Medical Research Council

Institute for Environment and Health

IEH report on

RECENT UK BLOOD LEAD SURVEYS

1998 **REPORT R9**

The Institute for Environment and Health was established by the Medical Research Council at the University of Leicester in 1993. The Institute is partly funded by the Department of the Environment, Transport and the Regions, the Department of Health and other Government Departments and Agencies by way of specific research and consultancy contracts.

This report, prepared by the Institute for Environment and Health under a Department of Health and a Department of the Environment contract, is based on a workshop held in Leicester in March 1997. The Institute gratefully acknowledges the contribution of all those who attended the workshop and provided material for inclusion in the Report, but assumes no endorsement from these scientists for the conclusions and recommendations contained here.

The views expressed here do not necessarily represent those of any Government Department or Agency.

Workshop chaired by Professor Dame Barbara Clayton

Edited by D Gompertz
Published by the Institute for Environment and Health, 1998

Printed by Page Bros, Norwich

ISBN 1 899110 13 5

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Contents

Executive summary

In 1994 the Department of the Environment (DoE) and Department of Health (DH) commissioned the Institute for Environment and Health (IEH) to manage the part of the 1995 Health Survey for England designed to investigate blood lead levels across the country. The study was also to review the association between blood lead level and blood pressure. The study was performed by the Department of Epidemiology and Public Health, University College London in conjunction with Social and Community Planning Research. The measurements of lead in blood were made by the Department of Clinical Biochemistry, Southampton General Hospital.

At the end of the study, IEH hosted an expert workshop to review the survey results and to compare them with a study of blood lead levels in children performed in the Avon area (Avon Longitudinal Study of Pregnancy and Childhood). The expert group was also asked to review these results in the context of recent international studies and to relate them to trends in environmental lead levels.

This IEH Report contains the summary reports, presented at the workshop, of the Health Survey for England blood lead study and the relationships between blood lead and blood pressure found during this study. Other papers presented at the workshop which describe the findings of the Avon study and review current knowledge concerning the effects of the lead on IQ and blood pressure are included, as are reviews of trends in blood and environmental lead levels in the UK and elsewhere.

The results of the Health Survey for England and other UK studies show that blood lead levels have fallen considerably over the eight years following earlier DoE surveys and the majority of the measurements are well below the recently accepted guidance value of 10 µg/dl. This fall has been ascribed to reduction of lead in petrol and paint and removal of leaded solder from food cans and lead plumbing from the home. Although the average blood lead levels reported are low there are occasional individuals with significantly raised blood lead. Lead in water derived from lead service pipes and household plumbing in areas with water of high plumbosolvency may be an important source of these higher levels. Lead in old paint may be a source associated with redecorating in older properties.

There is also evidence that even at the relatively low blood lead levels found in the Health Survey for England there is a relationship between blood lead and blood pressure.

A number of recommendations are made for further research, including a comparison of blood and tooth lead and development of breast or bottle fed children living in one of the remaining areas with a high content of lead in drinking water.

1 Introduction

During the years 1984 to 1987 the Department of the Environment (DoE) developed a UK Blood Lead Monitoring Programme to cover the period when the maximum permissible content of lead in petrol was reduced from 0.4 to 0.15 g/l (DoE, 1990). In fact, from the early 1970s there appears to have been a long-term downward trend in blood lead levels and there was a relatively small additional decline in blood lead levels associated with the reduction in petrol lead in 1986. Control of lead from other sources was also thought to contribute to the fall in blood lead levels seen during this Monitoring Programme. Projections for a further decline in blood lead levels were such that it was suggested that such a survey need not be repeated for five to ten years (DoE, 1990).

Meanwhile surveys from other countries have shown major decreases in population blood lead levels (Ducoffre *et al.,*1990; Pirkle *et al.,*1994; Strömberg *et al.,*1995). These falls have been associated primarily with the reduction of lead in petrol and in paints. In 1995 the Department of Health (DH) and the DoE commissioned a new survey of population blood lead levels. This new survey was part of the 1995 Health Survey for England, an annual nationwide DH survey that started in 1991 and was designed to address various aspects of the nation's health. From 1994, the Health Survey for England has been performed jointly by the Department of Epidemiology and Public Health at University College London Medical School and by Social and Community Planning Research (SCPR). The blood lead part of the 1995 Survey was managed by the Institute for Environment and Health (IEH) and the analyses of the blood samples were performed at the University of Southampton. The DH also asked the organisers to review whether any statistical relationship could be detected between blood lead levels and blood pressure in this population.

This Blood Lead Study within the 1995 Health Survey for England mainly involved adult subjects although 5% of the samples were from children (over 11 years). A parallel study of children born in the Avon region in a six month period in 1992, part of the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC), had taken the opportunity to measure blood lead levels in 584 younger children (aged 31 months). This study also aims to assess the impact of lead levels on subsequent IQ.

The IEH was commissioned by the DH and DoE to arrange an expert workshop in March 1997 to review the results of the Health Survey for England study and the implications for government. It was decided that it would be important to consider the blood lead results from ALSPAC at the same time and also the findings from the Glasgow 1993 Lead Study, which looked at lead in tap water and maternal blood lead levels (Watt *et al.,* 1996). Specialist summary reviews of international trends in blood lead levels, trends in environmental levels, and the effects of lead on children's IQ and on hypertension were also prepared for this workshop.

The workshop, chaired by Dame Barbara Clayton, was hosted by IEH on March 4th 1997. The papers summarising the results of the Health Survey for England and ALSPAC Blood Lead Surveys and the specialist reviews are presented in this report.

REFERENCES

DoE (1990) *UK Blood Lead Monitoring Programme, 1984–1987. Results for 1987* (Pollution Report No 28), London, UK, HMSO

Ducoffre G, Claeys F & Bruaux P (1990) Lowering time trend of blood levels in Belgium since 1978. *Environ Res, 51,* 25–34

Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM & Matte TD (1994) The decline in blood lead levels in the United States. *J Am Med Assoc, 272,* 284–291

Strömberg U, Schütz A & Skerfving S (1995) Substantial decrease of blood lead in Swedish children, 1978–94, associated with petrol lead. *Occup Environ Med, 52,* 764–769

Watt GCM, Britton A, Gilmour WH, Moore MR, Murray GD, Robertson SJ & Wormersley J (1996) Is lead in tap water still a public health problem? An observational study in Glasgow. *Brit Med J, 313,* 979–981

2 Blood lead surveys

2.1 SURVEY OF BLOOD LEAD LEVELS IN THE POPULATION IN ENGLAND, 1995

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2.1.1 INTRODUCTION: BACKGROUND AND PURPOSE

Lead is one of the metals present in the earth's surface to which man is naturally exposed. It has proven useful to society since ancient times; nevertheless, environmental contamination has become a cause for concern, and the clinical consequences of lead poisoning have long been known (Graebner, 1988). The possible adverse effects of lower levels of exposure remain more controversial (Shingal & Thomas, 1980).

Before major changes were introduced to limit lead exposure, the main sources of lead in the environment in the industrialised countries were considered to be leaded petrol and house paints (Rosner & Markowitz, 1985). Since these changes, the relative impact of other identified sources, such as food (crops grown in soils of high lead content, food in soldered tins; Moore *et al.,* 1979), water (lead piping

being the principal source of contamination in tap water; Watt *et al.,* 1996) and other beverages which contain lead, has tended to increase.

The source dictates whether lead is absorbed principally by the respiratory tract or the gastrointestinal tract into the blood stream. In the blood stream, lead is carried on the surface of red cells and around 95% of the total body lead of adults is stored in bone (Wittmers *et al.,* 1988). Lead competes with calcium for transport and for binding sites, is mobilised from the bone in a similar way to calcium and is released, along with calcium, when bone is reabsorbed.

The best known health hazards associated with environmental lead exposure are effects on the central nervous system and haematological effects (Royal Commission on Environmental Pollution, 1983). Encephalopathy is the most severe result of lead poisoning, leading to coma and death in acute cases of exposure, which are usually occupational. Lead interferes with globin synthesis, and causes anaemia by shortening the life span of the erythrocyte.

Blood lead is the best available indicator of recent exposure to lead and it is used to evaluate the impact of different environmental sources of current lead exposure. However, effects of low-level lead exposure and cumulative effects of exposure are difficult to evaluate mainly because blood lead does not reflect long-term exposure (DHSS, 1980).

Hitherto it has been accepted that the brain is the critical target organ for the adverse effects of low-level exposure, particularly in young children (DHSS, 1980). Fetal and infant exposures have been reported to affect cognitive development (Needleman & Gatsonis, 1990). The mechanisms for the effects of lead on the central nervous system are unclear, but appear to involve lead interactions with calcium-mediated intracellular messenger systems and neurotransmission.

Blood pressure may be affected by exposure to low levels of lead, possibly through its influence on calcium-mediated control of vascular smooth muscle contraction and on the renin–angiotensin system, but the effects remain difficult to assess and are controversial (Hertz-Picciotto & Croft, 1993; see also Sections 2.6 and 2.7).

Associations between blood lead levels and both obesity and smoking have been reported (Szymanski & Hertz-Picciotto, 1995). Both contribute to bone mineral loss and affect the retention of lead stored in bone. Smoking is also well known to be associated with higher blood lead levels (Hense *et al.,* 1992).

Increasing evidence that skeletal lead can disrupt bone mineral metabolism raises the possibility that women with larger stores of lead are at increased risk of bone loss and of developing osteoporosis (Szymanski & Hertz-Picciotto, 1995).

Blood lead levels in general population surveys around the world showed a major decrease after measures were introduced to reduce lead exposure (Hinton *et al.,* 1986; Christensen & Holst, 1988; Elwood *et al.,* 1990; Strömberg *et al.,* 1995). These measures included reducing or banning lead in petrol, house paint, pipes and soldered cans containing food. In the USA the National Health and Nutrition Examination Survey for 1988–91 (NHANES III) showed that public health efforts to reduce lead in petrol and soldered cans containing food were associated with a 78% decrease in blood lead levels in the US population compared with the 1976–80 period (Pirkle *et al.,* 1994).

In 1984 the UK DoE commissioned a lead monitoring programme. A survey was conducted annually over the period 1984–87 when blood lead levels were measured in particularly 'exposed' areas (urban areas with heavy traffic) and professions (traffic police and taxi drivers), and 'control' (rural) areas (DoE, 1986, 1987, 1988, 1990). Average blood lead levels fell over the period, and more so in the exposed than in the unexposed (control) group.

The purpose of the present survey was to obtain information on the distribution of blood lead levels in the population in England and its relation to possible environmental determinants of lead levels. This was achieved using a subsample of the 1995 Health Survey for England (Prescott-Clarke & Primatesta, 1997).

MATERIALS AND METHODS

The Health Survey for England is an annual nationwide survey, commissioned by the DH, which began in 1991 and includes data about various aspects of the nation's health. Data are collected in two visits: an interviewer's visit (which includes height and weight measurements), followed by a visit from a nurse, who measures blood pressure, records current use of prescribed medicines and takes a blood sample.

A multi-stage stratified probability sampling design has been adopted in all surveys in the series to date. The survey population is drawn from those living in private households and hence institutionalised people are not included in the sample. The sampling frame is the small user Postcode Address File (PAF).

Postcode sectors are the primary sampling units (PSU). In 1995, 18 delivery points (addresses) were systematically selected from each selected PSU, yielding a total selected sample of 12 960 addresses (Prescott-Clarke & Primatesta, 1997).

Five stratification levels were used:

- the 'old' 14 Regional Health Authorities;
- % of the population aged 16+ who have a limiting long-term illness;
- % of households with household head in non-manual occupation;
- % of households with no car; and
- % of population who are non-white.

The use of region as the first stratifier ensured the correct regional balance of the sample. The data to classify postal sectors for the second to fifth stratifiers came from the 1991 Census of Population.

The sample was designed so that fieldwork conducted in each quarter of the year was carried out with a fully representative subset of the total sample.

Every adult (aged 16 years or over) and child (aged 2 to 15) from each selected household was eligible for the survey. In order to limit the burden on households where there were three or more children aged 2–15, two of these children were randomly selected for the survey. No interviews were attempted with the other children. As only about 15% of households containing children aged 2–15 have three or more children in this age group, only a small proportion (12%) of all children were thus omitted from the sample. The application of weights was nevertheless required to compensate for the omissions. Blood samples were not requested from those under 11 years of age.

At the request of the DH and the DoE, an additional sample of blood to allow for the analysis of blood lead was obtained from people who participated in the survey in two quarters of the year (April–June and September–December). This ensured full representativeness of the sample.

LABORATORY METHODS

Blood lead analyses were performed using microsampling flame atomic absorption spectrometry (Delves, 1970). The protocol used for controlling the accuracy of these analyses was based on that used successfully in the DoE UK Blood Lead Monitoring Programme (1984–87) in order to allow a valid comparison of current blood lead levels with the data from this earlier exercise (DoE, 1986). This protocol consisted of:

- the use of low lead blood collection tubes for all survey specimens, with testing of ten blank tubes per batch;
- calibration of every analytical run using matrix matched standards prepared from portions of the same pool of a low lead bovine blood specimen, using invariant acceptance limits for each of the five calibrating standards;
- control of every analytical run by analysing portions of the same internal quality control (IQC) specimens immediately following the calibrating standard and thereafter following every set of five survey specimens — invariant acceptance limits are assigned to each set of three IQC specimens;
- within-laboratory re-analysis by a different analytical method of a subset of at least 20% of all survey specimens which included all specimens with lead content greater than 10 µg/dl;
- re-analysis by an external laboratory of at least 10% of all survey specimens; and
- participation in external quality assessment programmes for blood lead analysis.

STATISTICAL METHODS

Firstly, descriptive statistical analyses were performed. These included age–sex distribution, and the analysis of lead distribution by demographic and lifestyle factors. Frequency distributions of blood lead level were positively skewed; lead measurements were therefore log transformed to normalise the distribution and stabilise the variance. Geometric mean (the antilogarithm of the mean of the log

transformed values) is therefore used here as well as the arithmetic mean as a summary measure.

Secondly, to assess the independent effect of several influencing factors on blood lead level, multiple regression analysis was used. This analysis was restricted to adults. Sex specific multiple linear regression models were developed: log blood lead was entered as the dependent variable, and age, social class, smoking status, alcohol consumption, region of residence (grouped into north, midlands and south) were introduced as explanatory variables. Differences from the reference category and 95% confidence intervals are reported.

2.1.2 RESULTS

RESPONSE RATES

In the 1995 Health Survey for England, interviews were obtained from 78% of sampled households (16 055 adults and 3733 children aged 2–15); in these households 80% of adults and 84% of children saw a nurse.

Of the 8659 people visited by a nurse over the periods April–June and September–December, 8483 (98%) were eligible for a blood sample, and valid lead measurements were obtained from 81% of those eligible (83% of males, 79% of females), as shown in Table 2.1.1. A total of 6857 samples were analysed, 6517 adults (3119 men, 3398 women) and 340 children (180 boys, 160 girls). Thirteen per cent refused a blood sample (12% of males, 14% of females) and in 4% the nurse was unable to obtain a sample (3% of males, 5% of females). From both sexes, blood was obtained from a smaller proportion of children than of adults (60% of eligible boys, 56% of girls). Some caution is therefore needed in the interpretation of the results for children, as the subjects from whom the sample was obtained may not be fully representative of the total sample.

BLOOD LEAD BY AGE AND SEX

The mean and geometric mean blood lead by age groups and sex are shown in Table 2.1.2.

Table 2.1.1 Blood lead sampling: response rates

Blood sample	Age (years)								All ages	
	11–15		16–44		45–64		65+			
	N	%	N	%	N	%	N	%	N	%
Males										
Obtained and analysed	180	60	1520	83	956	87	643	89	3299	83
Obtained, not analysed	7	2	29	2	22	2	20	3	78	2
Attempted, not obtained	15	5	35	2	31	3	29	4	110	3
Not attempted	–	–	1	0	–	–	–	–	1	0
Refused	99	33	246	13	86	8	32	4	463	12
Females										
Obtained and analysed	160	56	1597	76	1075	84	726	84	3558	79
Obtained, not analysed	15	5	57	3	39	3	23	3	134	3
Attempted, not obtained	16	6	99	5	63	5	48	6	226	5
Not attempted	–	–	1	0	–	–	–	–	1	0
Refused	97	34	341	16	107	8	68	8	613	14

– not applicable

Table 2.1.2 Blood lead (μg/dl) by age and sex

	Age (years)					
	11–15	**16–24**	**25–44**	**45–64**	**65+**	**All ages**
Males						
Mean	2.5	3.3	4.3	4.9	5.0	4.4
SE	0.1	0.15	0.09	0.12	0.13	0.06
Geometric mean	2.2	2.7	3.6	4.1	4.3	3.6
Females						
Mean	1.9	2.7	2.6	3.4	3.8	3.1
SE	0.06	0.21	0.05	0.07	0.08	0.04
Geometric mean	1.8	2.1	2.3	2.9	3.3	2.6
Total						
Mean	2.2	3.0	3.4	4.1	4.4	3.7
SE	0.06	0.13	0.05	0.07	0.07	0.03
Geometric mean	2.0	2.4	2.8	3.4	3.7	3.0

Table 2.1.2 Study population by age and sex

	Age (years)					
Sex	**11–15***	**16–24**	**25–44**	**45–64**	**65+**	**Total**
Males	204	324	1196	956	643	3323
Females	185	321	1276	1075	726	3583
Total	389	645	2472	2031	1369	6906

* The bases (study population) shown are weighted. Unweighted bases are about 12% smaller than the weighted bases (see Section 2.1.1)

The distribution of blood lead levels was higher in males than in females, at all ages. The geometric mean for males of all ages was 3.6 μg/dl and for females was 2.6 μg/dl. Lead concentration increased consistently and significantly ($p<0.01$) with age in both sexes, being highest in the 65 and over age group (geometric mean 4.3 μg/dl in men, 3.3 μg/dl in women) and lowest in children under 16 (2.2 μg/dl in boys and 1.8 μg/dl in girls).

Table 2.1.3 shows the distribution of blood lead levels by age and sex. No children had blood lead levels above 10 μg/dl (the action level proposed by the Centers for Disease Control; CDC, 1991), and among adults 5% of men and 1.1% of women had levels above 10 μg/dl. Of the 6517 adults included in the survey only 11 (1.7 per 1000) had levels above 25 μg/dl, the level above which investigations and interventions are recommended (WHO, 1987).

Table 2.1.3 Percentage distribution of blood lead levels by age and sex*

Blood lead levels (μg/dl)	Age (years) 11–15	16–24	25–44	45–64	65+	All ages
Males						
<2.5	61.6	49.1	29.5	20.7	17.7	28.6
2.5–5	32.3	38.0	43.3	45.8	43.9	42.9
5–10	6.1	10.8	22.7	27.0	31.7	23.5
>10	0	2.2	4.5	6.5	6.7	5.0
Females						
<2.5	80.3	69.8	61.3	40.8	32.8	51.1
2.5–5	18.6	24.6	32.1	42.7	44.6	36.4
5–10	1.1	4.7	6.0	14.8	21.1	11.3
>10	0	0.9	0.7	1.7	1.5	1.1
Total						
<2.5	70.5	59.4	45.9	31.4	25.7	40.3
2.5–5	25.8	31.3	37.5	44.2	44.3	39.6
5–10	3.7	7.8	14.0	20.5	26.1	17.2
>10	0	1.6	2.5	3.9	3.9	3.0

* Study population as for Table 2.1.2

BLOOD LEAD BY SOCIAL CLASS

The geometric mean lead level among men tended to be higher in individuals from lower social classes than in individuals from higher social classes (Table 2.1.4). Geometric means were 3.4, 3.5, 4.1 and 3.7 μg/dl in men in social classes I–II, IIINM, IIIM and IV–V respectively. Taken together, the geometric mean for men was significantly lower in non-manual social classes (I, II and IIINM) than in manual social classes (IIIM, IV and V) in all age groups ($p<0.01$). No statistically significant difference between manual and non-manual social classes was apparent among women.

BLOOD LEAD BY SMOKING STATUS

In both sexes, the geometric mean was higher in those who were current smokers than in those who were not ($p<0.01$). As shown in Table 2.1.5, in both sexes and at all age groups those who smoked regularly had higher geometric mean values than those who were ex- or non- smokers. In men, current smokers of 20 or more cigarettes a day had a geometric mean of 4.7 μg/dl, whereas those current smokers who smoked less than 20 cigarettes a day had a geometric mean of 4.0 μg/dl. The association was seen in all age groups except for the 16–24 year olds (where those who smoked 20 or more cigarettes a day were very few). In women the differences between smoking categories were less marked.

