persist in the global environment such that they are now referred to as 'legacy pollutants'. Examples of such chemicals include methoxychlor, DDT, hexachlorobenzene, mirex, heptachlor, chlordane, aldrin, dieldrin and endrin.

In general, these chemicals were released into the local environment (air, water and soil) during their manufacture, use and disposal. These chemicals are considered semi-volatile, meaning that these chemicals can partition into the atmosphere and travel long distances on wind currents across international boundaries and into remote regions of the globe. The major source of OC pesticide exposure in the general human population today is through the diet. They enter the human food-chain because the largest fraction of OCs are now stored in the world's soils, and due to their lipophilic (literally 'fat-loving') nature, OCs may accumulate in plants and in the fatty tissues of livestock or other wildlife. Other minor sources of exposure are by absorption, ingestion and inhalation of contaminated water and air. In our bodies, OC pesticides may accumulate in human breast milk, and can also cross the placenta [75,76,77]. In these ways, some level of OC pesticides can be passed to the fetus or to infants during pregnancy and lactation, respectively [77]. However, breastfeeding is encouraged due to the many associated health-benefits [54] that currently outweigh known risks.

In the present study, the following OC pesticides were measured in blood serum samples of pregnant women in Alberta: alpha-, beta-, gamma- and delta-hexachlorocyclohexane (HCH), heptachlor, aldrin, heptachlor epoxide, endosulfan, dieldrin, 4,4′-DDT, 4,4′-DDE, 4,4′-DDD, endrin, methoxychlor, chlordanes, hexachlorobenzene, trans-nonachlor, mirex and octachlorostyrene. However, only mirex, 4,4′-DDE and hexachlorobenzene were detected in the blood serum samples. Detailed information about these detected OCs can be found in the following sections.

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Mirex

GENERAL INFORMATION

Sources

Mirex was used world-wide as an insecticide to control termites, fire ants and harvester ants, and also as a flame-retardant in plastic and rubber products, paints and electrical components [78]. In Canada, mirex was never registered for pesticide applications; however, mirex was used extensively in the south-eastern U.S. and was also manufactured in the U.S. [78]. Thus it entered the Canadian environment in part through air, and also through water and sediments from the Niagara and Oswego Rivers [78,79,80].

Like other OC pesticides, mirex can accumulate in fatty tissues of aquatic organisms and enter the human food chain. Today, dietary intake of fish and shell fish is one of the potential sources of exposure to the general population. Absorption, ingestion and inhalation of water and air containing trace concentrations of mirex are other sources of minor exposure. The human fetus and infants are exposed to mirex during pregnancy, and via breastfeeding, respectively [75,76,81].

Regulations in Canada

Since 1978, all uses of mirex were banned in Canada under the Canadian Environmental Protection Act (CEPA) [82,83]. In 1989, mirex was added to the List of Toxic Substances in Schedule 1 of CEPA [83]. In 1997, the Toxic Substances Management Policy of the Canadian Federal Government decided that mirex should be virtually eliminated, but no new regulations were adopted as the exposure and uses of mirex were already considered to be sufficiently regulated in Canada [82].

Possible Health Effects

Human studies addressing possible health effects of mirex exposure to the background human population are very limited. Animal studies report effects on liver, kidneys, the nervous system, and the reproductive system [84,85].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall mean mirex concentrations in blood serum of pregnant Albertan women ranged from 0.1 to 0.8 ng/g of serum or, expressed on a serum lipid basis, from 22 to 166 ng/g lipid. Concentrations (lipid adjusted and whole serum) depended on age and geographic region as shown in Figures 36-37. There were no differences in concentrations between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. The estimated 50^{th} centile among individuals was 4.2 ng/g lipid. In the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third Report, CDC), the geometric

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mean of serum mirex concentrations for the U.S. populations was not calculated, as serum mirex concentrations were mostly below the limits of detection of 10.5 ng/g of lipid, and the 50th centile was also reported as non-detectable [27].

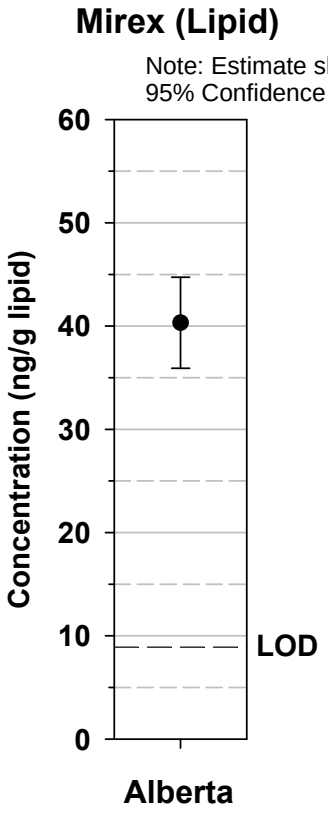

Figure 36

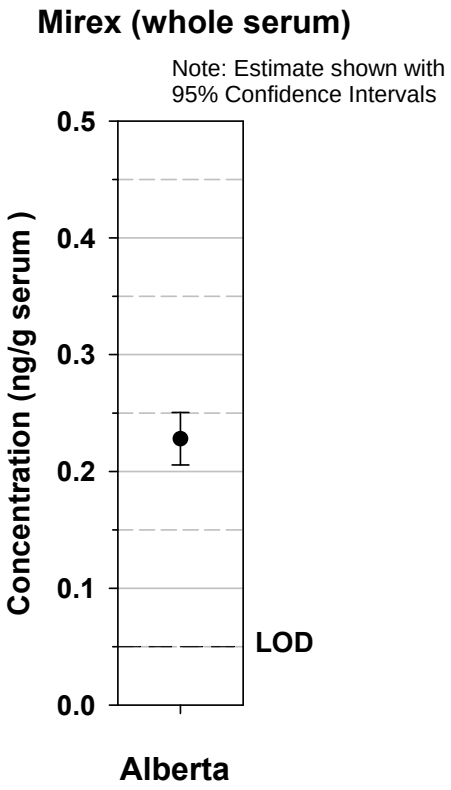

Figure 37

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Dichlorodiphenyldichloroethylene (DDE)

GENERAL INFORMATION

Sources

In the 1940s, dichlorodiphenyltrichloroethane (DDT) was widely applied as an agricultural insecticide and for control of mosquitoes to prevent diseases such as malaria. Due to eventual environmental and human health-related concerns, the uses of DDT were phased out in Canada and the U.S. by the mid-1970s. However, DDT still persists in the environment as a result of long half-lives in soil, water and animal tissues, and also due to its continuing use in some countries, primarily for malaria control.

In the environment, DDT rapidly transforms to a very stable, highly persistent and lipophilic (literally 'fat-loving') chemical form, dichlorodiphenyldichloroethylene (DDE) [86]. There are not, and never have been, any commercial uses for DDE, but it is now widely detected in soil, sediments, water and in the food chain throughout the world [87]. Diet is the main source of exposure to DDE in the general human population, and the human fetus and infants are exposed though the placenta and breastfeeding, respectively [75,76,86]. Absorption, ingestion and inhalation of water and air containing trace concentrations of DDE are other sources of minor exposure [86].

Regulations in Canada for DDT

DDT was not manufactured in Canada, but in 1946 was registered for insecticide use to control agricultural insects, and also for household and industrial uses [88]. Most uses of DDT were banned in Canada by the mid-1970s, and the remaining uses were phased out by 1985 [88]. According to the Pest Control Products Act in Canada, 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 [88].

Possible Health Effects

The human health effects of DDE depend on the dose, the length and timing of exposure, and other factors. Background concentrations of DDE in humans usually are not known to cause any adverse health effect. However, at high doses, such as in the case of accidental releases, unusual occupational exposures, or accidental consumption of highly contaminated food, DDE may cause adverse health effects including respiratory problems, impairment of the immune system, neurotoxicity, birth defects and reproductive toxicity [87,89,90,91,92].

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BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean DDE concentrations in blood serum of pregnant Albertan women ranged from 0.11 to 1.5 ng/g of serum or, expressed on a serum lipid basis, from 12 to 214 ng/g lipid. Concentrations (lipid adjusted and whole serum) depended on age and geographic regions as shown in Figures 38-39. The concentrations in the north were lower than those in the central region and in turn those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. The estimated 50^{th} centile among individuals was 14 ng/g lipid. In a recent study, the geometric mean serum DDE concentration in pregnant women from Southern Alberta (Calgary Health Region, during the period of 2001 to 2003) was reported as 109 ng/g lipid [74]. In the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third Report, CDC), the geometric mean of serum DDE concentrations in females were 305 ng/g of lipid and 1.8 ng/g of serum, and the 50^{th} centile was reported as 256 ng/g lipid, which is higher than in the present study [27].

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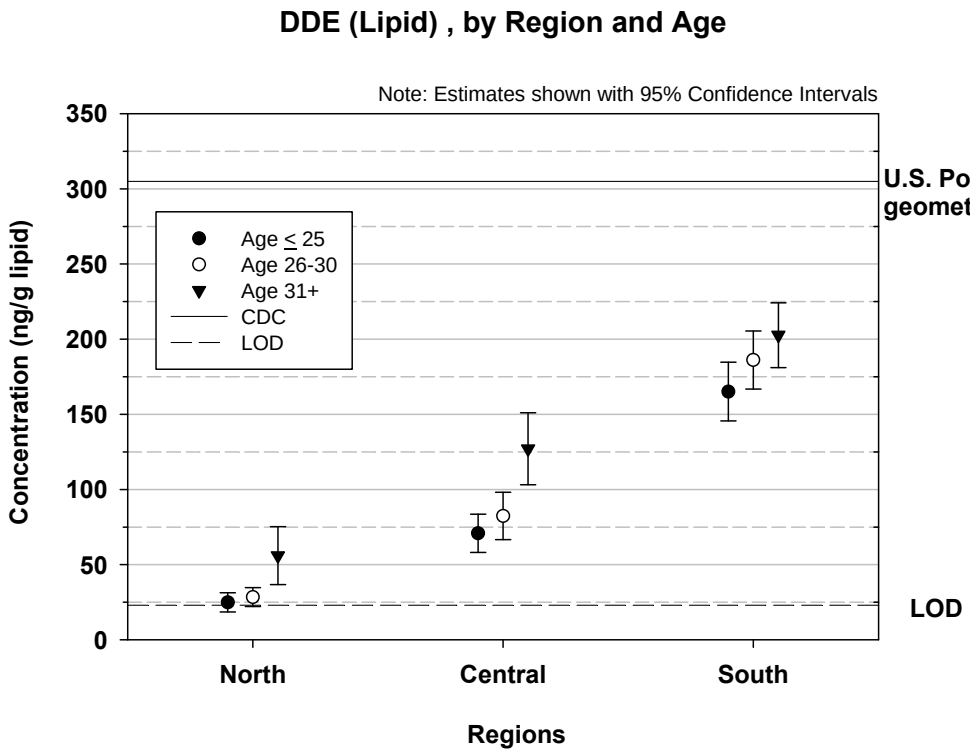
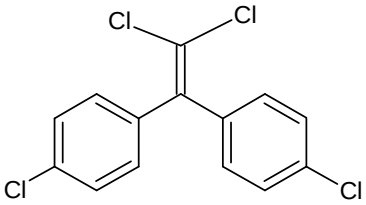


Figure 38

DDE



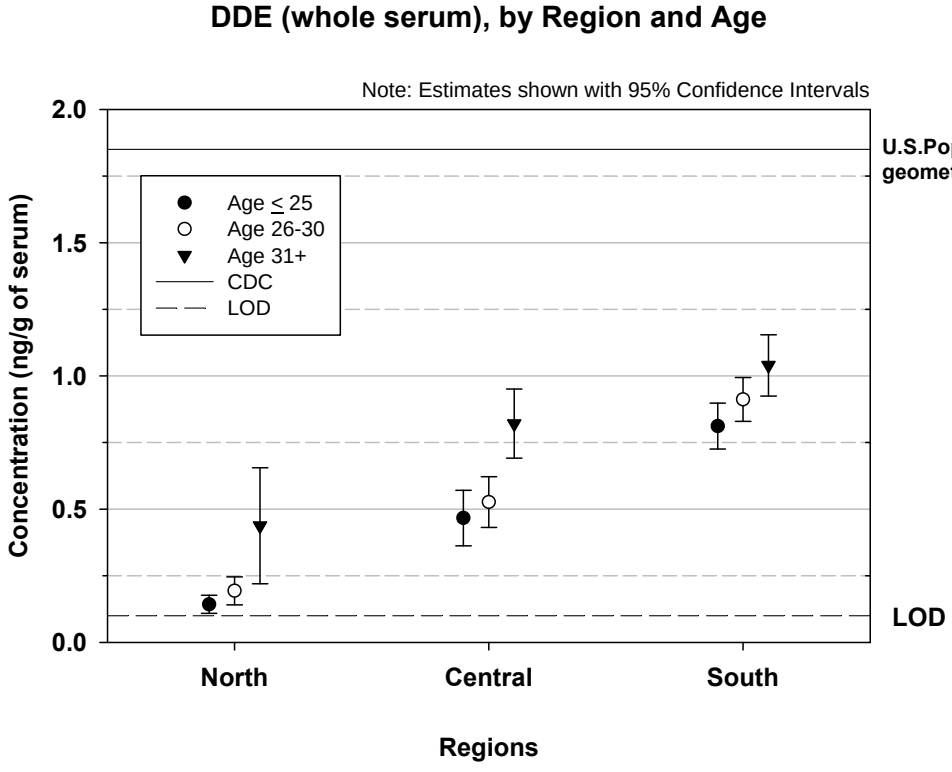


Figure 39

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Hexachlorobenzene (HCB)

GENERAL INFORMATION

Sources

Hexachlorobenzene (HCB) was widely used as a fungicide until the 1960s. In Canada, HCB was registered for pesticide use in 1940, and it was banned in 1972 due to concerns for environmental and human health [93]. However, it is still released in a small amount in the Canadian environment as a by-product of manufacture and use of chlorinated solvents and pesticides, emission from incinerators, and through long-range transport in air and water from other countries [94,95,96]. Diet is the main source of exposure of HCB to the general human population, and the human fetus and infants are exposed through the placenta and breastfeeding, respectively [75,76,94,97]. Other minor sources of exposure include absorption, ingestion and inhalation of water and air containing trace concentrations of HCB [94,98].

Regulations in Canada

In 1972, the use of HCB for fungicidal applications was banned in Canada [93], and the Federal Government stopped the production and commercial use of HCB in Canada in 1980 [93]. 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 [93].

Possible Health Effects

Background concentrations of HCB in humans are not known to cause any adverse health effects. At high doses, such as through unusual occupational exposure or by accidental consumption of highly contaminated food, HCB may cause several adverse health effects such as liver disease (porphyria cutanea tarda), neurotoxicity, immunotoxicity and skin lesion with discoloration [94,99,100]. The International Agency for Research on Cancer (IARC) considers HCB a possible human carcinogen [94].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends

Overall, mean HCB concentrations in blood serum of pregnant Albertan women ranged from 0.1 to 0.4 ng/g of serum or, expressed on a serum lipid basis, from 22 to 65 ng/g lipid. Concentrations (lipid adjusted and whole serum) depended on age and geographic regions as shown in Figures 40-41. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. The estimated 50^{th} centile among individuals was 3.7 ng/g lipid. In a recent study, the geometric mean of serum HCB concentration in pregnant women from Southern Alberta (Calgary Health Region, during the period of 2001 to 2003) was

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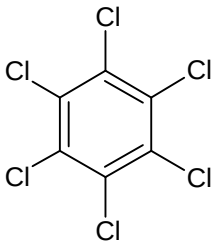
reported as 14 ng/g lipid [74]. In the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third Report, CDC), the geometric mean of serum HCB concentrations for the U.S. populations was not calculated, as serum HCB concentrations were mostly below the limits of detection of 31 ng/g of lipid, and the 50th centile was also reported as non-detectable [27].



HCB

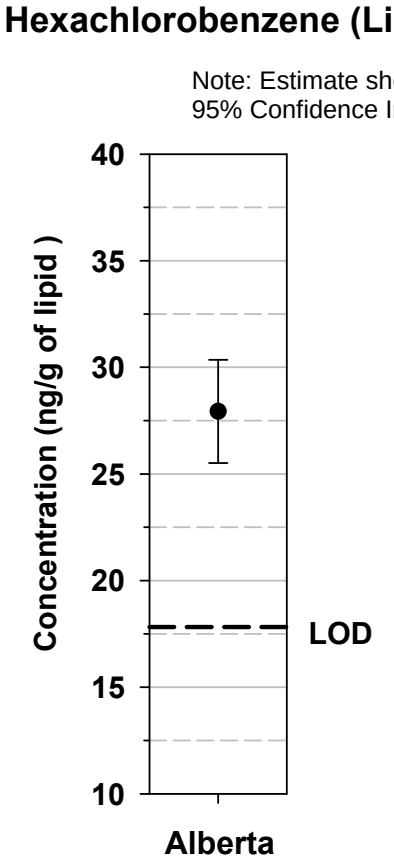


Figure 40

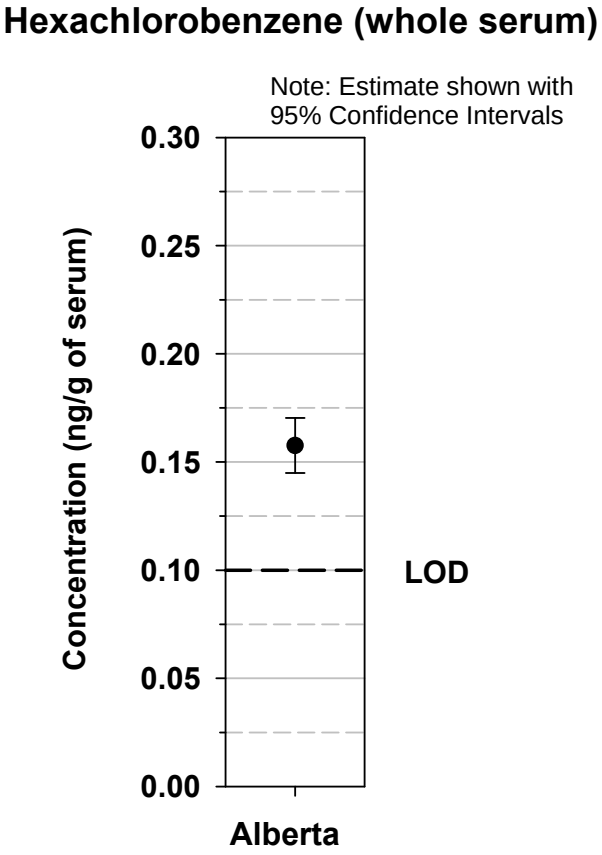


Figure 41

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Polybrominated Diphenyl Ethers (PBDEs)

GENERAL INFORMATION

Sources

Polybrominated diphenyl ethers (PBDEs) are additive brominated flame retardants that have been used for many decades in commercial products including computers, televisions, foam furniture, carpet underlay, and electrical appliances [101,102]. They are incorporated into these products to minimize the risk of fires, and hence save lives. PBDEs are manufactured as commercial mixtures, and three main types have been used historically: penta-BDE, octa-BDE and deca-BDE, so named based on the average bromine content. Penta-BDE was used mainly in foam products for stuffing (such as household furniture and seat cushions), octa-BDE for high-impact plastic products (such as telephone, FAX machines and computers), and deca-BDE is used primarily in plastics for electric components (such as wire and cable insulation) [101,103]. PBDEs can be released from these products into our homes or into the environment. PBDEs are rather persistent in the environment and can travel long-distances, such that they are detectable all over the world, including in remote regions far from their source.

