LEAD CONCENTRATIONS IN THE BLOOD OF CHILDREN FROM POTTERY-MAKING FAMILIES EXPOSED TO LEAD SALTS IN A MEXICAN VILLAGE[1]

Gilberto Molina-Ballesteros,[2] Miguel A. Zúñiga-Charles,[3] Adolfo Cárdenas Ortega,[4] Pedro Solís-Cámara R.,[5] and Pedro Solís-Cámara V.[6]

Pottery-making, a trade that involves significant exposure to lead salts, is a major household industry in many parts of Mexico. To study potential lead poisoning, the authors examined blood lead levels and other indicators of lead intoxication in a group of 153 Mexican children whose families made pottery in home workshops. This article reports the results of that study.

Introduction

Lead poisoning has been known since at least the fifteenth century, when a European law prohibited the preservation of wine with lead or lead salts. However, the problem has probably been around much longer, presumably since the time lead was discovered and used for various purposes by the Egyptians around 1500 BC.

Present-day exposure to lead occurs in many ways—during the smelting and refining process; through contact with leaded gasoline, engine fumes, storage batteries, fumes from burning paint, and so forth; and ingestion in various ways, in the form of lead paint or lead dissolved in water exposed to lead pipes *(1-7)*. There are, of course, many other ways lead can enter the body, one of particular concern to us being in the form of lead salts taken in during the preparation of glazed pottery or during the use of badly fired glazed pottery *(8, 9)*. Pottery-makers in Mexico are commonly exposed to lead in these ways *(10, 11)*.

Large-scale programs in the United States have detected increased lead absorption or lead poisoning in substantial numbers of children. In 1978, for example, 25,000 children out of 400,000 examined were found to have abnormally high levels of the metal in their bodies *(12)*. It is true that these data contrast with those from the United Kingdom, where only about 100 cases of lead poisoning per year are detected *(13)*. However, this low detection rate is probably due to the fact that no appropriate detection programs have been conducted. The situation in other countries, including Mexico, is uncertain.

Lead poisoning can be either chronic or acute, the chronic form being more common. According to the statistics of the Mexican Social Security Institute, chronic lead poisoning, known as saturnism, is the third most common occupational disease in Mexico, accounting for 12 per cent of all recorded occupational disease cases *(11)*. The insidious nature of the disease and the absence of distinctive symptoms frequently hinder diagnosis. However, a blood lead concentration of 40 μg/100 ml is considered normal for the general population. For occupationally exposed

[1]Also appearing in Spanish in the *Boletín de la Oficina Sanitaria Panamericana* 92(1):33-39, 1982.

[2]Chief of the Research Services Subdivision, Northeastern Biomedical Research Unit, Mexican Social Security Institute (IMSS), Apartado Postal 020-E, Monterrey, Nuevo León, Mexico.

[3]Investigator, Northeastern Biomedical Research Unit, IMSS.

[4]Chief of the Clinical Analysis Laboratory, Western Biomedical Research Unit, IMSS.

[5]Psychologist, Western Biomedical Research Unit, IMSS.

[6]Director, Western Biomedical Research Unit, IMSS.

adults, 40 to 80 μg/100 ml is generally considered acceptable, 80-120 μg/100 ml is considered excessive, and over 120 μg/100 ml is considered dangerous. The foregoing levels do not hold for children, who can be visibly affected at much lower concentrations *(14, 15)*.

Poisoning due to organic lead salts produces similar symptoms in children and adults. The commonest symptoms are abdominal pain, asthenia, constipation, diarrhea, paresthesia, psychological alterations, and vomiting. Other frequent symptoms include changes in sleep patterns, anorexia, dyspepsia, nausea, headaches, dizziness, muscular weakness, trembling, joint pains, weight loss, and a metallic taste in the mouth. The commonest signs are lack of muscular coordination, atrophy of the most used muscles, parotiditis, visual disorders, ulcerative stomatitis, blue gum, gray patches on the gingival mucosa, trembling of the extended tongue, and furred tongue *(16)*.

Inorganic lead salts produce different symptoms in children—including confusion, irritability, vomiting, gastrointestinal symptoms, ataxia, stupor, and fatigue *(16)*. It has also been demonstrated that lead causes poor intellectual performance and various behavioral changes at relatively low concentrations in the blood *(17)*.

Pottery-making is very widespread in Mexico, which is the world's fifth-largest pottery producer. This trade is still practiced throughout the country by thousands of families at small workshops installed in their own homes. These home workshops are scattered through the states of Puebla, Hidalgo, Guanajuato, México State, Morelos, Oaxaca, Michoacán, and Jalisco. The vast majority have primitive low-temperature kilns and use a lead oxide-silica oxide mixture for glazing. Because they are unregistered, there is no easy way to accurately ascertain their number *(11)*.

