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EXPOSURE AND PRELIMINARY HEALTH ASSESSMENTS OF THE OUJÉ-BOUGOUMOU CREE POPULATION TO MINE TAILINGS RESIDUES

REPORT OF THE SURVEY

INSTITUT NATIONAL DE SANTÉ PUBLIQUE DU QUÉBEC

Québec

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JANUARY 2005

McMaster University

CHUQ
CENTRE HOSPITALIER UNIVERSITAIRE DE QUÉBEC

Conseil Cri de la santé et des services sociaux de la Baie James
Cree Board of Health and Social Services of James Bay

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EXECUTIVE SUMMARY

BACKGROUND

The Cree community of Oujé-Bougoumou is located approximately 60 kilometres west of Chibougamau and comprises 622 residents. There was, for this community, a potential exposure to toxic substances derived from tailings residues left behind from mining operations in the mid 1950s.

Confronted with this potential exposure, the Grand Council of the Crees commissioned an environmental contamination study, which was conducted by Christopher L. Covel from CL COVEL PG LLC and Roger D. Masters from Dartmouth College, Hanover, New Hampshire. This study indicated mobilization of toxic elements from mine tailings residues and suggested possible impacts on human health.

The report produced by this study was subsequently critically reviewed by Evert Nieboer of McMaster University who endorsed the environmental conclusions, but not the interpretation of the data on contaminants in hair on which the human health risk judgement had been based. A 2001 survey by the Quebec Ministry of the Environment confirmed the presence of toxic elements in sediments near mine tailings sites. Subsequently, the Oujé-Bougoumou Council accepted E. Nieboer's recommendations that an environmental risk assessment and a human health study be initiated.

To address the Oujé-Bougoumou community's needs, the ministry of Health and Social Services mandated the Quebec National Institute of Public Health to conduct a study in order to characterize the possible exposure of the Cree community of Oujé-Bougoumou to environmental toxic elements. This report presents the data of the project conducted during the autumn of 2002 in the Cree communities of Oujé-Bougoumou and Nemaska.

OBJECTIVES

The main objective of this study was to assess the exposure of the Cree community of Oujé-Bougoumou to various toxic elements associated with mine tailings residues. At the same time, the study goal was to measure a battery of clinical chemistry parameters that can be used in the assessment of the general health of the population.

Three specific objectives were defined in the scope of this study. The first one consisted of assessing human exposure to inorganic elements associated with the mine tailings (arsenic, copper, selenium and zinc), life-style issues (lead and cadmium) or persistent pollutants often associated with fish consumption (e.g., mercury and PCBs). The second specific objective aimed to compare the biological contaminant exposure results with reference data obtained concurrently in a control Cree community (Nemaska), as well as with the concentrations observed in a Southern Quebec population previously studied and those recommended (published) as reference ranges. Finally a third specific objective was to measure and interpret an array of biochemistry parameters of clinical interest that relate to individual and/or general health.

POPULATIONS UNDER STUDY

A total of 225 participants in Oujé-Bougoumou (the study community) and 100 in Nemaska (the control community) participated in the study. Age and gender subgroups were considered in the recruitment and the different groups were respectively composed of children aged between 0 and 14 years old, women 15 to 39 years, men 15 to 39 years, and men and women over 40. The number of study subjects in each group but the second was proportional to the age distribution in each community.

STUDY METHODS

Three questionnaires were designed, tested and adapted for the specific purpose of this study. In the exposure/life-style questionnaire, questions were posed about the following issues: general socio-demographics and residency, specific information about the household property, occupational details, outdoor activities and hobbies and questions concerning life-style issues, stress and psychological well-being. The food frequency questionnaire addressed the consumption of traditional subsistence and the health questionnaire self-reported health outcomes.

Blood, urine and hair samples were collected and levels of contaminants and clinical biochemistry parameters were determined employing proven laboratory measurements.

FINDINGS

Cardiovascular disease (CVD) risks factors such as obesity, cigarette smoking, and diabetes were found to be frequent in both communities. Only diabetes was more frequent in Oujé-Bougoumou. Fish consumption was associated with a more favorable omega-3 fatty acid status and appeared to improve the CVD risk factors of low level of plasma HDL cholesterol, high level of LDL cholesterol and high level of total cholesterol.

Folate, vitamin B12, thyroid stimulating hormone (TSH) and free thyroxin (T4) levels were essentially in their normal ranges and not different between the two communities.

Total fish consumption was comparable in both communities, but predatory (piscivorous) fish were favoured in Oujé-Bougoumou, while insectivorous fish were more often consumed in Nemaska. Game offal was eaten more frequently Oujé-Bougoumou.

Inorganic and total arsenic concentrations in urine were generally higher in Nemaska than Oujé-Bougoumou. Exceedances of the normal reference range of copper in plasma occurred, as expected, among pregnant women and those taking oral contraceptives. Copper in urine and hair were significantly higher in Oujé-Bougoumou females of reproductive age. Selenium in plasma was higher in Nemaska than Oujé-Bougoumou, and this likely reflects differences in dietary habits. Zinc in plasma was higher in Oujé-Bougoumou and might also reflect differences in dietary habits.

Cadmium level in whole blood of all participants was 2.5 to 3.0-fold higher compared to the Southern Quebec comparison group. Smoking was the main factor, but exposure through consumption of game liver and kidney remains plausible. Lead whole-blood concentrations were lower in both Cree

communities compared to other native communities, but exposure was still related to hunting and consumption of wildfowl and game.

Mercury exposure in the two Cree communities may be termed mild to moderate and was comparable to levels found in Canadian Inuit populations and was related to fish, bird and game consumption; the observed hair-to-blood mercury ratio was 242:1. PCBs exposure might be designated as moderate to high and was related to fish and game consumption. Liver enzymes and levels of TSH were normal among the most exposed individuals (>100 µg/L of total PCBs). Exposure to DDT and its metabolite DDE was high in the over 40 age group, especially in Ouje-Bougoumou, and was dependent on the consumption of fish and game. The DDE/DDT ratio was 57±33, suggesting little current use or sources of DDT. Concentrations of persistent contaminants were generally higher in the over 40 age group and reflect the role of diet as a major source of exposure.

CONCLUSIONS

Based on the observed concentrations of the signature elements arsenic, copper, selenium and zinc in body fluids, we conclude that the residents of Oujé-Bougoumou are not at risk of internal (systemic) exposure. Hair levels of the mine tailings signature elements were moderately elevated in Oujé-Bougoumou relative to Nemaska and a possible indirect source through chronic low-level contact might be explored in the case of arsenic and copper.

Cigarette smoking is the major source of exposure to cadmium in both Oujé-Bougoumou and Nemaska. Lead exposure is related to hunting activities and consumption of wildfowl and game in both Cree communities.

Exposure to mercury and PCBs was higher in Oujé-Bougoumou than in Nemaska; higher consumption of piscivorous (predator) fish such as walleye and trout might be the explanation. The observed concentrations of the p,p'-DDE (a metabolite of the insecticide DDT) are found to be relatively high in the over 40 age group, especially in Oujé-Bougoumou.

In both communities, the status of the essential elements copper, selenium and zinc are judged to be normal and adequate to sustain proper health. The iron status also appears adequate.

No new information about health status was identified in the two study communities.

RECOMMENDATIONS

The impact on the general environment of the elements related to mine tailing residues should be assessed as part of the ongoing environmental risk assessment, even though there is no evidence of unusual intake by humans.

The source of the organochlorines PCBs and DDT/DDE should be investigated as part of the ongoing environmental risk assessment.

Replacement of leaded ammunition should continue to be encouraged.

It is recommended that consumption guidelines for subsistence foods be reviewed, updated and their use by the Cree communities should continue to be promoted. The following factors should be incorporated into a more formal consumption guideline program. Routine monitoring of local fish tissues, as well as kidney, liver and fatty tissues of game and of piscivorous fowl, should be initiated. Consumption guidelines should be based on the biomonitoring results obtained for fish caught in local lakes and rivers and for wildfowl and game bagged in the communities' hunting grounds. The importance of consuming traditional foods to maintain health should be considered in the risk management.

The regular consumption of game liver and kidney should be avoided since a concern arises because persistent contaminants accumulate in kidney (especially cadmium) and in liver (e.g., cadmium, mercury and PCBs).

Anti-smoking interventions are likely to yield significant health benefits.

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1 INTRODUCTION

This report summarizes and interprets the data for the project conducted during the autumn of 2002 in the Cree community of Oujé-Bougoumou located in the James Bay Cree Nation Territory of Quebec. This community of 622 residents is located approximately 60 km Northwest of Chibougamau. The primary focus of the study was the potential exposure to toxic substances derived from tailings left behind from mining operations in the mid 1950s. The community of Nemaska was selected as the control community. It is an inland community with approximately the same age distribution and number of inhabitants as Oujé-Bougoumou and is located 323 km north of Oujé-Bougoumou. It has no history of mining nor any related activities. By comparing the Oujé-Bougoumou results with those from Nemaska, a determination can be made as to whether the exposures to arsenic, cadmium, copper, selenium, zinc and polychlorinated biphenyls (PCBs) experienced by the Oujé-Bougoumou population differ significantly from those of the control population.

The project was a follow-up to two recent reports about environmental contaminants and health implications for the Cree population. The first study was conducted in March 2001 by CL COVEL in Oujé-Bougoumou in Eeyou Istchee (Covel and Masters, 2001) and was supported by the Grand Council of the Cree. The primary objective was to determine if the tailings deposits and surface water runoff from 3 mines operating within the traditional Oujé-Bougoumou territory, namely Joe mann Mine (JMM), Campbell Point Mine (CPM) and Copper Rand Mine (CRM), may have contaminated or are contaminating the sediment, surface water and fish population of the area. A second objective was to determine if such occurrences have the potential to influence the health of the Oujé-Bougoumou Cree. A review and interpretation of historical aerial photographs from 1953 through 1998 clearly revealed that since 1953 some portions of both Doré and Chibougamau Lakes have been filled with mine-tailing waste from CPM and CRM operations. Mine tailings residues, sediment, surface water, fish samples and hair samples from 23 Oujé-Bougoumou Cree participants were collected for analysis and showed elevated levels of some toxic metals. The data were compared to toxicity guideline levels promulgated by the Canadian Council of Ministers of the Environment (CCME) documents *Canadian Environmental Quality Guidelines* (CEQG) and *Interim Sediment Quality Guidelines* (ISQG). The second report was a critical review of the Covel Report prepared by Dr. E. Nieboer dated April 9, 2002 and revised June 15, 2002 (Nieboer, 2002). Dr. Nieboer identified some of the strengths and weaknesses of the study, especially the misinterpretation of hair data. Moreover, the ecological observation of the mobilization of signature elements from the tailings pointed out by Covel and endorsed by Nieboer appeared to have been confirmed by a more recent survey conducted by the Quebec Ministry of the Environment (MEF) in the Oujé-Bougoumou Cree Nation Territory during 2001 (Laliberté and Tremblay, 2002). Subsequently, the recommendations made by Nieboer that an environmental risk assessment be initiated for the Oujé-Bougoumou territory and a health study be conducted were adopted by the community. Biological monitoring of the contaminants in body fluids and hair was to constitute the major component of the suggested human health survey, but an assessment of the self-reported health status supplemented by routine clinical chemistry/biochemistry measurements was to be included.

The health study was conducted under the co-direction of the Cree Board of Health and Social Services of James Bay (CBHSSJB) and the Quebec National Public Health Institute (INSPQ-CHUQ) through a mandate received from the Ministry of Health and Social Services of Quebec. The actual work was jointly supervised by Dr. Eric Dewailly of the INSPQ-CHUQ and Dr. Evert Nieboer of McMaster University who represented both the CBHSSJB and the Oujé-Bougoumou community, respectively. Suzanne Coté of the URSP-CHUL-CHUQ supervised the field work and coordinated the preparation of this report as Project Leader.

2 BACKGROUND, OBJECTIVES AND SCOPE

2.1 THE COVEL REPORT

Sixteen sediment and 13 surface water samples were collected and analyzed from the Doré, Obatogamau and Chibougamau Lakes and the Nemenjiche River. Among these, 12 sediment samples and 11 surface water samples contained metal concentrations exceeding the Canadian Council of Ministers of the Environment (CCME) / Interim Sediment Quality Guidelines (ISQGs) and the Aquatic Life Water Quality Guidelines (ALWQGs) of the Canadian Environmental Quality Guidelines (CEQGs). Furthermore, the 10 fish samples collected and analyzed from the same river and lakes contained measurable concentrations of some inorganic elements. However, no evidence is reported to suggest that the signature elements arsenic (As), copper (Cu), and zinc (Zn) had bioaccumulated in the fish samples analyzed. Mercury levels begin to approach the Health Canada and Ontario Ministry of Environment consumption guideline concentrations of 0.5 mg/kg wet weight in a number of cases. Iron and zinc concentrations were uniformly high in all samples and likely reflect their biological essentiality. Moreover, for the 23 Oujé-Bougoumou Cree participants, it was suggested that 6 toxic and 3 essential elements were high in human hair. Ratios were taken between the average concentrations observed in the 23 Oujé-Bougoumou Cree and those reported for 10,000 US individuals. No information is given about the demographic distribution of this reference group: age, race, gender, place of residence (urban, rural), etc. For the individual hair results reported, the observed concentrations are compared to the upper limit of the American range (corresponding to the 95^{th} percentile value). When this is used in the group comparison, all but the averages for cadmium (1.08 µg/g compared to 0.5 µg/g or 0.7 µg/g for females and males, respectively) and mercury (6.4 µg/g compared to 2 µg/g or 3 µg/g for females and males, respectively) exceeded the expected range. Clearly, mercury levels are substantially elevated relative to the upper limit of the American reference values. The highest concentrations reported were 27 µg/g and 29 µg/g. Only for lead is the dependence on age examined and, in this case, there was no observed trend. Plotting of the mercury hair data compiled in Appendix D of the COVEL Report is suggestive of some age dependence.

2.2 COVEL RECOMMENDATIONS

(i) "A more comprehensive investigation of the soils, sediment, surface and groundwater, fish and other biota in Oujé-Bougoumou Cree Nation Territory needs to be performed. This investigation should include evaluation of heavy metal contamination of all current and former mine locations in the area;

(ii) Consumption of fish from suspect areas should be carefully monitored and the Oujé-Bougoumou Cree should conduct environmental education programs in the Village of Oujé-Bougoumou to educate the community and members in the bush on the risks associated with long term health effects of fish consumption from these areas;

(iii) Undertake an independent human health risk assessment of all suspected areas as soon as possible;

(iv) Implementation of a program for identification and detoxification of heavy metals in the Oujé-Bougoumou Cree as soon as possible;

(v) Explore the option of remediation regarding the contamination in order to cap, control and monitor existing mine tailings waste to prevent further contamination from entering the ecosystem for all current and former mine sites within the Cree Nation Territory;

(vi) Retain external legal council with an expertise in environmental law to assist in the contamination remediation issue;

(vii) Conduct an assessment of the regulatory regime, that permitted the environmental damages documented in this report to occur."

2.3 CONCLUSIONS AND RECOMMENDATIONS FOR A CRITICAL ASSESSMENT OF THE COVEL REPORT

Following the COVEL study, Dr. Evert Nieboer conducted a critical assessment of the COVEL Report (Nieboer, 2002), and his conclusions/recommendations are reproduced below.

A. Critical Assessment of the Sediment, Surface Water and Fish Quality Investigation: Conclusions and Recommendations.

1. Some deficiencies were pointed out in the description of the sampling sites and the analytical details provided (e.g., in-laboratory sampling, sample pretreatment and quality assurance).
2. Based on their prevalences and concentrations in sediments, arsenic, copper and perhaps zinc, may be designated signature elements.
3. Based on the Canadian Council of Ministers of the Environment (CCME) probable effect levels (PELs) for sediments, it was concluded that arsenic and copper aquatic toxicity seems probable.
4. For each of arsenic, copper (and perhaps selenium) the Canadian Drinking Water Quality Guidelines were exceeded for three of 12 surface water samples collected on/nearby tailings; for other sites, water samples exceeded the CEQG for aquatic toxicity most consistently for Cu and Zn.
5. Since the COVEL report provides evidence of the presence and/or mobilization of toxic tailings constituents that may result in aquatic toxicity, it seems prudent to compile all available environmental data (concentrations in soil, sediments, surface water, vegetation, fish and other biota) for the entire Oujé-Bougoumou Cree Nation Territory. Potential sources of these data likely include the Quebec Ministry of the Environment and the owners (or previous owners) of the 20 or so copper and gold mines. Such an activity could constitute the first step in an environmental site assessment (ESA).

B. Critical Assessment of the Human Hair Study: Overview and Recommendations

The following recommendations were made after a detailed review of structural features of hair, trace metal deposition pathways and patterns, and factors and sources that influence levels of trace elements in hair.

1. Deficiencies are pointed out in terms of study design and the analytical details provided (hair sample collection, cleaning procedure, in-laboratory sampling, chemical treatment and quality assurance).

2. The evidence for hair concentrations as an index to exposure and body burden is critically examined for the elements of concern identified in the COVEL Report, as well as for the signature elements arsenic and copper. For the elements other than mercury, the scientific basis is not in place for hair concentrations alone to trigger health-related interventions among environmentally exposed individuals.
3. Based on Items 1 and 2 above, the significance of the human hair findings are assessed and it is concluded that of the elements measured in the COVEL study, mercury and lead are of some concern and a follow-up seems warranted. The level of concern is moderate, as is the case in other Native communities in Canada and elsewhere. Establishing the source of cadmium also seems prudent, since smokers constitute a susceptible population when environmental or occupational cadmium exposure occurs.
4. The region-wide environmental site assessment/classification process recommended might conceivably end-up defining the need for a more detailed site-specific risk assessment, which could include a human health component. Such a process would take some time and thus it is recommended that a preliminary health study be implemented concurrently with the environmental assessment. Such an initiative would supplement the latter, but may also help to reduce the level of anxiety in the Oujé-Bougoumou communities. Biological monitoring initiatives would constitute the major component of the suggested survey, and the questionnaire required for this could begin to address the environment and health issues.

The Oujé-Bougoumou Council accepted the Nieboer recommendations and solicited a health study proposal from Drs. Dewailly and Nieboer, that was adopted and received financial support as explained in Section 1.

2.4 HEALTH SURVEY OBJECTIVES AND SCOPE

The main objective of this study was to assess the exposure of the Cree community of Oujé-Bougoumou to various toxic elements associated with mine tailings residues and to measure a battery of clinical chemistry parameters that can be used in an assessment of general health. In addition, the report by Laliberté and Tremblay (2002) indicated that PCBs and mercury concentrations in fish caught in Lakes Chibougamau and Doré were relatively elevated. Consequently, the three specific objectives were: (i) to assess human exposure to inorganic elements associated with the mine tailings (arsenic, copper, selenium and zinc), life-style issues (lead and cadmium) or persistent pollutants often associated with fish consumption (mercury and PCBs); (ii) to compare the biological contaminant exposure results with reference data obtained concurrently in a control Cree community (Nemaska), the levels observed in a Southern Quebec population previously studied, and recommended (published) reference concentrations; (iii) to measure and interpret an array of clinical chemistry parameters that relate to individual and/or general health.

The contaminants of concern measured in blood, urine and/or hair are listed in Table 1. The primary toxicological/ health outcome(s) for each is also indicated in the table. A detailed toxicological profile for each individual contaminant is provided in Appendix 1. Similarly, the various clinical chemistry (biochemistry) parameters measured in relation to general health are outlined in Table 2. A brief statement of the associated health outcome(s) is also provided.

Cyanide was not included in the suite of contaminants as it is chemically unstable and does not bioaccumulate. Further its levels in blood, and its metabolite thiocyanate in urine, are difficult to interpret (Lauwerys and Hoet, 2001).

3 STUDY METHODS

3.1 STUDY POPULATIONS

The cross-sectional study was conducted in the James Bay First Nation Cree Territory of Quebec in the communities of Oujé-Bougoumou and Nemaska, which respectively constitute the study and control populations. Sample collection took place in Oujé-Bougoumou from October 17th until October 31st 2002 and in Nemaska from November 4th until November 13th 2002. In each community, the Cree Beneficiary List was employed to randomly select potential participants (see Section 3.4 for further details). A first subgroup was recruited in the community of Oujé-Bougoumou consisting of 78 women of childbearing age in the range of 15 to 39 years old (see Table 3). The selection of this group of women was justified by the fact that exposure to the contaminants can be especially harmful to the fœtus. Moreover, an additional 147 subjects (see Table 3) were recruited from the three following age categories: 0-7 (n=35, 13 females and 22 males); 8-14 (n=21, 7 females and 14 males); males 15-39 (n=40); males and females $\geq$40 years (n=51, 28 females and 23 males). The selection was proportional to the age distribution in the community. The total number of 225 included 44 non-recruited volunteers upon a request from Oujé-Bougoumou Council on October 22nd. These individuals or their families originally resided around Lakes Chibougoumau and Doré prior to the establishment of the present Oujé Bougoumou community. The age and gender distribution of the 44 non-recruited volunteers were: 0 to 14 years of age (n=3 males); women of childbearing age, 15 to 39 years (n=3); men aged 15 to 39 (n=9); and individuals 40 years and over (n=29, 17 females and 12 males).

Moreover, a control group of 100 individuals was randomly recruited from the community of Nemaska to match the age distribution of the Oujé-Bougoumou participants: 0-7 years old (n=18, 9 females and 9 males); 8-14 years (n=11, 2 females and 9 males); women of childbearing age, 15 to 39 years (n=43); men 15 to 39 years (n=15) and individuals 40 and over (n=13, 5 females and 8 males).

Consequently, the total number of residents of Oujé-Bougoumou and Nemaska who were recruited or participated as volunteers and completed the study was 325. The recruitment and participation details are summarized in Table 4.

3.2 ETHICS AND CONFIDENTIALITY

The research project, information sheets and age-specific informed consent forms (see Appendix 2) were submitted and approved by the Ethics Committees of the Faculty of Medicine, Laval University (Quebec, Canada), and the Cree Board of Health and Social Services of James Bay; the McMaster University Research Ethics Board was also informed about the project activities.

All information collected in this study was kept strictly confidential. A numeric code was used to catalogue and identify questionnaires and biological samples. No name appears on any of the documentation, except on the master list that permits the linking of data records to the names of individuals. Only one of the two principal investigators (Dr. Dewailly) and his designated official had access to the identification list. The latter is to be destroyed once the medical follow-up has been

completed. All questionnaires and test results were kept in a secure (locked) filing cabinet at the Public Health Research Unit in Quebec for the duration of the study. At the end of the project, all of the (questionnaires and tests results) are to be returned to the CBHSSJB through the INSPQ-CHUQ Research Office and are to be destroyed after 5 years (scheduled for 2008)[1]. All the specimens (blood, hair, urine) are to be kept at the Public Health Research Unit in Quebec until the report is finalized to allow for verifications; unused portions of the samples are to be destroyed no later than December 31st 2003. No identifiers appear in the report nor in any future publications.

3.3 STAFF TRAINING

The training session for the local interviewers, who were recommended by the Band Council of both communities, was conducted by the field coordinator (a registered nurse). She was assisted by an epidemiologist from the Public Health Research Unit**,** Laval University, who also had qualifications in nutrition.

The training of eight interviewers took place in Oujé-Bougoumou on October 15th and 16th 2002, and a group of four in Nemaska, on November 4th 2002. This training consisted of explaining the purpose of the study and going through the documents to be used by the interviewers (namely the project information sheets, the age-specific informed consent forms, and study questionnaires). Furthermore, the local nurses, laboratory technician and research assistant (who had previously been trained by the field coordinator) participated in the training of the local interviewers, especially in the practice sessions involving the food frequency questionnaire. The latter sessions were supervised by the qualified nutritionist. One of the local interviewers was designated to focus on recruitment.

3.4 RECRUITMENT AND GENERAL ORGANIZATION

As already indicated (Section 3.1.), most of the recruitment was achieved by the designated recruiter by randomly selecting participants from the Cree beneficiary lists. This person was fluent in Cree and English and carried out the participants selection in both communities. Local radio announcements were made and tracks were distributed in both English and Cree to inform the community about the research project. Moreover, a poster was designed to indicate what was expected from the participants during the study. The local recruiter was responsible for phone contacts and invited the randomly selected residents to participate in the research project. She also arranged and supervised the appointments.

During the first visit to the healing centre, the local interviewers assisted each participant in completing the appropriate written consent form and questionnaires, which were available either in English or in Cree. All of these steps were supervised by the field coordinator assisted by the research assistant, both of whom were responsible for the quality control of all documents completed. If the participant was under 18 years of age, one of the parents or the guardian was invited to sign the informed consent form; moreover, if the participant was a child or a baby, the parent or the guardian answered the questionnaire for them. For all participants over 7 years of age, body weight, height, and

[1] The communities of Oujé-Bougoumou and Nemaska may be asked to extend this deadline if an anticipated study "Exposure and Baseline Health Assessments of the Eeyouch of Eeyou Istchee in relation to the Environmental Impacts of Mercury and Other Contaminants" is implemented in the context of the New Mercury Agreement.

waist and hip circumferences were measured and recorded (National Institute of Health, 1998; Lemieux et al., 1996) by the research nurse using standardized techniques. During the first visit, the research nurse also collected the blood and hair samples and blood pressure was measured. The participant was also asked to provide a first-morning urine sample, for which instructions, a collection cup and storage bottle were provided. He or she was asked to bring the specimen to the clinic the next morning. All pertinent information was recorded on the Specimen and Measurement Collection Sheet (see Appendix 3). Furthermore, during the same visit, the research nurse asked a number of questions about the participant's medical health status and psychological well-being. The questions were directed to the parent or guardian if the participant was too young to answer. This visit lasted approximately one hour and a half, and it was possible to schedule it at any time during the day (morning, afternoon or evening). A few visits were done at home for participants who asked for it or for whom it was not possible to come to the clinic (e.g., seniors).

3.5 QUESTIONNAIRES

Questionnaires were designed, tested and adapted for the specific purpose of this study. In the exposure/life-style questionnaire, questions were asked about the following issues: general socio-demographics (e.g., gender, age, family size, etc.); residency and household property information (e.g., places and duration lived there, age of present house, type of building materials, etc.); occupational details (job responsibilities, physical aspects, potential contaminant exposure); outdoor activities and hobbies (time spent outside, hunting details, play areas for youngsters, etc.); and life-style issues (concerning exercise, smoking, alcohol use, etc.); stress and psychological well-being. The food frequency questionnaire addressed the consumption of traditional subsistence and store-bought foods and the health questionnaire self-reported health outcomes (including use of medicines) (Copies of the questionnaires are provided in Appendix 4). The contents of these questionnaires were derived from validated survey instruments used by Santé Quebec for the health surveys conducted among the James Bay Cree in 1991 (Daveluy et al., 1991; Santé Quebec, 1994) and from the exposure/reproductive health studies conducted by the McMaster team in the west coast James Bay Cree communities of Fort Albany and Kashechewan (Nieboer et al., 2001 and 2003).

In the food frequency questionnaire consumption of fish, waterfowl and other birds, and game consumption received special focus; specifically the frequency during the last year and portion size (food model Po 3 was used, for which one portion is equivalent to 90 grams). This component of the questionnaire was prepared by the epidemiologist-nutritionist from the Quebec Public Health Research Unit. Community input was sought and received about culturally relevant issues and was field tested in a workshop held in Oujé-Bougoumou.

The exposure life-style and food frequency questionnaires were tested before the beginning of the study on October 1st and 2nd 2002, in Oujé-Bougoumou, by the nurse field coordinator. For the eight volunteers who agreed to participate, 2 participants were aged between 0-14 years old, 3 women and 1 man in the 15-39 group, and 2 others were over 40. The administration of the questionnaire required around 50 minutes. Appropriate adjustments were made to clarify/adjust any of the questions that were judged to be unclear or confusing.

3.6 BIOLOGICAL ASSESSMENTS

Six vacutainers of blood for a total of 37 ml were collected from each participant aged 15 years and over. Three of the vials of blood (for a total of 22 ml) were collected for the determination of the contaminants of concern and omega-3 fatty acids. For the participants 7 years old and younger, one vacutainer of 3 ml was collected for lead determination only. The 3 remaining vials were required for haematology and biochemistry analyses. Specific details have already been provided in Tables 1 and 2.

All participants over 7 years old were asked to collect a first-morning urine sample the day after their blood collection; collection cups and an 80 ml storage bottle were provided during the first visit to the clinic. The urine specimen was divided into 3 portions: 30 ml for the determination of contaminants (8 years old and over), 5ml for the clinical chemistry (biochemistry) and the leftover portion for dip-stick analysis (15 years old and over). These steps were completed by the laboratory technician immediately upon receiving the samples at the clinic.

Some of the blood specimens were centrifuged onsite for plasma isolation at 3000 rpm/10 minutes. All the biological samples (whole blood, plasma and urine) were stored in a freezer at -20^{o}C at the community nursing station, except for the 3-ml of blood from the children was kept at 4^{o}C.

For the hair samples, strands (about the length of a pen) were cut close to the scalp from the occipital region of the head by the research nurse. A haemostatic clamp was used to squeeze the hair sample to avoid any movement when cutting and when inserting the specimen into a plastic bag. The latter was stapled to avoid any movement during transportation and handling and was tagged with the participant's identification number, his or her birth date and the sampling date. The proximal end of the hairs (near the scalp) was clearly identified to allow the identification of the segments to be analyzed. Hair samples were obtained from every age group.

For the 325 individuals who participated in the study, 271 had their blood taken for all the contaminants of concerns. For the participants aged 0 to 7 years old, blood was analysed only for lead (total of 46 specimens with 7 missing). Urinary samples were not collected for participants aged 0 to 7 years; we obtained 266 urinary specimens (6 were missing). The urinary dip-stick analyses, which yielded biochemistry parameters, were done only for participants $\geq$15 years; 234 of such tests were carried out (6 urinary specimens were missing). Hair sampling was done for every participant; for the 0 to 7 year olds, only mercury was determined and all specimens were shorter than 2 cm (see below). The 0-2 cm hair segment was analysed for 258 participants (10 were missing, 4 specimens were too short). When the strand of hair was not long enough, only mercury was analysed. In total, 315 strands of hair were analysed for mercury (10 were missing), and 258 for arsenic, cadmium, copper, selenium and zinc. The 4-5 cm strands were analyzed only for mercury (n=229).

Biological samples were kept frozen in insulated containers with ice packs during their transfer to Quebec City, either by car or by bus at the end of the stay in the communities. In the laboratory, they were kept in a freezer at minus 20^{o}C, at the Public Health Research Unit, Research Center CHUL-CHUQ, Quebec.

3.7 LABORATORY ANALYSES

Laboratory determination of metals (cadmium, copper, lead, mercury and zinc), metalloids (arsenic and selenium) and organochlorines (OCs) were performed at the Direction de la toxicology humaine de l'Institut national de santé publique de Quebec, under the supervision of Mr. A. Leblanc. This facility is the reference laboratory for human toxicology in the Province of Quebec. Their expertise in the determination of toxic metals and persistent organic pollutants (POPs) in human fluids and tissues is recognized internationally. This laboratory participates in the QA/QC program of the Canadian Northern Contaminants Program and the Artic Monitoring Assessment Programme (AMAP).

The clinical chemistry (biochemistry) determinations were performed at the Hôpital Laval under the supervision of Dr. R. Lavoie, and the omega 3 fatty acids analyses in the Department of Nutritional Sciences, University of Guelph, Ontario, under the supervision of Dr. B. Holub.

Brief descriptions of the analytical methods/protocols employed are provided in Appendix 5. Because inductively coupled plasma mass spectrometry (ICP-MS) was used for the elemental determinations and a standard screen for organochlorines, additional elements were determined at no extra cost in blood, urine and hair and a number of pesticides in plasma. A list is provided in Appendix 5.

3.8 SAMPLE SIZE AND STATISTICAL ANALYSES

The sample size needed was estimated using the urinary arsenic database at the Unité de toxicologie du Quebec. The arithmetic mean concentrations (0.25 μmol/L) and standard deviation of 0.11 μmol/L corresponding to non-exposed individuals were used in the calculation with the requirements of a statistical power of 80% (a 0.20 risk of type II error), a 0.05 risk of type I error, a minimum detectable difference of 0.03 and a participation rate of 75% among Cree (Daveluy et al., 1991) (two-sided test). Based on these specifications, we estimated that 200 exposed (Oujé-Bougoumou) and 100 non-exposed (Nemaska) participants would allow us to detect significant differences in element concentrations between these two groups. The same calculation when applied to the organochlorines yielded a comparable estimate.

Statistical analysis were performed using SAS System for Windows: Release 8.02 Cary, NC: SAS Institute Inc, 1999-2001 (SAS, 2001). Univariate summary statistics reported include: geometric and arithmetic means, range, standard deviation and minimum; 10^{th}, 50^{th}, 90^{th} percentiles, and maximum for each contaminant. The data were examined (stratified) according to age and sex, to consumption of fish, birds, ducks and game and community; in addition, smoking status was considered for cadmium.

The geometric mean and its 95% confidence interval (95% CI) for each contaminant were compared between the 2 communities using the Student's *t* test. For concentrations below the detection limit (DL), a value of DL/√2 was used in the statistical analyses. Adjustment for potential confounders was achieved by multivariate analysis of variance. Potential confounders were selected from variables distributed differently between the 2 communities; the Chi-square (for categorical data) and the Student's *t* tests (for continuous data) were used in the community comparisons. The dependent variables were the concentrations of each contaminant. As they were not normally distributed, they were log-transformed prior to the analyses. The independent variables were age, sex and smoking; as well as the consumption of fish, wildfowl and game. Moreover, Pearson correlations were assessed for

the associations between contaminant concentrations and consumption of fish, wildfowl and game. In addition, results available from ongoing studies in Southern Quebec were used in the comparisons.

As indicated in Section 3.1, 44 non-selected volunteers from Oujé Bougoumou participated in the study. It was decided to include them in the statistical analysis to obtain a total number of 225 participants in this community. This was justified for two reasons. First, all women of age 15-39 years were contacted and invited to participate; only 3 individuals were added to this subgroup as non-recruited volunteers. Second, by including the non-recruited volunteers we achieved a more representative sample across all age groups. Consequently, an adjustment for this inclusion was not felt to be necessary.

4 RESULTS

4.1 OVERVIEW

The results presented in this report are stratified by age, sex, gender and community. First, descriptions of the participants and selected sociodemographic and lifestyle characteristics derived from the questionnaire are presented. Prevalences are then provided for the level of anxiety experienced in relation to environmental contaminants, breast-feeding and various self-reported health problems. Average concentrations of selected biochemical indicators of anaemia, hyper/hypo-thyroidism, diabetes and cardiovascular disease risk factors are also presented. Moreover, the results of a detailed food frequency questionnaire are reported, which provides information on the consumption of wild fish, wildfowl and eggs, game and liver or kidneys derived from them, including seasonal dependence. Plasma concentrations of various fatty acids as a proportion of the total in plasma phospholipids are compiled for consumers and non-consumers of wild fish or piscivorous birds. As for the biochemical parameters, the findings for the biomonitoring component of the study are stratified by age and gender and include concentrations of the following chemicals of concern: copper, selenium, zinc and PCBs in plasma; cadmium, lead and mercury in whole blood; arsenic, cadmium and copper in urine; and arsenic, cadmium, copper, lead, mercury, selenium and zinc in hair. Observed associations between contaminant levels and intake of wild fish, wildfowl and game are also reported.

4.2 SOCIODEMOGRAPHIC INFORMATION

The sociodemographic variables addressed in the questionnaire were selected to obtain information pertinent to the study. For the same reasons, personal life-style issues and related attributes were also pursued.

The distribution of the Cree populations of Oujé Bougoumou and Nemaska by sex and age are summarized in Table 3. Clearly, the size and age structure of the two communities were similar; both show that children 0-14 years of age constitute the largest proportion. A perusal of Table 1 indicates that all women between 15 and 39 years old were contacted and invited to participate in the study. It also suggests that the population lists were probably not up-to-date, since more women were invited than those listed. In Table 4, the participation and refusal rates are listed, stratified by gender and age, as well as the number no longer living in the community and those temporarily absent. The latter individuals did however receive an invitation and were counted as such. The refusal rate was higher in Nemaska (106/242=43.8%) than Oujé-Bougoumou (78/329=23.7%) resulting, respectively, in participation rates of 41.3% (100/242) and 68.4% (225/329). Weighting factors for the study populations are presented in Table 5.

Sociodemographic and lifestyle characteristics in each community are summarized in Table 6. Breakdown by gender and the proportion who were married were similar. In both communities, the Cree language was the most common by used at home, followed by English and French. A greater proportion of the Cree in Nemaska declared speaking English at home, whereas more Cree of Oujé-Bougoumou declared speaking French ($p<0.05$). The number of adults per household was significantly higher in Nemaska than Oujé-Bougoumou ($p < 0.001$). Moreover, the number of homes with children was higher in Nemaska (84.2%) than in Oujé-Bougoumou (78.2%) and the difference between the two

populations was significant (p=0.029). There were no significant differences between the two populations for the level of education and working status.

Details about housing are outlined in Table 7. Since the community of Oujé-Bougoumou was relocated in 1992, most residences were built during the same period using similar construction materials. While most of the housing information for Nemaska was derived from the questionnaire, that for Oujé-Bougoumou was supplemented/verified using a housing register. Most participants from both communities lived in a house; other possibilities being an apartment and/or senior residence/apartment. Moreover, recent renovations were rare in Oujé-Bougoumou (0.7%) as compared to Nemaska (14.5%). The type of drinking-water piping in the homes was documented and the use of copper piping was higher in Oujé-Bougoumou (99.7%) compared to Nemaska (73.1%), where PVC and plastic piping were also used (26.9%). The heating system in the houses of Oujé-Bougoumou was mostly derived from a central system employing hot water, whereas electrical heating was prominent in Nemaska. Only 5.7% of the houses were built during the last five years in Oujé-Bougoumou, and this might explain the low use of PVC and plastic piping and electric heating. All the differences mentioned were statistically significant ($p<0.001$).

The types of drinking water used by the two populations are described in Table 8. Most residents of both communities declared tap water as the main source while at home (68.8% in Oujé-Bougoumou and 89.9% in Nemaska, $p<0.001$). More people of Oujé-Bougoumou drank bottled water when they were in the community or in the bush, and the difference was significant between the two communities ($p<0.001$). The proportion consuming water from a spring was similar in both communities, whereas a greater proportion consumed lake or river water in Nemaska than in Oujé-Bougoumou (p=0.006).

Some features of bush-related activities and the use of firearms are compared in Table 9. The number of days spent in the bush during the year before the survey, appeared to be higher in Oujé-Bougoumou than in Nemaska. Hunting activities and the use of firearms were similar in both communities. A greater proportion of hunters in Oujé-Bougoumou used lead bullets compared to Nemaska (p=0.029). The use of lead and steel shells was significantly more popular in Nemaska than in Oujé-Bougoumou ($p<0.05$). About 10% of hunters from both communities declared washing their hands before smoking, following firearm use. A greater proportion of Oujé-Bougoumou hunters (p=0.012) also declared washing their hands before eating following the use of a firearm. In both communities, the ammunition and guns were mainly stored either inside the tent or in the house. A greater proportion of Nemaska hunters indicated keeping their clothes inside the tent when in the bush (p=0.034), whereas more Oujé-Bougoumou hunters (71.9%) than in Nemaska (9.6%) stored clothing and footwear in a sealed case/container ($p<0.001$).

A low proportion of participants in both communities reported boat repair or building, making their own lead bullets or sinkers for fishing, using lead-containing materials, or carrying out home renovations. In both communities, most children participated in outdoor activities (92.9% in Oujé-Bougoumou and 93.1% in Nemaska), and most of them had contact with pets, dirt, sand or rocks.

4.3 PREVALENCE OF ANXIETY RELATED TO ENVIRONMENTAL CONTAMINANT EXPOSURE

About 30% of the participants in both communities were considerably worried about environmental pollution. However, when asked if they were worried about possible risks to their health from mine tailings, 81.6% declared being so in Oujé-Bougoumou. The latter question was not asked of the Nemaska residents as it was judged not applicable.

4.4 PREVALENCE OF BREAST-FEEDING AND VARIOUS SELF-REPORTED HEALTH PROBLEMS

When the survey was conducted, no female participants in Oujé-Bougoumou breast-fed, while 3.1% did so in Nemaska (Table 12). Regarding the self-reported health problems, the most frequently declared in Oujé-Bougoumou were (in decreasing order): allergies, diabetes, respiratory troubles and high blood pressure. In Nemaska, allergies followed by high blood pressure and hypercholesterolemia were the most commonly reported outcomes. None of the reported conditions were significantly different (p 0.05) between the communities except diabetes. It was reported to be more prevalent in Oujé-Bougoumou (p=0.032).

4.5 PREVALENCE OF SELECTED CLINICAL CHEMISTRY (BIOCHEMISTRY) OUTCOMES

The prevalences of the biochemical indicators iron, folate, vitamin B12, thyroid hormones, and glycated haemoglobin (HbAlc) for the individuals 15 years of age and over interviewed 15 in Oujé-Bougoumou and Nemaska are summarized in Table 13. The various parameters measuring iron status yield a mixed message when comparing the results between Oujé-Bougoumou and Nemaska. Iron saturation was lower in Nemaska (p=0.007), while the mean red blood cell counts were lower in Oujé-Bougoumou ($p \leq 0.003$). The other iron-status measures (i.e., mean iron, iron-binding capacity, transferrin, ferritin, and haemoglobin) were not different between the two communities. Concentrations of folate, vitamin B12, TSH and Free-T4 were mostly normal (93-100%) in both communities. Glycated levels of haemoglobin were higher for the Oujé-Bougoumou respondents (p=0.054), which supports the higher prevalence of diabetes noted in Table 12.

4.6 PREVALENCE OF CARDIOVASCULAR DISEASE RISK FACTORS

More than fifty percent of Cree aged 15 years and over in both Oujé-Bougoumou and Nemaska smoked cigarettes every day (Table 14). This prevalence is considerably higher (perhaps > 3-fold) than that observed among Quebecers in 1992 (Table 15). In Nemaska, only 7.1% were non-smokers as compared to 19.4% in Oujé-Bougoumou (p=0.040). By contrast, a greater proportion (34.2%) of ex-smokers was found in Nemaska than in Oujé-Bougoumou (23.2%). Among current smokers, individuals from Nemaska were the heavier smokers (p=0.023). The prevalence of passive smoking (data not shown) was 21.5% in Oujé-Bougoumou and 11.6% in Nemaska, but the difference was not significant.

Prevalences of obesity (BMI ≥30) were not different between Oujé-Bougoumou and Nemaska; 60.4% and 71.0%, respectively (p>0.05). Only 12.9% of Oujé-Bougoumou participants and 7.4% in Nemaska had a BMI in the normal range. Over 75% of both populations, aged 15 years and over, had elevated

waist girth ($p \geq 0.05$). When comparing the prevalences of weight and abdominal obesity in the Cree communities with that of Quebecers (Table 15), it is clear that these outcomes are considerably more common than among the general Quebec population (perhaps as much as 5-fold).

A prevalence of high blood pressure of 23.1% was found in Oujé-Bougoumou and 20.2% in Nemaska ($p > 0.05$). When compared with the prevalence observed among Quebecers, it appears that high blood pressure occurs more frequently in Cree Communities than among the general Quebec population (perhaps 2-fold).

The prevalences of abnormal blood lipids were similar in the two Cree communities, except for the HDL cholesterol concentration. In Oujé-Bougoumou, more individuals had lower HDL levels (7.1% versus none in Nemaska ($p=0.026$); and 7.7% in Quebecers, Table 15). Generally speaking, the prevalences of high levels of total and LDL cholesterol were lower among the study participants than that observed among the general Quebec population (Table 15), but not so for triglyceride which appears somewhat higher in the Cree community.

Although the prevalences of elevated plasma glucose levels ($\geq$6.1 nmol/L) were comparable in the Cree communities ($p \geq 0.05$), they were considerably higher (perhaps 4-fold or more) than that reported for the general Quebec population (Table 15).

4.7 CONSUMPTION OF WILD FISH, WILDFOWL AND GAME

Food consumption data were obtained using the food frequency questionnaire administered during the survey. The survey revealed that 30.3% of consumers usually fish or hunt themselves. Other consumers declared obtaining fish, wildfowl or game mainly from relatives (64%), parents (52%) and friends (31%).

Food intakes were calculated by multiplying the frequency consumption on an annual basis by usual serving size in grams. Weekly mean intakes in grams of various wild fish species are provided in Table 16. The category "other" includes wild fish occasionally consumed by Cree populations such as red sucker and burbot. Walleye appears to be the most commonly consumed species. The total fish intake per week was 38.3 g in Oujé-Bougoumou and 35.6 g in Nemaska ($p \geq 0.05$). Mean intake of walleye was significantly higher in Oujé-Bougoumou than in Nemaska ($p=0.012$), whereas mean intakes of northern pike, lake sturgeon and white sucker were higher in Nemaska ($p \leq 0.033$). The proportion of fish consumers in Oujé-Bougoumou and Nemaska were 93.1% and 94.6%, respectively (Table 17). A greater proportion of consumers in Oujé-Bougoumou ate walleye, brook trout and lake trout compared to Nemaska ($p \leq 0.004$). Furthermore, there were significantly more consumers of lake sturgeon, northern pike, lake whitefish and white sucker in Nemaska ($p \leq 0.02$) than in Oujé-Bougoumou. However, the total fish intake per week did not vary between consumers in the two communities ($p \geq 0.05$).

Weekly mean intakes in grams of wildfowl among populations of Oujé-Bougoumou and Nemaska are reported in Table 18. The category "other" includes wildfowl occasionally consumed by Cree populations such as merganser and loon. Quantitatively, the most consumed wildfowl species were (in decreasing order): goose, partridge and willow ptarmigan. Wildfowl intake did not vary significantly between the two populations, except for partridge with a weekly mean intake higher in Oujé-Bougoumou ($p=0.040$). The total mean intake of wildfowl was 62.6 grams per week in Oujé-

Bougoumou and 48.3 grams in Nemaska ($p \geq 0.05$). The proportions of wildfowl consumers in Oujé-Bougoumou and Nemaska were 98.5% and 100%, respectively. There were significantly more consumers of golden eye duck in Oujé-Bougoumou ($p<0.001$) while more people consumed willow ptarmigan in Nemaska ($p<0.001$). For other wildfowl, the proportions of consumers were similar in both communities ($p \geq 0.05$). With the exception of the golden eye duck, the mean weekly intake of wildfowl species did not vary between consumers of both communities ($p \geq 0.05$); consumption in Nemaska of golden eye duck was two-fold higher ($p=0.013$; Table 19).

Mean intakes of game per week in the two communities are summarized in Table 20. The category "other" includes game occasionally consumed by Cree populations such as squirrel, lynx, marten, mink, weasel and muskrat. The most consumed game meat was (in decreasing order): moose, rabbit, American beaver, caribou and bear. The mean intake of rabbit was significantly higher in Oujé-Bougoumou ($p=0.005$), whereas that of caribou was higher in Nemaska ($p<0.001$). No significant differences were noted for other game species. Moreover, the total mean intake of game per week was similar in both populations ($p \geq 0.05$). The proportion of game consumers in Oujé-Bougoumou and Nemaska were 97.1% and 100%, respectively (Table 21). There were more consumers of American beaver ($p=0.045$) and caribou in Nemaska than in Oujé-Bougoumou ($p<0.001$). For other game species, the proportions of consumers were similar in both communities. With the exception of rabbit, for which the weekly mean intake was higher among consumers of Oujé-Bougoumou ($p=0.006$), mean intake of other game did not vary significantly between consumers of Oujé-Bougoumou and Nemaska.

Seasonal frequencies show that fish was mainly consumed during the summer in both populations, and walleye contributed the most to the consumption in Oujé-Bougoumou (Table 22); in Nemaska, lake sturgeon, northern pike and lake whitefish were the most common. More than 60% of both populations also consumed fish in spring and fall. The total fish consumption frequency during winter was significantly lower ($p=0.0003$) in Nemaska than in Oujé-Bougoumou. For other seasons, total fish consumption frequencies did not vary significantly between the two populations. Wildfowl (total) appeared to be consumed at similar levels in all seasons by both communities; goose contributed the most to the consumption, particularly in summer. Moreover, the total consumption frequency of wildfowl during summer was significantly higher in Nemaska ($p=0.001$) than in Oujé-Bougoumou. For other seasons, total wildfowl consumption frequency did not vary between the two populations ($p \geq 0.05$). Game was mainly consumed in fall and winter, and moose contributed the most to the diet. The total consumption frequency of game in Nemaska was significantly higher than in Oujé-Bougoumou during the summer ($p=<0.001$). For other seasons, the total game consumption frequencies did not vary significantly between the two populations.

In Oujé-Bougoumou, mean intake of fish and wildfowl per week did not vary significantly according to age groups (Table 23), but for game it did. Individuals over 40 roughly ate twice as much as did the 15-39 age group ($p=0.003$). In Nemaska, weekly mean intakes of fish, wildfowl and game differed between these two age groups. In each case, the consumption was higher in the older group ($p \leq 0.05$). Mean intakes of fish, wildfowl and game did not vary significantly between the Oujé-Bougoumou and Nemaska participants aged 15-39 years. However, those over 40 in Nemaska consumed more wild fish ($p=0.021$).

4.8 CONSUMPTION OF EGGS, LIVER OR KIDNEYS OF FISHING AND HUNTING PRODUCTS

Fish eggs were not often consumed in the two populations with the consumption frequency being less than 10% among fish consumers (data not shown). The consumption of fish skin varied according to species. The fish species for which the skin was most often consumed by the Cree were (in decreasing order): brook trout (66%), lake trout (40%), white sucker (33%), lake whitefish (30%) and northern pike (27%).

Regarding wildfowl, 20% of goose consumers declared eating goose liver. For other wildfowl species, the liver was not often consumed with the consumption frequency varying between 0 to 6%. There were very few consumers of wildfowl eggs, although 5% of goose consumers declared eating goose eggs.

The consumption of game organs was most popular and appeared to be more frequent among consumers in Oujé-Bougoumou than of Nemaska. The game species for which the liver was most often consumed were (in decreasing order): American beaver (34%), rabbit (22%) and moose (22%). Moreover, the game species for which the kidneys were most often eaten were moose (51%), rabbit (33%) and beaver (31%).

4.9 CONCENTRATIONS OF FATTY ACIDS IN PLASMA PHOSPHOLIPIDS

The relative concentrations of fatty acids in plasma phospholipids of the Oujé-Bougoumou and Nemaska participants for consumers and non-consumers of fish and piscivorous birds (such as loon, merganser, etc.) are shown in Table 24. The plasma phospholipid concentrations of fatty acids are expressed as relative percentages of total fatty acids by weight. Concentrations of EPA, DHA and total n-3 fatty acids were higher in Oujé-Bougoumou compared to Nemaska ($p \leq 0.012$). In Oujé-Bougoumou, these compounds were most prevalent among consumers of fish and piscivorous birds ($p \leq 0.045$). In Nemaska, concentrations of n-3 fatty acids did not vary significantly between consumers and non-consumers. By contrast, concentrations of n-6 fatty acids and total PUFAs were higher in Nemaska than in Oujé-Bougoumou ($p < 0.001$). Consequently, the ratio of n-3 to n-6 fatty acids was higher in Oujé-Bougoumou than in Nemaska ($p = 0.003$). Concentrations of monounsaturated fatty acids (MUFA) and saturated fatty acids (SFA) were also higher among the population of Oujé-Bougoumou than with the population of Nemaska ($p \leq 0.012$).

