

# Prenatal Lead Exposure in Israel: an International Comparison

Yona Amitai MD<sup>1</sup>, Daniel Katz MD<sup>1</sup>, Matityahu Lifshitz MD<sup>3</sup>, Rosa Gofin MD<sup>2</sup>, Maya Tepferberg MSc<sup>4</sup> and Shlomo Almog PhD<sup>4</sup>

<sup>1</sup>Department of Pediatrics, Hadassah University Hospital, Mt. Scopus, and <sup>2</sup>Department of Social Medicine, Hebrew University-Hadassah School of Public Health and Community Medicine, Ein Kerem, Jerusalem; and <sup>3</sup>Department of Pediatrics, Soroka Medical Center, Beer Sheva; and <sup>4</sup>Institute of Toxicology, Sheba Medical Center, Tel-Hashomer, Israel

**Key words:** lead poisoning, fetus, umbilical cord, epidemiology

## Abstract

**Background:** Prenatal lead exposure (umbilical cord blood lead concentration >10 µg/dl) may impair cognitive development. Childhood lead poisoning is infrequent in Israel, and there are no data on lead exposure in immigrants to Israel from the former Soviet Union.

**Objectives:** To evaluate prenatal blood lead concentrations in Israeli newborns whose mothers were born in Israel and in those whose mothers recently immigrated from Russia, and to compare data of prenatal lead exposure in Israel with those reported from other countries.

**Methods:** We compared the UCBLC of 35 newborns of new immigrants from Russia with a group of 35 newborns whose mothers were born in Israel. Venous BLC was also measured in 50 mothers. Data are compared with similar reports on prenatal lead exposure internationally.

**Results:** The UCBLC in all 70 newborns (mean±SD) was 3.53±1.6 µg/dl, and mothers' BLC (mean±SD) was 3.90±1.39 µg/dl. UCBLC and BLC in the 50 mother-newborn pairs correlated ( $r=0.36$ ,  $P<0.01$ ). All newborns except one had UCBLC <8.0 µg/dl. There was no significant difference between UCBLC in the two groups.

**Conclusions:** Prenatal lead exposure among the study subjects in both groups was low. In this sample the newborns of mothers born in Israel and those whose mothers recently immigrated from Russia were not found to be at risk for lead poisoning. Prenatal lead exposure in this sample was low compared to that reported from various parts of the world.

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Lead poisoning, quite frequent in children around the world including the United States, may result in impaired cognitive development, particularly in young children [1]. Since lead also crosses the placenta, it has a deleterious neurological effect on newborns [2-5].

Prenatal lead exposure is assessed by determination of maternal blood lead concentrations at birth as well as by umbilical cord blood lead concentrations. Bellinger et al. [2]

demonstrated a significant impairment in cognitive functions at 1-2 years of age in a cohort of infants with UCBLC >10 µg/dl (mean 14.6), and Deitrich and co-workers [3] observed a reduction in Bayley Mental Developmental Index at the ages of 3 and 6 months in infants with mild elevations of UCBLC. Based on these studies, UCBLC >10 µg/dl is regarded as evidence for significant prenatal lead exposure. Recently de Caceres et al. [5] demonstrated a negative correlation between UCBLC and the Brazelton Neonatal Behavior Assessment, administered as early as 3 days after delivery in newborns with a mean UCBLC of 6.48 µg/dl.

Prenatal lead exposure has been extensively studied in many countries [2-23]. Childhood lead poisoning is not considered a public health problem in Israel because the most dangerous source for lead poisoning in children, namely deteriorated paint with high lead content in old buildings, is uncommon. Thus, lead poisoning has been found in only a limited number of children with exposure to lead from other sources, such as the use of khol as an eye cosmetic in Druze children [24] and lead poisoning from contaminated flour [25].

The recent immigration of approximately 900,000 Jews from the former Soviet Union raises the question of lead exposure in this population. Although there are no reliable data on lead exposure in Russia, lead exposure due to industrial and environmental pollution and to housing conditions in East Europe and Russia is probably worse than in the U.S. [26]. The slow elimination of lead from the body over years [27] suggests that the body burden of lead may still exist in emigrants who were exposed to lead in their country of origin.

In view of this possibility we undertook a study to address the following question: Are babies of mothers who immigrated to Israel from Russia after 1990 particularly at high risk for prenatal lead exposure? Data on prenatal lead exposure in our subjects are compared with data on prenatal lead exposure reported from other countries.

