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Lead exposure in children: Levels in blood, prevalence of intoxication and related factors

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Lead is a highly toxic metal, the main source of which is contamination from combustion of unleaded petrol. The aims of this work were to detect the degree of lead exposure in a large sample of children; determine the relationship between blood lead levels (BPb) and age, sex, habitat and season of the year; and correlate BPb with zinc protoporphyrin (ZPP) values. A cross-sectional study was carried out. Blood from routine extractions drawn at our centre was used. BPb and ZPP were measured by atomic absorption spectrophotometry and haematofluorimetry, respectively. We analysed 1158 blood samples from children. BPb (mean ± SEM): 0.22 ± 0.04 µmol l^{-1}. Correlation BPb–age: BPb = 0.19 + 0.086 x age (months), r = 0.129, P < 0.0001. BPb was greater in boys (0.23 ± 0.007 versus 0.20 ± 0.006 µmol l^{-1}, P < 0.0002). No differences were observed between habitats (urban versus rural). BPb were higher in the warm months (0.24 ± 0.013 versus 0.21 ± 0.007 µmol l^{-1}, P < 0.0001). Prevalence of lead intoxication (BPb > 0.48 µmol l^{-1}) was 4.2%. No differences in prevalence were found among the different groups. The correlation between BPb and ZPP showed r = 0.0969, P = 0.0024. Utility for screening: sensitivity of 53.7% and specificity of 59.3% (cut-off point of 60 µmol ZPP mol^{-1} haem). We can conclude that lead exposure in children in our sample was in the range reported in similar studies in other areas and countries, and below the toxic limit. None of the factors analysed significantly influenced lead intoxication prevalence. There was no good correlation between ZPP and BPb in our sample and the ZPP cut-off point used did not present good specificity and sensitivity values.

Keywords: children, lead, toxicity

Introduction

Lead is a ubiquitous, highly toxic metal to which we are exposed in normal life, mainly because of the atmospheric contamination from leaded petrol. The clinical symptoms of lead poisoning, known as Saturnism, have long been known. Although the classic picture of lead poisoning is currently rare, noxious effects of the metal at very low exposure levels have been observed, particularly on the developing nervous system of children (Landrigan & Graef 1987, Folinsbee 1992, Needleman 1993).

Certain characteristics in children render them vulnerable to lead poisoning: greater absorptive capacity by the digestive tract, increased absorption in states of ferropenia or deficient calcium intake frequent at these ages, great hand-to-mouth activity of children, and the rapid development of the nervous system which occurs in this period of life (Sayre *et al*. 1974, Charney *et al*. 1983, Lockitch 1993).

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Since Needleman *et al.* (1979) reported that lead concentrations in dentine of children correlated inversely with intellectual development and behaviour in school-age children, many other studies have corroborated these deleterious effects of low-level lead exposure on the central nervous system in children, effects which have been shown to be persistent and irreversible, with no treatment having so far proved useful (Dietrich *et al.* 1987, 1992, 1993, Faust & Brown 1987, Fulton *et al.* 1987, McMichael *et al.* 1988, Needleman & Gatsonis 1990, Rabinowitz *et al.* 1991, 1992, Baghurst *et al.* 1992, Bellinger *et al.* 1992, Sciarillo *et al.* 1992, Wasserman *et al.* 1992, Leviton *et al.* 1993).

Exposure levels regarded as safe have decreased with the discovery of toxic effects even in blood lead levels considered low. Thus, as in the 1970s blood lead levels of 1.44 μmol l^{-1} were considered safe, current knowledge shows that toxic manifestations are already beginning with exposures higher than 0.48 μmol l^{-1}, and as the innoculty of exposure at lower lead doses cannot be guaranteed, the only really safe blood lead level is 0 μmol l^{-1} (American Academy of Pediatrics 1987).

Considering that no blood lead level can be regarded as safe, no treatment is available to alleviate the effects of low-dose lead exposure and that harmful effects in children are persistent and irreversible, the only really efficacious measure will be prevention, acting on sources avoiding the exposure of the individual to lead (Centers for Disease Control 1991).

