A Survey of Blood Lead Levels in Pregnant Women attending two Public Prenatal Clinics in Nairobi City, Kenya

Owago*[1] Joshua Odhiambo, Jane Murungi[2] and Caroline Lang'at-Thoruwa[2]

[1] Department of Environmental Science and Engineering in the School of Environmental Studies, China University of Geosciences (Wuhan), 388 Lumo Road, Wuhan, 430074, P.R. China.
[2] Department of Chemistry in the School of Pure and Applied Sciences at Kenyatta University, P.O. Box 43844-00100 GPO Nairobi, Kenya
* Corresponding Author: Tel. +8613720135523, +254722821146; Fax +86-27-87515956 +254-20-4449072; email: joowago@hotmail.com

Abstract

Exposure to lead remains an under-recognized public health problem in the developing countries such as Kenya despite well documented toxic effects of the metal at elevated blood levels. The objective of the study was to measure the blood lead levels in pregnant women of Nairobi city so as to assess the extent of exposure of the subjects to the toxic heavy metal and to evaluate the toxicological significance of the findings. Blood lead levels (BLLs) of 223 women in Nairobi City: 152 from Eastleigh Clinic and 71 from Pumwani maternity hospital were examined. The two public hospitals serve mainly disadvantaged communities. Overall BLLs ranged from non-detectable (nd) or zero to 295.0 μg dL^{-1}. Women from Eastleigh clinic had slightly higher mean BLL than those from Pumwani (29.5μg dL^{-1} vs 25.9μg dL^{-1}) but Student t-test found no significant difference between them (t-stat=0.80; df=186; $p>0.05$). The blood lead levels were above typically cited as average and may pose threat to the mothers and fetuses, as 70.4 % samples had values greater than 10 μg dL^{-1} (the US Centers for Disease Control cut off level for raised blood lead level in children and pregnant women). Clearly this work gives additional urgency to Kenyan efforts to reduce and prevent lead exposure to her population and provides a rationale for deeper investigation to identify sources of exposure in the country. A periodic surveillance of blood lead levels in vulnerable individuals such as children, pregnant women and occupationally exposed individuals is advocated.[Journal of American Science 2009:5(3) 41-51] (ISSN: 1545-1003)

Key words: Blood lead levels, pregnant women, health risk, Nairobi City

1.0 Introduction

Lead is a widely distributed heavy metal in the environment particularly in urban areas that display older housing stock with lead-containing paint, high concentration of industry and heavy traffic [CDC, 1991; US EPA, 1998]. Exposure to lead occurs mainly through either inhalation of contaminated air and dust or ingestion of food, water, soil or any other substances containing lead. Motor vehicle exhaust fumes from leaded fuel, smelters, lead manufacturing and recycling industries, waste sites (e.g. contaminated landfills), old lead water piping and lead-combining solders, and leaded paint are common sources of lead contamination [UNEP/FAO, 2003]. In humans, lead can damage the nervous and reproductive systems, and the kidneys, and it can cause high blood pressure and anaemia [UNEP/FAO, 2003]. Exposure to the metal is associated with a wide range of negative pregnancy outcomes including developmental delays, early membrane rapture, low birth weight, spontaneous abortion, complications during pregnancy, increased prenatal mortality and inhibited postnatal growth [US EPA, 1986]. In addition, prenatal exposure is a major cause of childhood lead poisoning [Weizsaecker, 2003]. Lead is a neurotoxic agent that may cause severe impairment of the nerve tissue, particularly in the developing central nervous system resulting in impairment of cognitive function and the induction of behavioural disorders in young children [Counter et al., 1997]. The blood-brain barrier is poorly developed in the embryo and neonate, and the developing neural tissue may be expected to be particularly vulnerable to lead [Ratcliffe, 1981]. The risk of lead intoxification may be higher during pregnancy on both the mother and foetus because there is evidence that lead is mobilized from its storage sites in the maternal skeleton and drawn into the bloodstream and soft tissues during

gestation [Gulson et al., 1997]; pregnancy induces anaemia by decreasing iron levels resulting in increased absorption and toxicity of lead [Mahaffey, 1990]; and lead passes across the placenta almost without hindrance [Todd et al., 1996].

