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Iron deficiency anaemia and blood lead concentrations in Brazilian children

Patricia Helen Carvalho Rondó[a,*], Andréia Conde[a], Miriam Coelho Souza[b], Alice Sakuma[c]

[a] Nutrition Department, School of Public Health, University of São Paulo, Avenida Dr. Arnaldo 715, SP, Brazil
[b] Faculty of Nutrition, Methodist University, Piracicaba, SP, Brazil
[c] Adolfo Lutz Institute, São Paulo, SP, Brazil

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ABSTRACT

This study investigated the relationship between iron deficiency/iron deficiency anaemia, assessed by several parameters, and blood lead concentration in children.

This cross-sectional study involved 384 Brazilian children, aged 2–11 years, who lived near a lead-manipulating industry. Complete blood counts were obtained by an automated cell counter. Serum iron, total iron binding capacity (TIBC) and ferritin were determined respectively, by colorimetric, turbidimetric methods and chemiluminescence. Blood lead was measured by atomic absorption spectrophotometry. The impact of several parameters for assessment of iron status (haemoglobin, serum iron, TIBC, transferrin saturation, ferritin, red cell indices and red cell distribution width) and variables (gender, age, mother's education, income, body mass index, iron intake, and distance from home to lead-manipulating industry) on blood lead concentration was determined by multiple linear regression.

There were significant negative associations between blood lead and the distance from home to the lead-manipulating industry ($P < 0.001$), Hb ($P = 0.019$), and ferritin ($P = 0.023$) ($R^2 = 0.14$). Based on these results, further epidemiological studies are necessary to investigate the impact of interventions like iron supplementation or fortification, as an attempt to decrease blood lead in children.

1. Introduction

The interaction between nutritional deficiencies and toxic metals, especially lead, has been of great interest in view of the concerns about environmental pollution. Children are considered a high risk group for iron deficiency and lead poisoning. Iron deficiency is the most common nutritional disorder in the world and the main cause of anaemia in childhood, with a high prevalence in developing countries. Children are at particular risk of iron deficiency due to their high demands for iron during a period of rapid growth and because their diet is often too low in available iron.[1]

Lead is a toxic metal with no physiological function in the body. Lead poisoning has resulted from fast industrialization, including mining, food can solders, dyes (mainly paints), and the manufacture and use of glazed household pottery, in developing countries without environmental controls. In a lead contaminated environment children are more exposed to lead because they have more hand-to-mouth activity and absorb lead more efficiently than do adults.[2]

Both iron deficiency and lead toxicity are detrimental to the growth and development of children causing behavioural and cognitive problems, and poor school performance.[1,3,4] Even blood lead concentrations below

* Corresponding author. Tel./fax: +55 11 3061 7867.
E-mail address: phcrondo@usp.br (P.H.C. Rondó).

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10 μg/dL (0.48 μmol/L) have been associated with negative outcomes in infancy and childhood.[3,5] Deficient iron stores not only seem to augment the risk of lead absorption considering the existence of a common intestinal iron-lead transporter (divalent metal transporter 1),[6] but also increase lead retention in tissues as well as toxicity of this metal.[7]

There are still controversies regarding the association between iron deficiency/iron deficiency anaemia and blood lead concentrations; some studies have shown an association[8–12] whereas others have not.[13,14] Therefore, the objective of this study was to assess the relationship between blood lead concentration and several parameters for assessment of iron status.