Relative concentrations of fatty acids in plasma phospholipids are compared by age groups in Table 25. Plasma concentrations of EPA, DHA, EPA+DHA and total n-3 fatty acids increased significantly with age in both populations ($p \leq 0.001$). Conversely, the younger groups in both communities showed higher concentrations of n-6 fatty acids ($p \leq 0.002$). Accordingly, the ratio of n-3 to n-6 fatty acids in both communities was significantly higher among the over 40 age group compared to those 15-39 years of age. Concentrations of total PUFAs did not exhibit a with age in either population. In Oujé-Bougoumou, higher concentrations of MUFAs and lower concentrations of SFAs were observed among individuals between 15-39 years of age as compared to the older individuals; in Nemaska, no significant difference was found between age groups for both these fatty-acid series. When comparing concentrations of fatty acids of the two age groups between the communities, n-3 fatty acids, n-3 to n-6 ratios, MUFAs and SFAs were higher among the younger individuals in Oujé-Bougoumou ($p \leq 0.001$ to 0.034). Conversely, concentrations of n-6 fatty acids and total PUFAs were

higher among the younger group in Nemaska ($p \leq 0.001$). For Cree aged 40 years and over, concentrations of fatty acids were not linked to community ($p \geq 0.05$).

Finally, the mean relative n-3 fatty acid concentrations of the Oujé-Bougoumou and Nemaska study populations were compared with other populations of Quebec (Dewailly 2001a, 2001b, 2002). Mean concentrations of n-3 fatty acids among the Cree of Oujé-Bougoumou and Nemaska approached those of James Bay Cree, surveyed in 1991 with a relative concentration of 5.00%, but are higher than those observed among the general population of Quebec in 1990 (mean = 2.57%). By contrast in 1992, the Inuit of Nunavik showed considerably higher relative concentrations of n-3 fatty acids (mean = 9.71%).

4.10 MEAN PLASMA CONCENTRATIONS OF PCBS (MEASURED AS AROCLOR 1260)

Results for the plasma PCBs concentrations measured as Aroclor 1260 in μg/kg lipids and in μg/L are reported in Tables 26 and 27, respectively. For the over 40 age group the geometric mean for total PCBs was 37.12 μg/L [95% confidence interval (CI) 28.15-48.94] in Oujé-Bougoumou and 22.84 μg/L (95% CI; 13.60-38.36) in Nemaska (p=0.122). Moreover, the observed PCBs concentrations in this group were comparable to what has been found in Quebec Inuit population for which geometric means were 16.1 μg/L for the entire population and 50.0 μg/L in the older group (Dewailly et al., 1994). Although the Oujé-Bougoumou mean plasma concentrations were generally higher compared to Nemaska, they were only significantly different for the group of women aged 15 to 39 years old (p=0.022). As indicated in Table 28, both males and females 15-39 years old and over 40 showed some exceedances of the levels of concern. More specifically, for the women of reproductive age 29.5% (Oujé-Bougoumou) and 23.3% (Nemaska) had concentrations above 5μg/L (see Table 28); while 7.5% (Oujé-Bougoumou) and 6.7% (Nemaska) of men aged 15-39 exceeded the 20μg/L level of concern guideline, while 72.6% (Oujé-Bougoumou) and 61.5% (Nemaska) of men and women over the age of 40 were above this concentration. By contrast, one male from Oujé-Bougoumou in the 15-39 age bracket exceeded the action level of 100 μg/L while 17.7% (Oujé-Bougoumou) and 7.7% (Nemaska) of the men and women 40 years and older did so.

4.11 MEAN PLASMA CONCENTRATIONS OF PCB CONGENER 153

In Appendix 7, the detection frequencies and mean concentrations are presented for all 14 PCB congeners and 11 chlorinated pesticides are presented (see Tables A1 to A10). P values for community differences in the concentrations of these contaminants were only calculated if the detection frequency $\geq$70%; the latter constitutes an arbitrary cut-off point, although it is consistent with current practices (Demers et al., 2000; Van Oostdam and Tremblay, 2002). This occurred for PCB congeners 99, 118, 138, 153, 170, 180 and 187, as well as 3 chlorinated pesticides (p,p'-DDE, hexachlorobenzene and trans-nonachlor). The geometric means for the concentration of these compounds are plotted in Figures 1 and 2. There is a trend to higher concentrations in Oujé-Bougoumou relative to Nemaska and this reaches significance ($p \leq 0.05$) for the PCB congeners 99, 118, 170 and 187. Moreover, we observed that wild fish and game consumption were positively associated with plasma Aroclor 1260 concentrations ($r=0.23$, $p<0.0002$ and $r=0.32$, $p<0.0001$, respectively) and congener 153 plasma levels ($r=0.23$ and $r=0.32$, $p<0.0001$, respectively) (data not shown).

Congener 153 is the most prevalent of the 15 measured (see Table A1); the observed plasma concentrations of PCB congener 153 are presented in Table 29. The group ≥40 years old, shows a higher arithmetic mean than the other groups, with 1197 µg/kg lipids (and a standard deviation (SD) of ±1033) in Oujé-Bougoumou, and 736±832 µg/kg lipids in Nemaska. These results are considerably higher than what was found in a cohort of 305 women from the Quebec city area, for which the arithmetic mean was 55.6±23.4 µg/kg lipids (Demers et al., 2000). The Oujé-Bougoumou mean plasma concentrations of congener 153 were generally higher than in Nemaska and were significantly different for the 8 to 14 year old group ($p=0.035$) and women aged 15-39 years old ($p=0.019$).

4.12 WHOLE BLOOD CONCENTRATIONS OF CADMIUM

As illustrated in Table 14, the current prevalence of smoking among the participants in both communities was close to 60%, with ex-smokers constituting another 23% (Oujé-Bougoumou) or 34% (Nemaska). The prevalences of non-smokers were 19% (Oujé-Bougoumou) and 7.1% (Nemaska). The link between cadmium exposure and smoking is clearly illustrated by a perusal of the results in Tables 30, 31 and 32. This compares with prevalences of 68.2% (non-smokers and ex-smokers) and 31.8% (smokers) for the Southern Quebec group (Table 15).

For smokers and non-smokers combined (see Table 30), the blood cadmium concentrations were similar in the two study communities ($p>0.05$). By contrast, a comparison with a Southern Quebec comparator community reveals that for all age groups over 15 years except the over 40 group in Nemaska, cadmium levels were considerably higher in the Cree communities ($p<0.001$). The difference is maintained for non-smokers only for Oujé-Bougoumou ($p=0.028$), implying that higher concentrations were observed in this community compared to Nemaska, as observed ($p=0.039$) (Table 31). As demonstrated in Table 32, a substantially larger proportion of non-smokers in Oujé-Bougoumou compared to Nemaska exceeded the level of concern of 4.98 nmol/L, with none above the action level of 44.5 nmol/L. For smokers in both Cree communities, there were quite a few exceedances of both the level of concern of 25 nmol/L and the action level of 44.5 nmol/L.

The involvement of smoking as a source of cadmium is supported by the strong positive association observed between whole-blood cadmium and plasma cotinine concentrations, with $r=0.76$ and $p<0.0001$.

4.13 WHOLE-BLOOD CONCENTRATIONS OF MERCURY

The mercury concentrations were found to be higher in most age groups in Oujé-Bougoumou compared to Nemaska (Tables 33 and 34), and the difference reached significance for women aged 15 to 39 years, those over 15 years old and the total study population ($p\leq0.013$). The observed concentrations were considerably higher than the values reported for the Southern Quebec reference group for all adult populations ($p<0.001$). Mercury was moderately elevated and increased with age. The maximum percentile levels were below the level declarable to Public Health in Quebec (Table 35) for the Southern Quebec population and for those aged 8-14 years in both Cree communities. The percentages above the concern and action levels are shown in Table 35. For both communities, no individual child exceeded these concentrations although it is clear that there were quite a few exceedances of the levels of concern and only three individuals were above the pertinent action level.

4.14 WHOLE BLOOD CONCENTRATIONS OF LEAD

Lead concentrations were significantly higher (p=0.009) in Oujé-Bougoumou only for the 0 to 14 year olds (Table 36) and only for the over 40 group in Nemaska (p=0.005). However, the levels were significantly higher in the Cree communities compared to the Southern Ontario comparator community only for the 40 years and older group ($p \leq 0.002$); they were marginally lower in the over 15 group in Nemaska (p=0.008). It is clear from the data in Table 37 that there are relatively few exceedances over the level of concern and one above the action level.

4.15 PLASMA CONCENTRATIONS OF COPPER, SELENIUM AND ZINC

From the data in Table 38, it is clear that there were no differences in plasma copper concentrations between the two communities, nor were they different from the values reported for the Quebec comparator group. Although there were some exceedances of the level of concern among the women in the 15-39 years age bracket, the study questionnaire revealed that 7 of the 15 women acknowledged being pregnant and 6 were taking oral contraceptives.

Plasma selenium concentrations (Table 40) were higher in Nemaska compared to Oujé-Bougoumou, reaching significance for the women 15-39 years (p=0.015), the over 40 group (p=0.036), and all participants over 8 or 15 years of age ($p \leq 0.002$). In Oujé-Bougoumou, the measured concentrations were lower than those for the Quebec comparator group in the over 15 and over 40 categories ($p<0.001$), while for Nemaska this only occurred for the total group ($\geq$15 years) (p=0.025). It is clear from the data in Table 41 that only one individual exceeded the level of concern and not one was over the action level.

Zinc plasma levels (Table 42) were generally higher in Oujé-Bougoumou, reaching significance for the following groups: children 8-14 (p=0.009); all individuals over 8 years (p=0.003) or 15 years (p=0.023). The observed concentrations were marginally lower in the Cree communities compared to the Southern Quebec comparator group, reaching significance only for two Nemaska groups: women 15-39 years (p=0.012) and all individuals over 15 ($p<0.001$). As indicated below (Section 4.20.), few individuals exceeded the level of concern of 22 μmol/L and none were above the action level.

4.16 URINARY CONCENTRATIONS OF INORGANIC (NON-DIETARY) ARSENIC, TOTAL ARSENIC, CADMIUM AND COPPER

As indicated in footnote 1 of Table 43, inorganic arsenic includes arsenate, arsenite and their metabolites (monomethylarsenic acid, MMA; and dimethylarsinic acid, DMA). Traditionally this was referred to as non-dietary arsenic as most of the excreted arsenic is present as organic arsenic which is derived primarily from seafood. By contrast, inorganic arsenic was believed to be linked to environmental inorganic arsenic forms. This is more fully explained in the discussion (section 5.2.2).

Inorganic arsenic in urine were generally higher in Nemaska (Table 43), reaching concentrations significance for children 8-14 years (p=0.008) and the entire study population (p=0.007); they approached significance when all study subjects older than 15 were considered (p=0.053). Because of low detection frequencies due to a higher detection limit, means could not be calculated for the Quebec comparator community. Thus comparing the Cree communities' data with Quebec is not very

helpful. Because of the higher detection limit for total arsenic relative to the observed concentrations (Table 44), intercommunity comparisons are not very certain. Qualitatively speaking, the observed concentrations for Nemaska again appear to be higher than for Oujé-Bougoumou, and those in Nemaska comparable to the Southern Quebec group. It is obvious from the data in Table 45, that there were very few exceedances of the level of concern and none above the action level. Interestingly, there was a moderate log scale correlation between urinary inorganic and total arsenic concentrations for all participants ($n=266$, $r=0.45$, $p<0.0001$). A weak relationship between total urinary and whole-blood arsenic (data not reported) was also noted ($n=266$, $r=0.32$, $P<0.0001$).

Observed cadmium concentrations in urine are summarized in Tables 46 (independent of smoking status), 47 (for non-smokers), and 48 (exceedances of the concern and action levels). As for total arsenic, the relatively high detection limit and low detection frequencies renders inter-community comparisons uncertain. This is especially so for the subgroup of women 15-39 years of age. Nevertheless, the data are suggestive that for smokers and non-smokers combined there are very little intercommunity differences between the Cree and Southern Quebec groups. For the same reason there is little merit in intercommunity comparisons for non-smokers (Table 47). Prudence also appears warranted when interpreting the seven exceedances of the level of concern reported in Table 48. Interestingly, there was a weak association for all participants between urinary and whole-blood cadmium ($n=266$, $r=0.31$, $p<0.001$).

From the urinary copper data summarized in Table 49, it is evident that relative to Nemaska and the Southern Quebec groups, females aged 15-39 in Oujé-Bougoumou exhibited higher concentrations ($p\leq0.021$). This was not seen in other subgroups. Consequently, the plasma and urinary copper results corroborate each other. When considering all participants in both communities, there was a weak link between serum and urinary concentrations ($n=265$, $r=0.22$ and $p=0.0003$). As pointed out in Section 4.15., pregnancy and the use of oral contraceptives appears to account for these observations. As illustrated in Table 50, there were few exceedances of the concern level.

4.17 HAIR CONCENTRATIONS IN THE 0-2 CM SEGMENT OF TOTAL ARSENIC, CADMIUM, COPPER AND LEAD

A perusal of Table 51 indicates that the detection frequencies of arsenic in hair samples ranged 69.2-90.0%, being slightly higher in the Oujé-Bougoumou study group. In spite of this, a consistent pattern of no inter-community differences is evident. Some of the maximum concentrations reported in Table 51 for Oujé-Bougoumou exceed the suggested level of concern of 4.0 nmol/g, and such values occur in each age subgroup; those for the Nemaska subjects are below the concern concentration. None of the 90th percentile values in Nemaska exceeds the concern level, while in Oujé-Bougoumou all but one do. No association was evident between arsenic in hair and that in urine (inorganic or total), not with whole blood arsenic.

Low detection frequencies of cadmium in hair make intercommunity comparisons uncertain. It is noticeable, however, that fewer samples in Nemaska had detectable cadmium compared to Oujé-Bougoumou. Nevertheless, for the age groups 8-14 and men 15-39 years of age community differences are not apparent. Only one of the maximum concentrations reported exceed the suggested level of concern of 4.4 nmol/g, and none of the 90th percentile values does so. For the data set of all

participants in both communities, there was a weak negative relationship between cadmium in hair and whole blood (n=258, r=-0.22, p=0.0005), but none with urine.

Copper levels in hair (Table 53), like those in urine, were elevated for the Oujé-Bougoumou subgroup of females 15-39 years of age ($p<0.001$), and this might have been expected. Two of the maximum concentrations reported exceed the suggested concern value of 0.63 µmol/g, but none of the 90th percentile values do. However, there was no correlation between copper in hair and urine, nor with that in plasma.

For three of the subgroups, lead in hair concentrations (Table 54) was numerically higher in Oujé-Bougoumou and the differences were significant ($p<0.001$, women 15-39 years), or nearly so ($p=0.052$, children 8-14 years; $p=0.056$, men 15-39 years). Three of the maximum levels reported in Table 54 exceeded the suggested level of concern of 26.5 nmol/g and one of the 90th percentile values; all were in Oujé-Bougoumou. A highly significant association that explained only 10% of the variation occurred between lead in hair and that in whole blood when considering the participants in both Cree communities (n=258, r=0.31, $p<0.0001$).

4.18 HAIR CONCENTRATIONS OF TOTAL AND METHYL MERCURY

Concentrations of total mercury in the 0-2 cm hair segments are reported in Tables 55 (in units of nmol/g) and 56 (in µg/g). Inter-community comparisons are not possible for the 0-14 and females 15-39 subgroups since the detection frequencies fall below 60%. For the remaining groups, there was no difference between Oujé-Bougoumou and Nemaska, although when considering all study subjects the mean was higher in Oujé-Bougoumou ($p<0.001$).

Health Canada (HC 1995) has set the basal total hair mercury level at 1 µg/g, the "no risk" level at 6 µg/g, 10 µg/g as maternal levels with observed motor and CNS effects in infants, the 6-30 µg/g range as at "increased risk", and 30 µg/g as the "at risk" level, corresponding respectively to 4, 20, 40, 40-100 and 100 µg/L in whole blood. Interestingly, the Cree Board of Health and Social Services of James Bay in Quebec (Dumont et al., 1998) has used a somewhat similar grouping: <6.0 µg/g as the "no risk" level; ≥15 µg/g as the intervention level for women of childbearing age; and ≥30 µg/g as the intervention level for all others. In both whole blood and hair, mercury is primarily present as methyl mercury. Based on these guidelines, the following categories for total mercury in hair are used in the present study: <29.9 nmol/g (<6 µg/g) as the "no risk" level; 29.9-69.8 nmol/g (6-14 µg/g) as "at increased risk"; and ≥74.9 nmol.g (≥15 µg/g) as "at risk". Samples are subdivided into these three concentration categories in Table 57. Clearly, the majority of the results fall into the "no risk" group. Only for the over 40 age group can a substantial number of mercury hair concentrations be characterized as "at increased risk" (33.3% in Oujé-Bougoumou and 15.4% in Nemaska). None of the reported values across the subgroups in the two communities reached the "at risk" concentrations.

It should be noted that the absorption of mercury vapour released from amalgam fillings do make small contributions to the observed blood (Berglund and Molin, 1996; Skare and Engqvist, 1994), urine (Levy et al., 2004) and hair (Lindow, 2003) mercury concentrations. Little methyl mercury is excreted by way of urine, and urine mercury primarily reflects exposure to inorganic forms of mercury.

A perusal of the data in Tables 58 and 59 suggest that the total mercury levels in the 4-5 cm segments were somewhat lower than in the 0-2 cm proximal segment. A statistical analysis showed this to be so for Oujé-Bougoumou (mean differences of -2.0$\pm$2.6, $p \leq 0.0001$), but not for Nemaska (mean difference of 0.35$\pm$1.3 with p=0.25). There were no intercommunity differences for the total mercury in the 4-5 cm hair segments, not even when considering all subjects as was the case for the 0-2 cm samples.

For 10% of the hair samples, methyl mercury was also determined in the 0-2 cm and 4-5 cm segments. The results are provided in Table 60. Like for total mercury, there were no significant intercommunity differences, although the mean values were numerically higher in Oujé-Bougoumou. In absence of age stratification, it is not possible to compare the relative magnitudes of total mercury and methyl mercury other than that they appear comparable.

Positive associations were observed between whole-blood and hair total mercury levels. For the 0-2 cm hair segments r=0.86 and p<0.0001, while for the 4-5 cm segments r=0.77 and p<0.001. The hair/blood concentration ratio was 242:1, near the value of 250 previously observed (WHO, 1990).

4.19 HAIR CONCENTRATIONS OF SELENIUM AND ZINC

Hair selenium levels were higher (Table 61) in Oujé-Bougoumou for three of the subgroups (women 15-39 years, p=0.012; over 40 group, p=0.040; all subjects, p<0.001). All but one of the maximum concentrations reported exceeded the suggested level of concern of 20.3 nmol/g. By contrast, the 90th percentile values were comparable to or lower than this value. A weak relationship between selenium in hair and whole blood (data not shown) was evident when considering all participants (n=258, r=0.23, p=0.002). This association was not observed for hair and plasma, even though selenium in plasma and whole blood were correlated (n=271, r=0.41, p<0.001).

Zinc-in-hair concentrations are reported in Table 62. The observed levels were higher in Oujé-Bougoumou for females 15-39 years (p=0.001) and for all subjects (p<0.001). The maximum concentrations indicated were of comparable magnitude to the suggested level of concern of 4.6 μmol/g, while all the 90th percentile values were below it. No relationship between zinc in hair and plasma was evident.

4.20 SUMMARY OF EXCEEDANCES

Contamination and medium-specific exceedances are summarized in Table 63, along with the suggested concern and action levels. Quite a few individuals had concentrations above that level of concern for all contaminants except lead, selenium and zinc. Exceedances were most numerous for cadmium in whole blood (OJ=27, N=2, non-smokers; OJ=58, N=32, smokers), mercury in hair (OJ=20, N=2) and whole blood (OJ=30, N=7), and PCBs in plasma (OJ=23, N=10, women of reproductive age; OJ=40, N=9, all men and women over 40) with most occurring for individuals in Oujé-Bougoumou. Action level exceedances occurred for cadmium in whole blood for smokers in both communities (OJ=14, N=12), and for PCBs in plasma (OJ=10, N=1, all men and women over 40) were most prominent in Oujé-Bougoumou; one man over 40 in Nemaska exceeded the mercury-in-whole-blood action level and one female of reproductive age in Oujé-Bougoumou for lead in whole blood.

4.21 ASSOCIATION BETWEEN CONTAMINANT CONCENTRATIONS AND CONSUMPTION OF FISH, WILDFOWL AND GAME AND OTHER EXPOSURE RISK FACTORS

The summary of correlation coefficients and associated p values provided in Table 64 for the univariate analysis emphasizes a number of trends. Of these, the dependence of mercury in hair and whole blood on consumption of fish, wildfowl and game is the most prominent (nearly all p values are <0.001). This occurs in both Cree communities. Lead in whole blood also shows a similar dependence on these three traditional food sources (p values range from <0.001 to 0.029). For the Oujé-Bougoumou participants, there is a suggestion of a positive dependence for whole-blood cadmium in non-smokers on fish (p=0.015) and game (p=0.053) consumption. There were insufficient data points for Nemaska to examine this trend. Only in Oujé-Bougoumou was plasma selenium associated with fish consumption (p=0.024). Total PCBs correlated strongly with consumption of fish and game in both communities (p<0.001 or 0.026), but with wildfowl only in Nemaska (p=0.028).

Not shown in Table 64 are the results for methyl mercury in the 0-2 cm and 4-5 cm hair segments. A univariate analysis was carried out on data for non-consumers of fish and individuals with methyl mercury below the detection limit. For the 0-2 cm segments, r=0.46, with p=0.0074 and n=33; similarly, r=0.38, p=0.0288 and n=33 for the 4-5 cm hair segments.

The comparison of mean contaminant concentrations for various exposure risk factors in Table 65 for the Oujé-Bougoumou suggests that participation in hunting is a predictor of whole-blood lead (p<0.001) as well as smoking status (with smokers and ex-smokers having higher levels, p=0.040). The sources of drinking water had no apparent effect on plasma zinc and urinary arsenic concentrations, but consumption of drinking water collected from a spring did influence lead in whole blood (p=0.036). These lead-related observations are presented in Table 66 for Nemaska: hunting activities (p=0.014); smoking (p<0.001) and consumption of lake/river water (p=0.033).

Multivariate analysis (see Table 67) indicates that the statistical significance and the actual differences in means corresponding to the higher total mercury concentrations in Oujé-Bougoumou compared to Nemaska in whole-blood (p=0.003) and the 0-2 cm hair segments (p<0.001) showed little dependence on adjustment for age, gender and consumption of wild fish, wildfowl and game. The same conclusion applies to the difference in plasma zinc (p=0.003), which was also higher in Oujé-Bougoumou, and for the higher plasma selenium (p=0.001) and urinary inorganic arsenic (p=0.007) observed for the Nemaska participants. Adjustment enhanced the significance somewhat of the higher total PCBs in plasma found for the Oujé-Bougoumou participants (p=0.037 to 0.020). By contrast, significance was reduced (i.e., larger p values) and the differences between mean concentrations in the two communities were decreased on adjustment of total mercury in the 4-5 cm hair segments, methyl mercury in both hair segments, and lead in whole blood. For the latter, additional adjustments for participation in hunting and smoking status were considered.

5 DISCUSSION

5.1 PREAMBLE

5.1.1 Limitations of the Study

Sample size (power) calculations using the observed geometric means for inorganic arsenic in urine, lead and mercury in whole blood and PCBs in plasma for the total populations studied in Oujé-Bougoumou and Nemaska indicate that caution is warranted in interpreting the differences between the subgroups of these two communities. In the calculation, the usual parameters were specified (statistical power of 80% corresponding to a 0.20 risk of type II error and 0.05 risk of type I error). A sample size of 30 or 40 is needed for each of the groups compared, depending on the contaminant. This condition is fulfilled only for the comparison of the subgroup of women 15-39 years of age and for all groups combined (total; ≥8 years or ≥15 years), as well as for the 0-14 year olds in the case of whole-blood lead and mercury in hair because these contaminants were also measured for children 0-7 years of age. Therefore, any conclusions based on the comparison between Oujé-Bougoumou and Nemaska for the 8-14 year olds, men 15-39 years, and the over 40 years of age groups are to be deemed uncertain. This does not apply to mercury in whole blood for the comparison between the Cree communities and the southern Quebec comparison group, because of the large differences in the observed mean concentrations.

Quantitative estimates of fish consumption appear relatively low among the participants of the present study. Subsequent to the study, some consultations were pursued with knowledgeable individuals in Oujé-Bougoumou, that revealed that fish consumption is in fact low among residents, in particular among individuals under the age of 50. We should also take note that the quantitative estimates only include wild fish intake and not that of store-bought fish. Further, fish consumption may be influenced by the amount of other traditional foods eaten, specifically game and wildfowl. It should also be reiterated that concentrations of n-3 fatty acids, in particular EPA and DHA for which fish is the main source, were higher among fish consumers than non-consumers only in Oujé-Bougoumou. Nevertheless, the reported fish intakes appear incongruent with the observed concentrations of n-3 fatty acids based on previous surveys of non-native Quebécers (Dewailly et al., 2001a) and James Bay Cree (Dewailly et al., 2002). For the latter, the mean daily intake of fish for the inland Cree communities in 1991 was 34 g, based on a 24-hour dietary recall. How different this is from the consumption data reported in the present study is difficult to assess, due to the dissimilarity of the dietary recall questionnaires employed. Because of these uncertainties, a follow-up assessment of the consumption of traditional foods is encouraged.

5.1.2 Toxicological Profiles

To simplify and contain the length of the discussion, the toxicological details for the essential and toxic elements measured are provided in Appendix 1. The individual toxicological profiles review the salient public health, occupational health and toxicological issues as well as providing background on normal (background) concentration ranges reported in the published literature. Some background on concern and action levels is also included where appropriate.

5.2 CONTAMINANTS POSSIBLY RELATED TO MINE TAILINGS RESIDUES

5.2.1 Overview

It is concluded that there is little evidence to support the notion that the Oujé-Bougoumou residents are unduly exposed to the elements for which there is evidence of mobilization from mine tailings, namely arsenic, copper, selenium and zinc.

5.2.2 Arsenic

The fact that inorganic arsenic in urine was generally higher in Nemaska, rather than Oujé-Bougoumou, suggests that arsenic released from tailings does not result in an unusual exposure of the residents of the latter community. The total urinary arsenic concentrations observed support the same trend, and further, are suggestive of the fact that the levels noted for the Nemaska participants are of comparable magnitude to those previously found for the Southern Quebec comparator group. The observed associations between inorganic and total arsenic in urine, and between total arsenic in whole blood and urine (Section 4.16), reinforce that arsenic in urine reflects that in the blood compartment. The implication of this is that, on average, inorganic and total arsenic have similar sources. Conversely, the absence between a link between arsenic in urine (or whole blood) and that in hair suggests that this measure does not reflect the amount of arsenic in blood. It is also encouraging that there are very few exceedances of the level of concern in either community and none of the action level (Table 63), and that the measured hair arsenic levels were no different between the two Cree communities. The upper 95% confidence limit of 2.11 nmol/g observed in hair (see Table 51) agrees with the range of 1.0-4.0 nmol/g reported by others (Yamauchi et al., 1989; Das et al., 1995; Wilhelm and Idel, 1996).

As pointed out in Section 4.16, in the past, dietary arsenic was believed to originate primarily from the consumption of seafood in the form of organoarenicals such as arsenobetaine and arsenosugars (Le et al., 1994; Appendix 1). These compounds are considered to be non-toxic and are excreted rapidly and predominantly unchanged in the urine. Environmental (in air, water and soil) and occupational exposures were understood to be the primary sources of inorganic compounds (mostly arsenates and arsenites). The latter are metabolized in the body to MMA and DMA (see Section 4.16). The sum of arsenate, arsenite, MMA and DMA was therefore designated as non-dietary or the inorganic fraction. Even though fish and shellfish remain the primary sources of arsenic intake (Dabeka et al., 1993), other foods do contribute to both organic and inorganic forms as outlined below (Schoof et al., 1999).

Speciation of arsenic in foods is still in the early stages of development. However, recent publications have confirmed that seafood contains little inorganic arsenic, although shrimp contain a small fraction of arsenic as DMA (Yost et al., 1998; Schoof et al., 1999). Rice appears to contain significant proportions of inorganic forms, including DMA, as do edible mushrooms (Slekovec et al., 1999). Further, edible parts of vegetables contain significant amounts of inorganic arsenic (mostly arsenite and arsenate), estimated to be between 25 -100% in contrast to meats (Schoof et al., 1999).

Based on the above information and of total arsenic in Canadian foods and their relative consumption (Dabeka et al., 1993), possible candidates other than seafood are: (wild rice), mushrooms, meat and poultry, bakery goods, cereals, sugar and candies. Individuals who exceeded the level of concern of

0.25 µmol/L in urine (Tables 45 and 63), might take the opportunity to review the balance of the consumption of these food items. Urinary arsenic (inorganic or total) showed no dependence on consumption of fish, wildfowl, nor game (Section 4.21, Table 64). It also seems logical to look for an external source of arsenic for the individuals in Oujé-Bougoumou that have hair arsenic concentrations above the level of concern of 4.0 nmol/g (see Table 51). One possibility is working with pressure-treated wood or being in contact with leachate from it. In Oujé-Bougoumou, treated wood was used in home construction and arsenic in soil near the home might well be a source. Further, tap water as a chronic low-level contact source is unlikely as the arsenic concentration in a 2002 survey was below the detection limit of 1.0 µg/L (E Robinson, personal communication).

5.2.3 Copper

Concentrations of copper in urine and hair were significantly increased in the Oujé-Bougoumou females of reproductive age relative to the same subgroups in Nemaska and Southern Quebec. Although this trend was not evident for plasma, there were 11 females in Oujé-Bougoumou compared to 4 in Nemaska with plasma copper concentrations that exceeded the level of concern of 25.2 µmol/L (see Table 63). As indicated in Section 4.15 and Table 39 (footnote 1), this level of concern does not apply to pregnant women near term or women taking oral contraceptives. Indeed for 13 of the 15 women, this limitation applied, and thus affords a logical interpretation. The contribution of an environmental source of copper therefore needs not be hypothesized.

In the absence of an association between copper in hair and that in plasma or urine, and the observation of a link (be it weak) between this metal in plasma and urine (see Section 4.16), suggest that copper in the latter media constitutes a more reliable index to its body burden. As explained in the copper toxicological profile (Appendix 1), copper homeostasis (i.e., metabolic control) explains why indices of copper status such as plasma copper, ceruloplasmin and erythrocyte superoxide dismutase (SOD) activity are relatively insensitive to changes in copper intake. Rather extensive exposures to copper are required to disturb this inherent copper balance. However, pregnancy, use of oral contraceptives, estrogen therapy such as in menopausal women, infections or inflammatory conditions are known to increase plasma copper, while corticosteroids and adrenocorticotropic hormone (ACTH) have the opposite effect (Burtis and Ashwood, 1994; Milne, 1998).

As suggested in Table 63, individuals who do not exhibit the conditions known to stimulate plasma copper levels might seek a confirmation of the enhanced copper status by more reliable tests the copper in plasma and urine such as plasma ceruloplasmin or superoxide dismutase (SOD) activity in erythrocytes. Persistent elevation of circulating copper levels may well reflect underlying diseases (e.g., dilated cardiomyopathy, or interruption of bile flow) (Milne, 1998; Beshgetoor and Hambidge, 1998), or induce tissue damage especially of the liver (IOM, 2002). Of course, the possibility of an unusual exposure should not be excluded *a priori*. This is especially relevant to external contamination of hair which can occur in certain non-occupational or occupational settings. For example, copper ions (Cu^{2+}) can be sequestered from water by hair and bind externally to it (Goldsack and Nieboer, 1975). It is indeed interesting that copper drinking-water pipes are more prevalent in Oujé-Bougoumou than Nemaska (Table 7). Tap water might thus be considered as a source, as well as other water sources coming into contact with hair.

It may be concluded that the copper status of the participants in both Cree communities as a group appears to be normal. The lower end of the normal range is 11.0 μmol (adult men) and 12.7 (non-pregnant women) (Burtis and Ashwood, 1994, 1996) and few individuals are below these concentrations (see Table 38). This is important since copper is an essential metal required for many important biochemical reactions (see copper toxicological profile; Appendix 1). Copper deficiency is a rare occurrence.

5.2.4 Selenium

As for urinary arsenic, selenium in plasma was higher in Nemaska than in Oujé-Bougoumou in four of the age/gender groups (Section 4.15 and Table 40). However, there was a trend towards lower values in the Cree communities relative to the Southern Quebec group. An additional environmental source for Oujé-Bougoumou is therefore unlikely. Plasma selenium as an index to selenium status is better established than selenium in hair. Hence it is not so obvious how to interpret the higher hair concentrations in Oujé-Bougoumou relative to Nemaska (Table 61). Some dependence of hair selenium content on age has been recognized and external contamination is not well-documented (Sky-Peck, 1990).

Accumulation of selenium in plasma depends on the form in which this element is present in the diet. It is known that selenomethionine, an amino acid, is incorporated randomly in protein, while other forms (e.g., selenate, and selenocysteine) are believed to be incorporated into the body's functional pool which appears to be under homeostatic (metabolic) control (Thomson, 1998). Fish selenium is known to increase plasma levels and the form of this element in fish appears to vary in different species. It is suspected that selenomethionine is the chemical form most common in wheat and cereals, although this requires further verification (Thomson, 1998). Thus the higher plasma selenium concentrations observed in Nemaska might well be related to the different fish-species consumption patterns. It is interesting that only in Oujé-Bougoumou was plasma selenium linked to fish consumption (Section 4.21 and Table 64). Differences in meat consumption, such as caribou in Nemaska (Table 20) might also be relevant. As pointed out in Section 4.7, lake sturgeon, northern pike, lake whitefish and white sucker were preferred in Nemaska, compared to walleye, brook trout and lake trout in Oujé-Bougoumou. If indeed the Oujé-Bougoumou fish or other foods contain more selenomethionine for example, this would afford a credible (but unproven) hypothesis for the higher hair concentration found in the female 15-39 subgroup providing this amino acid showed some preference for incorporation in hair protein. The weak relationship observed between selenium in hair and whole blood, but not with plasma (Section 4.19), is consistent with the experimental evidence that selenomethionine incorporates into haemoglobin and likely accounts for the fact that whole-blood selenium is about 30% higher than plasma levels (Wang et al 1995a; Thomson, 1998).

Since there is only one exceedance of the plasma level of concern and none of the action level, follow-up is not required. It is also known that pregnancy tends to repress plasma selenium (Burtis and Ashwood, 1994; Navarro et al., 1996) and this is consistent with the relative patterns observed in the Cree communities. The lower-end of the normal range is 0.7 μmol/L (Odland et al., 1999). Since selenium is present in proteins, hair shampoo/rinsing preparations may well be an external source affecting hair levels, providing there is a difference in use or product type between the communities compared. Shampooing and hair treatments are known to affect hair levels of the elements (Sky-Peck, 1990; Wilhelm and Idel, 1996). Further, tap water as a low-level contact source is unlikely as the

selenium concentration in a 2002 survey was below the detection limit of 1.0 µg/L (E. Robinson, personal communication).

It seems appropriate to conclude that the selenium status in both communities is adequate. Differences such as seen between the three communities compared have been observed before and are usually related to different availabilities of dietary forms of this element (Wang et al., 1995b). Proper dietary selenium intake is essential as deficiencies have been linked to increase risk of cardiovascular disease and cancer (Aro et al., 1995; Wang et al., 1995b), although this area of study is controversial (Thomson, 1998). Selenium is an essential element and a proper balance is required for good health (see selenium toxicological profile, Appendix 1).

5.2.5 Zinc

The community distribution pattern observed for zinc in plasma is the opposite from selenium, with a trend to higher concentrations in Oujé-Bougoumou and the Southern Quebec group when compared to Nemaska. Nevertheless, all community means reflect the existence of normal zinc status (Wang et al., 1995b; Burtis and Ashwood, 1994), with very few exceedances. The hair zinc levels corroborate this finding, although no association was found between it and plasma concentrations (Section 4.19). The lower end of the normal reference interval in plasma is around 10 µmol/L, but mean levels below this lower limit have been observed in groups of pregnant women (Odland et al., 1999). Proper zinc intake is important as it is an important essential metal that especially influences growth and development among many other basic functions (see zinc toxicological profile, Appendix 1). It is known that pregnant women are at risk of acquired zinc deficiency because of its requirement by the developing fetus, and this metal appears to be normally suppressed during pregnancy (Odland et al., 1999). Good dietary sources of zinc are red meat, liver, eggs and certain seafoods, whereas whole grain products contain the element in less available form (Appendix 1). It may well be that the greater consumption of certain food items in Oujé-Bougoumou, such as game liver (see Section 4.8), accounts for the slightly higher plasma and hair zinc levels there, although there were no associations with consumption of fish, wildfowl or game (Table 64).

In conclusion, as for arsenic, copper and selenium, there is no basis for invoking an environmental source such as mine tailings residues.

5.3 CONTAMINANTS ASSOCIATED WITH LIFE-STYLE FACTORS

5.3.1 Cadmium

A primary source of cadmium is smoking, but others are recognized including: industrial emissions (past and present) and associated soil and vegetables contamination; significant consumption of kidney or liver from marine mammals or game; aquatic macrophytes such as wild rice (for summary see cadmium toxicological profile in Appendix 1).

The influence of smoking on the observed whole-blood cadmium concentrations can be deduced from various lines of evidence. First, for smokers and non-smokers combined (≥15 years of age) there is a 2.5 to 3.0-fold higher concentration of cadmium in whole blood in the Cree communities compared to the Southern Quebec comparison group, which reflects the ratio of smoking prevalences (see Section

4.12 and Tables 14, 15, and 30). Second, for the three groups there is a 2-to 4-fold drop in whole blood cadmium when comparing the means to nonsmokers (c.f. Tables 30 and 31). Third, a strong association was observed between whole-blood cadmium and plasma cotinine, a known biomarker of nicotine exposure (Dewailly et al., 1994; Benowitz, 1999; Moyer, 2002). Fourth, urinary cadmium levels are less sensitive to current exposures such as in smokers and the intercommunity differences are not seen; nor that between the combined group and nonsmokers (c.f. Tables 46 and 47). The modest association observed between cadmium in urine and whole blood supports this (Section 4.16). Further, the weak negative association between cadmium in hair and whole blood (Section 4.17) suggests that the metal levels in hair do not derive from smoking.

As discussed in some detail in the cadmium toxicological profile (Appendix 1), cadmium is a potent renal toxicant. Environmental exposures, including smoking, can induce measurable subclinical renal dysfunction and increase the rate of bone fractures in women. For example, whole-blood cadmium concentrations of 25 nmol/L for 25 years increase abnormal renal function by 5% and 50 nmol/L is responsible for a 10% increase. Similarly, at urinary cadmium levels of 35.6-47.4 nmol/L there is a 10% probability of mild tubular dysfunction. Cadmium accumulates in the liver and kidney, and for smokers the concentrations in these organs have been shown to be elevated compared to non-smokers.

It is tempting to interpret the slight elevation of whole-blood cadmium for non-smokers in Oujé-Bougoumou relative to Nemaska and the Southern Quebec Groups ($\geq$ 15 years; Table 31). Higher levels of secondary smoke is one possibility. A slightly higher prevalence of the consumption of game liver and kidney affords another explanation (see Section 4.8). The positive association for the non-smoking Oujé-Bougoumou participants between whole-blood cadmium and consumption of game supports this notion (see Section 4.21; Table 64). The concurrent correlation with fish consumption likely reflects confounding because those who eat game regularly might be expected to do the same for fish.

It seems prudent to inform the Cree communities about the risk of cadmium exposure from cigarettes and that frequent consumption of game liver or kidneys might be a health risk for those with renal insufficiency and osteoporosis.

5.3.2 Lead

Of all the metals, lead is one of the most systemic toxicants. Its primary targets are the synthesis of haemoglobin, the kidney, the nervous system, reproduction, and development. Of these impairments of cognition, behavior or development in the unborn and young children has had the biggest impact in public health. Lead in whole blood constitutes the primary gold standard for linking exposure to health outcomes. Although 0.48 μmol/L (100 μg/L or 10 μg/dL) has been set as the level of concern (review) by international agencies, including Health Canada and the Quebec Public Health Department, there is renewed evidence that this may need to be lowered (Rogan and Ware, 2003; see the lead toxicological profile in Appendix 1 for details of this and all the issues mentioned above).

By comparison to other native communities (Dewailly et al., 1994; Van Oostdam, 1999; AMAP, 2003; Nieboer et al., 2002), the whole-blood lead levels observed are lower in the Cree communities of Oujé-Bougoumou and Nemaska. There is no clear trend in differences between these two communities: being higher in children in Oujé-Bougoumou and in the over 40 groups in Nemaska, and

with no difference for the remaining gender/age groups. This mixed trend also applies to the comparison with the Southern Quebec data: they were significantly higher for the 40 and over group in both Cree communities, but marginally lower for the 15 years and older participants in Nemaska. These patterns suggest that the 40 and older members of the Cree communities participate more in hunting and/or consume more wildfowl and game bagged with leaded ammunition. Indeed, whole-blood concentrations were associated with the consumption of wildfowl and game (see Section 4.21, Table 64). The similar dependence on fish intake observed is to be expected as individuals who consume wildfowl and game also include fish in their diet. Radiographic, analytical and isotopic tracing evidence has established that edible portions of game harvested with lead shot are contaminated with lead (Levesque et al., 1998; Nieboer et al., 2002; see lead toxicological profile Appendix 1). Inhalation of lead-containing aerosols can also occur during gun firing. Indeed, this older age group in both communities consumes considerably more game and wildfowl (especially in Nemaska) (Section 4.7 and Table 23), and leaded ammunition is used in both communities (Section 4.2; Table 9). Furthermore, hunting activities constituted a risk factor for blood lead concentrations in both study communities (Section 4.21; Tables 65 and 66). Even though drinking water from a spring (Oujé-Bougoumou) or lake and/or river (Nemaska) also constituted a risk factor for lead in blood, it is likely a confounder of being in the bush during hunting. Smoking also was a risk factor for higher whole-blood lead concentrations in both communities, and this association is well established (Lauwerys and Hoet, 2001). The observed reduction in the difference in blood-lead means for the two communities and the increase in the p value on adjustment for age, sex, consumption of fish, wildfowl and game, participation in hunting, smoking status and obtaining drinking water from a spring, lake or river suggest that these independent variables were indeed exposure risk factors.

Lead in hair was somewhat higher in Oujé-Bougoumou, although the mean levels were well below the suggested level of concern or means reported for non-occupationally exposed individuals (Sky-Peck, 1990; Wilhelm and Idel, 1996) (Section 4.17; Table 54). As reviewed in detail in Nieboer (2002), hair cut close to the scalp like the 0-2 cm segment in this study can reflect blood lead concentration. The weak correlation observed between lead in hair for the occipital segment and that in whole blood attests to this (Section 4.17). However , tiny particulates containing lead can be trapped in the interstial spaces of hair and lead ions (Pb^{2+}) can adhere (adsorb) strongly to the hair surface. Such external contributions to the hair lead concentration have been documented (Nieboer, 2002). Lead fumes or microparticles of lead generated during the use of firearms and lead leached from solder into tap water are examples. The fact that the highest mean lead-in-hair concentrations occur among the men aged 15-39 years and in the over 40 age group can in part be interpreted to reflect the consumption of lead contaminated meats, by analogy to blood lead. In addition, internal exposure (by inhalation) and external contamination (adsorption/incorporation of particulates) during the use of firearms likely contribute (Nieboer et al., 2002). For these various reasons, it is hard to separate the contributions to lead in hair from the peripheral blood and those that occur externally. Further, higher lead-in-hair concentrations in women of reproductive age in Oujé-Bougoumou may reflect adsorption of lead ions from tap water during shampooing, since copper piping (and thus lead solder) was more prevalent in the community compared to Nemaska (Section 4.2; Table 7). The relatively low blood lead concentrations confirm that this tap water as a source of lead is not high enough to result in health effects.

As mentioned above and discussed in detail in the toxicological profile of lead (Appendix 1), 0.48 µmol/L of lead in whole blood is the trigger for public health review. Such review is mostly advisory and is concerned with education about lead sources up to double this concentration. It is evident from the summaries in Tables 37 and 63 that only one child needed the advisory review, as well as 2 men in the 15-39 age group and 7 individuals in the over 40 subgroup. Only one female of reproductive age needed the more medical review.

5.4 CONTAMINANTS ASSOCIATED WITH FISH CONSUMPTION

5.4.1 Mercury

As a metal, mercury is somewhat unique in that it can form compounds with a mercury-carbon bond referred to as organometallics that are very stable in aqueous solution. Methyl mercury is the prime example. It arises by two pathways: microbial and chemical synthesis (AMAP, 1998). It is quite soluble in both aqueous and more organic media such as fats (lipids). Methyl mercury, as opposed to inorganic forms of mercury, is preferentially biomagnified in the aquatic food chain and hence accumulates especially in fish-eating (piscivorous) species. [Biomagnification refers to increases in concentration with each trophic level.] Methylmecury is incorporated (secreted) into human hair and is the primary form of mercury present in fish tissue and human blood in individuals not inadvertently exposed to inorganic forms of mercury. Mercury in hair or whole blood is a good indice of exposure to methyl mercury.

Inorganic forms of mercury can be considered to be systemic toxicants. Methyl mercury primarily attacks the nervous system, and low-level dietary exposures have been associated with developmental delays, mild neurological impairments and increases in blood pressure in infants exposed through breast milk, as well as developmental and cognitive deficits due to prenatal exposure. Cardiovascular risk factors in a Finnish study of men have also been associated with concentrations of mercury in hair with fish consumption as the suspected source of methyl mercury. Further, progressive nervous system effects have been linked to consumption of highly mercury-contaminated fish. Additional details are provided in the mercury toxicological profile (Appendix 1).

Exposure to mercury in the two Cree communities may be termed mild to moderate, with means comparable to those reported for Canadian Inuit populations (Dewailly, 1994; Van Oostdam, 1999; AMAP, 2003). Although there are some exceedances of the levels of concern of mercury in whole blood and hair (0-2 cm), only 3 individuals exceeded the whole-blood action level and none for hair. Individuals exceeding the level of concern are advised to voluntarily review their consumption (amount and type) of fish, wildfowl and game, especially in the context of women planning a pregnancy. Exceedance of the action level should involve a health worker in this review process. Both hair and whole-blood concentrations of mercury are strongly associated with the consumption of fish, wildfowl and game (Section 4.21; Table 64). For fish, the consumption of all tissues are relevant to mercury intake, while liver and kidney consumption are likely of greater importance for wildfowl and game. Since community differences of mercury in whole blood and hair were rather insensitive to adjustments for consumer age, gender and consumption of fish, wildfowl and game, this suggests that the exact species, type of tissues consumed and the extent of lake-specific contamination are important. Indeed the preferential consumption of predator (piscivorous) fish such as lake trout and walleye in Oujé-Bougoumou, compared to insectivorous species such as lake whitefish, white sucker,

and sturgeon in Nemaska, is consistent with the trend in higher exposure indices in Oujé-Bougoumou (Section 4.7). The Quebec Ministry of Environment Report (Laliberté and Tremblay, 2002) suggests that the following relative accumulation pattern for Lakes Chibougaumau and Dorés are: lake trout >> walleye, northern pike, burbot >> lake whitefish. The more popular practice of consuming liver and kidney from game animals in this community (Section 4.8) is also supportive of this interpretation.

5.4.2 Polychlorinated Biphenyls (PCBs)

Production of PCBs has been banned in North America and ceased in 1977, although they are still present in older electrical transformers that have not been replaced in recent times. As outlined in the toxicological profile (Appendix 1), there were many commercial uses of PCBs. They are environmentally persistent, chemically stable and fat soluble. Like methyl mercury, they biomagnify in the aquatic food chain and accumulate especially in fish eating (piscivorous) species such as: walleye, burbot, northern pike and lake trout; birds of prey; auks, loons, gulls, and mergansers; seals, walrus, and whales; mink, marten, and polar bears; and humans (AMAP, 1998). They also accumulate, but to a much lesser extent, in the lichen → caribou → wolf food chain, residing in the fatty tissues including the liver of the animals.

As explained in the toxicological profile (Appendix 1), the health impact of chronic low-level exposures is difficult to assess. Neurological and developmental impairments due to prenatal and perinatal exposure through breast feeding are suspected in children, as well as hormone disruption in children and adults, and subclinical liver function perturbations.

Exposure to PCBs in the two Cree communities might be designated as moderate to high. The geometric means of the age/gender groups over 15 years were in the range of 1.6 to 37.2 µg/L; with minima falling between < DL to 2.9 and maxima 20 to 222 µg/L (as Aroclor 1260). This compares to what has been found in a Quebec Inuit population for which the geometric mean was 16 µg/L (entire population) and 50 µg/L in the over 40 group (Dewailly et al., 1994). It is also enlightening to compare the geometric means for women of reproductive age in Oujé-Bougoumou of 2.8 µg/L and a concentration range of 0.3-26.6 µg/L and the corresponding Nemaska values (1.6 µg/L and 0.3-20.2 µg/L) with maternal plasma Aroclor 1260 concentrations reported for Inuit mothers from 1994 to 2000 of 2.4-8.0 µg/L (range of geometric means for five communities), 0.10-2.0 µg/L (range of minima) and 8-60 µg/L (range of maxima) (AMAP, 2003). Similarly, men from five communities in Greenland, who are believed to be at very high risk of exposure, had geometric means ranging between 18-107 µg/L (AMAP, 2003), which are of comparable magnitude to the over-40 age group geometric mean concentrations in the present study of 37.1 µg/L (Oujé-Bougoumou) and 22.8 µg/L (Nemaska). In the Greenland groups maxima ranged from 71 to 331 µg/L, compared to 222 µg/L (Oujé-Bougoumou) and 126 µg/L (Nemaska).

Plasma PCBs (as Aroclor 1260) were strongly dependent on consumption of fish and game in both communities, but on wildfowl only in Nemaska. This observation is consistent with the discussion of accumulation patterns of PCBs in fish, fish-eating birds and mammals presented above (also see Section 4.21 and Table 64). Since the significance of the difference in mean concentrations in the communities increased somewhat on adjusting for age, sex and consumption of fish, wildfowl and game (see Section 4.21 and Table 67), other factors must be at play. As for methyl mercury, consumption of the exact species and the type of tissue appears relevant. The food frequency

questionnaire information indeed indicates that piscivorous fish species were preferred by Oujé-Bougoumou participants and more insectivorous fish in Nemaska. Because of the known bioaccumulation/biomagnification of PCBs, this is a very important factor that affords one plausible explanation for the intercommunity difference. Of course, an important issue is the extent to which the fish are contaminated. According to the Quebec Ministry of Environment report (Laliberté and Tremblay, 2001), relative to Waconichi Lake and Lake Obatogamau, PCBs concentrations in all fish species were considerably higher for Lakes Dorés and Chibougamau. It is not known where the fish caught in Lake Champion near Nemaska fit into the relative contamination pattern. We understand that results for this are pending.