Determination of blood lead is an indicator of recent or chronic exposure to the metal. Knowledge of blood levels in a given population would give an idea of the magnitude of exposure and orient adequate preventive methods for each case.

Lead pollution levels in the Metropolitan Area of Barcelona are higher than in other parts of Catalonia (Generalitat de Catalunya 1995), probably due to the greater density of traffic. A higher number of individuals at risk for lead exposure may exist in this zone and therefore increased measures of exposure control and monitoring of individuals at risk may be required.

The aims of the present study were: (1) to determine exposure to lead in a wide sample of children from the Metropolitan Area of Barcelona and compare it with other regions of Catalonia with less lead pollution; (2) to observe the possible association between blood lead levels and the prevalence of intoxication with various factors that could be considered of risk (age, sex, area of residence, childhood illness, time of year when extraction was made); and (3) to ascertain the relationship between blood lead level (BPb) and zinc protoporphyrin (ZPP) levels in the same individuals, since ZPP determination has been recommended as a screening method for lead exposure.

Subjects, materials and methods

The study was conducted between January 1 and December 31, 1993 in Hospital Materno-Infantil Vall d'Hebron, Barcelona, Spain.

Subjects

Convenience sampling was used for subject selection: all consecutive routine samples drawn on days when blood lead determinations could be made were used to assess blood lead and zinc protoporphyrin levels, without previous case selection.

Data on age, sex, habitat and reason for blood extraction were obtained from clinical records of the children. Age was quantified in days from date of birth to the date when the extraction was made. To evaluate the influence of habitat on children's blood lead concentrations, individuals were assigned to one of four residence groups: (1) the city of Barcelona; (2) towns of the Metropolitan Area of Barcelona; (3) other towns; and (4) populations of less than 20 000 inhabitants. Children were divided into two groups according to the period of the year in which the extraction was made: warm months (spring and summer) or cold months (autumn and winter).

A total of 1158 blood samples was analysed. Age of the individuals was 6.32 ± 4.6 years (mean ± SD). The sample showed a slight predominance of males ($n = 637$; 55%) versus females ($n = 521$; 45%). Of the samples, 245 were obtained in spring (21.2%), 224 in summer (19.3%), 345 in autumn (29.8%) and 344 in winter (29.7%). By residence zones, 393 samples were from children living in the city of Barcelona (33.9%), 390 from the Metropolitan Area of Barcelona (33.7%), 122 from other towns (10.5%) and 253 from rural areas (21.8%). Reasons for blood extractions were: pre-operative analysis in 70 previously healthy children (6%); infectious diseases (gastro-enteritis, pneumonia, urinary tract infections) in 169 (14.6%); respiratory disorders (asthma, bronchitis, cystic fibrosis) in 212 (18.3%); cardiac diseases (congenital heart diseases) in 110 (9.5%); digestive diseases (malabsorption, diarrhoea, hepatic diseases) in 134 (11.6%); endocrine disorders (diabetes mellitus, growth retardation) in 81 (7%); haematologic problems (anaemia) in 95 (8.2%); nephrologic diseases (renal failure, arterial hypertension) in 195 (16.8%); oncologic diseases (leukaemia, lymphoma) in 65 (5.6%); and rheumatologic diseases (chronic juvenile arthritis) in 27 (2.3%).

Instrumentation

A Perkin-Elmer 3030 B atomic absorption spectrometer equipped with an HGA-400 electrothermal and an AS-40 autosampler, and a PR-100 printer-sequencer were used. Pb EDL was used as the radiation source. Absorbance was measured at a wavelength of 283.3 nm, using a band-pass of 0.7 nm. Pyrolytic graphite-coated tubes with inserted graphite platforms and deuterium background corrector were used throughout. Solutions were pipetted by adjustable-volume Gilson pipettes with disposable tips.

Chemicals and reagents

Ultra-pure water Milli-Q (Millipore Corp., Bedford, MA, USA) with 0.2 % HNO_3 was used to clean the autosampler and prepare standards and solutions. Nitric acid (Merck, Darmstadt, Germany), $NH_4H_2PO_4$ and Triton X-100 (BDH Chemicals, Poole, Dorset, England, UK) were of special quality for atomic absorption. All necessary material (polyethylene tubes, volumetric flasks and tips) was previously cleaned by soaking in 10% nitric acid, washed several times in ultrapure water and kept closed in bags or containers of polyethylene until use.

