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Objectives To confirm whether blood lead concentrations in Karachi were as high as reported in 1989 and to identify which types of exposure to lead contribute most to elevated blood lead concentrations in children in Karachi.

Methods A total of 430 children aged 36–60 months were selected through a geographically stratified design from the city centre, two suburbs, a rural community and an island situated within the harbour at Karachi. Blood samples were collected from children and a pretested questionnaire was administered to assess the effect of various types of exposure. Cooked food, drinking-water and house dust samples were collected from households.

Findings About 80% of children had blood lead concentrations >10 µg/dl, with an overall mean of 15.6 µg/dl. At the 5% level of significance, houses nearer to the main intersection in the city centre, application of surma to children's eyes, father's exposure to lead at workplace, parents' illiteracy and child's habit of hand-to-mouth activity were among variables associated with elevated lead concentrations in blood.

Conclusion These findings are of public health concern, as most children in Karachi are likely to suffer some degree of intellectual impairment as a result of environmental lead exposure. We believe that there is enough evidence of the continuing problem of lead in petrol to prompt the petroleum industry to take action. The evidence also shows the need for appropriate interventions in reducing the burden due to other factors associated with this toxic element.

Keywords Lead/blood; Child, Preschool; Risk factors; Environmental exposure; Vehicle emissions; Cosmetics/chemistry; Dust/analysis; Occupational exposure; Socioeconomic factors; Cross sectional studies; Pakistan (*source: MeSH, NLM*).

Mots clés Plomb/sang; Enfant âge pré-scolaire; Facteurs risque; Exposition environnement; Gaz échappement automobile ; Cosmétiques/composition chimique; Poussière/analyse; Exposition professionnelle; Facteurs socio-économiques; Etude section efficace; Pakistan (*source: MeSH, INSERM*).

Palabras clave Plomo/sangre; Infante; Factores de riesgo; Exposición a riesgos ambientales; Emisiones de vehículos; Cosméticos/química; Polvo/análisis; Exposición ocupacional; Factores socioeconómicos; Estudios transversales; Pakistán (*fuente: DeCS, BIREME*).

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Voir page 774 le résumé en français. En la página 774 figura un resumen en español.

Introduction

Lead is a pervasive chemical that is well known for its toxicity (*1*). Lead causes neurological, physiological and behavioural problems in children, ranging from raised hearing threshold and decrease in intelligence quotient (IQ) at low blood lead concentrations to acute encephalopathy, memory loss and death at high blood lead concentrations (*2*). The harmful effects of lead, at even relatively low levels of exposure, have led WHO and the US Centers for Disease Control and Prevention (CDC) to consider lead concentrations in blood ⩾10µg/dl as elevated (*2*). Sources of lead in the environment that have been shown to contribute greatly to elevated blood lead concentrations include petrol, paint, water, food, cosmetics and lead-glazed ceramics (*3, 4*).

Automobile emissions are recognized as an important source of lead exposure for urban residents, particularly in areas with congested traffic. The lead from emissions is deposited in dust, soil and other ecosystems (*5*) and subsequently ingested by children (*6, 7*). Nutritional deficiencies of iron, zinc, copper, calcium and phosphorus enhance the absorption of lead (*8, 9*).

Leaded paint is another well-established cause of lead poisoning and elevated blood lead concentrations in children, often in association with pica (*10*). In New Zealand, the blood lead concentrations fell by 42% between 1978 and 1985 after lead-based paints and varnishes were abandoned and lead-soldered food and drinks cans were replaced by seamless-welded containers (*11*).

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Two studies from Glasgow and Canada identified drinking-water as a potential source for elevated blood lead concentrations (*12*, *13*). Although concentrations of lead levels in ground and surface water are typically low (*14*), levels may increase after water from surface drainage enters the distribution system. The type of utensils and containers used for storing and boiling water or for cooking and storing food can also contribute to elevated blood lead concentrations in children (*6*).

