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EXPOSURE TO ENVIRONMENTAL CONTAMINANTS IN NUNAVIK: PERSISTENT ORGANIC POLLUTANTS AND NEW CONTAMINANTS OF CONCERN

NUNAVIK REGIONAL BOARD OF HEALTH AND SOCIAL SERVICES
RÉGIE RÉGIONALE DE LA SANTÉ ET DES SERVICES SOCIAUX NUNAVIK
Québec

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BACKGROUND OF THE NUNAVIK INUIT HEALTH SURVEY

The monitoring of population health and its determinants is essential for the development of effective health prevention and promotion programs. More specifically, monitoring must provide an overall picture of a population's health, verify health trends and how health indicators vary over distance and time, detect emerging problems, identify priority problems, and develop possible health programs and services that meet the needs of the population studied.

The extensive survey conducted by Santé Québec in Nunavik in 1992 provided information on the health status of the Nunavik population (Santé Québec, 1994). The survey showed that health patterns of the population were in transition and reflected important lifestyle changes. Effectively, the Inuit population has undergone profound sociocultural, economic, and environmental changes over the last few decades. The Inuit have changed their living habits as contact with more southerly regions of Quebec increased. A sedentary lifestyle, the switch to a cash-based domestic economy, the modernization of living conditions and the increasing availability and accessibility of goods and foodstuffs imported from southern regions have contributed to these changes. These observations suggest the need for periodic monitoring of health endpoints of Nunavik Inuit to prevent the negative impact of risk factor emergence and lifestyle changes on subsequent morbidity and mortality from major chronic diseases.

In 2003, the Nunavik Regional Board of Health and Social Services (NRBHSS) decided to organize an extensive health survey in Nunavik in order to verify the evolution of health status and risk factors in the population. The NRBHSS and the Ministère de la Santé et des Services sociaux (MSSS) du Québec entrusted the Institut national de santé publique du Québec (INSPQ) with planning, administering and coordinating the survey. The INSPQ prepared the survey in close collaboration with the Unité de recherche en santé publique (URSP) of the Centre hospitalier universitaire de Québec (CHUQ) for the scientific and logistical component of the survey. The Institut de la statistique du Québec (ISQ) participated in methodology development, in particular the survey design.

The general aim of the survey was to gather social and health information on a set of themes including various health indicators, physical measurements, and social, environmental and living conditions, thus permitting a thorough update of the health and well-being profile of the Inuit population of Nunavik. The survey was designed to permit a comparison of the 2004 trends with those observed in 1992. Data collected in 2004 also allowed researchers to compare the Inuit to other Quebecers.

Target population

The health survey was conducted among the Inuit population of Nunavik from August 27 to October 1, 2004. According to the 2001 Canadian census, the fourteen communities of Nunavik have a total of 9632 inhabitants, 91% of whom identified themselves as Inuit. The target population of the survey was permanent residents of Nunavik, excluding residents of collective dwellings and households in which there were no Inuit aged 18 years old or older.

Data collection

Data collection was performed on the Canadian Coast Guard Ship Amundsen, thanks to a grant obtained from the Canadian Foundation for Innovation (CFI) and the Network of Centres of Excellence of Canada (ArcticNet). The ship visited the fourteen villages of Nunavik, which are coastal villages. The study was based on self-administered and interviewer-completed questionnaires. The study also involved physical and biological measurements including clinical tests. The survey was approved by the Comité d'éthique de la recherche de l'Université Laval (CERUL) and the Comité d'éthique de santé publique du Québec (CESP). Participation was voluntary and participants were asked to give their written consent before completing interviews and clinical tests. A total of 677 private Inuit households were visited by interviewers who met the household respondents to complete the identification chart and the household questionnaire. A respondent was defined as an Inuit adult able to provide information regarding every member of the household. The identification chart allowed demographic information to be collected on every member of the household. The household questionnaire served to collect information on housing, environment, nutrition and certain health indicators especially regarding young children.

All individuals aged 15 or older belonging to the same household were invited to meet survey staff a few days later, on a Canadian Coast Guard ship, to respond to an interviewer-completed questionnaire (individual questionnaire) as well as a self-administered confidential

questionnaire. Participants from 18 to 74 years of age were also asked to complete a food frequency questionnaire and a 24-hour dietary recall, and to participate in a clinical session. The individual questionnaire aimed to collect general health information on subjects such as health perceptions, women's health, living habits and social support. The confidential questionnaire dealt with more sensitive issues such as suicide, drugs, violence and sexuality. During the clinical session, participants were invited to answer a nurse-completed questionnaire regarding their health status. Then, participants had a blood sample taken and physical measurements were performed including a hearing test, anthropometric measurements, an oral glucose tolerance test (excluding diabetics) and toenail sampling. Women from 35 to 74 years of age were invited to have a bone densitometry test. Finally, participants aged 40 to 74 could have, after consenting, an arteriosclerosis screening test as well as a continuous measure of cardiac rhythm for a two-hour period.

Survey sampling and participation

The survey used a stratified random sampling of private Inuit households. The community was the only stratification variable used. This stratification allowed a standard representation of the target population. Among the 677 households visited by the interviewers, 521 agreed to participate in the survey. The household response rate is thus 77.8%. The individual response rates are obtained by multiplying the household participating rate by the individual collaboration rate since the household and individual instruments were administered in sequence. The collaboration rate corresponds to the proportion of eligible individuals who agreed to participate among the 521 participating households. In this survey, about two thirds of individuals accepted to participate for a response rate in the area of 50% for most of the collection instruments used in the survey. A total of 1056 individuals signed a consent form and had at least one test or completed one questionnaire. Among them, 1006 individuals answered the individual questionnaire, 969 answered the confidential questionnaire, 925 participated in the clinical session, 821 had a hearing test, 778 answered the food frequency questionnaire, 664 answered the 24-hour dietary recall, 282 had an arteriosclerosis test, 211 had a continuous measure of their cardiac rhythm for a two-hour period and 207 had a bone densitometry test. More details on the data processing are given in the Methodological Report.

INTRODUCTION [1]

Human exposure to environmental contaminants is a well-known phenomenon in the Canadian Arctic. The Inuit of Nunavik are exposed to a plethora of toxic substances that are carried from southern to northern latitudes by oceanic and atmospheric transport and biomagnified in Arctic food webs. As the Inuit traditional diet comprises large amounts of tissues from marine mammals, fish and terrestrial wild game, the Inuit are more exposed to metals and persistent organic pollutants (POPs) than populations living in southern regions. Metals of interest include mercury, lead and cadmium whereas organic contaminants of concern are classical POPs and new contaminants of interest such as perfluorooctanesulfonate (PFOS), halogenated phenolic compounds (HPCs) and polybrominated diphenyl ethers (PBDEs).

