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Assessing the Influence of Age and Ethnicity on the Association Between Iron Status and Lead Concentration in Blood: Results from the Canadian Health Measures Survey (2007–2011)

Gerard Ngueta [1,2]

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Abstract Inverse association has been reported between iron intake and blood lead concentration (PbB) in epidemiological studies. Data on this association at a low dose of lead exposure are scarce, and the potential influence of ethnicity and age has not been previously reported. This study aimed to estimate the relation between serum ferritin, haemoglobin, haematocrit, and mean corpuscular volume and PbB among 6–18-year-old individuals. Data from Canadian Health and Measures Survey (CHMS), cycle 1 (2007–2009) and cycle 2 (2009–2011), were accessed. A household interview followed by a physical examination (including collection of blood) was performed. The quantification of lead and trace elements in blood was performed by inductively coupled plasma mass spectrometry. The mean PbB was 0.79 μg/dL (95 % confidence interval (95 % CI) 0.75–0.82). Except for haemoglobin levels, no association was found between PbB and any of the parameters of iron status, independently of age. A significant interaction was observed between ferritin levels and ethnicity in relation to PbB ($p = 0.07$). We found a little evidence of an association between iron status and PbB in the whole sample of subjects aged 6–18 years exposed to low levels of environmental lead. The significant interaction observed between ferritin levels and ethnicity in relation to BPb suggests that the influence of ferritin levels on lead uptake may change by ethnicity, even at low exposure.

Keyword Iron status · Blood lead levels · Ethnicity · Age

Introduction

Over the last century, the harmful effects of lead have been largely published in the medical literature. Reducing the human exposure to lead through primary prevention has been a main focus of public health authorities, especially in western countries. In 2013, exposure to lead continues to be a public health concern, particularly as a safe level for its toxicity remains unknown. Although the major public health actions undertaken since the early 1970s yield to marked decline in children's blood lead concentration (PbB) in North America [1, 2], recent studies suggest that even at a low dose (<5 μg/dL), lead remains a major concern for human health. Chronic exposure to a low dose at an early age is associated with a significant decline in reading, writing and spelling scores at 7–8 years of age [3, 4]. Evidences from pooled data suggest that PbB increase from <1 to 10 μg/dL is associated with an IQ decrement of about 6.2 (95 % confidence interval (95 % CI) 3.8–8.6) [5].

The gastrointestinal uptake of lead represents a key step in the process of lead kinetic and toxicity. The role of iron in altering lead uptake and susceptibility to lead toxicity has long been suspected and reported. Indeed, the inverse association has been previously observed between iron intake and PbB in both cross-sectional [6] and follow-up studies [7–10]. Authors suggested that increased dietary iron intake may decrease lead absorption and that competitive inhibition of gastrointestinal uptake between iron and lead as previously observed in animal studies [11, 12] may hold in humans.

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✉ Gerard Ngueta
Ngueta@hotmail.com

[1] Population Health and Optimal Health Practices Research Unit, CHUQ Research Center, Faculty of Medicine, Laval University, 2875 Bld Laurier, Delta II (Floor 6), Quebec, QC G1V 2M2, Canada

[2] Department of Occupational and Environmental Health, School of Public Health, Université de Montréal, Montreal H3T 1A8, QC, Canada

A recent hypothesis suggests that the gastrointestinal uptake of both iron and lead may differ by ethnicity [13]. The hypothesis stipulates that, among iron-deficient children, the gastrointestinal uptake of lead may be more pronounced in non-Hispanic Blacks. Assessing the influence of ethnicity on the association between iron levels and PbB may be a first step to validate such a hypothesis. Most of studies assessing the relation between iron status and PbB were conducted when the mean PbB at a population level was higher than values currently observed. Furthermore, they mainly included children aged below 6 years, with a large proportion from lower socioeconomic class. Considering the scarcity of recent investigations at a low dose of lead exposure, and given that the potential influence of ethnicity on the association between iron status and PbB has not been previously reported, it would be interesting to address these questions. The present study aimed to estimate the relation between PbB and parameters of iron status among 6–18-year-old individuals living in Canada, with a specific emphasis on serum ferritin. Ferritin is known as an iron-storage protein that maintains sufficient blood iron in case of inadequate dietary intake, with a high sensitivity to changes in iron intake. Data from Canadian Health and Measures Survey (CHMS), cycle 1 (2007–2009) and cycle 2 (2009–2011), were accessed to address this issue.