## Patients and Methods

The study took place during the periods January-November 1994 and June-September 1995 in the delivery rooms of the two Hadassah University Hospitals (Mt. Scopus and Ein Kerem), Jerusalem, and at the Soroka Medical Center, Beer

UCBLC = umbilical cord blood lead concentration  
BLC = blood lead concentration

Sheva. The study was approved by the Human Investigation Committee of these institutions, and all participating mothers signed an informed consent.

Prenatal lead exposure was compared between babies of mothers who immigrated to Israel from Russia after January 1990 (Group 1) and a matched group of Israeli-born mothers and their babies (Group 2). This group was formed by selecting the consecutive delivery of a mother who was born in Israel for each mother from Group 1. In both groups premature and multiple deliveries were excluded. The mothers were interviewed for details on maternal health, obstetric history, number of school years, past and present occupation, hobbies, and residential addresses. They were asked about the housing conditions and location, whether the building was old (>15 years), and whether it was located close to a city center. The course of delivery, Apgar scores, birthweights and special neonatal health problems were also recorded.

Umbilical cord blood samples were collected at delivery and venous blood samples were taken from the mothers. Blood samples were collected in heparinized test tubes and kept at 4°C until analyzed. Lead concentrations were analyzed at the Institute of Toxicology at the Sheba Medical Center, Tel-Hashomer, an accredited laboratory for occupational medicine testing of lead in blood. Lead was measured in whole blood by atomic absorption spectrophotometry, in duplicates. Calibration is done with certified standards from MERCK, and the CV of the method is 7%.

### Statistical analysis

The correlation between the maternal BLC and UCBLC in neonate–maternal pairs was studied by means squares regression analysis. Differences between the mean values of demographic data and BLC between groups were analyzed by the paired or unpaired Student's *t* test, as appropriate. The chi-squared test was used to compare differences in proportions of categoric parameters. A *P* value <0.05 was considered significant.

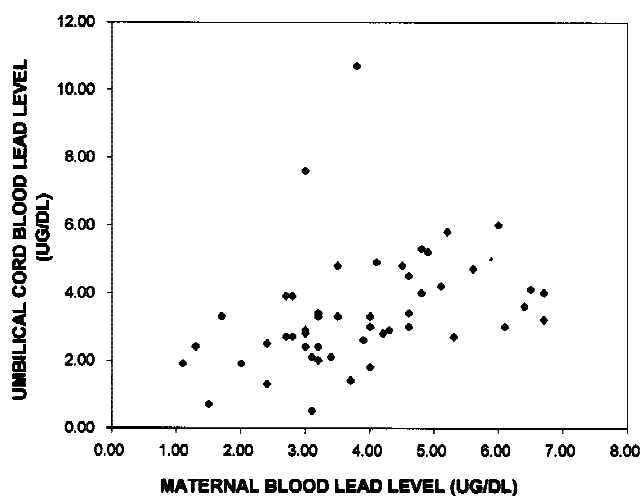
### Results

The study included 184 mothers: 90 emigrants from the former Soviet Union and 94 born in Israel. The interval between immigration to Israel and the study was 1–4 years (mean 3.2 years). All 184 mothers were interviewed, and umbilical cord blood samples were collected from all their newborns. UCBLC was determined in the first 70 newborns (35 in Group 1 and 35 in Group 2). Since the initial evaluation of the results of these samples provided satisfactory answers to the study questions we did not determine lead levels further. Mothers of the babies with or without determination of lead levels were comparable with regard to all the epidemiological parameters (age, number of pregnancies, school years, weeks of gestation, birthweight of the babies, percentage of old houses, and percentage of houses in city centers). Details on occupation and hobbies indicated that none of the mothers had any occupation or hobby that put them at risk for lead exposure.

**Table 1.** Comparison of demographic data and blood lead levels in umbilical cord and in maternal venous blood between babies of new immigrants from the ex-USSR and Israeli-born mothers

	Russia (n=35)	Israel (n=35)	<i>P</i>
Age (yr) (mean±SD)	28.0 (5.4)	27.5 (5.4)	
No. of pregnancies (mean±SD)	2.6 (1.9)	2.6 (1.9)	0.91
Schooling (yr, mean±SD)	10.4 (3.6)	10.0 (3.4)	0.37
% old houses (>15 yr)	16	12	0.69
% houses in city centers	30	24	0.24
Weeks of gestation (mean±SD)	40.0 (1.7)	39.8 (1.6)	0.66
Birthweight (kg, mean±SD)	3.214 (0.4)	3.223 (0.4)	0.89
UCBLC (µg/dl, mean±SD)	3.56 (1.6)	3.50 (1.6)	0.80
Maternal BLC (µg/dl, mean±SD)	3.92 (1.6)	3.88 (1.5)	0.88

### CORRELATION BETWEEN BLOOD LEAD LEVEL IN MOTHER-INFANT PAIRS



**Figure 1.** Relation between maternal blood lead levels and umbilical cord blood lead levels in 50 mother–infant pairs ( $r=0.36$ ,  $P<0.01$  by Pearson's correlation coefficient).