The place selected for the work reported here, a town named Tonalá in the state of Jalisco, had a population of approximately 35,054 inhabitants in 1980 according to the census of that year, of which some 423 were pottery workers. The fact that the pottery-makers with home workshops were fairly well concentrated in certain areas made it feasible to study the health effects of this handicraft by comparing children belonging to pottery-making families with home workshops to children from other Tonalá families.

Previous study of Tonalá pottery-makers had shown the blood lead levels of selected adult pottery workers to be abnormally high *(10)*. In addition, some children in pottery-making families who could have been exposed to lead salts were found to have high blood lead concentrations. It was therefore deemed advisable to make a study of children belonging to pottery-making Tonalá families. This article presents the results of that study.

Materials and Methods

The study included 153 children belonging to pottery-making families with home workshops; it also included 80 children belonging to other families that did not appear to be exposed to the metal through their occupations. These latter children, selected by simple random sampling from three primary schools in the vicinity, were used as a control group. The children from pottery-making families were identified by data obtained from the previous study *(10)*, by information that schools provided, and by comments received from the families involved regarding other families with home workshops. All such children identified who were between 5 and 15 years of age were included in the study. The children in the control and pottery-family groups, each of which included both girls and boys, were not matched by sex or age.

Following authorization by each child's parents and the school authorities, clinical histories were drawn up for the purpose of identifying pathologies characteristic of lead poisoning. In addition, 10 ml specimens of peripheral venous blood were obtained with disposable syringes and placed in heparinized test tubes;

and urine specimens (from a single urination by each child) were collected. Blood lead concentrations were then measured by atomic absorption spectroscopy *(18)*; blood protoporphyrin IX levels were measured with a hematofluorometer (Hemafluor ZP, 4,000 Model) *(19)*; hemoglobin levels and hematocrits were determined by the usual methods; and levels of urinary delta-aminolevulinic acid were measured according to the modified method of Davis and Andelman *(20)*.

From a clinical point of view, the most important direct tests for diagnosing bodily absorption of lead or lead poisoning are those determining lead levels in the blood and urine. And even though the precise diagnostic value of blood lead determinations is being questioned *(21-24)*, blood lead tests are still considered the tests of choice.

Of the various methods available for direct determination of blood lead levels, the most commonly used are atomic absorption methods such as that employed in this study. These latter methods have a number of advantages, among them the ability to make many determinations quickly.

The various indirect methods used depend on the inhibiting effect lead has upon certain enzymes active in the biosynthesis of heme, the Fe-protoporphyrin that comprises the protein-free part of the hemoglobin molecule *(25)*. One of these methods, measurement of zinc protoporphyrin in erythrocytes *(26)*, involves determination of fluorescence with a hematofluorometer. The test, which we chose for our study, employs no special reagents, is both quick and economical, and requires only a single drop of blood. Another indirect method that we used was to determine the level of delta-aminolevulinic acid in the urine *(20)*. Besides being considered very useful *(27, 28)*, this ion-exchange chromatography test can be performed in a reasonably expeditious manner.

Because of lead's adverse impact on heme biosynthesis, hemoglobin and hematocrit determinations were performed to provide complementary information.

Results

The results obtained by testing blood and urine specimens from the children belonging to pottery-making families and the control group are shown in Table 1. It should be noted that highly significant differences were observed between the two groups with regard to levels of blood lead, erythrocytic zinc protoporphyrin, and urinary delta-aminolevulinic acid. In this same vein, the top levels of blood lead (98 μg %) and zinc protoporphyrin (225

Table 1. Test results obtained with all the study children of both sexes, expressed in terms of the average result (± 1 standard deviation) and the range of results recorded. The statistical significance of the difference between figures for exposed children (from pottery-making families) and those for control children is shown at the far right.