Traditional remedies or cosmetics can also be important sources of lead exposure. "Surma" and "kohl", which are preparations of powders, gels or water-based fluids used for eye make-up, contain 16–80% lead (*15*). A study in Faisalabad, Pakistan, in 1988 reported that 80% of 20 samples of surma had a lead content >65% (*16*). In Israel, blood lead concentrations were significantly higher in infants to whom kohl was applied than in those who did not have kohl applied (11.2 vs 4.3 μg/dl). Among infants not directly exposed to kohl, blood lead concentrations were significantly higher in infants whose mothers used kohl than in those whose mothers did not (5.2 vs 2.8 μg/dl) (*17*).

Family members of workers exposed to lead at their workplace are believed to be at additional risk of elevated blood lead concentrations. For example, blood lead concentrations of up to 280 μg/dl were reported in workers at a lead-smelting plant in the USA (*7*) and concentrations of 28–70 μg/dl were found among families living next to a battery factory in Bombay, India (*18*).

In 1989, Manser et al. reported mean blood lead concentrations of 38 μg/dl among children at the Karachi Grammar School and 38.2 μg/dl among children at a school in a less congested area of the city (*19*). In contrast, a study conducted in Bombay, India, in 1991, reported a mean blood lead concentration in children of 11.3 μg/dl (*20*). These differences and the similarities between Karachi and Bombay in terms of concentrations of lead in the air derived from petrol, sociocultural status and socioeconomic status raise a few research issues, including the need to reassess blood lead concentrations in children from Karachi and the need to identify factors associated with high concentrations. This study tackles these research issues and considers their importance in improving the health of the public in Karachi — the most populated city in Pakistan.

Methods

During August–December 2000, a cross-sectional study was conducted through a geographically stratified design in five diverse communities of Karachi: the city centre (Sadar), two suburbs (Malir and North Karachi), a rural community 50 km away from Karachi (Gadap) and a fishing community located within the harbour in Karachi (Baba Island). The sites were chosen on the basis of several factors, including distance from the city centre, wind direction and area of residence (rural or urban).

In the city centre, we started from the main intersection — Empress Market — and moved in all four cardinal directions. An apartment was randomly chosen from every fifth building (a one-in-five systematic sample). The criteria for inclusion of a child in the study needed the parents' consent to recruit the eldest child aged 36–60 months in the household who had lived in the same house or neighbourhood for at least one year before the study. This age group was chosen as a trade-off between the fact that younger children face greater harm from elevated blood lead concentrations and the fact that families in Karachi were reluctant to consent to blood samples being taken from very young children. We excluded children who had a history of any chronic or congenital disease. A similar sampling strategy was used for the two suburbs and Baba Island. In Gadap, however, four villages were randomly chosen from the area and all households with at least one child who met the selection criteria were included in the sample.

Blood lead concentration was the main outcome variable. Trained interviewers used a pretested questionnaire in the local language (Urdu) to collect information on demographic and socioeconomic characteristics, household behaviour in the context of potential lead exposures, distance of the house from the main intersection and direction in which the house opened. Cooked food, drinking-water and house dust samples were collected from each household.

Sample size

The sample size calculation was intended to detect a mean difference in blood lead concentrations of 3 μg/dl between exposed and non-exposed groups at the 5% level of significance, with at least 80% power. We assumed equal sample sizes in the exposed and non-exposed groups, and we calculated that a minimum of 175 children was needed for each group. We considered the need for an additional 22.5% to take into account refusals and potential losses, so we targeted a total sample of 430 children, of which 400 were analysed.

Laboratory analysis

We used lead-free syringes to collect blood samples into heparinized, lead-free, vacuum, blood collection tubes, which were then transported in iceboxes to the Pakistan Council for Scientific and Industrial Research (PCSIR) laboratory for lead assessment. To standardize the assessment procedure, CDC validated 20 adult blood samples at both the PCSIR and CDC laboratories. After a correlation of 0.98 between the two laboratory results was obtained, the lead assessment of the 400 blood samples started. We followed the graphite furnace atomic absorption spectrophotometer procedure, as described by Flajnik & Shrader, to assess lead levels (*21*). Cooked food, drinking-water and dust samples from the houses of children with blood lead concentrations above the third quartile and below the first quartile were analysed with standard procedures.

