BLOOD LEAD LEVELS IN CHILDREN AGED 24 TO 36 MONTHS IN VANCOUVER

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Abstract • Résumé

Objectives: To determine the blood lead levels in children and to identify risk factors for elevated levels.

Design: Cross-sectional study.

Setting: Vancouver.

Participants: Random sample of children aged 24 to 36 months, born and still resident in Vancouver. The sample was stratified proportionally by the median annual family income in the census tract where each family resided.

Outcome measures: Blood lead levels and risk factors for elevated blood lead levels, determined from a questionnaire administered to parents.

Results: Of the children in the sample, 42% (178/422) were ineligible or could not be located. Of the remaining children, 73% (177/244) participated and adequate blood specimens were obtained from 172. The mean blood lead level was 0.29 μmol/L (standard deviation 0.13 μmol/L). (A blood lead level of 1 μmol/L is equivalent to 20.7 μg/dL.) The lowest level was 0.06 μmol/L, and the highest was 0.85 μmol/L. Of children with adequate samples, 8.1% (14/172) had blood lead levels of 0.48 μmol/L or higher, and 0.6% (1/172) had a level higher than 0.72 μmol/L. The logarithms of the levels were normally distributed, with a geometric mean (GM) of 0.26 μmol/L (geometric standard deviation 1.56). Of approximately 70 possible predictors of blood lead levels analysed, those that showed a statistically significant association ($p < 0.05$) with increased blood lead levels were soldering performed in the home as part of an electronics hobby (GM blood lead level 0.34 μmol/L, 95% confidence interval [CI] 0.27 to 0.39 μmol/L), aboriginal heritage (GM blood lead level 0.33 μmol/L, 95% CI 0.28 to 0.39 μmol/L), dwelling built before 1921 (GM blood lead level 0.32 μmol/L, 95% CI 0.28 to 0.37 μmol/L), age of water service connection to dwelling (predicted blood lead level 0.00087 μmol/L [95% CI 0.00005 to 0.00169 μmol/L] higher per year since service connection) and decreased stature (predicted blood lead level 0.018 μmol/L [95% CI 0.0353 to 0.0015 μmol/L] higher for every standard deviation below the age-specific mean height).

Conclusions: This study found much lower blood lead levels in children than those found in previous Canadian studies. The authors believe that this result is not an artefact due to differences in population sampling or methods of collection of blood specimens. The study showed no clear risk factors for elevated blood lead levels: although a few factors had a statistically significant association with increased blood lead levels, the differences in levels were small and unimportant.

Objectifs : Déterminer les taux de plombémie chez les enfants et identifier les facteurs qui risquent de causer des taux élevés.

Conception : Étude transversale.

Contexte : Vancouver.

Participants : Échantillon aléatoire d'enfants âgés de 24 à 36 mois nés à Vancouver et qui y résident encore. L'échantillon a été stratifié proportionnellement au revenu familial annuel médian du secteur de recensement où résidait chaque famille.

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Mesures des résultats : Taux de plombémie et facteurs qui risquent de causer des taux de plombémie élevés, déterminés à la suite d'un questionnaire administré aux parents.

Résultats : Parmi les enfants de l'échantillon, 42 % (178/422) n'étaient pas admissibles ou n'ont pu être localisés. Des enfants restants, 73 % (177/244) ont participé et l'on a obtenu des spécimens de sang suffisants de 172 d'entre eux. Le taux de plombémie moyen était de 0,29 µmol/L (écart type de 0,13 µmol/L). (Un taux de plombémie de 1 µmol/L équivaut à 20,7 µg/dL.) Le taux le plus faible était de 0,06 µmol/L et le plus élevé, de 0,85 µmol/L. Parmi les enfants qui ont fourni des spécimens suffisants, 8,1 % (14/172) avaient des taux de plombémie de 0,48 µmol/L ou plus et 0,6 % (1/172) avait un taux de plus de 0,72 µmol/L. Les logarithmes des taux étaient distribués normalement, leur moyenne géométrique (MG) s'établissant à 0,26 µmol/L (écart type géométrique de 1,56). Sur quelque 70 prédicteurs possibles des taux de plombémie analysés, ceux qui ont montré un lien important sur le plan statistique ($p < 0,05$) avec des taux de plombémie élevés étaient des travaux de soudure exécutés au foyer dans le cadre d'un passe-temps en électronique (MG du taux de plombémie de 0,34 µmol/L, intervalle de confiance [IC] à 95 % de 0,27 à 0,39 µmol/L), l'ascendance autochtone (MG du taux de plombémie de 0,33 µmol/L, IC à 95 % de 0,28 à 0,39 µmol/L), les logements construits avant 1921 (MG du taux de plombémie de 0,32 µmol/L, IC à 95 % de 0,28 à 0,37 µmol/L), l'âge du raccordement du service d'aqueduc au logement (taux de plombémie prévu augmentant de 0,00087 µmol/L [IC à 95 % de 0,00005 à 0,00169 µmol/L] chaque année depuis le raccordement du service) et la petite taille (taux de plombémie prévu de 0,018 µmol/L, [IC à 95 % de 0,0353 à 0,0015 µmol/L] plus élevé par écart type au-dessous de la taille moyenne selon l'âge).

Conclusions : Cette étude a révélé des taux de plombémie beaucoup plus faibles chez les enfants que ceux qu'ont révélés des études canadiennes antérieures. Les auteurs sont d'avis que ce résultat n'est pas un artefact attribuable à des différences au niveau de l'échantillonnage de la population ou des méthodes de collecte des spécimens de sang. L'étude n'a indiqué aucun facteur évident qui risque de hausser les taux de plombémie : même s'il y avait un lien important sur le plan statistique entre quelques facteurs et des taux de plombémie élevés, les écarts entre les taux étaient minces et sans importance.