Analytical procedure

A first series of standard solutions in the range of 0.48–2.41 mmol l^{-1} was prepared by dilution with 0.7% nitric acid from a Pb stock solution of 4.82 mmol l^{-1} (Spectrosol, BDH). Working standards were prepared daily by diluting the standard solutions above appropriately with the matrix modifier. The matrix-matched Pb calibration curve ranged from 0.048 to 0.24 mmol l^{-1}. A matrix modifier consisting of 0.2% (w/v) $NH_4H_2PO_4$, 0.5% Triton X-100, 0.5% HNO_3 and ultrapure water was used. The volume of the injected samples was always 10 µl. Accuracy and precision of Pb analyses were checked by running certified Pb content standards (CRDL Standard 2 solution; Radian Corp, Austin, TX, USA & Canada) intercalated between every 10 samples in the autosampler with each new analysis batch. Samples which did not differ by more than 2% of the standard value were considered acceptable (Banno *et al*. 1994). A direct calibration curve was performed in each assay and was considered not valid if the linear regression coefficient was less than 0.99 and if intra-assay coefficients of variation were above 3%. All samples were analysed in triplicate and results refer to the standard curve made previously.

Samples

Blood samples were collected in polyethylene 1 ml tubes containing EDTA (Microtainer, Becton Dickinson, USA). Syringes and tubes were previously tested for Pb content. Haematological determinations were carried out on the same day as the extraction. For blood Pb determinations blood samples were stored at +4ºC for no more than a week; we had previously proved the stability of blood Pb levels when samples are stored at +4ºC. For measurement, samples were prepared by dilution of 100 µl of blood in 900 µl of the matrix modifier (0.5% Triton X-100, 0.2% $NH_4H_2PO_4$). The initial Pb result was corrected by the haematocrit (HCT) of each sample to avoid errors produced by extreme HCT values.

Graphite furnace conditions

We used the furnace temperature programme shown in Table 1, previously optimised in our laboratory. Under the described analytical conditions, the limit of Pb detection (signal equivalent to three times the background noise) was approximately 0.009 mmol l^{-1} .

The same samples were used to assess zinc protoporphyrin (ZPP) using haematofluorimetry (Helena Laboratories, Beaumont, TX, USA), following the usual technique. After the calibration with known values of protoporphyrins, the concentration of protoporphyrins is shown in µg dl^{-1}. These results were corrected with haemoglobin values transforming the results in µmol ZPP mol^{-1} haem. Accuracy was verified with samples with a known amount of ZPP. All samples were analysed in triplicate.

Statistical analysis was performed using the SPSS-PC+ software package. Simple linear regression was applied in testing for associations between study variables: BPb–age and BPb–ZPP. Significant differences in quantitative variables (BPb) compared with qualitative variables (residence, illness, period of year) were evaluated by analysis of variance with Duncan correction ($P < 0.05$). To evaluate the utility of ZPP for screening of lead exposure, values of sensitivity, specificity and predictive values were calculated. A P value < 0.05 was considered statistically significant.

Results are expressed as mean ± standard error for mean (mean ± SEM).

Table 1.

Step	Temperature (ºC)	Ramp (s)	Hold (s)	Gas flow (ml min^{-1})
Dry 1	130	10	5	300
Dry 2	200	15	20	300
Char	850	15	45	300
Atomisation	1800	0	2	0
Pyrolysis	2500	1	4	300
Cool	20	10	5	300

Results

Blood lead was 0.22 ± 0.04 μmol l^{-1}. Median was 0.19 μmol l^{-1}. Figure 1 shows the distribution histogram of blood lead values in the sample.