The traditional suite of legacy POPs comprises polychlorinated dibenzo *p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs) and chlorinated pesticides. Due to their lipophilic properties, these compounds accumulate in fatty tissues. They are also found in human milk and are able to cross the placental barrier. Several studies have shown that *in utero* exposure to PCBs may have detrimental effects on several aspects of cognitive development (Darvill et al., 2000; Jacobson et al., 1990; Patandin et al., 1999; Stewart et al., 2003; Walkowiak et al., 2001). Some of these organic compounds have endocrine disrupting properties and have the capacity of altering natural hormone-signalling pathways such as thyroid hormones (Brouwer et al., 1998; Cheek et al., 1999; Lans et al., 1993), estrogens and androgens (Bonefeld-Jorgensen et al., 2001). POPs have also been found to be carcinogenic, hepatotoxic and have reproductive and immunotoxic effects (Safe, 1994; Schecter et al., 2006; Schmidt, 1999). *In utero* exposure to POPs was shown to increase the risk of acute infection in the first year of life of Inuit infants (Dallaire et al., 2004).

The Inuit are exposed to these compounds through the consumption of contaminated traditional foods, particularly seal and beluga fats. Recently, declining trends in POP body burdens were observed among Inuit newborns (Dallaire et al., 2003) and Caucasian newborns

[1] For ease of readability, the expression "Inuit" is used throughout the theme paper to define the population under study even though a small percentage of individuals surveyed identified themselves as non-Inuit. Refer to "Background of the Health Survey" for further details regarding the definition of the target population.

residing along the Lower North Shore of the St. Lawrence River (Dallaire et al., 2002). Similar observations were reported among Arctic wildlife species (Braune et al., 2005). This decline could be explained in part by the implementation of the Stockholm Convention on POPs aiming to reduce and eventually eliminate the release of these legacy chemicals into the environment.

Although concentrations of organochlorines in human, wildlife and environmental matrices are decreasing, other compounds manufactured in industrialized regions of North America and Europe have been measured recently in environmental media, in tissues of wildlife species and in human tissues from the Arctic. POPs emerging more recently include perfluorooctanesulfonate (PFOS), halogenated phenolic compounds (HPCs) and brominated flame retardants such as polybrominated diphenyl ethers (PBDEs). PFOS is an end-product of fluorochemicals that have been produced since 1970 but have received less attention until now due to the difficulty in analytic measurements (Hekster et al., 2003). Animal studies have demonstrated that PFOS may alter thyroid hormone homeostasis, affect fatty acid transport and metabolism as well as membrane function (Lau et al., 2003; Thibodeaux et al., 2003). The presence of PFOS in biological matrices was reported in several wildlife species of the Arctic (Bossi et al., 2005; Giesy & Kannan, 2001; Smithwick et al., 2005; Tomy et al., 2004).

Halogenated phenolic compounds that include metabolites of PCBs (hydroxylated and methylsulfone), pentachlorophenol and other chlorophenols have also been recently measured in environmental media and tissues of wildlife from circumpolar regions (Sandala et al., 2004; Verreault et al., 2005). Significant amounts of some of these compounds have been detected in the blood of Inuit adults (Sandau, 2000) and in cord blood from Inuit newborns in Nunavik (Sandau et al., 2002). In the latter study, significant associations were reported between thyroid hormone levels and a composite measure of PCP and hydroxylated-PCBs metabolites.

PBDEs are a class of brominated flame retardants that are extensively used in various industrial and commercial products such as textiles and electronic devices (Alaee et al., 2003). They share similar physical and chemical properties with PCBs, PCDDs and PCDFs, which suggests that they may induce similar detrimental developmental, hormonal and reproductive effects (Birnbaum & Staskal, 2004; Gill et al., 2004; Hooper & McDonald, 2000; McDonald, 2002). An increasing concentration of PBDEs has been observed in human milk from the Canadian Arctic, although these levels are still lower than those in southern populations (Pereg et al., 2003; Ryan & Van Oostdam, 2004) and those in the Faroe Islands in the last decade (Fangstrom et al., 2005).

Given efforts during the past decade through the Stockholm Convention to reduce global use of metals and POPs and to reduce the exposure of Northern Aboriginal people to them, periodic re-assessments of exposure are needed to evaluate the efficiency of implemented programs and information campaigns (i.e. ban of lead shots, earlier publicity campaigns, etc.). As several new compounds sharing similar properties with POPs are entering the Arctic environment and its food webs, it is of prime importance to assess their levels in the Inuit in order to provide up-to-date information to the population and propose effective and viable public health advice respectful of their traditional lifestyle.

METHODOLOGICAL ASPECTS

Based on this perspective, one of the research themes in the Nunavik Inuit Health Survey 2004 addressed the following objectives: 1) to assess changes in contaminant exposure among the Inuit of Nunavik during the past decade by updating information available on contaminant levels measured in adult blood during the 2004 Nunavik Inuit Health Survey, and comparing this information with data collected in 1992 during the Santé Québec survey; 2) to begin monitoring the emerging contaminants of concern in northern regions. This theme paper only presents the results from POPs exposure assessment since another paper published in the context of this survey focuses on metals.

Laboratory methodology for organic compounds

Laboratory analysis for organic contaminants was performed at the human toxicology laboratory of the Institut national de santé publique du Québec (INSPQ) using the ASPE (Automated Solid Phase Extraction) method. This method is based on the fractionation of the plasma extract leading to three fractions (F1, F2, F3), followed by different purification and derivatization methods on F1, F2 and F3, that contain non-polar, non-planar compounds (F1), semi-polar, planar compounds (F2) and polar compounds (F3), respectively. Using this sample preparation, PCBs, OC pesticides, and brominated compounds such as BDEs, as well as other compounds can then be measured by mass spectrometry. The final list of compounds measured in the framework of the 2004

Nunavik Inuit Health Survey includes 86 analytes: 30 PCB congeners, 16 other OCs (pesticides, phenolic compounds and industrial pollutants) and 26 of their metabolites (15 hydroxy metabolites and 11 methylsulfones), 2 toxaphene congeners (Parlar # 26 and 50) and 12 brominated compounds including PBB-153, TBBA, 4 BDE congeners (congeners # 47, 99, 100 and 153) and brominated metabolites. Limits of detection for the 86 analytes are presented in Table A6 (Appendix).