Symptomatic lead poisoning is rare, and it is mainly confined to people who work or live in areas of environmental lead contamination. However, during the past decade, accumulated evidence has shown that long-term, low-level lead exposure has other, less obvious, effects on health; in particular, it has been shown to have subtle but measurable adverse effects on the mental development, and possibly the hearing and growth, of young children.[1-12] Such effects are dose-related and have been observed in children with blood lead levels much lower than 1.93 µmol/L, the level at which symptoms of toxicity appear. Some authorities believe that there is a threshold for adverse effects of lead; for example, the Royal Society of Canada's Commission on Lead in the Environment has suggested that a blood lead level of 0.97 µmol/L is the threshold for adverse effects.[2] However, the US Environmental Protection Agency has stated that a blood lead level of 0.48 to 0.72 µmol/L is the "lowest observed effect level," with the proviso that this does not mean that levels below 0.48 µmol/L are harmless.[1,3] In any case, levels of 0.72 to 0.97 µmol/L are thought to be consistent with the "background" lead contamination to which the general population, especially in cities, is exposed. For example, in the United States, the Agency for Toxic Substances and Disease Registry has estimated that about 2.4 million US children (17% of the urban population 6 months to 5 years of age) have a blood lead level of 0.72 µmol/L or higher.[3] In Canada, a 1984 survey of Ontario children 3 to 6 years of age found that 25% of urban children tested had a blood lead level of 0.72 µmol/L or higher, and 5.6% had a blood lead level of 0.97 µmol/L or higher.[13,14]

This article describes a cross-sectional study of the blood lead levels in children, conducted during the fall of 1989 in the City of Vancouver.[15] The study was prompted by results of earlier environmental investigations of lead levels in the soil and the drinking water in the Vancouver area.

In 1977 specimens of soil and street-gutter dust along main roads in Vancouver were collected as controls for a study of soil lead levels in residential neighbourhoods in Trail, BC, the site of a large primary lead and zinc smelter.[16] High soil lead levels (mean 6.4 µmol/g [1320 ppm]) were found in Trail, but levels in Vancouver were even higher (mean 7.5 µmol/g [1545 ppm]). The only apparent source of lead in the Vancouver soil specimens was emissions from motor vehicles. In a soil survey conducted by the City of Vancouver Health Department in 1987, measurements taken at the same sites as those taken during the 1977 study were generally lower (mean 3.5 µmol/g [716 ppm], standard deviation [SD] 4.6 µmol/g).[17] However, many sites still had soil lead levels above the thresholds for intervention identified by the Royal Society of Canada's Commission on Lead in the Environment: 2.4 µmol/g (500 ppm) for residential land and 4.8 µmol/g (1000 ppm) for parkland.[2] However, most of the sites of soil collection in Vancouver in the 1977 and 1987 studies were neither residential areas nor

parkland. In June 1988 the City of Vancouver Health Department conducted a further survey of soil lead levels in residential neighbourhoods near corridors of dense motor-vehicle traffic.[18] Soil specimens from 80 test sites had a mean lead level of 1.8 μmol/g (380 ppm, SD 2.4 μmol/g), 24% of sites had a level over 2.4 μmol/g and 9% had a level over 4.8 μmol/g.

In 1987–88, the Greater Vancouver Regional District conducted studies of lead levels in tap water in homes and schools.[19] These showed lead levels in excess of guideline levels set by the Department of National Health and Welfare,[20] but such levels were found only in the first litre of water poured when the tap was turned on, particularly when the tap was first used in the morning. This high level was due to the acidity and softness of the region's water supply, characteristics that promote the leaching of lead from pipes and solder, especially when water has been standing in pipes for long periods.

The significance of these soil and water findings for human absorption of lead was not clear. The Royal Society of Canada's Commission on Lead in the Environment had based its criteria for intervention on research on the correlation of blood lead levels with soil lead levels in residential areas surrounding lead smelters.[2] Such research is of questionable use in interpreting the Vancouver studies of lead levels in soil, which investigated the environmental distribution of lead from a diffuse source, motor-vehicle traffic, and consequently used very different strategies to select and map testing sites. It is also questionable whether the Canadian standard for lead in drinking water (a maximum allowable level of 0.24 μmol/L), which is based on a theoretic risk-assessment model,[21] is applicable. Although blood lead levels have been correlated with levels of lead in drinking water in epidemiologic studies,[2,22,23] differences in sampling strategies (the amount sampled, the timing of collection and the location of sites within the water-distribution system) between these studies and those in Vancouver also call into question the application of previous research. The results of the studies of lead levels in Vancouver soil and drinking water suggested a health hazard; however, it was recognized that the extent and magnitude of the risk could best be determined through a study of blood lead levels in the Vancouver population. Because children are at risk of suffering the subtle adverse effects of long-term low-level exposure to lead, they were the population of interest.

The specific objectives of the study were to determine blood lead levels in Vancouver children aged 24 to 36 months, to describe the frequency distribution (mean and percentiles) of these levels and to identify socioeconomic and environmental risk factors for elevated blood lead levels.