Owing to their use in commercial products, people are exposed through PBDE-containing products in their houses and workplaces via dust [104,105,106]. Another potential source of exposure is through dietary intake, primarily from meat, dairy, fish and eggs [107,108]. Human milk is a major source of PBDEs to infants [109]. As the PBDEs are lipophilic (literally 'fat-loving') compounds, they can build-up in our body over time, including in fatty tissues and in human breast milk. PBDEs can also cross the placenta [110]. In these ways, PBDEs can be passed to the fetus or to infants during pregnancy and lactation, respectively [111]. Recent human breast milk studies show that Canadians contain the second-highest concentrations of PBDEs in the world, which is five to ten times higher than in Europeans and Japanese [112]. However, breastfeeding is encouraged due to the many associated health-benefits [54] 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 used in Canada are imported from other countries, and these are not manufactured in Canada. In July 2006, the Federal Government proposed to recommend the addition of PBDEs to the List of Toxic Substances in Schedule 1 of the Canadian Environmental Protection Act (CEPA) [113]. In December 2006, penta-, tetra- and hexa-BDE congeners, were identified as 'toxic', as defined by CEPA, and thus these PBDEs were added to the 'List of Toxic Substances' [113]. Also by the end of 2006, the Canadian Government announced regulations to ban the manufacturing, use, sale, and

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import of commercial PBDE mixtures of penta-BDE and octa-BDE [113]. The use of deca-BDE is not currently regulated in Canada.

Some other countries also have regulations controlling PBDEs. In August 2004, the European Union (EU) banned the use of penta- and octa-BDEs. Deca-BDE was also banned in the EU for use in electronic products as of July 1st, 2006, and a complete ban for deca-BDE will be in place in the EU by 2008 [113]. Japan has also voluntarily phased out the use of penta-BDE [114]. Several U.S. states adopted bans on the manufacture and use of penta- and octa-BDEs since 2005 [113]. The main U.S. manufacturer of penta- and octa-BDE mixtures voluntarily stopped production of these two PBDEs at the end of 2004. Otherwise, the use of deca-BDE is not restricted in the U.S. except in Washington and Maine [113].

Possible Health Effects

There are 209 varieties of PBDEs (also known as 'congeners'), varying in the number and relative position of bromine atom substitution [108]. In general, smaller PBDE congeners (with lower numbers of bromine atoms, 1-5 atoms) are better absorbed into our bodies than larger PBDEs, and are also more toxic [115]. The larger congeners, such as deca-BDE, eliminates rapidly from the human body (half-life, 6.8 day), whereas smaller congeners such as hepta-BDE are more slowly eliminated (long half-life, 86 days) [115,116]. Deca-BDE is known to be less toxic than penta- and octa-BDEs, however recent studies indicate that it breaks down to lower brominated PBDEs in the environment, which may be an indirect exposure source to humans [117,102]. Currently there are no data to indicate that background human populations have suffered any health effects from PBDE exposure, albeit few studies have been undertaken to examine this [118,119,120]. The skin sensitization potential of commercial deca-BDE was examined in human volunteers, and did not reveal any evidence of skin sensitization [118]. According to some epidemiologic studies in workplaces, no adverse effects in workers could be related to PBDE exposures [118]. A recent study of male fish-eaters from the Baltic region, a number of hormones in the blood were measured and compared to the concentrations of PBDE congeners [119]. The plasma concentrations of pituitary, thyroid, or testosterone hormone concentrations were not affected by the high-consumption of PBDE-contaminated fish. In laboratory animals which are exposed to much higher concentrations of PBDEs than those to which humans are exposed, penta- and octa-BDEs cause brain and thyroid problems, hearing deficits and fetal malformations [121,122].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

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

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)

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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)

However, except for BDEs 77,138,183 and 209, all other PBDE congeners were detected, and ranges of mean concentrations are shown based on lipid weight in blood serum samples:

PBDEs	Lipid weight (ng/g lipid)
BDE 28	2.1 to 98
BDE 47	11 to 340
BDE 66	0.2 to 4.5
BDE 85	0.4 to 24
BDE 99	2.5 to 470
BDE 100	2.1 to 98
BDE 153	4.5 to 53
BDE 154	0.4 to 30

For all the BDE congeners, there were no differences in concentrations apparent between regions or across age groups, as shown in Figures 42-49. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In a recent study, PBDE concentrations in blood of pregnant women living in a Californian agricultural community were monitored, and total PBDE concentrations ranged from 5.3 to 320 ng/g lipid [123].

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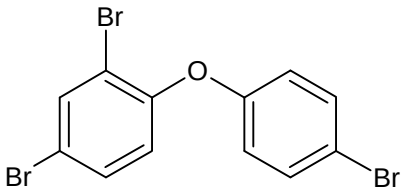


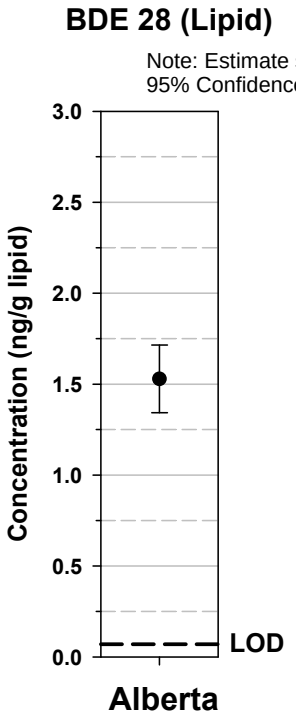
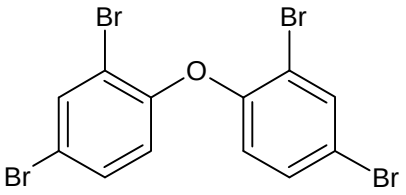


Figure 42



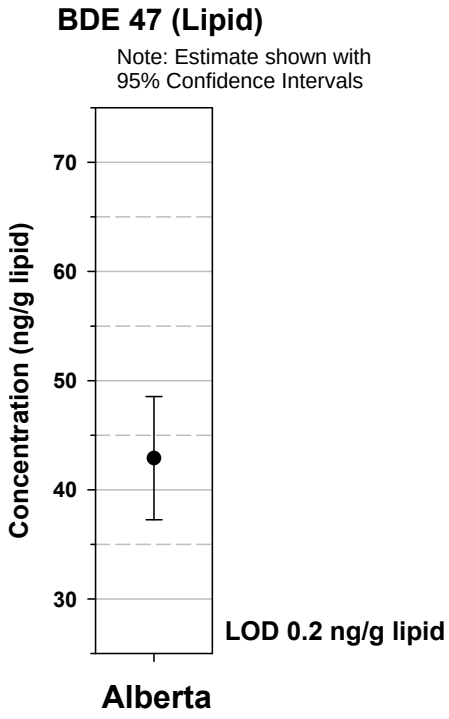


Figure 43

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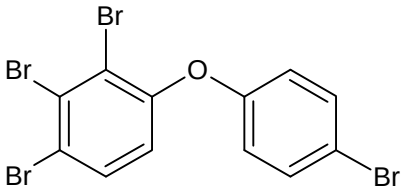

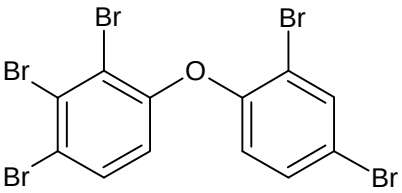

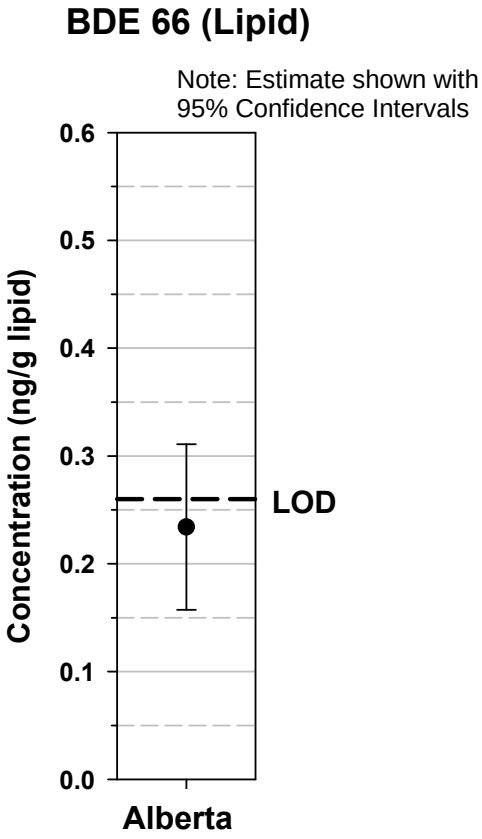

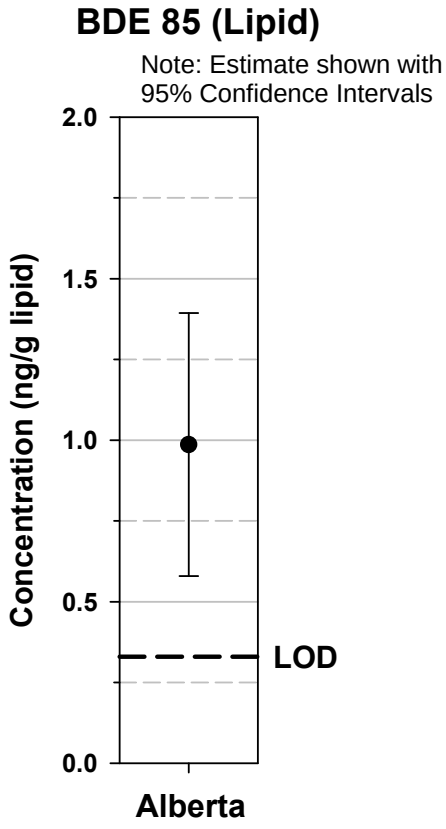

Figure 44

Figure 45

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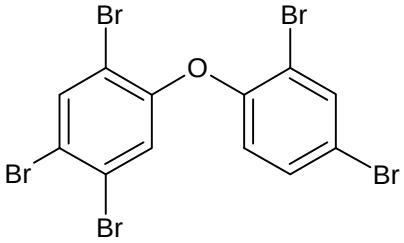

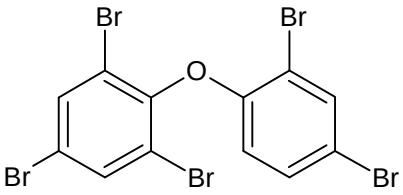

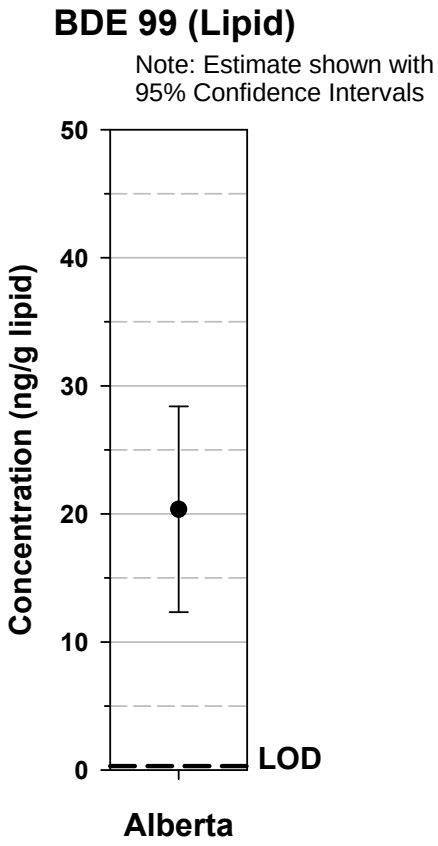

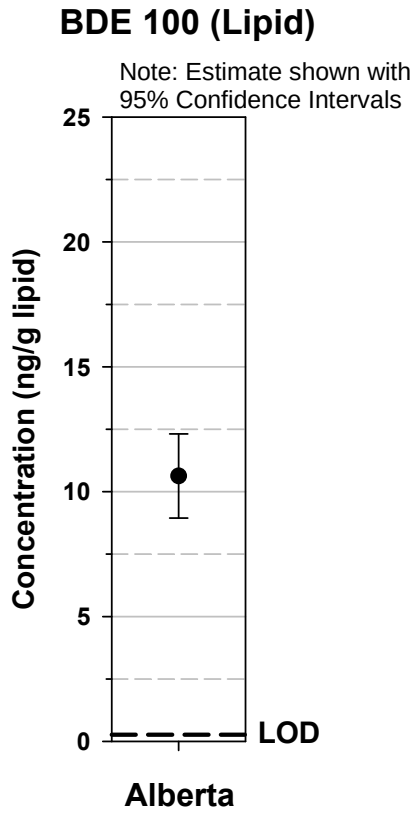

Figure 46

Figure 47

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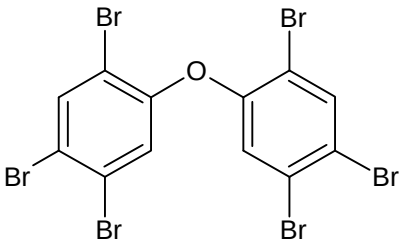


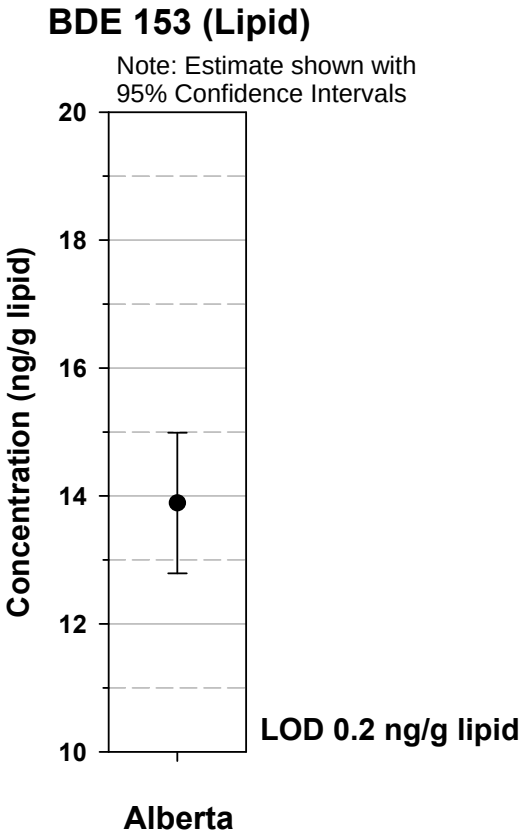
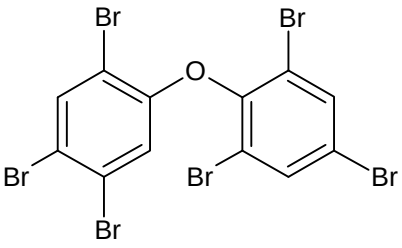


Figure 48



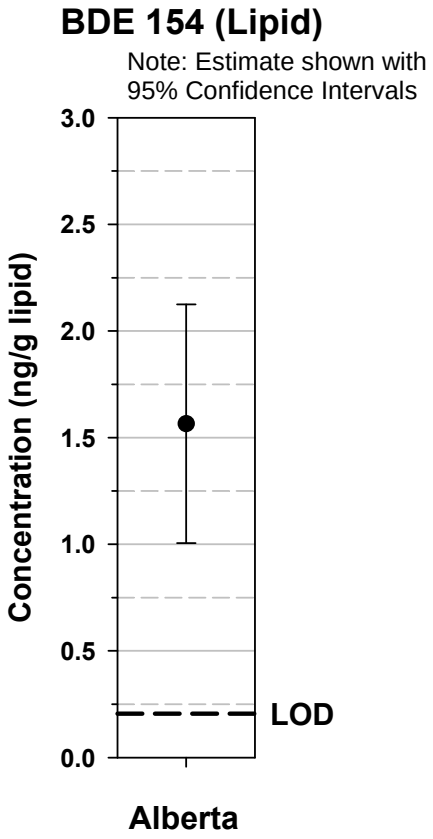


Figure 49

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Perfluorochemicals

GENERAL INFORMATION

Sources

Perfluorochemicals (PFCs) are a group of man-made fluorinated organic compounds that have been widely used for decades in industrial processes and various consumer and commercial products. These highly fluorinated molecules are unique in their ability to resist high temperatures and to repel oil, grease and water. A major use of PFCs has been as active ingredients in stain repellent polymer formulations for paper, textiles, food packaging, carpets, and leather, while other uses include their application as surfactants, fire-fighting foams, and fluoropolymer processing aids [124,125].

