Acute Lead Poisoning Associated with Backyard Lead Smelting in Jamaica

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ABSTRACT

Long term backyard smelting of lead in a district known as Mona Commons, Kingston, Jamaica, has produced lead burdens as high as 30 000 mg/kg in soils near to the smelter, and indoor dust loadings of 373 μg/f² in the residents' home. The blood lead levels (BPb) of 107 children from the district were in the range 2.2–202 μg/dL. Fifty-nine per cent of these had BPb levels above 10 μg/dL and the population mean was an unacceptably high 25.1 μg/dL. The highest levels were observed for five siblings, two of whom presented with lead encephalopathy. This severe chronic exposure to lead was exacerbated by a significant history of pica, and chronic nutritional anaemia. Chelation therapy significantly reduced the BPb levels but due to lead storage in other organs, the values after several months were still higher than desirable. This study emphasizes the importance of reducing the exposure of children to lead.

Envenenamiento Agudo con Plomo Asociado con la Fundición Casera de Plomo en Jamaica

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RESUMEN

La fundición casera a largo plazo de plomo en el distrito conocido como Mona Commons, Kingston, Jamaica, ha producido una alta acumulación de plomo, ascendente a 30 000 mg/kg en los suelos cercanos al horno de fundición, y la concentraciones interiores de 373 μg/f² de polvo en los hogares de los residentes. Los niveles de plomo en sangre (PbS) de 197 niños del distrito estuvieron en el rango de 2.2–202 μg/dL. El cincuenta y nueve por ciento de ellos, presentaron niveles de PbS por encima de 10 μg/dL, y la media poblacional tuvo 25.1 μg/dL, lo cual representa un nivel inaceptablemente alto. Los niveles más altos se observaron en cinco hermanos, dos de los cuales presentaron encefalopatía plúmbica. Esta exposición crónica severa al plomo, estuvo exacerbada por una historia de pica y anemia nutricional crónica. La terapia de quelación redujo significativamente los niveles de PbS, pero debido al almacenamiento de plomo en otros órganos, los valores luego de varios meses se hallaban todavía por encima de los límites deseables. Esto resalta la importancia de reducir la exposición de los niños al plomo.

West Indian Med J 2006; 55 (6): 394

INTRODUCTION

Lead exposure has been associated with numerous maladies including cognitive and behavioural deficits, hypertension, osteoporosis, and a range of non-specific constitutional symptoms (1). Lead is also a potent neurotoxin especially detrimental to the developing nervous system of the fetus, babies and young children (2). Most children with elevated blood lead levels are asymptomatic, but irreversible damage to the developing brain leads to a lowering of IQ and there are substantial statistical correlations between behavioural problems and the consumption of leaded gasoline over years (3). The risk of lead poisoning is especially high for children in poorer circumstances since exposure is also likely to be higher and accompanied by iron and calcium deficiencies that result in higher blood lead concentrations (4). Improved understanding of the dangers of lead exposures to young children have led to the reductions in the action levels recommended by the Centers for Disease Control (CDC) and summarized in Table 1.

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Table 1: Changes in CDC's blood lead levels of concern (μg/dL), *1960–1991*

Year	1960	1970	1975	1985	1991	Future
Level of concern	60	40	35	25	10	<5

This reduction over some 30 years, by a factor of six, reflects the growing concern about the effects of low concentrations of lead on brain development and as recent work shows, there now appears to be no safe threshold of exposure (5) and it appears that the cognitive development of children may be impaired at levels below 5 μg/dL (6).

Beginning with the report of the "Dry Belly Ache" which afflicted the English garrison in 1786 (7) when soldiers imbibed rum stored in leaden casks, there have been several reports of lead poisoning in Jamaica, mainly among children. Most of the severe cases have been a result of backyard smelting for the recovery of the lead content of lead-acid batteries. These are still being observed (8). The work reported in this paper is part of a programme to examine the blood lead levels of children islandwide, and deals primarily with severe lead poisoning and lead pollution in a residential district known as Mona Commons, near to the University Hospital of the West Indies (UHWI).

SUBJECTS AND METHODS

The study was approved by the UHWI/UWI Faculty of Medical Sciences Ethics Committee. Informed consent was obtained from parents/guardians.

Blood sampling and analysis

One hundred and seven children, virtually all those in the age range 2–12 years in Mona Commons, were examined in a door-to-door survey of blood lead levels, using finger prick blood samples. Values above 70 mg/dL were confirmed using venous samples. The analyses were carried out by anodic stripping voltametry using a LeadCare Analyzer (9). Quality control was maintained by frequent comparison with standard reference materials obtained from the National Institute of Standards and Technology. The comparison measurements agreed to within ± 2.5%.