We chose this age range because the age of greatest risk is thought to be 12 to 36 months.[10] Children of this age have a high sensitivity to the toxic effects of lead (highest in fetuses and infants and declining with age), many opportunities for exposure (increasing with age) and a high level of hand-to-mouth behaviour (greatest in those under 36 months of age), which facilitates ingestion of environmental lead. Prospective studies have shown that blood lead levels in children increase steadily after about 6 months of age, peak at about 24 months and decline thereafter.[7,24] We would have liked to include children aged 12 to 24 months, but previous surveys involving children under 2 years of age reported problems in the recruitment and cooperation of subjects.[25]

The chosen time of year for the study was based on seasonal variation in children's blood lead levels; levels are thought to be highest May through October and lowest during the winter in North America.[10] Cold, wet weather is believed to prevent children from playing outside in the dirt and to reduce dust dispersion in the environment. We would have liked to conduct the study entirely during the season of high blood lead levels; however, we anticipated difficulty in locating and contacting subjects during the summer. Therefore, we decided to conduct the study in the autumn.

Methods

The methods used were reviewed and approved by the University of British Columbia Clinical Screening Committee for Research and Other Studies Involving Human Subjects.

Sampling and recruitment

We sampled the population of children aged 24 to 36 months, born and still resident in the City of Vancouver. The sample was drawn from a frame consisting of 5520 births to families with an address in the City of Vancouver between Oct. 1, 1986, and Sept. 30, 1987, listed on the computerized birth-notice registry in the City of Vancouver Health Department. The registry receives notice of all births to families resident in the city through liaison with hospitals in the greater Vancouver area and with the vital-statistics divisions of the British Columbia government and other provincial governments. To represent fairly children from all socioeconomic levels, we used a stratified random-sampling technique. We divided the frame into six levels based on the median annual family income, according to the 1986 census, in the census tract in which each family resided.[26,27] We then drew randomly and proportionally from each level. We selected children by lottery, with the use of random numbers generated by the statistical software package SAS, Release 6.03 (SAS Institute Inc., Cary, NC). We drew 422 names altogether.

After a publicity campaign through the mainstream English-language media and selected ethnic media, a team of study nurses contacted the chosen families first by letter and then by telephone and in-person canvassing to verify eligibility and persuade them to participate. For 104 of the 422 families, the address and telephone number from the birth-notice registry were no longer valid, so the team used a standard search protocol, following leads from the telephone directory, the city directory[28] and current occupants and neighbours. A nurse visited the home of each family that agreed to participate and obtained formal written consent before data and specimen collection. Recruitment and home visits occurred from Oct. 17 to Dec. 13, 1989.

Of the names drawn, 16% (67/422) refused to participate, 20% (85/422) could not be located and 22% (93/422) were located but were ineligible because they had moved out of the City of Vancouver (83), there was an incorrect birth residence or birth date on the birth notice (4) or the child had died (6). We did not attempt to ascertain the cause of death for the six children who had died. We believe that most of the children we could not locate had moved out of the city, and so were likely ineligible. Excluding those children who were ineligible or likely ineligible, the participation rate was 73% (177/244). Characteristics (socioeconomic level, household language, child's sex and age, and mother's age and reproductive history) of participants and nonparticipants are given in Table 1.

Table 1: Characteristics of study participants and nonparticipants

	No. (and %), except where otherwise indicated			
		Nonparticipants		
Characteristic	Participants	Refused	Not found or ineligible	Total sample
Families				
Income level, $*				
47 687–116 264	31 (18)	8 (12)	11 (6)	50 (12)
35 733– 45 666	24 (14)	9 (13)	21 (12)	54 (13)
30 886– 35 438	35 (20)	11 (16)	38 (21)	84 (20)
28 199– 30 570	38 (21)	18 (27)	35 (20)	91 (22)
25 232– 27 555	25 (14)	12 (18)	30 (17)	67 (16)
9 943– 23 671	24 (14)	9 (13)	43 (24)	76 (18)
Language†				
English/other	121 (68)	30 (45)	121 (68)	272 (64)
Chinese	31 (18)	22 (33)	28 (16)	81 (19)
Punjabi	11 (6)	6 (9)	13 (7)	30 (7)
Spanish	6 (3)	5 (7)	5 (3)	16 (4)
Hindi	4 (2)	2 (3)	6 (4)	12 (3)
Vietnamese	4 (2)	2 (3)	5 (3)	11 (3)
Total	177 (100‡)	67 (100‡)	178 (100‡)	422 (100‡)
Children				
Sex (no. and % female)	88 (50)	33 (49)	83 (47)	203 (48)
Mean age, as of Oct. 1, 1989, mo	30.7	30.6	30.0	30.3
Mothers				
Mean age at birth of child, yr	29.5	30.2	27.6	28.8
Mean no. of pregnancies, including subject child	2.4	2.4	2.1	2.2
Mean no. of live births, including subject child	1.9	1.8	1.7	1.8

*Estimated as the median annual family income in the census tract in which the family resided.
†Language used when the family was contacted or interviewed; if no contact was made, the home language was inferred from the family name.
‡Percentages may not sum to 100 because of rounding.

Blood-specimen collection and laboratory analysis

The nurse collected blood from the children in their homes with the use of fingerprick capillary sampling. The protocol involved washing the child's hands with warm water and soap, rinsing the finger with distilled water, cleansing it with an alcohol swab and drying it with cotton gauze before the fingerprick.[15] The first drop of blood was discarded, then a free-flowing sample of 0.6 mL of capillary whole blood was collected in a heparinized Microtainer (Becton–Dickinson, Rutherford, NJ). Validation of this protocol in a laboratory, by comparison with venipuncture, has yielded a coefficient of determination (r^2) of 0.986.[29] This protocol is the same as the capillary sampling protocol later recommended by the US Centers for Disease Control and Prevention (CDC), Atlanta,[11] except that we rinsed the finger with distilled water and we did not apply a silicone-barrier spray to the skin before the fingerprick.