Table 2.1.4 Blood lead (μg/dl) by social class, age and sex

Sex / Age (years)	Social class I–II	IIINM	IIIM	IV–V	All social classes
Males					
16–24					
Mean	2.8	(3.1)	4.1	3.1	3.3
SE	0.20	(0.26)	0.47	0.20	0.17
Geometric mean	2.4	(2.8)	3.0	2.8	2.7
25–44					
Mean	3.8	3.8	4.9	4.7	4.3
SE	0.13	0.24	0.17	0.23	0.09
Geometric mean	3.2	3.2	4.1	3.9	3.6
45–64					
Mean	4.6	4.6	5.6	4.8	4.9
SE	0.16	0.35	0.27	0.28	0.12
Geometric mean	3.9	3.9	4.6	3.9	4.1
65+					
Mean	4.8	5.3	5.3	4.7	5.0
SE	0.17	0.49	0.25	0.24	0.13
Geometric mean	4.2	4.3	4.4	4.1	4.3
All males					
Mean	4.0	4.2	5.0	4.4	4.4
SE	0.08	0.17	0.12	0.13	0.06
Geometric mean	3.4	3.5	4.1	3.7	3.7
Females					
16–24					
Mean	2.7	(1.9)	2.8	2.5	2.6
SE	0.46	(0.12)	0.45	0.17	0.22
Geometric mean	2.1	(1.8)	2.1	2.2	2.1
25–44					
Mean	2.7	2.6	2.5	2.5	2.6
SE	0.08	0.14	0.09	0.09	0.05
Geometric mean	2.3	2.2	2.2	2.3	2.2
45–64					
Mean	3.6	3.2	3.3	3.3	3.4
SE	0.11	0.17	0.12	0.16	0.07
Geometric mean	3.1	2.8	2.9	2.8	2.9
65+					
Mean	3.9	3.8	3.4	3.9	3.8
SE	0.15	0.16	0.17	0.17	0.08
Geometric mean	3.4	3.4	2.9	3.3	3.3
All females					
Mean	3.2	3.1	2.9	3.1	3.1
SE	0.07	0.09	0.07	0.08	0.04
Geometric mean	2.7	2.6	2.5	2.6	2.6

Table 2.1.4 continued

Sex / Age (years)	Social class I–II	IIINM	IIIM	IV–V	All social classes
Males and females					
16–24					
Mean	2.8	2.6	3.4	2.8	3.0
SE	0.26	0.18	0.33	0.13	0.14
Geometric mean	2.3	2.3	2.5	2.5	2.4
25–44					
Mean	3.2	3.1	3.8	3.5	3.4
SE	0.08	0.13	0.11	0.13	0.05
Geometric mean	2.7	2.6	3.1	2.9	2.8
45–64					
Mean	4.1	3.8	4.4	4.0	4.1
SE	0.10	0.18	0.15	0.16	0.07
Geometric mean	3.4	3.2	3.6	3.3	3.4
65+					
Mean	4.4	4.3	4.5	4.2	4.4
SE	0.12	0.19	0.17	0.14	0.08
Geometric mean	3.8	3.6	3.7	3.6	3.7
Total					
Mean	3.6	3.5	4.0	3.7	3.7
SE	0.05	0.09	0.08	0.07	0.04
Geometric mean	3.0	2.9	3.2	3.1	3.1

( ) are shown where the number of people in the subset is <50

Table 2.1.4 Study population by social class, age and sex

Sex / Age (years)	Social class I–II	IIINM	IIIM	IV–V	All social classes
Males					
16–24	90	37	83	55	265
25–44	454	132	377	194	1157
45–64	403	80	282	180	945
65+	212	72	231	118	633
All males	1159	321	973	547	3000
Females					
16–24	97	27	94	61	279
25–44	467	201	317	212	1197
45–64	430	135	288	193	1046
65+	203	164	150	184	701
All females	1197	527	849	650	3223
Males and Females					
16–24	187	64	177	116	544
25–44	921	333	694	406	2354
45–64	833	215	570	373	1991
65+	415	236	381	302	1334
Total	2356	848	1822	1197	6223

Table 2.1.5 Blood lead (μg/dl) by smoking status, age and sex

Sex Age (years)	Smoking status Never smoked regularly	Former regular smoker	Less than 20 a day	20 or more a day	Total
Males					
16–24					
Mean	2.7	(3.9)	3.9	(3.5)	3.3
SE	0.14	(0.81)	0.36	(0.37)	0.15
Geometric mean	2.4	(2.9)	3.2	(3.1)	2.7
25–44					
Mean	3.9	4.0	4.6	5.4	4.3
SE	0.12	0.19	0.19	0.26	0.09
Geometric mean	3.2	3.4	3.9	4.6	3.5
45–64					
Mean	4.4	4.8	5.3	6.5	4.9
SE	0.20	0.18	0.31	0.43	0.12
Geometric mean	3.6	4.0	4.5	5.4	4.1
65+					
Mean	4.2	5.2	5.3	(5.6)	5.0
SE	0.18	0.18	0.38	(0.42)	0.13
Geometric mean	3.7	4.4	4.6	(5.1)	4.3
All males					
Mean	3.9	4.8	4.7	5.7	4.5
SE	0.08	0.11	0.14	0.21	0.06
Geometric mean	3.2	4.0	4.0	4.7	3.7
Females					
16–24					
Mean	2.1	(2.7)	3.4	(4.7)	2.7
SE	0.08	(0.52)	0.56	(1.72)	0.21
Geometric mean	1.9	(2.3)	2.5	(3.0)	2.1
25–44					
Mean	2.4	2.6	3.1	2.9	2.6
SE	0.06	0.10	0.14	0.16	0.05
Geometric mean	2.1	2.3	2.6	2.5	2.3
45–64					
Mean	3.2	3.4	3.9	3.7	3.4
SE	0.09	0.12	0.19	0.24	0.07
Geometric mean	2.8	2.9	3.3	3.2	2.9
65+					
Mean	3.7	3.7	4.3	(4.2)	3.8
SE	0.10	0.14	0.27	(0.60)	0.08
Geometric mean	3.2	3.2	3.8	(3.7)	3.3
All females					
Mean	2.9	3.2	3.5	3.5	3.1
SE	0.04	0.07	0.12	0.20	0.04
Geometric mean	2.5	2.8	2.9	2.9	2.6

( ) are shown where the number of people in the subset is <50

Table 2.1.5 Study population by smoking status, age and sex

Sex Age (years)	Smoking status Never smoked regularly	Former regular smoker	Less than 20 a day	20 or more a day	Total
Males					
16–24	170	22	98	28	318
25–44	570	223	239	163	1195
45–64	340	376	117	122	955
65+	162	385	63	32	642
All males	1242	1006	517	345	3110
Females					
16–24	188	17	88	23	316
25–44	674	232	258	112	1276
45–64	548	267	158	101	1074
65+	393	249	67	16	725
All females	1803	765	571	252	3391

BLOOD LEAD BY DRINKING HABIT

The results for blood lead by alcohol consumption are shown in Table 2.1.6. Very few respondents reported being ex-drinkers: those who did were therefore considered together with the non/occasional drinkers. In men, ex/non/occasional drinkers tended to have lower geometric mean values than drinkers; among those who drank regularly the geometric mean lead level tended to increase with the number of units drunk per week. This was true for all age groups and the overall geometric mean rose from 3.5 μg/dl in ex/non/occasional drinkers to 4.4 μg/dl in those who drank more than 21 units per week. The difference in lead levels among drinking categories was statistically significant ($p<0.01$).

A similar pattern was observed among women. The geometric mean for all age groups rose from 2.5 μg/dl for ex/non/occasional drinkers to 3.2 μg/dl in those drinking more than 14 units per week ($p<0.01$).

BLOOD LEAD BY SMOKING AND DRINKING

The unadjusted results for adults by smoking and drinking habits showed that within each drinking category average blood lead levels tended to rise with increasing smoking rates, especially in men. Similarly, within each smoking category among men, the heaviest drinkers had the highest blood level concentration, and a similar tendency was seen among women.

Table 2.1.6 Blood lead (µg/dl) by alcohol consumption, age and sex

Males Age (years)	Alcohol consumption: Non/ex/ occasional drinker	1–10 units per week	10–21 units per week	>21 units per week	Total
16–24					
Mean	(2.6)	2.9	3.5	3.6	3.3
SE	(0.23)	0.22	0.40	0.25	0.15
Geometric mean	(2.3)	2.5	2.7	3.0	2.7
25–44					
Mean	4.4	4.0	4.1	4.7	4.3
SE	0.35	0.14	0.16	0.16	0.09
Geometric mean	3.5	3.3	3.4	4.0	3.5
45–64					
Mean	4.5	4.1	4.8	6.1	4.9
SE	0.51	0.16	0.23	0.23	0.12
Geometric mean	3.5	3.5	4.2	5.1	4.1
65+					
Mean	4.5	4.4	5.2	6.9	5.0
SE	0.23	0.15	0.26	0.44	0.13
Geometric mean	3.8	3.9	4.6	5.9	4.3
All males					
Mean	4.3	4.0	4.4	5.3	4.5
SE	0.19	0.08	0.12	0.12	0.06
Geometric mean	3.5	3.4	3.7	4.4	3.7

Females Age (years)	Alcohol consumption: Non/ex/ occasional drinker	1–7 units per week	7–14 units per week	>14 units per week	Total
16–24					
Mean	2.9	2.3	2.4	3.4	2.7
SE	0.67	0.13	0.17	0.72	0.21
Geometric mean	2.0	2.0	2.1	2.5	2.1
25–44					
Mean	2.4	2.4	2.6	3.4	2.6
SE	0.10	0.06	0.08	0.14	0.05
Geometric mean	2.0	2.1	2.4	3.0	2.2
45–64					
Mean	3.0	3.3	3.8	4.4	3.4
SE	0.10	0.09	0.19	0.20	0.07
Geometric mean	2.6	2.8	3.3	3.8	3.0
65+					
Mean	3.6	3.7	4.3	4.8	3.8
SE	0.11	0.14	0.24	0.26	0.08
Geometric mean	3.0	3.2	3.8	4.5	3.3
All females					
Mean	3.0	2.9	3.2	3.9	3.1
SE	0.07	0.05	0.08	0.13	0.04
Geometric mean	2.5	2.5	2.8	3.2	2.6

( ) are shown where the number of people in the subset is <50

Table 2.1.6 Study population by alcohol consumption, age and sex

	Alcohol consumption				
Males Age (years)	**Non/ex/ occasional**	**1–10 units per week**	**10–20 units per week**	**>21 units per week**	**Total**
16–24	38	87	77	117	319
25–44	100	414	289	393	1196
45–64	111	331	210	304	956
65+	158	263	109	113	643
All males	407	1095	685	927	3114
Females Age (years)	**Non/ex/ occasional**	**1–7 units per week**	**7–14 units per week**	**>14 units per week**	**Total**
16–24	69	106	78	61	314
25–44	268	546	239	223	1276
45–64	321	428	165	161	1075
65+	370	217	81	58	726
All females	1028	1297	563	503	3391

BLOOD LEAD BY ETHNIC ORIGIN

Only 5% of those sampled were not of white European origin. The numbers were therefore too small for each ethnic group to be analysed separately and so all other ethnic groups were combined. White people tended to have higher blood lead levels than other ethnic groups combined in both sexes, and in males this difference was statistically significant ($p = 0.04$; data not shown).

BLOOD LEAD BY AREA OF RESIDENCE

Very small differences were observed in geometric mean levels by area of residence (Table 2.1.7). In males, the geometric mean was 3.8 µg/dl for those living in urban areas and 3.5 µg/dl for those living in rural areas. In females the overall geometric mean was 2.6 µg/dl in both groups.

BLOOD LEAD BY AGE OF DWELLING

Age of dwelling, stratified as before and after 1945, was recorded in only 51% of the sample and associated blood lead levels are shown in Table 2.1.8. In males the geometric mean lead level was 3.9 µg/dl for those who lived in a house built before 1945 and 3.4 µg/dl for those who lived in a house built after that date. In females the corresponding figures were 2.8 and 2.4 µg/dl. These differences were not statistically significant.

Table 2.1.7 Blood lead (μg/dl) by rural/urban area, age and sex

Sex	Region			
Age (years)	Urban	Suburban	Rural	All regions
Males				
11–15				
Mean	(2.6)	2.6	(2.3)	2.5
SE	(0.23)	0.13	(0.24)	0.10
Geometric mean	(2.3)	2.3	(2.0)	2.2
16–24				
Mean	3.3	3.2	(3.2)	3.3
SE	0.22	0.20	(0.52)	0.15
Geometric mean	2.8	2.7	(2.5)	2.7
25–44				
Mean	4.8	4.1	4.0	4.3
SE	0.21	0.11	0.17	0.09
Geometric mean	3.9	3.5	3.4	3.6
45–64				
Mean	5.3	5.0	4.6	4.9
SE	0.31	0.18	0.19	0.12
Geometric mean	4.2	4.1	3.9	4.1
65+				
Mean	5.2	5.0	4.9	5.0
SE	0.23	0.18	0.28	0.13
Geometric mean	4.4	4.2	4.2	4.3
All ages				
Mean	4.7	4.3	4.2	4.4
SE	0.13	0.08	0.11	0.06
Geometric mean	3.8	3.6	3.5	3.6
Females				
11–15				
Mean	(1.8)	2.0	(1.9)	1.9
SE	(0.11)	0.09	(0.10)	0.06
Geometric mean	(1.7)	1.8	(1.8)	1.8
16–24				
Mean	2.5	2.7	2.9	2.7
SE	0.15	0.31	0.77	0.21
Geometric mean	2.2	2.1	2.1	2.1
25–44				
Mean	2.7	2.6	2.5	2.6
SE	0.08	0.07	0.10	0.05
Geometric mean	2.4	2.3	2.1	2.3
45–64				
Mean	3.5	3.4	3.6	3.4
SE	0.16	0.08	0.13	0.07
Geometric mean	2.9	2.9	3.1	2.9
65+				
Mean	4.0	3.7	3.7	3.8
SE	0.17	0.10	0.16	0.08
Geometric mean	3.4	3.2	3.3	3.3
All ages				
Mean	3.1	3.0	3.1	3.1
SE	0.07	0.05	0.09	0.04
Geometric mean	2.6	2.6	2.6	2.6

( ) are shown where the number in the subset is <50

Table 2.1.7 Study population by rural/urban area, age and sex

Sex	Rural/urban			
Age (years)	Urban	Suburban	Rural	All regions
Males				
11–15	49	111	45	204
16–24	93	188	43	324
25–44	275	697	224	1196
45–64	202	502	252	956
65+	159	342	142	643
All ages	778	1840	706	3323
Females				
11–15	32	110	42	185
16–24	107	157	57	321
25–44	322	724	230	1276
45–64	232	572	271	1075
65+	189	394	143	726
All ages	882	1957	743	3583

Table 2.1.8 Blood lead (μg/dl) by age of dwelling*, age and sex

Sex	Age of dwelling		
Age (years)	Before 1945	1945 or after	All dwellings
Males			
11–15			
Mean	(2.4)	2.3	2.3
SE	(0.15)	0.16	0.12
Geometric mean	(2.3)	2.0	2.1
16–24			
Mean	3.2	2.8	3.0
SE	0.19	0.19	0.13
Geometric mean	2.9	2.4	2.6
25–44			
Mean	4.5	4.0	4.2
SE	0.18	0.14	0.11
Geometric mean	3.8	3.3	3.5
45–64			
Mean	5.2	4.6	4.8
SE	0.24	0.24	0.17
Geometric mean	4.4	3.8	4.0
65+			
Mean	5.9	4.7	5.1
SE	0.35	0.22	0.19
Geometric mean	5.0	3.9	4.3
All ages			
Mean	4.7	4.1	4.3
SE	0.12	0.10	0.08
Geometric mean	3.9	3.4	3.6

Table 2.1.8 continued

Sex	Age of dwelling		
Age (years)	Before 1945	1945 or after	All dwellings
Females			
11–15			
Mean	(2.3)	1.9	2.0
SE	(0.20)	0.09	0.09
Geometric mean	(2.1)	1.8	1.9
16–24			
Mean	3.1	2.1	2.5
SE	0.65	0.12	0.27
Geometric mean	2.3	1.9	2.1
25–44			
Mean	2.8	2.4	2.6
SE	0.13	0.06	0.06
Geometric mean	2.4	2.1	2.2
45–64			
Mean	3.6	3.2	3.3
SE	0.14	0.11	0.09
Geometric mean	3.1	2.7	2.9
65+			
Mean	4.3	3.4	3.7
SE	0.21	0.12	0.11
Geometric mean	3.7	3.0	3.2
All ages			
Mean	3.3	2.8	3.0
SE	0.10	0.05	0.05
Geometric mean	2.8	2.4	2.5

* Age of dwelling was recorded in 51% of the total sample

( ) are shown where the number of people in the subset is <50

Table 2.1.8 Study population by age of dwelling, age and sex

Sex	Age of dwelling		
Age (years)	Before 1945	1945 or after	All dwellings
Males			
11–15	39	71	110
16–24	82	97	179
25–44	247	389	636
45–64	198	317	515
65+	122	226	348
All ages	688	1100	1788
Females			
11–15	34	75	109
16–24	68	103	171
25–44	264	430	694
45–64	219	342	561
65+	133	262	395
All ages	718	1212	1930

BLOOD LEAD BY REGION

Among children of either sex there was no suggestion of a difference in blood lead levels by broad region of residence (north, midlands, south). However, from 16 years upwards those living in the north tended to have higher blood lead levels than those living in the midlands or the south of England. These differences were statistically significant ($p<0.01$) in both sexes (Table 2.1.9).

MULTIPLE REGRESSION ANALYSIS

Age, smoking, drinking, social class and area of residence (north, midlands, south) all had an independent significant effect on blood lead levels, in both sexes, after allowing for the effects of the other variables (Table 2.1.10).

Among these variables, age showed the largest impact on blood lead levels. Compared with those of people aged 16–24 years, blood lead levels increased with increasing age bands in both sexes.

Compared with non smokers, men who smoked 20 or more cigarettes a day had an increase of 0.32 µg/dl (CI 0.23, 0.41) in mean blood lead. In women a similar but weaker association (0.18 µg/dl, CI 0.10, 0.26) was observed.

Alcohol consumption also showed a significant association with blood lead in both sexes ($p<0.01$). Compared with non drinkers, blood lead levels were 0.33 µg/dl (CI 0.24, 0.41) higher among men who drank more than 21 units of alcohol per week. Among women, levels in those who drank more than 14 units per week were 0.38 µg/dl (CI 0.30, 0.47) higher than in non-drinkers.

Social class showed a significant independent association with blood lead in both sexes ($p<0.01$). However, the association was different in the two sexes. Compared with social classes I and II combined, lead levels in men were higher in social class IIIM only. Among women, in contrast, levels were significantly lower in all other social classes than in I and II combined.

Those living in the south of England had significantly lower mean blood lead levels than the overall average ($p<0.01$ in both sexes), whilst those living in the north had significantly higher levels.

Table 2.1.9 Blood lead (µg/dl) by broad region, age and sex

Sex	Region			
Age (years)	North	Midlands	South	All regions
Males				
11–15				
Mean	(2.5)	(2.9)	2.4	2.5
SE	(0.21)	(0.21)	0.14	0.10
Geometric mean	(2.2)	(2.6)	2.1	2.2
16–24				
Mean	3.7	3.7	2.9	3.3
SE	0.32	0.28	0.20	0.15
Geometric mean	3.0	3.1	2.4	2.7
25–44				
Mean	5.0	4.5	3.9	4.3
SE	0.19	0.21	0.10	0.09
Geometric mean	4.2	3.7	3.3	3.6
45–64				
Mean	5.4	5.2	4.6	4.9
SE	0.23	0.26	0.17	0.12
Geometric mean	4.5	4.3	3.8	4.1
65+				
Mean	5.8	5.0	4.7	5.0
SE	0.32	0.23	0.16	0.13
Geometric mean	4.8	4.3	4.0	4.3
All ages				
Mean	5.0	4.6	4.0	4.4
SE	0.12	0.12	0.07	0.06
Geometric mean	4.1	3.8	3.4	3.6
Females				
11–15				
Mean	(2.0)	(1.8)	1.9	1.9
SE	(0.17)	(0.13)	0.07	0.06
Geometric mean	(1.8)	(1.7)	1.8	1.8
16–24				
Mean	2.7	(3.0)	2.6	2.7
SE	0.17	(0.50)	0.33	0.21
Geometric mean	2.3	(2.4)	2.0	2.1
25–44				
Mean	3.0	2.7	2.4	2.6
SE	0.13	0.09	0.05	0.05
Geometric mean	2.6	2.4	2.1	2.3
45–64				
Mean	4.0	3.5	3.1	3.4
SE	0.14	0.14	0.08	0.07
Geometric mean	3.4	3.0	2.7	2.9
65+				
Mean	4.4	3.8	3.4	3.8
SE	0.17	0.18	0.10	0.08
Geometric mean	3.8	3.3	3.0	3.3
All ages				
Mean	3.5	3.2	2.8	3.1
SE	0.08	0.08	0.05	0.04
Geometric mean	3.0	2.7	2.4	2.6

( ) are shown where the number of people in the subset is <50

Table 2.1.9 Study population by broad region, age and sex

Sex	Region			
Age (years)	North	Midlands	South	All regions
Males				
11–15	49	48	107	204
16–24	86	71	167	324
25–44	284	247	665	1196
45–64	246	212	498	956
65+	159	148	336	643
All ages	824	726	1773	3323
Females				
11–15	46	30	109	185
16–24	93	48	180	321
25–44	305	255	716	1276
45–64	298	227	550	1076
65+	195	149	382	726

Table 2.1.10 Sex-specific regression models of variables influencing blood lead in adults

	Men (Base 2991)			Women (Base 3212)		
	N	Differences in blood lead from reference category (μg/dl)	95% CI	N	Differences in blood lead from reference category (μg/dl)	95% CI
Age (p<0.01)						
16–24[a]	259	0		269	0	
25–44	1156	0.32	0.22, 0.42	1197	0.08	0.01, 0.15
45–64	944	0.52	0.41, 0.65	1045	0.43	0.33, 0.53
65+	632	0.66	0.53, 0.81	701	0.69	0.57, 0.82
Smoking status (p<0.01)						
Non-regular smoker[a]	1186	0		1712	0	
Ex-smoker	986	0.10	0.04, 0.15	721	0.02	-0.03, 0.07
<20 a day	484	0.22	0.05, 0.29	536	0.19	0.13, 0.25
>20 a day	335	0.32	0.23, 0.41	243	0.18	0.10, 0.26
Alcohol consumption level (p<0.01)						
Non-drinker[a]	307	0		857	0	
Ex-drinker	77	0.02	-0.11, 0.17	111	-0.11	-0.19, -0.02
Men [women] Units per week						
1–10 [1–7]	1054	0.03	-0.04, 0.11	1240	0.05	0.00, 0.09
10–21 [7–14]	672	0.14	0.05, 0.23	533	0.18	0.11, 0.24
>21 [>14]	881	0.33	0.24, 0.44	471	0.38	0.30, 0.47
Social class (p<0.01)						
I–II[a]	1155	0		1189	0	
IIINM	320	0.01	-0.06, 0.08	527	-0.04	-0.09, 0.01
IIIM	970	0.15	0.10, 0.21	847	-0.07	-0.11, -0.03
IV–V	546	0.04	-0.02, 0.10	649	-0.06	-0.10, -0.01
Region (p<0.01)[b]						
North	751	0.08	0.05, 0.11	838	0.09	0.06, 0.12
Midlands	562	0.02	-0.02, 0.05	648	0.03	0.00, 0.06
South	1588	-0.09	-0.11, -0.06	1726	-0.11	-0.13, -0.09

CI, confidence interval
[a] Reference category
[b] Differences from overall geometric mean

2.1.3 DISCUSSION

The DoE undertook an extensive programme to monitor blood lead level annually in the UK over the period 1984–87 in the context of the reduction in petrol lead in 1986. A fall in blood lead was observed over the period, and the report concluded that by 1987 all the effect of the reduction in petrol lead had been observed and the observed reduction in blood lead was not solely due to the reduction in petrol lead content (DoE, 1990). The final recommendation was not to continue monitoring the population annually, but to repeat the survey 5 to 10 years later (DoE, 1990).

The current survey is the first in 8 years to have monitored blood lead levels in the English population. In this study we found low blood lead levels in a stratified random sample of the English population, in keeping with the findings from most developed countries. The study also confirmed findings of previous investigations that blood lead levels are higher in males than females, increase with age and are higher in those adults who have higher consumption of cigarettes and alcohol.

Possible explanations for the observed sex difference in blood lead levels are that 95% of lead in blood is bound to haemoglobin, which is lower in females than in males, and that smoking rates and alcohol intakes, which increase blood lead (Shaper *et al.,* 1982), are lower among women.

Environmental factors such as degree of urbanisation result in variations in lead exposure (Berode *et al.,* 1991). In the present study, however, area of residence (urban and rural) and age of dwelling were not found to be associated with blood lead levels.

Adults, but not children, living in the north of England had higher blood lead levels than those living in the midlands or the south. This broad regional analysis shows some consistency with the distribution of lead in the soil in England, as shown in the Wolfson Geochemical Atlas of England and Wales (Webb *et al.,* 1978).

Small effects of social class on blood lead have been reported (Quinn, 1985). In the present study when the association with social class (defined in the health survey on the basis of the occupation of the head of the household; Office of Population Censuses and Surveys, 1980) was tested in a multiple regression

analysis, blood lead showed a significant increase in men in social class IIIM only compared with social classes I–II, whereas in women blood lead showed a small but significant decrease in lower social classes.

As stated in the preliminary results of this survey (Delves *et al.,* 1996), blood lead levels have fallen by a factor of 2.6–3.0 in adults and 3.6–5.0 in children since the 1984–87 period. From the early 1970s to the mid 1980s there appears to have been a long-term downward trend in blood lead of around 4% per year (DoE, 1988); since then the decrease has been on average over 10% per year.

Interpretation of the differences between lead levels recorded in the 1984–87 monitoring programme and those in the current survey has to take into account the differences in the selection criteria used. The two populations differ in that the 1984–87 programme included non-occupationally exposed subjects as well as people living in particularly exposed areas and working in particular professions, whereas the health survey covered a representative sample of the general population in England — a sample which included subjects from different geographical areas with different ambient lead levels and different, undetermined, levels of occupational exposure.