	Exposed children (both sexes)			Control children (both sexes)			
	(No. of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	(No of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	Statistical significance of difference
Age in years	(153)	9.9 ± 2.3 years	5-15 years	(80)	10.6 ± 2.7 years	5-15 years	$p<0.05$
Blood lead (μg %)	(153)	39.5 ± 19.6 μg %	12-98 μg %	(80)	24.8 ± 7.7 μg %	9-39 μg %	$p<0.001$
Delta-aminolevulinic acid (mg/l)	(150)	3.2 ± 4.1 mg/l	0-27 mg/l	(79)	1.7 ± 1.2 mg/l	0 3-7.1 mg/l	$p<0.001$
Erythrocytic zinc protoporphyrin (μg %)	(152)	42.3 ± 47.9 μg %	1-225 μg %	(80)	14.9 ± 13.9 μg %	1-83 μg %	$p<0.001$
Hemoglobin (g %)	(140)	13.3 ± 1.1 g %	10.8-16.8 g %	(70)	14.0 ± 1.1 g %	11.8-17.8 g%	$p<0.001$
Hematocrit (% v/v)	(140)	40.8 ± 2.9%	34-49%	(70)	42.3 ± 3.9%	31-54%	$p<0.005$

Table 2. Test results obtained with boys 5-15 years of age, expressed in terms of the average result (± 1 standard deviation) and the range of results recorded.

	Exposed children (boys)			Control children (boys)			
	(No of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	(No of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	Statistical significance of difference
Age in years	(82)	9.69 ± 2.3 years	5-15 years	(27)	11.2 ± 2.3 years	7-15 years	p<0.005
Blood lead (µg %)	(82)	40.6 ± 20.9 µg %	13-98 µg %	(27)	26 2 ± 7 5 µg %	14-38 µg %	p<0.001
Delta-aminolevulinic acid (mg/l)	(82)	3.5 ± 5.2 mg/l	0.4-27 mg/l	(27)	1 7 ± 0.9 mg/l	0.3-3.5 mg/l	p<0.01
Erythrocytic zinc protoporphyrin (µg %)	(82)	40 5 ± 47 4 µg %	1-225 µg %	(27)	12.0 ± 10 1 µg %	1-36 µg %	p<0.001
Hemoglobin (g %)	(82)	13.3 ± 1.0 g %	10.8-16.8 g %	(27)	14.2 ± 1 4 g %	12.3-17 8 g%	p<0.001
Hematocrit (% v/v)	(82)	40.7 ± 2.9%	34-49%	(27)	43.9 ± 3.9%	37-54%	p<0.001

µg %) observed in individual blood specimens from exposed children were quite high. No significant differences were found between the data for boys (Table 2) and girls (Table 3).

Table 4 subdivides the exposed children (from pottery-making families) and control children into four groups whose blood lead concentrations were in the ranges of 0-20, 21-40, 41-60, and over 60 µg %. As this division shows, considerable numbers of exposed and control children were found to have blood lead concentrations in the ranges of 0-20 and 21-40 µg %. However, none of the control children had blood lead concentrations above 40 µg %, considered the upper "normal" limit, while 41 per cent of the exposed girls and 43 per cent of the exposed boys had concentrations above that figure.

Discussion

Lead is an ubiquitous element with many domestic and industrial applications. Children are invariably exposed to the metal, small quantities of which are present in ordinary food, air, water, dust, and so forth; but they may also experience marked exposure to lead from specific and often unsuspected sources *(1-7)*.

Childhood lead poisoning is not diagnosed with notably greater frequency in Mexico than elsewhere; and so the high levels of lead, zinc protoporphyrin, and delta-aminolevulinic acid found in the blood and urine of Tonalá children from pottery-making families points to considerable lead exposure in their home environments. These findings also again confirm what has been known for centuries: that many population groups dedicated to making ceramic products run a high risk of contracting chronic lead poisoning.

Although the number of Tonalá children 5 to 15 years old who belonged to pottery-making families was about 449 at the time of our investigation, the 153 "exposed" children studied appeared to include nearly all those from families with home workshops. The rest of the pottery-connected children appeared to come from families that worked in small but relatively modern industrial shops separated from their households, and so these children did not seem exposed to the same risk of chronic lead poisoning as the study children.

Our opinion, in the light of the available data, is that the study children's exposure is related to the primitive pottery-making methods used by their families and the level of lead contamination that exists in the family workshops, since these workplaces are within the physical area of their households.

In most cases, the pottery kilns are in the patios of the children's homes; and the glazing process, which involves wetting pots fired for the first time with lead-containing litharge, is

Table 3. Test results obtained with girls 5-15 years of age, expressed in terms of the average result (± 1 standard deviation) and the range of results recorded.