Blood specimens were transported to the laboratory on ice within 48 hours. Blood lead was measured with the use of a graphite-furnace atomic-absorption spectrophotometer with an ammonium phosphate modifier,[29] the Varian SpectrAA-300 with a model GTA-96 graphite-tube atomizer and Zeeman background correction (Varian Canada Inc., Georgetown, Ont.). The coefficient of variation among runs was 2.2% at a blood lead concentration of 3.70 μmol/L and 7.3% at 0.23 μmol/L. According to results of the 1989 Quebec Interlaboratory Comparison Program, the accuracy and precision rating of the laboratory's whole-blood analyses was 96%.[30] (A lead concentration in whole blood of 1 μmol/L is equivalent to a blood lead level of 20.7 μg/dL.)

In specimens with a sufficient amount of blood, we also measured levels of zinc protoporphyrin. Elevated levels of zinc or erythrocyte protoporphyrin, which indicate impairment of heme synthesis, occur in mild or subclinical lead toxicity. Such levels also occur in iron deficiency, which increases the absorption of lead in the digestive tract and predisposes to pica. We measured the blood level of zinc protoporphyrin in order to assess its usefulness as a predictor of blood lead levels and to help us decide on appropriate follow-up for any children with elevated blood lead levels. We based our plan of response to test results on recommendations made by the CDC in 1985, in which blood lead and protoporphyrin levels are used to classify children for the purpose of investigation and intervention.[10]

Levels of zinc protoporphyrin were measured by hematofluorometry (Helena Laboratories, Beaumont, Tex.). For internal quality control, we used a whole-blood-hemolysate pool, frozen at −70°C in aliquot portions. For a mean zinc protoporphyrin level of 37 μmol/μmol of heme, the coefficient of variation was 8.5%. A concentration of zinc protoporphyrin of 70 μmol/μmol of heme is approximately equal to an erythrocyte protoporphyrin level of 0.62 μmol/L (35 μg/dL) of whole blood.[31]

Because of the possibility of spuriously high measurements of blood lead levels as a result of contamination of fingerprick specimens by lead on the skin,[10,11,32,33] we decided before testing the specimens to confirm any blood lead level of 0.97 μmol/L or higher by repeating the test with a blood specimen obtained by venipuncture.

Assessment of risk factors

For each participating child and his or her family, we obtained a sociodemographic profile and data on factors hypothesized to increase the risk of elevated blood lead levels. To obtain these data, the nurse administered to parents in each household a questionnaire on the child's age, sex and ethnic background, the mother's reproductive history (age, number of pregnancies and births), the child's diet and health, the family's social and economic characteristics (household language, number of parents and siblings in the home, parents' education, occupation and employment status, use of day care, presence of a telephone, home ownership and duration of residence in the home), housing characteristics (age of dwelling and type of heating), lead exposure from home hobbies and lead exposure in the parents' occupations. The items on the questionnaire were adapted from the 1986 Census of Canada[34] (for socioeconomic and housing characteristics and for description of occupation), Canada's Health Promotion Survey[35] (for socioeconomic characteristics), the Child and Family Questionnaire used in the US Environmental Protection Agency–CDC Lead Soil Abatement Project[36] (for hobbies and occupations involving lead), the 1984 Ontario blood lead survey questionnaire[13] (for hobbies) and the Risk Factor Questionnaire used by the City of Toronto Health Department in its 1984–88 surveys of the South Riverdale neighbourhood[37] (for housing characteristics).

The nurse also measured the child's height and weight as indicators of health and nutritional status as well as assessing the quality of housing for such problems as peeling paint or structural deterioration by visual inspection. We determined the age of water-service connections to the home from municipal tax records, and we obtained data on the volume of motor-vehicle traffic near the home from the records of municipal traffic-management and planning programs.

A vocational-rehabilitation consultant reviewed the parents' job descriptions and classified them by standard job titles and 4-digit codes according to the *Canadian Classification and Dictionary of Occupations*.[38] We used job ti-

tles to assign to fathers a socioeconomic status score based on the Blishen Index,[39] which rates job titles of men in Canada according to social prestige, income and educational requirement. We estimated parents' income on the basis of the average employment income in British Columbia, according to the 1986 census, for their sex and job category (the 4-digit code assigned earlier).[40]

Statistical analysis

We tested whether the frequency of the blood lead levels obtained was normally distributed with the Shapiro–Wilk *W* statistic. As previous studies had found,[1,2] to achieve a normal distribution it was necessary to transform the data by taking the natural logarithm of each measured blood lead level. This manoeuvre enabled us to describe succinctly the frequency distribution of the logarithms of the blood lead levels using the statistics of a normal distribution (mean and SD) and to use powerful parametric methods for comparisons among subgroups. To facilitate understanding of the transformed data, we followed certain descriptive conventions. The geometric mean (GM) of the blood lead levels is the antilogarithm of the arithmetic mean of the logarithms of these levels. Similarly, the geometric standard deviation (GSD) of blood lead levels is the antilogarithm of the arithmetic SD of the logarithms of these levels. The 95% confidence limits for the GM blood lead level are the antilogarithms of the 95% confidence limits for the arithmetic mean of the logarithms of the levels.