Analysis of PFOS was carried out according to a method recently developed by the INSPQ human toxicology laboratory. This method is based on alkaline extraction with methyl-tert butyl ether and tetrabutylammonium hydrogen-sulfate, followed by electrospray LC-MS-MS analysis. Quantification was carried-out using isotope-labelled internal standards. This analytic method has a detection limit of 0.1 µg/L.

Consumption of traditional food

Data on food and nutrient intakes was obtained using a food frequency questionnaire and a 24-hour dietary recall. The food frequency questionnaire was administered to women and men and measured their consumption of traditional food for all four seasons during the year before the survey. Traditional food refers to food items derived from fishing and hunting. For each food item figuring in the questionnaire, daily consumption frequency for each season (number of times per day) and usual serving size in grams were measured. The frequency for each season has been added for the calculation of the average consumption frequency per day on an annual basis and the average intake in grams was calculated by multiplying the consumption frequency of the food item and the corresponding serving size (frequency x serving size)[2].

Statistical analysis

The analyses reported here are descriptive and were performed in order to present plasma concentrations of organic pollutant concentrations in plasma. All data were weighted so that estimations generated from the survey data would be representative of the entire population under study and not just the sample itself. Moreover, all the analyses were adjusted for survey design since this survey used a complex method of sampling that requires special attention in the calculation of variance. POPs levels were log-transformed in order to approximate a Gaussian distribution and geometric means were used in statistical analyses. Individuals with results under the limits of detection of the analytical method were included in the analysis by assigning them half the value of the detection limit. Pearson correlations between Aroclor 1260 expressed as total PCBs and the other legacy POPs were determined in order to evaluate if total PCBs could be used as a surrogate for exposure to legacy POPs. Overall, POPs were highly correlated with total PCBs. Therefore, variations between 1992 and 2004 data stratified according to gender, age category and coastal regions[3] are presented only for total PCBs concentrations.

Percent detection, geometric means, 95% confidence intervals and range of values were calculated for plasma concentrations of 86 organic contaminants measured in plasma samples for the first time in Nunavik. Comparisons of geometric means for pentachlorophenol, PBDE-47, PBDE-153 and PFOS according to gender, age categories, consumer of marine mammal fat and quartile of consumption of country fish were carried out using the Wald chi-square statistic with Satterthwaite correction for degrees of freedom (Aguirre-Torres, 1994). Statistical analyses for comparison purposes have been conducted at a threshold of $\alpha = 0.05$.

Comparisons of the samples with the 1992 Santé Québec survey group were performed for plasmatic concentrations of 11 legacy POPs (total PCBs determined as Aroclor 1260, aldrin, β-HCH, *p,p'*-DDE, *p,p'*-DDT, mirex, hexachlorobenzene, α-chlordane, cis-nonachlor, oxychlordane, transnonachlor). Given the sampling procedures in the different surveys, these comparisons include an adjustment in proportions or rates to take into account the change in the population's age structure. Moreover, the comparisons with other surveys also included an adjustment for survey design (Aguirre-Torres, 1994).

[2] The total consumption of country fish is defined as consumption of the following food items: artic char, cod, whitefish, trout, salmon, other fish (pike, cisco, walleye), dried fish and mollusc. The total consumption of marine mammal fat is defined as the consumption of beluga and seal fat.

[3] It should be noted that the Nunavik territory has been divided into these two regions because place of residence could influence life habits. The Hudson coast includes the villages of Kuujjuarapik, Umiujaq, Inukjuak, Puvirnituq, Akulivik, Ivujivik and Salluit while the Ungava coast includes Kangiqsujuaq, Quaqtaq, Kangirsuk, Aupaluk, Tasiujaq, Kuujjuaq and Kangiqsualujjuaq.

Accuracy of estimates

The data used in this module come from a sample and is thus subject to a certain degree of error. The coefficient of variation (CV) has been used to quantify the accuracy of estimates and the Statistics Canada scale was used to qualify the accuracy of estimates. The presence of an "E" footnote next to an estimate indicates a marginal estimate (CV between 16.6% and 33.3%). Estimates with unreliable levels of accuracy (CV > 33.3%) are not presented and have been replaced by the letter "F".

RESULTS

Descriptive statistics for plasmatic concentrations of 11 legacy POPs detected among the Inuit adult population aged 18 to 74 during the 1992 Santé Québec health survey and the 2004 Nunavik Inuit Health Survey are presented in Table A1 (Appendix). Statistically significant declines ($p < 0.001$) in plasmatic levels for all organochlorines are observed between 1992 and 2004. Mean plasma concentration of *p,p'*-DDT shows the highest decline with 67% over the 12-year period, whereas trans-nonachlor levels have decreased by only 37%.

Table A2 (Appendix) presents the Pearson correlation coefficient between plasmatic concentrations of total PCBs expressed as Aroclor 1260 and concentrations of the other classical POPs. Total PCB plasma concentrations were highly correlated with all POPs with the exception of alpha-Chlordane that shows a weaker correlation ($r = 0.21$). Tables A3, A4 and A5 (Appendix) show the 1992 and 2004 mean blood concentrations of total PCBs, used as a surrogate for exposure to legacy POPs, stratified according to gender, age and region, respectively. Significant decreases in mean total PCB concentrations ($p < 0.001$) occurred in both genders between the two surveys, without a significant difference between men and women ($p = 0.82$) (Table A3, Appendix). Total PCB concentrations decreased for all age categories between 1992 and 2004 (Table A4, Appendix). Adults aged 25 to 44 have significantly higher levels ($p < 0.001$) of total PCBs than younger adults, whereas adults aged 45 to 74 have significantly higher levels than the other two age categories ($p < 0.001$). A significant decrease in total PCB levels occurred in communities along the Hudson coast and Ungava coast during the 12-year period ($p < 0.001$) (Table A5, Appendix). However, total PCB concentration is still significantly higher in participants from Hudson Bay communities ($p = 0.003$).

Table A6 (Appendix) shows descriptive statistics for concentrations of organic contaminants measured in the plasma of participants from the Nunavik Inuit Health Survey 2004. Percent detection, geometric means, 95% confidence intervals and range are shown. Means were computed only for analytes detected in more than 50% of samples. For other analytes, we only report percent detection and the range of detected values. For PCBs, a total of 29 congeners were measured, including the traditional suite of PCB congeners measured in earlier studies (IUPAC # 99, 101, 118, 128, 138, 153, 156, 170, 180, 183, 187). The most abundant congeners are IUPAC # 153, 180 and 138. Most congeners were detected in more than 50% of samples, except for IUPAC # 128, 189 and 209.