According to the Ministry of Environment report, the concentrations of PCBs were in the following order: lake trout >>> walleye, lake whitefish, burbot and northern pike. The position of the insectivorous lake whitefish is unusual in this grouping as PCBs are generally present in relatively low concentrations (AMAP, 1998). Perhaps this affords a clue as to the source of the PCBs. However, the absence of PCB residues in the sediments of Lakes Chibougamau and Dorés (Laliberté and Tremblay, 2002) is inconsistent with the fish accumulation pattern and requires follow up.

As for methyl mercury, there was a significant number of exceedances of the concern levels (Section 4.20 and Tables 28 and 63). In such instances, the individuals are advised to voluntarily review their consumption (amount and type) of fish, wildfowl and game, especially in the context of women planning a pregnancy. Exceedance of the action level should involve a health worker in this review process. Liver enzymes have already been assessed in the 11 individuals who were above the plasma PCBs 100 μg/L action level (Tables 28 and 63). No one was outside the appropriate male or female reference interval. There were no exceedances of the plasma thyroid stimulating hormone (TSH) and thyroxine (T4) among the same individuals. Additional clinical chemistry measurements might be pursued such as selected hormones (e.g., plasma estrogen, testosterone, leuteinizing hormone (LH), growth hormones, adrenocroticotrophic hormone, prolactin), and perhaps a more detailed general health assessment.

The distribution patterns and intercommunity differences of the most prevalent PCB congener 153 (Section 4.11; Tables 29 and A1; Figure 2) follow that discussed for Aroclor 1260, including the concentration dependence on fish and game consumption. Qualitatively speaking, PCB 153 is representative of the other congeners measured (see Figure 2; Tables A1 to A7 in Appendix 7). The concentrations found for the pesticides hexachlorobenzene and transnonachlor (Tables A8 to A10) appear somewhat lower than those reported for other native communities, including Cree and Inuit populations (Dewailly et al., 1994; AMAP 2003; Nieboer et al 2001). By contrast, p,p-DDE levels tend to be comparable or higher than in other communities, suggesting the possibility of a local source, especially in the past since this metabolite of p,p-DDT is most prominent for the 40 years and older group and is two-fold higher in Oujé-Bougoumou in this age group than in Nemaska ($p=0.051$). The p,p-DDE/ p,p'-DDT ratio was 57 ± 33 and the p,p'-DDE concentration was dependent on the consumption of fish and game ($p<0.001$) and less so in wildfowl ($p=0.061$).

5.5 SELF-REPORTED HEALTH STATUS AND CLINICAL CHEMISTRY OUTCOMES

5.5.1 Self-reported health problems

As pointed out in Section 4.4, of the self-reported health outcomes, diabetes differed between Oujé-Bougoumou and Nemaska. The reported prevalences were 10.6% in Oujé-Bougoumou and 3.4% in Nemaska (p=0.032, Table 12). For A1c-glycated haemoglobin (HbA1c), the prevalence of individuals with a proportion over 0.065 was also higher in Oujé-Bougoumou; 14.5% compared to 5.3% in Nemaska (p=0.054, Table 13). HbA1c is a measure of the average concentrations of blood glucose over the past 2 or 3 months. This clinical measurement therefore confirms the self-reported diabetes results. By contrast, the plasma glucose levels were not different between the communities. This is not surprising as the glucose measurement requires overnight fasting, a condition which could not be met; HbA1c does not have this requirement. By comparison, the CBH 2001 diabetes registry (Elizabeth Robinson, private communication) indicated that the prevalences in the study populations were 12.8% (Oujé-Bougoumou) and 10.2% in Nemaska. Even though the weightings of individuals in each age/gender participant group were quite similar for the two communities, and thus representative of their respective populations, the smaller Nemaska sample size may have influenced the results. Nevertheless, the sociodemographic breakdowns of the participants were similar (Tables 6 and 7).

5.5.2 Clinical chemistry measurements

5.5.2.1 Iron, vitamins and hormones

Iron is essential for the formation of haemoglobin and many intracellular heme-containing enzymes. Reduced body iron occurs for various reasons, including inadequate dietary iron, excessive blood loss, or both. Ferritin is the principal iron storage protein and serum ferritin levels provide the best single indicator of iron stores. Transferrin saturation is measured as the ratio of serum iron and total iron-binding capacity. This protein transports iron to the tissues. More severe and long-standing iron deficiency results in reduced haemoglobin level and anemia. A vitamin B12 deficiency can also be responsible for lowered haemoglobin levels. Inadequate folate intake during pregnancy may cause megaloblastic anemia and neural tube defects in infants. The effects of thyroid hormones such as the thyroid stimulating hormone (TSH) and the thyroxine (T4) on the body are complex. They influence growth, development, protein synthesis, energy metabolism, processes of genetic control, etc.

As pointed out in Section 4.5, most iron-status measures were not different between the two communities, especially those considered the most reliable (i.e., serum ferritin; Table 13). The isolated occurrences of more individuals below the suggested reference intervals in Oujé-Bougoumou for erythrocyte count and iron saturation in Nemaska are therefore inconsistent. It is difficult from the data in Table 13 to make a judgment of the prevalence of anemia. Diagnosis of this disease in individuals is a complex matter that takes into account many factors, including gender-specific reference ranges employed for the various red-blood-cell related indices (Cotran et al., 1999; Adamson, 2001; Adamson and Longo, 2001). Nevertheless, it appears that the iron status is adequate.

Folate, vitamin B12, TSH and free T4 levels were essentially in their normal ranges and not different between the two communities.

5.5.2.2 Cardiovascular risk factors

Cardiovascular disease (CVD) is the leading cause of death and hospitalization in both Quebec and Canada. Research in cardiovascular health has shown that the use of tobacco, high blood pressure, obesity, abnormal blood lipid concentrations and diabetes are major risk factors. The relatively high prevalence of smoking in the two study communities (Section 4.6 and Tables 15 and 16) not only constitutes a cardiovascular risk factor but also a factor of respiratory cancers, other respiratory diseases, pregnancy complications and poor prenatal development (Burns, 2001). Detrimental interactions of smoking with prescription drugs are also known. Anti-smoking intervention initiatives are likely to yield significant health benefits.

Overweight and obesity lead to adverse metabolic effects on blood pressure, cholesterol, triglycerides and insulin resistance. Risks of ischemic heart disease (IHD) and type 2 diabetes increase steadily with increasing BMI. The body mass index (BMI) is the most useful indicator of health risk associated with obesity. Similarly, waist girth is an indicator of abdominal obesity which is associated with several risk factors that influence the development of diabetes and coronary heart disease. It is evident from the results summarized in Section 4.6 and Tables 14 and 15 that, according to the BMI and waist girth measures employed, obesity constitutes a health issue in both communities. Relative to the Southern Quebec comparator community, high blood pressure appears to be more prevalent as well (about 20-23% in the study communities compared to 14%). Elevated blood pressure produces a variety of structural changes in the arteries that supply blood to the brain, heart, kidneys and elsewhere. High blood pressure increases the risks of stroke, IHD, renal failure and other diseases.

High values of total cholesterol low-density lipoprotein (LDL) cholesterol or triglycerides, and low values of high-density lipoprotein (HDL) cholesterol are associated with increased risk of CVD. Cholesterol is a key component in the development of atherosclerosis, the accumulation of fatty deposits on the inner lining of arteries. Mainly as a result of this, cholesterol increases the risks of IHD, ischaemic stroke and other vascular diseases. Also apolipoprotein B is the main protein of LDL and its concentration in plasma is a strong predictor of IHD. From these perspectives, in native communities, the cardiovascular risk appears to be somewhat less than in the Quebec reference group. Specifically, the proportion with good HDL cholesterol concentrations was comparable (Oujé-Bougoumou) or higher (Nemaska); and more individuals in the Cree communities had lower levels of total cholesterol and LDL cholesterol. By contrast, the distributions of low and high concentrations of triglycerides were comparable in the Cree and Southern Quebec groups. These outcomes are interpreted in terms of differences in fish consumption as discussed below.

5.5.2.3 Fatty acids status

Considerable evidence exists on diets rich in fish and marine mammals and their effect protective on cardiovascular disease (CVD) (Whitney et al., 1998; Dewailly et al., 2002). Epidemiological evidence has indicated that an unbalanced intake of the various lipids can lead to disease (Whitney et al., 1998). Saturated fatty acids have no double bonds, are derived primarily from animal fats, and constitute a risk for CVD by raising LDL cholesterol levels. LDL transports triglycerides and cholesterol to cells and appears to be involved in the development of atherosclerosis. By contrast HDL is a scavenger of cholesterol and is protective of CVD. Polyunsaturated fatty acids (PUFAs) have more than one double bond and tend to lower LDL when of the omega-3 (or n-3) type but not in the omega-6 (n-6) form. [The n-3 and n-6 are notations to identify the position of the double bond in the fatty acid.] Omega-6

PUFAs occur primarily in vegetable and plant oils and the omega-3 types in fish and marine oils. Monounsaturated fatty acids only have a single double bond and also appear to raise the HDL concentration. They occur in most fatty food sources. Omega-6 fatty acids, like the omega-3 type, are required by the body, but nutritionists suggest that a proper balance be maintained for good health. A value of the n-3/n-6 ratio of 0.25 has been suggested as optimum. In foods, fatty acids are primarily combined with glycerol to give triglycerides. In the body, triglycerides are the form in which fatty acids are transported between organs and tissues.

Consumption of fish and piscivorous birds in Oujé-Bougoumou was associated with higher concentrations of n-3 fatty acids and a slightly higher n-3/n-6 ratio, but not in Nemaska (Section 4,9, Table 24). Again, the species of fish is likely important here by analogy to mercury and PCBs accumulation. This is also consistent with the somewhat lower n-3/n-6 ratio observed in Nemaska. These finding are similar to an extensive and recently published study of n-3 fatty acids among James Bay Cree (Dewailly et al., 2002). In this study, like in the present survey, older individuals had higher values of the n-3 fatty acids and n-3/n-6 ratio than younger individuals (Section 4.9, Table 25), which reflects higher consumption of fish (Table 23). Dewailly et al. (2002) concluded that n-3 fatty acids appeared to favourably influence some CVD risk factors. A positive association was observed with HDL, and this is consistent with the present finding of improved HDL cholesterol levels in Oujé-Bougoumou relative to Nemaska, and between the Cree communities and the Southern Quebec reference group. Dewailly et al. (2002) also noted an inverse relationship of n-3 fatty acids with triglycerides, but only for coastal villages as opposed to inland communities. Thus differences between Oujé-Bougoumou and Nemaska would not be expected on this basis. However, the improved concentrations of the various cholesterol parameters (i.e., total, HDL and LDL) in the two study communities relative to the Southern Quebec group as noted above is most likely the result of higher fish consumption by the Cree. However, as pointed out in Section 4.6, there was no relative improvement in blood pressure in this comparison. Indeed, Dewailly et al. (2002) observed that n-3 fatty acids were positively associated with both systolic and diastolic blood pressure. Dewailly et al. (2002) conclude that "The Cree population must be encouraged to maintain their traditional fish-based diet, which may be one of the factors protecting them against mortality from CVD". Fish consumption appears to mitigate somewhat the CVD risks associated with obesity, cigarette smoking, and diabetes observed in the present and previous surveys (e.g., Santé Quebec, 1994).

6 CONCLUSIONS AND RECOMMENDATIONS

6.1 IMPACT OF MINE TAILINGS RESIDUES

Conclusion 1

Based on the observed concentrations of the signature elements arsenic, copper, selenium and zinc in body fluids, it is concluded that the residents of Oujé-Bougoumou are not at risk of internal (systemic) exposure.

The justification for this conclusion is based on the following factors discussed in Section 5.2: body fluids as opposed to hair concentrations are the gold biomonitoring standards; levels of arsenic and selenium were higher in Nemaska; the mean concentrations were not unusual; no dependence was observed on consumption of fish, wildfowl or game consumption, except for selenium on fish.

It is important to emphasize that Conclusion 1 does not imply that there is no impact of mining residues on the environment. They may well impact indirectly on the Oujé-Bougoumou community by affecting biota. This dimension and related issues need to be addressed in the ongoing environmental risk assessment.

Conclusion 2

Hair levels of the mine tailings signature elements were moderately elevated in Oujé-Bougoumou relative to Nemaska and a possible indirect source through chronic low-level contact might be explored in cases of arsenic and copper.

This conclusion is based on the fact that except for arsenic, the relative increases of the other signature elements of copper, selenium and zinc were statistically significant. By comparison, selenium and zinc hair levels were judged to be above average in the COVEL report (Covel and Masters, 2001); arsenic and copper were not. Although a number of explanations are possible for this conclusion as outlined in Section 5.2, tap water as a chronic low-level source of external exposure is not likely as arsenic and selenium concentrations were below the detection limit of 1 μg/L in a 2002 drinking water survey (E.R. Robinson, personal communication). Nevertheless, some copper may be released into tap water from the piping if taps are not used (flushed) regularly. Leaching of arsenic and copper from pressure-treated wood into nearby soil is a possible source, especially for playing children. A potential external source of zinc does not readily come to mind, and is likely not needed to explain its relative elevation in hair in Oujé-Bougoumou. As discussed earlier, similar reasoning might be applied to selenium. Again, it needs to be emphasized that there is no evidence of enhanced intake (absorption) into the body of the four signature elements associated with mine tailings residues as stated in Conclusion 1.

6.2 EXPOSURE TO CADMIUM AND LEAD

Conclusion 3

Cigarette smoking is the major exposure source of cadmium in both Oujé-Bougoumou and Nemaska.

Smoking is mostly recognized to be linked to increased risk of CVD, respiratory cancers, other respiratory diseases, pregnancy complications and poor pre-natal development. These outcomes are primarily related to organic toxic compounds in cigarettes. Because cadmium has a very long half life in liver and kidney ($\geq$10 years), it accumulates with age and thereby can result in kidney damage and perhaps also enhance a related risk of bone fractures in females. Consequently as pointed out in Section 5.5.2.2, anti-smoking interventions are likely to yield significant health benefits.

Conclusion 4

Lead exposure is related to hunting activities and consumption of wildfowl and game in both Cree communities.

Even though the extent of lead exposure in the Oujé-Bougoumou and Nemaska communities is mild to moderate, a shift to non-leaded ammunition is encouraged. The reason for this is that new evidence is emerging that the level in blood at which medical concern begins for children and females of reproductive age needs to be lowered. There appears to be no safe threshold for this systemic toxicant.

6.3 EXPOSURE TO MERCURY, POLYCHLORINATED BIPHENYLS (PCBS) AND P,P'-DDE

Conclusion 5

Exposure to mercury and PCBs was higher in Oujé-Bougoumou than in Nemaska; higher consumption of piscivorous fish and birds affords one explanation.

Exposure to mercury in the two Cree communities may be deemed mild to moderate compared to other native communities (for details see Section 5.4.1). By contrast, PCBs exposure is designated as moderate to high. Fish consumption appears to be the primary source, although intake of wildfowl and game cannot be clearly identified as a separate source since consumption of all three traditional foods appears to be a common practice. As documented in the mercury toxicological profile (see Appendix 1), absorption of mercury vapour released from amalgam fillings is known to make a small contribution to the total whole blood and hair mercury concentrations. Such a contribution may be expected to be intensified for individuals who grind their teeth (bruxism).

It is recommended that consumption guidelines be reviewed, updated and their use by the Cree communities should continue to be encouraged. The following factors should be incorporated into a more formal consumption guideline program. Routine monitoring of local fish tissues and kidney, liver and fatty tissues of piscivorous fowl and of game should be initiated. Consumption guidelines should be based on the biomonitoring results obtained for fish caught in local lakes and rivers and for wildfowl and game bagged in the communities' hunting grounds. The importance of fish consumption in maintaining health (see Section 5.6.4) should also be factored in. Further, extensive sediment analysis for PCBs is encouraged to establish if there are unique sources of these contaminants in Lakes

Chibougaumau and Dorés related to mining and mineral beneficiation operations. Such follow-up work should be conducted as part of the ongoing environmental risk assessment. The recent conclusion that for all sediments total PCBs levels were below the detection limit of 0.4 µg/kg (Laliberté and Tremblay, 2002) seems incongruent with the concentrations observed in fish and humans.

Conclusion 6

The observed concentrations of the p,p'-DDE metabolite of the insecticide p,p'-DDT are judged to be relatively high in the over 40 age group, especially in Oujé-Bougoumou.

The observed concentrations were compared to those reported for other native communities in Canada and world-wide. Consumption of fish and game is suggested as the likely source. As for PCBs, it seems prudent to investigate a possible local source.

6.4 COMMUNITY HEALTH STATUS, LIFE-STYLE ISSUES AND DIETARY ISSUES

Conclusion 7

In both communities, the status of the essential elements copper, selenium and zinc are judged to be normal and adequate for sustaining proper health. The iron status also appears adequate.

This conclusion is based on comparing the observed plasma concentrations of copper, selenium and zinc with the lower limit of the accepted reference levels. For iron, ferritin concentrations were most relevant, using 10 µg/L (women) and 15 µg/L (men) as the lower limit of the pertinent reference intervals (Adamson, 2001).

Conclusion 8

No new information about health status were identified in the two study communities.

It is clear from the Discussion (see Section 5.5) that diabetes and cardiovascular issues continue to be of paramount concern, as well as the associated complications and risks of smoking and obesity. Fish consumption appears to improve the CVD risk factors and thus it is crucial to encourage the maintenance of the traditional fish-based diet. As already indicated, any fish consumption and wildfowl/game guidelines need to take this factor into account.

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TABLES

Table 1 Contaminants of concern measured

Contaminant[1]	Primary Toxicological/Health Outcome	Subjects			Body Fluid/Tissue			
		Adult Female	Adult Male	Children 8-14	Whole Blood	Plasma/ Serum	Urine	Hair
Arsenic (As)	Skin effects (including cancer); neurological; cardiovascular	Yes	Yes	Yes	No	No	Yes	Yes
Cadmium (Cd)	Kidney disturbance	Yes	Yes	Yes	Yes	No	Yes	Yes
Copper (Cu)	Deficiency symptoms; gastrointestinal disturbance is the major toxic outcome	Yes	Yes	Yes	No	Yes	Yes	Yes
Lead (Pb)	Neurological; growth and development	Yes	Yes	Yes	Yes[2]	No	No	Yes
Mercury (Hg)	Neurological	Yes	Yes	Yes	Yes	No	No	Yes[2]
PCBs[3]	Endocrine disturbances	Yes	Yes	Yes	No	Yes	No	No
Selenium (Se)	Deficiency symptoms; hair loss/nail defects/neurological (toxic outcomes)	Yes	Yes	Yes	No	Yes	No	Yes
Zinc (Zn)	Deficiency symptoms; gastrointestinal, anemia, HDL decrease (toxic outcomes)	Yes	Yes	Yes	No	Yes	No	Yes

[1] A detailed toxicological profile for each contaminant is provided in Appendix 1

[2] Tests were also done for the participants less than 7 years old

[3] PCBs, polychlorinated biphenyls

Table 2 Clinical chemistry (biochemistry) parameters measured in relation to general health

Parameter[1, 2]	Related Health Outcome[c]	Subjects			Project Specific Reference[3]
		Adult Female	Adult Male	Children 0-14	
Plasma Vitamin B12	Prevention of anaemia	Yes	No	No	–
Plasma Folic acid	Multiple deficiency effects	Yes	No	No	–
Plasma iron, iron binding capacity, ferritin, transferin	Anaemia; chronic disorders	Yes	No	No	Willows et al. (2000)
Haemoglobin and hematocrit	Anaemia	Yes	No	No	Willows et al. (2000)
Plasma glucose[4], HbA1c	Diabetes	Yes	Yes	No	–
Peripheral blood cell counts (white cells and red cells, platelets) and cell measures; peripheral blood film morphology	Chronic disorders	Yes	Yes	No	–
Urinalysis (dip-stick analysis for glucose, blood, leukocytes, ketones, pH, protein); urinary creatinine	Kidney function and contaminants concentration normalization	Yes	Yes	No	–
Plasma BUN and creatinine	Kidney function	Yes	Yes	No	–
Bilirubin	Liver function	Yes	Yes	No	–
Plasma cholesterol , lipid profile (LDL, HDL, triglycerides)[4]	Cardiovascular and liver diseases	Yes	Yes	No	Dewailly et al. (2002)
Plasma omega-3 fatty acids	Cardiovascular and fish consumption	Yes	Yes	No	Dewailly et al. (2002)
Plasma TSH and T4	Thyroid function	Yes	Yes	No	–
Plasma cotinine	Measure of smoking	Yes	Yes	No	–

[1] Abbreviations: HbA1c, glycated haemoglobin; pH, measure of acidity; BUN, blood urea nitrogen; LDL, low density lipoprotein; HDL, high density lipoprotein; TSH, thyroid-stimulating hormone; T4, thyroxine.

[2] To be measured for the participants 15 years old and over

[3] A general reference is Burtis and Ashwood (1996)

[4] The sample collection should be preceeded by a 12-hour fast

Table 3 Total population size and participants in the Cree communities of Oujé-Bougoumou and Nemaska (stratified by gender and age).

	Oujé-Bougoumou			Nemaska		
Group	**Population**[1]	**Invited**[2]	**Participants**	**Population**[3]	**Invited**[2]	**Participants**
0-14 years	238	143	56	206	82	29
Women 15-39 years	150	159	78	136	103	43
Men 15-39 years	131	94	40	137	61	15
Men & women ≥40 years	103	81	51	137	38	13
Total	622	477	225	616	284	100

[1] From October 2001 population list

[2] Randomly selected using the Cree Beneficiary List for each community

[3] From October 2002 population list

Table 4 Recruitment and participation details for the communities of Oujé-Bougoumou and Nemaska (stratified by gender and age).

	Oujé-Bougoumou					Nemaska				
Group	**Contacted**	**Moved**	**Out of the village**	**Refused**	**Participant**	**Contacted**	**Moved**	**Out of the village**	**Refused**	**Participants**
	n	**n**	**n**	**n (%)**	**n (%)**	**n**	**n**	**n**	**n (%)**	**n (%)**
0-14 years	143	50	5	32 (34.4)	56 (60.2)	82	10	7	36 (50.0)	29 (40.3)
Women 15-39 years	159	65	6	10 (10.6)	78 (83.0)	103	15	6	39 (44.3)	43 (48.9)
Men 15-39 years	94	24	8	22 (31.4)	40 (57.1)	61	14	10	22 (46.8)	15 (31.9)
Men & women ≥40 years	81	9	7	14 (19.4)	51 (70.8)	38	3	13	9 (25.7)	13 (37.1)
Total	477	148	26	78 (23.7)	225 (68.4)	284	42	36	106 (43.8)	100 (41.3)

Table 5 Sampling proportion (weighting) of the Oujé-Bougoumou and Nemaska participants (stratified by gender and age).

Group	Oujé-Bougoumou			Nemaska		
	Population	Sample	W[1] (Group weight)	Population	Sample	W (Group weight)
0-14 years	238	56	0.383	206	29	0.334
Women 15-39 years	150	78	0.241	136	43	0.221
Men 15-39 years	131	40	0.211	137	15	0.222
Men & women ≥40 years	103	51	0.166	137	13	0.222
Total	622	225	1.000	616	100	1.000

[1] W was calculated by dividing the number in each group by the total population

Table 6 Socio-demographic characteristics of the Oujé-Bougoumou and Nemaska participants.

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Sex			
Male	52.8	56.7	0.517
Female	47.2	43.3	
Living with someone as a couple			
No	29.8	26.0	0.594
Yes	70.2	74.0	
Language spoken at home			
Cree	96.7	99.5	0.133
English	63.0	74.7	0.040
French	11.7	4.7	0.048
Number of adults in household			
1-2	76.2	50.4	<0.001
3-4	18.9	40.3	
≥5	4.9	9.3	
Number of children in household			
0	21.8	15.8	0.029
1-2	32.0	48.9	
3-4	39.1	27.9	
≥5	7.1	7.4	
Highest level of formal education completed			
No schooling	39.3	27.7	0.064
Elementary	45.9	47.5	
Secondary and over	14.8	24.8	
Working status			
Student	20.7	22.7	0.259
Work	53.4	61.1	
Jobless	25.9	16.2	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

Table 7 Housing details for the Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Type of residence			
House	68.8	97.3	<0.001
Other[2]	31.2	2.7	
Live in the residence since			
<1 year	13.9	7.8	<0.001
1-4 years	20.3	31.8	
5-9 years	37.4	17.9	
≥10 years	28.4	42.5	
Age of residence			
<5 years	5.7	19.0	<0.001
5-9 years	42.9	16.5	
≥10 years	51.3	64.5	
Recent renovation			
Yes	0.7	14.5	<0.001
No	99.3	85.5	
Material of drinking-water piping			
PCV and plastic	0.3	26.9	<0.001
Copper	99.7	73.1	
Heating system			
Electric	0.3	96.9	<0.001
Hot Water	99.7	0.0	
Oil or firewood	0.0	3.2	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

[2] Other includes: apartment; senior citizen's (elder) apartment

Table 8 Sources of drinking water used by the Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Tap water[2]			
No	31.5	10.1	<0.001
Yes	68.5	89.9	
Bottled water[3]			
No	22.4	61.7	<0.001
Yes	77.6	38.3	
Water from a spring[3]			
No	82.1	86.4	0.330
Yes	17.9	13.6	
Water from a lake/river[3]			
No	31.9	17.3	0.006
Yes	68.1	82.8	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

[2] In the community only

[3] When in the community and in the bush

Table 9 Bush-related activities and use of firearms by Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Number of days in bush during previous year			
0	9.7	11.7	0.039
<2 weeks	15.6	9.4	
<1 month	29.4	44.0	
≥1month	45.4	35.0	
Hunting activity			
No	64.9	63.8	0.854
Yes	35.2	36.2	
- If yes, use of firearm			
No	6.3	1.4	0.257
Yes	93.8	98.6	
- If yes, kind of bullets used			
Lead bullets	73.5	51.6	0.029
Steel bullets	26.1	44.0	0.071
Lead shells	58.0	78.4	0.045
Steel shells	24.2	45.0	0.034
- After handling a firearm, hand washing before smoking			
Yes	11.2	10.9	0.331
Sometimes	28.1	42.9	
Never	60.7	46.2	
- After handling a firearm, hand washing before eating			
Yes	58.2	28.7	0.012
Sometimes	29.6	45.6	
Never	12.3	25.7	
- Ammunition stored inside tent or house			
No	8.0	0.0	0.081
Yes	92.1	100.0	
- If yes, stored in a sealed case/container			
No	22.5	32.6	0.258
Yes	77.5	67.4	
- Clothing and footwear stored inside tent			
No	24.6	7.7	0.034
Yes	75.4	92.3	
- If yes, stored in a sealed case/container			
No	28.1	90.4	<0.001
Yes	71.9	9.6	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

Table 10 Other activities with potential lead exposure to lead by the Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Boat repair/building (≥15 years)			
No	92.1	89.7	0.582
Yes	7.9	10.3	
Making of bullets/fishing sinkers (≥15 years)			
No	98.0	97.6	0.872
Yes	2.0	2.4	
Use of materials with lead (≥15 years)			
No	96.7	100.0	0.149
Yes	3.3	0.0	
Home renovation (≥15 years)			
No	86.5	91.3	0.338
Yes	13.5	8.7	
Outdoor activities (for children: 0-14 years of age)			
No	7.1	6.9	0.962
Yes	92.9	93.1	
Contact with pets, dirt, sand or rocks (for children: 0-14 years of age)			
No	7.7	10.7	0.605
Yes	92.3	83.3	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

Table 11 Level of personal anxiety related to environmental contamination reported by the Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=178)	Nemaska (n=71)	
Worried about:	%	%	p value[1]
Pollution			
Not at all	30.6	32.9	0.241
Somewhat/Fairly	36.3	40.4	
Very much	33.1	26.7	
Potential risk for health[2]			
Not at all	18.4	-	-
Somewhat/Fairly	40.3	-	
Very much	41.3	-	

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

[2] This question was not considered applicable to Nemaska residents

Table 12 Prevalence of breast-feeding and various self-reported health problems among the Oujé-Bougoumou and Nemaska participants

	Oujé-Bougoumou (n=225)	Nemaska (n=100)	
	%	%	p value[1]
Present breastfeeding			
No	100.0	96.9	0.120
Yes	0.0	3.1	
Health problems			
Anaemia	3.8	3.9	0.960
Cancer	-	-	-
Diabetes	10.6	3.4	0.032
High blood pressure	9.2	12.3	0.396
Heart disease	3.8	1.7	0.319
Hypercholesterolemia	4.2	8.3	0.126
Goitre or thyroid problem	5.4	4.0	0.597
Respiratory problems	10.0	4.6	0.105
Liver problems	1.3	0.0	0.258
Kidney problems	3.2	0.5	0.143
Osteoporosis	1.3	0.0	0.252
Allergy	14.0	20.0	0.169
Other	12.3	15.8	0.390

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

Table 13 Prevalence of selected clinical chemistry (biochemistry) outcomes among Oujé-Bougoumou and Nemaska participants (≥15 years of age)

		Oujé-Bougoumou (n=169)	Nemaska (n=71)	
Indicator[1]	**Reference interval**	**%**	**%**	**p value**[2]
Iron (P)	10-30 µmol/L			
<10 µmol/L		23.4	33.1	0.142
≥10 µmol/L		76.6	66.9	
Iron binding capacity (P)	45-80 µmol/L			
<80 µmol/L		93.6	91.6	0.603
≥80 µmol/L		6.4	8.4	
Iron saturation (P)	0.20-0.55%			
<0.2%		32.9	52.6	0.007
≥0.2%		67.1	47.4	
Transferrin (P)	2.00-3.60 g/L			
<2.0 g/L		1.6	0.0	0.303
≥2.0 g/L		98.4	100.0	
Ferritin (P)	20-300 µg/L			
<20 µg/L		18.5	26.3	0.198
≥20 µg/L		81.5	73.7	
Haemoglobin (WB)	M (140-180 g/L) W (120-160 g/L)			
<140(M) or 120(F) g/L		15.7	9.1	0.196
≥140(M) or 120(F) g/L		84.3	90.9	
Red blood cells (WB)	$(4.70\text{-}6.10)*10^{12}$/L			
$<4.70 * 10^{12}$/L		51.4	29.0	0.003
$\geq 4.70 * 10^{12}$/L		48.6	71.0	
Folate (P)	5.5-50.0 nmol/L			
<5.5 nmol/L		0.0	0.0	-
≥5.5 nmol/L		100.0	100.0	
Vitamin B12 (P)	110-675 pmol/L			
<110 pmol/L		1.6	0.0	0.307
≥110 pmol/L		98.4	100.0	
TSH (P)	0.27-4.2 mU/L			
<0.27 mU/L		1.0	0.8	0.859
≥0.27 mU/L		99.0	99.2	
Free-T4 (P)	10-22 pmol/L			
<10 pmol/L		2.0	6.8	0.089
≥10 pmol/L		98.0	93.2	
Alc-type Hb (P)	0.043-0.065			
<0.065		85.5	94.7	0.054
≥0.065		14.5	5.3	

[1] P, plasma; WB, whole blood

[2] p value corresponds to the chi-square test for the inter-community comparison of proportions

Table 14 Prevalence of cardiovascular disease risk factors among the Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Oujé-Bougoumou (n=169)	Nemaska (n=71)	
Indicator[1]	**%**	**%**	**p value**[2]
Smoking status			
Non-smoker	19.4	7.1	0.040
Ex-smoker	23.2	34.2	
Smoker	57.4	58.7	
- If smoker, number of cigarettes/day:			
≤10	83.0	64.9	0.023
11-25	13.1	29.5	
>25	3.9	5.6	
BMI category (kg/m^2)			
Normal weight (18.5-24.9)	12.9	7.4	0.535
Overweight (25-29.9)	26.8	21.7	
Obesity (30-39.9)	45.4	54.8	
Severe obesity (≥40)	15.0	16.2	
Waist girth (abdominal obesity)			
Normal	23.3	19.9	0.618
Elevated	76.8	80.1	
Blood pressure			
High[3]	23.1	20.2	0.607
Normal	76.9	79.8	
Total cholesterol (P)			
<5.2 mmol/L	74.8	73.3	0.599
5.2-6.1 mmol/L	18.7	23.0	
≥6.2 mmol/L	6.5	3.8	
LDL cholesterol (P)			
<4.1 mmol/L	96.7	99.2	0.287
≥4.1 mmol/L	3.3	0.8	
Apolipoprotein-B (P)			
<1.20 g/L	88.7	85.6	0.531
≥1.20 g/L	11.3	14.4	
HDL cholesterol (P)			
<0.9 mmol/L	7.1	0.0	0.026
≥0.9 mmol/L	92.9	100	
Triglycerides (P)			
<2.3 mmol/L	79.8	75.0	0.430
≥2.3 mmol/L	20.2	25.0	
Glucose (P)			
<6.1 mmol/L	64.2	51.6	0.084
≥6.1 mmol/L	35.8	48.4	

[1] P, plasma

[2] p value corresponds to the chi-square test for the inter-community comparison of proportions

[3] Diastolic ≥ 90 mmHg and systolic ≥ 140 mmHg

Table 15 Prevalence of cardiovascular disease risk factors among Quebecers (≥15 years of age)[1]

Indicator[2]	%
Smoker status	
Non-smokers + Ex-smokers	68.2
Smoker	31.8
BMI category (kg/m^2)	
<30	87.2
≥30	12.8
Waist girth (abdominal obesity)	
Normal	84.7
Elevated	15.3
Blood pressure	
High	13.8
Normal	86.2
Total cholesterol (P)	
≤6.1 mmol/L	79.9
≥6.2 mmol/L	19.1
LDL cholesterol (P)	
<4.1 mmol/L	83.3
≥4.1 mmol/L	16.7
HDL cholesterol (P)	
<0.9 mmol/L	7.7
≥0.9 mmol/L	92.3
Triglycerides (P)	
<2.3 mmol/L	83.2
≥2.3 mmol/L	16.8
Glucose (P)	
<6.1 mmol/L	92.1
≥6.1 mmol/L	7.9

[1] Source: Santé Quebec, 1994.
[2] P, plasma

Table 16 Mean weekly consumption of wild fish (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Oujé-Bougoumou		Nemaska		
	Mean	(sd)	Mean	(sd)	p value[1]
Walleye	17.5	(13.3)	8.0	(9.6)	0.012
Lake sturgeon	3.7	(4.0)	7.9	(6.4)	0.005
Northern Pike	3.0	(4.1)	6.3	(6.9)	0.033
Brook trout	5.9	(6.7)	3.3	(5.5)	0.174
Lake whitefish	2.5	(4.2)	5.0	(6.8)	0.102
Lake trout	4.5	(6.5)	2.8	(4.5)	0.343
White sucker	0.3	(0.7)	1.4	(3.1)	0.033
Other[2]	10.0	(2.0)	0.8	(2.6)	0.807
Total	38.3	(29.9)	35.6	(33.2)	0.767

[1] p value corresponds to the Student's t test for the inter-community comparison of means
[2] Includes red sucker and burbot, among other species

Table 17 Mean weekly intake of wild fish (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥15 years of age) who consumed fish

	Oujé-Bougoumou			Nemaska				
	% of consumers	Mean for consumer	(sd)	% of consumers	Mean for consumer	(sd)	p value[1]	p value[2]
Walleye	89.9	19.7	(13.7)	52.3	16.9	(12.8)	<0.001	0.620
Lake sturgeon	53.2	7.3	(5.0)	76.7	11.5	(7.1)	0.001	0.066
Northern Pike	33.1	10.5	(6.3)	63.0	12.2	(8.9)	<0.001	0.629
Brook trout	54.8	11.7	(8.5)	31.3	13.0	(9.7)	0.002	0.787
Lake whitefish	28.1	9.2	(7.0)	44.8	14.6	(10.3)	0.018	0.227
Lake trout	53.9	8.4	(8.4)	32.6	10.3	(7.8)	0.004	0.678
White sucker	6.1	5.7	(1.6)	17.6	10.1	(7.2)	0.009	0.394
Other	14.2	7.3	(4.5)	5.4	11.3	(9.3)	0.061	0.493
Total	93.1	41.8	(30.7)	94.6	39.5	(34.5)	0.695	0.822

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

[2] p value corresponds to the Student's t test for the inter-community comparison of means

Table 18 Mean weekly intake of wildfowl (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Oujé-Bougoumou		Nemaska		
	Mean	(sd)	Mean	(sd)	p value[1]
Goose	25.4	(14.7)	32.6	(12.8)	0.094
Partridge	22.8	(33.9)	5.0	(3.0)	0.040
Willow ptarmigan	5.9	(19.8)	6.0	(4.5)	0.992
Black scoter	1.9	(2.1)	1.0	(1.5)	0.161
Mallard	1.8	(2.0)	0.9	(1.0)	0.128
Golden eye duck	1.5	(1.8)	0.6	(1.6)	0.104
Northern pintail	0.8	(1.3)	0.9	(1.4)	0.926
American black duck	0.9	(2.0)	0.7	(1.0)	0.701
Other[2]	0.4	(0.9)	0.3	(0.6)	0.587
Total	61.5	(48.9)	48.3	(21.2)	0.306

[1] p value corresponds to the Student's t test for the inter-community comparison of means

[2] Includes merganser and loon

Table 19 Mean weekly intake of wildfowl (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥15 years of age) who consumed wildfowl

	Oujé-Bougoumou			Nemaska				
	% of consumers	Mean for consumers (sd)		% of consumers	Mean for consumers (sd)		p value[1]	p value[2]
Goose	98.0	26.0	(14.7)	100.0	32.6	(12.8)	0.240	0.127
Partridge	87.0	26.6	(36.2)	77.6	7.1	(3.1)	0.086	0.078
Willow ptarmigan	38.1	15.7	(31.3)	67.6	9.4	(5.1)	<0.001	0.539
Black scoter	32.1	6.4	(2.9)	22.6	6.2	(2.5)	0.159	0.919
Mallard	33.2	6.0	(2.8)	25.9	3.5	(1.3)	0.288	0.087
Golden eye duck	26.3	6.1	(2.6)	5.2	14.9	(4.7)	<0.001	0.013
Northern pintail	15.7	6.0	(2.4)	19.4	5.7	(2.8)	0.506	0.848
American black duck	14.9	7.7	(4.4)	19.7	4.7	(1.7)	0.386	0.330
Other	14.4	3.3	(2.0)	9.9	5.5	(1.0)	0.376	0.247
Total	98.5	62.6	(49.2)	100.0	48.3	(21.2)	0.312	0.270

[1] p value corresponds to the chi-square test for the inter-community comparison of proportions

[2] p value corresponds to the Student's t test for the inter-community comparison of means

Table 20 Mean weekly intake of game (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥ 15 years of age)

	Oujé-Bougoumou		Nemaska		
	Mean	(sd)	Mean	(sd)	p value[1]
Moose	24.4	(16.1)	21.6	(15.0)	0.552
Rabbit	18.7	(16.6)	6.5	(3.2)	0.005
American beaver	10.9	(7.4)	9.3	(5.9)	0.470
Caribou	1.5	(1.9)	13.7	(19.2)	<0.001
Bear	3.6	(2.9)	1.9	(1.9)	0.032
Other[2]	0.6	(0.9)	0.1	(0.2)	0.032
Total	59.7	(37.6)	53.2	(35.9)	0.564

[1] p value corresponds to the Student's t test

[2] Includes squirrel, lynx, marten, mink, weasel and muskrat

Table 21 Mean weekly intake of game (in grams, on an annual basis) by the Oujé-Bougoumou and Nemaska participants (≥15 years of age) who consumed game

	Oujé-Bougoumou			Nemaska				
	% of consumers	Mean for consumers (sd)		% of consumers	Mean for consumer (sd)		p value[1]	p value[2]
Moose	93.9	25.9	(16.3)	99.2	21.9	(15.0)	0.080	0.409
Rabbit	80.6	23.0	(17.8)	81.1	8.6	(3.1)	0.934	0.006
American beaver	78.7	14.2	(7.8)	90.1	11.0	(6.1)	0.045	0.200
Caribou	26.5	5.8	(2.8)	80.2	19.1	(22.1)	<0.001	0.065
Bear	53.0	7.3	(3.3)	44.0	6.0	(2.6)	0.231	0.419
Other[3]	15.4	4.6	(1.4)	10.4	1.7	(0.0)	0.331	0.042
Total	97.1	61.4	(37.9)	100	53.2	(35.9)	0.161	0.468

[1] p value corresponds to the chi-square test for the comparison of proportions

[2] p value obtained by Student's t test for comparison of mean

[3] Includes squirrel, lynx, marten, mink, weasel and muskrat

Table 22 Seasonal consumption frequency (%) of wild fish, wildfowl and game by the Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Last summer (2002)		Last spring (2002)		Last winter (2001/2002)		Last fall (2001)	
	Oujé-Bougoumou	Nemaska	Oujé-Bougoumou	Nemaska	Oujé-Bougoumou	Nemaska	Oujé-Bougoumou	Nemaska
	%	%	%	%	%	%	%	%
Wild fish:								
Walleye	83.1	50.8	44.6	20.3	35.7	6.0	46.9	18.7
Lake sturgeon	26.1	61.7	28.6	47.8	5.5	9.0	15.4	34.5
Northern Pike	23.4	58.4	18.5	38.7	8.7	17.2	15.4	35.1
Brook trout	49.7	28.3	11.1	13.5	7.5	0.8	27.7	12.9
Lake whitefish	14.2	33.2	12.8	17.2	7.8	10.4	13.7	40.2
Lake trout	42.7	18.8	11.9	19.1	4.6	6.0	26.8	15.3
White sucker	3.1	10.5	1.1	13.5	3.5	6.0	1.1	9.3
Other	6.5	3.1	3.1	2.2	3.6	2.2	4.2	2.2
Total	89.4	90.7	63.3	65.7	45.2	23.9	65.3	61.5
Wildfowl:								
Goose	70.8	97.0	98.0	100.0	61.1	68.8	59.5	79.3
Partridge	42.9	46.0	35.1	29.6	50.8	30.5	73.2	50.6
Willow Ptarmigan	6.2	10.5	3.5	12.9	33.5	62.0	11.5	11.0
Black scoter	2.4	3.0	31.1	22.6	1.4	0.0	1.9	0.0
Mallard	3.0	0.0	32.2	25.9	1.7	0.8	5.1	0.0
Goldeneye Duck	2.6	0.0	24.9	5.2	0.9	0.0	0.5	0.0
Northern pintail	-	-	14.8	19.4	1.4	0.0	0.5	0.0
American black Duck	0.5	0.0	14.4	19.7	-	-	0.5	0.0
Other	1.4	7.0	12.7	7.0	0.0	0.8	1.4	3.3
Total	81.3	97.8	98.5	100.0	77.1	80.8	86.7	84.8
Game:								
Moose	49.7	82.7	56.5	62.0	78.9	78.5	77.5	77.7
Rabbit	23.4	46.0	39.0	39.2	74.3	54.8	48.9	54.1
American Beaver	27.0	60.3	40.4	33.0	60.2	59.6	59.4	64.0
Caribou	3.2	28.0	6.7	31.7	21.2	60.0	3.8	59.4
Bear	22.1	36.2	23.1	21.1	15.4	18.1	40.6	18.9
Other	0.0	3.3	9.5	2.2	8.5	2.6	2.9	2.2
Total	65.0	91.0	75.8	75.2	90.5	93.9	87.3	93.1

Table 23 Mean weekly intake (in grams, on an annual basis) of wild fish, wildfowl and game by age among the Oujé-Bougoumou and Nemaska participants (≥15 years of age)[1]

	Oujé-Bougoumou			Nemaska				
	15-39 years	≥40 years	p value[2]	15-39 years	≥40 years	p value[2]	p value[3]	p value[4]
Wild fish	36.0 (23.0-49.0)	46.6 (35.6-57.5)	0.374	23.1 (9.3-37.0)	89.8 (23.7-155.9)	0.002	0.232	0.021
Wildfowl	60.9 (39.3-82.4)	64.0 (47.9-80.0)	0.872	43.3 (31.8-54.8)	70.4 (42.3-98.5)	0.049	0.281	0.683
Game	50.1 (37.8-62.4)	91.4 (59.7- 123.0)	0.005	43.7 (24.0-63.5)	95.2 (53.5-136.9)	0.026	0.572	0.897

[1] Arithmetic mean (95% CI)

[2] p value corresponds to the Student's t test for comparison between age groups

[3] p value corresponds to the Student's t test for comparison between Oujé-Bougoumou and Nemaska for the 15-39 years of age group

[4] p value corresponds to the Student's t test for comparison between Oujé-Bougoumou and Nemaska for the ≥40 years of age group

Table 24 Relative concentrations of fatty acids[1] in plasma phospholipids by consumption of wild fish and piscivorous birds among Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Oujé-Bougoumou				Nemaska				
Fatty acids	**Consumers**	**Non-consumers**	**Total**	**p value[7]**	**Consumers**	**Non-consumers**	**Total**	**p value[8]**	**p value[9]**
EPA[2]	0.64 (0.58-0.69)	0.48 (0.36-0.60)	0.63 (0.57-0.68)	0.094	0.51 (0.44-0.58)	0.51 (0.25-0.77)	0.51 (0.44-0.58)	0.990	0.012
DHA[2]	2.76 (2.63-2.89)	2.28 (1.99-2.58)	2.72 (2.60-2.84)	0.032	2.44 (2.28-2.60)	2.23 (1.81-2.66)	2.41 (2.27-2.56)	0.394	0.004
EPA +DHA	3.40 (3.24-3.56)	2.76 (2.50-3.02)	3.34 (3.20-3.49)	0.020	2.95 (2.74-3.16)	2.74 (2.15-3.34)	2.92 (2.73-3.11)	0.512	0.002
PUFA, n-3 series[2,3]	5.18 (4.93-5.43)	4.32 (3.99-4.65)	5.11 (4.88-5.34)	0.045	4.53 (4.26-4.79)	4.25 (3.35-5.15)	4.50 (4.25-4.74)	0.492	0.002
PUFA, n-6 series[2,4]	36.23 (35.97-36.50)	36.54 (35.92-37.15)	36.26 (36.0-36.51)	0.506	37.90 (37.46-38.33)	38.44 (37.11-39.77)	37.96 (37.55-38.37)	0.405	<0.001
Total PUFA	41.42 (41.15-41.68)	40.86 (40.25-41.46)	41.37 (41.12-41.62)	0.219	42.42 (42.07-42.77)	42.69 (41.98-43.41)	42.45 (42.14-42.77)	0.595	<0.001
n-3/n-6 ratio	0.14 (0.14-0.15)	0.12 (0.11-0.13)	0.14 (0.13-0.15)	0.054	0.12 (0.11-0.13)	0.11 (0.08-0.14)	0.12 (0.11-0.13)	0.481	0.003
MUFA[2,5]	15.06 (14.80-15.31)	15.16 (14.59-15.74)	15.07 (14.83-15.30)	0.810	14.49 (14.19-14.79)	14.52 (13.74-15.31)	14.49 (14.22-14.77)	0.933	0.005
SFA[2,6]	43.53 (43.29-43.77)	43.98 (43.16-44.80)	43.57 (43.34-43.80)	0.288	43.09 (42.77-43.41)	42.78 (42.47-43.10)	43.06 (42.77-43.34)	0.506	0.012

[1] Arithmetic mean (95% CI) of the percentage by weight of total fatty acids

[2] EPA, eicosapentanoic acid; DHA, docosahexanoic acid; PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids

[3] PUFA, n-3 series: (C18:3 + C18:4 + C20:3 + C20:4 + C20:5 + C22:5 + C22:6)

[4] PUFA, n-6 series: (C18:2 +C18:3 + C20:2 + C20:3 + C20:4 + C22:2 + C22:4 + C22:5)

[5] MUFA: (C14:1 + C16:1 + C18:1 + C20:1 + C22:1 + C24:1)

[6] SFA: (C14:0 + C16:0 + C17:0 + C18:0 + C20:0 + C22:0 + C24:0)

[7] p value corresponds to the Student's t test for differences by consumption of fish or piscivorous birds in Oujé-Bougoumou

[8] p value corresponds to the Student's t test for differences by consumption of fish or piscivorous birds in Nemaska

[9] p value corresponds to the Student's t test for differences between Oujé-Bougoumou and Nemaska

Table 25 Relative concentrations of fatty acids[1] in plasma phospholipids by age among the Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	Oujé-Bougoumou			Nemaska				
Fatty acids	**15-39 years**	**≥40 years**	**p value[7]**	**15-39 years**	**≥40 years**	**p value[7]**	**p value[8]**	**p value[9]**
EPA[2]	0.57 (0.51-0.62)	0.82 (0.70-0.94)	<0.001	0.45 (0.40-0.49)	0.79 (0.52-1.06)	<0.001	0.006	0.831
DHA[2]	2.53 (2.42-2.64)	3.33 (3.04-3.61)	<0.001	2.25 (2.12-2.39)	3.13 (2.75-3.51)	<0.001	0.003	0.469
EPA +DHA	3.10 (2.97-3.23)	4.14 (3.78-4.50)	<0.001	2.70 (2.54-2.86)	3.92 (3.37-4.48)	<0.001	<0.001	0.522
PUFA, n-3 series[2,3]	4.82 (4.56-5.08)	6.06 (5.64-6.47)	<0.001	4.21 (4.01-4.41)	5.76 (5.04-6.49)	<0.001	0.004	0.468
PUFA, n-6 series[2,4]	36.49 (36.22-36.77)	35.51 (35.01-36.01)	0.002	38.35 (37.96-38.74)	36.21 (35.23-37.19)	<0.001	<0.001	0.165
Total PUFA	41.31 (41.00-41.61)	41.57 (41.16-41.97)	0.384	42.56 (42.22-42.90)	41.98 (41.11-42.85)	0.154	<0.001	0.324
n-3/n-6 ratio	0.13 (0.13-0.14)	0.17 (0.16-0.19)	<0.001	0.11 (0.10-0.12)	0.16 (0.14-0.18)	<0.001	<0.001	0.365
MUFA[2,5]	15.27 (14.99-15.55)	14.40 (13.97-14.84)	0.002	14.51 (14.22-14.80)	14.42 (13.52-15.31)	0.800	<0.001	0.979
SFA[2,6]	43.42 (43.14-43.71)	44.03 (43.67-44.39)	0.026	42.93 (42.61-43.25)	43.61 (42.97-44.25)	0.068	0.034	0.236

[1] Arithmetic mean (95% CI) of the percentage by weight of total fatty acids

[2] EPA, eicosapentanoic acid; DHA, docosahexanoic acid; PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids

[3] PUFA, n-3 series: (C18:3 + C18:4 + C20:3 + C20:4 + C20:5 + C22:5 + C22:6)

[4] PUFA, n-6 series: (C18:2 +C18:3 + C20:2 + C20:3 + C20:4 + C22:2 +C22:4 + C22:5)

[5] MUFA: (C14:1 + C16:1 + C18:1 + C20:1 + C22:1 + C24:1)

[6] SFA: (C14:0 +C16:0 + C17:0 + C18:0 + C20:0 + C22:0 +C24:0)

[7] p value corresponds to the Student's t test for comparison between age groups

[8] p value corresponds to the Student's t test for comparison between Oujé-Bougoumou and Nemaska for the 15-39 age group

[9] p value corresponds to the Student's t test for comparison between Oujé-Bougoumou and Nemaska for ≥40 age group