Data management

Data were double entered into EpiInfo software, and 10% of the forms were checked randomly to ensure that the error rate for data entry was <0.3%, with a 95% level of confidence.

Statistical analysis

Analysis of variance (ANOVA) was used to compare the mean concentrations of lead in blood, cooked food, drinking-water and house dust for children from the five sites. Two independent sample *t*-tests were used to identify factors associated with elevated blood lead concentrations. When the assumptions of the *t*-test were not met, its nonparametric counterpart — the Mann–Whitney test — was used. Finally, we used a multiple linear regression model to investigate the simultaneous and independent effects of various exposures on the lead concentrations in blood.

Ethical issues

Parents or guardians provided informed consent. The protocol for the study was reviewed and approved by the Aga Khan University Human Subjects Protection Committee.

Results

The overall response rate was 93%. About 85% of respondents were mothers, 10% fathers and the remaining 5% guardians. The median age of the children was 54 months. The level of literacy of mothers was generally lower than that of fathers at all five sites. The socioeconomic and demographic characteristics of the households by sites are shown in Table 1.

Overall, 80.5% of the children had blood lead concentrations >10 μg/dl. The overall mean blood lead concentration was 15.6 μg/dl (median 13.94 μg/dl). When the five sites were compared with ANOVA, significant differences were found in the mean concentrations of lead in blood ($P<0.0005$), cooked food ($P<0.05$) and house dust ($P<0.05$); however, ANOVA did not find any significant differences in lead concentrations in drinking-water from the five sites ($P=0.73$). Fisher's least significant difference procedure showed that the mean blood lead concentration in children from Baba Island (21.60 μg/dl) was significantly higher than that for children from the other four sites ($P\leqslant 0.002$). Baba Island also had the highest mean concentration of lead in cooked food (3.90 μg/g) and the second highest mean concentration of lead in house dust (91.30 μg/g) (the highest mean concentration in house dust (101.76 μg/g) was found in samples from Sadar). Area-specific mean and median concentrations of lead in blood, cooked food, drinking-water and house dust are shown in Table 2.

Factors associated with elevated blood lead concentrations

Children who were exposed to surma at least twice a week had a significantly higher median blood lead concentration (15.48 μg/dl) than others (12.66 μg/dl) ($P=0.001$). Children with a habit of eating food bought from vendors had a significantly higher median blood lead concentration than children who did not have such a habit (14.90 vs 13.10 μg/dl ($P=0.001$). Furthermore, children who used closed vehicles as their mode of transport had significantly lower median blood lead concentrations than those who used other means of transport, such as motorized rickshaws (10.41 vs 14.02 μg/dl). In the inner city stratum (Sadar), we found a negative correlation between the blood lead concentrations and the distance from the main intersection ($r = -0.26$, $P=0.02$).

Children whose fathers were exposed to lead at their workplace had a median blood lead concentration of 15.19 μg/dl; this was significantly higher than the 12.69 μg/dl found for other children ($P<0.005$). Specifically, children with a household member involved in a battery processing job had a higher median blood lead concentration than children whose household members were not involved in such work (18.17 vs 13.72 μg/dl, $P=0.016$). Children who lived in houses that opened to the west and were thereby exposed to the prevailing wind had a median blood lead concentration of 15.92 μg/dl

Table 1. **Distribution of socioeconomic and demographic characteristics by area of residence**

