THE MONITORING AND ASSESSMENT OF HUMAN EXPOSURE TO LEAD IN THE ENVIRONMENT

by

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ABSTRACT

A comprehensive study of a group of two year old urban children (n = 97), designed to provide quantitative information simultaneously for lead intakes via all identified pathways, has been carried out in Birmingham (UK). This was based on the analysis of environmental, dietary and biological samples which included housedust, pavement and road dust, soil, air, handwipes, water, food and blood. A behavioural section was also incorporated in order to assess hand-to-mouth activity.

Results showed that for children whose blood levels and exposure to environmental lead were within the normal ranges for the UK, blood lead concentration was significantly related to a combination of housedust lead loading and an overall rate of touching objects, to water lead concentration and to the parents' smoking habits.

On the basis of assumptions used by the Royal Commission on Environmental Pollution, the estimated average total uptake of lead was 36 μg/day; of this, 97% was from ingestion and only 3% from inhalation.

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This was a collaborative study and the behavioural measures were assessed at the Environment, Health and Behaviour Research Group (EHBRG), University of Birmingham, blood lead analysis was carried out at the Trace Element Unit (TEU), University of Southampton, and food and water lead analysis at the Laboratory of the Government Chemist. I am particularly grateful to Dr. Peter Harvey (EHBRG) and Dr. Trevor Delves (TEU) for the useful discussions on data interpretation. Thanks are also due to Mr. Mike Quinn, Department of the Environment, in this respect.

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There are several members of AGRG who have provided an input to the work presented here. The environmental sampling programme incorporated into the study was developed jointly with John Watt, to whom I extend my grateful thanks. The excellent technical abilities of Krystyna St. Clair-Gribble proved to be invaluable and I am most grateful to Barrington Coles for his help with processing the air filter data. Thanks again go to Krys

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STATEMENT REGARDING CONJOINT WORK

I was responsible for the development of methods for sampling and analysing handwipes, while refinements to the air and dust sampling methodology were carried out jointly with my colleague, John Watt. I personally collected, prepared and analysed a proportion of the dust, soil, handwipe and air samples obtained during the course of this study. Assistance with sample collection, preparation and analysis was provided by field officers and technicians (who were trained jointly by John Watt and myself) due to the detailed nature of the study and the large number of samples required. All data interpretation reported in this thesis is solely mine.

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1. INTRODUCTION

Lead has been used by man for thousands of years and is one of the most widely dispersed environmental pollutants. Although it is doubtful whether lead performs any essential biological function in man or other animals, it is clear that, in substantial amounts, it is toxic to most forms of life. Indeed, the clinical manifestations of lead poisoning have been well known for centuries. However, in recent years there has been a growing concern that the intake of lead from general environmental exposure may result in subtle, subclinical adverse effects and, in this context, particular attention has been paid to the possible effects on the intelligence and behaviour of young children.

Children are exposed to lead in the environment from a number of sources, along a variety of pathways but, despite the concerns about possible adverse health effects, there is a general lack of understanding about the significance of the various pathways to their total lead intake. Several studies have been carried out which have addressed this issue but few have been comprehensive. Indeed, it was noted by the Royal Commission on Environmental Pollution in its Ninth Report on Lead in the Environment that there were major gaps in knowledge concerning the intakes of lead by young children. In response to this identified need, a detailed study has been carried out in Birmingham to provide quantitative information on the lead intakes of young children in the UK via all the major pathways, simultaneously.

This study is considered in detail in this thesis, which is structured in the following manner. In Chapter 2, the major sources of lead in the environment are identified and discussed and the literature on lead concentrations in environmental media is reviewed.

Chapter 3 deals with the uptake, distribution, retention and excretion of lead in man and summarises the extensive literature on human health effects. The pathways of lead from the environment to man are considered in Chapter 4 and the sampling protocol and analytical techniques employed in the Birmingham Study are detailed. The results from this study are presented in Chapter 5 and discussed in detail in Chapters 6 and 7. The main conclusions are summarised in Chapter 8.

2. LEAD IN THE ENVIRONMENT

2.1 INTRODUCTION

Lead (Pb) is a dense (11.34 g/cm^3), soft, malleable metal which has a shiny silver lustre in its pure metallic state. More commonly, however, its appearance takes on a dull grey/blue colour due to surface oxidation in the presence of air. Lead, atomic number, 82, is a member of Group IVB of the periodic table. Lead has two stable oxidation states, +2 and +4, although the inorganic chemistry of the element is dominated by the divalent state. The relative atomic weight of lead is 207.2 and it has four stable isotopes, ^{204}Pb, ^{206}Pb, ^{207}Pb and ^{208}Pb. The origin of the first of these, ^{204}Pb, is uncertain as it is not the product of any known radioactive decay series. However, ^{206}Pb, ^{207}Pb and ^{208}Pb are the final products of the radioactive decay of ^{238}U, ^{235}U and ^{232}Th, respectively. There are four radioactive isotopes included in these decay series, ^{210}Pb, ^{211}Pb, ^{212}Pb and ^{214}Pb. Only the first of these, ^{210}Pb, is long lived, with a half-life of 22 years.

Metallic lead does not occur in nature but is obtained by extraction from its ores, the most abundant of which is galena (lead sulphide). Other ores include cerussite (lead carbonate) and anglesite (lead sulphate). Lead melts at the relatively low temperature of 327^{o}C and is, thus, easily extracted from its ores by smelting. It boils at 1744^{o}C.

Lead has been known to man since prehistoric times, and it is thought possible that it was the first metal to be smelted. Small beads of metallic lead have been found with gold and copper objects dating from 7000-6500 BC, in excavations on the central Anatolian Plain, indicating an anthropogenic source even at this early date (Krysko,

1979). In the ancient world, Egyptians and other early races used lead for small decorative objects (figurines, rings, beads, etc.), as coins, standard weights and sinkers (for fishing) and used lead oxides or compounds of lead as cosmetics. Also, lead was used in glass making to increase its brilliance, make it easier to cut or engrave and also to colour it. It was not, however, until the Roman era that lead was used in large quantities. The main use was for sanitary engineering, providing piping and cisterns to supply water to towns and houses throughout the Roman Empire. Lead sheet was also used for roofing and for covering and protecting the keels of ships. Domestically, most of the cooking pots, utensils, plates and drinking vessels were made of pewter which, at that time, comprised 50 per cent lead, 50 per cent tin. After the fall of Roman civilisation, the need for lead declined and did not rise again substantially until the advent of the Industrial Revolution, around the second half of the eighteenth century. Then, in the early 1920's, it was discovered that tetra-ethyl lead could be used as an anti-knock agent in petrol and this led to a marked increase in consumption. Worldwide consumption continued to rise until it peaked in 1978, at around 5.5 x 10^9 kg (World Metal Statistics, 1983). The increase in consumption during the twentieth century is summarised in Table 2.1.

The principal user of lead today is the automotive industry, where it is employed mainly in the manufacture of storage batteries and additives for petrol, although it is also used in smaller quantities for solder and bearings in car engines. A significant proportion of lead is used in the building industry in the form of roofing, guttering and flashing and lead is also used for the sheathing of telephone and power cables. In smaller quantities, it is used in paints, radiation shielding, plastics, in the manufacture of ammunition and in the production of printing metals. Recent lead consumption figures for the

Table 2.1 All-time worldwide consumption of lead

Period	Lead consumption ($\times 10^9$ kg)
Pre-1900	80
1901-1910	10.7
1911-1920	11.2
1921-1930	14.2
1931-1940	14.6
1941-1950	14.9
1951-1960	24
1961-1970	33
1971-1980	38
Total, all-time	241

Source: Nriagu (1979)

UK indicate that 30 per cent of the total is used in the manufacture of batteries, 30 per cent in a variety of building applications, 22 per cent in lead based chemicals, 16 per cent in "other" metallic uses (such as radiation shielding, cable sheathing and solder) and only 2 per cent in petrol, paint and pipes (Lead Development Association, 1988).

2.2 SOURCES

2.2.1 Natural

The average lead content of the earth's crust has been reported by Taylor (1964) to be around 13 µg/g, although other estimates range from 2 to 16 µg/g (Moeller, 1952; Lead Development Association, 1974; Heslop and Jones, 1976). In the natural cycle, lead is released to the earth's surface through a variety of processes which include weathering of rocks, volcanic activity and uptake,

and subsequent release, by plants. Accurate assessments of the quantities emitted naturally are difficult, particularly when considering sources such as vegetative exudates and forest fires, where there is a general lack of reliable information and relevant data. However, a number of attempts have been made to quantify the worldwide emission of lead to the atmosphere from natural sources (Nriagu, 1979; Lantzy and Mackenzie, 1979; Jaworowski et al., 1981; Weisel, 1981). The resultant estimates have recently been summarised by Pacyna (1986) and these are presented in Table 2.2.

Table 2.2 Estimated worldwide natural emissions of lead to the atmosphere

Source category	Emission rate ($\times 10^6$ kg/y)	
Windblown dust	10.0	(3-35)*
Volcanoes	6.4	(0.4-96)
Vegetation	1.6	(1.6-21)
Forest fires	0.5	(0.04-6.8)
Sea spray	0.1	(0.001-5)
Total	18.6	

* range of literature values in parenthesis
Source: Pacyna (1986)

The estimated total annual worldwide natural emission of lead to the atmosphere is 18.6 x 10^6 kg, with windblown dust accounting for over half of this total. This estimated total is somewhat lower than that estimated earlier by Nriagu (1979) of 24.5 x 10^6 kg.

Attempts have also been made to quantify the concentrations of lead derived from natural sources in a

variety of environmental media. The US National Research Council (1980) has estimated the natural concentrations of lead in air, soil, water and food in the USA and these are presented in Table 2.3.

Table 2.3 Lead concentrations estimated as natural in the USA

Medium	Estimated natural concentration
Air	
Rural/remote	0.01-0.1 ng/m^3
Inhabited	0.1-1.0 ng/m^3
Soil	
Rural/remote	5-25 μg/g
Inhabited	5-25 μg/g
Water	
Fresh	0.005-10 μg/l
Marine	0.001 μg/l
Food	0.0001-0.1 μg/g

Source: National Research Council (1980)

Such an exercise is, however, extremely difficult and estimates, often based on concentrations measured in remote areas, are difficult to validate. When lead is released into the environment, it has a long residence time. For example, estimates by Tyler (1978) suggest that 90 per cent of the total amount of lead present in the upper layers of a moorland soil today would still be there in 70 to 200 years time. The effect of a long environmental residence time combined with the fact that man has been mining and processing lead for thousands of years is that lead is now one of the most widely dispersed environmental pollutants. Concentrations of lead measured

in remote areas do not, therefore, necessarily reflect natural concentrations and may well include a component of anthropogenic origin. This point has been well illustrated by Murozumi et al. (1969) who analysed recent snow and ancient ice in Greenland and reported that lead concentrations had increased by a factor of 200 (from around 1 to around 200 pg/g) over the preceeding 3000 years. They also found a corresponding 100-fold increase in the lead:silicate dust ratio during the same interval and, on this basis, claimed that in earlier times most natural lead in the troposphere came from wind-entrained soil dust, while more than 99 per cent of the lead now in the troposphere of the Northern Hemisphere originates from human activities. More recent studies by Ng and Patterson (1981) and Boutron and Patterson (1986) resulted in similar estimates of current anthropogenic inputs (i.e. greater than 99 per cent) but indicated that natural lead emissions from volcanoes were also likely to have made significant contributions to the air lead burden in ancient times.

In addition to the problems posed by long range transport of anthropogenic lead and subsequent deposition in remote areas, there are many opportunities during sample collection, processing and analysis for contamination. Indeed, some workers suggest that most of the reported values from remote area studies are too high because of such contamination and conclude, therefore, that most of the estimates of natural lead concentrations are also too high (Settle and Patterson, 1980; Peel, 1986).

2.2.2 Anthropogenic

Lead is released into the environment during the mining and smelting of its ores, from the production, use, recycling and disposal of lead-based products and from the burning of fossil fuels and wood. Historical changes in

anthropogenic emission rates of lead to the atmosphere, estimated by Nriagu (1979), are presented in Table 2.4.

Table 2.4 All-time worldwide anthropogenic emissions of lead to the atmosphere

Period	Lead emission (x 10^9 kg)
Pre-1900	3.52
1901-1910	0.47
1911-1920	0.49
1921-1930	1.12
1931-1940	1.64
1941-1950	1.67
1951-1960	2.69
1961-1970	3.70
1971-1980	4.26
Total, all-time	19.6

Source: Nriagu (1979)

The marked increase in emissions since the 1920's is associated with the widespread use of lead additives as anti-knock agents in petrol and is particularly noticeable from the 1950's onwards. Emissions in the three decades up to 1980 are estimated to account for over half of the total all time worldwide anthropogenic emission of 19.6 x 10^9 kg. More detailed assessments of emissions from individual anthropogenic sources have been made. Nriagu and Pacyna (1988) estimated the worldwide emissions of lead from a variety of such sources for the year 1983 and these estimates are presented in Table 2.5. Emissions from the combustion of petrol (given as "mobile sources" in the Table) provide the largest single source, accounting for around 75 per cent of the estimated total of 332 x 10^6 kg. The non-ferrous metal industry accounts

Table 2.5 Estimated worldwide anthropogenic emissions of lead to the atmosphere in 1983

Source category	Emission rate (x 10^6 kg/y)
Coal combustion	
- electric utilities	0.78-4.65
- industry and domestic	0.99-9.90
Oil combustion	
- electric utilities	0.23-1.74
- industry and domestic	0.72-2.15
Pyrometallurgical non-ferrous metal production	
- mining	1.70-3.40
- Pb production	11.70-31.20
- Cu-Ni production	11.05-22.10
- Zn-Cd production	5.52-11.50
Secondary non-ferrous metal production	0.09-1.44
Steel and iron manufacturing	1.07-14.20
Refuse incineration	
- municipal	1.40-2.80
- sewage sludge	0.24-0.30
Phosphate fertilisers	0.06-0.27
Cement production	0.02-14.24
Wood combustion	1.20-3.00
Mobile sources	248.03
Miscellaneous	3.90-5.10
Total:	288.70-376.00
Median value:	332.35

Source: Nriagu and Pacyna (1988)

for the second largest fraction. Nriagu (1979) compiled a similar emissions inventory for the year 1975 and estimated the total anthropogenic emissions to be around

450 x 10^6 kg for that year. It has been suggested that this apparent decrease in total emissions between 1975 and 1983 is the result of an overall global reduction in emissions of particulate lead from industry and a phase down in the consumption of leaded petrol over this period (Nriagu and Pacyna, 1988). A comparison of the estimated total anthropogenic emission rate in Table 2.5, 332 x 10^6 kg/y, with the estimated total natural emission rate in Table 2.2, 18.6 x 10^6 kg/y, indicates that the input of lead to the atmosphere from anthropogenic sources is around 18 times greater than that from natural sources.

In the UK, most atmospheric lead is derived from petrol combustion. A recent emissions inventory by Hutton and Symon (1986) indicates that atmospheric lead input from petrol combustion is around 6.8 x 10^6 kg/y, while that from all anthropogenic sources (including petrol) is around 7.6 x 10^6 kg/y. This suggests that only around 12 per cent of lead in air is of an industrial origin. This estimated proportion is slightly higher than that estimated for the UK by the Royal Commission on Environmental Pollution [RCEP] (1983) of 10 per cent. It should be noted that the industrial emission figures reported by the RCEP (1983) of 0.2 to 0.25 x 10^6 kg/y were based on estimates from the early 1970's (Department of the Environment [DoE], 1974). The estimated industrial emission rate of 0.8 x 10^6 kg/y reported by Hutton and Symon (1986) is based on more recent data and is, therefore, likely to be a more accurate reflection of the current situation.

Anthropogenic lead in soil is derived from a wide range of sources along a number of pathways. The sources of atmospheric lead, outlined above, also serve as sources of soil lead through the processes of wet and dry deposition. Run-off of contaminated water from mining and milling operations, both past and present, can increase soil lead levels. Thornton (1980) has estimated that

about 4000 km^2 of agricultural land in England and Wales are severely contaminated as a result of past metal mining and smelting, with lead being cited as a major contaminant of most of this land. Direct sources of lead into soil include lead arsenate insecticides, sewage sludge, impurities in fertilisers and other minor sources such as paint flakes, lead shot and lead fishing weights. The total worldwide input of anthropogenic lead into soils has been estimated to be between 479 and 1113 x 10^6 kg/y (Nriagu and Pacyna, 1988). In the UK, a total annual input of anthropogenic lead into arable soil of 1.6 x 10^6 kg/y has been estimated (Hutton and Symon, 1988). In both of these cases, atmospheric deposition provided by far the greatest input.

Inputs of lead to freshwater include deposition from the air, leaching from soils and mine wastes, surface run-off, direct industrial discharge and discharge from sewage works. Much of this lead ultimately reaches the sea bed via river flow, although some is transported as river dredgings and dumped at sea. The total worldwide input of anthropogenic lead into aquatic ecosystems (both freshwater and marine) has been recently estimated to be around 138 x 10^6 kg/y (Nriagu and Pacyna, 1988). Anthropogenic lead inputs to UK estuarine and coastal waters have been estimated to total 0.46 x 10^6 kg/y, with sewage and sludge disposal alone accounting for 0.43 x 10^6 kg/g (Hutton and Symon, 1986). However, in this UK study, inputs from atmospheric deposition were not quantified. An earlier study of anthropogenic inputs of lead into the North Sea from bordering countries indicated that such inputs can be large (Norton, 1982). Of a total estimated input of 7.7 x 10^6 kg/y, 4.6 x 10^6 kg/y was attributed to atmospheric deposition, 2.4 x 10^6 kg/y to inputs from rivers and the remaining 0.7 x 10^6 kg/y to a combination of direct discharge from sewage outfalls and the dumping of sewage sludge and other wastes.

2.3 LEAD IN AIR

In general, air lead concentrations in remote areas are low. It is apparent from an extensive review by Wiersma and Davidson (1986) of around 40 studies that remote areas most likely to be influenced by vehicular and industrial emissions, such as those only a moderate distance from populated areas in industrialised countries, show the greatest air lead concentrations. Most of the remote sites in the USA and Europe fall into this category and reported concentrations range from 10 to 100 ng/m^3. Remote continental locations in South America and Asia, and sampling sites over the Atlantic Ocean, show concentrations of around 1 to 20 ng/m^3. Air lead levels in the Arctic and Antarctic, and over remote areas of the Pacific Ocean, are mostly found to be below 1 ng/m^3 although values of less than 0.1 ng/m^3 have been noted (Wiersma and Davidson, 1986).

Air lead concentrations in rural areas of industrialised nations are generally higher than those in remote regions. In the United States, the annual lead concentrations at non-urban sites throughout the country, between 1970 and 1974 were reported to range from 47 to 139 ng/m^3 (US Environmental Protection Agency [EPA], 1986) while in Belgium, the lead level at two rural sites averaged 174 ng/m^3 between 1972 and 1975 (Kretzschmar et al., 1980). In another rural area of Belgium, Roels et al. (1980) reported a higher average lead concentration of 370 ng/m^3 for 1978. In the UK, the average lead concentration at a semi-rural site was reported to be 130 ng/m^3 over the period 1957 to 1974 (Salmon et al., 1978) while the mean concentration at four rural sites between 1976 and 1979 was found to be 76 ng/m^3 (Cawse, 1977, 1978, 1979, 1981). The most recent published data for rural areas in the UK show annual mean concentrations at seven sites ranging from 8 to 77 ng/m^3 during 1986 (McInnes, 1987).

Air lead concentrations in urban areas are usually higher than those found in rural areas. Studies in nine North American cities during the 1970's revealed mean lead levels ranging from 0.8 to 2.0 $\mu g/m^3$ (EPA, 1986), while UK studies at five urban locations between 1976 and 1981 showed mean values ranging from 0.48 to 0.85 $\mu g/m^3$, with a five year average of 0.65 $\mu g/m^3$ (McInnes, 1982). Higher values have been noted elsewhere, such as Turin, Italy, where the average over the period 1974 to 1979 was reported to be 4.5 $\mu g/m^3$ (Facchetti and Geiss, 1982). Elevated lead concentrations are also frequently found at roadside locations (RCEP, 1983; EPA, 1986). In the UK, Turner et al. (1980) noted levels ranging from 0.4 to 2.2 $\mu g/m^3$ alongside main roads in London during a three month period in 1978, while Colwill and Hickman (1981) reported monthly mean values ranging from 6 to 25 $\mu g/m^3$ over the period 1973 to 1979 from a site on the central reservation of the M4 motorway. Not surprisingly, high concentrations have also been noted in the vicinity of lead works. Lead concentrations within 1 km of a lead smelter in Belgium, monitored over five consecutive years (1974 to 1978), were found to range from 2.68 to 4.06 $\mu g/m^3$ (Roels et al., 1980). Extraordinarily high levels are occasionally reported. Two studies in the vicinity of lead smelters in Yugoslavia revealed concentrations ranging from 24.2 to 38.4 $\mu g/m^3$ (Fugas, 1977) and 21.2 to 29.2 $\mu g/m^3$ (Popovac et al., 1982).

Results generated from air monitoring networks in operation over a number of years have been used to assess temporal trends in airborne lead levels. In the United States, data from the EPA's National Filter Analysis Network, summarised in Table 2.6, show that mean air lead concentrations at urban locations have decreased over the period from 1970 to 1980, the greatest decrease occurring since 1977 (EPA, 1986). The 1980 level is around a quarter of the value in 1970. This trend is mainly attributed to the decreasing lead content of leaded petrol

and to the increasing usage of unleaded petrol. Between 1975 and 1980, lead consumption in petrol decreased by 52 per cent while the corresponding composite maximum quarterly averages of ambient lead decreased 51 per cent. It is also reported that promulgation of the 1978 National Ambient Air Quality Standard for Lead in the United States (1.5 $\mu g/m^3$ averaged over a calendar quarter) has had the effect of reducing the air lead concentration in major urban areas (EPA, 1986).

Table 2.6 Annual mean air lead concentrations in the USA, 1970 to 1980

Year	Number of sites	Arithmetic mean ($\mu g/m^3$)	Geometric mean ($\mu g/m^3$)
1970	797	1.19	0.99
1971	717	1.23	1.00
1972	708	1.13	0.93
1973	559	0.92	0.76
1974	594	0.89	0.75
1975	695	0.89	0.74
1976	670	0.85	0.72
1977	533	0.91	0.68
1978	282	0.68	0.50
1979	167	0.56	0.39
1980	220	0.32	0.24

Source: EPA (1986)

The situation in the UK is not quite as clear cut. Following the implementation of the Clean Air Act of 1956, which limited smoke emissions, there was a decrease in air lead levels recorded at a semi-rural site in England until the early 1960s (Salmon et al., 1978). The emission of lead from petrol combustion increased by about 12 per cent per year between 1960 and 1970 in the UK due to a doubling of consumption of petrol and an increase in the

average lead content in the fuel, resulting in an upward trend in lead levels during the decade at this location. During the 1970's, however, there is evidence of a decrease alongside some main roads and at rural sites in England and Wales (Colwill and Hickman, 1981; Cawse, 1974, 1975, 1976, 1977, 1978, 1979, 1981). Other data for urban and industrial sites show moderate to substantial reductions in air lead concentrations from 1976 to 1981 at two urban sites in Scotland but small or insignificant decreases over the same period at three urban sites in England (McInnes, 1982). Although there was a phased reduction in the lead content of petrol in the UK during the 1970's, from 0.84 g/l in 1972 to 0.45 g/l in 1978, national figures indicate that total lead emissions from petrol combustion remained fairly stable due to an increase in traffic levels (DoE, 1987). It has been suggested that a recession in industrial activity was responsible for the major proportion of the decrease in air lead concentration both in the rural areas and at the two Scottish urban sites (Cawse, 1981; McInnes, 1982). The three English urban sites were remote from industrial sources of lead and the air lead concentration was largely dependent upon vehicle emissions. There was a further small reduction in the maximum permitted lead content of petrol in 1981, from 0.45 to 0.40 g/l. In January, 1986 this maximum was reduced substantially to 0.15 g/l, its current level. Air lead concentrations measured at 18 monitoring stations in rural, urban and kerbside locations across the UK during 1986 have been compared by McInnes (1987) with those from the same sites measured between October, 1984 and September, 1985 (i.e. the last 12 month period prior to the introduction of deliveries of petrol with reduced lead content). Reductions of between 24 and 74 per cent were found at individual sites and the average reduction over all sites was 53 per cent, from 0.47 $\mu g/m^3$ to 0.22 $\mu g/m^3$. A similar exercise carried out by Page et al. (1988) using results from four urban sites in Wales, revealed reductions of between 52 and 61 per cent. It is

interesting to note that in the Federal Republic of Germany, which underwent the same reduction in the maximum permitted lead content of petrol (0.4 to 0.15 g/l) at the beginning of 1976, there was a parallel decrease in air lead concentration ranging from about 20 per cent in some rural areas to around 65 per cent in some inner city areas (Jost and Sartorius, 1979).

There have been a large number of studies into the dispersion of airborne lead from both line sources (roads) and point sources (e.g smelters, power plants, incinerators) which have resulted in the following observations. Lead concentrations show diurnal and day-to-day changes which are determined primarily by variations in rate and point of emission, wind direction and local ventilation factors (Janssens and Dams, 1975; Demuynck et al., 1976). Airborne lead concentrations decrease with distance from the emission source, the decrease being characteristically curvilinear (Yankel et al., 1977; Chamberlain et al., 1978) and the size of the airborne lead particles decreases with distance from the source (Daines et al., 1970). Vertical gradients of lead in the atmosphere can develop, giving higher concentrations at street level than roof top level (Schroeder, 1974) The physical and chemical characteristics of vehicular lead emissions in air are related to the nature of the fuel and the operating conditions of the vehicle (Nriagu, 1980). Marked seasonal variations in ambient lead levels are common, with maximum concentrations typically occurring in the winter. This may be attributed to seasonal differences in the intensity of lead emissions from different sources and/or to the variability in regional ventilation factors (Faoro and McMullen, 1977). Indoor air lead levels are generally lower than those found in external air, the lowest indoor:outdoor ratios being noted for air conditioned houses (Yocum, 1982).

Airborne lead is transferred to the earth's surface by means of dry and wet deposition. There are a number of factors which influence dry deposition fluxes. These include wind speed, particle size distribution and the texture of the surface onto which the deposition takes place (EPA, 1986). As a result, there may be large differences in dry depostion fluxes in areas with similar air lead concentrations. Wet deposition of lead occurs when particles are trapped by rain or snow. In the UK, the annual deposition rate of lead in rural areas is reported to be around 20 mg/m^2, while in typical urban areas values of up to around 360 mg/m^2 have been noted (RCEP, 1983). Extremely high annual deposition rates, up to around 3000 mg/m^2, can, however, be found near point sources (Turner et al., 1980).

2.4 LEAD IN SOIL

Soil is the major reservoir for lead in the environment and, as will be discussed in more detail in Section 2.7, forms the initial link of the food-chain through which the metal is transferred to man. Lead levels vary widely between different geographical regions due to differences in both natural factors (parent material, degree of weathering, organic matter content) and anthropogenic factors (proximity to pollutant source, land use, etc.).

Preliminary results from a national inventory of soils in England and Wales show the geometric mean (range) lead concentration for 2776 surface soils (0-15 cm) to be 48 µg/g (5-16400 µg/g) (McGrath, 1986). This value is similar to the median level of 42 µg/g reported by Archer (1980) for around 750 agricultural surface soil samples taken throughout England and Wales. A recent baseline survey of lead in Welsh soils by Davies and Paveley (1985) revealed a median lead content of 35 µg/g in surface soils

(n = 654) while Reaves and Berrow (1984) found a lower geometric mean value of 17 μg/g in around 470 Scottish surface soil samples. Some of these workers have attempted to calculate the likely background concentrations of lead in soils by employing a statistical sieving technique to separate these from other values resulting from natural or anthropogenic contamination. For soils in old lead mining areas in England and Wales, Davies (1983) concluded that the average background soil lead content was 42 μg/g. Using the same technique on the data from the national soil inventory, McGrath (1986) estimated the background concentrations of lead in soils in England and Wales to be 32 and 35 μg/g, respectively.

From throughout the conterminous USA, Shacklette et al. (1971) sampled soils at a depth of 20 cm to determine the elemental composition of soil materials derived from the earth's crust. Lead concentrations ranged from less than 10 μg/g to greater than 70 μg/g, with a geometric mean of 16 μg/g (n = 863). Although this range of values is likely to represent natural levels of lead in soils in the US, it has been suggested that contamination with lead could have occurred during sample collection and handling in this relatively early study (EPA, 1986). However, in a later survey of a further 355 soils, Shacklette and Boerngen (1986) again found a mean lead concentration of 16 μg/g. Data are also available for "background" soil lead levels in Canada. McKeague and Wolynetz (1980) found an arithmetic mean of 20 μg/g for 53 uncultivated soils and reported that there were no differences with depth between the A, B and C horizons in the soil profile. Analysis of over 400 agricultural surface soil samples collected by John (1975) throughout British Columbia revealed a lower average lead concentration of 10 μg/g.

It is apparent from the studies cited above that, in general, soils which are free from contamination (either natural or anthropogenic) contain lead at concentrations

usually well below 50 μg/g. In contaminated environments, however, very much higher concentrations, sometimes by several orders of magnitude, are found. In Great Britain, severe soil contamination has been documented in areas which have, historically, been centres of non-ferrous metal mining and smelting. In the old lead/zinc mining areas of the Halkyn Mountain, north-east Wales, Davies and Roberts (1975) reported an overall mean lead concentration of around 6600 μg/g, for 17 garden and 43 field soil samples, and an extremely high maximum of 48000 μg/g. Analysis of 43 soil samples from villages in mineralised areas of Derbyshire by Barltrop et al. (1975) revealed a mean lead level of around 5000 μg/g, with a wide range of values between 1000 and 28000 μg/g. In a more recent, more extensive, survey of Derbyshire village soils, Thornton et al. (1985) reported similar findings, with a geometric mean value of 5600 μg/g, range 1200 to 22000 μg/g, for 100 garden soils. The old zinc mining area of Shipham, Somerset has also been studied and data of Thornton et al. (1980) showed a high mean lead concentration of around 2400 μg/g (range 104-6880 μg/g, n = 607) in surface soils.

It is generally accepted that roadside soils are often contaminated with lead from petrol-driven vehicles and that the effect is most marked within 25-30 m of the road. Examples of such contamination have been reported in many publications and typical levels in surface roadside soil have been given as 150 to 540 μg/g by Lagerwerff and Specht (1970) and as 500 to 1100 μg/g by Ward et al. (1977). The EPA (1986) concluded from a review of the available literature that roadside soils may contain atmospheric lead from 30 to 2000 μg/g in excess of natural levels within 25 m of a road, all of which is in the upper layer of the soil profile. Levels of contamination are mainly dependent upon the amount of traffic and the distance from the road. Work of Quarles et al. (1974) showed decreases in soil lead from 550 to 40

μg/g within 25 m alongside a highway with 12500 vehicles per day in Virginia. Their findings were confirmed by Wheeler and Rolfe (1979), who observed an exponential decrease linearly correlated with traffic volume and by several workers in other parts of the world (Little and Wiffen, 1978; Wong and Tam, 1978; Agrawal et al., 1981; Garcia-Miragaya et al., 1981).

Urban soils are often contaminated with lead. Since the early work in Scotland by Purves (1968) and Purves and MacKenzie (1969) which demonstrated this phenomenon, there have been countless other studies throughout the world which have confirmed these findings. These have been reviewed elsewhere (Khan, 1980; Harrison and Laxen, 1981; Ratcliffe, 1981; Duggan and Inskip, 1985; Chaney and Mielke, 1986; Davies and Thornton, 1988) and suggest that lead in vehicle exhaust fumes is the major contributing factor resulting in elevated lead levels in urban soils, although inputs from industrial emissions, the burning of fossil fuels in domestic situations and flaking paint can also be important factors in certain circumstances.

Higher lead concentrations are usually found in soils from central areas of cities than in those from suburban areas. In the UK, Davies et al. (1979) reported that distance from the city centre of London (Marble Arch) had a major influence on soil lead levels. Three zones within Greater London were studied, central London (0-4 km), inner London (4-10 km) and outer London (10-30 km) and revealed mean soil lead concentrations of 523, 242 and 142 μg/g (EDTA extractable), respectively. The mean soil lead level in an adjacent rural zone (>30 km) was reported by these workers to be 30 μg/g (EDTA extractable). This trend in the lead content of London soils has been confirmed by further work (Davies, 1983a; Denton, 1988). A similar decrease in soil lead concentration with increase in distance from the city centre has also been noted in Birmingham, UK by Davies and Houghton (1984) and

in the US by Mielke et al. (1983, 1984) from studies in Baltimore and Minneapolis-Saint Paul, by Kinard et al. (1976) in Columbia, South Carolina and by Brams (1977) in Houston and Galveston, Texas. The phenomenon of elevated soil lead levels alongside major roads has also been noted in urban communities (Davies and Holmes, 1972) and it has been suggested by Chaney and Mielke (1986) that the zonal soil lead patterns discussed above could be a reflection of the uneven distribution of traffic in cities, whereby the greatest quantity of petrol lead is emitted in city centres.

A recent comprehensive survey of 100 households in each of 47 representative towns and city boroughs in the UK has confirmed that elevated concentrations of lead exist in surface garden soils compared with agricultural soils (Thornton et al., 1985). Lead concentrations ranged from 12 to over 14000 μg/g, with a geometric mean of 230 μg/g, in 3550 garden soils from throughout England, Scotland and Wales, excluding London and areas affected by mining and smelting. In the 579 gardens sampled in seven London boroughs, the geometric mean value was very much higher, 647 μg/g. Significantly lower lead values were noted in soils from households of less than 30 years than in those from households of more than 100 years. These authors concluded that this was likely to be a reflection of a shorter history of soil contamination from bonfires and fossil fuel residues (e.g. soot) commonly used as soil improvers and ameliorants. Davies (1978) has also noted an ageing process, whereby older gardens contain more lead. Several workers have reported that lead in soil adjacent to a house decreases with distance from the house and it is thought likely that this is due to paint chips flaking off from exterior surfaces of the house or to dust of atmospheric origin washing down from the rooftop (Ter Haar and Aronow, 1974; Getz et al., 1979; Wheeler and Rolfe, 1979; Preer et al., 1984). It has been suggested that urban soil lead could also be derived from abraded

tyre material, plastics, rubber and battery factories, insecticides and old car batteries (Fleming and Parle, 1977).

Contamination with lead has also been noted in soil around smelters and is reported to be dependent upon the smelter emission rate, length of time the smelter has been in operation, prevailing wind speed and direction, regional climatic conditions and local topography (Roberts, 1975). In Great Britain, Little and Martin (1972) observed decreases from 125 to 10 μg/g in a 6 km zone around a smelting complex, with all of the excess lead in the upper 6 cm of the soil profile, while Adamson (1980) reported a maximum value of 1173 μg/g near a smelter in north Humberside. Extremely high levels of lead in soil can accumulate and Roberts (1975) reported concentrations between 15000 and 20000 μg/g near a smelter in Toronto. Kerin (1975) found 5000 to 9000 μg/g adjacent to a Yugoslavian smelter, with a contamination zone of 7 km in radius, and Ragaini et al. (1977) reported a soil lead level of 7900 μg/g near a smelter in Kellog, Idaho, observing a 100 fold decrease at a depth of 20 cm in the soil profile. Palmer and Kucera (1980) found soil lead in excess of 60000 μg/g near two smelters in Missouri, decreasing to 10 μg/g at 10 km and near a smelter in New Zealand, Ward et al. (1977) reported a maximum value of 126000 μg/g.

Agricultural land in industrialised countries is frequently used for the disposal of sewage sludge and it has been reported that in the UK about 45 per cent of the sludge produced annually is utilised in agriculture (Davis and Coker, 1979). The lead content of sewage sludge is very variable and depends upon the origin of the sewage. For example, when the run-off from streets enters the sewerage system, this may be a major pathway of lead to the sludge. Surveys of sewage sludges in the UK have shown lead concentrations ranging from 19 to 45400 μg/g

(dry weight), with half of the sludges having lead concentrations above 300 μg/g (DoE, 1981; Sterritt and Lester, 1981). Application of such sludge to agricultural soil can result in an accumulation of lead. Richardson (1980) reported an increase from 60 to 245 μg/g of lead in soil (0-15 cm) which received sludge application over a period of 75 years. A similar observation was made by Andersson and Nilsson (1972) who showed an increase of 18 μg/g of lead in Swedish soil (0-20 cm) treated with sludge for 12 years. In the UK, the maximum permissible soil lead concentration arising from sewage sludge application is 300 μg/g (DoE, 1981). This is similar to the limit value given in EEC Directive 86/278/EEC due to come into force by June, 1989 (European Economic Community [EEC], 1986).

2.5 LEAD IN DUSTS

Both within buildings and outdoors, dust may contain appreciable amounts of lead, particularly in urban areas. The role played by such lead-bearing dust in the exposure of children to environmental lead will be explored in some detail later, in Chapter 4. The purpose of the current section is to provide a summary of the more recently published data on lead levels in dust in a number of different situations. Results are presented for indoor and outdoor dusts from rural, urban, industrialised and mineralised areas from a variety of geographical locations. It must be stressed from the outset, however, that it is virtually impossible to make valid comparisons between results reported by different workers because, at present, there are no widely accepted standard procedures employed for sample collection and pre-treatment prior to analysis or for the analysis itself. Further interpretive difficulties arise from some workers reporting arithmetic means and others geometric means. Despite these problems, such an exercise is useful in that it provides an

indication of the relative magnitudes of lead in dust in the different situations.

Reported lead concentrations in outdoor dusts are set out in Table 2.7 while those reported in indoor dusts are listed in Table 2.8. Where possible, geometric rather than arithmetic means are cited. Typically, the frequency distribution of dust lead concentration in a sample population is positively skewed and approximately log-normal, i.e. transformation of the data to logarithms will result in a Gaussian or "normal" distribution. For such a log-normal distribution, the most appropriate measure of central tendency is the geometric mean. This is the antilogarithm of the simple or arithmetic mean of the log-transformed values. Where workers have quoted the arithmetic mean and the median but not the geometric mean, the median is included in the Tables. For a log-normal distribution, this is closer in value to the geometric mean than is the arithmetic mean.

With reference to Table 2.7, it can be seen that external dust lead levels are fairly low in rural areas, with reported mean concentrations ranging from 35 to 144 μg/g. Generally, higher lead concentrations are found in urban areas. Reported mean/median values range from 289 μg/g, in 220 samples from playgrounds throughout England, Scotland and Wales (Culbard et al., 1988), to 2611 μg/g, in 30 road dust samples from Hong Kong (Lau and Wong, 1982). A direct comparison between levels in rural and urban situations is possible where workers have taken both types of samples in the same study. This is the case in the studies of Day et al. (1975), Duggan and Williams (1977) and Strehlow and Barltrop (1987) and all of these workers found that dust lead concentrations in urban areas were at least one order of magnitude higher than those in rural areas. It is not altogether surprising to find higher lead concentrations in urban dusts than in those from rural locations. Air lead concentrations are also

Table 2.7 Concentrations of lead in outdoor dusts

SAMPLE SITE	LOCATION	n	CONCENTRATION (ug/g) MEAN[a]	RANGE	REFERENCE
RURAL					
Playgrounds/parks	Cheshire, UK	25	85	-	Day et al., 1975
Footpath	Surrey, UK	7	35	-	Duggan & Williams, 1977
Roads near schools	Suffolk, UK - 1984	133	144[b]	30-1037	Strehlow & Barltrop, 1987
	,, ,, - 1985	94	83[b]	10-450	,, ,, ,,
URBAN					
Various	Manchester, UK	350	860[c]	90-10200	Day et al., 1975
Major roads ("A")	Birmingham, UK	151	1800[c]	160-10000	Archer & Barratt, 1976
Minor roads ("B")	,, ,,	91	1300[c]	300-4600	,, ,, ,,
Residential roads	,, ,,	85	950[c]	220-4300	,, ,, ,,
Residential pavements	,, ,,	180	700[c]	200-5800	,, ,, ,,
Roads/pavements	London, UK	79	1200	430-3500	Duggan & Williams, 1977
Roads	Glasgow, UK	-	960	150-2300	Farmer & Lyon, 1977
Roads near petrol station	Christchurch, NZ	12	1970	144-2850	Fergusson et al., 1980
Playgrounds	London, UK	330	1230	50-26500	Rundle & Duggan, 1980
Major roads	Lancaster, UK	19	1890	740-4880	Harrison & Laxen, 1981
Roads	Hong Kong	30	2611	2000-3800	Lau & Wong, 1982
Roads	Brighton, UK	138	794[b]	16-28370	Davies & Thornton, 1987
Roads near schools	London, UK - 1984	115	1881[b]	582-5720	Strehlow & Barltrop, 1987
,, ,, ,,	,, ,, - 1985	90	1552	353-5430	,, ,, ,,
Roads	Various, UK	400	786[b]	45-9664	Culbard et al., 1988
Playgrounds	,, ,,	220	289[b]	11-6860	,, ,, ,,
Playgrounds/pavements	London, UK - 1982/83	77	509[b]	85-17900	Schwar et al., 1988
,, ,,	,, ,, - 1985	209	574[b]	84-15300	,, ,, ,,

contd./

Table 2.7 contd.

SAMPLE SITE	LOCATION	n	CONCENTRATION (ug/g) MEAN[a]	RANGE	REFERENCE
INDUSTRIAL					
Near lead smelter	Toronto, Canada	4	69000	5800-99000	Roberts et al., 1974
Roads	Birmingham, UK	117	1300[c]	160-50000	Archer & Barratt, 1976
Roads near lead users	Birmingham, UK	127	3000[c]	230-128000	Archer & Barratt, 1976
Roads near lead works	London, UK	12		20000-75000	Turner et al., 1980
Roads near lead smelter	Arnhem, Netherlands	35	761	25-2667	Keizer et al., 1982
Entrance to lead works	Glasgow, UK	-	-	26000-66000	Hampton & McKirdy, 1985
Pavements near print works	London, UK	6	7500	3700-13500	Jensen & Laxen, 1985
Roads near lead users	Birmingham, U.K.	58	791[b]	142-21000	Davies et al., 1987

KEY: n = number of samples
a = arithmetic mean
b = geometric mean
c = median

Table 2.8 Concentrations of lead in indoor dusts

SAMPLE SITE	LOCATION	n	CONCENTRATION (ug/g) MEAN[a]	RANGE	REFERENCE
RURAL					
Homes	Henllan, UK	31	177[b]	28-813	Davies et al., 1985
Homes	Suffolk, UK - 1983	119	333[b]	22-26620	Strehlow & Barltrop, 1987
Homes	,, ,, - 1984	104	374[b]	7-27148	,, ,, ,,
Homes	,, ,, - 1985	112	291[b]	20-8170	,, ,, ,,
URBAN					
Homes	Omaha, USA	16	572		Angle et al., 1974
Homes	Hartford, USA	59	11000	4900-17000	Lepow et al., 1975
Homes, offices, shops and schools	Birmingham, UK	806	1000[c]	100-280000	Archer & Barratt, 1976
Homes	Illinois, USA	239	600	170-1440[d]	Solomon & Hartford, 1976
Homes	New York, USA	8	1240		Rice et al., 1977
Homes	Birmingahm, UK	117	3500[c]	30-157000	DoE, 1982
Homes	Various, USA	30	250		Milar & Mushak, 1982
Homes	Leicester, UK	45	430	100-2100	Bowman & Musgrove, 1984
Homes	Christchurch, NZ	11	734	287-1408	Ferguson et al., 1986
Homes	York, UK	210	342[b]	83-8240	Lane, 1986
Homes	Brighton, UK	135	705[b]	27-24120	Davies & Thornton, 1987
Homes	Edinburgh, UK	495	308[c]	48-13600	Laxen et al., 1987
Homes	London, UK - 1983	146	893[b]	51-26755	Strehlow & Barltrop, 1987
Homes	London, UK - 1984	109	889[b]	59-18865	,, ,, ,,
Homes	London, UK - 1985	112	790[b]	58-29980	,, ,, ,,
Homes	Various, UK	4638	561[b]	5-36900	Culbard et al., 1988

contd./

Table 2.8 contd.

SAMPLE SITE	LOCATION	n	CONCENTRATION (ug/g) MEAN[a]	RANGE	REFERENCE
INDUSTRIAL					
Homes of lead workers	Tennessee, USA	14	2687[b]		Baker et al., 1977
Homes of lead workers	New York, USA	15	3310[b]		Rice et al., 1977
Homes near lead works	London, UK	60	-	500-8700	Turner et al., 1980
Homes near lead works (0-1.6 km)	Texas, USA	53	22190	2800-103750	Landrigan & Baker, 1981
Home burning old battery cases	N. Carolina, USA	4	-	5862-41283	Dolcourt et al., 1981
Homes near lead works	Arnhem, Netherlands	107	1054	63-4741	Brunekreef et al., 1981
Homes near paint removal operation	USA	4	7580	4930-10000	Landrigan et al., 1982
Homes of lead workers	Leeds, UK	17	1024	107-2275	Leeds City Council, 1982
Homes of lead workers	Various, USA	17	3000	-	Milar & Mushak, 1982
Homes with lead works	Japan	13	-	260-750480	Kawai et al., 1983
MINERALISED AREA					
Homes	Derbyshire, UK	64	1189[b]	190-25000	Barltrop et al., 1975
Homes	Halkyn, UK	59	340[b]	8-2943	Davies et al., 1985
Homes	Derbyshire, UK	100	1870[b]	606-7020	Culbard et al., 1988

KEY: n = number of samples a = arithmetic mean b = geometric mean
c = median d = range of means

higher in urban areas (for a number of reasons, including increased emissions from vehicles, from coal and refuse burning and from various industrial processes) and it is to be expected that this will result in higher lead deposition rates in these areas. This is well illustrated in the study of Strehlow and Barltrop (1987) who reported a mean air lead concentration of 0.50 $\mu g/m^3$ in London compared with a mean of 0.10 $\mu g/m^3$ in rural Suffolk and corresponding total lead deposition rates of about 800 and 50 $\mu g/m^2$ for the urban and rural sites, respectively. Differences in dust lead concentration have also been found within cities. In a recent study by Schwar et al. (1988), lower dust lead levels were noted in the outer areas of Greater London than those in inner-city areas. The results of Archer and Barratt (1976) (Table 2.7) show a fall in the median road dust lead concentration from 1800 $\mu g/g$ at major arterial, "A" road sites, through 1300 $\mu g/g$ at "B" road sites to 950 $\mu g/g$ at residential road sites and suggest that vehicle emissions are an important source of lead in urban dusts.

As outlined earlier, it is virtually impossible to judge whether or not differences in dust lead concentrations reported by different workers are real or whether they are simply a reflection of differences in the sampling and analytical techniques employed. Such problems have been highlighted by Jones and McDonald (1983) who examined the efficiency of different methods of extracting lead from street dust. Four techniques were evaluated (including those employed by Day et al., 1975; Archer and Barratt, 1976; and Farmer and Lyon, 1977 cited in Table 2.7) and differences of between 250 and 450 $\mu g/g$ were found which could be attributed solely to differences in analytical techniques. Such problems will persist until agreement is reached on a standard method of lead extraction from dust and, indeed, on a standard method of dust sampling.

Dust lead concentrations in industrialised areas, particularly in the vicinity of premises in which lead is processed or used, are usually higher than those found in general urban environments. Mean/median values included in Table 2.7 range widely from around 760 to 69000 μg/g, with individual values ranging from 25 to 128000 μg/g. It is likely that these elevated lead levels are the result of stack or low level emissions from a variety of industrial processes and, in some cases, a result of the movement of lead rich dust out of works on the wheels of departing vehicles (Turner et al., 1980).

With reference to Table 2.8, it can be seen that the reported mean lead concentrations of dusts from within rural homes range from 177 to 374 μg/g and are greater than those reported for external rural dusts (Table 2.7). Results from the study by Strehlow and Barltrop (1987) reveal that housedust lead concentrations in a rural Suffolk community are approximately four times higher than those in corresponding road dust samples. This general phenomenon of lead enrichment of indoor dusts relative to those found outdoors strongly suggests that there are internal sources of lead contributing to the indoor dust lead pool.

Whereas there have only been a limited number of indoor dust lead studies carried out in rural areas, there has been an abundance of such studies undertaken in urban and industrialised areas. The results from a selection of these are included in Table 2.8. Within urban homes, reported mean/median values range widely from 250 to 11000 μg/g. These levels are generally higher than those found in rural housedusts and the results from Strehlow and Barltrop (1987) indicate that concentrations in London housedusts are around three times higher than those in housedusts from Suffolk.

A detailed comparison of results from the different urban studies is not only hampered by the use of different sampling procedures and analytical techniques, as outlined earlier, but also by the fact that the studies were designed to meet differing objectives. For example, in the study in Birmingham reported by DoE (1982), the houses sampled were not randomly selected but were selected specifically because the children living in them had elevated body burdens of lead. The environments in which these children were living would have been likely also to contain elevated lead levels and the reported median value of 3300 μg/g should not, therefore, be considered typical for urban housedusts in general. Similarly, the extremely high mean value of 11000 μg/g in Hartford, Connecticut, reported by Lepow et al. (1975) is atypical and likely to reflect the biased nature of sample selection procedure, whereby only houses of children with chronically elevated blood lead levels were investigated. If the results from these two studies are excluded, the range of mean/median values is very much narrower, 250 to 1250 μg/g, and probably more indicative of typical lead levels in urban housedust. The extensive study of Culbard et al. (1988) was designed to provide baseline data on, among other things, lead levels in urban dusts throughout the mainland of Great Britain. The geometric mean value cited in Table 2.8, 561 μg/g, is derived from the analysis of 4638 randomly selected housedust samples from 40 towns and cities and 7 London boroughs and can, therefore, be considered typical for urban housedusts in England, Scotland and Wales. In this study, geometric mean values for the 47 urban locations ranged from 205 μg/g to 1348 μg/g, while the lead concentration of individual samples ranged from 5 to 36900 μg/g. The median lead concentration of 1000 μg/g reported by Archer and Barratt (1976) for around 800 dust samples from offices, shops, schools and homes is towards the top end of the range of mean values reported from studies in which only housedusts were sampled.

Lead levels in indoor dusts from urban areas are generally lower than those found in urban road dusts. With reference to Tables 2.7 and 2.8, it can be seen that Strehlow and Barltrop (1987) found that lead levels in housedust were only around half the value of those in corresponding road dusts in inner London, while Culbard et al. (1988), in their study of urban areas throughout England, Scotland and Wales, found that lead concentrations in housedust were about 30 per cent lower than those in road dust. Lower lead values in housedust than in road dusts were also reported by Davies and Thornton (1987) in their study in Brighton.

Lead concentrations in dusts from houses situated in the vicinity of industries which use or process lead are often higher than those found in urban housedusts from non-industrialised situations (Table 2.8). For example, Landrigan and Baker (1981) reported an extremely high mean value of 22190 μg/g in housedust samples from properties within 1.6 km of a smelter in El Paso, Texas, but noted lower levels, mean of 973 μg/g, in houses situated at a distance of 6.4 km, or more, from the same smelter. Turner et al. (1980) found housedust lead concentrations ranging from 500 to 8700 μg/g in houses near works in London, while Brunekreef et al. (1981) reported levels ranging from 463 to 4741 μg/g, mean 1054 μg/g, in houses similarly situated in Arnhem, The Netherlands. From the results reported by Turner et al. (1980), it would appear that indoor dust lead concentrations in industrialised areas are lower than corresponding road dust concentrations (Table 2.7).

Elevated lead levels have been noted in dusts from the homes of lead workers. Reported mean values for such dusts listed in Table 2.8 range from around 1000 to around 3300 μg/g. It is apparent from the study of Kawai et al. (1983) that in Japan such lead-related activities as quench-hardening of cutlery in baths of molten lead and

type-printing are actually carried out within people's homes. This seems extraordinary but is reported to be an extensive practice in this country. The highest lead concentration found in the 13 housedust samples analysed was around 750480 μg/g, the sample having been swept from the windowsill in the living room of a house containing a cutlery tempering workshop. The study of Dolcourt et al. (1981) indicates that the somewhat unusual practice of burning old battery cases in stoves within homes can also result in elevated dust lead concentrations.

The concentration of lead in dusts from homes in mineralised areas can also be high. Barltrop et al. (1975) and Culbard et al. (1988) in their studies of houses in such areas in Derbyshire found geometric mean housedust lead concentrations of 1189 and 1170 μg/g, respectively. Davies et al. (1985), however, reported a geometric mean of only 340 μg/g in their study of housedust in an old lead mining area of north Wales.

2.5 LEAD IN WATER

The major inputs of lead to surface freshwater, identified earlier (Section 2.2.2), include deposition from the air, leaching from soils and mine wastes, surface run-off, direct industrial discharge and discharge from sewage works. The major source of lead in ground water, on the other hand, is the bedrock through which the water passes, although it has been suggested that the careless application of fertilisers or sewage sludge to agricultural land may provide a further input (EPA, 1986). Lead is present in rainwater and in untreated surface and ground water in either disolved or particulate form. The solubility of lead in water is a function of pH and hardness, which is a function of calcium and magnesium concentration (EPA, 1986). At neutral pH, much of the lead in freshwater becomes adsorbed onto particles and is

deposited in sediments, whereas at low pH, the process is usually reversed. The solubility of lead is reported to decrease as water hardness increases (EPA, 1986).

The concentration of lead in rainwater has been measured both in the UK (Cawse, 1980) and the USA (Lazrus et al., 1970) and results reveal similar mean concentrations, 27 and 34 µg/l, respectively, in the two countries. These concentrations are generally higher than those reported in surface waters. An extensive survey of surface waters in the US showed average lead concentrations of around 4 µg/l (Durum et al., 1971). In the UK, an examination of the lead concentration in some river and estuarine waters indicated that around 50 per cent had concentrations of less than 10 µg/l (DoE, 1974) and a survey of surface waters draining unmineralised areas of south-west England (Aston and Thornton, 1977) revealed mean levels ranging from <1 to 3 µg/l. Reviews by the National Research Council (NRC, 1980) and Chow (1978) suggest that the typical lead concentration of surface waters is around 4 µg/l.

It is not surprising to find that waters draining areas of lead mineralisation can have higher lead levels than surface waters in unmineralised areas. Elevated levels were reported in rivers flowing through mineralised areas of Wales by Abdullah and Royle (1973) and Aston and Thornton (1977) reported concentrations ranging from 11 to 200 µg/l, mean 73 µg/l, from such areas in south-west England. Waters receiving polluted discharges can also show elevated lead concentrations, the magnitude of which will be dependent upon the quantity of lead discharged and the dilution capacity of the receiving water (Harrison and Laxen, 1981). Direct discharge of industrial effluent to surface waters does occur but, more commonly, such discharges enter sewerage systems and are transported to sewage treatment plants. In addition to industrial effluents, sewage treatment plants receive domestic

wastewater and storm water run-off, both of which are likely to contain lead. Although sewage treatment will significantly reduce the lead content of these effluents, some lead will still be discharged with the final effluent from the works into surface waters (Lester et al., 1979). Run-off waters from motorways and major roads can also provide a significant direct lead input to surface waters (Laxen and Harrison, 1977; Hewitt and Rashed, 1988).

It is reported that lead concentrations in ground water are unlikely to be influenced by surface run-off or atmospheric deposition (EPA, 1986). In the USA, concentrations typically range from 1 to 100 µg/l (NAS, 1980). Lead concentrations are generally lower in ocean waters than in inland waters. Concentrations as low as 4 ng/l have been found in deep ocean waters, although in coastal waters which receive surface run-off, levels in excess of 5 µg/l can occur (Abdullah and Royle, 1973; Harrison and Laxen, 1981).

In the context of human exposure to lead, it is the concentration of lead in drinking water which is of prime interest. That is, the concentration of lead in water which has passed through the distribution system and has been drawn from a drinking water tap. There are a number of factors that can modify the lead concentration of water as it passes through the distribution system and these include the quality of the water itself (including the degree of softness), the amount of lead with which it is in contact, the time of contact and the water temperature (RCEP, 1983). For example, it is well known that where soft, acidic waters pass through lead plumbing systems (pipes, lead lined tanks), the lead concentration in the resultant tap water will be elevated (RCEP, 1983; EPA, 1986; Royal Society of Canada, 1986). In this context, it has been found that the addition of lime to such acidic waters (to obtain a pH value in excess of 8.5) can markedly reduce the plumbosolvency of the water and that,

in certain cases, the addition of orthophosphate will further reduce the solubility of lead by buffering the pH of the water supply (Richards and Moore, 1984). Tap water that has been standing in pipes overnight usually has a higher lead concentration than tap water obtained after a period of flushing. This is not only the case where lead pipes are in service (e.g. Moore, 1977; DoE, 1982a) but also where lead solder has been used to join copper pipes (Moore, 1977; Sharrett et al., 1982). It is apparent that the age of the "plumbing" is an important factor in the latter case and it is reported that the lead on the inner surface of the joints is leached away over a period of years (EPA, 1986).

The leaching of lead into drinking water from PVC pipes has also been noted where lead stearate has been used as a stabiliser in the PVC (Heusghem and De Graeve, 1973). Where roof catchment cistern systems are used, lead from atmospheric deposition will be washed from the roof and may accumulate in the cistern. Although use of these sytems is not a common practice, a recent survey of drinking water from 40 such systems in the US indicated that the water lead concentration exceeded 50 µg/l in nine cases (Young and Sharpe, 1984).

In Great Britain, a large drinking water survey, carried out during 1975 and 1976, showed that in around two-thirds of the random daytime samples collected, lead concentrations were less than 10 µg/l (DoE, 1977). Overall, around 10 per cent of the samples exceeded 50 µg/l, while around 4 per cent of the samples exceeded 100 µg/l. Looking at the results separately for England, Scotland and Wales reveals that in Scotland, where soft, acidic waters were, at that time, frequently encountered in combination with lead plumbing, around 34 per cent of the samples were greater than 50 µg/l and 21 per cent were above 100 µg/l. In 1980, EEC Directive 80/778/EEC on water quality, established a Maximum Admissible

Concentration (MAC) for lead in running water intended for human consumption of 50 μg/l (EEC, 1980). It is not valid, however, to make comparisons between results from the 1975/76 survey and the MAC because remedial treatment of water supplies and replacement of lead pipes, which has been widespread since the mid-1970's both in Scotland and in other parts of Britain, has resulted in dramatic decreases in water lead concentration in some areas (e.g. Moore et al., 1985; see Section 6.5). It is unfortunate that water lead data collected in recent years by the various water authorities has not been collated and published to provide a more up-to-date and relevant national picture of water lead concentrations in the UK. However, water lead concentrations were measured in random daytime samples in 24 British towns by Pocock et al. (1983) (as part of the British Regional Heart Study) between 1978 and 1980 and revealed that around 6 per cent of the samples exceeded 50 μg/l.

Other, more recent data on water lead levels in the UK have been reported in the literature. For example, Sherlock et al. (1985) found that about two-thirds of random daytime tap water samples collected in London had lead concentrations below 5 μg/l, 22 per cent had concentrations between 5 and 10 μg/l and 4 per cent had concentrations around 20 μg/l. Strehlow and Barltrop (1987) reported the combined results of studies in London and in a rural area of Suffolk which showed that 80 per cent of samples (random daytime) had less than 3 μg/l and 95 per cent, less than 10 μg/l. Raab et al. (1987) found a median water lead concentration (30 minute stagnation samples) of 17 μg/l in a recent study in Edinburgh.

It is reported that lead pipes are seldom in service in the US, except in certain northeastern states (EPA, 1986) and that the median lead concentration in municipal drinking water in the US is around 10 μg/l (EPA, 1986). A recent national survey in Canada revealed a low median

lead concentration of less than 1 µg/l (Royal Society of Canada, 1986).

2.7 LEAD IN FOOD

Lead reaches food crops through uptake from the soil and through deposition onto aerial surfaces both from the air and by soil splash. Lead in the soil, as mentioned previously (Section 2.4), may be of natural origin, from contaminated waste material such as sewage sludge or from aerial deposition. Not all soil lead, however, is in a form available for uptake by plants. There are many factors which influence the release of lead from soil to crops and these include inorganic and organic colloidal constituents of the soil, cation exchange capacity, pH and the concentration and form of lead in the soil (EPA, 1986). In comparison with other metals, the availability of lead in soil is reported to be low and, thus, even in moderately contaminated soils, plants may be growing mormally (RCEP, 1983). From a review of the literature, Davies and Thornton (1988) reported that, in broad terms, there is a direct relationship between the concentration of lead in the soil and that in the plant. The relative contribution made by the uptake of lead via aerial surfaces of plants has proved difficult to assess and work in this area is continuing. Harrison and co-workers (Harrison and Johnston, 1987; Harrison and Chirgawi, 1988) suggest that such uptake can contribute significantly to the lead content of plant tissues while Zimdahl and Koeppe (1977) concluded from a review of available data that less than one per cent of aerially deposited lead is likely to penetrate through the leaf cuticle in a variety of crops. Entry via stomata has been considered as a possible means of absorption but this has not been quantified.

Although many of the reported studies of the lead content of plants have been concerned with crops or

vegetables grown in areas of high soil lead concentration, surveys have been carried out to establish the more typical lead content of fruits, cereals and vegetables eaten by man. In a study to determine the "background" concentrations of lead and other metals in agricultural crops, the US Food and Drug Administration (FDA), Department of Agriculture and the EPA analysed over 1500 samples of the most common crops taken from a cross-section of geographical locations in the USA (Wolnik et al., 1983). Collection sites were remote from mobile and stationary sources of lead and soil lead concentrations were within the "normal" background range for the US, 8-25 μg/g. Mean concentrations ranged from 0.003 μg/g in sweet corn to 0.100 μg/g in peanuts (values cited on a fresh weight basis). Levels of lead in the other crops analysed, wheat, potatoes, soybeans and lettuce, fell within this range.

The Ministry of Agriculture, Fisheries and Food (MAFF) has also carried out analysis of the lead concentration in fruit and vegetables in the UK and Table 2.9 shows "background" values for the lead contents of fruits and vegetables unaffected by any elevated concentration of lead in the air or soil (MAFF, 1982). The values are broadly similar to those cited above from the US and similar values have also been reported for fruits and vegetables in France (Servant and Delapart, 1981). The current general limit for lead in food intended for sale for human consumption in the UK, set by the Lead in Food Regulations (1979), is 1 μg/g (fresh weight) and it can be seen that the "background" levels given in Table 2.9 fall well below this limit.

Relatively high lead levels, however, have been found in vegetables grown in household gardens or allotments in large conurbations and areas contaminated by past mining or smelting activity. A variety of vegetables grown on contaminated urban soils in London (Davies et

Table 2.9 Lead in fresh fruit and vegetables in the UK

Food Crop	Sample Number	Mean (μg/g fresh wt.)	Range (μg/g fresh wt.)
Cabbage	23	0.08	<0.01-0.51
Carrot	8	0.02	<0.01-0.04
Leek	4	0.06	0.04-0.07
Mushroom	6	0.06	0.03-0.13
Onion	11	0.06	<0.01-0.38
Pear	17	0.10	<0.01-0.23
Potato	19	0.04	<0.01-0.14
Plum	5	0.11	0.06-0.16
Rhubarb	5	0.07	0.04-0.15
Sprout	16	0.05	<0.01-0.24
Swede	8	0.02	<0.01-0.05
Tomato	10	0.06	0.01-0.14
Watercress	4	0.15	0.08-0.33

al., 1979; Davies, 1983a; Denton, 1988) and Birmingham (Davies and Houghton, 1984) have been shown to contain excess levels of lead. In the US, vegetable lead concentrations were also shown to be raised when grown on urban soils in Boston (Preer and Rosen, 1977) and Washington, DC (Preer et al., 1984).

A number of areas in Great Britain have a high lead content in the soil due to past mining or smelting activities and two of these areas, north-east Wales and Somerset, have been investigated in detail. Davies and Roberts (1975) reported that radish grown in gardens in the Halkyn Mountain area of Wales commonly contained lead in excess of 2 μg/g (fresh weight) and Davies and White (1981) confirmed these results for other vegetables (onion, lettuce, carrot, Brussels sprout), noting that lead tended to be retained in plant roots but that enough

could still be translocated to edible leaves to exceed the legal limit. In Somerset, England, the village of Shipham has been studied by Davies and Ginnever (1979) who reported that although soil lead values ranged from 597 to 2612 μg/g in the eight garden soils studied, the associated home grown vegetables did not generally contain excess levels of lead. The same community has been extensively investigated by Thornton et al. (1980) who found a wider range of soil lead concentration, 104 to 6880 μg/g, and reported lead levels in 168 samples of winter vegetables ranging from 0.12 to 890 μg/g (dry weight). Further work in Shipham by Sherlock (1983) revealed elevated levels of lead in a variety of vegetables, with very much higher levels in winter crops, range of means from 1.07 to 2.79 μg/g (fresh weight), than in the same vegetables grown in the summer, 0.09 to 0.29 μg/g (fresh weight). However, these elevated levels were not reflected in the analysis of duplicate diets from the area and the mean lead intake of Shipham residents, 0.64 mg/week (range 0.32-1.58 mg/week) was similar to that from a normal British diet, estimated from the MAFF Total Diet Study, of 0.66 mg/week (range 0.43-1.55 mg/week). Diaries kept by volunteers indicated that vegetables comprised 20 per cent of most diets and that a significant proportion came from outside the village. From the above discussions, it is apparent that even in areas of significant soil lead pollution, dietary lead intake is not necessarily elevated.

Several studies have shown that fallout from smelters can cause elevated levels in crops grown in surrounding soils. In the UK, a study near a smelter carried out by MAFF (1982) between 1974 and 1980, indicated elevated lead levels both in the outer leaves of Brussels sprouts, means ranging from 2.28 to 3.67 μg/g (fresh weight), and in the outer leaves of lettuce, range of means 2.64 to 7.76 μg/g (fresh weight). The concentrations of lead on the inner leaves were all well

below 1 μg/g (fresh weight) for Brussels sprouts although in the case of lettuce, concentrations in excess of this were noted on occasion. It was reported that a large proportion of this contamination could be removed by washing. Near a large smelter in the US, Yankel et al. (1977) reported lead concentrations of up to 770 μg/g (dry weight) in unwashed lettuce grown within a mile of the source. Lagerwerff and Brower (1974) concluded that persons living in the vicinity of a smelter in Kansas who consumed home produced vegetables and milk were likely to ingest at least 50 per cent more lead than if they had purchased the same items of food in Washington DC and Crecilius et al. (1974) suggested that the consumption of plants coated with dust derived from a smelter near Tacoma, Washington, was a cause for concern. Also, in an Australian study, elevated lead levels were noted in lettuce (mean = 23 μg/g dry weight) and other leaf vegetables (mean = 10.3 μg/g dry weight) grown in domestic gardens within 500 m of a copper smelter (Beavington, 1975). Other studies of non-food plants growing in the vicinity of smelters have also revealed elevated lead concentrations (Burkitt et al., 1972; Little and Martin, 1972; Gale and Wixson, 1979; Martin et al., 1982)

It is well established that plants growing near roads are often contaminated by lead and that the major effect is contained within 30-50 m of the road (Davies and Thornton, 1988). It has been reported that the extent of contamination of roadside crops depends on the following factors: (a) distance from the road; (b) traffic density; (c) duration of exposure; (d) plant species and (e) climatic factors such as wind direction (Page et al., 1971; MAFF, 1982; EPA, 1986). Much of the work on vegetation alongside roads has been concerned with non-food plants and is of limited value in the context of human dietary intake of lead. Such work does, however, provide an indication of the likely contamination of food crops should they be grown in such an environment. For

example, studies by Motto et al. (1970) revealed a clear distance-decline effect in the lead content of roadside grass sampled at several sites alongside motorways in New Jersey, as did those of Lagerwerff and Specht (1970) from sites in Maryland, Missouri and Ohio. Similar findings were reported for roadside grasses in Birmingham, England, by Davies and Holmes (1972). Animals grazing on, or given feed from, such grasses could provide an indirect pathway to human intake. Studies by Ward et al. (1978) showed an increase in the body burden of lead both in sheep allowed to graze alongside roads and in those fed forage from the roadside. It is unlikely, however, that this route would have any great significance to humans because of the poor absorption of ingested lead in animals and the fact that the bulk of retained lead is in the bones (Ratcliffe, 1981). Also, animals are very rarely fed exclusively on herbage grown very close to main roads (MAFF, 1982).

Although several studies of the lead content of plants grown near major roads and motorways have revealed elevated levels, it is difficult to ascertain the relative amounts of lead actually in plants compared with that on the surface of plants (Dedolph et al., 1970; Motto et al., 1970; Schuck and Locke, 1970; Ter Haar, 1970; Davies and Holmes, 1972; Rabinowitz, 1974; Welch and Dick, 1975; Crump and Barlow, 1980). The published data are unsystematic and indicate that the percentage of lead removed from the surface of fruit and vegetables by washing ranges from almost zero to 100 per cent, although 50 per cent is generally taken as an average value (RCEP, 1983). For example, data from Motto et al. (1970) and Davies and Holmes (1972) on roadside grasses indicate that around half of the plant lead content could be removed by washing. Schuck and Locke (1970), from their study of roadside cauliflowers, cabbages, tomatoes and oranges, concluded that at least 50 per cent of the surface lead could be removed by washing. Ter Haar (1970) found that inedible portions of several plants (bean leaves, corn

husks, soybean husks and chaff from oats, wheat and rice) had two to three times the lead concentration when grown near a busy highway compared with similar plants grown in a greenhouse supplied with filtered air. The edible portions of these and other plants, however, showed little or no difference in lead content between those grown in ambient air and those grown in filtered air. This author concluded that, except for lettuce, there was no measurable effect on the lead concentration of the edible portion due to lead in air. From a comprehensive review of the literature, Chamberlain (1983) reported that foliar deposition of airborne lead accounts for most of the lead on grass and on exposed leaves of other plants but that most plant tissues which enter the human diet are protected from direct fallout.

Another potential source of lead for food crops is from sewage sludge applied to agricultural land, although it has been reported that the extra lead introduced into soil from such applications has only a low availability to plants (Davies and Thornton, 1988). If sewage sludge is sprayed directly onto field plots, however, it has been suggested that the resulting sludge adhering to the surface of the plants could cause significant problems (Chaney and Lloyd, 1979).

The above discussions have focused on the lead content of crops in different environmental situations. Dietary lead can, however, also be derived from fish, meat, dairy products, fats, oils and drinks. Data on these other dietary sources have been provided for the UK by MAFF (1982). It was reported that the concentration of lead in the edible tissue of marine fish caught around the British Isles is unlikely to exceed 0.05 μg/g (fresh weight). Lead levels in fresh water fish were slightly higher but usually less than 0.5 μg/g (fresh weight) except in the case of eels which were found to exceed 1 μg/g (fresh weight) on occasion. The mean lead

concentration of meats ranged from 0.06 to 0.30 μg/g (fresh weight), with the highest values being noted in lamb liver. Dairy products generally contained less than 0.03 μg/g (fresh weight). An interesting example of the influence exerted by a geochemically lead rich environment on lead levels in grazing livestock is also provided within this report (MAFF, 1982). About 30 dairy farms in the old mining areas of north Derbyshire were estimated to have high lead concentrations in their soil. Most of the milk from these farms went to dairies in Sheffield and it was noted that there was a significantly higher lead concentration in milk from this city than for the rest of the UK, although the actual concentration was fairly low, 0.02 μg/g (fresh weight). Concentrations of lead in two batches of milk sampled in 1979 from cattle grazing in this area, on land affected by the operation of lead workings, showed mean values of 0.14 and 0.04 μg/g (fresh weight) and were elevated in comparison with control samples. Further sampling in 1980 and 1981 revealed mean values of 0.04 and 0.03 μg/g (fresh weight), respectively. The report indicates that it is because this milk is bulked with other milk from uncontaminated areas that the Sheffield milk samples do not contain lead at concentrations greater than 0.02 μg/g (fresh weight). It is concluded that this concentration of lead in milk does not give cause for concern. However, such a conclusion ignores the possibility that milk with a lead concentration of up to 0.14 μg/g (fresh weight) may well be consumed by a farmer and his family and, particularly in the case of a very young child, could provide a significant proportion of the total dietary lead intake.

