Environmental Mercury Exposure and Blood Pressure Among Nunavik Inuit Adults

Beatriz Valera, Éric Dewailly, Paul Poirier

Abstract—Epidemiological evidence suggests a negative impact of methylmercury on the cardiovascular system, but findings regarding the effect on blood pressure (BP) are not consistent. We aimed to study the impact of mercury levels on BP among Nunavik Inuit adults. The health survey Qanuippitaa? was conducted in Nunavik (northern Quebec, Canada), and data were obtained from 732 Inuit ≥18 years of age. Anthropometric blood samples, as well as systolic BP and diastolic BP, were assessed. Pulse pressure (systolic BP−diastolic BP) was calculated. Mercury blood concentration was used as a biomarker of recent exposure. Simple relations between mercury and BP parameters were studied by using the Pearson correlation, whereas multiple regressions were performed to control for confounders. Mean age of the participants was 34.3 years (95% CI: 33.6 to 34.9 years). Systolic BP, diastolic BP, and pulse pressure means were 117 mm Hg (95% CI: 116 to 118 mm Hg), 73 mm Hg (95% CI: 72 to 74 mm Hg), and 43 mm Hg (95% CI: 42 to 44 mm Hg), respectively. Mercury mean was 50.2 nmol/L. In multivariable analyses, mercury was associated with systolic BP ($\beta=2.14; P=0.0004$), whereas the association with diastolic BP was near the significance level ($\beta=0.96; P=0.069$). In conclusion, mercury is associated with increasing BP and pulse pressure among Nunavik Inuit adults after considering the effect of fish nutrients (n-3 fatty acids and selenium) and other confounders. (Hypertension. 2009;54:981-986.)

Key Words: mercury ■ blood pressure ■ Inuit ■ Nunavik ■ adults

Mercury is normally present in the soil, but inorganic mercury is also released into the aquatic environment, where it is transformed by algae and bacteria into methylmercury (MeHg). Thus, it can climb up the food chain and be accumulated in predator fish and marine mammals. Populations inhabiting Arctic regions are highly exposed to MeHg, because the traditional diet is mainly based on predator fish and marine mammal consumption. Among Nunavik Inuit adults (northern Quebec, Canada), a health survey conducted in 1992 reported a mean blood mercury concentration of 109 nmol/L (21.8 $\mu$g/L). This concentration was higher than levels found in the southern part of Quebec and other Arctic populations. Epidemiological evidence suggests a negative impact of MeHg on the cardiovascular system, but effects on blood pressure (BP) are not consistent.

Sørensen et al observed increasing systolic BP (SBP) in 7-year-old children from the Faeroe Islands, who had been exposed to high mercury levels during the prenatal period. In contrast, this effect was not observed when the same cohort was re-evaluated at 14 years old. The effect of MeHg on BP in subjects exposed in utero was also studied by Oka et al. In that study, individuals suffering from fetal Minamata disease had lower pulse pressure (PP) than those in the control group. However, no statistical difference was observed in SBP and diastolic BP (DBP). Although the fetus seems susceptible to a mercury impact, recent studies suggest that mercury could also affect BP in environmentally exposed adults. Pedersen et al observed an increase in PP and a decrease in DBP in Greenlanders whose diet is based on traditional food. On the other hand, Fillion et al reported a higher risk of increased SBP among subjects with higher mercury hair levels. A positive association between blood mercury levels and SBP and DBP was reported by Choi et al among Faroese whaling men. Furthermore, a negative impact of mercury on SBP was observed by Vupputuri et al, but only among nonfish consumers.

Taking into account that Inuit from Nunavik are highly exposed to environmental mercury, which could influence BP, we assessed the association between mercury levels and BP considering possible confounding factors, such as age, sex, obesity, cholesterol levels, insulin sensitivity (measured as homeostasis model assessment [HOMA] of insulin resistance [IR]), smoking, alcohol consumption, physical activity, lead, and socioeconomic status. In addition, we aimed to assess the confounding/modifier effect of fish nutrients, such as omega-3 fatty acids and selenium.
as n-3 fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]) and selenium.

