

TOXICITY AND MONITORING OF LEAD IN TAP WATER

By

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ABSTRACT

The guideline value of 0.05mg/L. has been recommended for lead in drinking water as reported by WHO 1984 while the Egyptian guideline value is 0.1 mg/litre.

Man's exposure to lead through water is generally low in comparison with the exposure through air and food as reported by WHO 1973. Monitoring of lead in tap water was conducted in 8 governorates in Egypt to through high lights about the interaction between the level of lead in drinking water and the public health taking in consideration that the Egyptian people drink daily 2.8 litres of water.

Data indicate that all the samples contained concentrations of lead varied between 0.118 to 0.945 mg/litre exceeding the Egyptian and WHO levels.

The variation of water contents from lead varied according to the age of tube lines, the absence of water tanks on buildings and the source of water (Nile water or underground water).

The main source of lead in tap water may be attributed to several reasons i.e. the contents of water tubes from lead, the lead part of tube which is normally connected in each house between the water meter and the main net of tubes in houses, the high content of water from chlorine compounds and due to the insertion of water pumps which is normally made from lead in Egypt.

Rats which received a repeated daily dose of lead nitrate (40 µg/kg) for 20 days as drinking water showed high concentration of lead in serum (8.16 ± 0.68 µg/ml) when compared with control (3.76 ± 0.14 µg/ml). While the concentration of lead in urine was found 4.01 ± 0.41 µg/ml in treated animals compared to the normal control (11.17 ± 0.21 µg/ml).

INTRODUCTION

The concentration of lead in the domestic water supply was 2-3 mg/litre. In this case, the reason for the extreme contamination was that the water was stored in lead tanks. Homes with both lead-lined water storage tanks and lead pipes had the highest concentration. The plumbosolvency of water standing in lead pipes is influenced significantly by several factors. The solvency increases about four-fold with increasing acidity over the pH range from 6 to 4. Increases of a somewhat lesser degree were also noted with increasing alkalinity over the range of pH from 8 to 10 (Moore, 1973). The same author also pointed out the increasing plumbosolvency of water with increasing temperature and with decreasing calcium concentration. Quite recently it was shown that lead concentrations in tap water were highly dependent on the volume of water flushed through the system before sampling. The concentrations were also considerably lower when a 95/5 (tin/lead) solder had been used in the copper piping instead of the 50/50 or 60/40 solders (Wong & Berrang, 1976).

When water was left standing overnight in plastic pipes, some degree of leaching of lead into the water was observed (Heusgem & DeGraeve, 1973). The source of lead in this case was probably lead stearate which is used as a stabilizer in the manufacture of polyvinyl plastics.

Smith and Flegal, 1992 reported that rats that have been reared on a low level diet (lead intake approximately 80 ng Pb/g body/day) were fed 206 Pb - enriched drinking water (210 ng Pb/m) for 1.5 days and then were chelated with a single injection of a 0.11 m mol/kg dose of DMSA. Blood, kidney, brain, urine and feces were collected 24 hrs after chelation and analyzed for lead concentration. These analysis demonstrated that DMSA chelation significantly increased (15 fold) the diuresis of labile soft tissue lead, but not skeletal lead. DMSA also appeared to effect a redistribution and impot of a comparable amount of lead to skeletal and smaller relative amounts of lead to the soft tissues (blood, kidney) of the chelated animals.

Bogden et al., 1992 reported that rats were fed diets containing 0.1, 0.5 or 0.5 Ca for 52 wk and were simultaneously given either 0, 50 or 100 mg/L in their drinking water. Rats fed the 0.1 % Ca diet had organ lead concentrations that were 2 to 20 fold greater than the correspondy animals fed 0.5% Ca. Lead inhibited an increase in renal calbindin in the rats fed 0.1 % Ca, but paradoxically increased renal calbindin levels in animals fed 2.5 % Ca.

Khalil Menesh et al., 1992 indicated that urinary excretion of lead, the enzymes N-acetyl- beta-D-glucosaminidase (NAG) and glutathions -S-transferase (GST) and brush border antigens (BB 50, CG 9 and HF 5)

centrifuged at 3000 rpm for 15 minutes to obtain serum. Samples were kept at (-20°C) until analysis.

RESULTS AND DISCUSSION

Results for lead level in drinking water in Great Cairo - (Cairo, Giza and Qalubia) are postulated in table 1. Data indicate that 100% of the samples tested contained lead in levels more than the acceptable levels adopted by both the Ministry of Public Health (0.1 mg/L) and WHO (0.05 mg/L). The mean level of lead in urban tap water was (0.4923 mg/L) and the maximum level was (0.709 mg/L), while the minimum level was 0.236 mg/L.