In the category of chlorinated pesticides and other industrial compounds, *p,p'*-DDE, trans-nonachlor, pentachlorophenol (PCP) and oxychlordane show the highest levels. Other compounds previously measured in the traditional suite of POPs were also measured (cis-nonachlor, HCB, mirex, *p,p'*-DDT, β-HCH).

Additional compounds were included in the suite of POPs measured. These included two congeners of toxaphene, brominated compounds, methylsulfone metabolites and hydroxylated metabolites. Toxaphenes were detected in more than 90% of samples, with levels ranging from 3 to 5913 ng/L. Among the brominated compounds, PBDEs # 47, 99, 100 and 153 were measured, but only # 47 and 153 were detected in more than 50% of samples, with levels ranging from 15 to 2400 ng/L and 5 to 620 ng/L, respectively. PBB # 153, pentabromophenol (PBP), tetrabromobisphenol A as well as two tetrabromophenols were measured, but only 2,3,4,6-TBP was detected in more than 50% of samples with levels ranging from 1 to 1395 ng/L. PFOS, a perfluorinated compound, was measured and detected in the blood of all participants. The mean concentration was 18 386 ng/L with levels ranging from 480 to 470 000 ng/L. Nine methylsulfone metabolites of PCBs and DDE were measured, and five of them were detected in more than 50% of samples. The most abundant ones were 3-Methylsulfonyl-PCB-49 and 3-Methylsulfonyl PCB-101. Seventeen hydroxylated metabolites of PCBs and PBDEs and heptachlorostyrene were measured and all of them except 2-OH-PBDE-68, 2-OH-PBDE-75 and 4-OH-PCB-193 were detected in more than 50% of samples. The most abundant metabolites are 4-OH-PCB-107, 4-OH-PCB-146 and 4-OH-PCB-187.

Concentrations of PCP, PBDE-47, PBDE-153 and PFOS stratified by gender and age groups are presented in Tables A7 and A8 (Appendix), respectively. Blood concentrations of PCP, PBDE-153 and PFOS were significantly higher in men than women ($p < 0.001$), whereas PBDE-47 levels were similar in both genders ($p = 0.284$) (Table A7, Appendix). There was a significant decrease in concentrations of PCP ($p = 0.004$) and a significant increase in PFOS ($p < 0.001$) in relation to age groups (Table A8, Appendix), with the highest mean concentration found in people aged 45 to 74. PBDE-47 did not show an age-related increase or decrease ($p = 0.081$), whereas PBDE-153 show a non-significant increase with age ($p = 0.061$). PBDE-47 showed slightly higher levels in women aged 18 to 39 years, but this difference with other age groups was not statistically significant.

Potential associations between PCP, PBDE-47, PBDE-153 and PFOS concentrations with traditional food consumption are presented in Tables A9 and A10 (Appendix). Consumers of marine mammal fat have significantly higher concentrations of PFOS ($p = 0.0001$) and slightly higher, but not statistically significant, PCP levels ($p = 0.054$) compared to non-consumers (Table A9, Appendix). However, consumption of marine mammal fat was not associated with plasma concentration of PBDE-153 and PBDE-47. In fact, consumers of marine mammal fat have significantly lower PBDE-47 blood levels ($p = 0.03$) compared to non-consumers. Blood concentrations of PCP, PBDE-47 and PBDE-153 were not associated with the consumption of fish expressed in quartiles (Table A10, Appendix), whereas PFOS levels increased with quartile of fish consumption ($p < 0.0001$). However, there is no difference in PFOS mean concentrations between the third and fourth quartile of fish consumption.

DISCUSSION

Legacy POPs

A decrease in exposure to legacy organochlorines is observed in adults aged 18 to 74 compared to 1992. The mean plasma concentration of Aroclor 1260 expressed as the total PCB concentrations in 2004 was 7720 ng/L, while the mean level reported in the Santé Québec study in 1992 was 16 118 ng/L, which represents a decline of more than 50% of the initial blood concentrations observed in 1992. Among participants in 2004, 11% had total PCB concentrations above the level of concern determined by Health Canada (20 µg/L whole blood). This proportion was 23% in 1992. Concerning women of childbearing age (18-39 years), in 2004, 14% had levels of total PCBs over the concern level for maternal blood (5 µg/L whole blood) set by Health Canada compared to 45% in 1992. The decline in mean blood concentration was 50% above the mean level reported in 1992 for all the other organochlorines, except for oxychlordane and trans-nonachlor for which the decreases were 49% and 37%, respectively.

These results are in agreement with previous observations reported in temporal trend studies addressing organochlorine exposure in the Nunavik population (Dallaire et al., 2003; Pereg et al., 2003) and in other populations (Dallaire et al., 2002; LaKind et al., 2001; Meironyte et al., 1999). It is likely that these decreases in plasma concentrations are attributable to the combination of changes in diet and a reduction of POPs released in the environment, since the use of these organic compounds is severely restricted in several parts of the world (Dallaire et al., 2003). Dallaire and colleagues (2003) have suggested that consumption of less contaminated traditional food, rather than a dietary shift, accounted in large part for the rapidly descending POP concentrations observed in the cord blood of Inuit neonates born in Nunavik between 1994 and 2001. In fact, reductions in the POP body burden have been reported in several species of Arctic wildlife in the last decade (Braune et al., 2005).

As expected, mean plasmatic concentrations of total PCBs were not different between men and women, but increased significantly with age. Also, participants from communities along the Hudson coast had significantly higher total PCB concentrations compared to participants from the Ungava coast. Similar results can be extrapolated to the other POPs since mean levels were all highly correlated with the mean total PCB concentrations. Augmentation with age is principally related to three factors. First, POPs are persistent compounds that tend to accumulate with age, since elimination is slower than exposure through traditional food consumption. Second, young adults consume less traditional Inuit food than the previous generations. Third, POP environmental levels are following a decreasing trend, so that younger birth cohorts were not as exposed as the older cohorts. The difference observed in total PCB levels between participants from the two regions is probably related to a more frequent consumption of fish and marine mammals

in individuals living along the Hudson coast as reported in the 1992 Santé Québec survey (Santé Québec, 1994).

Despite this decrease, levels of exposure to PCBs are still above the concern level for 14% of women of childbearing age. Chronic exposure to PCBs and other organochlorines has been related to adverse effects on the immune function in Nunavik children (Dallaire et al., 2004; Dallaire et al., 2006; Dewailly et al., 2000). Therefore, monitoring of these contaminants should continue and information on sources of exposure should be provided to the population in order to attempt to reduce exposure to background levels.