BPb–age relationship. Blood lead levels showed a slight relationship with age of the children (BPb = 0.19 + 0.086 × age in months, $r = 0.129$, $P < 0.0001$). When individuals were grouped in one-year intervals, an increase in BPb between children under one year of age (BPb = 0.14 ± 0.01 μmol l^{-1}, median 0.14) and children over three years of age (BPb = 0.21 ± 0.01 μmol l^{-1}, median 0.19) was observed; this difference was statistically significant ($P < 0.005$) (Figure 2).

BPb–sex relationship. Blood lead levels were higher in males (0.23 ± 0.007, median 0.19 μmol l^{-1}) than in females (0.20 ± 0.006, median 0.19 μmol l^{-1}, $P < 0.0002$). These differences were observed in all age groups except in children under one year of age, in whom BPb values were higher in females (0.14 cf. 0.13 μmol l^{-1}).

BPb–illness relationship. No significant differences were observed in BPb in relation to the illness of the individual. In all cases, BPb values were far below the toxicity threshold of 0.48 μmol l^{-1}. Maximum values corresponded to children with respiratory and kidney disease (0.24 μmol l^{-1}) (Figure 3).

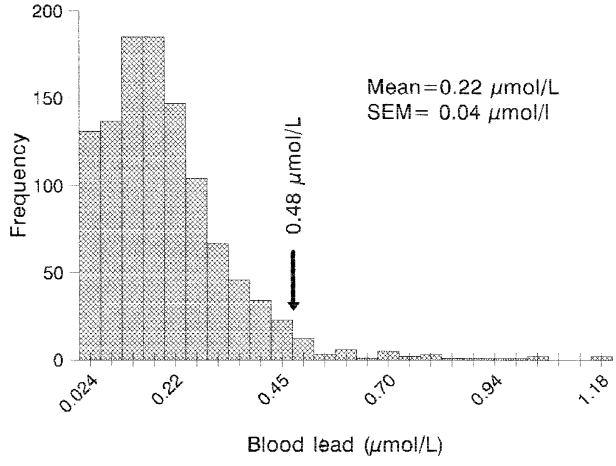

Figure 1. Distribution histogram of blood lead values in children of our sample (1158 cases). Arrow shows the currently considered toxicity limit (0.48 μmol l^{-1}). Each bar represents an interval of 0.048 μmol l^{-1} in BPb.

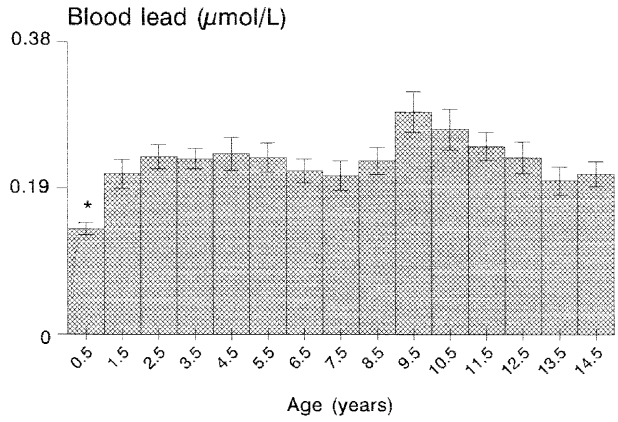

Figure 2. Distribution histogram of blood lead values in different age groups in the sample (1158 cases). Bars represent mean ± SEM of blood lead for each one-year interval. *Statistically significant differences ($P < 0.005$).

Relationship with zone of residence. No differences were found in BPb in relation to the zone of residence: Barcelona 0.24 ± 0.01 μmol l^{-1} (median 0.19); Metropolitan Area of Barcelona 0.22 ± 0.009 μmol l^{-1} (median 0.19); other cities 0.19 ± 0.014 μmol l^{-1} (median 0.19); and rural areas 0.23 ± 0.015 μmol l^{-1} (median 0.19).

Relationship with season of the year. Higher blood lead levels were found in samples drawn in spring and summer (0.24 ± 0.013 μmol l^{-1}) compared with samples drawn in autumn and winter (0.21 ± 0.007 μmol l^{-1}); this difference was statistically significant ($P < 0.0001$) (Figure 4).