Materials and Methods

Study Population and Sampling

In the fall of 2004, the Qanuippitaa? health survey was conducted in the 14 Nunavik communities. The target population included all of the permanent residents, except for non-Inuit households and individuals living full-time in public institutions. The survey plan was a complex, 2-stage stratified random sampling. The first stage involved selecting a stratified random sample of private Inuit households with proportional allocation. The community was the only stratification variable used. Because home addresses (civic numbers) in some municipalities are consecutive, the survey frame was sorted first by home addresses, followed by a systematic draw of a predetermined number of households to avoid selection of 2 immediate neighbors. Because many Inuit regularly move from one house to another, it was decided to sample households instead of individuals. To obtain a good representation of each community, a proportional allocation of sample units corresponding with the size of each village was chosen. In the second stage, all of the eligible individuals were asked to participate according to the survey steps or instruments. A total of 670 Inuit households were eligible, and 521 agreed to participate, which corresponds with a household response rate of 77.8%. Among 1330 adults ≥18 years of age who were eligible for the clinical session, 889 agreed to participate. Taking into account the household response rate, the participation rate for the clinical session was estimated at 52% (77.8×889/1330). A complete set of data was obtained from 806 adults ≥18 years of age. We excluded 74 individuals who were treated for systemic hypertension during the data collection period. Thus, the final sample was composed of 732 individuals. Each individual who agreed to participate in the survey was asked if a subject’s waist was not sufficiently defined, he or she was considered as non-inclusion in the survey.

Data Collection

All of the information was gathered onboard the Amundsen research icebreaker. Questionnaires were used to collect information regarding age, sex, smoking, alcohol consumption, physical activity, and socioeconomic status (measured as total income). During a clinical session, blood samples and anthropometric and physiological measurements were taken. The height of participants was obtained using a health scale. Waist circumference (WC) was measured after exhaling the breath from the mouth placed horizontally to where the abdomen curves in. If a subject’s waist was not sufficiently defined, he or she was measured at the last floating rib. Hip circumference was assessed by placing the measuring tape horizontal to the hips at the pubic symphysis and the most prominent part of the buttocks. All of the foregoing measurements were recorded to the nearest centimeter. Waist:hip ratio (WHR) and body mass index (BMI = weight/height^2) were calculated. BP was measured according to the Canadian Coalition for High Blood Pressure technique using mercury sphygmomanometers, 15-in stethoscopes, and cuffs sized to the subjects’ arms. Before having their BP taken, subjects had to have rested for 5 minutes and not eaten or smoked for ≥30 minutes. Each subject had 3 BP readings, and means of SBP and DBP were calculated using the last 2 readings. SBP and DBP intra-individual variations were 1.9% and 2.4%, respectively. PP (PP = SBP – DBP) was calculated.

Laboratory Analyses

Mercury, lead, and selenium were determined in whole blood, which is a biomarker of recent exposure. Determination was performed by inductively coupled plasma mass spectrometry. For mercury and lead determinations, samples were diluted 20-fold in a solution containing ammonium hydroxide before analysis. The detection limit for mercury, selenium, and lead was 0.5 nmol/L, 0.1 μmol/L, and 1 nmol/L, respectively, and each run of samples included a standard. The interassay variabilities for the mercury, lead, and selenium measurements were 2.1%, 2.8%, and 6.1%, respectively. Analyses were performed by the Institut national de santé publique du Québec Human Toxicology Laboratory, which is accredited International Organization for Standardization 17025 by the Standards Council of Canada. This laboratory is also an international leader in analytic toxicology applied to human and environmental studies and a reference institution for interlaboratory comparison programs in heavy metals measurements. Concentrations of triglycerides, low-density lipoprotein (LDL) cholesterol, and high-density lipoprotein (HDL) cholesterol were determined according to methods of the Lipid Research Clinics (US Department of Health). Determination of insulin was performed with a Roche Modular Analytics E170 (Elecsys module) autoanalyzer using a commercial double-antibody radioimmunoassay. Insulin sensitivity was estimated from the HOMA model as follows: fasting insulin×fasting glucose/22.5. The fatty acid composition of the erythrocyte membranes was measured after membrane purification, chloroform/methanol lipid extraction, and methylation of fatty acids, followed by capillary gas-liquid chromatography using a DB-23 column (39.0×0.25 mm ID×0.25 μm thickness) in a Hewlett-Packard gas chromatograph.