Nevertheless, despite differences in selection criteria, the observed decline in lead concentrations between the surveys is large and therefore seems indicative of a real decline.

Previous action taken to reduce exposure to lead from petrol, water, canned food, decorative paint and industrial processes seems to have been effective in limiting the exposure to lead in the general population. It seems reasonable, assuming acceptable cost-effectiveness, to encourage the current trend in falling blood lead levels.

REFERENCES

Berode M, Wietlisbach V, Rickenbach M & Guillemin MP (1991) Lifestyle and environmental factors as determinants of blood lead levels in a Swiss population. *Environ Res, 55*, 1–17

CDC (1991) *Preventing Lead Poisoning in Young Children*: *A statement by the Centers for Disease Control*, Atlanta GA, USA, US Department of Health and Human Services

Christensen JM & Holst E (1988) Evaluation of blood lead levels in Danes for the period 1976–1987. *Fresenius A Anal Chem, 332*, 710–713

Delves HT (1970) A microscopy sample method for the rapid determination of lead-blood by atomic absorption spectroscopy. *Analyst, 95*, 431–438

Delves HT, Diaper SJ, Oppert S, Prescott-Clarke P, Periam J, Dong W, Colhoun H & Gompertz D (1996) Blood lead concentrations in United Kingdom have fallen substantially since 1984. *Brit Med J, 313,* 883–884

DoE (1986) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1984* (Pollution Report No 22), London, UK, HMSO

DoE (1987) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1985* (Pollution Report No 24), London, UK, HMSO

DoE (1988) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1986* (Pollution Report No 26), London, UK, HMSO

DoE (1990) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1987* (Pollution Report No 28), London, UK, HMSO

DHSS (1980) *Lead and Health: the Report of a DHSS Working Party on Lead in the Environment,* London, UK, HMSO

Elwood PC, Jones M, James K & Toothill C (1990) Evidence of a fall in cord blood levels in South Wales (UK). *Environ Geochem Health, 13*, 253–257

Graebner W (1988) Private power, private knowledge, and public health: science, engineering and lead poisoning, 1900–1970. In: Bayer, R ed., *The Health and Safety of Workers*, Oxford, UK, Oxford University Press, pp 15–71

Hertz-Picciotto I & Croft J (1993) Review of the relation between blood lead and blood pressure. *Epidemiol Rev, 19,* 352–373

Hense H-W, Filipiak B, Novak L & Stoeppler M (1992) Nonoccupational determinants of blood lead concentrations in a general population. *Inter J Epidemiol, 21*, 753–762

Hinton D, Coope PA, Malpress WA & Janus ED (1986) Trends in blood lead levels in Christchurch (NZ) and environs 1978–85. *J Epidemiol Community Health, 40,* 244–248

Moore MR, Hughes MA & Goldberg DJ (1979) Lead absorption in man from dietary sources. The effect of cooking upon lead concentrations of certain food and beverages. *Inter Arch Occup Environ Health, 44*, 81–90

Needleman HL & Gatsonis CA (1990) Low-level lead exposure and the IQ of children. *New Engl J Med, 236*, 673–678

Office of Population Censuses and Surveys (1980) *Classification of Occupation,* London, UK, HMSO

Pirkle JL, Brody DJ, Gunter EK, Kramer RA, Pascal DC, Flegal KM & Matte ID (1994) The decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys (NHANES). *J Am Med Assoc, 272,* 284–291

Prescott-Clarke P & Primatesta P, eds (1997) *Health Survey for England 1995* (Series HS no. 5), London, UK, HMSO

Quinn MJ (1985) Factors affecting blood lead concentrations in the UK. *Inter J Epidemiol, 14,* 420–431

Royal Commission on Environmental Pollution (1983) *Lead in the Environment* (Royal Commission on Environmental Pollution, Ninth Report), London, UK, HMSO

Rosner D & Markowitz G (1985) A gift of God? The public health controversy over leaded gasoline during the 1920s. *Am J Public Health, 75*, 344–352

Shaper AG, Pocock SJ, Walker M, Wale CJ, Clayton B, Delves HT & Hinks L (1982) Effects of alcohol and smoking on blood lead in middle-aged British men. *Brit Med J, 284*, 299–302

Shingal RL & Thomas JA, eds (1980) *Lead Toxicity*, Baltimore MD, USA, Urban and Schwarzenberg

Strömberg U, Schütz A & Skerfving S (1995) Substantial decrease of blood lead in Swedish children, 1978–94, associated with petrol lead. *Occup Environ Med, 52*, 764–769

Szymanski E & Hertz-Picciotto I (1995) Blood lead levels in relation to menopause, smoking and pregnancy history. *Am J Epidemiol, 141*, 1047–1058

Watt GCM, Britton A, Harper Gilmour W, Moore MR, Murray GD, Robertson SJ & Wormersley J (1996) Is lead in tap water still a public health problem? An observational study in Glasgow. *Brit Med J, 313,* 979–981

Webb JS, Thornton I, Thomson M, Howarth RJ & Lowenstein PL (1978) *The Wolfson Geochemical Atlas of England and Wales,* Oxford, UK, Oxford University Press

WHO (1987) *Air Quality Guidelines for Europe* (WHO European Series No 23), Copenhagen, Denmark, World Health Organization Regional Office for Europe, pp 242–261

Wittmers LE, Wallgren J, Alich A, Aufderheide AC & Rapp G (1988) Lead in bone. IV. Distribution of lead in the human skeleton. *Arch Environ Health, 43*, 381–391

2.2 THE ALSPAC STUDY ON LEAD IN CHILDREN

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2.2.1 BACKGROUND AND AIMS OF THE STUDY

There have been a number of studies that have shown that blood lead levels in children around the age of two are inversely associated with later IQ measures (Pocock *et al.,* 1994). Blood lead tends to reach its peak in children at around two years of age, but no geographical studies in the UK have been carried out prospectively in order to assess whether these earlier findings remain valid now that lead levels are thought to have dropped considerably. The aim of this study therefore was to build on the resource of the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) to determine blood lead levels at two years of age so that future studies could assess their impact on subsequent IQ.

METHODOLOGY

ALSPAC is set up with the specific aim of identifying features of the environment that affect the health, development and well-being of children (Golding *et al.,* 1996). It has collected information on environmental exposures from early pregnancy and continues to do so throughout the child's early life. The children themselves had an expected date of delivery between April 1991 and December 1992 inclusive and were born to mothers resident in the Avon area. This study comprises over 14 000 children. It is proposed that all children undergo a complete physical and cognitive examination at ages 7–8 years.

A randomly chosen set of ALSPAC children born between June 1992 and December 1992 have been examined in a clinic setting at frequent intervals from four months of age. The only exclusions from this subsample of the cohort were very immature babies (less than 33 weeks gestation) and their randomly selected controls. At 31 months, 1305 children were invited to attend this clinic (known as Children in Focus) and 1135 did so (87% of those invited). Of these children, 96.6% were seen in the age period 30 months 3 weeks to 31 months 3 weeks.

Blood sampling was by venepuncture, only undertaken with parental permission. A local anaesthetic cream (ELMA) was applied at least 50 minutes before the blood was taken. Parents of 81% of the 1135 children attending gave consent and of these children blood samples were obtained from 71% (N = 653). Of these, 69 samples were too small to allow lead analyses to be carried out.

The total concentration of lead in the blood samples was measured in duplicate by atomic absorption spectrometry using microsampling flame atomisation at Southampton General Hospital (Delves, 1970; DoE, 1986).

According to the pre-determined ethics protocol, when blood lead levels of 25 µg/dl or more were detected, the community paediatrician contacted the child's parents and provided them with a letter to take to their general practitioner to enable appropriate investigations to be carried out.

As the lead data were not normally distributed, for the purposes of statistical analysis the data were transformed using natural logarithms.

2.2.2 RESULTS

Blood lead results were obtained from 584 children. The distribution of blood lead levels is shown in Figure 2.2.1. The levels ranged from 0.8 to 27.6 μg/dl, and showed a skewed distribution. The arithmetic mean was 4.20 μg/dl, the median 3.30 μg/dl and the geometric mean 3.44 μg/dl. The proportion of blood lead levels exceeding different cut-off points is shown in Table 2.2.1.

Unadjusted analysis showed no significant association with maternal educational qualifications, with damp, condensation or mould in the home, with the use of day-care, with the number of other children in the household, the parenting score, the frequency with which the child was bathed or the month of the test.

There were, however, statistically significant unadjusted associations with (i) maternal age ($p = 0.001$), with children of younger mothers having higher lead levels; (ii) the ownership of the home, with children living in rented

Figure 2.2.1 Blood lead levels of children in the ALSPAC study

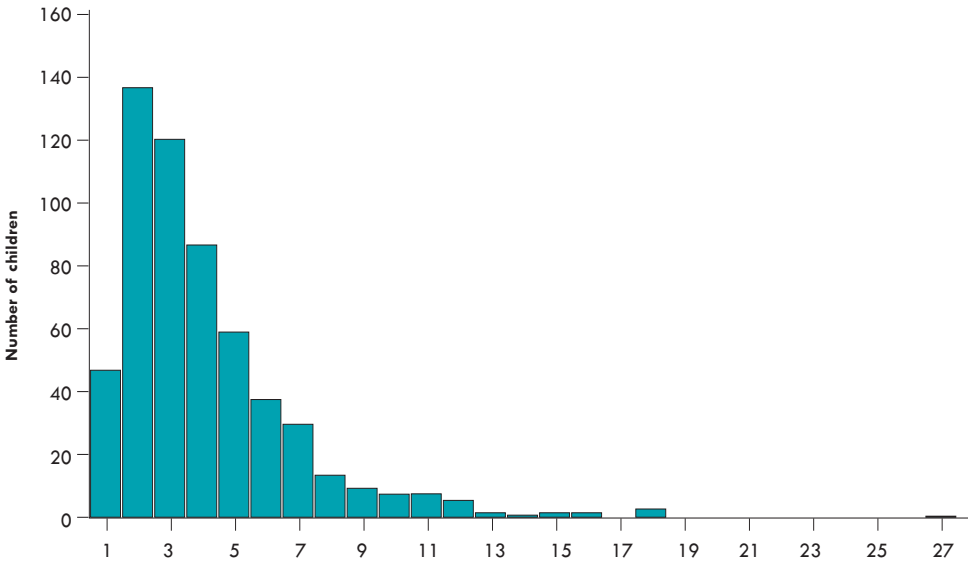

Table 2.2.1 The proportion of blood lead levels exceeding various cut-off points

Cut-off point (µg/dl)	Prevalence (%)
≥10	5.4
≥11	3.5
≥15	1.6
≥20	0.3
≥25	0.3

accommodation having higher lead levels ($p = 0.038$); (iii) exposure of the child to environmental tobacco smoke, those exposed having significantly higher levels ($p<0.001$); (iv) the presence of pets in the household, ($p = 0.002$); and (v) level of traffic ($p = 0.002$). There was an association with breast feeding such that the children who had been breast fed for at least 6 months had lower lead levels than those who had either never been breast fed or had been breast fed for less than 6 months ($p = 0.023$). In addition there was substantial geographical variation within Avon, with residents in central Bristol having the highest lead levels, and those in the outer areas of Bristol having the lowest levels ($p<0.0001$).

Adjustment of data for the geographic variable showed that the housing tenure ceased to be significant, but all other associations remained statistically significant, with the prevalence of pets in the home ($p = 0.0005$) and in particular of cats in the home ($p = 0.007$) becoming stronger in association. The relationship between the level of traffic and the postcode of the mother was strong, but level of traffic still remained statistically significant ($p = 0.03$). The geographical variation remained practically unchanged under the influence of the various items considered.

2.2.3 CONCLUSION

Although blood lead levels in children aged 2½ years are generally lower than in previous studies (see Section 2.3), some high levels were identified in this study. Although the mean blood lead level has dropped considerably, the range found is still comparable to that of 15 years ago. The key question is how the lead levels of the children may affect their health and development, and this will be tested with long-term follow-up studies of the children within the ALSPAC cohort.

REFERENCES

DoE (1986) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1984* (Pollution Report No 24), London, UK, HMSO

Delves HT (1970) A microscopy sample method for the rapid determination of lead-blood by atomic absorption spectroscopy. *Analyst, 95,* 431–438

Golding J and the ALSPAC Study Team (1996) Children of the Nineties: a resource for assessing the magnitude of long-term effects of prenatal, perinatal and subsequent events. *Contemp Rev Obstet Gynaecol,* 8, 89–92

Pocock SJ, Smith MA & Baghurst P (1994) Environment lead and children's intelligence: a systematic review of the epidemiologic evidence. *Brit Med J, 309*, 1189–1197

2.3 OVERVIEW OF UK AND INTERNATIONAL STUDIES ON TRENDS IN BLOOD LEAD, AND THE USE OF LEAD ISOTOPE RATIOS TO IDENTIFY ENVIRONMENTAL SOURCES

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2.3.1 OBSERVED DECREASES IN BLOOD LEAD LEVELS

The substantial, threefold to fivefold, decline in blood lead levels in the UK since 1994 (Delves *et al.,* 1996) is part of a continuing process which began in the mid to late 1970s (Figure 2.3.1). One of the earliest UK studies (Moncrieff *et al.,* 1964) reported an upper limit of 'normal' for children of 36 µg/dl and a median concentration of 23 µg/dl. By the mid 1980s, median blood lead levels for children had halved, and by 1995, for children over 6 years, they were only

1.7 µg/dl for girls and 2.3 µg/dl for boys (Delves *et al.,* 1996). For younger children aged 2½ years the median blood lead level in 1995, recorded in the ALSPAC study, was only 3.3 µg/dl (see Section 2.2.2) compared with 15.6 µg/dl for pre-school children in 1983 (Harvey *et al.,* 1984). Currently, blood lead levels of 10 µg/dl or more are unusual in UK children and only 1.2% of women and 5.3% of men had concentrations exceeding this in the 1995 Health Survey for England (Delves *et al.,* 1996).

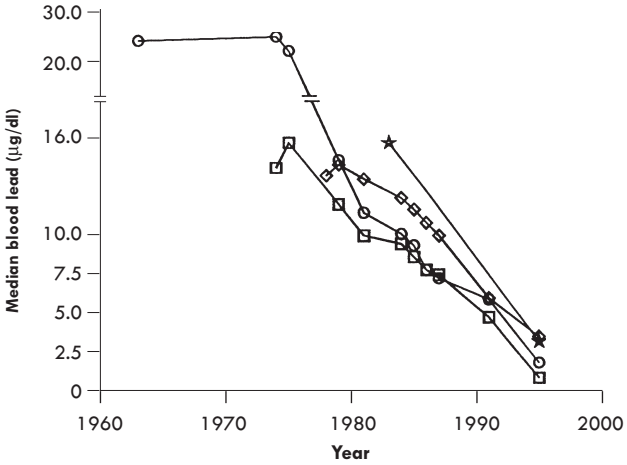
These observations suggest that blood lead levels above 10 µg/dl in UK subjects indicate increased exposure to lead and increased uptake, and that further investigations are warranted, especially for children.

The downward trend in UK blood lead levels (Figure 2.3.1) is similar to that seen in many other industrialised countries (Figures 2.3.2–2.3.4). Concentrations of lead in umbilical cord blood are generally lower than those seen in infants or in older children or adults, but even so, the general fivefold decline since 1980 is obvious for subjects in the USA (Figure 2.3.2). Similarly, blood lead levels for children and adults in other countries were about three to five times higher in the late 1970s to early 1980s, than they are currently (Figures 2.3.3 and 2.3.4). Notable exceptions to the low blood lead levels usually observed nowadays are those for inhabitants of Mexico City (Olaiz *et al.,* 1996) and urban areas of Korea (Yang *et al.,* 1996).

2.3.2 POSSIBLE CAUSES OF DECLINE IN BLOOD LEAD LEVELS

A wide range of measures to reduce lead were implemented in Britain from the late 1970s and throughout the 1980s. These included the removal of lead solder from cans containing food, the reduction of lead in some drinking water supplies (Sherlock *et al.,* 1984), and the restriction of the use of lead in paint, especially for children's toys and furniture. In addition to these measures, lead added to petrol was reduced from 0.8 g/l in 1971 to 0.4 g/l in 1981 and to 0.14 g/l in 1986.

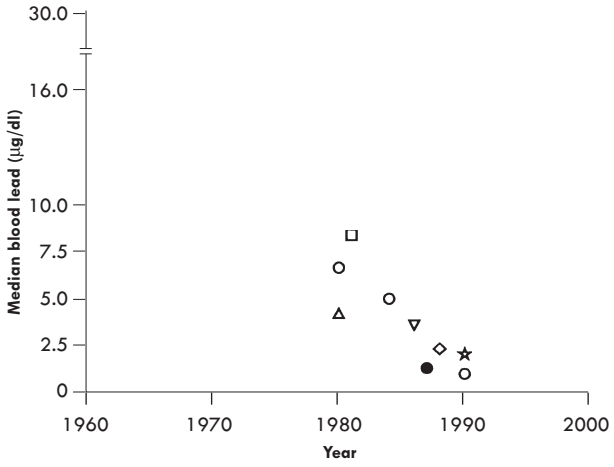
Figure 2.3.1 Biological monitoring of environmental lead in UK: changes in median blood lead levels 1964–1995



Children, ○ <16years; ★ <3 years; □ Adult females; ◇ Adult males

All analyses conducted by Great Ormond Street Hospital (1964–78) and Southampton General Hospital (1979–95). From Moncrieff *et al.* (1964); Elwood *et al.* (1977); Pocock *et al.* (1983); DoE (1981, 1983); Harvey *et al.* (1984); DoE (1990); Delves *et al.* (1996); and from the ALSPAC study (*Blood Lead Levels at Two and a Half Years,* Report to the DoE) and a study at a contaminated land site at Portsmouth, 1991

Figure 2.3.2 Temporal changes in cord blood lead levels



UK	△ From Zarembski *et al.* (1983)	Canada	☆ From Rhainds & Levallois (1993)
Denmark	◇ From Milman *et al.* (1988)	USA	○ From Rabinowitz & Needleman (1982); Satin *et al.* (1991); Hu *et al.* (1996)
Faeroe Islands	● From Grandjean *et al.* (1992)	Australia	□ From Tong *et al.* (1996)
Finland	▽ From Karpella *et al.* (1986)		

Figure 2.3.3 Temporal changes in children's blood lead levels

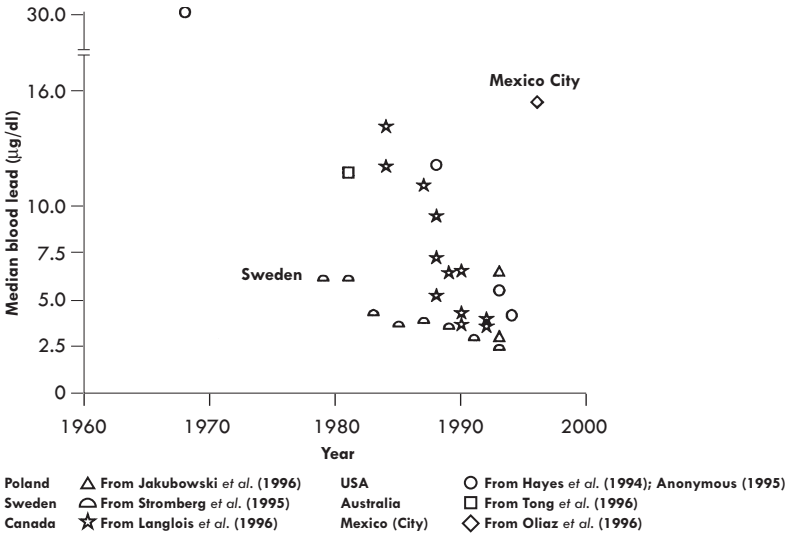
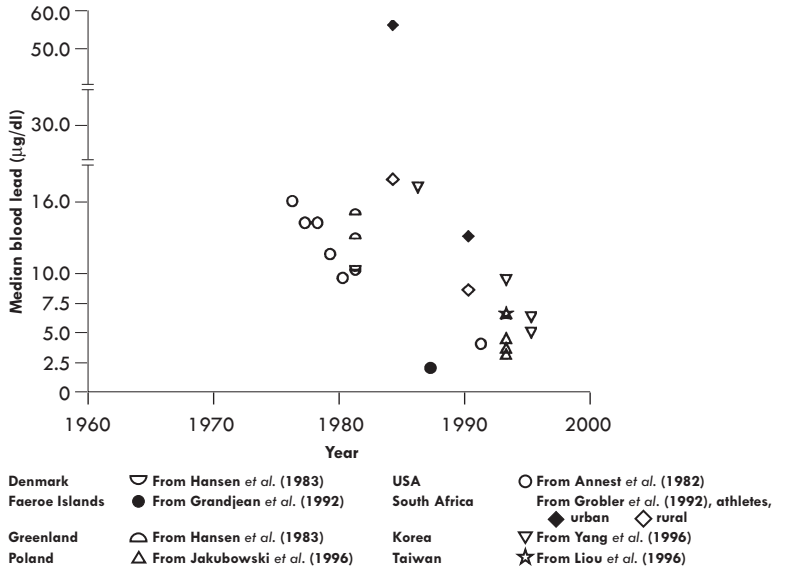


Figure 2.3.4 Temporal changes in adult blood lead levels



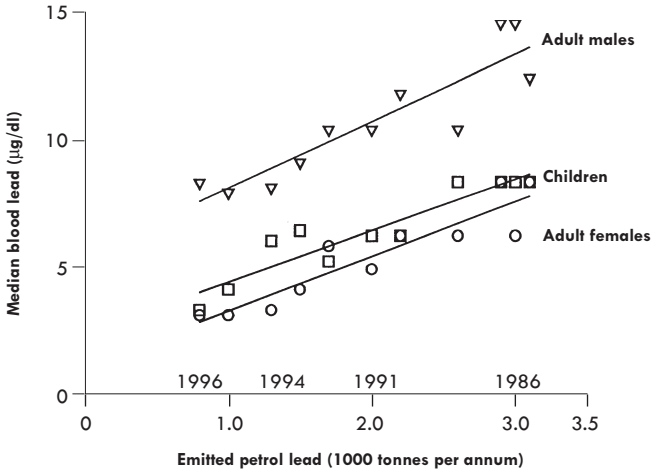
Since this last reduction the sales of lead-free petrol have increased from <0.1% of the total sales in 1986 to 64.5% of the total in 1995. The estimated emissions of lead from petrol-engine road vehicles has consequently fallen from more than 8000 tonnes per annum in 1972–74 to 3000 tonnes per annum in 1987, and to 1000 tonnes per annum in 1995 (DoE, 1995). This eightfold decline in petrol lead emissions will have played an important role in the lowering of UK blood lead levels. It would be difficult to quantify the effect for the period 1970–85 when many sources of lead were being simultaneously reduced. However, it might be possible to evaluate the effect of the reduction in petrol lead from 1986 onwards.

Blood lead data from the laboratory at the Department of Clinical Biochemistry, Southampton General Hospital, obtained for children screened for suspected lead poisoning and from adults screened either for toxicity or for monitoring occupational exposure, have shown a reduction over the past 20 years similar to that seen for environmentally exposed subjects. Median blood lead data for these subjects show highly significant correlations with emitted petrol lead over the period 1986 to 1995 (Figure 2.3.5) as do the data for environmentally exposed subjects, albeit with fewer data points (Figure 2.3.6). There are similarities in the slopes of the regression lines for all subjects in both figures. These data suggest that for each reduction of 1000 tonnes per annum in petrol lead emissions there will be a reduction in blood lead of 2.0 to 2.4 μg/dl for subjects screened for toxicity or occupational exposure, and of 2.6 to 3.4 μg/dl for subjects exposed only to environmental lead. Furthermore the regression data suggest that in the absence of other major sources of exposure, as petrol lead emissions approach zero then so will blood lead levels for subjects exposed only to environmental lead.

