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Lead poisoning from contaminated flour

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Abstract

Lead widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world. Young children are particularly vulnerable to the toxic effects of lead and can suffer profound and permanent adverse health impacts. Lead also causes long-term harm in adults, including increased risk of high blood pressure and kidney damage. Exposure to lead can occur by contaminated air, water, dust, food, or consumer products. Lead is a cumulative poison, which produces a series of effects on blood forming tissues, the digestive and nervous systems, and the kidneys. The aim of this paper was the determiation of lead in blood of poisoning persons, as well as on contaminated flour and in contaminated milk. Between 1992 and 1998 some rural districts of north and southern Albania reported several massive outbreaks of lead intoxication. Intoxications were associated with the use of contaminated flour originating from rural old mills, whose cracked grindstones were repaired by using lead as a filling material. Almost 6,000 individuals living in the affected areas were exposed to the toxic pollutant. However, only 236 individuals were checked for lead blood exposure levels. Of these, almost half revealed clinical signs of lead poisoning. Lead analytical determinations were performed in blood, milk, and flour samples from the area population by using atomic absorption spectrometry. Lead levels in blood varied from 7.1 μg/dl to 91.6 μg/dl. Mean blood lead concentrations according to the age-groups varied from 39.18 μg/dl to 44.99 μg/dl, whereas the average lead levels in flour samples were 42 ppm, while in blended bovine milk 0.6 ppm. The levels of lead in blood samples, as well as in flour and milk have resulted higher than TLVs levels acceptable by WHO.

Keywords: Lead poisoning, Lead blood, Contaminated flour, Contaminated milk

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1 Introduction

Lead is a naturally occurring toxic metal found in the Earth's crust. Its widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world.

Important sources of environmental contamination include mining, smelting, manufacturing and recycling activities, and, in some countries, the continued use of leaded paint and leaded aviation fuel. More than three quarters of global lead consumptioQn is for the manufacture of lead-acid batteries for motor vehicles. Lead is, however, also used in many other products, for example pigments, paints, solder, stained glass, lead crystal glassware, ammunition, ceramic glazes, jewellery, toys and some cosmetics and traditional medicines. Drinking water delivered through lead pipes or pipes joined with lead solder may contain lead. Much of the lead in global commerce is now obtained from recycling.

Lead is a cumulative poison, which produces a series of effects on blood forming tissues, the digestive and nervous systems, and the kidneys. People affected by lead poisoning become pallid, moody, and irritable, they lose their appetite and may become anemic. In late stages they may suffer dizziness, headaches, visual disturbances and occasionally paralysis of the hands and wrists (Merian and Clarkson, 1991; Simms, 1986; Codex Standard 193-1995).

Lead is easily taken into the body by inhaling lead dust, absorbing lead based chemicals through the skin or digesting lead present in food and water (Merian and Clarkson, 1991; Palloti et al., 1983).

For the first time Albanian public health service was faced to lead poisonings from contaminated flour during period Spring - Summer 1992. The first signs of poisonings in the area population appeared in May 1992 in a young boy hospitalized after presenting with renal colic associated with severe pain. In the coming days other family members also developed signs of lead poisoning and were promptly hospitalized, while other area families also subsequently showed clinical signs of intoxication. This lead intoxication scenario reappeared in June 1992, February 1996 (Tabaku and Panariti, 1996), May 1996 and April 1997 (Panariti and Berxholi, 1998) and resulted in four deaths, two children and two adults. The present paper presents an investigation of the lead levels in blood, flour, and milk samples from the affected area.

2 Materials and Methods

In the three areas where clinical symptoms of lead poisoning were observed, human blood as well as milk and flour samples were collected. Two hundred and thirty six (236) blood samples (130 in May 1992, 51 in June 1992, and 55 samples in May 1996) were collected by venipuncture in polypropylene tubes containing heparin as an anticoagulant. Blood samples were stored in deep-freeze until analysis was performed. Blood samples were diluted with sol. Triton X-100 0.2% 1:10 and lead was determined by Graphic Furnace Atomic absorption spectrometry (Varian 10 Plus Instrument).

The accuracy and precision of the assays were checked by standard quality control procedures (Lind et al., 1988; Vahter et al., 1991a; Vahter et al., 1991b). The results were in agreement with accepted criteria and their average of repeated evaluations was accepted as "true" value. The present acceptability criteria dictated that samples reading lead values below 200 μg/L had to vary in repeated assays no more than ± 15 μg/L from the mean, while those from 210 to 250 μg/L varied no greater than ± 20 μg/L from the mean, and above 250 μg/L the maximum variation was ± 25 μg lead /L from the samples mean.

The flour samples were collected from the families of the intoxicated individuals. Bovine milk samples were collected only from families which had used tainted flour as cattle-feed. Five gram samples of flour and 10 ml of milk were digested in concentrated nitric acid at 75^0C for at least 3 hours; measurements were performed by Atomic Absorption Spectrometry with air acetylene flame.

3 Results and Discussions

Table 1 and 2, and figure 1 indicate the determined lead levels in human blood. The obtained data were submitted to statistical elaboration through STATGRAPHICS Version 5.0.