New contaminants of concern

In 2004, as many as 86 organic compounds were measured including legacy POPs and new compounds of concern in the Arctic. Hydroxylated and methylsulfonyl metabolites of PCBs as well as one methylsulfonyl of DDE and two hydroxylated metabolites of PBDEs were measured in Inuit adults in Nunavik for the first time. Also, concentrations of two toxaphene congeners were determined for the first time in this population.

Toxaphenes

Plasmatic levels of toxaphene parlar 26 in this study were lower than the concentrations reported among the general population of the Chukotka Peninsula in Russia (Sandanger et al., 2003), but higher than those reported in Inuit from the Baffin and Kivallik area in a study addressing maternal and umbilical cord blood in Arctic Canada (Butler Walker et al., 2003). Parlar 50 concentrations in Nunavik were similar to levels observed in the Chukotka Peninsula (Russia), but remain higher than in the Baffin and Kivallik regions. Toxaphene was shown to be a weak mutagen and possibly a tumour promoter (Schrader et al., 1998); it was also shown to induce immunologic effects and weight lost in newborns (Tryphonas et al., 2001). Dietary surveys from five Inuit regions excluding Nunavik have revealed that high consumers of traditional food had blood concentrations exceeding the provisional tolerable daily intake for toxaphene (0.2 µg/kg body weight/day) (Van Oostdam et al., 2005). Therefore, efforts will be made in other projects to document temporal changes in toxaphene exposure.

Pentachlorophenol (PCP)

Pentachlorophenol was detected in all samples, with an arithmetic mean plasmatic concentration of 1115.7 ng/L. Levels in Nunavik are three to fourfold lower than concentrations measured in 1992 in the context of the Santé Québec health survey (Sandau, 2000) and lower than a southern sample of post-menopausal women from the Quebec region (Sandanger, 2007, personal communication). Higher concentrations of PCP in men compared to women were found in 2004, as was observed in the same population in 1992. Sources of exposure to PCP seem to be different from those of classical POPs since PCP concentrations in our sample decreased with age and were not associated with marine mammal fat and fish consumption. Surprisingly, PCPs significantly increased with age in a sub-sample (n = 30) randomly selected from the 1992 Santé Québec health survey (Sandau, 2000). Small sample size in the first study may explain the discrepancy between results. Since mean PCP concentration is the third largest among measured organic compounds and prevalent phenolic compounds in the Nunavik population, an investigation of sources of exposure is particularly crucial, especially among young adults.

Polybrominated diphenyls ethers (PBDE)

All PBDE congeners, except PBDE-153, were detected in less than 70% of samples. However, the mean plasmatic concentration of PBDE-47 was higher than the mean PBDE-153 level. Prevalent concentrations of PBDE-47 are an indication of exposure to the commercial penta-PBDE formulation that was predominantly used in North America. Production of this commercial mixture in the USA ended in 2004 and has been phased-out in Canada since 2006. Arithmetic mean concentration of the congener PBDE-47 on a lipid basis was 10.91 ng/g lipids, which is below levels reported in most US populations (Schecter et al., 2005), but approximately twofold higher than concentrations in European (Thomas et al., 2006) and Asian populations (Harrad & Porter, 2006; Kim et al., 2005). Also, mean PBDE-47 concentrations in Inuit women were three times lower than levels measured in post-menopausal women from Quebec's southern population (Sandanger et al., 2007). Mean plasmatic concentrations of PBDE-47 were similar between men and women, whereas there was a significant difference between genders for congener PBDE-153. The difference observed could be attributable to a higher metabolizing capacity among women for this congener compared to men or to elimination related to breastfeeding. Neither

congener was associated with age, although there was a borderline increase for PBDE-153 that could be linked to its higher bioaccumulative property. Concentrations of these two PBDEs were not associated with fish and marine mammal fat consumption. In fact, significantly lower PBDE-47 concentrations were measured in non-consumers of marine mammal fat compared to consumers. Indoor dust has been reported to be a significant source of exposure to PBDEs (Jones-Otazo et al., 2005).

Therefore, our results suggest that non-consumers of traditional food have different lifestyle habits than consumers, leading to higher PBDE exposure. Some individuals in our sample had very high levels of PBDE-47 in the blood as observed in other populations. Since there are no close industrial sources in Nunavik, specific lifestyle habits could be the most probable explanation. Levels of PBDEs are still well below those of PCBs but increasing trends have been reported in Arctic populations and biota (Ikonomou et al., 2002; Pereg et al., 2003). Therefore, monitoring of these compounds should be maintained and efforts to identify sources of exposure in Inuit populations should be increased.

Perfluorooctanesulfonate (PFOS)

Surprisingly, Inuit participants had higher mean plasmatic PFOS concentrations than mean total PCB levels, followed by *p,p'*-DDE and pentachlorophenol. We could not compare the current levels of PFOS with those prevailing in 1992 because this compound was not measured previously. Plasmatic concentrations in Nunavik were generally higher than levels measured in Asia (Harada et al., 2007; Taniyasu et al., 2003; Yang et al., 2004), similar to European levels (Falandysz et al., 2006; Kärrman et al., 2004) but lower than concentrations reported in US populations (Kannan et al., 2004; Olsen et al., 2003).

Inuit men were significantly more exposed than women. Also, PFOS concentrations show an age-related increase that could be attributable to higher traditional food intake in people aged 45 to 74. Indeed, PFOS plasmatic levels were higher among consumers of marine mammal fat compared to non-consumers and with increasing quartile of fish consumption. Fish intake has been reported to be a source of exposure to PFOS and other fluorinated compounds in two fish-eating populations (Falandysz et al., 2006; Thomsen et al., 2006). Also, PFOS have been shown to biomagnify along Arctic food webs (Bossi et al., 2005; Haukas et al., 2007; Tomy et al., 2004). However, other sources of exposure such as the use of manufactured products containing PFOS are highly probable since non-consumers of fish have a geometric mean plasmatic concentration of 15 µg/L. The maximum concentration of 470 000 ng/L detected in two men aged between 40 and 50 was a thousand-fold higher than the lowest level measured. Reasons for such high exposure are not clear, but those two individuals were found to be in the third quartile of fish intake. Investigations of routes of exposure to PFOS and other perfluorinated compounds in humans, as well as thorough toxicological assessments, are being conducted by several research teams.