2. Materials and methods

This cross-sectional study involved 384 pre-school and school children, aged 2–11 years, who participated in a prospective cohort epidemiological study carried out in Santo Amaro da Purificação city, Northeast Brazil, from 2001 to 2003. The city was chosen for its vicinity to a lead-manipulating metal industry which had been in activity for 33 years until its closure at the end of 1993. According to a previous study carried out in Brazil, lead poisoning seems to constitute a public health problem in this region of the country.[15]

All the children aged 2–11 years attending a pre-school or school in Santo Amaro city were invited to participate in the study. Their parents or guardians received detailed information about the study, and gave formal consent prior to data collection. They were interviewed before blood collection to obtain information on demographic and socioeconomic factors, and area of residence. The anthropometric data were obtained according to Jelliffe and Jelliffe[16] recommendations. The children were weighed, after having fasted for 10–12 hours, by a portable electronic scale (Sohnle®, model 7500, Nassau, Germany), with accuracy of 100 g. Their height was measured using a SECA® stadiometer (Leicester Portable Height measure model, Hamburg, Germany), with accuracy of 0.1 cm. The body mass index (BMI) was classified according to the Centers for Disease Control and Prevention (CDC):[17] low weight (<5th percentile); normal weight (5–85th percentile); risk of overweight (>85–95th percentile); overweight (≥95th percentile).

Fasting venous blood samples were collected from the children by a trained nurse, in the main laboratory of the city, into dry tubes, and tubes with EDTA and trace metal-free heparin. Complete blood counts were performed on the samples within 4–6 hours of collection using a CellDyn 3000 CD® (Abbott Laboratory, Maidenhead, Berkshire, UK) automated cell counter. Serum iron and total iron binding capacity (TIBC) were determined by colorimetric and turbidimetric methods respectively, using the SYNCHRON CX® system (Beckman Coulter, Miami, USA). Serum ferritin was assessed by chemiluminescence in the DPC 1000 IMMULITE® equipment (Diagnostic Products Corporation - DPC, Los Angeles, USA). All the measurements were performed in duplicate, including lead determination (coefficient variation = 3%).

The blood samples for lead determination were stored in a refrigerator at 6 °C, properly transported to the Toxicology section of Adolfo Lutz Laboratory in São Paulo city, and analysed within 15 to 30 days. Blood lead was measured by graphite furnace atomic absorption spectrophotometry method with Zeeman background correction (Model SIMAA 6000AA Spectrometer, Perkin-Elmer, Norwalk, CT, USA). The samples were diluted 1:10 with 1% Triton X-100 in 0.1% nitric acid, and a mixture of ammonium dihydrogen phosphate and magnesium nitrate was used as chemical modifier.[18] The quantification limit obtained for lead was 0.01 μmol/L (0.2 μg/dL) in 1:10 blood dilution, corresponding to 0.10 μmol/L in total blood. For determination of the quantification limit, a blood sample was obtained from a non-exposed person. Lead concentrations were determined in 10 preparations, and the calculation was made according to the International Union of Pure and Applied Chemistry (IUPAC) recommendations.[19] We gave the blood samples that showed lead levels below the quantification limit a value corresponding to 0.048 μmol/L (half of the limit value of the quantification method). To determine the accuracy of the method, we used a lead reference material in bovine blood (NIST 955b, level 2), obtaining a 96% recovery. Children were considered as being lead contaminated if their blood lead concentrations were equal to or greater than 0.48 μmol/L (10 μg/dL).[5]

The cut-off points adopted for the diagnosis of iron deficiency anaemia were haemoglobin (Hb) <11 g/dL for children aged less than 6 years, and <12 g/dL for those children aged 6 years or more, ferritin values <12 μg/L, TIBC ≥410 μg/dL, serum iron <40 μg/dL, and transferrin saturation (TS) <10%.[20] Reference ranges for mean corpuscular volume (MCV) (75–87 fl for children <6 years; 77–95 fl for children ≥6 years), mean corpuscular haemoglobin (MCH) (24–30 pg for children <6 years; 25–33 pg for children ≥6 years) and mean corpuscular haemoglobin concentration (MCHC) (31–37 g/dL for children <12 years) were established according to the age of the children.[20] There is no clear cut-off point for red cell distribution width (RDW) in infancy. Therefore, the <15% cut-off point established by the equipment was adopted.