Tap water samples from new houses without storage tanks contained Pb less (0.4216 mg/L) than the same houses with storage tanks (0.4843 mg/L). Tap water samples from very old houses contained more lead levels than samples from new buildings.

Table 1: Levels of lead found in tap water in Great Cairo

Site	Level (mg/L)	Mean
Urban area	* Maximum 0.433 ** Minimum 0.236 0.473	0.4216 ± 0.1052
New houses without tanks	* Maximum 0.433 ** Minimum 0.351 0.512	0.4843 ± 0.049
New houses with tanks	* Maximum 0.473 ** Minimum 0.433 0.551	0.5711 ± 0.083
Very old houses	* Maximum 0.551 ** Minimum 0.512 0.709	0.3256 ± 0.107
Rural area	* Maximum 0.394 ** Minimum 0.603	0.2314 ± 0.068
Giza	* Maximum 0.197 ** Minimum 0.473	
Qalubia	* Maximum 0.197 ** Minimum 0.473	

* Samples from houses with a connection of lead tube.
** Samples from houses without a connection of lead tube.

All samples taken from houses which the net pipes were connected with the water meter with a piece of lead contained high levels of lead than the others which have not this piece.

Tap water samples from rural areas showed that the level of lead in water was lower in Giza (0.3256 mg/L) than in Qalubia (0.2314 mg/L) and in all cases urban areas contained lead level in water samples more than in rural areas.

Samples of tap water which were taken from Sharikia governorate (Table 2) show that urban samples contained high levels of lead (0.604 mg/L) than the samples of rural areas (0.610 mg/L). Another trend was obtained in the case of El-Behera governorate. Samples from rural area contained lead levels more (0.421 mg/L) than the samples from urban areas (0.298 mg/L) (Table 2).

In Sewiss, where is no rural areas, the chemical analysis of water showed a high level of lead in water. The mean level of lead in samples was (0.322 mg/L) and the maximum lead level was estimated to be 0.473 mg/L, (table 2).

The water samples from Minia governorate showed the

were utilized to explore possible markers of kidney injury in male rats which administered lead acetate (0.5%) continuously in drinking water for periods ranging from 1-12 months GFR was increased significantly after three months of lead exposure, but was decreased significantly after 18 months.

Smith et al., 1992 reported that the elevated concentration of lead in kidney of rats (fresh weight) relative to levels in blood are consistent with the presence of specific lead-binding sites in the kidney a very low levels of exposure.

Singh et al., 1991 reported that lead administration (250, 500, 1000 and 2000 ppm as lead acetate) in drinking water during fetal development (from 15 to 28 days of gestation), in normal and iron-deficient pregnant rats, revealed dose-dependent increases in the lead content of maternal blood that was marked in iron-deficient animals. The placenta and fetus did not show a dose-dependent increase in lead content.

Tomokurti et al., 1991 reported that concentrations of both beta-aminobutyric acid (ABA) and urinary delta-aminolevulinic acid (ALA) increased significantly in the lead - exposed mice to 500 ppm in drinking water for 14 days.

Korsrud and Meldrum 1988 reported that the concentrations of lead in the livers and kidneys of rats increased in proportion to the dose of lead acetate that the rats were given orally or in the drinking water.

Lead is a poison of enzymes, binding to the sulfhydryl groups of proteins. In high concentration, lead alters the tertiary structure of intracellular proteins, denaturing them and causing cell death and tissue inflammation (Harrison's 1991).

MATERIALS AND METHODS

A - Water Samples and Lead Analysis:

Samples were taken from 8 governorates, Cairo, Giza, Qalubia, Ismailia, Minia, Sharikia and Behera.

In each local governorate, samples were collected from rural and urban areas, also from new and old houses, from houses with storage tanks and without tanks. The samples from houses which have a lead part which connected the water meter and the house net pipes were put in consideration. The total number of tested samples were 116. All the samples were stored at 4°C until analysis. Lead was analyzed by Flam Atomic Absorption (Spectrophotometer Buck Scientific 2000) according to the method of Weiz, 1985.

B - Experimental Animals:

The present work was conducted on 12 adult male albino rats (250 - 300 g). They fed on a diet of rat chow and water *ad libitum* for one week before starting the experimental work. The animals were allocated into two groups, the first group (6 rats) each was treated with a dose of 40 mg/kg b.w lead nitrate in drinking water through gastric oral intubation. The dose given daily for 20 days. The second group (6 rats) as control group received water only. Animals were kept in metabolic cages all over the period of treatment. Urine samples were collected daily.