PFCs do not occur naturally, and can be released into our homes and into the environment (air and water) from these various consumer products, industrial or manufacturing processes, and accidental spills [126]. The general population is exposed to some extent to PFC in our houses and in the workplace via dust [127]. Another potential route of exposure is through the diet, mainly from fish and food products of animal origin which are exposed to PFC containing air, water and feed [128,129,130]. The use of PFCs in grease and water repellent coatings for food packaging is also known to be a source of exposure [131,132]. In the environment, PFCs can travel long-distances, such that they are detectable all over the world, including in remote regions far from their sources of production or use.

Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are the two most abundant PFCs in humans and in the environment, but many related PFCs are also known to co-occur. These are highly stable in the environment and in our bodies. The half-lives in humans are 5.4 years for PFOS and 3.8 years for PFOA [133]. PFCs are widely found in wildlife and in human blood [134,135,136,137], and PFCs are known to cross the placental barrier [138,139].

Regulations in Canada

PFCs used in Canada are imported from other countries, and are not manufactured in Canada [140]. In 2002, the predominant global manufacturer, and the only manufacturer of PFOS in the U.S., voluntarily phased out the production of PFOS and other related chemicals. As a result, its uses decreased significantly in Canada after 2002 [140]. In July 2006, the Federal Government proposed to recommend the addition of PFOS to the List of Toxic Substances under Schedule 1 of the Canadian Environmental Protection Act (CEPA) [140]. In December 2006, the Canadian Government proposed a regulation to prohibit the manufacture, use, sale, offer for sale, and import of PFOS and its salts with certain minor exemptions including for use in semiconductors, photographic films, or for use in laboratories for scientific research [140]. The Canadian Government has also taken actions on fluorotelomers, which are PFCs that can degrade to PFOA in the atmosphere and in organisms. In June 2006, the Canadian Federal Government

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proposed a regulation to prohibit the introduction of four new fluorotelomer-based substances into Canada, and also recommended the addition of these substances to the List of Toxic Substances under Schedule 1 of CEPA [141].

Possible Health Effects

Human studies relating to possible health effects of PFC exposure are very limited. A few studies suggested negative associations between PFOS or PFOA concentrations in pregnant women or cord blood and the infant's birth weight or size [142,143]. There are comparably more data from animal studies at higher concentration, and adverse health effects in animals include developmental and reproductive effects, as well as general systemic toxicity and effects on the liver [144,145,146].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

The following PFCs were measured in the blood serum samples of pregnant women in Alberta:

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)
6. Perfluorotetradecanoate (PFTA)

However, except for PFDS, all other PFCs were detected, and ranges of mean concentrations are shown based on wet weight in blood serum samples:

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PFCs	Wet weight (ng/g serum)
PFHxS	0.7 to 9.8
PFOS	0.3 to 14
PFOA	1.7 to 3.8
PFNA	0.3 to 0.7
PFDA	0.02 to 0.3
PFUA	0.02 to 0.8
PFDoA	0.07 to 1.1
PFTA	0.1 to 1.2

Concentrations depended on age and geographic regions as shown in Figures 50-57. For PFHxS and PFOS (Figures 50 and 51), the concentrations in the north were equal to those in the central region and in turn those in the central region were greater than those in the south. For PFOA (Figure 52), the concentrations in the north were equal to those in the central region and in turn those in the central region were greater than those in the south. The concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn greater than those in the 31+ age group. For PFNA (Figure 53), the concentrations in the ≤25 year age group were higher than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For PFDA (Figure 54), the concentrations in the ≤25 year age group were equal to those in the 26-30 year age group which were in turn lower than those in the 31+ age group. For PFUA and PFDoA (Figures 55 and 56), the concentrations in the north were equal to those in the central region and in turn those in the central region were greater than those in the south. For PFTA (Figure 57), there were no differences in concentrations apparent between regions or across age groups. The seasonal variation of PFC concentrations was examined in the south, but there was no apparent temporal or seasonal trend observed.

In the pooled serum samples from the participants of the U.S. National Health and Nutrition Survey (NHANES 2001-2002), the estimated least-square mean concentrations of PFHxS, PFOS, PFOA and PFNA in non-Hispanic white females were reported as 4.3, 24, 3.9 and 0.5 ng/mL, respectively, which are consistent with the present study [147].

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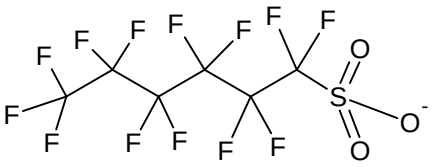

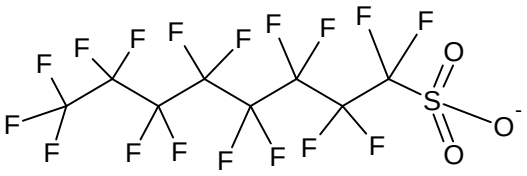

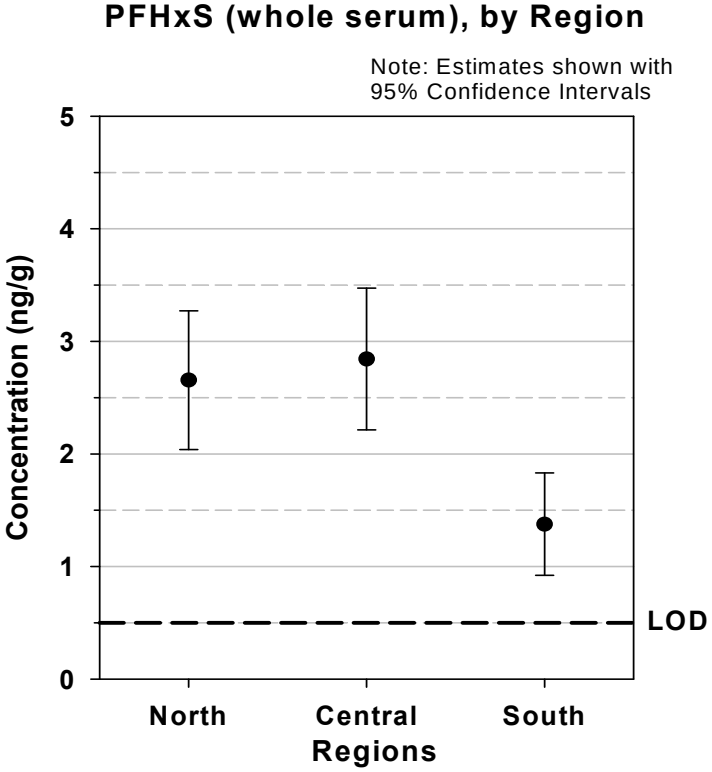

Figure 50

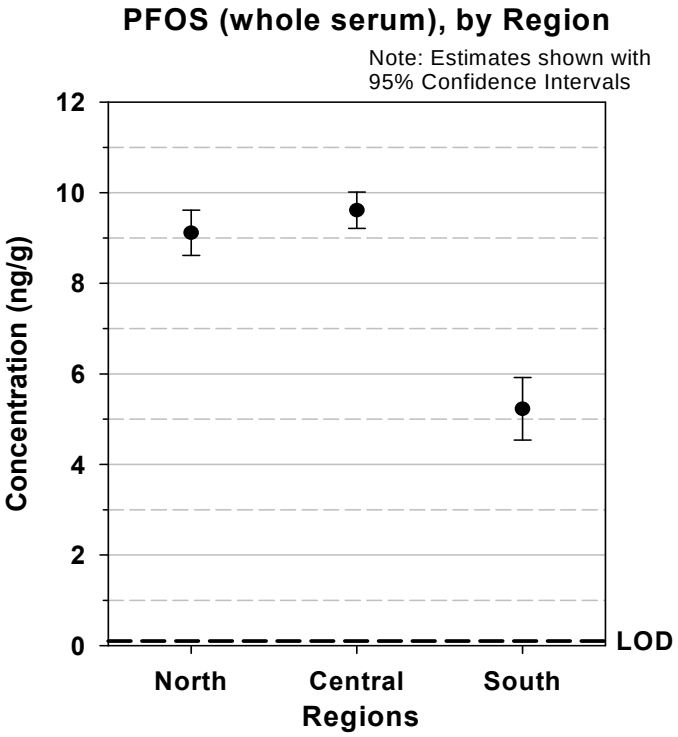

Figure 51

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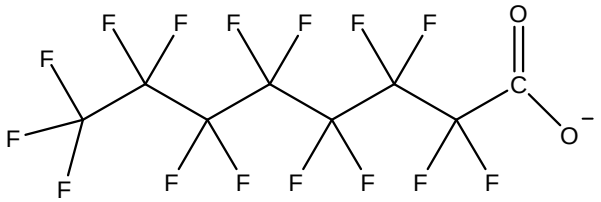


PFOA (whole serum), by Region and Age

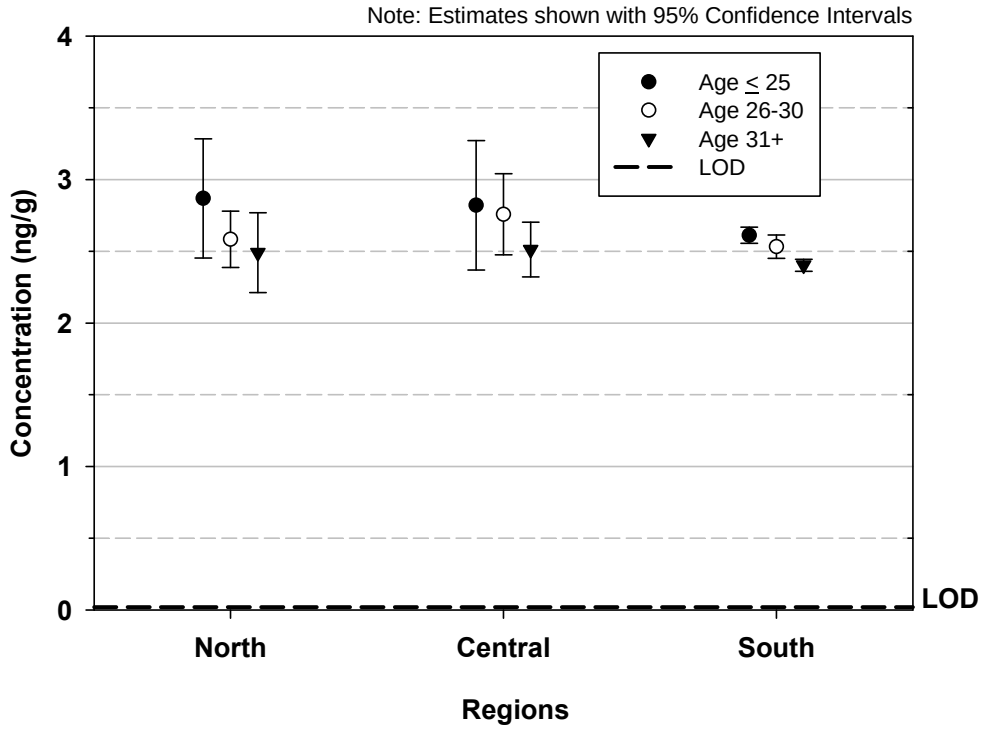
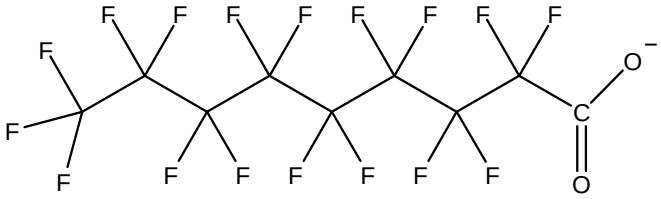


Figure 52



PFNA (whole serum), by Age

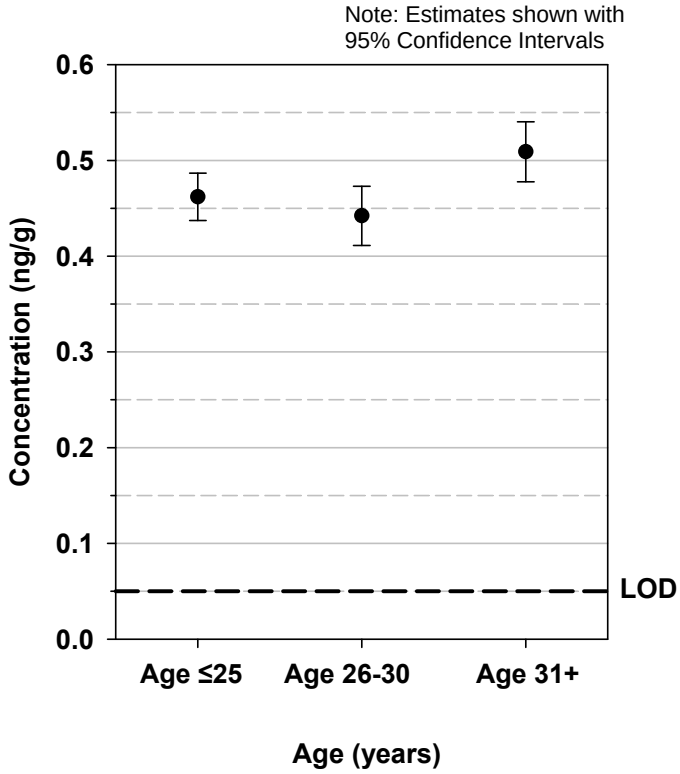


Figure 53

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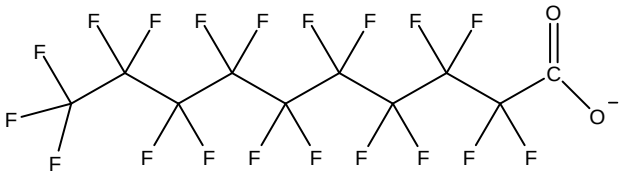

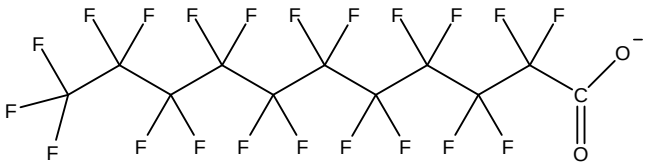

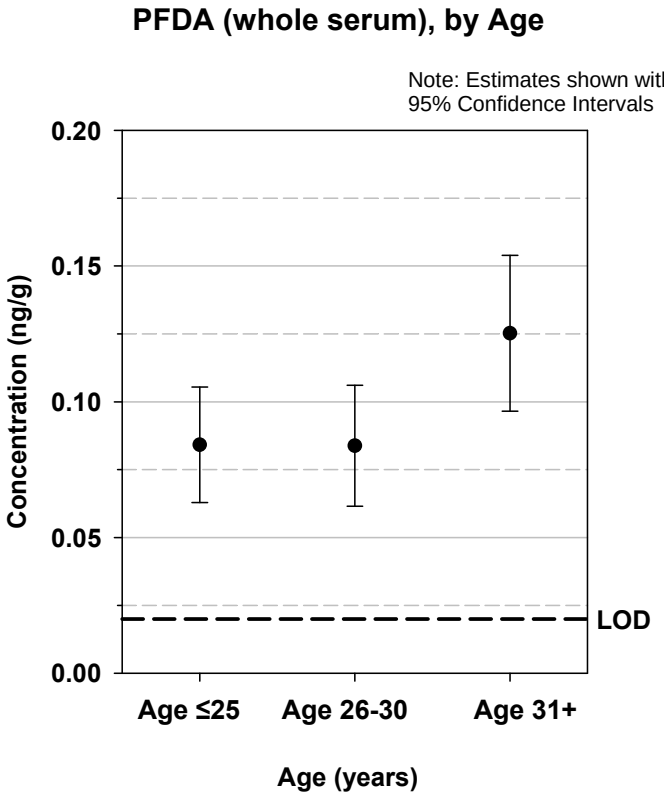

Figure 54

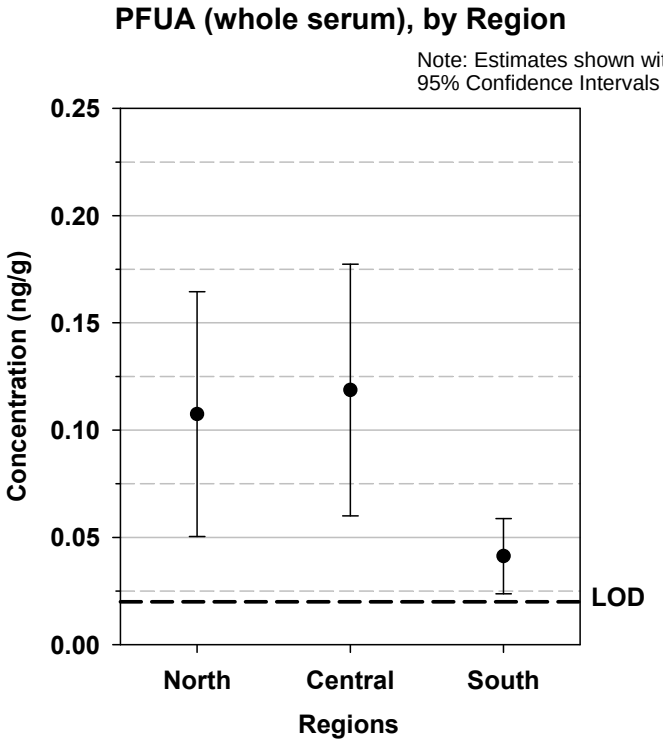

Figure 55

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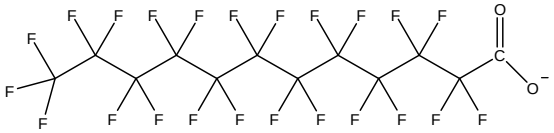