The use of lead solder in the manufacture of cans for foodstuffs constitutes a major source of lead in the diet and it is reported that much of the lead in such foodstuffs derives from solder particles deposited on the internal surface whilst the can is in the vicinity of the soldering equipment (RCEP, 1983; EPA, 1986). Estimates by

the US FDA suggest that lead in solder contributes more than 66 per cent of the lead in canned food where a lead solder seam is used. This lead was thought to represent a contribution of 20 per cent to the total lead consumption in food (Federal Register, 1979). It was reported by the RCEP (1983) that the average lead concentration in the UK in uncanned food was around 0.06 μg/g (fresh weight) compared with 0.22 μg/g (fresh weight) in canned food and that from an estimated total dietary lead intake of 100 μg/day in adults, 15 μg/day was derived from solder in cans. In the UK, the maximum permitted concentration of lead in canned food intended for sale for human consumption by adults was recently lowered from 2 μg/g (fresh weight) to 1 μg/g (fresh weight) although in foods specially intended for infants and young children the limit is 0.2 μg/g (fresh weight) (RCEP, 1983; Lead in Food (Amendment) Regulations, 1985)

The contamination of foodstuffs with dust lead from preparation surfaces and the uptake of lead by foodstuffs during cooking provide additional potential sources of human exposure to lead. Although Gallacher et al. (1984) found a significant correlation between lead on food preparation surfaces and children's blood lead, this issue has yet to be addressed thoroughly in the literature. Indications are, however, that the uptake of lead during cooking can be significant. Little et al. (1981) reported that the amount of lead absorbed from cooking water varied between around 3 and 79 per cent. Rinsing the food after cooking resulted in losses of less than 1 per cent, indicating that the lead was firmly attached to the food. The authors concluded that even when water lead concentrations were relatively low (50 μg/l), the intake of lead resulting from boiling vegetables in this water may be 10 to 50 μg/day. Lead uptake by the foodstuffs during cooking has also been demonstrated by Smart et al. (1981) and Smart et al. (1983).

3. LEAD IN MAN

3.1 INTRODUCTION

The absorption, distribution, retention and excretion of lead in humans has been extensively documented from experimental studies on adults and it is from this literature base that most of the following discussions are drawn. However, as will become apparent in Section 3.3, which deals with the human health effects of lead, it is young children who constitute the population most at risk from lead. For obvious reasons, ethical considerations preclude the use of children in experimental studies and, as a consequence, the metabolism of lead in this section of the population is a poorly understood phenomenon.

3.2 METABOLISM OF LEAD

3.2.1 Absorption

There are two main routes by which lead is absorbed into the bloodstream - via inhalation into the respiratory system and via ingestion into the alimentary system. A third, less important, route, direct absorption through the skin, or percutaneous absorption, is significant only with certain organic compounds of lead and intake by this route is usually negligible in the general population (RCEP, 1983).

Beginning with the inhalation route, not all lead inhaled from ambient air reaches the lungs. Some particles are trapped on the moist surface of the trachea and subsequently transferred, by muco-ciliary action, to the oesophagus. Of the lead which reaches the lungs, only a proportion is deposited, the remainder being exhaled.

The International Commission for Radiological Protection (ICRP) provided an early model for the deposition and removal of lead from the lungs and upper respiratory tract (ICRP, 1966). Briefly, the model predicts that, for a typical adult male, 35 per cent of lead inhaled from ambient air is deposited in the airways, with most of this going to the lungs. The ICRP model predicts a total deposition of 40-50 per cent for particles with a mass median aerodynamic diameter (MMAD) of 0.5 μm and indicates that the absorption rate would vary, depending on the solubility of the particular chemical form.

From a number of experimental studies on the deposition of inorganic lead in the respiratory system, it appears that the deposition of airborne lead, as encountered by the general population, is approximately 30-50 per cent, depending on particle size and ventilation rates (Kehoe, 1961, 1961a, 1961b; Nozaki, 1966; Chamberlain et al., 1978; Morrow et al., 1980; Gross, 1981). Ventilation rate is reported to be particularly important with submicrometre particles where Brownian diffusion governs deposition, as a slower breathing rate enhances the frequency of collisions of particles with the alveolar wall. Chamberlain et al. (1978) collated data from a number of studies of the effect of particle size on lead deposition rate in the lung and showed that with increasing particle size, the deposition rate decreases to a minimum over the range where Brownian diffusion predominates, followed by an increase in deposition with size (> 0.5 μm MMAD) as impaction and sedimentation become the main deposition factors. It also appears that essentially all of the lead deposited in the lower respiratory tract is absorbed, irrespective of chemical form (Rabinowitz et al., 1977; Chamberlain et al., 1978; Morrow et al., 1980). This latter finding differs from the earlier predictions by the ICRP. Supporting evidence for total absorption of deposited lead is provided by post mortem tissue analysis for lead content. Barry (1975)

found that lead was not accumulated in the lungs of lead workers and data from Gross et al. (1975) also showed that there was no lead accumulation in non-occupationally exposed subjects.

No measurements have been reported on the proportional deposition of air lead in, and absorption from, the lungs of children. It is difficult to estimate these from the results of experiments on adults as the respiratory rate is greater in young children and their airways are smaller than in adults. Although two year old children weigh around one-sixth as much as adults, it has been reported that they inhale 40 per cent as much air lead as adults (Barltrop, 1972). Differences in airway dimensions between adults and 10 year old children have been investigated by James (1978) who estimated that a child of this age has a deposition rate 1.6 to 2.7 times higher than an adult. It is likely, therefore, that a greater proportion of inhaled lead is deposited in the lower respiratory tract of children than of adults. A value of 70 per cent deposition, or retention, was suggested by the Lawther Working Party (Department of Health and Social Security [DHSS], 1980) with 100 per cent of the deposited lead being absorbed. The resulting combined retention and absorption factor of 0.7 was also endorsed by the RCEP (1983) as being an appropriate figure for two year old children.

Gastrointestinal absorption of lead in adults primarily involves uptake from food and beverages, although absorption of lead originally deposited in the upper respiratory tract and then swallowed, also occurs. Further lead uptake can occur, particularly in children, from the ingestion of non-food items, such as dust, soil and paint, through both mouthing activity and pica. Based on long term metabolic studies with adult volunteers, Kehoe (1961a, 1961b) estimated that approximately 10 per cent of dietary lead is absorbed. However, these studies

did not take into account the contribution of biliary clearance of lead into the gut and a higher estimate of 15 per cent absorption was obtained by Chamberlain et al. (1978) when this was taken into consideration.

Although no experimental studies have been carried out using children, an indication of the gastrointestinal absorption of lead in this group can be obtained from balance studies. In such studies, the lead excreted in faeces and urine, as well as that ingested in the diet, is assessed and the net gain or loss of lead calculated. Eleven balance studies were carried out by Alexander et al. (1973) on eight normal children, aged three months to eight and a half years, which indicated an average absorption of around 53 per cent. Ziegler et al. (1978) carried out a number of three day balance studies on each of nine infants aged two weeks to two years, who were on ordinary diets and found an average absorption of 43 per cent. In 104 balance studies on 29 hospitalised children aged between three weeks and 14 years, Barltrop and Strehlow (1978) reported that around one half of the children were in negative balance. Also wide individual variations in net retention of lead were observed ranging from a negative balance of 57 per cent to a positive balance of 99 per cent, with an average negative balance of around 40 per cent of the dietary intake. It should be noted that as these children were hospitalised, the results may not necessarily reflect the response of children living in a "normal" environment.

A number of recent studies indicate that lead ingested under fasting conditions is absorbed to a much greater extent than when it is taken with, or incorporated into, food. Blake (1976) found that lead uptake (^{203}Pb-labelled lead chloride) was 45 per cent after a fasting period compared to 6 per cent when administered with food, while Heard and Chamberlain (1982), using a similar procedure, noted around 63 per cent absorption after

fasting. Rabinowitz et al. (1980) reported an absorption of 35 per cent when ^{204}Pb was ingested after 16 hours of fasting. Studies by Barltrop (1975) and Garber and Wei (1974) indicate that beverage lead is absorbed to a greater extent than is lead in food. The isotope studies outlined above support these observations as the beverages were consumed between meals and, therefore, when the food content of the gastrointestinal tract was low. If the results from these experimental studies on adults are also valid for young children, the implication is that lead consumed with drinking water and non-food items, such as dust, soil and paint, is more important than equal amounts of lead consumed with solid food, as they are usually ingested between meals. However, as there are no suitable data available for young children, this is speculative at present.

The question of bioavailability of lead in ingested soil and dust has been addressed by several workers. Various studies on wildlife unable to avoid the ingestion of soil or dust, e.g. feral pigeons (Hutton and Goodman, 1980) have indicated that bioavailability may be appreciable. Also, livestock grazing on soils rich in lead, e.g. those contaminated with mine wastes or geochemically lead rich, have often shown signs of lead toxicity thought to be the result of direct ingestion of soil (RCEP, 1983). One study has indicated that between 40 and 70 per cent of the lead intake of cattle may be derived from the ingestion of soil (Thornton and Abrahams, 1983). Other studies have involved the administration of lead contaminated dust and soil to laboratory animals. Stara et al. (1973) reported studies of rats fed either tunnel, motorway or smelter dusts. Accumulation of lead in body tissue was non-linear with dose, with a lower percentage absorption at higher doses. The studies showed that the smelter dust (6700 μg/g) had an appreciably lower effect on blood, bone and kidney than either tunnel dust (22200 μg/g) or highway dust (10400 μg/g). Dacre and Ter

Haar (1977) conducted an evaluation of the bioavailability in rats of lead in soil from alongside houses (990 μg/g) and alongside roads (2300 μg/g) compared with lead acetate. A high fibre, high nutrient basal diet was used which was amended with the two soils and lead acetate to contain 50 mg lead per kg diet. Results from the analysis of bone tissue indicated that soil lead was about 70 per cent as bioavailable as lead acetate.

A somewhat more detailed evaluation of the effect of soil on dietary lead absorption has been completed by Chaney et al. (1984). Rats were fed purified complete casein-sucrose diets with and without 5 per cent uncontaminated soil and with and without 50 μg/g lead in the diet as lead acetate to test the effect of dietary soil on lead absorption. Rats were also fed urban garden soil of varying concentration to compare bioavailability of lead rich soils with that of lead acetate. All lead was added at 50 mg lead per kg of dry diet, i.e. unequal amounts of soil were added. Bone lead concentrations were used to evaluate the diet lead bioavailability. The addition of 5 per cent uncontaminated soil to the diet reduced lead absorption to 53 per cent of the lead acetate control. Four soils with lead concentrations around 1000 μg/g resulted in bone lead levels about 25 per cent of those found using lead acetate, while using soil with a lead concentration of around 10000 μg/g resulted in lead levels of 70 per cent of those found with a lead acetate diet. The general trend showed increased soil lead bioavailability at higher soil lead concentrations. It should be remembered, though, that when soil lead concentrations were higher, the percentage of soil in the diet was correspondingly lower. The workers (Chaney et al., 1984) concluded that the soil was probably acting like a fibre and a calcium and phosphorus source in the diet and therefore would be expected to adsorb lead in the lumen of the intestine and reduce lead absorption. An indication of the influence of a high fibre, high nutrient

diet on the absorption of dietary lead can also be gained by a comparison of the results from the studies of Chaney et al. (1984) and Dacre and Ter Haar (1977). The experimental protocols used by both groups of workers were similar although, as noted above, the basal diets differed. Tibia lead concentrations reached 247 μg/g in rats fed 50 mg lead (as lead acetate) per kg purified diet while femur lead levels of only 5-7 μg/g were noted in rats fed the same level of lead (as lead acetate) in a high fibre, high nutrient diet. Although these experimental results are not truly comparable because the work was carried out in different laboratories and different bone types were analysed, the differences do appear to be quite marked.

Other, more indirect, work has also been carried out to investigate the bioavailability of ingested dust and soil lead. Samples have been subjected to chemical extraction procedures thought to simulate the natural processes which operate in the gut and results have indicated that soil and dust lead can be very soluble under such conditions (Day et al., 1979; Harrison et al., 1981; Chaney et al., 1984; Gibson and Farmer, 1984). However, solubility does not necessarily directly equate with bioavailability and it is difficult to draw firm conclusions from such studies regarding the absorption of such ingested lead from the gastrointestinal tract. What is clear is that more research is needed to characterise the bioavailability of dust and soil lead.

It is apparent that the chemical form in which lead is administered has a minimal influence on its uptake from the gut (Harrison et al., 1969; Karhausen, 1973: Barltrop and Meek, 1975; Rabinowitz et al., 1980; Heard and Chamberlain, 1982; Mahaffey, 1983). The physical size, however, appears to be of some relevance, with small particles being more effectively absorbed than large ones (Barltrop and Meek, 1979). Various nutritional factors

have been shown to influence the absorption of ingested lead. Using adult subjects, Heard and Chamberlain (1982) monitored the uptake of ^{203}Pb from the gut, as a function of the amounts of dietary calcium and phosphorus. Without supplementation with either of these minerals in fasting subjects, uptake averaged around 60 per cent, compared with an uptake of around 10 per cent with 200 mg of calcium plus 140 mg of phosphorus, the amounts present in an average meal. Calcium alone reduced uptake by a factor of 1.3 to around 52 per cent and phosphorus alone by a factor of 1.2 to around 47 per cent. It has been suggested that when calcium and phosphorus are present together insoluble calcium phosphate is formed which co-precipitates any lead present (EPA, 1986). Several other studies have indicated that blood lead concentration is inversely related to the intake of calcium and phosphorus. Mahaffey et al. (1976) found that children (1 to 4 years old) with blood lead concentrations greater than 1.92 µmol/l (40 µg/dl) had significantly ($p < 0.01$) lower intakes of calcium and phosphorus compared with a control group. Sorrell et al. (1977), in a study of 1 to 4 year old children, observed that blood lead concentration was inversely correlated with calcium intake and that children with blood lead levels greater than 2.88 µmol/l (60 µg/dl) had significantly ($p < 0.001$) lower intakes of calcium and vitamin D. Milk consumption has also been shown to be inversely related to blood lead in two observational studies (Johnson and Tenuta, 1979; Brunekreef et al., 1983) and in one human experiment (Blake et al., 1983), a phenomenon likely to be a reflection of the high calcium content of milk. Iron and zinc deficiency have also been associated with increased lead absorption from the gut. Watson et al. (1980) reported that adult subjects who were iron deficient showed two to three times greater lead absorption than subjects who were iron replete. Yip et al. (1981) found that children with elevated blood lead levels (1.44 µmol/l, 30 µg/dl) had an increased prevalence of iron deficiency as lead concentrations

increased, while Chisholm (1981) demonstrated an inverse relationship between chelatable iron and chelatable lead levels in a group of children with elevated blood lead levels. The mean serum zinc levels in children with plumbism were found to be significantly lower than the values in normal children in a study by Markowitz and Rosen (1981) and an inverse relationship between body lead levels, as indexed by urinary aminolaevulinic acid (ALA) levels, and the amount of systematically active zinc was noted by Chisholm (1981) in a group of children with blood lead levels ranging from 2.16 to 2.88 μmol/l (45 to 60 μg/dl).

There is some confusion over the role played by vitamin D in the absorption of lead from the gut. Hunter (1977, 1978) suggested that the seasonal variation of children's blood lead (that of higher levels in the summer months) could be the result of exposure to sunlight. The proposed mechanism was that in the summer, vitamin D increased the absorption of lead from the gastrointestinal tract. It has been demonstrated elsewhere, however, that the active vitamin D metabolite in this respect is likely to be 1,25 dihydroxy ($(OH)_2$) vitamin D, which has no seasonal variation, and that the metabolite 25 hydroxy (OH) vitamin D which does show seasonal variation, is reported not to affect lead metabolism (Rosen et al., 1980; Chesney et al., 1981; Mahaffey et al., 1982). From a study of Asian children in the UK, however, a significant ($p < 0.001$) negative correlation was observed between plasma 25-OH vitamin D and blood lead concentration (DoE, 1982) although in a more recent UK study of both Asian and Caucasian children, no significant correlation was found between these two measures (Sherlock et al., 1985). It was noted by the latter authors that such correlations have been found only when some of the children studied had blood lead concentrations greater than those observed in their study (geometric mean = 0.41 μmol/l, 8.5 μg/dl).

It is apparent from the above discussions that there are many factors which can affect the absorption of lead from the gut in both adults and children and that the processes involved are complex and not yet fully understood, particularly in the latter group. However, for the purposes of assessing general population exposure to lead it is useful to have an estimate of the average absorption from the gastrointestinal tract. The Lawther Working Party (DHSS, 1980) reported the net average absorption of ingested lead by adults to be around 10 per cent, while that for children to be higher, around 40 per cent. The RCEP (1983) chose to use the same estimate of 10 per cent absorption for adults in their calculations but, citing the work of Alexander et al. (1973), suggested that a higher estimate of 53 per cent was more appropriate for two year old children.

3.2.2 Distribution

Initially, lead taken up into the bloodstream from the gastrointestinal tract or the respiratory tract is distributed according to the rate of delivery of blood to the various organs and systems. Lead is then redistributed to organs and systems in proportion to their respective affinities for the element. With constant exposure, for an extended period of time, a near steady-state of intercompartmental distribution is achieved.

More than 99 per cent of blood lead is reported to be associated with the erythrocytes, under normal conditions (Manton and Cook, 1979; Everson and Patterson, 1980; De Silva, 1981), although it is the small fraction of lead transported in plasma and extracellular fluid which provides lead to the various body organs (Baloh, 1974). Most of the erythrocyte lead is bound within the cell, primarily associated with haemoglobin (Raghavan et al., 1981). From the literature, it appears that whole blood lead has a biological half-life of between 25 and 28

days. The mean residence time of whole blood lead was found to be 35 days, equivalent to a 25 day half-life, in studies by Rabinowitz et al. (1976). Chamberlain et al. (1978) established a similar half-life for ^{203}Pb in blood when the labelled lead was administered via ingestion, inhalation or injection. Studies described by Griffin et al. (1975) reveal half-lives of 28 and 26 days for the inhalation of 10.4 and 3.1 μg Pb/m^3, respectively.

Alterations in blood lead levels in response to abrupt changes in exposure apparently occur over somewhat different periods, depending on whether exposure has increased or decreased. With increased lead intake, blood lead achieves a new value in approximately 40 to 60 days (Tola et al., 1973; Griffin et al., 1975), while a decrease in exposure may involve a longer period of time, depending on the magnitude of the past exposure (Rabinowitz et al., 1977; Gross, 1981; O'Flaherty et al., 1982). Lead in the small but biologically significant pool in blood plasma is virtually all bound to albumin, with only trace amounts bound to high weight globulins (Ong and Lee, 1980). Recent studies of the erythrocyte-plasma relationship in humans indicate that there is an equilibrium between these compartments such that levels in plasma rise with levels in whole blood (Cavalleri et al., 1978; Everson and Patterson, 1980; De Silva, 1981). There appears to be little change in blood lead with age during adulthood, although in children there is usually a peak at two to three years, followed by a decline (De Silva and Donnen, 1980; DoE, 1981a, 1983; Annest et al., 1982; McMichael et al., 1985). In older children and adults, levels are sex-related, with females showing lower levels than males, even at comparable levels of exposure (see Section 6.6).

Soft tissue lead levels generally stabilise in early adult life and show a turnover rate similar to blood which is sufficient to prevent accumulation. There is, however,

continuing accumulation in the renal cortex which may be a reflection of the formation of lead containing nuclear inclusion bodies (Cramer et al., 1974; Indraprasit et al., 1974). Also, aortic levels appear to rise with age, although it has been suggested that this may be due to the entrapment of lead in atherosclerotic deposits (Barry, 1975; Gross et al., 1975). In general, the lead content of soft tissue is below 0.5 μg/g wet weight, although higher levels are found in the renal cortex and aorta (Horiuchi et al., 1959; Gross et al., 1975; Barry, 1975, 1981). The relatively constant lead concentration reported in lung tissue across age groups suggests no accumulation of respired lead and is consistent with data for deposition and absorption discussed previously. In a small group of samples from subjects with known or suspected occupational exposure, Barry (1975) found elevated lead levels in liver, brain, skin, pancreas, prostate and aorta. The distribution of lead across brain regions has been studied in both adults and children. The data of Niklowitz and Mandybur (1975) and Grandjean (1978) for adults and those of Okazaki et al. (1963) for post-mortem samples from young children who died of lead poisoning, are consistent in showing that lead is selectively accumulated in the hippocampus. Subcellular distribution of lead in soft tissue is not uniform, with high amounts of lead being sequestered in the mitochondria and nucleus. Cramer et al. (1974) studied renal biopsy tissue in lead workers and observed mitochondrial changes and lead-binding nuclear inclusion bodies in renal proximal tubules.

Lead becomes localised and accumulates in human calcified tissue, a process which can begin during foetal development (Horiuchi et al., 1959; Barltrop, 1969). Various workers have documented that around 95 per cent of total body lead is present in bone and that lead levels increase until around 60 years of age, when a decrease occurs with changes in intake as well as in bone mineral

metabolism (Horiuchi et al., 1959; Schroeder and Tipton, 1968; Horiguchi and Utsonomiya, 1973). The proportion of body lead in bone is reported to be lower in children (around 73 per cent) than in adults, although concentrations of lead in bone increase more rapidly than in soft tissue during childhood (Barry, 1975, 1981). Total accumulation in bone by 60 years of age ranges up to approximately 200 mg, although occupational exposure can raise this figure several fold. A mean value of 9 mg has been reported from studies of 23 children aged from 16 years and under (Barry, 1975, 1981).

In adults, bone lead is the most inert pool and accumulation can serve to maintain elevated blood lead levels years after past exposure, particularly occupational, has ended. This is likely to account for the observation that duration of exposure correlates with rate of reduction of blood lead after termination of exposure (O'Flaherty et al., 1982). The biological half-life of lead in bone has been estimated to be in the order of several decades (Rabinowitz et al., 1976; Holtzman, 1978) although it has been suggested that there may be two bone compartments, one of which is a repository for relatively labile lead (Rabinowitz et al., 1977). Tooth lead levels increase with age at a rate proportional to exposure (Steenhaut and Pourtois, 1981) and are also roughly proportional to blood lead levels (Winneke et al., 1981). Dentine zones of lead enrichment related to abrupt changes in exposure have been documented and it has been suggested that dentine is the most responsive component of teeth to lead exposure (Needleman and Shapiro, 1974; Fremlin and Edmonds, 1980). The rate of lead deposition in teeth appears to vary with tooth type, being highest in the central incisors and lowest in the molars (Mackie et al., 1977; Delves et al., 1982).

3.2.3 Retention and Excretion

Dietary lead which is not absorbed passes through the gastrointestinal tract and is eliminated in faeces, as is that deposited fraction of air lead which is swallowed and not absorbed. Lead absorbed into the bloodstream and not retained is excreted through the renal and gastrointestinal tracts, the latter by biliary clearance. Based on the metabolic balance data and isotope excretion findings of various workers, it appears that short-term lead excretion in adults amounts to 50 to 60 per cent of the absorbed fraction, the balance moving primarily to bone, with some subsequent fraction (approximately half) of this stored amount eventually being excreted (Kehoe, 1961, 1961a, 1961b; Rabinowitz et al., 1976; Chamberlain et al., 1978). The rapidly excreted fraction was determined by Chamberlain et al. (1978) to have an excretion half-life of about 19 days, which is consistent with other estimates (Rabinowitz et al., 1976). By use of the stable isotope, ^{204}Pb, Rabinowitz et al. (1973) reported that urinary and faecal lead excretion accounted for 76 and 16 per cent, respectively, of the measured recovery, the remainder being excreted via hair, sweat and nails. Following either injection or oral intake of ^{203}Pb, the amounts of labelled lead in urine and faeces were compared for the two administration routes by Chamberlain et al. (1978). Endogenous faecal lead was 50 per cent of that in urine, that is a 2 to 1 ratio of urinary to faecal lead was found. The age dependency of lead excretion in humans is poorly understood due to the fact that there are few relevant data for children available. It has been reported, however, that infants appear to have a lower total excretion rate than adults, although the excretion of endogenous faecal lead may be higher than for adults (EPA, 1986).

In normally exposed humans, it has been calculated that lead is retained in bone with a biological half-life

of around 17 years (Holtzman, 1978), although other estimates put this in the order of several decades (Rabinowitz et al., 1976). Lead labelling experiments, using adult subjects, indicate a short-term or initial retention of around 40 to 50 per cent of the fraction absorbed, much of which is by bone (Chamberlain et al., 1978) and it has been estimated that 25 per cent of the lead absorbed daily undergoes long-term bone storage (EPA, 1986). Studies on children suggest that lead retention can be higher in this group than in adults. By means of metabolic balance studies, Ziegler et al. (1978) obtained a retention figure (as a percentage of total intake) of 31.5 per cent for infants, while Alexander et al. (1973) provided an estimate of 18 per cent. The balance studies of Barltrop and Strehlow (1978), however, indicated a net negative retention in their hospitalised subjects. As the lead exposure of these children could have changed on admission, the results may have been a reflection of lower lead intakes in this situation.

3.3 EFFECTS

3.3.1 Introduction

There is a vast body of literature on the toxicity of lead and this has recently been comprehensively reviewed by the EPA (1986) and the US Public Health Service (Agency for Toxic Substances and Disease Registry [ATSDR], 1988). The purpose of this section, therefore, is not to duplicate such work with detailed assessments of individual studies, but rather to summarise the major findings reported.

Most studies on the human health effects of lead have used blood lead concentration as the indicator of body lead burden to which these effects have been related. The lowest-observed-effect levels, in terms of blood lead

concentrations associated with particular human health effects, have been summarised by the EPA (1986) for both adults and children and these are presented in Tables 3.1 and 3.2, respectively.

With reference to these tables, it can be seen that at low blood lead levels, biochemical changes such as the disruption of certain enzyme activities involved in haem synthesis are detectable. At increasing blood lead concentrations, there are sequentially more pronounced effects on haem synthesis and a broadening of effects to additional biochemical and physiological mechanisms in various tissues, such that increasingly more severe disruption of the normal functioning of many different organ systems becomes apparent. In addition to impairment of haem synthesis, signs of disruption of normal functioning of the erythropoietic and nervous systems are among the earliest effects observed in response to increasing blood lead concentrations. At increasingly higher blood lead levels, more severe disruption of these systems occurs along with renal effects, a disruption of reproductive functions, an impairment of immunological functions and many other biological effects. At sufficiently high levels, the damage to the nervous system and other effects can be severe enough to result in permanent mental retardation or, in some cases, death.

The biological basis for lead toxicity is linked to the ability of lead, on contact with a tissue, to bind with some ligand in that tissue. This binding can interfere with the normal physiological functioning of the molecule in question and result in, for example, the inhibition of enzyme activity and the inhibition or other alterations of essential ion transport.

The main target organelle for lead toxicity, in a variety of cell and tissue types, is the mitochondrion. Mitochondrial effects take the form of structural changes

Table 3.1 Summary of lowest-observed-effect levels for key lead-induced health effects in adults

Lowest-observed-effect level (PbB) µg/dl	µmol/l	Haem synthesis and haematological effects	Neurological effects	Effects on the kidney	Reproductive function effects
100-120	4.80-5.76		Encephalopathic signs and symptoms	Chronic nephropathy	
80	3.84	Frank anaemia			
60	2.88				Female reproductive effects
50	2.40	Reduced haemoglobin production	Overt subencephalopathic neurological symtoms		Altered testicular function
40	1.92	Increased urinary ALA and elevated	Peripheral nerve dysfunction		
25-30	1.20-1.44	EP elevation in males			
15-20	0.72-0.96	EP elevation in females			
<10	<0.48	ALA-D inhibition			

Key: PbB = Blood lead concentrations
ALA = Delta-aminolaevulinic acid
ALA-D = Delta-aminolaevulinic acid-dehydratase
EP = Erythrocyte protoporphyrin

Source: EPA (1986)

Table 3.2 Summary of lowest-observed-effect levels for key lead-induced health effects in children

Lowest-observed-effect level (PbB) µg/dl	µmol/l	Haem synthesis and haematological effects	Neurological and related effects	Renal system effects	Gastrointestinal effects
80-100	3.84-4.80		Encephalopathic signs and symptoms	Chronic nephropathy	Colic and other overt gastrointestinal symptoms
70	3.36	Frank anaemia			
60	2.88		Peripheral neuropathies		
50	2.40				
40	1.92	Reduced haemoglobin synthesis	Peripheral nerve dysfunction		
		Elevated coproporphyrin	CNS cognitive effects		
		Increased urinary ALA			
30	1.44			Vitamin D metabolism interference	
15	0.72	EP elevation	Altered CNS electrophysiological responses		

contd./

Table 3.2 contd.

Lowest-observed-effect level (PbB) µg/dl	µmol/l	Haem synthesis and haematological effects	Neurological and related effects	Renal system effects	Gastrointestinal effects
10	0.48	ALA-D inhibition	MDI deficits, reduced gestational age and birth weight (prenatal exposure)		
<10	<0.48 ?	Py-5-N activity inhibition			

Key: PbB = Blood lead concentrations
ALA = Delta-aminolaevulinic acid
ALA-D = Delta-aminolaevulinic acid-dehydratase
EP = Erythrocyte protoporphyrin
Py-5-N = Pyrimidine-5'-nucleotidase
MDI = Mental development index

Source: EPA (1986)

and marked disturbances in mitochondrial function within the cell, especially energy metabolism and ion transport. These effects have been associated with demonstrable accumulation of lead in mitochondria in both in vivo and in vitro animal studies (Castellino and Aloj, 1969; Barltrop et al., 1971; Walton and Buckley, 1977; Silbergeld et al., 1977). Also, structural changes have been documented in lead workers, across a wide range of exposure levels, which include mitochondrial swelling in renal biopsy tissue, along with distortion and loss of cristae (infoldings of the inner membrane) (Wedeen et al., 1975; Biagini et al., 1977). From an extensive review of the literature concerning the effects of lead on mitochondrial function in animals (both in vivo and in vitro) by Bull (1980), it is apparent that uncoupled energy metabolism, inhibited respiration and altered kinetics of intracellular calcium can result. Given the diverse evidence of lead's impairment of mitochondrial structure and function, as viewed from a subcellular level, it is not surprising that these effects are thought to be the basis of dysfunction of haem synthesis, erythropoiesis and the central nervous system, discussed below.

Lead exposure also provokes a typical cellular reaction in humans and other animal species that has been morphologically characterised as a lead containing nuclear inclusion body (Goyer and Moore, 1974). These bodies can appear rapidly after lead exposure and have been postulated to serve a protective role in the cell (Choie et al., 1975). However, any such role appears to be an imperfect one as both subcellular organelle injury and lead uptake can occur simultaneously with lead-inclusion body formation (Fowler et al., 1980; Osheroff et al., 1982).

3.3.2 Effects of lead on haem synthesis

The effects of lead on haem synthesis have been widely studied. In addition to being a constituent of haemoglobin, haem is also incorporated into other high molecular weight proteins such as the cytochrome P-450 component of the mixed function oxidase system and the various cytochromes of the respiratory or electron transport chain (ATSDR, 1988). As the disturbance of haem synthesis by lead can affect fundamental metabolic and energy-transfer processes, lead poses the potential for multiple organ toxicity. A summary of the principal steps in the haem synthesis pathway and the effects of lead on enzyme activity, as outlined recently by the EPA (1986), is given in Figure 3.1. While this simplified schematic will suffice as the basis for the following discussion, more detailed descriptions of this pathway have been documented elsewhere (e.g. Moore et al., 1980).

The first and final three steps in the haem synthesis pathway occur within the mitochondrion, while the intermediate stages occur in the cytoplasm. Lead interferes with haem synthesis by altering the activity of three enzymes, delta-aminolaevulinic acid synthase, delta-aminolaevulinic acid dehydratase and ferrochelatase[1] (EPA, 1986). It indirectly stimulates, through feedback derepression, the mitochondrial enzyme delta-aminolaevulinic acid synthase (ALA-S). This enzyme catalyses the condensation of glycine and succinyl-coenzyme A to form delta-aminolaevulinic acid (ALA) and its activity controls what is the rate limiting step in this pathway. Increases in ALA-S activity have been documented in lead workers by Takaku et al. (1973),

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1. Moore et al. (1980), however, cite evidence from Campbell et al. (1977) which indicates that the activity of a fourth enzyme in this pathway, coproporphyrinogen oxidase, is also inhibited by lead.

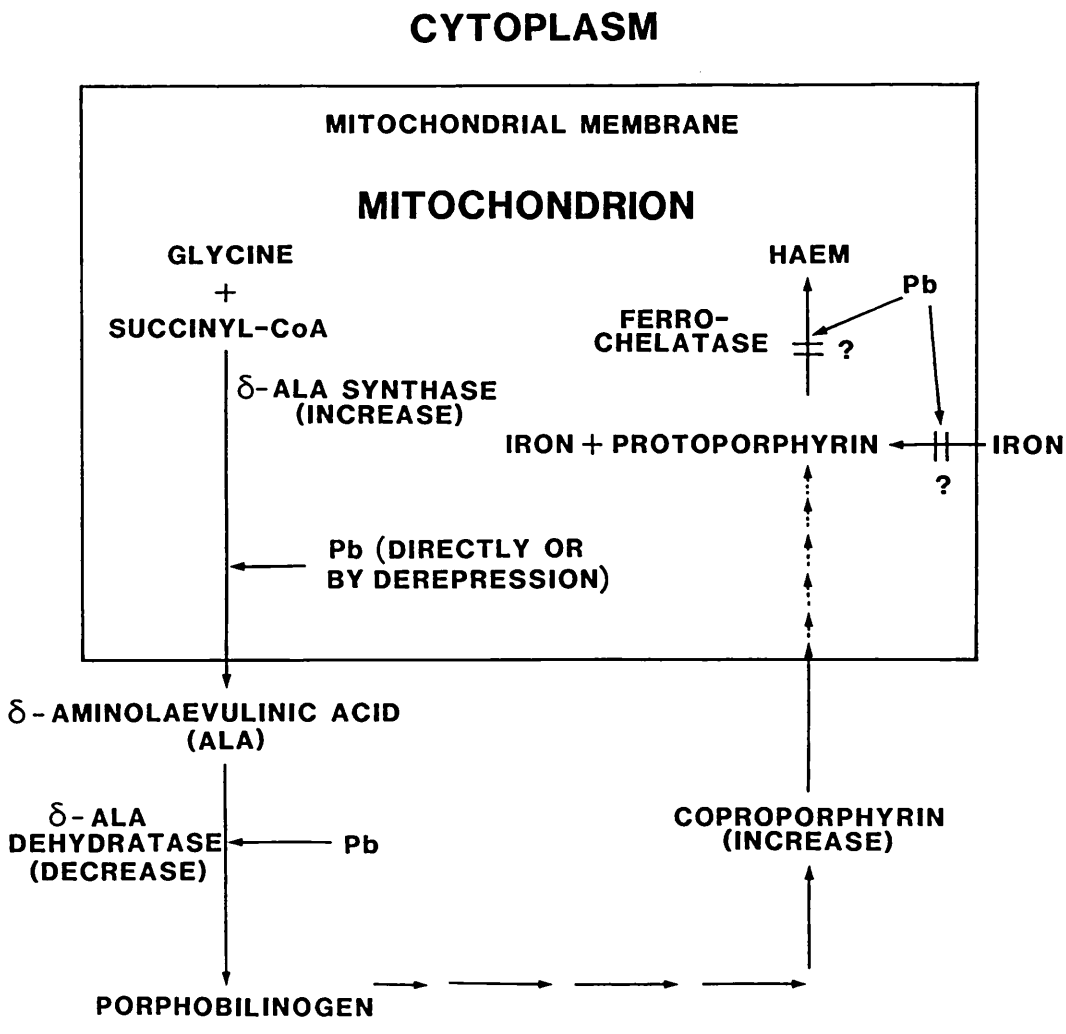


Figure 3.1 Effects of lead on haem synthesis

Campbell et al. (1977) and Meredith et al. (1978).

Lead also directly inhibits delta-aminolaevulinic acid dehydratase (ALA-D). This cytosolic enzyme catalyses the condensation of two units of ALA to form porphobilinogen. ALA-D activity has been shown to be inversely correlated with blood lead concentration in several studies of occupationally exposed individuals (ATSDR, 1988). General population studies indicate that the activity of ALA-D is inhibited at very low blood lead levels, with no threshold yet apparent. In a study of urban subjects not exposed occupationally to lead, Hernberg and Nikkanen (1970) found that ALA-D activity was inversely correlated with blood lead levels over the entire range of 3 to 34 μg/dl (0.14 to 1.63 μmol/l). Other studies have confirmed this inverse correlation and apparent lack of threshold (Roels et al., 1976; Chisholm et al., 1985). The resultant effect of the inhibition of ALA-D and the stimulation of ALA-S is an accumulation of ALA which is reflected in increased levels of ALA in blood or plasma and urine. A study of lead workers by Meredith et al. (1978) indicates an exponential relationship between blood lead and blood ALA. Numerous other studies of occupationally exposed individuals have shown a direct correlation between blood lead and the logarithm of urinary ALA, some at levels below 40 μg/dl (1.92 μmol/l) (EPA, 1986). A similar relationship has also been found in children, primarily at blood lead levels greater than 40 μg/dl (1.92 μmol/l) (ATSDR, 1988).

Lead also decreases the activity of the mitochondrial enzyme ferrochelatase. This enzyme catalyses the insertion of iron into the protoporphyrin ring to form haem, in the final step of the haem synthesis pathway (Figure 3.1). It is reported that the action of lead on this enzyme occurs either by direct inhibition or by alteration of intramitochondrial transport of iron (Moore and Goldberg, 1985; EPA, 1986). This disturbance

of the pathway results in an accumulation of protoporphyrin IX, which is present in the circulating erythrocytes as zinc protoporphyrin (ZPP) due to the placement of zinc, rather than iron, in the porphyrin moiety. ZPP is bound in the haem pockets of haemoglobin and remains there throughout the life of the erythrocyte (ATSDR, 1988). As accumulation of ZPP occurs only in erythrocytes formed during the presence of lead in erythropoietic tissue, this effect will be detectable in circulating erythrocytes only after a lag time reflecting maturation of erythrocytes (Moore and Goldberg, 1985; EPA, 1986). It should be noted here that assays used in studies of protoporphyrin accumulation measure ZPP or total erythrocyte protoporphyrin (EP). EP is also known as free erythrocyte protoporphyrin (FEP) because ZPP is converted to this form during extraction. The elevation of erythrocyte EP or ZPP in lead workers has been documented extensively and the EPA (1986) concluded from a review of a number of studies that there was an apparent threshold in male workers at blood lead concentrations of around 25 to 30 µg/dl (1.2 to 1.44 µmol/l)and in female workers at around 15 to 20 µg/dl (0.72 to 0.96 µmol/l). In children, levels of erythrocyte EP or ZPP are reported to be exponentially correlated with blood lead levels and there is an apparent threshold at a blood lead concentration of around 15 µg/dl (0.72 µmol/l) (ATSDR, 1988).

An increase in urinary coproporphyrin has long been recognised in workers with lead poisoning and used as an indicator of excessive exposure to lead (EPA, 1986). The EPA (1986) identified a lowest-observed-effect level for elevated urinary coproporphyrin at a blood lead level of 40 µg/dl (1.92 µmol/l) but did not present the basis for this conclusion nor discuss the possible biochemical basis for such an elevation. Campbell et al. (1977) have reported that lead inhibits the mitochondrial enzyme coproporphyrinogen oxidase (which catalyses the conversion

of coproporphyrinogen to protoporphyrinogen) and concluded that it is this inhibition which results in elevated levels of coproporphyrin in urine.

One of the results of a marked interference with haem synthesis is a reduction of the haemoglobin concentration in blood. Decreased haemoglobin production, coupled with an increase in erythrocyte destruction, results in a hypochromic, normocytic anaemia with associated reticulocytosis. It has been suggested that the increase in erythrocyte destruction may be partly due to the inhibition by lead of another enzyme, pyrimidine-5'-nucleotidase, which results in an accumulation of pyrimidine nucleotides in the erythrocyte or reticulocyte (Angle et al., 1982; EPA, 1986). This enzyme inhibition and nucleotide accumulation affect erythrocyte membrane stability and survival by alteration of cellular energetics. Decreased haemoglobin concentrations and anaemia have been noted in lead workers. The EPA (1986) concluded that the threshold for a decrease in haemoglobin in occupationally exposed adults is a blood lead level of 50 µg/dl (2.4 µmol/l), while the threshold for frank anaemia in such adults is around 80 µg/dl (3.84 µmol/l). In children, the blood lead threshold for a decrease in haemoglobin levels is estimated to be around 40 µg/dl (1.92 µmol/l) and that for frank anaemia around 70 µg/dl (3.36 µmol/l)(World Health Organisation, 1977; EPA, 1986). These threshold levels are slightly lower than the corresponding levels in occupationally exposed adults cited above.

Erythrocyte pyrimidine-5'-nucleotidase activity has also been shown to be inhibited in lead workers and the EPA (1986) suggested that the threshold for inhibition in adults is a blood lead concentration of around 44 µg/dl (2.11 µmol/l). The activity of this enzyme is reported to be inhibited in children at very low blood lead levels but there is no evidence to-date that a threshold exists. For

example, significant negative linear correlations between pyrimidine-5'-nucleotidase activity and blood lead level have been found both in a study of 21 children with blood lead levels ranging from 7 to 80 µg/dl (0.34 to 3.84 µmol/l) and in another of 42 children with levels ranging from less than 10 to 72 µg/dl (0.48 to 3.46 µmol/l) (Angle and McIntire, 1978; Angle et al., 1982).

As mentioned earlier in this section, haem is also incorporated into cytochrome P-450. Thus, disruption of the haem synthesis pathway by lead can also adversely affect the formation of this protein. Indeed, studies in children have shown that lead inhibits its formation, as reflected in decreased activity of hepatic mixed function oxygenases (Alvares et al., 1975; Saenger, 1984).

3.3.3 Neuropsychological effects of lead

In addition to the reviews of the neuropsychological effects of lead by the EPA (1986) and the ATSDR (1988), there have been a number of other reviews in recent years which have specifically focused on the neuropsychological effects of lead in children, such as those by Rutter (1980), Bornschein et al (1980), Medical Research Council (MRC, 1985), Yule and Rutter (1985), Harvey (1986) and Lansdown (1986). Again, the major findings have been extracted and these are summarised below.

Lead impairs both the peripheral and central nervous systems. The effect of lead on peripheral nerve function has been studied intensively in adults. The EPA (1986) reviewed and evaluated 28 studies of peripheral nerve function that measured the conduction velocity of electrically stimulated nerves in the arm or leg of lead workers. It was concluded that decreased nerve conduction velocities could occur in adults at blood lead levels of less than 70 µg/dl (3.36 µmol/l) and possibly at levels as low as 30 µg/dl (1.44 µmol/l). Effects of lead on

peripheral nerve function have also been documented in children. Frank peripheral neuropathy has been observed in children with blood lead levels ranging from 60 to 80 µg/dl (2.88 to 3.84 µmol/l) and changes in peripheral nerve conduction velocities have been reported at lower blood lead levels of around 20 to 30 µg/dl (0.96 to 1.44 µmol/l) (ATSDR, 1988). However, it should be noted that these observations have been drawn from only a limited number of studies. Firm conclusions cannot, therefore, be made at present about the effects of lead on the peripheral nervous system in children and further work will be necessary to clarify the situation.

The most severe neuropsychological effect of lead in humans is encephalopathy. Early symptoms that may develop within weeks of initial exposure include dullness, irritability, poor attention span, headache, muscular tremour, loss of memory and hallucinations. The condition may then worsen, sometimes abruptly, to delirium, convulsions, paralysis, coma and death (ATSDR, 1988). Histopathological findings in fatal cases of lead encephalopathy are similar in both adults and children and are reported to include cerebral oedema, altered capillaries and perivascular glial proliferation. Neuronal damage is variable and may be caused by anoxia. In adults, encephalopathy may be associated with blood lead levels as low as 100 µg/dl (4.8 µmol/l), although it is generally observed only at levels well in excess of this. Encephalopathy has been reported in some children with blood levels as low as 80 µg/dl (3.84 µmol/l), whereas in other children with levels up to 300 µg/dl (14.4 µmol/l), signs or symptoms of encephalopathy have been absent (EPA, 1986). It has been suggested that such variation may be due to individual susceptibilty, changes in levels over time, increased tolerance for gradually accumulating burdens or interacting factors such as nutritional status (Lansdown, 1986).

According to the EPA (1986), numerous studies have clearly shown that childhood lead poisoning with encephalopathy results in a greatly increased incidence of permanent neurological and cognitive impairments. Indeed, children with symptomatic lead poisoning without encephalopathy (with blood lead levels ranging from around 80 to 100 μg/dl, 3.84 to 4.8 μmol/l) are also reported to have increased incidence of lasting neurological and behavioural impairments (EPA, 1986). Adults have been found to exhibit other overt neurological signs and symptoms (e.g. loss of appetite, paresthesia of lower limbs, weakness in upper limbs, dropping objects) and impaired performance on neurobehavioural tests (e.g. visual-motor coordination tasks, verbal reasoning ability) at blood lead levels ranging from 40 to 60 μg/dl (1.92 to 2.88 μmol/l). These blood lead levels are similar to those at which other symptoms of lead intoxication, such as gastrointestinal complaints, occur (ATSDR, 1988).

A review of a number of studies of apparently asymptomatic children with relatively high blood lead levels led the EPA (1986) to conclude that cognitive deficits (lower IQ values and other neuropsychological deficits) can occur in children at blood lead concentrations ranging from around 40 (1.92 μmol/l) to around 80 μg/dl (3.84 μmol/l). It was further stated that average decrements of around 5 IQ points (on the Stanford-Binet Scale and McCarthy General Cognitive Index) are likely to be associated with markedly elevated blood lead concentrations of around 50 to 70 μg/dl (2.4 to 3.36 μmol/l) in asymptomatic children. The question of whether or not adverse neuropsychological effects are associated with body lead burdens lower than this has attracted a good deal of attention and controversy in recent years. Since the pioneering study in the US by Needleman et al. (1979), there have been over 20 studies worldwide which have addressed this issue. However, several of the early studies were plagued by inadequacies in methodology and

many of the others have resulted in questionable interpretation. These problems are considered in detail in the reviews cited at the beginning of this section, although a useful additional assessment of the methodological and statistical issues involved is provided by Yule (1986).

It was reported by the EPA (1986) that none of the general population studies reviewed individually provided definite evidence for or against neuropsychological deficits being associated with relatively low body lead burdens in asymptomatic children representative of the general paediatric population. In the Needleman et al. (1979) study, shed deciduous teeth of six and seven year old children, collected by school teachers, were used to assess body lead burdens. From an initial study population of over 2000 children, two sub-groups (<10th and >90th percentiles of tooth lead concentrations) were selected for in-depth neuropsychological testing. It was reported that significant differences ($p<0.05$) existed between these two extreme groups for certain IQ scores (Wechsler Intelligence Scale for Children - Revised [WISC-R]), certain classroom behaviour scale items and several experimental measures of perceptual motor behaviour. However, this study has been severely criticised in the literature for a variety of reasons which include the fact that a middle group was omitted, making it impossible to examine any dose-effect relationship properly, behaviour rating results were not corrected for social factors or parental IQ and no attempt was made to control for variations in lead levels in teeth by tooth type or position in the mouth, a factor that has been shown to be of great importance elsewhere (Smith et al., 1983: Lansdown, 1986).

In response to such criticisms, the data were further analysed by Needleman and co-workers (Needleman, 1982, Needleman et al., 1982, Bellinger and Needleman,

1983) and others (e.g. Marshall, 1983). The EPA (1986) also re-analysed the data and reported that average IQ decrements of about four points do appear to be associated with lead exposure levels experienced by the Needleman high-lead group. Among that group, statistically significant ($p<0.05$) IQ decrements appear to remain (after controlling for confounding variables) for children with dentine lead levels of 30-39.9 µg/g but not for children with 20-29.9 µg/g or lower dentine lead levels, as was reported by Bellinger and Needleman (1983). It was estimated that the equivalent blood lead levels at which these effects may occur were likely to range from 30 to 50 µg/dl.

From a review of the remaining general population studies, the EPA (1986) reported a lack (with a few exceptions) of statistically significant effects on IQ or other neuropsychological measures. Most of the studies showed slightly lower IQ scores for higher lead exposure groups than for low lead control groups before correcting for confounding variables but the differences were typically reduced to one or two IQ points and were not statistically significant after correction for confounding variables. An exception to this overall pattern is to be found in the results from a pilot study of London children by Yule et al. (1981). These workers reported deficits of 6 to 7 IQ points in a group of children having blood lead concentrations in the top quartile, 17 to 32 µg/dl, (0.82 to 1.54 µmol/l)compared with a group with blood lead levels in the bottom quartile, 7 to 10 µg/dl (0.34 to 0.48 µmol/l). However, it is reported that the study provided only relatively crude control for socio-economic factors and failed to take parental IQ into account (EPA, 1986). Two further studies have subsequently been carried out by these workers (controlling appropriately for socio-economic factors and parental IQ) in which no statistically significant association between blood lead and IQ was found (Yule and Lansdown, 1983).

In reviewing the literature, Harvey (1986) has highlighted the difficulties in attempting to make direct comparisons between studies carried out in different countries. Children in studies from the USA and Mexico all have considerably higher mean lead concentrations (and ranges) in blood and teeth than those in studies from the UK, West Germany, Australia and New Zealand. For example, in tooth lead studies carried out by Smith et al. (1983) in the UK and Winneke et al. (1983) in West Germany, all of the so-called "high" lead groups were around the same as Needleman's (1979) "low" group. Furthermore, a lack of consistency between definitions in different countries can lead to confusion. In its review of the health effects of lead, the DHSS (1980) asserted that although it appears to be widely held that an association has been demonstrated between high body lead levels and hyperactivity in children, there is no satisfactory evidence which confirms this view. It was stated that American workers use the terms "hyperactivity" and "hyperkinesis" much more loosely than clinicians in the UK. It was further stated that recent evidence suggests that children's levels of activity must be monitored in at least two different settings before a diagnosis of hyperactivity can be considered and that few of the existing rating scales stand close psychometric scrutiny (DHSS, 1980).

In addition to the studies of Yule and co-workers outlined above, other major British studies include that of Smith et al. (1983), those of Harvey et al. (1984, 1988) and a recent study by Fulton et al. (1987). In the first and largest of these (Smith et al., 1983), a total of 2663 eligible 6 to 7 year old children from three London boroughs donated teeth (incisors). Four hundred and three children in three pre-defined ranges of tooth lead (low lead, <2.5 μg/g, medium lead, 5-5.5 μg/g, high lead, >8.0 μg/g) were then selected for extensive neuropsychological testing. Before correcting for confounding factors, there were statistically significant

associations between tooth lead and full scale IQ scores (using WISC-R). However, after allowance for confounding factors, the associations lost statistical significance. The average IQ scores for the medium and high lead groups were 2.2 and 2.3 points, respectively, lower than the low lead group.

Harvey and co-workers studied two groups of children, aged 2.5 (Harvey et al., 1984) and 5.5 years (Harvey et al., 1988), in Birmingham. Blood lead concentration was used as the indicator of body lead burden in both studies. In the study of 2.5 year olds, IQ was computed from tests of the British Ability Scales (BAS) whereas for the 5.5 year olds, the Wechsler Pre-School and Primary Scale of Intelligence (WPPSI) was used. For the 2.5 year olds, there was a statistically significant association between IQ and blood lead ($p<0.05$). However, IQ was also significantly associated with several non-lead variables and after allowance for these confounders, the association between blood lead and IQ was not statistically significant. Similarly, there was no statistically significant association between blood lead and behavioural measurements. The fact that these workers did not find evidence of an association between body burden and IQ at this age (after allowing for confounders) is a particularly important negative finding when one considers that lead levels tend to peak at around this age (Section 3.2.2) and that, as is generally agreed, any impairment in brain function is likely to have occurred by then (MRC, 1985). In the study of 5.5 year olds, no significant relationship was found between overall IQ and blood lead concentration (Harvey et al., 1988).

After reviewing the evidence most appropriate to British children (i.e. that provided by Yule et al., 1981, 1983; Smith et al., 1983; Harvey et al., 1984) the MRC concluded in 1985 (MRC, 1985) that a moderate elevation in

body lead burden, as found in some British children, has little or no effect on IQ. Since that time, results from two other major British studies have been published. One of these studies, that of 5.5 year old children in Birmingham by Harvey et al. (1988) outlined above, provided evidence to support this conclusion. Results from the other study, carried out by Fulton et al. (1987), led these workers to conclude that lead at low levels of exposure probably has a small harmful effect on the performance of children in ability and attainment tests. As this is the first British study to report statistically significant adverse effects of lead at low levels of exposure (apart from that of Yule et al., 1981, discussed above), it is examined in some detail below.

Close inspection of other publications connected with this study indicates that this conclusion should be treated with caution. These workers studied children aged six to nine years from primary schools in central Edinburgh. From an initial sample of 855, a stratified subsample of 501 children completed individual tests of cognitive ability and educational attainment from the BAS, between August, 1983 and June, 1985. Blood lead was selected as the main measure of lead exposure although naturally shed milk teeth were also collected to provide additional information on long term exposure (Raab et al., 1985). Analysis of results showed there to be a significant relation between tests of ability and attainment (BAS combined score [BASC], number score, reading score) and blood lead when confounding variables were taken into account (Fulton et al., 1987). However, for the BASC score, less than one per cent of a total of 45.5 per cent variance explained by the covariates in the optimal regression model could be attributed to the effect of lead. In order to examine the possible dose-response relation of blood lead against ability scores, the children were ordered by their blood lead values and put into groups of about 50. The mean blood lead

concentration of the lowest group was 5.6 μg/dl, while that of the highest group was 22.1 μg/dl (1.06 μmol/l). The difference in the adjusted BASC score between these two groups (lowest and highest) was -5.8 points. It was reported that the data showed a dose-response relation with no evidence of a threshold.

Although careful attention was paid to statistical methodology in this study, it is questionable whether blood lead concentration was the most appropriate indicator of body burden for these study children. In an early paper detailing the study design, Raab et al. (1985) indicate that Edinburgh was chosen because it had a plumbosolvent water supply and a high proportion of the houses had lead plumbing. One can infer from this that the water lead concentration was also likely to have been elevated. However, it was also stated that plumbing changes were likely to take place in the city "over the next few years". The inference here is that such changes would probably have resulted in a decrease in water lead concentration in Edinburgh over the two year period of the study. Although this issue has not been specifically addressed by these workers, the general picture which emerges from an appraisal of the published results is that of a probable decline in water lead concentrations during the study period.

In each home a 30 minute stagnation sample of kitchen cold water was taken. Results from the first 129 households, presented in an interim report by Raab et al. (1985), revealed a median water lead concentration of 35 μg/l, with an inter-quartile range of 8-88 μg/l. However, summary results published at the end of the study for all 495 households (including the first 129) showed that the median water lead concentration was only 17 μg/l and the inter-quartile range was 6-43 μg/l (Raab et al., 1987). It is important to note here that not only has the median value decreased substantially but the top end of the

inter-quartile range has also fallen markedly, indicating that the whole distribution has shifted downwards. Of course, had the remaining 361 households been treated as a separate group and compared with the first group of 129, the differences would have been even more striking. It is possible that these differences were due to some sort of sampling bias e.g. all of the early samples having been obtained from one particular geographical area with higher water lead levels. This is, however, unlikely as sampling was carried out on a random basis. It is probable then that the drinking water lead intake of at least some of the study children dropped markedly at some point prior to testing. Blood lead concentration provides an indication of current or recent lead exposure but is a poor indicator of the level of lead to which a child may have been exposed in the past. Tooth lead concentration, on the other hand, gives an indication of the cumulative exposure to lead from the prenatal period when the teeth are formed until their time of shedding. It is felt, therefore, that tooth lead concentration is likely to be the more appropriate indicator of body lead burden for these Edinburgh children. Indeed, comparison of the blood lead and tooth lead results from this study adds weight to this point. The geometric mean blood lead concentration for the 501 study children, 11.5 μg/dl (0.55 μmol/l) (Fulton et al., 1987), is similar to values noted in other UK cities for children of similar ages (Smith et al., 1983; DoE, 1986; Harvey et al., 1988). However, the mean tooth lead concentration of the samples so far analysed (n=150) is reported to be 9.3 μg/g (Keating et al., 1987) which is around twice as high as the mean values noted for children of this age in seven other UK urban areas (Delves et al., 1982). Thus, the tooth lead results indicate elevated body lead burdens whereas the blood lead results do not. This finding will need to be reappraised when the full data set for the teeth becomes available.

To summarise, the implication is that some of the

study children may have had high lead intakes at some time during their lives which are not adequately reflected in the measure of body burden which has been related to their ability and attainment by Fulton et al. (1987). It is felt that any assessment of the validity of the conclusion from this study, that lead at low levels of exposure probably has a small harmful effect on the performance of children in ability and attainment tests, should take this important factor into account.

In addition to the cross-sectional studies described above, there have recently been a number of prospective studies undertaken to assess the possible neuropsychological effects of prenatal exposure to lead. These studies have been reviewed by EPA (1986), Davis and Svensgaard (1987) and ATSDR (1988) and it was concluded that the evidence suggests that neurobehavioural deficits in infants are associated with maternal or cord blood lead concentrations of around 10 to 15 μg/dl (0.48 to 0.72 μmol/l). The most frequently used index of mental development in such studies has been the Bayley Mental Development Index (BMDI) and Davis and Svensgaard (1987) report that the magnitude of deficits range from two to eight points on the BMDI for every 10 μg/dl (0.48 μmol/l) increment in blood (maternal/cord) lead. However, these authors admit that the predictive ability of the BMDI is debatable and that it is difficult to assess the implications of a deficit in infant BMDI scores in terms of later childhood IQ test scores. Also, results from one of the studies reviewed, carried out in Australia by Baghurst et al. (1987), showed that deficits in BMDI scores were more closely related to postnatal blood lead levels than to prenatal levels. The only prospective study conducted to-date in the UK is that of Moore et al. (1982, 1988). This study was based in Glasgow and coincided with a water treatment programme to reduce water lead concentrations in this city (see Section 6.5). One hundred and fifty one pregnant women were selected for

study and divided into three discrete groups, on the basis of blood lead concentration (low, <10 μg/dl [<0.48 μmol/l]; medium, 15-25 μg/dl [0.72-1.2 μmol/l]; high, >30 μg/dl [>1.44 μmol/l]). Cord blood samples were obtained at birth. Blood was sampled annually thereafter and psychometric testing using the Bayley Scales was carried out on the first and second birthdays. From an analysis of the results, it was concluded that there was no evidence to suggest that lead made direct or indirect contributions to BMDI scores at either age.

It would be premature to draw firm conclusions from the results reported so far from the various prospective studies, as many of these are still in progress. In many of the studies, it will be several more years before the children can be investigated using tests of IQ and other psychometric indices with known validity and predictability.

3.3.4 Other human health effects of lead

It has been known for over half a century that kidney disease can result from lead poisoning (EPA, 1986). The characteristics of acute nephropathy include nuclear inclusion bodies, mitochondrial changes and cytomegaly of the proximal tubular epithelial cells, dysfunction of the proximal tubules (Fanconi's syndrome), manifested as an excessive excretion of amino acids, glucose and phosphates (i.e. aminoaciduria, glucosuria and phosphaturia), and increased sodium and decreased uric acid excretion. It is reported that these effects appear to be reversible (ATSDR, 1988). Characteristics of chronic lead nephropathy include progressive interstitial fibrosis, dilation of tubules and atrophy or hyperplasia of the tubular epithelial cells, few or no nuclear inclusion bodies, reduction in glomerular filtration rate and azotemia. These effects are reported to be irreversible

(ATSDR, 1988). Studies of occupationally exposed workers have provided evidence for lead induced chronic nephropathy being associated with blood lead concentrations ranging from 40 to over 100 µg/dl (1.92 to 4.8 µmol/l), although some studies indicate that the lower end of this range may be around 30 µg/dl (1.44 µmol/l). The EPA (1986) concluded from the relatively few studies of children that lead induced nephropathy in this section of the population only occurs at relatively high blood lead levels (greater than 80 µg/dl [3.84 µmol/l], although usually exceeding 120 µg/dl [5.76 µmol/l]).

Evidence from occupational, clinical and general population studies suggests that lead affects the cardiovascular system in humans, producing cardiac lesions and electrocardiographic abnormalities at high levels of exposure and increases in blood pressure, particularly in middle-aged men, at low blood lead levels, with no apparent threshold for this effect (ATRDR, 1988). The contribution of lead, compared with many other factors which affect blood pressure, appears to be relatively small, usually accounting for only one to two per cent of the variation explained by the various models employed (EPA, 1986).

Typical gastrointestinal symptoms caused by lead exposure include constipation or diarrhoea, epigastric pain, nausea and intermittent vomiting (Hernberg, 1980; ATSDR, 1988). Although mild symptoms may be associated with blood lead concentrations ranging from as low as 30 to 80 µg/dl (1.44 to 3.84 µmol/l), it is reported that there is currently insufficient information to establish a clear dose-effect relationship for the general population at ambient exposure levels (EPA, 1986).

In addition to the possible adverse effects of prenatal exposure to lead on neuropsychological development, discussed earlier, evidence from recent

studies indicates that gestational age may be reduced as a result of such an exposure. Moore et al. (1982), McMichael et al. (1986) and Dietrich et al. (1986) reported significant negative correlations between maternal or cord blood lead and gestational age. However, Needleman et al. (1984) and Bellinger et al. (1984) did not find a significant relationship between these variables. The EPA (1986) concluded from its review of this literature that gestational age is reduced as a result of prenatal exposure to lead, even at maternal or cord blood lead levels below 15 µg/dl (0.72 µmol/l)and the ATSDR (1988) concluded (from data of McMichael et al., 1986) that the risk of preterm delivery increases by at least fourfold as either maternal or cord blood lead level at delivery increases from less than 8 µg/dl (0.38 µmol/l) to greater than 14 µg/dl (0.67 µmol/l). In its assessment of the reproductive toxicity of lead, the EPA (1986) reported that there was sufficient qualitative evidence to support the conclusion that at high exposure levels, lead has significant adverse effects on human reproduction. Indeed, excessive occupational exposure to lead has long been known to be associated with a high probability of spontaneous abortion in pregnant women. However, the available data do not permit an estimate of the effect levels in women and only the tentative conclusion that adverse effects on sperm or the testes (e.g. depression of sperm count or peritubular fibrosis) may occur at blood lead levels of 40 to 50 µg/dl (1.92 to 2.4 µmol/l) in chronically exposed men, can be drawn (ATSDR, 1988).

Although evidence of an adverse effect of prenatal lead exposure on birth weight is limited (Dietrich et al., 1986 and Bornschein et al., 1987), strong evidence of an association between lead exposure and growth retardation in children is provided by Schwartz et al. (1986). Stepwise multiple regression analysis indicated that blood lead level (range 4 to 35 µg/dl, 0.19 to 1.68 µmol/l) was

a statistically significant predictor of children's (aged <7 years) height, weight and chest circumference. The strongest relationship was observed between blood lead level and height, with no threshold evident down to the lowest observed concentration of 4 µg/dl (0.19 µmol/l). These authors stated that the mean blood lead level of children with an average age of 59 months appeared to be associated with a reduction of 1.5 per cent in the height that would be expected if the blood lead level had been zero. The reported reduction in weight and chest circumference was of the same magnitude. The mechanism for such retardation is, however, unclear (ATSDR, 1988).

Hearing thresholds in children appear to be affected adversely by lead exposure (Robinson et al., 1987; Schwartz and Otto, 1987, 1987a). In the analysis of Schwartz and Otto (1987), the probability of elevated hearing thresholds increased significantly with increasing blood blood lead concentration across the entire range of blood lead levels studied (<4 to >50 µg/dl, <0.19 to >2.4 µmol/l) with no apparent threshold.

4. TRANSFER OF LEAD FROM THE ENVIRONMENT TO MAN: THE BIRMINGHAM STUDY

4.1 INTRODUCTION

In Chapter 2, consideration was given to the natural and anthropogenic sources of lead in the environment and to the concentrations of lead in various environmental compartments and it was concluded that lead is a ubiquitous environmental pollutant. From discussions in Chapter 3, which focused on both the metabolism of lead and its toxicity in humans, it was shown that the intake and subsequent uptake of lead by man can result in a wide spectrum of adverse effects, particularly in young children. In the present chapter, the transfer pathways of lead from the environment to man will be outlined and the study protocol and analytical techniques detailed for a comprehensive study carried out to ascertain the significance of the various environmental pathways to the total lead intake of a group of young children in Birmingham (UK).

4.2 PATHWAYS OF LEAD FROM THE ENVIRONMENT TO MAN

Man is exposed to lead in his environment from a number of sources along a multitude of pathways. Sources can be thought of as materials from which lead is first released into the environment, such as petrol, solder, paint, lead pipes or lead-bearing rock, whereas the media through which this liberated lead passes, such as water, air, food and housedust, are all stages in a variety of pathways from source to receptor which, in this case, is man (RCEP, 1983).

The major sources and transfer pathways of lead from the environment to man have been usefully summarised

recently by the EPA (1986) and this summary, with slight amendments, is presented in Figure 4.1. A more comprehensive flow chart could have been included but it was felt that this would only serve to confuse matters. Indeed, a comprehensive figure compiled by the US National Research Council (NRC, 1980) contains over one hundred arrows and interactions and is somewhat difficult to interpret. The EPA (1986) summary figure is, however, a slight oversimplification of the situation and has, therefore, been adapted here (Figure 4.1) to take account of two important transfer pathways and one important source which were omitted. As certain children are active soil eaters and others can inadvertently ingest soil through hand-to-mouth activity, an arrow has been included to indicate direct intake of lead from soil. Also, an arrow has been drawn leading directly from paint pigments to man to take account of those children who lick or suck painted objects (e.g. cots, toys) and those who actively peel off and consume paint from painted surfaces (e.g. windowsills). Lead water pipes can be an important source of lead in drinking water and such a source category has, therefore, been included in Figure 4.1. The transfer pathways in this case would be through drinking water to man and, where water is used in food preparation, through drinking water, via food, to man.

With reference to Figure 4.1, it can be seen that the major sources of lead are thought to be paint, petrol, industrial emissions, solder, water pipes and lead-bearing rock. Although a variety of the likely transfer pathways of lead from these sources through the environment to man have been given, the relative contribution made by the different pathways to the total lead intake of man and, in particular, of young children, is at present unclear. This uncertainty exists despite the fact that there have been a large number of studies which have addressed this issue.

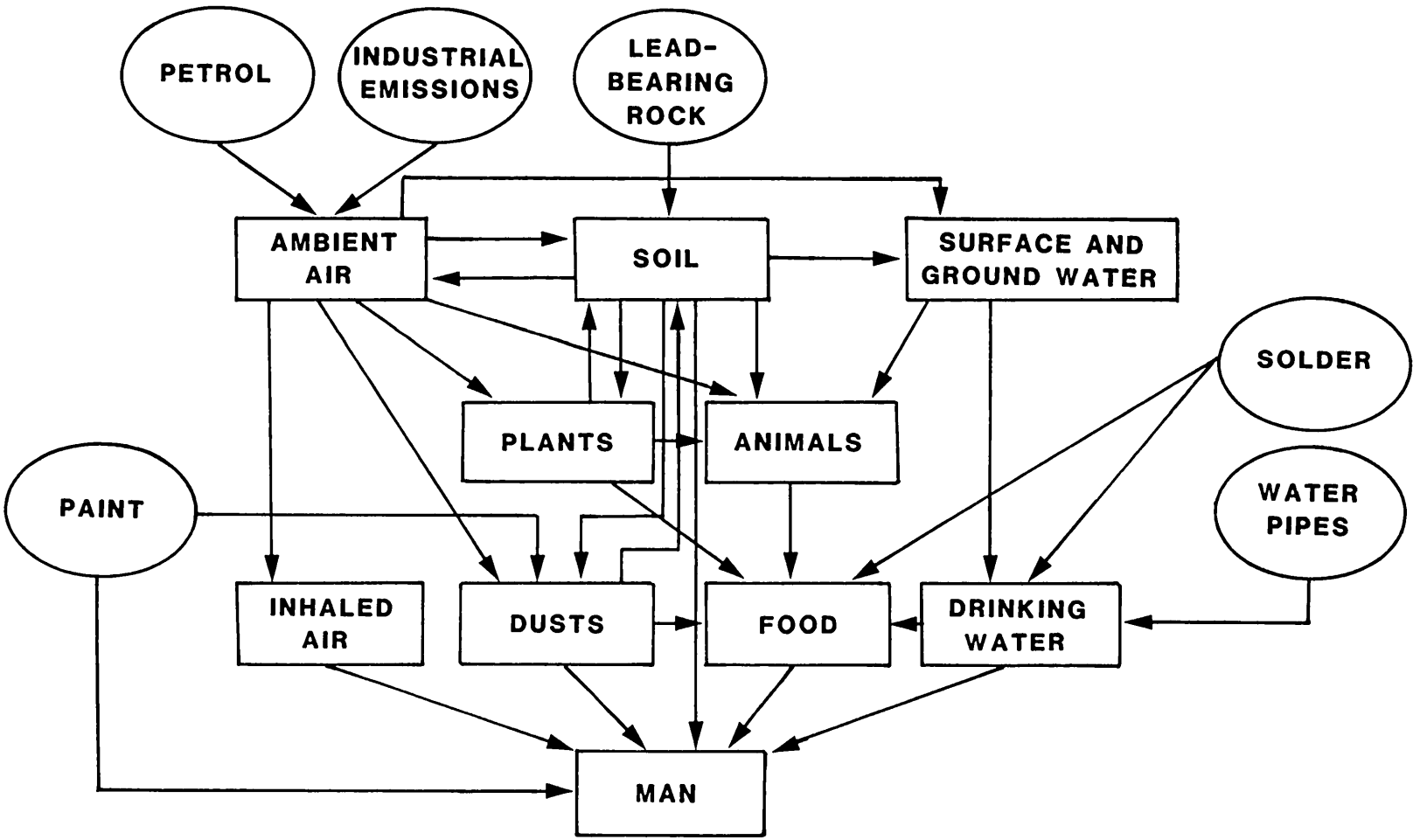


Figure 4.1 Major sources of lead in the environment and transfer pathways to man

Some of the earliest studies to indicate that intake of lead from the environment may contribute to the body burden of lead in children were those carried out by Barltrop et al. (1974, 1975) in lead mineralised areas of Derbyshire. Their data indicated that children living in areas with elevated soil lead concentrations exhibited elevated blood lead concentrations and also showed that the blood lead concentration of the children was consistently greater than that of their mothers. However, it was found that children who were reported by their parents to habitually put dirty fingers and toys in their mouths apparently received little or no extra lead burden when compared with other children living in the same area. It was noted that the mean housedust lead concentration increased with, but was lower than, the mean soil lead concentration. It was concluded by these workers that lead in soil and dust is a relatively minor source and, in itself, insufficient to cause a hazard.

Day et al. (1975), on the other hand, concluded from their study of lead concentrations in road and playground dusts, that children in urban areas may significantly increase their daily intake by the inadvertent ingestion of dirt or dust from their surroundings. Duggan and Williams (1977) reached a similar conclusion. Other work by Sayre et al. (1974), Vostal et al. (1974) and Lepow et al. (1975), where the actual amount of lead present on the hands of urban children was assessed, supported this assertion and indicated that an important route of transfer of lead into young children from the environment may involve hand-to-mouth activity. Indeed, Lepow et al. (1975) concluded, on the basis of "mouthing" behaviour observed among the ten subject children from an urban area in the US (all of whom had blood lead levels in excess of 1.92 µmol/l [40 µg/dl]) and on the basis of the concentrations of lead noted in soil, housedust and on the children's hands, that it was likely that the hand-to-mouth exposure route was the principal cause of the

excessive lead accumulation in these children. Charney et al. (1980) investigated a group of urban children in the US with high blood lead concentrations (40-70 μg/dl, 1.92-3.36 μmol/l) and a matched group with lower concentrations (<29 μg/dl, 1.39 μmol/l) and found that the high lead group had significantly higher hand lead levels, their homes had higher dust lead concentrations, they played more often in soil and this soil had a higher lead content, they mouthed objects more often and they sucked their fingers more often.

A study of children in an old lead mining area of Wales showed that although pica, as assessed by questionnaire, showed no relation with blood lead, the amount of lead removed from the children's hands was an important contributor to blood lead concentrations (Gallacher et al., 1984). Studies have also been carried out in the vicinity of industrial sources which suggest that this exposure route may be important for children. Roels et al. (1980) concluded from a study of children attending schools near a lead smelter in Belgium, that the contribution to blood lead made by the ingestion of lead via dirty hands was far greater than that made by the inhalation of airborne lead. In another smelter study, Brunekreef et al. (1981) found a correlation between the blood lead concentration of children living in the vicinity of the smelter and the frequency with which they mouthed dirty objects.