Monitoring for lead exposure generally involves measuring the concentration of whole blood lead. It is taken as a good indicator of exposure because of ease of sampling, homogeneity of the sample and especially because blood lead levels (BLL) appears to vary linearly with exposure. It is also useful in evaluating the likelihood of health effects and it may reflect the level of lead contamination in the total environment (air, water, soil and food). However, in view of the relatively short half-life of lead in blood, BLL measurements reflect only recent exposures [O'Neill, 1993]. The normal body burden for Lead in adults (blood lead levels ≤30-40 μg/dl) is of the same order as that at which harmful effects may occur to the foetus [WHO, 1995].

It is well recognized that BLLs have been decreasing in among the people living in countries where measures that aim to control or ban lead in gasoline have been implemented. Such decreases in BLLs have been reported in Canada, Germany, Norway and United Kingdom [WHO, 1995]. At the time of this study, fuel containing tetraethyl lead was the only type of gasoline available and so, the main source of environmental lead were internal combustion engines of vehicles that use the fuel. In Kenya, places with higher vehicular densities also had elevated lead in their soils [Onyari et al., 1991], plants (Makokha, 2004) and human nails [Were et al., 2008]. According to UNEP (2005), exposure to lead in Kenya is large, and the risks involved in human health and the environment are significant.

The purpose of this study was to conduct a preliminary blood lead survey of a small group of pregnant women reporting to prenatal clinics of Eastleigh and Pumwani, Nairobi, for assessing the extent of environmental lead absorption. The data was used to evaluate maternal and unborn baby's risk to lead toxicity and to evaluate the need for a deeper investigation for possible future use in the assessment of the impact on blood lead concentration of the phasing out of leaded fuel in Kenya, which was due in 2006.

2.0 Materials and Methods

2.1 Study Area

Nairobi is the largest town in Kenya with a population of about 2.1 million [CBS, 2000]. The city covers approximately 700 square kilometers. The city has got the greatest concentration of industries and the heaviest traffic density in Kenya. These and other sources introduce enormous qualities of pollutants such as lead into the environment. However, the city has over the years been having poor environmental management systems. For example, among the developing countries sampled for study on air quality management capabilities, it was the worst [UNEP/WHO, 1996]. Consequently, the city residents could be at a great risk of exposure to a myriad of pollutants including lead, hence the choice of Nairobi city for this survey.

2.1.1 Sampling Stations

Participants were recruited at two prenatal clinics in Nairobi City. Pumwani maternity hospital and Eastleigh clinic are both public hospitals situated to the east of the city centre, about 2 kilometers from each other. Public hospitals in Kenya serve primarily the poor and disadvantaged communities who rely on government subsidized medicare. Pumwani is a referral hospital that serves both patients from its neighbourhood and those from other parts of the city. Pregnant mothers with complications or who had had complications in their previous pregnancies may be referred Pumwani for specialized gaenecological attention. Eastleigh clinic on the other hand normally serves primarily the residents of the Eastleigh and the immediate neighbouring estates including slum dwellings such as Mathare valley and Mlango Kubwa.

Patients attending these clinics were likely to be among the poorest city residents in occupations and dwellings of high risk for lead exposure hence the basis for inclusion of the two hospitals in the study.

2.2 Sample Population, Recruitment and Sampling

In this cross sectional survey, all pregnant women seeking prenatal care at the two public prenatal clinics were eligible for recruitment. A total of 223 mothers aged between 15 and 40 years were recruited between April and June 1998. The Kenyatta National Hospital Ethics and Research Committee; Medical Officer of Health (MOH) in charge of Nairobi City Council Health Department; and the participating hospitals approved the research protocol. The mothers in both hospitals

were introduced to the study; adequately informed about the study in English, Swahili or in their mother tongues and their informed consents were obtained.

In order to relate the samples to their sources for possible follow-up, the patient's hospital personal file number was noted and blood lead value determined was recorded against the number for each patient.

2.2.1 Blood Sample Collection and Pre-treatment

About 5ml of blood was taken once from each mother to be used in routine prenatal checks and for the BLL determinations. The samples were taken by healthcare workers at the hospitals. Venous blood samples were collected into lead free (EDTA –containing) vacutainer tubes (Becton Dickinson VACUTAINER systems L10262-00 Franklin lakes NJ7414-1885). Each tube was inverted several times to mix the EDTA and the blood so as to prevent coagulation. After the portions for other purposes had been taken, the remaining samples were stored at about 4^0C until lead analysis.

2.3 Laboratory Procedure and Quality Control

Sample preparation and instrumental analysis were carried out at the Mines and Geology Research Laboratory, Nairobi, Kenya. Samples were prepared for analysis using standard methods (vanLoon, 1985; NIOSH, 1977).