We converted the height and weight measurements to height-for-age and weight-for-height *z*-scores (the number of SDs above or below the child's age-specific mean height or height-specific mean weight, based on the international growth reference curves of the World Health Organization[41]). These conversions were made with the use of the nutritional anthropometry programs in the statistical software package EpiInfo, Version 5 (USD Inc., Stone Mountain, Ga.). The purpose of these manoeuvres was to remove the confounding effect of age from our analyses of height and weight as predictors of blood lead levels.

We condensed questionnaire and other data items into about 70 variables to test their value as predictors of blood lead levels. We considered variables to be either numeric (continuous and interval-ratio) or categoric (nominal or ordinal). For numeric variables, we conducted simple linear regression analysis of their relation with the blood lead level and tested the null hypotheses that the regression and correlation coefficients were equal to zero. For categoric variables, we tested the null hypothesis of no difference between categories in mean logarithms of blood lead levels with the use of Student's *t*-test (for variables with two categories) or a one-way analysis of variance (for variables with three or more categories). In cases in which Bartlett's test suggested that the variances in the logarithms of blood lead levels between categories were unequal, we tested the null hypothesis of no difference in distributions of logarithms of blood lead levels between categories with the use of the nonparametric Wilcoxon rank-sum test for two samples or the Kruskal–Wallis test for comparison of three or more independent samples.

We explored the relation between the blood lead level and height-for-age with the use of multiple regression analysis in order to take into account three potential confounding variables: socioeconomic status, diet or health problems and race. We tested models with height-for-age as the dependent variable and the blood lead level, the confounding variables, and variables for the interactions between the blood lead level and each of the confounding variables as independent variables. Categoric variables were represented by dummy variables. We obtained the model with the best fit by sequential backward elimination of variables. We used the same process to explore the relation between the blood lead level and the age of the water-service connection to the home, taking into account the confounding variable of socioeconomic status.

We used an analysis of covariance to explore the relation between the blood lead level and the age of the subject's home, with an adjustment for socioeconomic status. In the covariance model, the logarithm of the blood lead level was the dependent variable, the Blishen Index of socioeconomic status (a numeric value) was the covariate and the period when the dwelling was constructed (a categoric variable) was the subgroup classification variable. We then conducted a multiple partial *F*-test of the null hypothesis of no difference in the covariate-adjusted least-squares means of the logarithm of blood lead levels between categories of the classification variable. We used the same analysis of covariance to study the relation between the blood lead level and race, with adjustment for socioeconomic status.

All statistical analyses were conducted with the use of the statistical software packages SAS, Release 6.03, and EpiInfo, Version 5.

Results

Blood lead levels

We obtained blood specimens adequate for analysis from 172 of the 177 participants. In only two children were the initial measurements of blood lead levels 0.97 μmol/L or higher (4.41 μmol/L and 1.24 μmol/L). Both children seemed well and had no obvious source of exposure to excess lead. Repeat testing (13 and 17 days

later, respectively) by venipuncture showed much lower levels: 0.66 μmol/L and 0.14 μmol/L, respectively. For analysis of the survey data we discarded the initial high measurements and retained the results of the repeat test.

In the 172 participants with adequate specimens, the mean blood lead level was 0.29 μmol/L, with an SD of 0.13 μmol/L. The lowest observation was 0.06 μmol/L, and the highest was 0.85 μmol/L. Of all of the observations, 8.1% (14/172) were 0.48 μmol/L or higher, and 0.6% (1/172) was higher than 0.72 μmol/L.

The frequency distribution of the blood lead levels is shown in Table 2. From analysis of the distribution of log-transformed data, we found that the Shapiro–Wilk W statistic was 0.987331 ($p = 0.84$), which is consistent with the null hypothesis that the logarithms of the blood lead levels in the population were normally distributed (i.e., the blood lead levels were "log-normally" distributed).

The GM of the blood lead levels was 0.26 μmol/L (95% confidence interval [CI] 0.24 to 0.28 μmol/L) and the GSD 1.56.

Risk-factor analysis

We obtained sufficient blood for analysis of zinc protoporphyrin levels from 162 participants. The mean level was 46.1 μmol/μmol heme, and the SD 21.4 μmol/μmol heme (lowest observation 19 μmol/μmol heme, 25th percentile 35 μmol/μmol heme, median 42 μmol/μmol heme, 75th percentile 53 μmol/μmol heme and highest observation 238 μmol/μmol heme). We found a zinc protoporphyrin level of 70 μmol/μmol heme or higher in 7% (12/162) of the participants. A simple linear regression analysis with the zinc protoporphyrin level as the dependent variable showed no significant correlation between the zinc protoporphyrin level and the blood lead level, ($r^2 = 0.0036$, intercept = 43.4, regression coefficient = 9.7 [95% CI −16.6 to 36.0]).

Table 2: Frequency distribution of blood lead levels in participants from whom adequate specimens were obtained

Level, μmol/L	No. (and %) of participants	Cumulative % of participants
0.050–0.099	3 (1.7)	1.7
0.100–0.149	16 (9.3)	11.0
0.150–0.199	30 (17.4)	28.5
0.200–0.249	33 (19.2)	47.7
0.250–0.299	22 (12.8)	60.5
0.300–0.349	26 (15.1)	75.6
0.350–0.399	13 (7.6)	83.1
0.400–0.449	10 (5.8)	89.0
0.450–0.499	9 (5.2)	94.2
0.500–0.549	6 (3.4)	97.7
0.550–0.599	0 (0.0)	97.7
0.600–0.649	1 (0.6)	98.3
0.650–0.699	1 (0.6)	98.8
0.700–0.749	1 (0.6)	99.4
0.750–0.799	0 (0.0)	99.4
0.800–0.849	1 (0.6)	100.0
Total	172 (100.0)	