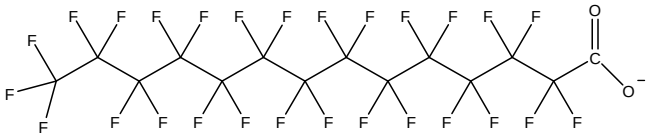

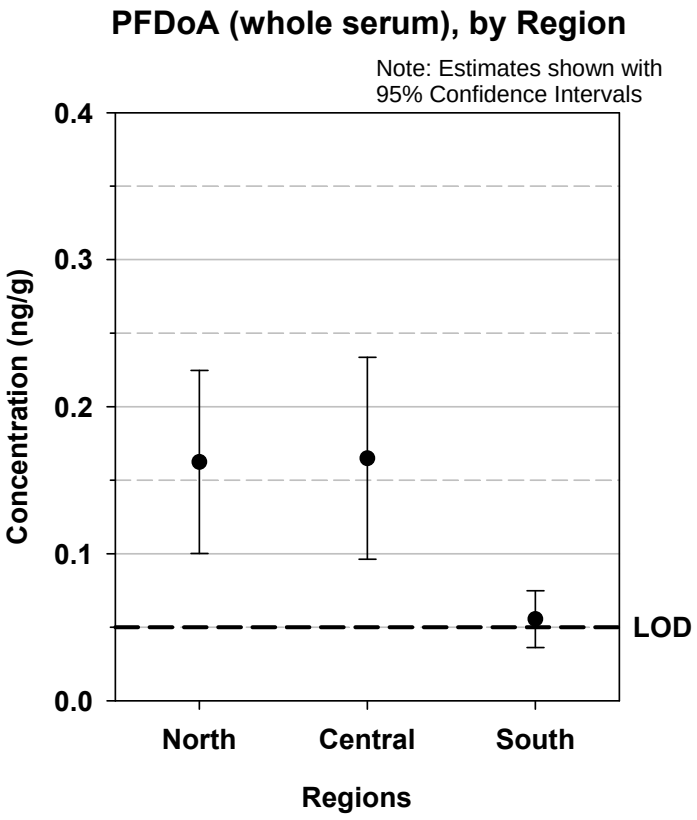

Figure 56

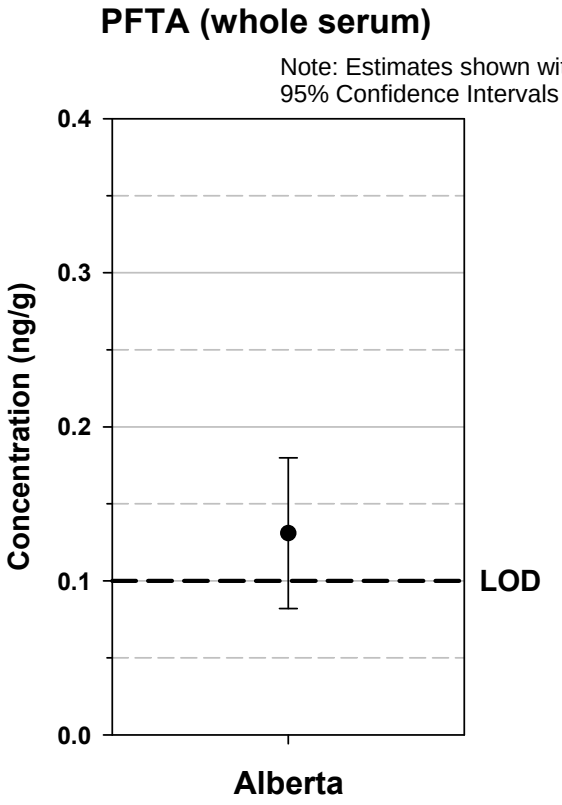

Figure 57

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Bisphenol A

GENERAL INFORMATION

Sources

Bisphenol A (BPA) is a synthetic chemical used to manufacture polycarbonate plastics and epoxy resins. BPA is an endocrine disruptor with estrogen-like activities [148,149] and its production volumes are among the highest of all chemicals worldwide [150]. These plastics and resins are used in various consumer products such as toys, food and beverage bottles and cans, plastic baby bottles, bottle tops, eyeglass lenses, medical equipment, dental sealants, and water pipes [151]. The leaching of BPA from plastic bottles, food cans and dental sealants are confirmed by several studies [152,153,154,155], and this has led to wide-spread human exposure to BPA [150].

Due to the extensive use of BPA in plastics and resins, the predominant source of BPA exposure in the general population is through the use of everyday plastic products, and consumption of contaminated canned and bottled foods and beverages [150,156]. Some studies have also shown that BPA leaches from municipal waste landfills into the surrounding ecosystem [157,158]. The leaching of BPA from infant formula cans into baby food, and in baby bottles, are sources of BPA exposures to infants [159,160,161]. Due to the semi-lipophilic (literally 'fat-loving') nature of BPA, it can accumulate in human breast milk [162], and BPA can also cross the placenta [163]. In these ways, BPA 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 [54] that currently outweigh known risks.

Regulations in Canada

In 2006, the Canadian Federal Government, as a part of the Chemicals Management Plan, selected BPA for a detailed safety review to assess the potential human and environmental effects. This process started in May 2007, and was recently completed. Canada is the first country to complete a risk assessment of BPA, and the draft Assessment Report was published in the Canada Gazette on April 19, 2008 for a 60-day public comment period in an effort to decide whether to ban the import, sale and use of baby bottles containing BPA. 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 (CEPA).

Possible Health Effects

Due to the high-volume production and wide-spread use of BPA, there is an increasing interest in investigating the effects of BPA exposure on human health. However, human studies are currently very limited, and most available data are from animal studies. As a hormone or endocrine disruptor, a high enough dose of BPA is associated with a number of adverse health effects in animals related to hormonal

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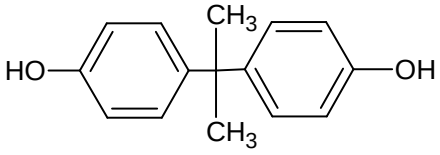
imbalance such as developmental and reproductive toxicity, neurotoxicity, ovarian dysfunction and recurrent miscarriages [155,164,165,166].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

In the present study, BPA was only measured for women of age 26-30 in Southern Alberta (Figure 58). Overall, mean concentrations for BPA ranged from 0.07 to 0.6 ng/g serum. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the Greenpeace study in the Netherlands, BPA concentration ranged from 0.5 to 1.7 ng/g serum [167], and in another study in Japan, it ranged from 0.2 to 0.8 ng/g in maternal serum [168]. In the U.S. National Health and Nutrition Survey (NHANES 2003-2004), the geometric mean urinary concentration of BPA in females was 2.4 μg/L [169]; however it is not possible to compare urinary levels to blood serum levels.



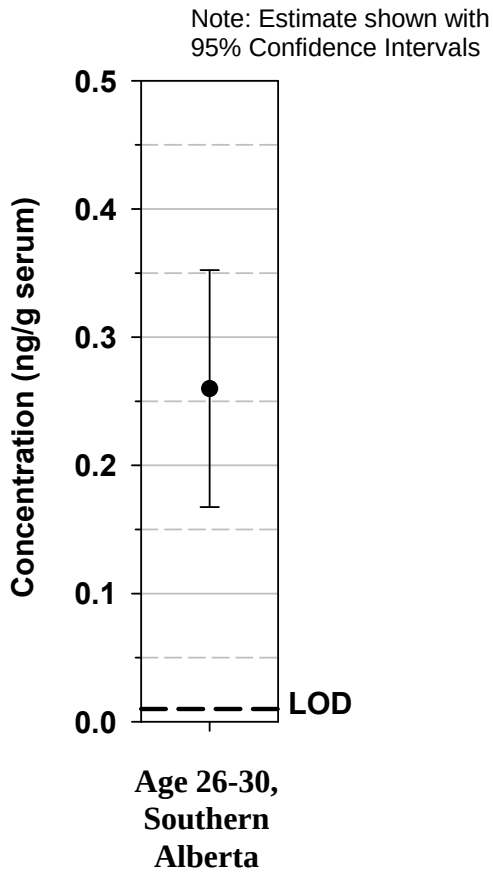


Figure 58

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Nonylphenol

GENERAL INFORMATION

Sources

Nonylphenol (NP) belongs to the family of organic alkylphenol (AP) chemicals which are the main components of widely used alkylphenol ethoxylate (APE) surfactants, such as nonylphenol ethoxylate (NPE). APEs have been used as surfactants for many decades in a wide variety of domestic, consumer and industrial products such as in cleaning products, paints, pesticides, textile processing, pulp and paper processing and cosmetics [170,171]. APEs are degraded in the environment to NP, and these are persistent and more toxic than the parent APEs [172,173,174]. APEs and their degradation products do not occur naturally, but are man-made chemicals having estrogen like activities [175,176,177].

These chemicals enter the aquatic environment through discharge from urban, municipal, and industrial wastewater and also by direct discharge in such activities as pesticide application. Absorption, ingestion and inhalation of water and air containing trace concentrations of these are sources of minor exposure to general population [178]. However, due to their lipophilic (literally 'fat-loving') nature, they accumulate in fatty tissues of wildlife and enter the human food chain, thus dietary intake is another potential source of exposure [179]. NP can also accumulate in breast milk [179] and can also cross the placenta [180]. In these ways, NP 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 [54] that currently outweigh known risks.

Regulations in Canada

In 2000, NP and NPEs were identified as 'toxic' by Environment Canada and Health Canada, and thus both were added to the 'List of Toxic Substances' in Schedule 1 of Canadian Environmental Protection Act (CEPA) [181]. As a result, since 2001 the use of NPEs in detergents has been banned in Canada [182].

Possible Health Effects

Human studies of possible health effects of AP or APE exposure are very limited, and most available data are from animal studies. As a hormone or endocrine disruptor, these are associated with a number of adverse health effects related to hormonal imbalance such as developmental and reproductive toxicity [183,184,185,186].

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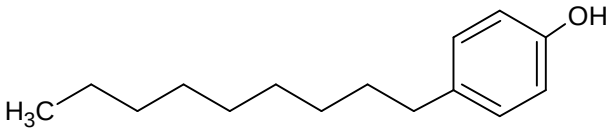
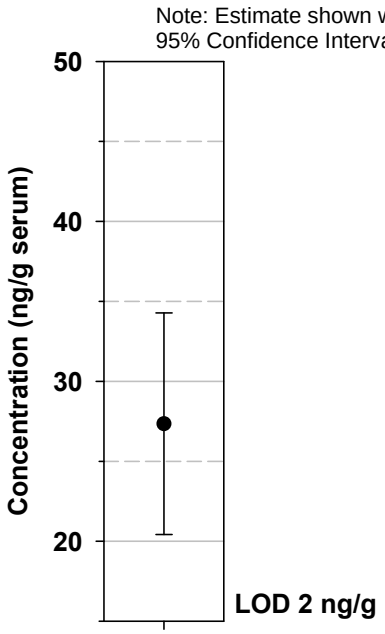
BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

In the present study, NP was measured only for the age group 26-30 in Southern Alberta (Figure 59). The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. Overall, mean concentrations for NP ranged from 12 to 80 ng/g serum. In a Greenpeace study in the Netherlands, NP concentrations ranged from 0.6 to 16 ng/g in maternal serum [187].



Nonylphenol (whole serum)



Age 26-30, Southern Alberta

Figure 59

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Methylmercury (CH_3Hg)

GENERAL INFORMATION

Sources and Guidelines

Mercury is widely distributed around the earth in its elemental, inorganic and organic forms. Elemental and inorganic mercury are released to air and water from the combustion of fossil fuels (mainly coal), mining, smelting, and other industrial processes. Inorganic mercury in the atmosphere (mercury vapour), is ultimately redeposited to the earth in precipitation. If incorporated into aquatic sediments, it may be transformed by microorganisms to methylmercury, an organic form of mercury that can accumulate in organisms. For example, plant and sedimentary materials containing methylmercury are consumed by small fish that are, in turn, consumed by progressively larger fish and finally by humans. At each stage in this food-chain, methylmercury concentrations can increase through the process of biomagnification, resulting in higher concentrations of methylmercury in our food sources [188,189].

The major source of methylmercury exposure in the general population is through the consumption of certain fish and seafood [190]. Large predatory fish at the top of aquatic food-chains generally have higher mercury concentration than small fish lower in the food-chain. Shark, large tuna, sword fish, marlin, and king mackerel contain 10-20 times higher concentrations of methylmercury than fish such as herring, cod, pollack, and shellfish such as shrimp or scallops [191]. Canned light tuna generally has lower methylmercury concentrations than canned albacore (white) tuna or fresh tuna steaks, and thus is a good alternative for limiting mercury intake while maintaining fish consumption.

Health Canada recently (March 28, 2007) updated the guidelines on fish consumption to limit mercury exposure, and has advised Canadians to limit their consumption to 150 grams per week of fresh and frozen tuna, shark, swordfish, escolar, and marlin. The new guidelines suggest a limit of 150 grams per month of these fish for pregnant women, breastfeeding mothers and women who might become pregnant. According to these guidelines, the recommended amount of these fish for children aged five to eleven is 125 grams per month, and for children aged one through four is 75 grams per month.

Behaviour in the Body

Methylmercury is rapidly absorbed into our blood from the gastrointestinal tract. Blood-borne methylmercury is present primarily in red blood cells and the blood can deliver methylmercury to the brain where it may accumulate while slowly being converted back to inorganic mercury [192]. Human pharmacokinetic studies indicate that the amount of time it takes to remove half of the body's methylmercury stores is approximately 70 days [190,193]. It is removed slowly through urine, feces, and breast milk.

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Possible Health Effects

The human health effects of mercury are diverse and can depend on the dose, the form of mercury present in our bodies, the length of exposure, and the timing of the exposure. Methylmercury is more toxic than inorganic or elemental mercury [194]. At high doses, such as in the case of accidental poisonings, methylmercury is well documented to be a human neurotoxin, and may cause adverse effects on the motor and sensory systems [195,196]. At lower doses during pregnancy, development of the fetus' central nervous system can be sensitive to methylmercury exposure, and for such reasons the above mentioned guidelines were developed for pregnant women [196]. Health Canada's guidelines for methylmercury in whole blood are as follows: (i) concentrations between 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 a "level of action" [197].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean methylmercury concentrations in blood serum of pregnant Albertan women ranged from 0.04 ng/g to 0.22 ng/g. Concentrations depended on age and geographic region as shown in Figure 60. The concentrations in the north were lower than those in the central region and in turn those in the central region were lower than those in the south. In general, the concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. In general, the difference in concentration between ages increased from north to central to south. This effect of age is consistent with results from a 1999/2000 U.S. study whereby whole blood methylmercury concentrations were ~1.5 times higher among women 30-49 years of age than among women 16-29 years of age [198].The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

Discussion and Interpretation.

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 [199] – red blood cells were excluded from the analysis here due to limitations in our study design. If we assume that our study design excluded 95% of methylmercury in red blood cells, the current data can be multiplied by 20 to arrive at an estimated whole blood concentration. Using our data from Northern Alberta, age 31+, mean whole blood concentrations are thereby estimated to be approximately 3 ng/g, which is well below Health Canada prescribed "level of concern" of 20 ng/g.

Few other data are available for methylmercury in serum, and a mean concentration of 2.4 nmol/L (equivalent to 0.51 ng/g) was reported for blood serum in a cohort of Swedish women, and an association with fish consumption was also evident [200]. In general, higher values are reported for people who have geographic proximity to a steady supply of fresh fish than more inland populations [201,202].

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Although these cannot be directly compared, a number of past studies have reported methylmercury concentrations in whole blood, or in red blood cells, from different populations [190]. For example, whole blood methylmercury concentrations among 1,709 female participants in the U.S. National Health and Nutrition Survey (NHANES) in 1999 and 2000 were 0.6 µg/L at the 50th percentile and ranged from concentrations that were non-detectable (5^{th} percentile) to 6.7 µg/L (95^{th} percentile) [198]. In Canada, the territorial baseline monitoring program covering the entire Northwest Territories and Nunavut for multiple contaminants, reported mean methylmercury of 2.2 µg/L in maternal blood, with a maximum maternal methylmercury concentration of 29 µg/L in one individual [203].

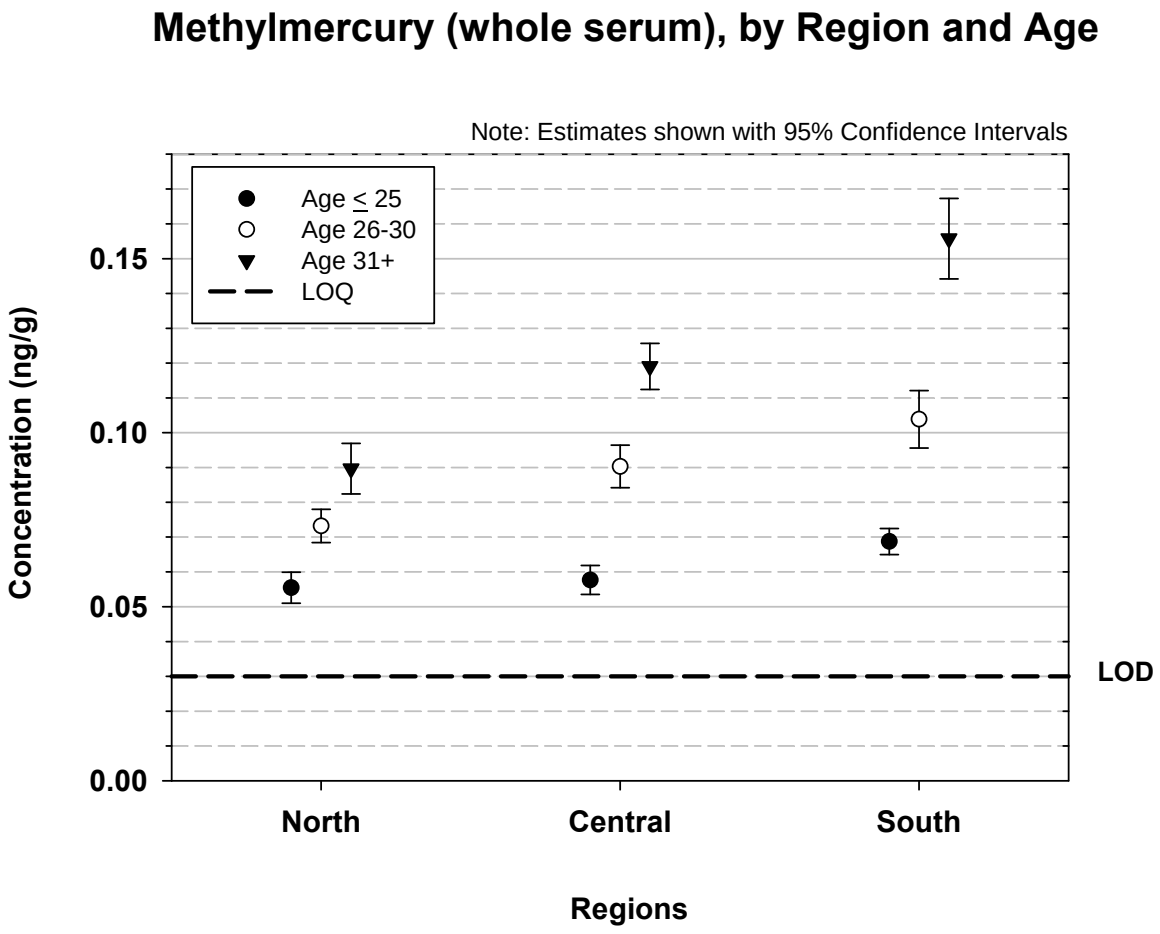

Figure 60

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Metals and Mineral Micronutrients

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

Mineral Micronutrients	Non-Micronutrients
boron	aluminum
cobalt	antimony
copper	arsenic
iron	barium
manganese	beryllium
molybdenum	cadmium
nickel	cesium
selenium	chromium
zinc	lead
	mercury
	platinum
	silver
	thallium
	tungsten
	uranium
	vanadium

All metals except for arsenic, beryllium, cadmium, thallium, platinum, tungsten and uranium, all other metals were detected. 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.