Variable	Area of residence					Total
	City centre, Sadar	Suburb, North Karachi	Suburb, Malir	Rural community, Gadap	Baba Island	
n	83	107	105	52	53	400
Sex						
Male	52 (63)[a]	57 (53)	60 (57)	21 (40)	23 (43)	212 (53)
Female	31 (37)	50 (47)	45 (43)	31 (60)	30 (57)	188 (47)
Age						
⩽48 months	39 (47)	40 (37)	43 (41)	24 (46)	23 (43)	168 (42)
>48 months	44 (53)	67 (63)	62 (59)	28 (54)	30 (57)	232 (58)
Father's educational status						
Illiterate	18 (22)	19 (18)	24 (23)	17 (33)	39 (74)	116 (29)
Up to intermediate diploma	51 (61)	67 (63)	63 (60)	31 (59)	14 (26)	228 (57)
Graduate/post graduate	14 (17)	21 (19)	18 (17)	4 (8)	0	56 (14)
Mother's educational status						
Illiterate	18 (22)	18 (17)	35 (33)	31 (60)	43 (81)	144 (36)
Up to intermediate diploma	59 (71)	76 (71)	62 (59)	21 (40)	10 (19)	228 (57)
Graduate/post graduate	6 (7)	13 (12)	8 (8)	0	0	28 (7)
Having refrigerator						
Yes	56 (68)	74 (69)	61 (58)	9 (17)	12 (23)	212 (53)
No	27 (32)	33 (31)	44 (42)	43 (83)	41 (77)	188 (47)
Having TV						
Yes	73 (88)	93 (87)	85 (81)	21 (41)	45 (85)	316 (79)
No	10 (12)	14 (13)	20 (19)	31 (59)	8 (15)	84 (21)
Having washing machine						
Yes	59 (71)	89 (83)	75 (71)	11 (21)	15 (28)	248 (62)
No	24 (29)	18 (17)	30 (29)	41 (79)	38 (72)	152 (38)

[a] Figures in parentheses are percentages.

Table 2. **Concentrations of lead in blood, cooked food, drinking-water and house dust samples by the area of residence**

Variable	Sadar	North Karachi	Malir	Gadap	Baba Island
Lead in blood					
n	83	107	105	52	53
Median (μg/dl)	13.53	13.11	13.41	12.20	20.81
Mean (SD) (μg/dl)	16.46 (15.72)	14.30 (5.30)	14.90 (5.76)	12.00 (4.50)	21.60 (6.36)
Lead in food					
n	43	47	52	23	36
Median (μg/g)	0.88	1.89	1.71	1.314	1.83
Mean (SD) (μg/g)	1.25 (1.33)	2.15 (1.50)	3.16 (3.28)	1.66 (1.40)	3.90 (5.86)
Lead in dust					
n	40[a]	47	52	23	36
Median (μg/g)	80.26	14.16	17.0	7.13	59.41
Mean (SD) (μg/g)	101.76 (96.0)	26.30 (34.70)	32.0 (55.83)	7.87 (2.84)	91.30 (88.40)
Lead in water					
n	43	47	52	23	36
Median (μg/l)	2.27	3.03	2.24	2.61	2.63
Mean (SD) (μg/l)	4.32 (8.19)	4.20 (5.17)	3.08 (3.28)	3.13 (1.85)	4.02 (5.23)

[a] Three outlier measurements from the dust samples were excluded in the calculation of the mean.

compared with 13.27 μg/dl for those children living in houses opening in other directions (P=0.008). It is important to note that there was an inverse association between the level of education for both parents and the blood lead concentrations in children. Factors associated with elevated blood lead concentrations are presented in Table 3.

A multivariate model was used to identify significant factors that controlled for the effect of other exposure variables. Adjusted P-values for factors associated with elevated blood lead concentrations are also given in Table 3. Father's education and his exposure to lead at his workplace, the child's habit of eating food from street vendors, the child's hand-to-mouth activity, the use of metal cooking utensils and living in "west-open" houses were identified as independent factors associated with elevated blood lead concentrations in children in Karachi.

Discussion

Overall, 80% of children with elevated blood lead concentrations (>10 μg/dl) were a cause for public health concern. The mean blood lead concentration in children from Sadar was 16.5 μg/dl. An earlier study conducted by Manser et al. more than a decade ago reported a higher mean concentration of lead in blood (38 μg/dl) among older children from two Karachi schools in Sadar. This difference could be due to differences in the methods of sample collection and analysis. We collected samples using lead-free syringes and equipment, and we standardized our laboratory tests with the CDC laboratory; however, Manser et al. used ordinary syringes and did not report on whether they standardized the laboratory methods (*22*).