Statistical Analyses

Description of all of the variables was carried out, and those not normally distributed were log-transformed. Participants taking medication for systemic hypertension at the moment of data collection were excluded from the analysis. We tested the interaction between mercury and selenium, as well as between mercury and DHA and EPA. Pearson correlation and simple regression were used to study the relationship between mercury levels and BP parameters, whereas multiple regressions were carried out to control for confounders. Variables considered as potential confounders were age, sex, HDL cholesterol, LDL cholesterol, triglycerides, insulin sensitivity, physical activity, socioeconomic status (measured as total income), smoking, alcohol consumption, n-3 fatty acids (DHA and EPA), selenium, and lead levels. We also verified the confounding effect of obesity (WC, BMI, and WHR) by using independent models that included one of the obesity indices and the other potential confounders. To assess the confounding effect, the change-in-estimate method was used. All of the variables were included in the initial models and considered as confounders if they modulated the regression coefficient ≥10%. We also used the 1-way ANOVA to test differences in PP by quartiles of mercury distribution. Means were also adjusted for the confounding factors detected in the multiple regression models. A P<0.05 was considered of statistical significance. Analyses were performed using SAS version 9.1 and SUDAAN version 9.3 software (SAS Institute).

Taking into account the complex sampling design used in this study, population weights were incorporated into all of the statistical analyses. Weighting participant answers takes into account the probability of selecting each individual as induced by the design of the survey, the rates of nonresponse, and differences observed between the sample and the population. The weight corresponds with the number of persons in the entire population who are represented by the respondent and are adjusted for age, sex, and community. Thus, the estimates generated by using population weights could be generalized to the entire population of Nunavik. To determine variances in the estimates, the bootstrap technique was used. This technique provides precision measurements for estimates obtained from a complex sample design. Details on methodology are available in the methodologic report of the survey.