Table 26 Plasma concentrations of total PCBs (measured as Aroclor 1260 in µg/kg) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% det.[1]	Mean (sd)		Geometric mean (95% CI)		p value[2]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	81.0	86.7	(69.9)	69.1	(51.9-91.9)	0.097	<DL	<DL	74.5	134.2	329.6
	Nemaska	11	72.7	260.8	(251.7)	144.0	(67.8-305.8)		<DL	<DL	189.6	628.1	636.7
Women	Oujé-Bougoumou	78	100.0	759.6	(841.8)	456.9	(362.1-576.5)	0.013	51.4	120.0	568.4	1 845.0	4 101.3
15-39 years	Nemaska	43	100.0	560.7	(746.3)	267.5	(184.9-387.1)		42.6	65.5	262.7	1 529.6	3 292.1
Men	Oujé-Bougoumou	40	100.0	1 566.3	(3 416.3)	689.2	(472.1-1 006.2)	0.066	54.1	126.8	701.0	2 720.4	21 653.1
15-39 years	Nemaska	15	93.3	792.9	(1 343.5)	335.6	(167.2-673.6)		<DL	70.3	512.4	1 729.8	5 383.0
Men & women	Oujé-Bougoumou	51	100.0	8 817.0	(7 683.4)	5 926.8	(4 525.1-7 762.5)	0.081	388.4	1 654.3	7 012.8	19 960.7	36 138.9
≥40 years	Nemaska	13	100.0	5 238.3	(5 852.4)	3 469.4	(2 105.0-5 718.3)		531.7	1 744.5	3 308.7	14 001.3	21 314.2
Total	Oujé-Bougoumou	190	97.9	2 348.0	(2 351.4)	573.3	(449.5-731.3)	0.037	<DL	57.8	585.7	7 012.8	36 138.9
(≥8 years)	Nemaska	82	95.1	1 240.9	(1 393.1)	362.4	(258.6-507.8)		<DL	50.9	290.0	3 292.1	21 314.2

[1] % of detection; detection limit (DL): 0.2 µg/L

[2] Comparison of the geometric means between Oujé-Bougoumou and Nemaska

Table 27 Plasma concentrations of total PCBs (measured as Aroclor 1260 in µg/L) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% of det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	81.0	0.43 (0.32)	0.35 (0.26-0.46)	0.065	<DL	<DL	0.3	0.7	1.5
	Nemaska	11	72.7	1.47 (1.35)	0.81 (0.38-1.75)		<DL	<DL	1.0	3.3	3.4
Women	Oujé-Bougoumou	78	100.0	4.99 (6.01)	2.76 (2.14-3.54)	0.022	0.3	0.6	3.0	11.6	26.6
15-39 years	Nemaska	43	100.0	3.70 (5.10)	1.62 (1.10-2.40)		0.3	0.3	1.4	8.6	20.2
Men	Oujé-Bougoumou	40	100.0	9.92 (21.47)	4.06 (2.72-6.06)	0.115	0.3	0.7	4.1	17.9	132.5
15-39 years	Nemaska	15	93.3	6.37 (12.28)	2.10 (0.97-4.55)		<DL	0.4	2.5	17.3	47.8
≥40 years	Oujé-Bougoumou	51	100.0	48.94 (56.68)	37.12 (28.15-48.94)	0.122	2.1	10.4	41.7	127.5	221.7
	Nemaska	13	100.0	34.62 (35.87)	22.84 (13.60-38.36)		2.9	11.8	24.7	96.5	125.6
Total	Oujé-Bougoumou	190	97.9	15.09 (15.43)	3.36 (2.60-4.33)	0.069	<DL	0.3	3.2	44.1	221.7
(≥8 years)	Nemaska	82	95.1	8.31 (8.96)	2.21 (1.55-3.15)		<DL	0.3	2.3	22.4	125.6

[1] % of detection; detection limit: 0.2 µg/L

[2] Comparison of the geometric means between Oujé-Bougoumou and Nemaska

Table 28 Exceedances of the concern and action levels of total PCBs (measured as Aroclor 1260 in µg/L) among the Oujé-Bougoumou and Nemaska participants

Group	Community	> Concern level[1]			> Action level[1] of 100 µg/L		
		Concern level	n (%)	p value[2]	n (%)		p value[2]
8-14 years	Oujé-Bougoumou	5 µg/L	0	-	0		-
	Nemaska		0		0		
Women 15-39 years	Oujé-Bougoumou	5 µg/L	23 (29.5)	0.527	0		-
	Nemaska		10 (23.3)		0		
Men 15-39 years	Oujé-Bougoumou	20 µg/L	3 (7.5)	1.000	1	(2.5)	1.000
	Nemaska		1 (6.7)		0	(0.0)	
Men & women ≥40 years	Oujé-Bougoumou	20 µg/L	37 (72.6)	0.503	9	(17.7)	0.672
	Nemaska		8 (61.5)		1	(7.7)	

[1] Source: AMAP 2002 (see Table 63, Appendix 6 and the PCBs toxicological profile in Appendix 1 for background and more details; based on occupational exposures likely with liver function changes as the primary outcome)

[2] p value corresponds to the Fisher exact test for comparison of proportions

Table 29 Plasma concentrations of PCB congener 153 (µg/kg lipids) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% det.[1]	Mean (sd)		Geometric mean (95% CI)		p value[2]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	95.2	11.5	(9.1)	9.3	(7.0-12.3)	0.035	2.6	4.4	9.5	17.9	43.7
	Nemaska	11	100.0	37.8	(36.4)	22.4	(11.4-44.3)		4.1	4.8	25.1	89.0	97.6
Women	Oujé-Bougoumou	78	100.0	101.0	(114.2)	59.9	(47.3-75.9)	0.019	6.2	15.4	72.9	242.8	577.6
15-39 years	Nemaska	43	100.0	76.5	(102.3)	35.9	(24.7-52.3)		5.2	8.3	35.8	217.9	455.2
Men	Oujé-Bougoumou	40	100.0	213.9	(449.6)	94.1	(64.0-138.4)	0.076	7.0	16.7	93.8	372.0	2 842.4
15-39 years	Nemaska	15	100.0	109.2	(180.2)	46.7	(23.3-93.4)		3.6	9.5	71.2	251.1	718.6
Men & women	Oujé-Bougoumou	51	100.0	1 197.0	(1 032.8)	806.5	(616.4-1 055.3)	0.094	52.6	228.8	951.4	2 683.4	4 687.1
≥40 years	Nemaska	13	100.0	736.4	(831.7)	482.4	(290.5-801.2)		71.8	240.2	474.4	2 002.6	3 010.2
Total	Oujé-Bougoumou	190	99.5	318.0	(317.0)	76.7	(60.0-98.0)	0.060	2.6	8.0	78.2	951.4	4 687.1
(≥8 years)	Nemaska	82	100.0	173.1	(196.8)	50.6	(36.2-70.6)		3.6	8.3	39.2	455.2	3 010.2

[1] % of detection; detection limit (DL): 0.02 µg/L (see Table 27)

[2] Comparison of the geometric means between Oujé-Bougoumou and Nemaska

Table 30 Whole-blood concentrations of cadmium (nmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)		Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100.0	7.12	(8.57)	5.60	(4.43-7.08)	0.754	-	3.56	3.56	4.45	7.12	43.60
	Nemaska	11	100.0	9.22	(13.75)	5.05	(2.80-9.12)		-	1.78	2.67	3.56	21.35	47.15
Women	Oujé-Bougoumou	78	100.0	24.85	(17.53)	18.32	(15.18-22.10)	0.686	<0.001	3.56	5.34	23.58	46.26	77.40
15-39 years	Nemaska	43	100.0	26.36	(16.65)	19.58	(15.00-25.55)		<0.001	2.67	5.34	27.58	49.82	58.72
	Quebec	104	100.0	7.39	(10.76)	4.51	(3.83-5.30)			1.19	2.16	3.67	16.40	58.60
Men	Oujé-Bougoumou	40	100.0	23.22	(15.07)	17.41	(13.36-22.69)	0.424	<0.001	2.67	5.34	22.24	42.71	56.05
15-39 years	Nemaska	15	100.0	31.02	(23.49)	21.62	(13.20-35.41)		<0.001	4.45	4.45	29.36	59.61	83.63
	Quebec	37	100.0	11.65	(15.99)	5.72	(3.96-8.25)			1.52	1.75	3.92	42.10	61.20
Men & women	Oujé-Bougoumou	51	100.0	11.27	(10.18)	8.52	(7.03-10.32)	0.886	0.050	3.56	4.45	7.12	24.02	47.15
≥40 years	Nemaska	13	100.0	14.71	(18.02)	8.81	(5.13-15.14)		0.334	2.67	2.67	8.01	37.37	64.95
	Quebec	331	99.4	11.46	(14.75)	6.83	(6.18-7.55)			<DL	2.73	5.23	30.80	81.50
Total	Oujé-Bougoumou	169	100.0	21.25	(7.54)	15.10	(13.24-17.22)	0.290	<0.001	2.67	5.34	16.90	43.60	77.40
≥15 years	Nemaska	71	100.0	25.21	(8.94)	17.26	(13.78-21.63)		<0.001	2.67	4.45	22.24	51.60	83.63
	Quebec	472	99.6	10.58	(14.14)	6.15	(5.65-6.69)			<DL	2.29	4.87	30.50	81.50
Total	Oujé-Bougoumou	190	100.0	18.66	(7.77)	12.58	(11.06-14.32)	0.512	-	2.67	4.45	11.57	42.71	77.40
(≥8 years)	Nemaska	82	100.0	22.18	(9.29)	13.68	(10.82-17.28)		-	1.78	2.67	18.68	50.71	83.63

[1] Unpublished data. INSPQ, Leblanc et al., 2003

[2] % of detection; detection limit (DL): 1 nmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 31 Whole-blood concentrations of cadmium (nmol/L) among non-smoking Oujé-Bougoumou and Nemaska participants (≥15 years of age), and a comparator population from Southern Quebec[1]

Community	n	% det.[2]	Mean (sd)	Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
Oujé-Bougoumou	32	100.0	6.83 (1.76)	6.21	(5.39-7.15)	0.039	0.028	2.67	4.45	5.34	10.68	22.24
Nemaska	5	100.0	4.27 (0.55)	4.13	(3.19-5.35)		0.914	2.67	-	-	-	5.34
Quebec	244	99.2	4.73 (4.06)	3.89	(3.62-4.19)			<DL	2.09	3.76	7.24	29.90

[1] Unpublished data. INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 1 nmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 32 Exceedances of the concern and action levels for whole-blood cadmium among the Oujé-Bougoumou and Nemaska participants (>15 years of age)

		> Concern level[1]			> Action level[1]		
Category	Community	Concern level (nmol/L)	n (%)	p value[2]	Action level (nmol/L)	n (%)	p value[2]
Non-smoker	Oujé-Bougoumou	5.0	27 (85.1)	0.003	44.5	0 (0,0)	-
	Nemaska		2 (21.7)			0 (0,0)	
Smokers	Oujé-Bougoumou	25.0	58 (42.3)	0.452	44.5	14 (10.2)	0.121
	Nemaska		32 (48.5)			12 (18.2)	

[1] Source: CTQ 1990-1995; or Järup et al., 1988; Elinder and Järup (1996); also see Table 63, Appendix 6 and the cadmium profile in Appendix 1 for background details. Kidney dysfunction is the target outcome considered.

[2] p value corresponds to the chi-square test for inter-community comparison of proportions.

Table 33 Whole-blood concentrations of total mercury (nmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)		Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	95.2	12.9	(13.5)	8.1	(5.2-12.6)	0.093	-	<DL	3.0	8.0	22.9	49.9
	Nemaska	11	72.7	7.1	(6.9)	4.0	(1.9-8.4)		-	<DL	<DL	6.0	18.9	19.9
Women	Oujé-Bougoumou	78	100.0	20.5	(22.0)	13.8	(11.5-16.7)	0.009	<0.001	3.0	5.0	14.0	39.9	109.2
15-39 years	Nemaska	43	95.3	13.5	(16.9)	8.9	(6.7-11.7)		<0.001	<DL	4.0	9.0	23.9	105.2
	Quebec	103	95.1	4.8	(3.9)	3.6	(3.1-4.2)			<DL	1.0	4.0	10.0	23.0
Men	Oujé-Bougoumou	40	100.0	37.8	(40.9)	23.1	(16.8-31.9)	0.160	<0.001	2.0	6.5	24.4	90.2	174.0
15-39 years	Nemaska	15	100.0	31.7	(48.2)	14.5	(7.8-26.8)		0.002	2.0	5.0	11.0	117.1	168.0
	Quebec	37	100.0	5.3	(3.2)	4.5	(3.7-5.4)			1.0	2.0	5.0	11.0	13.0
Men & women	Oujé-Bougoumou	51	100.0	74.2	(68.0)	51.2	(39.8-65.8)	0.291	<0.001	6.0	16.9	52.8	160.0	374.9
≥40 years	Nemaska	13	100.0	110.9	(138.5)	69.5	(40.9-118.4)		<0.001	9.0	38.9	62.8	196.9	538.4
	Quebec	330	93.6	5.2	(5.7)	3.7	(3.4-4.0)			<DL	1.0	4.0	10.0	50.0
Total	Oujé-Bougoumou	169	100.0	37.3	(21.5)	21.3	(18.2-25.0)	0.013	<0.001	1.99	6.0	19.9	95.2	374.9
(≥15 years)	Nemaska	71	97.2	35.2	(34.3)	14.4	(10.7-19.3)		<0.001	<DL	4.0	11.0	70.8	538.4
	Quebec	470	94.5	5.1	(5.1)	3.7	(3.5-4.0)			<DL	1.0	4.0	10.0	50.0
Total	Oujé-Bougoumou	190	99.5	32.8	(21.0)	17.8	(15.2-20.9)	0.003	-	<DL	4.0	16.9	82.8	374.9
(≥8 years)	Nemaska	82	93.9	29.9	(32.3)	11.2	(8.4-15.1)		-	<DL	2.0	10.0	66.8	538.4

[1] Unpublished data: INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 1 nmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 34 Whole-blood concentrations (µg/L) of total mercury among the Oujé-Bougoumou and Nemaska and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)	Geometric mean (95% CI)	p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	95.2	2.59 (2.71)	1.63 (1.05-2.53)	0.093	-	<DL	0.60	1.60	4.60	10.00
	Nemaska	11	72.7	1.42 (1.38)	0.79 (0.37-1.69)		-	<DL	<DL	1.20	3.80	4.00
Women	Oujé-Bougoumou	78	100.0	4.11 (4.41)	2.78 (2.30-3.36)	0.009	<0.001	0.60	1.00	2.80	8.00	21.90
15-39 years	Nemaska	43	95.3	2.70 (3.38)	1.78 (1.35-2.35)		<0.001	<DL	0.80	1.80	4.80	21.10
	Quebec	103	95.1	0.96 (0.77)	0.72 (0.62-0.84)			<DL	0.20	0.80	2.01	4.61
Men	Oujé-Bougoumou	40	100.0	7.58 (8.20)	4.64 (3.36-6.39)	0.160	<0.001	0.40	1.30	4.90	18.10	34.90
15-39 years	Nemaska	15	100.0	6.36 (9.67)	2.90 (1.56-5.38)		0.002	0.40	1.00	2.20	23.50	33.70
	Quebec	37	100.0	1.06 (0.64)	0.89 (0.73-1.09)			0.20	0.40	1.00	2.21	2.61
Men & women	Oujé-Bougoumou	51	100.0	14.89 (13.64)	10.27 (7.99-13.19)	0.291	<0.001	1.20	3.40	10.60	32.10	75.20
≥40 years	Nemaska	13	100.0	22.24 (27.79)	13.95 (8.20-23.74)		<0.001	1.80	7.80	12.60	39.50	108.00
	Quebec	330	93.6	1.05 (1.13)	0.74 (0.67-0.81)			<DL	0.20	0.80	2.01	10.03
Total	Oujé-Bougoumou	169	100.0	7.48 (4.32)	4.27 (3.65-5.01)	0.013	<0.001	0.40	1.20	4.00	19.10	75.20
(≥15 years)	Nemaska	71	97.2	7.07 (6.87)	2.88 (2.15-3.86)		<0.001	<DL	0.80	2.20	14.20	108.00
	Quebec	470	94.5	1.03 (1.03)	0.74 (0.69-0.80)			<DL	0.20	0.80	2.01	10.03
Total	Oujé-Bougoumou	190	99.5	6.58 (4.21)	3.58 (3.06-4.19)	0.003		<DL	0.80	3.40	16.60	75.20
(≥8 years)	Nemaska	82	93.9	6.00 (6.49)	2.26 (1.68-3.02)			<DL	0.40	2.00	13.40	108.00

[1] Unpublished data: INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 0.1 µg/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 35 Exceedances of the concern and action levels for whole-blood total mercury among the Oujé-Bougoumou and Nemaska participants

Group	Community	> Concern level			> Action level		
		Concern level	n (%)	p value[1]	Action level	n (%)	p value[1]
8-14 years	Oujé-Bougoumou	74.8 nmol/L[2]	0 (0.0)	-	99.7 nmol/L[3]	0 (0.0)	-
	Nemaska		0 (0.0)			0 (0.0)	
Women 15-39 years	Oujé-Bougoumou	74.8 nmol/L[2]	5 (6.3)	0.421	99.7 nmol/L[3]	1 (1.3)	-
	Nemaska		1 (2.3)			1 (2.3)	
Men 15-39 years	Oujé-Bougoumou	74.8 nmol/L[2]	5 (12.5)	1.000	498.5 nmol/L[4]	0 (0.0)	-
	Nemaska		2 (13.3)			0 (0.0)	
	Oujé-Bougoumou	99.7 nmol/L[3]	3 (7.5)	0.606			
	Nemaska		2 (13.3)				
Men & women ≥40 years	Oujé-Bougoumou	74.8 nmol/L[2]	20 (39.2)	0.751	498.5 nmol/L[4]	0 (0.0)	0.203
	Nemaska		4 (30.8)			1 (7.7)	
	Oujé-Bougoumou	99.7 nmol/L[3]	12 (23.5)	0.721			
	Nemaska		4 (30.8)				

[1] p value corresponds to the Fisher exact test for comparison of proportions

[2] Source: Level declarable to Public Health authorities in Quebec (also see Table 63, Appendix 6 and the mercury toxicological profile in Appendix 1)

[3] Source: WHO 1972, HWC 1979, Walker 1996 (also see Table 63 and Appendix 6)

[4] Source: WHO 1972, HWC 1979 (also see Table 63 and Appendix 6)

Table 36 Whole-blood concentrations of lead (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)		Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
0-14 years	Oujé-Bougoumou	51	100	0.127	(0.110)	0.099	(0.082-0.119)	0.009	-	0.029	0.048	0.082	0.251	0.652
	Nemaska	27	100	0.078	(0.060)	0.067	(0.055-0.081)		-	0.024	0.029	0.068	0.101	0.357
Women	Oujé-Bougoumou	78	100	0.108	(0.190)	0.072	(0.061-0.085)	0.812	0.370	0.024	0.034	0.063	0.169	1.593
15-39 years	Nemaska	43	100	0.087	(0.073)	0.070	(0.058-0.084)		0.628	0.019	0.034	0.068	0.174	0.430
	Quebec	104	100	0.072	(0.031)	0.066	(0.062-0.071)			0.031	0.041	0.063	0.114	0.205
Men	Oujé-Bougoumou	40	100	0.164	(0.122)	0.129	(0.104-0.160)	0.467	0.034	0.039	0.055	0.111	0.369	0.531
15-39 years	Nemaska	15	100	0.145	(0.126)	0.110	(0.076-0.161)		0.573	0.039	0.043	0.106	0.319	0.507
	Quebec	37	100	0.106	(0.045)	0.098	(0.087-0.111)			0.048	0.067	0.094	0.180	0.262
Men & women	Oujé-Bougoumou	51	100	0.214	(0.154)	0.170	(0.141-0.206)	0.005	0.002	0.039	0.077	0.179	0.425	0.700
≥40 years	Nemaska	13	100	0.354	(0.193)	0.309	(0.230-0.416)		<0.001	0.154	0.159	0.328	0.579	0.772
	Quebec	331	100	0.138	(0.073)	0.123	(0.117-0.130)			0.031	0.068	0.122	0.224	0.538
Total	Oujé-Bougoumou	169	100	0.146	(0.079)	0.101	(0.090-0.114)	0.987	0.625	0.024	0.039	0.092	0.314	1.593
(≥15 years)	Nemaska	71	100	0.149	(0.071)	0.101	(0.083-0.123)		0.008	0.019	0.039	0.087	0.367	0.772
	Quebec	472	100	0.121	(0.070)	0.106	(0.101-0.111)			0.031	0.054	0.107	0.203	0.538
Total	Oujé-Bougoumou	220	100	0.139	(0.077)	0.100	(0.091-0.111)	0.115	-	0.024	0.043	0.087	0.294	1.593
(≥0 years)	Nemaska	98	100	0.123	(0.066)	0.087	(0.075-0.101)		-	0.019	0.039	0.077	0.319	0.772

[1] Unpublished data: INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 0.001 µmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 37 Exceedances of the concern and action levels of whole-blood lead among Cree populations of Oujé-Bougoumou and Nemaska

Group	Community	> Concern level				> Action level			
		Concern level	n	(%)	p value[1]	Action level	n	(%)	p value[1]
0-14 years	Oujé-Bougoumou	0.48 µmol/L[2]	1	(2.0)	1.000	0.96 µmol/L[3,4]	0	(0.0)	-
	Nemaska		0	(0.0)			0	(0.0)	
Women 15-39 years	Oujé-Bougoumou	0.48 µmol/L[2]	1	(1.3)	1.000	0.96 µmol/L[3,4]	1	(1.3)	1.000
	Nemaska		0	(0.0)			0	(0.0)	
Men 15-39 years	Oujé-Bougoumou	0.48 µmol/L[2]	1	(2.5)	0.475	0.96 µmol/L[3,4]	0	(0.0)	-
	Nemaska		1	(6.7)			0	(0.0)	
Men & women ≥40 years	Oujé-Bougoumou	0.48 µmol/L[2]	4	(7.8)	0.142	0.96 µmol/L[3,4]	0	(0.0)	-
	Nemaska		3	(23.1)			0	(0.0)	

[1] p value corresponds to the Fisher exact test for comparison of proportions

[2] Source: CEOH 1994, level declarable to public health authorities in Quebec (also see Table 63, Appendix 6 and lead toxicological profile in Appendix 1)

[3] Source: CBH, 2003 (also see Table 63 and Appendix 6)

[4] Source: US CDC, 1997 (also see Table 63 and Appendix 6)

Table 38 Plasma concentrations of copper (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)		Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100	16.10	(2.18)	15.95	(15.03-16.94)	0.811	-	11.49	13.54	16.29	19.12	19.52
	Nemaska	11	100	16.53	(3.19)	16.20	(14.24-18.43)		-	10.47	11.18	17.71	19.28	19.68
Women	Oujé-Bougoumou	78	100	19.54	(5.31)	18.92	(17.90-20.00)	0.711	0.161	11.81	14.40	18.02	28.57	36.99
15-39 years	Nemaska	43	100	19.00	(4.36)	18.62	(17.56-19.73)		0.065	14.48	15.27	17.79	22.59	35.57
	Quebec	4	100	23.25	(5.87)	22.68	(17.55-29.29)			16.7	-	-	-	29.1
Men	Oujé-Bougoumou	39	100	13.99	(2.29)	13.81	(13.13-14.52)	0.098	0.139	8.97	11.81	13.54	18.18	18.89
15-39 years	Nemaska	15	100	15.03	(1.89)	14.92	(14.02-15.88)		0.498	12.83	12.83	14.40	17.00	19.28
	Quebec	4	100	15.73	(1.81)	15.65	(13.98-17.51)			14	-	-	-	17.7
Men & women	Oujé-Bougoumou	51	100	16.11	(2.71)	15.87	(15.13-16.65)	0.062	0.152	9.99	13.30	15.90	19.12	22.27
≥40 years	Nemaska	13	100	17.85	(3.23)	17.60	(16.00-19.35)		0.506	14.17	14.40	18.18	20.46	25.18
	Quebec	38	100	17.24	(4.14)	16.84	(15.74-18.02)			12	13.3	16.65	23.5	32.1
Total	Oujé-Bougoumou	168	100	17.44	(2.22)	16.87	(16.24-17.52)	0.214	0.585	8.97	12.67	16.53	23.22	36.99
(≥15 years)	Nemaska	71	100	17.95	(1.90)	17.58	(16.79-18.40)		0.613	12.83	14.17	17.24	21.80	35.57
	Quebec	46	100	17.63	(4.45)	17.17	(16.09-18.32)			12	13.7	16.75	25.1	32.1
Total	Oujé-Bougoumou	189	100	17.19	(2.15)	16.70	(16.15-17.27)	0.226	-	8.97	12.83	16.53	22.19	36.99
(≥8 years)	Nemaska	82	100	17.68	(1.90)	17.31	(16.57-18.08)		-	10.47	14.09	17.31	21.80	35.57

[1] Unpublished data: INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 0.01 µmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 39 Exceedances of the concern level of plasma copper among the Oujé-Bougoumou and Nemaska participants

Group	Community	> Concern level[1,2] of 25.2 µmol/L		
		n	(%)	p value[3]
8-14 years	Oujé-Bougoumou	0	(0.0)	-
	Nemaska	0	(0.0)	
Women 15-39 years	Oujé-Bougoumou	11	(14.1)	0.570
	Nemaska	4	(9.3)	
Men 15-39 years	Oujé-Bougoumou	0	(0.0)	-
	Nemaska	0	(0.0)	
Men & women ≥40 years	Oujé-Bougoumou	0	(0.0)	0.203
	Nemaska	1	(7.7)	

[1] This concern level does not apply to pregnant women near their term or women taking oral contraceptives. In this instance, the recommended level of concern is 47.4 µmol/L (see Table 63) (Burtis and Ashwood, 1996)

[2] Source: Ellenhorn 1999

[3] p value corresponds to the Fisher exact test for comparison of proportions.

Table 40 Plasma concentrations of selenium (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)	Geometric mean (95% CI)		p value[3]	p value[4]	Percentiles Minimum	10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100	1.34 (0.15)	1.34	(1.28- 1.40)	0.210	-	1.16	1.18	1.30	1.54	1.73
	Nemaska	11	100	1.41 (0.17)	1.41	(1.32- 1.50)		-	1.24	1.25	1.37	1.46	1.87
Women	Oujé-Bougoumou	78	100	1.43 (0.15)	1.42	(1.39- 1.46)	0.015	0.760	1.05	1.25	1.43	1.63	1.82
15-39 years	Nemaska	43	100	1.50 (0.15)	1.50	(1.45- 1.54)		0.974	1.16	1.30	1.49	1.73	1.84
	Quebec	4	100	1.53 (0.36)	1.49	(1.14- 1.93)			1.00	-	-	-	1.77
Men	Oujé-Bougoumou	39	100	1.54 (0.16)	1.53	(1.48- 1.58)	0.293	0.244	1.20	1.32	1.54	1.71	2.00
15-39 years	Nemaska	15	100	1.59 (0.13)	1.58	(1.52- 1.65)		0.531	1.38	1.39	1.61	1.75	1.76
	Quebec	4	100	1.65 (0.23)	1.64	(1.44- 1.87)			1.43	-	-	-	1.97
Men & women	Oujé-Bougoumou	51	100	1.45 (0.20)	1.44	(1.38- 1.49)	0.036	<0.001	0.77	1.27	1.44	1.68	1.95
≥40 years	Nemaska	13	100	1.58 (0.16)	1.58	(1.49- 1.66)		0.278	1.33	1.41	1.54	1.77	1.95
	Quebec	38	100	1.66 (0.21)	1.65	(1.58- 1.72)			1.00	1.45	1.69	1.93	2.06
Total	Oujé-Bougoumou	168	100	1.46 (0.08)	1.45	(1.42- 1.48)	0.002	<0.001	0.77	1.27	1.46	1.68	2.00
(≥15 years)	Nemaska	71	100	1.54 (0.07)	1.53	(1.49- 1.56)		0.025	1.16	1.35	1.52	1.73	1.95
	Québec	46	100	1.65 (0.22)	1.63	(1.56- 1.70)			1.00	1.43	1.68	1.93	2.06
Total	Oujé-Bougoumou	189	100	1.44 (0.08)	1.43	(1.40- 1.45)	0.001		0.77	1.24	1.43	1.66	2.00
(≥8 years)	Nemaska	82	100	1.51 (0.08)	1.50	(1.47- 1.54)			1.16	1.32	1.49	1.73	1.95

[1] Unpublished data: INSPQ, Leblanc *et al*., 2003

[2] % of detection; detection limit (DL): 0.1 µmol/L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 41 Exceedances of the concern and action levels of plasma selenium among Oujé-Bougoumou and Nemaska participants

		> Concern level[1] (2 µmol/L)			> Action level[2] (of 3.0 µmol/L)	
Group	**Community**	**n**	**(%)**	**p value[3]**	**n (%)**	**p value[3]**
8-14 years	Oujé-Bougoumou	0		-	0	-
	Nemaska	0			0	
Women 15-39 years	Oujé-Bougoumou	0		-	0	-
	Nemaska	0			0	
Men 15-39 years	Oujé-Bougoumou	1	(2.6)	1.000	0	-
	Nemaska	0	(0.0)		0	
Men & women ≥40 years	Oujé-Bougoumou	0		-	0	-
	Nemaska	0			0	

[1] Source: CTQ, 2003; upper end of the laboratory reference range

[2] Source: Nantel *et al.*, 1985; based on the follow-up of acute poisoning in a child

[3] p value corresponds to the Fisher exact test for comparison of proportions.

Table 42 Plasma concentrations of zinc (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1,2]

Group	Community	n	% det.[3]	Mean (sd)	Geometric mean (95% CI)	p value[4]	p value[5]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100	14.41 (1.42)	14.34 (13.74-14.96)	0.009	-	11.70	12.90	14.50	16.40	16.71
	Nemaska	11	100	13.03 (1.12)	12.98 (12.33-13.67)		-	11.20	11.60	13.10	14.40	14.40
Women 15-39 years	Oujé-Bougoumou	78	100	12.84 (1.97)	12.69 (12.27-13.13)	0.067	0.184	8.64	10.01	13.01	15.19	18.80
	Nemaska	43	100	12.21 (1.38)	12.13 (11.74-12.54)		0.012	9.44	10.50	12.10	14.31	16.00
	Quebec	4	100	14.13 (1.21)	14.09 (12.98-15.29)			13.10	-	-	-	15.80
Men 15-39 years	Oujé-Bougoumou	39	100	13.62 (2.56)	13.39 (12.62-14.20)	0.139	0.887	8.94	10.50	13.69	16.80	20.79
	Nemaska	15	100	12.47 (1.77)	12.35 (11.47-13.30)		0.236	9.23	10.21	12.19	14.50	14.50
	Quebec	4	100	13.60 (1.06)	13.57 (12.59-14.63)			12.60	-	-	-	15.00
Men & women ≥40 years	Oujé-Bougoumou	51	100	13.93 (2.67)	13.70 (13.04-14.40)	0.789	0.454	10.30	11.09	13.60	17.40	22.90
	Nemaska	13	100	13.81 (3.56)	13.49 (11.99-15.17)		0.535	10.60	11.00	12.39	15.30	24.40
	Quebec	38	100	14.14 (1.67)	14.04 (13.49-14.62)			8.66	12.40	14.30	15.80	17.80
Total (≥15 years)	Oujé-Bougoumou	168	100	13.28 (1.07)	13.09 (12.75-13.43)	0.023	0.001	8.64	10.50	13.10	16.29	22.90
	Nemaska	71	100	12.56 (0.98)	12.42 (12.00-12.85)		<0.001	9.23	10.60	12.19	14.50	24.40
	Quebec	46	100	14.09 (1.57)	14.00 (13.53-14.49)			8.66	12.60	14.00	15.80	17.80
Total (≥8 years)	Oujé-Bougoumou	189	100	13.49 (1.07)	13.31 (13.00-13.62)	0.003		8.64	10.60	13.40	16.40	22.90
	Nemaska	82	100	12.65 (0.94)	12.52 (12.16-12.90)			9.23	10.69	12.19	14.50	24.40

[1] Unpublished data: INSPQ, Leblanc et al., 2003

[2] A level of concern of >22 µmol/L is suggested and an action level of >30 µmol/L (CTQ, 2003)

[3] % of detection; detection limit (DL): 0.05 µmol/L

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[5] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 43 Urinary concentrations of inorganic arsenic ("non-dietary" arsenic)[1] among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[2]

Group	Community	n	% det.[3]	Mean (sd)	Geometric mean (95% CI)	p value[4]	p value[5]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100.0	0.070 (0.041)	0.059 (0.045-0.077)	0.008	-	0.016	0.024	0.067	0.134	0.158
	Nemaska	11	100.0	0.097 (0.030)	0.093 (0.078-0.111)		-	0.059	0.067	0.088	0.147	0.155
Women	Oujé-Bougoumou	74	98.6	0.095 (0.050)	0.082 (0.071-0.094)	0.510	-	0.007	0.043	0.085	0.176	0.224
15-39 years	Nemaska	42	95.2	0.122 (0.091)	0.091 (0.069-0.119)		-	0.007	0.025	0.100	0.235	0.409
	Quebec	79	16.5	-	- -			<DL	<DL	<DL	0.13	0.59
Men	Oujé-Bougoumou	40	97.5	0.088 (0.076)	0.067 (0.054-0.085)	0.300	-	0.007	0.035	0.071	0.140	0.390
15-39 years	Nemaska	15	100.0	0.096 (0.045)	0.084 (0.063-0.113)		-	0.020	0.043	0.093	0.168	0.178
	Quebec	25	40.0	-	- -			<DL	<DL	<DL	0.13	0.23
Men & women	Oujé-Bougoumou	50	92.0	0.072 (0.047)	0.056 (0.044-0.070)	0.150	-	0.007	0.023	0.062	0.139	0.199
≥40 years	Nemaska	13	100.0	0.092 (0.055)	0.080 (0.059-0.107)		-	0.035	0.039	0.076	0.186	0.218
	Quebec	259	23.6	-	- -			<DL	<DL	<DL	0.16	1.45
Total	Oujé-Bougoumou	164	96.3	0.088 (0.026)	0.071 (0.064-0.079)	0.053	-	<DL	0.035	0.077	0.166	0.390
(≥15 years)	Nemaska	70	97.1	0.111 (0.037)	0.087 (0.073-0.104)		-	<DL	0.039	0.093	0.186	0.409
	Quebec	363	23.1	-	- -			<DL	<DL	<DL	0.14	1.45
Total	Oujé-Bougoumou	185	96.8	0.085 (0.026)	0.069 (0.062-0.076)	0.007	-	0.007	0.032	0.075	0.154	0.390
(≥8 years)	Nemaska	81	97.5	0.108 (0.035)	0.088 (0.076-0.103)		-	0.007	0.043	0.093	0.182	0.409

[1] Inorganic arsenic includes its metabolites and traditionally is referred to as "non-dietary" arsenic (see text)

[2] Unpublished data: INSPQ, Leblanc et al., 2003

[3] % of detection; detection limit (DL): 0.01 µmol/L for Oujé-Bougoumou and Nemaska and 0.1 µmol/L for the Southern Quebec population

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[5] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 44 Urinary concentrations of total arsenic (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean[3] (sd)	Geometric mean[3] (95% CI)		p value[4]	p value[5]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	47.6	-	-	-	-	-	<DL	<DL	<DL	0.187	0.774
	Nemaska	11	100.0	0.158 (0.034)	0.155	(0.137-0.175)		-	0.120	0.120	0.160	0.200	0.227
					-	-							
Women	Oujé-Bougoumou	74	62.2	0.220 (0.449)	0.136	(0.113-0.162)	0.019	-	<DL	<DL	0.120	0.307	3.738
15-39 years	Nemaska	42	85.7	0.489 (1.749)	0.198	(0.152-0.258)		-	<DL	<DL	0.174	0.401	11.481
	Quebec	79	59.5	-	-	-			<DL	<DL	0.116	0.878	3.46
Men	Oujé-Bougoumou	40	57.5		-	-	-	-	<DL	<DL	0.100	0.194	1.802
15-39 years	Nemaska	15	86.7	0.154 (0.050)	0.145	(0.119-0.176)		0.117	<DL	<DL	0.160	0.214	0.227
	Quebec	25	68.0	0.519 (0.938)	0.220	(0.137-0.352)			<DL	<DL	0.142	1.350	4.400
Men & women	Oujé-Bougoumou	50	40.0	-	-	-	-	-	<DL	<DL	<DL	0.280	0.587
≥40 years	Nemaska	13	46.2	-	-	-		-	<DL	<DL	< DL	0.254	0.654
	Quebec	259	61.0	0.475 (1.817)	0.176	(0.154-0.200)			<DL	<DL	0.129	0.868	24.800
Total	Oujé-Bougoumou	164	54.3	-	-	-	-	-	<DL	<DL	0.107	0.280	3.738
(≥15 years)	Nemaska	70	78.6	0.355 (0.639)	0.168	(0.139-0.202)		0.873	<DL	<DL	0.174	0.307	11.481
	Quebec	363	61.2	0.446 (1.580)	0.175	(0.156-0.195)			<DL	<DL	0.128	0.868	24.800
Total	Oujé-Bougoumou	185	53.5	-	-	-	-	-	<DL	<DL	0.107	0.280	3.738
(≥8 years)	Nemaska	81	81.5	0.317 (0.595)	0.165	(0.141-0.193)		-	<DL	<DL	0.160	0.294	11.481

[1] Unpublished data: INSPQ, Leblanc et al., 2003

[2] % of detection; detection limit (DL): 0.1 µmol/L

[3] Means calculated when the detection frequency was at least 60%

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[5] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 45 Exceedances of the concern and action levels of inorganic "non-dietary"urinary arsenic among the Oujé-Bougoumou and Nemaska participants

Group	Community	> Concern level[1] of 0.25 µmol/L		> Action level[2] of 0.47 µmol/L	
		n (%)	p value[3]	n (%)	p value[3]
8-14 years	Oujé-Bougoumou	0 (0.0)	-	0 (0.0)	-
	Nemaska	0 (0.0)		0 (0.0)	
Women 15-39 years	Oujé-Bougoumou	0 (0.0)	0.045	0 (0.0)	-
	Nemaska	3 (7.1)		0 (0.0)	
Men 15-39 years	Oujé-Bougoumou	2 (5.0)	1.000	0 (0.0)	-
	Nemaska	0 (0.0)		0 (0.0)	
Men & women ≥40 years	Oujé-Bougoumou	0 (0.0)	-	0 (0.0)	-
	Nemaska	0 (0.0)		0 (0.0)	

[1] Source: CTQ, 2003; upper end of the laboratory reference range

[2] Source: ACGIH 2002; occupational guideline or biological exposure index

[3] p value obtained by Fisher exact test for comparison of proportions.

Table 46 Urinary concentrations of cadmium (nmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean[3] (sd)	Geometric mean[3] (95% CI)	p value[4]	p value[5]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	33.3	-	-	-	-	<DL	<DL	<DL	4.45	5.34
	Nemaska	11	36.4	-	-		-	<DL	<DL	<DL	4.45	4.45
Women	Oujé-Bougoumou	74	82.4	7.15 (4.99)	5.66 (4.83-6.65)	0.738	-	<DL	<DL	6.23	14.24	24.02
15-39 years	Nemaska	42	76.2	7.82 (5.97)	5.94 (4.70-7.51)		-	<DL	<DL	5.78	15.12	29.36
	Quebec	79	59.5	-	-			<DL	<DL	3.33	7.39	11.20
Men	Oujé-Bougoumou	40	70.0	6.26 (4.86)	4.78 (3.79-6.04)	0.926	0.508	<DL	<DL	4.89	11.57	24.91
15-39 years	Nemaska	15	80.0	5.42 (2.83)	4.69 (3.49-6.30)		0.631	<DL	<DL	5.34	8.90	11.57
	Quebec	25	64.0	5.54 (4.96)	4.23 (3.21-5.56)			<DL	<DL	4.01	16.00	19.80
Men & women	Oujé-Bougoumou	50	78.0	7.50 (6.78)	5.68 (4.61-6.99)	0.389	0.547	<DL	<DL	6.23	12.90	44.49
≥40 years	Nemaska	13	69.2	11.05 (10.17)	7.08 (4.00-12.54)		0.348	<DL	<DL	8.01	18.68	37.37
	Quebec	259	78.8	7.04 (6.68)	5.31 (4.88-5.79)			<DL	<DL	5.03	14.70	42.80
Total	Oujé-Bougoumou	164	78.0	7.01 (2.49)	5.44 (4.87-6.08)	0.509	0.236	<DL	<DL	5.34	14.24	44.49
(≥15 years)	Nemaska	70	75.7	7.91 (3.12)	5.83 (4.84-7.03)		0.241	<DL	<DL	6.23	16.90	37.37
	Quebec	363	73.6	6.29 (6.00)	4.79 (4.47-5.14)			<DL	<DL	4.61	11.20	42.80
Total	Oujé-Bougoumou	185	73.0	6.18 (2.50)	4.66 (4.19-5.19)	0.527	-	<DL	<DL	4.45	12.46	44.49
(≥8 years)	Nemaska	81	70.4	6.92 (3.08)	4.97 (4.17-5.92)		-	<DL	<DL	5.34	15.12	37.37

[1] Unpublished data: INSPQ, Leblanc et al., 2003

[2] % of detection; detection limit (DL): 3 nmol/L

[3] Means calculated when the detection frequency was at least 60%

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[5] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and a population from South of Quebec

Table 47 Urinary concentrations of cadmium (nmol/L) among non-smoking Oujé-Bougoumou and Nemaska participants, and non-smokers in a comparator population from Southern Quebec[1]

Community	n	% det.[2]	Mean[3] (sd)	Geometric mean[3] (95% CI)		p value[4]	p value[5]	Minimum	Percentiles 10th	50th	90th	Maximum
Oujé-Bougoumou	31	67.7	6.90 (2.70)	5.02	(3.76-6.70)	0.120	0.197	<DL	<DL	5.34	14.24	24.02
Nemaska	5	40.0	-	-	-		0.497	<DL	-	-	-	5.34
Quebec	196	66.8	4.49 (2.77)	3.87	(3.60-4.17)			<DL	<DL	3.96	7.47	19.80

[1] Unpublished data: INSPQ, Leblanc et al., 2003

[2] % of detection; detection limit (DL): 3 nmol/L

[3] Means calculated when the detection frequency was at least 60%

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[5] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and a population from South of Quebec

Table 48 Exceedances of the concern and action levels of urinary cadmium among non-smoking Oujé-Bougoumou and Nemaska participants (≥15 years of age)

	> Concern level[1] of 9.96 nmol/L			> Action level[2] of 44.5 nmol/L	
Community	n	(%)	p value[3]	n (%)	p value[3]
Oujé-Bougoumou	7	(22.6)	0.250	0 (0.0)	-
Nemaska	0	(0.0)		0 (0.0)	

[1] Source: CTQ, 2003; corresponds to the upper end of the laboratory reference range

[2] Source: ACGIH, 2003; occupational guideline or biological index; estimated to correspond to a 5-10% probability of the prevalence of kidney dysfunction (Elinder and Järup, 1996)

[3] p value obtained by Fisher exact test for comparison of proportions.

Table 49 Urinary concentrations of copper (µmol/L) among the Oujé-Bougoumou and Nemaska participants, and a comparator population from Southern Quebec[1]

Group	Community	n	% det.[2]	Mean (sd)		Geometric mean (95% CI)		p value[3]	p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	21	100.0	0.300	(0.086)	0.288	(0.253-0.327)	0.394	-	0.137	0.211	0.299	0.409	0.488
	Nemaska	11	100.0	0.268	(0.046)	0.264	(0.238-0.293)		-	0.189	0.220	0.269	0.326	0.349
Women	Oujé-Bougoumou	74	100.0	0.331	(0.253)	0.294	(0.265-0.326)	0.021	0.005	0.046	0.186	0.288	0.455	2.298
15-39 years	Nemaska	42	100.0	0.263	(0.102)	0.236	(0.201-0.277)		0.917	0.046	0.120	0.253	0.387	0.519
	Quebec	79	100.0	0.265	(0.128)	0.234	(0.208-0.263)			0.057	0.106	0.244	0.443	0.620
Men	Oujé-Bougoumou	40	100.0	0.252	(0.089)	0.229	(0.196-0.268)	0.769	0.201	0.046	0.120	0.261	0.363	0.439
15-39 years	Nemaska	15	100.0	0.270	(0.114)	0.240	(0.179-0.322)		0.496	0.042	0.148	0.260	0.386	0.519
	Quebec	25	100.0	0.300	(0.138)	0.270	(0.223-0.326)			0.098	0.127	0.273	0.521	0.627
Men & women	Oujé-Bougoumou	50	100.0	0.278	(0.175)	0.239	(0.204-0.280)	0.497	0.075	0.038	0.113	0.243	0.430	1.118
≥40 years	Nemaska	13	100.0	0.326	(0.255)	0.270	(0.193-0.377)		0.094	0.072	0.168	0.279	0.433	1.118
	Quebec	259	99.6	0.251	(0.371)	0.202	(0.188-0.218)			<DL	0.089	0.217	0.385	5.900
Total	Oujé-Bougoumou	164	100.0	0.299	(0.095)	0.263	(0.243-0.284)	0.284	0.019	0.038	0.165	0.269	0.419	2.298
(≥15 years)	Nemaska	70	100.0	0.276	(0.068)	0.243	(0.214-0.277)		0.351	0.042	0.148	0.257	0.403	1.118
	Quebec	363	99.7	0.258	(0.321)	0.213	(0.200-0.226)			<DL	0.098	0.228	0.398	5.900
Total	Oujé-Bougoumou	185	100.0	0.299	(0.091)	0.268	(0.250-0.287)	0.213		0.038	0.173	0.275	0.419	2.298
(≥8 years)	Nemaska	81	100.0	0.275	(0.064)	0.247	(0.221-0.275)			0.042	0.168	0.263	0.386	1.118

[1] Unpublished data: INSPQ, Leblanc et al., 2003

[2] % of detection, detection limit (DL): 0.01 µmol /L

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

[4] Comparison of geometric means between Oujé-Bougoumou, or Nemaska, and data for a population from Southern Quebec

Table 50 Exceedances of the concern level of urinary copper among the Oujé-Bougoumou and Nemaska participants

		> Concern level[1] of 0.5 µmol/L		
Group	**Community**	**n**	**(%)**	**p value[2]**
8-14 years	Oujé-Bougoumou	0	(0.0)	-
	Nemaska	0	(0.0)	
Women 15-39 years	Oujé-Bougoumou	3	(4.1)	1.000
	Nemaska	1	(2.4)	
Men 15-39 years	Oujé-Bougoumou	0	(0.0)	0.273
	Nemaska	1	(6.7)	
Men & women ≥40 years	Oujé-Bougoumou	3	(6.0)	1.000
	Nemaska	1	(7.7)	

[1] Unpublished data: INSPQ, Leblanc et al., 2003; corresponds to the upper end of the laboratory reference range.

[2] p value corresponds to the Fisher exact test for comparison of proportions.