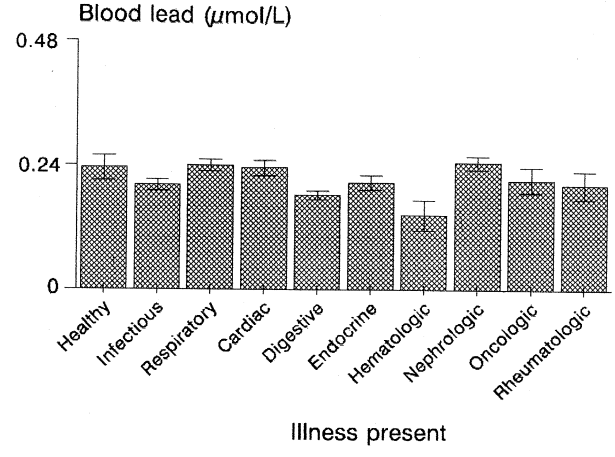

Figure 3. Blood lead values in children grouped according to illness present at the time of extraction ($n = 1158$ cases). Each bar represents mean ± SEM. No statistically significant differences are seen among groups. Blood lead values are below the toxicity limit in each group (0.48 μmol l^{-1}).

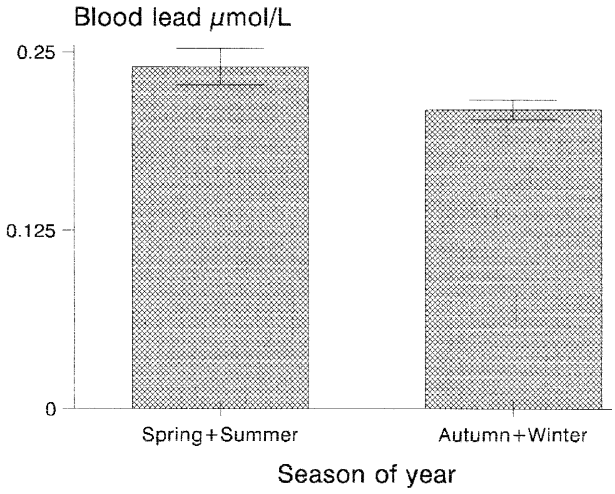

Figure 4. Blood lead values in children according to the season of the year when the sample was obtained ($n = 1158$ cases). Each bar represents mean ± SEM. Differences between groups are statistically significant ($P < 0.0001$).

Table 2. Analysis of variance. Influence of variables on blood lead levels

Source of variation	*F*	*F* significance
Covariables		
Age	13.047	0.000
Main effects	4.489	0.000
SEX	11.098	0.001
RESIDENCE	3.060	0.028
SEASON	4.151	0.006
Explained variance	2.34	0.000
Multiple classification analysis		
	Adjusted deviation	Beta
SEX		
Male	0.04	
Female	–0.05	0.13
RESIDENCE		
Barcelona	0.03	
Metropolitan Area	0.00	
Cities	–0.10	
Rural	–0.01	0.12
SEASON		
Spring	0.06	
Summer	0.02	
Autumn	–0.08	
Winter	0.01	0.14
Multiple *r*		0.253
Multiple r^2		0.064

Influence of variables on blood lead levels. The data of analysis of variance of all parameters taken together are shown in Table 2. A highly significant influence of the parameters on blood lead levels was observed ($F = 0.000$).

Prevalence of lead toxicity. Following current Centers for Disease Control (CDC) recommendations, an individual with a BPb value over 0.48 µmol l^{-1} was considered as intoxicated. In our sample, 49 children exceeded this threshold; thus, the intoxication prevalence was 4.2% of children. No significant differences in prevalence were observed according to age (Figure 5), residence area (Barcelona 4.4 ± 1.3%, Metropolitan Area 4.8 ± 1.3%, other cities 2.8 ± 2%, rural 7.5 ± 2.1%), sex (males 5.2 ± 0.88%, females 3.6 ± 0.82%), illness, or season of the year when extraction was made (summer and spring 5.7 ± 1.5%, winter and autumn 4.2 ± 0.66%).