Results

Characteristics of the participants are presented in Table 1. Mean age of the participants was 34.3 years (95% CI: 33.6 to 34.9 years), and the sample was composed of 319 men (34.1 years [95% CI: 33.2 to 35.1 years]) and 413 women (34.4 years [95% CI: 33.6 to 35.2 years]). Mercury levels did not
Table 1. Characteristics of the Participants and BP Parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>Mean (95% CI)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>731</td>
<td>34.3 (33.6 to 34.9)</td>
<td>18.0 to 71.0</td>
</tr>
<tr>
<td>Sex, N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>319 (49.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>413 (50.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mercury, mmol/L*</td>
<td>732</td>
<td>50.2 (46.6 to 54.1)</td>
<td>1.0 to 1200.0</td>
</tr>
<tr>
<td>Lead, μmol/L*</td>
<td>732</td>
<td>0.19 (0.18 to 0.20)</td>
<td>0.03 to 2.4</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>624</td>
<td>2.85 (2.77 to 2.92)</td>
<td>0.4 to 6.1</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>624</td>
<td>1.65 (1.61 to 1.69)</td>
<td>0.7 to 3.4</td>
</tr>
<tr>
<td>Triglycerides, mmol/L*</td>
<td>732</td>
<td>1.18 (1.13 to 1.24)</td>
<td>0.3 to 5.5</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L*</td>
<td>732</td>
<td>4.35 (4.29 to 4.40)</td>
<td>2.5 to 8.5</td>
</tr>
<tr>
<td>Fasting insulin, pmol/L*</td>
<td>732</td>
<td>46.9 (45.0 to 46.9)</td>
<td>13.0 to 336.0</td>
</tr>
<tr>
<td>HOMA-IR*</td>
<td>621</td>
<td>9.1 (8.6 to 9.5)</td>
<td>1.7 to 82.1</td>
</tr>
<tr>
<td>BMI, kg/m²*</td>
<td>692</td>
<td>26.4 (26.0 to 26.8)</td>
<td>17.2 to 46.1</td>
</tr>
<tr>
<td>WHR*</td>
<td>701</td>
<td>0.88 (0.87 to 0.89)</td>
<td>0.7 to 1.2</td>
</tr>
<tr>
<td>WC, cm</td>
<td>701</td>
<td>90 (89 to 91)</td>
<td>63 to 131</td>
</tr>
<tr>
<td>EPA, % fatty acid*</td>
<td>732</td>
<td>1.12 (1.07 to 1.18)</td>
<td>0.2 to 7.3</td>
</tr>
<tr>
<td>DHA, % fatty acid</td>
<td>732</td>
<td>5.16 (5.04 to 5.29)</td>
<td>0.7 to 9.9</td>
</tr>
<tr>
<td>Selenium, μmol/L*</td>
<td>732</td>
<td>3.70 (3.58 to 3.82)</td>
<td>1.5 to 4.5</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>732</td>
<td>117 (116 to 118)</td>
<td>85 to 187</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>732</td>
<td>73 (72 to 74)</td>
<td>50 to 111</td>
</tr>
<tr>
<td>PP, mm Hg</td>
<td>732</td>
<td>43 (42 to 44)</td>
<td>19 to 91</td>
</tr>
</tbody>
</table>

Alcohol consumption, N (%)  
Drinkers 633 (89.7)  
Nondrinkers 76 (10.3)

Total income, N (%)  
Less than $20 000 341 (54.1)  
$20 000 to $40 000 180 (26.9)  
$40 000 to $60 000 101 (15.5)  
$60 000 or more 24 (3.5)

Physical activity, N (%)  
Active 126 (18.1)  
Moderate active 49 (6.9)  
Somewhat active 47 (6.3)  
Sedentary 493 (68.7)

Smoking habits, N (%)  
Smokers 544 (74.6)  
Ex-smokers 45 (5.9)  
Nonsmokers 140 (19.2)

To convert to micrograms per liter, mercury concentration in nanomoles per liter must be divided by 5.0.
*For log-transformed variables, the geometric means are presented.

Differ between men and women (P = 0.07). Blood mercury concentrations increased with age (r = 0.48; P < 0.0001) and were positively correlated with selenium (r = 0.67; P < 0.0001), EPA (r = 0.67; P < 0.0001), and DHA (r = 0.61; P < 0.0001). Means of obesity indices (WC, BMI, and WHR) were 90 cm (95% CI: 89 to 91 cm), 26.4 kg/m² (95% CI: 26.0 to 26.8 kg/m²), and 0.88 (95% CI: 0.87 to 0.89), respectively. WC and BMI did not differ by sex, whereas WHR was slightly higher in men than in women (0.89 vs 0.88; P = 0.046).