2.3.1 Equipment and Reagent

Blood lead measurements were performed using atomic absorption spectrometer (AAS-Spectr-AA-10, Varian-Techron, pty. Ltd, Austria). All the reagents used were of high standard analytical grade supplied by BDH Chemicals Limited, Poole, England. Water used was distilled-deionised. All the glassware used in this study were decontaminated by soaking overnight in 5% HNO_3 and rinsed thoroughly in deionised water. The lead standard solutions were obtained from spectroscopic grade lead nitrate stock solution (1000 mg/L). The standard solutions were freshly prepared daily and checked for constancy of absorption before taking the readings.

2.3.2 Acid Digestion

About 2 ml of whole blood samples were transferred to labeled digesting tubes and 4ml of concentrated nitric acid added to them. The samples were placed on a hot plate in the fume chamber and subsequently heated to just below boiling point. The samples were digested slowly for about 3h. When the volumes had reduced to about one third, 2 ml of 30% hydrogen peroxide solution was added. The samples were then evaporated slowly, residues dissolved in 1% nitric acid and filtered to provide sample solutions ready to be aspirated into the AAS.

2.3.3 AAS Analysis

The digested sample solutions were assayed in duplicate by use of AAS with acetylene flame. The lead metal was under detection limit of 0.20 μgdL^{-1}. A series of standard solutions of lead were freshly prepared for instrumental calibration by diluting in commercial standard containing 1000 μgL^{-1} of the metal. Adequate quality control was ensured. Standard and blank samples were analyzed for every ten samples analyzed in a day. Lead was not detected in any of the blank samples analyzed.

3.0 Results

The average blood level among the 223 pregnant women who participated in the study was 28.4$\mu g\ dL^{-1}$ and ranged from zero or non-detectable (nd) to 295.0$\mu g\ dL^{-1}$. Figure 1 presents BLL distribution by hospital. The blood lead levels were slightly higher in women recruited at Eastleigh hospital than those of Pumwani hospital. The mean (± standard error) BLL for Eastleigh clinic subjects (n=152) was 29.5 ± 3.1 (median 20) while the mean for Pumwani maternity hospital subjects (n=71) was 25.9 ± 3.3 (median 15). There was no significant difference between the mean BLLs in the two hospitals (p=0.21; df=186; t-stat. =0.80; t-test). Lead was not detected in 10.5% (n=16) and 16.9% (n=12) subjects from Eastleigh and Pumwani clinics respectively. Seventy three per cent (n=111) of the women from Eastleigh and 70.4% (n=50) had blood lead concentrations above the international action level of 10 $\mu g\ dL^{-1}$ for children. Some 31.5% (n=48) of the mothers from Eastleigh and 35.3% (n=25) from Pumwani had BLLs above 30$\mu g\ dL^{-1}$, which is the universal upper limit of acceptable BLL for women in reproductive age (WHO, 1980). Also, BLLs of 27 (17.7%) and 16 (22.5%) mothers from Eastleigh and Pumwani respectively, were above the US threshold of 45 μgdL^{-1} for lead poisoning treatment with chelation therapy. There were also 6 women; 4 from

Eastleigh and 2 from Pumwani, whose blood lead levels were ≥100 μgdL^{-1}. Only 2 women who were both recruited at Eastleigh hospital had their blood lead concentrations ≥200 μgdL^{-1}. However, none of the subjects had any noticeable symptoms of lead intoxification at the time of their recruitment.