To estimate the intake of foods rich in iron a 24 h diet recall was used three times, together with a food frequency questionnaire, applied to the parents or guardians of the children. The values obtained were compared with the dietary reference intake (DRI) for iron.[21] The Recommended Dietary Allowances (RDA) cut-off points for children aged 1–3 years, 4–8 years, and 9–13 years were respectively 7, 10 and 8 mg/day of iron. To estimate the foods most ingested by the children, a total of 100 women in the region were interviewed three times (two weekdays and one day in the weekend), by two dieticians from our team, in the rainy and dry seasons, with a month interval, using a 24 h diet recall. In a study carried out by the same authors in Santo Amaro city (unpublished data), we asked how often on average fruits and vegetables (with seasonal variation) were consumed in season. A list of seasonal fruits and vegetables and the length of season for each item were calculated, allowing us to take seasonality into

account to calculate the average daily intakes of such foods. The ingestion of foods rich in iron did not vary greatly by season in this region of Brazil. Based on the data obtained on the 24 h diet recall, a food frequency questionnaire was developed to assess iron intake by the 100 children. The food frequency questionnaire included 42 food items, food names, food portions and how often the children consumed them, to determine the quantity and the regularity of the foods consumed. The NutriSurvey for Windows 95, an easily accessible software (http://www.nutrisurvey.de) including Brazilian food[22] was used to analyze data of the questionnaire, particularly iron intake, and compared with the DRIs.[21]

The STATA-10 software (College Station, TX, USA) was used for storage and statistical analysis of the data. The relationship between the following iron parameters: serum iron, TIBC, TS, ferritin, Hb, MCV, MCH, MCHC, RDW, and the confounding variables (gender, age, education of the mother, per capita income, BMI, iron intake, and distance from home to the lead-manipulating industry) on blood lead concentrations were determined using Pearson's or Spearman's correlation. Finally the impact of all the independent variables cited above (selected according to their importance, correlation coefficient and collinearity) on blood lead concentrations was determined by multiple linear regression, using the backward stepwise selection method. The outcome variable, blood lead, was analyzed separately for each independent variable. The variables with descriptive level $P \leq 0.20$ were selected for entry into the multiple linear regression model. In the final model, the variables with a $P \leq 0.05$ were considered as being statistically significant.

Informed written consent was obtained from all parents/guardians and the protocol was approved by the Ethical Committee of the University of São Paulo and Santo Amaro city Health Secretariat.

3. Results

The characteristics of the children are presented in Table 1. The children included in this study were of a low socioeconomic status, considering that the majority of their mothers had <4 years of education and a per capita income of ≤R$50 (approximately US$45). Most of the children presented a normal weight (79.9%), adequate iron intake (60.6%), concentrations of blood lead ≥5 μg/dL (62.5%), anaemia (55.7%) based on Hb concentrations, normal red cell indices [MCV (84.4%), MCH (85.1%), MCHC (90.3%)] and RDW (85.7%). Although the parameters serum iron, TIBC, TS and ferritin were not suggestive of iron deficiency anaemia according to the cut-off points proposed, most of the values were compatible with iron depletion or iron deficient erythropoiesis.[20] Table 2 shows a multivariate linear regression model considering blood lead of the children as the dependent variable. There were statistically significant negative associations between blood lead and distance from home to the lead-manipulating metal industry (P <0.001), Hb (P = 0.019), and ferritin (P = 0.023) (R^2 = 0.15; adj.R^2 = 0.14).

4. Discussion

According to the results of this study, blood lead concentration in children from 2–11 years of age was negatively associated with the distance from home to the lead-manipulating metal industry (P <0.001), Hb (P = 0.019) and ferritin (P = 0.023). The linear regression model explained 14% (R^2) of the variation in blood lead concentration (Table 2) suggesting that there are other factors associated with lead in blood, or that it is not an adequate parameter to assess lead toxicity in this population.

Blood lead is a good indicator of acute lead toxicity/poisoning, but chronic exposure to lead is better assessed by determination of lead in bone.[4] However, lead in blood is widely used in epidemiological studies, probably due to the difficulty of determining bone lead in addition to blood lead in this type of study. In our study 18.2% of the children presented lead concentrations ≥10 μg/dL and 44.3% of them concentrations from 5–9.9 μg/dL, indicating that these children had acute lead poisoning and toxicity, respectively, but probably, they were also chronically exposed to lead.