Results of simple and multiple analyses between mercury and BP parameters are shown in Table 2. The interaction terms between mercury and selenium, as well as between mercury and EPA and DHA, were not statistically significant (P > 0.05 in all of the models). After adjusting for confounders, the association with SBP remained significant (β = 2.14, P = 0.0004). In this model, EPA and selenium were negatively associated with SBP (β = −1.75, P = 0.049 and β = −2.80, P = 0.025, respectively). With respect to WC, adjustments for BMI and WHR decreased the regression coefficients between mercury and SBP, but the association remained significant. WC, BMI, and WHR explained 7.9% (P < 0.0001), 7.1% (P < 0.0001), and 3.4% (P < 0.0001), respectively, of the SBP variance. DBP tended to increase with blood mercury concentrations (β = 0.96; P = 0.069) in multivariable analyses. In this model, EPA was negatively associated with DBP (β = −2.08; P = 0.005), whereas the association with selenium was near the significance level (β = −1.73; P = 0.072). Taking into account that mercury was log-transformed, a 1% increase in blood mercury concentration translates into an increase of 0.02 mm Hg in SBP after controlling for confounders. The analysis of quartiles of mercury concentrations revealed significant differences in PP means between quartiles 2 and 4 (42 mm Hg [95% CI: 41 to 43 mm Hg] versus 45 mm Hg [95% CI: 44 to 47 mm Hg]; P = 0.0017) after adjusting for age, age², selenium, EPA, LDL cholesterol, and HOMA-IR.

Discussion

In the present study, we observed a positive association between mercury levels and BP among Nunavik Inuit adults, whose diet is still mainly based on traditional food. Blood mercury concentrations were associated with increasing SBP and PP after controlling for the confounding factors. Fish nutrients, such as n-3 fatty acids and selenium, did not show a modifier effect on the relationship between mercury and BP parameters. However, they had a strong confounding effect, suggesting that not adjusting for these substances could lead to an underestimation of the effect size. To our knowledge, this study is the first to evaluate the influence of levels of n-3 fatty acids and selenium on the association between mercury and BP.

Regarding studies conducted in adults, we have previously reported a positive association between mercury and SBP and PP in a smaller group of only 205 Inuit adults ≥40 years of age with valid Holter data. However, in the current study including more adults ≥40 years of age, as well as younger adults, we obtained consistent results. Our results are also in accordance with those published by Fillion et al. They observed that individuals with higher hair mercury levels had higher risk of increased SBP. A positive association between mercury and SBP and DBP was also reported by Choi et al., which concurs with our results. In contrast, our results partially agree with those published by Pedersen et al. They observed a positive association between blood MeHg concentrations and PP, whereas a negative association was observed with DBP, and no significant association was observed with SBP. In that study, statistical models were...
Mercury and BP Parameters

<table>
<thead>
<tr>
<th>BP Parameters</th>
<th>Simple Correlation, Crude β (95% CI)</th>
<th>Multiple Regression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Adjusted β (95% CI)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Model ( R^2 )</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>0.10*</td>
<td>2.14 (0.94 to 3.33)†§</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>1.27 (0.30 to 2.25)*</td>
<td>1.87 (0.68 to 3.06)§</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>1.79 (0.59 to 2.99)*§</td>
<td>0.20†</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>0.96 (–0.08 to 1.99)</td>
<td></td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>0.02</td>
<td>0.17 (–0.46 to 0.79)</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>0.91 (–0.14 to 1.95)</td>
<td></td>
</tr>
</tbody>
</table>

Confounders were selected using the change-in-estimate method (≥10% change in the regression coefficient).

*P<0.05.
†P<0.001.
‡P<0.0001.
§SBP model was adjusted for age, age\(^2\), sex, EPA, selenium, alcohol consumption, and WC. In the second and third models, WC was replaced by BMI and WHR, respectively.
||DBP model was adjusted for age, age\(^2\), sex, selenium, EPA, lead, LDL cholesterol, HDL cholesterol, alcohol intake, and WC. In the second and third models, WC was replaced by BMI and WHR, respectively.

adjusted only for age and BMI. However, no adjustment for other BP risk factors, as well as fish nutrients, such as n-3 fatty acids and selenium, could have influenced the results. n-3 fatty acids and selenium are correlated with MeHg blood concentrations because of the same exposure source (fish and marine mammals), and, also, they can influence BP.23–25 It has been suggested previously that no adjustment for these fish nutrients could result in a negative confounding effect and, consequently, underestimates the effect of mercury.26,27