Of the other 70 potential predictors of blood lead levels analysed, only a few had a statistically significant ($p < 0.05$) association with increased geometric mean blood lead levels. Children in homes where soldering was performed as part of an electronics hobby had a GM blood lead level of 0.34 μmol/L (95% CI 0.27 to 0.39 μmol/L). Children with aboriginal heritage had a GM blood lead level of 0.33 μmol/L (95% CI 0.28 to 0.39 μmol/L); after adjustment for socioeconomic status (measured by the Blishen Index), the GM blood lead level was 0.36 μmol/L. Children living in dwellings built before 1921 had a GM blood lead level of 0.32 μmol/L (95% CI 0.28 to 0.37 μmol/L); after adjustment for socioeconomic status, the GM blood lead level was 0.31 μmol/L. Also, blood lead levels were a predicted 0.00087 μmol/L (95% CI 0.00005 to 0.00169 μmol/L) higher for every year since the water service to the home was first connected. There was an association between increased blood lead levels and decreased height-for-age measurements: blood lead levels were a predicted 0.018 μmol/L (95% CI 0.0353 to 0.0015 μmol/L) higher for every SD below age-specific mean height. However, this association disappeared in the multivariable regression analysis, which controlled for socioeconomic status, race and a dietary or health problem.

Because the validity of the Blishen Index (or any index of socioeconomic status) is debatable, we studied four other indicators of socioeconomic status as well: the parents' employment income, education level and employment status as well as the median income in the census tract. Of the five indicators, the Blishen Index score showed the strongest association with the blood lead level. A simple regression analysis with the logarithm of the blood lead level as the dependent variable and the Blishen Index score as the independent variable yielded a correlation coefficient of −0.14 ($0.05 < p < 0.10$ in a two-tailed test). We therefore selected the Blishen Index score to represent socioeconomic status in the multivariable analyses.

Discussion

The participation rate in this survey was 73%. Participants were similar to the total sample in terms of lan-

guage group, socioeconomic level and mother's reproductive history. It appears that the participants represented fairly the diverse cultures, socioeconomic backgrounds and lifestyles of the target population.

Most previous surveys of blood lead levels in Canada have studied groups in areas of exceptional environmental contamination; for example, workers in lead-related industries, residents of areas near smelters or other lead-pollution sources and communities built on former industrial landfill sites. Only two previous surveys in Canada studied a sample of children selected from the general population; these were the Physical Measures component of the 1978–79 Canada Health Survey[42] and a survey conducted in Ontario in 1984.[13,14] In Table 3 the results of our study are compared with those of the Canadian and other surveys.

The blood lead levels we found were much lower than those found in the Canada Health Survey, in the Ontario survey and in the United States National Health and Nutrition Examination Survey II (1976–80).[3,44] In fact, the levels were as low as those found in some studies of blood lead levels in nonindustrialized societies.[43] We do not believe that the low levels found in our study were due to differences in methods between our study and the other Canadian studies. Of the three Canadian studies, ours had the youngest subjects (aged 24 to 36 months) and the highest proportion of city dwellers (100%). These factors would lead one to expect the results to show higher blood lead levels than those found in the 1984 Ontario survey or the Canada Health Survey. Similarly, time of year cannot explain the low levels we found. We collected blood specimens between Oct. 19 and Dec. 13, 1989, a period of mid-level exposure. The 1984 Ontario survey was conducted only slightly earlier in the year, from Sept. 12 to Nov. 21. The Canada Health Survey took place over 8 months, from July to March, that cover periods of high, mid-level and low exposure. Most important, we used a fingerprick blood-collection method, which could have biased our measurements upwards as a result of contamination of the fingerprick blood specimens by lead on the skin.[10,11,32,33]

The chief limitation of blood lead measurement as an indicator of exposure to environmental lead is that it provides no information about exposure sources or absorption routes. Because we did not conduct any concurrent testing of the environment (e.g., soil, water, house dust, food or house paint), we can only speculate about the reasons for the low levels of blood lead that we found. There was a marked decline in the use of leaded gasoline from 1979 to 1989.[2] Other possible explanations include Vancouver's relative lack of heavy industry and its mild climate, heavy rainfall and lush vegetation, which may reduce dust dispersion and limit children's contact with soil. Although we could not determine the relative contributions of soil, drinking water and other environmental sources to children's total lead intake, the total intake from all sources was much lower than expected.

Table 3: Comparison of results of surveys of blood lead levels in children

Study location (and date, if known)	Age of participants	Type of blood specimen	Mean blood lead level in sample, μmol/L	% of sample with blood lead level above that given		
				0.72 μmol/L*	0.97 μmol/L†	1.21 μmol/L‡
Canada (1978–79)[42]	3–5 yr	Venous	NA§	NA	2.9‖	NA
Nepal[43]	Children	NA	0.25	NA	NA	NA
Ontario (1984)[14]						
Urban residents	3–6 yr	Capillary	0.58 (GM¶)	25.0	5.6	1.8
Suburban residents	3–6 yr	Capillary	0.48 (GM)	9.9	3.7	1.6
Rural residents	3–6 yr	Capillary	0.43 (GM)	10.3	3.5	0.8
Papua New Guinea[43]	Children	NA	0.25	NA	NA	NA
United States (1976–80)[3,44]						
White children	6–35 mo	Venous	0.72	NA	19.7	NA
Black children	6–35 mo	Venous	1.01	NA	49.5	NA
Vancouver (1989)	24–36 mo	Capillary	0.26	0.6	None	None

*"Lowest observed adverse effect" level set by the US Environmental Protection Agency.[1]
†Adverse-effect threshold established by the Royal Society of Canada's Commission on Lead in the Environment.[2]
‡Medical intervention threshold set by the US Centers for Disease Control and Prevention.[10]
§NA = not available. The mean blood lead level in participants in the Canada Health Survey was impossible to calculate because the lower limit of detection for the method used was 0.24 μmol/L.[45]
‖The true percentage may be higher; some lead may have been lost to the glass walls of the storage containers.[45]
¶GM = geometric mean.