Petroleum products

Our findings show that children who lived in areas with high levels of traffic congestion in urban Karachi had higher blood lead concentrations than those who lived in a rural community outside Karachi. The inverse association between elevated lead concentrations in blood and distance from the main intersection in Sadar suggest that the problem of leaded petrol in Karachi continues. In 1990, a study in Hungary reported mean blood lead concentrations of 20 μg/dl in children who lived near traffic and main roads(*23*). After lead was eliminated from petrol, a study in 1994 showed that the mean blood lead concentrations had fallen to 6.9 μg/dl. Similarly, the removal of lead from petrol in the United States resulted in a decrease of 37% in the mean blood lead concentrations between 1976 and 1980 (*24*). The present study shows that leaded petrol in Pakistan is directly harming children's health and that action should be taken by the petroleum industry in Pakistan.

Baba Island — a fishing community — had the highest blood lead concentrations in children. The seawater in the harbour is contaminated due to the presence of commercial and navy ships and numerous other boats in the area. The leaded petroleum products in the water contaminate not only the fish but also the soil in this area. Baba Island had the highest mean concentrations of lead in cooked food and the second highest in house dust — only 10% less than that in Sadar. This suggests that lead is present in fish — the main item in the daily diet of the residents in Baba Island. Studies from Ahmadabad, India, and West Bengal have also reported elevated blood lead concentrations in children for whom fish was a large part of their daily food content (*25*). Other possible factors include the use of lead-based paints for boats and houses, but a lack of such data does not allow us to further comment on the mechanism by which lead may have found its way into the children's blood in Baba Island.

Surma

Surma is available as a fine powder or heavy crystals of lead sulfide; the colour varies from shiny deep black to dull grey brown. An analytical study found that the concentration of lead in different types of surma available in Pakistan ranged from 0.03% to 81.37% (*26*). Eye rubbing and finger licking could further enhance the absorption of lead, causing elevated blood lead concentrations in surma-exposed children. In our study, children who were exposed to surma at least twice a week had median concentrations of lead about 3 μg/dl higher than those in other children. As shown in Table 3, however, when we adjusted for other variables in the multivariate model, the effect of surma was not statistically significant. Nevertheless, in

Table 3. **Factors associated with elevated blood lead concentrations in children in Karachi, Pakistan (n = 400)**

Exposure variable	Exposed[a]		Non-exposed[b]		P-value[c]	Adjusted P-value
	Median (µg/dl)	Percentage of children exposed	Median (µg/dl)	Percentage of children non-exposed		
Application of surma to eyes[d]	15.48	52	12.66	48	0.001	NS[e]
Father's exposure to lead at work	15.19	40	12.69	60	0.004	0.06
Family involved in battery processing	18.17	2	13.72	98	0.016	NS
Father's education (⩽ 10th grade)	14.83	75	12.68	25	0.003	0.06
Mother's education (⩽ 10th grade)	14.35	85	12.56	15	0.006	NS
Habit of putting things in mouth	16.50	13	13.61	87	0.001	0.03
Child eats things from vendors	14.90	69	13.10	31	0.001	0.06
Going to school by closed vehicle	10.41	4	14.02	96	0.007	NS
Cooking food in aluminium utensils	14.00	89	12.23	11	0.027	0.08
Cleaning of the house more than twice a day	15.50	31	13.45	69	0.036	NS
House opens to the west	15.92	32	13.27	68	0.008	0.002

[a] High exposure to lead at workplace (such as painting, car mechanic, driver, etc.).
[b] Low exposure to lead at workplace (teacher, officers, businessman, etc.).
[c] P calculated using nonparametric test (Mann-Whitney).
[d] Exposure to surma indicates application at least twice a week.
[e] NS means ''not statistically significant at 5% level''.

a study conducted in California among children of Pakistani/Indian origin, the average blood lead concentration in children who used eye cosmetics was 12.9 µg/dl — significantly higher than that in children who did not use such cosmetics (4.3 µg/dl) (*15*). Given that more than half of the children in our study were exposed to surma, we believe that educating mothers about the potential hazards of leaded surma could discourage application of surma to children's eyes. It is important to note that most mothers who applied surma to their children (54%) did not have any formal education, and this might mean that alternative strategies, including removal of lead from surma at production, are needed.