Based on these data, we assume that the 35 immigrant mothers, the 35 Israeli-born mothers, and their babies with UCBLC data are representative of the whole population participating in the study. Table 1 presents the data of the 70 babies (35 of Group 1, and 35 of Group 2) with actual determinations of UCBLC. As shown, mean UCBLCs for Group 1 and 2 are comparable, 3.56 and 3.53 µg/dl, respectively. The range of UCBLC in Groups 1 and 2 was 0.5–7.6 and 1.3–10.7 µg/dl, respectively. Only three babies in each group had UCBLC >6.0 µg/dl. Maternal BLC was also comparable in the two groups, with mean levels of 3.93 and 3.88 µg/dl for Groups 1 and 2, respectively. All BLCs in both groups were <7.0 µg/dl. In the 50 mother–infant pairs (25 mothers in each group) there was a statistically significant correlation ( $r=0.36$ ,  $P<0.01$ ) between maternal BLC and the matched UCBLC [Figure 1]. Data of prenatal lead exposure in our study are compared with those reported from various parts of the world during the last 20 years [Table 2].

**Table 2.** International comparison of umbilical cord blood lead levels during the last 20 years.

Author	Reference	Site & year of study	No. of subjects	Mean UCBLI ( $\mu\text{g}/\text{dl}$ )	Comment
Rothenberg	6	Mexico City 1979–80	50	13.0	High population
Berlinger	2	Boston, USA 1979–82	11,837	6.6	
McMichael	4	Port Pirie, Australia 1979–82	523	8.3	Lead smelter area
Dietrich	3	Cincinnati, USA 1979–84	305	6.3	Inner city, high risk
Zarembski	7	England 1980–81	1,209	4.06	
Ernhart	8	Cleveland, USA 1981–82	178	5.7	High risk
Satin	9	California, USA 1984	723	4.9	
Meyer	10	Braunschweig, Germany 1985–86	9,189	3.76	
Hwang	11	Taipei, Taiwan 1985–87	205	7.48	High population
Graziano	12	Mitrovica, Yugoslavia	602	17.1	Lead smelter area
		Pristina, Yugoslavia	900	5.1	Reference area
Schucard	13	Buffalo, USA 1987–88	802	3.8	
Koren	14	Toronto, Canada 1989	95	1.66	
Saxena	15	Lucknow, India 1990	243	16.5	
Rhains	16	Quebec, Canada 1990	423	1.95	
Lagerkvist	17	Sweden 1989–92	290	3.11	Lead smelter area
			190	2.69	Reference area
Hu	18	Boston, USA 1990	223	1.19	
Plockinger	19	Austria 1991	51	2.63	
Al-Saleh	20	Riad, Saudi Arabia	126	4.14	
Mayan	21	Porto, Portugal 1992	248	8.9	Rural area
			124	12.4	Urban area
de Caceres	5	Barcelona, Spain	30	6.48	
Shen	22	Shanghai, China 1993	348	9.2	
Gonzales-Cassio	23	Mexico City, Mexico 1996	238	7.1	
Present study		Israel 1994–95	70	3.53	

## Discussion

Lifetime environmental exposure to lead results in its accumulation in the body. Since lead freely crosses the placenta, maternal exposure results in fetal exposure as reflected in UCBLI. Indeed, our study showed a significant correlation between maternal BLC and neonatal lead concentrations, indicated by UCBLI.

In our study UCBLI measurements of all newborns with one exception were  $<8.0 \mu\text{g}/\text{dl}$ , indicating that in this sample the babies of mothers born in Israel as well as babies of new immigrants from Russia do not seem to be at risk for prenatal lead exposure.

The international comparison of reports on prenatal lead exposure during the last 20 years [Table 2] shows that the variation between UCBLI in these studies is mainly due to differences in environmental exposure. However, a general trend of a reduction in UCBLI over time is noted, due most likely to a tighter control of lead exposure. Particularly high levels were found in a vicinity of lead smelters in Kosovo, Yugoslavia [12] and Australia [4], and in heavily polluted areas in Mexico City [6,23], Porto, Portugal [21], Taipei [11] and Shanghai [22]. The main sources of lead in those polluted areas are leaded gasoline, lead in deteriorated paint in old residencies, and lead-glazed ceramics (in Mexico) [23].