Table 51 Hair (0-2 cm) concentrations of total arsenic (nmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean (sd)	Geometric mean (95% CI)		p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	20	90.0	2.45 (3.69)	1.20	(0.69-2.11)	0.547	<DL	0.25	1.20	4.27	16.95
	Nemaska	11	81.8	1.49 (1.19)	0.90	(0.42-1.90)		<DL	<DL	1.20	3.20	3.47
Women	Oujé-Bougoumou	78	78.2	0.78 (2.29)	0.34	(0.27-0.44)	0.659	<DL	<DL	0.27	1.20	19.76
15-39 years	Nemaska	42	71.4	0.54 (0.38)	0.38	(0.28-0.50)		<DL	<DL	0.53	1.07	1.34
Men	Oujé-Bougoumou	31	87.1	1.82 (2.43)	0.93	(0.60-1.45)	0.413	<DL	<DL	1.34	3.87	12.28
15-39 years	Nemaska	12	83.3	0.95 (0.68)	0.67	(0.37-1.20)		<DL	<DL	0.73	1.87	2.27
Men & women	Oujé-Bougoumou	51	76.5	2.11 (2.48)	0.88	(0.58-1.34)	0.146	<DL	<DL	1.20	5.07	11.08
≥40 years	Nemaska	13	69.2	0.74 (0.62)	0.45	(0.24-0.85)		<DL	<DL	0.53	1.74	1.87
Total	Oujé-Bougoumou	180	80.6	1.52 (1.31)	0.61	(0.50-0.75)	0.227	<DL	<DL	0.67	3.87	19.76
(≥8 years)	Nemaska	78	74.4	0.82 (0.37)	0.50	(0.39-0.64)		<DL	<DL	0.67	1.87	3.47

[1] The suggested level of concern is >4.0 nmol/g (>0.30 µg/g) (CTQ, 2003); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 0.1 nmol/g

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 52 Hair (0-2 cm) concentrations of cadmium (nmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean[3] (sd)	Geometric mean[3] (95% CI)		p value[4]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	20	100.0	0.645 (0.776)	0.378	(0.239-0.598)	0.436	0.089	0.089	0.311	1.512	3.292
	Nemaska	11	72.7	0.486 (0.513)	0.273	(0.136-0.549)		<DL	<DL	0.267	1.157	1.512
Women	Oujé-Bougoumou	78	66.7	0.709 (4.396)	0.148	(0.117-0.186)	-	<DL	<DL	0.089	0.445	38.969
15-39 years	Nemaska	42	38.1	-	-	-		<DL	<DL	<DL	0.178	0.890
Men	Oujé-Bougoumou	31	93.5	0.397 (0.321)	0.286	(0.211-0.387)	0.697	<DL	0.089	0.267	0.890	1.246
15-39 years	Nemaska	12	83.3	0.589 (1.044)	0.251	(0.125-0.505)		<DL	<DL	0.222	1.246	3.737
Men & women	Oujé-Bougoumou	51	82.4	0.674 (0.845)	0.353	(0.256-0.487)	-	<DL	<DL	0.356	1.690	3.381
≥40 years	Nemaska	13	53.8	-	-	-		<DL	<DL	0.178	2.313	2.847
Total	Oujé-Bougoumou	180	79.4	0.641 (1.438)	0.233	(0.198-0.274)	-	<DL	<DL	0.178	1.068	38.969
(≥8 years)	Nemaska	78	52.6	-	-	-		<DL	<DL	0.089	1.068	3.737

[1] The suggested level of concern is >4.4 nmol/g (>0.5 µg/g); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 0.085 nmol/g

[3] Means calculated when the detection frequency was at least 60%

[4] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 53 Hair (0-2 cm) concentrations of copper (µmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean (sd)	Geometric mean (95% CI)		p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	20	100.0	0.187 (0.053)	0.181	(0.160-0.204)	0.194	0.100	0.137	0.174	0.270	0.304
	Nemaska	11	81.8	0.172 (0.128)	0.057	(0.011-0.289)		<DL	<DL	0.161	0.259	0.481
Women	Oujé-Bougoumou	78	100.0	0.249 (0.208)	0.219	(0.199-0.241)	<0.001	0.102	0.142	0.217	0.348	1.898
15-39 years	Nemaska	42	76.2	0.156 (0.149)	0.037	(0.015-0.089)		<DL	<DL	0.146	0.260	0.875
Men	Oujé-Bougoumou	31	100.0	0.190 (0.056)	0.183	(0.166-0.202)	0.674	0.108	0.135	0.181	0.256	0.381
15-39 years	Nemaska	12	100.0	0.199 (0.072)	0.191	(0.162-0.224)		0.137	0.142	0.182	0.249	0.408
Men & women	Oujé-Bougoumou	51	100.0	0.208 (0.123)	0.190	(0.172-0.211)	0.086	0.111	0.138	0.172	0.306	0.887
≥40 years	Nemaska	13	76.9	0.151 (0.091)	0.041	(0.008-0.205)		<DL	<DL	0.176	0.238	0.254
Total	Oujé-Bougoumou	180	100.0	0.220 (0.075)	0.200	(0.189-0.211)	<0.001	0.100	0.138	0.182	0.320	1.898
(≥8 years)	Nemaska	78	80.8	0.164 (0.062)	0.052	(0.029-0.094)		<DL	<DL	0.167	0.259	0.875

[1] The suggested level of concern is >0.63 µmol/g (>40 µg/g) (CTQ, 2003); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 0.0003 µmol/g

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 54 Hair (0-2 cm) concentrations of lead (nmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean (sd)	Geometric mean (95% CI)	p value[3]	Mini-mum	Percentiles 10th	50th	90th	Maxi-mum
8-14 years	Oujé-Bougoumou	20	100.0	21.86 (55.03)	4.81 (2.35-9.85)	0.052	0.24	0.84	3.67	59.14	241.98
	Nemaska	11	81.8	2.95 (2.40)	1.28 (0.41-3.98)		<DL	<DL	2.61	6.03	6.32
Women	Oujé-Bougoumou	78	100.0	1.97 (3.16)	1.33 (1.13-1.57)	<0.001	0.29	0.58	1.30	2.94	25.05
15-39 years	Nemaska	42	76.2	0.99 (1.15)	0.43 (0.27-0.69)		<DL	<DL	0.63	2.32	5.12
Men	Oujé-Bougoumou	31	100.0	5.27 (6.62)	3.32 (2.40-4.59)	0.056	0.77	1.25	2.70	11.97	34.55
15-39 years	Nemaska	12	100.0	2.77 (3.37)	1.81 (1.10-2.96)		0.72	0.82	1.66	6.08	12.35
Men & women	Oujé-Bougoumou	51	100.0	6.18 (8.35)	3.63 (2.75-4.80)	0.175	0.34	1.06	3.38	13.66	47.15
≥40 years	Nemaska	13	76.9	7.63 (14.71)	1.37 (0.37-5.04)		<DL	<DL	2.08	17.90	53.52
Total	Oujé-Bougoumou	180	100.0	7.02 (11.88)	2.39 (2.02-2.83)	<0.001	0.24	0.68	1.93	10.81	241.98
(≥8 years)	Nemaska	78	80.8	2.67 (3.09)	0.79 (0.53-1.17)		<DL	<DL	0.92	5.74	53.52

[1] The suggested level of concern is >26.5 nmol/g (>5.5 µg/g); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 0.05 nmol/g

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 55 Hair (0-2 cm) concentrations of total mercury (nmol/g) among the of Oujé-Bougoumou and Nemaska participants

Group	Community	n	% of det.[1]	Mean[2] (sd)	Geometric mean[2] (95% CI)	p value[3]	Mini-mum	Percentiles 10th	50th	90th	Maxi-mum
0-14 years	Oujé-Bougoumou	54	63.0	3.31 (4.16)	1.89 (1.44-2.47)	-	<DL	<DL	1.50	7.98	17.45
	Nemaska	29	41.4	-	-		<DL	<DL	<DL	3.99	7.48
Women	Oujé-Bougoumou	78	80.8	5.43 (6.52)	3.10 (2.45-3.94)	-	<DL	<DL	3.49	13.96	36.89
15-39 years	Nemaska	42	57.1	-	-		<DL	<DL	1.00	5.48	13.46
Men	Oujé-Bougoumou	35	97.1	9.41 (8.50)	6.46 (4.78-8.74)	0.081	<DL	1.99	5.98	21.93	34.90
15-39 years	Nemaska	13	92.3	6.65 (7.47)	3.67 (1.97-6.83)		<DL	1.00	2.49	18.94	20.94
Men & women	Oujé-Bougoumou	51	98.0	24.31 (18.04)	16.40 (12.26-21.94)	0.397	<DL	4.49	19.44	53.84	69.29
≥40 years	Nemaska	13	100.0	16.53 (11.96)	12.52 (8.00-19.60)		3.49	3.49	13.96	32.40	43.87
Total	Oujé-Bougoumou	218	83.0	8.05 (5.76)	3.67 (3.10-4.34)	<0.001	<DL	<DL	3.99	21.44	69.29
	Nemaska	97	62.9	4.35 (3.48)	2.05 (1.63-2.57)		<DL	<DL	1.50	13.46	43.87

[1] Detection limit (DL): 1 nmol/g

[2] Means calculated when the percentage of detection was at least 60%

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 56 Hair (0-2 cm) concentrations of total mercury (µg/g) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% of det.[1]	Mean[2] (sd)	Geometric mean[2] (95% CI)	p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
0-14 years	Oujé-Bougoumou	54	63.0	0.66 (0.83)	0.38 (0.29-0.50)	0.037	<DL	<DL	0.30	1.60	3.50
	Nemaska	29	41.4	-	-		<DL	<DL	<DL	0.80	1.50
Women	Oujé-Bougoumou	78	80.8	1.09 (1.31)	0.62 (0.49-0.79)	0.003	<DL	<DL	0.70	2.80	7.40
15-39 years	Nemaska	42	57.1	-	-		<DL	<DL	0.20	1.10	2.70
Men	Oujé-Bougoumou	35	97.1	1.89 (1.70)	1.30 (0.96-1.75)	0.081	<DL	0.40	1.20	4.40	7.00
15-39 years	Nemaska	13	92.3	1.33 (1.50)	0.74 (0.40-1.37)		<DL	0.20	0.50	3.80	4.20
Men & women	Oujé-Bougoumou	51	98.0	4.88 (3.62)	3.29 (2.46-4.40)	0.397	<DL	0.90	3.90	10.80	13.90
≥40 years	Nemaska	13	100.0	3.32 (2.40)	2.51 (1.61-3.93)		0.70	0.70	2.80	6.50	8.80
Total	Oujé-Bougoumou	218	83.0	1.61 (1.16)	0.74 (0.62-0.87)	<0.001	<DL	<DL	0.80	4.30	13.90
	Nemaska	97	62.9	0.87 (0.70)	0.41 (0.33-0.52)		<DL	<DL	0.30	2.70	8.80

[1] Detection limit (DL): 0.2 µg/g

[2] Means calculated when the percentage of detection was at least 60%

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 57 Exceedances of the concern and action levels of hair mercury (0-2 cm) among the Oujé-Bougoumou and Nemaska participants

Group	Community	Categories of hair mercury[1] <6 ppm[2] n	(%)	6-14 ppm n	(%)	≥15 ppm n	(%)	p value[3]
0-14 years	Oujé-Bougoumou	54	(100.0)	0	(0.0)	0	(0.0)	-
	Nemaska	29	(100.0)	0	(0.0)	0	(0.0)	
Women 15-39 years	Oujé-Bougoumou	77	(98.7)	1	(1.3)	0	(0.0)	1.000
	Nemaska	42	(100.0)	0	(0.0)	0	(0.0)	
Men 15-39 years	Oujé-Bougoumou	33	(94.3)	2	(5.7)	0	(0.0)	1.000
	Nemaska	13	(100.0)	0	(0.0)	0	(0.0)	
Men & women ≥40 years	Oujé-Bougoumou	34	(66.7)	17	(33.3)	0	(0.0)	0.312
	Nemaska	11	(84.6)	2	(15.4)	0	(0.0)	

[1] Source: Dumont et al., 1988 (also see Table 63, Appendix 6 and the mercury toxicological profile in Appendix 1); 6-14 ppm constitutes the concern level and ≥15 ppm the action level (see text)

[2] 6 ppm=29.9 nmol/g and 15 ppm=74.78 nmol/g

[3] p value corresponds to the Fisher exact test for comparison of proportions.

Table 58 Hair (4-5 cm) concentrations of total mercury (nmol/g) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% of det.[1]	Mean[2] (sd)	Geometric mean[2] (95% CI)	p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
0-14 years	Oujé-Bougoumou	27	51.9	-	- -	0.902	<DL	<DL	1.00	3.49	13.96
	Nemaska	18	55.6	-	- -		<DL	<DL	1.25	5.48	7.98
Women	Oujé-Bougoumou	77	70.1	3.36 (4.71)	2.04 (1.65-2.51)	0.478	<DL	<DL	1.99	6.48	31.90
15-39 years	Nemaska	42	66.7	2.64 (2.48)	1.80 (1.38-2.34)		<DL	<DL	1.50	5.98	10.97
Men	Oujé-Bougoumou	13	76.9	9.94 (12.00)	4.64 (2.18-9.88)	0.727	<DL	<DL	4.49	32.40	36.89
15-39 years	Nemaska	4	75.0	6.78 (7.33)	3.48 (0.83-14.68)		<DL	<DL	4.99	16.45	16.45
Men & women	Oujé-Bougoumou	38	97.4	17.73 (14.81)	11.77 (8.51-16.27)	0.738	<DL	2.99	13.71	36.89	62.31
≥40 years	Nemaska	10	100.0	16.15 (14.30)	10.39 (5.34-20.19)		1.99	2.49	14.96	39.88	43.87
Total	Oujé-Bougoumou	155	74.2	5.88 (4.69)	2.60 (2.15-3.15)	0.221	<DL	<DL	2.49	15.95	62.31
	Nemaska	74	68.9	4.27 (3.51)	2.13 (1.66-2.72)		<DL	<DL	1.99	8.47	43.87

[1] Detection limit (DL): 1 nmol/g

[2] Means calculated when the percentage of detection was at least 60%

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 59 Hair (4-5 cm) concentrations of total mercury (µg/g) among the Oujé-Bougoumou and Nemaska participants

Group	Community	n	% of det.[1]	Mean[2] (sd)	Geometric mean[2] (95% CI)		p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
0-14 years	Oujé-Bougoumou	27	51.9	-	-	-	0.902	<DL	<DL	0.20	0.70	2.80
	Nemaska	18	55.6	-	-	-		<DL	<DL	0.25	1.10	1.60
Women	Oujé-Bougoumou	77	70.1	0.67 (0.94)	0.41	(0.33-0.50)	0.478	<DL	<DL	0.40	1.30	6.40
15-39 years	Nemaska	42	66.7	0.53 (0.50)	0.36	(0.28-0.47)		<DL	<DL	0.30	1.20	2.20
Men	Oujé-Bougoumou	13	76.9	1.99 (2.41)	0.93	(0.44-1.98)	0.727	<DL	<DL	0.90	6.50	7.40
15-39 years	Nemaska	4	75.0	1.36 (1.47)	0.70	(0.17-2.94)		<DL	<DL	1.00	3.30	3.30
Men & women	Oujé-Bougoumou	38	97.4	3.56 (2.97)	2.36	(1.71-3.26)	0.738	<DL	0.60	2.75	7.40	12.50
≥40 years	Nemaska	10	100.0	3.24 (2.87)	2.08	(1.07-4.05)		0.40	0.50	3.00	8.00	8.80
Total	Oujé-Bougoumou	155	74.2	1.18 (0.94)	0.52	(0.43-0.63)	0.221	<DL	<DL	0.50	3.20	12.50
	Nemaska	74	68.9	0.86 (0.70)	0.43	(0.33-0.55)		<DL	<DL	0.40	1.70	8.80

[1] Detection limit (DL): 0.2 µg/g

[2] Means calculated when the percentage of detection was at least 60%

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 60 Hair concentrations of methylmercury (nmol/g) among the Oujé-Bougoumou and Nemaska participants

	Community	n[1]	Mean (sd)	n[2]	Geometric mean (95 % CI)	p value[3]
0-2 cm	Oujé-Bougoumou	23	5.43 (4.53)	18	3.99 (2.33-6.84)	0.290
	Nemaska	10	2.72 (1.77)	8	2.47 (1.38-4.41)	
4-5 cm	Oujé-Bougoumou	23	3.93 (4.50)	16	2.78 (1.53-5.02)	0.263
	Nemaska	10	2.38 (1.82)	9	1.67 (0.96-2.90)	

[1] n for arithmetic mean

[2] n for geometric mean (values of 0 are excluded)

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 61 Hair (0-2 cm) concentrations of selenium (nmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean (sd)	Geometric mean (95% CI)	p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	20	100.0	19.62 (27.52)	13.96 (10.39-18.76)	0.074	7.60	8.23	11.39	30.38	132.93
	Nemaska	11	81.8	11.21 (7.99)	7.70 (3.97-14.95)		<DL	<DL	10.13	16.46	30.38
Women	Oujé-Bougoumou	78	100.0	9.84 (11.95)	8.43 (7.69-9.25)	0.012	3.80	6.33	7.60	12.66	111.41
15-39 years	Nemaska	42	76.2	7.87 (5.31)	5.41 (3.93-7.45)		<DL	<DL	8.86	12.66	27.85
Men	Oujé-Bougoumou	31	100.0	12.74 (4.26)	12.09 (10.76-13.59)	0.690	5.06	8.86	11.39	16.46	25.32
15-39 years	Nemaska	12	100.0	13.19 (4.33)	12.64 (10.69-14.94)		8.86	8.86	12.03	16.46	24.05
Men & women	Oujé-Bougoumou	51	100.0	20.31 (39.39)	14.33 (12.11-16.95)	0.040	3.80	8.86	12.66	22.79	291.18
≥40 years	Nemaska	13	76.9	10.04 (6.50)	6.62 (3.48-12.59)		<DL	<DL	8.86	17.72	18.99
Total	Oujé-Bougoumou	180	100.0	14.24 (11.09)	10.91 (10.06-11.84)	<0.001	3.80	6.33	10.13	18.99	291.18
(≥8 years)	Nemaska	78	80.8	9.64 (3.01)	6.77 (5.37-8.53)		<DL	<DL	8.86	16.46	30.38

[1] The suggested level of concern is >20.3 nmol/g (>1.6 µg/g) (CTQ, 2003); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 1 nmol/g

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 62 Hair (0-2 cm) concentrations of zinc (μmol/g) among the Oujé-Bougoumou and Nemaska participants[1]

Group	Community	n	% of det.[2]	Mean (sd)	Geometric mean (95% CI)	p value[3]	Minimum	Percentiles 10th	50th	90th	Maximum
8-14 years	Oujé-Bougoumou	20	100.0	2.284 (0.545)	2.220 (1.991-2.475)	0.261	1.346	1.492	2.341	3.052	3.381
	Nemaska	11	81.8	2.304 (1.219)	0.893 (0.200-3.980)		<DL	<DL	2.739	3.320	3.703
Women 15-39 years	Oujé-Bougoumou	78	100.0	2.694 (0.547)	2.645 (2.535-2.760)	0.001	1.438	2.188	2.624	3.198	5.156
	Nemaska	42	76.2	2.130 (1.379)	0.615 (0.273-1.387)		<DL	<DL	2.440	3.534	4.743
Men 15-39 years	Oujé-Bougoumou	31	100.0	2.960 (0.939)	2.815 (2.504-3.164)	0.887	0.995	1.943	2.800	4.100	5.722
	Nemaska	12	100.0	2.947 (0.737)	2.858 (2.462-3.318)		1.591	2.463	2.678	3.902	4.192
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	2.558 (0.774)	2.441 (2.238-2.663)	0.121	1.132	1.637	2.555	3.290	5.202
	Nemaska	13	76.9	2.282 (1.416)	0.679 (0.151-3.048)		<DL	<DL	2.861	3.596	3.825
Total (≥8 years)	Oujé-Bougoumou	180	100.0	2.633 (0.334)	2.544 (2.447-2.645)	<0.001	0.995	1.790	2.586	3.381	5.722
	Nemaska	78	80.8	2.306 (0.627)	0.840 (0.484-1.456)		<DL	<DL	2.601	3.703	4.743

[1] The suggested level of concern is >4.6 μmol/g (>300 μg/g) (CTQ, 2003); corresponds to the upper end of the laboratory reference range.

[2] Detection limit (DL): 0.007 μmol/g

[3] Comparison of geometric means between Oujé-Bougoumou and Nemaska

Table 63 Concern and action levels for the major contaminants, number of people affected and actions to be taken[1]

Contaminant	Level of concern[2]	Number of exceedances	Action[3]	Action level[2]	Number of exceedances	Action[3]
Arsenic (U) (non-dietary; i.e., inorganic including metabolites)	0.25 µmol/L	2 (OJ) 3 (N)	VR of sources	0.47 µmol/L	None	Interview and retest
Cadmium (WB)	5.0 nmol/L (NS)	27 (OJ); 2 (N)	VR of secondary smoke	44.5 nmol/L	14 (OJ); 12 (N)	Interview
	25 nmol/L (S)	58 (0J); 32 (N)	VR of smoking			
Cadmium (U)	10.0 nmol/L (NS)	7 (OJ)	VR of secondary smoke	47.4 nmol/L	none	Interview
	35.6 nmol/L (S)	none	VR of smoking			
Lead (WB)	0.48 µmol/L (children & women of RA)	2 (OJ)	VR	0.96 µmol/L (children & women of RA)	1 (OJ)	Interview and retest
	0.48 µmol/L (all men; women ≥40)	5(OJ); 4(N)	VR	0.96 µmol/L (all men & women >40)	none	
Mercury (WB)	75 nmol/L (declarable in Quebec)	30 (OJ); 7 (N)	VR	100 nmol/L (children & women of RA)	1 (OJ); 1 (N)	Interview and retest
				500 nmol/L (all men and women >40)	1 (N)	Interview and retest
Mercury (H)	6-14 µg/g	20 (OJ) 2 (N)	VR	>15 µg/g	none	Interview and retest

Table 63 (continued) Concern and action levels for major contaminants, number of people affected and actions to be taken[1]

Contaminant	Level of Concern[2]	Number of exceedances	Action[3]	Action level[2]	Number of exceedances	Action[3]
Copper (P)	>25.2 µmol/L	11 (OJ) 4 (N) (all female 15-39)	Check questionnaire if pregnant or use of oral contraceptive	>47.4 nmol/L (if not pregnant or not on oral contraceptive nor estrogen therapy)	2 (OJ & N) (females 15-39)	Check for infections, inflammations, other prescription drugs. Confirm by other tests of copper status (plasma ceruloplasmin & erythrocyte SOD activity?)
Selenium (P)	>2 µmol/L	1 (OJ)	VR	>3 µmol/L (child)	none	Check for selenosis and source; interview
Zinc (P)	>22 µmol/L	1 (OJ) 1 (N)	VR	≥30 µmol/L	none	Retest; check for reduced HDL & Cu, anemia; interview
PCBs	5 µg/L (children & women of RA)	23 (OJ); 10 (N)	VR	100 µg/L	10 (OJ); 1 (N)	Interview; repeat liver enzyme tests; other hormone tests than TSH and T4 (FSH, LH, prolactin, T3, testosterone)
	20 µg/L (all men & women >40)	40 (OJ) 9 (N)	VR			

[1] Abbreviations: U, urine; P, plasma; WB, whole blood; H, hair; OJ, Oujé-Bougoumou; N, Nemaska; VR, voluntary review; RA, reproductive age.

[2] Sources for the concern and action levels are given in the contaminant specific tables: 28 (PCBs); 32 and 48 (cadmium); 35 and 57 (mercury); 37 (lead); 39 (copper); 41 (selenium); 42 (zinc).

[3] VR, voluntary review; interview implies that the person would be visited by a health worker or would be consulted by his/her doctor.

Table 64 Associations between intakes of wild fish, wildfowl and game, and concentrations of contaminants among the Oujé-Bougoumou and Nemaska participants

Log (Concentration)	Community	n	Wild fish r (p value)[1]	Wildfowl r (p value)[1]	Game r (p value)[1]
Whole blood cadmium among non-smokers(≥15 years)	Oujé-Bougoumou	32	0.427 (0.015)	0.150 (0.413)	0.346 (0.053)
	Nemaska	5	-	-	-
Urinary cadmium among non-smokers (≥15 years)	Oujé-Bougoumou	31	-0.007 (0.972)	-0.070 (0.708)	0.051 (0.785)
	Nemaska	5	-	-	-
Whole blood mercury	Oujé-Bougoumou	190	0.361 (<0.001)	0.219 (0.002)	0.386 (<0.001)
	Nemaska	82	0.525 (<0.001)	0.399 (<0.001)	0.493 (<0.001)
Hair total mercury (0-2 cm)	Oujé-Bougoumou	218	0.368 (<0.001)	0.242 (<0.001)	0.453 (<0.001)
	Nemaska	97	0.535 (<0.001)	0.431 (<0.001)	0.480 (<0.001)
Hair total mercury (4-5 cm)	Oujé-Bougoumou	155	0.360 (<0.001)	0.365 (<0.001)	0.413 (<0.001)
	Nemaska	74	0.413 (<0.001)	0.291 (0.012)	0.325 (0.005)
Whole blood lead	Oujé-Bougoumou	220	0.173 (0.010)	0.148 (0.029)	0.246 (<0.001)
	Nemaska	98	0.461 (<0.001)	0.254 (0.012)	0.406 (<0.001)
Plasma selenium	Oujé-Bougoumou	189	0.164 (0.024)	0.084 (0.251)	0.109 (0.135)
	Nemaska	82	0.163 (0.147)	-0.124 (0.269)	0.003 (0.977)
Plasma zinc	Oujé-Bougoumou	189	0.061 (0.405)	-0.100 (0.173)	-0.028 (0.740)
	Nemaska	82	0.023 (0.838)	0.029 (0.797)	0.027 (0.810)
Urinary inorganic arsenic	Oujé-Bougoumou	185	0.061 (0.409)	0.098 (0.185)	-0.074 (0.318)
	Nemaska	81	-0.113 (0.317)	-0.137 (0.221)	-0.194 (0.083)
Urinary total arsenic	Oujé-Bougoumou	185	0.059 (0.426)	0.027 (0.714)	-0.052 (0.479)
	Nemaska	81	-0.118 (0.296)	-0.074 (0.509)	-0.096 (0.393)
Total PCBs (measured as Aroclor 1260)	Oujé-Bougoumou	190	0.161 (0.026)	0.084 (0.249)	0.313 (<0.001)
	Nemaska	81	0.475 (<0.001)	0.243 (0.028)	0.381 (<0.001)

[1] Pearson's correlation coefficient

Table 65 Concentrations of contaminants according to different risk factors among the Cree population of Oujé-Bougoumou

Concentration (µmol/L)	Risk factor	n	% of det.	Mean (sd)	Geometric mean (95% CI)	p value[1]
Whole blood lead	Practice of hunting activities					
	Yes	78	100.0	0.198 (0.069)	0.156 (0.133-0.182)	<0.001
	No	142	100.0	0.114 (0.078)	0.083 (0.074-0.093)	
	If yes use of firearm					
	Yes	71	100.0	0.200 (0.069)	0.157 (0.133-0.185)	0.710
	No	7	100.0	0.183 (0.071)	0.142 (0.081-0.247)	
	Smoking status					
	Non-smoker	32	100.0	0.110 (0.045)	0.084 (0.066-0.107)	0.040
	Ex-smoker	42	100.0	0.182 (0.068)	0.133 (0.105-0.170)	
	Smoker	95	100.0	0.145 (0.092)	0.097 (0.083-0.115)	
	Drink tap water					
	No	71	100.0	0.157 (0.072)	0.110 (0.090-0.134)	0.245
	Yes	149	100.0	0.131 (0.079)	0.096 (0.086-0.108)	
	Drink water from a spring					
	No	175	100.0	0.133 (0.079)	0.096 (0.086-0.107)	0.036
	Yes	44	100.0	0.175 (0.068)	0.128 (0.101-0.162)	
	Drink water from a lake/river					
	No	74	100.0	0.137 (0.059)	0.104 (0.089-0.123)	0.645
	Yes	145	100.0	0.141 (0.085)	0.010 (0.087-0.113)	
Plasma zinc	Drink tap water					
	No	60	100.0	13.57 (1.045)	13.39 (12.84-13.96)	0.734
	Yes	129	100.0	13.45 (1.086)	13.27 (12.90-13.65)	
	Drink water from a spring					
	No	147	100.0	13.40 (1.058)	13.23 (12.89-13.58)	0.335
	Yes	42	100.0	13.83 (1.115)	13.62 (12.91-14.37)	
	Drink water from a lake/river					
	No	64	100.0	13.45 (1.199)	13.21 (12.62-13.84)	0.688
	Yes	125	100.0	13.50 (1.004)	13.35 (13.00-13.71)	
Urinary inorganic arsenic	Drink tap water					
	No	59	94.9	0.083 (0.026)	0.066 (0.055-0.080)	0.623
	Yes	126	97.6	0.085 (0.026)	0.070 (0.062-0.079)	
	Drink water from a spring					
	No	142	96.5	0.085 (0.028)	0.068 (0.061-0.077)	0.906
	Yes	43	97.7	0.081 (0.020)	0.069 (0.058-0.084)	
	Drink water from a lake/river					
	No	63	95.2	0.078 (0.021)	0.064 (0.054-0.076)	0.376
	Yes	122	97.5	0.088 (0.029)	0.071 (0.062-0.080)	
Urinary total arsenic	Drink tap water					
	No	59	52.5	- -	- --	0.448
	Yes	126	54.0	- -	- --	
	Drink water from a spring					
	No	142	52.8	- -	- --	0.687
	Yes	43	55.8	- -	- --	
	Drink water from a lake/river					
	No	63	50.8	- -	- --	0.976
	Yes	122	54.9	- -	- --	

[1] p value corresponds to the chi-square test for comparison of proportions.

Table 66 Concentrations of contaminants according to different risk factors among the Cree population of Nemaska

Concentration (µmol/L)	Risk factor	n	% of det.	Mean (sd)	Geometric mean (95% CI)		p value[1]
Whole blood lead	Practice of hunting activities						
	Yes	27	100.0	0.160 (0.065)	0.119	(0.089-0.161)	0.014
	No	71	100.0	0.109 (0.065)	0.078	(0.066-0.092)	
	If yes. use of firearm						
	Yes	26	100.0	0.165 (0.065)	0.127	(0.096-0.168)	-
	No	1	100.0	-	-	-	
	Smoking status						
	Non-smoker	5	100.0	0.168 (0.051)	0.138	(0.073-0.263)	<0.001
	Ex-smoker	20	100.0	0.228 (0.087)	0.172	(0.122-0.243)	
	Smoker	46	100.0	0.112 (0.060)	0.077	(0.062-0.097)	
	Drink tap water						
	No	9	100.0	0.087 (0.027)	0.072	(0.046-0.112)	0.446
	Yes	89	100.0	0.126 (0.068)	0.089	(0.075-0.104)	
	Drink water from a spring						
	No	82	100.0	0.126 (0.069)	0.088	(0.075-0.105)	0.608
	Yes	16	100.0	0.103 (0.044)	0.079	(0.056-0.113)	
	Drink water from a lake/river						
	No	17	100.0	0.074 (0.032)	0.062	(0.048-0.082)	0.033
	Yes	81	100.0	0.135 (0.069)	0.094	(0.079-0.112)	
Plasma zinc	Drink tap water						
	No	7	100.0	13.30 (0.752)	13.23	(12.16-14.38)	0.274
	Yes	75	100.0	12.59 (0.956)	12.46	(12.07-12.86)	
	Drink water from a spring						
	No	70	100.0	12.71 (0.976)	12.58	(12.17-13.00)	0.483
	Yes	12	100.0	12.27 (0.709)	12.20	(11.43-13.02)	
	Drink water from a lake/river						
	No	9	100.0	12.13 (0.514)	12.09	(11.41-12.80)	0.403
	Yes	73	100.0	12.72 (0.979)	12.58	(12.18-13.00)	
Urinary inorganic arsenic	Drink tap water						
	No	7	100.0	0.111 (0.040)	0.084	(0.045-0.159)	0.855
	Yes	74	97.3	0.108 (0.034)	0.089	(0.076-0.104)	
	Drink water from a spring						
	No	70	97.1	0.111 (0.035)	0.092	(0.079-0.108)	0.147
	Yes	11	100.0	0.086 (0.029)	0.066	(0.041-0.108)	
	Drink water from a lake/river						
	No	8	100.0	0.087 (0.022)	0.073	(0.045-0.120)	0.421
	Yes	73	97.3	0.111 (0.036)	0.090	(0.077-0.106)	
Urinary total arsenic	Drink tap water						
	No	7	85.7	0.332 (0.207)	0.217	(0.112-0.420)	0.298
	Yes	74	81.1	0.316 (0.620)	0.161	(0.137-0.189)	
	Drink water from a spring						
	No	70	82.9	0.344 (0.639)	0.170	(0.143-0.203)	0.302
	Yes	11	72.7	0.146 (0.028)	0.133	(0.101-0.176)	
	Drink water from a lake/river						
	No	8	75.0	0.132 (0.023)	0.124	(0.094-0.163)	0.220
	Yes	73	82.2	0.339 (0.626)	0.171	(0.144-0.202)	

[1] p value corresponds to the chi-square test for comparison of proportions.

Table 67 Mean concentrations of contaminants among Cree populations of Oujé-Bougoumou and Nemaska

		Crude[1]			Adjusted[2]		
Concentration	**Community**	**Geometric mean (95% CI)**		**p value[3]**	**Geometric mean (95% CI)**		**p value[4]**
Whole blood mercury (nmol/L)	Oujé-Bougoumou	17.8	(15.2-20.9)	0.003	19.1	(16.6-21.9)	0.002
	Nemaska	11.2	(8.4-15.1)		13.0	(10.6-15.9)	
Hair total mercury (0-2 cm) (nmol/g)	Oujé-Bougoumou	3.67	(3.10-4.34)	<0.001	4.78	(4.18-5.47)	<0.001
	Nemaska	2.05	(1.63-2.57)		2.92	(2.40-3.56)	
Hair total mercury (4-5 cm) (nmol/g)	Oujé-Bougoumou	2.60	(2.15-3.15)	0.221	3.27	(2.74-3.90)	0.591
	Nemaska	2.13	(1.66-2.72)		3.05	(2.39-3.88)	
Hair methyl mercury (0-2 cm) (µmol/g)	Oujé-Bougoumou	3.99	(2.33-6.84)	0.290	4.80	(2.59-8.91)	0.895
	Nemaska	2.47	(1.38-4.41)		4.57	(2.07-10.12)	
Hair methyl mercury (4-5 cm) (µmol/g)	Oujé-Bougoumou	2.78	(1.53-5.02)	0.263	4.34	(2.56-7.37)	0.941
	Nemaska	1.67	(0.96-2.90)		4.44	(2.33-8.48)	
Whole blood lead[5] (µmol/L)	Oujé-Bougoumou	0.100	(0.091-0.111)	0.115	0.113	(0.099-0.129)	0.889
	Nemaska	0.087	(0.075-0.101)		0.115	(0.094-0.140)	
Plasma selenium (µmol/L)	Oujé-Bougoumou	1.43	(1.40-1.45)	0.001	1.43	(1.41-1.45)	0.003
	Nemaska	1.50	(1.47-1.54)		1.51	(1.47-1.55)	
Plasma zinc (µmol/L)	Oujé-Bougoumou	13.31	(13.00-13.62)	0.003	13.51	(13.20-13.81)	0.003
	Nemaska	12.52	(12.16-12.90)		12.72	(12.30-13.16)	
Urinary inorganic arsenic (µmol/L)	Oujé-Bougoumou	0.069	(0.062-0.076)	0.007	0.066	(0.059-0.073)	0.009
	Nemaska	0.088	(0.076-0.103)		0.084	(0.072-0.098)	
Urinary total arsenic (µmol/L)	Oujé-Bougoumou		-	-		-	-
	Nemaska	0.165	(0.141-0.193)		0.155	(0.132-0.182)	
Total PCBs (measured as Aroclor 1260) (µg/kg lipids)	Oujé-Bougoumou	573.3	(449.5-731.3)	0.037	615.2	(522.1-724.9)	0.020
	Nemaska	362.4	(258.6-507.8)		438.7	(343.4-560.6)	

[1] Adjusted for age and sex

[2] Adjusted for age, sex and consumption of wild fish, wildfowl and game

[3] p value corresponds to the Student's t test

[4] p value corresponds to analysis of variance

[5] Adjusted for age, sex and consumption of wild fish, wildfowl and game, participation in hunting, smoking status and consumption of water from a spring or a lake/river

FIGURES

Figure 1 Plasma concentrations of chlorinated pesticides among Cree populations of Oujé- Bougoumou and Nemaska (Quebec, Canada, 2003)

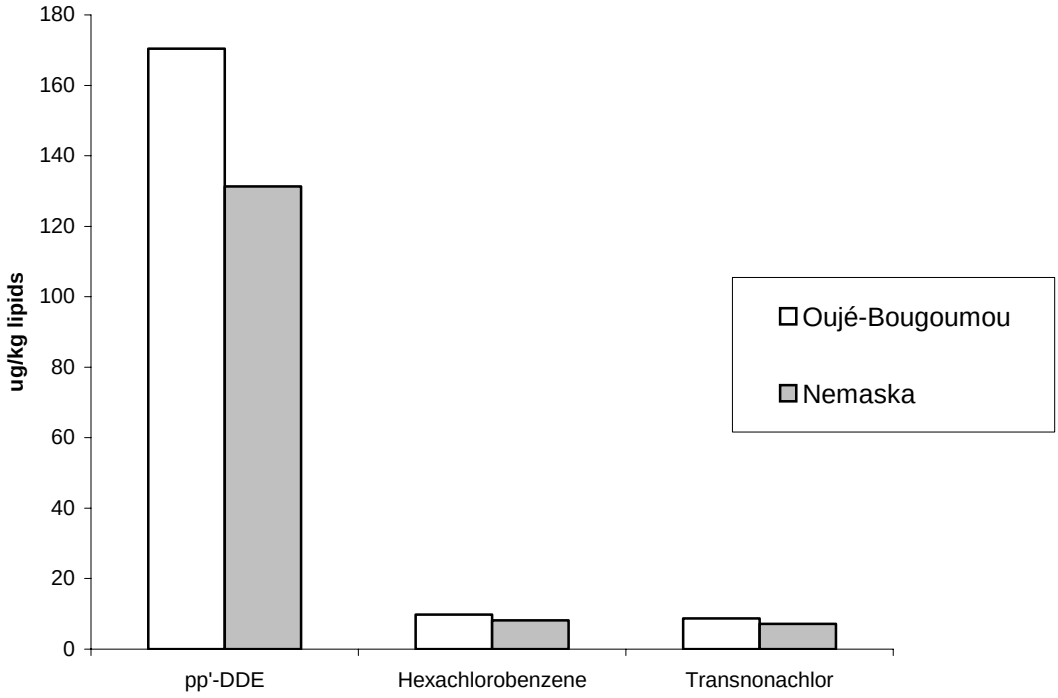
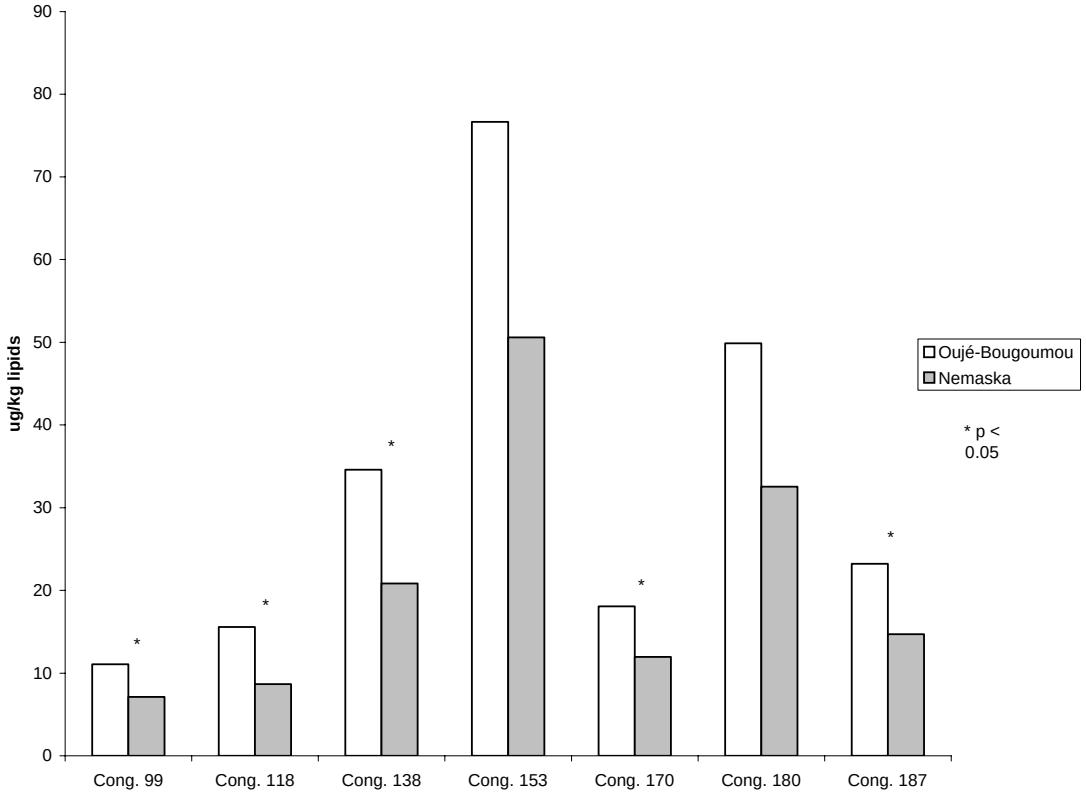


Figure 2 Plasma concentrations of PCB congeners among Cree populations of Oujé-Bougoumou and Nemaska (Quebec, Canada, 2003)



APPENDIX 1

Toxicological Profiles and Related Health Issues for Arsenic, Cadmium, Copper, Lead, Mercury, Polychlorinated Biphenyls (PCBs), Selenium and Zinc[2]

[2] July 24, 2003, Revised: May 21, 2004

Abridged Toxicological Profiles and Related Health Issues: Inorganic Arsenic (for Physicians)

Revised May 19, 2004

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Inorganic Arsenic

Public Health Issues (for high level, chronic ingestion)
- Skin and internal cancers
- Skin lesions
- Neurological outcomes
- Cardiovascular disease
- Gastrointestinal disorders

Chronic Occupational Health Issues
- Lung cancer and cancers at other sites
- Cardiovascular disease
- Neurological outcomes

Exposure Guidelines

Occupational (total aerosols; 8-hr TLV-TWA)

• Arsenic, elemental and inorganic compounds, as As	0.01 mg/m3

General Public

• Oral Reference Dose (US EPA) (non-carcinogenic effects)	0.3 μgAs/kg/day
• Oral Risk-specific Dose (US EPA) (cancer; risk of 10^{-6})	0.00067 μgAs/kg/day
• Oral Risk-specific Soil Quality Guideline (CCME) [cancer; risk of 10^{-6}(10^{-5})]	12 (31) μg/g
• WHO Provisional Tolerable Daily Intake (PTDI)	2.0 μg/kg-bw/day
• Soil Remediation Criteria (MOE) (generic, residential)	20 μg/g (25 μg/g for medium and fine textured soils)

Body Fluids Reference Values and Biological Exposure Indices
- Background Concentration
  Urine: 8±6 μg As/g creatinine (inorganic arsenic and its metabolites)

• End of workweek (inorganic arsenic plus methylated metabolites) (ACGIH)	35 μg As/L

Chronic Health Effects

Dermal Effects

Hyperkeratosis, hyperpigmentation and skin cancer have been associated with the consumption of drinking water containing inorganic arsenic. These clinicopathological findings have been documented for outbreaks in Taiwan, Japan, Argentina, Bangal, and Bangladesh (Bates et al, 1992; Chen et al, 1992; Hopenhayn-Rich et al, 1996; Mazumder et al, 1998; Smith et al, 2000). Concentrations above

50 µg/L in the drinking water were common, which is clearly in excess of the WHO maximum allowable level of 10 µg/L. Concentrations >500 µg/L were not uncommon. Similar outcomes have also been traced to the medical use of Fowler's solution during the period 1809-1950, which contained 1% of potassium arsenite (3120 µg As/L). It constituted therapy for an extensive list of diseases including nutritional disorders (e.g., anorexia), neuralgia, rheumatism, diabetes, and hematological disorders (Gorby, 1994).

Internal Cancers
Epidemiological assessments of the drinking-water arsenic contamination incidents and the medicinal use of arsenite strongly suggest that ingestion of inorganic arsenic is linked to elevated risks of bladder, kidney, lung, and liver cancers (Bates et al, 1992; Chen et al, 1992). Inhalation of arsenic trioxide (As_2O_3) in copper smelters has also been associated with enhanced respiratory cancer risks and at other sites (e.g., Axelson et al, 1978; Enterline et al, 1987, 1995; Järup and Pershagen, 1991; Bates et al, 1992). Good linear relationships between lung cancer risk (standard mortality ratios, SMRs, of 100-800) and total urinary arsenic (100-1200 µg/L, corresponding to mean air levels of 200-1500 µg/m^3) have been reported (Enterline et al, 1987); the average duration of exposure was about 20 years.

IARC (1987) designates arsenic and arsenic compounds as carcinogenic to humans (Group 1).

Neurological Outcomes
Chronic exposure to arsenic by ingestion or inhalation has been associated with peripheral neuropathy, axonal degeneration and encephalopathy (Gorby, 1994; Lagerkvist and Zetterlund, 1994; Morton and Caron, 1989; Lewis, 1997; ATSDR, 1993, 2000). It appears that neurological outcomes are not seen for arsenic intakes $\leq$10 µg/kg/day (ATSDR, 1993, 2000).

Cardiovascular Disease
Vascular disease has been associated with occupational exposure and ingestion of arsenic. Copper smelter workers exposed to arsenic trioxide appear to have had increased risk of cardiovascular disease, although this has not been consistently found (ATSDR, 1993; Engel et al, 1994). Increased vasospastic reactivity (e.g., dependence of finger blood pressure on temperature) and the presence of Raynaud's phenomenon have also been reported (Lagerkvist et al, 1986; Engel et al, 1994). Estimates of workplace exposure levels at which this occurred were 0.05-0.5 mg As/m^3 (ATSDR, 1993). By contrast, severe peripheral vascular disease with gangrene and amputations have consistently been reported with the drinking-water contamination incidents described earlier (Engel et al, 1994; Smith et al, 2000). Ingestion rates of 14 to 65 µg As/kg/day are suspected (ATSDR, 1993, 2000).

Gastrointestinal and Hepatic Symptoms
Nausea, vomiting, diarrhea, anorexia, weight loss, hepatomegaly, jaundice, pancreatitis and liver cirrhosis have been reported for chronic intake of high levels of inorganic arsenic (Gorby, 1994). Exposure levels above 10 µg As/kg/day appear to be responsible (ATSDR, 1993, 2000).

Reproductive and Developmental Outcomes
A critical review of the available data (ATSDR, 2000; IPCS, 2001; Nieboer, 2003) suggests that there is sufficient evidence to provide a strong presumption that human exposure to inorganic arsenic results in developmental toxicity in the presence of maternal toxicity. The same level of concern appears to apply to lactation. The available data (animal and human) provides no consistent evidence that inorganic arsenic compounds impair fertility.

Other Outcomes
Hematologic effects (e.g., anemia, leukopenia) appear to be common effects of chronic arsenic poisoning, while the kidney does not appear to be a major target organ (Gorby, 1994; ATSDR, 1993, 2000).

Biological Exposure Indices
Consumption of seafood can result in significant intake of arsenic because seafood contains organoarsenicals such as arsenobetaine and arsenosugars (Le et al, 1994). These compounds are considered to be nontoxic and are excreted rapidly and predominantly unchanged into the urine. Assessments of exposures to inorganic arsenic by monitoring urinary arsenic must take this into account. Consequently, chemical speciation must be considered. The major human metabolic pathway for inorganic arsenic is methylation, with dimethylarsinic acid (DMA) and monmethylarsonic acid (MMA) the major products. Consequently, these species need to be determined in urine along with unchanged inorganic arsenic consisting of arsenate and arsenite. The sum of these compounds is designated iAs-met. Typical background concentrations of iAs-met for non-exposed individuals is 8 ± 6 µg As/g creatinine (roughly equal to 10±8 µg/L) (Vahter, 1986). The recommended biological exposure index for As-met at the end of the workweek for workers is 35 µg As/L (ACGIH, 2000). The excretion half-life of organic arsenic into urine appears to be about one day, while that for inorganic arsenic is estimated to be around four days (Lauwerys and Hoet, 2001).

Hair and nail arsenic concentrations have also been used as indices of arsenic exposure (Lauwerys and Hoet, 2001). In acute arsenic poisoning cases, the distribution of this element along the length of a hair can be used to distinguish between chronic and acute exposures (see Wilhelm and Idel, 1996). The bulk of the arsenic associated with hair for both low-level and high-level exposures appears to be inorganic arsenic (Yamauchi et al., 1989). However, exogenously accumulated arsenic is difficult to distinguish from that secreted into the hair.Because of this uncertainty, the fact that measurable increases in hair arsenic have not been well defined in terms of intake or exposure, and its apparent dependence on gender, race and dietary habits, the consensus is that hair arsenic analysis is only suitable as a screening method on a group basis (Sky-Peck, 1990; de Peyester and Silvers, 1993; Das et al., 1995; Wilhelm and Idel, 1996). Reported mean hair arsenic concentrations are generally below 0.5 µg/g (Sky-Peck, 1990; Wilhelm and Idel, 1996).

Concluding Remarks
Like lead, arsenic compounds constitute systemic poisons. The intake levels at which no noncancer adverse outcomes are known appear to be $\leq$10 µg/kg/day. Gastrointestinal absorption rates of inorganic arsenic are not well established, but appear to be extensive for some compounds (ATSDR, 1993, 2000). Health Canada (1996) accepts the WHO provisional tolerable daily intake (PTDI) of 2.0 µg/kg-bw/day as inorganic arsenic. The oral risk-specific CCME soil quality guidelines of 12 and 31 µg/g, corresponding to cancer risks of 10^{-6} and 10^{-5} respectively, suggest that for contaminated residential properties (e.g., Port Colborne and Sudbury, ON) that there may be a risk of skin and other cancers. Furthermore, using the slope factor of 1.5×10^{-3} $(\mu g/kg\text{-}bw/day)^{-1}$ (EPA, 1998), the WHO PTDI is assigned an associated risk of 3/1000. It can be argued that the uncertainties in the calculation of the risk-specific soil guidelines and actual cancer risk estimates are significant. In fact, about one-third of the 3/1000 risk may be expected to occur for the normal background intake of inorganic arsenic of 0.12-0.70 µg/kg-bw/day by Canadians (PSL, 1993), which seems unrealistic. Risks of one per million or one-hundred thousand are usually considered acceptable. This criterion cannot be met in case of arsenic for the average Canadian.

Actual exposures to arsenic can be ascertained by measuring the inorganic arsenic and its metabolites in urine.

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Abridged Toxicological Profiles and Related Health Issues: Cadmium (for Physicians)

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Physician Copy - Cadmium
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Cadmium

Public Health Issues
- Renal function perturbations
- Bone fracture risk

Chronic Occupational Health Issues
- Chronic obstructive lung disease
- Reduced lung function
- Renal damage
- Bone diseases
- Lung cancer

Exposure Guidelines

Occupational (8-hr TLV-TWA)

• Cadmium, elemental and compounds, as Cd	0.01 mg/m3 (total) 0.002 mg/m3 (respirable fraction)

General Public

Ambient Air Quality Criteria (MOE)	2.0 µg/m3 (cadmium and compounds)
• Soil Remediation Criteria (MOE) (generic, residential)	12 µg/g
• Reference dose (US EPA) 1.0 µg/kg/day for food	

Biological Exposure Indices

• BEIs (ACGIH)	5 µg/g creatinine in urine (or 5 µmol/mol creatinine) 5 µg/L in blood (or 44.5 nmol/L)

Chronic Health Effects

The chronic health issues associated with exposures to cadmium and its compounds are well documented in the following sources: Friberg et al, 1985, 1986; IPCS, 1992; IARC, 1993; ATSDR, 1998; EPA, 1998; Järup et al, 1998. Only the salient features are summarized below and a special focus is provided on the public health implications.

High level chronic exposure in occupational settings has been associated with respiratory diseases (reduced lung function, chronic obstructive lung disease), kidney damage, bone lesions and lung cancer. Of these adverse health outcomes, nephrotoxicity is most relevant to public health. It involves both renal tubular dysfunction and glomerular permeability (Lauwerys and Hoet, 1993; Lauwerys et al, 1994). In cross-sectional population studies (Buchet et al, 1990; Staessen et al, 1994) and a prospective population study (Staessen et al, 1999), the renal function of residents of six districts (total population of approximately 10,000) surrounding three zinc smelters was compared to that of inhabitants of four control districts (also total population of approximately 10,000) that were more than 10 km away from the smelters and were less polluted by cadmium. "After standardization for several possible confounding factors, five variables (urinary excretion of retinol-binding protein, N-acetyl-beta-glucosaminidase, beta2-microglobulin, amino acids and calcium) were significantly associated with the urinary excretion of cadmium (as a marker of cadmium body burden), suggesting

the presence of tubular dysfunction" (Buchet et al, 1990). In a follow-up study (Staessen et al, 1994), these nephrotoxic markers in urine were again found to be elevated in the six polluted areas, as well as a reduction in creatinine clearance and serum zinc and an increase in serum creatinine,. "In all 10 districts, cadmium in the soil was positively correlated with cadmium in celery (r=0.77), in beans (r=0.67), and in residents' urine (r=0.76). The creatinine clearance was inversely correlated with cadmium in soil (r= -0.78), in celery (r= -0.90), and in beans (r=-0.70)" (Staessen et al, 1994). In a prospective 7-year follow-up study of the same populations, Staessen et al (1999) found that "cadmium excretion in the districts near smelters was 22.8% higher (p=0.001) than in other districts", and there was a significant difference in bone fracture rates in women (p<0.007) and a non-significant increase (p=0.08) of height loss in men. Across the 10 districts, mean cadmium concentrations in soil ranged 0.8 to 14.7 µg/g and 0.1 to 4.0 µg/g (dry weight) in vegetables.

Biological Exposure Indices

Cadmium concentrations in whole blood and urine are good, but different indices of exposure. Because of its unique metabolism involving the protein metallothionein, cadmium accumulates in the liver and kidneys and is released from these organs with a long half-life of ≥10 years (Friberg et al, 1985, 1986). Cadmium in urine reflects the accumulated cadmium and thus the body burden. By contrast, cadmium in blood represents more recent exposure and exhibits a half-life of 40-90 days, although there is evidence for a slower phase as well (Nieboer et al, 1999; Lauwerys and Hoet, 2001).

For individuals with blood and urinary cadmium levels within the reference intervals established for the general Canadian population (i.e., <2 µg Cd/g creatinine or 2 µmol/mol creatinine in urine and <6 µg (54 nmol) Cd/L in blood for smokers, compared to <1 µg/g or 1 µmol/mol creatinine in urine and <2 µg/L or 18 nmol/L in blood for non-smokers) (Nieboer and Fletcher, personal assessment; also see Health Canada, 1995), the risk of cadmium-related renal dysfunction is low or nonexistent only for non-smokers. Empirical models predict up to 10% probability of subclinical renal dysfunction (e.g., micro proteinuria) for individuals environmentally exposed with urinary cadmium exceeding 2.0-3.0 µg Cd/g creatinine (2.0-3.0 µmol/mol creatinine) and blood levels ≥5.6 µg/L or 50 nmol/L (Järup et al, 1988; Buchet et al, 1990; ACGIH, 1991). It is apparent that the largest single source of cadmium exposure is through cigarette smoking and that this alone can affect renal function. The ACGIH (2000) continues to recommend the biological exposure index (BEI) of cadmium in urine of occupationally exposed individuals as 5 µg/g creatinine (or 5 µmol/mol creatinine) and 5 µg/L (44.5 nmol/L) in blood. In promulgating this BEI, it was concluded that even though subclinical renal changes such as micro-proteinuria remain within the normal range, they are predictive of exacerbating the age-related decline in renal function (ACGIH, 1991).