Materials and Methods

General Information

The CHMS is a population-representative direct health measures survey that is being conducted by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada. This large cross-sectional survey aims to collect new and important data on Canadian's health status, from a sample of approximately 5000 Canadians aged 6 to 79 years. For cycle 1 (2007–2009) and cycle 2 (2009–2011), the sampling protocol covered approximately 96 % of the Canadian population and resulted in data collection sites in five provinces. Further details of the sampling strategy are provided elsewhere [14]. Within each site, dwellings were stratified and randomly selected using the sampling frame. Initial contact with selected dwellings was made through a mail-out containing information about the CHMS. Subsequently, a roster of all residents from each participating dwelling was obtained, and one or two eligible respondents were selected per dwelling. The probability of a respondent being selected varied by stratum, depending on the age group being targeted, and was designed to achieve the desired age and sex stratification. The CHMS was voluntary and included respondents who agreed to participate. Ethics approval to conduct the survey was obtained from Health Canada's Research Ethics Board [15].

Data Collection

Data collection was performed in two stages: a household interview followed by a physical examination (including collection of biological samples).

Household Interview

Interviewers contacted the sampled household to create a roster of household members, and selected a respondent, based on a computerized sampling vector that was designed to provide the desired age distribution [14]. During the household interview, a health questionnaire was administered in the respondent's home by Statistics Canada interviewers through a computer-assisted personal interview. The household questionnaire was designed to provide background and contextual information as well as modules on general health, height and weight, chronic conditions, activity restrictions, health and lifestyle behaviours, nutrition, activity levels, medication use and ethnicity. The information about race/ethnicity was self-reported.

Clinic Visit

One to 6 weeks after the household interview, direct physical measurements and biospecimen collection were performed in a mobile clinic. At each site, the clinic staff consisted of a manager, two senior health measures specialists, four laboratory technicians/technologists, four clinic coordinators, two licenced dentists, two dental recorders, and a site logistics officer. All health measurement specialists were certified exercise physiologists. The staff travelled from site to site and lived at each location for 6 to 7 weeks. The use of mobile clinics required a clustered sample design.

Blood and urine specimens were collected in the mobile clinics as well as the subsequent processing, including aliquoting and centrifuging of serum and plasma. Two −20 °C freezers were used to temporarily store the biospecimens until shipping. Once a week, the specimens were shipped to the reference laboratory for analysis. Standardized operating procedures were developed for the collection of blood and urine specimens. BecDickinson Vacutainers and VWR urine specimen were used for collection of blood and urine, respectively.

Laboratory Analyses

Complete Blood Count Analysis

Whole blood was analysed for the complete blood count at the mobile clinic laboratory using the Beckman Coulter HmX Hematology Analyzer. The complete blood count analysis included determination of haemoglobin and of mean corpuscular volume.

Lead in Blood

The quantification of lead and trace elements in blood was performed by the "Centre de Toxicologie du Québec (Institut National de Santé Publique du Québec (INSPQ))".

Blood samples were diluted in a basic solution containing octylphenol ethoxylate and ammonia. They were analysed for total lead by inductively coupled plasma mass spectrometry (ICP-MS), Perkin Elmer Sciex, Elan DRC II (M-572). Matrix-matched calibration was performed using blood from a non-exposed individual [16].

INSPQ followed standardized operating procedures that were developed for every assay and technique performed in their laboratory. The laboratory, which is accredited under ISO 17025, uses numerous internal and external quality control programmes. The limit of detection (LOD) for lead in whole blood was 0.02 μg/dL.

Blood Lipids, Ferritin and Calcium

The Health Canada Nutrition laboratory (Bureau of Nutritional Sciences, Nutrition Research Division, Ottawa, Ontario) was the reference laboratory for serum lipids, calcium and iron analyses. Serum aliquots were frozen at −20 °C and shipped once a week on dry ice to the laboratory. Serum was then analysed for ferritin by solid-phase, two-site chemiluminescent immunometric assay using the Immulite 2000 (Siemens HealthCare Diagnostics). The measurement of serum ferritin was not included in the cycle 1 of CHMS, but just in the cycle 2. Triglycerides and total cholesterol were measured by enzymatic methods using the VITROS TRIG Slide method based on the procedure described by Spayd and co-workers [17], and total cholesterol was measured using the VITROS CHOL Slide method based on the procedure proposed by Allain and colleagues [18]. The colorimetric reflectance spectrophotometry (arsenazo III) was used to measure serum levels of calcium. The reproducibility of this method is very high (coefficient of variation ≈1.5 %). The application range as reported by the manufacturer was from 0.25 to 3.49 mM.