Clearly, whilst these are strong associations, they do not prove causality. Further studies using stable lead isotopic compositions would be of value in assigning relative contributions to body lead (see Section 2.3.3). Also, in certain locations in the UK, other sources may be important. For example, recent data from Glasgow have shown a reduction in blood lead associated with a reduction in water lead (Watt *et al.,* 1996).

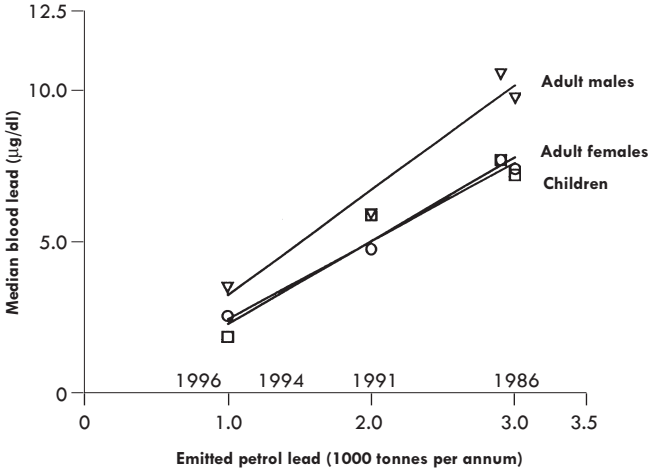
In other countries, the effect of petrol lead is more obvious. In South Africa in 1984, the median blood lead levels in athletes who trained in urban areas were 55.5 μg/dl compared with 17.7 μg/dl for athletes training in rural areas (Grobler *et al.,* 1996). At that time the petrol lead concentration was 0.8 g/l and this was subsequently reduced to 0.4 g/l in 1994, by which time the blood lead levels in urban and rural athletes had fallen to 13.0 μg/dl and 8.5 μg/dl respectively. Strömberg *et al.* (1995) are confident that their observed reductions in blood lead

Figure 2.3.5 Petrol lead in the UK and blood lead levels in subjects screened for toxicity or occupational exposure at Southampton General Hospital (1986–96)



Adult males ▽; $y = 2.6x + 5.4$; $r = 0.90$, $p = 0.0001$
Adult females ○; $y = 2.1x + 1.1$; $r = 0.92$, $p = 0.00005$
Children □; $y = 2.0x + 2.4$; $r = 0.93$, $p = 0.00002$

Figure 2.3.6 Petrol lead in the UK and blood lead levels in subjects screened for environmental exposure to lead at Southampton General Hospital (1986–96)



Adult males ▽; $y = 3.4x - 0.2$; $r = 0.98$, $p = 0.02$
Adult females ○; $y = 2.6x - 0.1$; $r = 0.99$, $p = 0.007$
Children □; $y = 2.7x - 0.4$; $r = 0.97$, $p = 0.03$

in Sweden from 1979 to 1994 are a consequence of reducing petrol lead. They observed a two year lag between the reduction in petrol lead and the reduction in blood lead of 3 to 19-year-olds and attributed this to the dampening effect of the slow release of skeletal lead in equilibrium with circulatory lead. Earlier studies from the USA (Annest, 1983) have reported correlations between blood lead and petrol lead.

2.3.3 STABLE LEAD ISOTOPIC ANALYSIS AND SOURCE APPORTIONMENT

Lead is one of the few elements that have stable isotopes produced by the decay of radioactive parents. The radiogenic isotopes ^{206}Pb, ^{207}Pb, and ^{208}Pb are produced respectively from ^{238}U, ^{235}U and ^{232}Th. The fourth stable lead isotope, ^{204}Pb, is not radiogenic but is a measure of primeval lead (Ault *et al.,* 1970). By measuring the stable lead isotope ratios in different sources and in body tissues and fluids it is possible to determine the relative contributions made by the sources to body lead. For this to be achieved, the different sources must be isotopically distinct and the increased uptake from a given source must be sufficient to produce a measurable change in isotopic composition of body lead.

This technique has been used successfully to identify sources of childhood lead poisoning (Yaffe *et al.,* 1983) and in environmental studies (Facchetti & Geiss, 1982; Delves & Campbell, 1993).

The most ambitious programme to assign the contribution of petrol lead to blood lead was that undertaken in Italy in the period 1974–81 (Facchetti & Geiss, 1982). An almost complete (~90%) exchange of the usual petrol lead distributed in the Piedmont region was effected during 1977–79. The total lead content of petrol was kept constant at 0.6 g/l but its isotopic composition was changed from North American lead with a ^{206}Pb:^{207}Pb ratio of 1.20 to Australian lead with a ratio of 1.04. The former ratio was similar to that found in Italian sources of lead (1.18) but the latter was unusually low. Following this exchange the ^{206}Pb:^{207}Pb ratios in airborne lead in Turin fell to around 1.06 indicating that about 90% was Australian lead. The ratios in the blood of all the subjects studied fell from around

1.16 in 1975 to around 1.13 to 1.14 during 1979, with little change in total lead concentrations. These data indicated that the contribution of petrol lead to blood lead for urban dwelling Italians was 24–27%.

Some of the observations made by Facchetti and Geiss are illustrated in Figure 2.3.7a. For comparison, some data from the UK (Delves & Campbell, 1993) are given in Figure 2.3.7b. Old English lead, for example that from the Mendips, has a ^{206}Pb:^{207}Pb ratio of approximately 1.18; this is reflected in UK water lead being supplied via lead piping and is also similar to the ratio seen in Italian subjects and sources before the exchange of petrol lead in 1977–79. The petrol lead supplied to the UK is predominantly Australian with a ^{206}Pb:^{207}Pb ratio of 1.065 (Figure 2.3.7b). This ratio in low-level sources and in body tissues and fluids of UK subjects with low lead concentrations is around 1.12 to 1.13. Fortuitously, then, the situation in the UK is qualitatively similar to that engineered for the expensive Italian study. It was concluded from the UK data in Figure 2.3.7b, that petrol lead contributed about 30–40% to the body lead of inner London children, but for some Scottish subjects the dominant source was water lead which contributed about 60% to body lead (Delves & Campbell, 1993). It is interesting that the ^{206}Pb:^{207}Pb ratios found in tissues of UK subjects from the Franklin expedition in 1840 (Farrer, 1993) are similar to those found in Roman rib remains and in modern-day (1970s) Italian subjects, with values around 1.18. The decline in UK ratios to 1.12–1.13 at low lead uptake indicates a substantial contribution to body lead, possibly up to 60–70%, from a source with a ratio of 1.04–1.06, such as petrol lead.

A more accurate assignment of source contributions to body lead may be achieved by measuring all four stable lead isotopes. Some improved discrimination has been achieved for the above UK study from measurements of three isotopes ^{206}Pb:^{207}Pb and ^{208}Pb (Figure 2.3.8). It was not possible to measure the ^{204}Pb isotope accurately because of its low abundance. If there were just two sources of environmental lead in the UK (say, water lead and petrol lead) then their relative contributions to body fluids and tissues could be assigned by simple proportionation using the loci of the data points along the line joining the two sources in Figure 2.3.8. Deviations from the line indicate either additional sources of lead or analytical errors or both. Measurements of all four isotopes on recently collected UK blood samples, in deciduous teeth (yet to be collected) and in environmental sources would be of great value in assigning the relative contributions of petrol lead and other sources to body lead.

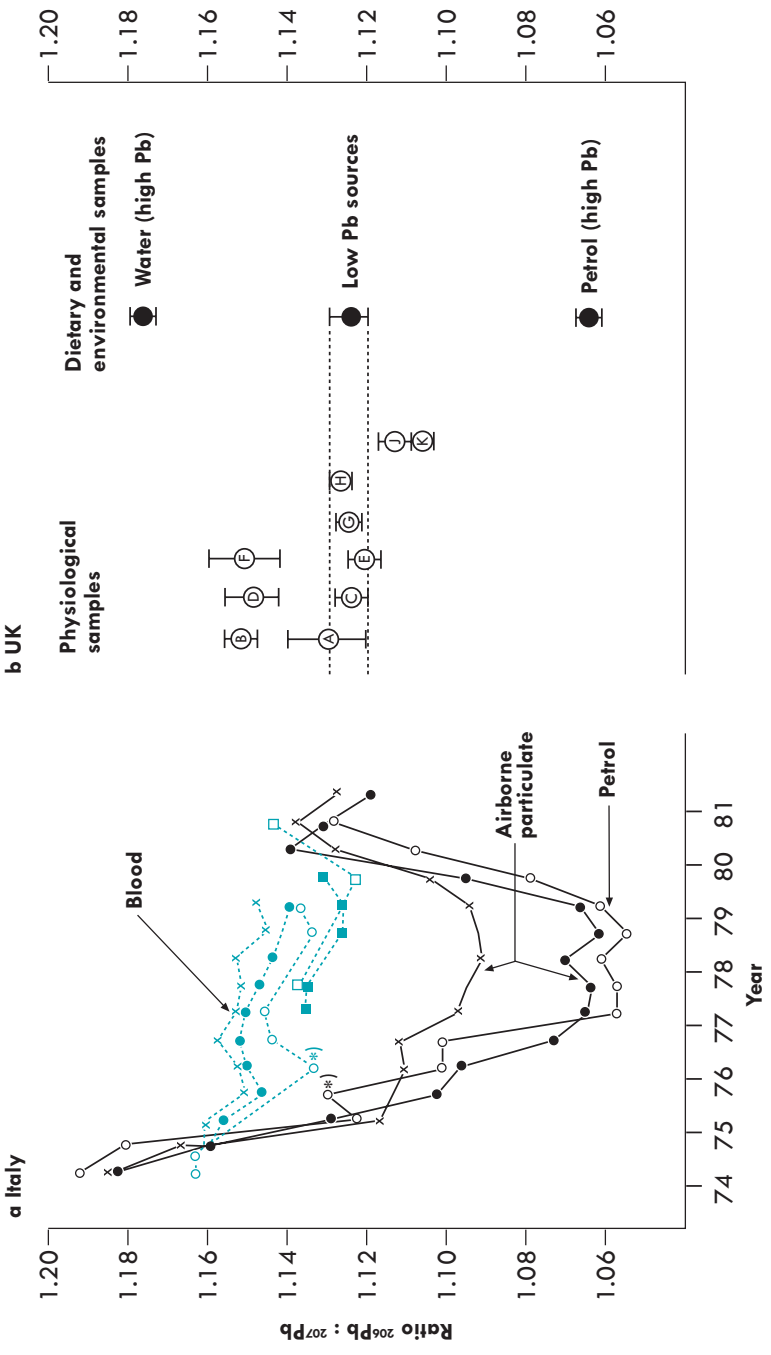


Figure 2.3.7 Comparison of lead isotope studies in Italy and the UK

Figure 2.3.7a Change in ^{206}Pb:^{207}Pb ratios in petrol, airborne particulates and blood 1974–81 in Italy (Adapted from Facchetti & Geiss, 1982)

Blood samples
- ● Adults, <25km Turin
- x Adults, >25km Turin
- ○ Adults, Turin
- □ Traffic wardens, Turin
- ■ School children, Turin

Airborne particulates
- x Countryside
- ● Turin

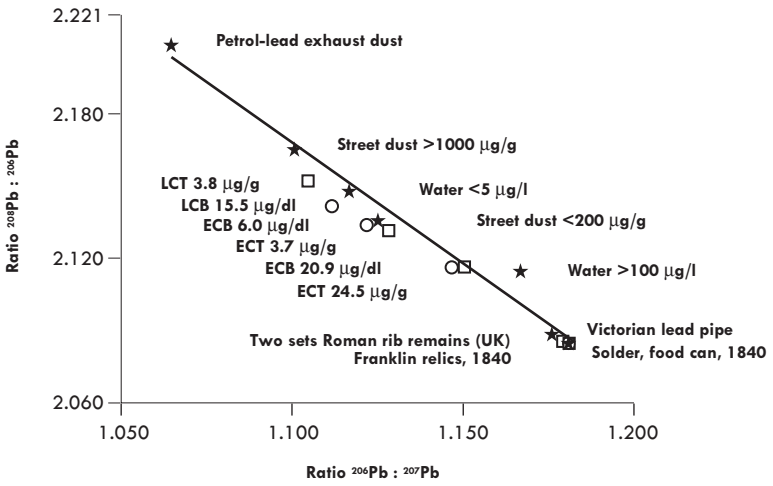
* based on a limited number of samples

Figure 2.3.7b Variations in ^{206}Pb:^{207}Pb in body tissues of Scottish and English subjects in some dietary and environmental samples (Adapted from Delves & Campbell, 1993)

Physiological samples

A Edinburgh, children's teeth 3.7 µg/g;
B Edinburgh, children's teeth 24.5 µg/g;
C Edinburgh, children's blood 6.0 µg/dl;
D Edinburgh, children's blood 20.9 µg/dl;
E Ayr, mothers' blood 7.2 µg/dl;
F Ayr, mothers' blood 21.3 µg/dl;
G Dundee, maternal/cord blood 6.5 µg/dl;
H Outer London, adult blood 11.2 µg/dl;
J Inner London, children's blood 15.5 µg/dl;
K Inner London, children's teeth 3.8 µg/g

Figure 2.3.8 Stable lead isotope ratios in body tissues and environmental sources of lead



All data are from Southampton except 1840 samples from Farrer (1993)

Sources of lead ★ ; blood samples ○ ; teeth or bone samples □
L = London; E = Edinburgh; C = children; B = blood; T = teeth.
$y = -1.007x + 3.27$ $r = -0.988$, $p < 0.0001$

2.3.4 CONCLUSIONS

Current blood lead levels in the UK, as in many industrialised countries, are only 1–3 μg/dl for subjects exposed only to environmental lead. Concentrations of 10 μg/dl are unusual and indicate increased lead uptake.

Although exposure to lead from a variety of sources has been substantially reduced, it is likely that the major cause of reductions in blood lead since 1986 is the reduction of petrol lead. Stable lead isotopic analysis bears this out, and more detailed isotopic analysis would help test this hypothesis further.

The uptake of lead by man from drinking water, derived from lead plumbing in areas where the water is very plumbosolvent, is likely to be most easily detected by total and isotopic analysis of lead in blood because of the low concentrations currently found in the absence of such uptake. This could be important in the northwest of England and in other areas in the UK.

REFERENCES

Annest JL, Mahaffey KR, Cox DH & Roberts J (1982) *Blood Lead Levels for Persons 6 Months–74 Years of Age: United States, 1976–80* (Advanced Data from Vital and Heath Statistics No.79. DHHS Pub No. (PHS) 82-1250), Public Health Service, Office of Health Research Statistics and Technology, US Department of Health and Human Services

Annest JL (1983) Trends in the blood lead levels of the US population. In: Rutter M & Jones RR, eds, *Lead versus Health,* Chichester and New York, John Wiley and Sons, pp 33–58

Anonymous (1995) Blood lead levels among children — Rhode Island, 1993–1995, *Morbid Mortal Weekly Report, 44,* 788–791

Ault WU, Senechal RG & Erlebach WE (1970) Isotopic composition as a natural tracer of lead in the environment. *Environ Sci Technol*, *4*, 305–313

Delves HT & MJ Campbell (1993) Identification and apportionment of sources of lead in human tissue. *Environ Geochem Health*, *15*, 75–84

Delves HT, Diaper SJ, Oppert S, Prescott-Clarke J, Dong W, Colhoun H & Gompertz D (1996) Blood-lead concentrations in United Kingdom have fallen substantially since 1984. *Brit Med J*, *313*, 883–884

DoE (1981) *European Community Screening Programme for Lead: United Kingdom. Results for 1979–1980* (Pollution Report No 18), London, UK, HMSO

DoE (1983) *European Community Screening Programme for Lead: United Kingdom. Results for 1981* (Pollution Report No 10), London, UK, HMSO

DoE (1990) *UK Blood Lead Monitoring Programme 1984–1987. Results for 1987* (Pollution Report No 28), London, UK, HMSO

DoE (1995) *Digest of Environmental Statistics No. 17,* London, UK, HMSO

Elwood WJ, Clayton BE, Cox RA, Delves HT, King E, Malcolm D, Ratcliffe JM & Taylor JF (1977) Lead in human blood and in the environment near a battery factory. *Brit J Prevent Soc Med*, *31*, 154–163

Facchetti S & Geiss F (1982) *Isotopic Lead Experiment Status Report*, Luxembourg, Commission of European Communities

Farrer KTH (1993) Lead and the last Franklin expedition. *J Archaeol Sci*, *20*, 399–409

Grandjean P, Weihe P, Jorgensen PJ, Clarkson T, Cernichiari E & Videro T (1992) Impact of maternal seafood diet on fetal exposure to mercury, selenium and lead. *Arch Environ Health*, *47*, 185–195

Grobler SR, Maresky LS & Kotze TJ (1992) Lead reduction of petrol and blood lead concentrations of athletes. *Arch Environ Health*, *47*, 139–142

Hansen JC, Kromann N, Wulf HC & Alboge K (1983) Human exposure to heavy metals in East Greenland II lead. *Sci Tot Environ*, *26*, 245–254

Harvey PG, Hamlin MW, Kumar R & Delves HT (1984) Blood lead, behaviour and intelligence test performance in preschool children. *Sci Tot Environ*, *40*, 45–60

Hayes EB, McElvaine MD, Hyman G, Orbach G, Fernandez AM, Lyne S & Matte TD (1994) Long-term trends in blood lead levels among children in Chicago: relationship to air lead levels. *Pediatrics*, *93,* 195–200

Hu H, Hashimoto D & Besser M (1996) Levels in lead in blood and bone of women giving birth in a Boston hospital. *Arch Environ Health*, *51*, 52–58

Jakubowski M, Trzcinka-Ochocka M, Razniewska G, Christensen JM & Starek A (1996) Blood lead in the general population in Poland. *Inter Arch Occup Environ Health*, *68,* 193–198

Karpella H, Loueniva R, Yrjänheikki E & Kaupila (1986) Lead and cadmium concentrations in maternal and umbilical cord blood, amniotic fluid, placenta and amniotic membranes. *Am J Obstetr Gynecol*, *155,* 1086–1089

Langlois P, Smith L, Fleming S, Gould R, Goel V & Gibson B (1996) Blood lead levels in Toronto children and abatement of lead-contaminated soil and house dust. *Arch Environ Health*, *51,* 59–67

Liou SH, Wu TN, Chiang HC, Yand T, Yang GY, Wu YQ, Lai JS, Ho ST, Guo YL, Ko YC, Ko NK & Chang PY (1996) Three-year survey of blood lead levels in 8828 Taiwanese adults. *Inter Arch Environ Health, 68*, 80–87

Milman N, Christensen JM & Ibsen KK (1988) Blood lead and erythrocyte protoporphyrin in mothers and new born infants. *Eur J Paediatr*, *147*, 71–73

Moncrieff AA, Koumides OP, Clayton BE , Patrick AD, Renwick AGC & Roberts GE (1964) Lead poisoning in children. *Arch Dis Childhood*, *39*, 1–13

Olaiz G, Fortoul TI, Rojas R, Doyer M, Palazuelos E & Tapia CR (1996) Risk factors for high levels of lead in blood of schoolchildren in Mexico City. *Arch Environ Health, 51*, 122–125

Pocock SJ, Shaper AG, Walker M, Wale CJ, Clayton B, Delves HT, Lacey RF, Packham RF & Powell P (1983) Effect of tap water lead, water hardness, alcohol, and cigarettes on blood lead concentrations. *J Epidemiol Commun Health*, *37*, 1–7

Rabinowitz MB & Needleman HL (1982) Temporal trends in the lead concentrations of umbilical cord blood. *Science, 216,* 1429–1431

Rhainds M & Levallois P (1993) Umbilical cord blood lead levels in Quebec City area. *Arch Environ Health*, *48*, 421–427

Satin KP, Neutra RR, Guirguis G & Flessel P (1991) Umbilical cord blood lead levels in California. *Arch Environ Health*, *46*, 167–173

Sherlock JC, Ashby D, Delves HT, Forbes GI, Moore MR, Patterson WJ, Pocock SJ, Quinn MJ, Richards WN & Wilson TS (1984) Reduction in exposure to lead from drinking water and its effect on blood lead concentrations. *Human Toxicol*, *3*, 383–392

Strömberg U, Schütz A & Skerfving S (1995) Substantial decrease of blood lead in Swedish children, 1978–94, associated with petrol lead. *Occup Environ Med*, *52*, 764–769

Tong S, Baghurst P, McMichael A, Sawyer M & Mudge J (1996) Lifetime exposure to environmental lead and children's intelligence at 11–13 years: the Port Pirie cohort study. *Brit Med J*, *312*, 1569–1575

Watt GCM, Britton A, Gilmour WH, Moore MR, Murray GD, Robertson SJ & Wormesley J (1996) Is lead in tap water still a public health problem? An observational study in Glasgow. *Brit Med J, 313,* 979–981

Yaffe Y, Flessel CP, del Rasano A, Matias V, Degarmo TE, Coleman GC, Granlich J & Kelly WR (1983) Identification of lead sources in Californian children using the stable isotope ratio technique. *Arch Environ Health*, *38*, 237–245

Yang JS, Kang SK, Park IJ, Rhee KY, Moon YH & Sohn DH (1996) Lead concentrations in blood among the general population of Korea. *Inter Arch Occup Environ Health*, *68*, 199–202

Zarembski PM, Griffiths PD, Walker J & Goodall HB (1983) Lead in neonates and mothers. *Clinica Chimica Acta*, *134*, 35–49

2.4 STUDIES OF LEAD AND CHILDREN'S IQ WITH REFERENCE TO BLOOD LEAD LEVELS OF THE POPULATIONS STUDIED

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2.4.1 INTRODUCTION

Much of the methodology for investigating the impact of single influences on child development or outcome has been developed and refined during studies of the impact of lead exposure. Lead is the toxin for which the effect on child outcome has been most researched; it is arguably the single most researched influence on child outcomes such as intelligence, educational attainment and behaviour. Despite this, there remains considerable uncertainty as to the real impact, if any, on children's intelligence of exposure to low levels of environmental lead.

That there is an association between body lead burden and measures of child intelligence is no longer in doubt. Curiously the association was much less clearly

evident in early lead studies, carried out on children living around smelters or lead industries, or children identified by urban screening programmes as having high levels of body lead. The blood lead levels of the 'exposed' or high lead groups in these early studies were typically more than 40 μg/dl, but despite these very high exposures, although some studies found an association of lead measures with IQ, others did not. One of the early British studies carried out in an area surrounding a lead smelter found a positive association between lead measures and child IQ, the children with higher lead levels also having significantly higher IQ scores (Lansdown *et al.,* 1974). The probable reasons for this finding will be discussed later.

In the past 20 years, however, since epidemiological studies of the relationship of body lead burden to child outcome have been carried out, the large majority of lead studies have reported an association, in uncontrolled data, between measures of lead exposure in children and measures of intelligence. The association is a negative one, with higher body lead measures generally associated with lower intelligence scores.