CONCLUSION

Several brominated POPs exert toxic effects that are similar to those exerted by PCBs, dioxin-like chemicals and organochlorine pesticides (Branchi et al., 2003; Darnerud et al., 2001; Zhou et al., 2002), whereas PFOS have different toxicological effects particularly on fatty acid transport and metabolism. Levels of PBDEs in the environment (Ikonomou et al., 2002) as well as in human tissues and fluids (Meironyte et al., 1999; Noren & Meironyte, 2000; Pereg et al., 2003) have been reported to rise dramatically in the Arctic over the past decade, whereas the temporal trend for PFOS is unclear. Therefore, monitoring exposure to these contaminants and investigating sources of exposure are of utmost importance to avoid undue exposures, despite the fact that guidelines regarding tolerable daily intake and levels of concern are still at the initial stage of formulation (Gill et al., 2004). Additionally, more experimental and epidemiological studies are needed to assess the toxic effects exerted by methylsulfone and hydroxylated metabolites of PCBs and to determine levels of concern for human plasma levels of metabolites. However, we should take into account that emerging POPs are not particularly high in the Arctic and most of them (except PFOS) are not associated with traditional food consumption. The levels reported here for these emerging compounds will serve as a baseline for further studies.

Further analyses will complete the exposure assessment component regarding POPs. Many questions and hypotheses are raised regarding the sources of exposure, the influence of diet and the possible toxic effects of emerging contaminants. Further analyses of the data collected during the Nunavik Inuit Health Survey 2004 will help provide answers to some of these questions.

KEY ISSUES

Trend since 1992

- Concentrations of all legacy POPs have declined since 1992. This decreasing trend is probably related to the reduction of POPs in the Arctic environment.
- However, 14% of women of childbearing age still have level above the concern level set by Health Canada.

PBDEs

- PBDE concentrations are below levels reported in other North American populations, but higher than European and Asian populations.
- Young adults aged 18-24 years are the most exposed group to PBDE-47, the prevalent congener.
- Consumption of traditional food is not the source of exposure in the Inuit population.

PFOS

- PFOS levels in Inuit of Nunavik are similar to concentrations measured in southern Canadian and European populations, higher than in Asia but lower than in USA.
- Consumption of marine mammals fat and fish is a source of exposure to PFOS in this population. Other sources of exposure, such as uses of manufactured products containing PFOS, probably account for the concentration measured in this population.

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APPENDIX

Table A1
Average blood concentrations (ng/L) for 11 legacy POPs, population aged 18 to 74 years, Nunavik, 1992 and 2004

Contaminant	1992					2004								
	n	% detected	Arithmetic mean	Geometric mean	95% confidence interval (lower-upper limit)	n	% detected	Detection limit (ng/L)	Arithmetic mean	Geometric mean	95% confidence interval (lower-upper limit)	Minimum	Maximum	% variation
Total PCBs	491	100.0	26978	16118	14736-17629	899	100		15818	7720*	7220-8255	360	310000	-52
Aldrin	492	2.2				169	0	10				5	5	
β-HCH	492	96.8	172	127	118-137	898	95.6	10	92	50*	47-53	5	1200	-61
p,p'-DDE	492	100.0	11229	6858	6308-7456	899	100	15	5154	2856*	2708-3011	76	54000	-58
p,p'-DDT	491	95.1	376	212	192-235	899	90.6	20	117	69*	65-73	10	1600	-67
Mirex	492	96.8	352	166	150-183	828	91.7	10	160	65*	60-70	5	3636	-61
Hexachlorobenzene	486	100.0	1416	937	866-1014	899	99.5	20	685	351*	332-372	10	8500	-63
Alpha-Chlordane	492	7.7				899	3.2[E]	5				3	22	
Cis-Nonachlor	492	98.8	393	231	210-254	899	98.1	5	236	105*	98-112	3	3500	-55
Trans-Nonachlor	492	100.0	2121	1142	1034-1261	899	100	1	1708	717*	670-768	8	23000	-37
Oxychlordane	492	99.6	1695	789	705-882	899	100	2	1027	405*	377-435	5	16000	-49

* Significant variation between health surveys, $p < 0.001$.

E Interpret with caution.

Sources: Nunavik Inuit Health Survey 2004 and Santé Québec survey 1992.

Table A2
Pearson's correlations between total PCBs and 10 legacy POPs, population aged between 18 to 74 years, Nunavik, 2004

Contaminant (log) (ng/L)	Total PCBs	
	Pearson's coefficient	P-value
Aldrin	-	-
β-HCH	0.83	< 0.0001
p,p'-DDE	0.91	< 0.0001
p,p'-DDT	0.77	< 0.0001
Mirex	0.91	< 0.0001
Hexachlorobenzene	0.85	< 0.0001
Alpha-Chlordane	0.21	< 0.0001
Cis-Nonachlor	0.89	< 0.0001
Trans-Nonachlor	0.91	< 0.0001
Oxychlordane	0.93	< 0.0001

Source: Nunavik Inuit Health Survey 2004.

Table A3
Average blood concentrations (ng/L) for total PCBs according to gender, population aged 18 to 74 years, Nunavik, 1992 and 2004

Contaminant (ng/L)	Gender	1992			2004					
		n	Geometric Mean	95% confidence interval (lower-upper limit)	n	Geometric Mean	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value
Total PCBs	Men	210	18115	15930-20601	404	8260*	7556-9030	400	93000	0.82
	Women	281	14228	12844-15760	495	7189*	6666-7754	360	310000	

* Significant variation between health surveys, $p < 0.001$.

Sources: Nunavik Inuit Health Survey 2004 and Santé Québec survey 1992.

Table A4

Average blood concentrations (ng/L) for total PCBs according to age group, population aged 18 to 74 years, Nunavik, 1992 and 2004

Contaminant (ng/L) / Age group	1992			2004					
	n	Geometric Mean	95% confidence interval (lower-upper limit)	n	Geometric Mean	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value[a]
Total PCBs									
18-24 years	107	7851	6897-8937	198	3386*	3033-3780	410	20000	< 0.001
25-44 years	232	13774	12360-15439	463	5866*	5320-6467	360	78000	
45-74 years	152	49834	44500-55807	238	27683*	24475-31310	520	310000	
Women of childbearing age (18 to 39 years)	173	9120	8182-10166	302	3741*	3401-4114	360	29000	

[a] Variation with age in 2004; based on the Satterthwaite adjusted χ^2 test.

* Significant variation between health surveys, $p < 0.001$.

Sources: Nunavik Inuit Health Survey 2004 and Santé Québec survey 1992.