Variable Under Study

The outcome variable for the present study was the PbB (μg/dL). The exposure variables were those related to iron status, namely serum ferritin levels (ng/mL), haemoglobin (g/L), haematocrit (%) and mean corpuscular volume (fL). We used the ferritin cutoff value of ≤15 ng/mL for defining iron deficiency [9]. The cutoffs for defining participants with low haemoglobin, low haematocrit and low mean corpuscular volume were <112 g/L, <34 % and <73 fL, respectively [19–21]. The potential covariates were age, sex, ethnicity (Black, Whites, and others), smoking status (never, former and current smokers), alcohol consumption (never, former, occasionally and regularly consumers), annual income levels (<10,000$, 10,000–14,999$, 15,000–29,999$, 30,000–59,999$, ≥60,000$) and blood calcium concentration. In addition, the body mass index was categorized according to the age- and sex-specific values previously defined by Cole and co-investigators [22]. The CHMS cycles 1 and 2 do not include measurement of lead concentration in environmental media (water, dust, soil, etc). In the absence of this data, the house age was forced into the regression model.

Statistical Analyses

SAS software (Version 9.4; SAS Institute Inc., Cary, NC) was used to perform all analyses. The association between iron parameters and blood lead concentrations was assessed using the analysis of covariance (ANCOVA) through the GLM procedure, with Bonferroni correction for multiple comparisons. Because the outcome variable was skewed, a natural-log transformation was used to normalize the distribution before analyses. The adjusted means of Log(PbB) as well as their 95 % confidence intervals (95 % CI) were then exponentiated to obtain the geometric means and 95 % CI. We evaluated whether the association between iron parameters and PbB varied by age categories (6–10, >10–18 years) or ethnicity (Whites, Blacks, others) by including their multiplicative terms in ANCOVA models. The stratification applied for age was done for making our study comparable with previous ones [7, 23]. The likelihood ratio test with 0.10 cutoff was then applied to define the statistically significant interaction. Sex-stratified analyses were also undertaken to gauge whether or not the strength and direction of association between ferritin levels and outcomes in young (6–10 years) and older (<10–18 years) were similar between boys and girls. Data on environmental lead concentrations were not available from the CHMS. As a consequence, for all analyses, the age of residence was forced into the regression models given that this variable is generally associated with dust and water lead exposure.

Results

Characteristics of the Study Population

The study population for the present study consisted of 3799 participants 6–18 years of age. Table 1 lists characteristics of the population under study stratified by sex.

About 80 % of the Canadian population sampled in CHMS self-identified with race/ethnicity that could be deemed "White" and ~20 % does not, and is a varied mixture of which a very small proportion (4 %) could be called "Black". The mean PbB was very low in this population with a geometric mean of 0.79 μg/dL (95 % CI 0.75–0.82). The median PbB was 0.75 μg/dL (interquartile range (Q1–Q3) 0.57–1.05 μg/

Table 1 Characteristics of participants from the Canadian Health Measures Survey according to sex

	CHMS cycles 1 (2007–2009) and 2 (2009–2011)	
	Girls	Boys
Total	1858	1941
Age (years)	11.8 (11.7–12.0)	11.8 (11.6–12.0)
Age groups		
6–10 years	692 (37.2)	738 (38.0)
>10–18 years	1166 (62.8)	1203 (62.0)
Ethnicity[b]		
White	1387 (74.6)	1503 (77.4)
Black	79 (4.2)	87 (4.5)
Others	394 (21.2)	351 (18.1)
Body mass index		
Normal	1402 (75.5)	1425 (73.4)
Overweight	318 (17.1)	320 (16.5)
Obesity	138 (7.4)	196 (10.1)
Income class		
Very low	36 (1.9)	16 (0.8)
Low	88 (4.7)	77 (4.0)
Medium	286 (15.4)	367 (18.9)
High	525 (28.3)	521 (26.8)
Very high	923 (49.7)	960 (49.5)
Blood calcium levels (mmol/L)	2.5 (2.4–2.5)	2.5 (2.4–2.5)
Ferritin levels (ng/mL)[a]	64.8 (43.6–96.3)	77.1 (59.1–100.5)

Values are *n*(%) or geometric mean (95 % confidence interval)

[a] Available for CHMS cycle 2 only (*n* = 1921)

[b] About 20 % of the Canadian population sampled in CHMS self-identified with race/ethnicity that could be deemed "non-Whites" and is a varied mixture of which a very small proportion (4 %) could be called "Black"

dL). The measurement of serum ferritin levels was only performed in the cycle 2 of the CHMS, including 1921 participants.