Cross-sectional epidemiological studies have now been conducted in Europe, including Eastern Europe, and in the USA, Australia, New Zealand and China. More recently a number of prospective studies have been carried out in the USA (in Cleveland, Boston and Cincinnati) and Australia (Port Pirie and Sydney), identifying a sample of children at or before birth, and following them as they develop. A recent systematic review (Pocock *et al.,* 1994), referred to in more detail later, identified a total of 26 studies of the lead and IQ relationship, all of which satisfied four specified criteria: they were published after 1978; they had a sample size of at least 100; they used a recognised measure of IQ; and the exposure measure was blood lead, whole tooth or dentine lead.

Before discussing some of the factors and inconsistencies that pose problems in interpreting the association between body lead burden and child intelligence, the main 'facts' of the association are presented below.

The lead and IQ association has been observed when the body lead burden has been assessed through single blood lead estimations (e.g., Harvey *et al.,* 1984; Fulton *et al.,* 1987) and serial blood lead estimations (e.g., Baghurst *et al.,* 1992; Dietrich *et al.,* 1993), and also with dentine lead (e.g., Needleman *et al.,* 1979), whole tooth lead (Smith *et al.,* 1983) and hair (Thatcher *et al.,* 1983). It has been noted with a variety of different measures of intelligence in addition to the most commonly used scale (the WISC-Wechsler Intelligence Scale for Children and its modifications and translations). In intelligence measures such as the WISC, where

performance IQ is distinguished from verbal IQ, and where this information is given, there is an association with both performance and verbal IQ, but it is usually greatest with the verbal components.

Intelligence is the most consistently measured outcome in lead studies, and it is also the one most consistently found to be associated with lead. Nonetheless, it should be noted that in attempts to illuminate the mechanism of lead effects, many lead studies have employed extensive test batteries of other psychometric tests in addition to intelligence tests. Tests of educational attainment, behaviour, gross and fine motor skills and coordination, visuospatial and auditory attention and memory, and attention and vigilance performance have all been included. Despite the number and scope of these other tests, most do not show any significant association with lead. Although lead associations have been found with outcomes other than IQ, there is little consistency between studies in the outcomes (either the skills or the particular tests) that are found to be lead associated. The exceptions to this are reading tests (which are themselves highly correlated with IQ measures), which have usually been found to be lead associated, and two specific tests — one a particular shape copying test and the other a complex reaction time task. In both these tests it is the error scores that have been found consistently to be lead associated.

Some studies have reported subgroup effects, for example that the lead–IQ relationship was only evident in socially disadvantaged children (Yule *et al.,* 1981), or only in boys (Pocock *et al.* 1989), but none of these subgroup effects have been found to be consistent between studies.

2.4.2 STRENGTH OF THE RELATIONSHIP BETWEEN BODY LEAD AND IQ

Before discussing the cumulated data on the strength of the lead and IQ relationship, it is relevant to note that the magnitude of the association varies between studies. It is not always of significant magnitude (e.g. Silva *et al.,* 1988) and sometimes is barely evident (Lansdown *et al.,* 1986). (An additional three studies that otherwise satisfied the criteria specified by Pocock *et al.* (1994) were

identified but not included in their meta-analyses, as in each case they were essentially 'negative' studies, where there was no initial lead–IQ association, and so further regression analyses were not carried out, and no regression coefficients were available.)

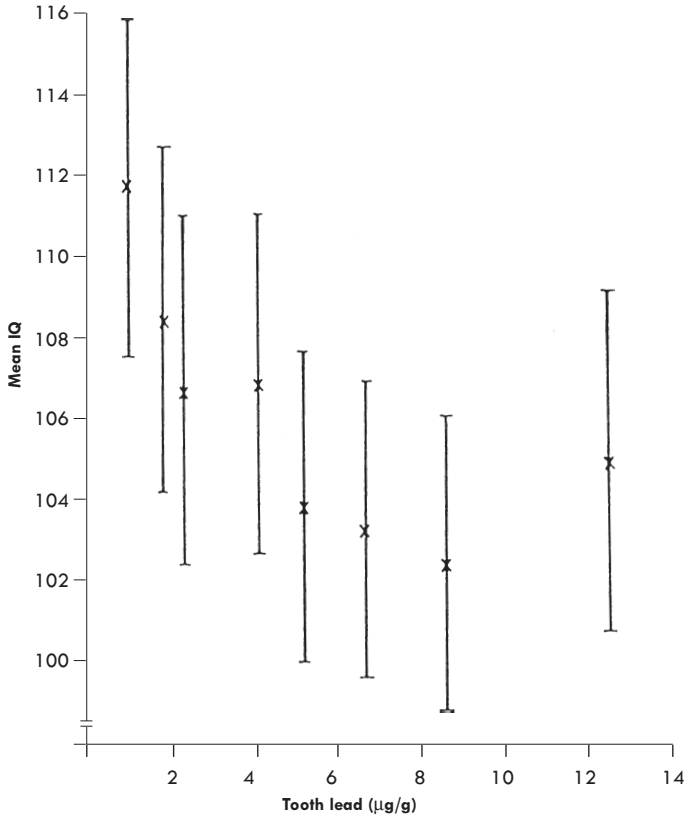
In most studies, however, the difference between groups with high and low blood lead, or from the extremes of lead distribution, varies between one and seven IQ points, but is usually between four and six IQ points in uncontrolled data. There is little evidence of a threshold or exposure level below which the lead–IQ association is not found. The association has, for example, been noted in a study in Denmark where the mean blood lead levels of the sample were around 5 μg/dl (Hansen *et al.,* 1989), and in an advantaged Boston sample (Bellinger *et al.,* 1992), where mean blood lead levels were only slightly higher at around 7 μg/dl.

This observation relates to one of the problems of the lead–IQ association: there is little evidence of a dose–response relationship between studies. Despite wide variation in the environmental exposure to lead of the populations studied, the size of the 'effect' on IQ is remarkably consistent, and inconsistently related to blood lead. To give an example, one of the studies with the lowest exposure, and a very restricted distribution of blood lead levels (which were not the primary lead measure used), also produced one of the largest IQ discrepancies — an eight point difference in verbal IQ between high and low lead groups (Hansen *et al.,* 1989). This compares with the four point IQ difference between high and low lead groups reported in the original population studied by Needleman *et al.* (1979). Again, blood was not the primary measure of lead body burden, but some blood lead data were available, and indicate that the mean blood lead of children in the high lead group was in excess of 30 μg/dl, while that for the 'low' lead group was over 20 μg/dl.

Within individual studies dose–response relationships have been reported, with increasing lead levels associated with a larger impact on IQ (e.g., Pocock *et al.,* 1989; Raab *et al.,* 1989). The pattern evident in Figure 2.4.1 has been found in several studies, with an upturn at the upper end of the lead distribution, indicating that the lead–IQ relationship differs in the extreme group.

In contrast, in one study carried out in the vicinity of an old lead mine in Greece (Hatzakis *et al.,* 1989), where the blood lead levels of the children were very high with a mean of 24 μg/dl, there is clear evidence of a dose–response relationship, but only at blood lead levels above 25 μg/dl (Figure 2.4.2).

Figure 2.4.1 Apparent dose–response relationship between IQ and tooth lead

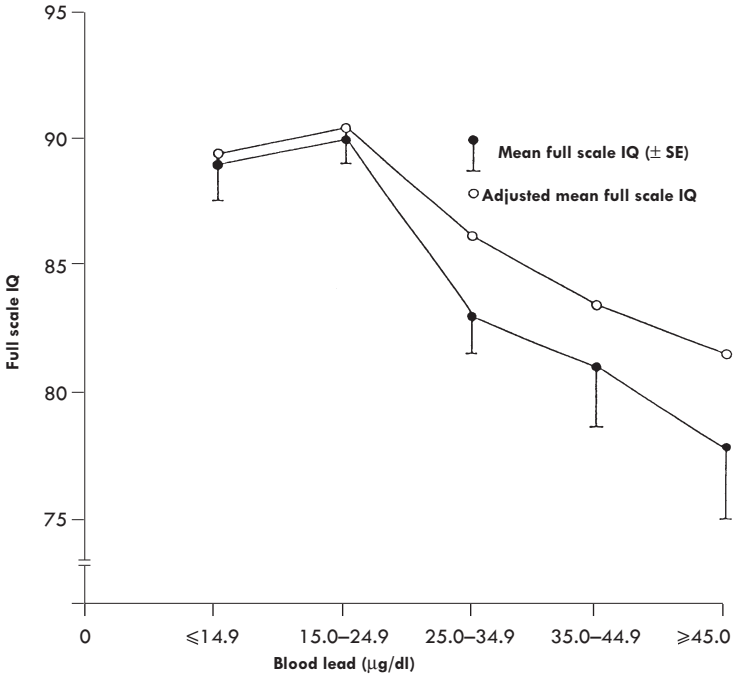


From Pocock *et al.* (1989)
Unadjusted mean IQ and 95% confidence limits for children in eight ordered groups of tooth lead
Reproduced by kind permission of Kluwer Academic Publishers, Lancaster, England, from Smith MA, Grant LD & Sors AI, eds (1989) *Lead Exposure and Child Development: an International Assessment*, London, Kluwer

2.4.3 INTERPRETATION

Most of the difficulties in interpreting studies showing lead–IQ relationships arise from observations, made in the same studies, that lead is an effective marker for social disadvantage: children in more disadvantaged circumstances have higher lead levels. One possible interpretation for the apparent dose–response relationships observed in some studies is that they reflect the increasing 'dose' of disadvantaging social factors (for which lead is a dose-sensitive marker) rather

Figure 2.4.2 Relationship between IQ and blood lead in children living near an old lead mine



Data from Hatzakis *et al.* (1989)
Adjusted and unadjusted mean WISC-R (revised version) full scale IQ values by blood lead levels in the Lavrion children
Reproduced by kind permission of Kluwer Academic Publishers, Lancaster, England, from Smith MA, Grant LD & Sors AI, eds (1989) *Lead Exposure and Child Development: an International Assessment*, London, Kluwer

than lead effects. The relationship between social factors and lead measures appears to hold even within environments where the exposure is atypical. For example, although children living near a lead smelter will generally have above-average body lead, their home circumstances are still important in explaining some of the variation in body lead levels. One of the probable exceptions is the study by Lansdown and colleagues (1974) mentioned earlier. As might be expected, children living nearer the lead works had significantly higher blood lead levels than children living further away. But there was some evidence that the families living further from the smelter were more socially disadvantaged than the more settled, stable families who had lived all their lives under the shadow of the lead works. The reverse confounding of social factors may explain the unusual result in this study. At the same time it demonstrates the importance of social factors in the relationship between lead and IQ.

There are few other exceptions to the normally observed relationship between lead and social disadvantage. The association has been noted for both demographic and environmental variables (such as social class, income, or overcrowded or poor housing), but lead is also an effective marker for parenting disadvantage to the child. For example, the child's lead level is an effective marker for the mother's mental state, and in particular, for maternal depression. It is probable that at least part of this association is mediated through dust or dirt in the home. The association between the state of cleanliness of the house and maternal depression can be demonstrated — it is not hard to imagine that house cleaning is not a priority for a depressed mother — and as there is evidence that the main ingestion source and route for young children is dust and dirt, via hand to mouth contact, this provides a probable mechanism. Poorer supervision of the child, and other factors such as diet, may also play a part. Other variables indicative of low parental interest in the child and a lack of child-centredness have also been demonstrated to be associated with lead levels. The problem is that variables such as maternal depression and parental interest also affect the child's intelligence, independently of any lead effect.

This finding, that lead uptake by children in urban populations is not random but highly collinear with disadvantage, has posed the major methodological problem in studies of the effects of lead on children, and remains the major problem in interpreting them. Studies have varied enormously in the extent to which they have collected information on, and controlled for, the influence of potentially confounding factors. The collinearity brings with it the problem of shared variance. Statistical control may remove variance properly attributable to lead — the problem of over-controlling. Equally, as one can never hope to measure all the complex of parental, social and environmental factors other than lead that influence a child's intelligence, control for confounding factors can never be complete — the problem of under-controlling. At most about 50% of the variance in a child's IQ can be explained by the most comprehensive inclusion of confounding variables. That, of course, leaves an equal proportion unexplained. In practice, in the large majority of studies control for potentially confounding variables has resulted in a far smaller proportion of explained variance in the IQ measures. The association of social variables with IQ measures, where they are quoted, is always stronger than the lead–IQ association, and the effect of controlling for social variables has been, in almost all studies, to reduce the strength of the lead–IQ association. This indicates that at least part of the observed relationship between lead measures and IQ is attributable to social variance rather than lead.

A further clue to the possible residual influence of social factors may lie in the nature of the lead and IQ relationship. As stated earlier, the lead association is usually most strongly evident with verbal, rather than performance IQ. It is known that performance IQ is more vulnerable to neurotoxic insult (and indeed, the performance element of the most widely used IQ test, the WISC, has been shown to be particularly sensitive to brain damage or dysfunction of any sort), whereas verbal IQ is more sensitive to socioeconomic and socially disadvantaging factors. Thus social class differences in IQ scores are most evident in the verbal components of the test.

2.4.4 META-ANALYSIS OF EVIDENCE FROM DIFFERENT STUDIES

Meta-analyses have been carried out (Pocock *et al.,* 1994) with the aim of examining the cumulated evidence on the lead–IQ association, from both prospective and cross-sectional studies. Five prospective studies were planned collaboratively, with the result that although there was still some variation in their design and methodology, there was also some consistency in the allowance for confounders. The purpose of the prospective studies was to investigate the relationship of lead levels *in utero*, or the earliest years of life (body lead levels tend to peak at around 2 years of age), with later outcome measures. All five studies have now reached the stage where the children are of school age. Measures of IQ have been obtained, and the data from them can be compared with the data from cross-sectional studies. Each of the prospective studies made repeated measures (a range of 5 to 25 measurements) of blood lead in each child, and most made frequent developmental assessments as well. Mean blood lead measurements at 2 years of age in these five studies ranged from 7 μg/dl in the Boston study to 21 μg/dl for the children living in the lead smelter area in Port Pirie, Australia.

In order to present the data from the different studies in a comparable format, for each study the multiple regression of IQ on lead (blood lead or tooth lead) and included confounding variables was used to obtain a regression coefficient for lead and IQ. This coefficient was then used to derive, for each study, an estimated change in IQ for either a specific doubling of blood lead (from 10 to 20 μg/dl) or tooth lead (from 5 to 10 μg/g).

None of the results from the prospective studies show any statistical association between blood lead levels of neonates and later measures of IQ (Figure 2.4.3a). (Furthermore, for the four studies that obtained them, maternal antenatal blood lead levels also showed no such association.) The estimated mean change in IQ for a doubling of blood lead (from 10 to 20 μg/dl) is +0.18 IQ points.

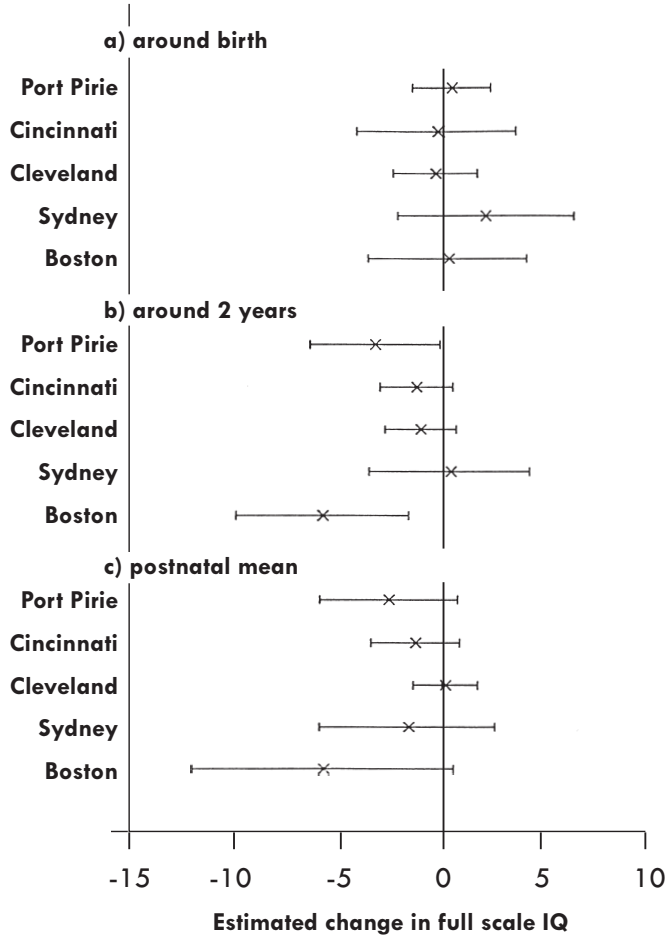
Blood lead measures taken at around the time of peak exposure, at 2 years, did show fairly strong evidence of an inverse association with IQ. Two of the studies showed significant associations (the 95% confidence intervals do not include zero) but the three others did not (Figure 2.4.3b). The estimated mean change was -1.85 IQ points for a doubling in blood lead. A mean of all postnatal blood measures (Figure 2.4.3c) shows rather less convincing evidence of an association than that of the measures on 2 year olds (an estimated mean change of -0.88 IQ points).

The 14 cross sectional studies using blood lead that were included in the analyses represent a range of mean blood lead values, from 7–8 μg/dl in Dusseldorf, Germany, to 24 μg/dl in the ancient mining area of Lavrion, Greece. The UK studies included in this analysis, all now reflecting blood lead samples obtained a decade or more ago, had mean blood lead values of around 11–13 μg/dl (Edinburgh: 11 μg/dl; London: 13 μg/dl; Birmingham: 12 μg/dl; and Greenwich: 13 μg/dl). The cross-sectional studies show more convincing evidence of an association, but greater variability (or less statistical heterogeneity), mainly due to one extreme value (in the Shanghai study). The order in which the studies are shown (Figure 2.4.4) is based on the size of the study (the largest at the top) and the degree of control for confounders (better or more comprehensive control at the top). The estimated IQ change for a blood lead doubling is -2.53 points (or -1.74 IQ points excluding Shanghai).

The seven tooth lead studies analysed showed greater homogeneity, but a smaller effect size, with an estimated change in IQ of -0.95 points for a specific doubling of tooth lead.

An attempt has been made to cumulate all the evidence (Figure 2.4.5). The type of study is indicated by a letter: P for prospective, C for cross-sectional blood lead, and T for cross-sectional tooth lead. Three sizes of letter indicate the sample size (>400, 200–400 and <200), and the square or circle indicates the degree to which confounders were controlled (a square for substantial control, or a circle for partial control). The influence of adjustment is shown by the arrowed lines. Thus the big letters in squares represent the biggest, best controlled studies.

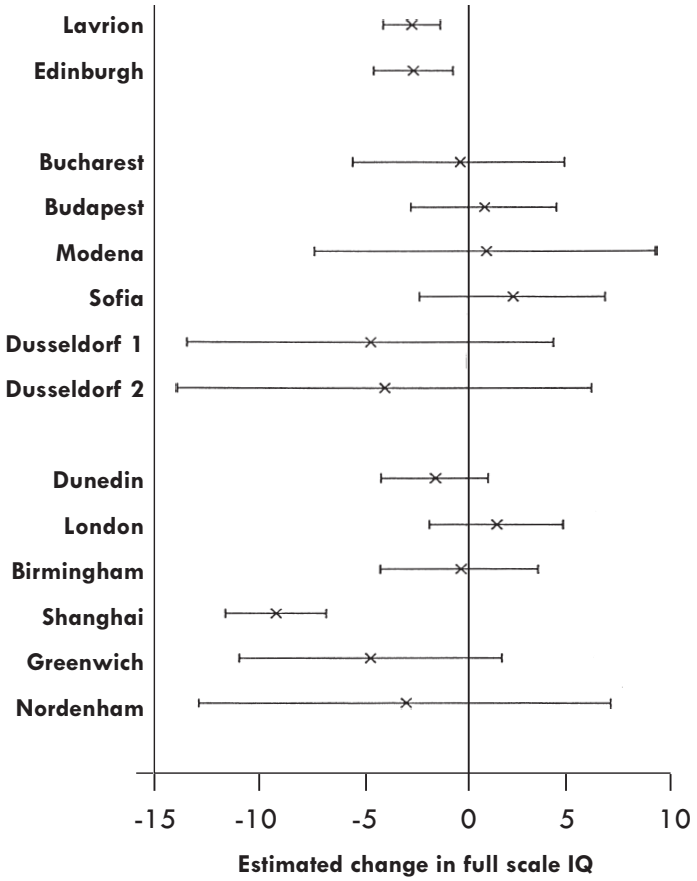
Figure 2.4.3 Results of prospective studies on blood lead and IQ



From Pocock *et al.* (1994)
Prospective studies estimated change in full scale IQ (and 95% confidence interval) for increase in blood lead from 10 to 20 µg/dl, using three measures of blood lead in each study

It can be seen that most studies show an inverse association — that is, they are below the horizontal zero line. The overall estimate was that a typical doubling of body lead burden (from 10 to 20 µg/dl blood lead or 5 to 10 µg/g tooth lead) was associated with a mean deficit in full scale IQ of around 1–2 IQ points. All the larger studies show a reduction in IQ of around 0.5 to 3 IQ points for a specific

Figure 2.4.4 Results of cross-sectional studies on blood lead and IQ



From Pocock *et al.* (1994)
Cross-sectional blood lead studies estimated change in full scale IQ (and 95% confidence interval) for increase in blood lead from 10 to 20 μg/dl
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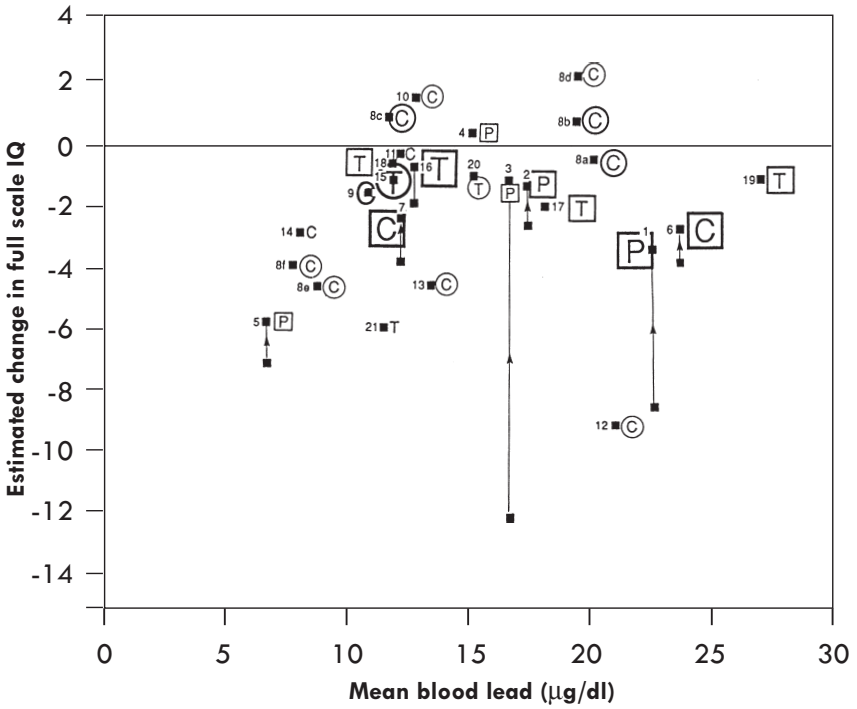
doubling of blood or tooth lead. And thirdly, but importantly, there is no striking relation between the mean blood lead level in the study and the magnitude of lead–IQ association, except that the three studies with the highest mean blood leads (Boston, Lavrion and Port Pirie) all had highly significant associations.