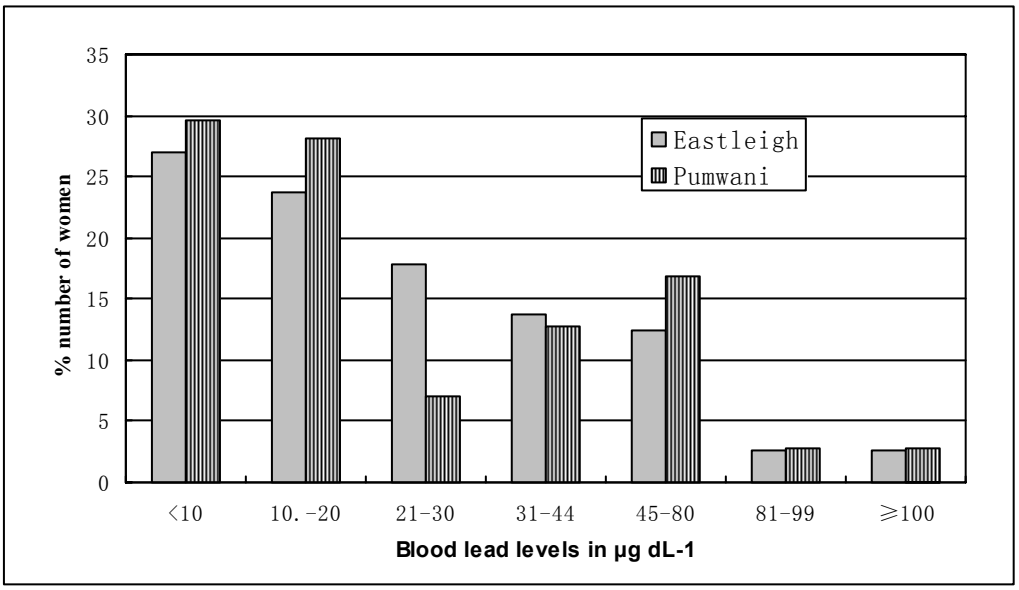

Figure 1: Distribution of blood lead levels (μg dL^{-1}) of pregnant women reporting to Eastleigh and Pumwani hospitals in Nairobi (1998)

4.0 Discussion

4.1 The blood leads levels

To our knowledge this was the first survey of blood lead levels in pregnant women in Kenya. The average BLL of women in this survey were higher than typically cited as average in pregnant women and may pose threat to the mothers and especially fetal development. The report, therefore, documents that exposure to lead among pregnant women attending public hospitals in Nairobi is a significant problem. The mean BLLs of Eastleigh (29.5 μg dL^{-1}) and Pumwani (25.9μg dL^{-1}) pregnant women were essentially similar as there was no significant difference between them ($p>0.05$; t-test). The somewhat higher BLLs exhibited by the Eastleigh mothers may have to do with socio-economic characteristics of the subjects. Within the proximity of Eastleigh clinic are informal settlements such as Mathare, Mlangokubwa, Korokocho. Mothers from these poor backgrounds could be living in dusty poorly ventilated shanties that may increase their chances of indoor and outdoor sources of lead. Some studies [Farias et al., 1996; Lee et al., 2005] reported that BLLs of pregnant women are affected by sociodemographic and nutritional factors even after controlling for environmental exposure. The women from low socio-economic status were more disadvantaged (had higher BLLs). Elsewhere in Africa, raised blood lead values were associated with dusty houses, houses in poor state of repair, overcrowding, low educational and income levels, and other factors related to family structures and socio-economic status [von Schirnding, 1991]. Being a referral hospital, Pumwani maternity hospital could be serving some mothers from the middle class with better living conditions and dietary habits, hence the lower BLLs. However, owing to the services rendered by the hospitals, their catchment areas may overlap or extend farther than assumed.

The World Health Organization or WHO (1980) recommended 40 μg dL^{-1} for male workers and 30 μg dL^{-1} for women in child-bearing age as maximal tolerable BLL; and that no more than 50% of a population's blood lead values should exceed 20 μg dL^{-1}.The US Centers for Disease Control cut off level for raised BLL for children and pregnant women is 10 μg dL^{-1} [CDC, 1991]. Blood lead concentration of 45 μg dL^{-1} or higher demands treatment by chelation therapy [DOHMH, 2007]. More than 70 μg dL^{-1} presents a medical emergency while over 120 μg dL^{-1} is highly toxic and potentially lethal [UNEP/UNICEF, 1997].

We were unable to locate published blood lead data on pregnant women measured before 1998, the date of this study, to compare with these results. However, the higher umbilical BLLs (mean 39.9 μg dL^{-1}; range: range: 1-124 μg dL^{-1}) reported among women from Thika, a satellite industrial town in the outskirts of Nairobi [Makokha, 2004], appear to confirm the higher exposure to lead of mothers in Kenyan urban centres. The report further indicates that pregnant women BLLs could be on the increase in Nairobi and environs. The few data available on BLL on the general population suggest that the general population in Nairobi may not be at as high risk as their pregnant counterparts. Wanjie (1991) reported a mean BLL of 9.3 μg dL^{-1} (range: nd-256.7 μg dL^{-1}; n=100), while Njoroge and UNEP (2005) determined a much lower mean BLL of 8.8 μg dL^{-1} (range:1.4-65 μg dL^{-1}) among Nairobi residents (including children aged below 1 year) with only 28% and 15% of the residents having blood lead concentration ≥10 μg dL^{-1} and >15 μg dL^{-1} respectively. The lower mean BLL among the general population of Nairobi than of pregnant women may be attributed to possible endogenous sources of lead among the pregnant subjects. It was reported that 45%-70% of lead in the blood of reproductive age women were from the long-term tissue stores [Gulson et al., 1995].