To understand the differences in results, we conducted a sensitivity analysis by including the same confounders used by Pedersen et al.11 After adjusting for BMI and age, the regression coefficients for SBP (β=0.01; P=0.99), DBP (β=–0.48; P=0.18), and PP (β=0.49; P=0.19) were not statistically significant. In further analyses, we included other traditional BP risk factors (sex, smoking, triglycerides, cholesterol, insulin resistance, alcohol, physical activity, and socioeconomic status), and no significant results were obtained. However, when we included n-3 fatty acids and selenium, mercury was significantly associated with increasing SBP. In our data, we observed that the exclusion of EPA and selenium from the statistical models had a strong influence on the association between MeHg and BP parameters. In the SBP model, the regression coefficient decreased from 2.14 to 1.63 (24%) after removing EPA and from 2.14 to 1.33 (38%) after removing selenium. In the DBP model, the regression coefficient decreased from 0.96 to 0.45 (53%) after removing EPA and from 0.96 to 0.40 (58%) after removing selenium. Finally, our results partially agree with those reported by Vupputuri et al.,15 where an adverse association between mercury and SBP was observed only among nonfish consumers. In that study, authors suggest that fish consumers could be protected by n-3 fatty acids. However, these substances were not controlled as confounders.

From experimental studies, Wakita28 observed that rats chronically exposed to MeHg developed systemic hypertension that persisted for many months after exposure. Increased BP was also detected in rats exposed to MeHg for 26 days.29 Mercury could modulate BP by different mechanisms. As suggested by Sakamoto et al.,30 toxicity of MeHg could be related to calcium homeostasis. They observed that administration of calcium channel blockers decreased the toxicity of MeHg in vivo and in vitro studies. Their results suggest that MeHg causes excessive calcium influx and/or increased opening of voltage-sensitive calcium channels. Also, the capacity of MeHg to increase intracellular calcium and/or to activate some calcium-dependent reactions has been reported previously.31–33 Moreover, chronic exposure to low concentrations of MeHg increases oxidative stress, which leads to reduction in NO bioavailability, endothelial dysfunction, and decreased smooth muscle relaxation.34 Administration of MeHg also results in decreased acetylcholine levels in the brain, liver, kidney, and serum of rats.35 A decrease in acetylcholine levels could lead to decreased relaxation, indirectly increasing BP.

Limitations and Strengths of the Study

With a response rate to the clinical session of 52%, a selection bias could have been introduced if individuals who did not agree to participate differed from those included in the analyses. However, the weighting method used in this study took into account differences in age, sex, and community between respondents and nonrespondents, which minimized the risk of a selection bias. Another potential limitation of our study is the use of casual BP measurement instead of ambulatory BP measurement. Ambulatory BP measurement provides a profile of BP away from the medical environment and shows BP behavior over a 24-hour period during the patient’s usual daily activities. However, in the context of our study, 24-hour ambulatory BP measurements were not possible, because data collection was carried out on board an icebreaker, and, in some communities, the research team stayed <24 hours. Knowing this limitation, we tried to minimize the bias caused by the stress of being in a doctor’s
office, and we observed only slight intraindividual variations in SBP and DBP (1.9% and 2.4%, respectively). Furthermore, the method used for measuring BP is in agreement with recent European and American guidelines. Another possible limitation is that level of physical activity takes into account only leisure-time physical activity. This measure may not represent the real level of physical activity, because domestic, work-based, transportation-based and other activities were not considered. In our study, leisure-time physical activity was not a confounder in BP models. However, it is possible that a residual confounding bias attributed to physical activity other than leisure time affects the results. Another limitation of this study is that individual salt intake could not be assessed, because only a 24-hour dietary recall was used. However, taking into account information on food consumed the day before the survey, we described sodium intake by sex and groups of age. Sixty-one percent of the population had excessive sodium intake (≥2300 mg/d). The median intake of sodium was higher in men than in women, and men aged 30 to 49 years had the highest sodium intake among all sex/age groups. Not controlling for sodium intake could result in residual confounding bias. However, we compared BP by quartile of sodium intake, and no significant differences were observed in SBP, DBP, and PP means. Also, mercury was inversely correlated with sodium intake (Spearman $r = -0.11$; $P = 0.0064$). Consequently, it is unlikely that the association observed between mercury and BP is attributable to sodium intake. We also deal with the limitation of cross-sectional designs, where it is not possible to establish a cause-effect relationship. However, to minimize the bias attributed to the temporality, we excluded the hypertensive participants, which avoids the overestimation of the association between mercury and BP. Hopefully, the associations obtained in this cross-sectional study could be tested in a cohort study that will be conducted in this population in a near future. This study will allow us to determine whether mercury exposure is associated with the development of hypertension.