Distance from the homes of the children to the lead-manipulating metal industry was highly associated with blood lead concentrations, despite the closure of the industry almost 10 years earlier. Children living within a 1650 meters radius from the industry had higher blood lead concentrations [8.95 (± 4.61) μmol/L] compared with children living within 1650–3300 [5.44 (± 3.72) μmol/L] meters radius or longer. A study carried out in the same city, involving pregnant women and their respective newborns, showed similar results.[23] It is well known that due to its chemical stability in soil, lead can persist on the ground for several years, even after the closure of the main source of contamination.[24]

In this study ferritin and Hb concentrations were also negatively associated with blood lead. Watson,[9] employing a double-isotopic technique, reported that iron-deficient (low serum ferritin) adults had increased absorption of lead and iron in contrast to iron-replete (normal serum ferritin) adults. However, Serwint et al.[15] investigating children 11–33 months old did not find a correlation between blood lead and ferritin concentrations in children with low and moderate lead exposure. Choi and Kim[25] only found an influence of ferritin on blood lead when serum iron declined. Tripathi et al.[26] observed a decreasing trend in Hb with increasing blood lead in 3–6 year old Indian children, and Rondó et al.[27] detected an inverse association between Hb and blood lead concentration in anaemic children aged 2–11 years who were living near a lead smelter in the south of Brazil. Muwakkit et al.[13] investigated the influence of dietary and socioeconomic factors, and blood lead on iron deficiency anaemia (based on measurements of Hb, TS and RDW). The authors referred to associations between iron deficiency anaemia and increased blood lead concentrations, lack of iron supplementation, and cultural dietary habits.

Few studies have controlled for the association between blood lead and iron status by the level of environmental contamination. Bradman et al.[10] found that children 1–6 years of age with low ferritin had higher blood lead

Table 1
Characteristics of the children ($n = 384$)

Variables	*n*	%	Mean (SD)
Gender			
Male	203	52.9	
Female	181	47.1	
Age (years)			7.23 (1.93)
<4	15	3.9	
4–6	167	43.5	
7–9	119	31.0	
>9	83	21.6	
Per capita income (Brazilian Real – R$)[a]			38.052 (28.10)
<20	85	23.8	
20–30	78	22.0	
30–50	96	27.0	
>50	97	27.2	
Body Mass Índex (BMI)[17]			15.17 (1.16)
Low weight (<5th percentile)	55	15	
Normal weight (5–85th percentile)	292	79.9	
Risk of overweight (>85–95th percentile)	14	3.8	
Overweight (≥95 percentile)	5	1.3	
Iron intake (mg/day)[21] [b]			13.28 (9.67)
Low	132	39.4	
Adequate	203	60.6	
Distance home-lead manipulating industry (meters)			
Near (<1650)	169	44	
Medium (1650–3299)	93	24.2	
Far (3300–5000)	122	31.8	
Blood lead (μg/dL)			6.99 (4.39)
<5.0	144	37.5	Median = 6.0
5.0–9.9	170	44.3	
≥10.0	70	18.2	
Haemoglobin (g/dL)[20]			11.58 (0.88)
Anaemic	214	55.7	
Non-anaemic	170	44.3	
Serum Iron (μg/dL)			86.28 (25.64)
<40	13	3.5	
40–60	47	12.5	
≥60	316	84.0	
Total Iron Binding Capacity (TIBC) (μg/dL)			398.41 (45.16)
<360	72	19.1	
360–410	159	42.3	
≥410	145	38.6	
Transferrin Saturation (%)			21.79 (6.36)
<10	13	3.5	
10–30	331	88.0	
≥30	32	8.5	
Ferritin (μg/L)			39.73 (24.92)
<12	22	5.9	Median = 34.4
≥12	354	94.1	
Mean Corpuscular Volume-MCV (fl)[20]			80.9 (4.99)
Microcytic	56	14.6	
Normocytic	324	84.4	
Macrocytic	4	1.0	
Mean Corpuscular Haemoglobin-MCH (pg)[20]			26.52 (2.03)
Decreased	54	14.1	
Normal	327	85.1	
Increased	3	0.8	
Mean Corpuscular Haemoglobin Concentration-MCHC (g/dL)[20]			32.76 (0.88)
Hypochromic	36	9.4	
Normochromic	347	90.3	
Hyperchromic	1	0.3	
Red blood cell Distribution Width-RDW (%)[c]			13.57 (1.63)
Decreased	4	1.0	
Normal	329	85.7	
Increased	51	13.3	