In comparison, Kenyan women had higher biological lead burden than their counterparts in other developing countries (Table 1). In the studies conducted between 1989 and 1999, BLLs in Nairobi were higher by a factor of between 2 and 5 except in Tehran, Iran and Durban, South Africa whose values were comparable to those of Nairobi. However, another study in Durban in 1996 when leaded fuel had been phased out in South Africa showed a much lower average BLL of 7.4 μg dL^{-1} [Karimi et al. 1999]. Recent studies reported lower mean BLLs, below 10μg dL^{-1} among women of childbearing age. For example a recent study in Muhumbili Hospital, Dar es Salaam, Tanzania a much lower mean umbilical cord blood lead concentration of 4.1 μg dL^{-1} (range: 0.1-18.1μg dL^{-1}) was determined [Azayo, et al., 2008], which is 10 times as low as the mean of 39.9 μg dL^{-1} reported in cord blood in Kenya [Makokha, 2004]. This suggests higher exposure in Kenya and therefore need for action.

Table 1: Reported blood lead levels in pregnant women in developing countries (1989-1999)

Country	City/Area	No. of subjects	Mean (range) (μg dL-1)	Year of Study	Reference
China	Central Urban, industrial & rural	89	6.8[a] (2.0-24.6)	1997-99	Wang et al. (2004)
India	Lucknow slums	500	14.3 (NR)	1994-95	Awasthi et al. (1996)
Iran	Tehran	80	26.8 (3.7-57.6)	1997-98	Akbarian et al. (2000)
Mexico	Mexico city	668	11.0 (1.3-29.0)	1994-96	Borja-Aburto et al. (1999)
Mexico	Mexico city	2100	11.2 (0.50-58.3)	1991-93	Hernandez-Avila et al. (1997)
Saudi Arabia	Abha, S. Saudi Arabia	172	11.3 (NR)	NR	Mirghani (1997)
Singapore	Urban	36	5.3 (1.4-9.9)	1989	Ong et al. (1993)
South Africa	Durban city	21	21.7 (NR)	1993	Chetty et al. (1993)
South Africa	Durban City	296	7.4 (NR)	1996	Karimi et al. (1999)
Kenya	Nairobi, Eastleigh hosp	152	29.5 (0-295.0)	1998	This study
Kenya	Nairobi, Pumwani Hosp.	71	25.9 (0-131.0)	1998	This study

Note: NR (Not reported in the literature cited); [*]Geometric mean

4.2 Risk Evaluation

The data from our study may suggest that apparent toxic impact of lead could be expected in the subjects. At blood lead level above 40μg dL^{-1} reduced nerve conduction, reduced haemoglobin synthesis (anemia), impaired kidney function and even ancephalopathy may be observed [Fergusson, 1990]. High levels of maternal exposure (>40 μg dL^{-1} or > 25μg dL^{-1} for a period of years) appear to reduce fertility and increase the risks of spontaneous abortion and reduced fetal growth (preterm delivery, low birth weight); maternal BLLs approximately 10μgdL-1 have been linked to increased risks of pregnancy hypertension, spontaneous abortion and reduced offspring neurobehavioral development [Bellinger, 2005]. Although the damage is greatest at higher levels of accumulation, no level has been found to be safe [Hackley and Katz-Jacobson, 2003]. Therefore, in

the present study, perhaps only the 28 mothers in whose bloods lead was not determined could be considered safe from lead effects. However, the fact that no woman had obvious clinical symptoms attributable to very high exposure to lead (encephalopathy), even among the subjects who had BLLs >200 μg dL^{-1} (n=2), agrees with the observation by Goyer (1996) that the signs occur at blood lead concentrations exceeding 300 μg dL^{-1}.