Occupational exposure

Exposure of family members to lead at their workplace was also associated with an increase in blood lead concentrations among their children. Jobs classified as having a risk of exposure to lead include those involved in battery processing, painting, car repairs, ship repairs, work in cement factories, plastic manufacturing, radiator repairs and soldering (*6*, *27–29*). A study in Alexandria, Egypt, reported a higher risk of lead toxicity among children working in battery workshops (*30*). A study from India reported blood lead concentrations of 30–69 µg/dl among those who worked as car mechanics; another study conducted in a ship repair yard reported a median concentration of 26 µg/dl among painters, fitters and other ship repair workers (*31*, *32*). These findings highlight the need for policy development and the implementation of stricter health and safety measures at such workplaces.

Contaminated dust

Children who lived in "west-open" houses had a higher median blood lead concentration than those who lived in houses with openings in other directions. Based on the information provided by the meteorological department of Karachi, the wind blows from the west for approximately 70–80% of the time. West-open houses in Karachi are popular because they receive cool winds. In our study, nearly half of children's room windows were kept open throughout the day. It is likely that the high levels of lead in petrol contaminate the dust that is blown into homes and the lead is subsequently ingested by children. Furthermore, children who had the habit of eating food from street vendors had higher blood lead concentrations than those who did not exhibit such habits (*33*). A likely explanation for this is potential contamination of food by street dust, which increases the likelihood of elevated blood lead concentrations in children.

Study limitations

The sample size of 400 was powered to identify factors associated with elevated blood lead concentrations in children in Karachi. The limited number of children from each stratum did not provide enough power to identify the area-specific factors associated with elevated blood lead concentrations. As a result of our limited funds, we only analysed cooked food, drinking-water and dust samples from children with blood lead concentrations above the third quartile and below the first quartile.

Conclusions

We believe that there is already enough evidence for concern about the continuing problem of lead in petrol, and that action should be taken by the petroleum industry. In addition, our results highlight the need for stricter food and health safety regulations for street vendors and manufacturers of household appliances and other items that deal with lead. The population of Karachi is largely unaware of the hazards and health consequences of lead exposure, and they therefore lack prevention strategies. ■

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Conflicts of interest: none declared.

Résumé

Facteurs associés à une plombémie élevée chez les enfants de Karachi (Pakistan)

Objectif Confirmer si la plombémie est aussi élevée à Karachi aujourd'hui qu'en 1989 et déterminer quels sont les types d'exposition au plomb qui jouent le plus grand rôle dans cette plombémie élevée relevée chez les enfants de Karachi.

Méthodes Au total, 430 enfants âgés de 36 à 60 mois ont été choisis selon un plan géographiquement stratifié incluant le centre ville, deux banlieues, une communauté rurale et une île située dans la rade de Karachi. Des prélèvements de sang ont été effectués chez les enfants et un questionnaire testé au préalable a été distribué afin d'évaluer l'effet des différents types d'exposition. Des aliments cuits, de l'eau de boisson et des échantillons de poussière de maison ont été recueillis dans les ménages.

Résultats Près de 80 % des enfants avaient une plombémie >10 µg/dl, avec une moyenne générale de 15,6 µg/dl. Au seuil de signification de 5 %, les maisons proches de la principale intersection du centre ville, l'application de surma sur les yeux des enfants, l'exposition du père au plomb sur son lieu de travail, l'illettrisme des parents et l'habitude de l'enfant de porter des choses à sa bouche figuraient parmi les variables associées à une forte plombémie.

Conclusion Ces résultats sont une source de préoccupation pour la santé publique, puisque la plupart des enfants à Karachi risquent de présenter un certain déficit intellectuel par suite d'une exposition environnementale au plomb. Nous pensons qu'il y a suffisamment de preuves du problème persistant posé par le plomb présent dans l'essence pour inciter l'industrie pétrolière à prendre des mesures. Les données montrent également la nécessité de disposer d'interventions appropriées pour réduire le poids d'autres facteurs associés à cet élément toxique.