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METALS
(Non-Micronutrients)

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Aluminum (Al)

GENERAL INFORMATION

Sources

Aluminum (Al) is the third most abundant chemical element in mineral rocks, and is therefore widely distributed in the environment [204]. It exists in various distinct forms including as aluminum oxide, hydroxide, chloride, sulphate, phosphate and borate. These may be used in, or found in, in a wide variety of industrial, medicinal and consumer products such as glass and ceramic, pulp and paper products, roofing, airplanes, antacids, astringents, food additives, beverage cans, foil and in cooking utensils. Aluminum can be released into our homes and into the environment (air, water and soil) through use or disposal of such products, and also through mining, and various industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of aluminum are the background sources of aluminum exposure to the general population. Other sources of exposure include its uses in pharmaceuticals and also through occupational activities where aluminum containing products are made [204,205,206].

Possible Health Effects

The human health effects of aluminum depend on the dose, the form of aluminum present in the environment, the length and timing of exposure, and other physiological factors. Background concentrations of aluminum in humans are not known to cause any adverse health effects [204]. 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 [204,207]. The role of aluminum in the development of 'Alzheimer-type' disease is controversial, and no definitive conclusions have been reached [204,207].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration for Al ranged from 12 μg/L to 56 μg/L. There were no differences in concentrations apparent between regions or across age groups, as shown in Figure 61. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

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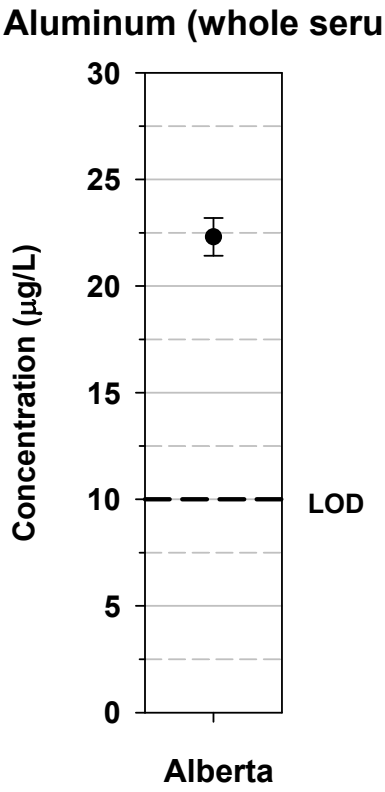

Figure 61

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Antimony (Sb)

GENERAL INFORMATION

Sources

Antimony (Sb) is a naturally occurring chemical element that enters the environment from natural processes such as weathering of rocks and minerals. In the environment, it exists in the form of various antimony compounds such as oxides, fluorides and hydrides. Such antimony compounds are used in a wide variety of products including semiconductors, batteries, paints, ceramics and fireworks [208]. Antimony can be released into the environment (air, water and soil) through use or disposal of such products, and also through mining and various other industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of antimony are the background sources of exposure to the general population. People may be exposed to higher antimony concentrations in occupational settings where antimony-containing products are manufactured or used [208].

Possible Health Effects

The human health effects of antimony depend on the dose, the form of antimony present in the environment, the length and timing of exposure, and other physiological factors. Background concentrations of antimony in humans are not known to cause any adverse health effects [208]. Under a high dose long-term exposure scenario, such as in the case of accidental releases and unusual occupational exposures, antimony may cause adverse health effects such as problems with respiratory, cardiac and digestive systems [208].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration for antimony ranged from 3.0 μg/L to 15 μg/L. Concentrations depended on region as shown in Figure 62. The concentrations in the north were equal to those in the central region and in turn those in the central region were greater than those in the south. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), the geometric mean of antimony concentration in urine samples of females was 0.1 μg/L [27], but that result cannot be compared to the present results from serum samples.

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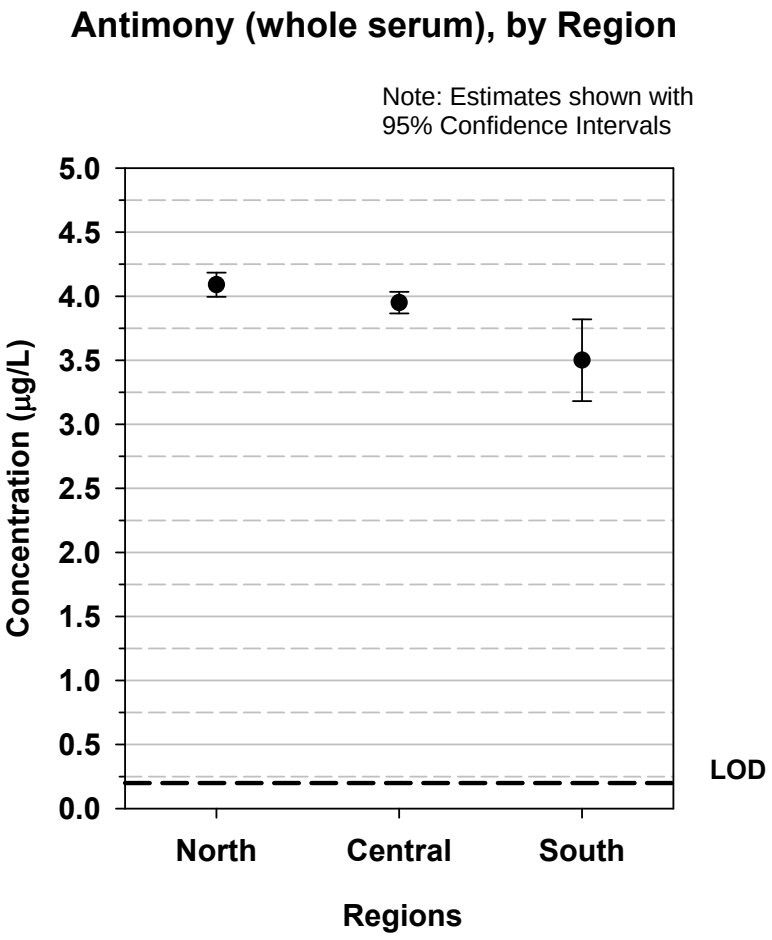

Figure 62

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Barium (Ba)

GENERAL INFORMATION

Sources

Barium (Ba) is an abundant naturally occurring chemical element that can be found in mineral rocks [209]. In the environment, it exists in the form of various barium salts such as sulphate, carbonate, nitrate and chlorate. These barium compounds are used in or found in a wide variety of products including drilling mud in the petroleum industry, bricks, glass, rubber, fluorescent lamps, and in medicine it is used for X-ray imaging of the digestive system. [209]. Barium can be released into the environment (air, water and soil) through use or disposal of such products, and also through mining, burning of coal and fossil fuels, and various other industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of barium are the background sources of exposure to the general population. People may be exposed to higher barium concentrations in occupational settings where barium-containing products are manufactured [209].

Possible Health Effects

The human 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. Background concentrations of barium in humans are not known to cause any adverse health effects [209]. At high dose long-term exposure scenarios, such as in the case of accidental releases and unusual occupational exposures, barium may cause adverse health effects such as problems with nervous, cardiac, respiratory and digestive systems, as well as general weakness, vomiting and muscular paralysis [209].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations for Ba ranged from 5.1 μg/L to 15 μg/L. Concentrations depended on region and age as shown in Figure 63. The concentrations in the north were equal to those in the central region, and in turn those in the central region were lower than those in the south. In general, the concentrations in the ≤25 year age group were greater than those in the 26-30 year age group which were in turn equal to those in the 31+ age group. However, the age trend was reversed in the north. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), the geometric mean of barium concentration in urine samples of females was 1.4 μg/L [27], but those results cannot be compared with the present results for serum.

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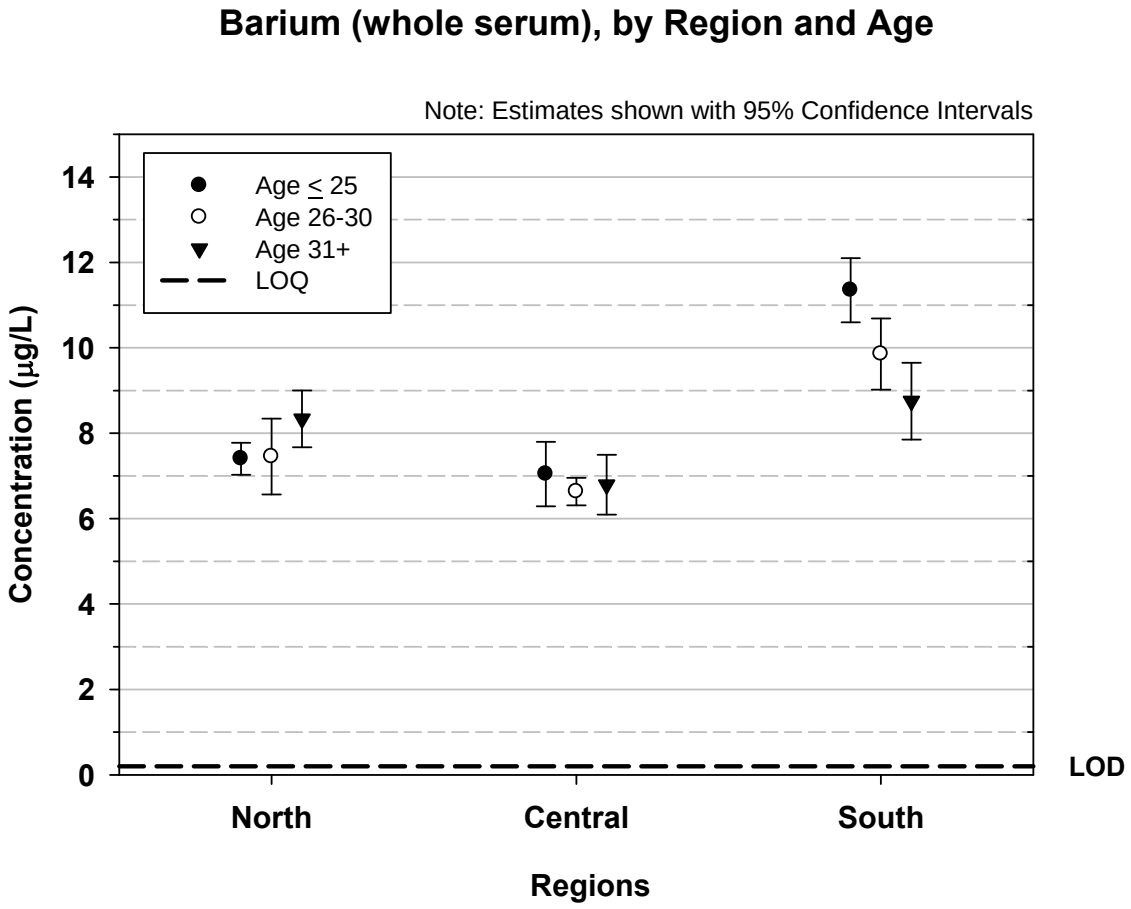

Figure 63

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Cesium (Cs)

GENERAL INFORMATION

Sources

Cesium (Cs) is a minor naturally occurring chemical element that enters the environment from natural processes such as weathering of rocks and minerals [210]. In the environment, natural cesium exists as a stable isotope (^{133}Cs) and as various naturally occurring compounds including hydroxides, carbonates, iodides and bromides. Such cesium compounds are used in a wide variety of products including alkaline storage batteries, photoelectric cells, optical instruments and glasses, and atomic clocks [210]. Cesium can be released into the environment (air, water and soil) through use or disposal of such products, and also through mining, and various industrial processes. After release to the environment, it can travel long distances in air including to remote regions far from the source. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of cesium are the background sources of cesium exposure to the general population. People may be exposed to higher amounts of cesium in occupational settings where cesium containing products are made.

Possible Health Effects

The human health effects of cesium depend on the dose, the length and timing of exposure, and other physiological factors. Background concentrations of cesium in humans are not known to cause any adverse health effects [210]. Human studies addressing possible health effects from long term exposure to higher concentrations of cesium are very limited [210].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations of cesium ranged from 0.4 μg/L to 0.7 μg/L. Concentrations depended on region and age as shown in Figure 64. The concentrations in the north were lower than those in the central region, and in turn those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group, which were in turn lower than those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), the geometric mean of cesium concentration in urine samples of females was 4.4 μg/L [27], but that result cannot be compared to the present results from serum samples.

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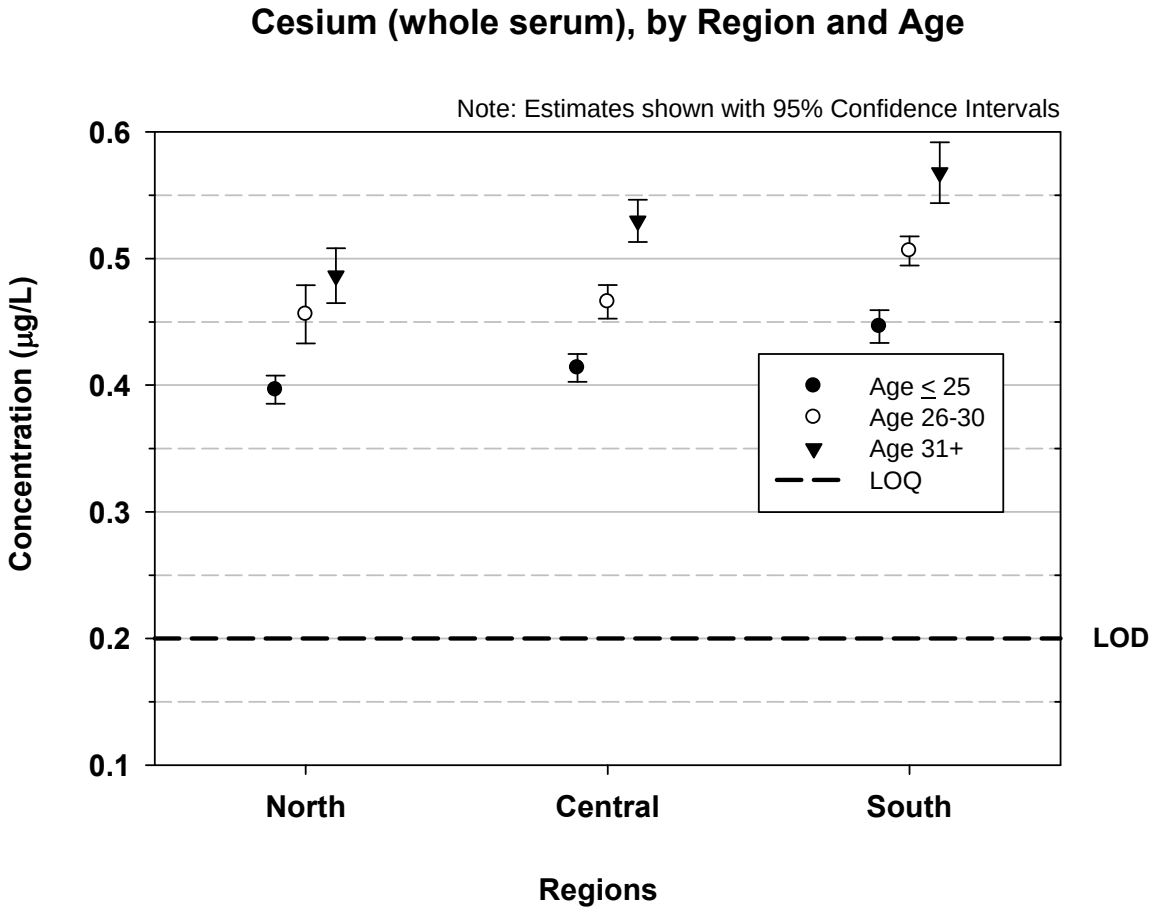

Figure 64

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Chromium (Cr)

GENERAL INFORMATION

Sources

Chromium (Cr) is a naturally occurring chemical element that enters the environment from natural processes and sources such as weathering of rocks and minerals, and volcanic eruptions. In the environment, it may exist in several chemical forms such as metallic chromium Cr(0), Cr(III) or Cr(VI). These are used in, or found in, a wide variety of products including steel products, dyes and paints, chrome plating and magnetic tape [211]. Chromium can be released into the environment (air, water and soil) through use or disposal of such products, and also through mining and various other industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of chromium are the background sources of exposure to the general population. People may be exposed to higher chromium concentrations in occupational settings where chromium-containing products are manufactured or used [211].