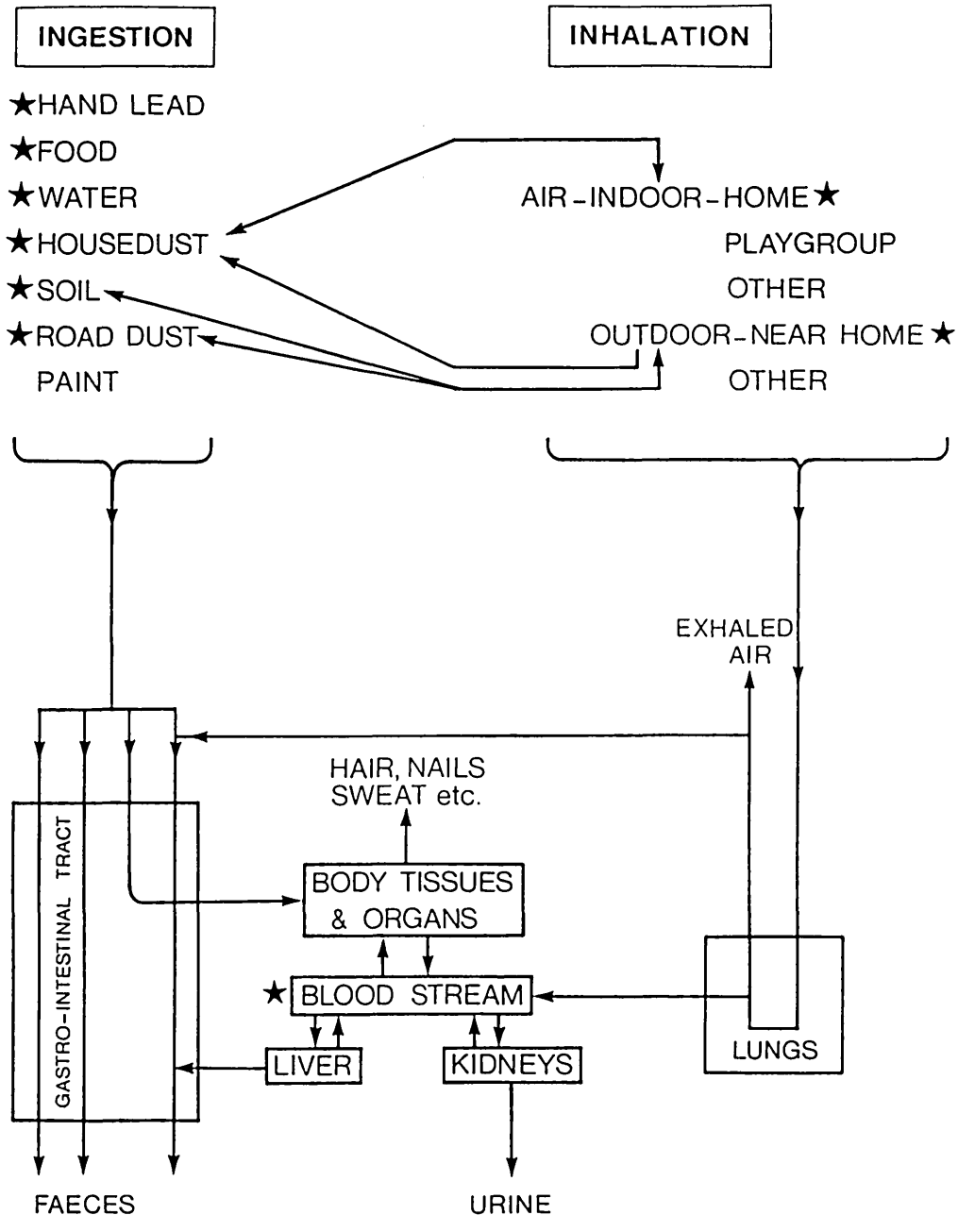
Thus, from the evidence cited above, it would appear that for some children, the intake of lead from the ingestion of dust and soil via hands can be significant. However, these studies have all been directed at populations with unusually high lead exposures and are difficult to interpret in the context of general population exposures.

From the extensive literature reviews of Duggan

(1983), Brunekreef (1984), Elwood (1986) and EPA (1986), it is apparent that many of the exposure assessment studies carried out in the past have not been comprehensive, often focusing on single pathways in isolation. However, it is not possible to fully assess the significance of any one particular transfer pathway (e.g. dusts to hands to mouth) to the total lead intake of a group of children unless intakes from all other pathways are assessed simultaneously.

In 1983, the RCEP stated in its Ninth Report on Lead in the Environment (RCEP, 1983) that "....although the limited data involve theoretical calculations, we believe that the intake of lead from dust can be substantial for young children". Furthermore, the Commission noted that there were major gaps in knowledge concerning such intakes. It was apparent at the time, therefore, that valid, comprehensive assessments were lacking. Also, results from a nationwide reconnaissance study carried out by the Applied Geochemistry Reseach Group, Imperial College, had shown that the geometric mean lead concentration in housedust from 47 urban locations throughout Britain was around 560 μg/g (range 5-36900 μg/g) (Culbard et al., 1988). If, as was suggested in the literature, children routinely ingested such dusts through hand-to-mouth acitivity, it was thought possible that the intake of lead from housedust could be making an important contribution to the body burden of "typical" British children living in urban areas.

In an attempt to ascertain the significance of this and other pathways to the total lead intake of children, a comprehensive study was commissioned by the UK Department of the Environment in the inner-city area of Birmingham. This study was designed to provide quantitative information on lead intakes via all the major pathways, simultaneously. Figure 4.2 shows the principal routes of exposure of young children to lead and indicates the



★ indicates parameters measured in Birmingham Study

Figure 4.2 Principal routes of exposure of young children to lead showing parameters measured in the Birmingham Study

parameters measured in this study. The study was based on the analysis of environmental, dietary and biological samples which included housedust, pavement and road dust, soil, handwipes, air filters, water, food and venous blood. Behavioural assessments were also made in order to quantify both mouthing and touching activities of the study children.

The study was carried out between November, 1984 and February, 1985 and the sections that follow provide a detailed account of the design and analytical techniques employed. The study was preceded by a pilot study, the results of which are presented and discussed in Appendix A. The results of the main study are presented in Chapter 5 and are discussed in detail in Chapters 6 and 7. The principal conclusions are summarised in Chapter 8.

4.3 THE BIRMINGHAM STUDY: PROTOCOL AND ANALYTICAL TECHNIQUES

4.3.1 Introduction

The study was approved by the Ethics Committee of the Queen Elizabeth Medical Centre, Birmingham and was carried out in accordance with the appropriate ethical guidelines (Medical Research Council, 1963; American Psychological Association, 1981; British Psychological Association, 1983).

4.3.1.1 Participating organisations

Due to the multi-disciplinary nature of the study it was necessary for a number of organisations to collaborate and these have been listed in Table 4.1.

Table 4.1 Organisations participating in the Birmingham Study

ORGANISATION	INPUT
Department of the Environment (DoE)	Funding and coordination of environmental, behavioural and biological sections
Ministry of Agriculture, Fisheries and Food (MAFF)	Funding and coordination of dietary section
Applied Geochemistry Research Group, Imperial College (AGRG)	Collection, analysis and interpretation of environmental samples
Environment, Health and Behaviour Research Group (EHBRG), University of Birmingham	Collection of blood samples and behavioural data. Analysis of behavioural data
Trace Element Unit,(TEU), University of Southampton	Analysis of blood samples
British Market Research Bureau (BMRB)	Collection of food/beverages and water samples
Laboratory of the Government Chemist	Analysis of food/beverages and water samples

4.3.1.2 Study area

The study was carried out within the inner ring-road and to the east of the city centre of Birmingham. Birmingham is Britain's second largest city and was considered to represent a typical UK urban environment.

Also, the DoE, the EHBRG and the TEU had conducted previous studies on environmental and blood lead levels in this city with which the results from this study could be compared.

4.3.1.3 Subject selection

All families with children born in, and still resident in, the study area, who at the time of the study would be aged between 22 and 26 months, were identified from birth records, approached by letter and subsequently visited. Asian children (i.e. those with one or more parent originating from the Indian sub-continent) were not included, however, as previous studies had indicated that lead intakes could be markedly different for a proportion of this section of the population (DoE, 1982). This age group was chosen because of the well established peaking of blood lead concentration at around two years (De Silva and Donnen, 1980; DoE, 1981a, 1983; Annest et al., 1982; and supported by McMichael et al., 1985). Of those who were contacted and whose children were not in outside care for more than six hours per week, 77 per cent agreed to participate. One hundred and eighty three of the volunteered children eventually provided venous blood samples, although only 146 of these samples contained a sufficient volume of blood for analysis. The final study population was selected in a stratified manner, using the following scheme:-

(a) all the upper quartile of the blood concentration distribution (or as close as possible to give n = 35);

(b) all the lower quartile (or as close as possible to give n = 35);

(c) a random sample of 35 from the remaining 76.

Thirty one children with higher blood lead levels (over 0.73 μmol/l [15.2 μg/dl]) and thirty four with lower values (under 0.44 μmol/l [9.2 μg/dl]), together with thirty three with intermediate values actually completed the study. One child with a blood lead level of 2.14 μmol/l (44.6 μg/dl) was found and investigated further by

the local health authority. Fifty six children provided a second blood sample at the end of the study.

4.3.2 Sampling protocol

The large nature of the study necessitated the recruitment and training of a number of field officers to carry out the sampling. Six officers were recruited to work on the environmental section, three on the dietary section and three on the behavioural section. Originally 105 children were selected for inclusion in the study and the sampling programme was therefore devised for this total, although, for various reasons, only 98 actually completed the study. The maximum number of households which could be monitored simultaneously was 15. The study, therefore, comprised of seven separate monitoring periods, with 15 households being monitored during any given period (7 x 15 = 105). Each block of 15 households was further sub-divided into three groups of five households, each group being the responsibility of one monitoring team. A monitoring team consisted of two environmental, one dietary and one behavioural officer. The following sampling protocol was implemented at each household.

4.3.2.1 Dusts

(i) Housedust

(a) One sample from the dust bag in the householder's own vacuum cleaner.

(b) One sample from the child's bedroom and one from the child's main playroom, using an adapted Electrolux 350 vacuum cleaner. The cleaner had a pre-weighed cellulose filter thimble (25 mm x 80 mm, Whatman 2800 258) mounted at the distal end of the suction tube, as shown in Figure 4.3. All of the exposed floorspace was sampled on Day 6 of the sampling period and the area was noted.

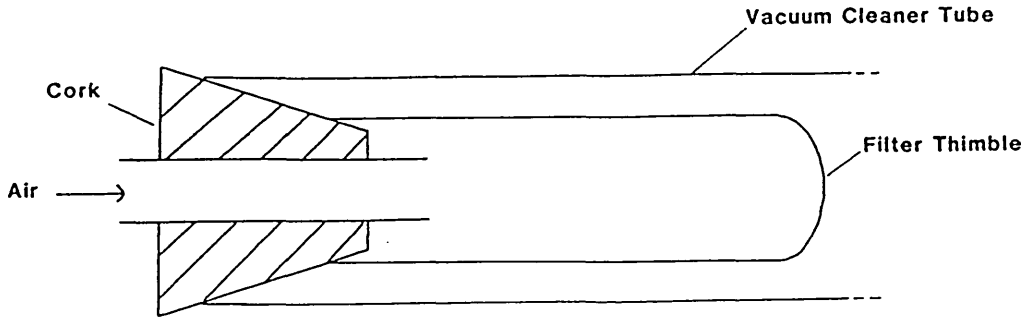


Figure 4.3 Schematic diagram of dust sampling apparatus

(c) One sample from under the doormat using an adapted Electrolux 350 cleaner.

(ii) Pavement and road dust

At both of these locations, immediately outside the house, a composite sample of 25 subsamples, collected using a plastic dust pan and brush, when conditions were dry.

4.3.2.2 Soil

One composite surface soil sample (0 to ~ 5cm), comprising 25 subsamples, was collected from areas of exposed soil in the front and back garden, using a stainless steel trowel.

4.3.2.3 Air

Air samples were obtained from three locations, the child's bedroom, the child's main playroom and immediately outside the house, for seven consecutive days. Aerosol

monitors (Millipore, MAWP037A0, containing 37 mm, 0.8 um cellulose acetate filters) were positioned at a height of 1.5 to 2 m and attached to aerosol adaptors (Millipore, XX6200004) incorporating 2 l/min flow limiting orifices (Millipore, XX5000020). These, in turn, were connected, through PVC adaptors, to clear plastic PVC tubing (3 mm internal diameter) which led back to one central Millipore vacuum pump (XXMJ22050) [see Figure 4.4]. Within the home the tubing was secured to the bottom of the skirting board using staples and the pump was located in an appropriate place, such as a basement or cupboard under the stairs, to minimise the intrusion of noise. Elapsed time meters (R.S. Components Ltd. 259-858) were fitted to the pumps to ensure that the actual operating time could be recorded. The aerosol monitors were changed daily, at which time the flow rate was checked using a rotameter.

4.3.2.4 Handwipes

Handwipe samples were taken daily during the study week. The sampler's hands were washed with soap and water and wiped with an "All-Fresh" wet-wipe which was placed in a self-sealing plastic bag. Both of the child's hands were then wiped thoroughly (including fingers, palm and back of the hand, up to the wrist) with a total of three wet-wipes and the wipes were saved for analysis, again in a self-sealing plastic bag. If the child objected strongly on the first day, only one wet-wipe was used so as not to discourage the child from participating during the rest of the week. Usually where this occurred, the child was sufficiently well acquainted with the sampler by the second day for the full three wet-wipes to be administered.

Figure 4.5 provides a summary of the environmental samples obtained at each location.

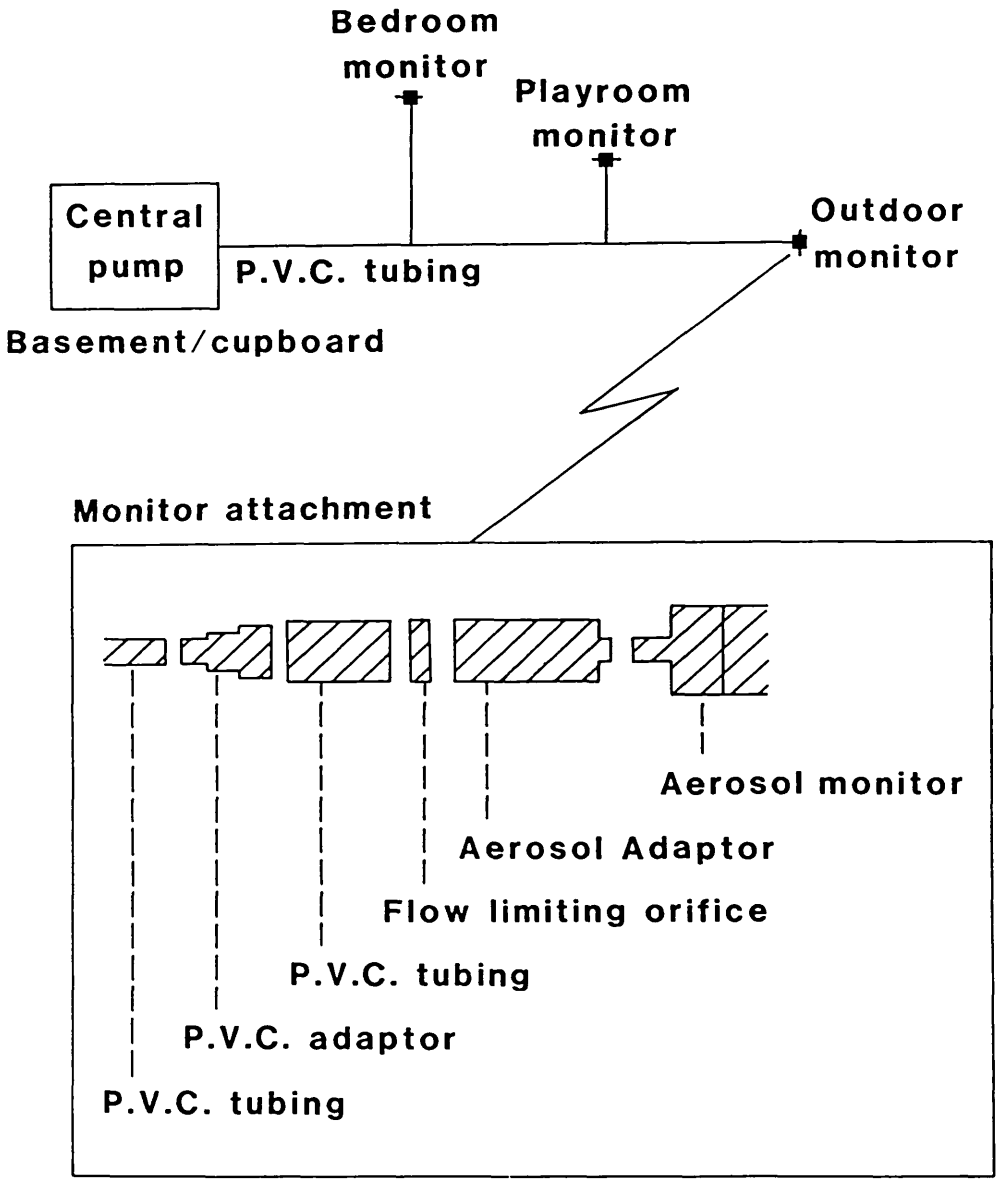
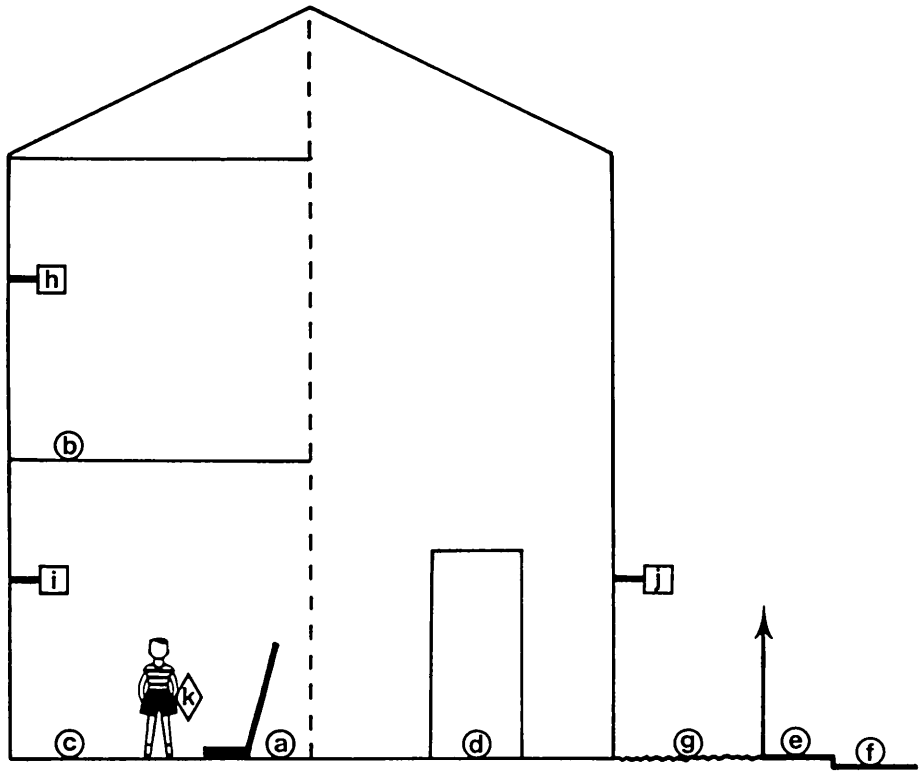


Figure 4.4 Schematic diagram of household air monitoring network



ⓐ Housedust - householder's vacuum cleaner

ⓑ Bedroom dust
ⓒ Playroom dust
ⓓ Housedust - under doormat

} Imperial College vacuum cleaner

ⓔ Pavement dust
ⓕ Road dust
ⓖ Soil
[h] Bedroom air
[i] Playroom air
[j] External air
◇k Handwipe

Figure 4.5 Summary of environmental samples obtained at each location

4.3.2.5 Food and water

Parents were asked by the BMRB field officers to collect and weigh duplicate samples of all food and drink (including water) consumed by their children over a seven day period. Containers were provided for duplicate samples of food and drink consumed inside and outside the home. A set of scales and a diary were provided for recording weights and for noting the different types of food consumed by the child. In addition, a composite tap water sample was obtained from each household by asking the parent to fill a graduated sample bottle with a 50 ml sample at a random time on each day of the week, from the tap(s) normally used for cooking or drinking.

The field officer called at the house two days after placement of the diary and equipment in order to check that weighings had been carried out correctly and that the diaries had been filled out properly. At the end of the study period, diet and water samples were collected and stored in a cold store near Birmingham and then transported to the Laboratory of the Government Chemist for analysis.

4.3.2.6 Blood

Parents and children were provided with transport to and from the University of Birmingham for the blood letting, which was carried out in autumn, 1984 and, where repeat samples were obtained, also in spring, 1985. All blood specimens were collected by venepuncture, by either a qualified doctor (initial sampling programme) or a qualified nurse (repeat sampling programme) and transferred to 2 ml polycarbonate tubes containing 3.5 mg of K_2H_2EDTA as anti-coagulant. All samples were stored at -20^0 and transported to the Trace Element Unit, University of Southampton, for analysis.

4.3.2.7 Hand-to-mouth activity

Each child was filmed using a portable video recorder (Sony F1 Betamax) and a low light, autofocus camera (JVC GXN-70) for approximately four hours, in a single session during the morning and early afternoon. As the aim was to collect data that were typical, the observer requested that the parent made as few alterations as possible to the child's normal routine. The only structure imposed was that the child was filmed in four different settings : free activity (play), being read a story, during its mid-day meal and while watching TV.

After the initial setting-up and testing of the apparatus, the child was recorded in a normal free play situation for 20 minutes. This recording was then played back because:

(a) it was generally of interest to the child and its family;

(b) it often had the effect of reassuring apprehensive children about the function of the camera and recorder;

(c) it provided the operator with a confirmation that the equipment was functioning correctly.

Once this had been done, the recorder was reactivated and the child filmed continuously for a period of 20 minutes. This was timed by a stopwatch displayed in the viewfinder and recorded onto the tape.

The next situation involved the parent reading a story to the child. In order to provide a standard setting, all children were read the same story, from the same book, provided by the filmer. As soon as the book was handed over, recording was resumed until either the child and/or parent clearly signified that they had

finished or until 10 minutes had elapsed.

The complete lunch period was also filmed. Recording began just prior to the food being placed in front of the child and continued until the child had obviously finished and left the table/high-chair/chair. Hand and face wiping after the meal were included in the recording.

After lunch, a second video recorder and monitor (Sony F1) were set up to play back a videotape of a popular childrens TV programme ("Playschool"). A standard 15 minute segment of the tape was played to each child during which time they were again video recorded. All children were filmed in this order.

Prior to leaving the house the observer ensured that the child's project number was noted on the tape and that, if there had been more than one child present during the filming, there was some unambiguous way of identifying the target child. Finally, the parent was asked a series of questions about how typical the child's behaviour was during the filming.

4.3.2.8 Questionnaire

A comprehensive, 140 item, questionnaire was administered by one of the environmental field officers towards the end of the monitoring period. This covered aspects of family and social background, along with the physical environment of the home and is shown in Appendix B. Parents were also interviewed by the dietary field officers at the end of the period to ascertain whether the child had been ill during the study week and to gain information on the use of hot and cold water taps for drinking and cooking purposes.

4.3.2.9 Logistics

The timetable of events in and around each house has been summarised in Table 4.2.

Table 4.2 Timetable for sample collection at each house

SAMPLE TYPE	DAY OF MONITORING PERIOD 1 2 3 4 5 6 7 8 9
Environmental	
- Dust - Road	[--------ANY DAY--------]
- Pavement	[--------ANY DAY--------]
- House (VAC)[1]	[-----ANY DAY-----]
- House (IC)[2]	C
- Soil	[--------ANY DAY--------]
- Air	PC C C C C C C CR
Handwipe	C C C C C C C C
Dietary	
- Food/drinks*	P C C C C C C C R
- Water*	PC C C C C C C R
Behavioural - Videotape	[--------ANY DAY--------]

(VAC)[1] Householder's vacuum cleaner sample
(IC)[2] IC vacuum cleaner (i.e. playroom, bedroom and doormat) samples
P Placement of equipment
R Removal of equipment
* Householder responsible for sample collection
C Day of sample collection

It was not possible for the environmental teams to install the air monitoring networks in all of their 5 houses in one day. Installation was therefore staggered,

with three networks being set up on the first day and the remaining two on the second day of the seven monitoring periods. As an example, on the first day of the study, November 27th, 1984, Team 1 set up Houses 1, 2 and 3, Team 2, Houses 6, 7 and 8 and Team 3, Houses 11, 12 and 13, this being Day 1 of the monitoring period in these houses. On the second day of the study, November 28th, 1984, Team 1 set up Houses 4 and 5, Team 2, Houses 9 and 10 and Team 3, Houses 14 and 15, this being Day 1 of the monitoring period in these houses but Day 2 of the monitoring period in Houses 1, 2, 3, 6, 7, 8, 11, 12 and 13.

The complexity of the sampling programme necessitated the compilation of diary sheets for each day of the project (see specimen in Appendix B). These were provided to ensure that each team member knew exactly what his duties were on each project day. In addition, check sheets were compiled for each individual house/child to ensure that all samples were taken at each location (see Appendix B).

4.3.3 Analytical techniques

All glassware used for the analysis of dust, soil, air filter and handwipe samples was soaked in 2% nitric acid overnight, rinsed four times with de-ionised water (DIW) and dried in a drying cabinet.

4.3.3.1 Dusts

All housedust, pavement and road dust samples were dried at 80°C for 24 hours and passed through a 1 mm sieve. The total weights of the thimble dust samples from the bedroom and playroom were noted to enable the calculation of loading figures. A 0.25 g subsample was then taken from each sample and placed in a test tube. Four ml of concentrated nitric acid (70%, Analar, BDH

Limited, Poole) were added, followed by 1 ml of concentrated perchloric acid (60%, Analar, BDH). The tube was then placed in a deep, programmable heating block and the sample digested and taken to dryness over 24 hours using the following programme:

Dwell time (hours)	Temperature (°C)
3	50
3	150
18	190

The sample was allowed to cool and 2 ml of 6M hydrochloric acid (Analar, BDH) were added. The tube was then placed in a shallow heating block and the sample leached at 60°C for 1 hour. Eight ml of DIW were added, the sample mixed in a vortex mixer, transferred to a disposable tube and centrifuged (2000 rpm for 5 minutes). The solution was then analysed for lead and a range of other elements by inductively coupled plasma atomic emission spectrometry (ICPAES) [ARL 34000]. A brief description of this technique and the standard operating conditions used is given in Appendix C.

4.3.3.2 Soil

All samples were dried at 80°C for 24 hours, passed through a 2mm sieve and milled in an agate mill ("Tema"). Subsamples (0.25 g) were then digested and leached by the same method as that described above for dust samples. The final solutions were analysed for lead and a range of other elements by ICPAES.

4.3.3.3 Air

To minimise the chance of contaminating any of the samples, analytical procedures were carried out in a clean laboratory with a filtered air supply. Each air filter

was carefully transferred from the sealed monitoring head into a beaker (50 ml) using plastic forceps. Four ml of concentrated nitric acid (70%, Aristar, BDH) were added and the sample was heated gently (at 60°C) until the filter had dissolved. The sample was then transferred into a test tube and the beaker was rinsed with a few ml of DIW which was also added to the test tube. One ml of concentrated perchloric acid (60%, Aristar, BDH) was then added and the test tube placed in a deep, programmable heating block. The following programme was used to complete the digestion and take the sample to dryness:

Dwell time (hours)	Temperature (°C)
1	100
6	125
3	150
16	190

Two ml of 1 M hydrochloric acid (70%, Aristar, BDH, with 5 mg/l lanthanum "spike" as an internal standard) were added and the test tube was placed in a shallow heating block and heated gently (at 60°C) for one hour. The solution was then transferred, without rinsing, to a disposable tube and analysed for lead by ICPAES.

4.3.3.4 Handwipes

Analytical procedures were carried out in a clean laboratory with a filtered air supply to minimise the chance of contamination. The handwipes were transferred into a conical flask (250 ml) using plastic forceps and 30 ml of concentrated nitric acid (70%, Analar, BDH) were added. Only 20 ml were used for a sampler's single handwipe. The flask was covered with a watchglass and heated gently (at around 60°C) until the wipes had disaggregated. The temperature was then increased to 110°C for a further 48 hours, during which time the sample

was occasionally shaken gently to aid digestion. The watchglass was then removed and the sample taken to dryness. Two ml of 6 M HCl (70%. Analar, BDH) were added, the sample was gently heated (at 60°C) for 5 minutes and the leachate decanted, with rinsing, into a disposable tube. The volume was made up to 10 ml with DIW and the solution analysed for lead by flame atomic absorption spectrophotometry (Perkin Elmer 5000) [see Appendix C for the standard operating conditions used].

4.3.3.5 Food/beverages and water

As outlined earlier, these samples were analysed by the Laboratory of the Government Chemist. The methods employed have been given in detail elsewhere (Evans et al., 1978) but were briefly as follows. Food/beverage samples were thoroughly homogenised and representative subsamples (around 10 g) were obtained. Both these samples and the water samples were digested in nitric and sulphuric acids. Lead was pre-concentrated by chelation with a mixture of ammonium tetramethylenedithiocarbamate and diethylammonium diethyl-dithiocarbamate and extracted with 4-methylpentan-2-one. All measurements were carried out by AAS.

4.3.3.6 Blood

These samples were analysed by the Trace Element Unit, University of Southampton, under the direction of Dr. H.T. Delves. Analyses were carried out in triplicate on 10 µl subsamples of the whole blood using microsampling flame AAS. The method has been described in detail elsewhere (Delves, 1970). Briefly, after partial oxidation with hydrogen peroxide in micro-crucibles made from nickel foil, the samples are volatilised by using an air-acetylene flame into a nickel absorption tube in the flame.

4.3.3.7 Hand-to-mouth activity

Pilot studies had been carried out on five families for five complete days prior to the main study. From extensive observation of these tapes it was possible to derive a full description of the two aspects of the child's behaviour of primary interest - what went into the mouth and what the hands touched. The number of behavioural categories that this produced was far too large to be practicable and so smaller, more inclusive categories were produced. This process resulted in 36 categories of mouthing behaviour (i.e. objects touching or going into the mouth) and 21 categories for hand contact (see Table 4.3). The categories were designed to be mutually exclusive and as unambiguous as possible. An uncodable category was included to take account of those occasions when the child's hands and /or mouth were not visible to the rater. A further category, unclassified, was included to take account of any behaviour not included in the original classification.

The observation procedure itself used a standard B.B.C. microcomputer programmed to allow each key on the keyboard to be coded to represent one of the behavioural categories. Labels with the appropriate behavioural code were affixed to the keys. When each key was pressed a counter was incremented and a timer started. The next time a key was pressed the first timer was stopped, another timer started and another counter incremented. The time from the first key press was stored, along with the value from the counter for that key code, so that at the end of the observation period a total frequency and duration for each behavioural category was available.

Two observers were used, one to rate the mouthing activity and one to rate the hand contact. Observation was carried out on the following portions of each tape:

Table 4.3 Behavioural categories

CONTACT WITH MOUTH

Code	Description
UC	Uncodeable
NC	No contact
SFV	Soft furnishings - vertical
SFH	Soft furnishings - horizontal
SMV	Smooth furnishings - vertical
SMH	Smooth furnishings - horizontal
FL	Floor
CL	Other person (body/clothes)
TOW	Flannel, dishcloth, towel
BLA	Security blanket
TAB	Tablecloth, mat
THF	Toy - hard (food related)
TSF	Toy - soft (food related)
TH	Toy - hard
TS	Toy - soft
PR	Pram, buggy
CRE	Living/dead animal
OO	Other object (not toy)
DUM	Dummy, pacifier
FOO	Food and drink
EAT	Eating utensils
FEH	Feeding with utensils
DRU	Drinking with utensils
DRW	Drinking without utensils
BOD	Body (own body/ clothes)
HAN	Hand (back, palm, wrist)
HF	1 hand 1 finger
HM	1 hand >1 finger
HT	1 hand 1 thumb
HSF	2 hands 1 finger of each hand
HSM	2 hands >1 finger
HST	2 hands 2 thumbs/ 1 finger + 1 thumb
HA	1 hand in mouth area
HSA	2 hands in mouth area
UCL	Unclassified

CONTACT WITH HAND

Code	Description
UC	Uncodeable
NC	No contact
SFV	Soft furnishings - vertical
SFV	Soft furnishings - horizontal
SMV	Smooth furnishings - vertical
SMH	Smooth furnishings - horizontal
CA	Carpet
LI	Lino
BO	Boards
CL	Clothes (own,sibs, parents)
BLA	Security blanket
TH	Toy - hard
TS	Toy - soft
PR	Pram, buggy
WAT	Water (playing)
OO	Other object
FOO	Food and drink
EAT	Eating utensils
BOD	Body (own, parents, sibs)
UCL	Unclassified

(a) free activity - final 10 minutes;
(b) book, meal, TV - complete period.

Each tape was rated for the whole of the appropriate time period and was not time-sampled within these periods.

Tapes were rated three times in all - once for the mouthing behaviour and twice for hand contact, each hand being rated separately. The first observer noted specific beginning and ending points on each tape to ensure that both observers rated the same section of tape. As all tapes had a stop-watch recorded on them, this indicator of when to start/stop rating was usually some index of elapsed time.

Both observers worked separately and only communicated essential information during the whole observation process in order to avoid possible biases.

4.3.3.8 Quality control

A rigorous quality control programme was devised and implemented during sample analysis. This is described in detail below for each section of the study.

(a) Environmental

For measurements made on the ICPAES and AAS, instrumental blanks and standard solutions were run at regular intervals (either every 15 minutes or after every 10 samples) during batch runs to monitor instrumental performance. If results for these check solutions fell outside predetermined limits, the machine was recalibrated. Samples which had been run between a previous successful check and an unsuccessful one were then re-run.

(i) Dusts and soils

Along with the reagent blanks and duplicates, three house reference materials (HRM) of differing lead concentrations were selected for inclusion. HRM 1 (soil) contained 13 µg/g Pb, HRM 2 (soil), 510 µg/g Pb and HRM 7 (housedust), 1590 µg/g Pb. The protocol followed is given in Table 4.4.

Table 4.4 Quality control protocol for dust and soil analysis

	Dusts	Soils
Number of samples	630	105
Number of solutions	743	124
Block batches	3 (250 tubes)	1
Instrument batches	6 (125 solutions)	1
Randomly within each block batch:		
Reagent blanks	5	3
Duplicated samples	25	10
HRM 1	5	3
HRM 2	5	3
HRM 7	5	0
Batch size	250	124

(ii) Air

Both reagent and procedural blanks were incorporated. The latter included unused air filters, from the same batch as those used for sampling, plus the analytical reagents. Blank filters were also "spiked" with 5 µg Pb (0.1 ml of HRM 9 stock solution containing 50 mg/l Pb) and with 2 µg Pb (0.1 ml of HRM 10 multi-element

stock solution containing 20 mg/l Pb) to assess recoveries. Due to the unique nature of the individual air filter samples, it was not possible to include duplicates. The full quality control protocol is given in Table 4.5.

Table 4.5 Quality control protocol for air filter analysis

Number of samples	2205
Number of solutions	2646
Block batches	11 (250 tubes)
Instrument batches	21 (125 solutions)

Randomly within each block batch:

Reagent blanks	12
Procedural blanks	12
HRM 9 "spike"	12
HRM 10 "spike"	12

(iii) Handwipes

As with the air filters, both reagent and procedural blanks were used. Blank wipes were "spiked" with 10 µg Pb (1.00 ml of HRM 8 stock solution containing 10 mg/l Pb) and with 50 µg Pb (1.00 ml of HRM 9). Dust "spikes" were also used and included 50 mg HRM7 (1590 µg/g Pb) and 10 mg of National Bureau of Standards Reference Material (NBSRM) 1648 (6550 µg/g Pb). It was not possible to include duplicates because each sample was unique. The protocol followed is given in Table 4.6.

Table 4.6 Quality control protocol for handwipe analysis

Number of samples	1470	
Number of solutions	1745	
Hotplate batches	35	(50 flasks)
Instrument batches	12	(150 solutions)
Randomly within each hotplate batch:		
Reagent blank	1	
Procedural blank	2	
HRM 7 "spike"	2	
HRM 8 "spike"	1	
HRM 9 "spike"	1	
Plus, within each instrument batch:		
NBSRM 1648 "spike"	1	

(b) Food/beverages and water

The protocol employed by the Laboratory of the Goverment Chemist was reported to be as follows. Reagent blanks were included with each analytical batch. Analysis of National Bureau of Standards' Reference Materials, Wheat Flour and Bowens Kale was used to assess accuracy and duplicate analyses were performed to assess precision (Smart et al., 1988).

(c) Blood

An extensive quality control assurance programme was implemented during the analysis of these samples at the University of Southampton. The programme was similar to

that used in the analysis of blood from a subsequent lead survey (UK Blood Lead Monitoring Programme, 1984-1987) by this laboratory, which has been described in detail elsewhere (DoE, 1986). In brief, three internal quality control (IQC) blood specimens were used, with lead concentrations of approximately 7, 14 and 27 μg/dl. These were analysed immediately following an accepted calibration and thereafter following every set of not more than five test samples. No analysis of sample specimens was carried out unless the immediately preceeding three IQCs were within predetermined acceptable limits and any subsequent set of test results were only accepted if they were followed by three IQCs within limits. Thus every set of accepted results was bounded by two sets of acceptable IQCs. All samples were analysed in triplicate. Within sample precision was considered acceptable if the standard deviation of the triplicate measurements was less than 0.05 μmol/l (1 μg/dl).

In addition to analysis by microsampling flame AAS, the samples that were collected in the second sampling programme (spring, 1985) were also analysed by electrothermal atomisation AAS and subjected to the same criteria for IQC limits and within sample precision described above.

(d) Hand-to-mouth activity

Reliability checks were made at the end of the main data collection phase. For intra-observer reliability, 10 tapes were randomly selected for each observer. For inter-observer reliability, a further 20 tapes were randomly selected, 10 for each observer, giving a total of 40 tapes which were re-rated.

5. THE BIRMINGHAM STUDY: RESULTS

The purpose of this chapter is to document and describe but not to discuss in detail the data generated by this study. The very large nature of the data set has justified the use of the following 2 chapters (Chapters 6 and 7) for the purpose of discussion and interpretation. Where possible, the raw data have been tabulated, which is the case for the dust, soil, handwipe and blood results. Otherwise, data summaries are presented, which is the case for the air, dietary and behavioural results.

5.1 DUSTS

5.1.1 Housedust

The results from the analysis of dust samples taken within each house are presented in Table 5.1. It should be noted that for the bedroom and playroom, dust lead loading figures have been calculated and included. These figures were derived using the following formula:-

$$\text{Dust loading} = \frac{\text{Dust lead conc. x Mass of dust collected}^{1}}{\text{Area sampled}}$$

1: <1mm fraction

As an example, the relevant information has been extracted from the data base for the bedroom of House 1 and the dust lead loading figure has been calculated below.

Location: House 1, bedroom
Dust lead concentration: 720 μg/g
Mass of dust collected: 1.21 g
Area sampled: 4 m^2

$$\text{Dust lead loading} = \frac{720\ \mu g/g \times 1.21\ g}{4\ m^2}$$

$$= 218\ \mu g/m^2$$

The population distributions of each sample type are presented in Figures 5.1 to 5.6. It can be seen that all of these show log-normal distributions, which suggests that the most appropriate measure of central tendency in all populations is the geometric, rather than arithmetic, mean. The data summaries at the bottom of Table 5.1 therefore include the geometric mean and 5th to 95th percentile range, which were as follows for each sample type: in terms of lead concentration, householder's vacuum cleaner, 336 µg/g (97-1440 µg/g); bedroom, 464 µg/g (109-2040 µg/g); playroom, 311 µg/g (105-1030 µg/g); doormat, 615 µg/g (120-4300 µg/g); and in terms of lead loading, bedroom, 51 µg/m^2 (3-417 µg/m^2); playroom, 42 µg/m^2 (1-614 µg/m^2).

5.1.2 Pavement and road dust

The results are presented in Table 5.2. Again, the data were log-normally distributed (Figures 5.7 and 5.8). Geometric means and 5th to 95th percentile ranges have therefore been included in the data summaries at the end of this Table and were 360 µg/g (127-1340 µg/g) and 527 µg/g (195-1170 µg/g) for pavement and road dust, respectively.

5.2 SOIL

The results from soil sample analyses are also presented in Table 5.2. Again, the population distribution showed a log-normal distribution (Figure 5.9) and so the geometric mean, 313 µg/g and 5th-95th percentile range, 92-1160 µg/g, have been included.

Table 5.1 Housedust lead levels

HOUSE NUMBER	DUST LEAD CONCENTRATION µg/g					DUST LEAD LOADING µg/m²		
	DOORMAT[1]	VACUUM[2]	BEDROOM[3]	PLAYROOM[4]	AVERAGE[5]	BEDROOM	PLAYROOM	AVERAGE
1	79	500	720	375	548	218	304	261
2	470	76	99	122	111	13	13	13
3	*	830	2700	440	1570	1250	610	930
4	15000	134	1870	160	1015	206	32	119
5	*	230	176	300	238	109	631	370
6	2100	23000	1620	4600	3110	325	1799	1062
7	4300	520	3500	600	2050	329	154	241
8	4300	1400	390	550	470	274	78	176
10	*	320	280	175	228	35	28	32
11	1430	350	770	410	590	31	14	22
12	340	460	1850	151	1001	117	50	84
13	350	137	300	189	245	45	17	31
14	320	230	510	480	495	13	5	9
15	*	570	200	126	163	5	3	4
16	420	370	620	230	425	88	21	54
17	810	*	107	230	168	13	77	45
18	860	660	240	920	580	104	827	466
19	*	470	530	5600	3065	201	1761	981
20	*	*	660	460	560	354	711	532
21	*	310	640	174	407	160	36	98
22	119	250	470	270	370	329	109	219
23	*	165	650	300	475	294	73	183
24	*	300	610	290	450	16	27	22
25	*	400	490	280	385	468	63	265
26	280	260	340	240	290	3	1	2
27	200	270	520	96	308	327	9	168
28	350	70	230	90	160	7	1	4
29	3100	1210	560	260	410	32	37	35

contd./

Table 5.1 contd.

HOUSE NUMBER	DUST LEAD CONCENTRATION µg/g				DUST LEAD LOADING µg/m²		
	DOORMAT[1]	VACUUM[2]	BEDROOM[3]	PLAYROOM[4]	AVERAGE[5]	BEDROOM	PLAYROOM	AVERAGE
30	*	96	470	182	326	9	5	7
31	660	400	760	320	540	38	154	96
32	178	320	740	330	535	8	118	63
33	*	*	250	290	270	313	435	374
34	*	210	630	290	460	151	94	122
35	*	280	330	250	290	19	15	17
36	*	280	320	690	505	8	26	17
37	9400	420	310	330	320	370	338	354
38	640	200	300	73	187	19	4	12
39	1290	200	169	210	190	2	9	5
40	*	240	210	230	220	2	6	4
42	2300	240	1090	1270	1180	61	33	47
43	*	530	4500	109	2305	23	1	12
44	*	1800	1090	1800	1445	34	393	214
45	270	6100	1080	750	915	196	6	101
46	130	220	780	340	560	75	126	101
47	320	500	450	520	485	75	90	83
48	*	147	290	270	280	40	95	68
49	*	270	110	560	335	93	461	277
50	950	450	540	370	455	674	66	370
51	*	510	420	1160	790	9	69	39
52	*	46	280	176	228	41	77	59
53	1190	310	250	620	435	42	17	30
54	*	410	1250	460	855	27	26	26
56	*	300	*	62	*	*	1	*
58	*	137	168	*	*	17	*	*
59	*	160	69	210	140	1	4	3
60	*	*	1280	450	865	50	4	27

contd./

Table 5.1 contd.

HOUSE NUMBER	DOORMAT[1]	DUST LEAD CONCENTRATION µg/g VACUUM[2]	BEDROOM[3]	PLAYROOM[4]	AVERAGE[5]	DUST LEAD LOADING µg/m² BEDROOM	PLAYROOM	AVERAGE
62	*	400	560	230	395	122	194	158
63	*	320	510	350	430	45	100	73
64	*	230	350	350	350	111	531	321
65	*	*	1670	410	1040	610	206	408
66	*	189	270	194	232	3	10	7
67	*	260	520	270	395	149	176	162
68	*	*	540	440	490	189	88	138
70	*	113	53	310	182	9	61	35
71	320	510	530	600	565	17	7	12
72	*	890	970	1010	990	23	456	240
73	420	290	500	260	380	1348	81	714
74	480	192	199	240	220	14	39	26
75	2500	390	240	144	192	8	*	*
76	670	890	570	149	360	350	71	210
78	125	470	720	308	510	137	66	101
79	*	970	2800	570	1685	110	14	62
80	180	550	4100	340	2220	164	82	123
81	*	310	360	440	400	380	333	357
82	*	173	440	250	345	176	57	116
83	*	310	1150	280	715	148	49	98
84	1010	1490	580	490	535	63	97	80
85	*	320	520	500	510	394	154	274
86	*	162	520	200	360	11	4	7
87	*	270	1830	350	1090	405	52	228
88	240	510	250	460	355	10	23	17
89	*	310	620	420	520	16	90	53
90	*	187	450	330	390	15	1	8
91	420	480	360	420	390	114	141	127

contd./

Table 5.1 contd.

HOUSE NUMBER	DUST LEAD CONCENTRATION µg/g DOORMAT[1]	VACUUM[2]	BEDROOM[3]	PLAYROOM[4]	AVERAGE[5]	DUST LEAD LOADING µg/m² BEDROOM	PLAYROOM	AVERAGE
92	*	300	310	250	280	99	247	173
93	360	320	560	290	425	59	68	63
94	*	2500	800	560	680	39	124	81
95	*	660	800	220	510	91	427	259
96	1230	380	208	220	210	10	4	7
99	*	220	162	107	135	38	13	25
100	*	104	122	185	154	15	36	25
101	*	140	120	147	134	19	8	13
102	*	210	600	119	360	152	27	90
103	*	133	131	126	129	3	6	5
104	*	270	210	195	203	13	25	19
105	*	280	330	210	270	121	53	87
106	183	560	*	550	*	*	41	*
Arithmetic mean	1436	713	693	455	578	141	150	149
Minimum	79	46	53	62	111	1	1	2
Maximum	15000	23000	4500	5600	3110	1348	1799	1062
Geometric mean	615	336	464	311	430	51	42	62
5th percentile	120	97	109	105	139	3	1	4
95th percentile	4300	1440	2040	1030	1780	417	614	486

* Missing data
1 I.C. cleaner - under doormat
2 Householder's own cleaner
3 I.C. cleaner - child's bedroom
4 I.C. cleaner - child's playroom
5 Average of bedroom and playroom

Figure 5.1 Frequency distribution of doormat dust concentrations

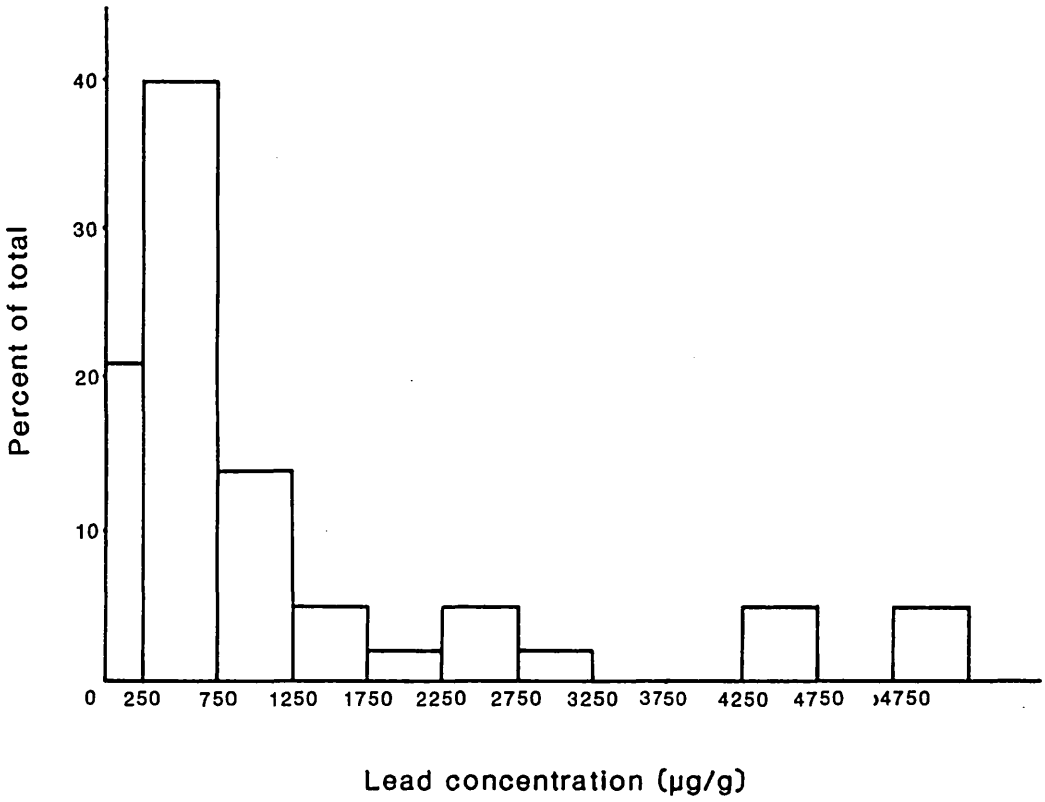


Figure 5.2 Frequency distribution of householder's vacuum cleaner dust lead concentration

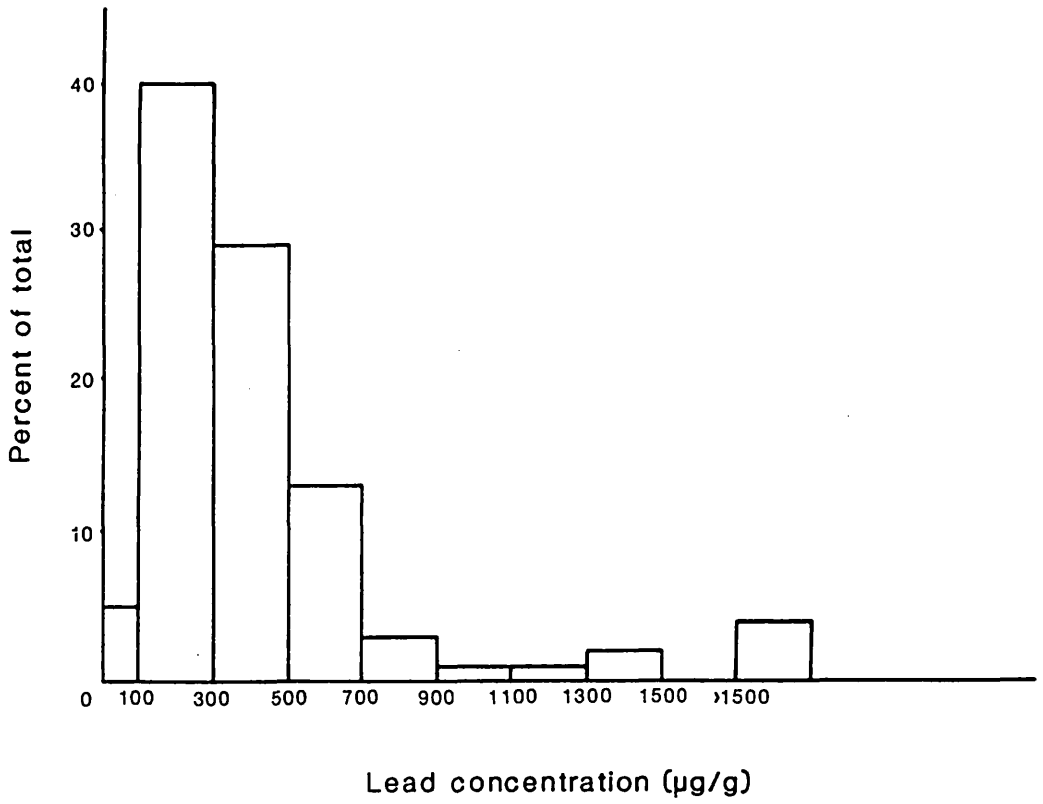


Figure 5.3 Frequency distribution of bedroom dust concentrations

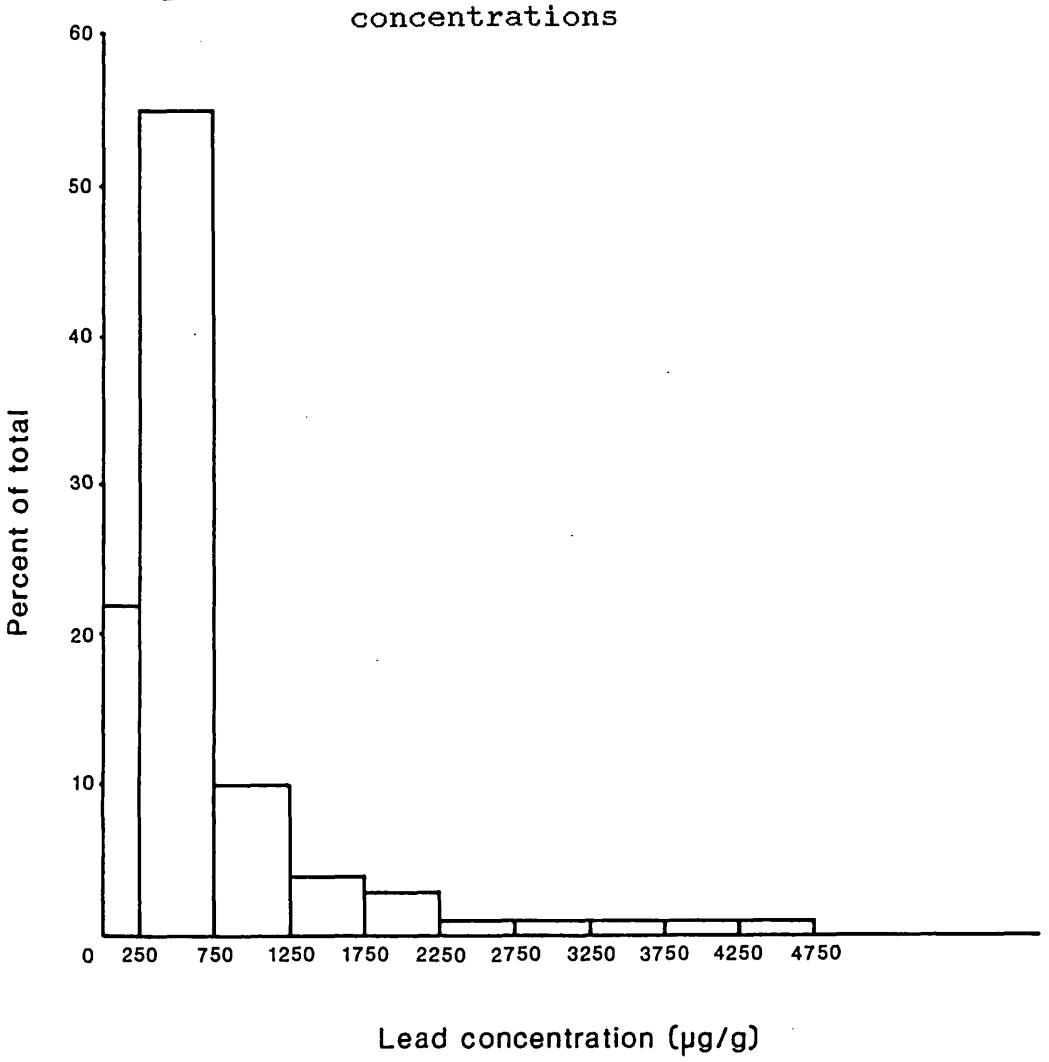


Figure 5.4 Frequency distribution of playroom dust lead concentrations

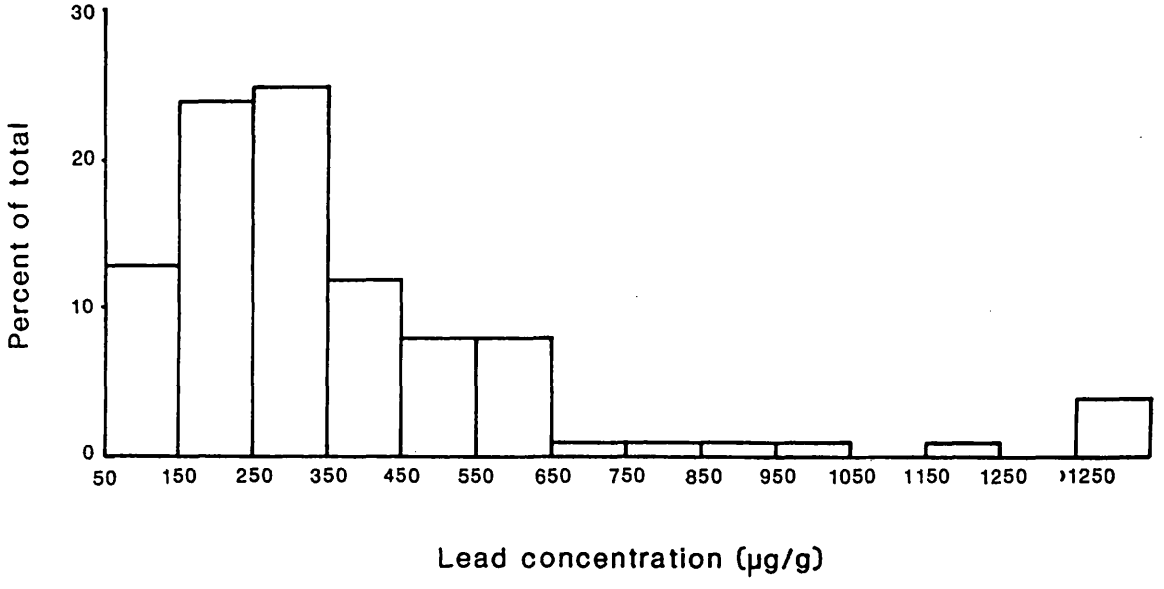


Figure 5.5 Frequency distribution of bedroom dust loadings

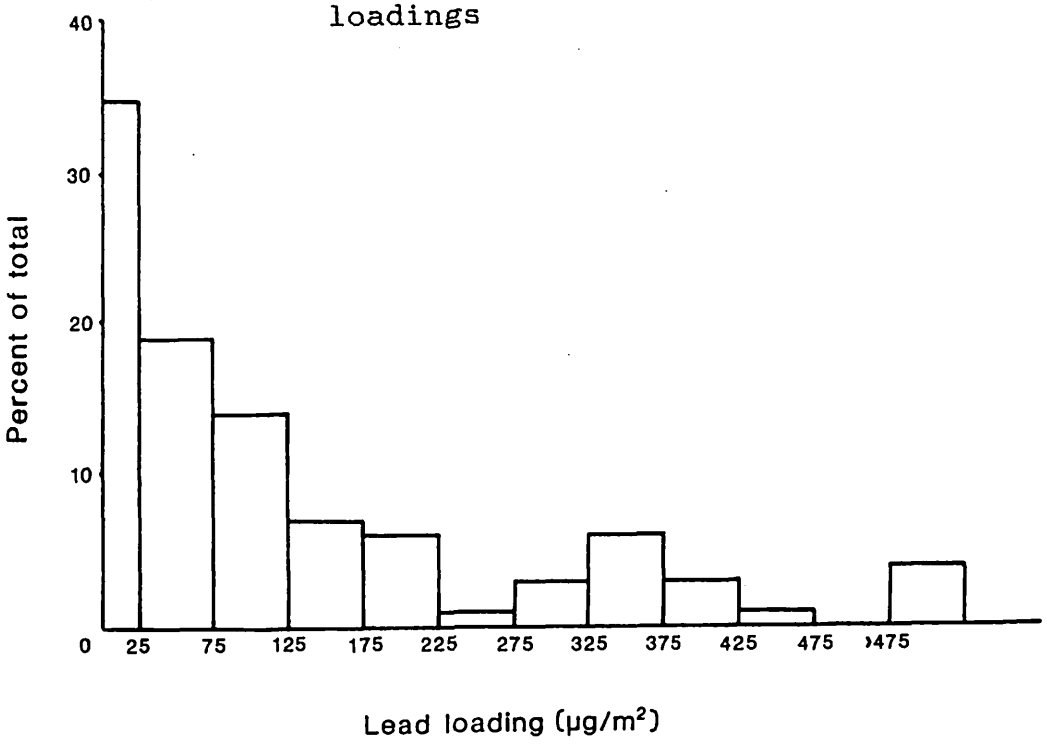


Figure 5.6 Frequency distribution of playroom dust lead loadings

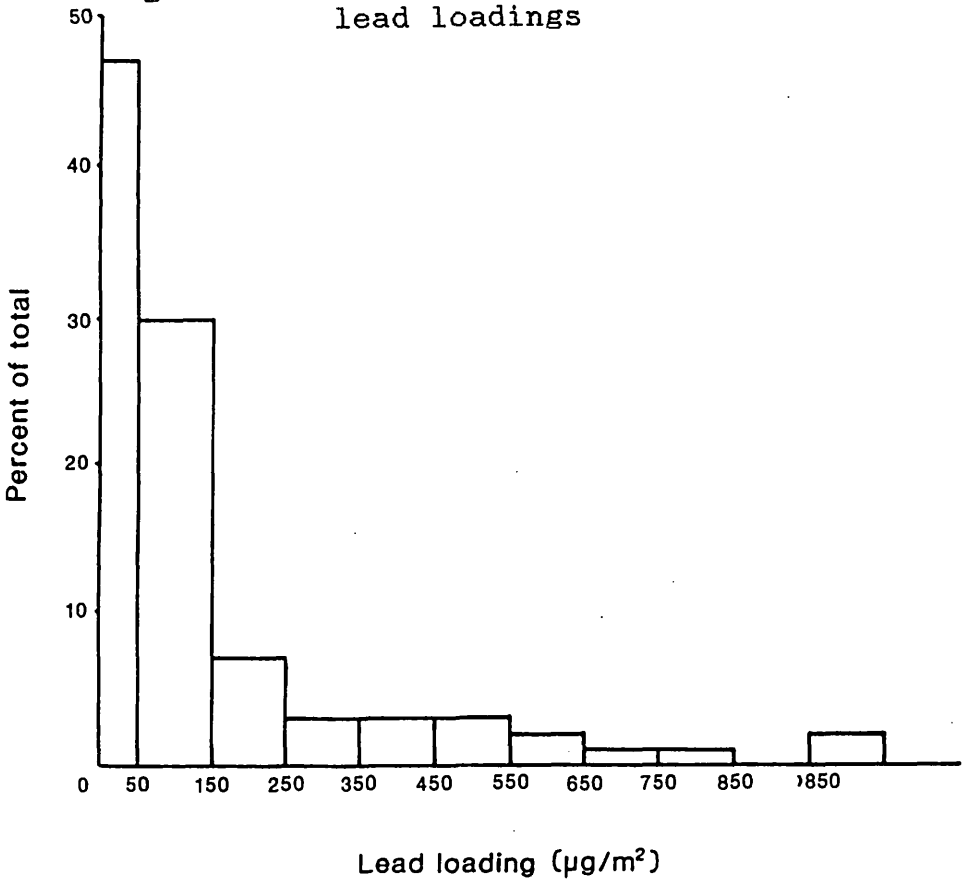


Table 5.2 Soil, pavement dust and road dust lead levels

HOUSE NUMBER	SOIL LEAD CONC. (µg/g)	DUST LEAD CONC. (µg/g) PAVEMENT	ROAD
1	640	560	240
2	360	220	730
3	1650	600	540
4	510	320	1110
5	1040	590	820
6	1180	520	760
7	1330	1190	720
8	390	1250	490
10	*	185	187
11	240	135	1030
12	230	370	210
13	320	270	670
14	200	740	450
15	460	490	560
16	680	750	300
17	56	171	310
18	770	670	630
19	320	430	760
20	*	400	660
21	290	300	480
22	*	380	270
23	*	340	240
24	640	210	740
25	960	470	860
26	200	5100	550
27	200	570	400
28	80	196	320
29	128	155	460
30	130	135	166
31	350	520	280
32	123	440	300
33	76	159	3200
34	470	3000	1080
35	1100	320	380
36	171	280	330
37	185	330	420
38	109	156	650
39	189	280	320
40	380	400	680
42	510	300	590
43	170	260	1650
44	*	330	810
45	210	270	530
46	161	270	420
47	320	500	290
48	174	1440	740
49	147	290	810
50	400	870	860
51	340	470	770
52	350	100	440
53	136	127	440
54	164	300	390

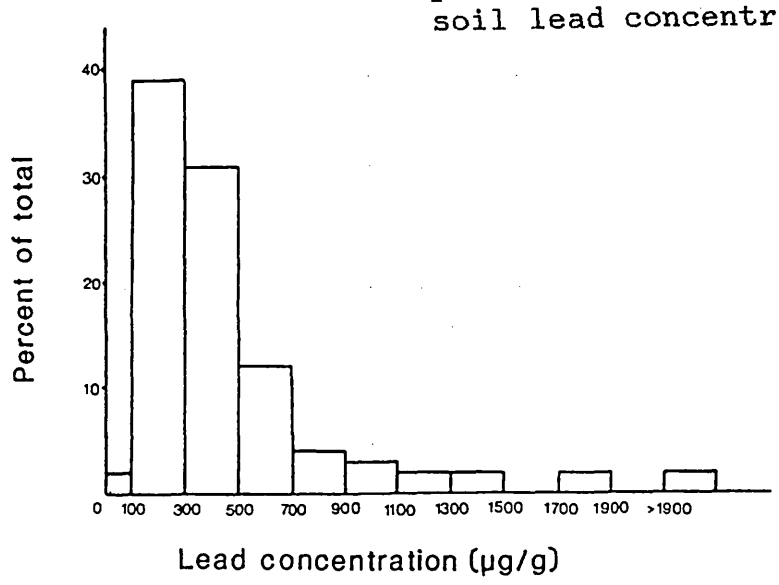
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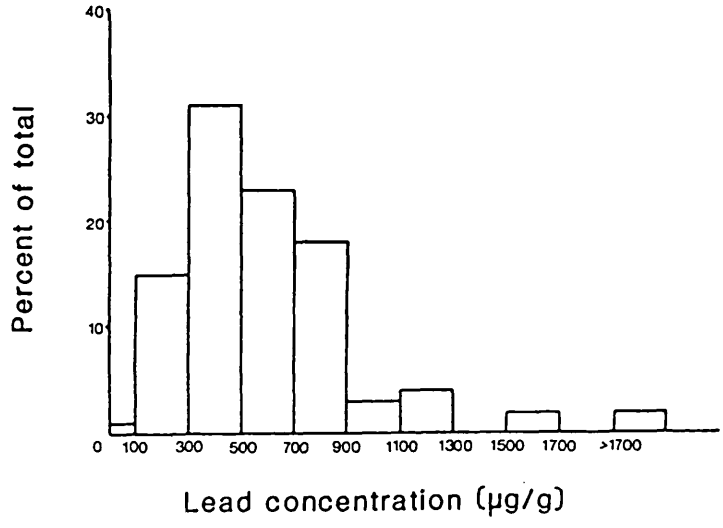
Table 5.2 contd.

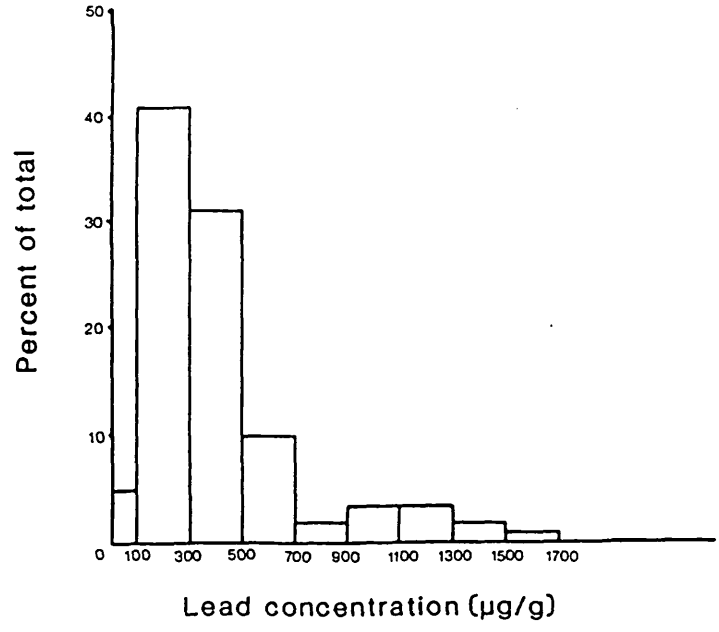
HOUSE NUMBER	SOIL LEAD CONC. (µg/g)	DUST LEAD CONC. (µg/g) PAVEMENT	ROAD
56	240	220	1000
58	122	240	360
59	132	320	650
60	230	480	530
62	480	430	590
63	950	360	370
64	156	144	460
65	650	320	1170
66	*	126	470
67	*	250	240
68	*	240	200
70	390	370	890
71	250	240	370
72	220	200	470
73	280	62	80
74	85	141	21000
75	*	280	340
76	*	1730	196
78	390	167	330
79	440	400	450
80	310	210	430
81	*	210	360
82	600	650	610
83	360	330	560
84	380	220	470
85	270	177	800
86	510	410	480
87	470	450	210
88	168	*	280
89	390	191	300
90	360	270	510
91	550	680	830
92	1260	730	640
93	1440	1030	1130
94	400	530	500
95	750	1320	1620
96	330	370	830
98	330	910	510
99	280	1830	1180
100	250	192	280
101	188	250	720
102	187	380	670
103	184	74	179
104	*	510	840
105	300	370	680
106	320	960	680
Arith. mean	405	506	805
Range	56-1650	62-5100	80-2100
Geo. mean	313	360	527
5th:95th %ile	92:1160	127:1340	195:1170

* Missing data

Figures 5.7-5.9 Frequency distributions of pavement dust, road dust and soil lead concentrations







5.3 AIR

The extensive data set generated by this section of the study is presented in full in Appendix D. Within this Appendix, there are 3 tables, Tables 1, 2 and 3, which contain the results from the bedroom, playroom and outdoor sites, respectively, for each day of the study week for each house. The results are also presented graphically in Appendix D where data from all sampling locations have been plotted against day number for each individual house.

The population distribution of all samples from the bedroom sites is shown in Figure 5.10, from the playroom sites in Figure 5.11 and from the external sites in Figure 5.12. Again, the familiar skewed distribution is apparent in all three cases. The geometric means (5th-95th percentile) for the bedroom (n = 599), playroom (n = 607) and external (n = 605) samples were 0.26 $\mu g/m^3$ (0.09-0.81 $\mu g/m^3$), 0.27 $\mu g/m^3$ (0.08-0.88 $\mu g/m^3$) and 0.43 $\mu g/m^3$ (0.12-1.53 $\mu g/m^3$), respectively, and these are included in a summary of the data (Table 5.3).

The weekly mean air lead concentrations for each sampling location, a derived mean internal air lead concentration and the ratio of the mean internal to mean external concentration for each house are presented in Table 5.4.