Based on the concentration ranges observed for cadmium in urine and whole blood of non-smokers by the Centre for Toxicology in Quebec City, Canada, the following concentrations are suggested as levels of concern when some review of exposure is warranted: 10.0 nmol/L in urine and 5.0 nmol/L in blood. Scrutiny of the predicted impact on kidney function of lifetime exposure to cadmium suggest that the concern levels for smokers are: 35.6 nmol/L in urine and 25.0 nmol/L in blood. These correspond to a 5% estimated probability of kidney dysfunction (Elinder and Järup, 1996; Järup et al, 1999). Based on the discussion above, action levels that require even closer scrutiny, perhaps including retesting and clinical assessment of renal function, are assigned the value of 44.5 nmol/L for both urine and blood cadmium concentrations.

In a world-wide survey, Canadian cigarettes had the second highest cadmium content (1.57±0.08 µg/cigarette) (Watanabe et al, 1987). Based on the Belgian studies reviewed, the general public may be at some minor risk in communities with zinc/cadmium mining/refining operations (past or present) or with extensive cadmium-products use or manufacturing (Kreis et al, 1992; Staessen et

al, 1994, 1999), since adverse renal function changes correlated positively with concentrations of cadmium in soil and vegetables. Some concern may also be warranted for subpopulations with unique dietary practices that include, for example, significant consumption of kidney or liver (e.g., from marine mammals or wildlife; Glooschenko et al, 1988; Crête et al, 1989) or aquatic macrophytes such as wild rice (Pip, 1993) that have a known potential for accumulating cadmium even in non-polluted environments. Consumption of fish fillet (i.e., muscle tissue) does not appear to make a significant contribution. In addition to smoking habits (past and present), age, and unique diets, statistical analysis of community-based studies must consider possible occupational exposure and environmental factors (i.e., place of residence) as predictors of cadmium levels in urine and whole blood (Nieboer, 1995; Sartor et al, 1992).

In the occupational setting, hair cadmium content can serve as a good indicator of airborne concentrations of this metal (Wilhelm and Idel, 1996). These authors concluded "that cadmium is a poor indicator of endogenous intake at low dose exposure" and, further, "hair cadmium analysis is not suitable to reflect the individual cadmium load." This conclusion receives support from a recent cross-sectional study of 40 residents of a remediated Cd-polluted area of Japan (Liu et al, 2001). While urinary and blood cadmium were both associated with perturbations of renal function, hair was not. Since cigarette smoke is responsible for the elevation of blood cadmium in smokers relative to non-smokers, second-hand smoke will likely constitute an important exogenous source for hair. In a 1990/1992 German environmental survey (Wilhelm and Idel, 1996; Seifert et al, 2000), the 95th percentile for hair cadmium was 0.30 μg/g for children (6-14 years) and 0.27 μg/g for adults (25-69 years); the respective geometric means were 0.048 and 0.046 μg/g.

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Toxicological Profile and Related Health Issues: Copper (for Physicians)

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Copper

Public Health Issues
- Dietary requirements as an essential metal
- Susceptible individuals (only a remote possibility)

Occupational Health Issues
- Metal fume fever
- Upper airways irritation

Acute Poisoning (Copper Salts)
- Rare, but with systemic symptoms

Exposure Guidelines

Occupational (total aerosols; 8-hr TLV-TWA)

• Fume	0.2 mg/m3
• Dusts and mists, as Cu	1.0 mg/m3

General Public

• WHO Minimal Dietary Requirement	1.4 mg/day (adults) 0.75 mg/day (children)
• Recommended Dietary Allowances	0.9-3.0 mg/day (adults) 1.0-2.0 mg/day (children)
• Tolerable Upper Dietary Intake Level (IOM)	10 mg/day (protection from liver damage)
• Ambient Air Quality Criteria (MOE)	50 µg/m3 (copper and its salts)
• Soil Remediation Criteria (MOE) (generic, residential)	225 µg/g (300 µg/g for medium and fine textured soils)

Essentiality

Copper is an essential trace metal. It is an important cofactor in oxidative proteins or enzymes (da Silva and Williams, 1991; IOM, 2001). For example, cytochrome-c oxidase. which is critical to respiration, contains copper; similarly tyrosinase is a copper-based enzyme involved in the oxidative catabolism of the amino acid tyrosine. Ceruloplasmin is the most important serum copper-transport protein.

Good dietary sources of copper are organ meats, especially liver, followed by seafood, nuts and seeds (US National Research Council, 1989). Human milk contains approximately 0.3 mg/L, while cows milk contains around 0.09 mg/L. An intake of 1.5-3.0 mg Cu per day for adults (between 1.0-2.0 mg per day for children) has been considered adequate and safe (Burtis and Ashwood, 1994; Whitney et al, 1998). Most recently, a daily intake of 900 µg/day has been recommended for US adults, with an upper tolerable intake of 10 mg per day to protect against liver damage (IOM, 2001). About a third of the copper taken in food is absorbed and the major excretion pathway is the bile (Wapnir, 1998).

Copper Deficiency

Although copper deficiency is not frequently reported, it produces a plethora of clinical symptoms (Burtis and Ashwood, 1994; Falchuk, 1998; IOM, 2002). Patients exhibit low plasma ceruloplasmin levels, neutropenia, and scurvy-like bone changes which respond to copper supplements. Deficiency is also illustrated by Menkes' disease, a sex-linked disorder of copper metabolism in which a defective copper transport gene in many tissues is responsible for systemic copper deficiency (Bull and Cox, 1994; Olivares and Uauy, 1996; Kaler, 1998). Individuals exhibit progressive cerebral degeneration, retarded growth and abnormally sparse and brittle hair ('Kinky hair syndrome').

Copper Toxicity

Copper toxicity is rare (Bremner, 1999; IOM, 2002), unless large amounts of a copper salt are ingested. Intake of high levels of copper results in gastrointestinal illness (abdominal, cramps, nausea, diarrhea) as has been demonstrated in a number of studies involving contaminated drinking water (Knobeloch et al, 1994; 1998; Pizarro et al, 1999a, 1999b). Copper concentrations of >2 mg/L are suspected. Liver damage appears to be limited to those with a genetic susceptibility such as patients with Wilson's disease, Indian childhood cirrhosis and idiopathic copper toxicosis (Scheinberg and Sternlieb, 1996; Olivares and Uauy, 1996; IPCS, 1998). Worry has also been expressed that infants can perhaps not adjust to high copper intakes making them potentially susceptible to overload and liver damage (Uauy et al, 1998). However, this does not appear to be a serious issue since in a recent study of infants there was no evidence of adverse or toxic effects (Olivares et al, 1998). Between ages 3 to 12 months the children were given drinking water daily with 2 mg/L of copper.

The genetically determined Wilson's disease clearly illustrates that copper is a systemic poison. In this inborn error of metabolism, there is a chronic accumulation of copper in all body tissues due to a defective gene encoding for a protein involved in copper transport in the liver (Bull and Cox, 1994; Scheinberg, 2001). A positive copper balance leads to deposition of copper in the liver where it produces cirrhosis; in the brain, causing mental disturbances, spasticity and tremor; in the cornea, where deposits of copper oxides are visible as the Kayser-Fleischer ring; and in the kidneys, causing renal tubular loss of amino acids, phosphate, bicarbonate and urate (Scheinberg and Sternlieb, 1996; IPCS, 1998).

Copper exposure in the workplace appears not to be associated with serious adverse health effects. Metal fume fever has been reported in workers exposed to copper oxide, metallic dust or fumes and is a 24-48 hour illness characterized by chills, fever, aching muscles, dryness and in the mouth and throat and headache (ATSDR, 1990; Balmes et al, 1997; IPCS, 1998). This response also occurs for exposure to fumes or dust of metallic zinc and magnesium or their oxides. Upper airways irritation is also known.

Maintenance of Copper Balance

Special tracer studies have shown that endogenous copper excretion into bile is a major point of regulation of the body's copper stores, as is regulation of gastrointestinal absorption (Turnlund, 1998; Turnlund et al, 1998). Relative absorption appears to be more efficient with lower dietary copper intake; conversely, bilary excretion seems to increase when dietary intake is enhanced. The latter pathway seems to be more important than regulation of absorption and thus in protection against deficiency and toxicity. Clearly, homeostasis explains why indices of copper stores such as plasma copper, ceruloplasmin and erythrocyte SOD activity are relatively insensitive to change, except under extreme dietary intake conditions. Since urinary copper excretion is low, it does not contribute significantly to regulating copper stores.

Biological Monitoring Issues

Plasma (serum) copper and ceruloplasmin concentrations, as well as urinary copper and erythrocyte superoxide dismutase (SOD) activity, are often employed as indices of copper status (Turnlund et al, 1997; Milne, 1998). These measures appear to respond to low copper diets and then respond to repletion (Turnlund et al, 1998). By contrast, there is very little change in the concentrations of these parameters when individuals with normal copper intake are given copper supplements (e.g., 1, 3 or 5 mg Cu/L as the sulphate salt (??) added to drinking water for 2 weeks, followed by 1 week of normal tap water, repeated for each dose, Pizarro et al, 1999a,b). It also appears that urinary copper is not very sensitive to occupational exposures to copper (Nieboer et al, unpublished).

The generally accepted serum Cu levels are between 601 and 1373 μg/L (Minoia et al, 1990; Burtis and Ashwood, 1996). In pregnant women, the concentration is generally higher, increasing during pregnancy. Accepted values at term are between 1180 and 3020 μg/L. The blood Cu concentration is known to increase in women using estrogen therapy or substitution. A variation through childhood and youth is recognized with low levels of 200-700 μg/L between 0-6 months, increasing to 900-1900 μg/L by 6-12 years of age. There are also gender differences, with slightly lower levels in adult males (700-1400 μg/L) than in adult females (800-1550 μg/L).

Copper excretion into urine by adults ranges 15-36 μg/day, roughly corresponding to concentrations of 8-18 μg/L, assuming a daily urine volume of 2 L. (Lauwerys and Hoet, 2001). In a copper refinery, concentrations ranged from 8-30 μg/L with the upper limit corresponding to cathode workers in a copper electrorefinery department (Nieboer et al, unpublished).

Copper in hair is evenly distributed across the hair shaft and does not show much dependence on distance from the root hair. The former constitutes evidence that copper is secreted into hair. Natural concentrations in hair are relatively high (20±10 μg/g) which reflects the substantial serum levels described above. Since the latter are homeostatically controlled, hair like serum copper is likely insensitive to internal exposures. Further, the inherently high concentrations will likely complicate the separation of exogenous (external) sources from the major endogenous (internal) contributions (Sky-Peck, 1990, Wilhelm et al, 1991, Seifert et al, 2000).

Concluding Remarks

Since in healthy individuals copper balance is regulated (i.e., intake, body tissue and fluid concentrations and excretion), moderate increases in uptake are tolerated. However, and somewhat hypothetically speaking, individuals with special susceptibilities may exist such as carriers of Wilson's disease mutations and, of course, Wilson's disease patients. The disease occurs in every ethnic and geographic population, with a worldwide prevalence of about 1 in 30,000, and the frequency of heterozygous carriers of a mutation is about 1 in 90 (Scheinberg, 2001). Carriers can have subclinical indications of impaired copper metabolism. Low serum ceruloplasmin levels, high liver biopsy copper concentrations and elevated urinary copper excretion are helpful diagnostically, among other measures (Scheinberg, 2001). Similar concerns exist for the other inherited diseases mentioned earlier, namely Indian childhood cirrhosis and idiopathic copper toxicosis. Individuals with other liver diseases may also represent another susceptible group.

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Toxicological Profile and Related Health Issues: Inorganic Lead (for Physicians)

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Physician Copy - Inorganic Lead
Prepared for the Regional Niagara Public Health Department and available online
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Lead

Public Health Issues
- Developmental impairment in utero
- Cognitive and behavioural impairments in children
- Hematological disturbances in children and adults (subclinical)

Occupational Health Issues
- Neuropathy (slowed nerve conduction)
- Developmental impairment in utero
- Adverse pregnancy outcomes
- Reduced sperm quality
- Anemia
- Hematological perturbances
- Hypertension
- Cognitive and behavioural impairments
- Neuropathy (slowed nerve conduction)

Acute Poisoning (Lead Fumes or Salts)
- Abdominal pain (colic)
- Encephalopathy
- Hemolysis
- Acute renal failure

Exposure Guidelines

Occupational (total aerosols; 8-hr TLV-TWA)
- Lead, elemental and inorganic compounds, as Pb — 0.05 mg/m3

General Public
- Ambient Air Quality Criteria (MOE)
  - -30 day — 0.7 µg/m^3
  - -24 hr — 2 µg/m^3
  - -point of impingement — 6 µg/m^3
- WHO Provisional Tolerable Daily Intake (PTDI) — 3.57 µg/kg/day(adults and children)
- Intake of Concern (MOE) — 1.85 µg/kg/day

Soil Cleanup Guidelines (MOE)
- residential/parkland — 200 µg/g
- agricultural — 60 µg/g
- industrial/commercial — 1000 µg/g

Biological Exposure Indices (of Medical Concern)
- Whole blood (children and females of reproductive age) — 100 µg/L

Note: 10 µg/dL=100 µg/L=0.48 µmol/L

Sources

Lead exposure has decreased steadily since the late 1970s because lead in the most common sources has been reduced or eliminated. Unleaded gasoline was introduced in 1975 and leaded gasoline has been banned in Canada since 1990. Air levels have dropped concomitantly. Leaching of lead from solder in food or drink cans has virtually been eliminated, solder used in plumbing now contains low lead levels, and lead pipes are no longer used. The latter two sources had the potential of contaminating drinking water. Paint chips and dust are significant sources of ingested lead by children, especially in the USA (Pirkle et al, 1994, 1998; Brody et al, 1994). Houses built before 1960 were likely painted (interior and exterior) with lead-based paint. Since 1980, lead in interior paint has been discontinued (Health Canada,1995). Ingestion of contaminated soil around industrial operations has also been shown to be an environmental source for children (e.g. Goulet et al, 1996; Langlois et al, 1996; Hilts et al, 1998). In a study of environmental contaminants in the western James Bay region of Ontario, cord and maternal blood lead correlated with the consumption of a traditional diet of wild game (fowl and mammal). Cord and maternal blood lead were highly correlated and of similar magnitude (Hanning et al, 1997, 2003). Subsequently, radiographic and analytical evidence has been presented for lead shot contamination of edible portions of game birds harvested with lead shot (Scheuhammer et al, 1998; Tsuji et al, 1999), as well as radiographic evidence of lead shot in the human gastrointestinal tract of First Nation Cree (Tsuji and Nieboer, 1997). The use of guns results in two exposure sources for hunters: aerosols generated from the fragmentation of leaded ammunition and from the gun powder which contains a lead compound as the primer (Anderson et al, 1977; Ozonoff, 1994; Tsuji et al, 1996). Examples of inadvertent sources of lead are certain ceramic glazes, poor quality crystal decanters and Asian folk remedies (Health Canada, 1995).

Absorption and Excretion

Within 24 hours of inhalation, 10-60% of lead particles of 0.01-5.0 um diameter are absorbed, unless the lead-containing substance has low solubility (Skerfving et al, 1995). Larger particles are deposited in the upper airways and most of them are cleared and swallowed. Absorption of ingested lead varies considerably and depends on the simultaneous intake of food. As much as 20% (e.g., a range of 4-21%) of soluble lead salts taken with meals may be absorbed; calcium, phosphate, iron, and phytate appear to reduce absorption (Skerfving, 1995). In a recent study (Maddaloni et al, 1998), soilborne lead was fed to six fasting adults (85.5 mg soil/70 kg body weight in a gelatin capsule, corresponding to 250 μg Pb; only the soil fraction <250 μm in size was employed). Under these conditions, 26±8 % was absorbed from the residential yard soil. When administered immediately after a standardized breakfast, the absorption rate dropped to 2.5±1.7%.

Iron deficiency and poor calcium intake are considered risk factors for lead uptake (Mahaffey, 1990; Sargent, 1994; Odland et al, 1999a). However, Ruff et al (1996) conclude that when iron is sufficient, changes in blood lead and changes in cognition in 2-year-old children are inversely related; when iron is deficient, other processes affect their perceptual-motor performance.

Multi-department toxicokinetic models of lead metabolism have been developed (e.g., Leggett, 1993). The half-life of lead in the peripheral blood and soft tissue compartments is around 1 month (Skerfving et al, 1995). Most blood lead is bound to erythrocyte intracellular proteins, while lead in the skeleton constitutes approximately 95% of the body burden. For bone, the most recent estimate of the half-life is 9-12 years (Brito et al, 2000), with a shorter time for trabecular bone (approximately 1 year; Skerfving et al, 1995). Lead stable isotope studies have shown that the mobilization of bone lead is enhanced during pregnancy, lactation, and trauma such as breaks (Mushak, 1998). Further, this experimental approach suggests that in cases where levels of environmental lead exposure and dietary lead intake are low, skeletal contribution is dominant, especially during pregnancy and postpartum (Smith et al, 1996; Gulsen et al, 1995, 1997, 1998, 1999). Urine is the major route of excretion.

Toxic Effects

Preamble

Clearly, lead is a systemic poison and there is convincing evidence that lead appears to have virtually no toxicological threshold (Todd et al, 1996; Finkelstein et al, 1998; Rogan and Ware, 2003). This is illustrated in the standard stick diagram relating blood lead levels to outcomes in children (see the attached figure). Negative impacts on the development of children appear to be evident at blood lead levels at or below the level of medical concern (10 µg/dL=100 µg/L=0.48 µmol/L).

The following reliable sources were consulted for the well-established knowledge described below regarding lead toxicity: Cullen et al, 1983; Mushak et al, 1989; Skerfving et al, 1995; IPCS, 1995; Goyer, 1996; Lewis, 1997; Levin and Goldberg, 2000. When used, these sources are identified and are supplemented by subject-specific references.

Acute Toxicity (Poisoning)

Gastrointestinal symptoms (cramping, colicky abdominal pain, nausea, vomiting, black stool) often present first. Neurological symptoms follow and can range from headache and confusion to stupor, coma and seizures (see attached figure). In severe cases, oliguria and acute renal failure may occur (Lewis, 1997). Treatment involves induced emesis and catharsis, hydration and chelation therapy (CDC, 1991, 1997; Lewis, 1997; Levin and Goldberg, 2000).

Developmental Effects

Children have been recognized for some time as the most sensitive receptor, with developmental outcomes (especially neurological impairments: both cognitive and behavioural) the primary concern (Needleman et al, 1979; Schwartz and Otto, 1991; Grant and Davis, 1989; Needleman and Bellinger, 1994; Winneke, 1995). Estimated loss in IQ by children from an increase of 100 µg/L to 200 µg/L in blood lead ranges from 1.3 to 5.8 (Rice and Silbergeld, 1996), with 2-3 IQ points being the best estimate based on meta-analysis (Winneke, 1995). Minor structural anomalies at birth have also been reported (Needleman et al, 1984). Considerable evidence exists (Goyer, 1990), including our own (Odland et al, 1999b), that the placenta does not attenuate the passage of lead to the fetus. Andrews et al (1994) have critically reviewed the available epidemiological studies on prenatal lead exposure in relation to premature rupture of membranes (PROM), preterm delivery, gestational age, low birth weight, mean birth weight and birth weight adjusted for gestational age. Many of the studies reviewed were vulnerable to community-related confounding factors, such as socio-economic status. Andrews et al (1994) conclude that the association between PROM and prenatal lead exposure has not been confirmed, nor has that for pre-term delivery and gestational age; by contrast, the data appear supportive of the hypothesis that prenatal lead exposure adversely effects birth weight and that this relationship may have a threshold. Maternal blood lead concentrations at which some impact was observed exceeded 60 µg/L. The birth weight finding showed some dependence on the method used in the statistical analysis. Consideration of gestational age did not clarify the relationships. Our own results support the conclusion about the likelihood of an association of prenatal lead exposure and reduced birth weight, based on univariate and multivariate linear regression analysis of maternal blood lead concentrations (range: 4-135 µg/L, N=262) and birth weight taking into consideration gestational age and confounders (Odland et al, 1999b). Osman et al (2000) report similar findings when examining the relationships between cord blood lead levels (1-122 µg/L) and fetal growth (i.e., reduced birth weight, length and head circumference). Recent studies of cognitive development in early childhood provide further evidence that low-level lead exposure represents a potentially preventable risk factor for developmental delay in young children (Mendelsohn et al, 1999). Neuropsychological decrements in adults appear to occur for blood lead concentrations above 400 µg/L, although based on neuropsychological testing elderly women had some loss in cognitive function at $\geq$80 µg/L (Muldoon et al, 1996; Levin and Goldberg, 2000). Neuroanatomic,

neurotoxicologic and behavioral studies in animals support the findings in humans (Verity, 1990; Rice and Silbergeld, 1996; Finkelstein et al, 1998).

Hematological Findings

The effect of lead on blood is extensive and ranges from subclinical effects such as inhibition of the enzyme delta-aminolevulinic acid dehydratase (ALAD), erythrocyte zinc protoporphyrin (ZPP) elevation, increased urinary delta-aminolevulinic acid (ALA), reduced hemoglobin production and frank enemia (see figure) (Cullen et al, 1983; Mushak et al, 1989; Goyer, 1996; Levin and Goldberg, 2000). Even in adults, ALAD inhibition is evident at 100 µg/L of lead in blood.

Renal Effects

Slight enzymuria (e.g. of N-acetyl-beta-glucosaminidase, NAG) and proximal tubular disturbances can be detected for blood lead levels in the range 300-400 µg/L (Verschoor et al, 1987; Skerfving et al, 1995; Goyer, 1996), and appears to be reversible. Acute lead poisoning can also result in reversible damage to the proximal tubules that includes aminoaciduria, glucosuria, hyperphospaturia, and enzymuria. By contrast, chronic high exposure to lead over many years (blood levels >500 µg/L) has led to interstitial nephritis and fibrosis, which results in reduced glomular filtration rate, azotemia (increase in blood urea nitrogen, BUN, and serum creatinine) and tubular reabsorption of uric acid. The latter effect may be responsible for the occurrence of gout (Skerfving et al, 1995; Levin and Goldberg, 2000). Correlation between blood lead concentrations in the range 20-725 µg/L with measures of glomerular impairment in a large general population has been reported (Staessen et al, 1992).

Hypertension

Blood pressure elevation may be among the most sensitive outcomes of lead exposure. A variety of studies suggest that both systolic and diastolic blood pressures may be affected by blood lead concentrations as low as 100-200 µg/L in both males and females (Goyer, 1996; Levin and Goldberg, 2000). However, meta-analyses suggest that the association is weak, with positive studies indicating that a doubling of blood lead levels (e.g., from 125 µg/L to 250 µg/L) may increase the systolic pressure by 1.0 mm Hg and the diastolic measurement by 0.6 mm Hg (Hertz-Picciotto and Croft, 1993; Staessen et al, 1995). Tibia bone lead measurements support this conclusion (Hu et al, 1996). Whether these observations translate into significant public health issues continues to be debated.

Reproductive Effects

Because of the relatively high background of spontaneous abortions (7-15%) and the many contributing factors, it has been difficult to clearly establish a link with environmental lead exposure (Winder, 1993; Hertz-Picciotto, 2000). Nevertheless, there is some suggestion that exposures in the range 100-250 µg/L might enhance the rate of spontaneous abortion (Hertz-Picciotto, 2000). Pregnancy outcomes such as PROM and reduced birth weight in relation to low levels of lead exposure have already been reviewed in an earlier section (Developmental Effects).

Impact of lead on male reproductive health is somewhat more certain according to the critical reviews by Winder (1993) and Apostoli et al (1998). Three main impacts have been discerned: decreased sperm count, volume, and density and abnormal sperm morphology; decreased libido; and endocrine disruption (reduced testosterone, thyroid function defects, and abnormal prostate function). Although such outcomes appear to be associated with blood lead levels of $\geq$ 400 µg/L, Levin and Goldberg (2000) point out that effects on sperm quality at lower concentrations have not been studied adequately. Recent studies of occupationally exposed individuals support this view (Alexander et al, 1996, 1998a; Viskum et al, 1999), suggesting effects at blood lead levels >140 µg/L. There is also the suggestion that reduction in sperm count due to lead exposure may be more evident in individuals with specific genotypes of the enzyme ALAD (Alexander et al, 1998b). Furthermore, recent studies of

time-to-pregnancy and non-occurrence of marital pregnancy provide limited evidence that paternal occupational exposure to lead contributes to infertility (Sallmén et al, 2000a,b).

Carcinogenicity
IARC (1987) denotes inorganic lead and lead compounds as Group 2B carcinogens with sufficient evidence in animals but insufficient in humans. Confounding factors such as smoking and exposure to other carcinogens have complicated the interpretation of human studies (Fu and Boffetta, 1995; Goyer, 1996). Exposure to arsenic trioxide during the refining of lead is a case in point. While animal experiments have suggested the possibility of renal tumours, this has not been born out in epidemiologic studies (Levin and Goldberg, 2000).

Biological Exposure Indices
Because of its interference with heme synthesis, a number of endpoints can serve as indices of exposure. Most prominent among these are ALAD acivity and ZPP accumulation in red blood cells, delta-aminolevulinic acid and coproporphyrinogen in urine (Lauwerys and Hoet, 1993). Of these, ZPP has had the widest application in monitoring lead exposure in children. Because it is relatively insensitive to exposures corresponding to the medical level of concern and lower ($\leq$100 µg/L), its use in screening has wained (Graziano, 1994; CDC, 1997; Froom et al, 1998). Blood lead has become the gold standard index for measuring exposure because of improved analytical sensitivity and the implementation of stringent quality-control programs for collection, handling and lead analysis (Graziano, 1994; CDC, 1997). Such precautions also permit the use of capillary blood sampling by fingerstick (CDC, 1997).

Empirically, relationships between blood lead levels in children and the concentration of lead in soil have been reported. Slope factors of 8 µg/L to 80 µg/L for 1000 µg/g (ppm) lead in soil have been observed for children as summarized by Stern (1994). Typically, the age group is 1-7 years and the ingestion rate of soil is around 100 mg per day. Based on theoretical risk assessments, Stern (1994) and the Ontario Ministry of Environment and Energy (OMEE, 1994) derive comparable relationships. Clearly, ingestion of soil can result in children reaching the medical level of concern for blood lead.

The CDC (1997) has made the following recommendations for clinical follow-up of children:

- $\leq$90 µg/L — Reassess or rescreen; no additional action unless exposure source changes;
- 100-140 µg/L — Provide education, follow-up testing and social services (if necessary);
- 150-190 µg/L — As for previous range, but if level persists proceed to the next range;
- 200-440 µg/L — Conduct clinical and environmental investigations and lead-hazard reduction and provide case management (i.e., coordination of care);
- 450-690 µg/L — As above, as well as initiate chelation therapy;
- $\geq$700 µg/L — Hospitalization and emergency medical treatment including chelation therapy.

Good correlations between hair and blood lead have been observed in the occupational setting (Foo et al, 1993; Wilhelm and Idel, 1996), as well as in communities with significant industrial sources (e.g., Chattopadhyay, 1977). In their critical analysis of the available lead-in-hair data, Wilhelm and Idel (1996) make the following two conclusions. (1) "If the first two cm of the proximal end of hair is employed, hair and blood lead concentrations represent the same exposure time period (2-3 months)." This is by virtue of the fact that the half-life of lead in the blood compartment is about one month and hair grows at the rate of 1 cm per month. However, because of the uncertainties due to confounding

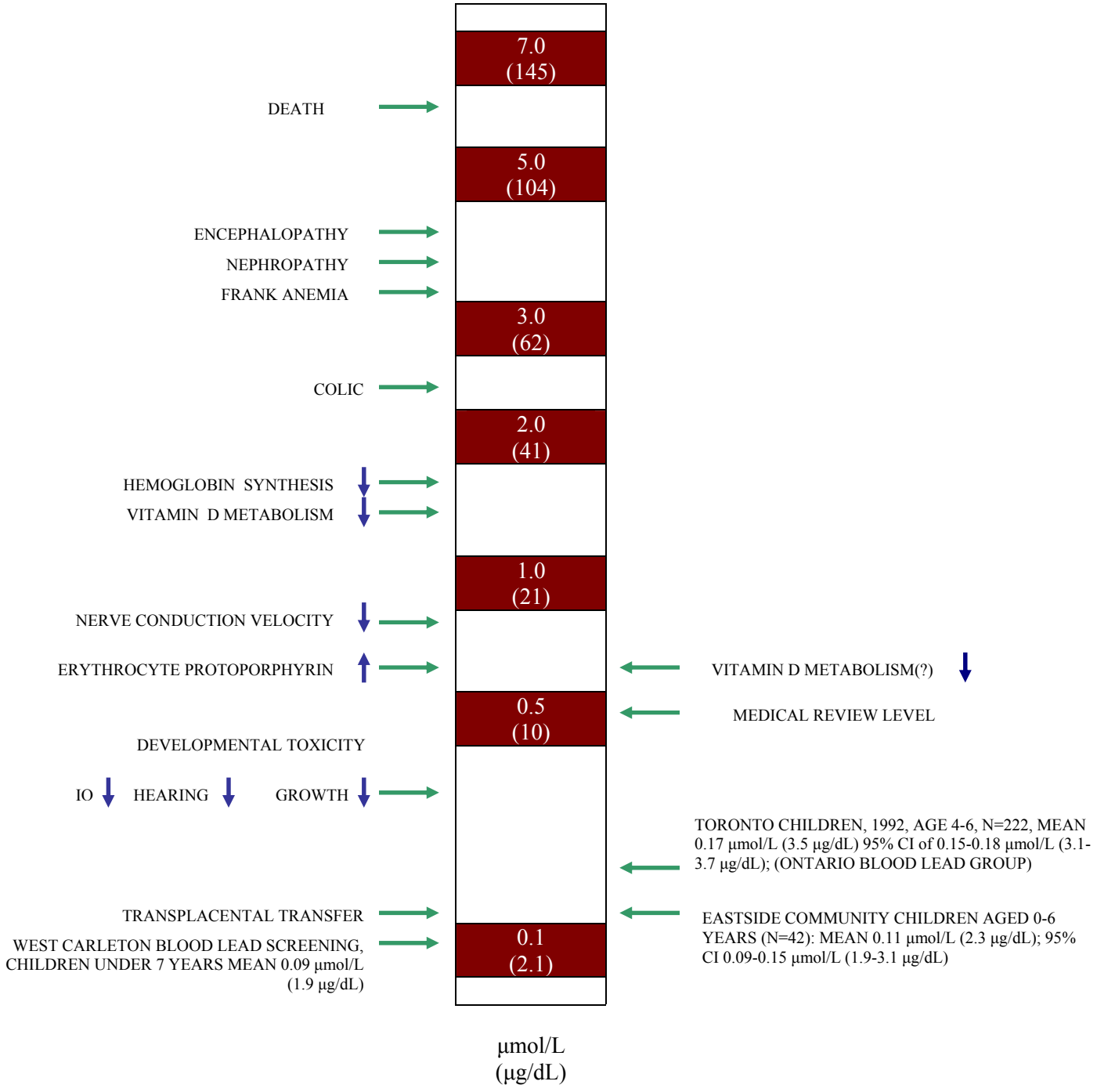
factors[3], there authors further recommend "that individual diagnosis based on hair lead determination has to be interpreted with caution and should be validated by blood analysis." Studies during 1990/1992 in Germany suggest that 5 µg/g in children's hair and 6 µg/g in adults correspond to the 95th percentile; respectively, the geometric means were 1.02 and 0.96 µg/g (Seifert et al, 2000).

Concluding Remarks

The blood lead levels in children considered to be of medical concern have undergone consistent downward adjustment from 400 µg/L (1970-1975), to 300 µg/L (1975-1985), 250 µg/L (1985-1990) and 100 µg/L (1990-present) (CDC, 1997). Epidemiological evidence again suggests that for effects on the central nervous system and developmental impairment (perinatal and childhood) the current guidelines might need be lowered again (Rogan and Ware, 2003). In addition, some researchers express the opinion that clinically apparent effects of lead at moderate to high exposures have subclinical analogs at chronic low-level exposures. This has been known for a long time for the inhibitory action of lead on heme synthesis, with effects on ALAD detectable below 100 µg/L in both children and adults. However, caution about sample size is warranted in designing studies hoping to detect a real clinical effect of the type discussed if it exists, since relative risks observed are bound to be small. Consequently, large numbers of subjects need to be studied to attain suitable statistical power. In addition, the debate will continue about the actual burden of illness about the observable clinical and subclinical outcomes and their public health implications.

[3] Relevant influencing factors are: season, gender, place of residence, permanent wave (females), date of last shampooing, occupation, smoking habits, alcohol consumption, milk consumption, lead in drinking water and indoor dust (Wilhelm and Idel, 1996). Use of leaded ammunition should be added and thus hunting (Tsuji et al, 1996).

Figure 1: LOWEST OBSERVED ADVERSE EFFECT LEVELS CORRESPONDING TO BLOOD LEAD CONCENTRATIONS IN CHILDREN (ADAPTED FROM TSUJI ET AL., 1996)



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Abridged Toxicological Profile and

Related Health Issues: Mercury

(with Emphasis on Methyl Mercury)

Revised May 19, 2004

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Mercury

Public Health Issues
- Methyl mercury levels in fish, related neurotoxcity and developmental effects
- Mercury vapour release from amalgam dental fillings

Occupational Heath Issues (Inorganic Forms of Mercury)
- Acute respiratory distress (high exposure)
- Gastointestinal effects
- Gingivitis
- Tremor and other neurological effects
- Erethism (shyness, emotional lability)
- Renal toxicity

Acute Poisoning
- Death (inorganic and alkyl mercury)
- Pulmonary edema (mercury vapour)
- Renal failure (mercury salts)
- Neurotoxicity (all forms)

Exposure Guidelines

Occupational (total aerosols: 8-h TLV-TWA)

• Alkyl compounds (ACGIH, 2002)	0.01 mg/m3
• Aryl compounds (ACGIH, 2002)	0.1 mg/m3
• Elemental and inorganic form	0.025 mg/m3

General Public

• WHO Drinking water guideline; Health Canada MAC	1 µg/L
• WHO and Health Canada Permissible tolerable weekly intake	5 µg/kg (total) 3.3 µg/kg (methyl mercury)
• USEPA Reference Dose (RfD)	0.1 µg/kg/day (methyl mercury)
• Fish Content (Health Canada)	0.45 mg/kg wet weight

Preamble

For the average Canadian, fish constitutes the major source of bioavailable mercury, as it is present primarily as methyl mercury. Dental amalgams and ambient air are the major sources of mercury vapour, while non-fish foods contain mercury ions (Hg2+) in complexed form (HC, 1995). Both types are designated as inorganic mercury, by contrast to organic mercury which includes alkyl mercury moieties such as methyl mercury. Mercury vapour is quite efficiently absorbed (around 80%), while mercuric ionic forms are poorly taken up. In communities where fish consumption is high, the main concern is that intake of methyl mercury. Consequently, the focus of the toxicological profile will be the health impact of methyl mercury and associated issues.

Absorption and Excretion

As already indicated, methyl mercury consumed in fish tissues is nearly wholly absorbed. Methyl mercury is mainly excreted into faeces and also in breast milk. It has a half-life in the body (including the blood compartment) of 32 to 70 days with a mean of 49 days (WHO, 1990; Clewell et al., 1999). Methyl mercury is incorporated into hair and its concentration constitutes a good index of intake (Airey, 1983; Katz and Katz, 1992; Cernichiari, 1995; Barbosa et al., 2001).

Biomarkers of Exposure and Effect

Both whole-blood mercury and hair mercury are good biomarkers of exposure to methyl mercury. Associations between fish consumption and blood mercury levels have been established (Brune et al., 1991). Eating of seabirds and seabird eggs have also been implicated as sources of methyl mercury (Dewailly et al., 1991). Health Canada (HC 1995) has set the basal total hair mercury level at 1 μg/g, the "no risk" level at 6 μg/g, 10 μg/g as maternal levels with observed motor and CNS effects in infants, the 6-30 μg/g range as at "increased risk", and 30 μg/g as the "at risk" level, corresponding respectively to 4, 20, 40, 40-100 and 100 μg/L in whole blood. Interestingly, the Cree Board of Health and Social Services of James Bay in Quebec (Dumont et al, 1998) has used a somewhat similar grouping: <6.0 μg/g as the "no risk" level; ≥15 μg/g as the intervention level for women of childbearing age; and ≥30 μg/g as the intervention level for all others. In both whole blood and hair, mercury is primarily present as methyl mercury. It should be noted that the absorption of mercury vapour released from amalgam fillings do make small contributions to the observed blood (Berglund and Molin, 1996; Skare and Engqvist, 1994), urine (Levy et al., 2004) and hair (Lindow, 2003) mercury concentrations. Little methyl mercury is excreted by way of urine, and urine mercury primarily reflects exposure to inorganic forms of mercury. In the Province of Quebec, Canada the whole blood total mercury level that is declarable for all individuals is 75 nmol/L (15 μg/L).

Toxic Effects

A perusal of the summary of observed health effects for workers exposed to inorganic mercury and for acute poisoning cases clearly illustrates that compounds of this metal are systemic poisons (LaDou, 1997; ATSDR, 1999). Low-level neurological disturbances, as well as ophthalmological abnormalities have also been reported for adult residents of northwest Quebec (Spitzer et al., 1988).

Much of the knowledge of organic mercury poisoning has been derived from studies of large populations affected by consumption of methyl mercury contaminated fish in Japan during the 1950s and 1960s and by consumption of methyl mercury treated wheat intended as seed in Iraq during the early 1970s (WHO, 1990; ATSDR, 1999). Progressive nervous system effects were most prominent. The earliest symptoms are of numbness and tingling of the lips, tongue, and distal extremities. Loss of motor coordination follows, with gait ataxia, tremor, loss of fine movement, muscular rigidity, spasticity, and seizures. Constriction of the visual fields, hearing loss, neurasthenia, dysphagia, dysarthria, coma, and death can result. Erythroderma, desquamation, and other skin rashes may occur. Behavioural changes, fits of laughter, and intellectual impairment may be prominent. Renal disease is rare.

Mild neurologic and developmental delays have been reported in infants exposed to methyl mercury by way of breast milk (WHO, 1990; ATSDR, 1999). Recent evidence suggests that prenatal exposure to methyl mercury may result in developmental and cognitive deficits that are detectable at 7 years of age (Grandjean et al, 1997, 1999). However, other studies have not confirmed these findings (Myers et al, 2003). Cord blood mercury concentrations constituted a risk factor, as well as maternal hair levels. For the same study group, diastolic and systolic blood pressures increased by 13.9 and 14.6 mmHg, respectively, as cord-blood Hg concentrations rose from 1 to 10 μg/L (Sorensen et al., 1999). Cardiovascular risk factors in a Finnish study of men have also been linked to mercury hair levels associated with fish consumption (Salonen et al., 1995). Cardiovascular irregularities have also been reported for the Iraqi epidemic (ATSDR, 1999).

Other than the neurological, developmental, and cardiovascular outcomes discussed, chronic, low-level dietary exposure to methyl mercury exposures do not appear to be associated with other health effects.

Concluding Remarks

The studies reviewed above support the Health Canada's rather stringent guidelines for hair and blood mercury levels related primarily to the consumption of fish contaminated with methyl mercury fetus are no doubt the most sensitive receptors.

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Abridged Toxicological Profile and Related Health Issues: Polychlorinated Biphenyls (PCBs)

Revised May 19, 2004

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Polychlorinated Biphenyls (PCBs)

Public Health Issues
- Fish consumption restrictions
- Prenatal exposure (neurological effects)
- Exposure of females of reproductive age

Occupational Heath Issues
- Weakness, weight loss
- Skin rash (chloracne)
- Elevated serum triglycerides
- Elevated liver enzymes

Acute Poisoning
- Skin rash (chloracne)
- Eye irriation
- Nausea, vomiting

Exposure Guidelines

Occupational (total aerosols: 8-h TLV-TWA)

• Arochlor 1242 (ACGIH, 2002)	1 mg/m3
• Arochlor 1254 (ACGIH, 2002)	0.5 mg/m3

General Public

• Oral Reference dose (RfD; USEPA)	
Arochlor 1254	0.2 µg/kg-day
Arochlor 1016	0.7 µg/kg-day
• Drinking Water Guidelines (PCBs; US EPA)	0.5 µg/L
• Fish Content (Ontario Ministry Environment)	consumption restrictions begin at 500 µg/kg

Preamble

The literature on PCBs is voluminous and critical appraisal of the available health data is essential. Two recent publications (Longnecker *et al*., 1997, ATSDR, 2000) have this focus, and the salient features of the conclusions reached are reflected in the current toxicological profile. A few more recent developments are also reported.

Background

In terms of their action, PCBs are representative of a class of organochlorines that includes the pesticide DDT and its metabolites. PCBs constitute a large family (209 members or congeners) of chlorinated hydrocarbons that were used commercially in electrical transformer fluids, heat exchange fluids, as paint additives, and in plastics among other uses. PCBs production in North America was stopped in 1977 and is now banned substances. They are environmentally persistent and stay around for a long time. Half-lives in humans vary with the congener, but are usually in years (see below); environmental persistence increases with the degree of chlorination with $t_{1/2}$ values of 10 days to 1.5 years for photo-degeneration. Aroclor (Monsanto Chemicals) is the most common tradename; each Aroclor mixture is assigned its own CAS number, as are families sharing the same chemical formula.

All Arocolor products are characterized by a four digit number; the first two digits represent the type of molecule (12= chlorinated biphenyl) and the last two digits give the weight percent of chlorine. Thus, Aroclor 1248 is a chlorinated biphenyl mixture with an average chlorine content of 48%. An exception is Aroclor 1016, which contains 41% chlorine but only 1% of pentachloro- and higher PCBs.

To identify individual PCB compounds, a numbering system has been designed that is consistent with the International Union of Pure and Applied Chemists (IUPAC) nomenclature for biphenyls. For example, the 42 hexachlorobiphenyl congeners have numbers 128-169; each number is used as a synonym for a specific PCB compound (e.g., congener 169 is 3,3'.4.4',5,5'-hexachlorobiphenyl).

Absorption and Excretion
Meat and especially fish are the main sources of exposure and mother's milk constitutes an important source for infants. PCBs are readily absorbed through the respiratory tract, gastrointestinal tract and skin. Distribution is primarily into fat and thus adipose tissue constitutes the primary body store; in plasma, they are present in the lipid fraction. Bilary excretion constitutes the primary elimination pathway.

Metabolism and Mechanisms of Action
The primary site of biotransformation is in the liver. PCBs induce hepatic microsomal monooxygenase (P-450) enzymes, which convert them to more polar (water-soluble) metabolites by hydroxylation and conjugation. PCBs are potent phenobarbital (PB) type and 3-methylcholanthrene (MC) type enzyme inducers; some induce both (Hansen, 1998). Highly chlorinated isomers are more resistant to metabolism and are therefore, generally speaking, more persistent and half-lives ($t_{1/2}$) are congener specific. In humans, estimates of $t_{1/2}$ for individual congeners vary between $\leq$1 to $\geq$20 years; congeners 138 and 153 are especially long-lived with some reported $t_{1/2}$ values exceeding 20 years. Assessments of $t_{1/2}$ values in humans for Aroclor mixtures range 0.9 to 2.6 years (Aroclor 1242), 8.6 years (Aroclor 1248), 3.3 to 65 years (Aroclor 1254) and 1-28 years (Aroclor 1260) (ATSDR, 2000; Lauwerys and Hoet, 2001). Half-life estimates appear to depend on the body burden (Lauwerys and Hoet, 2001).

Mechanistically speaking, planar PCBs have dioxin-like toxicity which involves receptor (Ah) mediated enhancement of gene expression by way of MC-type induction. This has been the most prominent model of interpreting their toxicity and has resulted in toxic equivalent factors (TEFs) for each specific congener. These are assigned relative to the experimental toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TEF=1.00; Van den Berg et al., 1998; ATSDR, 2002). A toxic equivalence factor (TEQ) is then calculated by multiplying the TEF by the congener concentration. This is usually summed for all PCB congeners present. Although MC-type induction/toxicity has received the primary focus, PB-type of induction/toxicity cannot be ignored.

More recently, epidemiologic evidence (reviewed below) suggests that disruption of endocrine axes by Ah-independent mechanisms occurs. Changes in the activities of Phase I and Phase II enzymes (by inhibition or induction) may well be an effective way of altering circulating/available hormone concentrations (e.g., estrogen, androgen, T3, T4), which through feedback loops may affect the release of a wide-range of metabolic regulators involved in the hypothalamous/anterior pituitary axis [e.g., follicle stimulating hormone (FSH), leuteinizing hormone (LH), thyroid stimulating hormone (TSH), growth hormone (GH), adrenocorticotropic hormone (ACTH), prolactin]. Such pathways seem more likely than estrogen/androgen/thyroid hormone mimicry, a model which has perhaps also received undue attention (e.g., Safe, 1995, 2000; Safe et al., 2000; ATSDR, 2000).

Biomarkers of Exposure

For the long-lived PCBs, concentration in adipose tissue and blood plasma reflect the body burden. The latter, of course, is favoured as biopsy tissue collection is invasive. Plasma or serum concentrations are expressed mg/kg lipid or µg/L. In addition, breast milk PCBs concentration is an important biomarker for infant exposure. In females, lactation is the most important method of eliminating body stores, and inverse associations between lactation and body burden are known (Furberg et al., 2002). Body mass index and age correlate positively with plasma concentrations. Recent plasma concentrations (as Aroclor 1260) for North Americans (all ages), who do not consume fish regularly, range 1-10 µg/L, compared to 2-60 µg/L for individuals who consume fish regularly and are not exposed at work such as Native peoples depending on subsistence food (ATSDR, 2000; Walker et al., 2003). Geometric means of Aroclor 1260 for six Canadian Inuit maternal populations during 1995-2000 ranged 2.4-8.0 µg/L with minima in the range 0.10-2.0 µg/L and maxima 7.9-60 µg/L (AMAP, 2002). Accumulation was age and exposure intensity dependent. By comparison, a recent study of 25 males and females not exposed occupationally and living in Hamilton, Ontario exhibited geometric means of Aroclor 1260 of 1.7 µg/L (men) and 1.3 µg/L (women), respectively with ranges of 0.30 to 7.90 µg/L and 0.40 to 3.80 µg/L (Nieboer et al., 2001). For occupationally exposed individuals, concentrations of 250 µg/L or higher have not been atypical (ATSDR, 2000).

Health Canada guidelines for PCBs in plasma or serum in units of µg/L are often quoted as summarized below (e.g., AMAP, 2002).

For Women of Reproductive Age	*For Men and Post-Menopausal Women*
<5: Tolerable	<20: Tolerable
5-100: Concern	20-100: Concern
>100: Action	>100: Action

However, the numerical values quoted were derived for whole blood (HC, 1995). Thus, when the same concentrations are applied to plasma or serum, an additional two-fold protection factor has been incorporated. This arises because PCBs primarily reside in the plasma lipid fraction of whole blood and the blood cells and plasma volumes are roughly equal.

Toxic Effects

Two accidents involving rice oil contaminated with PCBs and small amounts of polychlorinated dibenzofurans (PCDFs) and some lesser known contaminants occurred in Japan (the Yusho incident) and Taiwan (the Yu-Cheng accident). Since the exposures were primarily to PCBs, the acute toxicity reported is assigned to them. The PCBs affected the skin (chloracne, pigmentation), the eye (hypersecretion, abnormal pigmentation, conjectiva, narrow field of vision, the mucosal surfaces (irritative effects), the liver (fatty changes, abnormal liver function) and the peripheral nervous system (peripheral neuropathy). General malaise, fatigue, respiratory problems, and lowered resistance to infections also occurred, as well as altered immunoglobin levels and white cell changes (HC, 1995; ATSDR, 2000; Longnecker et al., 1997). After 15 years, increased risk of respiratory cancers in males was observed and of liver cancer in females in the Japan incident (Longnecker et al., 1997). Children born of affected mothers and who were exposed *in utero* had an increased risk of dying by age four. Children exposed *in utero* also had lower body weight for about 10 years and lower scores on intelligence test which persisted (Rogan et al., 1988; Guo, 1995; Longnecker et al., 1997). They were also hyperactive and exhibited disordered behaviour.

The evidence for health impairments associated with chronic low-level exposures is less convincing (ATSDR, 2000; Longnecker et al., 1997; AMAP, 2002). Liver function tests abnormalities and chloracne have been associated with occupational exposure, and increased risks of kidney and skin cancers may be termed suggestive but inconclusive. Breast cancer risk in individuals environmentally

exposed suggest no association, but this also remains inconclusive. Overall studies of children and newborns indicate that neurologic and developmental status are adversely affected by high prenatal and perinatal PCBs exposure (Jacobson and Jacobson, 1996; Longnecker et al., 1997; ATSDR, 2002; Walkowiak et al., 2001). There is a suggestion that enhanced exposure to PCBs through fish consumption may affect the gamma-glutamyl transferase (GGT) enzyme as seen in occupationally exposed individuals. A number of studies are now suggesting that endocrine hormone disruption appears to be associated with relatively low exposures. A suppression of T3 and T4 concentrations in mothers and stimulation of infant TSH levels have been reported (Koopman-Esseboom et al., 1994), as well as T3 suppression in adult women ($p<0.001$; Hagman et al., 2001). Similarly, Gerhard et al. (1998) have noted in women with repeated miscarriages positive associations between the sum of four PCB congeners (101,138, 153 and 180) and blood FSH, LH, and prolactin and a negative relationship for TSH and testosterone ($p<0.05$). In a menstrual cycle study, we (Nieboer et al, 2001) observed strong positive correlations between plasma concentrations of Aroclor 1260, as well as for congeners 138 and 153, and mid-luteal phase urinary FSH level ($p<0.05$) and a negative relationship with mid-luteal urinary $E_1$3G concentrations ($p=0.057$-0.064; $E_1$3G is a conjugate of estrogen). The latter relationship has been linked to infertility (Baird et al., 1999). Additional research is needed to verify that, at background concentrations, PCBs have the ability to disrupt circulating levels of hormones involved in the hypothalamus/anterior pituitary axis hormonal pathways. And finally, the relationship between the body burden of PCBs and immunological suppression remains unresolved.

Since strong intercongener correlations exist for background exposures, and apparently also between PCBs and dioxins, some uncertainty remains in attributing the various health outcomes described solely to PCBs.