	Exposed children (girls)			Control children (girls)			
	(No. of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	(No of children)	Average age or test result ± 1 standard deviation	Range of ages and test results	Statistical significance of difference
Age in years	(71)	10.1 ± 2.3 years	6-15 years	(53)	10.2 ± 2.9 years	6-15 years	Not significant
Blood lead (μg %)	(71)	38.2 ± 18.1 μg %	16-80 μg %	(53)	24.0 ± 7.7 μg %	9-39 μg %	$p<0.001$
Delta-aminolevulinic acid (mg/l)	(68)	2.7 ± 2.2 mg/l	0-8.2 mg/l	(52)	1.7 ± 1.4 mg/l	0.3-7.1 mg/l	$p<0.01$
Erythrocytic zinc protoporphyrin (μg %)	(70)	44.4 ± 48.7 μg %	1-186 μg %	(53)	15.8 ± 15.4 μg %	1-83 μg %	$p<0.001$
Hemoglobin (g %)	(58)	13.4 ± 1.1 g %	10.8-16.2 g %	(43)	13.8 ± 1.0 g %	11.8-16.2 g%	Not significant
Hematocrit (% v/v)	(58)	40.8 ± 2.9%	35-49%	(43)	41.4 ± 3.6%	31-48%	Not significant

Table 4. Percentages of study children with different blood lead concentrations, by sex.

Study children		Blood lead concentrations												
		0-20 μg %			21-40 μg %			41-60 μg %			>60 μg %			
Sex	Group	No. of children	% of group	Average ± 1 standard deviation	No. of children	% of group	Average ± 1 standard deviation	No. of children	% of group	Average ± 1 standard deviation	No. of children	% of group	Average ± 1 standard deviation	% of children with blood lead concentrations above 40 μg %
Female	Exposed	10	(14)	18 ± 2 μg %	32	(45)	28 ± 5 μg %	19	(27)	49 ± 7 μg %	10	(14)	72 ± 8 μg %	41
	Control	20	(38)	16 ± 3 μg %	33	(62)	29 ± 5 μg %	0	(0)	–	0	(0)	–	0
Male	Exposed	11	(13)	17 ± 3 μg %	36	(44)	29 ± 5 μg %	21	(26)	49 ± 6 μg %	14	(17)	79 ± 9 μg %	43
	Control	7	(26)	17 ± 2 μg %	20	(74)	30 ± 6 μg %	0	(0)	–	0	(0)	–	0

performed weekly. In most of these households, the wet pots bound for the kiln drip on the patio floor while in transit. The result is that small children are exposed to contaminated material. Therefore, it stands to reason that if the potter is trained to carry out the glazing and baking process at a place to which the rest of the family does not have access, this contamination can be reduced. The appropriate alteration of living quarters and workshops that this implies is most advisable. The ideal solution, however, would be to make a technical and economic study of the pottery-making process with the aim of eliminating litharge from that process. This would not only halt the chronic lead intoxication process underway in the child population studied, but would also prevent it in other younger children (113 in the case at hand) below five years of age.

SUMMARY

Pottery-making is a common Mexican handicraft, and thousands of home workshops devoted to this trade are scattered through central and southern Mexico. This entails a risk of chronic lead poisoning through exposure to lead oxide in glazing material, a matter of particular concern because chronic lead poisoning is Mexico's third most common occupational disease.

To investigate such exposure and follow up on earlier studies, a survey was conducted of the children 5 to 15 years of age in a town called Tonalá in Jalisco State. A total of 153 children belonging to pottery-making families with home workshops were examined, as were 80 children from non-pottery-making families who served as a control group.

Blood and urine specimens from these children showed that many of those from the pottery-making families had unusually high levels of lead and zinc protoporphyrin in their blood and high levels of delta-aminolevulinic acid in their urine. These findings indicate considerable lead exposure in the test children's home environments, and demonstrate a need to modify those environments or find a substitute glazing compound that contains no lead. They also confirm again the generally accepted proposition that many population groups dedicated to making ceramic products run a high risk of contracting chronic lead poisoning.

REFERENCES

(1) Baker, E. L., D. S. Folland, T. A. Tylor, M. Frank, W. Paterson, G. Lovejoy, D. Cox, J. Housworth, and P. J. Landrigan. Lead poisoning in children of lead workers. *N. Engl J Med* 296:260, 1977.

(2) Barltrop, D. The prevalence of pica. *Am J Dis Child* 112:116, 1966.

(3) Caprio, R. J., H. L. Margulis, and M. M. Joselow. Lead absorption in children and its relationship to urban traffic densities. *Arch Environ Health* 28:195, 1974.

(4) Landrigan, P. J., E. Baker, R. Feldman, D. Cox, K. Eden, W. Orenstein, J. Mather, A. Yankel, and I. Von Lindern. Increased lead absorption with anemia and slowed nerve conduction in children near a lead smelter. *J Pediatr* 89:904, 1976.

(5) McCord, C. P. Lead and lead poisoning in early America; pewter era. *Indust Med* 22:573-577, 1953.