The lack of concurrent environmental data also makes it difficult to generalize our findings to other populations. On the basis of other studies of the relation between age and blood lead level,[7,24,46] we believe that mean blood lead levels in children younger than 18 months or older than 36 months were likely even lower than in those in the age group we surveyed, at least in Vancouver.

We found some groups with a relatively higher mean blood lead level than that of the total sample; although these differences were statistically significant, the sizes of the differences were so small as to be of no practical importance. The Royal Society of Canada's Commission on Lead in the Environment suggested that a difference in mean blood lead levels of 0.14 μmol/L or more between the subgroup and the population should be the criterion for environmental intervention.[2] In addition, we tested hypotheses for 70 different variables thought to be risk factors; with a nominal 5% probability of a Type I error for each hypothesis test, "statistically significant" associations could occur by chance alone. Of the risk-measurement instruments we tested (zinc protoporphyrin levels, questionnaire, housing inspection, height and weight measurements, age of water-service connections and traffic counts), none were found to be useful in identifying groups at a high risk of elevated blood lead levels. However, such instruments could have greater discriminatory ability in populations in which the mean blood lead level is higher than in the population we studied.

On the basis of criteria for lead-hazard investigation and intervention prevailing in 1989,[2,10] we concluded that Vancouver did not have a lead-contamination problem that warranted a screening program or environmental investigation. Subsequent downward revisions of recognized thresholds for effects of blood lead and intervention could prompt reassessment of this position. For example, in 1991 the CDC[11] recommended that children with blood lead levels as low as 0.48 μmol/L be retested and that environmental and medical investigation be conducted for children with blood lead levels persistently higher than 0.72 μmol/L. The most appropriate form of reassessment may be a repeat survey of the population because, since our study was conducted, there has been an important community-wide intervention to reduce lead exposure: the implementation, in September 1990, of federal environmental regulations that almost eliminated the use of leaded gasoline in Canada.

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References

1. *Air Quality Criteria for Lead* [report nos EPA-600/8-83/028 AF (vol 1), EPA-600/8-83/028 BF (vol 2), EPA-600/8-83/028 CF (vol 3), EPA-600/8-83/028 DF (vol 4), NTIS PB87-142386 (vol 1), NTIS PB87-142394 (vol 2), NTIS PB87-142402 (vol 3), NTIS PB87-142410 (vol 4)], US Environmental Protection Agency, Research Triangle Park, NC, 1986
2. *Lead in the Canadian Environment: Science and Regulation* [final report], Commission on Lead in the Environment, Royal Society of Canada, Ottawa, 1986
3. Agency for Toxic Substances and Disease Registry: *The Nature and Extent of Lead Poisoning in Children in the United States: a Report to Congress*, Public Health Service, US Department of Health and Human Services, Atlanta, 1988
4. Dietrich KN, Krafft KM, Bornschein RL et al: Low level fetal lead exposure effect on neurobehavioural development in early infancy. *Pediatrics* 1987; 89: 721–730
5. McMichael AJ, Vimpani GV, Robertson EF et al: The Port Pirie Cohort Study: maternal blood lead and pregnancy outcome. *J Epidemiol Community Health* 1986; 40: 18–25
6. McMichael AJ, Baghurst PA, Wigg NR et al: Port Pirie Cohort Study: environmental exposure to lead and children's abilities at the age of four years. *N Engl J Med* 1988; 319: 468–475
7. Baghurst PA, Robertson EF, McMichael AJ et al: The Port Pirie Cohort Study: lead effects on pregnancy outcome and early childhood development. *Neurotoxicology* 1987; 8: 395–402
8. Bellinger D, Leviton A, Waternaux C et al: Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med* 1987; 316: 1037–1043
9. *Preventing Lead Poisoning in Young Children — A Statement by the Center for Disease Control* [report no US-DHEW 00–2629], Environmental Health Services Division, Bureau of State Services, Public Health Service, US Department of Health, Education and Welfare, Atlanta, 1978
10. *Preventing Lead Poisoning in Young Children — A Statement by the Centers for Disease Control* [report no US-DHHS 99-2230], Chronic Diseases Division, Center for Environmental Health, Public Health Service, US Department of Health and Human Services, Atlanta, 1985
11. *Preventing Lead Poisoning in Young Children — A Statement by the Centers for Disease Control — October 1991*, Lead Poisoning Prevention Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health and Injury Control, Centers for Disease Control, Public Health Service, US Department of Health and Human Services, Atlanta, 1991
12. Needleman HL, Bellinger D: The health effects of low level exposure to lead. *Annu Rev Public Health* 1991; 12: 111–140
13. Duncan C, Kusiak RA, O'Heany J et al: *Blood Lead and Associated Risk Factors in Ontario Children, 1984*, Ontario Ministry of Health, Ontario Ministry of Labour, Ontario Ministry of

the Environment, Toronto, 1985

14. O'Heany J, Kusiak R, Duncan CE et al: Blood lead and associated risk factors in Ontario children. *Sci Total Environ* 1988; 71: 477–483