In the USA, strict measures to control these sources — by massive screening programs in children, residential deleading, and phasing out of lead in gasoline — resulted in

a dramatic decline in lead exposure. Nationwide, the percentage of U.S. children aged 1–5 years with BLC  $>10 \mu\text{g}/\text{dl}$  decreased from 88.2% during 1976–80 to 4.4% during 1991–94! [28].

From this survey it appears that UCBLIs in our sample were lower than those found in the vast majority of other countries. Childhood lead exposure has never been researched on a large scale in Israel. We studied a limited number of patients in three hospitals located in two cities. Only 70 newborns underwent UCBLI analysis, but their demographic data and risk factors for lead exposure were comparable with the other 114 newborns who did not have UCBLI determinations. This allows us to deduct that the results of UCBLI determinations are applicable also to the other newborns in our sample, although not to the general newborn population in Israel. In our study none of the mothers and only a single newborn had UCBLI  $>10 \mu\text{g}/\text{dl}$ . These results suggest that lead exposure is insignificant in this sample. Of note, a recent study of 206 Arab children aged 1–4 years in East Jerusalem revealed a mean BLC of  $7.2 \mu\text{g}/\text{dl}$ , but only 3 children had BLC  $>10 \mu\text{g}/\text{dl}$  [25]. Also, in our study, all 35 newborns and 25 of their mothers who recently immigrated from Russia had UCBLI and BLC  $<10 \mu\text{g}/\text{dl}$ , and the average lead concentrations were identical to those of the Israeli-born mothers and their infants.

In conclusion, our study did not find lead exposure to be a problem in the sample of Russian immigrants and in a control group of Israeli-born mothers and their babies. A

search for childhood lead poisoning in Israel should be targeted to specific groups of children with known risk factors. Data on prenatal lead exposure in our study population indicate low exposure in comparison with a large number of studies worldwide.