Chelation Therapy

Chelation therapy was carried out mainly with British-AntiLewisite (BAL) and calcium disodium ethylene diamine tetra-acetate ($CaNa_2EDTA$), the recommended treatment for children who present in an acute encephalopathic state. Oral succimer was used for outpatient treatments. Four (two sets of twins) of the five siblings were hyperactive and at times aggressive, although formal developmental assessments are yet to be done, they were also not as verbally expressive as children of their own age. The oldest sibling was assessed as functioning at one year below her chronological age, but other factors *eg* genetic cognitive potential and other environmental influences may also contribute to this.

Three chelation treatments were performed over a period of about two months accompanied by regular BPb checks. After discharge, BPb measurements were continued over a period of five months.

Environmental sampling and analysis

Thirty soil samples were collected in Mona Commons using a 15m x 15m grid. Three composite dust samples were collected from the epiphytic plant, *Tillandsia recurvata*, growing on trees near to the contaminated areas, and the lead content of the adhering dust measured. The soil and dust samples were analysed by X-ray fluorescence (XRF) using a Kevex EDX-771 spectrometer. Indoor dust from the floor in the children's residence was collected from a 25 x 25 cm area using wipes. The wipes were digested in 25% nitric acid and the solution analysed by a Palintest SA 5000 anodic stripping voltametry system (10). The lead contents of foods and water were examined by atomic absorption spectrometry.

RESULTS

Medical evaluation and treatment

Blood lead levels results for a total of 107 children from the Mona Commons are summarized in Table 2.

Table 2: Blood lead levels of 107 children from Mona Commons

BPb Levels (μg/dL)		CDC Classification				
Range	Mean	I (<10)	II (10–19)	III (20–44)	IV (45–-69)	V (>70)
2.2–202	25.1	44	19	30	9	5

As many as 59% of these children showed BPb levels above the CDC intervention level of >10 μg/dL and the population mean is unacceptably high. Of these, 48 children received iron supplements; eight with BPb levels between 40 and 60 mg/dL were referred to the paediatric outpatient clinic at the UHWI. One child continued to visit the clinic for over a year and received oral chelation with succimer, with an overall decline in the BPb level of only 9%.

The five children with the highest BPb values (ranging from 89 μg/dL to 202 μg/dL), well above the present CDC emergency level of 70 μg/dL, were admitted to the UHWI. Two presented with lead encephalopathy; one of these (BPb = 126 μg/dL) went on to have status epilepticus, which is common in children with BPb above 100 μg/dL but has also been seen at levels of about 70 μg/dL (11). These children required acute seizure management prior to the initiation of chelation therapy. The severe chronic exposure of these children to lead was the result of backyard lead smelting exacerbated by significant pica and chronic nutritional anaemia. The radiograph shown as Fig. 1, of (A) upper limbs and (B) lower limbs of one of these children illustrates a marked

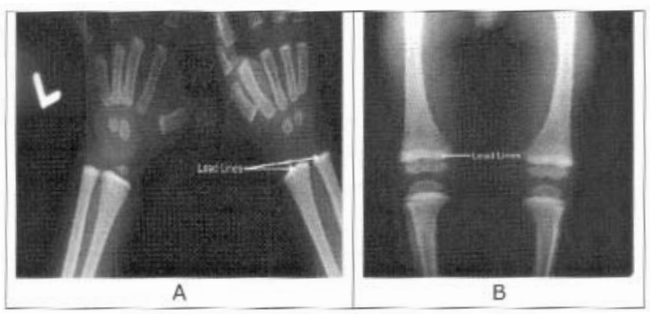

Fig. 1: X-ray images showing lead in bones of one of the lead poisoned children (Mo4).

linear increase in the density of the metaphyses, attributed to the significant lead uptake that had already occurred in the bones.

Chelation Therapy

A summary of the BPbs on chelation during hospitalization is shown in Table 3.

Table 3: Summary of chelated children's blood lead levels over a three-month period

		Blood Lead Levels (µg/dL)		
Identifier	**Age (mo)**	**Initial**	**On discharge**	**% Reduction**
Mo1*	30	126	22	83
Mo2*	30	158	28	83
Mo3!	20	202	25	87
Mo4!	20	99	26	73
Mo5	60	89	13	85

*Identical twins !fraternal twins; mo = months

The variations of BPb levels of the five children after chelation during hospitalization are shown in Fig. 2.

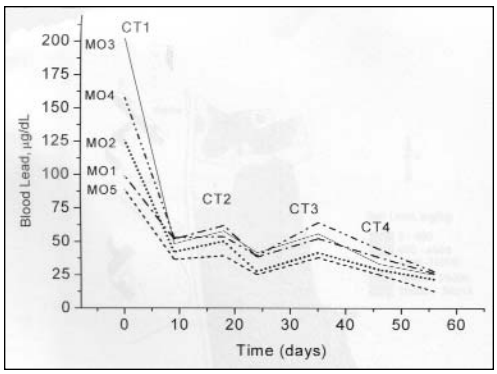

Fig. 2: Blood lead levels of the five emergency cases before and after chelation therapy treatment.