Table A5

Average blood concentrations (ng/L) for total PCBs according to coastal region, population aged 18 to 74 years, Nunavik, 1992 and 2004

Contaminant (ng/L) / Coastal region	1992			2004					
	n	Geometric Mean	95% confidence interval (lower-upper limit)	n	Geometric Mean	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value[a]
Total PCBs									
Hudson	273	19032	16875-20601	482	8614*	7726-9603	410	310000	0.003
Ungava	218	12675	11114-14455	417	6709*	6068-7419	360	86000	

[a] Variation between coastal regions in 2004; based on the Satterthwaite adjusted χ^2 test.

* Significant variation between health surveys, $p < 0.001$.

Sources: Nunavik Inuit Health Survey and Santé Québec survey 1992.

Table A6
Plasma concentrations of 86 organic contaminants (ng/L), population aged 18 to 74 years, Nunavik, 2004

	n	Detection limit	% Detected	Geometric Mean	95% confidence interval	Minimum	Maximum
PCB (IUPAC #)							
Aroclor 1260	899		100.0	7720	7220-8255	360	310000
74	898	10	93.5	52	49-55	5	2600
99	899	10	98.6	148	138-158	5	6000
101	899	10	66.9	17	16-18	5	540
105	899	10	75.8	25	23-26	5	1200
118	899	10	99.4	128	120-136	5	5300
128	899	10	34.3			5	200
138	899	10	100.0	463	434-494	25	19000
146	899	10	97.3	130	121-139	5	6100
153	899	10	100.0	1062	992-1138	42	41000
156	899	10	87.9	45	42-48	5	2400
157	899	10	60.6	17	16-18	5	610
163	899	10	98.4	158	148-169	5	6200
167	899	10	57.7	14	14-15	5	880
170	899	10	99.2	156	146-166	5	6182
172	899	10	76.9	29	27-31	5	1400
177	899	10	80.8	27	26-29	5	1700
178	899	10	88.3	51	47-54	5	1900
180	899	10	100.0	562	525-602	21	22727
183	899	10	92.9	56	52-60	5	2800
187	899	10	99.8	225	211-240	5	9100
189	899	10	39.1			5	270
194	899	10	94.5	98	91-105	5	5700
195	899	10	54.5	14	13-15	5	760
196	899	10	68.4	21	20-22	5	1500
201	899	10	95.5	97	91-104	5	4700
203	899	10	87.6	51	48-55	5	2500
206	899	10	76.4	30	28-32	5	1700
208	899	10	52.8	15	14-16	5	880
209	899	10	48.5			5	710

	n	Detection limit	% Detected	Geometric Mean	95% confidence interval	Minimum	Maximum
Chlorinated pesticides and other industrial compounds							
α-HCH	899	10	7.6			5	82
cis-Nonachlor	899	5	98.1	105	98-112	2.5	3500
Hexachlorobenzene	899	20	99.5	351	332-372	10	8500
Chlordecone	291	50	49.2	81	71-93	25	4400
Mirex	899	10	91.7	65	60-70	5	3636
Oxychlordane	899	2	100.0	405	377-435	5	16000
Octachlorostyrene	899	5	26.3			2.5	49
p.p'-DDD	292	50	5.0			25	180
p.p'-DDE	899	15	100.0	2856	2708-3011	76	54000
p.p'-DDT	899	20	90.6	69	65-73	10	1600
ß-HCH	898	10	95.6	50	47-53	5	1200
Trans-Nonachlor	899	1	100.0	717	670-768	8	23000
2,3,4,6-Tétrachlorophenol	817	10	80.2	21	20-23	5	790
Pentachloroanisole	899	10	1.9[E]			5	14
Pentachlorobenzene	899	10	16.9			5	83
Pentachloronitrobenzene	899	10	F			F	F
Pentachlorophenol	821	10	100.0	822	779-868	140	18000
Toxaphene							
Parlar # 26	899	5	95.8	76	71-82	2.5	3391
Parlar # 50	899	5	98.1	138	129-148	2.5	5913
Brominated flame retardants and other compounds							
PBB IUPAC # 153	896	10	41.3			5	380
PBDE IUPAC # 47	896	30	57.0	36	34-39	15	2400
PBDE IUPAC # 99	890	20	19.8			10	575
PBDE IUPAC # 100	896	20	18.3			10	580
PBDE IUPAC # 153	896	10	77.1	18	17-19	5	620
PBP	770	2	8.5			1	250
Tetrabromobisphenol-A	821	20	5.4			10	480
2.3.4.5-Tetrabromophenol	770	2	1.6[E]			1	41
2.3.4.6-Tetrabromophenol	770	2	58.3	7	6-8	1	1395
Perfluorinated compounds							
PFOS	913	100	100.0	18386	17675-19127	480	470000

	n	Detection limit	% Detected	Geometric Mean	95% confidence interval	Minimum	Maximum
Methylsulfone metabolites							
3-Methylsulfonyl-PCB 49	834	2	90.6	21	20-23	1	910
3-Methylsulfonyl-PCB 87	832	2	72.2	8	7-9	1	610
3-Methylsulfonyl-PCB 101	834	2	83.1	14	13-15	1	920
3-Methylsulfonyl-PCB 141	831	5	31.8			2.5	130
3-Methylsulfonyl-PCB 149	831	5	18.8			2.5	64
3-Methylsulfonyl-DDE	832	5	36.6			2.5	950
4-Methylsulfonyl-PCB 49	834	2	62.6	4	4-5	1	220
4-Methylsulfonyl-PCB 91	831	2	16.5			1	23
4-Methylsulfonyl-PCB 101	832	2	67.9	6	6-7	1	260
4-Methylsulfonyl-PCB 110	164	5	49.2			2.5	310
4-Methylsulfonyl-PCB 149	832	5	41.8			2.5	180
Hydroxylated metabolites							
2-Hydroxy-PBDE 68	821	10	6.1			5	120
2-Hydroxy-PBDE 75	821	5	49.9			2.5	150
3-Hydroxy-PCB 138	815	2	85.1	23	21-25	1	980
3-Hydroxy-PCB 153	776	2	96.0	33	30-36	1	1500
3-Hydroxy-PCB 180	798	2	68.6	5	4-5	1	550
4-Hydroxy-PCB 107	820	10	99.4	159	149-169	5	3200
4-Hydroxy-PCB 146	776	2	99.3	126	117-137	1	4900
4-Hydroxy-PCB 163	821	2	91.6	10	9-11	1	590
4-Hydroxy-PCB 172	796	2	95.0	17	16-19	1	790
4-Hydroxy-PCB 187	821	2	99.9	149	139-159	1	4300
4-Hydroxy-PCB 193	819	2	43.9			1	210
4-Hydroxy-PCB 199	822	2	93.6	17	16-18	1	1200
4-Hydroxy-PCB 200+198	821	2	67.9	5	4-5	1	560
4-Hydroxy-PCB 201	821	2	55.7	3	2-3	1	98
4-Hydroxy-PCB 202	820	2	84.7	7	7-8	1	340
4-Hydroxy-PCB 208	822	2	72.8	5	4-5	1	340
4-Hydroxy-Heptachlorostyrene	774	1	100.0	78	72-84	1	960

E Interpret with caution.