15. Jin A, Hertzman C, Peck S et al: *Blood Lead Levels in Vancouver Children, October-December 1989* [final report], City of Vancouver Health Department, Vancouver, 1992

16. Schmitt N, Philion JJ, Larsen AA et al: Surface soil as a potential source of lead exposure for young children. *Can Med Assoc J* 1979; 121: 1474–1478

17. Appendix 1: Soil lead levels in Vancouver — May 1987. In *Protocol for a Proposed Study: Assessment of Potential Health Hazard to Vancouver Children from Environmental Lead Contamination* [file no 4662-2], Health Department, City of Vancouver, Vancouver, 1989

18. Appendix 2: Soil lead levels in Vancouver — June 1988. In *Protocol for a Proposed Study: Assessment of Potential Health Hazard to Vancouver Children from Environmental Lead Contamination* [file no 4662-2], Health Department, City of Vancouver, Vancouver, 1989

19. Jin A, Peck S: *Lead Levels in School Drinking Fountain Water, June 1988: Discussion and Proposals for Intervention*, Health Department, City of Vancouver, Vancouver, 1988

20. *Guidelines for Canadian Drinking Water Quality*, Department of National Health and Welfare, Ottawa, 1987

21. *Supporting Documentation for the Guidelines for Canadian Drinking Water Quality*, Department of Natinal Health and Welfare, Ottawa, 1978

22. Bonnefoy X, Huel G, Gueguen R: Variation of blood lead level as a result of lead contamination of the subject's drinking water. *Water Res* 1985; 19: 1299–1303

23. Pocock SJ, Shaper AG, Walker M et al: Effects of tap water lead, water hardness, alcohol and cigarettes on blood lead concentrations. *J Epidemiol Community Health* 1983; 37: 1–7

24. Clark CS, Bornschein RL, Succop P et al: Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res* 1985; 38: 46–53

25. Ellis E, Erb J, McFarlane G: *South Riverdale Blood Lead Testing, 1984*, Department of Public Health, City of Toronto, Toronto, 1985

26. *Census Canada 1986, Profiles Series, Census Tracts, Vancouver: Part I* [cat no 95–167], Statistics Canada, Ottawa, 1988

27. *Census Canada 1986, Profiles Series, Census Tracts, Vancouver: Part II* [cat no 95–168], Statistics Canada, Ottawa, 1988

28. *1989 Vancouver BC City Directory*, RL Polk and Company, Vancouver, 1989

29. Jacobson B, Lockitch G, Quigley G: Improved sample preparation for accurate determination of low level lead in whole blood by graphite furnace analysis. *Clin Chem* 1991; 37: 515–519

30. Weber J-P: An interlaboratory comparison program for several toxic substances in blood and urine. *Sci Total Environ* 1988; 71: 111–123

31. Stanton NV, Gunter EW, Parson PJ et al: Empirically determined lead poisoning screening cutoff for the Protofluor-Z hematofluorometer. *Clin Chem* 1985; 35: 2104–2107

32. Cooke RE, Glynn KL, Ullmann WW et al: Comparative study of a micro-scale test for lead in blood, for use in mass screening programs. *Clin Chem* 1974; 20: 582–585

33. Sinclair DF, Dohnt BR: Sampling and analysis techniques used in a blood lead survey of 1241 children in Port Pirie, South Australia. *Clin Chem* 1984; 30: 1616–1619

34. *Census Canada 1986, Reference, Census Handbook* [cat no 99–104E], Statistics Canada, Ottawa, 1988

35. Rootman I, Warren R, Stephens T et al (eds): *Canada's Health Promotion Survey: Technical Report* [cat no H39–119/1988E], Department of National Health and Welfare, Ottawa, 1988

36. Schilling RJ, Bain RP: Prediction of children's blood lead levels on the basis of household specific soil lead levels. *Am J Epidemiol* 1988; 128: 197–205

37. Macpherson AS: *South Riverdale Blood Lead Testing, 1986*, Department of Public Health, City of Toronto, Toronto, 1987

38. *Guide: Canadian Classification and Dictionary of Occupations*, 9th ed, [cat no MP53–8/1989E], Occupational and Career Information Branch, Employment and Immigration Canada, Ottawa, 1989

39. Blishen BR, McRoberts HA: A revised socioeconomic index for occupations in Canada. *Can Rev Sociol Anthropol* 1976; 13: 72–79

40. *Employment Income by Occupation* [cat no 93–116], *The Nation* series, Statistics Canada, Ottawa, 1989

41. Dibley MJ, Goldsby JB, Staehling NW et al: Development of normalized curves for the international growth reference: historical and technical considerations. *Am J Clin Nutr* 1987; 46: 736–748

42. *The Health of Canadians: Report of the Canada Health Survey* [cat no 82–538E], Department of National Health and Welfare and Statistics Canada, Ottawa, 1981

43. Neri LC, Tessier J: Blood lead levels in children in Canada and other countries. *Chronic Dis Can* 1986; 7: 18–25

44. Mahaffey KR, Annest JL, Roberts J et al: National estimates of blood lead levels: United States, 1976–1980. *N Engl J Med*, 1982; 307: 573–579

45. Hotz MCB (ed): *Health Effects of Lead*, Commission on Lead in the Environment, Royal Society of Canada, Ottawa, 1986

46. Levallois P, Lavoie M, Goulet L et al: Blood lead levels in children and pregnant women living near a lead-reclamation plant. *Can Med Assoc J* 1991; 144: 877–885

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