One of the outcomes of this exercise in cumulating information was a demonstration of the heterogeneity of the evidence, and of the limitations of

Figure 2.4.5 Cumulated results of all studies on blood lead and IQ



From Pocock *et al.* (1994)
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Study type: P prospective; C cross-sectional blood lead; T cross-sectional tooth lead
Sample size: P >400; P 200–400; P <200 etc.
Control of confounders: □ substantial; O partial
Influence of adjustment shown by arrows
Identifying number next to each point corresponds to study's reference in original publication

observational epidemiology in making causal interpretations. Chance can readily be ruled out as an explanation as the association is almost always in one direction, with higher lead associated with poorer performance. The hypothesis that lead is causal in the observed association is highly plausible, but does not account for the lack of dose–response. This hypothesis is supported by animal models, but exposure levels, differences in outcome measures, and the impact of confounding factors mean that animal models can provide only indirect support of biological plausibility. Two other possible explanations must also be considered. First, there is reverse causality — that is, that children who are less intelligent behave in ways that increase their uptake of lead. Lending some plausibility to this are the

somewhat stronger associations of IQ with current blood lead, than with historical or cumulative measures, such as those obtained from the prospective or tooth lead studies. A second possibility, for which there is some evidence, is that the association is due, at least in part, to uncontrolled social factors. With evidence that lead exposure in children is falling both in the UK and elsewhere, it is likely to be increasingly difficult to separate these different hypotheses. In conclusion, uncertainty still remains as to the real impact that lead makes on children's neuropsychological development.

REFERENCES

Baghurst PA, McMichael AJ, Wigg NR, Vimpani G, Robertson EF & Roberts RJ (1992) Life-long exposure to environmental lead and children's intelligence at age seven: the Port Pirie cohort study. *New Engl J Med*, *327*, 1279–1284

Bellinger DC, Stiles KM & Needleman HL (1992) low-level lead exposure, intelligence and academic achievement: a long term follow-up study. *Pediatrics, 90*, 855–861

Dietrich KN, Berger OG, Succop PA, Hammond PB & Bornstein RL (1993) The development consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati lead study cohort following school entry. *Neurotoxicol Teratol, 15,* 37–44

Fulton M, Thomson G, Hunter R, Raab G, Laxen D & Hepburn W (1987) Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet, 1,* 1221–1226

Hansen ON, Trillingsgaard A, Beese I, Lyngbye T & Grandjean P (1989) Neuropsychological profile of children in relation to dentine lead level and socio-economic group. In: Smith MA, Grant LD & Sors AI, eds, *Lead Exposure and Child Development: an International Assessment,* London, UK, Kluwer Academic, pp 240–250

Harvey PG, Hamlin MW, Kumar R & Delves HT (1984) Blood lead, behaviour and intelligence test performance in preschool children. *Sci Tot Environ, 40*, 45–60

Hatzakis A, Kokkevi A, Maravelias C, Katsouyanni K, Salaminios F & Kalandidi A (1989) Psychometric intelligence deficits in lead-exposed children. In: Smith AD, Grant LD & Sors AI, eds, *Lead Exposure and Child Development: an International Assessment*. London, UK, Kluwer Academic, pp 211–223

Lansdown RG, Shepherd J, Clayton BE, Delves HT, Graham PJ & Turner WC (1974) Blood lead levels, behaviour, and intelligence: a population study. *Lancet*, *1,* 538–541

Lansdown R, Yule W, Urbanowicz MA & Hunter J (1986) The relationship between blood-lead concentrations, intelligence, attainment and behaviour in a school population: the second London study. *Inter Arch Occup Environ Health*, *57*, 225–235

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C & Barrett P (1979) Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *New Engl J Med, 300*, 689–695

Pocock SJ, Ashby D & Smith MA (1989) Lead exposure and children's intellectual performance: The Institute of Child Health/Southampton study. In: Smith MA, Grant LD & Sors AI, eds, *Lead Exposure and Child Development: an International Assessment,* London, UK, Kluwer Academic, pp 149–165

Pocock SJ, Smith MA & Baghurst P (1994) Environment lead and children's intelligence: a systematic review of the epidemiological evidence. *Brit Med J, 309*, 1189–1197

Raab G, Fulton M, Thomson G, Laxen DPH, Hunter R & Hepburn W (1989) Blood lead and other influences on mental abilities — results from the Edinburgh lead study. In: Smith MA, Grant LD & Sors AI, eds, *Lead Exposure and Child Development: an International Assessment,* London, UK, Kluwer Academic, pp 183–200

Silva PA, Hughes P, Williams S & Faed JM (1988) Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin. *New Zealand J Child Psychol Psychiatry*, *29,* 43–52

Smith M, Delves T, Lansdown R, Clayton B & Graham P (1983) The effects of lead exposure on urban children: the Institute of Child Health/Southampton study. *Develop Med Child Neurol, 25,* 1–54

Thatcher, RW, Lester ML, McAlaster R, Horst R & Ignasias SW (1983) Intelligence and lead toxins in rural children. *J Learning Disabilities*, *16,* 355–359

Yule W, Lansdown R, Millar I & Urbanowicz MA (1981) The relationship between blood lead concentration, intelligence and attainment in a school population: a pilot study. *Develop Med Child Neurol, 23*, 567–576

2.5 TRENDS IN ENVIRONMENTAL LEAD LEVELS: IMPLICATIONS FOR HUMAN EXPOSURE

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2.5.1 INTRODUCTION

Lead is ubiquitous in the environment, present usually in small amounts from natural geological sources in all rock, soil, dust, water and air. These sources may influence the composition of foodstuffs for human consumption, the composition of dusts, which may be inhaled or ingested, and the composition of water supplies. All of these sources contribute to human exposure.

In addition, lead mineralisation, coupled with mining and smelting of lead ores, has over many centuries given rise to extensive contamination of the environment in many parts of the world. For example, in Britain alone, it has been estimated that an excess of 4000 km^2 of land is affected as a result of mining activity commencing in Roman times or earlier (Davies, 1980; Thornton, 1980).

This paper presents current information on the sources and amounts of lead in the surface environment and on pathways leading to human exposure. The

importance of the speciation of lead is emphasised, as this controls solubility, migration and bioavailability, and should be considered in the process of quantitative risk assessment. For example, most lead in soils and dusts is biologically inert and it is only the more soluble phases that present a potential hazard to plants, animals and man.

2.5.2 SOURCES OF LEAD IN THE ENVIRONMENT

NATURAL SOURCES

Natural sources of lead in the surface environment arise from the weathering of geological materials and emissions to the atmosphere from volcanoes, wind-blown dust, sea spray, biogenic material and forest fires. Rasmussen (1996) discusses the difficulties in estimating lead emissions from natural sources. For example, estimates of lead flux from volcanoes range widely from 540 tonnes/year to 6000 tonnes/year; as volcanic activity is episodic, it is difficult to obtain representative data (Nriagu, 1989). Large uncertainties in estimating metal fluxes from wind-blown dust are due to order of magnitude variations in metal concentrations of unconsolidated surface materials (Darnley, 1995).

ANTHROPOGENIC SOURCES

Worldwide anthropogenic emissions of lead to the atmosphere have been presented for 1983 by Nriagu and Pacyna (1988; Table 2.5.1). Although these figures provide an overall perspective, it must be borne in mind that regional and local variations will occur, reflecting factors such as the locations of major industrial lead works and the consumption of lead. It is accepted that these emissions have decreased, especially for leaded petrol. The majority of lead emitted in 1983 (66%) arose from the combustion of leaded petrol.

Atmospheric emissions of lead from mining and metallurgical operations constitute up to 22% of all anthropogenic sources of the metal (Table 2.5.1). In 1990, 155 mines in 35 countries produced 3.3 million tonnes of lead. Over the past

Table 2.5.1 Worldwide anthropogenic emissions of lead to atmosphere for 1983

Source category	Emissions (tonnes/year)
Coal combustion	
Electric utilities	775–4650
Industry and domestic	990–9900
Oil combustion	
Electric utilities	232–1740
Industry and domestic	716–2150
Pyrometallurgical non-ferrous metal production	
Mining	1730–3400
Pb production	11 700–31 200
Cu, Ni production	11 050–22 100
Zn, Cd production	5520–11 500
Secondary non-ferrous metal production	90–1440
Steel and iron manufacture	1065–14 200
Refuse incineration	
Municipal	1400–2800
Sewage sludge	240–300
Phosphate fertilisers	55–274
Cement production	18–14 240
Wood combustion	1200–3000
Mobile (traffic) sources	248 030
Miscellaneous (other industrial metal applications)	3900–5100
Total emissions	288 700–376 000

From Nriagu & Pacyna (1988)

decade, recycling has continued to play an increasingly important role. Of the total world production from more than 50 countries in 1990, which exceeded 5.6 million tonnes, over 41% was derived from recycled materials*.

Other anthropogenic sources include airborne emissions and liquid effluents from other industries, disposal of sewage sludges and other organic waste materials, domestic and commercial use of lead-containing materials, including paints, and the disposal of industrial and urban refuse in landfill and by incineration.

* Data from the American Bureau of Metal Statistics, 400 North Main Street, Suite 16, Manahawkin, New Jersey 08050, USA

LEAD IN THE ATMOSPHERE IN THE UK

Estimated atmospheric emissions for lead in the UK for 1985 were given as 'best estimates' by Hutton and Symon (1986), although the authors did not feel able to cite even a plausible range. These again reflect the great importance of leaded petrol combustion (Table 2.5.2). A recent summary of releases to the atmosphere in the UK (AEA, 1996) indicates an estimated reduction from 7086 tonnes in 1985 to 1755 tonnes in 1994. By far the greatest reduction was for lead from petrol, which fell from 6555 tonnes in 1985 to 1295 tonnes in 1994.

Table 2.5.2 Estimated atmospheric trace metal emissions in the UK (ca. 1985)

Source	Emissions (tonnes/year)
Non-ferrous metals production	51
Production and use of articles containing lead*	6802
Iron and steel production	478
Fossil fuel combustion	80
Cement manufacture	36
Municipal waste incineration	142
Sewage sludge incineration	1.2
Total emission	7590

From Hutton & Symon (1986)

* Including lead alkyls in petrol

Although no effects on health have been linked to the release of heavy metals from incineration plants, it was recommended that Her Majesty's Inspectorate of Pollution (now the Environment Agency) should set a separate standard for emissions of lead to air from this source (Royal Commission on Environmental Pollution, 1993). The total estimated 1991 lead emission from UK incineration plants was 161 tonnes.

Global emissions of lead to the atmosphere have been presented (ECE, 1994) on a continental basis for 1989 (Table 2.5.3). Lead emissions within the European Community (EC) region for 1985 are presented by source in Table 2.5.4, although a more recent estimate for 1990 suggests the total emission lies between 32 000 and 54 000 tonnes/year.

Concentrations of airborne lead strongly reflect the proximity of local sources of emissions, since atmospheric disposal processes rapidly dilute the lead compounds as they are carried downward from the source. Although airborne concentrations arising from stack emissions may be calculated with some confidence, fugitive

Table 2.5.3 Global emission inventory for lead in 1989

Continent	Maximum emission estimate (tonnes)						
	Petrol combustion	Fossil fuel combustion	Non-ferrous metal production	Waste incineration	Cement production	Steel and iron	Total
Africa	13 316	628	4421		85	86	17 536
Asia	44 006	4209	21 323	207	867	3713	74 325
Australia	2000	411	2775	62	12	118	5378
Europe	47 579	3477	13 041	537	641	4278	69 553
North America	14 192	993	7613	2498	177	1316	36 789
South America	8796	195	5422		98	555	15 066
Total	128 889	9913	54 595	3304	1880	10 066	208 647

From ECE (1994)

Table 2.5.4 Lead emissions in Europe (1985)

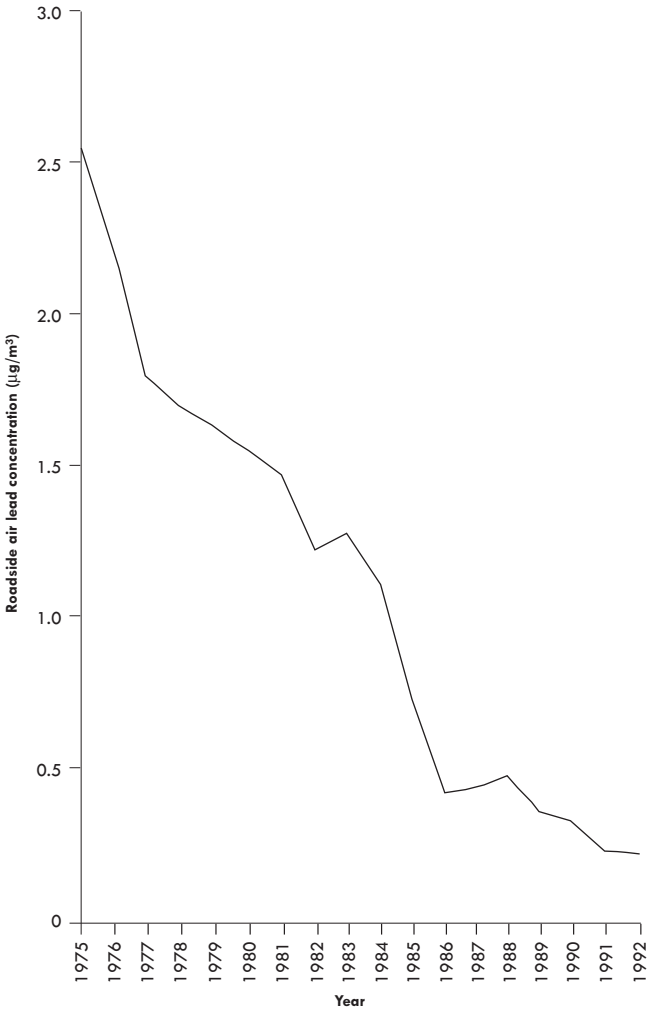
Category	Emissions (tonnes/year)
Fuel combustion in utility boilers	1300
Fuel combustion in industrial, commercial and residential units	1600
Petrol combustion	64 000
Non-ferrous metal industry	13 040
Iron and steel production	3900
Waste incineration	540
Other sources	1120
Total	85 500

From ECE (1994)

emissions, which are mostly from ground level sources, are very difficult to predict and require field measurement.

Concentrations of lead in air alongside roads are influenced both by vehicle density and the concentration of lead in petrol. At around 30–100 metres from a road, air lead concentrations are similar to background levels. Reduction of lead in UK petrol from 0.4 g/l to 0.15 g/l in 1985 and the subsequent introduction of lead-free petrol have lead to a decline in airborne levels. A study of 29 UK sites (Williams, 1990) showed air lead concentrations to have fallen by more than 50% in 1986 and 1987 from their pre-1986 levels. This decrease is further illustrated by measurements made in the city of Birmingham, which show a decline in air lead concentrations of around 90% over the period 1975 to 1992 (see Figure 2.5.1). The reduction in air lead due to the introduction of unleaded petrol means that a larger proportion of lead in air can be attributed to non-automotive lead sources.

Figure 2.5.1 Roadside air lead concentration in Birmingham



Data supplied by Environmental Services Department, Birmingham City Council, Aston Science Park, Birmingham B4 7NJ

Both wet and dry deposition processes remove lead from the atmosphere, resulting in contamination of surface waters (rivers, lakes and seas), soils, and vegetation, including food crops.

LEAD IN WATER

Concentrations of lead in river waters depend on local inputs, as residence times are short. In areas of lead mineralisation, rivers can contain up to ten times as much lead as those in unmineralised areas, in which background levels of lead tend to be well below 10 µg/l.

The main sources of lead in drinking water are lead service pipes and household plumbing. Other sources include lead–tin solder and brass fittings. The solubility of lead from pipes is dependent on the acidity of the water, temperature and residence time; generally acidic water is more plumbosolvent. A recent report of the House of Lords Select Committee on the European Communities (1996) concludes that there is no doubt that there are serious health risks from lead in drinking water, particularly for children, infants and fetuses, and that the elimination of lead from drinking water should be an ideal policy objective. Proposed EC legislation to reduce the lead standard for drinking water from 50 µg/l to 10 µg/l may prove difficult to comply with in the UK.

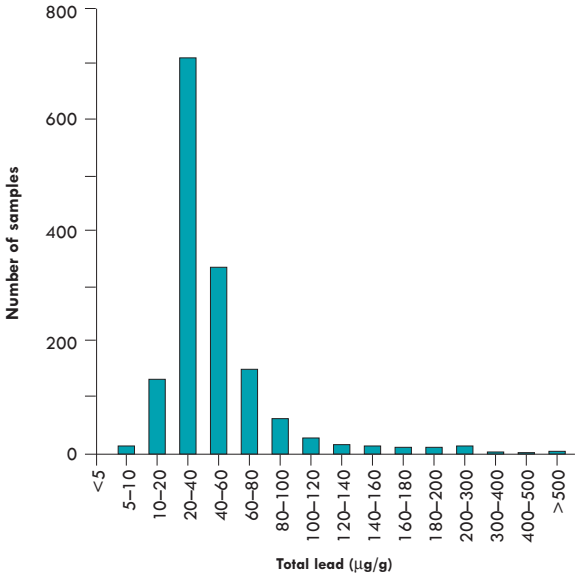
LEAD IN ROCKS AND SOILS

The average abundance of lead in the earth's crust is about 13 to 16 µg/g (Swaine, 1978). However, black shales, rich in organic matter, and sulphide minerals tend to have higher lead contents. The average concentration of lead in coal has been reported as 15 µg/g, ranging up to 60 µg/g.

It is difficult to obtain reliable information on typical lead concentrations in uncontaminated soils. Certainly in the UK and in many other parts of the world, there is widespread low-level contamination, reflecting long histories of urbanisation and industrialisation. A statistical study of soil lead for England and Wales has shown that in surface soils (0–15 cm) the concentration of lead ranges between 50 and 106 µg/g with a geometric mean of 42 µg/g (Davies, 1983). The frequency distribution of lead in 5000 agricultural soils for England and Wales is shown in Figure 2.5.2 (Archer & Hodgson, 1987). It is of interest that lead values for over 3000 surface soils from cropland in the USA were lower than those in Britain, with a median concentration of only 11 µg/g (Holmgren *et al.,* 1983).

The result of long-term contamination from atmospheric and other inputs has been greater accumulation of lead in surface soil than in lower depths. In normal agricultural soils an enhancement factor in the surface ranging from 1.2

Figure 2.5.2 Lead concentrations of soils in England and Wales



Adapted from Archer & Hodgson (1987)

to 2.0 is common, while in locations affected by historical mining and smelting, enhancement factors have been found to range between 4 and 20 (Colbourne & Thornton, 1978).

Lead accumulated in the surface soil is relatively immobile and is likely to remain for many hundreds of years. The vertical migration of lead in soils and rocks has been assessed at sites of three historical lead smelters in central Britain, ranging in age from around 600 to 1800 years (Maskall *et al.,* 1996). Of the total amount of lead present in surface soils, which often exceeds a concentration of 1% in these sites, only relatively small amounts appear to be in a mobile form. In this situation, it was concluded that lead is occluded in slag particles, precipitated as insoluble compounds and specifically absorbed to soil constituents.

Lead concentrations in urban soils are higher than in rural and agricultural soils. Results from the 1981–1982 UK nationwide survey of urban soils (see below) show a geometric mean of 230 µg/g for the majority of urban gardens and 650 µg/g in London (Thornton & Culbard, 1985). One of the important sources of lead in urban soils is paint, with additional inputs from fossil fuel and bonfire residues, fertilisers and atmospheric deposition.

LEAD IN DUSTS

Much work has been undertaken over the past 20 or more years to establish concentrations of lead in roadside and household dusts and it is not within the scope of this paper to review this extensive work. For example, results of the nationwide UK survey in 1981–82 showed lead concentrations in road dusts to range widely from 45 to 9660 µg/g, with geometric mean values of approximately 710 µg/g for all study locations excluding London, 1350 µg/g for London boroughs, and 2150 µg/g for Derbyshire mining villages; for comparison, values for household dusts were 500, 1010 and 1870 µg/g respectively (Thornton *et al.,* 1990). Household dust (floor dust) is likely to contribute around 50% of the total lead intake of the very young child.

LEAD IN FOOD

FAO/WHO (1987) proposed a Provisional Tolerable Weekly Intake for dietary lead of 3 mg for an adult, or 430 µg/day and a maximum of 25 µg/kg bodyweight per day for children and infants. The mean lead content of cereals, meat, offal, soft drinks, frozen foods, fruit and miscellaneous other foods examined during the 1980s in the UK (MAFF, 1989) was less than 100 µg/kg fresh weight and the majority contained less than 50 µg/kg. By 1987, the mean values were all below 90 µg/kg, for the various food categories examined. Many of the values were below the limit of determination and the lead content was therefore likely to be considerably lower than this limit.

In the vegetables examined, the lead concentration was generally less than 100 µg/kg fresh weight although spinach contained a mean concentration of 226 µg/kg — the spinach plant may be a lead accumulator. Samples of fish muscle contained too little lead for it to be detected.

However, concentrations of lead in gardens of mining areas in Derbyshire were two to four times as high as in other urban gardens, with 13% of the vegetables, in particular lettuce and spinach, exceeding the current statutory limit of 1 µg/g fresh weight in saleable food (Moir, 1992).

Most of the lead taken in with fluids comes from drinking water. It has been calculated that the lead in water contributes about 10% of the total dietary lead intake when the water contains 20 µg/l. Where the lead concentration reaches 100 µg/l, the contribution to the total dietary intake may then approach 40% (Smart *et al.,* 1981).

The absorption of lead from the intestine depends on many factors, including the amount of lead in the food and the presence of other elements, in particular calcium, phosphorus and iron. Thus, only a variable proportion of lead intake is retained by the body. This probably lies between 10% and 47% in adults (Moore *et al.,* 1979) and may be higher in children (Ryu *et al.,* 1983).

GEOCHEMICAL MAPS

The Wolfson Geochemical Atlas of England and Wales (Webb *et al.,* 1978) shows the regional distribution of lead to be largely governed by surface contamination from historical mining and smelting activities (Thornton, 1980). It has been estimated that some 4000 km^2 of agricultural land is contaminated with lead to various degrees. This includes an area of approximately 250 km^2 in the old mining and smelting area of Derbyshire in central England, where surface soils in agricultural land and in local village gardens contain from several hundred to several thousand µg/g lead, with those immediately adjacent to old workings having concentrations of 1–3% (Colbourne & Thornton, 1978; Cotter-Howells & Thornton, 1991). There is also an anomalous area around the city of Birmingham, Britain's second largest conurbation, where lead inputs from industry have contaminated the surface drainage system.

Similar geochemical atlases, based on the systematic sampling and analysis of stream sediments, have been published for 30 countries, each covering areas in excess of 5000 km^2 (Plant *et al.,* 1989). An International Geochemical Mapping Project (IGCP Project 259) has an ultimate goal of preparing geochemical maps of the world (Darnley, 1990).

The Soil Geochemical Atlas of England and Wales (McGrath & Loveland, 1992) was based on systematic sampling on a 5 km grid, yielding 5692 surface (0–15 cm) soil samples. The concentrations of lead ranged from 3 µg/g to over 16 000 µg/g with a geometric mean value of 46 µg/g.