Overall, the time variation patterns of BPb were similar in each child. After the first chelation, the levels fell by 46–76% and initially tended to increase between chelations (12). Twenty-one days after the third chelations the BPb levels were 13–28 µg/dL at which time the children were discharged from hospital. During the following weeks, there were significant increases in BPb shown in Table 4.

Table 4: Variation of blood lead levels (mg/dL) with time after discharge

	Days after discharge						
	0	**18**	**44**	**74**	**133**	**157**	**244***
Identifier			Blood Lead				
Mo1	25	45	57*	33		59	56
Mo2	22	43	41		45		49
Mo3	26	28	52*	42		51	58
Mo4	28	38	31				54
Mo5	13	38	31		39		42

*Chelated with oral succimer as outpatients at this point

On discharge, the children were placed in a government home for the care of children where they were monitored. Oral chelation treatment was provided with succimer for those whose BPb level had risen above 50 µg/dL. After the last chelation treatment, the children were returned by the parents to the original residence. Further follow-up was indicated.

Environmental evaluations

Some environmental assessment results for Mona Commons are summarized in Table 5.

Table 5: Lead contents of environmental samples in Mona Commons

Sample Type	**Number of samples**	**Range**	**US EPA safe limit**
Soil (mg/kg)	30	45–30 000	400
Aerial dust (mg/kg)	3	12–1255	–
House dust (µg/f^2)	1	373	40

The soil samples at the home of the lead-poisoned children and the immediate surroundings showed levels as high as 30 000 mg/kg. The range of soil lead concentrations in Canada and many European countries in 25 – 100 mg/kg (13). The lead concentration in uncontaminated Jamaican soils is 44 mg/kg (14). The dust sample collected from the floor of the children's home showed lead concentration of 373 µg/f^2, nearly ten times in excess of the United States Environmental Protection Agency PA limit of 40 µg/f^2. The lead content of the drinking water was below the sensitive detection limits of atomic absorption spectroscopy and that of all Jamaican foods so far examined is also low (15). The contaminating effect of the lead smelting operation in Mona Commons is illustrated in Fig. 3 by the concentration distribution of lead in the soils.

The extent of lead contamination is obvious; some 50% of the soil area had lead soil concentrations greater than 400 mg/kg, the US EPA lead limit for residential soils (16). The area near to the smelting operation, including the five lead-poisoned children's yard, was grossly contaminated, and despite the high density of lead and its compounds, even at

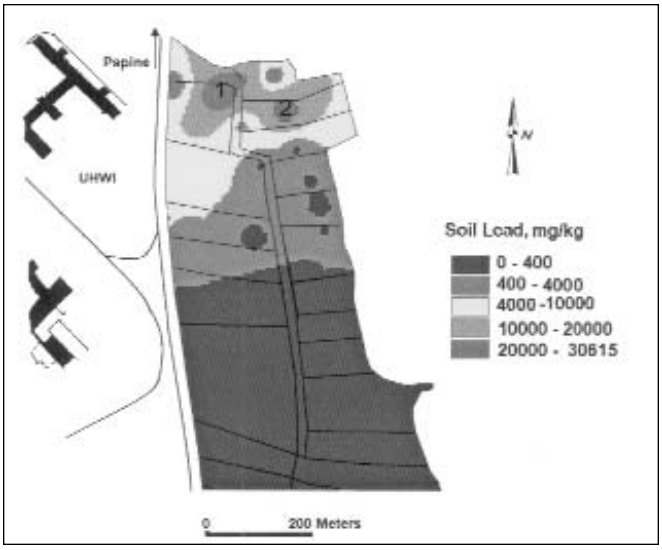

Fig. 3: Distribution of lead in Mona Commons' soil.

30 feet above ground level there were quite significant concentrations in the outdoor dust.

DISCUSSION

The standard treatment for lead poisoning at BPb levels in the emergency range is chelation therapy. This can quickly reduce the levels but because of releases from stores of lead in bones and other tissues, these do tend to rebound. For comparison, Table 6 shows some BPb data for two similarly

Table 6: Blood lead levels of two children from Maverly before and after chelation treatment

		Blood lead levels mg/dL			
Identifier	**Age (years)**	**Before CT 2003–11–7**	**20 days after CT**	**CT date 2004–7–21**	**5 days after CT**
M1	3	135	67	186	90
M2	9	61	45	56	23

CT = chelation therapy

lead-poisoned siblings, then aged three and nine years respectively, from another district who received similar therapy on two occasions at the Bustamante Hospital for Children.