Table 5.3 Summary of internal and external air lead concentrations and internal:external ratios

	AIR LEAD CONCENTRATION: $\mu g/m^2$				RATIO[2]
	BEDROOM	PLAYROOM	INTERNAL[1]	EXTERNAL	INT/EXT
N	599	607	1206	605	90
Mean	0.32	0.34	0.33	0.55	0.63
Range	<.05-1.52	<.05-1.96	<.05-1.96	<.05-2.98	0.39-1.33
Geo. mean	0.26	0.27	0.26	0.43	0.61
5th %ile	0.09	0.08	0.08	0.12	0.42
95th %ile	0.81	0.88	0.87	1.53	0.87

1 Mean of all samples taken in the bedroom and playroom
2 Ratio of mean internal air lead concentration to mean external air lead concentration

Table 5.4 Bedroom, playroom and external air lead concentrations at each location

HOUSE NUMBER	WEEKLY MEAN AIR LEAD CONCENTRATION: µg/m³ BEDROOM	PLAYROOM	INTERNAL[1]	EXTERNAL	RATIO[2] INT/EXT
1	0.25	0.21	0.23	0.37	0.62
2	0.19	0.21	0.20	0.39	0.51
3	0.17	0.20	0.18	0.27	0.67
4	0.21	0.24	0.22	0.27	0.81
5	0.28	0.36	0.32	0.61	0.52
6	0.39	0.56	0.48	0.36	1.33
7	0.25	0.29	0.27	0.49	0.55
8	0.22	0.23	0.22	0.26	0.85
10	0.23	0.25	0.24	0.32	0.75
11	0.46	0.31	0.37	0.37	1.00
12	0.32	0.33	0.33	0.53	0.62
13	0.34	0.34	0.34	0.72	0.47
14	0.33	0.38	0.36	0.82	0.44
15	0.60	0.57	0.58	1.15	0.46
16	0.29	0.35	0.32	0.54	0.59
18	0.36	0.39	0.38	0.57	0.67
19	0.37	0.50	0.43	0.69	0.62
20	0.52	0.63	0.57	0.65	0.88
21	0.38	0.45	0.42	0.66	0.64
22	0.53	0.61	0.57	0.90	0.63
23	0.40	0.40	0.40	0.60	0.67
24	0.50	0.65	0.58	0.77	0.75
25	0.60	0.56	0.58	1.12	0.52
26	0.46	0.40	0.43	0.73	0.59
27	0.51	0.37	0.44	0.81	0.54
28	0.46	0.40	0.43	0.94	0.46
29	0.41	0.45	0.43	0.75	0.57
30	0.47	0.54	0.50	0.87	0.57
39	0.39	0.77	0.56	1.32	0.42
40	0.82	0.55	0.70	1.37	0.51
42	0.57	0.71	0.62	1.06	0.58
43	0.48	0.40	0.43	0.89	0.48
44	0.74	0.70	0.72	1.64	0.44
45	0.67	0.86	0.76	1.15	0.66
46	0.16	0.23	0.20	0.52	0.38
47	0.32	0.38	0.36	0.47	0.77
48	0.39	0.33	0.36	0.60	0.60
49	0.25	0.28	0.26	0.43	0.60
50	0.32	0.30	0.31	0.50	0.62
51	0.38	0.33	0.35	0.50	0.70
52	0.26	0.27	0.26	0.44	0.59
53	0.30	0.32	0.31	0.60	0.52
54	0.39	0.34	0.35	0.47	0.74
56	0.19	0.21	0.20	0.49	0.41
58	0.22	0.25	0.24	0.41	0.59

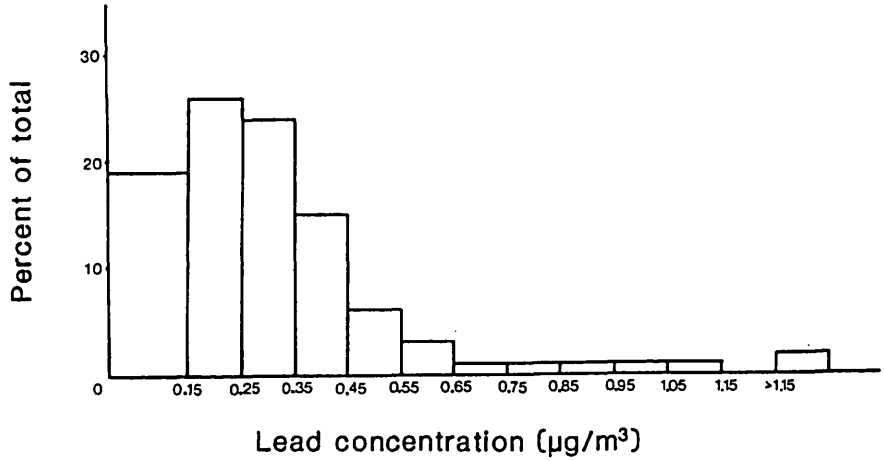
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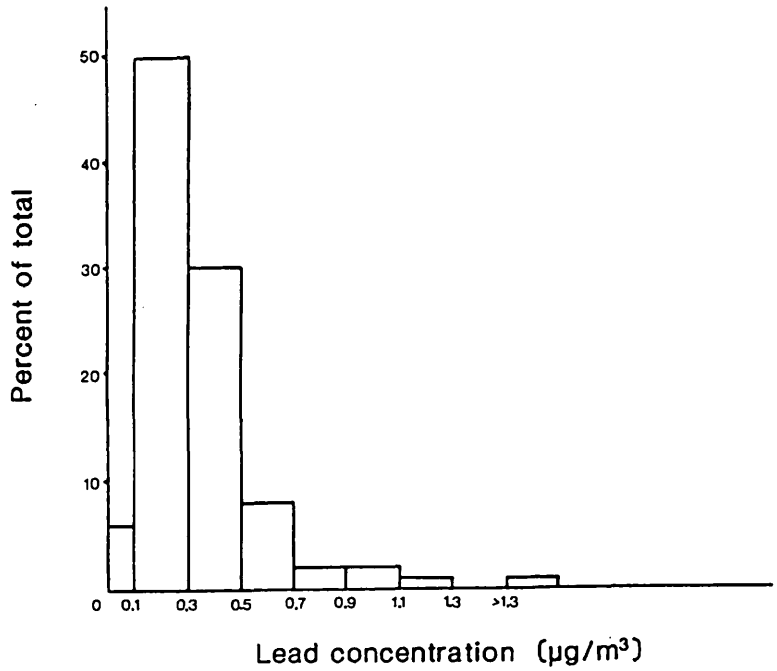
Table 5.4 contd.

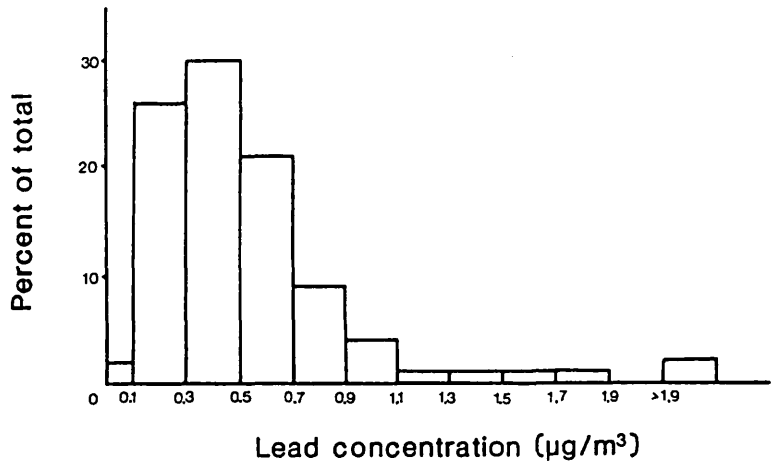
HOUSE NUMBER	WEEKLY MEAN AIR LEAD CONCENTRATION: µg/m³ BEDROOM	PLAYROOM	INTERNAL[1]	EXTERNAL	RATIO[2] INT/EXT
59	0.23	0.23	0.23	0.51	0.45
60	0.33	0.35	0.34	0.45	0.76
62	0.27	0.26	0.26	0.47	0.55
63	0.27	0.16	0.22	0.34	0.65
64	0.20	0.22	0.21	0.38	0.55
65	0.21	0.23	0.22	0.39	0.56
66	0.28	0.24	0.26	0.43	0.60
67	0.26	0.30	0.28	0.42	0.67
68	0.19	0.27	0.23	0.44	0.52
70	0.22	0.31	0.27	0.46	0.59
71	0.33	0.27	0.30	0.46	0.65
72	0.23	0.42	0.32	0.47	0.68
73	0.20	0.29	0.24	0.52	0.46
74	0.31	0.28	0.29	0.54	0.54
75	0.24	0.27	0.26	0.46	0.57
76	0.19	0.20	0.19	0.32	0.59
78	0.27	0.29	0.28	0.43	0.65
79	0.24	0.28	0.26	0.35	0.74
80	0.24	0.22	0.23	0.41	0.56
81	0.21	0.27	0.24	0.35	0.69
83	0.19	0.49	0.33	0.31	1.06
84	0.34	0.36	0.35	0.80	0.44
85	0.30	0.32	0.31	0.40	0.78
86	0.28	0.40	0.34	0.76	0.45
87	0.47	0.36	0.42	0.59	0.71
88	0.26	0.22	0.24	0.47	0.51
89	0.20	0.26	0.23	0.30	0.77
90	0.21	0.25	0.23	0.43	0.53
91	0.20	0.29	0.24	0.28	0.86
92	0.26	0.27	0.27	0.39	0.69
93	0.25	0.25	0.25	0.35	0.71
94	0.19	0.18	0.19	0.29	0.66
95	0.22	0.22	0.22	0.28	0.79
96	0.20	0.25	0.22	0.36	0.61
98	0.20	0.24	0.22	0.38	0.58
99	0.20	0.22	0.21	0.43	0.49
101	0.29	0.25	0.27	0.32	0.84
102	0.22	0.30	0.26	0.25	1.04
103	0.18	0.22	0.20	0.33	0.61
104	0.17	0.18	0.18	0.38	0.47
105	**	0.23	0.23*	0.33	0.70
106	0.21	0.26	0.24	0.32	0.75

1 Mean of all samples taken in the bedroom and playroom
2 Ratio of mean internal air lead concentration to mean external air lead concentration
* Playroom only
** Missing data

Figures 5.10-5.12 Frequency distributions of bedroom, playroom and external air lead concentrations







5.4 HANDWIPES

The results from the analyses of handwipes from each child for each day of the study week are presented in Table 5.5. These data showed a log-normal distribution (Figure 5.13). The geometric mean (5th-95th percentile) hand lead level (total for both hands) was 5.7 μg (1.9-15.1 μg). The data from the analysis of the samplers' handwipes are given in Appendix E.

5.5 FOOD AND WATER

Unfortunately, it has not been possible to present the raw data from the dietary section of the study in this thesis. However, a summary of the results is presented in Table 5.6. The geometric mean (5th-95th percentile) lead concentration in the diet (i.e. food and beverages) was 0.025 μg/g (<0.02-0.06 μg/g) while that for water was 0.019 mg/l (<0.005-0.100 mg/l). The mean lead intake from the diet was 161 μg/week (82-389 μg/week).

5.6 BLOOD

The results from both the first sampling programme, carried out in September and October, 1984 and the second programme, carried out in March, 1985 are presented in Table 5.7 and Figures 5.14 and 5.15. The geometric mean (5th-95th percentile) blood lead concentration for the whole study population, i.e. using the results from the first sampling programme, was 0.56 umol/l (0.24-1.15 umol/l) while that for the 56 repeat samples was 0.59 umol/l (0.34-1.02 umol/l).

The result from one child has been omitted from Table 5.7. Child number 3 was found to have a very high blood lead level, 2.14 umol/l and was referred to the

Table 5.5 Hand lead: µg total both hands

CHILD NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	8	MEAN
1	*	11.7	13.9	15.6	9.1	14.7	7.1	47.5	17.1
2	6.9	4.9	*	3.9	6.7	6.1	5.9	4.1	5.5
3	3.7	1.7	7.9	6.8	3.5	3.5	4.6	4.3	4.5
4	14.3	4.6	8.4	2.6	5.4	7.1	*	7.0	7.1
5	7.3	3.9	*	2.1	*	2.8	13.3	*	5.9
6	16.1	7.0	5.5	6.2	4.2	6.0	8.1	6.4	7.4
7	*	11.6	6.1	10.5	7.1	9.3	9.3	9.8	9.1
8	23.9	14.4	10.8	13.5	13.2	10.4	20.3	8.2	14.3
10	5.1	2.2	2.6	4.3	3.0	3.6	3.8	*	3.5
11	*	*	6.7	4.8	3.5	4.6	2.1	8.0	5.0
12	*	3.6	5.0	6.4	5.7	6.9	6.9	4.5	5.6
13	*	6.7	5.9	6.5	8.8	8.9	*	12.4	8.2
14	8.6	<1.2	7.7	2.9	<1.2	6.7	10.4	8.6	5.9
15	*	5.8	6.0	3.7	6.6	5.4	*	7.4	5.8
16	*	2.2	7.7	4.9	4.1	13.0	2.0	5.6	5.6
17	7.9	1.7	5.7	5.0	1.6	*	4.1	4.5	4.4
18	*	6.1	5.5	5.4	4.5	6.8	*	9.0	6.2
19	*	4.9	6.5	4.3	4.7	5.0	6.3	5.7	5.3
20	15.6	8.2	2.6	8.8	9.6	7.2	*	6.9	8.4
21	3.7	6.3	2.2	*	4.6	3.2	9.2	*	4.3
22	12.7	*	2.0	3.9	*	5.1	*	1.9	5.1
23	16.7	*	9.7	6.5	6.0	5.4	9.6	10.4	9.2
24	4.9	4.9	3.7	5.0	3.1	2.8	5.3	6.9	4.6
25	7.7	4.3	*	3.9	7.7	*	3.8	4.0	5.2
26	*	8.6	*	3.9	<1.2	3.6	7.8	5.2	5.1
27	*	12.6	5.6	5.4	6.4	6.3	8.2	11.6	8.0
28	23.2	7.5	4.0	4.2	3.8	5.2	3.0	3.7	6.8
29	*	9.8	9.3	11.5	9.2	15.2	7.6	9.3	10.3
30	5.5	3.8	4.4	4.4	1.7	2.7	5.1	*	3.9
31	6.1	4.2	6.9	5.5	3.1	7.1	9.4	3.3	5.7
32	6.1	5.9	6.5	5.3	5.6	6.4	13.4	5.4	6.8
33	11.6	7.8	2.4	13.1	6.9	9.0	12.1	5.0	8.5
34	4.3	2.0	2.7	4.2	3.4	3.6	5.5	4.6	3.8
35	6.4	2.6	4.3	8.4	4.8	4.7	*	4.9	5.2
36	3.8	3.1	9.7	5.4	6.2	6.5	8.4	2.7	5.7
37	15.5	7.7	5.9	8.2	19.0	7.5	8.2	13.1	10.6
38	5.2	2.1	1.4	2.4	4.2	2.7	4.9	4.1	3.4
39	5.6	3.7	5.0	2.2	4.1	1.6	2.8	3.2	3.5
40	2.1	1.6	4.1	2.3	2.7	1.2	<1.2	2.9	2.3
42	15.4	8.0	4.3	7.6	12.1	11.3	11.4	8.5	9.8
43	*	5.2	9.1	6.4	5.1	5.0	5.0	3.7	5.6
44	*	7.3	7.9	7.4	10.2	5.4	15.8	8.0	8.9
45	*	92.8	14.7	57.2	13.6	24.1	9.6	8.1	31.4
46	10.5	5.8	5.8	9.9	9.0	11.7	12.1	6.3	8.9
47	5.7	4.7	4.6	5.3	5.5	4.3	9.8	5.8	5.7
48	7.4	3.9	8.8	3.1	10.1	7.1	5.2	4.1	6.2
49	15.2	7.1	6.6	10.2	8.3	11.2	9.9	10.2	9.8
50	*	6.7	3.5	9.3	5.7	10.4	10.6	7.5	7.7
51	6.6	4.4	3.1	4.0	5.2	2.6	2.6	4.5	4.1

contd./

Table 5.5 contd.

CHILD NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	8	MEAN
52	2.8	2.7	7.0	<1.2	1.8	3.5	2.4	1.4	2.9
53	3.1	3.0	4.3	2.0	<1.2	4.4	3.4	1.9	2.9
54	3.7	3.2	4.5	3.9	4.3	1.9	1.6	5.1	3.7
56	6.0	2.3	1.6	2.7	4.6	4.0	9.4	5.1	4.5
58	9.4	5.2	7.1	3.2	5.0	4.5	10.8	9.4	6.8
59	5.6	5.3	2.1	2.2	2.5	2.1	5.1	4.4	3.7
60	*	9.4	4.0	2.6	3.4	3.3	2.6	2.8	4.0
62	10.5	*	8.8	5.2	5.3	4.2	2.9	5.1	6.0
63	8.6	5.7	3.9	3.2	7.1	3.3	6.2	6.0	5.5
64	5.1	4.4	5.9	6.0	6.2	9.7	8.4	7.1	6.6
65	5.5	4.8	6.3	6.3	4.5	9.8	10.5	6.2	6.7
66	1.8	1.7	<1.2	2.9	3.6	2.7	3.3	2.9	2.7
67	4.7	1.3	3.1	4.3	<1.2	*	*	*	3.4
68	8.3	1.4	1.6	4.2	3.8	*	2.4	7.0	3.7
70	2.3	4.3	2.6	5.7	3.8	1.7	1.5	7.9	3.7
71	*	2.2	3.0	3.3	5.6	2.5	2.4	3.1	3.2
72	10.3	13.4	10.8	13.0	11.1	13.0	15.2	10.6	12.2
73	*	9.0	9.5	<1.2	7.4	3.1	8.5	7.3	6.6
74	7.7	6.3	7.9	7.8	2.4	2.7	3.3	4.3	5.3
75	*	6.7	7.2	7.1	3.6	4.3	9.8	9.4	6.9
76	13.3	4.2	5.4	9.1	13.6	5.4	8.4	8.3	8.5
78	*	12.1	4.7	11.4	11.3	8.9	23.6	11.6	11.9
79	11.0	2.8	2.9	6.6	2.5	8.7	6.4	38.0	9.9
80	7.0	3.4	*	9.1	13.6	6.5	*	4.0	7.3
81	20.6	6.5	10.5	16.3	8.9	6.7	7.3	3.3	10.0
82	6.3	*	*	*	3.8	*	11.3	8.0	8.5
83	3.7	5.9	13.7	7.0	8.1	6.4	10.2	16.9	9.0
84	12.2	8.5	5.2	8.2	23.4	6.8	4.4	<1.2	8.7
85	11.0	<1.2	10.9	1.9	8.0	12.0	5.6	14.2	8.1
86	15.1	8.3	7.9	4.4	13.9	8.9	8.8	7.7	9.4
87	12.3	9.9	3.9	12.7	6.9	3.6	6.0	7.9	7.9
88	12.2	11.1	10.1	3.3	9.1	3.0	5.9	4.1	7.4
89	16.9	8.4	10.7	5.4	<1.2	11.3	8.2	9.4	8.9
90	*	8.0	2.8	6.5	12.0	6.4	7.2	8.0	7.3
91	19.1	18.7	13.0	18.4	14.4	6.4	16.2	11.8	14.8
92	*	4.2	13.6	11.5	8.1	406.1	4.7	5.9	65.0
93	3.5	*	4.7	8.2	7.0	10.4	*	4.7	6.4
94	12.9	6.2	9.4	11.5	11.1	6.3	7.0	9.5	9.2
95	*	17.1	11.4	6.9	9.8	7.4	5.6	6.3	9.2
96	4.4	7.9	4.6	3.2	6.6	1.8	2.1	3.4	4.3
98	4.3	2.8	2.5	3.6	4.2	3.1	3.3	2.4	3.3
99	5.0	2.7	2.2	9.5	2.5	2.0	4.3	1.6	3.7
100	3.3	7.7	7.7	4.6	18.3	3.7	3.7	1.6	6.3
101	*	6.0	7.3	13.0	1.4	*	<1.2	4.0	5.6
102	10.5	5.7	9.3	5.6	5.1	*	4.5	6.4	6.7
103	11.2	5.9	4.7	3.1	6.8	*	3.2	3.9	5.5
104	8.7	4.3	5.8	2.3	*	3.1	1.8	9.7	5.1
105	19.1	10.4	11.1	38.8	*	*	39.7	13.1	22.0
106	*	3.4	11.2	*	5.4	8.8	5.2	*	6.8

* Missing data

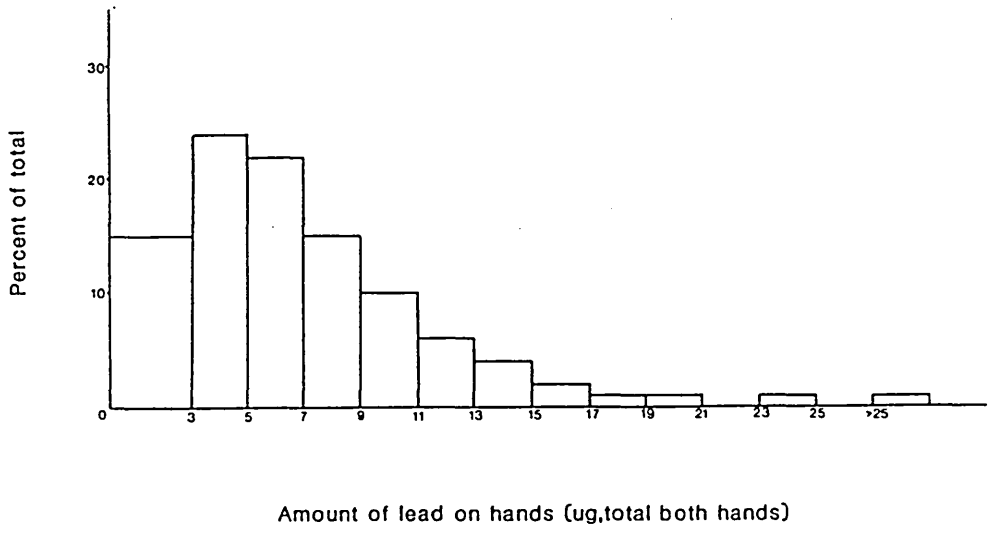


Figure 5.13 Frequency distribution of hand lead level

Table 5.6 Summary of dietary and water lead levels and weekly lead intakes for the study population

	N	A.M.	RANGE	G.M.	PERCENTILE 5th	PERCENTILE 95th
LEAD CONC.:						
Diet[1] (µg/g)	97	0.028	<0.02-0.16	0.025	<0.02	0.06
Water (mg/l)	97	0.029	<0.005-0.14	0.019	<0.005	0.100
LEAD INTAKE:						
Diet[1] (µg/wk)	97	185	62-1104	161	82	389

N = number of samples
Diet[1] = food and beverages
A.M. = arithmetic mean
G.M. = geometric mean

Table 5.7 Blood lead concentration (µmol/l) from first (Pb.B 1) and second (Pb.B 2) samples

CHILD	PB.B 1	PB.B 2	CHILD	PB.B 1	PB.B 2	CHILD	PB.B 1	PB.B 2
1	0.66	0.71	35	0.23	*	72	0.43	0.41
2	0.42	*	36	0.46	0.52	73	0.76	0.95
4	1.23	1.10	37	0.68	0.61	74	0.35	*
5	0.60	*	38	0.57	0.57	75	0.27	*
6	0.87	*	39	0.34	*	76	0.56	0.56
7	1.04	1.01	40	0.17	0.29	78	0.82	*
8	1.39	0.96	42	0.34	0.80	79	0.60	0.64
10	0.50	*	43	0.42	0.57	80	0.46	0.46
11	0.88	0.82	44	1.48	1.00	81	0.81	0.65
12	0.36	0.33	45	0.70	*	82	0.71	*
13	0.92	*	46	0.66	0.63	83	0.34	0.37
14	0.48	0.56	47	0.29	0.36	84	0.36	*
15	0.90	0.84	48	0.56	0.68	85	0.55	0.61
16	0.79	*	49	0.60	*	86	0.47	0.41
17	1.44	1.48	50	0.55	*	87	0.88	0.74
18	0.34	0.46	51	0.60	0.67	88	0.34	0.37
19	0.83	0.84	52	0.60	0.60	89	0.99	*
20	0.61	0.60	53	0.29	*	90	0.41	0.52
21	0.56	*	54	0.36	*	91	0.74	0.74
22	0.76	*	56	0.32	0.34	92	0.82	0.74
23	0.63	*	58	0.20	0.38	93	0.57	0.59
24	0.38	*	59	0.32	*	94	0.77	*
25	0.57	0.48	60	0.50	*	95	1.17	*
26	0.37	0.40	62	0.42	0.46	96	0.89	0.81
27	0.88	*	63	0.92	*	98	0.38	0.39
28	0.35	0.40	64	0.78	*	99	0.44	*
29	1.04	*	65	0.89	*	100	0.45	0.42
30	0.32	*	66	0.38	*	101	0.41	0.43
31	0.56	0.67	67	1.02	*	102	0.63	*
32	0.37	*	68	0.73	0.68	103	0.80	0.68
33	1.03	0.75	70	0.63	0.63	104	0.45	*
34	0.43	*	71	0.63	0.63	105	0.55	1.01
						106	0.40	*

Number of samples	97	56
Arithmetic mean	0.62	0.63
Minimum	0.17	0.29
Maximum	1.48	1.48
Geometric mean	0.56	0.59
5th percentile	0.24	0.34
95th percentile	1.15	1.02

* Missing data

Conversion Factor: 1 µmol/l = 20.7 µg/dl

Figure 5.14 Frequency distribution of blood lead concentrations from first sampling programme

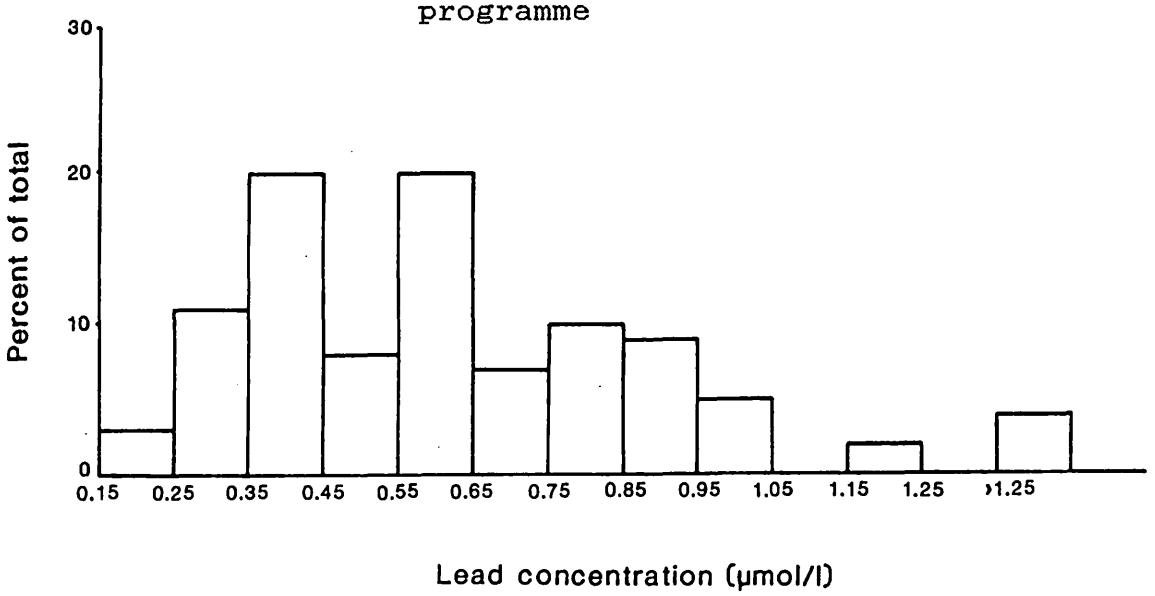
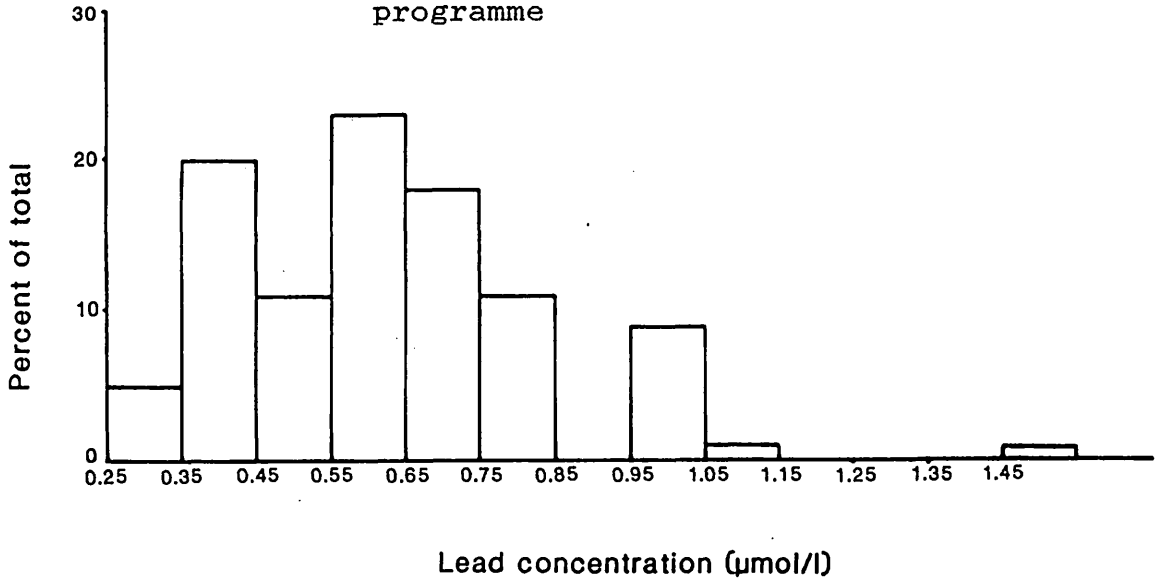


Figure 5.15 Frequency distribution of blood lead concentrations from second sampling programme



Birmingham Children's Hospital for further investigation. If this 'outlier' is included in the data set the geometric mean (5th-95th percentile) is 0.57 umol/l (0.29-1.24 umol/l).

5.7 HAND-TO-MOUTH ACTIVITY

The final data set generated by the rating of video tapes was extremely large (97 subjects, 4 settings [play, book, meal, TV], 78 behavioural categories [36 mouthing, 21 for each hand] and 2 behavioural dimensions [frequency and duration] giving a matrix of 60528 cells) and was therefore reduced to a more manageable size, in the following manner. For the mouthing behaviour, five major categories were derived to include fixed objects (large static objects in the environment, e.g. furniture), mobile objects (any non-static object ranging from toys to prams), hands at the mouth (all or a part of the hand in or around the mouth area) and feeding (any food, drink or eating/drinking-related object). A sub-grouping of behaviours of hands in the mouth was subsumed under the third category to give more detailed information on this aspect. A similar procedure was followed for the hands. The behaviours were summed for both hands to give a single figure for what the hands touched. The major categories were again fixed and mobile objects together with a third category of flooring (this category was also subsumed under the fixed category). The actual figures in these categories are presented as rates. That is, each behaviour total frequency figure is divided by the time period for which the observation was made (in seconds) and this product then multiplied by 60 to give the frequency of behaviour per minute. The mean rates of mouthing behaviour are shown in Table 5.8 and those of hand contact in Table 5.9. Data for each subject for a variety of mouthing and hand contact behaviors are presented in Appendix F.

Table 5.8 Mean rates of mouthing behaviours (behaviours per minute)

Setting	Collapsed categories				
	Fixed	Moveable	Feeding	Hand in mouth	Hand at mouth area[1]
Play	0.02	0.45	0.34	0.13	0.44
Book	<0.01	0.19	0.17	0.36	0.56
Meal	0.10	0.24	3.56	0.11	1.53
TV	0.03	0.26	0.16	0.31	0.54
Mean	0.02	0.29	1.05	0.23	0.76

[1] The "Hand at mouth area" category includes the more specific "Hand in mouth" category.

Table 5.9 Mean rates of hand contact (behaviours per minute)

Setting	Collapsed categories		
	Moveable	Fixed[1]	Floor
Play	14.29	3.86	1.17
Book	16.81	0.65	0.10
Meal	6.45	3.22	0.12
TV	10.03	4.78	1.85
Mean	11.92	3.13	0.81

[1] The fixed category includes the flooring category.

5.8 QUALITY CONTROL

The overall quality control results from the analysis of reference materials for the environmental samples (dust, soil, air filters and handwipes) have been

summarised in Table 5.10. It can be seen from this Table that, in general, accuracies were less than 5 per cent and relative standard deviations were less than 10 per cent in all cases. Precisions on the duplicate soil and dust analyses, assessed by the use of precision control charts (Thompson and Howarth, 1976), were found to be around 10 and 40 per cent, respectively (95 per cent confidence limits). For all instrumental batch runs, blank corrections were made when necessary.

Accuracy and precision of blood lead analyses were reported to be satisfactory (Delves, personal communication). In addition to the analysis by MSF-AAS, 55 samples re-analysed by ETA-AAS showed a mean difference in results of +0.46 µg/dl (0.02 µmol/l) with 94.5% of results agreeing within 2 µg/dl (0.10 µmol/l). The maximum difference was +2.9 µ/dl (0.14 µmol/l). Accuracy and precision of food/beverages and water analyses were reported to be adequate (Smart et al., 1988).

5.9 QUESTIONNAIRE

Firstly, the data on lead levels from the environmental and blood samples were partitioned into a number of subgroups identified from the questionnaire responses. Intra-parameter comparisons were then carried out on the subgroups by means of analysis of variance and t-tests and the results from this exercise have been summarised in Table 5.11. Only the subgroups showing statistically significant differences have been included.

Table 5.10 Overall quality control results for dust, soil, air filters and handwipes from the analysis of reference materials

	DUSTS AND SOILS Concentration (µg/g)			AIR FILTERS Conc. (µg/g)		HANDWIPES Weight (µg)			
REFERENCE MATERIAL	HRM 1	HRM 2	HRM 7	HRM 9	HRM 10	HRM 7	HRM 8	HRM 9	NBSRM
Number of samples	22	22	22	117	117	69	34	34	12
Established value	13.0	510.0	1590.0	2.50	1.00	79.5	10.0	50.0	66.4
Observed mean	13.5	500.0	1628.6	2.38	1.00	91.3	10.0	49.5	65.6
SD	1.1	17.3	40.6	0.18	0.06	3.5	0.6	1.5	2.8
RSD (%)[1]	8.2	3.5	2.5	7.6	6.0	3.8	6.0	3.0	4.3
Accuracy (%)[2]	3.8	2.0	2.4	4.8	0	14.8	0	1.0	1.2

1 Relative standard deviation (RSD) (%) = $\frac{\text{Standard deviation (SD)}}{\text{Observed mean}} \times 100$

2 Accuracy (%) = $\frac{\text{Established value - observed mean}}{\text{Established value}} \times 100$

Table 5.11 Lead levels in blood (PbB), internal air (PbAI), on hands (PbH), in housedust (concentration PbD, loading PbL), soil (PbS) and road dust (PbR) in a number of subgroups identified from questionnaire responses

QUESTION	PbB µmol/l	PbAI µg/m³	PbH µg	PbD µg/g	PbL µg/m²	PbS µg/g	PbR µg/g
HOUSE TYPE:							
Council?	-	0.27	4.8	289	-	-	-
Other?	-	0.32*	6.2*	478**	-	-	-
HOUSE TYPE:							
Rented?	-	-	5.0	313	-	-	-
Owned?	-	-	6.4*	510**	-	-	-
HOUSE AGE:							
< 35 years?	0.41	-	4.6	221	15.4	176	-
> 35 years?	0.60***	-	6.2*	498***	81.2***	342**	-
DECORATING:							
Currently?	0.68	-	-	547	-	-	750
Not current?	0.52**	-	-	393*	-	-	463**
PETS:							
One or more dogs?	-	0.34	-	-	-	-	-
No dogs?	-	0.29*	-	-	-	-	-
CAR MAINTENANCE:							
Carry out decarbs?	0.68	-	-	-	98.3	-	-
Don't carry out decarbs?	0.52**	-	-	-	49.3*	-	-
CLOSE PROXIMITY TO:							
Commercial garages?	0.61	-	-	506	92.1	365	-
No commercial garages?	0.49*	-	-	313**	26.8***	224**	-

contd./

Table 5.11 contd.

QUESTION	PbB µmol/l	PbAI µg/m³	PbH µg	PbD µg/g	PbL µg/m²	PbS µg/g	PbR µg/g
CLOSE PROXIMITY TO:							
Waste land, demolition sites, tips, etc.?	-	-	-	-	-	364	-
No waste land, demolition sites, tips, etc.?	-	-	-	-	-	267*	-
CLOSE PROXIMITY TO:							
Metal using industry?	-	-	-	-	-	407	-
No metal using industry?	-	-	-	-	-	248***	-
DISTANCE FROM ROAD:							
< 10 m?	-	-	-	-	78.0	389	-
> 10 m?	-	-	-	-	42.0*	216***	-
SMOKING HABITS:							
Smokers?	0.62	-	-	-	-	-	-
Non-smoker?	0.51*	-	-	-	-	-	-
MOUTHING:							
Exterior railings?	0.57	-	-	-	-	-	-
No exterior railings?	0.38*	-	-	-	-	-	-
HAND WASH:							
With running water?	0.49	-	-	-	-	-	-
Not with running water?	0.60*	-	-	-	-	-	-

Significance levels: * $p<0.05$; ** $p<0.01$; *** $p<0.001$
"-" Denotes no significant difference

6. THE BIRMINGHAM STUDY: DISCUSSION 1

This is the first of two chapters (Chapters 6 and 7) in which the Birmingham data are discussed. In this chapter, the results for each of the individual parameters measured (dust, soil, air, hand, blood, diet and water lead) are discussed separately and compared with the literature.

6.1 DUSTS

The results from the analysis of housedust, pavement and road dust samples are summarised in Table 6.1. Due to the log-normal distribution of the sample populations (Figures 5.1 to 5.6) the mean values referred to in the following discussion are geometric means. The highest mean lead concentration in dust from within the home, 615 μg/g, was noted in samples from under the doormat and was almost twice as high as those from the householder's vacuum cleaner, 336 μg/g (significant difference, $p<0.01$) and higher (although not significantly) than the mean from the bedroom and playroom samples, 430 μg/g. This is not a surprising result when one considers that, in urban areas, external dusts, generally, have higher lead concentrations than housedusts (Section 2.5) and that the doormat is the most likely location within a house for the removal of such external dusts. In the national reconnaissance study, the mean lead concentration in road dusts was 786 μg/g compared with 561 μg/g in housedust (excluding data from "geochemical hotspots", Culbard et al., 1988). From Table 6.1, it can be seen that the mean road dust concentration in Birmingham, 527 μg/g, is lower than the national average but again higher than the concentrations found in the bedroom, playroom and vacuum cleaner samples. Birmingham was one of the cities included in the national survey and the geometric mean housedust lead concentration

found in 1981/2 was 419 μg/g (n = 159). This figure is similar to the mean concentration found in the present study using the Imperial College sampler, 430 μg/g, although slightly higher than that noted for the householder's vacuum cleaner sample, 336 μg/g.

Table 6.1 Summary data for lead in housedust, soil, pavement and road dust

	N	AM	RANGE	GM	GSD	PERCENTILE 5th	PERCENTILE 95th
LEAD CONC. (μg/g)							
Doormat	42	1436	79-15000	615	3.30	120	4300
Vacuum[1]	92	713	46-23000	336	2.42	97	1440
Bedroom	96	693	53-4500	464	2.38	109	2040
Playroom	97	455	62-5600	311	2.08	105	1030
Average	95	578	111-3110	430	2.06	139	1780
Soil	86	405	56-1650	313	2.04	92	1160
Pavement	97	506	62-5100	360	2.12	127	1340
Road	97	805	80-2100	527	1.97	195	1170
LEAD LOADING (μg/m^2)							
Bedroom	96	141	1-1348	51	4.84	3	417
Playroom	96	150	1-1799	42	6.32	1	614
Average[2]	94	149	2-1062	62	4.43	4	486

1 From householder's own vacuum cleaner
2 Bedroom and playroom
GSD Geometric standard deviation

Other data pertaining to internal dusts in Birmingham do appear in the literature but are difficult to compare directly with the results from this study. For example, an extremely high median value of 3500 μg/g has been reported for 117 samples collected in 28 Birmingham houses during 1979/1980 (DoE, 1982). However, the houses

included in this study were selected because the children living in them had been found to have high blood lead concentrations and/or high erythrocyte protoporphyrin concentrations and cannot be considered as representative. In another Birmingham study, a high median lead concentration of 1000 μg/g was reported for 806 internal dust samples (Archer and Barratt, 1976). These, however, were not solely housedust samples but were collected from offices, shops and schools as well as houses in the area.

A somewhat unexpected result from the present study was that the mean lead concentration in pavement dust, 360 μg/g, was significantly lower than that found in road dust ($p<0.001$), even though the sampling sites were adjacent in all cases. These data suggest that there may have been a greater input of non-lead bearing dust and soil particles onto the pavement sites, perhaps by pedestrian movement, and/or that the processes of deposition and removal were operating at different rates in the two situations.

The concentrations of lead in residential road dusts in Birmingham have been measured previously. Archer and Barratt (1976) reported a median value of 950 μg/g for 85 such road dust samples collected between 1972 and 1975. The mean value found in this study, 527 μg/g (Table 6.1), is only around one half (56%) of this value. Although it is appreciated that, because the sample collection procedures and analytical techniques employed by these workers were different from those used in the present study, the data are not strictly comparable, the results do suggest that lead levels have decreased over the intervening period (mid-1970's to mid-1980's). A drop in industrial lead emissions could have resulted in such a finding but, unfortunately, it is difficult to confirm this as there is little information concerning changes in lead emissions from such sources over this period. There was a phased reduction in the lead content of petrol from 0.84 g/l in 1972 to 0.4 g/l in 1981, although national

figures indicate that the total lead emissions from petrol combustion remained fairly stable, despite this reduction, due to an increase in traffic levels (DoE, 1986). A further reduction, to a maximum of 0.15 g/l, came into effect at the beginning of 1986 and it will be of interest to continue monitoring dust lead levels to establish whether or not there is a corresponding decrease in these values.

In addition to the bedroom and playroom dust lead concentration figures in Table 6.1, dust lead loadings are also included. The geometric mean of the average bedroom and playroom lead loading in 94 homes was 62 $\mu g/m^2$. The few data in the literature on house dust lead loadings range widely. Similar results to those found in this study have been reported for 507 Edinburgh homes, where the median lead loading value was 45 $\mu g/m^2$ (Laxen et al., 1987) and for the Netherlands, where geometric mean lead loadings ranged from 30 to 81 $\mu g/m^2$ in 166 homes in four different areas (Brunekreef et al., 1983). A higher median value of 418 $\mu g/m^2$ was found in 65 homes in Christchurch, New Zealand (Fergusson and Schroeder, 1985) and a mean of 680 $\mu g/m^2$ has been reported for 12 homes in Champaign-Urbana, USA (Solomon and Hartford, 1976). A study of 15 London homes revealed an extremely high median lead loading of 3500 $\mu g/m^2$, although these properties were situated near to known lead sources (Turner et al., 1980).

The indication from pilot studies that the exposure of a child to lead in housedust is expressed more realistically by lead loading than simply lead concentration (Appendix A) is supported by data from the main study. For example, in house 12 the lead concentration in the bedroom is approximately 12 times as high as that in the playroom (1850 and 151 $\mu g/g$, respectively) while the lead loading is only around twice as high (117 and 50 $\mu g/m^2$, respectively, Table 5.1). Looking at concentration alone, therefore, could be

deceptive as the total mass of lead available for ingestion by a young child from housedust is not reflected in this figure. Although the lead concentration is high in the bedroom dust in this house, there is very little actual dust present and, therefore, relatively little lead to which the child is exposed. The usefulness of this index of exposure (lead loading) is explored further in the following chapter (Section 7.2).

The dust data can be further interpreted using information gained from the questionnaire. The relevant sections have been extracted from Table 5.11 and are shown in Table 6.2.

Newer properties (<35 years old) were found to have significantly lower levels of lead in housedust than older properties (>35 years old) in terms of both lead concentration (221 μg/g versus 498 μg/g, $p<0.001$) and lead loading (15 μg/m^2 versus 81 μg/g, $p<0.001$). These findings complement earlier observations from the reconnaissance study by Thornton et al. (1985) who suggested that this phenomenon may be due to voluntary reductions in the lead content of household paints over the past 40 years. If the property was being renovated/decorated at the time of sampling, the housedust lead concentration was significantly higher than if no decorating was taking place, geometric means being 547 μg/g and 393 μg/g, respectively ($p < 0.05$). This is in agreement with the findings of other workers who have reported significant elevation in household dust lead levels during home renovation work (Inskip and Atterbury, 1983; Inskip and Hutton, 1987; Laxen, 1987). One somewhat surprising finding which cannot be readily explained was that the road dust lead levels were also elevated outside such houses (Table 5.11).

Table 6.2 Lead levels in housedust and garden soil for a number of subgroups identified from questionnaire responses

	GEOMETRIC MEAN LEAD VALUE		
	HOUSEDUST		SOIL
	CONCENTRATION ($\mu g/g$)	LOADING ($\mu g/m^2$)	CONCENTRATION ($\mu g/g$)
House age <35y?			
Yes	221***	15***	176**
No	498	81	342
Decorating?			
Yes	547*	94	331
No	393	52	300
Near commercial garage?			
Yes	506**	92***	365**
No	313	27	224
Near waste land/ demolition site/ tip?			
Yes	443	80	364*
No	417	48	267
Near metal using industry?			
Yes	453	81	407***
No	408	47	248
House <10m from nearest road?			
Yes	481	78*	389***
No	361	42	216

Significance levels : * $p<0.05$
** $p<0.01$
*** $p<0.001$

Properties located within a 500 m radius of a commercial garage were found to have significantly higher housedust lead levels than those further away. The respective means were 506 μg/g and 313 μg/g ($p < 0.01$) in terms of concentrations and 92 μg/m² and 27 μg/m² ($p < 0.001$) in terms of loadings. This was also true for soil lead levels (Section 6.2) and while these results might suggest that commercial garages were acting as point sources for lead in the surrounding houses and gardens, no elevations in lead concentrations were noted in the road or pavement dust samples or in the external air samples obtained from alongside these houses (Table 5.11). This is surprising as the most likely transfer mechanism from such a source would inolve aerial transport and deposition.

Properties within 10 m of a road were found to have significantly higher lead loadings than those situated at a greater distance than this, respective means being 78 μg/m² and 42 μg/m² ($p < 0.05$). This was also the case for the soil lead levels (Section 6.2) and these data suggest that the roads could have been acting as "line" sources for lead in this situation. As with properties located near to commercial garages, however, there was no corresponding increase in air lead levels alongside these properties (Table 5.11).

In the light of the quality control results from the dust analyses (Section 5.8 and Table 5.10), a word of caution is necessary. Rarely are the results from quality control programmes published in the scientific literature. This is particularly so with the literature concerning lead levels in dust, reviewed earlier (Section 2.5). Where authors have reported the precision of their analyses, this has been based on replicate analyses of reference materials. Results of duplicate or replicate analysis of the environmental dust samples themselves are absent. Such an approach with the results from this study

would be somewhat misleading. For example, the relative standard deviation on the replicate analyses of the dust reference material, HRM 7, was 2.5%, which is an indication of excellent precision. However, this cannot provide a good estimate of the precision on the samples themselves, which overall was 40%. This poor precision is an unfortunate relection of the extremely heterogeneous nature of housedusts. Ideally, all samples should have been analysed in duplicate. However, with the very small sample weight of the sieved dusts from many of the thimble samples, such an approach would not have been possible. Looking at the precision of the duplicate analyses of the thimble samples alone, on which the lead loading calculations were based, the precision is somewhat better, 30%, although still poor. It is felt that the precision could be improved by sieving housedusts to a finer fraction than that used in this study, which was the < 1 mm fraction. While sieving to the < 1 mm fraction will remove much of the extraneous material which is present in housedust (pet hairs, carpet "fluff", etc.), it is likely that a certain amount of other material (organic debris such as skin, food particles, insects) will still be present and the heterogeneity will persist. Sieving to successively finer fractions should result in successively more homogeneous samples. It is clear that further work is necessary in this area.

6.2 SOIL

The results from the analysis of soil samples have been summarised in Table 6.1. The mean soil lead concentration found in the present study, 313 µg/g, was similar to that noted for Birmingham in the reconnaisance study, 278 µg/g (n = 159). Both of these values, however, are slightly higher than the national average, 266 µg/g (n = 4126). In another study of Birmingham garden soils, a high median value of 570 µg/g (n = 27) was found (DoE,

1982). These soils were sampled, though, because children associated with them had above average body burdens of lead and cannot be considered as representative samples.

The relevant sections regarding lead levels in garden soil for a number of subgroups identified from questionnaire responses have been extracted from Table 5.11 and are shown in Table 6.2. Older properties (> 35 years) were found to have significantly higher soil lead levels than newer properties (< 35 years), 342 μg/g compared with 176 μg/g, respectively. This finding is similar to observations in earlier studies and may, in part, be due to a longer history of soil contamination from bonfires and fossil fuel residues, often used as soil improvers and ameliorants, in the older properties. Also, there is likely to have been a greater accumulation of lead from deposited particulate matter (e.g. from industrial and vehicular emissions) in these gardens.

Properties located within a 500 m radius of a commercial garage were found to have significantly higher levels in garden soil than those further away (365 μg/g and 224 μg/g, respectively, $p<0.01$). As mentioned earlier (Section 6.1), this was also the case for housedust lead levels and although these results suggest that such garages were acting as point sources, there were no parallel elevations in lead concentrations in the road, pavement dust or air samples obtained from alongside these properties.

Elevated soil lead levels were also noted in gardens in close proximity to (within a 500 m radius of) waste land, demolition sites and tips, 364 μg/g compared with 267 μg/g ($p<0.05$) and those in close proximity to a metal using industry, 470 μg/g compared with 248 μg/g ($p<0.001$). The highly significant elevation in lead levels near to metal using industries is not too surprising and has been reported elsewhere (JURUE, 1982). Garden soils from

houses within 10 m of the nearest road were also found to have significantly higher lead concentrations than those from houses situated at a greater distance than this, respective means being 389 μg/g and 216 μg/g ($p<0.001$). This finding is in agreement with previous work which has shown that lead emitted by petrol-engined vehicles is usually deposited close to roads, resulting in elevated soil lead concentrations in narrow strips on each side of the road (Section 2.4).

6.3 AIR

The results from the analysis of air filter samples have been summarised in Table 6.3.

Table 6.3 Summary of internal and external air lead concentrations and internal:external concentration ratios

	AIR LEAD CONCENTRATION: μg/m³				RATIO
	BEDROOM	PLAYROOM	INTERNAL	EXTERNAL	INT/EXT
N	599	607	1206	605	90
Arith. mean	0.32	0.34	0.33	0.55	0.63
Minimum	<0.05	<0.05	<0.05	<0.05	0.39
Maximum	1.52	1.96	1.96	2.98	1.33
Geo. mean	0.26	0.27	0.26	0.43	0.61
5th %ile	0.09	0.08	0.08	0.12	0.42
95th %ile	0.81	0.88	0.87	1.53	0.87

The mean air lead concentration in the bedroom was 0.26 μg/m³ ($n = 599$) and similar (no statistically significant difference, $p > 0.05$) to that found in the playroom, 0.27 μg/m³ ($n = 607$). The mean external air lead concentration, 0.43 μg/m³ ($n = 605$), was

significantly higher than those found indoors ($p < 0.001$) although not as high as other figures published previously for outdoor air lead in Birmingham. In 1983/4 the city-wide annual average from 9 sites was reported to be 0.79 $\mu g/m^3$ (City of Birmingham Environmental Health Department, 1985). Archer and Barratt (1976) reported a median value of 0.75 $\mu g/m^3$ from 15 sites across the city in 1975, with average weekly means ranging from 0.4 to 1.4 $\mu g/m^3$. Still higher concentrations were recorded at a site alongside the Gravelly Hill motorway interchange during the same year and resulted in an annual mean (average of monthly means) of 1.79 $\mu g/m^3$ (DoE, 1978). These values can be compared with a five year average (1976 to 1981) of 0.65 $\mu g/m^3$ from five urban sites in England and Scotland (McInnes, 1982).

A total of 1811 air filters were successfully analysed and the results have been tabulated in full in Appendix D. Plots have also been included for each house showing the daily air lead concentration at each sampling site (bedroom, playroom and outdoors). With reference to this Appendix, it can be seen that most of the data for Houses 31-38 are missing. The loss of these samples was the unfortunate result of a malfunction in the heating block.

For the purposes of this discussion, selected examples have been drawn from Appendix D to illustrate the situation in individual houses and are presented in Figure 6.1. The general pattern that emerged is shown in Figures 6.1 (a), (b) and (c) where lead concentration has been plotted against sampling day for Houses 19, 29 and 30, respectively. There are two important points to note. Firstly, that the internal air lead concentration plots closely follow those for external air but are usually lower. This suggests that the external lead level has a large influence on the lead level found indoors and also suggests that the physical structure of the house (walls,

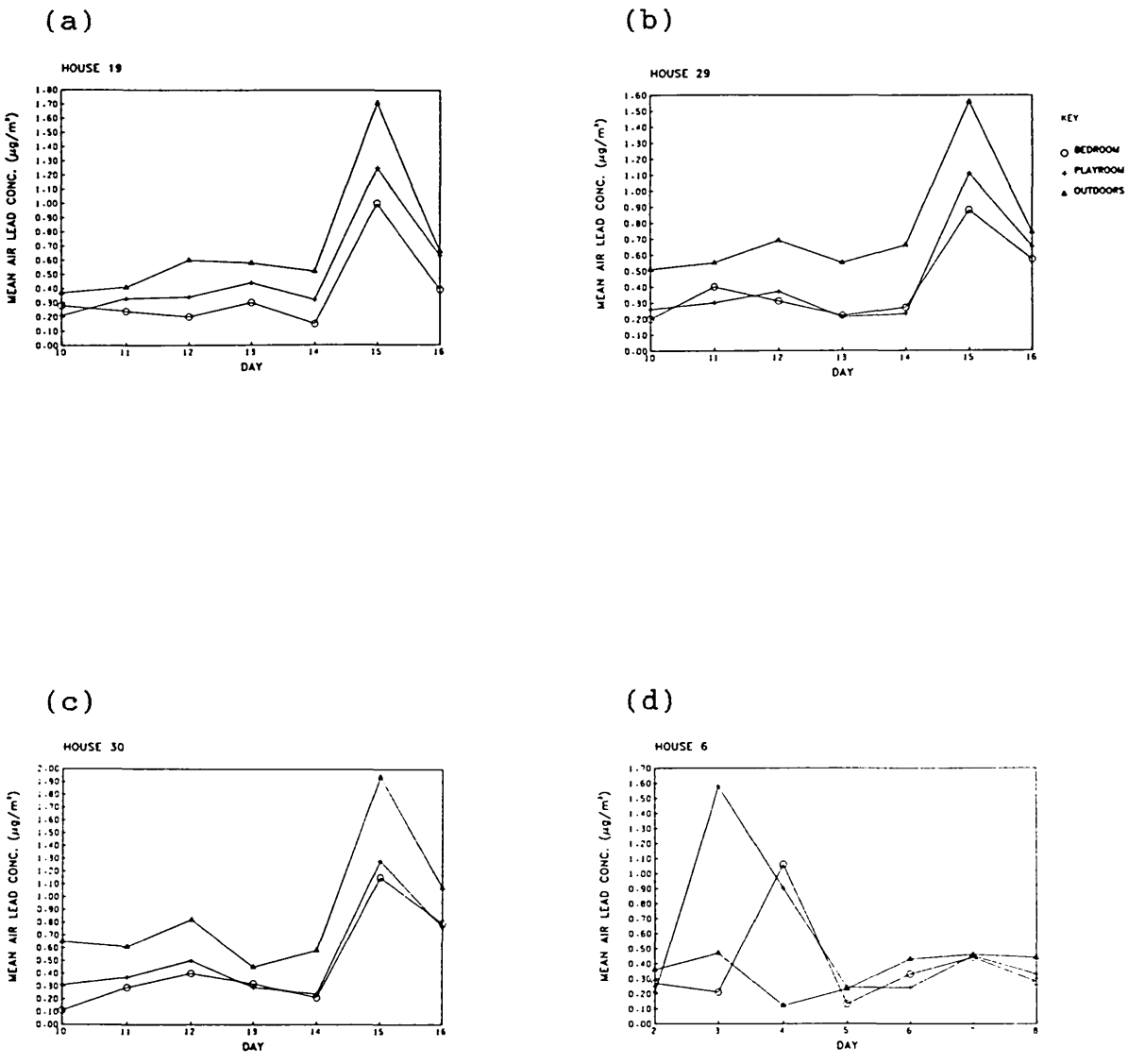


Figure 6.1 Bedroom, playroom and outdoor air lead concentrations for (a) House 19, (b) House 29, (c) House 30 and (d) House 6

doors, windows) prevents some of the lead-bearing suspended particulate matter reaching the indoor air. Secondly, that the plots for each house are very similar to one another, each showing for example, a small peak on day 12 and a larger peak on day 15. This suggests that the same air mass was being sampled, even though the houses were scattered throughout the study area. It is thus likely that meteorological factors, such as wind speed and direction or rainfall, were influencing the air lead concentration in the study area. The data from House 6 are shown in Figure 6.1 (d) and provide an interesting deviation from the norm. Here it can be seen that the internal air lead concentrations are very much higher than the external concentrations on days 3 and 4, which suggests that there was an internal source of lead contributing to the indoor air lead burden on these days. This property was being redecorated at the time of the study and old paint was being removed using a machine sander. It has been demonstrated elsewhere that stripping paint in this manner can result in large increases in environmental lead levels within a home. Inskip and Hutton (1987) found that air lead and deposited dust lead levels were highest after sanding although "burning off" and "hot air" paint removal methods also produced significant contamination. The dust lead concentration from this householder's vacuum was higher than in any other house sampled, at 23000 μg/g.

The internal:external air lead ratio was calculated for each house (Table 5.4) and revealed a mean (5th-95th percentile) of 0.61 (0.47-0.87) (Table 6.3). It is interesting, but not surprising, to find that the largest ratio, 1.33, occurred in House 6. There is not a vast literature on internal:external air lead ratios but from a review of the available data, Yocum (1982) suggested that ratios of 0.6 to 0.8 were typical for houses without air conditioning (none of the houses in this study was air conditioned). The few such studies carried out in the

United Kingdom have indicated, from limited data bases, ratios of between 0.6 and 0.7 (Lawther et al., 1973; Chamberlain et al., 1978; and DoE, 1978).

The mean internal air lead concentration has been plotted against the mean external air lead concentration for each house, and reveals a positive correlation (Figure 6.2 (a)). The regression equation was calculated by the least squares method to be:

Int. air Pb conc. = 0.096 + 0.42 (Ext. air Pb conc.) (I)

The regression line has been fitted to the data and is included in Figure 6.2 (a). The variance ratio (F) was 287.7 ($p < 0.001$, $R^2 = 76\%$) which provides evidence that the slope is probably not zero and implies that the weekly mean external air lead concentration is a useful predictor of the weekly mean indoor air lead concentration.

Although the data have been handled on the basis of individual houses above, it is also possible to pool the data from all sampling locations for individual days during the study period to gain information on average levels throughout the city on any given day. The results of such a treatment of the data are presented in Figure 6.3 (there are no results for days 17 to 42 due to the Christmas break). The relationship between external and internal levels, outlined above, is confirmed by this plot and the similarity between bedroom and playroom levels, already noted, is very apparent. Daily internal and external means have been compared in Figure 6.2 (b). The straight line regression equation was calculated from these data to be:-

Int. air Pb conc. = 0.055 + 0.50 (Ext. air Pb conc.) (II)

and the regression line has been included on this graph ($p<0.001$). The large R^2 value, 97%, indicates that the

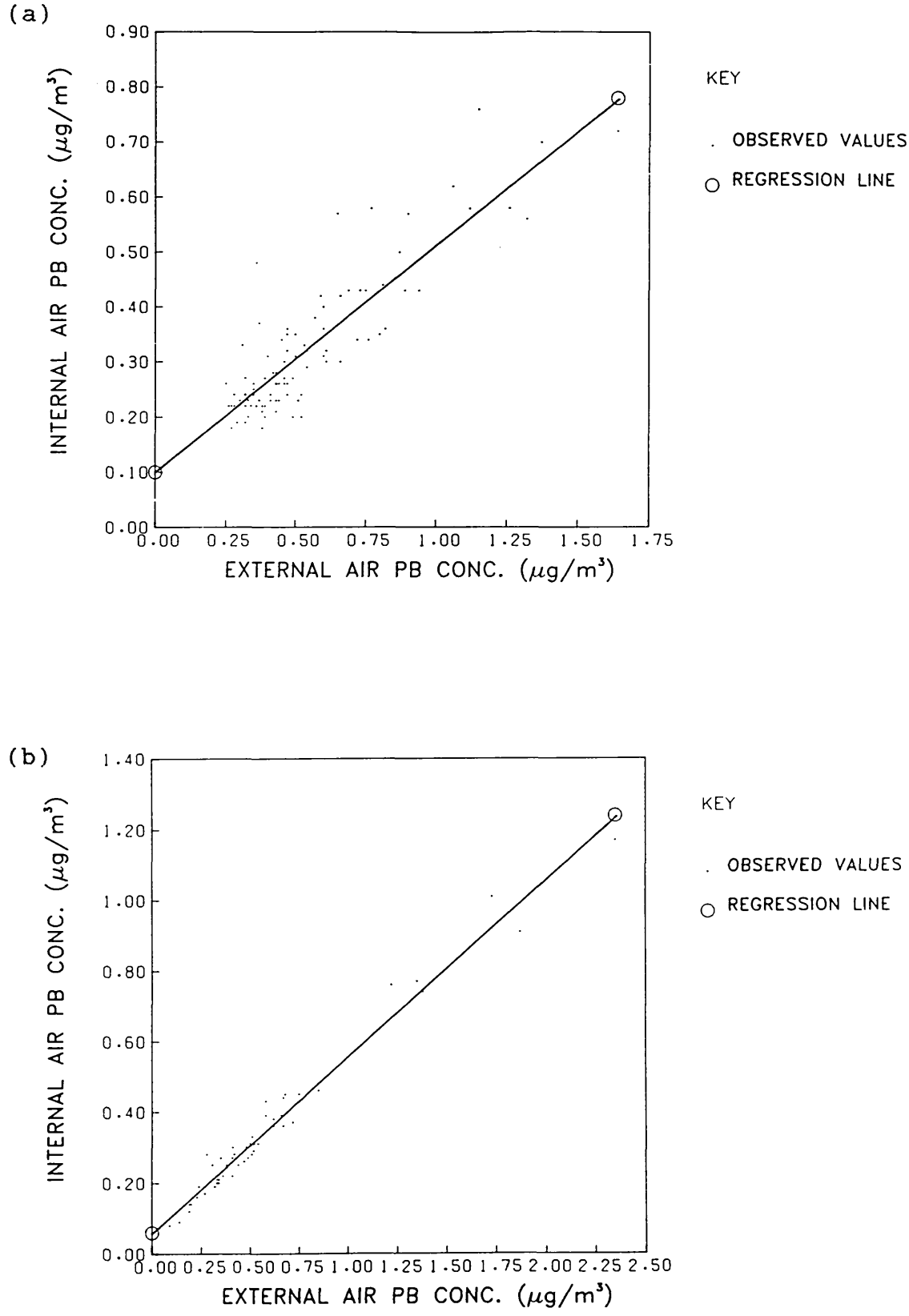


Figure 6.2 Mean internal vs mean external air lead concentration for (a) individual houses and (b) individual days

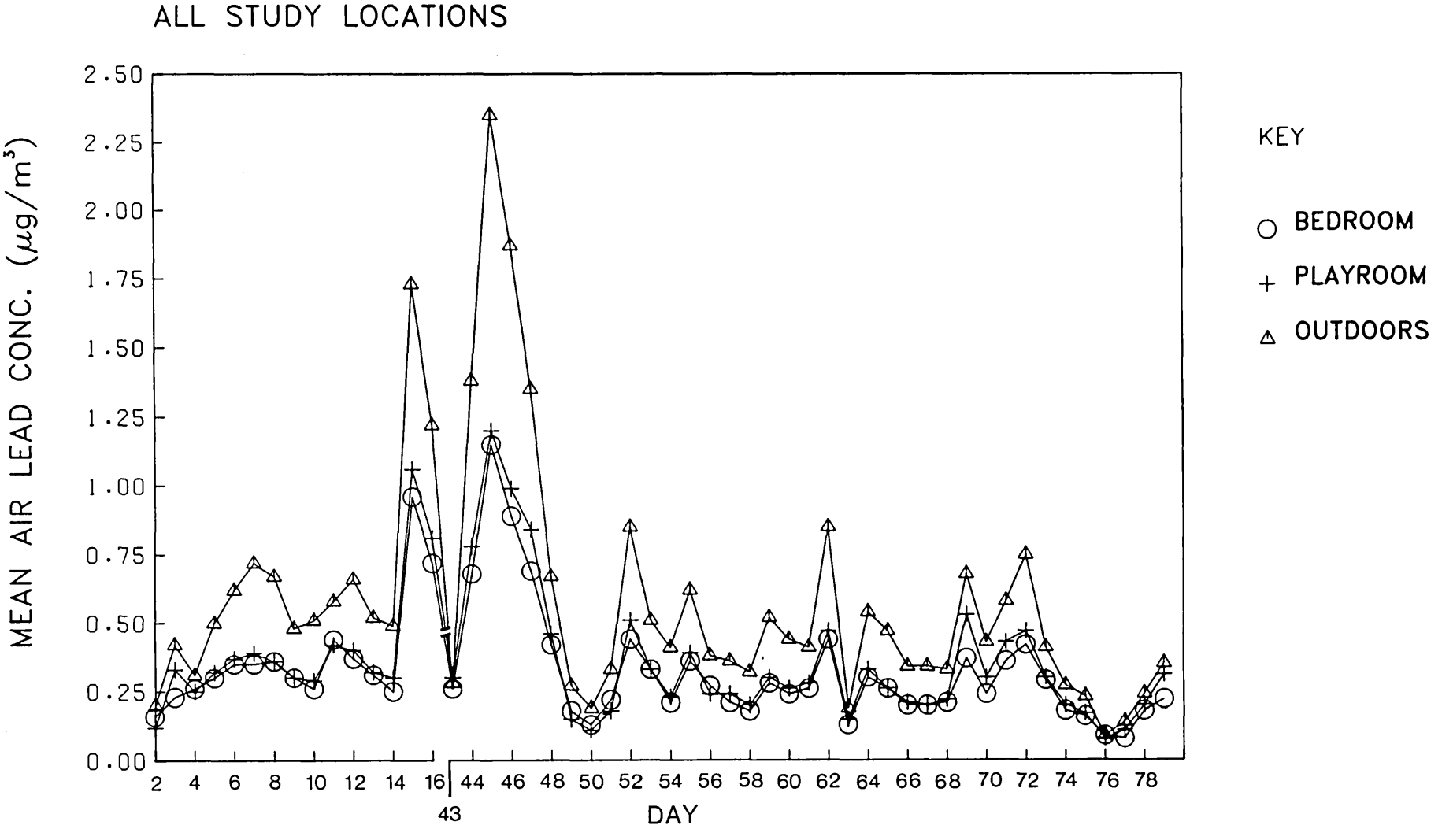


Figure 6.3 Mean bedroom, playroom and outdoor air lead concentrations from all sampling locations on each day of the study

mean daily external air lead concentration across the study area is an excellent predictor of the mean daily internal air lead concentration. This finding could have important implications in the design of future exposure assessment studies as it is cheaper and less complicated to implement an "outdoor only" air monitoring programme. The data generated could then be used to estimate indoor concentrations.

From the two regression lines fitted to the data (Figures 6.2 (a) and (b) and equations I and II) it can be seen that neither of the intercepts are zero. This indicates that if the external air lead concentration is zero there will still be lead in the internal air and suggests that there is another source (or other sources) contributing to the indoor air lead burden. It is felt that the most likely source is housedust (which this and other studies have shown to contain lead to varying degrees) which has been resuspended by the day to day activities of the householders and, where appropriate, their pets. In this context, the questionnaire responses revealed that households with one or more dogs had a slightly higher mean internal air lead concentration than those with no dogs, 0.34 $\mu g/m^3$ and 0.29 $\mu g/m^3$, respectively ($p<0.05$) (Table 5.11). This is not an unexpected finding and Brunekreef et al. (1983) have also shown a positive correlation between presence of pets and lead deposition indoors. There is no ready explanation, however, as to why council houses were found to have slightly lower lead levels than other types of property (Table 5.11).

6.4 HANDWIPES

The results from the analysis of each child's handwipe samples are presented in Table 5.5. If less than three wipes were obtained, the samples were discarded as

being incomplete. Data from the analysis of the samplers' handwipes are given in Appendix E. These samples were taken to indicate situations where the sampler had not cleaned his hands thoroughly enough before wiping the child's hands. If the lead level on a sampler's handwipe was greater than 10 μg, the result from the analysis of the child's wipes for that sampling session was omitted because of possible contamination from this source (this was the case for around 4% of the samples). This figure was felt to be appropriate in the light of recovery trials, which had indicated that around 70 per cent of the total removed with five wipes is recovered with the first wipe, and the fact that only part of the sampler's hand (finger tips) came into contact with the child's handwipe.

The geometric mean (5th-95th percentile) hand lead level (ug Pb, both hands) was 5.7 μg (1.9-15.1) and lower than others reported in the literature (Table 6.4). It should be noted, however, that a comparison of the results from this study with those from other investigations is difficult because several different sampling techniques have been used. Most workers have employed only one handwipe (Vostal et al., 1974; Charney et al., 1980; Roels et al., 1980; Gallacher et al., 1984). Duggan et al. (1985), however, used two, while Bornschein et al. (1986) used a total of six. Brunekreef et al. (1983) did not use handwipes at all, but rinsed the children's hands in slightly acidic water.

Bearing these difficulties in mind, the lower lead levels found in this study could be a reflection of the young age of the children investigated. Vostal et al. (1974) reported a median value of 20 μg Pb per hand for urban children aged 2-6 years old, while Roels et al. (1980) found a range of 13-20 μg per hand for an urban group aged 10-14 years old. Charney et al. (1980) reported a mean of 21 μg per hand for urban children aged 1-6 years old (with blood lead levels < 30 μg/100 ml),

Table 6.4 Summary data for lead on children's hands from a number of investigations

REFERENCE	LEAD ON HANDS IN µg/HAND	REMARKS
Vostal et al. 1974	20	Median in urban children (2-6 years)
	5	Median in suburban children (2-6 years)
Charney et al. 1980	49	Children with PbB 40-70 µg/dl (1-6 years)
	21	Children with PbB < 29 µg/dl (1-6 years)
Roels et al. 1980	244-436	School age children near smelter (10-14 years)
	13-20	Urban children (10-14 years)
Brunekreef et al. 1983	12	Inner-city children (4-6 years)
	5	Suburban children (4-6 years)
Gallacher et al. 1984	7.6	Roadside dwellings
	6.5	Cul de sac dwellings
	10.2	Old lead mining area
	7.1	Control village (all 1-3 years)
Duggan et al. 1985	6-22	Geometric means, urban school children (5-6 yr)
Bornschein et al. 1986	3.9	Geometric mean, urban children (1.5 years)
This study	2.8	Geometric mean, urban children (2 years)

Brunekreef et al. (1983), a mean of 12 μg per hand for inner-city children aged between 4 and 6 years old and in a study of 1-3 year olds living in roadside dwellings (near major roads in Wales), Gallacher et al. (1984) found a mean hand lead level of 7.6 ug. From a recent survey of 5 and 6 year old infant school children in London, Duggan et al. (1985) reported means ranging from 6-22 μg per hand. A mean hand lead level of 3.9 μg per hand, reported by Bornshein et al. (1986) for 18 month inner-city children, is, however, lower than others in the literature and closer to that found in this study (2.8 μg per hand).

Although the studies discussed above generated data on hand lead levels for children of different ages, in a variety of different settings, no information was obtained on the variability over time of hand lead for individual children. Such information is available from the results of this study, as the children's hands were wiped daily for eight consecutive days. In general, there was little day-to-day variation and selected examples have been drawn from the full data set (Table 5.5) to illustrate this point and these are presented in Table 6.5 . Hand lead levels for children 10, 24 and 72 were quite stable over the study period, as indicated by the raw data and the small standard deviations on the means. It is no surprise, however, that there were exceptions to this general consistency, as shown by the results for children 91 and 92. For child 91, the lead levels were relatively high over the study period except on day 6. For child 92, the results were erratic and it is not clear why such an extraordinarily high level of 406 μg of lead was present on one particular day, although "outliers" are to be expected in such a large data set (n = 712). A small paint flake or piece of solder adhering to the hands could have resulted in such a finding.

