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Blood lead levels in Dunedin 11 year old children

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Abstract

Blood lead levels were determined for 579 eleven year old children. The range of blood lead levels was from 0.19 to 2.41 μmol/l with a geometric mean of 0.49 μmol/l (geometric SD 0.07) and an arithmetic mean of 0.54 μmol/l (arithmetic SD 0.24). Two children had levels above 1.45 μmol/l. There was no significant correlation between blood lead levels and socio-economic status. Ten children with elevated blood lead levels (greater than 1.11 μmol/l) were reassessed and the results from all but one child had returned to a lower level. In nine out of ten of these cases recent inside paint stripping activities had been carried out in the child's home.

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Introduction

The problem of environmental contamination from lead and its possible effects on health are a matter of concern to the general public and health professionals alike. While acute lead poisoning is now relatively rare, evidence has been advanced to suggest that there is a small but significant association between body lead burden and cognitive deficits and behaviour problems in children, even at levels well below the accepted thresholds for frank poisoning. This evidence, however, is by no means universally accepted because of differences in methodologies and conclusions from various studies [1]. In Great Britain, mounting public interest in, and concern about, lead and health has resulted in two recent important investigations and public reports on the problem of lead and health [1,2]. Also, a number of recent reviews on the state of knowledge concerning lead and its effect on health, particularly child health, have appeared [3-6].

A number of important studies of blood lead levels have been carried out. As a result of a European Economic Community directive [7], the United Kingdom was required to carry out two series of surveys, the first in 1979 and the second in 1981. The surveys showed that the arithmetic mean blood lead concentration was 0.62μmol/l in inner city populations and 0.53μmol/l in outer city populations. Overall, 3.1% of the inner city population and 1.9% of the outer city population were reported to have blood lead levels greater than 1.21μmol/l. A related study, as part of the response to the EEC directive [8], involved a sample of 166 London children from predominantly low social class, who lived near a lead smelter. The children's age varied from 6 to 12 years, with a mean of 8.5 years. The arithmetic mean blood level was 0.65 μmol/l.

The largest survey reported was part of the US second National Health and Nutrition Examination Survey (NHANES II) [9]. The sample comprised nearly 10 000 people aged between 6 months and 74 years from across the country. The arithmetic mean blood lead concentration was 0.67 μmol/l with 1.9% having blood lead levels greater than 1.45μol/l. The data were collected between 1976 and 1980, and over that period there was a 37% decrease in blood lead levels from an arithmetic mean of 0.76μmol/l to 0.48 μmol/l. Annest [9], in his report of the NHANES II results, suggested that the reduction in mean lead levels over the five year period could have reflected increased community awareness programmes on lead toxicity, federally funded screening programmes, occupational safety programmes, efforts to remove lead from diet, and/or a phase down in the use of lead in petrol. The correlation between blood lead concentrations and the total lead used in petrol was 0.95 over the five year period.

There has been only limited research in New Zealand on lead levels in samples from the general population. The studies that have been carried out have used samples of children as they are generally believed to be most at risk for adverse effects of high lead levels [1]. Two reports on blood lead levels in children have come from Auckland [10,11]. Small groups (n's = 13-48) of preschool children in differing locations have been studied. The geometric mean blood lead levels range from 0.56 μmol/l in Glenfield, a new suburb, to 1.07 μmol/l in Freemans Bay, an old inner city suburb. The

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Auckland research group has recently completed a further series of studies in a variety of other geographic locations in Auckland [Kjellstrom T, personal communication]. The only other New Zealand study of a sample from the general population was reported from Christchurch by Shellshear and others [12]. They studied the blood lead levels of 170 preschool children and found an arithmetric mean blood lead level 0.84 μmol/l (range 0.19 μmol/l to 8.21 μmol/l). Sixteen children (9.4%) had blood lead levels above 1.69 μmol/l, the level above which follow up investigation is recommended in the United Kingdom [7] and 21 children (12.4%) had levels above 1.45 μmol/l, the level at which children in the USA are recommended for follow up investigation [13].