4.3 Possible Sources of Exposure

According to UNEP (2005), people in Kenya can be exposed to lead through many sources including certain foods, especially plant-based foods, industrial pollution, cigarette smoke and contaminated drinking water. The higher blood lead values noted in the present study in Nairobi could be attributed to the heavy automobile traffic and use of petrol containing tetraethyl and tetramethyl lead as an anti-knock additive. Leaded gasoline is widely considered as the main contributor to the exposure of urban populations to lead, in countries where unleaded gasoline was still in use. By the time of the present study, leaded petrol which was to be phased out in Kenya by 2006, was still the only petrol available. Lead may be inhaled with suspended particulate matter or ingested with contaminated edibles. However, according to Tong et al (2000), exposure attributable to miscellaneous sources may be even more significant than universal exposure associated with leaded petrol, especially for people living in poverty.

Traffic together with biomass and waste burning have been implicated as the main sources of air pollution in Nairobi city [Gatari et al., 2005; Gatebe, et al., 1996] and could be a major source of inhaled lead. In a recent study, van Vliet and Kinney (2007) estimated that in Nairobi City, individuals who live, work or travel for long periods on roadways may be exposed, on a daily basis, to upto 44.6 μgm^{-3} of particulate matter that are less than 2.5 μm in diameter ($PM_{2.5}$). Those with only background levels could be taking in upto 20μgm^{-3} per day. However, these fine particles in Nairobi have been found to contain lead in the range of 0.055μgm^{-3}-0.419μgm^{-3} [Gatebe et al., 1996]. From the above data it can be estimated that Nairobi residents exposed to background levels of particulate matter may be inhaling a minimum of >1μgm^{-3} of lead per day. This is a high rate of exposure considering that without prolonged exposure to lead, each 1 μg m^{-3} increase in ambient air lead increases the mean BLL by approximately 1 μg dL-1 [US EPA, 1977]. Atmospheric lead in Nairobi could also be emanating from poor solid waste management which has seen Dandora dumpsite in Nairobi's east lands listed among the "dirty thirty" of the world's most polluted places [Blacksmith Institute, 2007]. Lead may volatilize into the atmosphere during combustion at open dumpsites or leach into groundwater and surface water and enter the food chain. Children from the vicinity of Dandora dumpsite have been found with BLL upto 30 μg dL^{-1} [Njoroge and UNEP, 2005]. Other possible contributors to airborne lead and exposure to the metal in Nairobi may include cottage and manufacturing industrial emissions. Also the following are hazards in individual households: cigarette smoke, burning of paper products, discarded rubber, battery casings, and painted wood for cooking and cooking.

Air inhalation of particulate lead accounts for only 10-30% of blood lead levels and the oral route of lead intake is most significant [WHO/UNEP, 1985]. More than 80% of the daily intake of lead is derived from the ingestion of food, dirt, and dust [WHO, 2003]. Maximum limits for lead content of most food substances are in the range of 0.02-1.0 μg g^{-1} [see WHO, 2000]. These values are considerably lower than typically reported for foodstuffs in Nairobi (Table 2). Of greater concern are the contaminants in maize (*Zea mays*), beans (*Phaseolus vulgaris*) and Kale (*Brassica oleracea var. acephala*; locally known as *sukumawiki)*. A combination of maize, beans and sukumawiki constitutes the most common subsistence meals in Kenya. Considering the large quantities of these food substances consumed, even low levels of lead in them spell high risk of lead poisoning. The high lead contents of the crops suggest ingestion rates of lead in Nairobi could be several factors in excess of the current WHO's provisional tolerable weekly intake of 25 μg g^{-1} [WHO, 2000]. Food lead content is increased when the water used for cooking or cooking utensils contain lead, or the food, especially if acidic, has been stored in lead ceramic pottery or lead -soldered cans [WHO, 2003]. Average lead levels in tap and rain waters in Nairobi have been determined to be below the World Health Organization's maximum safe limits for drinking water of 10 μg L^{-1}, while the mean lead levels in bore-hole and river waters were in excess of maximum limit [Mebratu, 2004]. Ceramics are cheaply obtainable in Kenya and used to serve or store food. However, food canning has reduced following a voluntary change from canning or tinning to plastic packaging in the past two

decades. Enforcements are weak of public health regulations on lead solder, lead paint and lead cosmetics which may be sources of ingested lead.