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Abridged Toxicological Profile and Related Health Issues: Selenium

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Selenium

Public Health Issues
- Dietary requirements as an essential element
- Selenium poisoning (selenosis; see below)

Occupational Heath Issues
- Hair, nail brittleness; hair loss; neurological symptoms; respiratory tract and dermal irritation
- Garlic odoured breath

Acute Poisoning (Selenosis)
Selenium poisoning can occur for daily intakes ≥0.5 mg (e.g., in areas of high-selenium soils)
- Severe respiratory irritation if inhaled

Exposure Guidelines
Occupational (total aerosols: 8-h TLV-TWA)

• Selenium compounds (ACGIH, 2002)	0.2 mg/m3

General Public

• WHO Recommended daily intake	0.9 µg/kg body wt. (adults)
• WHO Guideline for drinking water	0.01 mg/L
• Recommended Dietary Allowance (IOM, 2000)	55 µg/day (adults)
• Tolerable Upper Dietary Intake Level (IOM, 2000)	400 µg/day (adults; protection from selenosis)

Essentiality
Selenium is a requirement for a selected number of proteins (selenoproteins), the activity of which is dependent on the incorporation of the amino acid selenocysteine. Although selenomethionine is randomly incorporated in proteins, its presence does not appear to be different from methionine. Two families of selenoprotiens are enzymes involved in the removal of iodine atoms (deiodinases) or scavenging of hydrogen peroxide (glutathione peroxidases). The functions of two other selenoproteins (referred to as P and W) are not known (da Silva and Williams, 1991; Brody, 1999; IOM, 2000). Meat and seafood are reliable dietary sources of selenium, in the form of selenomethionine (initially synthesized in plants) and selenocysteine. These forms are highly bioavailable, as well as the inorganic selenate and selenite salts used in dietary supplements.

Deficiency
It is suggested that selenium deficiency does not alone cause disease but predisposes individuals to illness when other stressors are present such as viral infection or chemical exposure (Brody, 1999; IOM, 2000). Keshan disesase is a cardiomyopathy observed in selenium-deficient children in the Keshan region of China. Kashin-Beck disease also appears to occur in low-selenium areas of Asia; it constitutes a slowly progressive and disabling degenerative disease of peripheral joints and spine. By contrast, clinical thyroid disorders have not been reported in association with selenium-deficient individuals. Epidemiologic evidence suggests that selenium intakes above the normal dietary requirements may have an anticancer effect, presumably because if its antioxidant activity.

Toxicity
Selenium poisoning (selenosis) can occur in areas of high-selenium soils, where the daily intake of Se is about 0.5 mg or greater (Brody, 1999; IOM, 2000; ATSDR, 2001), or due to chronic exposure in the

workplace (LaDou, 1997). This disease involves hair and nail brittleness and loss, gastrointestinal disturbances, skin rash and dermatitis, fatigue, irritability, and other nervous system disturbances, as well as garlic breath odour (IOM, 2000; ATSDR, 2001). Acute exposure by inhalation results in severe respiratory irritation (LaDou, 1997).

Reference Intervals and Body Fluids

Plasma (serum) selenium concentrations are somewhat lower than whole blood levels, typically 56-105 µg/L (0.70-1.30 µmol/L) and 76-140 µg/L (0.96-1.80 µmol/L), respectively (Minoia et al., 1990; Burtis and Ashwood, 1996; Herber et al., 1999). The observed concentrations are suitable indices of both body burden and intake. Urinary selenium can also confirm over exposure, with normal concentrations usually ≤100 µg/L (LaDou, 1997). The relationship of human hair concentrations to body burden is not clear, although animal studies suggest a possible link between dietary intake and nail and hair selenium concentrations (Salbe and Levander, 1990; IOM, 2000). However, there are indications that external deposition of selenium in hair does not occur in absence of a well-delivered point source (Sky-Peck, 1990). Typically, the mean ±sd hair selenium concentrations appear to be 0.60±0.40 µg/g (Sky-Peck, 1990; Lauwerys and Hoet, 2001)

Concluding Remarks

Selenium is typical of essential elements. Both deficiency and excess can be detrimental. Its biochemical role is related to the incorporation of the amino acid selenocysteine into proteins and enzymes. Mechanistically, it seems to facilitate oxygen atom transfer. Its toxicity is likely due to its inorganic forms selenate and selenite.

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Abridged Toxicological Profile and Related Health Issues: Zinc

Revised May 19, 2004

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Zinc

Public Health Issues
- Dietary requirements as an essential element
- Individuals with malabsorption syndromes are susceptible to reduced intake

Occupational Heath Issues
- Metal fume fever (zinc oxide fumes)
- Skin, eye and respiratory irritation (zinc salts)

Acute Poisoning
- Pulmonary edema and pneumonia can develop after excessive exposure by inhalation of zinc salts

Exposure Guidelines
Occupational (respirable: 8-h TLV-TWA)

• Fume of zinc oxide (ACGIH, 2002)	2 mg/m3
• Zinc chloride fume (ACGIH, 2002)	1 mg/m3

General Public

• CCME Community Health Guide for Drinking Water	<5 mg/L
• Recommended Dietary Allowance (IOM, 2002)	8 mg/day (women); 11 mg/day (men)
• Tolerable Upper Dietary Intake Level (IOM, 2002)	40 mg/day (reduction in erythrocyte copper-zinc superoxide dismutase activity as outcome)

Essentiality
There are as many as 100 zinc metalloenzymes and they occur in all six enzyme classes. Zinc-dependent enzyme catalysis is therefore very diverse. rominent examples are: RNA polymerases, alcohol dehydrogenases, carbonic anhydrase, carboxypeptidases, superoxide dismutase, and alkaline phosphatase. rominent zinc-containing proteins are insulin and metallothionein. Zinc insulin is a slow-release hormone and metallothionen is involved in the transport of both copper and zinc. The latter also appears to assist in the detoxification of metals such as cadmium and mercury.

Good dietary sources of zinc are red meat, liver, eggs and certain seafoods, whereas whole grain products contain the element in a less available form (IOM, 2002). Since it is primarily located in the germ and bran portions, about 80% of the zinc is lost in milling. Low iron status appears to increase zinc uptake, while phytate and phosphorus-rich proteins reduce it.

Deficiency Symptoms
Impaired growth is a feature of mild zinc deficiency. Indeed, pregnancy outcome responds to zinc supplementation, as does immune function (IOM, 2002). Clinical symptoms are often quite basic and non-specific: loss of appetite, growth retardation, poor skeletal development, skin changes, reduced healing and immunological abnormalities (e.g., recurrent infections) (Odland et al., 1996; Brody, 1999).

Toxicity

"Adverse effects associated with chronic intake of supplemental zinc include suppression of immune response, decrease in high-density lipoprotein (HDL) cholesterol, and reduced copper status" (IOM, 2002). For acute poisonings, the usual immediate symptoms occur: epigastric pain, nausea, vomiting, abdominal cramps, diarrhea and headaches (IOM, 2002).

Occupationally, the toxicity of zinc compounds appears to be limited to inhalation as the route of exposure. Zinc oxide fume induces a reversible effect referred to as fume fever. Hours after exposure the worker develops headache, fatigue muscle and joint pains, fever chills, profuse cough and chest pain. These symptoms resolve themselves overnight (ATSDR, 1994; La Dou, 1997). Contact with zinc salts (e.g., zinc chloride) may cause skin and eye burns, eczematous dermatitis, while their inhalation irritates the mucous membrane. Pulmonary edema and pneumonia may occur (ATSDR, 1994; LaDou, 1997).

Body Fluids Reference Intervals

Zinc levels in whole blood are considerably higher than in serum: 4100-7600 µg/L (62.7-116.3 µmol/L) compared to 600-1200 µg/L (9.2-18.4 µmol/L) (Minoia *et al*., 1990; Burtis and Ashwood, 1996). There are suggestions that low zinc hair levels reflect low intake. However, the use of hair in establishing body burden needs more research (Sky-Peck, 1990; IOM, 2002). The 95th percentile concentration of zinc in hair is reported as 240 µg/g, with arithmetic mean values around 155 µg/g (children 6-14 years) and 170 µg/g (adults 25-69 years) (Seifert et al., 2000) or slightly higher (Sky-Peck, 1990)

Concluding Remarks

The four to five-fold difference between the tolerable upper dietary intake level (40 mg/day) and the recommended dietary allowance (8-11 mg/day) is consistent with the infrequent occurrence of zinc toxicity due to oral intake. For this reason, environmental exposures to this metal are not likely to be of serious adverse consequence.

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APPENDIX 2

Written Consent forms

INFORMATION SHEET (0 –7 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

Principal Investigators: **Éric Dewailly**, Public Health National Institute of Quebec, Department of Social and Preventive Medicine, Faculty of medicine, Laval University, Quebec (Canada). **Evert Nieboer**, Department of Biochemistry, Occupational Health Program, Institute of Environment and Health McMaster University, Hamilton, Ontario.

Sponsoring organizations: The Cree Nation of Oujé-Bougoumou, the Cree Board of Health & Social Services of James Bay, the Québec Ministry of Health and Social Services

Funding organization: Québec Ministry of Health and Social Services.

Background:

This project is a follow-up to two recent reports about contaminants and health in the Oujé-Bougoumou population:

- the study by Christopher Covel in 2001, and
- the critical review of the Covel report by Dr. Evert Nieboer in April 2002.

The Joe Mann Mine, the Campbell Point Mine and the Copper Rand Mine operated in the Oujé-Bougoumou area. The Covel project took place to see if the tailing piles from these mines, along with the surface water runoff from those tailing piles, might be creating problems for the environment and human health.

The project took samples of sediments and surface water from the Doré, Obatogamau and Chibougamau lakes and from the Nemenjichi River. When these were analyzed, they were found to have levels of arsenic, copper and zinc that were higher than the maximum recommended in the Canadian Environmental Quality Guidelines (CEQGs). The project also took hair samples from 23 Cree living in Oujé-Bougoumou. The analysis of these hair samples showed elevated levels of some toxic metals.

However, many details were missing in the Covel project. For this reason, the Covel report was reviewed by Dr. Nieboer, who identified its strengths and weaknesses. For example, Dr. Nieboer showed that some of the toxic metals (such as mercury) found in the hair samples needed to be further studied; while other metals that were reported present in hair (such as arsenic) are not known to be measurable in hair.

The Cree Nation of Oujé-Bougoumou, the Cree Board of Health and the Government of Québec decided that a comprehensive health study was needed. This more complete study will fill in the details that were missing in the Covel project and will develop an

understanding of whether contaminants from these mines have been harmful to the environment and to the health of people in this region.

The purpose of the present study is:

- to find out if people living in the community of Oujé-Bougoumou have been exposed to different substances related to mine tailings (arsenic, copper, zinc and selenium),
- to understand if some people's lifestyle is exposing them to some toxic substances (for example, smoking exposes people to cadmium and hunting to lead);
- to determine if eating fish is exposing some people to some environmental pollutants (for example, mercury and organochlorines).

The project will also carry out some tests on people's health to understand the quality of their general health. These will include tests for such problems such as anaemia, heart diseases and diabetes.

In order to understand whether people in Oujé-Bougoumou are more exposed to toxic substances than people in other communities, the results from Oujé-Bougoumou will be compared to the results from a similar study carried out with the residents of Nemaska. Nemaska is not close to any mines.

What you and your child will be asked to do to participate in the study

1. Step 1, Day 1:
   - You and your child will be invited to come to the Health (Healing) Center in your community.
   - During this visit, you will be interviewed about your child by a local research interviewer.
   - The interview will last approximately one hour.
   - You will be asked detailed questions about your child's lifestyle, health status and dietary habits.
   - During this visit, in your presence, the research nurse will ask you a few questions about your child's health.
   - During this visit the nurse will take:
     i. a blood sample (3 ml)
     ii. a small hair sample (about the size of a pen)

If you and your child are not able to come to the clinic for the interviews, an interviewer and a nurse can come to your home to ask your child the questions and take his or her blood, hair and urine samples.

Benefits

By participating in this study your child will be helping the community of Oujé-Bougoumou, the Cree Board of Health, the Cree Regional Authority and the Government of Quebec to better understand whether the mines have contaminated the environment and had an effect on people's health. The results of the study will also give you more understanding of any possible risks to your child's own health.

By participating, you will also receive an assessment of your child's general health.

Risks

It is not expected that being in this study will harm your child. However, the fact that your child has been asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed at the end of this form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken.

Confidentiality

All information collected in this study will be kept confidential. Your child's questionnaires and test samples will only be identified with a number (numeric) code. Your child's name will not appear on them. Your child's name will be on the 'master' identification sheets that link his or her name to each of these numbers. These 'master' sheets are kept in a locked file and only the two principal investigators, Dr. Éric Dewailly and Dr. Evert Nieboer, will have access to these files. Once the study has been completed and your child's individual test results sent back to you or to the clinic, these 'master' identification sheets will be destroyed.

While the study is taking place, all questionnaires and test results will be kept in a secure locked file cabinet, first, at your Health Center, and then, at the office of Dr. Dewailly at the Quebec National Institute of Public Health. At the end of the project, all of the data (questionnaires and tests results identified only with a number) will be returned to the Cree Board of Health through the Research Office. Your child's name will not be any materials that are returned. After 5 years, those returned materials will also be destroyed.

Your child's specimens of blood and hair and will be kept at the office of Dr. Dewailly at the Quebec National Institute of Public Health until the final report has been printed, hopefully at the beginning of May 2003. After that, they will be destroyed.

Your child's name will not appear on any publication or report.

Information

When the final report is being printed, a letter will be send to you informing you about your child's test results and telling you who to contact for further information. This letter could be sent to you either at your home, at the clinic of your community or at both places.

The results of the study will be explained in popular language reports that will be widely distributed in the community. You may also obtain a copy of the complete final report.

Withdrawing from the study

You are free to withdraw your child from the study at any time without any ill effect to yourself or your child. Even after you have agreed to have your child participate in the study, you can decide you do not want him or her to continue. For example, you can withdraw after your child has already completed the first interview; or after he or she has completed the second interview. If you decide to have your child stop participating and to withdraw him or her from the study, his or her interviews and medical tests will not be used, but will be destroyed. To withdraw your child from the study, please talk to one of the people listed at the end of this form.

Honorarium

An amount of $20.00 will be given to you as an honorarium for your time and involvement. It will be given to you after your child has completed the second interview.

Contact persons

At any time during the study, you may call the following people to get more information about the study, including copies of the reports; to make any comments about the study; or, to withdraw from the study.

While the study is taking place, please call:

Suzanne Côté, nurse field coordinator:
at the Healing Center in Oujé-Bougoumou call (418) 745-3901 ext. 237
at the Health Center in Nemaska, call (819) 673-2511.

Kenny Mianscum, for the community of Oujé-Bougoumou
at Headquarters Office, Oujé-Bougoumou call (416) 745-3911

Matthew Tanoush, for the community of Nemaska
at Administration Office, Nemaska call (819) 673-2512

After the study has been completed, please call:

Jill Torrie, Public Health Research Office, Cree Board of Health
Call: (514) 861-2352 (31) or e-mail: torrie.jill@ssss.gouv.qc.ca

INFORMED CONSENT FORM (0-7 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

The Research Committee of the Cree Board of Health and Social Services of James Bay and le Comité d'éthique de la recherche de l'Université Laval (Québec, Canada) have reviewed and accepted the study proposal, the questionnaires, the information sheet and this consent form. As well, the Research Ethics Board of McMaster University in Ontario has also been informed about this project.

It is not expected that being in this study will harm your child. However, the fact of being asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed on the information form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken

- I agree to allow my child to complete, with the help of an interviewer, a questionnaire.
- I authorize a research nurse to draw 3 ml of venous blood from my child and to cut a small sample of my child's hair.
- I authorize the research team to perform all the medical tests mentioned in the information sheet on my child's test samples.
- I understand that one risk of having my child's blood drawn is that he or she may develop a light bruise on the site of the puncture. I also understand that the fact of being chosen for this study may cause me and my child some anxiety.
- I understand that some advantages of my child participating in the study are that his or her exposure to environmental contaminants will be evaluated.
- I understand that all information collected during the study will be kept strictly confidential. No names or individual data will be communicated with anyone outside of the study.
- I understand that all of my child's personal medical test results will be sent to me at the end of the study, either at my address or through the local clinic.
- I agree that all procedures included in this study have been explained to me in person, all my questions have been answered and a copy of this document was given to me.

- I understand that I and my child will not suffer any ill effects or lose any benefits if I refuse to allow my child to participate in this study or if I first agree to allow him or her to participate and then later withdraw him or her.
- I understand that if any medical problem is found, I will be informed and a report will be sent to the community clinic staff.

- **I have explained all aspects of this study to my child and I have discussed how he or she feels about participating. I believe my child is comfortable with participating in this study.**

- **I have read and understood what is involved in the study and hereby consent and voluntarily agree to:**
  allow my child to participate in this study Yes ☐

- **I authorize a research nurse to review my child's medical file for detailed information about his or her health status** (you do not need to agree to this to have your child participate in this research project. If you accept, you can change your mind at any time without any ill effect to you or your child.)

  Yes ☐ **No ☐**

_____
Name of the participant

_____	_____	__/____/____
Name of one parent/tutor (the participant is under 18 years old)	Signature of one parent/tutor (the participant is under 18 years old)	Date (y/m/d)
_____	_____	____/____/____
Name of the witness	Signature	Date (y/m/d)
_____	_____	____/____/____
Name of the principal investigator /or his designee	Signature	Date (y/m/d)

The informed consent form has been read and explained to the participant by the research interviewer:

Name: _____

Address: _____

Phone number: _____

Signature: _____

INFORMATION SHEET (8-14 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

Principal Investigators: **Éric Dewailly**, Public Health National Institute of Quebec, Department of Social and Preventive Medicine, Faculty of medicine, Laval University, Quebec (Canada). **Evert Nieboer**, Department of Biochemistry, Occupational Health Program, Institute of Environment and Health McMaster University, Hamilton, Ontario.

Sponsoring organizations: The Cree Nation of Oujé-Bougoumou, the Cree Board of Health & Social Services of James Bay, the Québec Ministry of Health and Social Services

Funding organization: Québec Ministry of Health and Social Services.

Background:

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However, many details were missing in the Covel project. For this reason, the Covel report was reviewed by Dr. Nieboer, who identified its strengths and weaknesses. For example, Dr. Nieboer showed that some of the toxic metals (such as mercury) found in the hair samples needed to be further studied; while other metals that were reported present in hair (such as arsenic) are not known to be measurable in hair.

The Cree Nation of Oujé-Bougoumou, the Cree Board of Health and the Government of Québec decided that a comprehensive health study was needed. This more complete study will fill in the details that were missing in the Covel project and will develop an

understanding of whether contaminants from these mines have been harmful to the environment and to the health of people in this region.

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- to understand if some people's lifestyle is exposing them to some toxic substances (for example, smoking exposes people to cadmium and hunting to lead);
- to determine if eating fish is exposing some people to some environmental pollutants (for example, mercury and organochlorines).

The project will also carry out some tests on people's health to understand the quality of their general health. These will include tests for such problems such as anaemia, heart diseases and diabetes.

In order to understand whether people in Oujé-Bougoumou are more exposed to toxic substances than people in other communities, the results from Oujé-Bougoumou will be compared to the results from a similar study carried out with the residents of Nemaska. Nemaska is not close to any mines.

What you and your child will be asked to do to participate in the study

2. Step 1, Day 1:
   - You and your child will be invited to come to the Health (Healing) Center in your community.
   - During this first visit, in your presence, your child will be interviewed by a local research interviewer.
   - The interview will last approximately one hour.
   - Your child will be asked detailed questions about his or her lifestyle, health status and dietary habits.
   - At the end of your interview, the interviewer will give you a collection cup for your child's urine sample

3. Step 2, Day 2:
   - When your child wakes up on Day 2, he or she will be asked to take a 'first-in-the-morning' urine sample (80ml) in the collection cup.

4. Step 3, Day 2:
   - Then you and your child will return to the clinic with the urine sample for your second visit.
   - This visit will take approximately 15 minutes.
   - During this visit, in your presence, the research nurse will ask your child a few questions about his or her health.
   - During this visit the nurse will take:
     - a blood sample (22 ml or approximately 1½ tablespoons)
     - a small hair sample (about the size of a pen)
     - your child's body weight
     - your child's height
     - your child's waist and hip circumferences
     - your child's blood pressure.

If you and your child are not able to come to the clinic for the interviews, an interviewer and a nurse can come to your home to ask your child the questions and take his or her blood, hair and urine samples.

Benefits

By participating in this study your child will be helping the community of Oujé-Bougoumou, the Cree Board of Health, the Cree Regional Authority and the Government of Quebec to better understand whether the mines have contaminated the environment and had an effect on people's health. The results of the study will also give you more understanding of any possible risks to your child's own health.

By participating, you will also receive an assessment of your child's general health.

Risks

It is not expected that being in this study will harm your child. However, the fact that your child has been asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed at the end of this form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken.

Confidentiality

All information collected in this study will be kept confidential. Your child's questionnaires and test samples will only be identified with a number (numeric) code. Your child's name will not appear on them. Your child's name will be on the 'master' identification sheets that link his or her name to each of these numbers. These 'master' sheets are kept in a locked file and only the two principal investigators, Dr. Éric Dewailly and Dr. Evert Nieboer, will have access to these files. Once the study has been completed and your child's individual test results sent back to you or to the clinic, these 'master' identification sheets will be destroyed.

While the study is taking place, all questionnaires and test results will be kept in a secure locked file cabinet, first, at your Health Center, and then, at the office of Dr. Dewailly at the Quebec National Institute of Public Health. At the end of the project, all of the data (questionnaires and tests results identified only with a number) will be returned to the Cree Board of Health through the Research Office. Your child's name will not be any materials that are returned. After 5 years, those returned materials will also be destroyed.

Your child's specimens of blood, hair and urine will be kept at the office of Dr. Dewailly at the Quebec National Institute of Public Health until the final report has been printed, hopefully at the beginning of May 2003. After that, they will be destroyed.

Your child's name will not appear on any publication or report.

Information

When the final report is being printed, a letter will be send to you informing you about your child's test results and telling you who to contact for further information. This letter could be sent to you either at your home, at the clinic of your community or at both places.

The results of the study will be explained in popular language reports that will be widely distributed in the community. You may also obtain a copy of the complete final report.

Withdrawing from the study

You are free to withdraw your child from the study at any time without any ill effect to yourself or your child. Even after you have agreed to have your child participate in the study, you can decide you do not want him or her to continue. For example, you can withdraw after your child has already completed the first interview; or after he or she has completed the second interview. If you decide to have your child stop participating and to withdraw him or her from the study, his or her interviews and medical tests will not be used, but will be destroyed. To withdraw your child from the study, please talk to one of the people listed at the end of this form.

Honorarium

An amount of $20.00 will be given to you as an honorarium for your time and involvement. It will be given to you after your child has completed the second interview/research project.

Contact persons

At any time during the study, you may call the following people to get more information about the study, including copies of the reports; to make any comments about the study; or, to withdraw from the study.

While the study is taking place, please call:

Suzanne Côté, nurse field coordinator:
at the Healing Center in Oujé-Bougoumou call (418) 745-3901 ext. 237
at the Health Center in Nemaska, call (819) 673-2511.

Kenny Mianscum, for the community of Oujé-Bougoumou
at Headquarters Office, Oujé-Bougoumou call (416) 745-3911

Matthew Tanoush, for the community of Nemaska
at Administration Office, Nemaska call (819) 673-2512

After the study has been completed, please call:

Jill Torrie, Public Health Research Office, Cree Board of Health
Call: (514) 861-2352 (31) or e-mail: torrie.jill@ssss.gouv.qc.ca

INFORMED CONSENT FORM (8-14 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

The Research Committee of the Cree Board of Health and Social Services of James Bay and le Comité d'éthique de la recherche de l'Université Laval (Québec, Canada) have reviewed and accepted the study proposal, the questionnaires, the information sheet and this consent form. As well, the Research Ethics Board of McMaster University in Ontario has also been informed about this project.

It is not expected that being in this study will harm your child. However, the fact of being asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed on the information form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken

- I agree to allow my child to complete, with the help of an interviewer, a questionnaire.
- I agree to allow my child to provide a 80 ml sample of his or her first morning urine.
- I authorize a research nurse to draw 22 ml (approximately 1½ tablespoons) of venous blood from my child and to cut a small sample of my child's hair.
- I authorize the research team to perform all the medical tests mentioned in the information sheet on my child's test samples.
- I understand that one risk of having my child's blood drawn is that he or she may develop a light bruise on the site of the puncture. I also understand that the fact of being chosen for this study may cause me and my child some anxiety.
- I understand that some advantages of my child participating in the study are that his or her exposure to environmental contaminants will be evaluated.
- I understand that all information collected during the study will be kept strictly confidential. No names or individual data will be communicated with anyone outside of the study.
- I understand that all of my child's personal medical test results will be sent to me at the end of the study, either at my address or through the local clinic.

- I agree that all procedures included in this study have been explained to me in person, all my questions have been answered and a copy of this document was given to me.
- I understand that I and my child will not suffer any ill effects or lose any benefits if I refuse to allow my child to participate in this study or if I first agree to allow him or her to participate and then later withdraw him or her.
- I understand that if any medical problem is found, I will be informed and a report will be sent to the community clinic staff.

- **I have explained all aspects of this study to my child and I have discussed how he or she feels about participating. I believe my child is comfortable with participating in this study.**

- **I have read and understood what is involved in the study and hereby consent and voluntarily agree to:**
  allow my child to participate in this study Yes ☐

- **I authorize a research nurse to review my child's medical file for detailed information about his or her health status** (you do not need to agree to this to have your child participate in this research project. If you accept, you can change your mind at any time without any ill effect to you or your child.)

  Yes ☐ **No ☐**

_____
Name of the participant

_____	_____	___/___/___
Name of one parent/tutor (the participant is under 18 years old)	Signature of one parent/tutor (the participant is under 18 years old)	Date (y/m/d)
_____	_____	___/___/___
Name of the witness	Signature	Date (y/m/d)
_____	_____	___/___/___
Name of the principal investigator /or his designee	Signature	Date (y/m/d)

The informed consent form has been read and explained to the participant by the research interviewer:

Name: _____

Address: _____

Phone number: _____

Signature: _____

INFORMATION SHEET (15 – 17 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

Principal Investigators: **Éric Dewailly**, Public Health National Institute of Quebec, Department of Social and Preventive Medicine, Faculty of medicine, Laval University, Quebec (Canada). **Evert Nieboer**, Department of Biochemistry, Occupational Health Program, Institute of Environment and Health McMaster University, Hamilton, Ontario.

Sponsoring organizations: The Cree Nation of Oujé-Bougoumou, the Cree Board of Health & Social Services of James Bay, the Québec Ministry of Health and Social Services

Funding organization: Québec Ministry of Health and Social Services.

Background:

This project is a follow-up to two recent reports about contaminants and health in the Oujé-Bougoumou population:

- the study by Christopher Covel in 2001, and
- the critical review of the Covel report by Dr. Evert Nieboer in April 2002.

The Joe Mann Mine, the Campbell Point Mine and the Copper Rand Mine operated in the Oujé-Bougoumou area. The Covel project took place to see if the tailing piles from these mines, along with the surface water runoff from those tailing piles, might be creating problems for the environment and human health.

The project took samples of sediments and surface water from the Doré, Obatogamau and Chibougamau lakes and from the Nemenjichi River. When these were analyzed, they were found to have levels of arsenic, copper and zinc that were higher than the maximum recommended in the Canadian Environmental Quality Guidelines (CEQGs). The project also took hair samples from 23 Cree living in Oujé-Bougoumou. The analysis of these hair samples showed elevated levels of some toxic metals.

However, many details were missing in the Covel project. For this reason, the Covel report was reviewed by Dr. Nieboer, who identified its strengths and weaknesses. For example, Dr. Nieboer showed that some of the toxic metals (such as mercury) found in the hair samples needed to be further studied; while other metals that were reported present in hair (such as arsenic) are not known to be measurable in hair.

The Cree Nation of Oujé-Bougoumou, the Cree Board of Health and the Government of Québec decided that a comprehensive health study was needed. This more complete study

will fill in the details that were missing in the Covel project and will develop an understanding of whether contaminants from these mines have been harmful to the environment and to the health of people in this region.

The purpose of the present study is:

- to find out if people living in the community of Oujé-Bougoumou have been exposed to different substances related to mine tailings (arsenic, copper, zinc and selenium),
- to understand if some people's lifestyle is exposing them to some toxic substances (for example, smoking exposes people to cadmium and hunting to lead);
- to determine if eating fish is exposing some people to some environmental pollutants (for example, mercury and organochlorines).

The project will also carry out some tests on people's health to understand the quality of their general health. These will include tests for such problems such as anaemia, heart diseases and diabetes.

In order to understand whether people in Oujé-Bougoumou are more exposed to toxic substances than people in other communities, the results from Oujé-Bougoumou will be compared to the results from a similar study carried out with the residents of Nemaska. Nemaska is not close to any mines.

What you and your child will be asked to do to participate in the study

Step 1, Day 1:

- You and your child will be invited to come to the Health (Healing) Center in your community.
- During this first visit your child will be interviewed by a local research interviewer.
- The interview will last approximately one hour.
- Your child will be asked detailed questions about his or her lifestyle, health status and dietary habits.

5. Step 2, Day 1:
   - At the end of your interview, the interviewer will give your child instructions about Step 2.
   - Your child will also be given a collection cup for a urine sample.

6. Step 3, Day 2:
   - When your child wakes up on Day 2, he or she will be asked to take a 'first-in-the-morning' urine sample (80ml) in the collection cup.

7. Step 4, Day 2:
   - Then you and your child will return to the clinic with the urine sample for your second visit.
   - This visit will take approximately 15 minutes.
   - During this visit, the research nurse will ask your child a few questions about his or her health.
   - During this visit the nurse will take:
     i. a blood sample (37 ml or approximately 2½ tablespoons)
     ii. a small hair sample (about the size of a pen)
     iii. your child's body weight
     iv. your child's height
     v. your child's waist and hip circumferences
     vi. your child's blood pressure.

If you and your child are not able to come to the clinic for the interviews, an interviewer and a nurse can come to your home to ask your child the questions and take his or her blood, hair and urine samples.

Benefits

By participating in this study your child will be helping the community of Oujé-Bougoumou, the Cree Board of Health, the Cree Regional Authority and the Government of Quebec to better understand whether the mines have contaminated the environment and had an effect on people's health. The results of the study will also give you more understanding of any possible risks to your child's own health.

By participating, you will also receive an assessment of your child's general health.

Risks

It is not expected that being in this study will harm your child. However, the fact that your child has been asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed at the end of this form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken.

Confidentiality

All information collected in this study will be kept confidential. Your child's questionnaires and test samples will only be identified with a number (numeric) code. Your child's name will not appear on them. Your child's name will be on the 'master' identification sheets that link his or her name to each of these numbers. These 'master' sheets are kept in a locked file and only the two principal investigators, Dr. Éric Dewailly and Dr. Evert Nieboer, will have access to these files. Once the study has been completed and your child's individual test results sent back to you or to the clinic, these 'master' identification sheets will be destroyed.

While the study is taking place, all questionnaires and test results will be kept in a secure locked file cabinet, first, at your Health Center, and then, at the office of Dr. Dewailly at the Quebec National Institute of Public Health. At the end of the project, all of the data (questionnaires and tests results identified only with a number) will be returned to the Cree Board of Health through the Research Office. Your child's name will not be on any materials that are returned. After 5 years, those returned materials will also be destroyed.

Your child's specimens of blood, hair and urine will be kept at the office of Dr. Dewailly at the Quebec National Institute of Public Health until the final report has been printed, hopefully at the beginning of May 2003. After that, they will be destroyed.

Your child's name will not appear on any publication or report.

Information

When the final report is being printed, a letter will be send to you informing you about your child's test results and telling you who to contact for further information. This letter could be sent to you either at your home, at the clinic of your community or at both places.

The results of the study will be explained in popular language reports that will be widely distributed in the community. You may also obtain a copy of the complete final report.

Withdrawing from the study

You are free to withdraw your child from the study at any time without any ill effect to yourself or your child. Even after you have agreed to have your child participate in the study, you can decide you do not want him or her to continue. For example, you can withdraw after your child has already completed the first interview; or after he or she has completed the second interview. If you decide to have your child stop participating and to withdraw him or her from the study, his or her interviews and medical tests will not be used, but will be destroyed. To withdraw your child from the study, please talk to one of the people listed at the end of this form.

Honorarium

An amount of $20.00 will be given to you as an honorarium for your time and involvement. It will be given to you after your child has completed the second interview.

Contact persons

At any time during the study, you may call the following people to get more information about the study, including copies of the reports; to make any comments about the study; or, to withdraw from the study.

While the study is taking place, please call:

Suzanne Côté, nurse field coordinator:
at the Healing Center in Oujé-Bougoumou call (418) 745-3901 ext. 237
at the Health Center in Nemaska, call (819) 673-2511.

Kenny Mianscum, for the community of Oujé-Bougoumou
at Headquarters Office, Oujé-Bougoumou call (416) 745-3911

Matthew Tanoush, for the community of Nemaska
at Administration Office, Nemaska call (819) 673-2512

After the study has been completed, please call:

Jill Torrie, Public Health Research Office, Cree Board of Health
Call: (514) 861-2352 (31) or e-mail: torrie.jill@ssss.gouv.qc.ca

INFORMED CONSENT FORM (15-17 years old)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

The Research Committee of the Cree Board of Health and Social Services of James Bay and le Comité d'éthique de la recherche de l'Université Laval (Québec, Canada) have reviewed and accepted the study proposal, the questionnaires, the information sheet and this consent form. As well, the Research Ethics Board of McMaster University in Ontario has also been informed about this project.

It is not expected that being in this study will harm your child. However, the fact of being asked to participate in a study of possible exposure to environmental contaminants might cause you and your child some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed on the information form.

When your child gives the blood sample, he or she may develop a light bruise where the sample is taken

- I agree to allow my child to complete, with the help of an interviewer, a questionnaire.
- I agree to allow my child to provide a 80 ml sample of his or her first morning urine.
- I authorize a research nurse to draw 37 ml (approximately 2½ tablespoons) of venous blood from my child and to cut a small sample of my child's hair.
- I authorize the research team to perform all the medical tests mentioned in the information sheet on my child's test samples.
- I understand that one risk of having my child's blood drawn is that he or she may develop a light bruise on the site of the puncture. I also understand that the fact of being chosen for this study may cause me and my child some anxiety.
- I understand that some advantages of my child participating in the study are that his or her exposure to environmental contaminants will be evaluated and his or her general health will be assessed.
- I understand that all information collected during the study will be kept strictly confidential. No names or individual data will be communicated with anyone outside of the study.
- I understand that all of my child's personal medical test results will be sent to me at the end of the study, either at my address or through the local clinic.

- I agree that all procedures included in this study have been explained to me and my child in person, all our questions have been answered and a copy of this document was given to me.
- I understand that I and my child will not suffer any ill effects or lose any benefits if I refuse to allow my child to participate in this study or if I first agree to allow him or her to participate and then later withdraw him or her.
- I understand that if any medical problem is found, I will be informed and a report will be sent to the community clinic staff.

- **I have explained all aspects of this study to my child and I have discussed how he or she feels about participating. I believe my child is comfortable with participating in this study.**

- **I have read and understood what is involved in the study and hereby consent and voluntarily agree to:**
  allow my child to participate in this study Yes ☐

- **I authorize a research nurse to review my child's medical file for detailed information about his or her health status** (you do not need to agree to this to have your child participate in this research project. If you accept, you can change your mind at any time without any ill effect to you or your child.)

  Yes ☐ **No ☐**

_____	_____	____/____/____
Name of one parent/tutor (the participant is under 18 years old)	Signature of one parent/tutor (the participant is under 18 years)	Date (y/m/d)
_____	_____	____/____/____
Name of the witness	Signature	Date (y/m/d)
_____	_____	____/____/____
Name of the principal investigator /or his designee	Signature	Date (y/m/d)

My mother or father and the people involved with the study have explained it to me and I agree to participate.

_____	_____	____/____/____
Name of the participant (child)	Signature	Date (y/m/d)

The informed consent form has been read and explained to the participant by the research interviewer:

Name: _____

Address: _____

Phone number: _____

Signature: _____

INFORMATION SHEET (18 years old and over)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

Principal Investigators: **Éric Dewailly**, Public Health National Institute of Quebec, Department of Social and Preventive Medicine, Faculty of medicine, Laval University, Quebec (Canada). **Evert Nieboer**, Department of Biochemistry, Occupational Health Program, Institute of Environment and Health McMaster University, Hamilton, Ontario.

Sponsoring organizations: The Cree Nation of Oujé-Bougoumou, the Cree Board of Health & Social Services of James Bay, the Québec Ministry of Health and Social Services

Funding organization: Québec Ministry of Health and Social Services.

Background:

This project is a follow-up to two recent reports about contaminants and health in the Oujé-Bougoumou population:

- the study by Christopher Covel in 2001, and
- the critical review of the Covel report by Dr. Evert Nieboer in April 2002.

The Joe Mann Mine, the Campbell Point Mine and the Copper Rand Mine operated in the Oujé-Bougoumou area. The Covel project took place to see if the tailing piles from these mines, along with the surface water runoff from those tailing piles, might be creating problems for the environment and human health.

The project took samples of sediments and surface water from the Doré, Obatogamau and Chibougamau lakes and from the Nemenjichi River. When these were analyzed, they were found to have levels of arsenic, copper and zinc that were higher than the maximum recommended in the Canadian Environmental Quality Guidelines (CEQGs). The project also took hair samples from 23 Cree living in Oujé-Bougoumou. The analysis of these hair samples showed elevated levels of some toxic metals.

However, many details were missing in the Covel project. For this reason, the Covel report was reviewed by Dr. Nieboer, who identified its strengths and weaknesses. For example, Dr. Nieboer showed that some of the toxic metals (such as mercury) found in the hair samples needed to be further studied; while other metals that were reported present in hair (such as arsenic) are not known to be measurable in hair.

The Cree Nation of Oujé-Bougoumou, the Cree Board of Health and the Government of Québec decided that a comprehensive health study was needed. This more complete study will fill in the details that were missing in the Covel project and will develop an understanding of whether contaminants from these mines have been harmful to the environment and to the health of people in this region.

The purpose of the present study is:

- to find out if people living in the community of Oujé-Bougoumou have been exposed to different substances related to mine tailings (arsenic, copper, zinc and selenium),
- to understand if some people's lifestyle is exposing them to some toxic substances (for example, smoking exposes people to cadmium and hunting to lead);
- to determine if eating fish is exposing some people to some environmental pollutants (for example, mercury and organochlorines).

The project will also carry out some tests on people's health to understand the quality of their general health. These will include tests for such problems such as anaemia, heart diseases and diabetes.

In order to understand whether people in Oujé-Bougoumou are more exposed to toxic substances than people in other communities, the results from Oujé-Bougoumou will be compared to the results from a similar study carried out with the residents of Nemaska. Nemaska is not close to any mines.

What you will be asked to do to participate in the study

8. Step 1, Day 1:
   - You will be invited to come to the Health (Healing) Center in your community.
   - During this first visit you will take part in a face-to-face interview with a local interviewer.
   - The interview will last approximately one hour.
   - It will involve detailed questions about your lifestyle, health status and dietary habits.

9. Step 2, Day 1:
   - At the end of your interview, the interviewer will give you instructions about Step 2.
   - You will also be given a collection cup for your urine sample.

10. Step 3, Day 2:
    - When you wake up on Day 2, you will be asked to take a 'first-in-the-morning' urine sample (80ml) in the collection cup.

11. Step 4, Day 2:
    - Then you will return to the clinic with your urine sample for your second visit.
    - This visit will take approximately 15 minutes.
    - During this visit, the research nurse will ask you a few questions about your health.
    - During this visit the nurse will take:
      i. a blood sample (37 ml or approximately 2½ tablespoons)
      ii. a small hair sample (about the size of a pen)
      iii. your body weight
      iv. your height
      v. your waist and hip circumferences
      vi. your blood pressure.

If you are not able to come to the clinic for the interviews, an interviewer and a nurse can come to your home to ask you the questions and take your blood, hair and urine samples.

Benefits

By participating in this study you will be helping the community of Oujé-Bougoumou, the Cree Board of Health, the Cree Regional Authority and the Government of Quebec to better understand whether the mines have contaminated the environment and had an effect on people's health. The results of the study will also give you more understanding of any possible risks to your own health.

By participating, you will also receive an assessment of your general health.

Risks

It is not expected that being in this study will harm you. However, the fact of being asked to participate in a study of possible exposure to environmental contaminants might cause you some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed at the end of this form.

When you give the blood sample, you may develop a light bruise where the sample is taken.

Confidentiality

All information collected in this study will be kept confidential. Your questionnaires and test samples will only be identified with a number (numeric) code. Your name will not appear on them. Your name will be on the 'master' identification sheets that link your name to each of these numbers. These 'master' sheets are kept in a locked file and only the two principal investigators, Dr. Éric Dewailly and Dr. Evert Nieboer, will have access to these files. Once the study has been completed and your individual test results sent back to you or to the clinic, these 'master' identification sheets will be destroyed.

While the study is taking place, all questionnaires and test results will be kept in a secure locked file cabinet, first, at your Health Centre, and then, at the office of Dr. Dewailly at the Quebec National Institute of Public Health. At the end of the project, all of the data (questionnaires and tests results identified only with a number) will be returned to the Cree Board of Health through the Research Office. Your name will not be on any materials that are returned. After 5 years, those returned materials will also be destroyed.

Your specimens of blood, hair and urine will be kept at the office of Dr. Dewailly at the Quebec National Institute of Public Health until the final report has been printed, hopefully at the beginning of May 2003. After that, they will be destroyed.

Your name will not appear on any publication or report.

Information

When the final report is being printed, a letter will be send to you informing you about your test results and telling you who to contact for further information. This letter could be sent to you either at your home, at the clinic of your community or at both places.

The results of the study will be explained in popular language reports that will be widely distributed in the community. You may also obtain a copy of the complete final report.

Withdrawing from the study

You are free to withdraw from the study at any time without any ill effect to yourself. Even after you have agreed to participate in the study, you can decide you do not want to continue. For example, you can withdraw after you have already completed the first interview or after you have completed the second interview. If you decide to stop participating and to withdraw from the study, your interviews and your medical tests will not be used, but will be destroyed. To withdraw from the study, please talk to one of the people listed at the end of this form.

Honorarium

An amount of $20.00 will be given to you as an honorarium for your time and involvement. It will be given to you after you have completed the second interview.

Contact persons

At any time during the study, you may call the following people to get more information about the study, including copies of the reports; to make any comments about the study; or, to withdraw from the study.

While the study is taking place, please call:

Suzanne Côté, nurse field coordinator:
at the Healing Center in Oujé-Bougoumou call (418) 745-3901 ext. 237
at the Health Center in Nemaska, call (819) 673-2511.

Kenny Mianscum, for the community of Oujé-Bougoumou
at Headquarters Office, Oujé-Bougoumou call (416) 745-3911

Matthew Tanoush, for the community of Nemaska
at Administration Office, Nemaska call (819) 673-2512

After the study has been completed, please call:

Jill Torrie, Public Health Research Office, Cree Board of Health
Call: (514) 861-2352 (31) or e-mail: torrie.jill@ssss.gouv.qc.ca

INFORMED CONSENT FORM (18 years old and over)

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

The Research Committee of the Cree Board of Health and Social Services of James Bay and le Comité d'éthique de la recherche de l'Université Laval (Québec, Canada) have reviewed and accepted the study proposal, the questionnaires, the information sheet and this consent form. As well, the Research Ethics Board of McMaster University in Ontario has also been informed about this project.

It is not expected that being in this study will harm you. However, the fact of being asked to participate in a study of possible exposure to environmental contaminants might cause you some anxiety. If you experience this, please talk to someone involved with the study about your concerns. Their names are listed on the information form.

When you give the blood sample, you may develop a light bruise where the sample is taken

- I agree to complete, with the help of an interviewer, a questionnaire.
- I agree to provide a 80 ml sample of my first morning urine.
- I authorize a research nurse to draw 37 ml (approximately 2½ tablespoons) of venous blood and to cut a small sample of my hair.
- I authorize the research team to perform all the medical tests mentioned in the information sheet on my tests samples.
- I understand that one risk of having my blood drawn is that I may develop a light bruise on the site of the puncture. I also understand that the fact of being chosen for this study may cause me some anxiety.
- I understand that some advantages of participating in the study are that my exposure to environmental contaminants will be evaluated and my general health will be assessed.
- I understand that all information collected during the study will be kept strictly confidential. No names or individual data will be communicated with anyone outside of the study.
- I understand that all of my personal medical test results will be sent to me at the end of the study, either at my address or through the local clinic.

- I agree that all procedures included in this study have been explained to me in person, all my questions have been answered and a copy of this document was given to me.
- I understand that I will not suffer any ill effects or lose any benefits if I refuse to participate in this study or if I first agree to participate and then later withdraw.
- I understand that if any medical problem is found, I will be informed and a report will be sent to the community clinic staff.
- **have read and understood what is involved in the study and hereby consent and voluntarily agree to:**
  participate in this study **Yes** ☐

- **I authorize a research nurse to review my medical file for detailed information about my health status** (you do not need to agree to this to participate in this research project. If you accept, you can change your mind at any time without any ill effect to you.)

  Yes ☐ **No** ☐

_____	_____	____/____/____
Name of the participant	Signature	Date (y/m/d)
_____	_____	____/____/____
Name of the witness	Signature	Date (y/m/d)
_____	_____	____/____/____
Name of the principal investigator /or his designee	Signature	Date (y/m/d)

The informed consent form has been read and explained to the participant by the research interviewer:

Name: _____

Address: _____

Phone number: _____

Signature: _____

APPENDIX 3

Specimen and Measurement Record Sheet

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree Population to Mine Tailings Residues

TO BE COMPLETED BY THE NURSE Reference number: Id. |___|___|___|

Group: Id. |___| groups **1**=0-14 years old,
2=only women 15-39,
Age: |__|__| **3**=only men 15-39,
4=40 and over

1. Collection of blood samples?

Yes |__| →**How many hours since the last meal?** _____hours. if under 0-14 years old na |__|

No |__| →**If NO**, give the reason: _____

Nurse initiales: _____

2. Measurements of blood pressure if 0-7 years old na |__|

Blood pressure (mmHg)**:**

Systolic: _____ Diastolic**:** _____ ***Nurse initiales:*** _____

3. Anthropometric measurements: if 0-7 years old na |__|

Weight : _____kg Waist girth :_____cm

Height : _____cm Hip girth :_____cm

Nurse initiales: _____

6. Collection of urinary sample? if 0-7 years old na |__|

Yes |__| No |__| →**If NO**, give the reason : _____

Nurse initiales: _____

7. Collection of hair?

Yes |__| No |__| →**If NO**, give the reason : _____

Nurse initiales: _____

8. The health questionnaire (nurse section) is completed yes |__| no |__|

→**If NO**, give the reason: _____

Thank you very much for your participation and collaboration

APPENDIX 4

Exposure, Lifestyle, Health and Food Frequency Questionnaires

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree population to Mine Tailings Residues Questionnaire

Subject Identification

Last Name: _____

First Name: _____

Middle Name (if applicable): _____

House Number: _____

Address: _____

Telephone (s) number (s): (_____)- _____-_____

Community: _____

Name of guardian (for minors): _____

At the end of the study, your results can be mail to:

Your home **Yes** ☐ 1 **No** ☐ 0

To the clinic **Yes** ☐ 1 **No** ☐ 0

Both places **Yes** ☐ 1 **No** ☐ 0

Exposure and Preliminary Health Assessments of the Oujé-Bougoumou Cree population to Mine Tailings Residues Questionnaire

Reference number:	Id. |___|___|___|
Group:	1.0-14 years ☐ 1 2.only women 15-39 ☐ 2 3.only men 15-39 ☐ 3 4.40 and over ☐ 4
Date of the interview:	| 2 | 0 | 0 | 2 | |___|___| |___|___| Year Month Day
The interview is held in:	☐ 1 English ☐ 2 Cree ☐ 3 French
Name of the interpreter:	

Thanks for agreeing to answer the following questions.

I would like to ask you some questions about you, your household and lifestyle

Write *the suitable answer or* ***notch*** *the corresponding box or* ***circle*** *the answer*
If the participant is a baby, indicate na (not applicable) when appropriate

Socio-demographic information

1. Gender:

Male ☐ 1
Female ☐ 2

2. What is your birth date?

Date:_____ / _____ / _____ → How old are you? |__|__| years

Year month day

3. What is your current marital status?

Not applicable (children)	☐ 1
Never married and not living with someone as a couple	☐ 2
Divorced/separated and not living with someone as a couple	☐ 3
Married or living with someone as a couple	☐ 4
Widow/widower and not living with someone as a couple	☐ 5

4. What language do you usually speak at home?

Cree ☐ 1
English ☐ 2
French ☐ 3
All of them ☐ 4
2 of them ☐ 5 → specify:_____

5. How many persons live in your household?

Number of adults: _____
Number of children: _____

6. What is the highest level of schooling you have completed?

Not applicable	☐ 1
No formal schooling	☐ 2
Some years of elementary school	☐ 3
Elementary school completed	☐ 4
Some years of secondary school	☐ 5
Secondary school completed	☐ 6
Partial training in community college (a trade school or a private commercial college, a technical institute, a CEGEP, a nursing school or a normal school)	☐ 7
Diploma or certificate from a community college (a trade school or a private commercial college, a technical institute, a CEGEP, a nursing school or a normal school)	☐ 8
Some university (not completed)	☐ 9
University degrees completed (Certificate, Bachelor, Masters, Ph.D)	☐ 10
Refused	☐ 11

7. Which of the following best describes your present working status?

Not applicable	☐ 1
Student	☐ 2
Work full time	☐ **3**
Work part time	☐ **4**
Work occasionally	☐ **5**
Income Security Program	☐ **6**
Housework	☐ **7**
Retired or on pension	☐ **8**
Unemployment insurance	☐ **9**
Social welfare	☐ **10**
Not working for health reasons	☐ **11→ specify:**_____
Other	☐ **12→ specify:**_____

8. Do you work at the present time?

Not applicable ☐ **1**

Yes ☐ **2** → **What kind of work do you do? (Give full description)**

_____

No ☐ **3**

9. Do you have to follow some health and safety guidelines for your work?

Not applicable ☐ **1**

Yes ☐ **2** → **specify which ones?**_____

No ☐ **3**

The following questions concern your domestic house/apartment

10. Where do you live?

In a house	☐ 1
In an apartment	☐ 2
In an elders residence	☐ 3
In an elders apartments	☐ 4
Other	☐ 5 → **specify:** _____

11. How long have you been living in your house/apartment?

Months:/_____/ Years: /_____/ Don't know: ☐ 9

Theses questions do not apply to the people from Oujé-Bougoumou
(for people from Oujé-Bougoumou go to question 17)

12. How long ago was your house/apartment built?

Months:/_____/ Years: /_____/ Don't know: ☐ 9

13. Has your house/apartment been recently renovated?

Yes ☐ 1 → **specify the type of renovation and indicate the year** (painting, typing, insulation)?