Association Between Iron Deficiency and PbB

After adjustment for age (continuous), gender, ethnicity, income level, body mass index, age of residence and blood calcium concentration, the iron status was not associated with PbB, independent of the parameter used to define iron status. However, a significant difference in mean PbB was observed between participants with haemoglobin <112 g/L (GM 0.69 μg/dL (95 % CI 0.56–0.83)) and those with haemoglobin ≥112 g/L (GM 0.89 μg/dL (95 % CI 0.85–0.93)) (see Table 2). Stratified analyses showed that low haemoglobin levels were associated with low PbB in young participants (6–10 years) and in Whites (see Table 3). As a whole, iron status was not associated with BPb in this population. However, a significant

Table 2 Adjusted[a] geometric means (95 % CI) of blood and urinary lead concentrations according to iron status

	Blood lead (μg/dL)
Haemoglobin (g/L)[b]	
≥112	0.89 (0.85–0.93)
<112	0.69 (0.56–0.83)
p value	0.0085
Haematocrit (%)[b]	
≥34	0.89 (0.85–0.93)
<34	0.77 (0.65–0.92)
p value	0.1203
Mean corpuscular volume (fL)[b]	
≥73	0.88 (0.84–0.92)
<73	0.97 (0.83–1.14)
p value	0.2378
Ferritin (ng/mL)[c]	
>15	0.86 (0.80–0.92)
≤15	0.83 (0.72–0.95)
p value	0.5810

[a] Means were obtained by ANCOVA. Models were adjusted for age (continuous), gender, ethnicity (non-Hispanic Whites, non-Hispanic Blacks, others), body mass index, age of residence, and blood calcium concentration and income level

[b] Based on CHMS cycles 1 and 2 combined (*n* = 3799)

[c] Based on CHMS cycle 2 (*n* = 1921)

interaction was observed between ferritin levels and ethnicity in relation to PbB ($p = 0.07$). The difference in mean PbB between iron-deficient and iron-sufficient participants was more important in Blacks. Further analyses showed that there was a significant interaction between serum ferritin and age in relation to PbB in boys, but not in girls (See Appendix, Table A.1).

Discussion

This is the first nationally representative cross-sectional study to assess the relation between iron parameters and both PbB in children aged above 6 years and adolescents at low lead exposure levels. The haemoglobin, haematocrit, mean corpuscular volume and serum ferritin levels in relation to PbB were examined. The potential role of age and ethnicity in modifying the association on a multiplicative scale was also assessed.

The mean PbB observed was slightly low as compared with the mean value reported in the US individuals aged 6–19 years (1.7 μg/dL (95 % CI 1.5–1.8)) [24]. Surprisingly, for iron parameters under study, the mean PbB was slightly elevated in participants with iron-sufficient status when compared to those with poor iron status. This trend persisted

Table 3 Adjusted[a] geometric means (95 % CI) of blood lead concentrations (μg/dL) according to iron status by age and ethnicity groups

	Age groups (years)		P^b	Ethnicity groups			P^b
	6–10	>10–18		Non-Hispanic Whites	Non-Hispanic Blacks	Others	
Haemoglobin (g/L)[c]							
≥112	0.96 (0.90–1.04)	0.84 (0.79–0.89)	0.7605	0.78 (0.75–0.82)	1.03 (0.84–1.27)	0.92 (0.86–0.99)	0.3308
<112	0.72 (0.58–0.89)	0.73 (0.46–1.18)		0.54 (0.42–0.69)	0.87 (0.51–1.51)	0.87 (0.53–1.41)	
p value	0.0053	0.5879		0.0031	0.5460	0.7987	
Haematocrit (%)[c]							
≥34	0.96 (0.89–1.02)	0.84 (0.79–0.89)	0.5314	0.78 (0.75–0.82)	1.01 (0.83–1.24)	0.92 (0.86–0.99)	0.2264
<34	0.84 (0.70–1.02)	0.65 (0.36–1.18)		0.63 (0.52–0.76)	1.48 (0.56–3.92)	1.04 (0.65–1.66)	
p value	0.1591	0.4035		0.0228	0.4398	0.6183	
MCV (fL)[c]							
≥73	0.95 (0.89–1.02)	0.83 (0.79–0.89)	0.6515	0.78 (0.75–0.82)	1.05 (0.85–1.28)	0.92 (0.86–0.98)	0.2154
<73	0.94 (0.77–1.15)	1.06 (0.81–1.39)		0.87 (0.66–1.15)	0.78 (0.50–1.21)	1.17 (0.91–1.51)	
p value	0.8918	0.0706		0.4352	0.1788	0.0523	
Ferritin (ng/mL)[d]							
>15	0.88 (0.79–0.97)	0.81 (0.73–0.90)	0.4512	0.79 (0.73–0.86)	0.96 (0.68–1.36)	0.86 (0.77–0.96)	0.0725
≤15	0.77 (0.56–1.08)	0.84 (0.72–1.00)		0.73 (0.63–0.85)	0.48 (0.16–1.46)	1.00 (0.74–1.36)	
p value	0.4428	0.5132		0.2753	0.1947	0.2892	