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## References

- Amitai Y, Hryhorczuk D. Lead poisoning. In: Strange GR, Ahrens W, Lelyveld S, Schelermeyer R, eds. *Pediatric Emergency Medicine*. New York: McGraw-Hill, 1996:548–51.
- Bellinger D, Leviton A, Waternaux C, Needelman H, Rabinowitz M. Longitudinal analysis of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med* 1987;316:1037–43.
- Dietrich KN, Krafft KM, Bornschein RL, Hammond PB, Berger O, Succop PA, Bier M. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics* 1987;80:721–30.
- McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ. Port Pirie cohort study: environmental exposure to lead and children's ability at the age of four years. *N Engl J Med* 1988;319:468–75.
- de Caceres ML, Botet F, Costas C, Rosales S. Umbilical cord blood lead levels and neonatal behaviour. *Behav Neurol* 1995;8:39–41.
- Rothenberg SJ, Schnaas L, Cansino-Ortiz S, Perroni-Hernandez E, de la Torre P, Neri-Mendez C, Hidalgo-Loperenna H, Svendsgaard D. Neurobehavioral deficits after low level lead exposure in neonates: The Mexico City Pilot Study. *Neurotoxicol Teratol* 1989;11:85–93.
- Zarembski PM, Griffiths PD, Walker J, Goodal HB. Lead in neonates and mothers. *Clin Chim Acta* 1983;134:35–49.
- Ernhart CB, Wolf AW, Sokol RJ, Brittenham GM, Erhard P. Fetal lead exposure: antenatal factors. *Environ Res* 1985;38:54–66.
- Satin KP, Neutra RR, Guirguis G, Flessel P. Umbilical cord blood lead levels in California. *Arch Environ Health* 1991;46:167–73.
- Meyer J, Geuenich HH, Robra BP, Windorfer A. Determinants of lead concentration in the umbilical cord blood of 9189 newborns of a birth cohort in the district of Braunschweig. *Zentralbl Hyg Umweltmed* 1992;92:522–33.
- Hwang YH, Wang JD. Temporal fluctuation of the lead level in the cord blood of neonates in Taipei. *Arch Environ Health* 1990;45:42–5.
- Graziano JH, Popovac D, Factor-Litvak P, ShROUT P, Kline J, Murphy MJ, Zhao Y, Mehmeti A, Ahmedi A, Rajovic B, Zvicer Z, Nenezic DU, Lolocono NJ, Stein Z. Determinants of elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. *Environ Health Perspect* 1990;89:95–100.
- Schucard JL, Schucard DW, Patterson R, Guthrie R. Prenatal lead exposure and its potential significance for developmental disabilities: a preliminary study of umbilical cord blood levels. *Neurotoxicology* 1988;9:317–26.
- Koren G, Chang N, Gonen R, Klein J, Weiner L, Demshar H, Pizzolato S, Radde I, Shime J. Lead exposure among mothers and their newborns in Toronto. *Can Med Assoc J* 1990;142:1241–4.
- Saxena DK, Singh C, Murthy RC, Mathur N, Chandra SV. Blood and placental lead levels in an Indian City: A preliminary study. *Arch Environ Health* 1994;49:106–10.
- Rhainds M, Levallois P. Umbilical cord blood lead levels in the Quebec City area. *Arch Intern Med* 1993;48:421–7.
- Lagerkvist BJ, Ekesrydh S, Englyst V, Nordberg GF, Soderberg HA, Wiklund DE. Increased blood lead and decreased calcium levels during pregnancy: A prospective study of Swedish women living near a smelter. *Am J Public Health* 1996;86:1247–52.
- Hu H, Hshimoto D, Besser M. Levels of lead of women giving birth in a Boston hospital. *Arch Environ Health* 1996;51:52–8.
- Plockinger B, Dadak C, Meisinger V. Lead, mercury and cadmium in newborn infants and their mothers. *Z Geburtshilfe Perinatol* 1993;197:104–7.
- Al-Saleh, Khalil MA, Taylor A. Lead, erythrocyte protoporphyrin and hematological parameters in normal maternal and umbilical cord blood from subjects of the Riyadh Region, Saudi Arabia. *Arch Environ Health* 1995;50:66–73.
- Mayan O, Paiva I, Henriques A, Da Silva MV, Magarinho R, Calheiros J. Exposicao a chumbo do recém-nascido. *Rev Port Saude Publica* 1995;12:25–30.
- Shen XM, Yan CH, Guo D, Wu SM, Li RQ, Huang H, Ao LM, Zuhou JD, Hong ZY, Xu JD, Jin XM, Tang JM. Low level prenatal lead exposure and neurobehavioral development of children in the first year of life; a prospective study in Shanghai. *Environ Res* 1998;79:1–8.
- Gonzales-Cossio T, Peterson KE, Sanin LH, Fischbein E, Palazuelos E, Aro A, Hernandez-Avila M, Hu H. Decrease in birth weight in relation to maternal bone lead burden. *Pediatrics* 1997;100:856–62.
- Nir A, Tamir A, Zelnik N, Iancu TC. Is eye cosmetic a source of lead poisoning? *Isr J Med Sci* 1992;28:417–21.
- El-Sharif N, Fischbein A, Kaul B, Konijn A, Richter ED. Reemergence of lead poisoning from contaminated flour in the West Bank: a case study in cooperation in environmental epidemiology [Abstract]. Presented at the ISEE, August 21, 1996, Edmonton, Alberta, Canada.
- Emerson T. Lead and your kids: lead is the No. 1 environmental threat to the young, public-health officials say, but America has been slow to respond — and Europe slower. *Newsweek*, Cover Story, February 17, 1992:32–7.
- Hryhorczuk DO, Rabinowitz MB, Hessl SM, Hoffman D, Hogan MM, Mallin K, Finch H, Orris P, Berman E. Elimination kinetics of blood lead in workers with chronic lead intoxication. *Am J Ind Med* 1985;8:33–42.
- Pirkle LJ, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the U.S. population to lead, 1991–1994. *Environ Health Perspect* 1998; 106:745–50.

**Correspondence:** Dr. Y. Amitai, Mother, Child & Adolescent Health Department, Ministry of Health, 20 King David St., Jerusalem 91010, Israel. Tel: (972-2) 622 8836; Fax: (972-2) 625 8136; email: dvdamitai@matat.health.gov.il

## Capsule

### Improvement in the acuity of infants after visual input

Visual acuity was assessed in 28 human infants who had been deprived of all patterned visual input by cataracts in one or both eyes until they were treated at 1 week to 9 months of age. Immediately after treatment, acuity was no better than that of normal newborns. Acuity improved significantly over the next month, with some improvement apparent after as little as 1 hour of visual input. Unlike

findings at older ages, the pattern of results was the same for eyes treated for monocular and for binocular deprivation. The results indicate that patterned visual input is necessary for the postnatal improvement of human visual acuity and that the onset of such input initiates rapid functional development.

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