URBAN GEOCHEMICAL MAPPING

Recent research into the impacts of urban development on the geochemical environment in the London Borough of Richmond on Thames and the industrial city of Wolverhampton has clearly shown an enrichment of lead in surface soils in developed locations compared with areas of open space. The highest

concentrations of lead in Richmond, those exceeding 1000 µg/g, tend to occur next to major road junctions and near roads with high traffic density. In addition, high concentrations of lead, of approximately 500 µg/g, occur in areas where housing is over 100 years old (Kelly *et al.,* 1996).

UK NATIONAL SURVEY OF LEAD IN DUSTS AND SOILS

A nationwide survey of metals in dusts and soils commissioned by the DoE was undertaken in 1981 and 1982 in 53 locations in England, Scotland and Wales (Thornton & Culbard, 1985). Cities, towns and villages were selected to reflect differences in geographical location and geology, degree of industrial and urban development, and population distribution. Seven London boroughs were included. Known 'geochemical hotspots' in areas of past mining and smelting activity were added to reflect likely acute exposures.

This survey gave the following results.

- Concentrations of lead in dusts and soils were high enough that some urban children were likely to be exposed to significant amounts of lead.
- Overall, 10% of homes had in excess of 2000 µg/g lead in house dust; 5% of garden soils had more than this level.
- London had significantly higher levels of lead, with 18% of homes and 10% of soils exceeding 2000 µg/g lead.
- 'Geochemical hotspots' showed significant lead contamination within the home. In Winster, Derbyshire, for example, 44% of house dusts had more than 2000 µg/g lead and 93% of soils exceeded this level, with 1% lead or more in several gardens.

A questionnaire for each household was used to provide information on the factors that might affect the accumulation of lead in house dust, including age of property, distance from roads, road type, occurrence of old lead paint and recent decorating history. In Brighton, England (Davies & Thornton, 1987), lead in both dust and garden soil increased with increasing age of property (Table 2.5.5).

Table 2.5.5 Geometric mean lead concentrations of dusts and soil in and around houses of various ages in the UK

Age of house	Lead concentration (μg/g)		
	Dust		Soil
	Vacuum	Road	
Pre-1870	982	1483	1146
1870–1919	1874	1607	1014
1920–1939	619	681	368
1940–1959	433	472	292
1960–1986	241	316	131

From Davies & Thornton (1987)

2.5.3 HUMAN EXPOSURE TO LEAD IN THE UK

Previously, the total intake of lead from food and drink by the average person in the UK was estimated at 220 μg/day (MAFF, 1972). By 1982 these estimates were lower. They are shown alongside intake from inhalation in Table 2.5.6. Davies and Thornton (1989) attempted to compare estimates of lead uptake from data from the US Environmental Protection Agency (EPA, 1986) with those estimated by the UK Royal Commission on Environmental Pollution (Royal Commission on Environmental Pollution, 1983), using absorption factors of 50% in the lung and 10% in the gut for adults, and 70% in the lung and 50% in the gut for the 2-year-old children, the percentage absorption used in the Royal Commission on Environmental Pollution Report (Table 2.5.7). In general, both total and daily estimates are lower for comparable groups in the USA than in the UK.

Table 2.5.6 Contribution to lead intake (UK) from air, water and food

Area	Air			Water			Diet
	Lead conc. (μg/m^3)	Air volume inhaled (m^3)	Daily intake (μg)	Lead conc. (μg/l)	Water volume inhaled/ ingested (l)	Daily intake (μg)	(including contribution from cooking water) (μg/day)
Rural area (low)	0.1	15	1.5	5	1.1	5.5	94
Urban area	2	15	30	100	1.1	110	140

From MAFF (1982)

Table 2.5.7 Comparison of estimated intake and uptake of lead in the USA and the UK

	USA			UK					
	Adult male	Adult with garden	2-yr-old child	Rural adult	Inner city adult	Inner city adult+ garden	Adult near main road	Rural 2-yr-old child	Inner city 2-yr-old child
Total lead intake (μg/day)	60.2	180.2	47.3	114–143	125–154	235	395	133	197
Total lead uptake (μg/day)	6.4	18.4	25.2	12–18	18–23	29	44	70	105
Lead uptake from petrol (μg/day)	2.8	5.8	15.9	2.0	6.8	17	6.5	3.3–45	5.9–80
Lead uptake from petrol (%)	44	32	63	11–16	30–39	59	15	5–64	6–76

From Davies & Thornton (1989) based on data from EPA (1986) and Royal Commission on Environmental Pollution (1983)

In 1985, a comprehensive study of environmental, dietary and blood lead was based on 97 2-year-old children in Birmingham, England. This provided detailed measurements allowing the calculation of total lead uptake based on lead intake from air, dust and diet (Davies, 1987). With an average indoor air lead concentration of 0.26 μg/m^3, an average time spent indoors of just over 23 hours per day and an assumed volume of respired air of 6 m^3/day, the lead intake from air would be approximately 1.5 μg/day; assuming a combined retention and absorption factor of 0.7, lead uptake would be just over 1 μg/day. In addition, a lead intake of 23 μg/day from the diet (food and beverages) and 42 μg/day from dust, assuming that ingested lead from dust and from the diet are equally bioavailable and that 53% of the lead ingested reaches the blood stream, would result in a total uptake by ingestion of 35 μg lead per day. The total estimated uptake (ingested and inhaled) of 36 μg/day is only about one third of that calculated by the Royal Commission on Environmental Pollution (1983) and much closer to that estimated for 2-year-old children in the USA (EPA, 1986). 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 the diet must be treated with caution.

A subsequent study of lead concentrations in dusts and soils was undertaken in 1996 in and around 85 of the households previously sampled in Birmingham in 1985. Lead concentrations and loading of lead (concentration of lead × weight of dust per area) in indoor and outdoor dusts were shown to have fallen by as much as 50% over the previous 11 years (Wang *et al.,* 1997). However, lead concentrations in garden soils had fallen to a much lesser extent. It was concluded that the fall in lead concentrations was largely due to reductions in atmospheric lead levels originating from traffic, as shown in Figure 2.5.1.

In an area of past lead–zinc mining in Derbyshire, a collaborative study by the Paediatric Unit, St Mary's Hospital Medical School and Imperial College showed that, when households were grouped according to the lead content of the soil in the gardens, the amount of lead in the blood and hair of children aged 2–4 years increased with that in the soil and house dust. None of the lead values in children were high enough to be considered hazardous at that time, even though the amounts in the soil and dust were extremely high, peaking at 2.8% and 2.5% lead, respectively. However, the children's lead status reflected that of the environment and, in the absence of evidence linking the human burden with lead in foodstuffs or water supplies, it was suggested that the major pathway of lead intake in the children was through the inhalation and the involuntary ingestion of dust particles (Barltrop *et al.,* 1975).

A more recent appraisal within one of these mining villages, in which the lead concentrations in garden soils and house dusts averaged 7000 and 1500 μg/g, respectively, showed that blood lead levels were within normal UK ranges and were certainly less than those predicted by some lead exposure models. Analysis of lead-rich soil grains, by scanning electron microscopy, has revealed that many are composed of pyromorphite, a stable soil–lead mineral formed by the weathering of galena and with an extremely low solubility, which may contribute to the low human bioavailability of lead from these soils, resulting in the lower than expected blood lead levels (Cotter-Howells & Thornton, 1991).

2.5.4 RESEARCH NEEDS

It is now important that attempts are made to determine current environmental lead levels in those towns and cities in which blood samples were taken in the recent UK survey (Delves *et al.,* 1996). The falling lead concentrations in dusts and soils in Birmingham, reported above, possibly represent the national picture, but this assumption will need to be verified. It would also seem prudent to undertake a limited survey of blood lead levels in young children in these locations, as it is the young child that is most at risk from lead in dust and soil as a result of hand-to-mouth activity.

Furthermore, there is an urgent need for the development and standardisation of procedures for lead analysis. Lead is present in soils, waters and air in a variety of chemical and mineral forms, many of which are largely biologically inert. In soils, most forms of lead are relatively immobile and it has been shown that, in old mining areas in both the UK and the USA, lead minerals weather to insoluble forms or are encapsulated in inert substances. More research is needed to increase our understanding of the species of lead in soils and plants, and how these different species affect human exposure and uptake. The processes responsible for producing these insoluble forms are likely to vary greatly under different climatic conditions (Thornton, 1996).

It is current practice in the regulatory process to make use of risk assessment strategies aimed at the protection both of human life and of the ecosystem. However, there are frequently insufficient reliable and relevant experimental data on which to base dose–response relationships. At present, there is no broadly accepted chemical method for analysing environmental materials to quantify

biologically active lead. Until this is accomplished, those responsible for risk assessment and the development of regulations will continue to ignore the subject of bioavailability.

REFERENCES

AEA (1996) *Estimated UK Emissions for Lead*, Harwell, UK, Atomic Energy Authority

Archer FC & Hodgson IH (1987) Total and extractable trace element contents of soils in England and Wales. *J Soil Sci, 38*, 421–431

Barltrop D, Strehlow CD, Thornton I & Webb JS (1975) Absorption of lead from dust and soil. *Postgrad Med J, 51*, 801–804

Colbourne P & Thornton I (1978) Lead pollution in agricultural soils. *J Soil Sci, 29*, 513–526

Cotter-Howells J & Thornton I (1991) Sources and pathways of environmental lead to children in a Derbyshire mining village. *Environ Geochem Health*, *13*, 127–135

Darnley AG (1990) International geochemical mapping: a new global project. *J Geochem Explor, 39*, 1–13

Darnley AG (1995) *A Global Geochemical Database for Environmental and Resource Management* (Final Report of IGCP Project 259), Paris, France, UNESCO, pp 122

Davies BE (1980) Base metal mining and heavy metal contamination of agricultural land in England and Wales. In: *Inorganic Pollution and Agriculture* (MAFF Reference Book 325), London, UK, HMSO, pp 142–158

Davies BE (1983) A graphical estimation of the normal lead content of some British soils. *Geoderma, 29*, 67–75

Davies DJA (1987) An assessment of the exposure of young children to lead in the home environment. In: Thornton I & Culbard E, eds, *Lead in the Home Environment*, London, UK, Science Reviews Ltd., pp 189–196

Davies DJA & Thornton I (1987) The influence of house age on lead levels in dusts and soils in Brighton, England. *Environ Geochem Health, 9*, 65–67

Davies BA & Thornton I (1989) *Environmental Pathways of Lead into Food: A Review*, International Lead Zinc Research Organisation, Research Triangle Park, North Carolina, USA

Delves HT, Diaper SJ, Oppert S, Prescott-Clarke P, Periam J, Dong W, Colhoun H & Gompertz D. (1996) Blood lead concentrations in United Kingdom have fallen substantially since 1984. *Brit Med J, 313,* 883–884

ECE (1994) *State-of-the-art report of Task Force on Heavy Metal Emissions*, Brussels, Belgium, Economic Commission for Europe

EPA (1986) *Air Quality Criteria for Lead* (EPA-600/8-83/028df), Research Triangle Park NC, USA, US Environmental Protection Agency

FAO/WHO (1987*) Toxicological Evaluation of Certain Food Additives and Contaminants* (WHO Food Additives Series 21), Cambridge, UK, Cambridge University Press

Holmgren GGS, Meyer MW, Daniels RB, Kubota J & Chaney RL (1983) Cadmium, lead, zinc, copper and nickel in agricultural soils of the United States. *Agronomy Abstracts*, 33

House of Lords, Select Committee on the European Communities (1996) *Drinking Water* (Session 1995–96, 4th Report)

Hutton M & Symon C (1986) The quantities of cadmium, lead, mercury and arsenic entering the UK from human activities. *Sci Tot Environ, 57*, 129–150

Kelly J, Thornton I & Simpson PR (1996) Urban geochemistry: a study of the influence of anthropogenic activity on the heavy metal content of soils in traditionally industrial and non-industrial areas of Britain. *Appl Geochem, 11*, 363–370

Maskall J, Whitehead K, Gee C & Thornton I (1996) Long-term migration of metals at historical smelting sites. *Appl Geochem, 11*, 43–51

McGrath SP & Loveland PJ (1992) *The Soil Geochemical Atlas of England and Wales*, London, UK, Blackie Academic

MAFF (1972) *Survey of Lead in Food* (MAFF Working Party on the Monitoring of Foodstuffs for Heavy Metals, 2nd Report), London, UK, HMSO

MAFF (1982) *Survey of Lead in Food* (2nd Supplementary Report), London, UK, HMSO

MAFF (1989) *Lead in Food Progress* Report (Food Surveillance paper No. 27), London, UK, HMSO

Moir AM (1992) *The Influence of Soil Factors and Atmospheric Deposition on the Cadmium and Lead Contents of Vegetables,* PhD thesis, University of London, UK

Moore MR, Hughes MA & Goldberg DJ (1979) Lead absorption in man from dietary sources. The effect of cooking upon lead concentrations of certain foods and beverages. *Arch Occup Environ Health, 44*, 81–90

Nriagu JO (1989) A global assessment of natural sources of atmospheric trace metals. *Nature, 338*, 47–49

Nriagu JO & Pacyna, JM (1988) Quantitative assessment of worldwide contamination of air, water and soils by trace metals. *Nature, 333*, 134–139

Plant JA, Hale M & Ridgeway J (1989) Regional geochemistry based on stream sediment sampling. In: Garland GD, ed., *Proceedings of Exploration '87* (Vol. 3), Ontario Geological Survey, pp 384–404

Rasmussen PA (1996) *Trace Metals in the Environment: A Geological Perspective*, Geological Survey of Canada, *Bulletin 492*

Royal Commission on Environmental Pollution (1983) *Lead in the Environment* (Royal Commission on Environmental Pollution, Ninth Report), London, UK, HMSO

Royal Commission on Environmental Pollution (1993) *Incineration of Waste* (Royal Commission on Environmental Pollution, Seventeenth Report), London, UK, HMSO

Ryu JE, Zielger EE, Nelson SE & Foron SJ (1983) Dietary intake of lead and blood lead concentration in early infancy. *Am J Dis Child, 137*, 886–891

Smart GA, Warrington M & Evans WH (1981) The contribution of lead in water to dietary lead intakes. *J Sci Food Agricult*, *32*, 129–133

Swaine DJ (1978) Lead in the environment. *J Proc Royal Soc New South Wales, 111*, 41–47

Thornton, I (1980) *Geochemical Aspects of Heavy Metal Pollution and Agriculture in England and Wales*, Inorganic Pollution and Agriculture, London, UK, HMSO, pp 105–125

Thornton I & Culbard E (1985) Metals in urban dusts and soils. *Environ Tech Lett, 6*, 137–144

Thornton I, Davies DJA, Watt JM & Quinn MJ (1990) Lead exposure in young children from dust and soil in the United Kingdom. *Environ Health Perspect, 89*, 55–60

Thornton, I (1996) Impacts of mining on the environment; some local, regional and global issues. *Appl Geochem, 11*, 355–361

Wang Y, Thornton I & Farago M (1997) Changes in lead concentrations in the home environment in Birmingham, England, over the period 1984 to 1996. *Sci Tot Environ, 207,* 149–156

Webb JS, Thornton I, Thomson M, Howarth RJ & Lowenstein PL (1978) *The Wolfson Geochemical Atlas of England and Wales,* Oxford, UK, Oxford University Press

Williams ML (1990) The role of motor vehicles in air pollution in the UK. *Sci Tot Environ, 93*, 1–8

2.6 THE RELATIONSHIP BETWEEN BLOOD LEAD AND BLOOD PRESSURE IN THE ENGLISH POPULATION

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2.6.1 INTRODUCTION

Studies on the relationship between blood lead and blood pressure have shown conflicting results (see Section 2.7). While some studies have found no significant association, others have found a variable increase in blood pressure with increasing blood lead. Studies carried out on subgroups of the British population have likewise found contradictory results (Beevers *et al.,* 1976; Pocock *et al.,* 1984; Staessen *et al.,* 1990), but no such study has been conducted among the English population as a whole. We have studied the influence of blood lead on blood pressure in a representative sample of the adult population of England, using a subsample of the 1995 Health Survey for England.

2.6.2 DATA AND METHODS

THE 1995 HEALTH SURVEY FOR ENGLAND AND THE MEASUREMENT OF BLOOD PRESSURE AND BLOOD LEAD

The 1995 Health Survey for England was a cross-sectional survey of a representative sample of the population living in private households in England, and was commissioned by the DH to monitor trends in the nation's health. The sampling method, including subsampling for blood lead, is described earlier (see Section 2.1). Methods of measurement of blood lead and blood pressure are detailed in a report to the DoE*.

Data were collected in two visits: an interviewer's visit (which included height and weight measurements), followed by a visit from a nurse (who measured blood pressure, took a sample of blood, performed a number of anthropometric measurements and obtained data on current medication). Blood pressure was measured using an automated device, the Dinamap 8100 monitor. Using an appropriately sized cuff, three blood pressure readings were taken on the right arm with the person in a seated position, after five minutes' rest. The data used in this study are based on the mean of the second and third measurements made on participants for whom three recordings were completed.

Following written consent from eligible participants, a 2 ml blood sample was collected from 6517 adults and 340 children into an EDTA (ethylene diamine tetra-acetic acid) tube for lead analysis. (The results presented here only refer to adults.) The blood lead level was measured by atomic absorption spectrometry using microsampling flame atomisation.

STATISTICAL METHODS AND VARIABLES USED

To examine the relationship between blood pressure and blood lead while controlling for various factors associated with blood pressure, stepwise linear and logistic regression analyses were carried out separately for men and women.

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* *Survey of Blood Lead Levels in the General Population in England*, Report to DoE

The outcome or dependent variables in the linear regression were the continuous variables, systolic and diastolic blood pressure, while that for the logistic regression was the odds of being hypertensive over being normotensive. Two definitions of hypertension were used, the first being the classical definition of having a systolic blood pressure ≥160 mm Hg, or diastolic blood pressure ≥95 mm Hg, or being on antihypertensive medication (hypertension I). The second more stringent, contemporary definition was a systolic blood pressure ≥140 mm Hg, or diastolic blood pressure ≥90 mm Hg, or being on antihypertensive medication (hypertension II).

The explanatory or independent variables used in both methods were blood lead, age, body mass index (BMI) defined as weight(kg)/height^2(m^2), smoking status (three categories: non-current smokers; <20 cigarettes per day; ≥20 per day), region of residence (north, midlands, south), social class, and alcohol consumption (non/ex/occasional drinkers; men drinking 21 units or less per week or women drinking 14 units or less per week; heavy drinkers, consuming over 21 units or 14 units per week, respectively).

Social class, in the 1995 survey as well as in previous Health Survey reports, is defined on the basis of the occupation of the head of household*. In this study the classes were further grouped into manual (skilled manual, partly skilled and unskilled occupations) and non-manual (professional, managerial and technical, and skilled non-manual occupations).

In stepwise regression each of the independent variables is added or removed according to a set of criteria (usually a significance level of 0.05 or less for addition, and 0.10 or more for removal of a variable), until a model is reached in which no more variables are eligible for entry or removal.

Systolic blood pressure (but not diastolic blood pressure), blood lead and BMI were log-transformed (to base 10) to obtain residuals with approximately normal distributions and equal variances for the linear regression analyses. Geometric mean, calculated by taking the antilogarithm of the mean of the logged original values, was used as the summary measure for these variables.

* The head of household owns or rents the property, or is a man married to or cohabiting with a women who is the owner/renter of a property. Heads of households who were in the armed forces, whose occupation was not adequately described or who were full-time students were not allocated a social class. The social class of those who were not employed at the time of the interview was based on previous employment.

SAMPLE SIZES

Altogether, 2563 men and 2763 women were included in these analyses. These comprise 82% of the 3119 men and 81% of the 3398 women aged 16 and over for whom blood lead measurements were made. The 556 men and 635 women who were excluded consisted of those who were not eligible for blood pressure and BMI measurements, those who refused to have their measurements taken, or those who did not have valid data.

Pregnant women were excluded since they were eligible neither for blood pressure to be measured nor for a blood sample to be taken. Respondents who had eaten, drunk alcohol or smoked in the half hour prior to measurement and those who did not have all three readings were considered not to have valid blood pressure measurements. Respondents who were chairbound or unsteady on their feet were not measured for height and weight.

Two types of additional analyses were carried out for reasons that are discussed in the final section of this paper: one which did not control for alcohol consumption and another which included only those not on antihypertensive medication (2254 men and 2398 women).

Only findings from the analyses that included all respondents (including those on treatment) and controlled for alcohol consumption (in addition to blood lead, age, BMI, region, social class, smoking status) are discussed in detail here. Findings from the additional analyses are shown in a report to DoE* and are summarised under 'other analyses', below.

2.6.3 RESULTS

CHARACTERISTICS OF THE STUDY GROUPS

The men and women in the study were similar in terms of age, smoking status, social class, and region of residence (Table 2.6.1). Around three quarters of the respondents were not current smokers, about half were in manual social classes

**Survey of Blood Lead Levels in the General Population in England*, Report to DoE

Table 2.6.1 Characteristics of the men and women in the study

Characteristic	Men	Women
Number	2563	2763
Mean age (years)	*47.5*	*47.7*
SE age	*0.34*	*0.33*
Geometric mean BMI	*26.1*	*25.6*
Smoking status (%)		
Non smokers	75	78
Smoked less than 20 cigarettes a day	15	16
Smoked 20 cigarettes or more a day	10	6
Social class (%)		
Manual classes	50	46
Non-manual classes	50	54
Residence (%)		
North	26	26
Midlands	21	20
South	53	54
Alcohol consumption (%)		
Non/ex/occasional drinkers	12	30
≤21 units per week for men; ≤14 units for women	58	56
>21 units per week for men; >14 units for women	29	14
Hypertensive status (%)		
On antihypertensive medication	12	13
Hypertensive I[a]	23	21
Hypertensive II[b]	46	37
Blood pressure (mm Hg)		
Geometric mean systolic blood pressure	*138.0*	*133.0*
Mean diastolic blood pressure	*77.8*	*73.2*
SE diastolic blood pressure	*0.24*	*0.23*
Blood lead concentration (µg/dl)		
Geometric mean	*3.7*	*2.6*

BMI, body mass index

[a] ≥160 mm Hg systolic blood pressure, ≥95 mm Hg diastolic blood pressure, or on antihypertensive medication
[b] ≥140 mm Hg systolic blood pressure, ≥90 mm Hg diastolic blood pressure, or on antihypertensive medication

and more than half resided in the south. The geometric mean blood lead was higher in men (3.7 μg/dl) than in women by 1.1 μg/dl; men were more likely to be regular or heavy drinkers than women. Mean blood pressures were higher among men than women, and more men were classified as hypertensive, particularly when we used the lower threshold definition (hypertension II). The proportions of those on antihypertensive medication, however, differed little between the sexes.

BLOOD LEAD AND BLOOD PRESSURE

Mean systolic and diastolic blood pressures and the proportion of hypertensives generally increased with increasing blood lead in both sexes (Figure 2.6.1, Table 2.6.2). The associations of blood lead with systolic and diastolic blood pressure for both sexes were all highly significant ($p<0.01$), although the correlation coefficients were small: 0.113 for men and 0.186 for women for systolic blood pressure, and 0.153 and 0.143, respectively, for diastolic blood pressure.

After controlling for the effects of the other variables (age, BMI, smoking status, social class, alcohol consumption), a significant positive association with blood lead was found only for diastolic blood pressure, and only in men ($p<0.01$), not in women ($p = 0.10$), as shown in Table 2.6.3. In addition, blood lead (and heavy drinking), as indicated by the standardised partial regression coefficient, proved less important than age or BMI.

Table 2.6.4 shows the estimated decreases in diastolic blood pressure with decreases in blood lead among men. A blood lead level of 10 μg/dl was used as one of the reference points, as it is often regarded as the cut off point for high blood lead and is the action level recommended by the Centers for Disease Control (CDC, 1991).