(6) Moore, M. R., B. Campbell, P. Meredith, A. Beattie, A. Goldberg, and D. Campbell. The association between lead concentrations in teeth and domestic water lead concentrations. *Clin Chim Acta* 87:77, 1978.

(7) Watson, W. N., L. E. Witherell, and G. C. Giguere. Increased lead absorption in children of workers in a lead storage battery plant. *J Occup Med* 20:759, 1978.

(8) Hughes, J. T., J. J. Horan, and C. P. Powles. Lead poisoning caused by glazed pottery: Case report. *N Z Med J* 84:266, 1976.

(9) Koplan, J. P., A. V. Wells, H.J.P. Diggory, E. L. Baker, and J. Liddle. Lead absorption in a

community of potters in Barbados. *Int J Epidemiol* 6:225, 1977.

(10) Molina-Ballesteros, G., M. A. Zúñiga-Charles, J. García de Alba, A. Cárdenas-Ortega, and P. Solis-Cámara. Lead exposure in two pottery handicraft populations. *Arch Invest Med (Mex)* 11: 147, 1980.

(11) Molina-Ballesteros, G., M. A. Zúñiga-Charles, F. J. Sánchez-Anzaldo, and R. Garza-Chapa. Plomo: sus implicaciones sociales y efectos sobre la salud. *Gac Med Mex* 115(2):57-64, 1979.

(12) United States Center for Disease Control. *Morbidity and Mortality Weekly Report* 28(10):117, 1979.

(13). Chisolm, J. J., and D. Barltrop. Recognition and management of children with increased lead absorption. *Arch Dis Child* 54:249, 1979.

(14) United Kingdom, Interdepartmental Working Group on Heavy Metals. *Lead in the Environment and Its Significance to Man.* Pollution Paper No. 2. Her Majesty's Stationary Office, London, 1974.

(15) Lansdown, R. Moderately raised blood lead levels in children. *Proc R Soc Lond (Biol)* 205: 145-151, 1979.

(16) Gordon, N. C., S. Brown, V. M. Khosla, and L. S. Hansen. Lead poisoning. *Oral Surg* 47: 500, 1979.

(17) Bryce-Smith, D., J. Mathews, and R. Stephens. Mental health effects of lead on children. *AMBIO* 7(5-6):192-206, 1978.

(18) Hessel, D. W. A simple and rapid quantitative determination of lead in blood. *Atomic Absorption Newsletter* 7:55, 1968.

(19) Blumberg, W. E., J. Eisinger, A. A. Lamola, and D. M. Zuckerman. The hematofluorometer. *Clin Chem* 23:270, 1977.

(20) Davis, J. R., and S. L. Andelman. Urinary delta-aminolevulinic acid (ALA) levels in lead poisoning: I. A modified method for the rapid determination of urinary delta-aminolevulinic acid using disposable ion-exchange chromatography columns. *Arch Environ Health* 15:53, 1967.

(21) Keppler, J. F., M. E. Maxfield, W. D. Moss, G. Tietjen, and A. Linch. Interlaboratory evaluation of the reliability of blood lead analyses. *Am Ind Hyg Assoc J* 31:412, 1970.

(22) Lauwerys, R., J.-P. Buchet, H. Roels, A. Berlin, and J. Smeets. Intercomparison program of lead, mercury, and cadmium analysis in blood, urine, and aqueous solutions. *Clin Chem* 21:551, 1975.

(23) National Institute for Occupational Safety and Health, U.S. Public Health Service, Department of Health, Education, and Welfare. *Criteria for a Recommended Standard: Occupational Exposure to Inorganic Lead.* Washington, D.C., 1972.

(24) Vitale, L. F., M. M. Joselow, R. P. Wedeen, and M. Pawlow. Blood lead: An inadequate measure of occupational exposure. *J Occup Med* 17:155, 1975.

(25) Baloh, R. W. Laboratory diagnosis of increased lead absorption. *Arch Environ Health* 28:198, 1974.

(26) U.S. Center for Disease Control; Department of Health, Education, and Welfare. *Preventing Lead Poisoning in Young Children: A Statement by the Center for Disease Control,* DHEW, PHS, CDC (00-2629). Atlanta, Georgia, 1978.

(27) Urbanowicz, H. Occupational exposure to inorganic compounds of lead: Investigation of delta-aminolevulinic acid and coproporphyrin excretion rates of persons exposed for the first time. *Arch Environ Health* 23:284, 1971.

(28) DeBruin, A., and H. Hollboon. Early signs of lead exposure: A comparative study of laboratory tests. *Br J Ind Med* 24:203, 1967.