Table 1. Pb- blood levels as μg/dl

Age (years)	Nr.	Mean	S D	Min.	Max.
1-5	33	39.693	16.815	13.0	75.1
6-15	83	44.995	19.377	7.1	91.6
16-30	53	41.613	19.495	10.0	91.5
31-50	51	39.186	16.040	10.3	87.4
over 50	16	40.337	16.070	14.2	74.8

Table 2. Blood lead levels μg/dl distribution in percentiles

Age (years)	P_{20}	P_{50}	P_{90}	P_{98}
1-5	21.0	40.0	66.5	75.0
6-15	25.0	45.5	67.5	83.0
16-30	21.0	41.3	65.0	82.0
31-50	22.5	42.5	53.9	66.5
over 50	25.0	40.0	67.0	74.8

Our observations were in agreement with those reported in two other lead poisoning from contaminated flour outbreaks studied in Israel, Spain, Western Bank arabs and Greece (Eisenberg et al., 1985; Carton et al., 1987; Hershko et al., 1989; Dona et al., 1999). This epidemic resulted from the improper use of lead to repair the old stone mills. These outbreaks were

characterized by the absence of a previous toxic contamination and by repeated ingestion of lead power, over a period of several days; the usual vehicle of the toxic was bread. However, several circumstances probably contributed to attenuate lead toxicity, such as the short duration of the highest contamination period, the poor digestive absorption of lead, some differences in diet habits, etc. These aspects helped lengthen the period of latency, and diversified the clinical and prognostic manifestations.

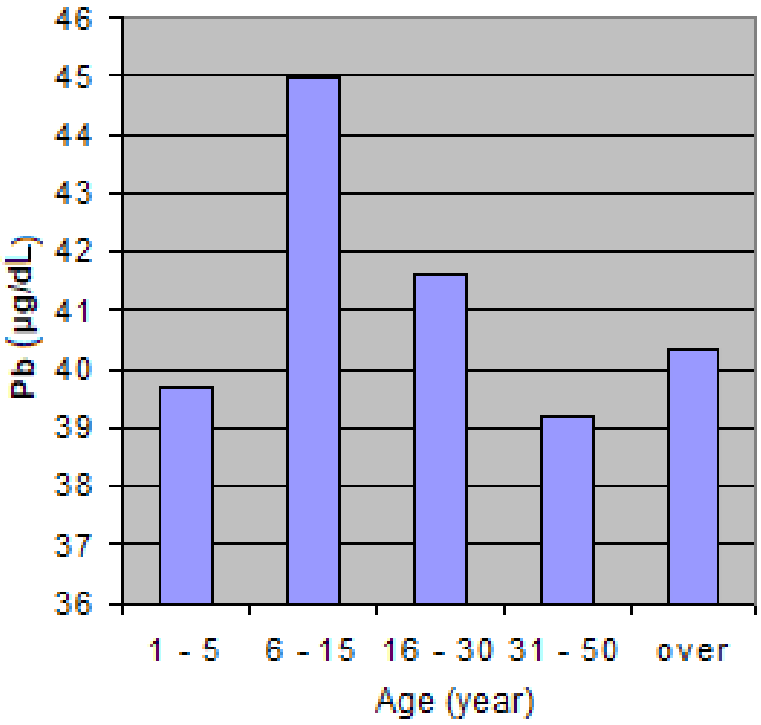

Figure 1. Lead in blood (µg/dL)

From the tables and the figure above it is apparent that most of the blood samples had lead levels above the population background levels (Christensen and Holst, 1988; Palloti et al., 1983; Claeys-Thoreau et al., 1983; Tabaku et al., 1990; Tabaku et al., 1998). The highest lead blood-levels were determined in samples collected in June 1992, which ranged from 22.5 - 91.6 µg/dl (mean 56.041 ± 13.857) whereas the lowest levels were determined in the samples collected in May 1996, and ranged from 10.0 - 44.1 µg/dl (mean 18.982 ± 9.030).

The symptoms of intoxication were more severe during the June 1992 period compared to those observed in May 1996. A good correlation was obtained between mean familial levels of lead in blood and doses of lead in contaminated flour. Also, was obtained a good correlation between the level of lead in blood and the severity of the patients in all groups of our classification.

Lead levels following determinations performed in 40 flour samples and 10 blended bovine milk samples (table 3), varied between 10.0 and 72.5 ppm (mean 42 ppm) and 0.45-0.85 ppm (mean 0.60 ppm) respectively. These levels are well above the acceptable levels in cereals and

milk as recommended by WHO (WHO Technical Reports Series 837; Codex Standard 193,1995) and should therefore be considered as highly toxic.

Table 3. Lead concentration in flour and milk (ppm)

	Number of samples	Min	Max	Mean ± SD
Flour	40	10.0	72.5	42.00 ± 22.40
Milk	10	0.45	0.85	0.60 ± 0.11

4 Conclusion

Data obtained by analysis of lead in blood of all age groups of poisoning population , as well as in flour and milk have resulted higher than threshold limits values. The temporal, clinical, and laboratory correlation of blood lead with the respective flour levels and with the onset of clinical signs characteristic of lead poisoning in humans, led to the establishment the diagnosis of lead poisoning.

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Conflict of interests

The author declares that there are no competing interests.

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