Resumen

Factores asociados al aumento de las concentraciones sanguíneas de plomo entre los niños de Karachi (Pakistán)

Objetivo Confirmar si las concentraciones sanguíneas de plomo en Karachi eran tan altas como las notificadas en 1989, e identificar el tipo de exposiciones al plomo que más contribuyen a aumentar esas concentraciones entre los niños de Karachi.

Métodos Se seleccionó en total a 430 niños de 36–60 meses mediante estratificación geográfica a partir del centro de la ciudad, dos barrios residenciales, una comunidad rural y una isla del interior del puerto de Karachi. Se extrajeron muestras de sangre de los niños y se empleó un cuestionario preensayado para evaluar el efecto de diversos tipos de exposición. En los hogares se tomaron muestras de alimentos cocinados, agua de bebida y polvo.

Resultados Aproximadamente un 80% de los niños presentaban concentraciones sanguíneas de plomo >10 µg/dl, con una media global de 15,6 µg/dl. A un nivel de significación del 5%, se identificaron como variables asociadas al aumento de las concentraciones sanguíneas de plomo la proximidad del hogar al cruce principal del centro de la ciudad, la aplicación de surma en los ojos de los niños, la exposición del padre a plomo en el lugar de trabajo, el analfabetismo de los progenitores y el hábito del niño de llevarse las manos a la boca.

Conclusión Estos resultados son preocupantes para la salud pública, pues significan que la mayoría de los niños de Karachi sufrirán probablemente algún grado de deterioro intelectual de resultas de la exposición al plomo ambiental. Consideramos que hay pruebas suficientes de que el plomo de la gasolina sigue constituyendo un problema que debería inducir a la industria petrolera a tomar algún tipo de medidas. La evidencia acumulada muestra asimismo la necesidad de emprender intervenciones apropiadas para reducir la carga atribuible a otros factores asociados a ese elemento tóxico.

References

1. John H, Cheryl H, Richard S, Christine S. *Toxics A to Z — a guide to everyday pollution hazards*. Berkeley: University of California Press; 1991. p. 47-104.
2. Manser WW, Khan MA, Hasan Z. Trace element studies in Karachi populations, Part III: blood copper, zinc, magnesium and lead levels in psychiatric patient with disturbed behavior. *Journal of the Pakistan Medical Association* 1989;39:235-8.
3. Lanphear BP, Burgoon DA, Rust SW, Eberly S, Galke W. Environmental exposures to lead and urban children's blood lead levels. *Environmental Research* 1998;76:120-30.
4. Brown MJ, Hu H, Gonzales-Cossio T, Peterson KE, Sanin LH, de Luz Kageyama M, et al. Determinants of bone and blood lead concentrations in the early postpartum period. *Occupational and Environmental Medicine* 2000;57:535-41.
5. Momeshora C, Osibanjo O, Ajayi SO. Pollution studies on Nigerian rivers. Toxic heavy metals status on surface waters in Ibadan city. *Environmental International* 1981;5:49-53.
6. Gloag D. Sources of lead. *British Medical Journal* 1981;281:41-4.
7. Von Schirnding Y, Bradshaw D, Fuggle R, Stokol M. Blood lead levels in South African inner-city children. *Environmental Health Perspectives* 1991;94:125-30.
8. Manser WWT. Lead: review of the recent literature. *Journal of the Pakistan Medical Association* 1989;39:296-302.
9. Lacasana M, Romieu I, Sanin LH, Palazuelos E, Hernandez-Avila M. Blood lead levels and calcium intake in Mexico City children under five years of age. *International Journal of Environmental Health* Research 2000;10:331-40.
10. Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environmental health perspectives* 1998;106 Suppl 1:217-29.
11. Hinton D, Coope PA, Malpress WA, Janus ED. Trends in blood lead levels in Christchurch (NZ) and environs 1978-1985. *Journal of Epidemiology and Community Health* 1986;40:244-8.