Zinc protoporphyrin determinations. ZPP values in the sample were 55.2 ± 0.72 µmol ZPP mol^{-1} haem. Geometric mean was 49.3 µmol ZPP mol^{-1} haem.

BPb–ZPP relationship. Correlation between BPb and ZPP values was poor ($r = 0.098$). The correlation was better when only intoxicated children were considered (Figure 6).

Utility of ZPP for screening of lead toxicity. Using a cut-off point of 60 µmol ZPP mol^{-1} haem, the method has shown sensitivity of 53.7%, specificity of 59.3%, a positive predictive value of 6.5% and a negative predictive value of 95.3%. Prevalence of children with ZPP values over the cut-off point was 62.7%.

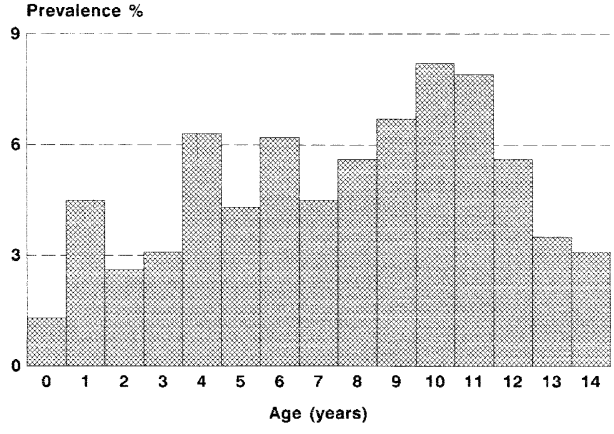

Figure 5. Prevalence of lead toxicity in children of our sample in different age groups ($n = 1158$ cases). No statistically significant differences are observed.

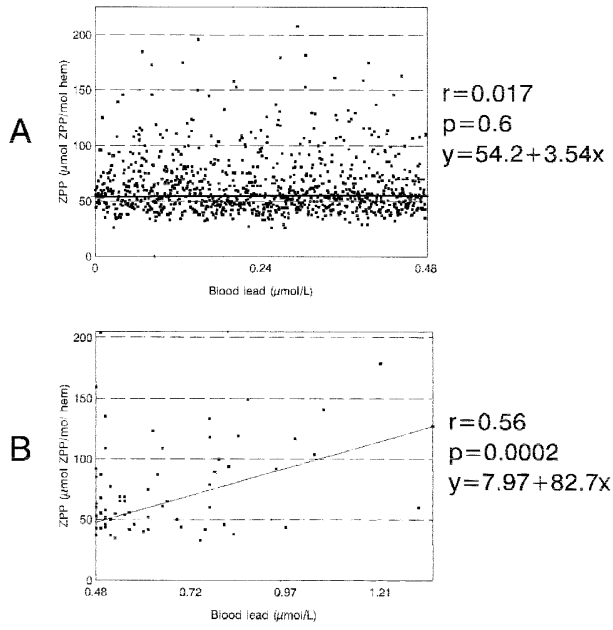

Figure 6. Correlation between blood lead and zinc protoporphyrin values in children in our sample ($n = 1158$ cases). A: Non-intoxicated children (BPb < 0.48 µmol l^{-1}) B: Lead-intoxicated children (BPb > 0.48 µmol l^{-1}). Dots represent ZPP and BPb values for each child in the sample.

Discussion

Our data show that lead exposure in infancy is not an important health problem in our area. Blood lead levels found were low in all age groups and below the toxicity threshold (0.48 µmol l^{-1}). Similar studies conducted in other areas of Spain obtained different results, depending on the year of the study and area of the country. Blood lead levels were higher in earlier studies and in areas with greater risk owing to industrial activity, in which mean blood lead values in children were found to be between 0.68 and 1.3 µmol l^{-1} (Cabeza *et al.* 1991, Rivas *et al.* 1993). Following European Community recommendations, maximum lead content in petrol in Spain was reduced in 1991 to a maximum of 0.15 g l^{-1} (Ministerio de Industria y Energía 1988). Studies conducted since 1991 show much lower blood lead levels of between 0.12 and 0.25 µmol l^{-1} in children depending on the area (Ordóñez *et al.* 1993, Cambra & Alonso 1995, Redondo *et al.* 1995), although all studies were limited by the low number of subjects in the sample, under 500 in all cases, and may therefore not be truly representative of the situation in the general child population.