_____

No ☐ 2
I don't know ☐ 3

14. Do you know the type of building material used for your house/apartment?

Yes ☐ 1 → **specify**: _____
No ☐ 2
I don't know ☐ 3

15. Do you know the type of material used for your drinking-water piping (copper, PVC, etc)?

Yes ☐ 1 → **specify**: _____
No ☐ 2
I don't know ☐ 3

16. Which heating system do you use in your house/apartment?

Electric ☐ 1
Oil ☐ 2
Gas ☐ 3
Firewood ☐ 4
Hot Water ☐ 5 → **If so, is it from village central heating?** Yes ☐ 1 No ☐ 2
Other ☐ 6 → **specify:** _____

17. Are there any pets living in your house/apartment?

Yes ☐ 1 → what kind of pets: _____
how many pets: _____
No ☐ 2

18. Do you drink water from the tap in your house?

Not applicable ☐ 1
I only drink tap water when in the community ☐ 2
I drink tap water most of the time ☐ 3
I drink tap water only occasionally ☐ 4
I never drink tap water ☐ 5

(Unless replying not applicable or only tap water to the above question, **ask**)
→ **When in the community, do you sometimes drink water from: (use the map)?**

Bottled water ☐ 1
From a spring ☐ 2 → **indicate from where:**_____
From a lake/river ☐ 3 → **indicate from where:**_____

→ **When in the bush, do you drink water from: (use the map)?**

Bottled water ☐ 1
From a spring ☐ 2 → **indicate from where:**_____
From a lake/river ☐ 3 → **indicate from where:**_____

19. How many glasses of water do you have a day (include hot or cold drinks)?

Not applicable ☐ 1

Number of glasses/cups **/_____/**

Activities in the bush

The following questions concern some lifestyle habits

20. How many days, weeks or months per season have you spent in the bush during the last year?

	Days	Weeks	Months	None
Summer	/_____/	/_____/	/_____/	☐
Spring	/_____/	/_____/	/_____/	☐
Winter	/_____/	/_____/	/_____/	☐
Fall	/_____/	/_____/	/_____/	☐

21. Do you hunt?

Yes ☐ 1 → **if yes, do you use a gun?** Yes ☐ 1 → **what kind of bullets do you use?**

No ☐ 2

- Lead bullets ☐ 1
- Steel bullets ☐ 2
- Lead shells ☐ 3
- Steel shells ☐ 4
- Some of the above ☐ 5 → specify: ___
- All of the above ☐ 6
- Other ☐ 7 → specify: ___

No ☐ 2 *If no, skip questions 22-24 and go to question 25*

22. Do you wash your hands after handling a gun or ammunition prior to the following?

smoking	Always ☐ 1	Often ☐ 2	Some time ☐ 3	Never ☐ 4
eating	Always ☐ 1	Often ☐ 2	Some time ☐ 3	Never ☐ 4

23. Are ammunition and guns usually stored inside the tent when in the bush (or in your house/apartment when not in the bush. This question concerns possible exposure to lead)?

	Yes	No	
Bush	☐ 1	☐ 2	→ If yes, are they stored in a sealed case/container? Yes ☐ 1 No ☐ 2
House/apartment	☐ 1	☐ 2	→ If yes, are they stored in a sealed case/container? Yes ☐ 1 No ☐ 2

24. Are clothing and footwear stored openly in the tent when in the bush (This question concerns possible exposure to lead) or in sealed containers?

Bush Yes ☐ 1 → If yes, are they stored in a sealed case/container? Yes ☐ 1 No ☐ 2

No ☐ 2

Cigarette smoking

25. Do you smoke cigarettes?

Not applicable ☐ 1
Yes ☐ 2
Ex-smoker ☐ 3 → **indicate the year you quit:** /_____/ (go to question 27)
No ☐ 4 (go to question 27)

26. If you smoke (or did), an average of how many cigarettes do (or did) you smoke per day?

Not applicable ☐ 1
1-10 ☐ 2
11-25 ☐ 3
26 and more ☐ 4
Other → specify: _____
I don't know ☐ 5

27. Do you currently live with someone who smokes cigarettes, pipes, or cigars inside the house on a regular basis?

Yes ☐ 1
No ☐ 2

Physical activities (questions 28 and 29 to be answered by children only)

28. Do you play outside?

Yes ☐ 1 → specify: _____
_____
No ☐ 2

29. When you play outdoors, are you in contact with:

Pets Yes ☐ 1 No ☐ 2
Dirt, sand, rocks Yes ☐ 1 No ☐ 2

Not applicable ☐ 8

30. Are you involved with the following activities?

Not applicable ☐ 8
Boat repairs/building boats Yes ☐ 1 No ☐ 2
Do you make your own bullets or fishing sinkers Yes ☐ 1 No ☐ 2
Do you use materials with lead such as solder, white lead filler, etc. Yes ☐ 1 No ☐ 2
Home renovations Yes ☐ 1 No ☐ 2
Other→ specify: _____ Yes ☐ 1 No ☐ 2

31. Which of the following sentences best describes your usual work activity pattern when <u>at home</u> (including housework)?

Not applicable	☐ 1
I am usually sitting during the day and do not walk around very much	☐ 2
I stand or walk around quite a lot during my day, but I do not have to carry or lift things very often	☐ 3
I usually lift or carry light loads, or I have to climb stairs or hills often	☐ 4
I do heavy work or carry very heavy loads	☐ 5

32. At the present time, which sentence below best describes your main pattern of activity during a day <u>at work</u>?

Not applicable	☐ 1
Not applicable (not formally employed)	☐ 2
I am usually sitting during the day and do not walk around very much	☐ 3
I stand or walk around quite a lot during my day, but I do not have to carry or lift things very often	☐ 4
I usually lift or carry light loads, or I have to climb stairs or hills often	☐ 5
I do heavy work or carry very heavy loads	☐ 6

33. Which sentences below best describes your usual pattern of activity when you are in the <u>bush</u>?

Not applicable	☐ 1
Not applicable (do not go to the bush)	☐ 2
I am usually sitting during the day and do not walk around very much	☐ 3
I stand or walk around quite a lot during my day, but I do not have to carry or lift things very often	☐ 4
I usually lift or carry light loads, or I have to climb stairs or hills often	☐ 5
I do heavy work or carry very heavy loads	☐ 6

34. In the last 3 months, how often did you participate in any fitness activities in your spare time? Only count activities that lasted at least 20 minutes.

Not applicable	☐ 1
Not once	☐ 2
Approximately once per month	☐ 3
Approximately 2-3 times per month	☐ 4
Approximately once per week	☐ 5
Approximately twice per week	☐ 6
Approximately 3 times per week	☐ 7
4 times and more per week	☐ 8

Thank you for your collaboration

Signature of the interviewer: _____

Date: _____

CLINICAL QUESTIONNAIRE (NURSE) ID number |___|___|___| Date: _____

Questions 1 to 7 to be completed only if the participant is a woman ≥15 years old (if not go to question 7)

1. Are you pregnant at the present time?

Yes ☐ 1
No ☐ 2

2. Are you breastfeeding at the present time?

Yes ☐ 1
No ☐ 2

3. Do you still have your periods?

Yes ☐ 1→ specify: regularly ☐ 1→ what was the start date of your last period?

____ / ____ / ____

irregularly ☐ 2 → what was the start date of your last period?

____ / ____ / ____

No ☐ 2 → **specify: At what age did you have your last period?** |_____|

I don't remember ☐ 3

If she still has her periods go to question 7

4. Do you use oral contraceptives (the pill) at the present time?

Yes ☐ 1→ **specify why:**
- birth control ☐ 1
- to regulate your menstrual cycle ☐ 2
- for another reason ☐ 3

No ☐ 2

5. Are you post-menopausal?

Yes ☐ 1
No ☐ 2
I don't know ☐ 3

6. Do you take hormonal medication for your menopausal status at the present time?

Yes ☐ 1 → **what is the medication name that the doctor has prescribed for your hormonal therapy (estrogen, provera, prometrium, etc)?**

_____
_____

No ☐ 2

The following questions are personal and concern psychological well-being and your health status

Psychological well-being

7. In general, compare to other persons of your age, would you say your health is …?

Not applicable	☐ 1
Very good	☐ 2
Good	☐ 3
Fair	☐ 4
Poor	☐ 5
I do not know	☐ 6

8. How satisfied are you with your health? Would you say you are …

Not applicable	☐ 1
Very satisfied	☐ 2
Somewhat satisfied	☐ 3
Not too satisfied	☐ 4
Not at all satisfied	☐ 5
I do not know	☐ 6

9. In general would say you are…

Not applicable	☐ 1
Very happy	☐ 2
Pretty happy	☐ 3
Not too happy	☐ 4
I do not know	☐ 5

10. Which of the following best describes you?

Not applicable	☐ 1
I am a person with no friends	☐ 2
I am a person with few friends	☐ 3
I am a person with some friends	☐ 4
I am a person with many friends	☐ 5
I do not know	☐ 6

11. How would you describe your relationship with other people in your community?

Not applicable	☐ 1
Very satisfactory	☐ 2
Somewhat satisfactory	☐ 3
Somewhat unsatisfactory	☐ 4
Very unsatisfactory	☐ 5
I do not know	☐ 6

12. Are you worried about the pollution of the environment, water and air in the Oujé-Bougoumou area?

Not applicable	☐ 1
Not at all	☐ 2
Somewhat	☐ 3
Fairly	☐ 4
Very much	☐ 5
I do not know	☐ 6

13. Are you worried about possible risks to health from mine tailings in the Oujé-Bougoumou area?

Not applicable	☐ 1
Not at all	☐ 2
Somewhat	☐ 3
Fairly	☐ 4
Very much	☐ 5
I do not know	☐ 6

14. During the past 12 months were you diagnosed with a serious illness (physical or mental, including depression)?

Not applicable	☐ 1			
Yes	☐ 2	→ **did you find it?**	Extremely stressful	☐ 1
			Somewhat stressful	☐ 2
			Slightly stressful	☐ 3
			Not at all stressful	☐ 4
			I do not know	☐ 5
No	☐ 3			

15. During the past 12 months did someone very close to you die?

Yes ☐ 2 → **did you find it?**

- Not applicable ☐ 1
- Extremely stressful ☐ 2
- More or less stressful ☐ 3
- Slightly stressful ☐ 4
- Not at all stressful ☐ 5
- I do not know ☐ 6

No ☐ 3

The following questions concern your health

16. At the present time, do you have any of the health problems listed below:

	Yes	**No**
Anemia	☐ 1	☐ 2
Cancer	☐ 1	☐ 2
Diabetes	☐ 1	☐ 2
High blood pressure	☐ 1	☐ 2
Heart disease	☐ 1	☐ 2
Hypercholesterolemia (high cholesterol)	☐ 1	☐ 2
Goitre or thyroid trouble	☐ 1	☐ 2
Respiratory trouble	☐ 1	☐ 2
Liver problems	☐ 1	☐ 2
Kidney problems	☐ 1	☐ 2
Osteoporosis	☐ 1	☐ 2

Allergy — Yes ☐ 1 No ☐ 2 → **specify:** _____

Other problem — Yes ☐ 1 No ☐ 2 → **if yes, specify:** _____

17. Please indicate the medications you are currently taking. None ☐ 0
(including non-prescription drug use and alternative treatment: homeopathic medicine, herbal drinks, plants etc.)

Medication name	Treatment indicated (year/month started)	Health problem
1._____	|__|__|__|__| |__|__| (year/ month)	_____
2. _____	|__|__|__|__| |__|__| (year/ month)	_____
3. _____	|__|__|__|__| |__|__| (year/ month)	_____
4. _____	|__|__|__|__| |__|__| (year/ month)	_____
5._____	|__|__|__|__| |__|__| (year/ month)	_____
6. _____	|__|__|__|__| |__|__| (year/ month)	_____
7. _____	|__|__|__|__| |__|__| (year/ month)	_____
8. _____	|__|__|__|__| |__|__| (year/ month)	_____
9. _____	|__|__|__|__| |__|__| (year/ month)	_____

18. Were you ever treated with radiation (for a cancer)?

Yes ☐ 1 → **please describe the treatment:**_____
When have you been treated? _____

No ☐ 2

Thank you for your collaboration

Signing of the nurse: _____

Date: _____

FOOD FREQUENCY QUESTIONNAIRE

I would like to ask you some questions about how often you eat certain foods such as fish, birds and ducks, and games.

First, I would like to know if you ate fish, birds and ducks, and games over the past 4 seasons. I will ask a series of questions for each animal species.

Let's start with brook trout (fresh or frozen or cooked). Did you eat brook trout over the past year?

Reference number:	Id. |_____|_____|_____|
Group :	Id. |_____|
	1. 0-14 years old, 2. 15-39 only women, 3. 15-39 only men, 4. 40 and over years old
Date of the	interview |_2_|_0_|_0_|_2_| |___|___| |___|___| Year Month Day

FISH SPECIES (1)

	Brook trout	**Walleye**	**Lake whitefish**	**Northern Pike**
	Maasimekw	***Ukaau***	***Atihkamekm***	***Atihkamekm***
1. Consumption: (past 12 months)	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Fish parts consumed:				
Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Skin	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Totally	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify (ex. head)				

Instructions:

Question 1. Did you eat brook trout over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat brook trout during summer? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat brook trout? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the lake or river at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer (e.g. flesh and skin).

FISH SPECIES (2)

	Lake trout	**Red sucker**	**White sucker**	**Lake sturgeon**
	Namekush	***Iyihaachaau***	***Mihkuchikaash***	***Nameu***
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring: Yes or No	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Fish parts consumed: Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Skin	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Totally	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify (ex. head)				

Instructions:

Question 1. Did you eat lake trout over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat lake trout during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat lake trout? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the lake or river at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

FISH SPECIES (3)

	Burbot	Other round white fish (1)	Other round white fish (2)	Other fish
	Miyaahkatuu			
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Fish parts consumed: Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Skin	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Totally	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify (ex. head)				

Instructions:

Question 1. Did you eat Burbot over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat burbot during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat burbot? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the lake or river at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

BIRDS and DUCKS SPECIES (1)

	Goose	Mallard	Wood duck	Golden eye duck
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Bird parts consumed: Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat goose over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat goose during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat goose? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

BIRDS and DUCKS SPECIES (2)

	Northern pintail	American black duck	Black scoter	Merganser
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Bird parts consumed: Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat northern pintail over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat northern pintail during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat northern pintail? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

BIRDS and DUCKS SPECIES (3)

	Loon	Willow ptarmigan	Partridges	Other
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Bird parts consumed: Flesh	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Eggs	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat loon over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat loon during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat loon? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

GAME (1)

	American beaver	Otter	Moose	Caribou
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Game parts consumed: Meat	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Kidney	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat american beaver over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat american beaver during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat american beaver? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

GAME (2)

	Rabbit	Bear	Squirrel	Lynx
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Game parts consumed: Meat	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Kidney	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat rabbit over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat rabbit during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat rabbit? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

GAME (3)

	Martins	Mink	Weasel	Muskrat
1. Consumption (past 12 months):	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2. Summer:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
2.1 How often during summer?				
2.2 Number of portions/meal:				
3. Spring:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
3.1 How often during spring?				
3.2 Number of portions/meal:				
4. Winter:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
4.1 How often during winter?				
4.2 Number of portions/meal:				
5. Fall:	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
5.1 How often during fall?				
5.2 Number of portions/meal:				
6. Place (s) of catch: 1st place				
2nd place				
3rd place				
7. Game parts consumed: Meat	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Liver	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Kidney	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐	Yes ☐ No ☐
Other part: specify				

Instructions:

Question 1. Did you eat martins over the past year? Enter Yes or No in the first column; do the same for questions 2 to 5.

Questions 2.1, 3.1, 4.1, 5.1. How often did you eat martins during this season? **A**: Twice a day **B**: Once a day **C**: 3-6 times a week **D**: 1-2 times a week **E**: 1-3 times a month **F**: 1-2 times during this season. Enter the chosen letter in the right line/column.

Questions 2.2, 3.2, 4.2, 5.2. Using this food model what is your usual serving when you eat martins? Show the food model. Enter the quantity under the right line/column. (food model Po 3)

Question 6. Places of catch: If the subject mentions more than one place, ask him the 3 main places in order of importance. Use the map to help the subject. Enter the name of the name of area at the right line/column.

Question 7. Enter Yes or No in the right line/column. You may enter more than one answer.

8. When you eat fish and game, have you usually caught or hunted it yourself?

Yes ☐ Sometimes ☐ No ☐

9. Where do you usually fish and hunt?

_____

_____

10. How do you usually fish:

Rod and reel ☐
Net ☐
Other (specify) : _____

11. If you answered "sometimes" or "no", can you tell me who catches or hunts the fish and game that you eat?

☐ **A parent: specify the name and phone number:** _____

_____

☐ **A relative: specify the name and phone number:** _____

_____

☐ **A friend: specify the name and phone number:** _____

_____

☐ **Other: specify the name and phone number:** _____

_____

THANK YOU VERY MUCH FOR YOUR COLLABORATION

Signature of the interviewer:_____Date: _____

APPENDIX 5

Laboratory Contaminants, Biochemistry and Omega-3 Methods

Laboratory Contaminants, Biochemistry and Omega-3 Fatty Acids Methods

Mercury

For the determination of mercury in hair samples (INSPQ method: M-111), hair samples are digested using nitric acid in a hot water bath. The digests are then analysed directly by cold vapor atomic absorption spectrometry (Pharmacia). Routine checks of accuracy and precision are accomplished using a certified reference material. The Centre de toxicologie du Québec also participates in Ottawa's Hair Mercury Interlaboratory Comparison Program.

For the determinations in blood (INSPQ method: M-109) and in urine (INSPQ method: M-110), total blood mercury and urine mercury concentrations are also determined by cold vapor atomic absorption spectrometry (Pharmacia). For total blood mercury concentrations, the inorganic mercury fraction is determined using the same methodology except that the use of cadmium chloride, as part of the reactant mixture, is omitted. Samples are microwave-digested using nitric acid and an aliquot is used for the analysis. For the urine, samples are digested using nitric acid in a hot water bath and an aliquot is used for the analysis. Accuracy and precision are measured using reference material from the Centre de toxicologie du Québec's Interlaboratory Comparison Program. Also, periodic evaluations are accomplished by the participation of the CTQ in the same program.

Others metals (cadmium, lead, etc): ICP-MS (Inductively coupled plasma mass spectrometry)

The trace metals testing in hair (INSPQ method: M-559), allows the determination of more than 30 elements (except for mercury) in a hair matrix. The hair is treated with concentrated nitric acid, diluted and analyzed. For the metal testing in whole blood (INSPQ method: M-557) and the trace metal testing in urine (INSPQ method: M-558), the method allows the determination of more than 20 elements (except for mercury and chromium) in whole blood, for which the samples are diluted in a solution containing ammonium hydroxide, and in urine, for which the samples are diluted in an acidic solution and analyzed.

A number of blanks, spikes, duplicates and certified reference materials are analyzed to control the validity of the results. The CTQ laboratory operates and participates in a multi-element ICP-MS interlaboratory comparison program for metals in biological samples. There are three test runs per year. Hair is occasionally included in that program.

Zinc

The method involves sample dilution followed by flame atomic absorption spectrometry with Analyst 100, Perkin-Elmer, as instrumentation. A 500 µl of specimen is diluted in a solution containing Triton-X. Calibration is performed in an aqueous glycerol solution. The limit of detection is 0.5 µmol/l for plasma/serum and 1 µmol/l for urine.

Arsenic

The determination of non-dietary urine arsenic (inorganic arsenic and its metabolites; INSPQ method: M-204) involves analyte reduction and complexation with potassium iodide followed by solvent extraction. Urine arsenic concentrations are determined by graphite furnace atomic absorption spectrometry. Processed extracts are then injected into the instrument. Routine checks of accuracy and precision are accomplished using reference material from the Centre de toxicologie du Québec's inter laboratory comparison program.

Organochlorines (OCs)

Blood samples (10 ml) collected in vial containing EDTA are centrifuged (10 min, 5000 rpm) and the plasma transferred in glass vials pre-washed with hexane. This standard screen includes the following: total PCBs, 14 specific constituents of PCBs called congeners, p,p′-DDT and its major metabolite p,p′-DDE, hexachlorobenzene and 10 other organochlorine pesticides. This OCs screen has been used extensively in the northern/arctic regions of the circumpolar countries, including Canada, under the auspices of the Arctic Monitoring and Assessment Programme (AMAP)(4). For organochlorine analysis, samples are thawed overnight at 4^{o}C and a 2-ml aliquot is extracted with hexane. The lipid extract is then cleaned-up on Florisil columns and taken to a final volume of 100 µL. OCs (polychlorinated biphenyls (PCBs) and chlorinated pesticides) will be quantified on a HP-5890 series II gas chromatograph equipped with dual-capillary columns and dual Ni-63 electron-capture detectors. Peaks will be identified by their relative retention times obtained on the two columns, using a computer program developed in-house. Quantification will be mainly performed on the Ultra-1 column. Total lipids were determined by routine clinical chemistry methods. Detection limits, based on 3 times the average standard deviation of noise were: 0.02 µg/L for PCB congeners and chlorinated pesticides; 0.04 for p,p′-DDT and ∃-BHC; 0.2 µg/L for total PCB as Aroclor 1260. The limit of detection, based on 3 times the standard deviation of the blank, is between 0.02 µg/L and 0.03 µg/L. The average percentage recoveries are greater than 95%.

Laboratory biochemistry methods

Most of the general biochemistry analyses were performed on an automated chemistry analyser, an Hitachi 917 with Roche reagents. This included plasma fasting glucose (reference limits (RL): 3.6-5.8 mmol/L), blood urea nitrogen (RL: 1.7-8.5), plasma creatinin (RL: male 50-110, female 40-90 umol/L), urinary creatinin (RL: male 7.1-15.9, female 5.2-14.1), bilirubin (RL: 0-17 umol/L).

The diabetes were confirmed if the determination of fasting glucose was higher than 7.8 mmol/L. The diabetes panel was completed with the measure of glycated hemoglobin fraction (HbA1c). This test was performed on the Varian system (Bio-Rad). After citrate lysis the whole blood hemoglobin fractions were separated and quantified photometrically using ionic resin-high pressure liquid chromatography (HPLC): normal reference values are 0.043-0.065. The HbA1c was an accepted indirect method for evaluating the mean glucose concentration over a 2-3 months period.

The iron status (Hitachi 917) was assessed with plasma total iron (RL: 10-30 umol/L), binding capacity (RL: 45-80 umol/L), saturation (RL: male 0.20-0.55, female 0.15-0.50) and plasma transferrin (RL: 2.00-4.00 g/L). Iron reserves were ascertained with the plasma ferritin measurement on the Abbott automated immunoassay system Axsym (RL: male 20-300, female 10-200 ug/L).

Besides the iron status, the anemia panel also includes the determination of hemoglobin, the cell morphology and the dosage of folates and vitamin B12. Hemoglobin was measured by the specific photometric absorbance value on the automated haematology system GEN-S (Beckman-Coulter). Anemia was suspected for hemoglobin values lower than 120 g/L for females or 140 g/L for males. The preparation of blood smears for cell morphology follows: after sampling a blood smear was prepared on site and dried. Upon arrival at the haematology lab and staining (May-Grünwald), the specimen was examined under the microscope. Vitamin dosages are done on the automated immuno-analyser Access (Beckman-Coulter). Reference values are 5.5-50 nmol/L for the folates and 150-550 for the vitamin B12.

The lipid panel (Hitachi 917) were completed with the measurement of HDL-cholesterol based on selective inhibition of the reaction with other lipoproteins, the calculated estimation of LDL-cholesterol (Friedwald formula) and the ratio Total-cholesterol/HDL-cholesterol (atherogenic index). For moderate risk individuals (10-20% risk of cardio-vascular diseases over a 10 year period) the expected values are >1 mmol/L for the HDL-cholesterol, <4 mmol/L for the calculated LDL-cholesterol and an atherogenic index lower than 6. For diabetic individuals expected values are <2.5 mmol/L for the calculated LDL-cholesterol with an atherogenic index <4. Apolipoprotein-B was determined by a immuno-nephelometric assay on the BN-Prospec (Dade-Behring): the normal reference value is <1.2 g/L.

The thyroid status was evaluated with a TSH and a free thyroxin (fT4). Whenever necessary the tri-iodothyronin (total T3) was determined. All three test were performed on the Abbott Axsym system. Reference values are 0.4-5.0 mU/L for the TSH, 10-27 pmol/L for the fT4, 0.9-2.7 pmol/L for the total T3. The thyroxin binding globulin (TBG) were analysed with a classic radioimmunoassay (DiaSorin): reference values 12-25 mg/L males, 14-30 mg/L females. This test will help to delineate hypo or hyperthyroxinemia due to TBG alterations.

For identification of habitual tobacco users the plasma nicotine metabolite cotinine was assayed. Following extraction of 2 mL of plasma with methylene chloride, the cotinine was measured by reversed-phase liquid chromatography with ultraviolet detection. Non-smokers or passively exposed have values lower than 45 pmol/L.

Plasma phospholipid fatty acid analysis; Omega 3

For the determination of the fatty acid composition of plasma phospholipid, 200 µl aliquots of plasma were extracted following the addition of chloroform: methanol (2:1, v/v), in the presence of a known amount of internal standard (diheptadecanoyl phospholipid). The total phospholipid was isolated from the lipid extract by thin-layer chromatography using heptane/isopropyl ether/acetic acid (60:40:3, v/v/v) as the developing solvent. Following transmethylation, using BF3/methanol, the fatty acid profile will be determined by capillary gas-liquid chromatography.

A list of additional substances determined in blood, urine and hair.

Participants 8 years old and over

Whole blood	antimony	silver	total arsenic
	bismuth	beryllium	cobalt
	tin	lithium	manganese
	molybdenum	nickel	tellurium
	thallium	uranium	zirconium
Plasma	Omega-3 fatty acids, total lipids		
Urine	creatinine	lead	antimony
	silver	bismuth	beryllium
	cobalt	tin	lithium
	manganese	molybdenum	nickel
	thallium	uranium	zirconium
	zinc		
Hair (0-2 cm)	antimony	silver	total arsenic
	bismuth	beryllium	calcium
	cobalt	gallium	lanthanum
	lithium	magnesium	manganese
	molybdenum	nickel	niobium
	gold	rubidium	strontium
	tellurium	thallium	uranium

Polychlorinated biphenyls (PCBs) and chlorinated pesticides (µg/L)

Plasma	Congeners; 28, 52, 99, 101, 105, 118, 128, 138, 170, 180, 183, 187.	
	Aldrin	alpha-chlordane
	gamma-chlordane	cis-nonachlor
	p,p'-DDE	p,p'-DDT
	Hexachlorobenzene	Mirex
	Oxychlordane	Trans-nonachlor
	Total lipids g/L	

APPENDIX 6

Reference Values for PCBs and Toxic Elements

6-A. Reference values of PCBs, measured in plasma or serum as Aroclor 1260 (Health Canada) (µg/L)

For women of reproductive age
<5: Tolerable
5-100: concern
>100: Action

For men and post-menopausal women
<20: Tolerable
20-100: concern
>100: Action

(AMAP, 2002)

Tolerable Daily Intake (TDI)
The current Health Canada Provisional Tolerable Daily Intake (pTDI) for PCB is 1 µg/.kg.bw/day and is based on developmental/reproductive effects in monkeys treated with Aroclor 1248 (WHO, 1976a; WHO, 1993). The LOEL in that study was 100 µg/kg/day and a 100 fold safety factor was applied to come up with a provisional TDI of 1 µg/kg/day. In fact the LOEL of the particular study was more accurately calculated to be 90 µg/kg/day (WHO, 1993) but Health Canada still maintains a pTDI of 1 µg/kg/day).

Blood Guidelines

In 1979, a Health Protection Branch Working Group was convened and reached a consensus on terminology and "working" PCB guidelines in whole blood for the general population (Wheatley, B., 1979. It should be noted that 1 ppb equates to 1 ng PCB/ml of fluid and that PCB refers to total PCB as determined by the current method of choice.

	Adult Males		Adult Females	Children (under 18 Years)	Pregnant and Lactating Women
Tolerable Level	<20	<5	(pre-menopause)	<5	<5
		<20	(post-menopause)		
Concern Level	20	5	(pre-menopause)	5	5
		20	(post-menopause)		
Action Level	100	100		20	>5 (Infant)

These values were derived on the following basis. First it was assumed that not all individuals were equally sensitive to the toxic effects of PCB and that there was a greater risk to infants, both in utero and suckling than to adults. Therefore, women of child-bearing age should be afforded the greatest protection. Based on the available information, a toxic level for humans, where clinical effects was seen, was considered to be 200 ppb PCBs in blood (this level was based on occupational exposure to Aroclor 1242 only). A safety factor of 10 was applied to this number to achieve a maximum acceptable level (Tolerable) of 20 ppb for adult males and post-menopausal women. To protect women of child-bearing age and children under 18, a calculation was made based on the currently accepted maximum level of PCB on human milk

of 50 ppb (In 1978 an external advisory committee to the ADM recommended that PCB levels in breast milk above 50 ppb should be viewed with "concern" (HWC, 1978). Allowing for a 10:1 ratio between milk and blood (an approximate figure based on fat content of each tissue, Grant, D.L., 1986), the figure of 50 ppb was divided by 10 to give a maximum tolerable PCB blood level of 5 ppb.

In 1986 the guidelines were reviewed (Grant, D.L., 1986) and remained unchanged. The review did provide some additional guidance as to how to advise clientele who have shown blood values that exceeded the tolerable levels. The guidelines as described above are still used presently.

PCB Guidelines for Other Canadian Jurisdictions

Province of Alberta

The Province of Alberta Department of Labour has established an Occupational Health and Safety Guideline for the medical monitoring of workers exposed to PCB at the Swan Hills Treatment Centre (Alberta Labour, 1994). Blood serum Action Levels were described as follows: Less than 10 ppb. No Action. From 10 ppb to less than 30 ppb, called for a review of potential sources and re-tested yearly. For blood PCB levels between 30 and 100 ppb, immediate re-testing is required and if results are confirmed, work tasks to be modified to reduce exposure and regular monitoring of levels and liver function should be carried out. If levels remain persistently high, a more detailed evaluation of hepatic function is called for. If serum levels exceeded 100 ppb, the employee is to be removed from the Waste Treatment Centre, and repeat blood serum testing is to be conducted monthly.

Northwest Territories

The Northwest Territories has established a set of Interim Contaminant Guidelines for Whole Blood Levels for women of reproductive age and for newborns (Walker, 1996). The levels for women of reproductive age are the same as described above , 5 ppb or lower are 'tolerable', higher than 5 ppb is termed 'level of concern/action'. However there is an important addition. Newborns, on the basis that blood lipid is 50% lower in cord blood than maternal blood, have been assigned a 'tolerable' level of 2.5 ppb and below, and a 'level of concern/action for any level that exceeds 2.5 ppb.

6-B. Mercury

TDI

The current Health Canada provisional TDI for total mercury is 0.71 µg/kg body weight/day and for methylmercury it is 0.47 µg/kg body weight/day (INAC, 1997, AMAP, 1998). These figures are based on the setting of a provisional tolerable weekly intake of 0.3 mg of total mercury of which no more than 0.2 mg should be present as methyl mercury (WHO, 1976b) by a Joint FAO/WHO Expert Committee on Food Additives. These figures divided by 60 kg as the weight for a person and 7 for the number of days in a week give rise to the numbers 0.71 and 0.47. The Japanese Research Committee on Minimata Disease, 1975 in WHO, 1976 concluded that the first clinical effect (paraesthesia) associated with the long-term exposure to methylmercury occurred at a level of approximately 5 µg/kg body weight/day. There would thus be approximately a 10-fold safety factor between this level and the current pTDI. More recently, a US EPA study (US EPA, 1997) has proposed a reference dose level of 0.1 µg/kg body weight/day for methylmercury. This value equates to a 1.1 ppm level in hair or 4.4 ppb level in whole blood.

Blood and Hair Guidelines

In humans, blood methylmercury and hair methylmercury levels are often used to indicate exposure and have been correlated to both total body burden and brain tissue levels (INAC, 1997). Hair values have an added advantage in that they can also reflect temporal trends mercury accumulation. The hair/blood mercury concentrations have been estimated at approximately 200-300/1 (INAC, 1997). The "standards" below were published in 1979 (HWC, 1979) and were derived from WHO (WHO, 1972).

Total Mercury

	Whole Blood (ppb) (SI, nmol/L)	**Hair (ppm) (SI, nmol/g)**
Normal Acceptable Range	<20 (99.7)	<6 (29.9)
Increasing Risk	20-100 (99.7-498.5)	6-30 (29.9-149.6)
At Risk	>100 (498.5)	>30 (149.6)

It was noted that symptoms may first appear in sensitive individuals with blood mercury concentration between 200 and 500 ppb (997.0 and 2492.5 nmol/L) or a hair concentration of between 50-125 ppm (249.3-623.1 nmol/g). It thus appears that a safety factor of 10 was applied to these levels to derive the "Normal, Increasing Risk and At Risk" categories described above. Charles Dumont (1986) used in reporting the 1993 results total mercury in

hair the following intervals; <6 ppm (29.9 nmol/g), 6<15 ppm (29.9<74.78 nmol/g), 15<30 ppm (74.78<149.6 nmol/g),

Mercury Guidelines for Other Canadian Jurisdictions

The Northwest Territories has established a set of Interim Contaminant Guidelines for Blood Levels for women of reproductive age and for newborns (Walker, 1996). The levels for women of reproductive age are somewhat different than the Health Canada figures. Levels of 20 ppb (99.7 nmol/L) or lower in whole blood are considered 'tolerable', values between 20-40 ppb (99.7 nmol/L- 199.40 nmol/L) are termed 'level of concern"and levels that exceed 40 ppb 'action levels' (199.40 nmol/L). In addition there are categories for newborns based on the fact that the newborn are 2-3 times more sensitive than adults to the neurodevelopmental effects of methylmercury. Newborns have been assigned a 'tolerable' level of 10 ppb, a 'level of concern' of 10 to 20 ppb (49.85 nmol/L to 99.7 nmol/L) in whole blood and an 'action level' at 20 ppb and above (>99.7 nmol/L).

Level declarable to public health in Québec for mercury >15 µg/L (74.8 nmol/L) in each age group

6-C. Lead

Level of lead in Whole Blood	Intervention
<10 µg/dL (0.48 µmol/L)	None
10-15 µg/dL (0.48 µmol/L-0.72 µmol/L)	Identify possible exposure source. Identify lead source responsible for the exposure and reduce or eliminate this source of exposure or modify behaviours that result in exposure.
>15-19 µg/dL (0.72 µmol/L-0.92 µmol/L)	Review exposure history to identify source of lead exposure. Exposure reduction counseling by medical personnel or public health authority and provision of advice on hygienic and nutritional measures.
20-44 µg/dL (0.97 µmol/L-2.12 µmol/L)	Medical assessment. Identify and eliminate environmental lead problem.
45-69 µg/dL (2.17 µmol/L-3.33 µmol/L)	Urgent medical and environmental assessment and possible hospitalization of children for chelation therapy.
70 µg/dL and greater (3.38 µmol/L)	Urgent medical assessment and possible hospitalization of children for chelation therapy.

The guidelines above were based on recommendations put forward by a Lead Working Group which reported to the Federal-Provincial Committee on Environmental and Occupational Health (CEOH, 1994). The level of 10 µg/dL (0.48 µmol/L) was chosen as the earliest intervention level because there has been evidence of cognitive and developmental effects in children, and blood pressure effects in adults at that level. Moreover, there is probably no threshold for these effects. As mentioned above, the pTDI of 3.57 µg/kg (0.02 µmol/L) body weight/day is associated with a lead level in whole blood of approximately 5.7 µg/dL (0.03 µmol/L). This means there is only a safety factor of about 2 between the pTDI and the adverse health effects observed at 10 µg/dL (0.48 µmol/L).

Level declarable to public health in Québec >100 µg/L (0.48 µmol/L) in each age group

Tolerable lead levels in hair

30 nmol/g (Centre de Toxicologie du Québec (CTQ), 1990-1995)

6-D. Other metals (Arsenic, Cadmium, Copper, Zinc, Selenium) tolerable levels (conventional and international system)

ELEMENT	HAIR	BLOOD	PLASMA(P)	URINE
	µg/g (SI)	µg/L (SI)	µg/L (SI)	µg/L (SI)
Arsenic	(0-2.670 nmol/g)[a] <0,001[b] (<10 nmol/g)	---	---	(0-0.25 µmol/L)[a] 5-50[b] (0.07-0.67 µmol/L) 10[e] (0.13 µmol/L)
Cadmium -Non-smokers	(0-4.45 nmol/g)[a] <0,7[d] (6.23 nmol/g)	(0-4.98 nmol/L)[a] <10[b] (<88.97 nmol/L) <2[c] (<17.79 nmol/L)	---	(0-9.96 nmol/L)[a]
-smokers		(8.90-80.07 nmol/L)[a] <6[c] (<53.38 nmol/L)		
Copper	(0.16-0.63 µmol/g)[a]	---	1200-1450[b] (18.89-22.82 µmol/L) 200-700[d,1] (3.15-11.02 µmol/L) 900-1900[d,2] (14.17-29.91 µmol/L) 700-1400[d,3] (11.02-22.04 µmol/L) 800-1500[d,4] (12.59-23.61 µmol/L) 1118-3020[d,5] (17.60-47.53 µmol/L) 700-1550[f] (11.02-24.40 µmol/L)	(0.01-0.50 µmol/L)[a]
Zinc	(1.91-4.59 µmol/g)[a]	(60.01-120.00 µmol/L)[a] 4100-7600[d] (62.73-116.28 µmol/L)	---	---
Selenium	(0-20 nmol/g)[a]	---	(1.00-2.00 µmol/L)[a] <150[b] (<1.90 µmol/L) 46-143[d] (0.58-1.81 µmol/L)	---

[a] Centre de Toxicologie du Québec (CTQ, all data in SI), 1990-1995 (urinary non-dietary arsenic)
[b] Goyer et Clarkson, 1996 et 2001 (urinary non-dietary arsenic)
[c] Health Canada, 1995
[d] Burtis et Ashwood, 1999
[e] Vahter, 1986, non-dietary arsenic
[f] Ellenhorn, 1999
--- not applicable
[1] children 0-6 mois
[2] children 6-12 ans
[3] men
[4] women
[5] pregnant women (near delivery)

Note: cadmium (Walker, 1996) Childbearing age women ≤5 ppb=tolerable value; >5 ppb=levels of concern/action

6-E. Other metals (Arsenic, Cadmium, Copper, Zinc, Selenium) action levels (conventional and international system)

ELEMENT	HAIR	BLOOD	PLASMA(P) SERUM(S)	URINE
	µg/g	**µg/L (SI)**	**µg/L (SI)**	**µg/L (SI)**
Arsenic (non-dietary)	(13.35 nmol/g)[a]	---	---	35[b] (0.47 µmol/L)
Cadmium	---	5[b] (44.49 nmol/L)	---	7.5[c] (66.73 nmol/L) (47.4 nmol/L)[f]
Copper	---	---	1600[d] (25.18 µmol/L)	---
Zinc	---	---	---	---
Selenium	---	---	285[e] (3.61 µmol/L)	---

[a] Goyer et Clarkson, 1996 et 2001 (surexposition)
[b] ACGIH, 2002, others
[c] Personal comment A. Leblanc (for 1.5 g creatinin/L)
[d] Ellenhorn, 1999
[e] Nantel et al., 1985
[f] Corresponding to 4 µmol/mol creatinine. Since a significant proportion of the general proportion of the general population displays early signs of toxicity already at urinary cadmium concentrations around 3 µmol/mol creatinine (Elinder and Jarup, 1996; Jarup and Bergland, 1998)

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APPENDIX 7

Percentages of Detection and Means of PCBs and Chlorinated Pesticides

Table A1. Average detection frequencies of polychlorinated biphenyls (PCBs) and chlorinated pesticides among the Oujé-Bougoumou and Nemaska participants

PCBs/chlorinated pesticides	Oujé-Bougoumou % of detection	Nemaska % of detection	p value[1]
Aroclor 1260	96.4	93.8	0.344
Congener 28	3.4	0.0	-
Congener 52	0.0	0.0	-
Congener 99	83.0	73.3	0.076
Congener 101	10.7	6.5	-
Congener 105	38.0	36.1	-
Congener 118	83.0	82.0	0.843
Congener 128	16.4	14.5	-
Congener 138	94.6	93.8	0.787
Congener 153	99.1	100.0	0.397
Congener 156	69.6	68.5	-
Congener 170	79.4	80.8	0.800
Congener 180	92.8	94.6	0.604
Congener 183	68.0	64.3	-
Congener 187	86.9	82.1	0.315
Aldrin	0.0	0.0	-
β-HCH	32.1	33.6	-
Alpha-chlordane	0.0	0.0	-
Gamma-chlordane	0.0	0.0	-
Cis-nonachlor	36.5	43.3	-
pp′-DDE	100.0	100.0	-
pp′-DDT	30.1	22.4	-
Hexachlorobenzene	100.0	99.4	0.292
Mirex	61.2	58.0	-
Oxy-chlordane	66.0	69.6	-
Transnonachlor	73.6	77.4	0.519

[1] p Value was obtained by the chi-square test for comparison of proportions calculated only if **%** of detection >70

Table A2. Plasma concentrations of congener PCB 99 among the Oujé-Bougoumou and Nemaska participants (μg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	28.6	-	- -	0.368
	Nemaska	11	45.5	-	- -	
Women 15-39 years	Oujé-Bougoumou	78	91.0	12.36 (12.43)	8.71 (7.28-10.41)	0.007
	Nemaska	42	69.0	8.40 (8.82)	5.66 (4.37-7.32)	
Men 15-39 years	Oujé-Bougoumou	40	97.5	19.34 (41.14)	10.44 (7.92-13.77)	0.057
	Nemaska	15	66.7	10.84 (18.25)	6.13 (3.82-9.84)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	102.76 (103.95)	65.14 (49.14-86.33)	0.105
	Nemaska	13	100.0	53.33 (46.47)	39.34 (25.46-60.80)	
Total (≥8 years)	Oujé-Bougoumou	190	87.9	29.53 (29.41)	11.08 (9.27-13.24)	0.006
	Nemaska	81	70.4	14.83 (12.58)	7.10 (5.59-9.00)	

[1] Detection frequency at the detection limit of 0.02 μg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A3. Plasma concentrations of congener PCB 118 among the Oujé-Bougoumou and Nemaska participants (µg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	33.3	-	- -	0.277
	Nemaska	11	63.6	4.46 (2.64)	3.91 (2.88-5.30)	
Women 15-39 years	Oujé-Bougoumou	78	94.9	20.44 (21.37)	13.00 (10.52-16.06)	0.002
	Nemaska	43	69.8	11.11 (16.13)	6.47 (4.86-8.62)	
Men 15-39 years	Oujé-Bougoumou	40	90.0	40.53 (151.48)	10.95 (7.53-15.92)	0.296
	Nemaska	15	86.7	12.37 (17.60)	7.61 (4.80-12.07)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	253.91 (292.89)	148.32 (108.74-202.32)	0.038
	Nemaska	13	100.0	103.39 (95.66)	72.58 (45.10-116.79)	
Total (≥8 years)	Oujé-Bougoumou	190	88.4	66.28 (82.34)	15.58 (12.50-19.42)	0.003
	Nemaska	82	76.8	23.82 (24.72)	8.67 (6.60-11.38)	

[1] Detection frequency at the detection limit of 0.02 µg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A4. Plasma concentrations of congener PCB 138 among the Oujé-Bougoumou and Nemaska participants (μg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	71.4	5.68 (4.02)	4.84 (3.84-6.10)	0.083
	Nemaska	11	72.7	13.26 (11.43)	8.85 (4.92-15.91)	
Women 15-39 years	Oujé-Bougoumou	78	100.0	45.12 (48.13)	27.82 (22.22-34.84)	0.005
	Nemaska	43	100.0	31.35 (41.38)	15.47 (10.83-22.09)	
Men 15-39 years	Oujé-Bougoumou	40	100.0	87.30 (208.00)	38.08 (26.50-54.74)	0.059
	Nemaska	15	93.3	43.35 (78.40)	18.93 (9.96-35.96)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	498.63 (450.02)	331.19 (251.86-435.52)	0.056
	Nemaska	13	100.0	270.94 (294.59)	183.29 (112.82-297.79)	
Total (≥8 years)	Oujé-Bougoumou	190	96.8	133.61 (136.03)	34.58 (27.42-43.62)	0.015
	Nemaska	82	95.1	65.69 (71.30)	20.82 (15.18-28.56)	

[1] Detection frequency at the detection limit of 0.02 μg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A5. Plasma concentrations of congener PCB 170 among the Oujé-Bougoumou and Nemaska participants (µg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	19.0	-	- -	0.028
	Nemaska	11	63.6	8.03 (7.52)	5.72 (3.50-9.34)	
Women 15-39 years	Oujé-Bougoumou	78	89.7	21.08 (25.25)	12.43 (9.87-15.65)	0.052
	Nemaska	43	72.1	16.70 (22.69)	8.31 (5.88-11.74)	
Men 15-39 years	Oujé-Bougoumou	40	92.5	49.80 (96.19)	21.69 (14.58-32.26)	0.077
	Nemaska	15	80.0	23.67 (36.21)	10.81 (5.70-20.50)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	289.18 (250.75)	191.66 (145.54-252.41)	0.102
	Nemaska	13	100.0	185.34 (215.09)	113.86 (64.79-200.09)	
Total (≥8 years)	Oujé-Bougoumou	190	85.3	75.11 (76.20)	18.07 (14.29-22.84)	0.050
	Nemaska	82	76.8	41.39 (50.10)	11.96 (8.73-16.39)	

[1] Detection frequency at the detection limit of 0.02 µg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A6. Plasma concentrations of congener PCB 180 among the Oujé-Bougoumou and Nemaska participants (μg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)		Geometric mean (95% CI)		p value[2]
8-14 years	Oujé-Bougoumou	21	61.9	6.11	(4.99)	4.95	(3.80-6.45)	0.025
	Nemaska	11	81.8	23.73	(26.20)	13.17	(6.48-26.78)	
Women 15-39 years	Oujé-Bougoumou	78	100.0	66.73	(82.33)	37.32	(29.04-47.96)	0.032
	Nemaska	43	97.7	53.03	(74.67)	22.61	(15.06-33.94)	
Men 15-39 years	Oujé-Bougoumou	40	100.0	162.58	(314.92)	68.05	(44.88-103.18)	0.054
	Nemaska	15	93.3	74.47	(112.10)	29.78	(13.97-63.47)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	951.20	(821.65)	628.75	(476.64-829.40)	0.110
	Nemaska	13	100.0	627.96	(760.69)	376.64	(212.54-667.43)	
Total (≥8 years)	Oujé-Bougoumou	190	95.8	244.93	(250.46)	49.87	(38.32-64.92)	0.072
	Nemaska	82	95.1	136.85	(174.32)	32.54	(22.66-46.72)	

[1] Detection frequency at the detection limit of 0.02 μg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A7. Plasma concentrations of congener PCB 187 among the Oujé-Bougoumou and Nemaska participants (µg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	42.9	-	- -	0.041
	Nemaska	11	63.6	9.18 (8.44)	6.39 (3.80-10.75)	
Women 15-39 years	Oujé-Bougoumou	78	94.9	28.42 (32.59)	16.79 (13.30-21.18)	0.022
	Nemaska	43	76.7	21.41 (28.69)	10.30 (7.17-14.79)	
Men 15-39 years	Oujé-Bougoumou	40	97.5	65.78 (129.00)	28.59 (19.27-42.40)	0.066
	Nemaska	15	80.0	32.99 (55.09)	13.72 (6.95-27.08)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	357.25 (306.38)	241.60 (184.92-315.65)	0.102
	Nemaska	13	100.0	224.78 (250.83)	146.50 (87.33-245.76)	
Total (≥8 years)	Oujé-Bougoumou	190	91.1	94.59 (94.06)	23.19 (18.29-29.40)	0.033
	Nemaska	82	79.3	51.39 (59.67)	14.68 (10.60-20.32)	

[1] Detection frequency at the detection limit of 0.02 µg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A8. Plasma concentrations of pp′-DDE among the Oujé-Bougoumou and Nemaska participants (μg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	100	43.78 (25.09)	38.04 (30.22-47.90)	0.014
	Nemaska	11	100	85.34 (59.36)	68.21 (44.81-103.82)	
Women 15-39 years	Oujé-Bougoumou	78	100	177.99 (141.39)	133.44 (112.65-158.08)	0.083
	Nemaska	43	100	150.80 (154.46)	102.38 (79.30-132.17)	
Men 15-39 years	Oujé-Bougoumou	40	100	385.86 (1 225.35)	178.06 (136.41-232.43)	0.276
	Nemaska	15	100	196.87 (219.17)	133.40 (85.13-209.05)	
Men & women ≥40 years	Oujé-Bougoumou	51	100	1 758.16 (1 757.15)	1 170.80 (903.83-1 516.63)	0.051
	Nemaska	13	100	862.79 (823.76)	671.40 (464.87-969.68)	
Total (≥8 years)	Oujé-Bougoumou	190	100	498.65 (541.28)	170.40 (141.02-205.91)	0.116
	Nemaska	82	100	252.42 (207.33)	131.30 (104.12-165.57)	

[1] Detection frequency at the detection limit of 0.02 μg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A9. Plasma concentrations of hexachlorobenzene among the Oujé-Bougoumou and Nemaska participants (µg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)	Geometric mean (95% CI)	p value[2]
8-14 years	Oujé-Bougoumou	21	100.0	6.01 (4.52)	5.23 (4.27-6.39)	0.756
	Nemaska	11	100.0	5.24 (1.75)	4.97 (4.05-6.09)	
Women 15-39 years	Oujé-Bougoumou	78	100.0	8.60 (3.56)	7.99 (7.34-8.69)	0.099
	Nemaska	43	97.7	8.06 (5.51)	6.80 (5.74-8.05)	
Men 15-39 years	Oujé-Bougoumou	40	100.0	11.86 (18.89)	8.96 (7.55-10.63)	0.890
	Nemaska	15	100.0	10.08 (6.71)	8.76 (6.76-11.35)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	40.38 (33.79)	30.34 (24.64-37.35)	0.397
	Nemaska	13	100.0	28.59 (16.21)	25.07 (18.79-33.45)	
Total (≥8 years)	Oujé-Bougoumou	190	100.0	14.89 (10.21)	9.78 (8.75-10.92)	0.066
	Nemaska	82	98.8	10.93 (5.25)	8.13 (6.96-9.49)	

[1] Detection frequency at the detection limit of 0.02 µg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska

Table A10. Plasma concentrations of transnonachlor among the Oujé-Bougoumou and Nemaska participants (µg/kg lipids).

Group	Community	n	% det.[1]	Mean (sd)		Geometric mean (95% CI)		p value[2]
8-14 years	Oujé-Bougoumou	21	4.8	-		-	-	0.038
	Nemaska	11	54.5	-		-	-	
Women 15-39 years	Oujé-Bougoumou	78	82.1	8.22	(6.73)	6.33	(5.42-7.40)	0.154
	Nemaska	43	65.1	7.38	(7.37)	5.19	(4.09-6.57)	
Men 15-39 years	Oujé-Bougoumou	40	90.0	15.32	(34.87)	8.46	(6.47-11.07)	0.461
	Nemaska	15	80.0	11.52	(17.61)	6.96	(4.44-10.90)	
Men & women ≥40 years	Oujé-Bougoumou	51	100.0	79.15	(76.87)	53.97	(41.96-69.41)	0.424
	Nemaska	13	100.0	59.14	(57.46)	43.20	(28.44-65.63)	
Total (≥8 years)	Oujé-Bougoumou	190	80.0	22.31	(22.33)	8.75	(7.37-10.37)	0.178
	Nemaska	82	72.0	15.24	(14.46)	7.12	(5.64-8.98)	

[1] Detection frequency at the detection limit of 0.02 µg/L
[2] Comparison of geometric mean between Oujé-Bougoumou and Nemaska