Lead intake in Nairobi can further increase due to ingestion of lead contaminated soil. A strong positive correlation is found between exposure to lead-contaminated soils and BLLs. Generally, BLLs rise 3-7 µg/dl for every 1000-ppm increase in soil or dust lead concentrations. Access to soil, behavior patterns, presence of ground cover, seasonal variation of exposure conditions, and other factors may influence this relationship [CDC, 1991]. Leaded gasoline, point sources such as manufacturing plants, and deteriorating exterior paint all can contaminate soil. The soil near roads and near the home can contain lead in high concentrations due to lead-based paint or leaded gasoline. Nairobi's soils in the city centre and residential areas and especially in close proximity to roadways have lead levels several factors higher than the WHO recommended levels (Table 2). Individuals who engage in pica are at particular risk. Pica, the deliberate ingestion of amounts of non-nutritive substances, has been identified as a risk factor for elevated BLLs among pregnant women [Abrahams et al., 2006]. In Kenya, it is common for pregnant women and lactating mothers to consume a lot of soil. A study of 275 pregnant women in Kenya revealed that 56% deliberately consumed soil regularly [Geissler et al., 1998]. The soils preferred were collected from hut walls, termite nests and 'soft stones' or from local shops where grey clay is readily available for purchase for the purpose of ingestion. Mean lead level of Kenyan consumable clays has been determined to be 24.5 µg Kg^{-1} [Wanjie, 1991].

The high blood lead levels also suggest there could be occupational exposure. Some women may have been occupied or lived in areas that are likely to receive high levels of lead from the automobile emissions or from factories that produce lead loaded fumes. In Nairobi, it is common to find women selling agricultural produce or textile goods at open-air markets or on the streets in the central business district. These are crowded and dusty locations, with numerous automobiles powered by leaded gasoline and poor exhaust systems. Women working in these areas are likely to suffer lead exposure. Others may be in "high-risk" occupations for lead exposure such as painting, metalwork or battery manufacturing factories. Njoroge and UNEP (2005) reported a mean BLL for the occupationally exposed (Ziwani cottage industries) as 22.6 ± 13.4µg dL-1 and among the workers, 89% had BLLs above 10µg dL-1, which is the action level.

The unusually high BLLs in the present study can partly be explained by factors related to the complexity of the toxikinetics of lead during pregnancy. Some previous reports have noted fluctuations of BLL during the trimesters, with a rise commonly noted during the later parts of the pregnancy to delivery [Moura and Valente, 2002; Hertz Picciotto, et al., 2000]. Others have found an association between blood lead and release from bone stores [Silbergeld et al, 1991]. In Kenya, women with normal pregnancies usually start seeking prenatal care when they are approaching delivery (second or third trimester). It is therefore likely that most subjects in the present study were in their second or last trimesters, when their BLLs were on the rise. Further, the high blood lead levels may reflect the poverty of the subjects. It has been shown that lead absorption is greatly increased during fasting and with diets deficient in iron, calcium and zinc or rich in fats [Mahaffey, 1995; Ratcliffe, 1981]. Having been recruited in public hospitals whose catchment areas include slums of Mathare, Majengo and Mukuru, some mothers in this study might have been from the poorest families in Nairobi, where iron and calcium deficiencies could be common. Njoroge et al (2008) noted a significant negative correlation (r=-0.80) between maternal blood lead concentration and maternal blood iron concentration among Kenyan mothers. It is also possible that some of the women could not afford regular meals, a condition similar to fasting, which may increase gastrointestinal lead absorption [Mahaffey, 1990].

Some 28 samples in this study had lead values below the method detection limit. This can be explained by the fact that, possibly, some of the study subjects may have not been residents in Nairobi and so travelled from less contaminated environments on the sampling day. Considering atmospheric lead levels in Nairobi were below the WHO standard in the 1990s [Gatari et al, 2005; Gatebe et al., 1996] it was possible to have such low levels given that BLLs reflect only recent exposure.

4.4 Limitations of the Study

It should be pointed out extreme blood lead values (<0.02-295.0 µg dL^{-1}) observed in this study were rather unusual, and may be attributed to the use of blood as a biomarker. One of the disadvantages of blood as an indicator has been noted to be that it is a circulatory system which deposits the lead particulates in various body organs within hours [Williams et al., 1998].

Therefore the times that the samples were taken may had had some effect on the levels that were actually determined.