[a] Means were obtained by ANCOVA. Models were adjusted for age (continuous), gender, ethnicity (for age-stratified analyses), body mass index, age of residence, and blood calcium concentration and income level

[b] *P* for interaction

[c] Based on CHMS cycles 1 and 2 combined ($n = 3799$)

[d] Based on CHMS cycle 2 ($n = 1921$)

across strata of age and race, suggesting that lead and iron would result from the same source in this population. The contaminated foods may be suspected to contribute largely to lead concentrations in blood as observed in this population.

Except for haemoglobin levels, no evidence was found for an association between PbB and any of iron parameters, independently of age. The absence of association between ferritin levels and PbB is not in line with previous studies including children aged below 6 years [6, 8, 10]. It is possible that the association as reported in the previous studies holds for children aged below 6 years, but not for older. Several prior works reported no association between iron intake and PbB in individuals aged between 10 and 18 years [7, 25] and those aged 6–7 years [26, 27]. Most of prior investigations were conducted when the mean PbB was more elevated than values currently observed. It is not excluded that iron status seriously impact gastrointestinal lead absorption at moderate or high levels of exposure, but not necessary at low levels. However, it should be noted that a recent study conducted in South Korea showed that the mean BPb in low serum ferritin group was significantly higher than that in the normal ferritin group in boys aged 10–19 years, but not in girls [23]. Such an association did not emerge from the CHMS data, even after stratification by sex (see Appendix, Table A.1). This discrepancy may be due to the fact that Sim and co-investigators considered three categories for serum ferritin levels, with the low ferritin category defined as being <15 μg/L and the reference group (normal ferritin) with ferritin levels at least equal to 30 μg/L. In the present study, normal ferritin was rather defined as being >15 μg/L.

A significant interaction was observed between ferritin levels and ethnicity in relation to PbB ($p = 0.07$). It appears that no previous study reported such an interaction. This interaction suggests that the direction and/or the strength of association between ferritin levels and PbB are not consistent across ethnicity strata. More recently, we relied on the prior works to postulate that divalent metal transporter 1 (DMT1) may contribute to the racial disparity in PbB [13]. It was suspected that non-Hispanic Blacks would be more sensitive to changes in iron status and that the more active DMT1 isoform (+1A/+IRE) would be more common in non-Hispanic Blacks compared with non-Hispanic Whites. Therefore, such an interaction between ferritin levels and ethnicity in relation to PbB was expected. Although the greatest difference in mean PbB between iron-deficient and iron-sufficient participants was observed in non-Hispanic Blacks, those with iron-deficiency did not show an elevated PbB when compared with iron-deficient non-Hispanic Whites. One explanation is the missing of adjustment for environmental lead in this study. Although the age of residence was forced in the regression models, it just remains a proxy of environmental lead. It would be surely fruitful to include measures of environmental

lead into the regression model. Unfortunately, available data from CHMS do not include measures of lead concentrations in the environmental media. On the other hand, the possibility that the main route of lead uptake in the gastrointestinal tract may be via passive transport cannot be ruled out. That is, DMT1 would not be involved in the lead uptake at very low levels of exposure. Future research activities should explore this possibility.

It appears that this is the first study assessing the potential influence of age and ethnicity on the association between iron parameters and PbB at low levels of exposure. The large number of biological measurements as well as the large sample size might be viewed as a study strength. However, there are several limitations to this study. First, the CHMS did not include the measure of environmental lead in cycles 1 and 2. The lack of adjustment could yield to underestimation of the association between iron status and PbB. However, by adjusting for the age of residence, which is reported to be strongly associated with dust and water lead exposure, the influence of the bias, if it exists, is limited. Second, given the low proportion of Blacks in the CHMS data, a separated analysis by ethnicity groups was not possible. As a result, the test of interaction was limited to the introduction of interaction terms into the regression models. Despite this limitation, results—though exploratory—are informative and future investigation involving a net stratified analysis by race/ethnic groups is needed.

Conclusions

An increasing amount of attention is being paid to the health effects from exposure to lead at very low levels. Altogether, our findings suggest that iron status is not associated with BPb in subjects aged 6–18 years exposed to low levels of environmental lead. Further studies including a large number of participants in each of the ethnic groups are needed.

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