This study reports the blood lead levels of 579 Dunedin 11 year old children. It then describes associations between blood lead levels and socio-economic status. Details are also provided on the stability of lead levels and the likely cause of elevated lead levels in those children with the highest levels at initial testing.

Method

Sample: The blood lead study sample comprised 579 or 63% of the 925 children assessed as part of the eleven year follow up of the Dunedin Multidisciplinary Health and Development Study. The study sample is known to be over-representative of the higher socio-economic levels and under-representative of Maori and other Polynesian children. The sample and the study have been described in detail elsewhere [14].

Of the 925 children either fully or partially assessed within two months of their eleventh birthdays, 803 attended the research unit. The remaining 122 children were assessed in other parts of New Zealand or overseas. Thus, the 579 children who participated in the blood lead study were 72% of the sample assessed at the research unit as eleven year olds. Ninety percent of the sample lived in the Dunedin metropolitan area. More detailed information on the sample included in the blood lead study compared with the those not included is given in the results sections.

Measures: All whole blood samples were analysed by one of the authors (PH), utilising the technique of graphite furnace atomic absorption spectrophotometry as described in detail elsewhere [15]. Particular attention was paid to contamination control. The reliability of the method was checked with a laboratory in Southampton and the correlation between independent assessments was 0.97. The method of analysis and quality assurance checks indicate that the Dunedin results can be compared with those from outside New Zealand where similar strict quality control is maintained. The blood lead results are reported as micro-moles of lead per litre of whole blood (μmol/l).

The socio-economic status of the sample was assessed by use of the index of socio-economic status for New Zealand [16]. This is based on the median income and education for particular occupations; in the present study the fathers' occupations when the children were eleven years of age were used.

Blood collection: The children were supine during venepuncture. A tourniquet was used. A disposable plastic 30 ml Terumo syringe was filled via a 21G 38 mm needle (Terumo Neolus). On completion of sampling, the needle was removed from the syringe and the first 2 ml of blood ejected into a polypropylene tube containing lead-free heparin. The tube was then capped and mixed by inversion. Tubes were stored at -20°C until analysed. All blood specimens were obtained between 8.45 and 9.45 am. The remainder of the blood was distributed to the appropriate tubes for other studies. Further details on the methods of blood collection and analysis and quality assurance studies are given elsewhere [15].

Follow-up: All children with initial lead levels of greater than 1.11 μmol/l and their families were brought back for further venepuncture between 3 and 12 weeks after the first assessment. This blood lead level is equivalent to the 98th percentile point on the distribution of results for the children in this study. At the same time, the parents and child were questioned to determine the possible source of the lead.

Expression of the results: Because the blood lead results were not normally distributed but conformed to a lognormal distribution, results are expressed as geometric means and standard deviations. However, in order to compare the results with others expressed as arithmetic means, these are also included.

Results

For the 579 eleven year old children, the range of blood lead levels was from 0.19 to 2.41 μmol/l with a geometic mean of 0.49 μmol/l (geometic SD 0.07). The arithmetric mean was 0.54 μmol/l (arithmetric SD 0.24). The geometric mean for 311 boys was 1.09 μmol/l (geometric SD 0.07; arithmetric mean 0.55; SD 0.22) and for 268 girls the geometric mean was 0.47 μmol/l (geometric SD deviation 0.07; arithmetric mean 0.50, SD 0.22). The sex difference was statistically significant ($p < 0.01$). Nine children (1.6%) had blood lead levels greater than 1.21 μmol/l and two children had blood lead levels greater than 1.45 μmol/l; one was 1.50 μmol/l and the other 2.41 μmol/l.

The distribution of results for the sample (omitting the child with a high lead level) is depicted in Figure 1. The figure shows the lognormal distribution of results with a small hump in the distribution from 0.97 to 1.45 μmol/l.