Possible Health Effects

The human health effects of chromium depend on the dose, the form of chromium present in the environment, the length and timing of exposure, and other physiological factors. Chromium (III) is an essential micro-nutrient, whereas chromium (VI) is considerably more toxic. However, background concentrations of chromium (VI) in humans are not known to cause any adverse health effects [211]. Under a high dose long-term exposure scenario, such as in the case of accidental releases and unusual occupational exposures, chromium (VI) may cause adverse health effects such as irritations to eyes, skin, mucous membranes and nose, and problems with the digestive system [211].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations for chromium ranged from 0.9 μg/L to 4.6 μg/L. There were no differences in concentrations apparent between regions or across age groups (Figure 65). The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

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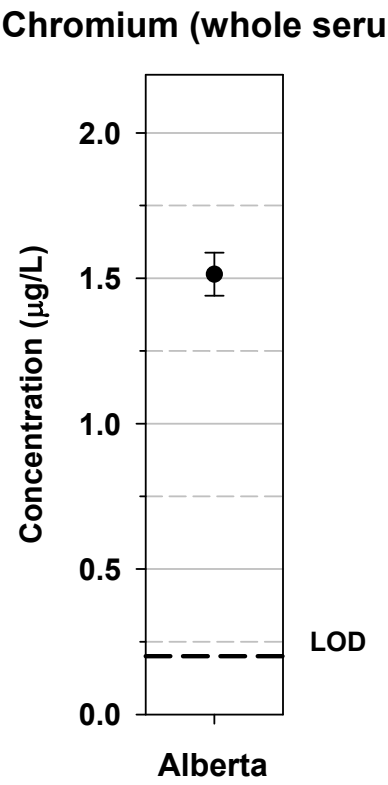

Figure 65

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Lead (Pb)

GENERAL INFORMATION

Sources

Lead (Pb) is a naturally-occurring heavy metal that is widely distributed in the environment. It is not considered a nutrient, rather it is considered as a toxic trace element. Lead has historically been present in a wide variety of industrial and commercial products such as paints, gasoline, plumbing fixtures, storage batteries, leaded glass, leaded crystal, and radiation shielding materials [212,213,214]. Lead can be released into our homes and into the environment (air, water and soil) through use or disposal of such products, and also through mining, and various industrial processes [215].

People are exposed to lead in their houses and workplaces via dust, particularly in older houses where lead-based paints are exposed, from drinking water coming into contact with old plumbing containing lead, and potentially from consumer products [214,215,216,217,218]. Absorption, ingestion and inhalation of air and water containing trace concentrations of lead are common sources of minor exposure to the general population. Another minor source of lead exposure is through food or drink, as it may be leached from lead-based glassware and ceramic used for storing, preparing and serving food. Like other metals, lead is also present in human breast milk and can cross the placenta [219,220,221]. In these ways, lead may be passed to the fetus and to infants during pregnancy and lactation, respectively [219,220,221]. However, breastfeeding is encouraged due to the many associated health-benefits, and these outweigh any known risks from lead for the general population [54,222].

Exposure to lead from various consumer products is regulated in Canada [223]. Since the early 1970s, the amount of lead in paints, gasoline, and solders were limited by Canadian law, and lead was phased out in these products by 1990. As a result, lead exposure to Canadians has decreased since the 1970s. According to Health Canada's blood-lead level guidelines for children and women of reproductive age, concentrations ≥ 100 μg/L are considered a "level of concern" [224,225].

Possible Health Effects

Lead is rapidly absorbed and distributed in the body via the blood, and can be slowly excreted through urine and breast milk. The human health effects of lead are diverse, and can depend on the dose, the length of exposure, and the timing of the exposure. At high doses, such as through unusual occupational exposures, lead is well documented to be a human neurotoxin, and may cause anemia, miscarriage, still birth, and several adverse effects on the motor and sensory systems, reproductive and immune systems, of fetus/infants [212,213,216,226, 227,228].

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BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Lead concentrations in blood serum of pregnant Albertan women were largely below quantification limits (< 0.2 μg/L), except for a few samples that ranged from 0.2 μg/L to 1.0 μg/L. In the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third CDC report), the geometric mean of lead concentrations in whole blood samples of females was 1.2 μg/dL (equivalent to 12 μg/L) [27]. This cannot be directly compared with the present study because serum is known to contain only a small fraction (1%) of total lead, leaving 99 % of the lead in red blood cells [229,230], which were excluded from the analysis here due to limitations in our study design.

Although too few samples were above detection limits in central and northern Alberta to allow detailed reporting, analysis of samples from Southern Alberta yielded noteworthy results due to the different manner by which samples were pooled by month to examine seasonal trends. Significantly higher lead concentrations were detected among all age groups in January, and in July for the age group ≤25 (Figure 66). The source of this temporal variability is unknown.

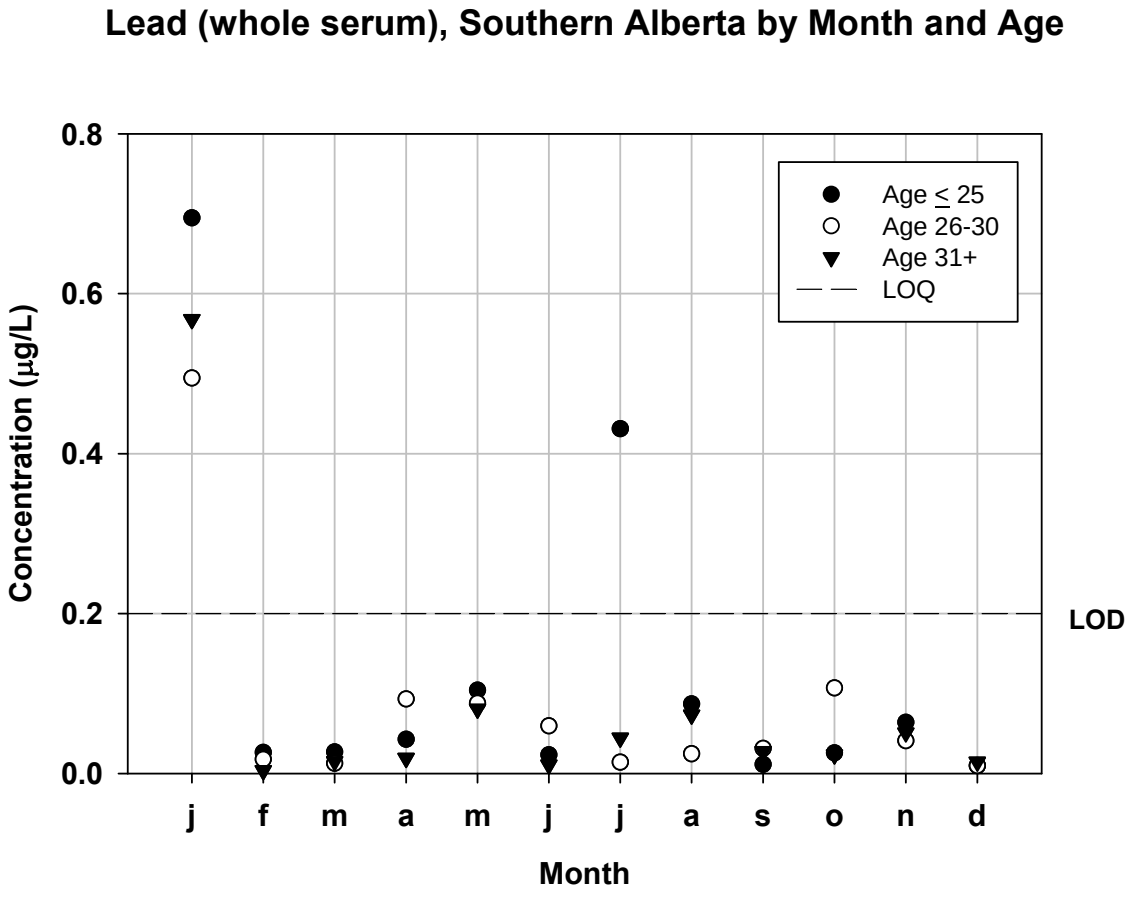

Figure 66

© 2008 Government of Alberta

Mercury (Hg)

GENERAL INFORMATION

Sources

Mercury is a naturally occurring chemical element that enters the environment from natural processes such as weathering of rocks and minerals, and volcanic activity. In the environment, it exists in several chemical forms including elemental, inorganic and organic mercury. Elemental and inorganic mercury compounds are used in or found in a wide variety of industrial, commercial and medicinal products such as electrical instruments, thermometers, batteries, cosmetics, dental fillings, antiseptics, laxatives, eye-drops and nasal sprays [231]. Mercury can be released into the environment (air, water and soil) through use or disposal of such products, burning of coal and waste, and also through mining and various other industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of mercury are the background sources of exposure to the general population. People may be exposed to higher mercury concentrations in occupational settings where mercury-containing products are manufactured or used [231].

Possible Health Effects

The human health effects of mercury depend on the dose, the form of mercury present in the environment, the length and timing of exposure, and other physiological factors. Background concentrations of elemental and inorganic mercury in humans are not known to cause any adverse health effects [231]. Under a high dose long-term exposure scenario, such as in the case of accidental releases and unusual occupational exposures, 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 [231].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration for Hg ranged from 0.2 μg/L to 0.9 μg/L. Concentrations depended on age as shown in Figure 67. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn lower than those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES, 2001-2002) (Third Report, CDC), the geometric mean of mercury concentrations in whole blood of females was 0.8 μg/L [27].

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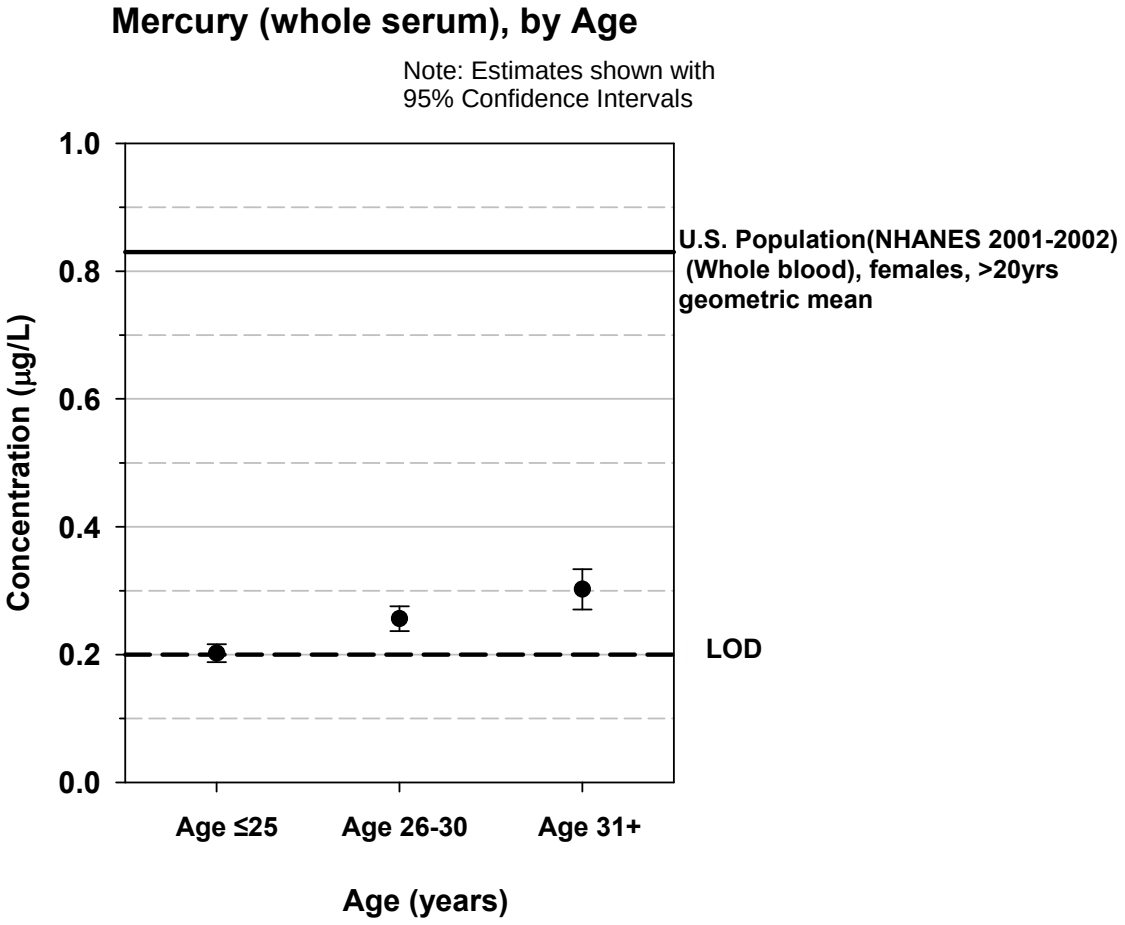

Figure 67

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Silver (Ag)

GENERAL INFORMATION

Sources

Silver (Ag) is a naturally occurring chemical element that enters the environment through natural processes, including the natural weathering of rocks [232,233]. In the environment, silver exists in various chemical forms including elemental silver, silver nitrate, silver chloride, silver sulphide and silver oxide. These silver compounds may also be used in a wide variety of industrial and commercial products such as jewellery, silverware, photographic materials, coins, dental fillings, electronic equipment, medicines and medical devices [232,233,234,235,236]. Silver can be released into our homes and into the environment (air, water and soil) through use or disposal of such products, through mining, and various industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace levels of silver are the common sources of background silver exposure to the general population.

Possible Health Effects

The human health effects of silver depend on the dose, the form of silver to which we are exposed, the length and timing of exposure, and other physiological factors. At background exposure concentrations, silver is not known to cause any adverse health effect [233]. Under a high dose long-term exposure scenario, such as in the case of accidental releases and unusual occupational exposures, silver may cause adverse health effects such as arygria (a blue-grey staining of the skin, eyes, nose and other body tissues), skin rashes, headaches, and coughing or other respiratory problems [233,237,238].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations for silver ranged from 0.2 μg/L to 0.6 μg/L. Concentrations depended on region and age as shown in Figure 68. The concentrations in the north were greater than those in the central region, and in turn those in the central region were greater than those in the south. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group, which were in turn lower than those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

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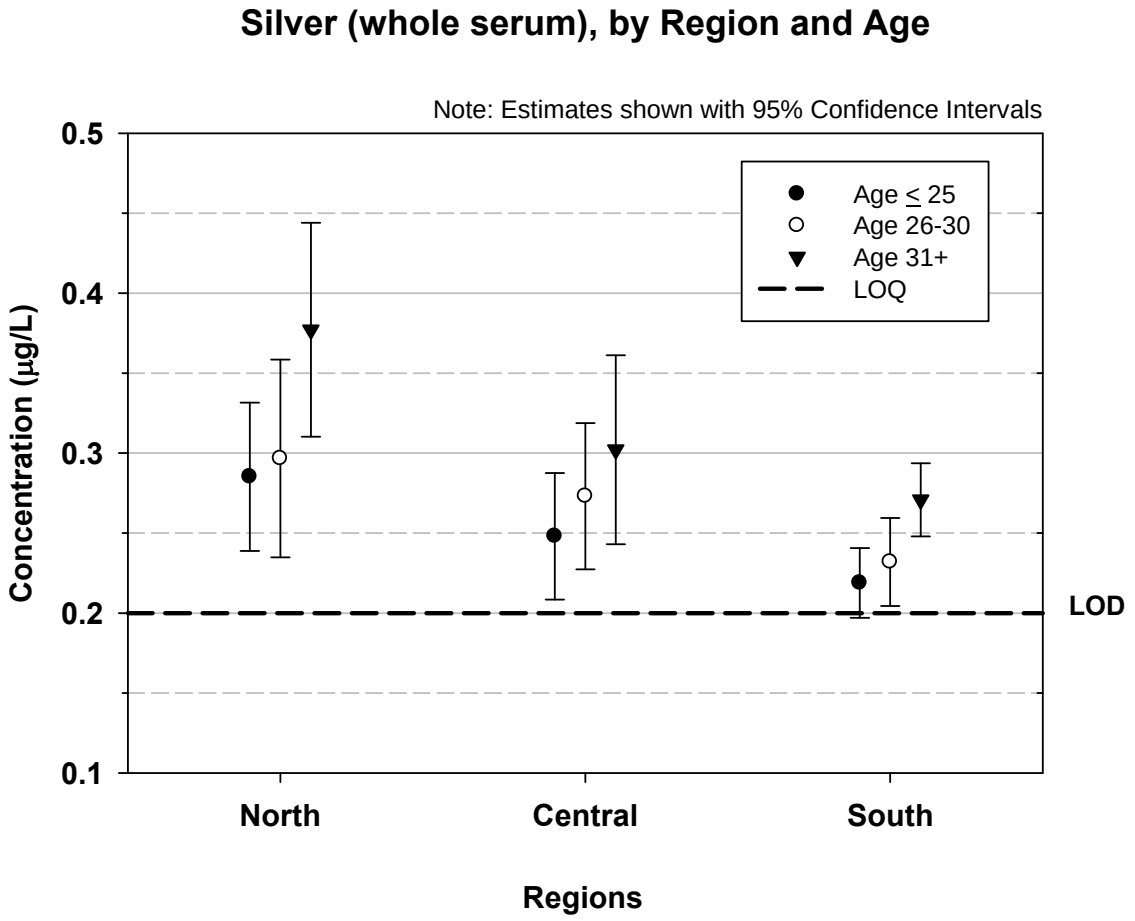

Figure 68

© 2008 Government of Alberta

Vanadium (V)

GENERAL INFORMATION

Sources

Vanadium (V) is a naturally occurring chemical element that can be found in mineral rocks. In the environment, it exists in the form of various vanadium compounds such as vanadium oxide, vanadium sulphate and vanadium chloride. These vanadium compounds are used in or found in a wide variety of products including steel products, aircraft engines, automobile parts, rubber, plastics and ceramics [239]. Vanadium can be released into the environment (air, water and soil) through use or disposal of such products, and also through mining, burning of fuel oils, and various other industrial processes. Absorption, ingestion and inhalation of food, water, soil and air containing trace concentrations of vanadium are the background sources of exposure to the general population. People may be exposed to higher vanadium concentrations in occupational settings where vanadium-containing products are manufactured or used [239].