Twenty days after the first chelation, the BPb levels had been lowered by 50% and 74% respectively and the children returned to their still-contaminated home. Within eight months, the BPb level of the three-year old, who was known to practise pica, had more than doubled, and that of the older had also increased significantly. Five days after the second chelation, their BPbs were 90 µg/dL and 23 µg/dL. At this stage, the yard was covered with marl and cemented over, but one year after this was done, their BPbs remained high at 89 and 45 µg/dL respectively. It would appear that these children were still receiving some exposure to lead and further follow-up was indicated.

These BPb results are less favourable than some, shown in Table 7, obtained without chelation on children in

Table 7: Blood lead levels of children from Kintyre and Fraser's Content (µg/dL)

	No. of children	**Range**	**Mean**
Kintyre			
Before	26	13–60	37.5
After 10 months	26	7–27	14
Fraser's Content			
Before	4	35–61	52
After 17 months	4	20–32	28

Kintyre and Fraser's Content in Jamaica. In Kintyre, mitigation was accompanied with iron and calcium nutritional improvements and the children were isolated from further exposure to lead (17).

Lead in the body is complexed by a variety of proteins including glutathione, and excreted in the urine (18), though at highly variable rates, so that even in the absence of chelation there can be significant BPb reductions reflecting environmental improvements.

These comparisons illustrate the potential complexity due to children's exposure, behaviour and responses. In Kintyre, the exposure was from mine-waste in the form of the insoluble sulphide, galena. In the other cases, it was from the oxide and lead sulphate in batteries and metallic lead and its vapour from smelting. Nevertheless, at the high lead concentrations that occur in some local environmental situations, lead poisoning is almost inevitable, although the necessary mitigation can be relatively simple.

The storage of lead in various body compartments adds to the complexity. For children, lead absorption is mainly *via* the gastrointestinal tract into the blood plasma from which it accumulates in the soft tissues (brain, kidneys, liver, and muscle) and especially in the skeleton, which contains approximately 73% of the body burden in children (18, 19). The BPb values are largely controlled by a series of equilibria between the compartments and the various reaction rates, neither of which are precisely known, and excretion. These determine the rates of increase of BPb after chelations.

As illustrated in Table 8, the half-life of lead in various adult tissues vary widely (20). Much less information is

Table 8: Half-lives of lead in adult human tissues (2)

Compartment	**Blood**	**Soft Tissues**	**Trabecular Bone**	**Cortical Bone**
Half-life	12–30 days	7–73 days	1.2 years	9.3–59 years

available on the kinetics of lead exchange in children, but these are likely to follow a similar pattern.

Although the blood lead levels are quickly lowered on chelation with $CaNa_2EDTA$, lead from the other compartments contributes to the generally slower increases shown in Fig. 2, to an extent controlled by the relative half-life values. The early contribution is determined mainly by lead release from the soft tissues and the labile component in trabecular bone, which exchanges with the blood; in cortical bone there is a much more inert pool. Such exchanges necessitate repeated courses of treatment, and even after complete removal from the source of lead exposure, the blood lead levels may increase significantly over time. These exchanges can impact the blood-lead levels of young children for a considerable period, even when each intervention is highly effective in reducing children's lead exposure (22).

Unfortunately, BPb levels that are well above the CDC recommended level are still being found in children in Jamaica, and there is now a growing consensus that there is intellectual impairment in children with BPb levels below the presently accepted 10 μg/dL (23). It does seem that more efforts ought to be made to eliminate a problem that is entirely preventable. This will require the cessation of lead smelting in communities.

In summary, although there is an increased appreciation in Jamaica of the dangers, backyard lead smelting is still being carried out. As such, there remains a substantial number of chronically lead poisoned children who require suitable clinical and environmental interventions. The consequent social and financial burdens, reduced intelligence and violent behaviour place demands on society that are far from trivial and are quite unnecessary since lead poisoning is an entirely preventable malady. This is becoming better understood and there are increased efforts to manage lead pollution in the Jamaican environment.

Medical and environmental interventions produce significant reductions in blood lead levels but such interventions, to be effective, do require longer term monitoring and support.

ACKNOWLEDGEMENT

This work was supported by the Environmental Foundation of Jamaica. Acknowledgements are also made to the staff of the Bustamante Hospital for Children. Mr Hydda McPherson, public health inspector of the Ministry of Health, Mr Kennett Blake from Kingston and St Andrew Health Department, Mrs Marva Brisett, Department of Community Health and Psychiatry, The University of the West Indies, Ms Kameaka Duncan, and Ms Pansy Matthews, principal of the Mona Commons Basic School.

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