There are a number of factors likely to influence the amount of lead on a child's hands, which include the

Table 6.5 Hand lead levels (μg total both hands) over eight consecutive days

DAY	CHILD				
	10	24	72	91	92
1	5.1	4.9	10.3	19.1	*
2	2.2	4.9	13.4	18.7	4.2
3	2.6	3.7	10.8	13.0	13.6
4	4.3	5.0	13.0	18.4	11.5
5	3.0	3.1	11.1	14.4	8.1
6	3.6	2.8	13.0	6.4	406.1
7	3.8	5.3	15.2	16.2	4.7
8	*	6.9	10.6	11.8	5.9
Mean	3.5	4.6	12.2	14.8	64.9
SD	1.0	1.3	1.7	4.3	151

* Missing data

frequency of hand washing, housedust lead concentration and/or loading and the activity pattern of the child. The relationships between these factors and hand lead levels are discussed fully in the following chapter (Chapter 7).

6.5 FOOD AND WATER

The geometric mean (5th-95th percentile) concentration of lead in the duplicate diets (which included food and beverages) was 0.025 μg/g (<0.02-0.06 μg/g, n = 97). Weekly lead intakes were calculated for each child, from a knowledge of the individual dietary weights and lead concentrations, which revealed a geometric mean (5th-95th percentile) of 161 μg/week (82-389 μg/week, n = 97). This gives an estimated average daily intake of 23 μg/day. There have been few estimates of the dietary lead intake of young children in the UK

with which these results can be compared. The RCEP (1983) noted that, at the time (pre-1983), there were no direct data on the average dietary lead intake of this section of the population. Using results from the MAFF Total Diet Study for 1975-1981 (MAFF, 1982), which indicated that for adults the average lead intake from food and beverages was 100 μg/day, the Commission estimated the daily dietary lead intake of two year old children to be 47 ug. This was based on a ratio of the average energy requirements of adults and 2 year old children of 0.47 (DHSS, 1980) and on the assumption that the children's diet is composed of foods of similar lead content to those in the diet of adults.

More recently, a duplicate diet study has been carried out in London (Sherlock et al., 1985). Three study groups, Caucasian, Asian vegetarian and Asian non-vegetarian, each numbering around 35, were recruited from children in the age range of 2.5-5 years. The sample collection procedure was the same as that employed in the present study and analyses were carried out in the same laboratory (Laboratory of the Government Chemist). The mean concentrations of lead in the diet of the three groups were reported to be very similar, 0.023 μg/g, 0.020 μg/g and 0.021 μg/g, respectively. It would appear that these values are arithmetic means and they should, therefore, be compared with the arithmetic mean value from this study of 0.028 μg/g. The arithmetic mean weekly intake in this study was 185 μg, which is slightly higher than that reported by Sherlock et al. (1985) for Caucasian children, 150 μg/week, and significantly higher than those reported for Asian children, 110 μg/week for each group. It should be noted that there were no Asian children included in the present study.

Other summary data are available for younger children (<2 years) in the UK. A small duplicate diet study on 11 infants (<4 months) in Ayr, Scotland showed

that 80% of the children had intakes of < 3 mg/week and the maximum intake was 6 mg/week (Sherlock et al., 1982). A larger study (n = 110) of infants (13 weeks) in Glasgow revealed intakes ranging from 0.04 mg/week to 3.4 mg/week (DoE, 1982a). These ranges are broader than that from the present study (0.062 mg/week - 1.1 mg/week) but the data are difficult to compare directly because of the difference in the ages of the study populations. A large proportion of the children in both of these infant studies were being bottle-fed with reconstituted milk. It is likely, therefore, that the dietary lead content would have been largely influenced by the water lead content which was known to be high and to vary widely in these two Scottish cities. A recent duplicate diet study by Strehlow and Barltrop (1987) of five and six year old children living in London and a rural area of Suffolk, indicated mean daily intakes (food and water) ranging from 25 to 50 µg/day.

Other estimates of the dietary lead intake of young children from studies outside the UK are generally higher than that estimated in the present study. In a study of pre-school children in the US, Bander et al. (1983) estimated the daily dietary lead intake of 1-2 year olds to be 55 µg/day and of 2-3 year olds to be 56 µg/day. Again in the US, Podrebarac (1984) reported that the daily lead intake for toddlers was 29 µg in 1977 and 35 µg in 1978, while Biddle (1982) found intakes ranging from 28 to 46 µg/day for such children between 1975 and 1980. Johnson et al. (1981, 1984) estimated intakes ranging from 20 to 30 µg/day for infants and toddlers in the US and in an early study in the Netherlands, Reith et al. (1974) reported a dietary lead intake of 42 µg/day for 1 to 4 year old children. To summarise, estimates of the dietary lead intake of young children range from around 20 to 60 µg/day. The estimate from this study, 23 µg/day, falls at the lower end of this range.

The geometric mean (5th-95th percentile) water lead concentration was 19 μg/l (<5-100μg/l, n = 97). This is around twice the median value reported for tap water throughout Great Britain, from a nationwide survey in 1975-6, of 10 μg/l (DoE, 1977; RCEP, 1983). It is also higher than values recently reported for around 100 London households. Sherlock et al. (1985) found that two-thirds of the tap water samples contained less than 5 μg/l of lead, around 20% contained 5-10 μg/l and only 4% contained 20 μg/l. Strehlow and Barltrop (1987) have also recently reported low water lead concentrations in London. The geometric mean value from the present study, 19 μg/l, is, however, similar to the median found in a study of 495 homes in Edinburgh, 17 μg/l (Raab et al., 1987). In the Edinburgh study, a 30 minute stagnation sample of kitchen cold water was taken in each home rather than random daytime samples, which was the case in this study.

It has been shown in a number of studies that the lead content of water supplies can be extremely high where soft, acidic waters pass through lead plumbing systems (Section 2.6). This point is well illustrated by the results from two Scottish studies (Moore et al., 1985). In Glasgow, a high mean value of 244 μg/l (n = 232) was found and the maximum was reported to be 2000 μg/l. Even higher levels were noted in Ayr, where the maximum value was 2570 μg/l and the mean was 474 μg/l (n = 96). The pH of the water was low in both cities, ranging from 4.5 to 6.3 and it is reported that this, in combination with existing lead plumbing and also in conjunction with soldered joints in copper piping, resulted in these markedly elevated concentrations of lead in water (Moore et al., 1985). It is interesting to note that successful abatement of excessive exposure from these water supplies was achieved by the addition of lime. This raised the pH to 9.0 and two subsequent sampling programmes showed that the mean lead concentrations in Glasgow and Ayr had decreased to 13 μg/l (n = 292) and 51 μg/l (n = 96),

respectively. Returning to the results from the present study, those houses with lead plumbing were found to have significantly higher water lead concentrations than those that did not. The respective geometric means being 30 µg/l (n = 21) and 16 µg/l (n = 60) ($p<0.01$).

6.6 BLOOD

The results from the blood sampling programme, presented in full in Table 5.7, are summarised in Table 6.6.

Table 6.6 Summary of blood lead concentrations (µmol/l), from first (Pb.B1) and second (Pb.B2) samples

	PbB1	PbB2
Number of samples	97	56
Arithmetic mean	0.62	0.63
Minimum	0.17	0.29
Maximum	1.48	1.48
Geometric mean	0.56	0.59
Geometric s.d.	1.56	1.42
5th percentile	0.24	0.34
95th percentile	1.15	1.02

The geometric mean (5th-95th percentile) blood lead concentration of the 97 participating children was 0.56 µmol/l (11.7 µg/dl) [0.24-1.15 µmol/l, 5-24 µg/dl]. Results from an earlier study, by Harvey et al. (1984) of a random sample of 2.5 year old children also living in the inner-city area of Birmingham, revealed an arithmetic mean blood lead level of 0.75 µmol/l (15.6 µg/dl) [n = 187]. This is higher than the arithmetic mean from the present study, 0.62 µmol/l (12.9 µg/dl). An extremely high mean level of 1.23 µmol/l (25.6 µg/dl)[n = 85] was

reported from a study of 2 to 3 year olds in the same city during 1979 and 1980 (DoE, 1982). This result is difficult to compare with those cited above because capillary, rather than venous, blood samples were obtained. The validity of results from capillary blood sample analysis has recently been brought into question because of the ease with which such samples can be contaminated (Johnson et al., 1978; Angle and McIntire, 1979; Mahaffey et al., 1979; RCEP, 1983).

Other blood lead data are available for Birmingham children as a result of the UK Blood Lead Monitoring Programme, 1984-1987, sponsored by the DoE (1986,1987a). The children studied were, however, older than those in this study, aged between 6 and 7 years old, and all attended schools situated on busy major roads. In 1984, the geometric mean lead level was 0.48 µmol/l (10 µg/dl) [n = 247] while that for 1985 was 0.43 µmol/l (9 µg/dl) [n = 240]. Both of these values are lower than that noted in the present study (0.56 µmol/l, 11.7 µg/dl). This could be a reflection of the general relationship between blood lead concentration and age in children whereby lead concentration reaches a peak around the age of 2 or 3 years and subsequently declines (Section 3.2). Mean lead levels in other urban areas included in the programme in 1984 and 1985 (Bristol, Sheffield and Bexley/Greenwich, Brent and Waltham Forest in London) ranged from 0.42 to 0.53 µmol/l (8.8-11 µg/dl) (DoE, 1986, 1987a). Other recent studies in London have revealed a mean of 0.41 µmol/l (8.5 µg/dl) in two and a half to five year old children (Sherlock et al., 1985) and means ranging from 0.36 µmol/l (7.6 µg/dl) to around 0.40 µmol/l (8.3 µg/dl) in five and six year olds (Strehlow and Barltrop, 1987). A slightly higher median value of 0.48 µmol/l (10.1 ud/dl) was reported recently by Raab et al. (1987) in six to nine year old children from central Edinburgh.

Several studies have indicated that adult men tend to have significantly higher blood lead levels than adult women, while sex differences in young children are reported to be negligible (DoE, 1981, 1982, 1983, 1986, 1987a; Mahaffey et al., 1982). For example, the latest results available from the UK Blood Lead Monitoring Programme, those for 1985, show no statistically significant difference between levels in boys, geometric mean = 0.45 μmol/l (9.4 μg/dl) [n = 503] and those in girls, geometric mean = 0.43 μmol/l (9.0 μg/dl) [n = 504] (DoE, 1987a). Also, data from a large survey in the USA (Second National Health and Nutrition Examination Survey - NHANES II) showed that, although young boys, aged six months to five years old, had slightly higher blood lead levels than girls of the same age, the differences were not statistically significant (Mahaffey et al., 1982). Grouping the blood lead data from the present study by sex revealed geometric means of 0.53 μmol/l (11.0 μg/dl) [n = 39] and 0.58 μmol/l (12.1 μg/dl) [n = 58] for boys and girls, respectively. There was no statistically significant difference between the two groups, which is in agreement with the findings discussed above.

As was mentioned earlier, where possible, a second blood sample was obtained at the end of the study period. The geometric mean (5th-95th percentile) blood lead concentration from the second sampling programme, 0.59 μmol/l (12.3 μg/dl) [0.34-1.02 μmol/l, 7.1-21.3 μg/dl] was slightly higher than that from the initial programme, 0.56 μmol/l (11.7 μg/dl) [Table 6.6]. The difference, however, was not statistically significant. Data from the two programmes have been compared in Figure 6.4. The results reveal a remarkable degree of temporal stability (r = 0.82) which suggests that lead exposure remained fairly constant over the study period. This indicates that environmental, dietary and behavioural measures obtained over the period can be usefully compared with the blood lead data. With reference to Figure 6.4, it can be seen,

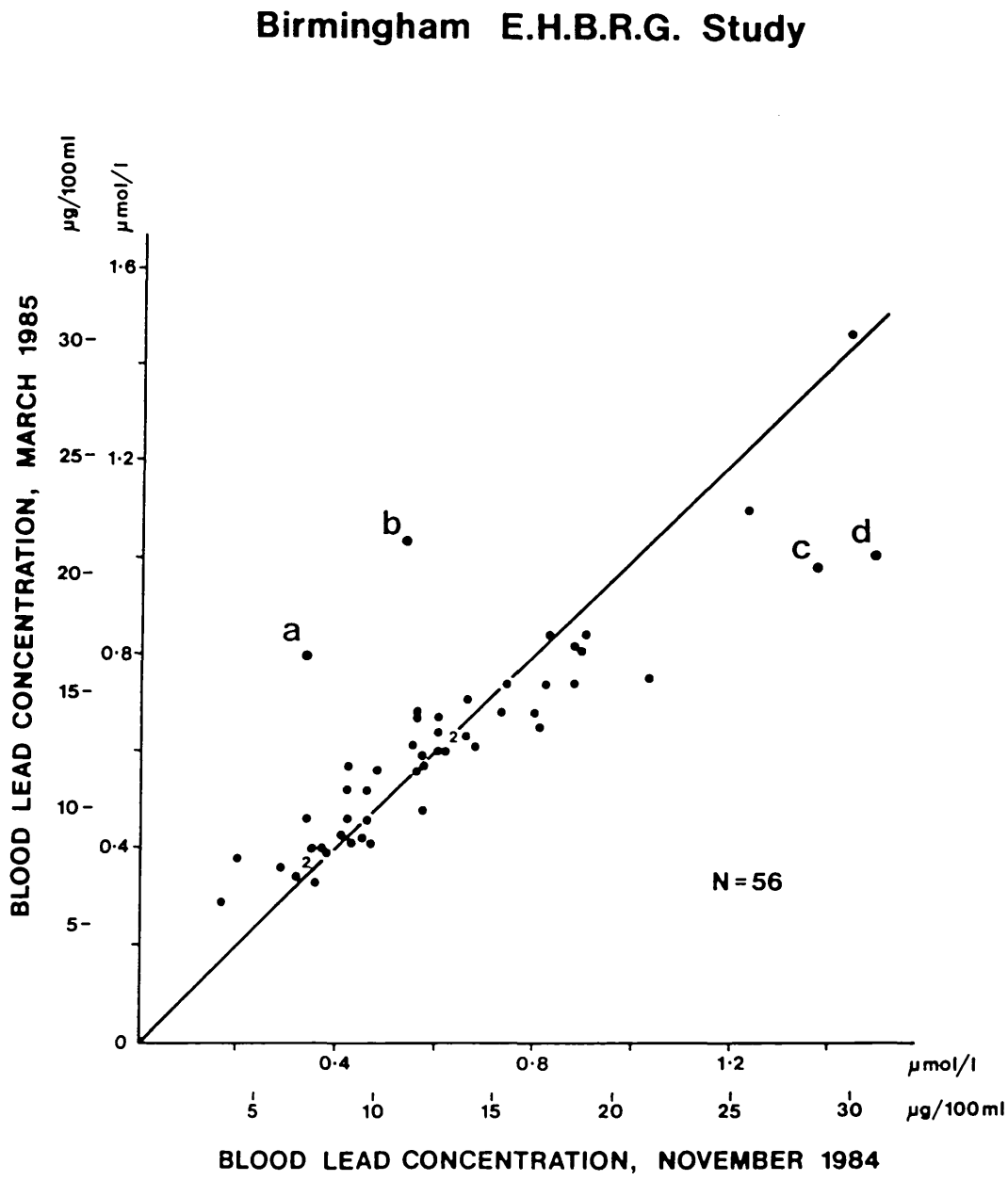


Figure 6.4 Comparison of blood lead concentrations from first and second sampling programme for 56 study children

however, that there were a few "outliers". It is interesting to note that the two children whose blood lead levels increased markedly over the period (a and b, Figure 6.4) had moved house just before the study. It is possible, therefore, that this could have resulted in a change in environmental lead exposure for these children, although to assume a causal relationship between this change and the change in blood lead level would be unwise. The two children whose blood lead levels decreased markedly (c and d, Figure 6.4) had not moved house.

The stability of blood lead concentration over time has also been evaluated in older children, aged around four to around eleven, by David et al. (1982). Samples were obtained from 29 children on four occasions, at one month intervals. The differences in the average blood lead concentrations were generally insignificant and correlations between measurements ranged from 0.72 to 0.81 and are similar to that from this study.

6.7 HAND-TO-MOUTH ACTIVITY

These data were supplied by Dr. Peter Harvey, Environment, Health and Behaviour Research Group, University of Birmingham, who has considered this aspect of the study in some detail elsewhere (Harvey et al., 1988). The results themselves present some difficulty in interpretation because there are no other data of this nature with which to compare them. From a methodological standpoint, it was shown to be practicable to obtain detailed, reliable and valid quantification of a specific aspect of a young child's behaviour in the home. A breakdown of data into different behavioural clusters and different settings gave a reasonably detailed picture.

An average taken over all behaviours and all settings indicates that something (objects or parts of the

body) touches the mouth area or goes into the mouth around two times per minute and that one or other hand touches something in the child's environment 16 times per minute.

7. THE BIRMINGHAM STUDY: DISCUSSION 2

7.1 INTRODUCTION

This is the second of two chapters (Chapters 6 and 7) in which the results from the Birmingham study are discussed. In the first section (Section 7.2), the relationships between the lead content of the various sample types is explored. The relative importance of the different exposure variables to blood lead is then assessed by means of multiple regression analysis (Section 7.3). In the final section (Section 7.4) a summary exposure assessment is completed for the study children.

7.2 RELATIONSHIPS BETWEEN BLOOD LEAD, ENVIRONMENTAL LEAD, DIETARY LEAD AND BEHAVIOURAL MEASURES: SIMPLE CORRELATION

Correlation coefficients between air lead, soil lead and all dust lead measurements are presented in Table 7.1. Given the number of locations sampled (n = 98[1]), a correlation coefficient of 0.20 would be required to achieve significance at the 5 per cent level, a correlation coefficient of 0.26 at the 1 per cent level and a correlation coefficient of 0.33 at the 0.1 per cent level.

With reference to this Table, there are a number of points of interest. As would be expected from other studies (Davies et al., 1985; Thornton et al., 1985; Davies and Thornton, 1987), soil lead concentration is significantly correlated with most measures of household

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1. The results from the environmental samples taken at the home of the one child with an elevated blood lead level have not been excluded here.

Table 7.1 Correlation coefficients for environmental lead measures (log-transformed data)

	DUST LEAD CONCENTRATION				DUST LEAD LOADING			LEAD CONCENTRATION			
	VAC	BED	PLAY	AV	BED	PLAY	AV	SOIL	PAVE	ROAD	EXT AIR
DUST LEAD CONC.-BED	.491[a]										
-PLAY	.081[a]	.308[b]									
-AV	.608[a]	.881[a]	.674[a]								
DUST LEAD LOAD -BED	.341[a]	.510[a]	.255	.481[a]							
-PLAY	.341[a]	.159	.566[a]	.350[a]	.603[a]						
-AV	.421[a]	.397[a]	.455[a]	.501[a]	.894[a]	.869[a]					
SOIL LEAD CONC.	.280[b]	.330[a]	.243[c]	.317[b]	.395[a]	.655[a]	.384[a]				
PAVE DUST LEAD CONC.	.910	.124	.088	.111	.144	.111	.122	.429[a]			
ROAD DUST LEAD CONC.	.035	-.043	.040	.027	.006	.043	.041	.036	.170		
EXT AIR LEAD CONC.	.000	-.029	.044	-.006	-.209[c]	-.198	-.190	-.283[b]	-.084	.028	
INT AIR LEAD CONC.	.184	.139	.230[c]	.180	-.007	-.020	-.010	-.090	.041	.004	.807[a]

VAC = Householder's own cleaner
BED = IC cleaner - bedroom
PLAY = IC cleaner - playroom
AV = Average of bedroom and playroom
PAVE = Pavement dust
ROAD = Road dust
EXT AIR = External air lead concentrations
INT AIR = Internal air lead concentrations

Significance levels:

a: $p < 0.1\%$
b: $p < 1\%$
c: $p < 5\%$

dust lead. It shows greater correlation with dust lead loading than with dust lead concentration. Soil and pavement dust are also significantly correlated (0.1%) and this may be indicative of a transfer of material between these two sites. Although there is a significant correlation (5%) between internal air lead concentration and dust lead concentration in the playroom and borderline significance with other dust concentration measures, there is virtually no correlation with dust lead loading figures. However, it is likely that if deposition rates had been measured then these would have been more closely correlated with dust lead loading than were the air lead concentration figures. There is no plausible explanation for the apparently significant negative correlation between either soil lead concentration and the external air lead concentration or dust lead loading and external air lead concentration. The significant correlation shown between most housedust measures is to be expected and the highly significant correlation between internal and external air lead concentrations has been discussed in detail earlier (Section 6.3)

The correlation coefficients for blood lead and a number of exposure variables are presented in Table 7.2 (n = 97). It should be noted here that all data relating to the one child with an elevated blood lead level (2.14 µmol/l, 44.6 µg/dl) have been excluded from these and subsequent statistical analyses. The study was designed to assess exposures of "typical" children and this child, who had a history of eating soil and a garden soil lead concentration of 1650 µg/g (maximum found in this study), was atypical. One "outlier" in the handwipe data set (406 µg from House 92, Day 6, see Section 6.4) has also been excluded for the purposes of these and subsequent analyses. This extraordinarily high value was not typical of the levels found on this child's hands on the other days (next highest value 13.6 µg, Day 3).

Table 7.2 Correlation coefficients for blood, dietary, water and selected environmental lead measures (log-transformed data)

	PbB	PbAI	PbAE	PbDC	PbDL	PbS	PbH	PbF
PbAI	-.069							
PbAE	-.147	.807[a]						
PbDC	.206[c]	.215[c]	.024					
PbDL	.463[a]	.020	-.165	.483[a]				
PbS	.180	-.049	-.252[c]	.282[b]	.350[a]			
PbH	.331[a]	.024	-.065	.285[b]	.453[a]	.135		
PbF	.053	.298[b]	.240[c]	.243[c]	.102	.140	.083	
PbW	.462[a]	.114	.058	.460[a]	.348[a]	.126	.351[a]	.366[a]

PbB = Blood lead concentration
PbAI = External air lead concentration
PbAE = Internal air lead concentration
PbDC = Average dust lead concentration (bedroom and playroom)
PbDL = Average dust lead loading (bedroom and playroom)
PbS = Soil lead concentration
PbH = Hand lead levels
PbF = Dietary (food/beverages) lead concentration
PbW = Water lead concentration

Significance levels: a $p < 0.1\%$; b $p < 1\%$; c $p < 5\%$

With reference to Table 7.2, it can be seen that hand lead is more highly correlated with dust lead loading ($r = 0.45$) than with dust lead concentration ($r = 0.29$). This is illustrated graphically by the scatter plots in Figures 7.1 and 7.2. Similarly, a greater correlation is found between blood lead concentration and dust lead loading ($r = 0.46$) than between blood lead concentration and dust lead concentration ($r = 0.21$). These two

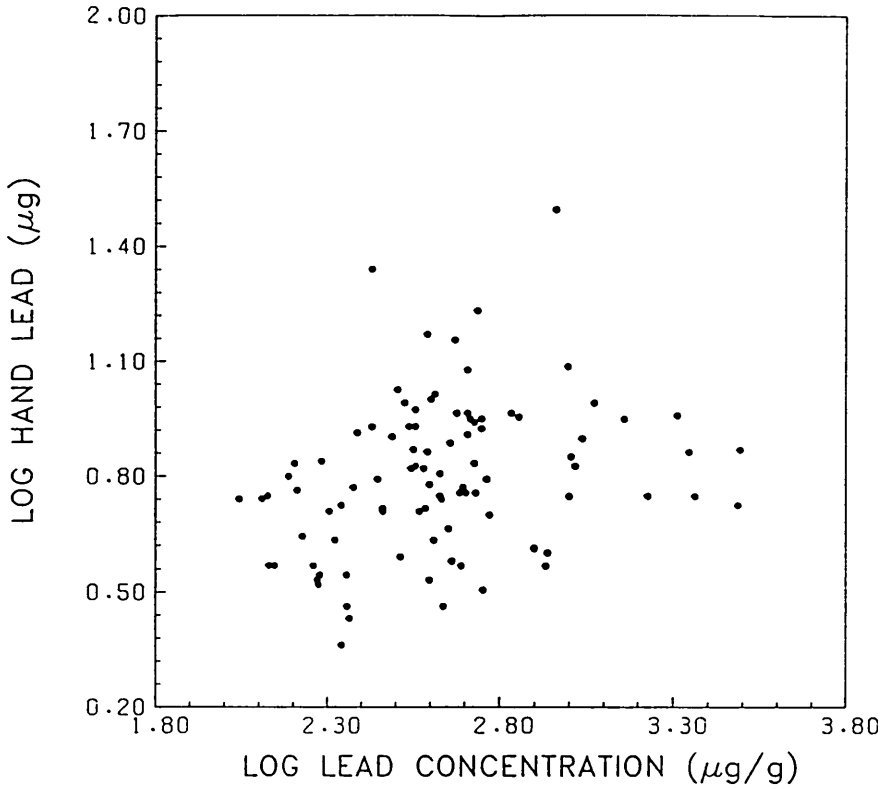


Figure 7.1 Scatter plot of hand lead level vs dust lead concentration (log-transformed data)

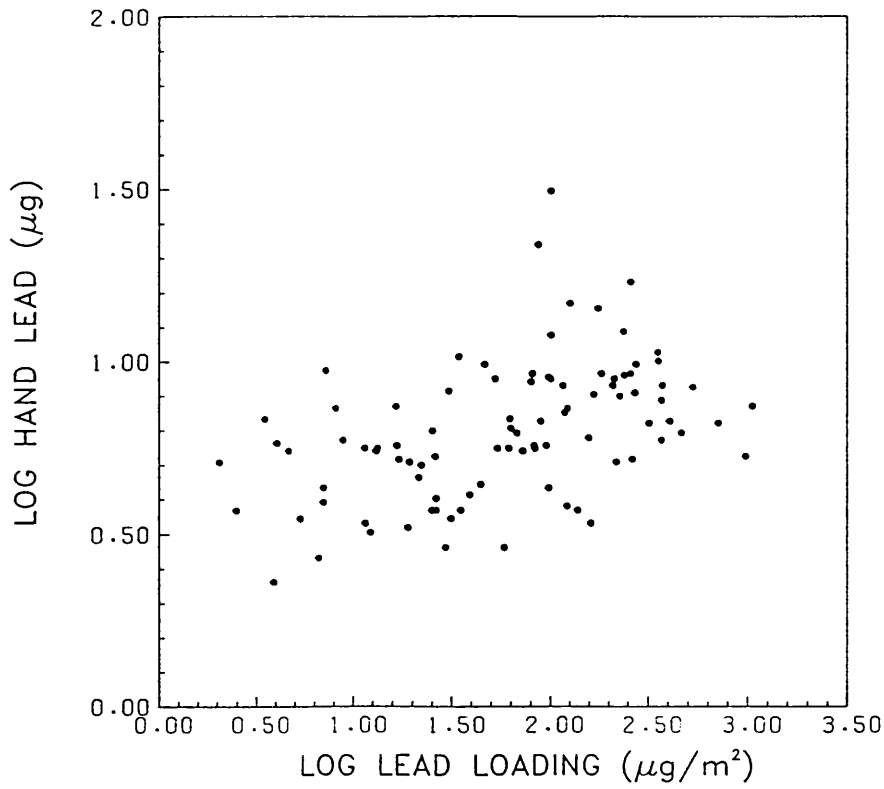


Figure 7.2 Scatter plot of hand lead level vs dust lead loading (log-transformed data)

findings support the indications from the Pilot Study (Appendix A) that looking at lead concentration alone could be deceptive as the total mass of lead available for ingestion by a young child from housedust is not reflected in this figure. The usefulness of calculating lead loading is further supported by the fact that the correlation between lead loading and blood lead ($r = 0.46$) is higher than that between hand lead and blood lead ($r = 0.33$) (see scatter plots in Figures 7.3 and 7.4). This finding is likely to be an indication that dust lead loading provides better temporal and spatial integration of lead exposure than the more temporally discrete hand lead measure. The only other variable to show a significant correlation with blood lead concentration is water lead concentration ($r = 0.46$) (see Figure 7.5). The lead concentration of the diet showed no correlation. However, the concentration of food and beverages does not properly reflect lead intake via this route. The weight of the total dietary sample was noted before analysis and lead intakes were subsequently calculated. The correlation between this measure and blood lead was also, however, insignificant ($r = 0.03$).

Again with reference to Table 7.2, it can be seen that there is a significant correlation between the concentration of lead in the diet and both the internal (1%) and external (5%) air lead concentrations. This finding is somewhat unexpected but could be a reflection of the contamination of food preparation surfaces with aerially deposited lead (Gallacher et al., 1984). The significant (5%) correlation between dust lead concentration and the concentration of the lead in the diet may also be a reflection of such contamination. The apparent strong correlations between dust lead (concentration and loading) and water lead concentration are difficult to explain, as is that between hand lead and water lead. One might hypothesise that houses where the water lead concentration is higher are likely to contain

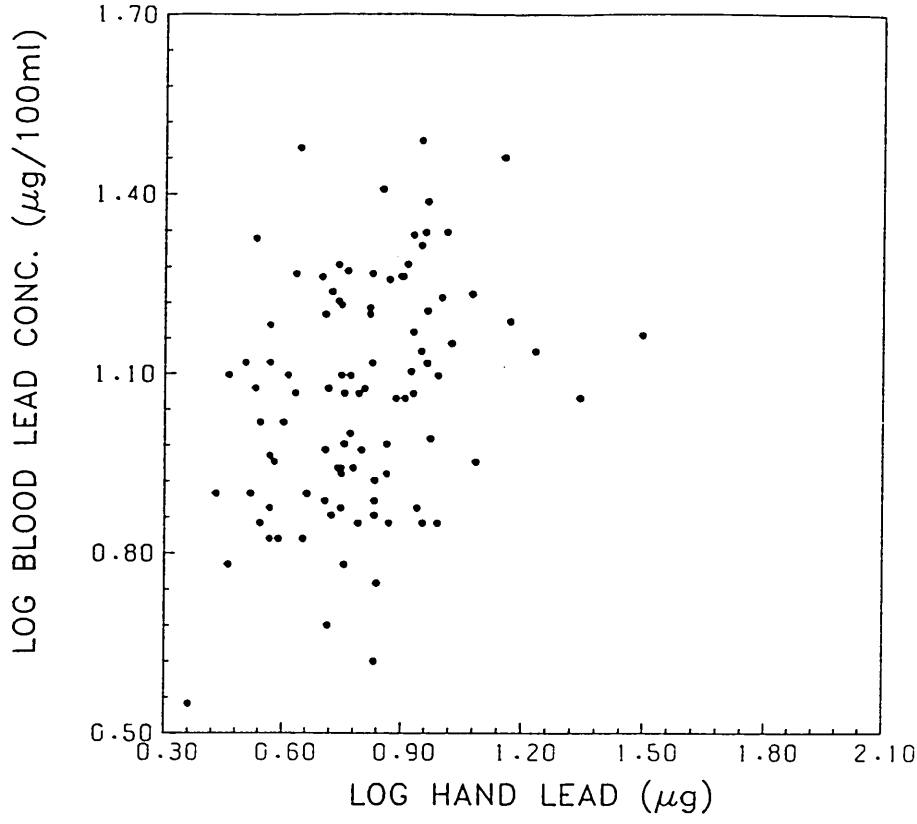


Figure 7.3 Scatter plot of blood lead concentration vs hand lead level (log-transformed data)

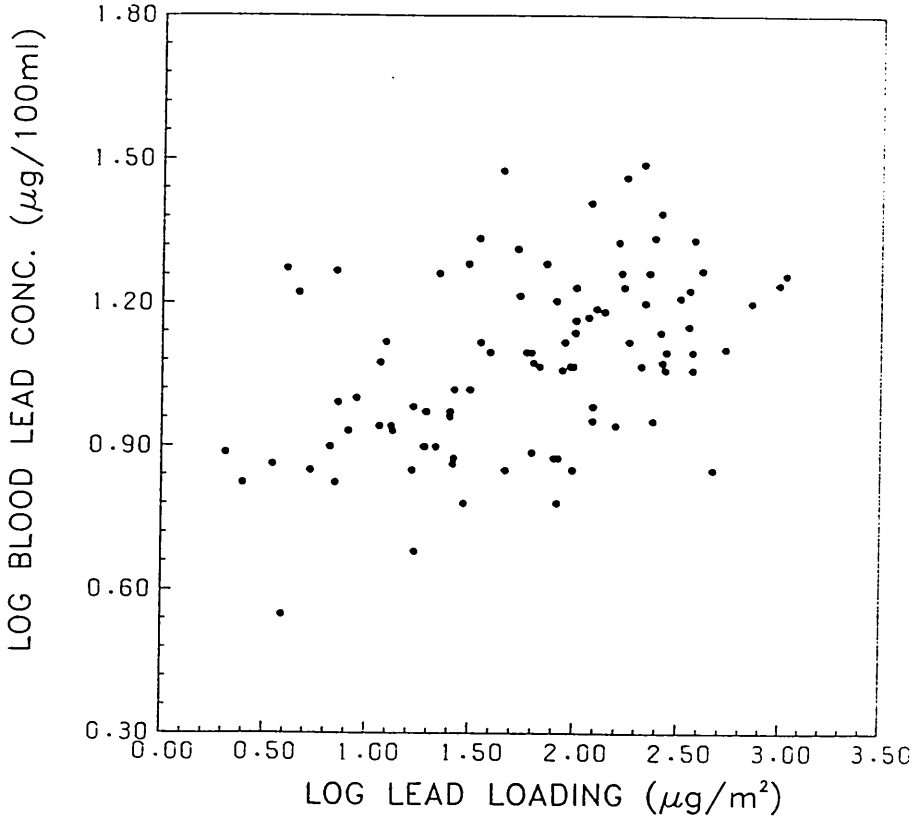


Figure 7.4 Scatter plot of blood lead concentration vs dust lead loading (log-transformed data)

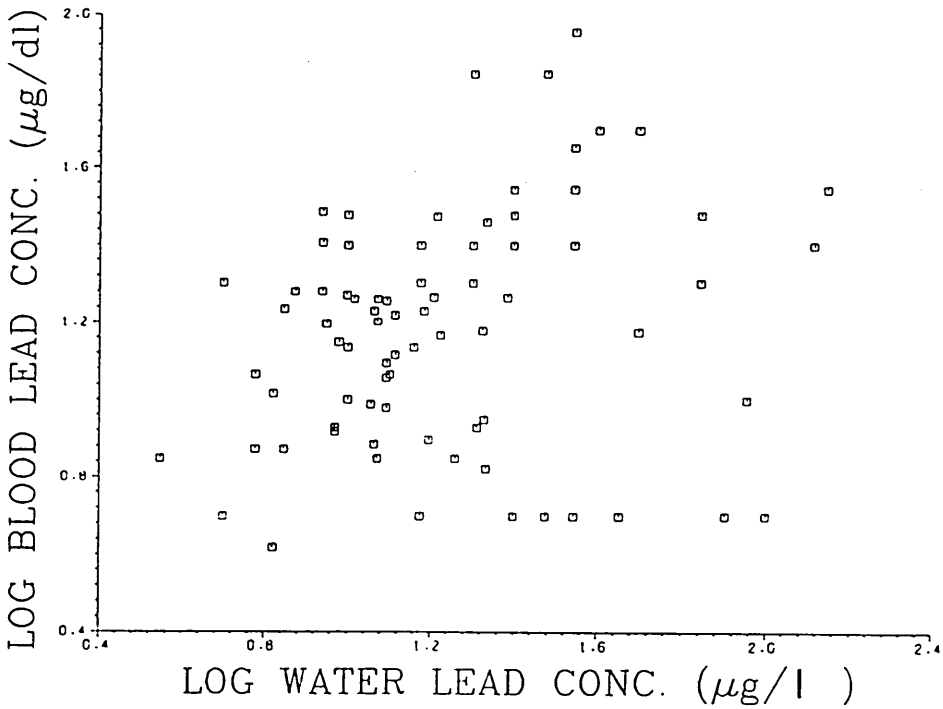


Figure 7.5 Scatter plot of blood lead concentration vs water lead concentration (log-transformed data)

contain lead pipes which may be contributing to the lead content of indoor dusts. However, from the questionnaire responses, no significant increase in dust lead concentration was noted in such houses. The significant correlation (0.01%) between lead concentrations in the water and the diet may be a reflection of the fact that the diet samples included not only food but also all beverages consumed by the children, several of which are likely to have either been water or other drinks containing water.

The only correlation coefficient to approach significance in a comparison of behavioural measures and blood lead was that for rate 15 (both hands touching all

objects), r = 0.18. This coefficient was not improved by combining rate 15 with any of the other rates before subsequent comparison with blood lead.

7.3 RELATIONSHIPS BETWEEN BLOOD LEAD, ENVIRONMENTAL LEAD AND BEHAVIOURAL MEASURES: MULTIPLE REGRESSION ANALYSIS

The relationships between the exposure variables discussed above and the blood lead concentration were further explored using multiple regression analysis. This was carried out using a backward elimination procedure, although results from the correlation exercise were used as a guide in the initial choice of the independent exposure variables for inclusion in the model. As discussed earlier (Section 4.2), one of the main hypotheses being tested in this study was that housedust lead influenced blood lead, with hand-to-mouth activity being a significant route of exposure. Hence, along with measures of housedust lead and hand lead, assessments were made of the mouthing and touching activities of the children. Thus, in addition to assessing the influence of either hand lead or dust lead on blood lead in any given model, the influence of the combined factors (hand lead x hand-to-mouth activity rating) and (dust lead x hand-to-mouth activity rating) were also independently assessed. The most appropriate combined factor was found to be (dust lead loading x rate 15), where rate 15 is the rate for hands touching all objects in the environment, including the floor. The "best" model (R^2 = 32%) was as follows:

$$\text{Log PbB } (\mu g/dl) = 0.57 + 0.11 \log X_1 + 0.13 \log \text{PbW } (\mu g/l)$$

PbB = blood lead concentration (µg/dl)
X1 = (dust lead loading [ug/m^2] x rate 15 [touches/min])
PbW = water lead concentration (µg/l)

The inclusion of air lead concentration, soil lead concentration or dietary lead intake, either separately or in any combinations gave non-significant regression coefficients for the variables concerned and only marginal improvements in the R^2 values.

Treatment of the data by multiple regression analysis was then taken one stage further. It was apparent from an assessment of the questionnaire responses that certain subgroups within the study population tended to have higher blood lead levels than the rest of the population (see Table 5.11). Those questionnaire items which resulted in subgroups with significantly different blood lead levels were: "age of house", "decorating", "pets", "car maintenance", "proximity to commercial garage", "smoking habits of the parents", "mouthing of exterior railings" and "hand washing technique". These can all therefore be considered as potential confounders as they are all potential determinants of the dependent variable, blood lead. In order to take account of this, these were entered into the model as dummy variables. The only variable to achieve a significant regression coefficient was parents' smoking habits. The adjusted model, with an improved R^2 value of 35% is given below:

$$\text{LogPbB } (\mu g/dl) = 0.55 + 0.10 \log X_1 + 0.14 \log PbW + 0.07S$$

where S = parents who smoke

This indicates that children of parents who smoke are likely to have a blood lead level around 1.2 µg/dl (0.06 µmol/l) higher than children of non-smoking parents. It is of interest that Sherlock et al. (1985) also noted a similar smoking effect in their study of around 100 two and a half to five year old London children.

There are few comprehensive exposure assessment studies which have been carried out on "typical" urban

children of this age, with which these results can be compared. Brunekreef et al. (1983) studied slightly older children, aged 4 to 6 years, and reported that the most important environmental factor influencing blood lead was lead deposition outdoors. Mouthing behaviour, hand dirtiness and intake of milk were also reported to influence blood lead levels significantly. In Cincinnatti, Bornschein et al. (1986) reported that, for 18 month old children, 38% of the observed variation in blood lead was accounted for by hand lead and dust lead. However, as is the case with many of the studies carried out in the US, they are difficult to relate to the situation in the UK because environmental and blood lead levels are consistently higher. For example, in Bornschein et al.'s (1986) study, the mean blood lead concentration was around 17 µg/dl (0.82 µmol/l)

One UK study does provide some useful data. Laxen et al. (1987) took water and dust samples from around 500 Edinburgh households and reported that both were significant predictors of blood lead (R^2 = 32%). However, in contrast to the findings in this study, they reported that dust lead concentration was a more useful predictor of blood lead than dust lead loading. The samples obtained by these workers were collected from a smaller area (1 m^2) than those in this study and are thought likely to have provided a less integrated sample. Also, because these samples were not sieved before analysis, carpet fluff, pet hairs and other extraneous material will have added to the weight of "dust" used in the calculation of the loading figures.

7.4 SUMMARY EXPOSURE ASSESSMENT

The above analyses relate the variability in blood lead to the variability in exposure measures. However, it is possible that a large proportion of blood lead might

have arisen from one particular, predominant intake (e.g. food) which either does not vary much between individuals or which is measured with large "within individual" errors relative to those in blood lead. The following exposure assessment of the relative importance of lead intakes and uptakes of via different pathways is based on assumptions used by the RCEP (1983). It should be noted here that the Commission stressed the importance of recognising the limitations of such an exercise, which involves many uncertainties and assumptions and makes no attempt to quantify lead excreted by the body.

Beginning with the inhalation route, the calculation of lead uptake from air is based on the air lead concentration, both indoors and outdoors, the amount of time spent at each location, and two main assumptions: that the volume of air respired per day is 6 m^3 and that 70 per cent of the inhaled lead is absorbed (RCEP, 1983). It is also assumed that the transfer of lead from trachea to oesophagus, by muco-ciliary action, is negligible. Unfortunately, there is considerable uncertainty regarding both of these two main assumptions due to the fact that it would be inappropriate to carry out the relevant experiments on this group of the population (Section 3.1). Not all of the lead inhaled is retained by the lungs, a proportion is exhaled. The percentage of inhaled lead which reaches the bloodstream from the lungs is dependent upon the physical and chemical form of the lead. Again due to a lack of data on children, the combined retention and absorption factor of 0.7 must be thought of as tentative.

The intake and uptake of lead via inhalation is shown in Table 7.3. The mean lead concentrations, both indoors and outdoors, found in this study were considerably lower than those previously estimated for typical inner city areas in the UK. The mean outdoor concentration was 0.43 $\mu g/m^3$ and the mean indoor

concentration 0.26 μg/m³, compared with the Commission's estimate of 1.0 and 0.75 μg/m³ respectively (Table 7.3). As this study was carried out in the winter it is not surprising to find that the average time spent indoors was high, 23.25 h/d. Assuming the volume of air respired per day was 6 m³, the calculated lead intake from air was 1.59 μg/d. Applying a combined retention and absorption factor of 0.7, lead uptake from the lungs was estimated to be only 1.1 μg/d, around a third of the Commission's estimate of 3.4 μg/d.

Table 7.3 Intake and uptake of lead via inhalation

	RCEP	THIS STUDY
Air lead conc. (μg/m³): Outdoor	1.00	0.43
Indoor	0.75	0.26
Time spent indoors (hrs/d)	20.00	23.25
Volume of air respired (m³/d)	6.00	6.00
Lead intake from air (μg/d)	4.8	1.59
Per cent absorption in lungs	70.0	70.0
Lead uptake from lungs (μg/d)	3.4	1.1

For the ingestion route, the main assumptions used are that the mass of dust ingested per day is 100 mg and that 53% of the total lead ingested reaches the bloodstream from the gut, the remainder passing directly to the faeces. It should be stressed that there is considerable variation in the dust ingestion rates cited in the literature (Davies and Thornton, 1988) and that the percentage absorption of lead varies widely, depending on the contents of the gut and the physical and perhaps chemical form of the ingested lead (Section 3.2.1).

The intake and uptake of lead via ingestion is shown in Table 7.4. There were no direct data on the average dietary lead intake of young children available to the Commission and it can be seen that the mean value from this study, 23 μg/d, is slightly less than half their estimate of 52 μg/d. The mean housedust lead

Table 7.4 Intake and uptake of lead via ingestion

	RCEP	THIS STUDY
Dietary lead intake (μg/d) [food and drinks]	52	23
Dust lead conc. (μg/g)	1400	424
Lead intake from dust (μg/d)	140	42.4
Total lead intake: ingestion (μg/d)	192	65.4
Per cent absorption in gut	53	53
Total lead uptake from gut (μg/d)	102	35

concentration was 424 μg/g[1], resulting in an estimated intake of 42.4 μg of lead per day via this pathway. Recent studies into the particle size distribution of dust adhering to children's hands indicate that this derived intake value may be an underestimate. Duggan et al. (1985) showed that the majority of dust particles found on hands were below 10 μm and that the maximum particle diameter was 100 μm. This is an important finding when one considers that, for a sieved dust, the lead concentration usually increases as the size fraction decreases. It may well be more appropriate to analyse the finer dust fractions rather than the less than 1000 μm fraction, which is the routine procedure of most workers currently. Further work is required to clarify this situation. The total lead intake from diet and dust was estimated to be around 65 μg/d (Table 7.4). Applying an absorption factor of 0.53 the total lead uptake from the gut was calculated to be 35 μg/d, approximately one third of the total uptake estimated for two year old children in inner-city areas of the UK as a whole.

To summarise, the total uptake from the lungs and gut was estimated to be 36 μg/d, compared with 105 μg/d

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1 excluding data from child with elevated blood lead level

estimated by the Commission. Only 3 per cent of the total uptake, in both cases, is attributed to inhalation, the other 97 per cent is due to ingestion (Table 7.5).

Table 7.5 Summary of lead uptakes from inhalation and ingestion

	RCEP	THIS STUDY
Total lead uptake from lungs and gut (µg/d):	105	36
Percentage of daily lead uptake from inhalation:	3	3
Percentage of daily lead uptake from ingestion:	97	97

The above assessment includes an estimate of lead intake from air via inhalation. Assessment of the intake of air lead via indirect, deposition pathways (e.g. diet, dust) is more difficult. In particular, there are no suitable data currently available concerning the proportion of housedust lead which is derived from air lead deposition. However, calculations by Chamberlain (1983) showed that approximately one tenth of total dietary lead intake could be due to fallout of lead from the atmosphere. The mean daily lead intake from the diet, 23 µg/day, would result in an estimated indirect intake from lead in air of 2.3 µg/day. Applying an absorption factor of 0.53 results in an estimated intake of 1.2 µg/day, which is similar to that via inhalation of 1.1 µg/day.

The calculations carried out so far have incorporated mean values from the various data sets to provide information on the likely exposures of a typical 2 year old child living in the inner-city area of

Birmingham. As actual exposures will differ, the relevant data from two households have been extracted and used in the following calculations to provide an indication of the ranges involved (Table 7.6). House 6 was being redecorated at the time of the study and, as well as having a relatively high mean indoor air lead concentration, 0.48 $\mu g/m^3$, had the highest housedust lead concentration, 3110 $\mu g/g$. House 2, on the other hand, had the lowest housedust lead concentration, 111 $\mu g/g$ and a mean indoor air lead concentration of 0.20 $\mu g/m^3$. Using the same assumptions as before, the intakes and uptakes have been estimated. Lead uptake from the lungs was calculated to be 2.0 $\mu g/d$ using data from House 6 compared with 0.65 $\mu g/d$ using data from House 2. The total intakes by ingestion, including diet and dust, were 332 and 18.1 ugd for Houses 6 and 2, respectively. This results in very different estimates of uptake of 176 and 10 $\mu g/d$ in the two situations. These figures are merely presented as an example of the possible uptakes in these types of environments and are not intended to be estimates of the uptakes of the two individuals actually living in these houses. There is also greater uncertainty associated

Table 7.6 Lead intake and uptake: selected examples

	HOUSE 6	HOUSE 2
INHALATION:		
Lead intake from air ($\mu g/d$)	2.7	0.92
Lead uptake from lungs ($\mu g/d$)	1.9	0.65
INGESTION:		
Dietary lead intake ($\mu g/d$) [food and drinks]	21.0	7.0
Dust lead conc. ($\mu g/g$)	3110	111
Lead intake from dust ($\mu g/d$)	311	11.1
Total lead intake: ingestion ($\mu g/d$)	332	18.1
Lead uptake from gut ($\mu g/d$)	176	10.0

with these figures than with those derived from earlier calculations, as very high or very low levels of lead in the gut may well modify the absorption factor.

Returning to the question of dust lead intake, the mean hand lead level was 5.7 μg (Section 5.4). The actual amount of lead present on the hands will vary throughout the day depending on the particular activity of the child and the frequency of washing. This figure is, thus, an estimate of the likely level at any given time on any given day. Looking again at the previously estimated value for lead intake from dust of around 42 μg per day,it would require the transfer of all hand lead from hand to mouth a total of 7 times per day to result in this intake. The results from the behavioural section of the study indicated that one or more fingers of either or both hands were placed in the mouth an average of 0.23 times per minute, or around 14 times per hour (Table 5.9). It would appear to be quite feasible, therefore, that such a daily transfer could take place.

In summary, the above assessment of the exposure of the study children to lead, based on assumptions used by the RCEP (1983), indicates that intake via the inhalation route accounts for only three per cent of the total daily lead uptake (Table 7.7).

Table 7.7 Summary of the intake and uptake of lead by young children in Birmingham

INTAKE ROUTE	PATHWAY	LEAD UPTAKE (μg/d)	TOTAL UPTAKE (%)
Inhalation	Air	1.1	3
Ingestion	Diet	12.2	34
	Dust	22.5	63
		----	---
	Total	35.8	100

For the ingestion route, the contribution to lead uptake made by the intake of dust is around twice that attributable to the diet, the estimates being 22.5 and 12.2 μg/d, respectively.

The EPA (1986) has recently provided an assessment of human lead intakes from air, food, water, beverages and dust with which the estimates from this study can usefully be compared. For a two year old child, a "baseline" exposure to lead would result in a total daily intake of lead of 46.6 μg. Such a child would typically live in a non-urban environment, eat a normal diet of food taken from a typical shop shelf and would have no habits or activities that would tend to increase lead exposure. For a two year old child living in a typical urban environment in the US, it is estimated that the total daily intake would rise by 91 to 137.6 μg/day. This is around twice as high as the estimated daily intake of 67 μg in this study for two year old children living in urban areas in the UK. The increase above "baseline" in the EPA's estimate is solely due to the intake of dust with higher lead concentrations in urban areas. The intake from the diet (food, water and beverages) in the urban situation, estimated to be 25.1 μg/day (the same as in the "baseline" situation) is, in fact, remarkably similar to the estimate of 23 μg/day in this study.

It is recognised that there are considerable uncertainties in the estimates of intake rates, retention and absorption factors used to derive the figures relating to this study and they should only be considered as general indicators of the significance of the various pathways.

8. SUMMARY, CONCLUSIONS AND RECOMMENDATIONS FOR FURTHER WORK

8.1 SUMMARY OF STUDY

8.1.1 Dust and soil

The mean lead concentration in housedust, 430 μg/g was slightly lower than the national figure, although soil lead, 313 μg/g was slightly higher. The average amount of lead in housedust (the "loading") was 60 $\mu g/m^2$. Soil lead concentration was more highly correlated with dust lead loading than dust lead concentration. The age of the house influenced lead levels in both housedust and garden soil, with older houses having higher concentrations. Houses being decorated had significantly higher lead concentrations than those that were not. Elevated lead levels were also noted in housedust and soil samples from houses located within 500 m of commercial garages. Increased lead concentrations were found in soil from gardens in close proximity to waste land (demolition sites and tips) and metal using industries, and from those within 10 metres of a road.

8.1.2 Air

In each house, the internal samples (playroom and bedroom) were very similar and the overall mean was 0.26 $\mu g/m^3$. The mean external air lead concentration was 0.43 $\mu g/m^3$. The internal:external ratio of 0.61 is similar to others cited in the literature. The correlation between the external and average internal air lead levels in individual houses was very high.

8.1.3 Handwipes

The mean amount of lead on the hands was 5.7 µg which is lower than values from several studies involving older children, but similar to that of 7.8 µg for 18 month old children in Cincinnatti. It has widely been assumed that a child ingests 100 mg of dust per day, on which basis the average intake of lead in this study would have been around 40 µg per day. This would require the transfer of all hand lead from hand to mouth seven times per day. The amount of lead on children's hands was more closely related to the dust lead loading than to the dust lead concentration.

8.1.4 Diet

The mean intake of lead in the diet (food and beverages) was 161 µg/week. This is higher than in a similar study in Harrow and could, in part, be due to the higher water lead concentrations in Birmingham where the geometric mean was 19 µg/l and 45 per cent were above 20 µg/l compared with only 5 per cent in Harrow. Nine per cent of the children exceeded the current Joint FAO/WHO Expert Committee on Food Additives (1987) provisional tolerable weekly intake for infants and young children (25 µg/kg body weight/week), although none of these exceeded the DHSS advisory action level for lead in blood (25 µg/dl).

8.1.5 Blood

The mean blood lead concentration, 0.56 µmol/l [11.7 µg/dl] was similar to that found previously among children of this age in central Birmingham. Agreement between repeated samples was very good.

8.1.6 Behavioural measures

These present some difficulty in interpretation because there are no other similar data available with which to compare them. However, it has been shown to be practicable to obtain detailed, reliable and valid quantification of a specific aspect of a young child's behaviour in the home environment. A breakdown of the data into different behavioural clusters and different settings gives a reasonably detailed picture. However, an average taken over all behaviours and all settings indicates that something (objects or parts of the body) touches the mouth area or goes into the mouth around two times per minute. Similarly, one or other hand touches something in the child's environment around 16 times per minute. While these averages smooth out the situational differences that clearly exist, they form a basis for fitting into other models.

8.1.7 Relationships between blood lead, environmental lead and behavioural measures

The correlation of blood lead concentrations with average indoor air lead concentrations was virtually zero. As with the results for handwipes, the correlation of blood lead concentrations with lead loading in housedust was much higher than for the lead concentrations. In addition, the correlation with the dust loading was higher than with the handwipe lead. The correlation with soil lead concentrations was similar to that with dust lead concentrations. Whilst the correlations of blood lead with both dietary lead concentration and intake were very small (and not statistically significant), that with water lead concentrations was highly significant.

The relative importance of the different exposure variables was assessed using multiple regression analysis. The most appropriate indicator of dust lead level found

(X_1) was the dust lead loading multiplied by the rate for hands touching all objects (including the floor) and the "best" model (R^2 = 32%) was:

$$\text{Log PbB } (\mu g/dl) = 0.57 + 0.11 \log X_1 + 0.13 \log \text{PbW } (\mu g/l)$$

where PbB = Blood lead concentration
PbW = Water lead concentration

The inclusion of air lead concentration, soil lead concentration or dietary lead intake, either separately or in combination, gave non-significant improvements in the R^2 values. It would therefore appear that as an indicator of lead exposure a combination of the dust lead loading and the overall rate of touching objects was better than the amount of lead actually on the hands, either on its own or in combination with the rate on hand-to-mouth activity. Water lead was also found to be a significant factor and further statistical analysis indicated that parents' smoking habits also influenced blood lead levels significantly. The adjusted model, with an improved R^2 of 35% was:

$$\text{LogPbB } (\mu g/dl) = 0.55 + 0.10 \log X_1 + 0.14 \log \text{PbW} + 0.07S$$

where PbS = Parents who smoke

8.1.8 Summary exposure assessment

An assessment of the relative importance of the different transfer pathways, based on assumptions used by the RCEP, indicates that lead uptake from inhalation would be only just over 1 µg/day, and assuming that lead from dust and from the diet are equally bioavailable, that by ingestion 35 µg/day. The resulting total estimated uptake (36 µg/day) is only about one third of that calculated by the RCEP, but the proportions of 3% from inhalation and

97% from ingestion are similar. However, given the uncertainties surrounding the figures for dust ingestion and absorption by the gut, both the estimated proportion and the total amounts of lead from dust and from the diet must be treated with caution. In addition, the average figures conceal a large amount of variability: on the same assumptions, uptake could have been only 10 μg/day where environmental lead levels were low, or, in exceptional circumstances, around 180 μg/day in a house being redecorated. Assessment of the importance of the contribution of air lead via indirect deposition and subsequent ingestion is more difficult. The proportion of lead in the house dust which is derived from aerial deposition is unclear, although source apportionment techniques are being developed. Chamberlain has calculatd that about one tenth of total dietary lead intake could be due to fallout from the atmosphere, which would give an estimated contribution to total dietary uptake of just over 1 μg/day, similar to that via inhalation.

8.2 CONCLUSIONS

This comprehensive study has demonstrated that it is possible to measure simultaneously the lead exposure of young children via all major pathways and to quantify specific aspects of a young child's behaviour. Blood lead was related principally to a combination of the amount of lead in the dust and the child's rate of "hand touching" activity, confirming the results from the pilot study which showed that exposure of children to lead in house dust was expressed more realistically by the "loading" than simply by the concentration. Water lead was also a significant factor and parents smoking habits were shown to influence blood lead levels. Although the estimates of intake and uptake of lead in this study were about one third of those made by the Royal Commission, both assessments indicate that for young children, only a very small proportion of the uptake was from inhalation.

8.3 RECOMMENDATIONS FOR FURTHER WORK

The major finding of this study was that, for children whose blood lead levels were within the normal range for the UK, blood lead concentration was significantly related to the lead content of housedust. In the light of this, further work is recommended on characterising the sources of lead in household dust. Two different approaches to this source apportionment work are showing promise. The first is the use of scanning electron microscopy, combined with energy dispersive X-ray microscopy, in which information on the size, shape and elemental composition of the individual particles of housedust is gathered and compared with information previously gathered on specific source type particles (e.g. paint, smelter) (Hunt et al., 1988). The second involves a comparison between the isotopic ratio of lead in dust and the isotopic ratio in potential source type materials. Indeed, the isotopic ratio of lead in blood can also be assessed by these techniques and usefully compared with those of a variety of potential environmental and dietary sources (Delves, personal communication). Source apportionment work, using both of these approaches, is ongoing with the samples collected in the present study. It will be of great interest to compare the results from the exposure assessment detailed in this thesis with those from an exposure assessment of the same study children based on the results from the isotope ratio work.

Finally, as this study demonstrated that the intake of lead from household dusts with lead contents considered typical for the UK could significantly contribute to the body burden of young children, further work is also recommended to assess the lead intakes from household dusts in areas where the lead content of such dusts has been shown to be highly elevated (e.g. lead mineralised areas of Derbyshire and north Wales).

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APPENDIX A BIRMINGHAM PILOT STUDY

A full scale pilot study was carried out in five houses in the proposed study area and this is considered in detail below.

A cross-section of the types of housing which might have been expected in the main study was selected by the Environment, Health and Behaviour Research Group (EHBRG). All five families approached agreed to participate and blood samples were obtained from the children (aged 22 to 26 months).

The following samples were taken during the pilot study: dust (house, pavement, road), soil, air and handwipes. In addition, a duplicate diet study was also carried out, water samples were obtained and a detailed questionnaire was administered. The sampling protocols and analytical techniques used were the same as those employed in the main study unless otherwise specified.

1. Dust

(a) Housedust

Samples were collected from the two rooms used for air sampling (child's bedroom and playroom) on the first and last day of the study period using the IC vacuum cleaner method described elsewhere (Watt et al., 1983). The entire exposed floor area was vacuumed into a pre-weighed filter thimble and the room was measured. A dust sample was also taken from the householder's own vacuum cleaner.

Results and Discussion

The results are presented in Table A1.

Table A1 Lead concentration, dust loading and lead loading in five Pilot Study homes

SAMPLE LOCATION	LEAD CONC. (μg/g)		DUST LOADING* (mg/m^2)		LEAD LOADING ($\mu g/m^2$)	
	DAY 1	DAY 8	DAY 1	DAY 8	DAY 1	DAY 8
HOUSE 1						
Bedroom	1170	945	326.3	227.1	387.3	214.6
Playroom	177	173	1334.2	312.4	437.9	54.0
HOUSE 2						
Bedroom	1310	980	91.8	73.8	120.2	68.7
Playroom	640	660	625.9	327.1	400.6	215.9
HOUSE 3						
Bedroom	250	1110	106.1	78.0	26.5	86.6
Playroom	240	175	59.3	45.6	14.2	8.0
HOUSE 4						
Bedroom	790	380	101.8	78.6	80.4	29.9
Playroom	410	420	105.0	99.5	43.0	41.8
HOUSE 5						
Bedroom	1190	970	150.2	164.2	178.7	159.2
Playroom	420	393	132.5	127.6	55.7	50.1

* <1mm fraction

The most striking feature of the data set (Table A1) is the relative difference between the values for lead concentration and lead loading. With reference to House 1, on Day 1, the concentration in the playroom is approximately one sixth of that in the bedroom (177 and 1170 μg/g, respectively) while the lead loading figure is higher for the playroom than for the bedroom (438 and 387 $\mu g/m^3$, respectively). This is an important finding when one considers the exposure of a child to lead in this

situation and indicates that looking at concentration alone could be deceptive. It also suggests that the total mass of lead to which the child is exposed from housedust is more accurately descibed by a lead loading figure. The pattern in House 2 is similar to that in House 1, while in Houses 3, 4 and 5, which were relatively clean, there is no great discrepancy between the lead concentration and the lead loading figures. Comparing the dust and lead loading data for Day 1 and Day 8, the dirtier houses, House 1 and House 2, show the biggest changes. It was felt that the children's exposure in these two houses had been altered radically because the samples obtained on day 1 had effectively removed the housedust to which they would normally be exposed. It was, therefore, decided to sample the housedust only once, at the end of the study week, in the main study.

(b) Pavement dust)
(c) Road dust) Collection and analysis of these.
) samples was straightforward.
)
2. Soil)

3. Air

Personal exposure monitors were considered for use in the study but were found to be unsuitable. Not only was the sampling device a heavy burden for such young children, but they also frequently tampered with the equipment (turning off the pump, removing the tubing, etc.).

Three locations were sampled, bedroom, playroom and immediately outside the house, using the method descibed in the main text. The pumps were situated as follows:-

House 1: Cupboard under the stairs
House 2: Garden shed

House 3: Cupboard in front room
House 4: Cloakroom off hall
House 5: Cupboard in hall

None of the householders found the noise from the pumps too disturbing. The situation in House 2 was ideal as the pump could not be heard in the house. When securing the tubing (linking the filter heads to the pump) staples were found to be preferable to sticky tape. Originally, the filter heads were placed at child-head height but this was found to be impractical because the children interfered with the filters. It was decided, therefore, to position the filter heads out of the their reach, at a height of 1.5 to 2 metres. Also, it was usually not possible to connect the pumps to an electrical socket out of the child's reach and on one occasion, a pump had been switched off for an undetermined period of time. Elapsed time meters were therefore fitted to the pumps for use in the main study so that the actual operating time could be noted daily.

Results and Discussion

The results have been plotted and appear in Figure A1. As expected, the internal levels reflect those found outdoors but are generally slightly lower, with the exception of House 4. The results for this house illustrate how careful one has to be when setting up the network. The external filter head was positioned alongside the front door and the tubing was fed through a small gap under the door and connected to the pump. When the flowrate was checked initially with the door closed it was found to be adequate. On subsequent days it was checked and also found to be adequate, but the door was not closed during this procedure. On Day 7 the front door was firmly closed when the flowrate was measured (due to bad weather) and it became immediately apparent that the tube was trapped in the door and that there was no air

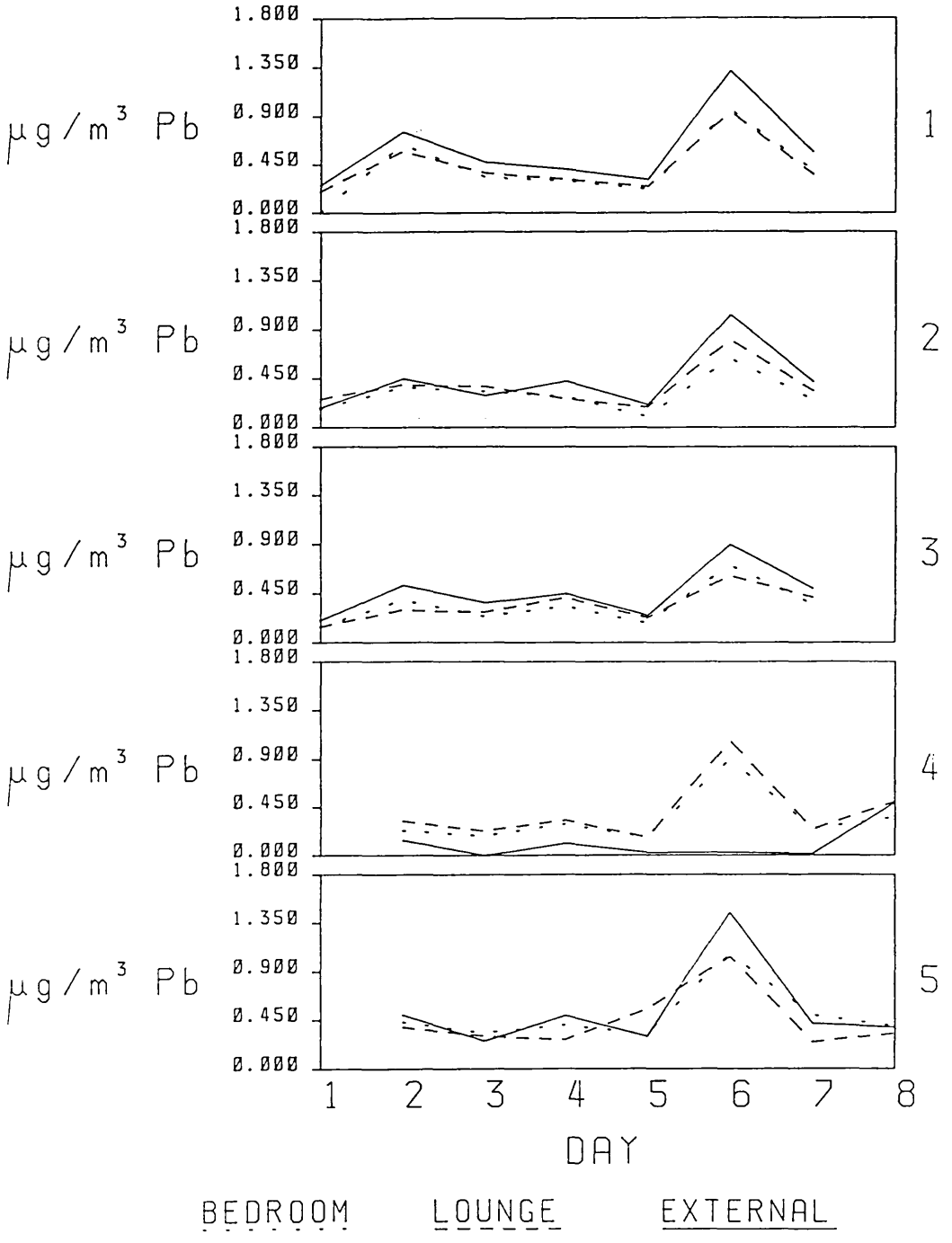


Figure A1 Daily air lead concentrations in the five Pilot Study houses

being drawn over the filter. This situation was then rectified, which accounts for the 'normal' reading on Day 8.

4. Handwipes

A series of experiments was carried out to determine the efficiency of "All Fresh" wet wipes in removing dust lead from hands. In the laboratory, around 0.05g (weighed on a 4 figure balance) of house reference material (HRM) 7 was applied to an adult subject's hands. The subject gently rubbed her hands together over a clean piece of pre-weighed paper, which was later re-weighed. One handwipe was then used to thoroughly wipe both hands and placed in a conical flask, which was immediately covered with a watchglass. This wiping procedure was then repeated 4 times, giving a total of 5 separate wipes. Each handwipe was analysed individually. This experiment was carried out a total of 5 times. The results presented in Table A2 show both the actual amount of lead (μg) on each wipe and the cumulative percentage of the total recovered for each wipe. On average (s.d.), 69% (9%) of the total was removed with the first wipe, 85% (6%) with the first two wipes, 94% (3%) with the first three wipes and 97% (2%) with the first four wipes. Similar trials by other workers (Que Hee et al., 1985) indicated 85% (7%) recovery with three wipes and 93% (4%) recovery with four wipes, although a different brand of handwipe (Walgreens Brand Wet Wipes) was used. Duggan et al. (1985) also investigated hand lead recoveries but using a slightly different method. After administering dust to the hands, 3 consecutive handwipes were used, followed by a rinse in dilute nitric acid. The results are difficult to compare with those above as the final acid rinse was far more efficient at removing hand lead then either the 2nd or 3rd wipe. It was not felt to be appropriate to amend the sampling technique in this study to include an acid rinse stage.

Table A2 Amount of lead removed from hands by consecutive handwipes

Experiment Number	Wipe Number	µg Pb	Cumulative % removed
1	1	58.1	59.3
	2	19.5	79.2
	3	13.4	92.9
	4	4.6	97.6
	5	2.4	100.0
	Total	98.0	
2	1	45.0	62.9
	2	13.7	82.1
	3	5.7	90.1
	4	2.8	94.0
	5	4.3	100.0
	Total	71.5	
3	1	87.2	85.7
	2	8.8	94.4
	3	5.7	100.0
	4	<DL	100.0
	5	<DL	100.0
	Total	101.7	
4	1	47.0	65.7
	2	11.4	81.7
	3	7.3	91.9
	4	3.9	97.3
	5	1.9	100.0
	Total	71.5	
5	1	57.0	71.7
	2	13.5	88.7
	3	4.9	94.8
	4	2.5	98.0
	5	1.6	100.0
	Total	79.5	
Mean (s.d.) of all experiments:			
	1		69 (9)
	2		85 (6)
	3		94 (3)
	4		97 (2)
	5		100

DL = 1.2 µg Pb

The total weight of lead applied during each experiment has been estimated from the dust lead concentration (1590 μg/g) and the weight of dust applied (Table A3). The apparent total weight of lead present on the hands has also been calculated. The percentage of the estimated totals actually recovered ranged from around 83 per cent to 119 per cent (mean = 101, s.d. = 14) and are similar to results found elsewhere (Que Hee et al., 1985).

Table A3 Total recovery of lead from hands using 5 "All-Fresh" wet wipes

Expt. No.	Wt. of dust (mg)	Estimated Pb content (μg)	Apparent Pb content (μg)	% recovery
1	53.2	84.6	98.0	115.8
2	51.6	82.0	71.5	87.2
3	53.9	85.7	101.7	118.7
4	54.0	85.9	71.5	83.2
5	50.4	80.1	79.5	99.3

It was concluded that the most appropriate number of handwipes to use, in what was to be a busy sampling programme, was three, as these and other trials indicated that around 90 per cent of the total lead removed with five handwipes could be recovered with three handwipes.

One child objected strongly to having her hands wiped on the first day of the study and, consequently, no sample was obtained. By the third day, the child was sufficiently well acquainted with the sampler to allow the full three wipes to be taken. It was decided, therefore, that in the main study if a child objected to this sample being taken on the first day, no sample should be obtained. This was done so as not to discourage the child from participating later in the study week. No other problems were encountered.

5. Diet and water

The collection of these samples was the responsibility of the parents. Although the tasks required of the parents were fairly straightforward (weighing of food samples, completion of a daily diary, filling of graduated bottle with water), several parents sought reassurance that they were doing their job correctly at some time during the study period. It was therefore decided that, in the main study, the dietary field officers should re-visit each parent early on in the study period to check that the tasks were being carried out adequately and/or provide reassurance.

6. Questionnaire

The administration of the questionnaire required around half an hour of the field officer's time. Where possible, the interview was carried out towards the end of the study period because it was felt that the parents were more likely to cooperate fully at this stage when they had become acquainted with the field officers than on the first or second day of the study period.

APPENDIX B

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B3.	House diary sheet	281
B4.	Sample check sheet	282

DUST & DIET STUDY

Part 1: PERSONAL DETAILS

THIS PART TO BE RETAINED BY EHBRG - DO NOT RETURN TO DOE

1.1 Surname of child _____

First name (s) _____

Home address _____

_____

_____

_____

Date of birth _____

Place of birth _____

Person (s) interviewed and relationship (s) to child _____

_____

_____

Names of parents (if different from above) _____

_____

address (s) _____

_____

_____

Name of General Practitioner _____

_____

address _____

_____

PART 2: THE HOME

2.1 How long have you lived at this address? (years) ___

Has X lived anywhere else since birth?