The lead concentration found in the atmosphere in our study area was below the maximum accepted limit of 2 µg m^{-3} annual mean, although values found were higher in the city of Barcelona and its Metropolitan Area, which reflects the greater contamination from traffic in these areas. A tendency towards decreased atmospheric lead concentrations has been observed in recent years thanks to the measures taken regarding the maximum lead content in petrol and the extended use of lead-free petrol (Table 3).

Children of our sample come from areas with heavy traffic but with no other lead sources (smelters, industries with high lead emissions); thus, there is no specific risk of lead poisoning. Although for selection we used a convenience sampling that may induce bias in the interpretation of results, we believe that the size of the sample (particularly compared with other studies conducted in Spain) and the objective of the study (to detect lead exposure in normal life but not in specific groups) permits us to assume that our results are extrapolable to the whole population from which our sample derives. Our data are similar to those obtained by Ordóñez in Madrid (Ordóñez *et al.* 1993), Cambra in Bilbao (Cambra & Alonso 1995) and Redondo in Valladolid (Redondo *et al.* 1995), who found mean blood lead values in children of 0.12, 0.27 and 0.27 µmol l^{-1}, respectively, with prevalence of lead intoxication between 1.4 and 3%.

No relevant differences in blood lead levels are observed between our results and those of studies from several European countries (Braithwaite & Brown 1988, Mauras *et al.* 1995) and America (Leung *et al.* 1993, Brody *et al.* 1994, Norman *et al.* 1994, Jin *et al.* 1995). All detected mean blood lead levels of 0.2 µmol l^{-1} in the child population. A reduction in blood lead levels is also found in most recent works, reflecting the effect of the widespread use of unleaded petrol on atmospheric contamination by lead (Davis *et al.* 1993) and a concomitant reduction in blood lead levels in the population (Shy

Table 3. Lead atmospheric concentrations in the study area expressed in µg m^{-3} annual mean. Source: Generalitat de Catalunya, 1995

Area	Year		
	1991	1992	1993
Barcelona	0.61	0.39	0.36
Metropolitan Area	0.54	0.39	0.35
Other cities	0.19	0.12	0.10
Rural areas	0.25	0.07	0.05

1990, Burke 1993, Wietlisbach *et al*. 1995). Between 1991 and 1994 mean geometric blood lead levels in the USA dropped from 12.8 μg dl^{-1} to 2.9 μg dl^{-1}, and the prevalence of values higher than toxicity level decreased from 77.8% to 4.4% (Brody *et al*. 1994, Norman *et al*. 1994, MMWR 1997). Similar results have recently been described in the city of Barcelona in a sample basically comprised of adults and following the reduction of lead in petrol (Rodamilans *et al*. 1996). Taking into account the prevalence of children with lead levels in the toxic range, our results are also similar to those of other recent studies from developed countries.

Age is the most important factor related to lead levels in children of our sample. An evident and significant increase in BPb values was observed in children during the first three years of life, rising from 0.14 to 0.21 μmol l^{-1}, and remaining stable throughout infancy (Figure 2). This rise is attributed to the increase in oral and open-air activities in children of these ages, and the greater exposure to lead from petrol in the environment (Rivas *et al*. 1993, Brody *et al*. 1994, Norman *et al*. 1994, Al-Saleh *et al*. 1995). This fact is important, since at these ages development of the nervous system in children is greater, and renders them more vulnerable to the harmful effects of the metal.

Sex and the season of the year when the extraction was made were also statistically related to BPb. The statistical significance for both factors is very high, but the magnitude of the difference in absolute values is scant: a difference of only 0.034 μmol l^{-1} in BPb was observed between boys and girls and between samples collected in warm and cold months. These high statistical significances therefore lack clinical importance. Other authors coincide in the findings of these differences and attribute them to the greater open-air activity of boys versus girls (Schuhmacher *et al*. 1992, Brody *et al*. 1994, Norman *et al*. 1994) and warm versus cold months (Jin *et al*. 1995); thus, in warm months of the year children appear to be more exposed to atmospheric contamination by the metal.