12. Sherlock JC, Quinn MJ. Relationship between blood lead concentration and dietary lead intake in infants: the Glasgow Duplicate Diet Study 1979-1980. *Food Additives and Contaminants*, 1986;3:167-76.
13. Health Canada. *Guidelines for Canadian drinking water quality — supporting documents. Lead.* Ottawa: Health Canada, 1992.
14. Centers for Disease Control. *Preventing lead poisoning in young children — United States*. Atlanta, GA: Department of Health and Human Services, 1991.
15. Sprinkle RV. Leaded eye cosmetics: a cultural cause of elevated lead levels in children. *Journal of Family Practice* 1995;40:358-62.
16. Ali S, Iqbal M, Yaqub M. Surma — a toxic cosmetic? *Journal of the Pakistan Medical Association* 1988;38:281-2.
17. Nir A, Tamir A, Zelnik N, Iancu TC. Is eye cosmetic a source of lead poisoning? *Israel Journal of Medical Science* 1992;28:417-21.
18. Shenoi RP, Khandekar RN, Jaykar AV, Raghunath R. Sources of lead exposure in urban slum school children. *Indian Pediatrics* 1991;28:1021-7.
19. Manser WW, Lalani R, Haider S, Khan MA. Trace element studies on Karachi Population, Part V: Blood lead levels in normal healthy adults and grammar school children. *Journal of the Pakistan Medical Association* 1990;40:150-4.
20. Alliance to End Childhood Lead Poisoning and Environmental Defense Fund. *The global dimensions of lead poisoning: an initial analysis*. Washington: Alliance to End Childhood Lead Poisoning and Environmental Defense Fund, 1994:43-60.
21. Flajnik C, Shrader D. Determination of lead in blood by GFAAS-Deuterium and Zeeman Background Correction. *American Clinical Laboratory* 1994; 13:45-7.
22. Manser WW, Khan MA. Trace element studies on Karachi Populations, Part I: Normal ranges for blood copper, zinc and magnesium for adults. *Journal of the Pakistan Medical Association* 1989;39:43-9.
23. Bitto A, Horvath A, Sarkany E. Monitoring of blood lead levels in Hungary. *Central European Journal of Public Health* 1997;5:75-8.
24. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. *New England Journal of Medicine* 1983;308:1373-7.
25. Pandya CB, Patel TS, Parikh DJ, Chatterjee SK, Ramanathan NL. Environmental lead exposure as health problem in India: An overview. *Journal of Environmental Biology*, 1983;4:127-48.
26. Haq I, Khan C. Hazard of a traditional eye-cosmetic — surma. *Journal of the Pakistan Medical Association*, 1982;1:7-8.
27. Grandjean P. Occupational lead exposure in Denmark: Screening with the Haematofluorometer. *British Journal of Industrial Medicine* 1979;36:52-8.
28. Corzo G, Naveda R. Occupational exposure to lead in production units in Maracaibo, Venezuela. *Investigative Clinical* 1998;39:163-73.
29. Ankrah NA, Kamiya Y, Appiah OR, Akyeampon YA, Addae MM. Lead levels and related biochemical findings occurring in Ghanaian subjects occupationally exposed to lead. *Eastern African Medical Journal* 1996;73:375-9.
30. Zaki A, el Shazly M, Abdel Fattah M, el Said K, Curtale F. Lead toxicity among working children and adolescent in Alexandria, Egypt. *Eastern Mediterranean Health Journal* 1998;4:520-9.
31. Kumar BD, Krishnaswamy K. Detection of occupational lead nephropathy using early renal markers. *Journal of toxicology. Clinical Toxicology*, 1995; 33:331-5.
32. Grandjean P and Kon SH. Lead exposure of welders and bystanders in a ship repair yard. *American Journal of Industrial Medicine* 1981;2:65-70.
33. Auermann E and Bortitz S. The contamination of vegetable foods by lead containing street dust. *Nahrung* 1997;21:793-7.