Strengths of our study include the minimization of the information and confounding bias, which increases the internal validity of the study. Mercury determination was carried out by the Institut national de santé publique du Québec Human Toxicology Laboratory, which is accredited International Organization for Standardization 17025 by the Standards Council of Canada. This laboratory is also an international leader in analytic toxicology applied to human and environmental studies and a reference institution for interlaboratory comparison programs in heavy metals measurements. Also, in this population, we have reported previously that mercury exposure is stable over time. For example, the Pearson $r$ correlation coefficient was 0.74 ($P<0.0001$) between maternal blood and last-trimester hair concentrations, suggesting that blood mercury is a good indicator of exposure for the last months. To minimize the confounding bias, we took into account traditional BP risk factors (age, sex, obesity [BMI, WC, and WHR], insulin sensitivity [measured as HOMA-IR], cholesterol [HDL and LDL cholesterol], and triglycerides levels, smoking habits, alcohol consumption, physical activity, and socioeconomic status). We also considered lead exposure, which was correlated with mercury and could influence BP. In addition, we included n-3 fatty acids and selenium levels, because their blood levels are high among Nunavik Inuit adults, especially considering that a protector effect of these substances on BP has been suggested. However, the associations between mercury and BP parameters remained significant, which may suggest that the negative impact of mercury on BP may be stronger than the possible protective effect of n-3 fatty acids and selenium.

Perspectives

The results of the present study obtained in a highly mercury-exposed population help us to better understand the cardiovascular mercury toxicity and to assess the risks/benefits of fish consumption. This aspect is very important for Nunavik Inuit, because traditional food is an important element of their culture. Moreover, this study constitutes the baseline of a cohort study that will allow us to determine whether mercury exposure is associated with the development of hypertension. Finally, the observation that WC, in comparison with BMI, was a better predictor of SBP is of interest in light of the refinement needed in obesity assessment in clinical practice.

Conclusions

Even in the setting of BP within the normal range and after controlling for fish nutrients (n-3 fatty acids and selenium) and other confounding factors, mercury is associated with higher BP and PP among Nunavik Inuit adults.

Acknowledgments

We thank Serge Déry, who was 1 of the scientific directors of the survey, as well as Danielle St-Laurent, who was the executive director. We also thank all of the professionals who worked in the data collection, as well as Elhadji A. Laouan Sidé for the statistical comments. Finally, we thank the residents of Nunavik.

Sources of Funding

This study was funded by the Québec Ministry of Health, the Nunavik Regional Board of Health and Social Services, the Canadian Institutes for Health Research, the Fonds en Recherche en Santé du Québec, the Northern Contaminants Program, and the ArcticNet network. B.V. is a doctorate scholar from Nasivik. P.P. is a clinician-research scholar from the Fonds de Recherche en Santé du Québec.

Disclosures

None.

References


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Hypertension. 2009;54:981-986; originally published online October 5, 2009;
doi: 10.1161/HYPERTENSIONAHA.109.135046

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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