Primary management of lead toxicity is source identification and exposure cessation. One weakness of the present study is perhaps that it was exploratory in nature and so did not investigate possible sources of exposure to lead or mitigating factors for low blood lead levels. As such, it is not possible to tell from our results whether the women were occupationally exposed or if the high levels observed were due to dietary or environmental sources. In addition, we did not include a control group. The addition of a control group, perhaps of pregnant women living in a rural setting would, probably, have provided evidence about the possible main sources of maternal lead during pregnancy in Nairobi. Similarly, inclusion of non-pregnant women from Nairobi may provide evidence about the behaviour of lead during pregnancy and explain the high or very low BLLs observed for the pregnant subjects. Having recruited subjects from public hospitals, which serve mainly mothers from low income groups, it is possible that women from higher socio-economic status were not included. It would therefore erroneous to assume that the subjects in this study were representative of pregnant mothers in Nairobi city.

In view of the above weaknesses of the present study, we strongly recommend that a more detailed study be conducted involving a sample size sufficient to permit any complex statistical analyses. The study should take into account previous findings about the possible sources of lead exposure as well as hygienic and dietary factors that may affect blood lead levels. There is also need to involve women from private hospitals and/or high socio-economic class in order to elucidate the extent of exposure to lead by women in Nairobi.

Table 2: Lead concentrations (μg g^{-1} or μg L^{-1}) reported for soil and foodstuffs from sites in the city centre and suburban of Nairobi

Substance[1]	Max. limit[a]	City centre sites		Suburban sites		Reference
		Mean	Range	Mean	Range	
Soil	0.1	44.6	14.9-196.0	16.0	12.5-18.4	Dickinson et al., 1987
		659.0	137.0-2196.0	624	148.0-4088.0	Onyari et al. 1991
	0.1	265.9	96.2-663.5	44.4	36.8- 63.9	Mebratu, 2004
Maize	0.2	1.9	1.6-2.2	1.0	0.7-1.7	Mebratu, 2004
″	″	1.7	1.4-1.9	0.8	0.6-1.5	Makokha, 2004
″	″	113.6[c]	-	126.8	-	Dickinson et al., 1987
Sorghum	0.2	-	69.9-77.5[b]	59.7	-	″
Sugarcane	-	-	-	14.8	-	
Cassava	0.1	82.6	-	26.7	-	″
Sweet potato	0.1		10.8-45.0[b]	69.5	-	″
Cocoyam	0.1	35.8	-	84.3	-	″
Kale	0.3		13.4-10.3[b]	46.6	-	″
	0.3	4.8	2.9-5.7	1.5	1.2-1.7	Makokha, 2004
Beans	0.2	2.4	2.2-2.5	1.1	0.8-1.6	″
Beans (leaf)	0.2	-	12.9-113.7[b]	24.3	-	Dickinson et al., 1987
Beans (seed)	0.2	-	34.8-35.8[b]	31.1	-	″
Pigeon peas	0.2	-	-	34.1	-	″
Cowpeas (leaf)	0.2	-	7.7-39.6[b]	21.3	-	″
Cowpeas (seed)	0.2	-	8.5-10.5[b]	127.2	-	″
Broad beans	0.2	49.5	-	77.7	-	″
Peas	0.2	64.8	-	-	-	″
Banana	-	-	19.3-23.3[b]	29.5	-	″
Tomato	0.2	1.6	1.6-1.7	1.1	0.7-1.7	Makokha, 2004
Spinach	0.3	4.5	3.8-5.4	1.5	1.2-1.7	″
Meat of cattle	0.05	-	-		0.16-0.39	Oyaro et al., 2007
Tap water	10			6	2-8	Mebratu, 2004
Rain water	10			6	1-10	″
Bore hole water	10			13	3-40	″
River water	10			19	3-35	″
Milk	20			46	30-80	″

Note: [a] maximum safe limit [WHO, 2000]; [b] lead concentrations for two sites; [c] lead concentration for one site; - not reported in the literature cited; [1] raw edible portions for crops; liquids measured in μg L^{-1}

4.0 Conclusion

Our results showed that blood lead concentrations measured in women during pregnancy were rather high, as more than 70% values were above 10 μg dL^{-1}, which is currently considered to be the benchmark for intervention. The samples from Eastleigh had slightly higher mean lead concentration than those from Eastleigh clinic, but Student t-test confirmed no significant difference between them. It can therefore be concluded that the high BLLs in both hospitals could be from similar sources of exposure. In addition, our findings indicate that in a developing country like Kenya, there are probably many unidentified or undiagnosed cases of lead poisoning. Clearly, this work gives a rationale for deeper investigation in order to identify the sources of lead exposure in Kenya. It may serve as a wake up call for the Kenya Government to implement programmes aimed at reducing the blood lead levels of reproductive age women so as to minimize transfer of maternal lead into the foetus and nursing infant.

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