One difference to be taken into account is in BPb levels between urban and rural areas. Although greater exposure in areas with high atmospheric contamination by lead was to be expected, our results not only revealed no statistically significant differences, but higher BPb levels in children from rural areas than in those from urban areas. These results concur with those of other studies (Rivas *et al*. 1993, Norman *et al*. 1994). The possibility of other sources of lead in rural areas should be considered, since a greater number of old houses with leaded pipes or walls painted with high-lead-content paint still exist, as well as certain marginal populations in whom poor living conditions may be added to nutritional deficiencies which favour absorption of lead by the digestive tract (Al-Saleh *et al*. 1995, Redondo & Guisasola 1995).

Consideration of these factors as a whole reveals that boys, residing in the city of Barcelona and during the warmer months of the year, are at greater risk of lead exposure.

Although lead exposure is not a public health problem in the area of the study, we found 49 children with blood lead values over 0.48 μmol l^{-1}. Since no relationship was detected between the factors analysed (age, sex, zone of residence, reason for extraction of the sample) and the prevalence of children with blood Pb levels above 0.48 μmol l^{-1}, parents and paediatricians of this reduced group were informed so that serial measurements of lead levels and individualised follow-up could be made. The reasons for these high lead levels in some children may be individual factors for increased lead absorption (anaemia, deficits in Ca) or inapparent sources of the metal (ceramics, lead in paintings or in soil). In any event, sources of lead in children were not studied in depth, since the aim of our study was to detect prevalence levels in our area.

The prevalence of children with lead values within the toxic range is similar to the prevalence found in other studies, and no differences in prevalence were detected following stratification of the group by age, sex or area of residence. With the prevalence found in the greater-risk group, i.e. children between 12 and 36 months of age, massive screening of the population would not be indicated according to the most recent recommendations, only individualised assessment of subjects at special risk (Centers for Disease Control 1997).

Determination of ZPP in whole blood has been recommended as a screening method to detect populations at risk for lead toxicity (Kammholz *et al.* 1972, Commission of European Communities 1983, Norman & Bordley 1995). Exposure to lead provokes alterations in different metabolic pathways, which are eventually manifested by the appearance of unusual metabolites that indicate the degree of exposure to the toxic metal. One of the earliest effects of lead exposure is haem synthesis inhibition. Pb impedes Fe binding to the porphyrinic ring, thereby provoking an accumulation of protoporphyrins. Thus, the finding of high ZPP values will indicate exposure to high doses of lead. The problem with this assessment is that ZPP rises when blood lead levels are considerably higher than the limit

currently accepted as toxic. In this study we observed that a ZPP cut-off point of 60 μmol ZPP mol^{-1} haem, does not present significant specificity and sensitivity values and therefore the test cannot be considered useful. Results of negative and positive predictive values indicate that the finding of a positive test will only reflect intoxication in 5% of cases. With these results, screening will not assist the decision-making process.

The poor correlation between ZPP and BPb values, particularly at low-level lead exposure, has rendered this method inaccurate for detecting individuals with BPb approaching the toxic range of 0.48 μmol l^{-1} (Blumberg *et al*. 1977, McElvaine *et al*. 1991, Rifai *et al*. 1992, Turk *et al*. 1992, Leung *et al*. 1993). Currently, the only accepted method for detecting the degree of lead exposure in a population is the determination of lead in blood. Our results also show this poor correlation between ZPP and BPb in children, which is worse in those with low blood levels (BPb < 0.48 μmol l^{-1}). Thus, ZPP cannot be considered a good screening technique for lead exposure.

Finally, this study shows that even if the prevalence of lead intoxication and lead values in children of our area are similar to those found in other studies and far below the toxic range, exposure to lead does exist and increases in the early years of life. In conclusion, measures to control and prevent exposure to lead must not be overlooked, bearing in mind that the only really safe blood lead level is 0 μmol l^{-1}.

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