Socio-economic status: Table 1 sets out the socio-economic status distribution for the blood lead study sample, the remainder of the Dunedin Multidisciplinary Health and Development Study sample, and the New Zealand urban population socio-economic status distribution. A chi square test showed that the Dunedin blood lead study sample distribution differed significantly from that in the remainder of the Dunedin sample in socio-economic status (χ^2 12.7; d.f. 5; $p < 0.05$). Inspection of the table shows that the blood lead sample had more members of the socio-economic status groups 1 and 4 but fewer members at other levels.

Comparison of the blood lead study sample with the New Zealand male work-force socio-economic status distribution showed greater differences. The Dunedin blood lead study sample was over-represented in the three highest socio-economic status levels and under-represented in the three lowest socio-economic status levels, particularly in the two lowest groups.

Table 1.—A comparison of the socio-economic status of those included in the blood lead study sample, the remainder of the Dunedin study sample, and the New Zealand urban distribution.

	Dunedin blood lead study sample		Remainder of Dunedin study sample		NZ male workforce*
	n	%	n	%	%
SES Level					
1 (High)	84	16.7	32	11.6	7.0
2	92	18.3	60	21.8	14.0
3	137	27.3	85	31.0	28.0
4	148	29.5	62	22.5	29.0
5	28	5.6	26	9.5	14.0
6 (Low)	13	2.6	10	3.6	8.0
Not known	77	–	71	–	–
Total	579	100.0	346	100.0	100.0

SES = socio-economic status
* From Johnson[17]

Table 2.—First and second results from children with elevated blood lead levels.

Child No	First blood lead level (μmol/l)	Second blood lead level (μmol/l)
1	1.16	0.63
2	1.21	0.77
3	1.25	0.34
4	1.25	0.29
5	1.30	1.21
6	1.30	0.82
7	1.30	0.68
8	1.45	1.25
9	1.50	1.25
10	2.41	0.53

Because the blood lead levels conformed to a lognormal distribution, a logarithmic transformation was undertaken to estimate the correlation with socio-economic status. The resulting correlation was −0.02 which was close to zero and not significant. This indicated that there was no association between variations of socio-economic status and blood levels in this sample.

Children with elevated blood lead levels: The parents of children with initial blood lead levels greater than 1.11 μmol/l (n = 12) were contacted and invited to bring the children to the Dunedin Hospital for a follow up blood analysis. Ten of the 12 children were reassessed 3 to 12 weeks after the first blood sample was taken. The results from both tests for the sample children are set out in Table 2.

The table shows that in the second assessment, every level was lower and only three remained above 1.11μmol/l. These three children were again invited to come for a further assessment and two accepted (child number 8 and number 9). Child no 9 had a blood lead level of 0.72μmol/l on reassessment but child no 8 still had an elevated blood lead level of 1.35μmol/l. This child has since been rechecked every three months through the general practitioner but her blood lead level remains in the range betweeen 1.11 to 1.45 μmol/l. No cause has been found and no other family member has had blood lead levels in excess of 0.97 μmol/l.

Every other member of the households of each of the 10 children with an initial lead level greater than 1.11 μmol/l also had their blood lead levels analysed but no other individual had a blood lead level in excess of 0.97 μmol/l.

The parents and the children who were followed up were asked about any activities the child and/or family were involved in that may have resulted in exposure to lead. All except the child whose lead level has remained persistently high, reported current or very recent paint stripping activities inside houses. Appropriate advice was given to all families of the hazards of paint stripping and protective methods outlined.

The father of child no 10, whose initial blood lead level was 2.41 μmol/l, reported that their cat had died and that a veterinarian had indicated that the cause of death was lead poisoning. The father and daughter were living alone in the house at the time and the father was involved in paint stripping, but did take precautions to protect himself from inhalation of dust. He was not aware of the need to protect his daughter. Hence, the father's blood lead level was below 0.97 μmol/l while the child's level was high.

Discussion

As pointed out by the Royal Commission on Environmental Pollution [1], studies of lead in man are needed and it is essential that they use reliable methods of analysis. Particular attention was paid to this issue in the present study and care was taken to examine the reliability of the Dunedin assessments by having some results checked with a laboratory at the University of Southampton. The resulting high agreement between the two sets of results indicates that the Dunedin results are comparable with values assayed in other places that used strict quality control.