- No 0
- Yes, within study area 1
- Yes, outside study area 2

If so where? give approximate dates and address (es)

_____

_____

_____

2.2 House type

- Own house 1
- Own flat 2
- Privately rented house 3
- Privately rented flat 4
- Council house 5
- Council flat 6
- Other, including shared

If flat, which floor? ___

2.3 How old is your house/flat?

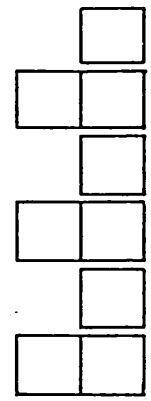
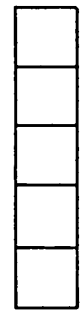
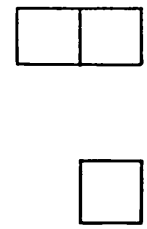
- Less than 5 years 1
- 5-15 years 2
- 15-35 years 3
- 35-80 years 4
- More than 80 years 5

2.4	Would you say there was any paint chipped or flaked or peeling anywhere <u>outside</u>, on window frames, gates, railings, lifts, stairs, coridoors, etc. No Some (less than 1/3 of surfaces) - specify main areas _____ _____ _____ Much (specify as far as possible)_____ _____ _____	0 1 2	
2.5	What about <u>inside</u>. Any paint chipped or peeling or flaked? No Some (less than 1/3 of surfaces specify as far as possible) _____ _____ _____ Much (specify as far as possible)_____ _____ _____	0 1 2	
2.6	Has there been any painting and decorating, or renovation work in your house? Since the arrival of X In the last 6 months Currently being renovated No Not known	1 2 3 4 9	

2.7	Has there been any decorating carried out in the last 3 years which involved any of the following techniques? Stripping with a blow torch Yes / No Stripping with a hot air stripper Yes / No Machine sanding Yes / No Hand sanding Yes / No If yes to any of these, how recently Since arrival of X 1 In last 6 months 2 Currently 3 Not known 4	
2.8	Are any windows or ventilators open during winter (note size, type, location, duration day, night) Yes / No _____ _____ _____	
2.9	Are the windows and doors draught sealed? All 1 Some 2 None 3	
2.10	Do you have double glazing? Yes / No Do you have roof insulation? Yes / No	
2.11	Do any of the windows have leaded lights? No 0 Some (less than 1/3) 1 Many 2	
2.12	Do you know of any lead flashings on the house? (Interviewer to check as far as possible) Yes / No	

2.13	Do you have any lead pipes in the house? Yes / No	☐
2.14	How many rooms are there? ☐☐	☐☐
2.15	How many rooms have carpet (covering more than 2/3 of their area?) ☐☐	☐☐
	Do you have any regular help with cleaning? (If yes, specify type and hours per week) Yes / No	☐
	_____	
	_____	
	How do you usually clean your carpets?	
	Brush 1 ☐	
	Sweeper 2 ☐	☐
	Vacuum (make & type) 3 ☐	
	How often do you usually get round to cleaning the carpet in the main play area?	
	Daily 1 ☐	
	2-3 times per week 2 ☐	
	Weekly 3 ☐	☐
	Less often 4 ☐	
	So you cleaned it_____ days ago	☐☐
2.16	Do you shampoo the carpet? Yes / No	☐
	If yes, how often	
	About once a month 1 ☐	
	Once every six months 2 ☐	☐
	Once a year 3 ☐	
	Less often than once a year 4 ☐	
2.17	How is the house heated?	
	Electricity 1 ☐	
	Coal 2 ☐	
	Gas-fire 3 ☐	☐
	Central heating, hot air 4 ☐	
	Central heating (other) 5 ☐	

2.18	What entrances are there? Front 1 Back 2 Other (please specify) 3 _____ _____	☐
2.19	Which entrances have door mats? Front 1 Back 2 Other 3	☐
2.20	Do you have any dogs? (give number) ☐☐ Do you have any cats? (give number) ☐☐	☐☐ ☐☐
2.21	Do you have any other pets (specify)_____ Yes / No Are your pets confined to any particular rooms? (specify) Yes / No _____ _____	☐ ☐
2.22	Do you have a car? Yes / No Do you have a motor cycle? Yes / No Where is the car/motor cycle kept (distance in metres) ☐☐ Do you maintain the car/motor cycle yourself? Yes / No Do you do oil changes Yes / No decarbs Yes / No exhaust system repairs Yes / No	☐ ☐ ☐☐ ☐ ☐ ☐ ☐

2.23	Do you have a front garden? Yes | No If yes, area of exposed soil in front garden (square metres) ____ Do you have a back garden? Yes | No If yes, area of exposed soil in back garden (square metres) ____ Do you grow any vegetables? Yes | No If yes, size of vegetable plot and/or allotment (square metres) ____	
2.24	Are any of the following within 1/4 mile (5 minutes walk)? Garages (lock up) 1 Garages (commercial) 2 Waste land, demolition sites, tips, etc 3 Metal using industry (specify as far as poss) 4 Public open space 5 _____ _____ _____ _____ _____	
2.25	How close is the nearest road? (as the crow files to main entrance of building) (in metres)? ____ What is the traffic density of this road? Light (domestic, deliveries only) 1 Medium (busy in rush hours, bus route short cut, etc) 2 Heavy (main road, through traffic, constantly busy) 3 Very heavy (Trunk road, major long route) 4	

Part 3: The Family

3.1	How many adults are there in the house? |___|___|	☐☐
3.2	What are the occupations of adults other than parents? 1. _____ 2. _____ 3. _____ 4. _____ What relationship (if any) do these other adults have to the child? _____ _____ _____	
3.3	Do any adults other than parents living in the house smoke? Yes | No | If yes, how many cigarettes per day in the house? |___|___| Adult 1 |___|___| Adult 2 |___|___| Adult 3 |___|___| Adult 4	☐ ☐☐ ☐☐ ☐☐ ☐☐
3.4	How many children are there in the house? |___|___| Ordinal position of X |___|___|	☐☐ ☐☐

Part 4: The Mother

4.1 Could you tell me how old you are Mrs. X? (years) ___

4.2 Where were you born?
- England, Scotland, N.Ireland, Wales 1
- Europe (outside UK) 2
- Other (specify) _____ 3

4.3 At what age did you leave school? (years) ___

Did you gain any qualifications?
- None 0
- CSE/O level 1
- A level 2
- Secretarial or technical qualifications 3
- Teacher training 4
- University training (not completed) 5
- Degree 6
- Post graduate 7
- Other (specify) _____ 8

4.4 Do you go out to work?
- No 0
- Full time, day 1
- Full time, night 2
- Part time, day - no of hours ____ 3
- Part time, evening - no of hours ____ 4

If yes, what is your job (in detail)

_____

_____

_____

Does it involve working with lead or other metals?
- No 0
- Yes (lead) 1
- Other metals (specify) _____ 2

4.5 Do you smoke in the house? Yes ___ No ___

If yes, how many cigarettes per day? ___

Part 5: The Father

5.1 And Mr. X how old are you? (years) ___

5.2 Where were you born?
- England, Scotland, N.Ireland, Wales 1
- Europe (outside UK) 2
- Other (specify) _____ 3

5.3 At what age did you leave school? (years) ___

Did you gain any qualifications?
- None 0
- CSE/O level 1
- A level 2
- Secretarial or technical qualifications 3
- Teacher training 4
- University (not completed) 5
- Degree 6
- Post graduate 7
- Other (specify) _____ 8

5.4 What is your job?

Give details of job (as fully as possible) if unemployed give details and dates of last job.

_____

_____

_____

Does (or did) it involve working with lead or other metals?
- No 0
- Yes (lead) 1
- Other metals (specify)_____ 2

_____

5.5 Do you smoke in the house? Yes / No

If yes, how many cigarettes per day? ___

Part 6 - The Child

6.1	Sex of child	M F	
6.2	Was X born on date due? On time Up to two weeks early More than two weeks early Up to two weeks late More than two weeks late Not known	1 2 3 4 5 9	
6.3	What did X weigh at birth? Less than 3lbs 3-5 lbs 5-6.5lbs 6.5-8lbs 8-10lbs More than 10lbs Not known	1 2 3 4 5 6 9	
6.4	Were there any complications of birth or delivery? No Yes Not known If yes, give details_____ _____ _____ _____	0 1 9	

6.5	Is **X** currently under the doctor for anything Yes | No | If yes, what type of treatment_____ _____ How often have you taken him/her to your GP in the last twelve months and what sorts of reasons? None 0 1-3 times 1 4-7 times 2 More than 7 times 3 Not known 9 _____ _____ _____	☐ ☐
6.6	How is his/her general health? Good, no problems 1 Occasional minor illnesses 2 Frequent minor illnesses (coughs, colds, stomach upsets) 3 Chronic problems (eczema, asthma, allergies) Chronic disability (diabetes) 4 Other (specify) 5 Not known 9	☐
6.7	Has **X** ever stayed overnight in hospital? Yes | No | If yes, reason_____ _____ length of stay (days)_____ At what age (months) _____ Over the last twelve months has **X** had to have outpatient hospital treatment at all? Yes | No |	☐ ☐☐ ☐☐ ☐

6.8	Has X ever had a head injury that worried you? Never 0 Minor, no loss of consciousness 1 Unconscious less than 15 minutes 2 Unconscious more than 15 minutes 3 Not known 9	
6.9	In the last twelve months have you given X any tablets or medicine for a period of more than a week? No 0 Yes, during the last 12 months 1 Yes, in use now 2 Not known 9	
6.10	Has X been immunised? Yes 1 No 0 Not known 9 If yes, which of the following have been given? Diptheria, tetanus, poliomyelitis, whooping cough (delete as necessary) 1st inoculation (approx. age 3-6 months) 1st booster (6 weeks later) 2nd booster (6 months later) Measles Inoculation (1-2 years)	D T P W

6.11 Did you have any problems feeding X as a baby?

No	0
Yes	1
Not known	9

If yes, what type of problems?

_____

_____

_____

Did you bottle or breast feed X?

Breast	1
Bottle	2

If breast fed, until what age?

Up to 6 weeks	1
Up to 12 weeks	2
Up to 6 months	3
Up to 12 months	4
More than 12 months	5
Not known	9

If bottle, until what age?

Up to 6 weeks	1
Up to 12 weeks	2
Up to 6 months	3
Up to 12 months	4
More than 12 months	5
Not known	9

6.12 Does X suck his/her thumb/finger/s? Yes | No

If yes:

at night, occasionally	1
at night, often	2
during the day, occasionally	3
during the day, often	4

If yes, does X

suck the thumb only?	1
or thumb and finger/s?	2

Does X use a dummy? Yes | No

If yes:

at night, occasionally	1
at night, often	2
during the day, occasionally	3
during the day, often	4

6.13	Some children eat or suck unusual things that aren°t food. Things like plasticine or soil or coal, or chewing small toys, or sucking their cots, etc. show list and record in box	
6.14	Has X got a good appetite? Yes / No No eating problems 0 Some difficulties (has some fads or poor appetite) 1 Marked difficulties (very poor appetite or refusal to eat all but a few items) 2 3 Does X eat snacks (not sweets) etc between meals? Yes / No Does X eat sweets between meals? Yes / No If yes, how many per day? Have you seen X eat sweets which have been dropped on the floor? Yes / No	
6.15	Are X's hands usually washed before meals? Yes 1 No 0 Not unless noticeably dirty 2 Don't know 9 And before snacks as well? Yes 1 No 0 Not unless noticeably dirty 2 Dont know 9 When X gets up? Yes / No X goes to bed? Yes / No Is the normal hand washing done at a basin with running water or with a flannel? Running water Yes / No Flannel Yes / No	

6.16	Does X have any sleep problems? No 0 Occasionally 1 Regularly 2 If yes, what sorts of problems? _____ _____ _____	☐
6.17	Does X have his/her own bedroom? sleeps alone/own room 1 sleeps with parents/own bed/cot 2 sleeps with parents/parents bed 3 sleeps with sibs/own bed/cot 4 sleeps with sibs/shares bed 5 sleeps with sibs in parents room 6 any other sleeping arrangements/own bed/cot 7 any other sleeping arrangements/shares bed 8 not known 9 Does X have a set bedtime? Yes / No	☐ ☐
6.18	Does X have any temper tantrums? How long do they last? No tantrums 0 Brief tantrums (less than 5 mintues) 1 Tantrums last 5-15 minutes 2 Tantrums last longer than 15 minutes 3 How often does he/she have them? Less than one a day 1 More than one a day 2 _____ _____	☐ ☐
6.19	How active is X? Underactive, spends most of his/her time with one toy 1 Not markedly active 2 Active 3 Very active 4 Hyperactive, constantly changing activities 5	☐

6.20	Is X restless when you take him shopping, does he/she rush around and touch things? No 0 Occasionally 1 Regularly 2 Needs to be restrained 3 Not known 4	☐
6.21	Usually, how long will X play with and concentrate on one toy, how long will he/she stick to one thing if he/she is really interested (not TV)? concentrates usually for more than 15 mins 1 concentrates usually for 5-15 mins 2 concentrates usually never more than than a few minutes 3	☐
6.22	What type of toys does X play with most (specify type, especially cuddly toys, and number) _____ _____	☐ ☐
6.23	In which room(s) does X play most? Living room 1 Kitchen 2 Bedroom 3 Other (specify) (include combinations in order of frequency) 4 _____ _____ _____	☐ ☐ ☐ ☐ ☐

6.24	Does X go out of the house to any playgroup nursery or childminder (hours p.w. and address) Yes No If yes, specify _____ _____ _____	

In the last 6 months has the child put any of these into his/her mouth

		never	occasionally (<1/week)	regularly (>1/week)	frequently (>1/day)
Painted wood objects:	furniture				
	cot				
	windowsill (interior)				
	windowsill (exterior)				
	railings (interior)				
	railings (exterior)				
	fences (exterior)				
	toys				
	other (specify)				
Unpainted wooden objects					
Painted metal objects					
Unpainted metal objects					
Soft objects:	clothing (own)				
	clothing (others)				
	blankets				
	soft toys				
	slippers/shoes				
	rags/clothes				
	other				
Plasticine/Playdoh					
Earth/soil					
Ashes (interior)					
Ashes (exterior)					
Coal dust (interior)					
Coal dust (exterior)					
Newspaper/comics					
Drawing paper					
Pencils					
Soap/toiletries					
Paint chips (domestic interior)					
Paint chips (domestic exterior)					

	Never	Occasionally (<1/week)	Regularly (>1/week)	Frequently (>/day)
Paints (childrens)				

Anything else (specify)

_____

_____

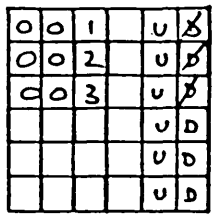
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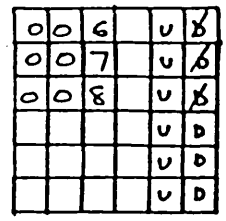
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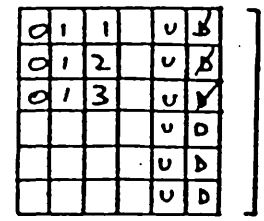
DATE: 27.11.84 DAY: M (T) W Th F S S

PROJECT DAY: 1 BLOCK: (1) 2 3 4 5 6 7

TEAM 1 TEAM 2 TEAM 3



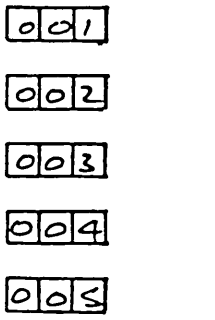


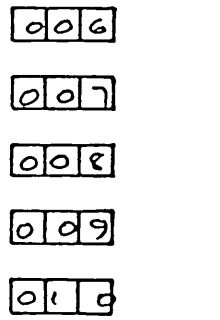


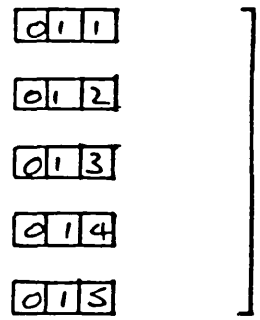
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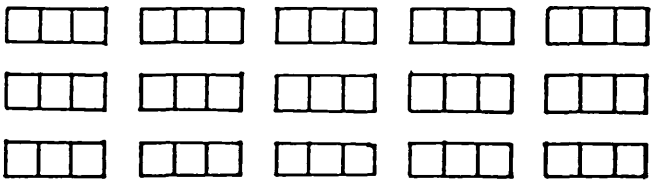
VT







BMRB



PAYMENT SCHEDULE

EHBRG

DIARY SHEET

NOTES:

DUST & DIET STUDY

FAMILY NAME: _____

CHILD'S NAME: _____

ADDRESS: MANOR FARM ROAD

ALOCKS GREEN

B11 2HT

(91 2E)

TELEPHONE: _____

EHBRG	0 0 1 1 5 2
IC	0 0 1
BMRB	0 0 1
DOE	0 0 1

TEAM: (1) 2 3

BLOCK: (1) 2 3 4 5 6 7

				TICK WHEN DONE	
DATE TO SET UP MONITORS:	2,7	1,1	8,4	☐	IC
DATE TO TAKE DOWN MONITORS:	0,4	1,2	8,4	☐	IC
DATE FOR BMRB BRIEFING	2,7	1,1	8,4	☐	BMRB
DATE FOR BMRB CHECK VISIT	2,9	1,1	8,4	☐	BMRB
DATE BMRB COLLECTION	0,5	1,2	8,4	☐	BMRB
DATE FOR BMRB DELIVERY TO STORE	0,6	1,2	8,4	☐	BMRB
DATE FOR VIDEOTAPING	2,8	1,1	8,4	☐	EHBRG
DATE FOR PAYMENT [CQ][CS]	1,3	1,2	8,4	☐	EHBRG

EHBRG CHECKLIST:

☐ INITIAL LETTER	☐ FOOD COLLECTED	☐ THANK YOU LETTER
☐ REMINDER LETTER	☐ VT RECEIVED	☐ SUMMARY RESULTS
☐ IC TOLD	☐ VT PASSED TO RATER	☐ IC FORMS
☐ BMRB TOLD	☐ VT RATED	☐ BMRB FORMS
☐ VT TOLD	☐ PAYMENT MADE	☐ VT FORMS

PLEASE RETURN TO EHBRG WHEN COMPLETE

PROJECT 178 D.O.E. DUST

HOUSE NUMBER

NAME

ADDRESS

CHILD'S NAME

AIR MONITORING

			BEDROOM		LIVING ROOM		OUTSIDE	
DAY	TIME	TIME ELAPSED	FLOW RATE	VOL	FR	VOL	FR	VOL
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....

HANDWIPES

DAY	NO. WIPES	TIME SINCE WASHING (HRS)	COMMENTS
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....
.....	.....	.....	.....

DUST LOADING

	DAY	AREA(m^2)	WEIGHT(g)			
			Thimble	Thimble & Sample	Sample	Dust Loading
BEDROOM (DSB)						
LIVING ROOM (DSL)						

ACTIVITY

DAY	TIME (Hrs)				
	AT HOME			AWAY FROM HOME	
	INDOORS		OUTDOORS (IN GARDEN)	INDOORS	OUTDOORS
	AWAKE	ASLEEP			
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....

ADDITIONAL SAMPLES

DAY

Household Cleaner (VAC).....

Soil (SOI).....

Door Mat (MAT).....

Road Dust (RDD).....

Pavement Dust (PAV).....

APPENDIX C INSTRUMENTAL OPERATING CONDITIONS

Table C1 ICPAES operating conditions

ICPAES instrument	Applied Research Laboratories, ARL 34000C, 1 m vacuum spectrometer with PDP computer
Forward power	1.2 kW at MHz
Viewing height	A 4.0 mm square window centred 14 mm above the load coil
Torch	Fassel type
Argon gas flow rates:	
Coolant	12.01 min^{-1}
Auxillary	0.41 min^{-1}
Injector	1.011 min^{-1}, humidified
Spray chamber	Scott double-pass type
Nebuliser	Concentric glass
Solution uptake rate	0.81 min^{-1} (unpumped)
Uptake tube	350 x 0.5 mm i.d. polyethylene
Nebuliser tip wash	0.5 ml of DIW containing 1% v/v Photo-flo between sample solutions
Pre-flush integration time	20 s and 3 x 5 s

Table C2 AAS operating conditions

AAS instrument	Perkin Elmer PE 5000
Analytical wavelength for Pb	217.0 nm
Slit width	0.7 nm
Lamp current	7 mA
Background correction	Deuterium arc
Detection limit using impact bead	0.05 µg/ml
Flame	Air-acetylene
Calibrators	S1 = 4.00 µg/ml
	S2 = 16.00 µg/ml
	S3 = 40.0 µg/ml
Number of determinations/sample	3

APPENDIX D BEDROOM, PLAYROOM AND EXTERNAL AIR LEAD CONCENTRATIONS

Figure D1 Air lead concentrations for Houses 1-6

Figure D2 Air lead concentrations for Houses 7-8, 10-13

Figure D3 Air lead concentrations for Houses 14-19

Figure D4 Air lead concentrations for Houses 20-25

Figure D5 Air lead concentrations for Houses 26-30, 39

Figure D6 Air lead concentrations for Houses 40, 42-46

Figure D7 Air lead concentrations for Houses 47-52

Figure D8 Air lead concentrations for Houses 53-56, 58-60

Figure D9 Air lead concentrations for Houses 62-67

Figure D10 Air lead concentrations for Houses 68, 70-74

Figure D11 Air lead concentrations for Houses 75-76,78-81

Figure D12 Air lead concentrations for Houses 82-87

Figure D13 Air lead concentrations for Houses 88-93

Figure D14 Air lead concentrations for Houses 94-96,98-100

Figure D15 Air lead concentrations for Houses 101, 103-106

Table D1 Bedroom air lead concentration

Table D2 Playroom air lead concentration

Table D3 External air lead concentration

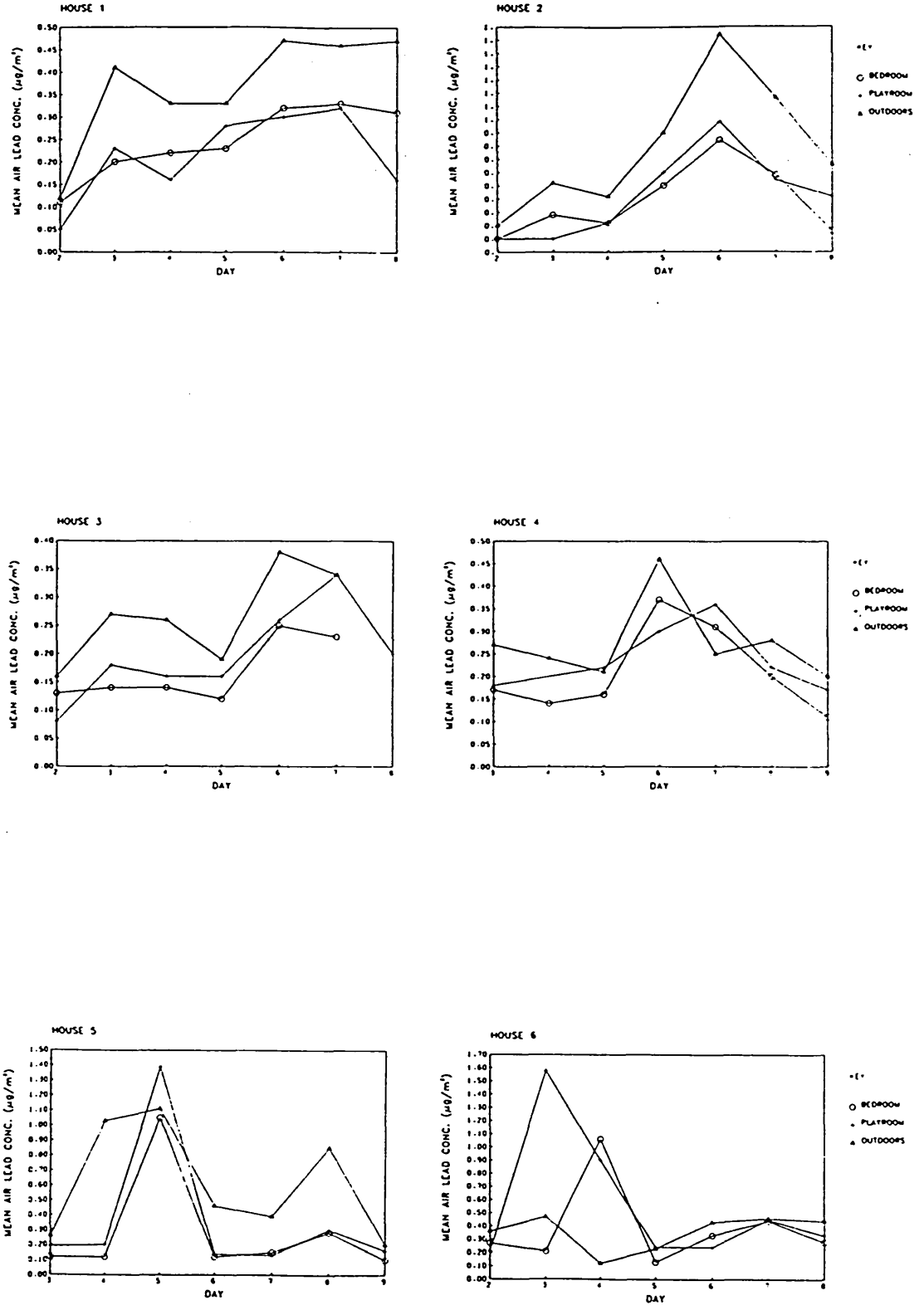


Figure D1 Air lead concentrations for Houses 1-6

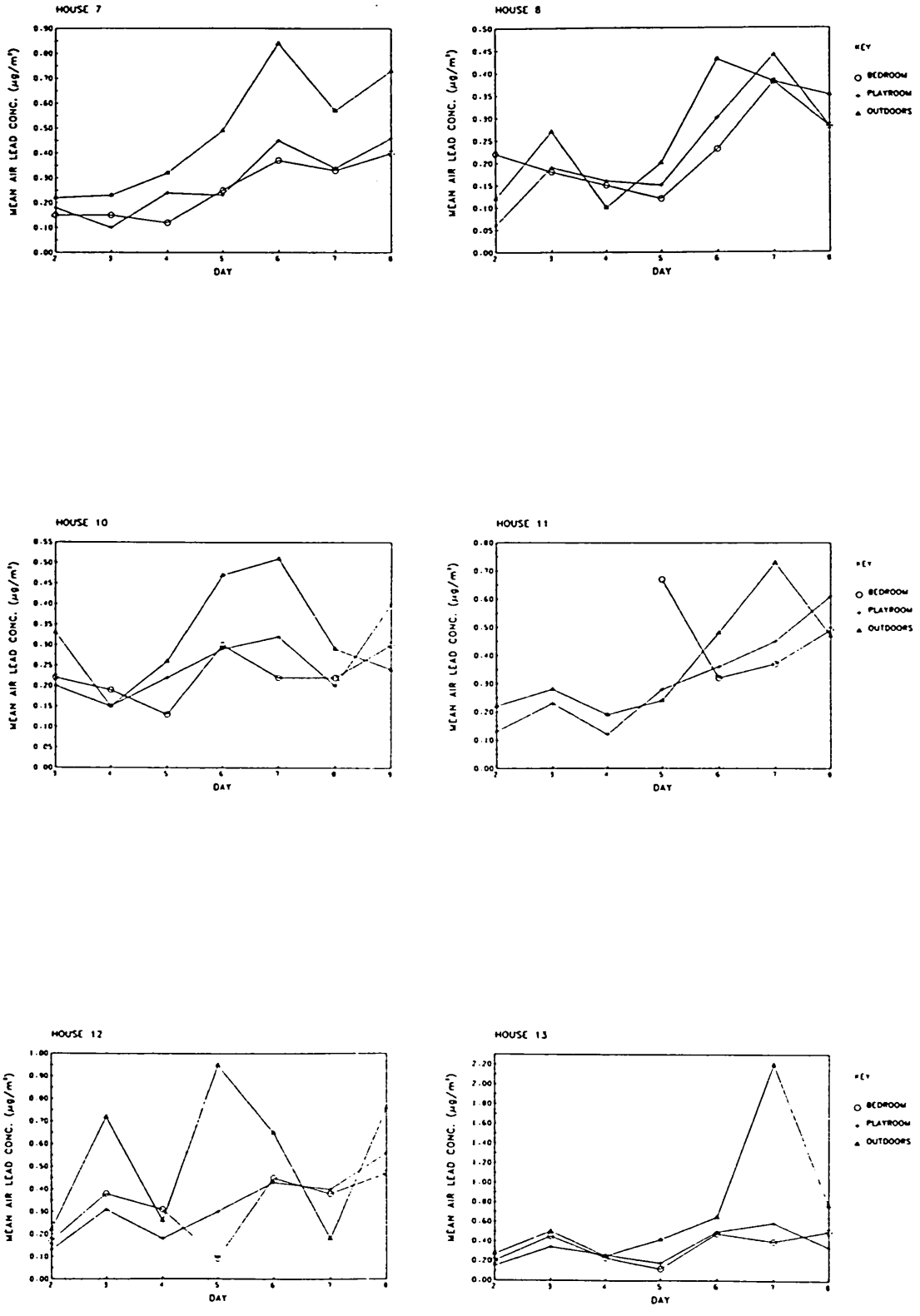


Figure D2 Air lead concentrations for Houses 7-8, 10-13

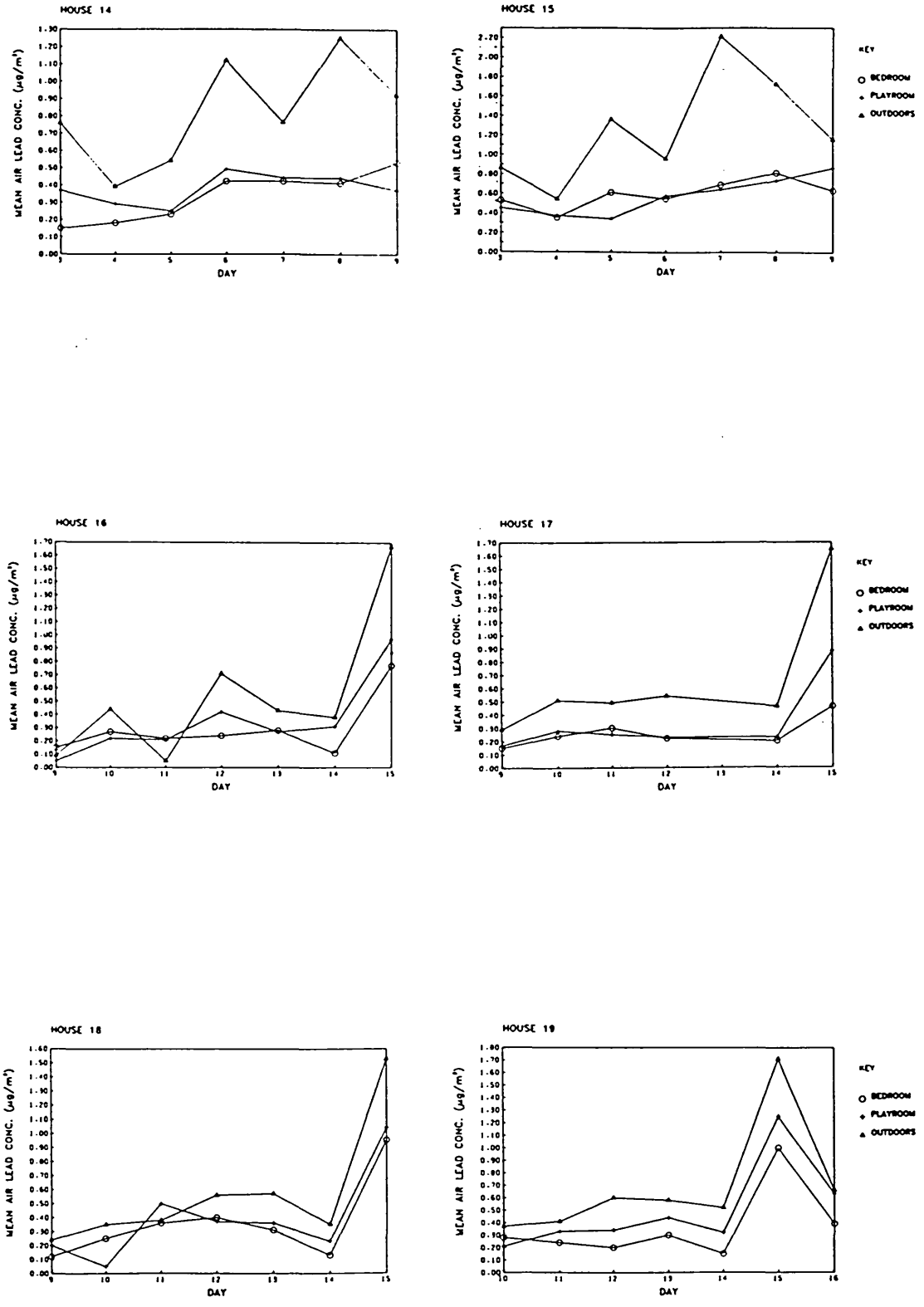


Figure D3 Air lead concentrations for Houses 14-19

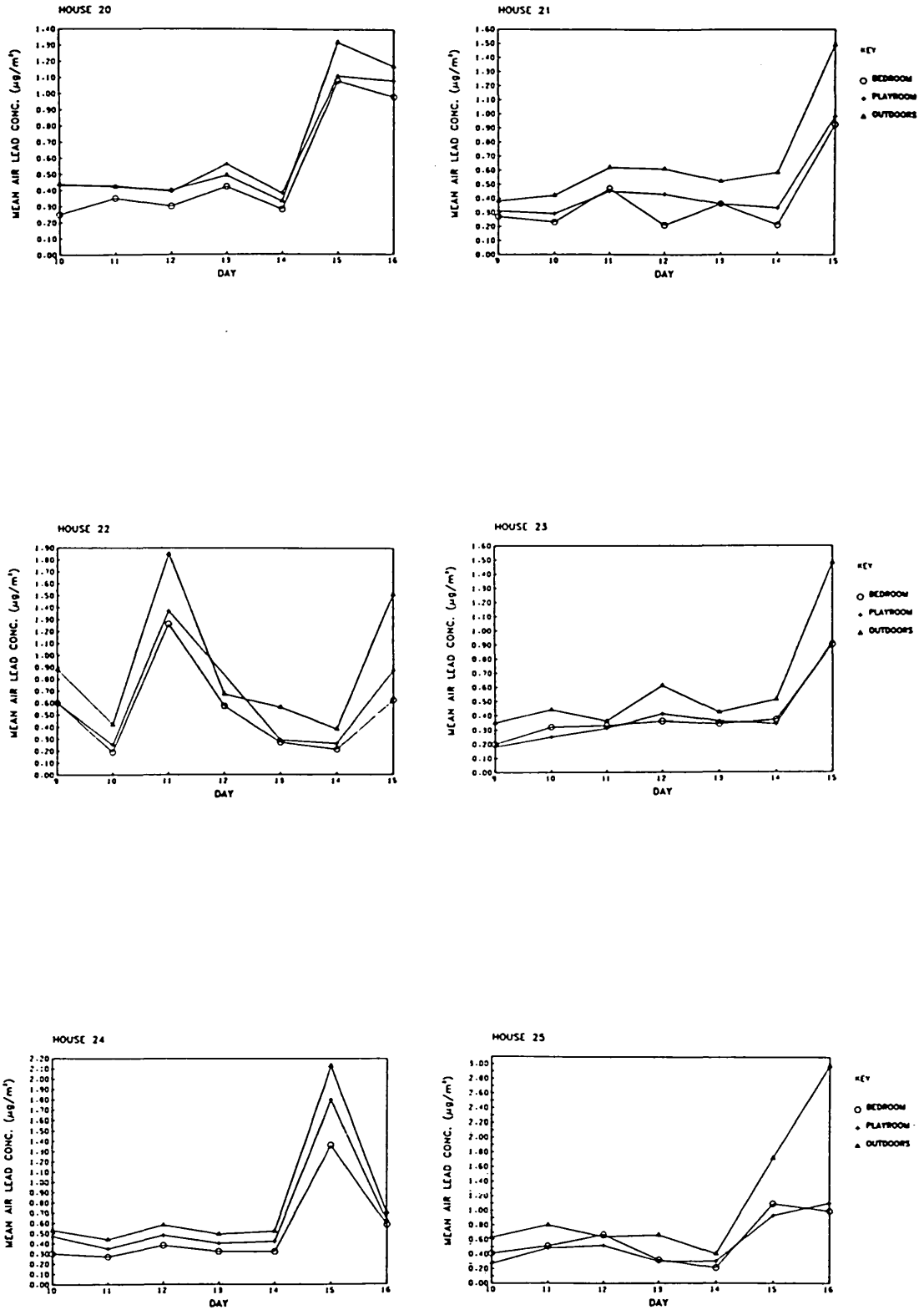


Figure D4 Air lead concentrations for Houses 20-25

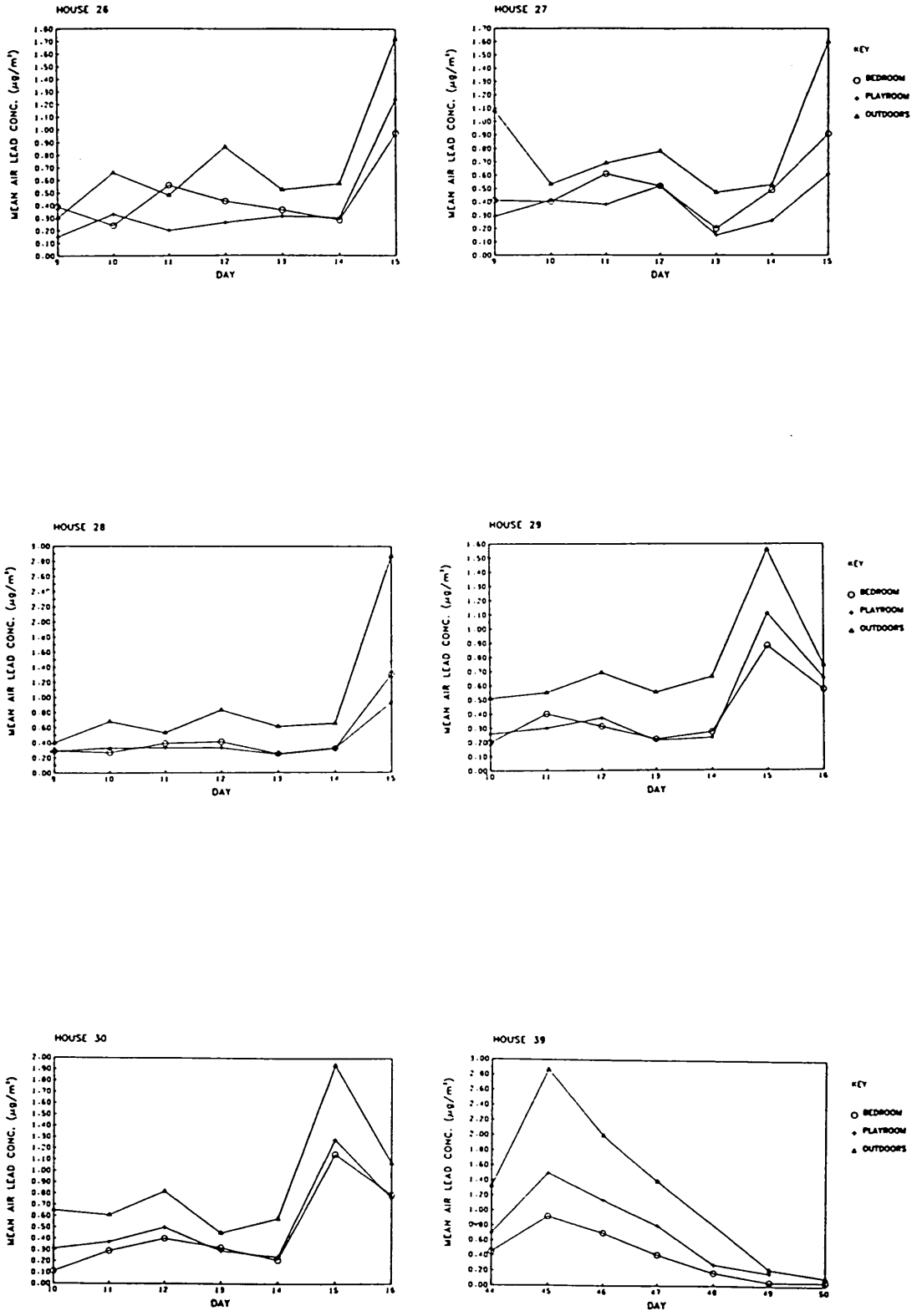


Figure D5 Air lead concentrations for Houses 26-30, 39

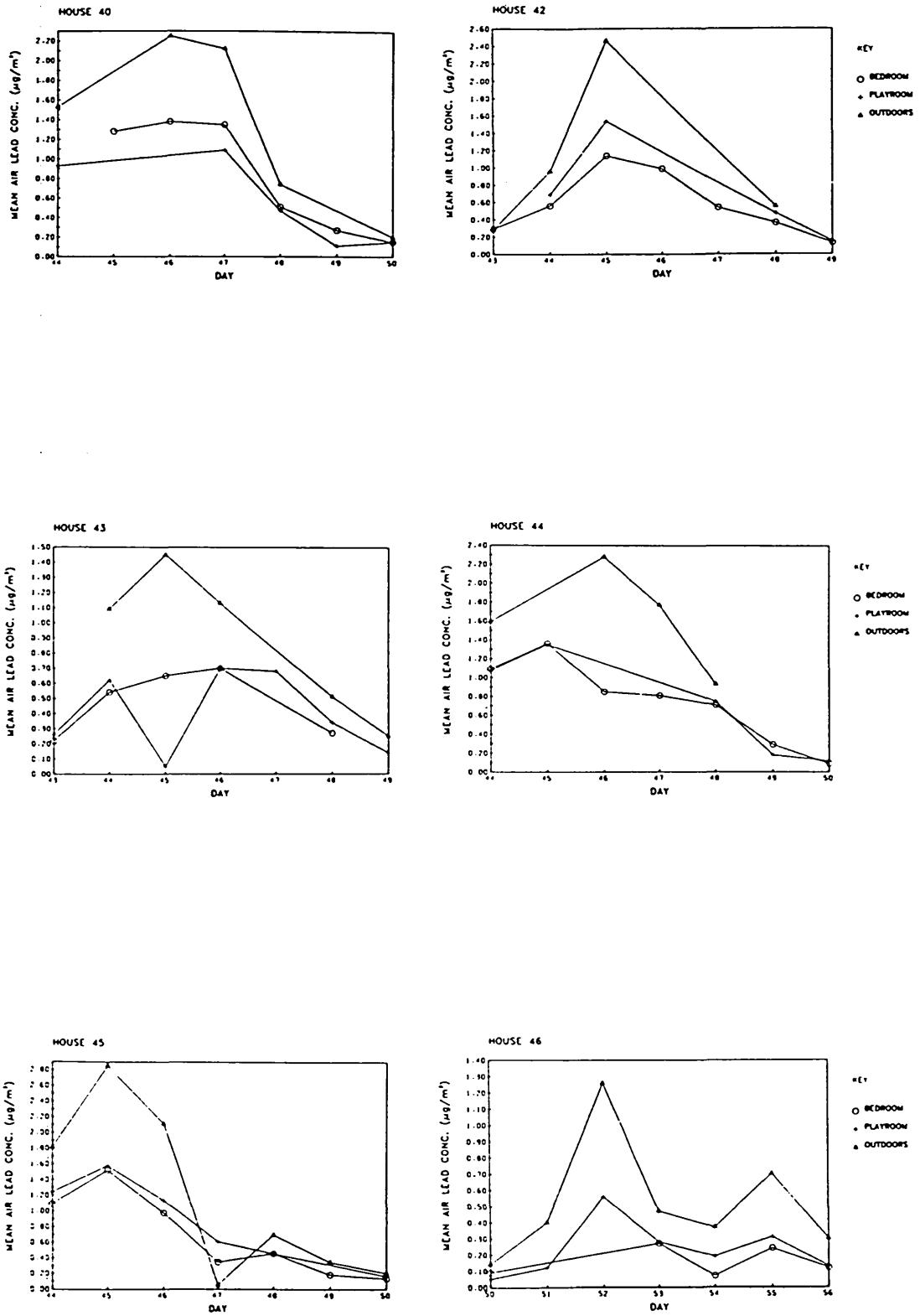


Figure D6 Air lead concentrations for Houses 40, 42-46

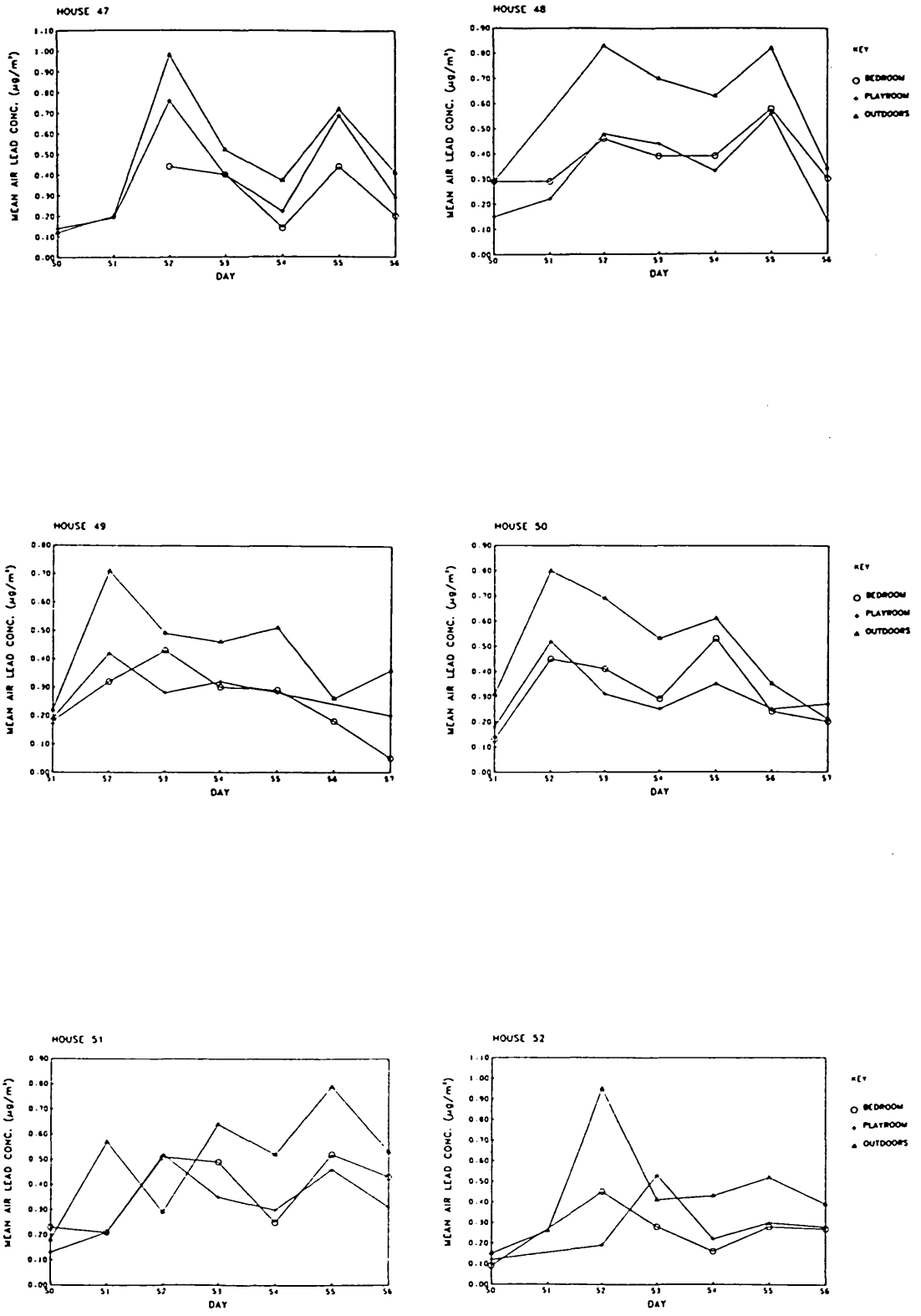


Figure D7 Air lead concentrations for Houses 47-52

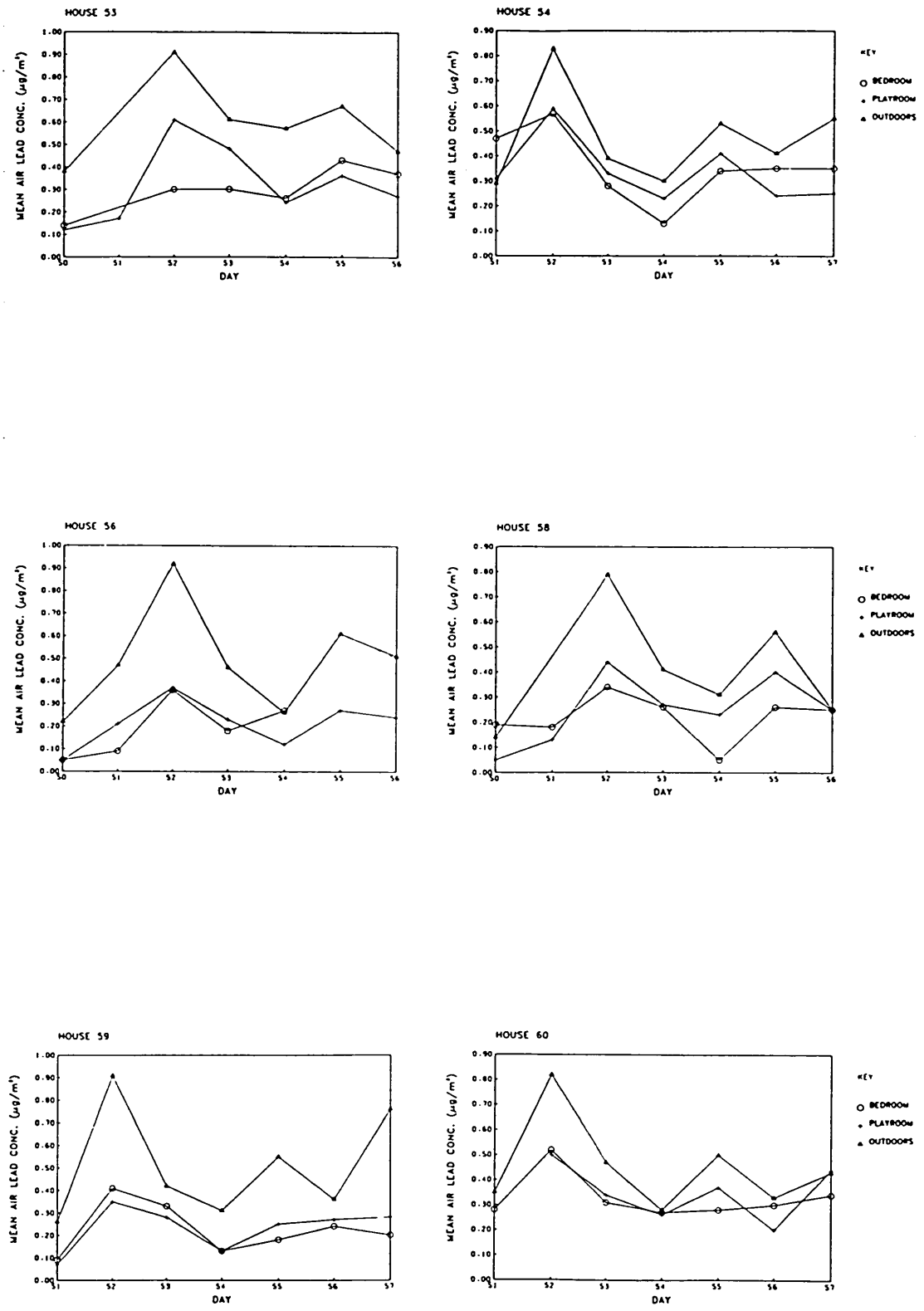


Figure D8 Air lead concentrations for Houses 53-56, 58-60

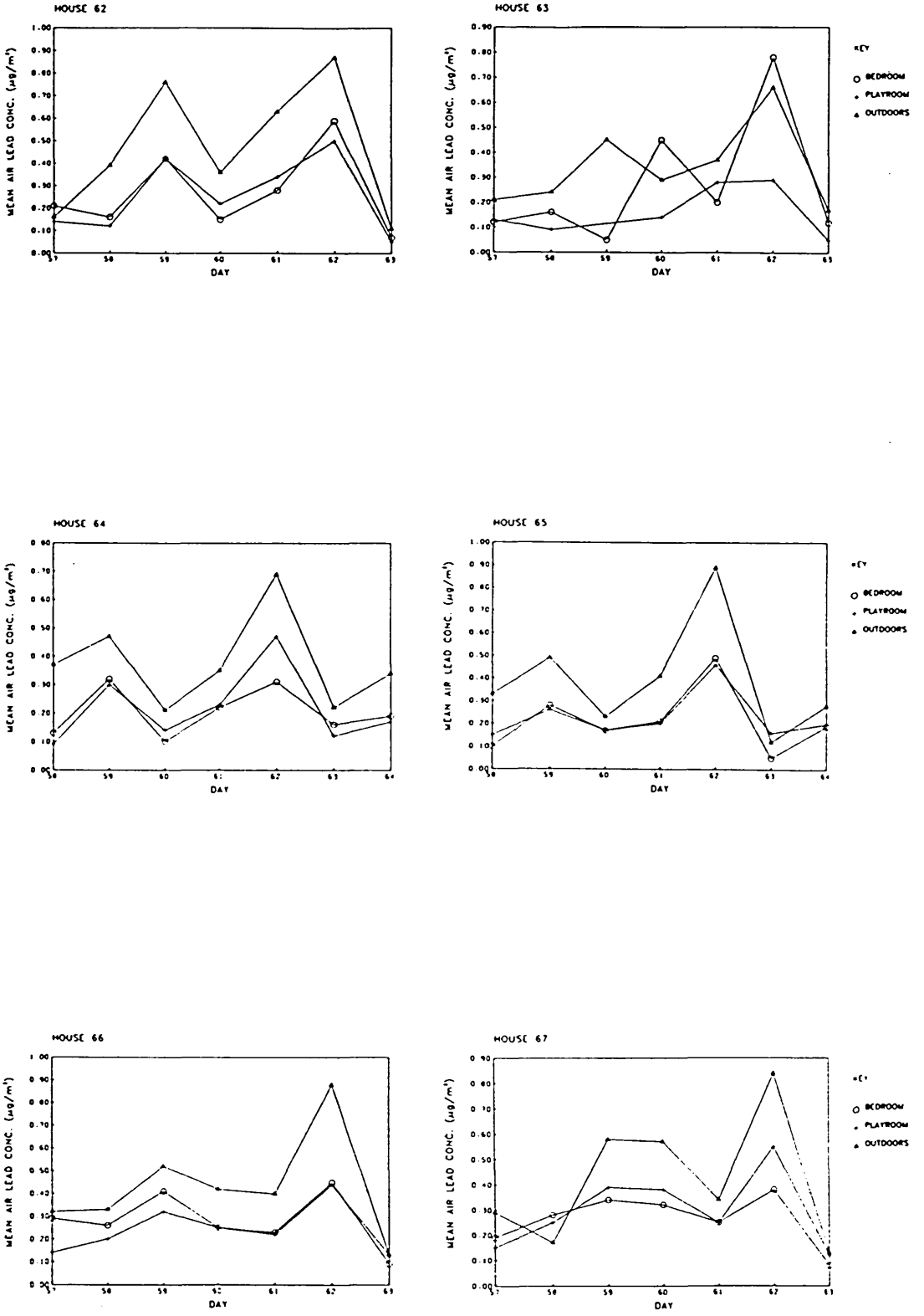


Figure D9 Air lead concentrations for Houses 62-67

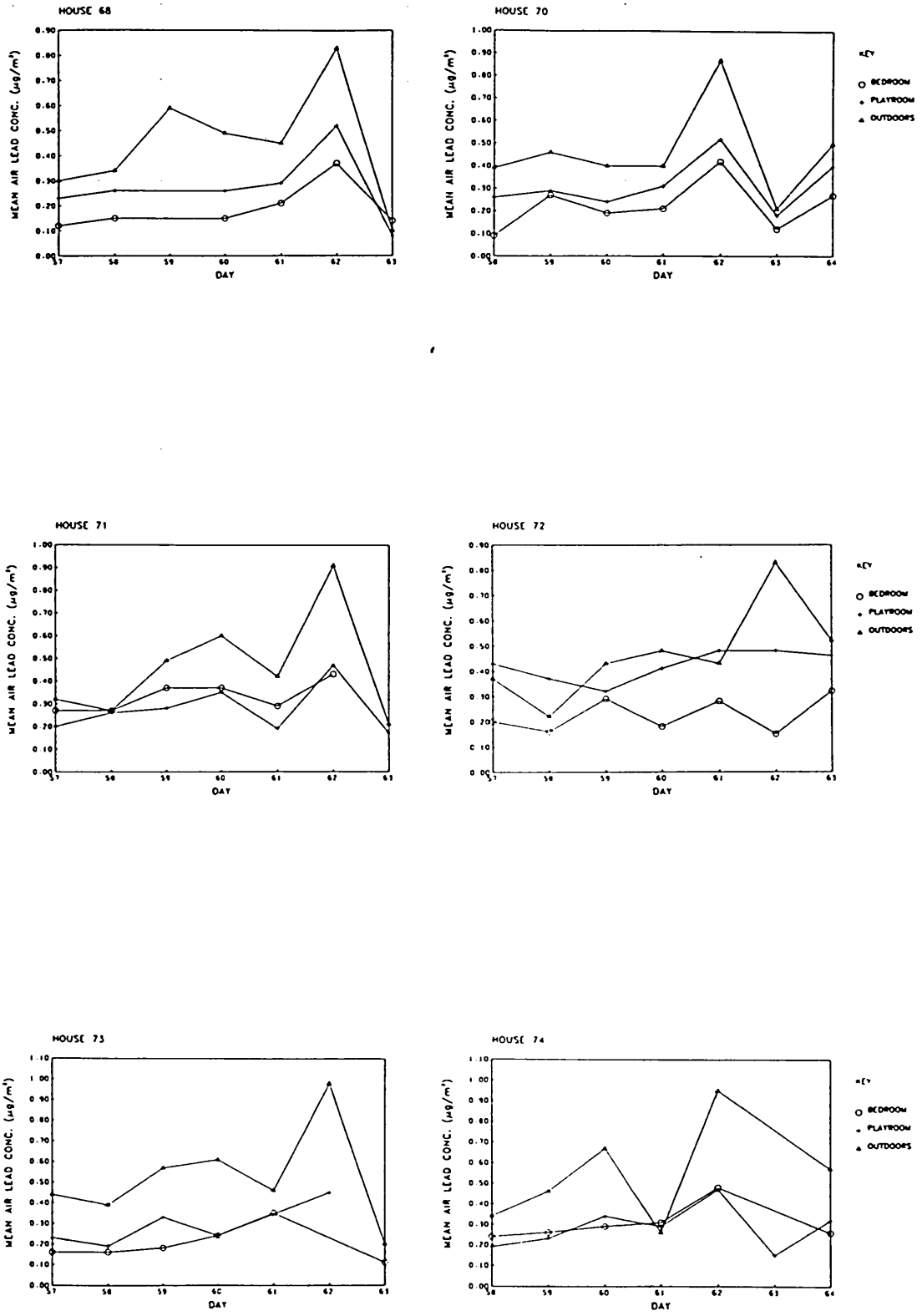


Figure D10 Air lead concentrations for Houses 68, 70-74

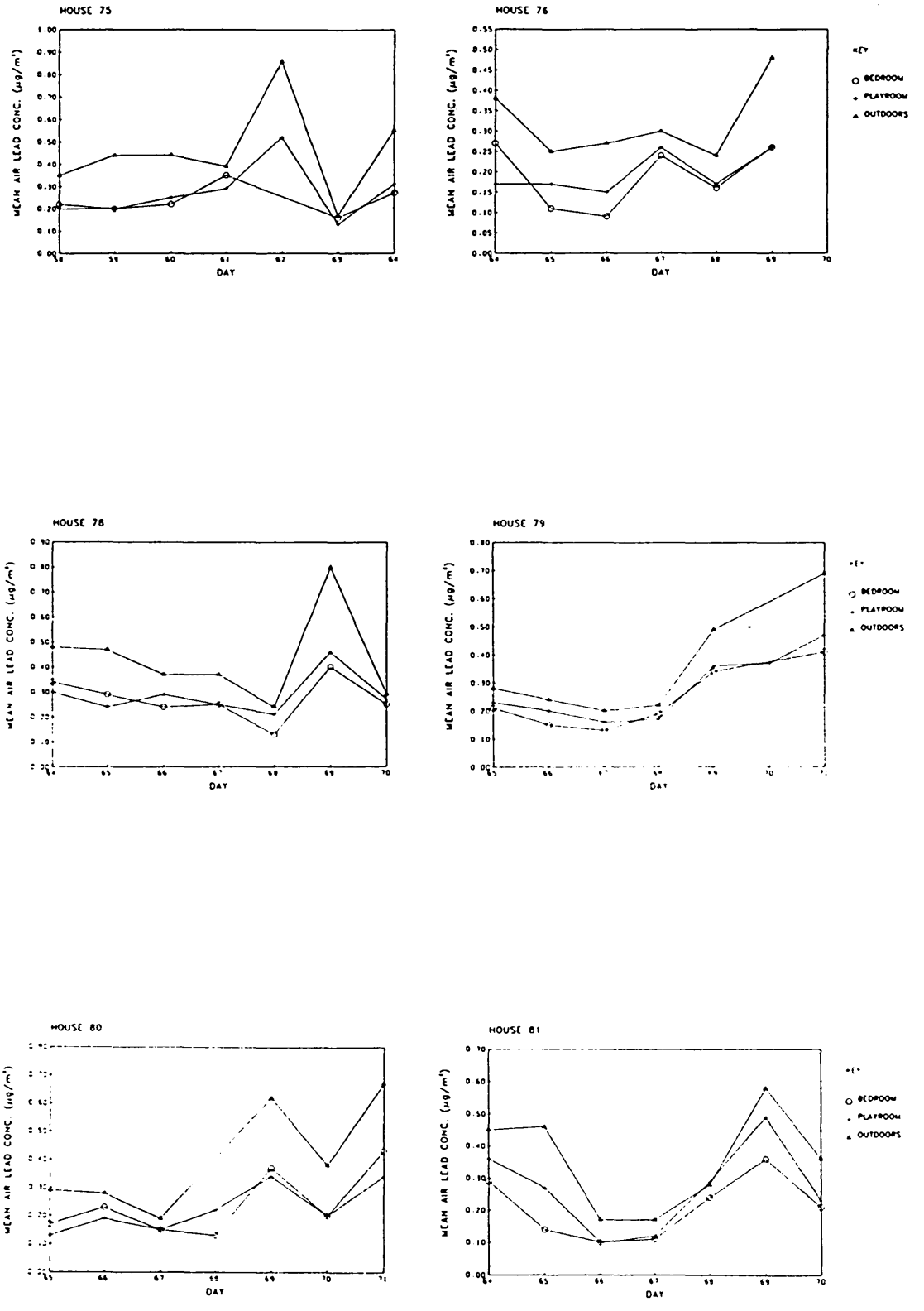


Figure D11 Air lead concentrations for Houses 75-76, 78-81

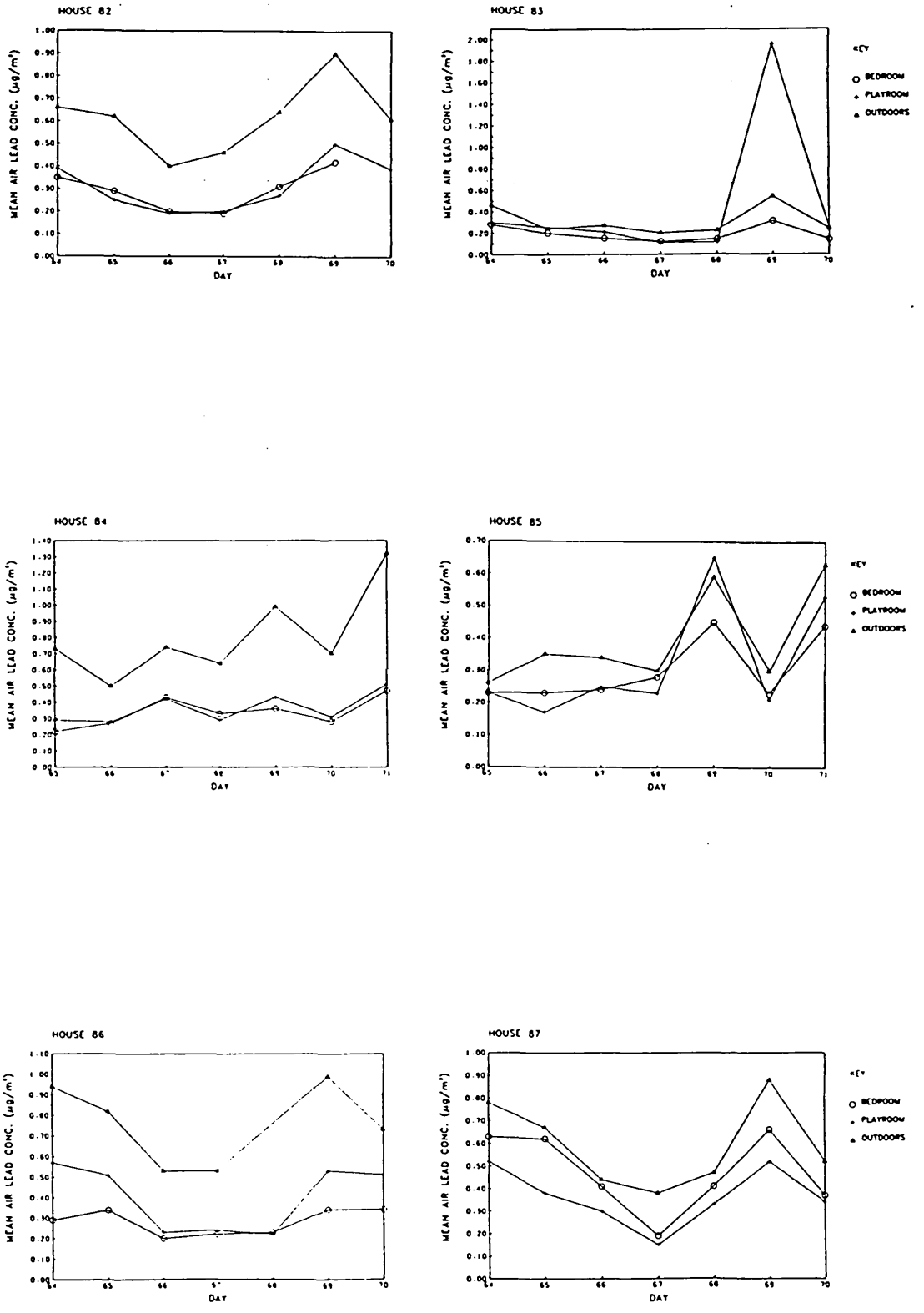


Figure D12 Air lead concentrations for Houses 82-87

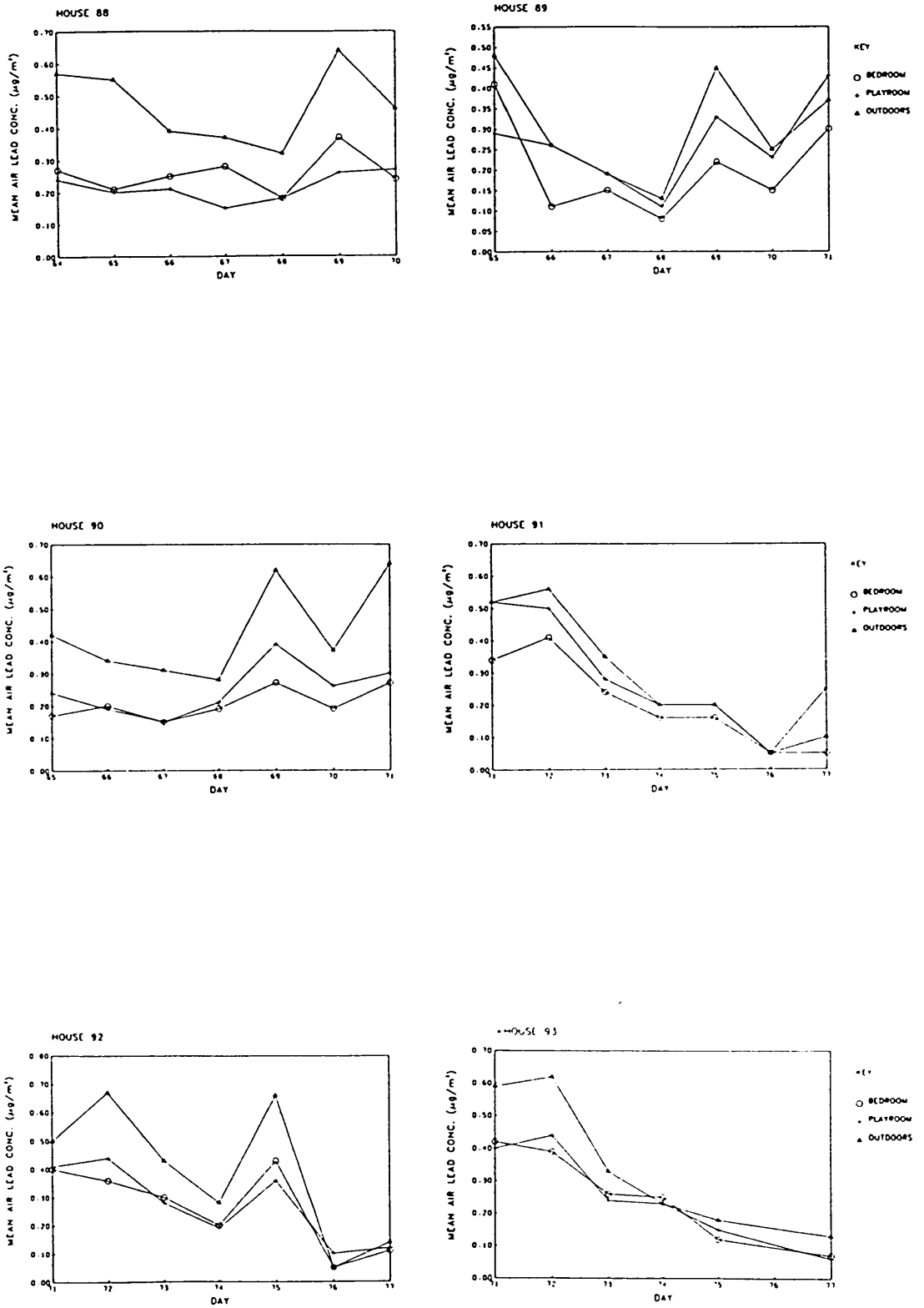


Figure D13 Air lead concentrations for Houses 88-93

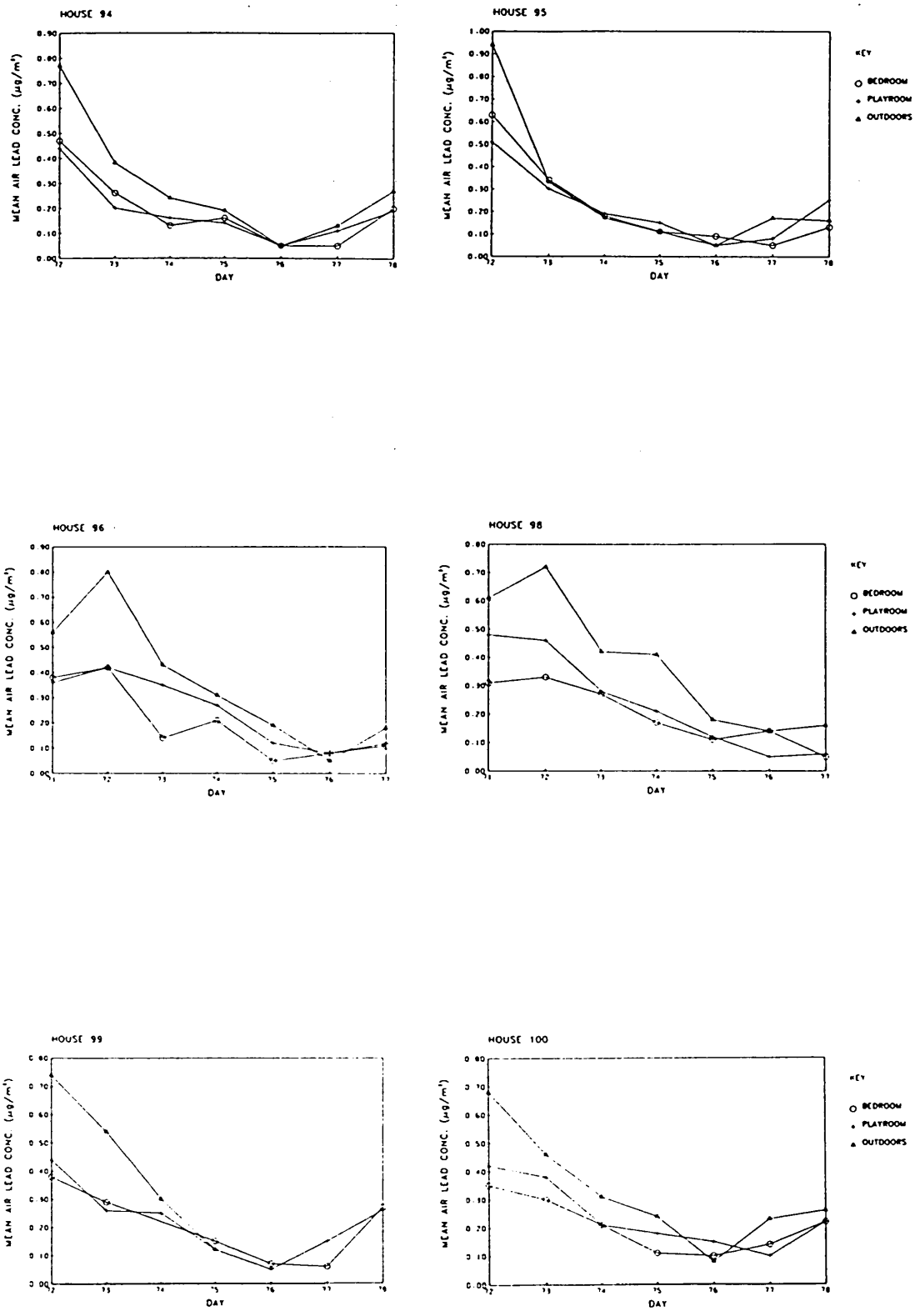


Figure D14 Air lead concentrations for Houses 94-96, 98-100

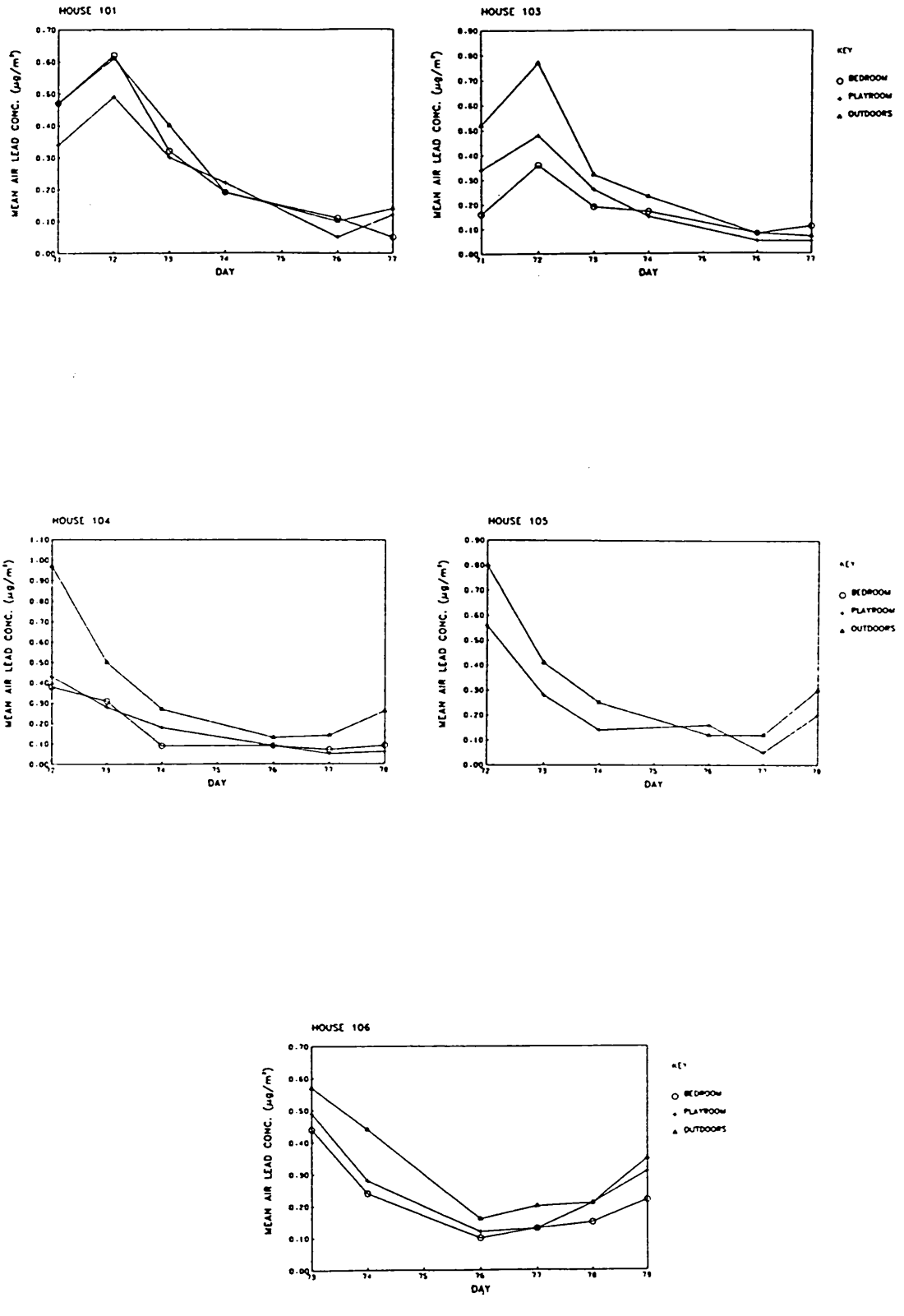


Figure D15 Air lead concentrations for Houses 101, 103-106

Table D1 Bedroom air lead concentration: μg/m³

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	MEAN
1	0.11	0.20	0.22	0.23	0.32	0.33	0.31	0.25
2	*	0.14	0.11	0.25	0.42	0.29	0.08	0.19
3	0.13	0.14	0.14	0.12	0.25	0.23	**	0.17
4	0.17	0.14	0.16	0.37	0.31	0.20	0.11	0.21
5	0.12	0.12	1.05	0.12	0.15	0.28	0.10	0.28
6	0.27	0.21	1.06	0.13	0.33	0.44	0.28	0.39
7	0.15	0.15	0.12	0.25	0.37	0.33	0.40	0.25
8	0.22	0.18	0.15	0.12	0.23	0.38	0.28	0.22
10	0.22	0.19	0.13	0.30	0.22	0.22	0.30	0.22
11	**	**	**	0.67	0.32	0.37	0.49	0.46
12	0.17	0.38	0.31	0.09	0.45	0.38	0.47	0.32
13	0.20	0.45	0.23	0.12	0.48	0.40	0.50	0.34
14	0.15	0.18	0.23	0.42	0.42	0.41	0.53	0.33
15	0.53	0.35	0.61	0.55	0.69	0.81	0.63	0.60
16	0.15	0.27	0.22	0.24	0.28	0.11	0.77	0.29
17	0.15	0.24	0.30	0.22	**	0.20	0.46	0.26
18	0.12	0.25	0.36	0.40	0.31	0.13	0.95	0.36
19	0.28	0.24	0.20	0.30	0.15	1.00	0.39	0.37
20	0.25	0.35	0.30	0.42	0.28	1.08	0.98	0.52
21	0.27	0.23	0.47	0.21	0.36	0.21	0.92	0.38
22	0.60	0.19	1.26	0.57	0.27	0.21	0.62	0.53
23	0.20	0.32	0.33	0.36	0.34	0.37	0.90	0.40
24	0.30	0.27	0.38	0.32	0.32	1.36	0.58	0.50
25	0.41	0.51	0.67	0.32	0.21	1.09	0.99	0.60
26	0.39	0.24	0.56	0.43	0.36	0.28	0.97	0.46
27	0.41	0.40	0.60	0.52	0.20	0.49	0.91	0.51
28	0.29	0.27	0.39	0.41	0.25	0.32	1.30	0.46
29	0.20	0.40	0.31	0.22	0.27	0.88	0.57	0.41
30	0.11	0.29	0.40	0.32	0.21	1.15	0.79	0.47
31	**	**	**	0.80	**	**	**	**
32	**	0.35	**	0.61	**	**	**	**
33	**	**	**	**	**	0.50	0.17	**
34	**	**	0.97	**	**	**	0.11	**
35	**	**	**	**	**	**	**	**
37	**	**	**	**	**	**	**	**
39	0.45	0.92	0.70	0.41	0.17	*	*	0.39
40	**	1.28	1.38	1.35	0.51	0.27	0.15	0.82
42	0.29	0.56	1.14	0.99	0.54	0.36	0.13	0.57
43	0.22	0.54	0.65	0.70	**	0.27	**	0.48
44	1.09	1.36	0.85	0.81	0.71	0.29	0.09	0.74
45	1.09	1.52	0.97	0.34	0.45	0.18	0.13	0.67
46	0.09	**	**	0.27	0.07	0.24	0.12	0.16
47	**	**	0.44	0.40	0.14	0.44	0.20	0.32
48	0.29	0.29	0.46	0.39	0.39	0.58	0.30	0.39
49	0.18	0.32	0.43	0.30	0.29	0.18	*	0.25
50	0.13	0.45	0.41	0.29	0.53	0.24	0.20	0.32
51	0.23	0.21	0.51	0.49	0.25	0.52	0.43	0.38
52	0.09	**	0.45	0.28	0.16	0.28	0.27	0.26
53	0.14	0.30	**	0.30	0.26	0.43	0.37	0.30

contd./

Table D1 contd.