Possible Health Effects

The human health effects of vanadium depend on the dose, the form of vanadium present in the environment, the length and timing of exposure, and other physiological factors. Background concentrations of vanadium in humans are not known to cause any adverse health effects [239]. Under a high dose long-term exposure scenario, such as in the case of accidental releases and unusual occupational exposures, vanadium may cause adverse health effects such as eye irritation, sore throat and coughing or other respiratory problems [239].

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration for vanadium ranged from 0.2 μg/L to 0.4 μg/L. Concentrations depended on region as shown in Figure 69. The concentrations in the north were equal to those in the central region, and in turn those in the central region were lower than those in the south. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

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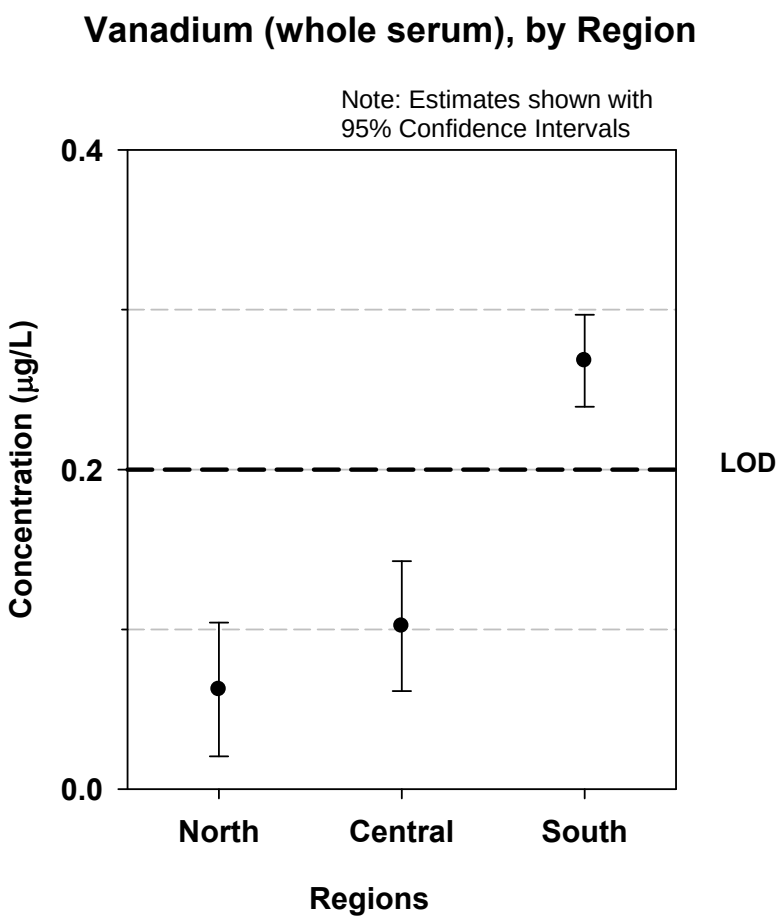

Figure 69

© 2008 Government of Alberta

Mineral Micronutrients

Mineral micronutrients are metals that are needed is small quantities to sustain life. As expected, all micronutrients examined in this study were detectable in serum, and all analytical results have been reported in the subsequent sections. Although high exposure to micronutrients may also represent health risks, and Health Canada sets allowable limits for many of these metals in natural health products, the normal public health focus is to ensure that populations are receiving an adequate supply. Thus, exposure sources and possible health effects arising from very high exposure to mineral micronutrients have not been reviewed here, and nutritional aspects are beyond the scope of the current report. In general, however, humans ingest mineral micronutrients in the diet, including fruits and vegetables, animal products, and drinking water. The ultimate source of mineral micronutrients is soil and water, thus human serum concentrations are anticipated to vary by region with local soil and drinking water chemistry.

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Boron (B)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean boron concentration in blood serum of pregnant Albertan women ranged from 13 μg/L to 34 μg/L. This is somewhat lower than previously reported concentrations of 68 μg/L (geometric mean) in women's blood serum samples collected in an urban area in Japan [240].

Boron concentrations in blood serum were dependant on age and geographic region (Figure 70). The concentrations in the north were lower than those in the central region and in turn those in the central region were lower than those in the south. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn equal to those in the 31+ age group. The interaction between age and region was not significant suggesting that the shape of the age relationships does not differ by region. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

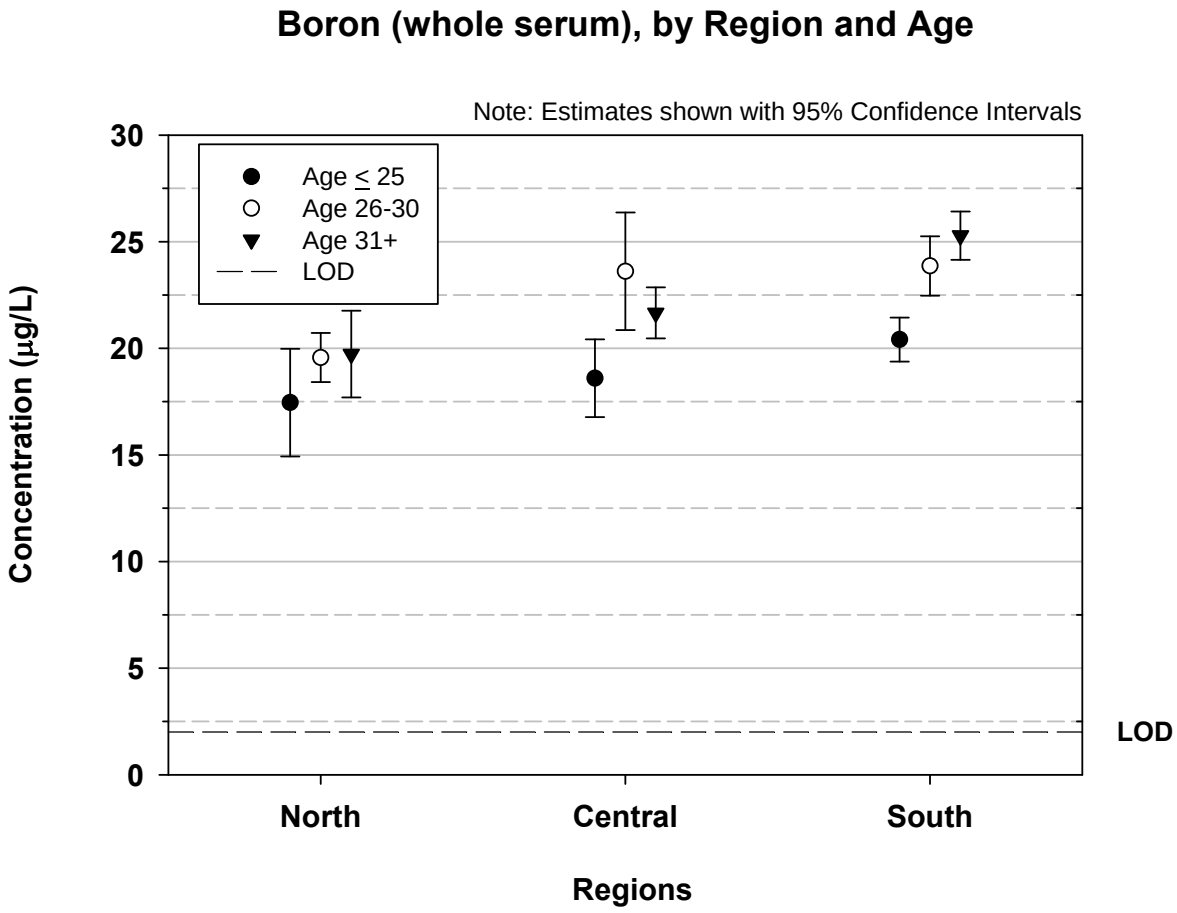

Figure 70

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Cobalt (Co)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations of cobalt (Co) ranged from 0.2 μg/L to 3.6 μg/L. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), cobalt was detectable in urine samples of U.S. population [27], but those results cannot be compared with the present results for serum samples.

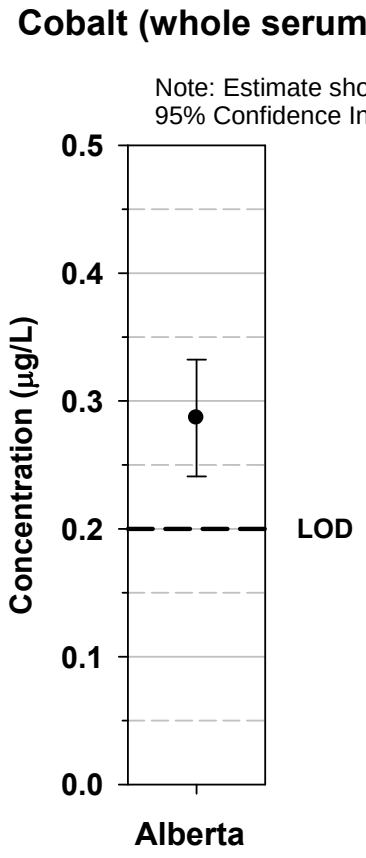

Figure 71

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Copper (Cu)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations of copper (Cu) ranged from 1700 µg/L to 2300 µg/L. Concentrations depended on region and age as shown in Figure 72. The concentrations in the north were equal to those in the central region and in turn those in the central region were greater than those in the south. The concentrations in the 26-30 year age group were lower than those in the ≤25 year age group which were in turn equal to those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

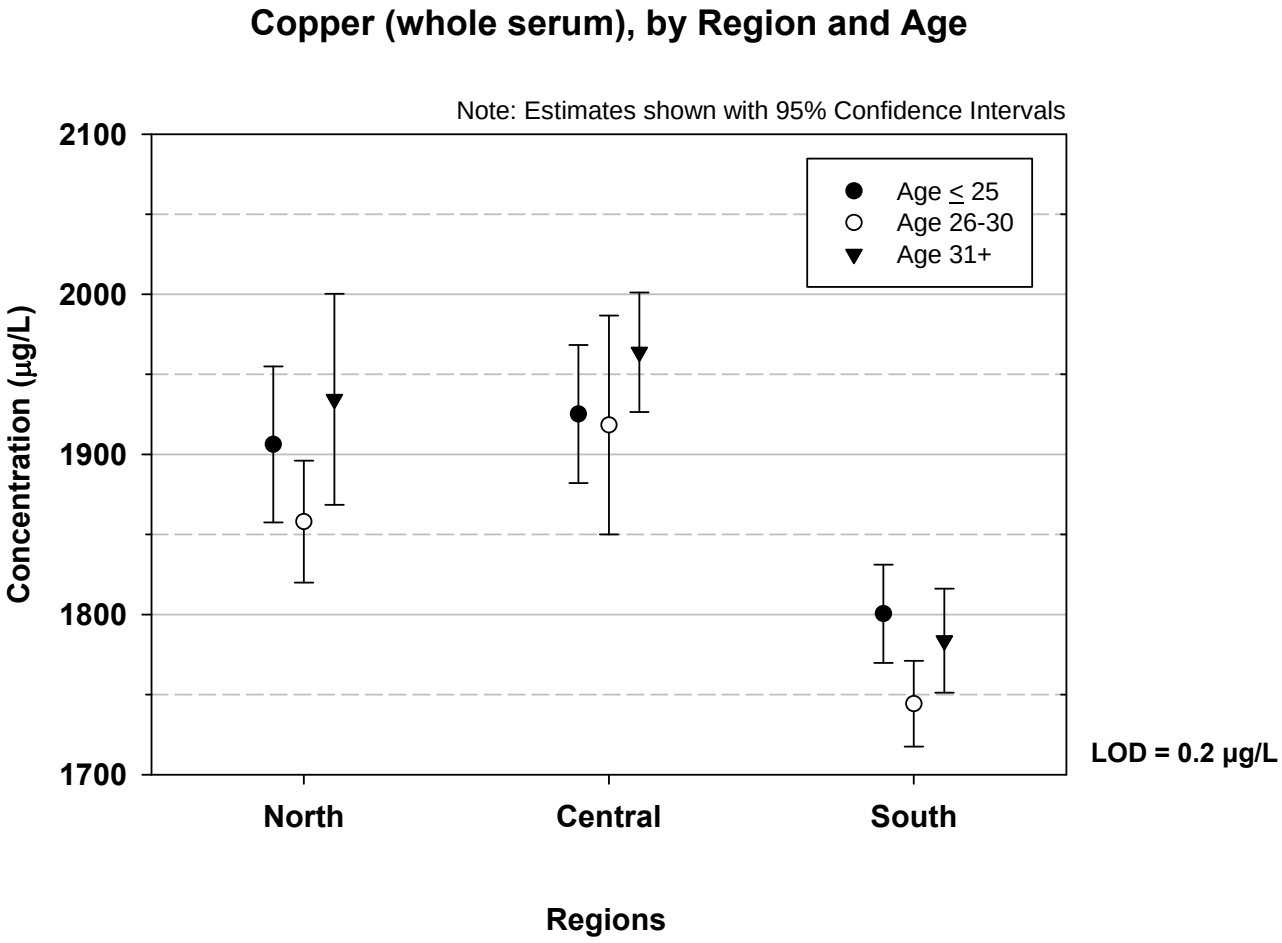

Figure 72

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Iron (Fe)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations for iron (Fe) ranged from 976 μg/L to 2320 μg/L. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

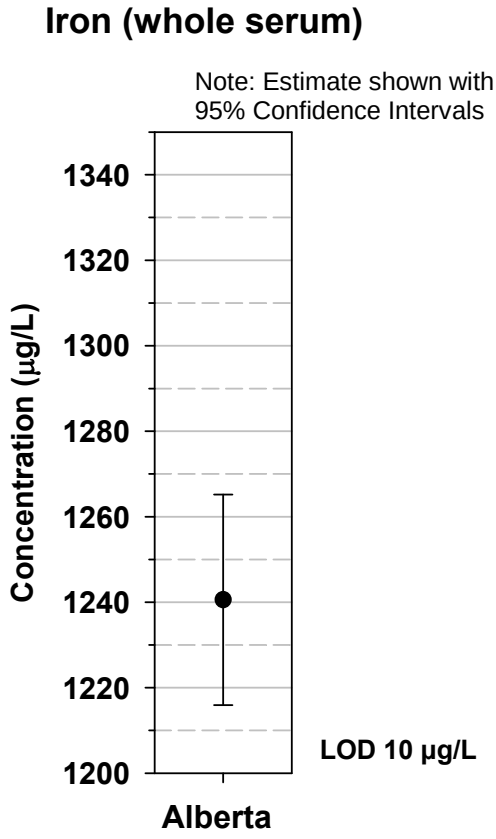

Figure 73

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Manganese (Mn)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations of manganese (Mn) ranged from 2 μg/L to 21 μg/L. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

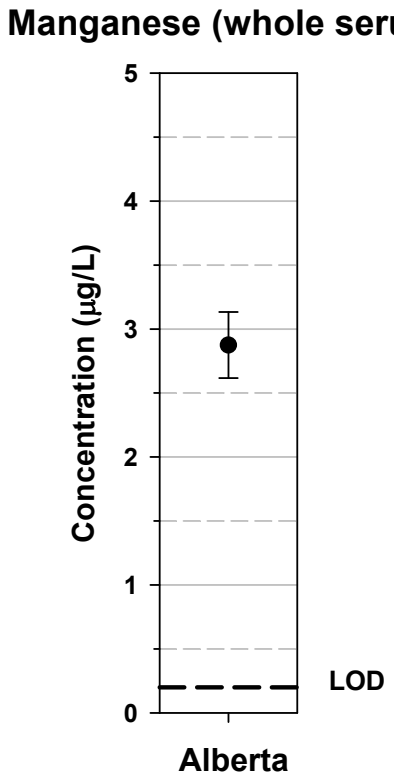

Figure 74

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Molybdenum (Mo)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentrations of molybdenum (Mo) ranged from 1.1 μg/L to 4.3 μg/L. Concentrations depended on age as shown in Figure 75. The concentrations in the ≤25 year age group were lower than those in the 26-30 year age group which were in turn equal to those in the 31+ age group. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed. In the U.S. National Health and Nutrition Survey (NHANES 2001-2002) (Third Report, CDC), molybdenum was detectable in urine samples of U.S. population [27], but those results cannot be compared with the present results for serum.