The arithmetic mean blood lead level of 0.54 μmol/l from the Dunedin sample was very similar to the British outer city population level (0.53 μmol/l) and a little lower than the inner city population level (0.62 μmol/l). One point six percent of the Dunedin sample had blood lead levels greater than 1.21 μmol/l in comparison with 1.9% for the British outer city populations and 3.1% for the inner city populations [1]. Dunedin had a lower mean blood lead level than that reported from London for 6-12 year old children [8]. Their distribution of results (ie lognormal) appears very similar to the Dunedin distribution. In comparison with the NHANES II [9] overall mean blood lead level of 0.67 μmol/l the Dunedin levels were lower. Altogther, 1.9% of the total American sample had blood lead levels greater than 1.45 μmol/l while there were only two such high levels in the Dunedin sample (0.3%). The Dunedin results were a little higher than the last year's results from the NHANES II study, where they recorded a blood lead arithmetic mean of 0.48μmol/l. Overall, the results from the Dunedin study showed that this sample tended to have slightly lower mean lead levels than those reported from the United Kingdom and the USA and considerably fewer children in the higher lead level groups.

It is not known whether progressive changes in methodology and improvements in sample collection may contribute to assay drift, or whether growing awareness of and action on the problem of lead in biosystems is reducing human exposure and thus the assayed values. Problems in standardisation of lead assays in the past are now regarded as being of sufficient magnitude to require re-evaluation of much of the older literature. In the event that a true fall in the mean lead levels has occurred, it will be important to evaluate whether this arises mainly from a reduction in the number of subjects in the upper range of lead values, or from a proportional shift downwards in the majority of the population. Depending on the number in a sample, the arithmetic mean can be particularly susceptible to small changes in the number of individuals with high values. If the number of individuals with high lead levels is reduced, this would represent a major public health gain for those most likely to experience adverse effects from body lead burden. If there has been a slight drop in lead levels by the majority of the population, this would indicate a small general improvement, but the frequency of individuals with higher levels may not have changed significantly. Further research on these issues is needed.

The Royal Commission on Environmental Pollution [1] pointed out that the concept of a threshold concentration of lead in the body below which there are no harmful effects is misleading and that it is not possible to give a meaningful value for a threshold concentration. However, the European Economic Community directive defined reference levels which, if

exceeded, had to be reported and had to trigger action to identify and reduce the exposure [7]. These reference levels were that no more than 2% of any group should have a blood lead concentration greater than 1.69 μmol/l, no more than 10% greater than 1.45 μmol/l, and no more than 50% above 0.97 μmol/l. In the Dunedin study, only one child had a blood lead concentration of greater than 1.45 μmol/l and only two children had a blood lead concentration greater than of 1.69 μmol/l. Five percent of the Dunedin sample had blood lead levels exceeding 0.97 μmol/l. Thus, the Dunedin results fell well below the European Economic Community directive's reference levels. Only one child in the Dunedin sample was above the 1.69 μmol/l level recommended by the European Economic Community directive as needing follow up investigation.

The Dunedin blood lead concentrations (geometric mean 0.49 μmol/l) were similar to those recorded for the newer suburbs such as Glenfield (0.56 μmol/l) by Kjellstrom and colleagues [11] but considerably lower than that found in the older suburbs such as Freemans Bay (1.07 μmol/l). The most recent work of the Auckland group (personal communication) should provide more information on some of the factors that account for variations in blood lead levels in their samples as they have selected their samples from a variety of areas and studied possible sources of lead. The Christchurch studies of Shellshear and others [12] found a considerably higher arithmetic mean lead level than Dunedin (0.84 μmol/l) and a large proportion of children with blood lead levels in excess of 1.45 μmol/l (12.4%) and 1.69 μmol/l (9.4%). That survey was carried out in 1973 and should now be repeated as the Christchurch figures breached the EEC reference levels [1].