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	MEAN
54	0.47	0.57	0.28	0.13	0.34	0.35	0.35	0.36
56	*	0.09	0.36	0.18	0.27	**	**	0.19
58	0.19	0.18	0.34	0.26	*	0.26	0.25	0.22
59	0.09	0.41	0.33	0.13	0.18	0.24	0.20	0.23
60	0.28	0.52	0.31	0.27	0.28	0.30	0.34	0.33
62	0.21	0.16	0.47	0.15	0.28	0.59	0.07	0.27
63	0.12	0.16	*	0.45	0.20	0.78	0.12	0.27
64	0.13	0.32	0.10	0.22	0.31	0.16	0.19	0.20
65	0.10	0.28	0.17	0.21	0.49	*	0.19	0.21
66	0.29	0.26	0.41	0.25	0.23	0.45	0.09	0.28
67	0.19	0.28	0.34	0.32	0.25	0.38	0.08	0.26
68	0.12	0.15	**	0.15	0.21	0.37	0.14	0.19
70	0.09	0.27	0.19	0.21	0.42	0.12	0.27	0.22
71	0.27	0.27	0.37	0.37	0.29	0.43	**	0.33
72	0.20	0.16	0.29	0.18	0.28	0.15	0.32	0.23
73	0.16	0.16	0.18	0.24	0.35	**	0.11	0.20
74	0.24	0.26	0.29	0.31	0.48	**	0.26	0.31
75	0.22	0.20	0.22	0.35	**	0.16	0.27	0.24
76	0.27	0.11	0.09	0.24	0.16	0.26	**	0.19
78	0.34	0.29	0.24	0.25	0.13	0.40	0.25	0.27
79	0.21	0.15	0.13	0.19	0.34	**	0.41	0.24
80	0.17	0.23	0.15	0.13	0.37	0.20	0.43	0.24
81	0.29	0.14	0.10	0.11	0.24	0.36	0.21	0.21
82	0.35	0.29	0.20	0.19	0.31	0.42	**	0.29
83	0.28	0.20	0.15	0.12	0.14	0.31	0.14	0.19
84	0.22	0.27	0.43	0.33	0.36	0.28	0.47	0.34
85	0.23	0.23	0.24	0.28	0.45	0.23	0.44	0.30
86	0.29	0.34	0.20	0.22	0.23	0.34	0.34	0.28
87	0.63	0.62	0.41	0.19	0.41	0.66	0.37	0.47
88	0.27	0.21	0.25	0.28	0.18	0.37	0.24	0.26
89	0.41	0.11	0.15	0.08	0.22	0.15	0.30	0.20
90	0.17	0.20	0.15	0.19	0.27	0.19	0.27	0.21
91	0.34	0.41	0.24	0.16	0.16	*	0.05	0.20
92	0.40	0.36	0.30	0.20	0.43	*	0.11	0.26
93	0.42	0.39	0.26	0.25	0.12	**	0.07	0.25
94	0.47	0.26	0.13	0.16	*	*	0.20	0.19
95	0.63	0.34	0.18	0.11	0.09	*	0.13	0.22
96	0.38	0.42	0.14	0.21	*	0.08	0.11	0.20
98	0.31	0.33	0.27	0.17	0.11	0.14	*	0.20
99	0.38	0.29	**	0.15	0.07	0.06	0.27	0.20
100	0.35	0.30	0.21	0.11	0.10	0.14	0.22	0.20
101	0.47	0.62	0.32	0.19	**	0.11	*	0.29
102	0.26	0.35	0.44	0.12	**	0.10	0.07	0.22
103	0.16	0.36	0.19	0.17	**	0.08	0.11	0.18
104	0.38	0.31	0.09	**	0.09	0.07	0.09	0.17
106	0.44	0.24	**	0.10	0.13	0.15	0.22	0.21

* Below detection limit
** Missing data

Table D2 Playroom air lead concentration: μg/m³

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	MEAN
1	*	0.23	0.16	0.28	0.30	0.32	0.16	0.21
2	0.05	*	0.11	0.30	0.49	0.27	0.21	0.21
3	0.08	0.18	0.16	0.16	0.26	0.34	0.20	0.20
4	0.18	**	0.22	0.30	0.36	0.22	0.17	0.24
5	0.19	0.20	1.39	0.14	0.13	0.30	0.16	0.36
6	0.21	1.58	0.90	0.24	0.24	0.45	0.33	0.56
7	0.18	0.10	0.24	0.23	0.45	0.34	0.46	0.29
8	0.06	0.19	0.16	0.15	0.30	0.44	0.28	0.23
10	0.20	0.15	0.22	0.29	0.32	0.20	0.40	0.25
11	0.13	0.23	0.12	0.28	0.36	0.45	0.61	0.31
12	0.13	0.31	0.18	0.30	0.43	0.40	0.56	0.33
13	0.15	0.34	0.26	0.18	0.50	0.59	0.34	0.34
14	0.37	0.29	0.25	0.49	0.44	0.44	0.37	0.38
15	0.45	0.37	0.34	0.58	0.64	0.73	0.86	0.57
16	*	0.22	0.21	0.42	0.27	0.31	0.97	0.35
17	0.17	0.28	0.25	0.23	**	0.23	0.88	0.34
18	0.20	*	0.50	0.37	0.36	0.23	1.04	0.39
19	0.21	0.33	0.34	0.44	0.32	1.25	0.63	0.50
20	0.43	0.43	0.39	0.56	0.38	1.11	1.08	0.63
21	0.31	0.29	0.45	0.43	0.36	0.33	0.98	0.45
22	0.60	0.25	1.37	**	0.29	0.26	0.87	0.61
23	0.18	0.25	0.31	0.41	0.36	0.34	0.92	0.40
24	0.47	0.35	0.48	0.40	0.42	1.80	0.62	0.65
25	0.27	0.48	0.52	0.30	0.30	0.93	1.10	0.56
26	0.15	0.33	0.20	0.26	0.31	0.30	1.24	0.40
27	0.29	0.41	0.38	0.52	0.15	0.26	0.61	0.37
28	0.29	0.33	0.33	0.33	0.24	0.32	0.93	0.40
29	0.26	0.30	0.37	0.21	0.23	1.11	0.65	0.45
30	0.31	0.37	0.50	0.29	0.24	1.28	0.76	0.54
31	**	0.57	**	**	**	**	**	**
32	**	0.42	**	**	**	**	0.14	**
33	0.34	**	**	**	**	**	**	**
34	**	**	**	**	**	**	**	**
35	**	**	**	**	**	**	**	**
37	**	**	**	1.04	**	**	**	**
39	0.70	1.50	1.14	0.80	0.28	0.17	**	0.77
40	0.93	**	**	1.09	0.47	0.11	0.15	0.55
42	**	0.69	1.54	**	**	0.47	0.15	0.71
43	0.26	0.62	*	0.70	0.68	0.34	0.14	0.40
44	1.07	1.35	**	**	0.75	0.18	0.12	0.69
45	1.24	1.58	1.13	0.60	0.45	**	0.16	0.86
46	*	0.12	0.56	0.28	0.19	0.31	0.13	0.23
47	0.14	0.19	0.76	0.40	0.22	0.69	0.29	0.38
48	0.15	0.22	0.48	0.44	0.33	0.56	0.13	0.33
49	0.19	0.42	0.28	0.32	0.28	**	0.20	0.28
50	0.18	0.52	0.31	0.25	0.35	0.25	0.27	0.30
51	0.13	0.21	0.52	0.35	0.30	0.46	0.31	0.33
52	0.12	0.19	0.53	**	0.22	0.30	0.28	0.27
53	0.12	0.17	0.61	0.48	0.24	0.36	0.27	0.32

contd./

Table D2 contd.

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	MEAN
54	0.31	0.59	0.33	0.23	0.41	0.24	0.25	0.34
56	*	0.21	0.37	0.23	0.12	0.27	0.24	0.21
58	*	0.13	0.44	0.27	0.23	0.40	0.25	0.25
59	0.07	0.35	0.28	0.13	0.25	0.27	0.28	0.23
60	**	0.50	0.34	0.26	0.37	0.20	0.44	0.35
62	0.14	0.12	0.42	0.22	0.34	0.50	*	0.26
63	0.13	0.09	**	0.14	0.28	0.29	*	0.16
64	0.09	0.30	0.14	0.23	0.47	0.12	0.17	0.22
65	0.15	0.26	0.17	0.20	0.46	0.16	0.20	0.23
66	0.14	0.20	0.32	0.25	0.22	0.44	0.13	0.24
67	0.15	0.25	0.39	0.38	0.25	0.55	0.12	0.30
68	0.23	0.26	**	0.26	0.29	0.52	0.08	0.27
70	0.26	0.29	0.24	0.31	0.52	0.18	0.40	0.31
71	0.20	0.26	0.28	0.35	0.19	0.47	0.17	0.27
72	0.43	0.37	0.32	0.41	0.48	0.48	0.46	0.42
73	0.23	0.19	0.33	0.24	**	0.45	**	0.29
74	0.19	0.23	0.34	0.29	0.47	0.15	0.32	0.28
75	0.20	0.20	0.25	0.29	0.52	0.13	0.31	0.27
76	0.17	0.17	0.15	0.26	0.17	0.26	**	0.20
78	0.30	0.24	0.29	0.25	0.21	0.46	0.27	0.29
79	0.23	0.20	0.16	0.17	0.36	0.37	0.47	0.28
80	0.13	0.19	0.15	0.22	0.34	0.20	0.34	0.22
81	0.36	0.27	0.10	0.12	0.29	0.49	0.23	0.27
82	0.39	0.25	0.19	0.20	0.27	0.50	0.39	0.31
83	0.30	**	0.21	0.11	0.11	1.96	0.25	0.49
84	0.29	0.28	0.42	0.29	0.43	0.31	0.51	0.36
85	0.23	0.17	0.25	0.23	0.65	0.21	0.53	0.32
86	0.57	0.51	0.23	0.24	0.22	0.53	0.51	0.40
87	0.52	0.38	0.30	0.15	0.33	0.52	0.34	0.36
88	0.24	0.20	0.21	0.15	0.18	0.26	0.27	0.22
89	0.29	0.26	0.19	0.11	0.33	0.23	0.43	0.27
90	0.24	0.19	0.15	0.21	0.39	0.26	0.30	0.25
91	0.52	0.50	0.28	0.20	0.20	*	0.25	0.29
92	0.41	0.44	0.28	0.19	0.36	0.10	0.12	0.27
93	0.40	0.44	0.24	0.23	0.15	**	0.06	0.25
94	0.44	0.20	0.16	0.14	*	0.11	0.19	0.18
95	0.51	0.30	0.19	0.15	*	0.08	0.25	0.22
96	0.36	0.42	0.35	0.27	0.12	0.08	0.12	0.25
98	0.48	0.46	0.28	0.21	0.12	*	0.06	0.24
99	0.44	0.26	0.25	0.12	0.05	0.15	0.26	0.22
100	0.42	0.38	0.21	0.18	0.15	0.10	0.22	0.24
101	0.34	0.49	0.30	0.22	**	0.05	0.12	0.25
102	0.52	0.59	0.36	*	**	0.10	0.17	0.30
103	0.34	0.48	0.26	0.15	**	0.05	*	0.22
104	0.43	0.28	0.18	**	0.09	*	0.06	0.18
105	0.56	0.28	0.14	**	0.16	*	0.20	0.23
106	0.49	0.28	**	0.12	0.13	0.21	0.31	0.26

* Below detection limit
** Missing data

Table D3 External air lead concentration: μg/m³

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK							MEAN
	1	2	3	4	5	6	7	
1	0.12	0.41	0.33	0.33	0.47	0.46	0.47	0.37
2	0.10	0.26	0.21	0.45	0.82	0.58	0.33	0.39
3	0.16	0.27	0.26	0.19	0.38	0.34	**	0.27
4	0.27	0.24	0.21	0.46	0.25	0.28	0.20	0.27
5	0.26	1.03	0.12	0.23	0.43	0.46	0.44	0.36
6	0.36	0.47	0.12	0.23	0.43	0.46	0.44	0.36
7	0.22	0.23	0.32	0.49	0.84	0.57	0.73	0.49
8	0.12	0.27	0.10	0.20	0.43	0.38	0.35	0.26
10	0.33	0.15	0.26	0.47	0.51	0.29	0.24	0.32
11	0.22	0.28	0.19	0.24	0.48	0.73	0.47	0.37
12	0.22	0.72	0.26	0.95	0.65	0.18	0.76	0.53
13	0.27	0.50	0.25	0.42	0.65	2.20	0.77	0.72
14	0.76	0.39	0.54	1.12	0.76	1.25	0.92	0.82
15	0.86	0.54	1.36	0.96	2.21	1.72	1.15	1.26
16	0.09	0.44	*	0.71	0.43	0.38	1.67	0.54
17	0.29	0.51	0.49	0.54	**	0.46	1.65	0.66
18	0.24	0.35	0.38	0.56	0.57	0.35	1.53	0.57
19	0.37	0.41	0.60	0.58	0.52	1.71	0.66	0.69
20	0.44	0.42	0.40	0.49	0.33	1.32	1.17	0.65
21	0.38	0.42	0.62	0.61	0.52	0.58	1.49	0.66
22	0.88	0.42	1.85	0.67	0.56	0.38	1.51	0.90
23	0.35	0.44	0.36	0.61	0.42	0.51	1.48	0.60
24	0.53	0.44	0.58	0.49	0.52	2.13	0.70	0.77
25	0.63	0.80	0.64	0.66	0.40	1.72	2.98	1.12
26	0.30	0.66	0.48	0.86	0.52	0.57	1.72	0.73
27	1.08	0.53	0.69	0.78	0.47	0.53	1.60	0.81
28	0.40	0.68	0.53	0.83	0.61	0.05	2.87	0.94
29	0.51	0.55	0.69	0.55	0.66	1.56	0.74	0.75
30	0.65	0.61	0.82	0.45	0.58	1.94	1.07	0.87
31	**	**	**	**	**	**	**	**
32	**	**	**	1.56	1.42	**	**	**
33	**	**	2.13	1.77	**	0.65	**	**
34	**	**	**	**	**	**	**	**
35	**	**	**	**	0.61	**	**	**
37	**	**	**	**	**	**	**	**
39	1.32	2.87	2.00	1.39	**	0.22	0.11	1.32
40	1.53	**	2.25	2.12	0.74	**	0.20	1.37
42	0.28	0.96	2.46	**	**	0.55	**	1.06
43	**	1.09	1.45	1.13	**	0.51	0.25	0.89
44	1.59	**	2.28	1.77	0.93	**	**	1.64
45	1.81	2.85	2.11	*	0.69	0.34	0.20	1.15
46	0.14	0.40	1.26	0.47	0.37	0.70	0.30	0.52
47	0.12	0.20	0.98	0.52	0.37	0.72	0.41	0.47
48	0.29	**	0.83	0.70	0.63	0.82	0.34	0.60
49	0.22	0.71	0.49	0.46	0.51	0.26	0.36	0.43
50	0.31	0.80	0.69	0.53	0.61	0.35	0.21	0.50
51	0.18	0.57	0.29	0.64	0.52	0.79	0.53	0.50
52	0.15	0.26	0.95	0.41	0.43	0.52	0.39	0.44
53	0.38	**	0.91	0.61	0.57	0.67	0.47	0.60

contd./

Table D3 contd.

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	MEAN
54	0.29	0.83	0.39	0.30	0.53	0.41	0.55	0.47
56	0.22	0.47	0.92	0.46	0.26	0.61	0.51	0.49
58	0.14	**	0.79	0.41	0.31	0.56	0.25	0.41
59	0.26	0.91	0.42	0.31	0.55	0.36	0.76	0.51
60	0.35	0.82	0.47	0.28	0.50	0.33	0.43	0.45
62	0.16	0.39	0.76	0.36	0.63	0.87	0.11	0.47
63	0.21	0.24	0.45	0.29	0.37	0.66	0.17	0.34
64	0.37	0.47	0.21	0.35	0.69	0.22	0.34	0.38
65	0.33	0.49	0.23	0.41	0.89	0.12	0.28	0.39
66	0.32	0.33	0.52	0.42	0.40	0.88	0.13	0.43
67	0.29	0.17	0.58	0.57	0.34	0.84	0.13	0.42
68	0.30	0.34	0.59	0.49	0.45	0.83	0.10	0.44
70	0.39	0.46	0.40	0.40	0.87	0.21	0.50	0.46
71	0.32	0.27	0.49	0.60	0.42	0.91	0.21	0.46
72	0.37	0.22	0.43	0.48	0.43	0.83	0.52	0.47
73	0.44	0.39	0.57	0.61	0.46	0.98	0.20	0.52
74	0.34	0.46	0.67	0.26	0.95	**	0.57	0.54
75	0.35	0.44	0.44	0.39	0.86	0.17	0.55	0.46
76	0.38	0.25	0.27	0.30	0.24	0.48	**	0.32
78	0.48	0.47	0.37	0.37	0.24	0.80	0.29	0.43
79	0.28	0.24	0.20	0.22	0.49	**	0.69	0.35
80	0.29	0.28	0.19	**	0.62	0.38	0.67	0.40
81	0.45	0.46	0.17	0.17	0.28	0.58	0.36	0.35
82	0.66	0.62	0.40	0.46	0.64	0.90	0.61	0.61
83	0.46	0.24	0.27	0.20	0.22	0.54	0.24	0.31
84	0.73	0.50	0.74	0.64	0.99	0.70	1.32	0.80
85	0.26	0.35	0.34	0.30	0.59	0.30	0.63	0.40
86	0.94	0.82	0.53	0.53	**	0.99	0.73	0.76
87	0.78	0.67	0.44	0.38	0.47	0.88	0.52	0.59
88	0.57	0.55	0.39	0.37	0.32	0.64	0.46	0.47
89	0.48	0.26	0.19	0.13	0.45	0.25	0.37	0.30
90	0.42	0.34	0.31	0.28	0.62	0.37	0.64	0.43
91	0.52	0.56	0.35	0.20	0.20	*	0.10	0.28
92	0.50	0.67	0.43	0.28	0.66	*	0.14	0.39
93	0.59	0.62	0.33	0.23	0.18	**	0.13	0.35
94	0.77	0.38	0.24	0.19	*	0.13	0.27	0.29
95	0.94	0.33	0.17	0.11	*	0.17	0.16	0.28
96	0.56	0.80	0.43	0.31	0.19	*	0.18	0.36
98	0.61	0.72	0.42	0.41	0.18	0.14	0.16	0.38
99	0.74	0.54	0.30	0.12	**	**	**	0.43
100	0.68	0.46	0.31	0.24	0.08	0.23	0.26	0.32
101	0.47	0.61	0.40	0.19	**	0.10	0.14	0.32
102	*	0.80	0.23	0.21	**	0.11	0.11	0.25
103	0.52	0.77	0.32	0.23	**	0.08	0.07	0.33
104	0.97	0.50	0.27	**	0.13	0.14	0.26	0.38
105	0.80	0.41	0.25	**	0.12	0.12	0.30	0.33
106	0.57	0.44	**	0.16	0.20	0.21	0.35	0.32

* Below detection limit
** Missing data

APPENDIX E SAMPLERS' HANDWIPE DATA

Table E Lead on samplers' hands: µg total both hands

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	8
1	7.6	1.2	2.2	2.6	<1.2	<1.2	1.9	<1.2
2	6.5	1.3	<1.2	<1.2	<1.2	<1.2	7.5	<1.2
3	8.0	3.4	4.6	1.9	<1.2	1.5	6.8	*
4	1.6	1.9	2.4	2.4	2.3	<1.2	15.9	2.3
5	2.3	<1.2	0.6	<1.2	12.0	2.6	1.8	12.4
6	3.6	<1.2	2.9	1.5	<1.2	3.6	7.2	2.1
7	2.3	2.0	<1.2	1.2	<1.2	1.2	3.1	4.0
8	8.0	1.4	2.2	<1.2	1.3	<1.2	1.3	<1.2
10	2.5	1.4	<1.2	2.4	1.6	<1.2	<1.2	*
11	15.1	1.2	7.8	4.6	2.9	2.3	1.4	6.7
12	26.5	<1.2	2.5	3.9	3.8	2.4	5.3	2.8
13	12.3	2.6	1.8	1.4	1.2	2.4	3.7	9.0
14	6.4	3.0	7.0	2.3	1.4	4.7	9.6	<1.2
15	14.8	3.6	9.8	2.3	2.9	1.4	5.0	4.8
16	20.1	1.4	1.8	<1.2	3.6	<1.2	<1.2	<1.2
17	6.1	<1.2	4.6	1.5	<1.2	*	1.8	2.9
18	20.4	1.6	6.2	4.7	<1.2	8.2	8.0	7.1
19	6.1	<1.2	<1.2	<1.2	<1.2	<1.2	2.1	1.3
20	1.8	<1.2	1.3	1.7	2.1	<1.2	<1.2	<1.2
21	2.9	1.9	2.0	<1.2	<1.2	2.3	<1.2	1.7
22	3.3	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	1.4
23	2.5	1.2	1.2	1.2	<1.2	<1.2	<1.2	<1.2
24	1.7	2.1	<1.2	3.0	1.5	1.0	1.4	1.3
25	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	2.1
26	15.4	*	2.0	7.0	1.3	4.0	3.2	1.7
27	*	*	1.5	<1.2	1.2	4.1	<1.2	2.4
28	8.8	6.1	<1.2	2.8	2.7	5.2	2.0	<1.2
29	10.4	2.9	4.9	1.2	1.7	5.7	3.8	5.6
30	3.2	1.9	<1.2	<1.2	2.3	<1.2	8.1	<1.2
31	1.8	2.9	3.2	5.3	4.9	2.7	<1.2	1.3
32	4.3	<1.2	3.3	9.0	2.8	2.1	1.3	<1.2
33	4.4	9.3	4.5	1.2	1.5	<1.2	2.3	4.9
34	9.4	<1.2	<1.2	<1.2	6.3	4.7	5.3	4.2
35	<1.2	2.7	1.3	7.3	2.4	2.8	3.1	<1.2
36	1.3	1.7	1.6	<1.2	<1.2	<1.2	1.2	<1.2
37	1.9	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	1.2
38	1.2	<1.2	1.4	<1.2	<1.2	<1.2	<1.2	2.0
39	<1.2	2.1	<1.2	<1.2	1.6	<1.2	1.3	2.1
40	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2
42	5.0	8.1	4.9	6.2	4.1	1.5	4.6	4.9
43	15.5	5.0	2.5	4.1	2.6	3.3	2.9	1.7
44	11.2	4.9	6.7	<1.2	4.3	4.7	7.4	7.8
45	27.5	4.9	3.1	4.9	3.0	1.8	<1.2	<1.2
46	7.7	<1.2	1.3	<1.2	3.3	3.1	1.7	4.0
47	1.4	3.5	<1.2	3.7	1.7	1.8	2.0	1.6
48	1.7	<1.2	1.3	<1.2	2.3	5.2	1.9	4.4
49	6.8	4.0	<1.2	2.5	<1.2	4.1	2.8	1.2

contd./

Table E contd.

HOUSE NUMBER	DAY OF SAMPLING DURING STUDY WEEK 1	2	3	4	5	6	7	8
50	11.7	<1.2	1.6	3.5	2.3	4.4	5.9	<1.2
51	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	1.2
52	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2
53	1.7	<1.2	<1.2	2.3	<1.2	<1.2	5.4	<1.2
54	<1.2	<1.2	<1.2	1.5	<1.2	<1.2	<1.2	<1.2
56	3.7	<1.2	3.0	1.7	5.5	1.2	2.0	1.8
58	7.9	<1.2	4.0	<1.2	4.0	1.4	<1.2	7.3
59	5.1	<1.2	1.2	<1.2	1.4	3.4	6.0	8.2
60	16.1	2.4	1.2	1.8	<1.2	1.2	<1.2	2.1
62	3.5	15.6	<1.2	6.1	4.2	3.0	<1.2	<1.2
63	6.8	2.1	2.4	2.0	1.9	1.4	2.6	8.0
64	1.7	1.9	<1.2	<1.2	2.0	2.0	8.5	<1.2
65	<1.2	2.5	<1.2	<1.2	1.9	<1.2	1.7	2.1
66	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2
67	1.9	1.3	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2
68	3.6	<1.2	<1.2	<1.2	<1.2	<1.2	1.4	1.6
70	1.4	2.0	<1.2	<1.2	<1.2	<1.2	<1.2	1.7
71	*	<1.2	<1.2	3.0	6.9	1.7	<1.2	3.4
72	8.2	10.0	4.3	5.3	3.8	3.5	<1.2	7.5
73	15.2	3.7	3.0	2.0	<1.2	1.7	<1.2	<1.2
74	3.7	1.4	<1.2	1.5	2.2	2.2	<1.2	2.8
75	11.2	5.7	6.7	2.6	1.3	3.8	2.7	1.9
76	<1.2	8.8	1.9	<1.2	8.4	<1.2	2.9	*
78	10.5	<1.2	2.8	8.6	<1.2	2.0	<1.2	<1.2
79	9.6	<1.2	2.4	<1.2	<1.2	1.2	*	2.6
80	<1.2	1.3	10.4	9.2	<1.2	<1.2	10.2	8.4
81	2.0	<1.2	<1.2	<1.2	1.6	1.9	2.7	<1.2
82	<1.2	14.8	1.9	*	<1.2	26.4	4.0	2.5
83	<1.2	2.6	1.9	1.2	5.3	1.7	5.9	9.6
84	4.0	2.2	3.0	3.5	2.4	1.9	<1.2	<1.2
85	2.7	<1.2	3.1	2.0	3.4	3.4	1.9	<1.2
86	5.5	4.6	4.8	<1.2	6.3	<1.2	2.3	3.1
87	2.9	8.3	<1.2	4.4	1.4	1.6	1.8	1.3
88	4.9	3.6	3.7	2.0	3.7	<1.2	<1.2	2.1
89	8.4	3.5	3.6	2.0	3.2	<1.2	3.0	*
90	14.5	3.9	1.8	<1.2	<1.2	1.4	<1.2	1.6
91	2.4	5.3	2.1	6.8	1.8	8.1	2.7	2.5
92	13.4	2.2	<1.2	6.3	2.8	3.8	8.0	2.5
93	<1.2	13.0	5.7	<1.2	4.4	1.9	*	1.2
94	1.9	5.4	<1.2	<1.2	1.3	<1.2	<1.2	<1.2
95	27.3	2.7	2.0	2.0	1.7	2.5	4.2	4.1
96	4.5	2.1	<1.2	<1.2	2.6	1.3	1.6	1.3
98	<1.2	<1.2	<1.2	<1.2	1.2	2.0	2.1	<1.2
100	<1.2	<1.2	1.2	<1.2	2.1	1.5	1.9	2.3
101	13.9	4.1	2.7	3.4	1.8	*	<1.2	3.6
102	6.8	2.2	4.3	4.7	3.2	*	<1.2	2.6
103	3.2	1.4	1.9	1.4	2.5	*	<1.2	2.9
104	3.5	1.2	4.4	3.2	*	<1.2	5.1	5.3
105	8.6	3.4	6.8	2.6	*	*	6.7	1.2
106	4.0	1.9	3.8	*	2.9	5.9	1.3	19.1

* Missing data

APPENDIX F MOUTHING AND HAND CONTACT BEHAVIOUR DATA FOR EACH SUBJECT

Table F Pica[1] and hand-to-mouth activity data[2]

CHILD NUMBER	PICA	RATE 3	RATE 4	RATE 6	RATE 15	RATE 18
1	11	0.00	0.83	1.45	13.57	1.08
2	7	0.00	0.25	0.81	6.26	0.00
4	0	0.29	0.54	1.21	11.96	0.15
5	4	0.03	0.08	0.62	14.46	1.28
6	7	0.42	1.39	3.61	13.46	1.05
7	2	0.16	0.34	2.05	15.79	2.01
8	17	0.12	1.17	1.72	17.77	1.55
10	6	0.21	0.64	1.30	15.00	1.20
11	4	0.00	0.00	0.55	11.32	0.73
12	9	0.34	1.28	1.97	5.49	0.03
13	30	1.38	2.09	2.47	13.53	0.08
14	9	0.01	0.06	0.79	21.79	0.33
15	8	0.54	1.89	2.60	15.63	0.24
16	11	0.08	0.51	1.27	13.77	0.90
17	8	0.05	1.62	2.83	18.02	0.40
18	1	0.02	0.42	1.20	12.03	0.78
19	30	0.13	0.60	1.49	11.74	0.28
20	9	0.32	0.94	1.58	12.09	0.20
21	8	0.20	0.79	1.45	16.50	2.87
22	8	0.00	0.15	1.12	16.42	0.10
23	8	0.00	0.45	2.91	14.24	0.48
24	11	1.22	1.55	2.43	14.30	0.32
25	14	0.11	0.93	1.97	11.92	0.15
26	22	1.38	1.59	2.90	17.26	0.96
27	4	0.05	0.41	1.15	13.77	1.38
28	4	0.20	1.38	2.10	17.85	1.38
29	10	0.95	1.86	2.69	16.46	1.51
30	20	0.70	1.06	2.24	11.90	1.46
31	2	0.70	1.73	1.93	16.44	0.03
32	2	0.41	1.19	3.05	18.34	0.20
33	4	0.03	0.60	1.97	15.13	0.85
34	0	0.00	0.00	0.34	12.99	0.15
35	6	0.38	1.59	2.00	12.87	0.05
36	8	0.88	0.98	1.61	13.63	0.13
37	7	0.00	0.02	1.86	15.76	1.11
38	7	0.03	0.25	1.04	12.22	1.83
39	13	0.37	0.98	1.42	16.42	1.72
40	4	0.17	0.43	1.14	6.74	0.28
42	7	0.02	0.24	2.10	14.60	0.53
43	13	0.07	0.67	2.10	16.85	0.32
44	5	0.09	0.60	1.00	16.33	1.77
45	5	0.08	0.84	1.99	21.04	2.83
46	7	0.11	0.67	1.30	14.45	0.25

contd./

Table F contd.

CHILD NUMBER	PICA	RATE 3	RATE 4	RATE 6	RATE 15	RATE 18
47	2	0.24	1.07	1.45	15.29	0.67
48	14	0.22	0.61	2.23	17.32	0.22
49	15	0.01	0.56	1.28	15.69	0.98
50	4	0.02	0.22	0.73	9.27	0.06
51	21	1.00	1.33	2.11	16.85	0.65
52	15	0.12	1.13	2.57	14.66	0.18
53	20	0.00	0.73	2.45	13.75	0.20
54	22	0.50	1.26	2.39	22.81	3.25
56	0	0.08	0.54	1.05	13.52	1.15
58	18	0.00	0.33	1.93	15.91	0.65
59	*	0.15	0.82	1.47	16.94	0.32
60	18	0.09	1.00	2.05	20.40	1.12
62	10	0.03	0.30	1.63	15.92	0.10
63	3	0.02	0.75	2.43	15.42	0.15
64	16	0.52	1.42	2.31	14.52	0.68
65	5	0.28	0.95	2.05	17.93	0.25
66	9	0.15	0.36	1.25	15.02	2.02
67	4	0.02	0.53	1.34	19.05	1.09
68	24	0.44	1.31	2.75	19.60	1.41
70	18	0.05	0.15	1.33	15.07	0.02
71	7	0.32	0.62	1.96	16.54	0.53
72	*	0.51	1.66	3.95	17.66	1.56
73	26	0.10	2.41	3.33	19.93	1.08
74	3	0.08	0.41	0.77	21.91	1.17
75	7	0.18	0.34	1.61	19.12	2.02
76	16	0.13	0.62	1.16	18.67	1.15
78	1	0.10	0.58	1.15	17.13	1.62
79	3	0.33	0.90	1.53	18.03	2.21
80	4	0.43	0.97	1.96	16.30	0.45
81	16	0.10	0.61	1.88	17.76	0.00
82	28	0.04	0.91	2.28	18.54	0.60
83	22	0.09	0.39	0.89	13.85	0.56
84	15	0.88	1.26	2.55	14.20	1.77
85	21	0.06	1.30	2.22	19.92	0.00
86	29	0.31	0.56	2.51	21.05	0.49
87	12	0.00	0.33	1.55	24.06	0.34
88	5	0.06	0.56	1.53	18.88	0.30
89	15	0.02	0.83	1.76	16.75	0.53
90	5	0.12	0.43	1.51	18.92	0.91
91	14	0.06	0.30	1.07	22.31	0.64
92	5	0.03	0.56	1.41	16.81	0.06
93	4	0.58	1.08	2.43	19.43	0.63
94	3	0.23	0.59	1.72	15.41	0.17
95	16	0.18	1.59	2.37	14.88	0.41
96	5	0.07	0.56	1.05	20.26	1.43
98	26	0.14	0.34	1.58	16.95	0.08
99	8	0.20	0.27	1.46	15.59	0.27
100	14	0.00	0.47	0.61	14.36	1.12
101	*	0.00	0.30	1.30	10.87	0.34

contd./

Table F contd.

CHILD NUMBER	PICA	RATE 3	RATE 4	RATE 6	RATE 15	RATE 18
102	6	0.30	0.68	1.95	21.95	2.06
103	7	0.00	0.12	0.79	14.91	0.43
104	11	0.04	0.19	0.94	14.49	0.48
105	6	0.00	0.16	0.71	12.41	2.20
106	6	0.00	0.00	2.51	13.92	0.80

KEY:

Rate 3: Hands in mouth rate (included in Rate 4)
Rate 4: Hands to mouth area rate
Rate 6: Overall rate of mouthing behaviours - all objects
Rate 15: Both hands touching all objects rate
Rate 18: Both hands touching floor rate (included in Rate 15)

1: Total score on pica scale completed by parental report. Higher scores indicate higher reported frequency of mouthing and number of objects mouthed.

2: All rates expressed in terms of behaviours per minute.

Pica: Scale based on questionnaire responses. Individual items were allocated a score based on the frequency of the behaviour (0 = never; 1 = occasionally; 2 = frequently; 3 = regularly) and the items summed to give a single figure.

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LEAD LEVELS IN BIRMINGHAM DUSTS AND SOILS

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ABSTRACT

Lead concentrations were measured in housedust, pavement dust, road dust and garden soil in and around 97 inner-city houses in Birmingham, England. The highest mean dust lead concentration within the home, 615 $\mu g\,g^{-1}$, was noted in samples from under the doormat. Generally, the housedust lead levels were lower than the national mean (507 $\mu g\,g^{-1}$), although soil lead concentrations were slightly higher. The age of the property was found to influence the lead levels in both housedust and garden soil, with older houses (> 35 years) having significantly higher concentrations than newer properties (< 35 years). Houses being decorated at the time of sampling were found to have significantly higher lead concentrations than those that were not. Elevated lead levels were also noted in housedust and garden soil from houses located within a 500 m radius of commercial garages. Increased lead concentrations were found in soil samples from gardens in close proximity to waste land (demolition sites and tips), metal-using industries and from those within 10 m of a road. Road dust samples from industrial areas had significantly higher lead concentrations than those from residential areas.

INTRODUCTION

The exposure of young children to environmental lead has received much attention in recent years (Department of Health and Social Security, 1980; Royal Commission on Environmental Pollution, 1983). In particular, the possible intake of lead from urban dusts and soils has been the focus of many investigations (Angle and McIntire, 1974; Barltrop et al., 1974, 1975; Roberts et al., 1974; Sayre et al., 1974; Ter Haar and Aronow, 1974; Vostal et al., 1974; Lepow et al., 1975; Duggan and Williams, 1977; Yankel et al., 1977; Duggan, 1980; Charney et al., 1980; Roels et al., 1980; Brunekreef et al., 1981; Diemel et al., 1981; Milar and Cooney, 1982). In the United Kingdom, a national survey of housedusts in 53 representative villages, towns and city boroughs revealed that in 10% of the 5228 homes tested, the lead concentration was in excess of 2000 $\mu g\,g^{-1}$ (Thornton et al., 1985). In an attempt to assess the significance of this and other pathways to the child's total lead intake, a study was commissioned by the U.K. Department of the Environment in the inner-city area of Birmingham, Britain's second largest city. This was designed to provide quantitative information on the intakes of young children via all the major

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pathways simultaneously and was based on the analysis of environmental, dietary and biological samples which included housedust, pavement and road dust, soil, water, food, blood, handwipes and air. A behavioural study was also incorporated in order to assess hand-to-mouth activity. The operational details and results from the dust and soil sampling programme are presented and discussed in this paper. Those from the other sections of this study have been published elsewhere (food and water: Smart et al., 1987; blood: Delves and Quinn, 1987; handwipes: Davies and Watt, 1986; air: Davies et al., 1987; behaviour: Harvey et al., 1987). In addition, road dust samples were collected from outside premises in which lead was being used for industrial purposes for comparison with those from residential areas.

STUDY DESIGN

A random selection of families with children born in the study area (within the inner ring road and to the east of the city centre of Birmingham) and at the time of the study aged 24 ± 2 months were approached by letter and subsequently visited. Of the 183 volunteered children who provided blood samples, a stratified sample of 106 was selected for participation (by the criteria described by Thomas et al., 1985) and 97 completed the study. Sampling was carried out between November, 1984 and February, 1985. From in and around each house the following dust and soil samples were obtained.

(1) Housedusts. (a) One sample from the dust bag in the householder's own vacuum cleaner. (b) One sample from the child's main playroom and one from the child's bedroom using an adapted Electrolux 350 vacuum cleaner. The cleaner had a pre-weighed cellulose filter thimble mounted at the distal end of the suction tube and has been described elsewhere (Watt et al., 1983). All of the exposed floorspace was sampled and the area noted. (c) One sample from under the doormat using an adapted Electrolux 350 cleaner.

(2) Pavement and road dust. At each of these locations, immediately outside the house, a composite sample of 25 subsamples, collected using a plastic dust pan and brush when conditions were dry.

(3) Soil. One composite surface soil sample (0–5 cm), made up from 25 subsamples, from areas of exposed soil in the front and back garden, using a stainless steel trowel.

In addition to these, one composite road dust sample, made up from 25 subsamples, was collected from outside each of 58 premises in which lead was being used for industrial purposes, with a plastic dust pan and brush when conditions were dry.

ANALYTICAL TECHNIQUES

Sample preparation

Dusts

All samples were dried at 80°C for 24 h and passed through a 1 mm sieve. The thimble dust samples were weighed (to enable the calculation of loading fig-

ures) and a 0.25 g subsample was obtained. A 0.25 g subsample was also taken from the householder's own vacuum cleaner dust sample and from the road and pavement dust samples

Soil

All samples were dried at 80°C for 24 h, passed through a 2 mm sieve and milled in an agate mill ("Tema"). A 0.25 g subsample was taken from each sample.

Digestion and analysis

Each sample was placed in a test tube and 4 ml of nitric acid were added, followed by 1 ml of perchloric acid. Tubes were then placed in a deep heating block and the samples were digested and taken to dryness over 24 h (3 h at 50°C, 3 h at 150°C, 18 h at 190°C). Samples were leached in 2 ml 6 *M* hydrochloric acid, made up to 10 ml with deionised water and analysed for lead and a range of other elements by inductively coupled plasma atomic emission spectroscopy.

Quality control

A rigorous quality control programme was implemented which consisted of reagent blanks, duplicates and reference materials and represented around 20% of the total number of samples analysed.

RESULTS AND DISCUSSION

The results are summarised in Table 1. As all sample populations showed log-normal distributions, geometric mean values have been calculated. The highest mean lead concentration in dust from within the home, 615 $\mu g\,g^{-1}$, was noted in samples from under the doormat and was almost twice as high as the mean value of those from the householder's vacuum cleaner, 336 $\mu g\,g^{-1}$ (significant difference, $p < 0.01$), and higher (although not significantly) than the mean from the bedroom and playroom samples, 430 $\mu g\,g^{-1}$. This is not a surprising result when one considers that external dusts, in general, have higher lead concentrations than housedusts and that the doormat is the most likely location within a house for the removal of such external dusts. In the national reconnaissance study, the mean lead concentration in road dust was 707 $\mu g\,g^{-1}$ compared with 507 $\mu g\,g^{-1}$ in housedust (excluding data from 'geochemical hotspots', Thornton et al., 1985). From Table 1, it can be seen that the mean road dust concentration in Birmingham, 527 $\mu g\,g^{-1}$, is lower than the national average, but again higher than the concentrations found in the bedroom, playroom and vacuum cleaner samples. Birmingham was one of the cities included in the national survey and the geometric mean housedust lead concentration found in 1981/2 was 419 $\mu g\,g^{-1}$ ($n = 159$). This figure is similar to the mean concentration found in the present study using the Imperial College sampler, 430 $\mu g\,g^{-1}$, although slightly higher than that noted for the householder's vacuum cleaner samples, 336 $\mu g\,g^{-1}$ (Table 1).

TABLE 1

Summary data for lead in housedust, soil, pavement and road dust

	N	AM	Range	GM	Percentile	
					5th	95th
Lead conc. ($\mu g\,g^{-1}$)						
Doormat	42	1436	79–15000	615	120	4300
Vacuum[a]	92	713	46–23000	336	97	1440
Bedroom	96	693	53–4500	464	109	2040
Playroom	97	455	62–5600	311	105	1030
Average[b]	95	578	111–3110	430	139	1780
Soil	86	405	56–1650	313	92	1160
Pavement	97	506	62–5100	360	127	1340
Road	97	805	80–2100	527	195	1170
Lead loading ($\mu g\,m^{-2}$)						
Bedroom	96	141	1–1348	51	3	417
Playroom	96	150	1–1799	42	1	614
Average[b]	94	149	2–1062	62	4	486

[a] From householder's own vacuum cleaner.
[b] Bedroom and playroom.

Other data pertaining to internal dusts in Birmingham do appear in the literature but are difficult to compare directly with the results from our studies. For example, an extremely high median value of $3500\,\mu g\,g^{-1}$ has been reported for 117 samples collected in 28 Birmingham houses during 1979/1980 (Department of the Environment, 1982). The houses included in this study, however, were selected because the children living in them had been found to have high blood lead concentrations and/or high erythrocyte protoporphyrin concentrations and cannot be considered as representative. In another Birmingham study a high median lead concentration of $1000\,\mu g\,g^{-1}$ was reported for 806 internal dust samples (Archer and Barratt, 1976). These, however, were not solely housedust samples but were collected from offices, shops and schools as well as houses in the area.

A somewhat unexpected result from the present study was that the mean lead concentration in pavement dust, $360\,\mu g\,g^{-1}$, was significantly lower than that found in road dust, $527\,\mu g\,g^{-1}$ ($p < 0.001$), even though the sampling sites were adjacent in all cases. These data suggest that there may have been a greater input of non-lead bearing dust and soil particles onto the pavement sites, perhaps by pedestrian movement, and/or that the processes of deposition and removal were operating at different rates in the two situations.

In addition to the bedroom and playroom dust lead concentration figures in Table 1, dust lead loadings have been calculated and included. Results from pilot studies had suggested that the exposure of a child to lead in housedust is expressed more realistically by lead loading than simply lead concentration in dust (Culbard et al., 1987). Although it is not possible to present the data for all houses individually in this paper, results from the present study do confirm

this. For example, in one of the houses sampled the lead concentration in the bedroom was approximtely 12 times that in the playroom (1850 and 151 $\mu g\,g^{-1}$, respectively), while the lead loading was only around twice as high (117 and 50 $\mu g\,m^{-2}$, respectively). Looking at lead concentration alone, therefore, could be deceptive as the total mass of lead available for ingestion by a young child from housedust is not reflected in this figure. The usefulness of calculating loadings is supported by data from the hand lead study which, as outlined earlier, was carried out at the same time and in the same houses as the dust survey (Davies and Watt, 1986). Looking at the association between housedust lead and hand lead in terms of correlation coefficients, there was a positive correlation of 0.44 between lead loading and hand lead and a positive correlation of only 0.24 between lead concentration and hand lead.

The mean soil lead concentration found in the present study, 313 $\mu g\,g^{-1}$, was similar to that noted for Birmingham in the reconnaissance study, 278 $\mu g\,g^{-1}$. Both of these values, however, are slightly higher than the national average, 230 $\mu g\,g^{-1}$ ($n = 3550$). Previous studies have shown soil lead levels to be correlated with housedust lead (Thornton et al., 1985) and this was also the case in this study. The correlation coefficient between dust lead loading and soil lead was 0.38. It is interesting to note that the highest soil lead value in the present study, 1650 $\mu g\,g^{-1}$, was found in a garden backing onto a factory which had been manufacturing guns and ammunition for many decades.

A comprehensive questionnaire was administered during the study which assisted in the interpretation of the environmental data (Table 2). Newer properties (< 35 years old) were found to have significantly lower levels of lead in housedust and garden soil than older properties (> 35 years old). These findings complement earlier observations by Thornton et al. (1985) who suggested that these phenomena may be due to voluntary reductions in the lead content of household paints over the past 40 years and to a shorter history of soil contamination from bonfires and fossil fuel residues, often used as soil improvers and ameliorants. If the property was being renovated/decorated at the time of sampling, the housedust lead concentration was significantly higher than if no decorating was taking place, geometric means being 547 and 393 $\mu g\,g^{-1}$, respectively ($p < 0.05$). This is in agreement with the findings of other workers who have reported significant elevation in household dust lead levels during home renovation work (Inskip and Atterbury, 1983; Inskip and Hutton, 1987). Properties located within a 500 m radius of a commercial garage were found to have significantly higher lead levels in both housedust and garden soil than those further away. The respective means for housedust were 506 and 313 $\mu g\,g^{-1}$ ($p < 0.01$) in terms of concentration and 92 and 27 $\mu g\,m^{-2}$ ($p < 0.001$) in terms of loading, and those for soil were 365 and 224 $\mu g\,g^{-1}$ ($p < 0.01$). While these results might suggest that commercial garages were acting as point sources for lead in the surrounding houses and gardens, no elevations in lead concentrations were noted in the road or pavement dust samples or in the external air samples obtained from alongside these houses. This is surprising as the most likely transfer mechanism from such a source would involve aerial transport and deposition. Elevated soil lead levels were

TABLE 2

Lead levels in housedust and garden soil for a number of subgroups identified from questionnaire responses

	Geometric mean lead value		
	Housedust		Soil concentration ($\mu g\,g^{-1}$)
	Concentration ($\mu g\,g^{-1}$)	Loading ($\mu g\,m^{-2}$)	
House age < 35 years?			
Yes	221***	15***	176**
No	498	81	342
Decorating?			
Yes	547*	94	331
No	393	52	300
Near Commercial garage?			
Yes	506**	92***	365**
No	313	27	224
Near waste land/ demolition site/ tip?			
Yes	443	80	364*
No	417	48	267
Near metal-using industry?			
Yes	453	81	407***
No	408	47	248
House < 10 m from nearest road?			
Yes	481	78*	389***
No	361	42	216

Significance levels: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

also noted in gardens in close proximity to (within a 500 m radius of) waste land, demolition sites and tips, 364 $\mu g\,g^{-1}$ compared with 267 $\mu g\,g^{-1}$ ($p < 0.05$), and those in close proximity to a metal using industry, 470 $\mu g\,g^{-1}$ compared with 248 $\mu g\,g^{-1}$ ($p < 0.001$). Garden soils from houses within 10 m of the nearest road were also found to have significantly higher lead concentrations than those from houses situated at a greater distance than this, respective means being 389 and 216 $\mu g\,g^{-1}$ ($p < 0.001$). Housedust lead loadings were also elevated in this situation, with means of 78 and 42 $\mu g\,m^{-2}$, respectively ($p < 0.05$).

The concentration of lead in urban street dusts is typically higher in the vicinity of premises which handle or process lead than in residential areas (Royal Commission on Environmental Pollution, 1983). In an earlier study in

Birmingham (1972–1975), Archer and Barrat (1976) reported a median lead value of 1300 $\mu g\,g^{-1}$ for 117 samples collected from industrial estates roads and a lower median value of 950 $\mu g\,g^{-1}$ for 85 residential road dust samples. The same trend was apparent from the results of the present study, although the actual values were very much lower. The geometric mean lead concentration of the industrial road dust samples was 791 $\mu g\,g^{-1}$ (n = 58, 5th–95th percentile = 220–3340 $\mu g\,g^{-1}$) and was significantly higher ($p < 0.01$) than that found for the residential road dust samples of 527 $\mu g\,g^{-1}$ (n = 97, 5th–95th percentile = 195–1170). Although it is appreciated that the sample collection procedures and analytical techniques employed by Archer and Barrat (1976) were different from those used in this study and that the data, therefore, are not strictly comparable, some useful observations can be made. The ratio of residential to industrial road dust lead in the earlier study (0.67) was similar to that found in this study (0.73). The level noted for industrial road dusts was 61% of that reported by Archer and Barratt (1976), while that noted for residential areas was 56% of the figure reported by these workers, which suggests that the lead levels in Birmingham road dust have decreased. A drop in industrial lead emissions could have resulted in such a finding but, unfortunately, it is difficult to confirm this as there is little information concerning changes in lead emissions from such sources over this period. There was a phased reduction in the lead content of petrol from 0.84 $g\,l^{-1}$ in 1972 to 0.4 $g\,l^{-1}$ in 1981, although national figures indicate that total lead emissions from petrol combustion remained fairly stable, despite this reduction, due to an increase in traffic levels (Department of the Environment, 1986). A further reduction, to a maximum of 0.15 $g\,l^{-1}$, came into effect at the beginning of 1986 and it will be of interest to continue monitoring dust lead levels to establish whether or not there is a corresponding decrease in these values.

CONCLUSIONS

Housedust and road dust lead concentrations in Birmingham were lower than the national means, although soil lead levels were slightly higher. Road dust samples from industrial areas had significantly higher lead concentrations than those from residential areas.

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Air lead concentrations in Birmingham, England – a comparison between levels inside and outside inner-city homes

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ABSTRACT

Air lead concentrations were monitored for seven consecutive days at two locations inside and one location immediately outside 97 houses in inner-city Birmingham. Concentrations in the bedroom and playroom were similar to each other, but usually lower than those found outdoors. The mean internal air lead concentration was 0.26 μg/m^3 (n=1206) and the mean external concentration, 0.43 μg/m^3 (n=605). The mean of the individual internal:external ratios was 0.61 and similar to others cited in the literature. The study indicates that, in the absence of any major internal sources of lead, the mean external air lead concentration is a good predictor of the mean air internal air lead concentration.

INTRODUCTION

Lead in air contributes to man's total lead intake both directly and indirectly. Inhalation of lead-bearing particulate matter and its subsequent retention in, and absorption from, the respiratory system provides the most direct route of transfer (Chamberlain, 1985). Other, less well defined and indirect, pathways result from the deposition of such airborne particulate matter. For example, plants grown in soil into which deposited lead has become incorporated are eaten by man. Also, deposited dust may inadvertently be ingested through hand-to-mouth activity, particularly by children (Department of Health and Social Security, 1980, Royal Commission on Environmental Pollution, 1983).

A study, recently commissioned by the U.K. Department of the Environment in the inner-city area of Birmingham, was designed to provide quantitative information on the intakes of young children *via* all the major pathways, simultaneously. Housedust, pavement and road dust, soil, water, food, blood, handwipe and air samples were all obtained. The operational details and results from the air monitoring programme incorporated into this study are presented and discussed in this paper.

STUDY DESIGN

The study was carried out between November, 1984 and February, 1985 in 97 inner-city Birmingham households (see Thomas et al., 1985 for the selection criteria). Air samples were obtained from three locations, the child's bedroom, the child's main playroom and immediately outside the house, for seven consecutive days. Aerosol monitors (Millipore, MAWPO37AO, containing 37 mm, 0.8 μm cellulose acetate filters) were positioned at a height of 1.5 to 2 m and attached to aerosol adaptors (Millipore, XX6200004) incorporating 2 l/min flow limiting orifices (Millipore, XX5000020). Ideally, the aerosol monitors should have been positioned lower than this, at around 1 m, to sample from within the child's breathing zone. Unfortunately, this was not a practical alternative as, during pilot trials, monitors at this height were continually interfered with and sometimes even removed by the child. The aerosol monitors were, in turn, connected, through P.V.C. adaptors, to clear plastic P.V.C. tubing (3 mm I.D.) which led back to one central Millipore vacuum pump (XXMJ22050) [see Figure 1]. Within the

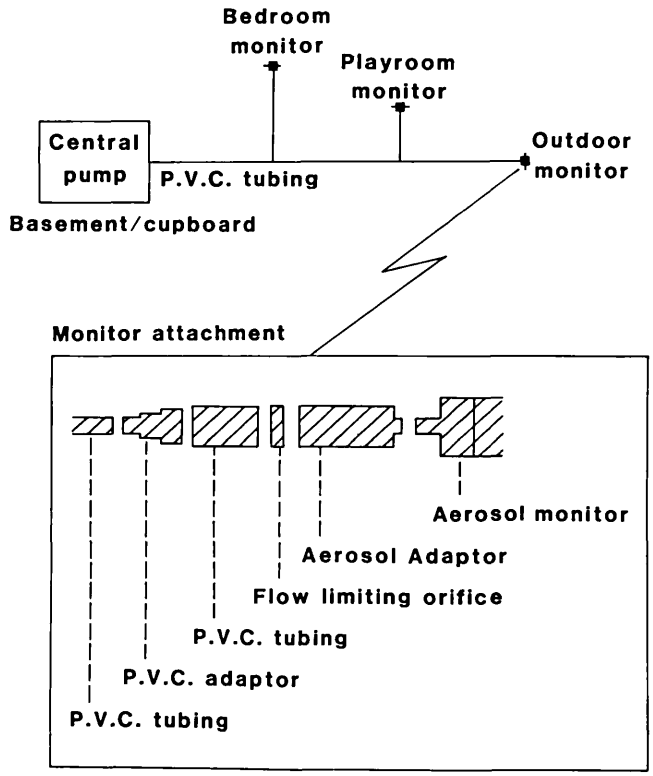


Figure 1 *Schematic diagram of household air monitoring network.*

home the tubing was secured to the bottom of the skirting board using staples and the pump was located in an appropriate place, such as a basement or cupboard under the stairs, to minimise the intrusion of noise. Elapsed time meters (R.S. Components Ltd. 259–858) were fitted to the pumps to ensure that the actual operating time could be recorded. The aerosol monitors were changed daily, at which time the flow rate was checked using a rotameter. Personal exposure monitors were considered for use in this study but were found to be unsuitable during pilot trials; not only was the sampling device a heavy burden for such young children, but the children frequently tampered with the equipment (turning off the pump, removing the tubing, etc.).

ANALYTICAL TECHNIQUES

To minimise the chance of contaminating any of the samples, analytical procedures were carried out in a clean laboratory with a filtered air supply. Each air filter was carefully transferred from the sealed monitoring head into a beaker (50 cm^3, acid washed, rinsed in deionised water [D.I.W.].) using plastic forceps. Four cm^3 of concentrated nitric acid (Aristar, BDH) were added and the sample was heated gently (at 60°C) until the filter had dissolved. The sample was then transferred into a test tube (acid washed, rinsed in D.I.W.) and the beaker was rinsed with a few cm^3 of D.I.W. which was also added to the test tube. One cm^3 of perchloric acid (Aristar, BDH) was then added and the test tube placed in a deep, programmable heating block. The following heating programme was used to complete the digestion and take the samples to dryness.

RISE TIME (SEC/DEG)	DWELL TIME (HRS)	TEMP. (°C)
001	1.0	100
300	6.0	125
180	3.0	150
001	16.0	190

Two cm^3 of 1 M hydrochloric acid (Aristar, BDH, with 5 mg/l La "spike" as an internal standard) were added and the sample was heated gently (60°C) for one hour. The solution was then transferred, without rinsing, to a disposable tube and analysed by Inductively Coupled Plasma Atomic Emission Spectroscopy (ICPAES). A rigorous quality control programme was implemented, which consisted of reagent blanks, procedural blanks, lead-only spikes and multi-element spikes and represented 20 percent of the total number of solutions analysed.

RESULTS AND DISCUSSION

The population distributions for samples taken in the bedroom, playroom and outdoors were all log-normal, therefore geometric means have been calculated and included in the summary table (Table 1). The log-transformed (log_{10}) data can be seen to show normal distributions (Figure 2).

The mean air lead concentration in the bedroom was 0.26 µg/m3 (n=599) and was similar (no statistically significant difference, $p>0.05$) to that found in the playroom, 0.27 µg/m3 (n=607). The mean external air lead concentration, 0.43 µg/m3 (n=605), was significantly higher than those found indoors ($p<0.001$) although not as high as other figures published previously for outdoor air lead in Birmingham. In 1983/4 the city-wide annual average from 9 sites was reported to be 0.79 µg/m3 (City of Birmingham Environmental Health Department, 1985). Archer and Barratt (1976) reported a median value of 0.75 µg/m3 from 15 sites across the city in 1975, with average weekly means ranging from 0.4 to 1.4 µg/m3. Still higher concentrations were recorded at a site alongside the Gravelly Hill motorway interchange during the same year and

Table 1 *Summary of internal and external air lead concentrations.*

	Air Lead Concentration: µg/m3			
	Bedroom	*Playroom*	*Internal*	*External*
N	599	607	1206	605
Arithmetic mean	0.32	0.34	0.33	0.55
Range	<.05–1.52	<.05–1.96	<.05–1.96	<.05–2.98
Geometric mean	0.26	0.27	0.26	0.43
5th percentile	0.09	0.08	0.08	0.12
95th percentile	0.81	0.88	0.87	1.53

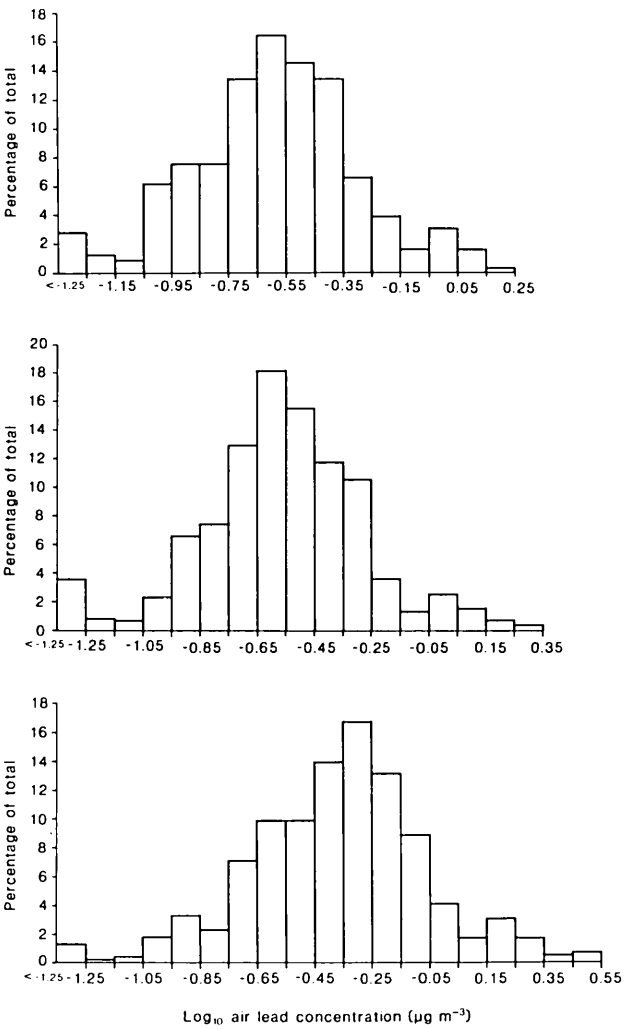


Figure 2 *Frequency distributions of log-transformed (log10) air lead data for a) Bedroom, b) Playroom, c) Outdoors.*

resulted in an annual mean (average of monthly means) of 1.79 μg/m3 (Department of the Environment, 1978). These values can be compared with a five year average (1976 to 1981) of 0.65 μg/m3 from five urban sites in England and Scotland (McInnes, 1982).

The large nature of the data base prohibits the inclusion of all the results in this paper and selected examples have therefore been drawn to illustrate the situation in individual houses. The general pattern that emerged is shown in Figure 3 (a, b and c) where the air lead concentration has been plotted against sampling day for houses 19, 29 and 30. There are two important points to note. 1: that the internal air lead concentration plots closely follow those for external air but are always lower. This suggests that the external lead level has a large influence on the lead level found indoors and also suggests that the physical structure of the house (walls, doors, windows) prevents some of the lead-bearing suspended particulate matter reaching the indoor air. 2: that the plots for each house are very similar to one another, each showing, for example, a small peak on day 12 and a larger peak on day 15. This suggests that the same air mass was being sampled, even though the houses were scattered throughout the study area. It is thus likely that meteorological factors, such as wind speed and direction or rainfall, were influencing the air lead concentration in the study area. Weekly climatological summaries were obtained from the Department of Geography, University of Birmingham, during the study period and a detailed analysis of these and the air lead data will be presented elsewhere. The data from house 6 have been plotted (Figure 3d) and provide an interesting deviation from the norm. Here it can be seen that the internal air lead concentrations are very much higher than the external concentrations on days 3 and 4 which suggests that there was an internal source of lead contributing to the indoor air lead burden on these days. This property was being redecorated at the time of the study and old paint was being removed using a machine sander. It has been demonstrated elsewhere that stripping paint in this manner can result in large increases in environmental lead levels within a home. Inskip and Hutton (1987) found that air lead and deposited dust lead levels were highest after sanding although 'burning off' and 'hot air' paint removal methods also produced significant contamination. The dust lead concentration from this householder's vacuum cleaner was higher than in any other house sampled, at 23000 μg/g.

The internal:external air lead ratio was calculated for each house and revealed a mean value of 0.61, range 0.39–1.33. It is interesting, but not surprising, to find that the highest ratio (1:33) occurred in house 6. There is not a vast literature on internal:external air lead ratios but from a review of the available data, Yocum (1982) suggested that ratios of 0.6 to 0.8 were typical for houses without air conditioning (none of the houses in this study was air conditioned). The few such studies carried out in the United Kingdom have indicated, from limited data bases, ratios of between 0.6 and 0.7 (Lawther *et al*, 1973, Chamberlain *et al*, 1978 and Department of the Environment, 1978).