At the last day of treatment, the animals were decapitated and samples of blood were collected and

mean concentration of lead in rural areas (0.146 mg/L) while, the mean in urban sample was (0.236 mg/L), table (2).

Table 2: Levels of lead found in tap water in the different 5 governorates.

Level	Governorates				
	Ismailia	El Minia	El Swiss	El Behera	Sharkia
Urban area					
Maximum	0.276	0.236	0.473	0.324	0.748
Mean	0.131 ±0.138	0.236 ±0.000	0.322 ±0.098	0.298 ±0.086	0.604 ±0.116
Minimum	0.000	0.236	0.197	0.167	0.433
Rural area					
Maximum	0.355	0.197	-	0.533	0.845
mean	0.66 ±0.067	0.146 ±0.037	-	0.421 ±0.07	0.610 ±0.129
Minimum	0.197	0.079		0.276	0.473

In Ismailia, samples from urban areas contained low lead level (0.131 mg/L) when compared by the samples from rural areas (0.66 mg/L).

The maximum lead level detected in rural areas in Sharkia (0.845 mg/L), followed by El-Behera (0.533 mg/L) followed by Qualiobia (0.473 mg/L), followed by Ismailia (0.355 mg/L) and the lowest was El-Minia rural areas (0.197 mg/L).

In the case of urban areas Sharkia headed all the governorates in the maximum lead level in tap water (0.748 mg/L) followed by Great Cairo (0.709 mg/L), Sewiss (0.473 mg/L), Ismailia (0.276 mg/L) and the lowest was El-Minia (0.236mg/L).

It is clear from the results that urban tap water samples were highly polluted with lead when compared with samples from rural areas in all governorates except Ismailia.

In the same time samples from homes with both lead-lined water storage tanks contained high levels of lead than home without storage tanks. The age of water pipe net is also responsible about the contamination of the tap water with heavy metal specially lead. The pollution was increased by the increase of the net age.

The main sources of lead in tap water in Great Cairo and the towns may be attributed to the water pipe net, storage tanks, connection of the water meter with water pipe nets with part of lead in most of the houses.

In village or in rural areas the main source of lead is the Sewage water and the underground water.

The mean daily intake of lead through drinking water is 1.316 mg/person (70 kg).

The mean values of lead in serum of the treated animals (Table 3) was found to be 8.16 ± 0.68 µg/ml compared to the control (3.76 ± 0.14 µg/ml).

Pb-B reflects the current state of the dynamic

equilibrium between the amounts of lead entering in the tissues.

Table 3: Levels of lead (µg/ml) in serum 2nd urine of rats giving 40 µg/kg lead nitrate.

Lead levels µg/ml	Serum mean ± S.D.	Urine mean ± S.D.
Control	3.76 ± 0.14	11.17 ± 0.21
Treated	8.16 ± 0.68	4.01 ± 0.41
% Change	+117.02	-68.85
T	15.7	42.1
P	<0.001	<0.001

After a single inhalation of a soluble lead compound, the concentration of lead in the body will change in the same was as after an intravenous injection i.e. there will be a rapid increase in Pb-B levels followed by a slower decrease; initially there will be a rapid elimination in the urine and a slow deposition in the tissues with subsequent redistribution according to the metabolism of lead in the term exposure at a constant rate are equilibrium between the amount of lead absorbed, deposited and excreted develops over a long period (weeks to months according to the daily doses received which can be considered as a steady state.

There are only limited data as to how quickly this equilibrium (and Pb-β) changes when irregular variations in the dose of lead recieved (e.g. air lead concentrations) occur.

The lead level in urine (Table 3) of control group was 11.17 ± 0.21 µg/ml while it was found 4.01 ± 0.41 µg/ml in treated animals.

An lowering rate of spontaneous lead excretion in the urine is an indication of high lead absorption in the tissues specially liver and kidney (organs of detoxication) these results were in good agreement with Smith, et al., (1992), but a normal rate of excretion does not serve as a reliable means of excessive absorption. Lead excretion in urine is dependent on the Pb-p level but it is also influenced by other, mostly unknown factors. So that no direct conclusions about exposure and the extent of absorption can be derived from lead levels in urine.

Harrison's (1991), demonstrated that adults may ingest up to $0.7 \mu\text{mol}$ (150 µg/d) of lead from normal exposure to food and drinking water. Positive lead balance may occur at these levels since renal excretion normally does not exceed $0.4 \mu\text{mol}$ (80 µg/d). In children, no more than $0.02 \mu\text{mol}$ (5 µg)/kg body weight. Also, he stated that only 10% of lead was excreted in urine which coincide with the present data.

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