[a] R$90 = approx. US$50;
[b] RDA cut-off points for children 1–3 years; 4–8 years; and 9–13 years were respectively 7, 10 and 8 mg/day of iron;
[c] RDW<15%, according to the equipment CellDyn 3000® automated cell counter (Abbott Laboratory, Maidenhead, Berkshire, UK).

Table 2
Multivariate linear regression model considering blood lead concentration in 384 children as the dependent variable

Blood Lead	Coefficient	Standard Error	95% CI	*P*
Distance from home to lead-manipulating industry	−1.885	0.242	−2.362 to −1.408	< 0.001
Haemoglobin	−0.589	0.249	−1.080 to −0.099	0.019
Ferritin	−0.018	0.008	−0.034 to −0.002	0.023
Constant	18.091	2.985	12.221 to 23.961	< 0.001

$R^2 = 0.15$; adj. $R^2 = 0.14$

concentrations than children with normal ferritin. The relationship persisted after stratification by the level of environmental contamination measured in their homes. A longitudinal study that evaluated the association between iron deficiency anaemia (assessed by Hb, MCV, and RDW) and lead poisoning, in children from 9–42 months of age, showed that iron deficiency precedes lead poisoning.[11] However, as the authors have emphasised there was no control for environmental contamination.

There are very few clinical trials assessing the impact of iron supplementation or iron fortification on blood lead concentrations, and the results are controversial. Wolf et al.[28] administered oral iron or a placebo to children aged 12–23 months and observed changes in blood lead concentrations which corresponded closely to changes in iron status. However, Rosado et al.[29] concluded that iron supplementation of lead-exposed children significantly improved iron status but did not reduce blood lead concentrations. Ruff et al.[30] referred that iron-supplemented children with pre-existing lead poisoning have a slower decline in blood lead compared with non-supplemented children. The recommendations of Wright et al.[31] regarding iron supplementation is to use it for lead-poisoned children with documented iron deficiency, and for iron-replete children with continued lead exposure. Zimmermann et al.[12] observed that improving iron status, through iron fortification, in iron deficient, lead exposed children from 5–9 years of age reduces their blood lead concentrations and may reduce chronic lead intoxication. However, according to Zimmermann et al.[12] iron fortification of food staples often does not effectively reach children younger than 2 years of age in developing countries, the ones most at risk for lead poisoning and iron deficiency.[8]

In conclusion, further epidemiological studies are necessary to investigate the impact of nutritional interventions like iron supplementation and iron fortification, as an attempt to decrease blood lead in children, associated to environmental and educational interventions.

Authors' contributions: PHCR designed the study protocol, secured funding, facilitated data collection, participated in the statistical analysis, interpreted data and did the main writing of the paper. AC coordinated all the data collection and participated in the interpretation of data and writing of the paper. MCdS designed the study protocol, participated in the interpretation of the results and discussion. AS participated in the interpretation of the results and writing of the paper. All authors read and approved the final version of the paper. PHCR is guarantor of the paper.

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Conflicts of interest: None declared

Ethical approval: The present study was carried out in accordance with the Declaration of Helsinki of the World Medical Association, and it was approved by the Research Ethics Committee of the School of Public Health, University of São Paulo.

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