It should be emphasised that the blood lead levels obtained from the Dunedin sample cannot be taken as representative of the whole of New Zealand or of any other age group. Also, the Dunedin sample was aged 11 years and would be expected, therefore, to have lower blood lead levels than preschool children or older adults [9]. Furthermore, the Dunedin sample was not representative of the country in terms of socio-economic status. Therefore, the lack of high levels reported from Dunedin should not be taken as evidence that these relatively low lead levels would be similar in other places. Further research, as recommended by the Royal Commission on Environmental Pollution [1] is needed to monitor blood lead levels in other samples in other parts of New Zealand.

It was of interest to find no correlation between blood lead levels in Dunedin and socio-economic status. Most other studies (eg NHANES II study [9]) have found that the more socio-economically disadvantaged tend to have higher lead levels. The lack of a significant association between socio-economic status and lead levels in Dunedin may reflect the higher socio-economic status of the Dunedin sample or other differences related to socio-economic status. Socio-economic status can be interpreted as a proxy variable which is an indicator of related environmental factors such as standards of housing, location, and other aspects of the environment. Future studies of lead in this country should attempt to elucidate the nature of other environmental correlates that co-vary with lead. At present, the only detailed research on this is being carried out in Auckland (personal communication). It is likely that the New Zealand results will differ from those overseas as many of the overseas studies have been carried out in very large cities and in areas characterised by low socio-economic status with their associated crowding, poor housing, and, sometimes, high road traffic concentrations.

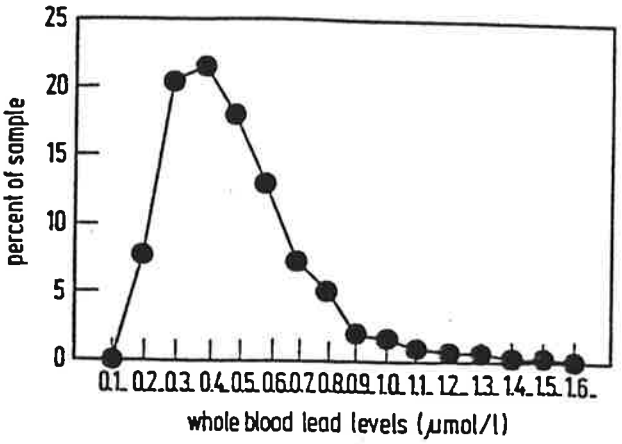

Figure 1. Whole blood lead levels in Dunedin 11 year old children.

The small but significant sex difference in blood lead levels between boys and girls was expected on the basis of the NHANES II study [9]. The higher blood lead levels in the boys were not due to their being taller and heavier as, over the whole sample, there were negative correlations (-0.07; -0.08; $p<0.05$) between blood lead levels and height and weight measures. This suggests the need for further investigation as to why the boys had significantly higher blood lead levels. It may be that girls are not as exposed to lead during their daily activities. Alternatively, there may be important differences between the sexes in the metabolic handling of lead.

The issue of the stability of blood lead levels has not been systematically investigated in this study, although it has been shown in other studies that test retest correlations have tended to range from 0.4 to 0.6 over periods of up to five years [18]. It was of interest, however, to find that among the ten children whose blood lead levels were reassessed, all but one had returned to a lower level. This may reflect either a recent acute ingestion or a chronic exposure to lead which was substantially influenced by intercurrent changes in body handling of lead. The fall in blood level between the initial and follow-up assessments suggests that redistribution of lead to other tissue pools or excretion had occurred. It is not known to what extent the upper ranges of the skewed distribution of lead values (Figure 1) below the 1.16 μmol/l level may also have reflected short term lead exposure with resulting temporary high lead levels as opposed to relatively stable high levels that reflect chronically high tissue levels of lead. Future studies of blood lead levels, therefore, should not restrict themselves to a single measure but should be longitudinal in design and involve serial lead level determinations on individual subjects. In this way, it may be possible to distinguish between acute episodes of raised blood lead levels as opposed to more stable high blood lead levels that reflect a continuing body lead burden. The latter condition may well have an important adverse effect on child health, cognitive development and behaviour but this remains to be investigated.

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