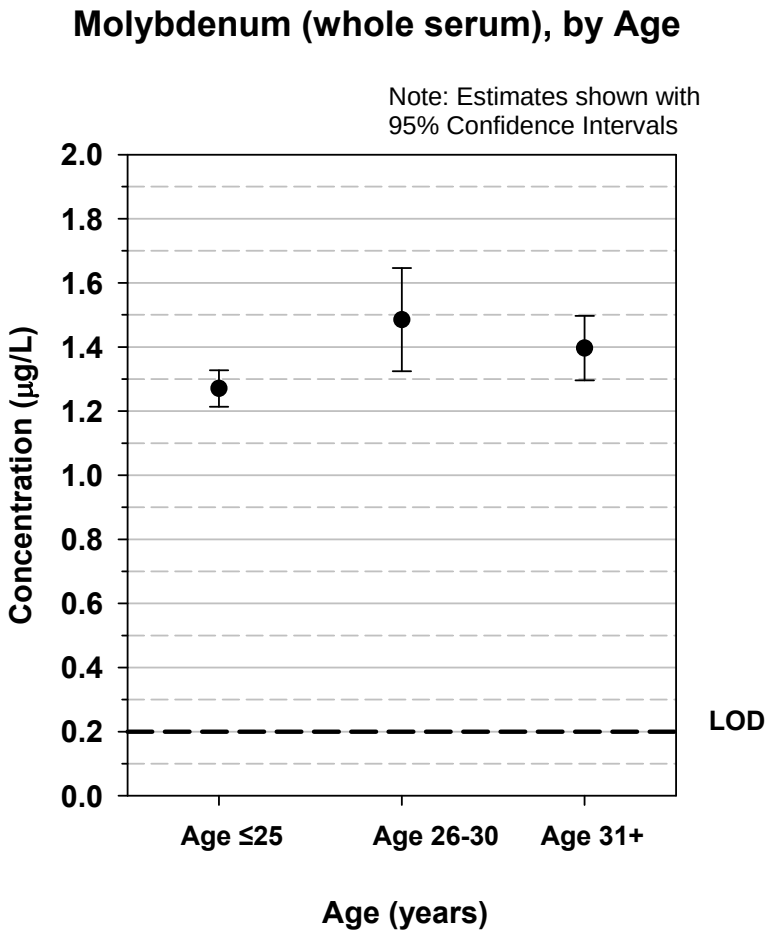

Figure 75

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Nickel (Ni)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration of Nickel (Ni) ranged from 0.4 µg/L to 5.5 µg/L. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

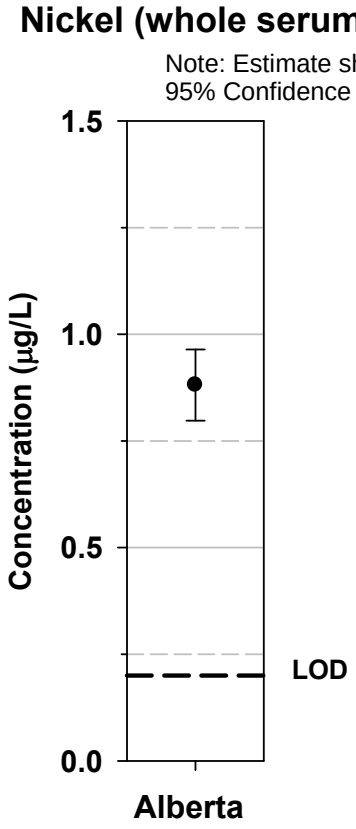

Figure 76

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Selenium (Se)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration of selenium (Se) ranged from 130 µg/L to 180 µg/L. There were no differences in concentrations apparent between regions or across age groups. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

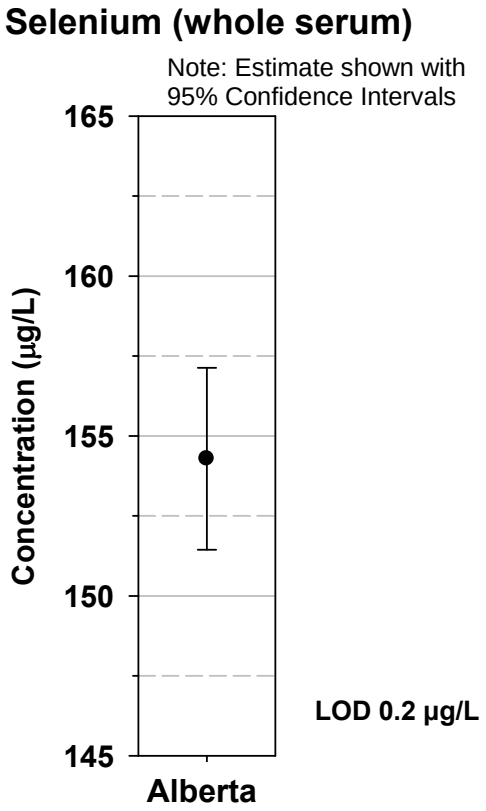

Figure 77

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Zinc (Zn)

BLOOD SERUM CONCENTRATIONS IN PREGNANT WOMEN IN ALBERTA

Concentrations and Trends.

Overall, mean concentration of zinc (Zn) ranged from 1200 µg/L to 1560 µg/L. Concentrations depended on region, but not age, as shown in Figure 78. The concentrations in the north were greater than those in the south region and in turn those in the south region were greater than those in the central region. The seasonal variation was examined in the south, but there was no apparent temporal or seasonal trend observed.

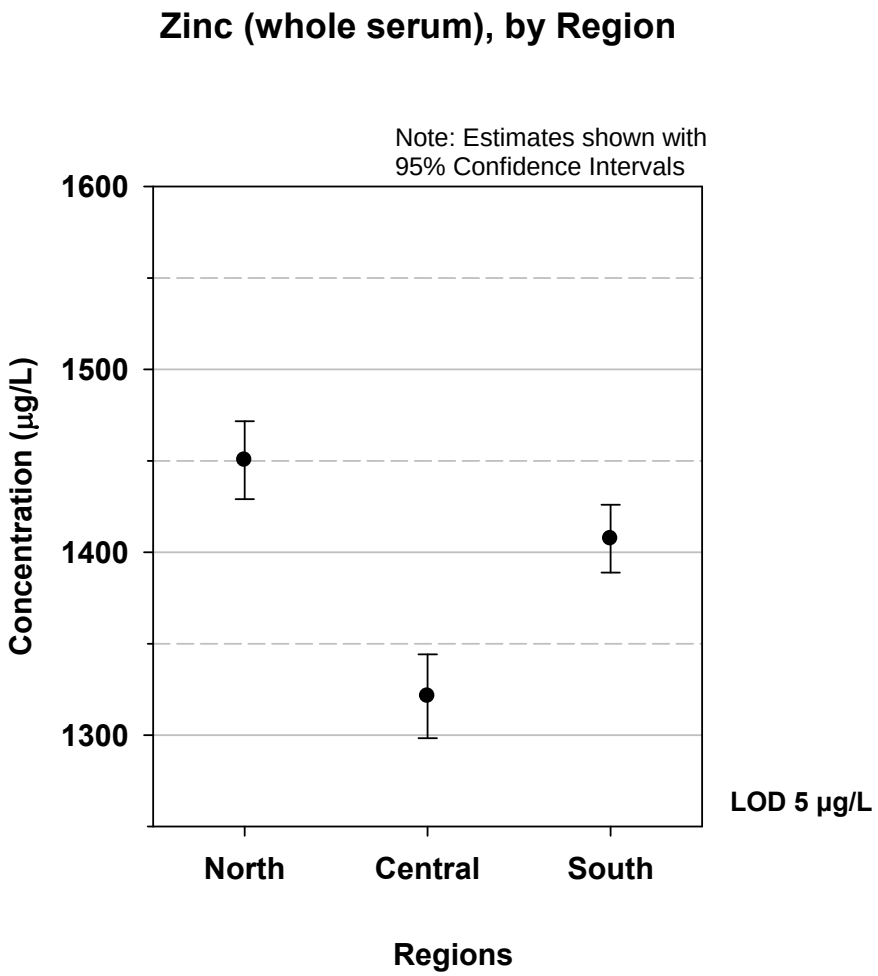

Figure 78

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Summary of the Results and Comparison with Other Studies

Chemical Name	Pregnant Women (Alberta, 2005)	Other Studies
	TOBACCO SMOKE	
Cotinine	Serum cotinine concentrations in pooled samples ranged from 5 to 55 ng/mL	U.S. population, NHANES (1988-1991), males and females, > 17 yrs., smokers, individual samples, Serum cotinine concentrations ranged from 22 to 113 ng/mL [28] U.S. population, NHANES (2001-2002), females, > 20 yrs., non-smokers, individual samples, CDC report, 50th centile 0.03 ng/mL [27]
	PHYTOESTROGENS	
Daidzein	Estimated distribution among individuals, 50th centile**,** 0.3 ng/mL; LOD 0.2 ng/mL	U.S. population, a subset of adults (208 samples, > 20 yrs, 61% females) from NHANES (1988-1994), mean concentration in serum was 3.9 ng/mL [46]

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies	
POLYCHLORINATED DIBENZO-P-DIOXINS AND POLYCHLORINATED DIBENZOFURANS (LIPID ADJUSTED)				
	Estimated distribution among individuals		U.S. population, NHANES (2001-2002), females, > 20 yrs., individual samples, CDC report [27]	
	50th centile	**LOD**	**50th centile**	**LOD**
1,2,3,6,7,8- HxCDD	1.5 pg/g	1.8 pg/g	40 pg/g	9.1 pg/g
1,2,3,4,6,7,8- HpCDD	4.0 pg/g	2.7 pg/g	43 pg/g	10 pg/g
OCDD	31 pg/g	7.4 pg/g	405 pg/g	319 pg/g
1,2,3,7,8- PCDF	0.09 pg/g	1.8 pg/g	<LOD	5.8 pg/g
2,3,4,7,8- PCDF	0.1 pg/g	1.8 pg/g	<LOD	5.5 pg/g
1,2,3,4,7,8- HxCDF	0.1 pg/g	1.9 pg/g	<LOD	6.5 pg/g
1,2,3,6,7,8- HxCDF	0.2 pg/g	2.2 pg/g	<LOD	6.1 pg/g
1,2,3,4,6,7,8- HpCDF	0.5 pg/g	2.4 pg/g	9.3 pg/g	7 pg/g
OCDF	0.09 pg/g	1.8 pg/g	<LOD	21 pg/g

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Chemical Name	Pregnant Women (Alberta, 2005)	Other Studies
	Ranges of mean concentrations	Swan Hills Waste Treatment Centre, long-term follow-up health assessment program (1997-2002), blood serum samples, 2001 survey, [60]
	Lipid Weight	**Mean Concentrations (lipid adjusted)**
1,2,3,6,7,8- HxCDD	2.8 to 23 pg/g	743 pg/g
1,2,3,4,6,7,8- HpCDD	5.5 to 55 pg/g	390 pg/g
OCDD	5.3 to 280 pg/g	3371 pg/g
2,3,4,7,8- PCDF	1.8 to 16 pg/g	34 pg/g
1,2,3,4,7,8- HxCDF	1.8 to 12 pg/g	33 pg/g
1,2,3,6,7,8- HxCDF	1.4 to 16 pg/g	46 pg/g
1,2,3,4,6,7,8- HpCDF	2.7 to 24 pg/g	142 pg/g
OCDF	3.2 to 17 pg/g	48 pg/g

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COPLANAR AND MONO-ORTHOSUBSTITUTED POLYCHLORINATED BIPHENYLS (LIPID ADJUSTED)

Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies	
	Estimated distribution among individuals		U.S. population, NHANES (2001-2002), females, > 20 yrs., individual samples, CDC report [27]	
	50th centile	**LOD**	**50th centile**	**LOD**
PCB 146	0.07 ng/g	0.9 ng/g	<LOD	10.5 ng/g
PCB 156	0.07 ng/g	0.9 ng/g	Not measured	
PCB 158	0.03 ng/g	0.9 ng/g	20.2 ng/g	10.5 ng/g
PCB 170	0.1 ng/g	0.9 ng/g	<LOD	10.5 ng/g
PCB 180	0.6 ng/g	0.9 ng/g	18.3 ng/g	10.5 ng/g
PCB 183	0.05 ng/g	0.9 ng/g	<LOD	10.5 ng/g
PCB 187	0.1 ng/g	0.9 ng/g	<LOD	10.5 ng/g
PCB 194	0.07 ng/g	0.9 ng/g	<LOD	10.5 ng/g
PCB 199	0.07 ng/g	0.9 ng/g	<LOD	10.5 ng/g
			Western Canada Study, Pregnant women from Southern AB (Calgary Health Region, 2001-2003, 209 samples, individual samples) geometric mean of total PCBs, 62 ng/g lipid [74]	

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Chemical Name	Pregnant Women (Alberta, 2005)	Other Studies
	Ranges of mean concentrations	Swan Hills Waste Treatment Centre, long-term follow-up health assessment program (1997-2002), blood serum samples, 2001 survey, [60]
	Whole weight (serum)	**Mean Concentrations (whole weight)**
PCB 156	5.1 to 13 pg/g	0.03 ng/g
PCB 170	5.1 to 21 pg/g	0.05 ng/g
PCB 180	6.3 to 67 pg/g	0.13 ng/g
PCB 183	5.1 to 44 pg/g	0.01 ng/g
PCB 187	5.1 to 62 pg/g	0.04 ng/g
PCB 194	5.3 to 11 pg/g	0.02 ng/g

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies	
ORGANOCHLORINE PESTICIDES (LIPID ADJUSTED)				
	Estimated distribution among individuals		U.S. population, NHANES (2001-2002), females, > 20 yrs., individual samples, CDC report [27]	
	50th centile	**LOD**	**50th centile**	**LOD**
Mirex	4.2 ng/g	8.9 ng/g	<LOD	10.5 ng/g
DDE	14 ng/g	23 ng/g	256 ng/g	8.3 ng/g
			Western Canada Study, Pregnant women from Southern AB (Calgary Health Region, 2001-2003, 209 samples, individual samples) geometric mean 110 ng/g lipid [74]	
HCB	3.7 ng/g	18 ng/g	U.S. population, NHANES (2001-2002), females, > 20 yrs., individual samples, CDC report [27]	
			50th centile	**LOD**
			<LOD	31.4 ng/g

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies
HCB			Western Canada Study, Pregnant women from Southern AB (Calgary Health Region, 2001-2003, 209 samples, individual samples) geometric mean 14 ng/g lipid [74]

POLYBROMINATED DIPHENYLETHERS (LIPID ADJUSTED)

	Estimated distribution among individuals		U.S. Study (California), pregnant women (24 samples were selected from a birth cohort study of 601 samples), individual study, >20yrs. [123]
	50th centile	**LOD**	**50th centile**
BDE 28	0.1 ng/g	0.07 ng/g	
BDE 47	3.7 ng/g	0.2 ng/g	11 ng/g
BDE 66	0.008 ng/g	0.2 ng/g	
BDE 85	0.03 ng/g	0.3 ng/g	0.3 ng/g
BDE 99	0.6 ng/g	0.3 ng/g	2.9 ng/g
BDE 100	0.8 ng/g	0.2 ng/g	1.8 ng/g

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies
	Estimated distribution among individuals		U.S. Study (California), pregnant women (24 samples were selected from a birth cohort study of 601 samples), individual study, >20yrs. [123]
	50th centile	**LOD**	**50th centile**
BDE 153	2.1 ng/g	0.2 ng/g	1.5 ng/g
BDE 154	0.05 ng/g	0.2 ng/g	0.3 ng/g
			U.S. Study (Texas), Pooled Samples of 100, 2003, serum samples [241]
	Concentrations (weighted mean) in pooled samples		**Concentrations**
BDE 28	1.5 ng/g		1.3 ng/g
BDE 47	43 ng/g		32 ng/g
BDE 66	0.2 ng/g		0.3 ng/g
BDE 85	1.0 ng/g		Not tested
BDE 99	20 ng/g		8.4 ng/g
BDE 100	11 ng/g		5.7 ng/g

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies	
			U.S. Study (Texas), Pooled Samples of 100, 2003, serum samples [242]	
	Concentrations (weighted mean) in pooled samples		**Concentrations**	
BDE 153	14 ng/g		12 ng/g	
BDE 154	1.5 ng/g		0.8 ng/g	
	PERFLUORINATED COMPOUNDS			
			U.S. population NHANES (2001-2002) (14 pooled samples with 34 individuals), serum samples, >12 yrs, females, NHW: non-Hispanic whites, [147]	
	Concentrations (weighted mean) in pooled samples	**LOD**	**Concentrations (weighted mean) in pooled samples**	**LOD**
PFHxS	2.1 ng/g	0.5 ng/g	4.3 ng/g	0.1 ng/g
PFOS	7.4 ng/g	0.1 ng/g	24 ng/g	0.05 ng/g

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies	
			U.S. population NHANES (2001-2002) (14 pooled samples with 34 individuals), serum samples, >12 yrs, females, NHW: non-Hispanic whites, [147]	
	Concentrations (weighted mean) in pooled samples	**LOD**	**Concentrations (weighted mean) in pooled samples**	**LOD**
PFOA	2.6 ng/g	0.02 ng/g	3.9 ng/g	0.05 ng/g
PFNA	0.2 ng/g	0.05 ng/g	0.5 ng/g	0.05 ng/g
	PHENOLS			
	Estimated distribution among individuals			
	50th centile	**LOD**		
Bisphenol A	0.02 ng/g	0.01 ng/g	U.S. population, subset from NHANES (2003-2004), 2517 people, individual study**,** geometric mean of urine concentration of BPA in females was 2.4 ng/g [169]	

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies
Bisphenol A			Greenpeace study in the Netherlands, individual study, BPA concentration ranged from 0.5 to 1.7 ng/g in maternal serum [167]
	Estimated distribution among individuals		
	50th centile	**LOD**	
Nonylphenol	3.0 ng/g	2 ng/g	Greenpeace study in the Netherlands, individual study, NP concentration ranged from 0.6 to 16 ng/g in maternal serum [187]
	MERCURY		
	Estimated distribution among individuals		
	50th centile	**LOD**	
Methylmercury	0.02 ng/g	0.03 ng/g	Swedish study, females, 142 samples, mean MeHg concentration in serum samples was 0.5 ng/g [200]

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Chemical Name	Pregnant Women (Alberta, 2005)		Other Studies
		METALS	
	Estimated distribution among individuals		
	50th centile	**LOD**	
Mercury	0.05 μg/L	0.2 μg/L	U.S. population, NHANES (2001-2002), females, > 20 yrs., non-smokers, individual whole blood samples, CDC report [27] geometric mean 0.8 μg/L, 95th centile 4.6 μg/L, 50th centile 0.7 μg/L
Lead	0.005 μg/L	0.2 μg/L	U.S. population, NHANES (2001-2002), females, > 20 yrs., non-smokers, individual whole blood samples, CDC report [27] geometric mean 12 μg/L, 95th centile 36 μg/L, 50th centile 11 μg/L

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Glossary of Terms

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 which are not normally found in the environment.
Naturally Occurring Chemicals	Chemicals that are present or produced naturally in the environment. Some man-made 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" – can be easily dissolved 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	Limit of detection, or limit of quantification

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Conversion of Measurement Units

Units	Abbreviations	Values/Conversions
litre	L	
decilitre	dL	10^{-1} L
millilitre	mL	10^{-3} L
microlitre	µL	10^{-6} L
gram	g	
microgram	µg	10^{-6} g
nanogram	ng	10^{-9} g
picogram	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/mL 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.	

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