F Unreliable estimate.

Source: Nunavik Inuit Health Survey 2004.

Table A7
Average blood concentrations (ng/L) for pentachlorophenol, PBDE-47, PBDE-153 and perfluorooctanesulfonate (PFOS) according to gender, population aged 18 to 74 years, Nunavik, 2004

	n	% Detected	Geometric Mean (ng/L)	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value
Pentachlorophenol							
Men	367	100	953	886-1024	170	10000	< 0.001*
Women	454	100	706	664-750	140	18000	
PBDE IUPAC # 47							
Men	402	55.9	36	33-40	15	610	0.284
Women	494	58.1	37	34-40	15	2400	
PBDE IUPAC # 153							
Men	402	88.0	24	22-27	5	620	< 0.001*
Women	494	65.6	13	12-14	5	406	
PFOS							
Men	412	100	20620	19504-21797	2800	470000	< 0.001*
Women	501	100	16280	15462-17142	480	200000	

* Significant difference between gender for 2004; based on the Satterthwaite adjusted χ^2 test.
Source: Nunavik Inuit Health Survey 2004.

Table A8
Average blood concentrations (ng/L) for pentachlorophenol, PBDE-47, PBDE-153 and perfluorooctanesulfonate (PFOS) according to age group, population aged 18 to 74 years, Nunavik, 2004

	n	% Detected	Geometric Mean (ng/L)	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value
Pentachlorophenol							
18 to 24 years	180	100.0	1017	921-1123	190	9300	0.004*
25 to 44 years	421	100.0	761	713-811	150	9900	
45 to 74 years	220	100.0	791	710-882	140	18000	
Women of childbearing age (18 to 39 years)	277	100.0	719	670-772	150	9300	
PBDE IUPAC # 47							
18 to 24 years	197	61.1	41	35-47	15	920	0.082
25 to 44 years	461	57.0	37	34-41	15	2400	
45 to 74 years	238	53.2	32	29-35	15	1300	
Women of childbearing age (18 to 39 years)	301	61.9	40	36-45	15	2400	
PBDE IUPAC # 153							
18 to 24 years	197	72.2	15	13-17	5	620	0.061
25 to 44 years	461	74.9	17	16-19	5	406	
45 to 74 years	238	85.6	22	20-25	5	620	
Women of childbearing age (18 to 39 years)	301	58.1	11	10-12	5	406	
PFOS							
18 to 24 years	205	100.0	14310	13406-15282	3000	97000	< 0.001*
25 to 44 years	471	100.0	16320	15314-17388	2300	470000	
45 to 74 years	237	100.0	29470	26743-32476	480	470000	
Women of childbearing age (18 to 39 years)	308	100.0	12900	12093-13760	2300	97000	

* Significant difference between age group for 2004; based on the Satterthwaite adjusted χ^2 test.
Source: Nunavik Inuit Health Survey 2004.

Table A9

Average blood concentrations (ng/L) for pentachlorophenol, PBDE-47, PBDE-153 and perfluorooctanesulfonate (PFOS) according to marine mammals fat consumption, population aged 18 to 74 years, Nunavik, 2004

	n	% Detected	Geometric Mean (ng/L)	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value
Pentachlorophenol							
Consumer	485	100.0	807	754-863	140	10000	0.055
Non-consumer	198	100.0	913	819-1019	170	18000	
PBDE IUPAC # 47							
Consumer	528	54.0	35	32-38	15	1300	0.03*
Non-consumer	222	61.8	41	35-46	15	850	
PBDE IUPAC # 153							
Consumer	528	76.3	18	16-20	5	620	0.90
Non-consumer	222	78.2	18	16-20	5	620	
PFOS							
Consumer	536	100.0	20188	19129-21306	480	47000	0.0001*
Non-consumer	227	100.0	15691	14229-17303	2900	47000	

* Significant difference between marine mammals fat consumption categories for 2004; based on the Satterthwaite adjusted χ^2 test.

Source: Nunavik Inuit Health Survey 2004.

Table A10

Average blood concentrations (ng/L) for pentachlorophenol, PBDE-47, PBDE-153 and perfluorooctanesulfonate (PFOS) according to quartile of consumption of fish (gr/day, annual), population aged 18 to 74 years, Nunavik, 2004

	n	% Detected	Geometric Mean (ng/L)	95% confidence interval (lower-upper limit)	Minimum	Maximum	P-value
Pentachlorophenol							
Low (0-25%)	175	100.0	947	844-1063	150	8000	0.09
Low-Moderate (25-50%)	174	100.0	794	715-881	170	9300	
High-Moderate (50-75%)	167	100.0	813	729-907	170	18000	
High (75-100%)	171	100.0	806	718-905	140	8300	
PBDE IUPAC # 47							
Low (0-25%)	189	54.1	36	32-42	15	850	0.40
Low-Moderate (25-50%)	188	59.8	40	34-47	15	920	
High-Moderate (50-75%)	188	56.1	35	30-41	15	900	
High (75-100%)	189	56.3	34	30-39	15	1300	
PBDE IUPAC # 153							
Low (0-25%)	189	73.6	17	15-19	5	620	0.27
Low-Moderate (25-50%)	188	74.4	17	15-19	5	168	
High-Moderate (50-75%)	188	82.3	20	17-22	5	620	
High (75-100%)	189	76.7	19	16-22	5	330	
PFOS							
Low (0-25%)	191	100.0	15195	13949-16551	3100	100000	< 0.0001*
Low-Moderate (25-50%)	192	100.0	16547	14805-18493	480	270000	
High-Moderate (50-75%)	192	100.0	22183	19963-24650	2900	470000	
High (75-100%)	192	100.0	21342	19190-23735	2300	200000	

* Significant difference between country fish consumption categories for 2004; based on the Satterthwaite adjusted χ^2 test.

Source: Nunavik Inuit Health Survey 2004.

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Qanuippitaa?

HOW ARE WE?