The mean internal air lead concentration has been plotted against the mean external air lead concentration for each house and reveals a positive correlation

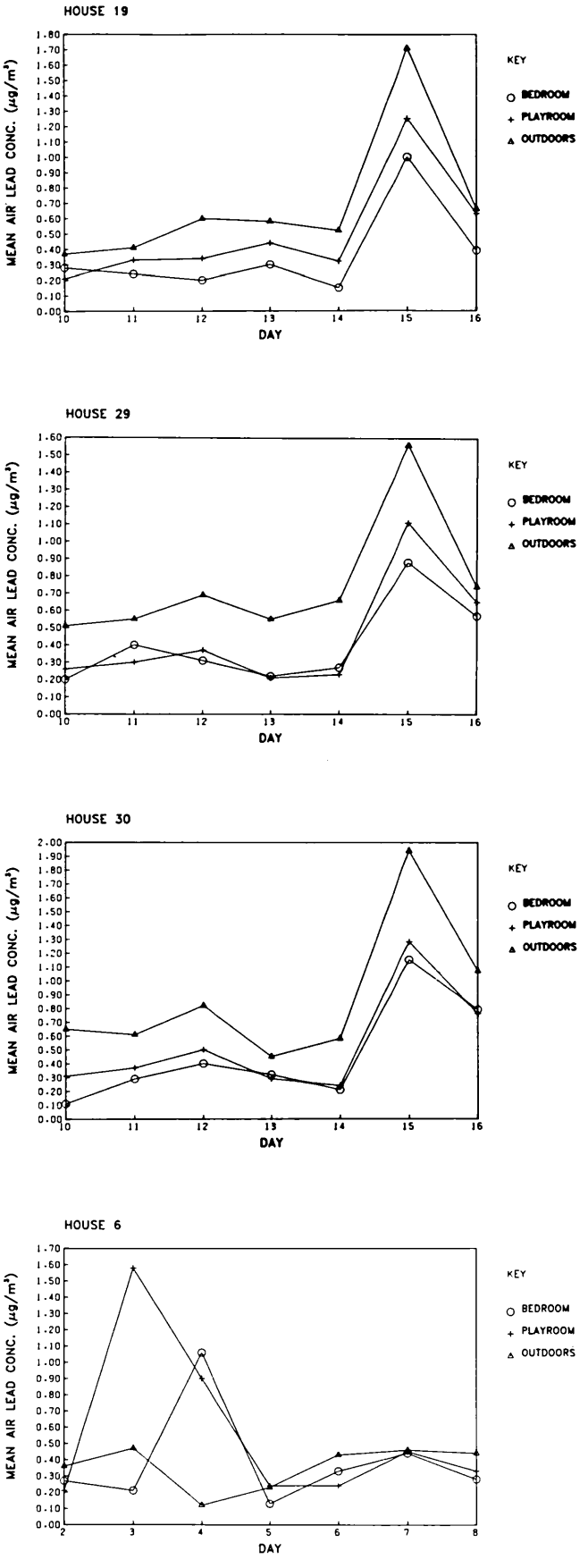


Figure 3 *Bedroom, playroom and outdoor air lead concentrations for a) House 19, b) House 29, c) House 30, d) House 6.*

(Figure 4a). Assuming a linear relationship, the regression equation was calculated by the least squares method to be:

Internal air lead conc. = 0.0963 + 0.419 (External air lead conc.) (I)

The regression line has been fitted to the data and is included in Figure 4a. The variance ratio (F) was 287.7 ($p<0.001$, r2 = 76%) which provides evidence that the slope is probably not zero and implies that the weekly mean external air lead concentration is a useful predictor of the weekly mean indoor air lead concentration.

Although the data have been handled on the basis of individual houses above, it is also possible to pool the data from all sampling locations for individual days during the study period to gain information on average levels throughout the city on any given day. The results of such a treatment of the data are presented in Figure 5 (there are no results for days 17 to 42 due to the Christmas break). The relationship between external and internal levels outlined above is confirmed by this plot and the similarity between bedroom and playroom levels already noted is very apparent. Daily internal and external means have been compared in Figure 4b. The straight line regression equation was calculated from these data to be:–

Internal air lead conc. = 0.0555 + 0.502 (External air lead conc.) (II)

and the regression line has been included on this graph ($p<0.001$). The large r^2 value, 97%, indicates that the mean daily external air lead concentration across the study area is an excellent predictor of the mean daily internal air lead concentration, a finding which could have important implications in the design of future

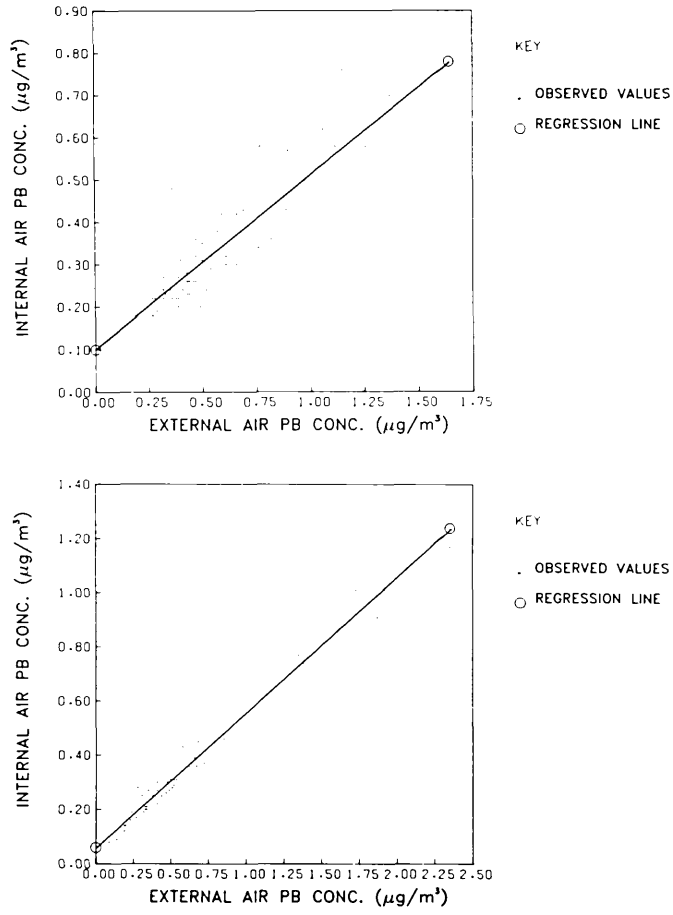


Figure 4 *Mean internal versus mean external air lead concentration for a) Individual houses, b) Individual days.*

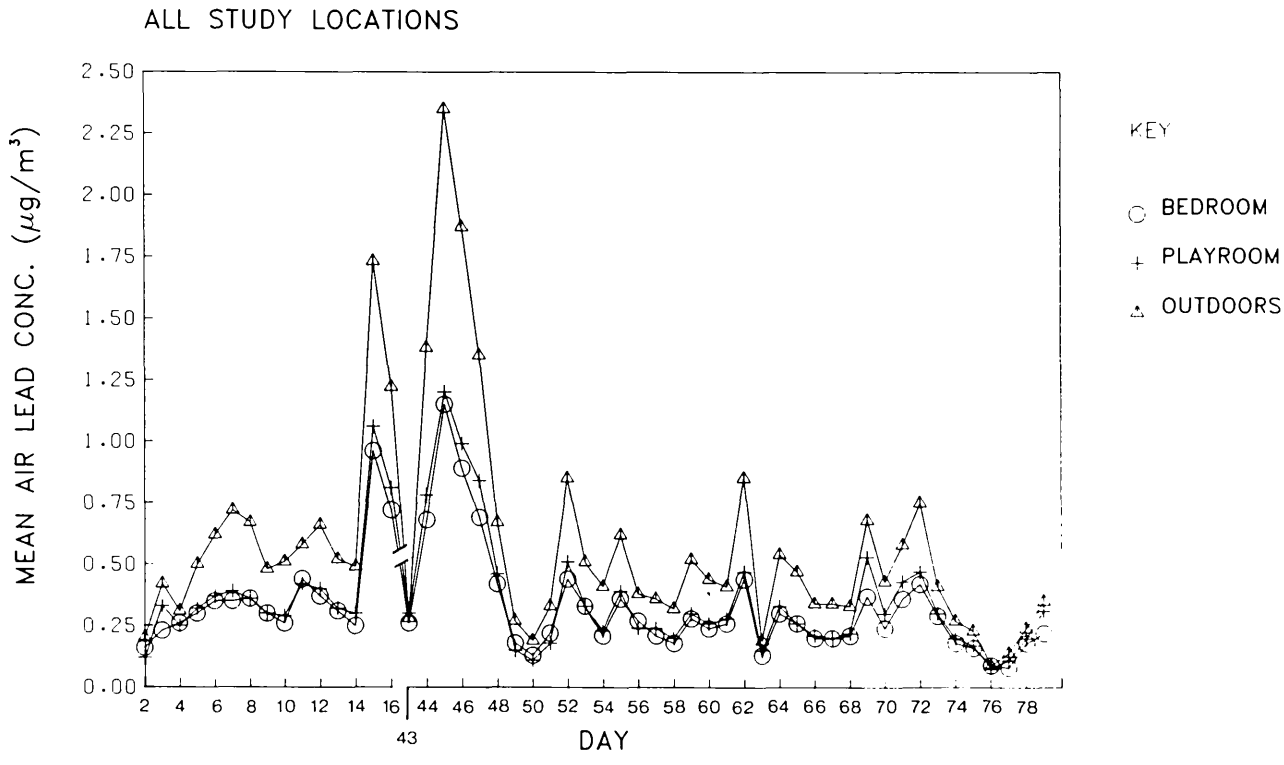


Figure 5 *Mean bedroom, playroom and outdoor air lead concentrations from all sampling locations on each day of the study.*

studies directed at assessing the exposure of young children to lead via inhalation. It is cheaper and less complicated to implement an 'outdoor only' air monitoring programme rather than to combine it with an indoor programme and the data generated could be used to estimate indoor concentrations by substitution into an appropriate regression equation.

From the two regression lines fitted to the data (Figures 4 a and b and equations I and II) it can be seen that neither of the intercepts are zero. This indicates that if the external air lead concentration is zero there will still be lead in the internal air and suggests that there is another source (or other sources) contributing to the indoor air lead burden. It is felt that the most likely source is housedust (which this and other studies have shown to contain lead to varying degrees) which has been resuspended by the day to day activities of the householders and, where appropriate, their pets. Further work is required in this area and could, for example, involve the spiking of housedust with a tracer and the subsequent mapping of the tracer's movement within a house.

CONCLUSIONS

The air lead concentrations recorded in inner-city Birmingham were relatively low, the mean external concentration being 0.43 $\mu g/m^3$ and the mean internal concentration, 0.26 $\mu g/m^3$. The mean of the individual internal:external ratios was 0.61 and similar to others cited in the literature. The study indicates that, in the absence of any major internal sources of lead, the mean external air lead concentration is a good predictor of the mean air internal air lead concentration.

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An Assessment of the Quantity and Significance of Lead on the Hands of Inner-City Children in the United Kingdom

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ABSTRACT

The exposure of young children to environmental lead has received much attention in recent years. The ingestion of lead contaminated dust from children's hands is thought to be an important route of intake for this group. A study was carried out to determine the quantity of lead present on the hands of around 100 children (approximately 1-3 years of age) attending playgroups in inner-city areas in the United Kingdom. The mean lead levels found (ug total both hands) ranged from 3.8-8.5 ug Pb.

The use of the handwipe method in a large, integrated study, recently completed by the Applied Geochemistry Research Group and others in 97 inner-city homes in Birmingham, U.K., is outlined. The significance of this pathway to the child's total intake is also discussed.

INTRODUCTION

The ingestion of lead contaminated dust from children's hands has been suggested as an important route of intake of lead for this group. The Royal Commission on Environmental Pollution in their 9th Report, on Lead in the Environment (12), conclude that...."Although the limited data involve theoretical calculations, we believe the intake of lead from dust can be substantial for young children". Several studies have been carried out in recent years to determine the quantity of lead present on children's hands in a number of different situations (3,6,8,10,13,14,15). Using a variety of assumptions attempts have then been made to quantify lead intake via this route. Estimates of daily lead intake appearing in the literature range widely between 8 and 1000 ug per day, although most

estimates fall between about 10 and 100 ug per day.

In the United Kingdom, very few such investigations have been carried out to date and data are lacking for pre-school children. It was decided, therefore, to initiate a small survey in playgroups in 3 British cities (Bath, Birmingham and London) to determine the quantity of lead present on the hands of this population. This exercise also served as a useful pilot study for the handwipe method which was incorporated into a large, integrated environmental, biological and behavioural study in Birmingham, England. This integrated study was designed to quantify the intake of lead by 2 year old children via both the inhalation and ingestion routes simultaneously.

Ninety-seven children were studied in their own homes over a seven day period and, along with handwipes, samples including housedusts, pavement and road dusts, air (both indoor and outdoor), soil, tap water, blood and food were obtained. Also video films of the children were taken in each home in order to quantify their mouthing behaviour.

The handwipe data from the playgroup surveys and the integrated study are presented in this paper and the significance of this pathway is discussed.

METHODS

The sampler's hands were washed with soap and water and wiped with an "All-Fresh" wet-wipe which was then placed in a self-sealing plastic bag. Both of the child's hands were then wiped thoroughly with a total of 3 wet-wipes and the wipes were saved for analysis, again in a self-sealing plastic bag. In the laboratory, the wipes were transferred into a conical flask and 30ml of concentrated nitric acid were added. The flask was covered with a watchglass and heated gently until the wipes had disaggregated. The temperature was then increased to 110^{o}C for a further 48 hours and the sample taken to dryness and leached with 2ml of 6M HCl. It was made up to a final volume of 10ml with de-ionized water and analysed by atomic absorption spectrophotometry.

An extensive quality control programme was implemented which included, randomly with each batch of 50 flasks:-

Reagent blank (1)

Procedural blank (2)

Wipes spiked with 10 ug Pb (1)

Wipes spiked with 50 ug Pb (1)

Wipes spiked with 50mg of in-house dust reference material (2).

Plus with each batch of 150 flasks:-

Wipes spiked with 10mg of National Bureau of Standards Reference Material 1648 - Urban Particulates (1).

RESULTS AND DISCUSSION

The frequency distribution of the data from the Bath playgroup has been plotted (Fig. 1). Due to the skewed nature of the distribution the geometric, rather than the arithmetric, mean was calculated and found to be 6.1 ug Pb total for both hands. It should be pointed out that a total of only 2

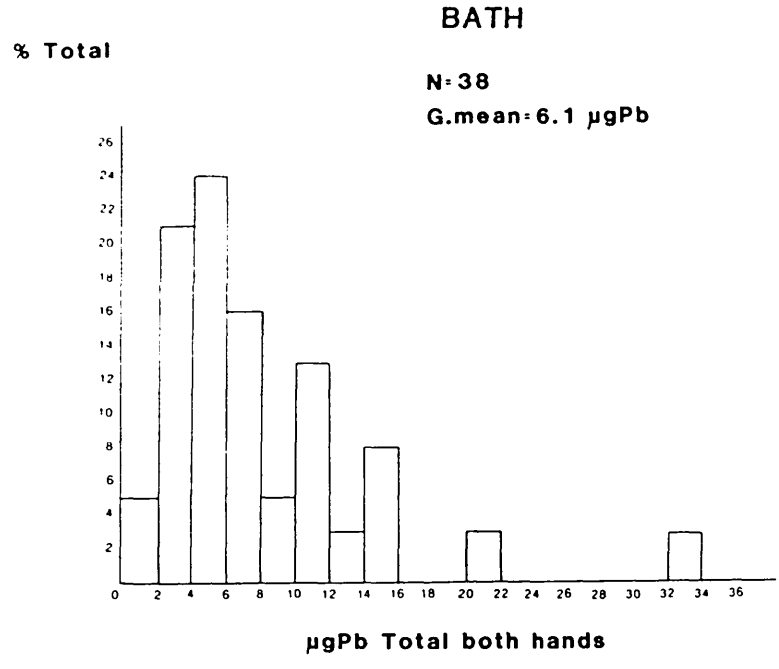


Figure 1

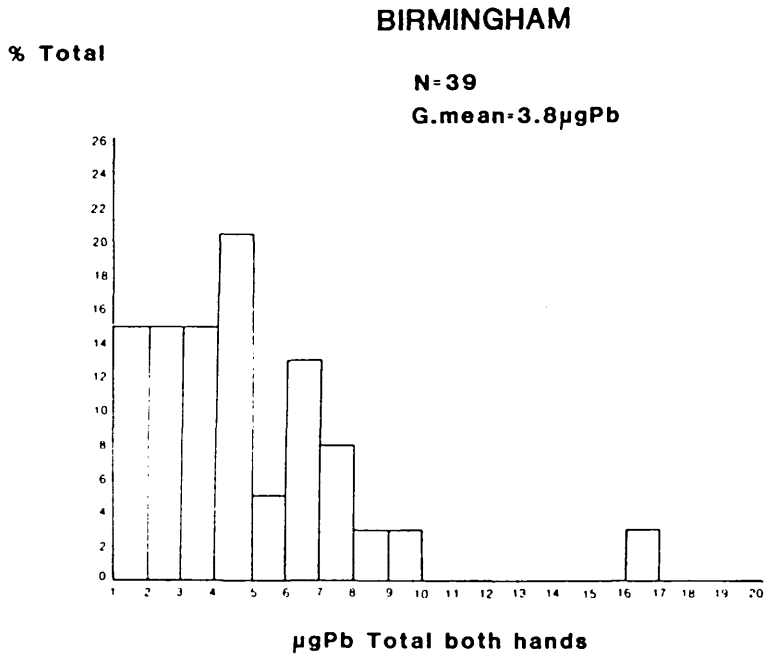


Figure 2

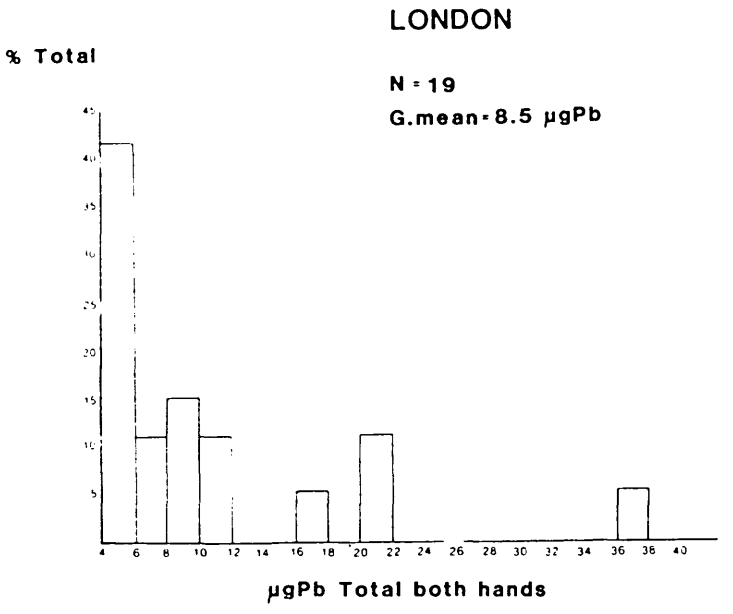


Figure 3

handwipes per child was used in this playgroup. The method was subsequently updated in the light of recovery trials which indicated that a total of 3 wipes was most appropriate. The rest of the data were derived from this updated method and are therefore not strictly comparable with these data. The results from the Birmingham playgroup have been plotted (Fig. 2) and show a similar distribution, although the geometric mean is lower, 3.8 ug Pb. Data from the London playgroup are more limited (Fig. 3) and reveal a geometric mean of 8.5 ug Pb. In the large data set from the integrated study (Fig. 4) again one finds this familiar distribution and the mean is 5.7 ug Pb.

The data have been summarised in Table 1. The mean values found are generally lower than others cited in the literature and this is likely to be due to the fact that the majority of children studied were 2 year olds and none were older than 3. Vostal et al (15) reported a median value of 20 ug Pb per hand for urban children aged 2-6, while Roels et al (11) found a range of 13-20 ug Pb per hand for an urban group aged 10-14 years. Brunekreef et al (2) have reported a mean

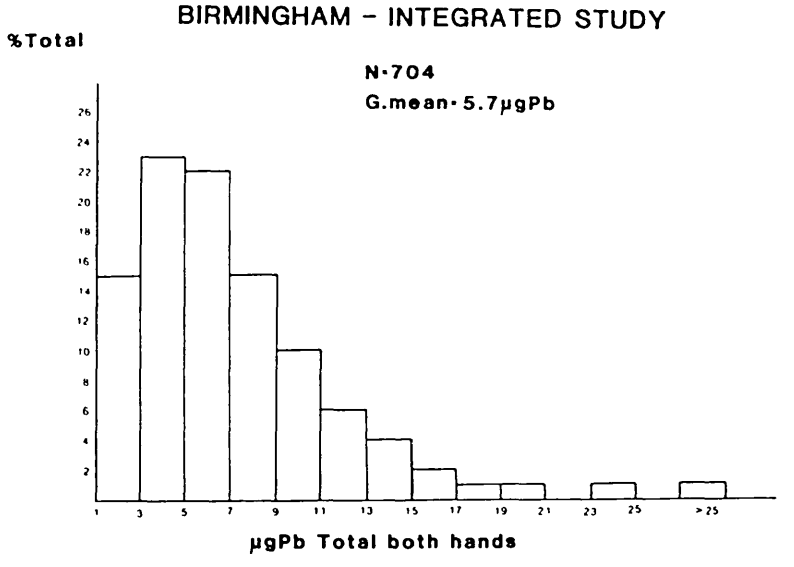


Figure 4

TABLE I - HAND LEAD (ug total both hands)
U.K. CHILDREN

	n	G. mean ug Pb	Percentile 5th	Percentile 95th	Situation
Bath	38	6.1	1.7	18.3	Playgroups
Birmingham	39	3.8	1.3	9.1	
London	19	8.5	4.4	30.2	
Birmingham	97	5.7	1.9	15.1	Households integrated study

of 12 ug Pb per hand for inner-city children aged between 4 and 6 in a recent study of 5 and 6 year olds in London, Duggan et al (8) found means ranging from 12-43 ug Pb per child in 11 infant schools. However, levels reported by Bornschein (1) of around 7.8 ug Pb per child for 18 month old children are low and closer to those found in our studies.

In the integrated study, the child's hands were wiped daily for 7 consecutive days and some of these data are presented (Table II). In general, there was little day to day variation and data from two typical Houses (10 and 72) are shown. There were exceptions such as in House 91 on day 4 and House 92 on day 5. It is not clear why such an extraordinarily

high level of 406 ug Pb was present on this child's hands in House 92 on this particular day although it is not surprising to find outliers in such a large data set (n = 704). If there had been a complete transfer of lead from hands to mouth, intake via this pathway would have been 20 times greater than that from the diet and around 200 times greater than that from the air.

TABLE II - TOTAL AMOUNT OF LEAD MEASURED DAILY ON BOTH HANDS (ug)

	House 10	House 72	House 91	House 92
Day 1	2.2	10.3	11.8	4.2
2	3.0	13.4	16.2	8.1
3	2.6	15.2	19.1	13.6
4	3.6	10.6	6.4	11.5
5	4.3	11.1	13.0	406.1
6	3.8	10.8	18.7	8.9
7	5.1	13.0	18.4	5.9

The data from the Bath, Birmingham and London playgroups (Table I) provide a useful indication of typical hand-lead levels in inner-city areas of the U.K., although in terms of assessing intake via this route they are of limited value. A more useful approach, with this goal in mind, is to incorporate an assessment of the hand to mouth activity of each individual child to supplement the measurement of hand lead. A child with a relatively low hand lead level exhibiting frequent hand to mouth activity may well ingest more lead via this pathway than a child with a high hand lead level that rarely places his hand in his mouth. In the Birmingham study, this was the type of approach used and video films were taken of each child in their home for approximately four hours. Each film included the

luncheon period, a 10 minute story, a 10 minute T.V. film and a period of free play. The films were subsequently analysed to quantify mouthing activity. The rating of these films has generated a vast body of data and the process of collapsing these data into a usable form is ongoing (9). The next stage will be to combine the resultant hand to mouth activity scores with the hand lead values obtained to provide a factor which reflects hand lead ingestion. This factor will then be incorporated, along with the other exposure variables measured, into a lead exposure model for the study population.

Many of the estimates in the literature concerning the amount of lead ingested by mouthing children are based on the assumption that a child ingests 100mg of dust per day (6,7,12,14). Data from the Birmingham study revealed a mean housedust lead concentration of 424 ug g^{-1} which would lead to an intake of 42.4 ug per day on this assumption. Noting that the mean hand lead level in this study was 5.7 ug, it would require the transfer of all hand lead from hand to mouth a total of seven times per day to result in this intake. This is only a crude estimate as the state of knowledge to date regarding dust ingestion is extremely poor. It should also be mentioned that recent studies by Duggan and co-workers (8) showed that the majority of particles found on hands were below 10 um and that the maximum particle diameter was 100 um. This is an important finding when one considers that, for a sieved dust, the lead concentration usually increases as the size fraction decreases. It may well be more appropriate to analyse the finer dust fraction rather than the less than 1000 um fraction which is the routine procedure followed by most workers currently, including ourselves.

Results from pilot studies suggested that the exposure of the child to lead in housedust is expressed more realistically by lead loadings than simply lead concentrations (4). This is supported by the results from the integrated study. If one looks at the association between housedust lead

and hand lead in terms of correlation coefficients, there is a positive correlation of 0.44 between lead loading and hand lead and a positive correlation of only 0.24 between housedust lead concentration and hand lead.

A summary exposure assessment has been carried out using the data from the integrated study and is discussed in detail elsewhere (5). The assessment indicates (Table III) that intake via the inhalation route accounts for only 3 percent of the total daily lead uptake. For the ingestion route, the contribution to lead uptake made by the intake of dust is around twice that attributable to the diet, the estimates being 22.5 and 12.2 ug per day, respectively. The assumptions used for this assessment are the same as those employed by the Royal Commission (12) and it is recognised that there are considerable uncertainties in the estimates of intake rates, retention and absorption factors used to derive these figures. They should, therefore, only be considered as general indicators of the significance of the various pathways.

TABLE III - INTAKE AND UPTAKE OF LEAD BY YOUNG CHILDREN IN BIRMINGHAM

Intake route	Pathway	Lead uptake (ug/d)	Total uptake (%)
Inhalation	Air	1.1	3
Ingestion	Diet	12.2	34
	Dust	22.5	63
Total		35.8	

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RELATIONSHIP BETWEEN BLOOD LEAD AND LEAD INTAKE IN TWO YEAR OLD URBAN CHILDREN IN THE UK

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ABSTRACT

A comprehensive study of a group of 97 two year old children in Birmingham (UK) included measurement of lead in samples of venous blood, internal and external air, house dust, pavement and road dust, garden soil, handwipes, diet (food and beverages) and water. Children's behaviour was recorded on video and quantified assessments made of both mouthing activity and hand contact. Regression analyses indicated that blood lead concentration was significantly related to a combination of dust lead loading and an overall rate of touching objects and to water lead concentration. A summary assessment of exposure, on the basis of assumptions used by the UK RCEP, gave an estimated total uptake of 36 ug/day (one third of that estimated by the RCEP); of this, 97% was from ingestion and only 3% from inhalation.

INTRODUCTION

The exposure of young children to environmental lead has received much attention in recent years, with emphasis on the effects on intelligence, attainment and behaviour. However, it is still unclear whether or not the intake of relatively small amounts of lead has a detrimental effect (1, 2). Various studies have investigated the magnitude of the different sources of lead in the environment and have attempted to assess the significance of the various pathways to the child's total lead intake, but often have looked only at single pathways in isolation. This paper reports the results from a comprehensive - environmental, biological, behavioural and dietary - study of 2 year old children in Birmingham (UK).

SUBJECTS AND METHODS

Subjects The full criteria for selection have been described elsewhere (3); in brief, from 183 randomly selected children who had been born in, and were still resident in, central Birmingham and who were then aged 24 ± 2 months, a stratified subset of 106 was selected; of these, 97 completed the study. Field work was carried out from November 1984 to February 1985. Venous blood samples were taken at the beginning of the study; 57 children also provided a second sample some 5 months later.

Environmental Measures (a) Air: 24 hour samples were obtained for each of 7 consecutive days from the child's main playroom, the child's bedroom and immediately outside the house (4, 5). (b) House dust: samples were taken from the household's own vacuum cleaner; and, using an adapted vacuum cleaner (6), from the child's main play room, the child's bedroom and under the doormat. (c) Pavement and Road Dust: composite samples were taken from immediately outside each house. (d) Soil: a composite surface (0-5 cm) sample was collected. (e) Handwipe: on each day, both hands were wiped thoroughly using 3 wet wipes (7). (f) Diet: a duplicate of the child's diet, as eaten and covering 7 days was collected along with a 7 day composite sample of drinking water. It was not possible to obtain all of the above samples for every child.

Behavioural Measures Each child's behaviour was recorded (using a standard portable autofocus colour camera and video recorder) over a session of 4 hours including a period of free play, lunch, a 10 minute story and a 10 minute video film (8).

Analytical Techniques Air (4, 5) and dust and soil (6) samples were analysed by inductively coupled plasma atomic emission spectroscopy; handwipes (7), food and water (9), and blood (10) by atomic absorption spectrophotometry. Samples of the video tapes were analysed, using a detailed set of behavioural categories for both mouthing activity and hand contact, with the aid of a specially written computer programme (8).

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RESULTS AND DISCUSSION

Blood The geometric mean blood lead concentration, 11.7 ug/100ml (5th and 95th percentiles 6 and 24 ug/100ml, n = 97) was similar to that found previously among children of this age in central Birmingham (11). Agreement between repeated samples was very good (r = 0.82) (12).

Air In each house, the internal samples (play room and bedroom) were very similar; the overall geometric mean was 0.26 ug/m^3 (0.08 - 0.87 ug/m^3, n = 1206). The geometric mean external air lead concentration was 0.43 ug/m^3 (0.12 - 1.53 ug/m^3, n = 605); the internal: external ratio of 0.61 is similar to others cited in the literature (13, 14). The correlation between the external and average internal air lead levels in individual houses was very high (r = 0.87) (4, 5).

Dust and Soil The geometric mean lead in house dust, 424 ug/g (138 - 2093 ug/g, n = 94) was slightly lower than the national figure (15), although soil lead, 313 ug/g (92 - 1160 ug/g, n = 87) was slightly higher. The correlation between dust and soil lead concentrations was fairly low (r = 0.28). The age of the house influenced lead levels in both house dust and garden soil, older houses (over 35 years) having higher concentrations. Houses being decorated had significantly higher lead concentrations than those that were not. Elevated lead levels were also noted in house dust and soil samples from houses located within a quarter of a mile of commercial garages. Increased lead concentrations were found in soil from gardens in close proximity to waste land (demolition sites and tips) and metal using industries, and from those within 10 yards of a road. Road dust samples from industrial areas had significantly higher lead concentrations than those from residential areas (6). The average amount of lead in house dust (the "loading") was 60 ug/m^2 (4 - 486 ug/m^2, n = 93).

Handwipes The geometric mean amount of lead on the hands was 5.7 ug (1.9 - 15.1 ug, n = 704); this is lower than values from several studies involving older children, but similar to that of 7.8 ug for 18 month old children reported by Bornschein et al (16). It has widely been assumed (17) that a child ingests 100 mg of dust per day, on which basis the average intake of lead in this study would have been around 40 ug per day; this would require the transfer of all hand lead from hand to mouth 7 times per day. The amount of lead on children's hands was more closely related to the dust lead loading (r = 0.44) than to the concentration (r = 0.24) (7).

Diet The geometric mean intake of lead in the diet (food and beverages) was 161 ug/week (82 - 389 ug/week, n = 96); this is higher than in a similar study in Harrow (18) and could, in part, be due to the higher water lead concentrations in Birmingham where the geometric mean was 19 ug/l (5 - 100 ug/l, n = 96) and 45% were above 20 ug/l compared with only 5% in Harrow (19). Nine percent of the children exceeded the current Joint FAO/WHO Expert Committee on Food Additives provisional tolerable weekly intake for infants and young children (25 ug/kg body weight/week), although none of these exceeded the DHSS advisory action level for lead in blood (25 ug/100ml).

Behavioural Measures Results have been described in detail elsewhere (8); they present some difficulty in interpretation because there are no other data available with which to compare them, but it is clear that it is both possible and practicable to obtain detailed, reliable and valid quantification of a specific aspect of the young child's behaviour in the natural environment. A breakdown of the data into different behavioural clusters and different settings gives a reasonably detailed picture. However, an average taken over all behaviours and all settings indicates that something - objects or parts of the body - touches the mouth area or goes into the mouth 1.8 times per minute; similarly, one or other hand touches something in the child's environment 15.8 times per minute. While these averages smooth out the situational differences that clearly exist, they form a basis for fitting into other models (8).

Relationships between Blood Lead, Environmental Lead and Behavioural Measures

The correlation of blood lead concentrations with average indoor air lead concentrations was virtually zero (4). As with the results for handwipes (7), the correlation of blood lead concentrations with lead loading in house dust (r = 0.46) was much higher than for the lead concentrations (r = 0.21). In addition, the correlation with the dust loading was higher than with the handwipe lead (r = 0.34). The correlation with soil lead concentrations (r = 0.18) was similar to that with dust lead concentrations. Whilst the correlations of blood lead with both dietary lead concentration and intake were very small (and not statistically significant), that with water lead concentration (r = 0.46) was similar to the correlation with dust lead loading.

The relative importance of the various sources of lead has been assessed using multiple linear regression (20). The indicator of dust lead levels used (X_1) was the dust lead loading multiplied by the rate for hands touching all objects (including the floor) and the "best" model (R^2 = 32%) was

$$\log \text{PbB (ug/100ml)} = 0.57 + 0.11 \log X_1 + 0.13 \log \text{PbW (ug/l)}.$$

The addition of air lead concentration, soil lead concentration or dietary lead intake (or any combination) gave non-significant regression coefficients for the variables concerned and only marginal improvements in the R^2 values. It would therefore appear that as an indicator of lead exposure a combination of the dust lead loading and the overall rate of touching objects was better than the amount of lead actually on the hand - either on its own or in combination with the rate of hand to mouth activity.

Assessment of Sources of Lead Exposure

The above analyses relate the varability in blood lead to the variability in the sources of lead. However, it is possible that a large proportion of blood lead might have arisen from one particular, predominant source which either does not vary much between individuals or which is measured with large "within individual" errors relative to those in blood lead (21). The following assessment of the relative importance of the sources of lead is based on assumptions used by the UK RCEP (17). With the average air lead concentrations described above, an average time spent indoors of just over 23 hours, and an assumed volume of air respired of 6 m^3/day, the lead intake from air would be 1.5 ug/day; assuming a combined retention and absorption factor of 0.7, lead uptake from air would be only just over 1 ug/day. On the assumption that 100 mg of dust per day is ingested, the intake from dust would be around 42 ug/day in addition to the 23 ug/day from the diet (food and beverages); assuming that lead from dust and from the diet are equally bioavailable, and that 53% of the total lead ingested reaches the bloodstream from the gut, the uptake by ingestion would be 35 ug/day. The total estimated uptake of 36 ug/day is only about one third of that calculated by the RCEP (17), but the proportions of 3% from inhalation and 97% from ingestion are similar (22). However, given the uncertainties surrounding the figures for dust ingestion and absorption by the gut, both the estimated proportions and the total amounts of lead from dust and from the diet must be treated with caution. In addition, the average figures conceal a large amount of variability: on the same assumptions, uptake could have been only 10 ug/day where environmental lead levels were low, or, in exceptional circumstances, around 180 ug/day in a house being redecorated (22). Work on both the regression models and the assessment of sources of lead exposure is continuing.

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INDOOR AND OUTDOOR AIR LEAD CONCENTRATIONS - IMPLICATIONS FOR THE HEALTH OF YOUNG CHILDREN

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ABSTRACT

Air lead concentrations were monitored for seven consecutive days at two locations inside and one location outside 97 houses in Birmingham, UK. Levels in the bedroom and playroom were similar, but usually lower than those found outdoors. The mean internal and external air lead concentrations were 0.26 ug/m^3 and 0.43 ug/m^3 respectively, and the mean ratio was 0.61. In the absence of any major internal sources of lead, the mean external air lead concentration is a good predictor of the mean internal values. The intake of lead via inhalation is of little significance to the total lead intake for two year old children.

INTRODUCTION

Lead in air contributes to man's total lead intake both directly and indirectly. Inhalation of lead bearing particulate matter and its subsequent retention in, and absorption from, the respiratory system provides the most direct route of transfer (1). Other, less well defined and indirect, pathways result from the deposition of airborne particulate matter. Crops, into which deposited lead has become incorporated, are eaten by man. Also, deposited dust may be ingested inadvertently through hand-to-mouth activity, particularly by children (2,3).

A comprehensive study designed to provide quantitative information on the intakes of young children, via all the major pathways simultaneously, has been carried out in Birmingham (4). Venous blood, air, house dust, pavement and road dust, soil, water, food, and handwipe samples were all obtained. The results from the air monitoring programme are presented in this paper, and are discussed in relation to an exposure model for two year old urban children.

STUDY DESIGN

The study was carried out between November 1984 and February 1985 in 97 households (for selection criteria see ref. 5). Twenty four hour air samples were obtained from the child's bedroom, the child's main playroom and immediately outside the house, for each of seven consecutive days. Aerosol monitors were positioned at a height of 1.5 to 2m and attached to aerosol adaptors incorporating 2 l/min flow limiting orifices (6).

ANALYTICAL TECHNIQUES

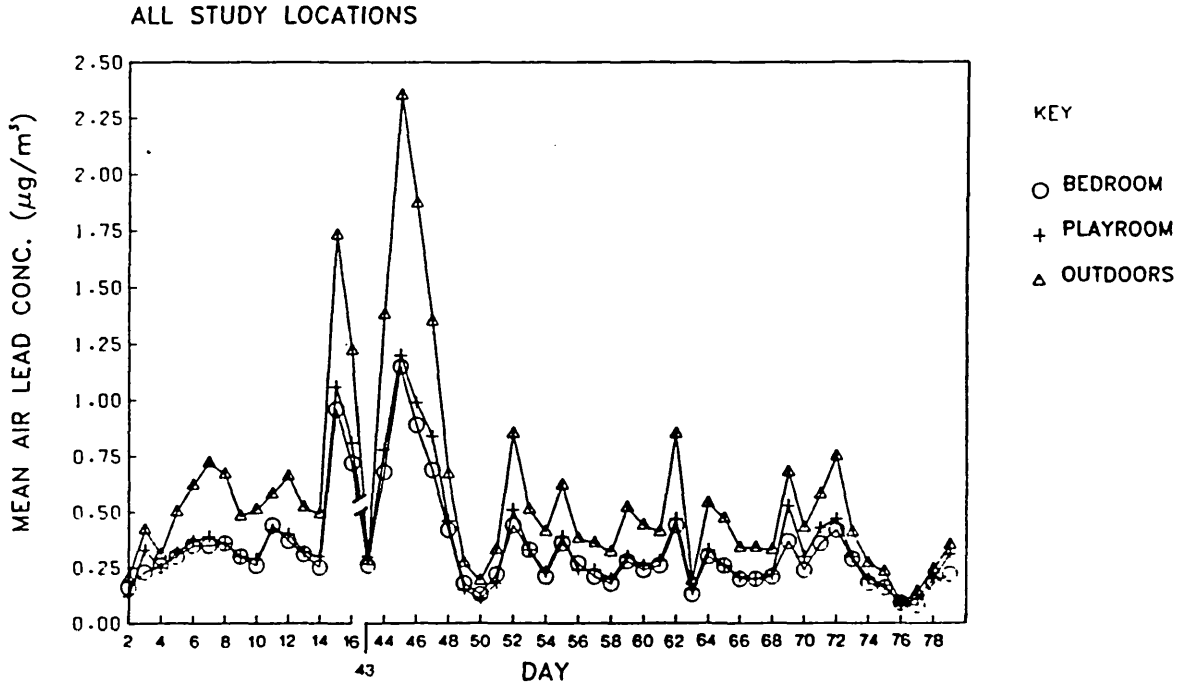
These have been detailed elsewhere (6). Briefly, the filters were digested in nitric:perchloric acid (in a ratio of 4:1), leached with hydrochloric acid and analysed by inductively coupled plasma atomic emission spectroscopy. A rigorous quality control programme was implemented.

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RESULTS AND DISCUSSION

The mean air lead concentration in the bedroom was 0.26 ug/m^3 (n=599), very similar to that in the playroom, 0.27 ug/m^3 (n=607). The mean external air lead concentration, 0.43 ug/m^3 (n=605), was significantly higher than those found indoors ($p < 0.001$) although not as high as other figures published previously for outdoor air lead in Birmingham (7,8,9).

Examination of the daily air lead concentrations in individual houses (6) reveals that 1: the internal concentration closely followed the external, but was usually lower. This suggests that the external air lead level has a large influence on that indoors, and that the physical structure of the house (walls, doors, windows) prevents some of the lead bearing suspended particulate matter reaching the indoor air. 2: the levels for houses sampled concurrently were very similar to one another, each peaking, for example, on the same days. This suggests that the same air mass was being sampled, even though the houses were widely scattered, and that meteorological factors, such as wind speed and direction or rainfall, were influencing the air lead concentration in the study area. The daily mean air lead concentrations are shown in the Figure below.



The mean internal:external air lead ratio was 0.61 (0.39-1.33). There is not a vast literature on internal:external air lead ratios but from a review of the available data, Yocum (10) suggested that ratios of 0.6 to 0.8 were typical. The few small studies carried out in the UK have indicated ratios of between 0.6 and 0.7 (9,11,12,).

The mean internal air lead concentration for each house was strongly correlated with the mean external concentration (6);

a linear regression gave:

$$\text{Internal PbA} = 0.096 + 0.42 * \text{External PbA}$$

with R^2=76% ($p < 0.001$). This indicates that the weekly mean external air lead concentration is a good predictor of the weekly mean indoor concentration. This finding could have important implications in the design of future exposure assessment studies as it is cheaper and less complicated to implement an "outdoor only" air monitoring programme; data could then be used to estimate indoor concentrations.

A summary exposure assessment for the study children (13) based on assumptions used by the Royal Commission on Environmental Pollution (3), gives an estimate of lead intake from air, via inhalation, of around 1.5 ug/day. This could result in an uptake of 1 ug/d or 3% of the estimated total daily uptake of 36 ug/day. Assessment of the intake of air lead via indirect, deposition pathways (e.g. diet, dust) is more difficult. In particular, there are no suitable data currently available concerning the proportion of house dust lead which is derived from air lead deposition. However, calculations by Chamberlain (14) showed that approximately one tenth of the total dietary lead intake could be due to fallout of lead from the atmosphere. The mean daily lead intake from the diet in the Birmingham study was 23 ug/day, which would result in an estimated indirect intake from lead in air of 2.3 ug/day. Applying an absorption factor of 0.53 (3) results in an estimated uptake of 1.2 ug/day, which is similar to that via inhalation.

The relative importance of the various sources of lead to blood lead has also been investigated using linear regression (4). The addition of air lead concentration to the different models tested gave non-significant regression coefficients and only marginal improvements in the R^2 values.

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Lead in the Home Environment

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10 Lead Levels in and around the Homes of Inner-city Children

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Introduction

Although several studies in recent years have investigated the exposure of young children to environmental lead, the relative contribution of the various pathways to their total lead uptake remains unclear (Royal Commission on Environmental Pollution, 1983; Department of Health and Social Security, 1980). In an attempt to assess the significance of the different pathways, a comprehensive study was commissioned by the UK Department of the Environment in the inner-city area of Birmingham. This was designed to provide quantitative information on the intakes of young children via all the major pathways simultaneously and was based on the analysis of environmental, dietary and biological samples which included house dust, pavement and road dust, soil, air, water, food, venous blood and handwipes. The results from the environmental section of the study are presented and discussed in this paper. The operational details and a full interpretation of all the results will be published elsewhere. A summary exposure assessment has been carried out and will also be presented at this meeting (Davies, 1987, this volume).

Study Design

The selection criteria for the study population have been described elsewhere (Davies, 1987, this volume). Sampling was carried out between November 1984 and February 1985. From in and around each house, the following samples were obtained:

1. House dust: a) from the householder's own vacuum cleaner; b) from the child's main playroom and bedroom and from under the doormat, using an adapted vacuum cleaner (Watt *et al.*, 1983).
2. Pavement and road dust: a composite sample of 25 subsamples.
3. Soil: a composite surface sample (0-5 cm) made up from 25 subsamples.
4. Air: 24 hour samples for each of 7 consecutive days from the child's main playroom and bedroom and from immediately outside the house.
5. Handwipe: both of the child's hands were wiped thoroughly, using 3 wet wipes, for 8 consecutive days.

Table 1. *Summary data for lead in house dust, soil, pavement and road dust*

	N	AM	Range	GM	Percentile 5th	Percentile 95th
Lead conc.(μg/g)						
Doormat	42	1436	79-15000	615	120	4300
Vacuum*	92	713	46-23000	336	97	1440
Bedroom	96	693	53-4500	464	109	2040
Playroom	97	455	62-5600	311	105	1030
Average**	95	578	111-3110	430	139	1780
Soil	86	405	56-1650	313	92	1160
Pavement	97	506	62-5100	360	127	1340
Road	97	805	80-2100	527	195	1170
Lead loading ($\mu g/m^3$)						
Bedroom	96	141	1-1348	51	3	417
Playroom	96	150	1-1799	42	1	614
Average**	94	149	2-1062	62	4	486

* From householder's own vacuum cleaner
**Bedroom and playroom

Analytical Techniques

Briefly, dust, soil and air filter samples were digested in a mixture of nitric and perchloric acids (ratio 4:1), leached with hydrochloric acid and analysed by inductively coupled plasma atomic emission spectroscopy. Handwipes were digested in nitric acid, leached with hydrochloric acid and analysed by atomic absorption spectrophotometry.

Results and Discussion

The results from the analysis of house dust, pavement dust, road dust and soil samples have been summarised in Table 1, while those for the air filters appear in Table 2. Data on hand lead levels are given in Table 3. Due to the log-normal distribution of all sample populations, geometric means have been included.

Dusts and Soils

The highest mean lead concentration in dust from within the home, 615mg/g, was noted in samples from under the doormat and was almost twice as high as those from

*[1]It should be noted that the mean house dust lead concentration used in the exposure assessment (see Davies, 1987, this volume), 424 μg/g, was calculated from the results for bedroom and playroom samples excluding those for the one child with an exceptionally high blood lead level of 2.14 umol/l (44.6 μg/dl).

Table 2. *Summary of internal and external air lead concentrations*

	Air lead concentration : $\mu g/m^3$			
	Bedroom	Playroom	Internal	External
n	599	607	1206	605
Arithmetic mean	0.32	0.34	0.33	0.55
Range	<0.05-1.52	<0.05-1.96	<0.05-1.96	<0.05-2.98
Geometric mean	0.26	0.27	0.26	0.43
5th Percentile	0.09	0.08	0.08	0.12
95th Percentile	0.81	0.88	0.87	1.53

Table 3. *Hand lead levels (µg total both hands) over 7 consecutive days*

Day	Child			
	A	B	C	D
1	5.1	4.9	10.3	*
2	2.2	4.9	*	4.2
3	2.6	3.7	10.8	13.6
4	4.3	5.0	13.0	11.5
5	3.0	3.1	11.1	8.1
6	3.6	2.8	13.0	406.1
7	3.8	5.3	15.2	4.7
8	*	6.9	10.6	5.9

*Missing data

the householder's vacuum cleaner, 336 µg/g (significant difference, ($p<0.01$) and higher (although not significantly) than the mean from the bedroom and playroom samples, 430 µg/g.*[1]. This is not a surprising result when one considers that external dusts, in general, have higher lead concentrations than house dusts and that the doormat is the most likely location within a house for the removal of such external dusts. In a national reconnaissance study in the UK, the mean lead concentration in road dusts was 786 compared with 561 µg/g in house dust (excluding data from "geochemical hotspots", Thornton *et al.*, 1985). From Table 1, it can be seen that the mean road dust concentration in Birmingham, 527 µg/g, is lower than the national average but again higher than the concentrations found in the bedroom, playroom and vacuum cleaner samples. Birmingham was one of the cities included in the national survey and the geometric mean house dust lead concentration found in 1981/2 was 419 µg/g (n = 159). This figure is similar to the mean concentration found in the present study using the adapted vacuum sampler, 430 µg/g, although slightly higher than that noted for the householder's vacuum cleaner samples, 336 µg/g.

Other data pertaining to internal dusts in Birmingham do appear in the literature but are difficult to compare directly with the results from our studies. For example, an extremely high median value of 3,500 μg/g has been reported for 117 samples collected in 28 Birmingham houses during 1979/1980 (Department of the Environment, 1982). However, the houses included in this study were selected because the children living in them had been found to have high blood lead concentrations and/or high erythrocyte protoporphyrin concentrations and cannot be considered as representative. In another Birmingham study, a high median lead concentration of 1,000 μg/g was reported for 806 internal dust samples (Archer and Barratt, 1976a). These, however, were not solely house dust samples but were collected from offices, shops and schools as well as houses in the area.

A somewhat unexpected result from the present study was that the mean lead concentration in pavement dust, 360 μg/g, was significantly lower than that found in road dust ($p<0.001$), even though the sampling sites were adjacent in all cases. These data suggest that there may have been a greater input of non-lead bearing dust and soil particles onto the pavement sites, perhaps by pedestrian movement, and/or that the processes of deposition and removal were operating at different rates in the two situations.

The concentrations of lead in residential road dusts in Birmingham have been measured previously. Archer and Barratt (1976a) reported a median value of 950 μg/g for 85 such road dust samples collected between 1972 and 1975. The mean value found in this study, 527 μg/g, is only around one half (56%) of this value.

In addition to the bedroom and playroom dust lead concentration figures in Table 1, dust lead loadings have also been included. An indication from pilot studies that the exposure of a child to lead in house dust may be expressed more realistically by lead loading than simply lead concentration in dust is supported by data from this study. For example, in one of the houses sampled, the lead concentration in the bedroom was approximately 12 times that in the playroom, while the lead loading was only around twice as high. Looking at concentration alone, therefore, could be deceptive as the total mass of lead available for ingestion by a young child from house dust is not reflected in this figure. Although the lead concentration is high in the bedroom dust in this house, there is very little actual dust present and, therefore, relatively little lead to which the child is exposed. The geometric mean of the average bedroom and playroom lead loading in 94 homes was 62 $\mu g/m^2$. The few data in the literature on house dust lead loadings range widely. Similar results to those found in this study have been reported for the Netherlands, where geometric mean lead loadings ranged from 30 to 81 $\mu g/m^2$ in 166 homes in four different areas (Brunekreef *et al.*, 1983). A higher median value of 418 $\mu g/m^2$ was found in 65 homes in Christchurch, New Zealand (Fergusson and Schroeder, 1985) and a mean of 680 $\mu g/m^2$ has been reported for 12 homes in Champaign- Urbana, USA (Solomon and Hartford, 1976). A study of 15 London homes revealed an extremely high median lead loading of 3,500 $\mu g/m^2$, although these properties were situated near to known lead sources (Turner *et al.*, 1980).

The mean soil lead concentration found in the present study, 313 μg/g, was similar to that noted for Birmingham in the reconnaissance study, 278 μg/g (n =

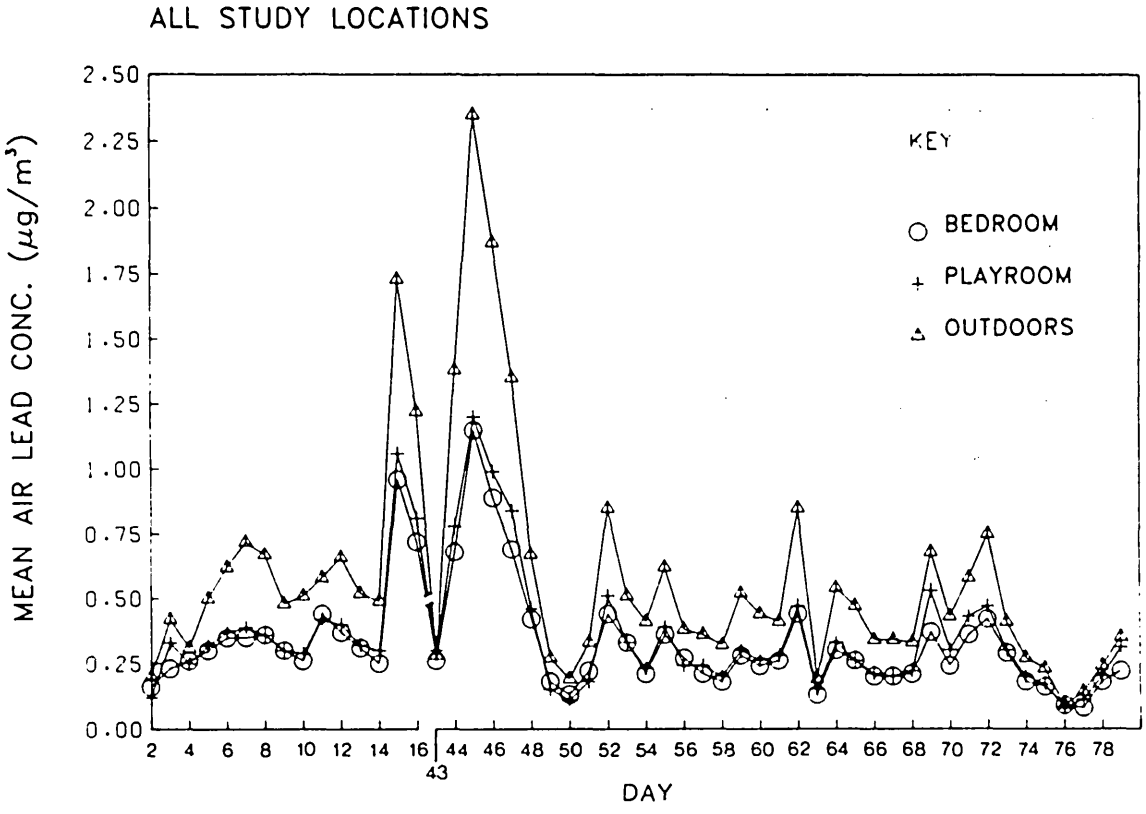


Figure 1. *Lead levels in and around the homes of inner-city children*

159). Both of these values, however, are slightly higher than the national average, 266 μg/g (n = 4,126). Previous studies have shown soil lead levels to be correlated with house dust lead (Davies *et al.*, 1985, Thornton *et al.*, 1985) and this was also the case in this study. The correlation coefficient between dust lead loading and soil lead was 0.38.

Air

The mean air lead concentration in the bedroom was 0.26 $\mu g/m^3$ (n = 599), very similar to that in the playroom, 0.27 $\mu g/m^3$ (n = 607) (Table 2). The mean external air lead concentration, 0.43 $\mu g/m^3$ (n = 605), was significantly higher than those found indoors ($p<0.001$) although not as high as other figures published previously for outdoor air lead in Birmingham. In 1983/4, the city wide annual average from 9 sites was reported to be 0.79 $\mu g/m^3$ (City of Birmingham Environmental Health Department, 1985). Archer and Barratt (1976b) reported a median value of 0.75 $\mu g/m^3$ from 15 sites across the city in 1975, with average weekly means ranging from 0.4 to 1.4 $\mu g/m^3$. Still higher concentrations were recorded at a site alongside the Gravelly Hill motorway interchange during the same year and resulted in an annual mean (average of monthly means) of 1.79 $\mu g/m^3$ (Department of the Environment, 1978). These values can be compared to a 5 year average (1976 to 1981) of 0.65 $\mu g/m^3$ from 5 urban sites in England and Scotland (McInnes, 1982).

Examination of the daily air lead concentrations in individual houses revealed that: 1) the internal concentration closely followed the external concentration, but was usually lower. This suggests that the external air lead level has a large influence on that indoors, and that the physical structure of the house (walls, doors, windows) prevents some of the lead bearing suspended particulate matter reaching the indoor air; 2) the levels for houses sampled concurrently were very similar to one another, each peaking, for example, on the same days. This suggests that the same air mass was being sampled, even though the houses were widely scattered and that meteorological factors, such as wind speed and direction or rainfall, were influencing the air lead concentration in the study area. The daily mean air lead concentrations are shown in Figure 1.

The mean internal:external air lead ratio was 0.61 (0.39-1.33). There is not a vast literature on internal:external air lead ratios but from a review of the available data, Yocum (1982) suggested that ratios of 0.6 to 0.8 were typical for houses without air conditioning (none of the houses in this study was air conditioned). The few such studies carried out in the UK have indicated, from limited data bases, ratios of between 0.6 and 0.7 (Lawther *et al.*, 1973; Chamberlain *et al.*, 1987; Department of the Environment, 1978).

The mean internal air lead concentration for each house was strongly correlated with the mean external concentration and a linear regression gave:

$$\text{Internal PbA} = 0.096 + 0.42 * \text{External PbA}$$

with $RP^2 = 76\%$ ($p<0.001$). This indicates that the weekly mean external air lead concentration is a good predictor of the weekly mean indoor concentration. This finding could have important implications in the design of future exposure assessment studies as it is cheaper and less complicated to implement an "outdoor only" air monitoring programme; data could then be used to estimate indoor concentrations.

Handwipes

The geometric mean (5th-95th percentile) hand lead level (total, both hands) was 5.7 µg (1.9 to 15.1 µg, n = 704). This is lower than other values cited in the literature and is probably a reflection of the young age of the study population. Vostal et al. (1974) reported a median value of 20 µg Pb per hand for urban children aged between 2 and 6 years, while Roels *et al.* (1980) found a range of 13 to 20 µg Pb per hand for an urban group aged 10 to 14 years. Brunekreef *et al.* (1983) have reported a mean of 12 µg Pb per hand for inner-city children aged between 4 and 6 and in a recent study of 5 and 6 year olds in London, Duggan *et al.* (1985) found means ranging from 12 to 43 µg Pb per child in 11 infant schools. In general, there was little day to day variation in hand lead levels and data from 3 typical children (A, B and C) are presented in Table 3. There were, however, exceptions, as was the case with child D. It is not clear why such an extraordinarily high level, 406 µg, was found on one of the days, although it is not surprising to find outliers in such a large data set (n = 704).

Conclusions

Environmental lead levels in and around the homes of 2 year old children living in the inner-city area of Birmingham were relatively low. Soil lead levels were correlated with house dust lead, confirming earlier findings. The highest lead concentration in dust from within the home was noted in samples from under the doormat. The external air lead concentration was strongly correlated with, and a good predictor of, the internal air lead concentration. Hand lead levels for individual children showed little variation over time.

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Lead in the Home Environment

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17 An Assessment of the Exposure of Young Children to Lead in the Home Environment

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Summary

An assessment of the exposure of two year old urban children to lead is presented. Using data generated from a large, integrated study recently completed in Birmingham, UK, the average total uptake from the lungs and gut was estimated to be 36 µg/day. Of the total daily uptake 3% was attributed to inhalation, 97% to ingestion.

Introduction

Man is exposed to lead in his environment from a number of sources along a multitude of pathways. Although both natural and anthropogenic sources exist, the former have little significance in today's industrialised society in which lead is the most widely used non-ferrous metal. In order to avoid confusion it is worth mentioning the differences between sources and pathways in this context. Sources can be thought of as materials from which lead is first released to the environment such as petrol, solder, paint, lead pipes or lead-bearing rock, whereas the media through which this liberated lead passes, such as water, air, food and house dust are all stages in a variety of pathways from source to receptor, which in this case is man.

Generally, the adverse health effects associated with the intake of large amounts of lead, such as may occur in certain occupational settings, are well defined. What is unclear at present is whether the intake of relatively small amounts of lead has a detrimental effect on human health, and in particular, on the health of young children. This subject area has been investigated in great detail by a number of workers in recent years and has also been discussed elsewhere at this meeting (Harvey 1986a, 1986b, Pocock 1986). An important point to note here is that even if the majority of experimental evidence suggests no adverse health effects at current levels of exposure, there is still only a narrow margin of safety between the resultant blood lead levels and those levels likely to cause adverse health effects in young children. For these reasons it is important to ascertain the significance of the various

environmental pathways to the total lead intake, and subsequent uptake, for the population most at risk - young children.

Several studies have been carried out with this basic objective in mind but in the main, these have not been truly comprehensive often looking only at single pathways in isolation. Lead enters the body via two main routes: inhalation into the respiratory system and ingestion into the alimentary system. A study, recently commissioned by the UK Department of the Environment in the inner-city area of Birmingham, was designed to provide quantitative information on lead intakes of young children via all the major pathways simultaneously. House dust, pavement and road dust, soil, air, water, food, blood and handwipe samples were all obtained. Due to the multidisciplinary nature of such a study it was necessary for a number of universities and government bodies to collaborate and these included the Universities of Birmingham and Southampton, Imperial College, the Ministry of Agriculture, Fisheries and Food and the Department of the Environment. The operational details and full interpretation of all results from this investigation will be published elsewhere. A summary exposure assessment has been carried out using the data generated from this study and is presented in this paper.

Population Studied and Samples Obtained

A random selection of families with children born in the area and at the time of the study aged between 24 and 27 months, was approached by letter and subsequently visited. Of those who were still resident and who could be contacted and were not in outside care for more than six hours per week, 77% agreed to participate. Of the volunteered children 183 eventually provided venous blood samples, of which 147 could be analysed; 56 children provided a second blood sample at the end of the study. Additionally, one child with a blood lead value of 2.14 µmol/l (44.6 µg/dl) was found but excluded from the analysis and investigated by the local health authority. 31 children with higher blood lead levels [over 0.73 µmol/l (15.2 µg/dl)] and 34 with lower values [under 0.44 µmol/l (9.2 µg/dl)] were selected for further study, together with 33 with intermediate values. The environmental and handwipe samples obtained have been described elsewhere at this meeting (Culbard *et al.* 1986). A duplicate of the child's diet, as eaten and covering seven days, was also collected, together with a composite seven day sample of drinking water. Also video films were taken in each home in order to quantify the child's mouthing activity (Harvey *et al.* 1986) and a comprehensive questionnaire was administered.

Results and Discussion

As an initial step in interpreting the data, it was thought that a comparison of the summary results from this study with those compiled recently for 2 year old urban children in the UK as a whole would be useful. The Royal Commission on Environmental Pollution, in their Ninth report on Lead in the environment, provided a model balance scheme for this section of the population, along with others, in order to give a broad outline of the actual and relative magnitudes of intakes and uptakes

of lead via different pathways (Royal Commission on Environmental Pollution 1983). The importance of recognising the limitations of such an exercise, which involved many uncertainties and assumptions and makes no attempt to quantify lead excreted by the body, is stressed by the Commission.

Beginning with the inhalation route, the calculation of lead uptake from air is based on the air lead concentration, both indoors and outdoors, the amount of time spent at each location, and two main assumptions: that the volume of air respired per day is 6 m^3 and that 70% of the inhaled lead is absorbed. It is also assumed that the transfer of lead from trachea to oesophagus by muco-ciliary action is negligible. Unfortunately, there is considerable uncertainty regarding both of these two main assumptions due to the fact that it would be inappropriate to carry out the relevant experiments on this group of the population. Experiments, mostly carried out on adult males, indicate that the volume of air breathed each day varies widely and is dependent upon the age, condition and activity of the subject. Not all of the lead inhaled is retained by the lungs; a proportion is exhaled. The percentage of inhaled lead which reaches the bloodstream from the lungs is heavily dependent upon the physical and chemical form of the lead (Nozaki 1966, Chamberlain *et al.* 1978, Morrow *et al.* 1980, Chamberlain and Heard 1981, Gross 1981). Again due to a lack of data on children, the combined retention and absorption factor of 0.7 used must be thought of as tentative.

With reference to Table 1 it can be seen that the mean lead concentrations, both indoors and outdoors, found in the Birmingham study were considerably lower than those previously estimated for typical inner city areas in the UK. The mean outdoor concentration was 0.43 $\mu g/m^3$ and the mean indoor concentration 0.26 $\mu g/m^3$ compared with the Commission's estimate of 1.0 and 0.75 $\mu g/m^3$ respectively. As this study was carried out in the winter it is not surprising to find that the average time spent indoors was high, 23.25 h/d. Assuming the volume of air respired per day was 6 m^3, the calculated lead intake from air was 1.59 µg/d. Applying a combined retention and absorption factor of 0.7, lead uptake from the lungs was estimated to be only 1.1 µg/d, around one third of the Commission's estimate of 3.4 µg/d.

For the ingestion route, the main assumptions used are that the mass of dust ingested per day is 100 mg and that 53% of the total lead ingested reaches the

Table 1. *Intake and uptake of lead via inhalation*

	Royal Commission	B'ham Study
Air lead conc.($\mu g/m^3$): Outdoor	1.00	0.4
Indoor	0.75	0.26
Time spent indoors (hrs/d)	20	23.25
Volume of air respired (m^3/d)	6	6
Lead intake from air (µg/d)	4.8	1.59
Percent absorption in lungs	70	70
Lead uptake from lungs (µg/d)	3.4	1.1

Table 2. *Intake and uptake of lead via ingestion*

	Royal Commission	B'ham Study
Dietary lead intake (μg/d) (food and drinks)	52	23
Dust lead conc.(μg/g)	1,400	424
Lead intake from dust (μg/d)	140	42.4
Total lead intake: ingestion (μg/d)	192	65.4
Percent absorption in gut	53	53
Total lead uptake from gut (μg/d)	102	35

Table 3. *Lead uptake from inhalation and ingestion*

	Royal Commission	B'ham Study
Total lead uptake from lungs and gut (μg/d):	105	36
Percentage of daily lead uptake from inhalation:	3	3
Percentage of daily lead uptake from ingestion:	97	97

bloodstream from the gut, the remainder passing directly to the faeces. The percentage absorption of lead is known to vary widely depending on the contents of the gut and the physical and chemical form of the ingested lead although, again, there are few data available for young children (Alexander *et al.* 1973, Garber and Wei 1974, Barltrop 1975, Blake 1976, Chamberlain *et al.* 1978, Ziegler *et al.* 1978, Rabinowitz *et al.* 1980, Gross 1981, Heard and Chamberlain 1982).

The intake and uptake of lead via ingestion is shown in Table 2. There were no direct data on the average dietary lead intake of young children available to the Commission and it can be seen that the results from the duplicate diet study in Birmingham revealed a mean value of 23 μg/d, which is slightly less than half their estimate of 52 μg/d. The mean house dust lead concentration was 424 μg/g, resulting in an estimated intake of 42.4 μg of lead per day, via this pathway. Recent studies into the particle size distribution of dust adhering to children's hands indicate though that this derived intake value may be an underestimate (Duggan *et al.* 1985). Duggan and co-workers showed that the majority of dust particles found on hands were below 10 μm and that the maximum particle diameter was 100 μm. This is an important finding when one considers that, for a sieved dust, the lead concentration usually increases as the size fraction decreases. It may well be more appropriate to analyse the finer dust fractions rather than the less than 1,000 μm fraction which is the routine procedure of most workers currently, including ourselves. Further work is

required to clarify this situation. The total lead intake from diet and dust was estimated to be around 65 µg/d (Table 2). Applying an absorption factor of 0.53 the total lead uptake from the gut was calculated to be 35 µg/d, approximately one third of the total uptake estimated for 2 year old children in inner city areas of the UK as a whole.

To summarise, the total uptake from the lungs and gut was estimated to be 36 µg/d, compared with 105 µg/d estimated by the Royal Commission. Only 3% of the total uptake, in both cases, is attributed to inhalation, the other 97% is due to ingestion (Table 3).

The calculations carried out so far have incorporated mean values from the various data sets to provide information on the likely exposure of a typical 2 year old child living in the inner city area of Birmingham. As actual exposures will differ, the relevant data from two households have been extracted and used in the following calculations to provide an indication of the ranges involved (Table 4). House 6 was bing redecorated at the time of the study and as well as having a relatively high mean indoor air lead concentration of 0.48 µg/m^3 it also had the highest house dust lead concentration, - 3,110 µg/g. House 2, on the other hand, had the lowest house dust lead concentration, 111 µg/g and a mean indoor air lead concentration of 0.2 µg/m^3. Using the same assumptions as before, the intakes and uptakes were estimated. Lead uptake from the lungs was calculated to be 2.0 µg/d using data from House 6 compared with 0.65 µg/d using data from House 2. The total intakes by ingestion, including diet and dust, were 322 and 18.1 µg/d for Houses 6 and 2 respectively. This results in very different estimates of uptake of 176 and 10 µg/d in the two situations. These figures are merely presented as an example of the possible uptakes in these types of environments and are not intended to be estimates of the uptakes of the two individuals actually living in these houses. There is also greater uncertainty associated with these figures than with those derived from earlier calculations very high or very low levels of lead in the gut may well modify the absorption factor.

Returning to the question of dust lead intake, the amount of lead present on the

Table 4. *Lead intake and uptake: selected examples*

	House 6	House 2
Inhalation:		
Lead intake from air (µg/d)	2.9	0.92
Lead uptake from lungs (µg/d)	2.0	0.65
Ingestion:		
Dietary lead intake (µg/d) (food and drinks)	21	7
Dust lead conc.(µg/g)	3,110	111
Lead intake from dust (µg/d)	311	11.1
Total lead intake: ingestion (µg/d)	332	18.1
Lead uptake from gut (µg/d)	176	10

child's hands was assessed daily and revealed a mean value of 5.7 µg (Culbard *et al.* 1986). The actual amount of lead present on the hands will vary throughout the day depending on the particular activity of the child and the frequency of washing. This figure is thus an estimate of the likely level at any given time on any given day. Looking again at the previously estimated value for lead intake from dust of around 42 µg per day, it would require the transfer of all hand lead from hand to mouth a total of seven times per day to result in this intake. This is only a crude estimate from the summary data, and a more sophisticated approach is planned which will incorporate data on the hand to mouth activity of the study children. It is important to gain information on the hand to mouth activity of individual children to supplement the measurement of hand lead. A child with a relatively low hand lead level exhibiting frequent hand to mouth activity may well ingest more lead via this pathway than a child with a high hand lead level that rarely places his hand in his mouth. As an example, one child with a mean hand lead level of 13 µg did not place his hands in his mouth at all during the period being rated whereas another with a low lead level of 2 µg did so four times.

During the study, the floor area over which the house dust samples were taken was measured in order to facilitate the calculation of the lead loading. The results from pilot studies had suggested that the exposure of the child to lead in house dust is expressed more realistically by lead loadings than simply lead concentrations (Culbard *et al.* 1986). This is supported by the results from this study. If one looks at the association between house dust lead and hand lead in terms of correlation coefficients there is a positive correlation of 0.44 between lead loading and hand lead and a positive correlation of only 0.24 between lead concentration and hand lead. Although the correlation coefficients are not high, the important point to note is the relative magnitude of the two (there is a much greater association between lead loading and hand lead than lead concentration and hand lead).

Some children exhibit pica, which is considered an abnormal tendency to chew and eat non-food items. This is likely to increase their lead intake by varying amounts, depending on the degree of pica. An indication of those children who exhibit this phenomenon, and to what extent, can be gained from the questionnaire responses and the videotapes. The one child mentioned earlier with a blood lead concentration of 2.14 µmol/l was found to be a soil eater. It is interesting to note also that the soil lead concentration in this child's garden was higher than any other at 1,600 µg/g. However, it would be inappropriate to assume a causal relationship from such limited data.

The blood lead concentrations determined from this study were similar to those previously found amongst children of this age in central Birmingham (Harvey *et al.* 1984). The geometric mean value was 0.56 µmol/l (11.7 µg/dl) and agreement between initial samples in November and repeats in March was very good (correlation coefficient = 0.82). This is most encouraging as it suggests that lead exposures remained reasonably constant during the study period and therefore that the data obtained on the lead exposure variables are appropriate for comparison with the blood lead data.

The next stage in the analysis of the data will involve the development of a lead

Table 5. *Intake and uptake of lead by young children in Birmingham*

Intake Route	Pathway	Lead Uptake (µg/d)	Total Uptake (%)
Inhalation	Air	1.1	3
Ingestion	Diet	12.2	34
	Dust	22.5	63
	Total	35.8	100

exposure model to relate blood lead to the exposure variables measured. This is not an easy task and there are several potential problem areas which will have to be addressed regarding the statistical approaches to be used. For example, standard regression techniques assume that the exposure variable is measured without error, which is not the case. With multiple regression analyses a further problem is that many of the co-variates are correlated with each other. These and other points will need careful consideration when developing the model.

Summary and Conclusions

In conclusion, the environmental lead levels in and around the homes of the study population were encouragingly low, as were the dietary lead levels. A summary assessment of the exposure of these children to lead indicates that intake via the inhalation route accounts for only 3% of the total daily lead uptake (Table 5). For the ingestion route, the contribution to lead uptake made by the intake of dust is around twice that attributable to the diet, the estimates being 2.5 and 12.2 µg/d respectively.

It is recognised that there are considerable uncertainties in the estimates of intake rates, retention and absorption factors used to derive these figures and they should only be considered as general indicators of the significance of the various pathways. As lead in house dust may have an important role in the exposure of children to lead, it is of interest to determine where the lead present in house dust comes from. An ongoing research programme at Imperial College addressing this issue, and described elsewhere (Watt and Johnson 1986), is making good progress.

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