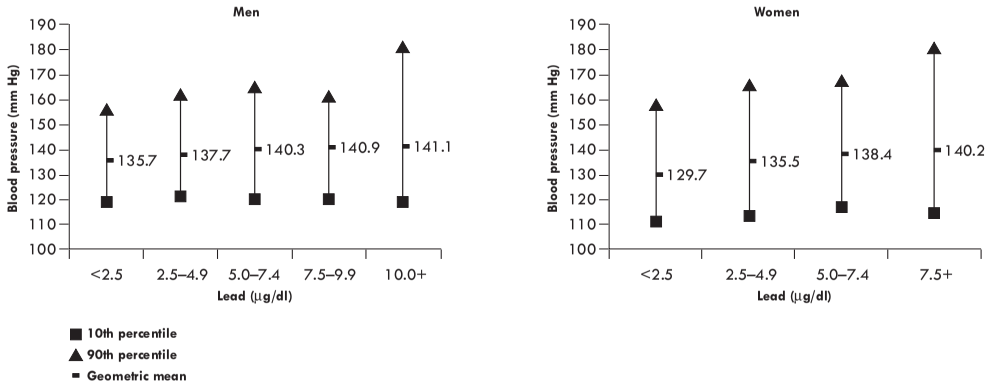
HYPERTENSION AND BLOOD LEAD

Geometric mean blood lead was higher among hypertensives (both classifications I and II) than normotensives for each sex by about 0.5 μg/dl (Figure 2.6.2, Table 2.6.5). Concomitantly, the proportion of men and women with high concentrations of blood lead was higher among hypertensives.

Blood lead was significantly associated with hypertension status II in men ($p = 0.01$; Table 2.6.6) and I in women ($p = 0.01$). Using classification II in men,

Figure 2.6.1 Systolic and diastolic blood pressure by blood lead level and sex

Systolic blood pressure



Diastolic blood pressure

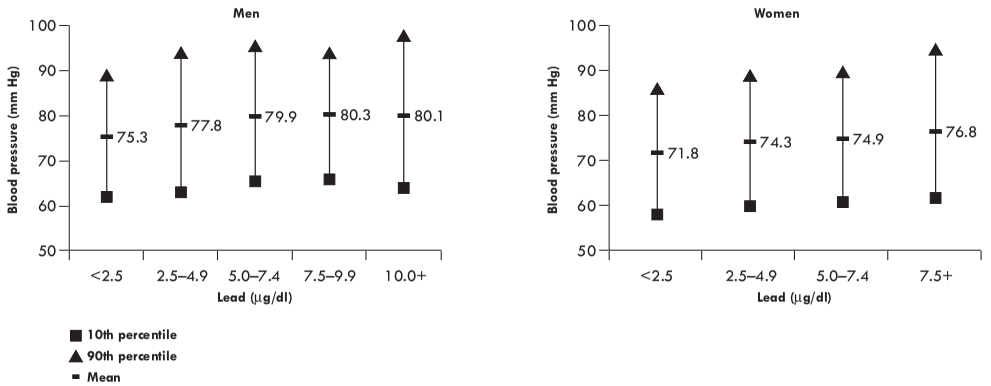


Table 2.6.2 Systolic and diastolic blood pressure* (in mm Hg) by blood lead level and sex

Men	Blood lead level (μg/dl)					Total
	<2.5	**2.5–4.9**	**5.0–7.4**	**7.5–9.9**	**≥10.0**	
Systolic blood pressure						
Percentage with pressure						
<140	67	62	54	46	51	60
140–160	26	27	30	40	33	28
>160	8	11	16	13	16	12
Mean pressure (mm Hg)	*136.5*	*138.8*	*141.5*	*142.0*	*142.7*	*139.1*
SE	*0.61*	*0.53*	*0.92*	*1.33*	*1.91*	*0.35*
Geometric mean	*135.7*	*137.7*	*140.3*	*140.9*	*141.1*	*138.0*
Diastolic blood pressure						
Percentage with pressure						
<90	90	83	81	77	80	84
90–95	4	7	8	13	8	7
≥95	6	9	11	10	12	9
Mean pressure (mm Hg)	*75.3*	*77.8*	*79.9*	*80.3*	*80.1*	*77.8*
SE	*0.44*	*0.37*	*0.59*	*0.91*	*1.22*	*0.24*

Women	Blood lead level (μg/dl)				Total
	<2.5	**2.5–4.9**	**5.0–7.4**	**≥7.5**	
Systolic blood pressure					
Percentage with pressure <140	75	63	55	50	68
140–160	16	22	29	27	20
≥160	9	15	15	23	12
Mean pressure (mm Hg)	*131.2*	*137.1*	*139.8*	*142.3*	*134.6*
SE	*0.54*	*0.70*	*1.32*	*2.51*	*0.41*
Geometric mean	*129.8*	*135.5*	*138.4*	*140.2*	*133.0*
Diastolic blood pressure					
Percentage with pressure					
<90	93	91	90	83	92
90–95	4	4	5	5	4
≥95	3	5	5	13	5
Mean pressure (mm Hg)	*71.8*	*74.3*	*74.9*	*76.8*	*73.2*
SE	*0.31*	*0.37*	*0.72*	*1.29*	*0.23*

* Diastolic blood pressure, unlike systolic blood pressure, showed a normal distribution so the geometric mean is not an appropriate average measure

Table 2.6.2 Study population by blood lead concentration and sex

Sex	Blood lead level (µg/dl) <2.5	2.5–4.9	5.0–7.4	7.5–9.9	>10.0	Total
Men	694	1117	440	181	131	2563
Women	1388	1022	249	104	*	2763

*These figures are included in 7.5–10.0 µg/dl due to the small number of subjects in this category

Table 2.6.3 Summary of the stepwise linear regression models[a]

Log_{10} (systolic blood pressure)	Standardised coefficient	Level of significance	Diastolic blood pressure	Standardised coefficient	Level of significance
Men					
Number = 2563					
R^2 = 0.207			R^2 = 0.209		
Age	0.39	<0.001	Age	0.37	<0.001
Log_{10} (BMI)	0.16	<0.001	Log_{10} (BMI)	0.18	<0.001
Alcohol >21 units/week	0.06	<0.001	Log_{10} (lead)	0.06	<0.001
Manual worker	0.04	0.021	Alcohol >21 units/week	0.06	<0.001
Log_{10} (lead)	[b]	0.460			
Women					
Number = 2763					
R^2 = 0.405			R^2 = 0.172		
Age	0.56	<0.001	Age	0.36	<0.001
Log_{10} (BMI)	0.20	<0.001	Log_{10} (BMI)	0.14	<0.001
Residing in north	0.04	0.010	Alcohol >14 units/week	0.04	0.022
Log_{10} (lead)	[b]	0.460	Log_{10} (lead)	[b]	0.099

[a] Only variables that showed significance in one or more models are reported

[b] Not estimated because not significant

Table 2.6.4 Estimated decreases in diastolic blood pressure with decreases in blood lead among men

Decrease in blood lead (μg/dl)		Decrease in diastolic blood pressure (mm Hg)
From	To	
10.0[a]	1.2[b]	2.6
10.0	5.0	0.9
3.1[c]	2.1	0.5

[a] Often regarded as the cut off point for high blood lead
[b] The combined 5th percentile for men and women in the study
[c] The combined geometric mean for men and women in the study

those in the highest lead group (7.5 μg/dl or more) were 1.7 times more likely to be hypertensive than those in the lowest group (less than 2.5 μg/dl; $p<0.01$). However, those in the intermediate lead groups were not significantly more likely to be hypertensive than those in the lowest group. In addition, the partial contribution of lead to the regression model was relatively small (0.04) compared with age (0.31) and BMI (0.15).

OTHER ANALYSES

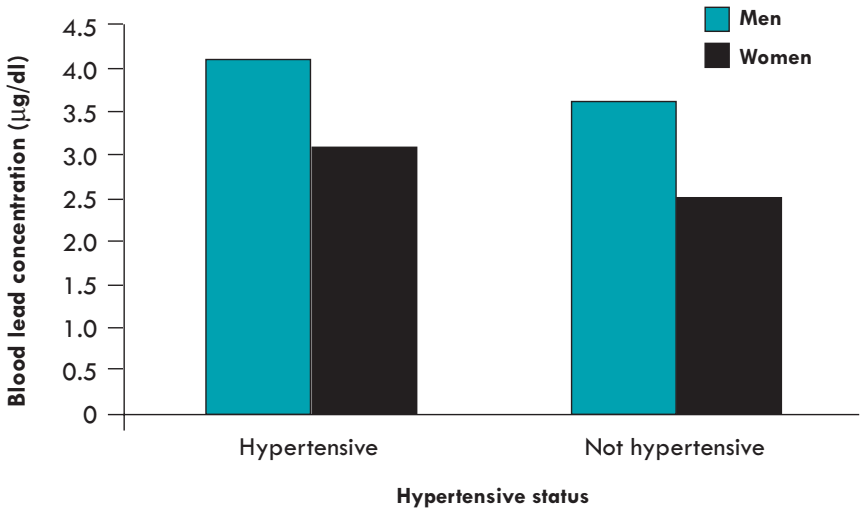
Table 2.6.7 summarises the findings of the various analyses, including the above, to examine the association between blood lead and blood pressure, and between blood lead and hypertension. Blood lead was significantly (positively) associated mainly with diastolic blood pressure in men and hypertension status II in men and I in women. For all the significant associations, the relative importance of blood lead was low compared with the other significant variables, particularly age and BMI, which are the main determinants of blood pressure in populations in developed countries.

In the analyses that included those on antihypertensive medication, controlling for alcohol consumption weakened the relationship between blood lead and diastolic blood pressure for women, from statistically significant ($p = 0.04$) to marginally significant ($p = 0.10$). It did not, however, change the associations between blood lead and hypertension in either sex, in that a significant association was found only for hypertension status II in men and I in women.

In contrast, in the analyses that excluded those on treatment, only diastolic blood pressure in men was significantly associated with blood lead, whether alcohol consumption was controlled for or not.

Figure 2.6.2 Hypertension status by geometric mean blood lead and sex

Hypertension I



Hypertension II

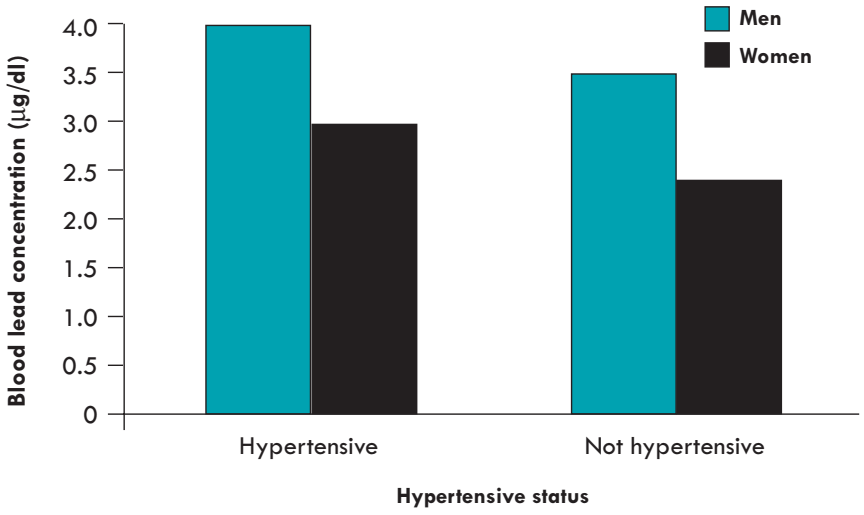


Table 2.6.5 Hypertension status by blood lead (μg/dl) and sex

	Hypertension status I		Hypertension status II		
	Normotensive	Hypertensive	Normotensive	Hypertensive	Total
Men					
Percentage with blood lead					
<2.5 μg/dl	29	19	31	23	27
2.5–4.9 μg/dl	43	44	44	43	44
5.0–7.4 μg/dl	16	22	15	19	17
≥7.5 μg/dl	12	15	9	15	12
Mean blood lead (μg/dl)	*4.3*	*4.9*	*4.2*	*4.8*	*4.5*
SE	*0.07*	*0.14*	*0.09*	*0.10*	*0.06*
Geometric mean	*3.6*	*4.1*	*3.5*	*4.0*	*3.7*
Women					
Percentage with blood lead					
<2.5 μg/dl	54	39	57	40	50
2.5–4.9 μg/dl	35	43	34	42	37
5.0–7.4 μg/dl	8	11	7	12	9
≥7.5 μg/dl	3	7	2	6	4
Mean blood lead (μg/dl)	*2.9*	*3.7*	*2.8*	*3.6*	*3.1*
SE	*0.05*	*0.10*	*0.05*	*0.07*	*0.04*
Geometric mean	*2.5*	*3.1*	*2.4*	*3.0*	*2.6*

Table 2.6.5 Study population by hypertension status and sex

	Hypertension status I		Hypertension status II		
	Normotensive	Hypertensive	Normotensive	Hypertensive	Total
Men	1984	579	1397	1166	2563
Women	2172	591	1744	1019	2763

Table 2.6.6 Summary of the stepwise logistic regression models

Hypertensive I/ Normotensive				Hypertensive II/ Normotensive			
	Partial correlation	Odds ratio	Level of significance		Partial correlation	Odds ratio	Level of significance
Men				**Men**			
Number=2563				*Number=2563*			
Age	**0.39**		**<0.001**	**Age**	**0.31**		**<0.001**
16–44[a]		1.0		16–44[a]		1.0	
45–54	0.16	4.5	<0.001	45–54	0.09	2.0	<0.001
55–64	0.26	10.4	<0.001	55–64	0.18	4.1	<0.001
65–74	0.32	18.4	<0.001	65–74	0.24	8.6	<0.001
75+	0.33	33.3	<0.001	75+	0.21	18.6	<0.001
BMI	**0.13**		**<0.001**	**BMI**	**0.15**		**<0.001**
≤20[a]		1.0		≤20[a]		1.0	
20–25	0.00	0.8	0.500	20–25	0.00	1.4	0.268
25–30	0.00	1.2	0.627	25–30	0.04	2.4	0.003
>30	0.03	2.4	0.024	>30	0.08	5.0	<0.001
Lead level	[b]		**0.212**	**Lead level**	**0.04**		**0.011**
<2.5[a]		1.0		<2.5[a]		1.0	
2.5–4.9	[b]	[b]	0.873	2.5–4.9	0.00	1.1	0.503
5.0–7.4	[b]	[b]	0.202	5.0–7.4	0.00	1.2	0.221
≥7.5	[b]	[b]	0.359	≥7.5	0.05	1.7	0.002
				Alcohol consumption			
					0.03		**0.035**
				Non/ex/occasional[a]		1.0	
				≤21units	-0.03	0.7	0.026
				>21units	0.00	0.9	0.357
Women				**Women**			
Number=2763				*Number=2763*			
Age	**0.41**		**<0.001**	**Age**	**0.43**		**<0.001**
16–44[a]		1.0		16–44[a]		1.0	
45–54	0.15	5.6	<0.001	45–54	0.18	4.3	<0.001
55–64	0.24	15.2	<0.001	55–64	0.29	12.2	<0.001
65–74	0.33	39.0	<0.001	65–74	0.35	28.1	<0.001
75+	0.35	88.4	<0.001	75+	0.29	58.9	<0.001
BMI	**0.12**		**<0.001**	**BMI**	**0.17**		**<0.001**
≤20[a]		1.0		≤20[a]		1.0	
20–25	0.00	1.0	0.500	20–25	0.00	1.3	0.378
25–30	0.00	1.5	0.627	25–30	0.05	2.2	0.002
>30	0.06	3.1	0.024	>30	0.10	5.0	<0.001
Lead level	**0.04**		**0.013**	**Lead level**	[b]		**0.079**
<2.5[a]		1.0		<2.5[a]		1.0	
2.5–4.9	0.00	1.0	0.822	2.5–4.9	[b]	[b]	0.987
5.0–7.4	-0.02	0.7	0.089	5.0–7.4	[b]	[b]	0.893
≥7.5	0.04	1.9	0.014	≥7.5	[b]	[b]	0.012

[a] Reference category

[b] Not estimated because not significant

Table 2.6.7 Summary of findings: blood lead, blood pressure and hypertension

Population and factors controlled for	Dependent variable	Significance of association with blood lead	
		Men	Women
All respondents including those on anti-hypertensive medication			
All factors* except alcohol consumption	SBP	0.14	0.46
	DBP	<0.01	0.04
All factors*	SBP	0.46	0.46
	DBP	<0.01	0.10
All factors* except alcohol consumption	HTI	0.21	0.01
	HTII	0.01	0.08
All factors*	HTI	0.21	0.01
	HTII	0.01	0.08
Respondents not on anti-hypertensive medication			
All factors* except alcohol consumption	SBP	0.08	0.13
	DBP	<0.01	0.08
All factors*	SBP	0.24	0.13
	DBP	<0.01	0.16

DBP, diastolic blood pressure; HTI, hypertension status I; HTII, hypertension status II; SBP, systolic blood pressure

* Age, body mass index, smoking status, region, social class, alcohol consumption, and blood lead

2.6.4 DISCUSSION

Various analyses were carried out in this study to address controversial issues in the relationship between blood lead and blood pressure. First of these issues is alcohol consumption (Hertz-Picciotto & Croft, 1993; Schwartz, 1995). The assumption behind the use of regression analysis to 'control' for the effects of certain variables is that these variables are simultaneously associated with the outcome and that they are independent predictors of the outcome, acting on a separate biological pathway. Whether alcohol meets these criteria is not clear cut.

First, alcoholic beverages, particularly wine, may actually contain significant amounts of lead (Rhainds & Levallois, 1997). Thus when alcohol consumption is high compared with other possible sources of lead, blood lead may only be an indicator of alcohol intake and the effect on blood pressure may actually be due to alcohol consumption. Second, alcohol may influence absorption, metabolism

in the liver, or secretion via the kidney; thus the assumption of separate pathways may be violated (Hertz-Picciotto & Croft, 1993).

Some earlier studies (see Section 2.7) did not control for alcohol consumption. However, most studies controlled for it as its relationship with lead is still not clear while its association with blood pressure has been established. In other studies (Wolf *et al.,* 1995), including this one, rather than the 'either-or' approach, both analyses were conducted, one controlling and another not controlling for alcohol intake.

Another controversial issue is whether to include respondents on antihypertensive medication, as these drugs lower blood pressure. Some studies have excluded those on treatment (Menditto *et al.,* 1994), but most studies have included individuals on treatment as they, on average, still have higher blood pressure than normotensives. In still other studies (Harlan *et al.,* 1985), including this one, two analyses have been made, one including and the other excluding, individuals on antihypertensive medication.

The present findings of a positive relationship between blood lead and blood pressure are consistent with the results of several cross-sectional and longitudinal studies on human subjects and with controlled experimental studies in animals. The relationship is, however, weak and is not consistent between the two measures of blood pressure and between the sexes.

Diastolic blood pressure consistently showed a significant association with blood lead in men, regardless of whether alcohol intake was controlled for or whether respondents on antihypertensive medication were excluded. On the other hand, systolic blood pressure showed no significant association in the various analyses for either sex. Other studies where the two blood pressure measurements were analysed, such as those of subgroups of the population in Austria (Wolf *et al.,* 1995) and Denmark (Kirkby & Gyntelberg, 1985), also found significant association only with diastolic blood pressure.

The association between blood lead and diastolic blood pressure was consistently found to be significant only in men, not in women. Controlling for alcohol consumption made the association with diastolic blood pressure for women non significant. A study using the second National Health and Nutrition Examination Survey in the USA, which stratified respondents according to blood pressure status and age, also found a positive relationship only in men (Harlan *et al.,* 1985). In contrast, the present study also found an association between blood lead and hypertension II in men and I in women, whether or not alcohol intake was controlled for.

The inconsistencies in the findings and the weakness of the relationship should not diminish the importance of minimising the public's exposure to lead. The possibility of a small direct effect of blood lead on blood pressure is suggested by this and several previous studies. Moreover, lead is toxic in large doses and has no known health benefit.

REFERENCES

Beevers DG, Erskine E & Robertson M (1976) Blood lead and hypertension. *Lancet, 2,* 1–3

CDC (1991) *Preventing Lead Poisoning in Young Children:* A Statement by the Centers for Disease Control, Atlanta GA, USA, US Department of Health and Human Services

Harlan WR, Landis JR, Schmouder RL, Goldstein NG & Harlan LC (1985) Blood lead and blood pressure: relationship in the adolescent and adult US population. *J Am Med Assoc, 253,* 530–534

Hertz-Picciotto I & Croft J (1993) Review of the relationship between blood lead and blood pressure. *Epidemiol Rev*, *15,* 352–373

Kirkby H & Gyntelberg F (1985) Blood pressure and cardiovascular risk factors of long-term exposure to lead. *Scand J Work Environ Health, 11,* 15–19

Menditto A, Morisi G, Spagnolo A, Menotti A, Cigna G, Giampaoli S, Lanti M, Mariotti S, Meli P, Scafoto E, Scagna M, Chessa A, Dicarlo G, Dima F, Farchi F, Giuli B, Pasquali M, Parriachi V, Santaquilani A & Seccareccia F (1994) Association of blood lead and blood pressure on men aged 55 to 75 years: effect of selected social and biochemical confounders. *Environ Health Perspect, 102,* 107–111

Pocock SJ, Shaper AJ, Ashby D, Delves T & Whitehead TP (1984) Blood lead concentration, blood pressure, and renal function. *Brit Med J*, 289, 872–897

Rhainds M & Levallois P (1997) Effects of maternal cigarette smoking and alcohol consumption on blood lead levels of newborns. *Am J Epidemiol, 145,* 250–257

Schwartz J (1995) Lead blood pressure, and cardiovascular disease in men. *Arch Environ Health, 50,* 31–37

Staessen J, Yeoman WB, Fletcher A, Markowe H, Marmot M, Rose G, Semmence A, Shipley M & Bulpitt CJ (1990) Blood lead concentration, renal function, and blood pressure in London civil servants. *Brit J Ind Med, 47,* 442–447

Wolf C, Wallnöfer A, Waldhör T, Vutuc C, Meisenger V & Rüdiger H (1995) Effect of blood lead on blood pressure in occupationally nonexposed men. *Am J Ind Med, 27,* 897–903

2.7 OVERVIEW OF OTHER STUDIES OF BLOOD LEAD AND HYPERTENSION

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2.7.1 BACKGROUND

A link between high level lead exposure and adverse effects on the cardiovascular and renal systems was recognised in the nineteenth century (Tanquerel des Planches, 1839; Batuman *et al.,* 1983). More recently the possibility that long-term low-level lead exposure may induce small but potentially important increases in blood pressure has been the subject of much investigation, debate and controversy.

In 1988 a consensus meeting (Victery *et al.,* 1988) concluded that the weight of evidence established an association between lead exposure, usually measured as blood lead level, and increased blood pressure, a view which has been supported by a more recent National Academy of Sciences panel in the USA (National Research Council, 1993). A comprehensive review of relevant studies published between 1980 and 1992 (Hertz-Picciotto & Croft, 1993) and a subsequent meta-analysis (Schwartz, 1995) also concluded that, although the data were not definitive, the association was likely to be causal. However, the author of an earlier meta-analysis which reviewed 23 studies (Staessen *et al.,* 1994), many of them included in the later meta-analysis (Schwartz, 1995), felt that the data did not support the hypothesis of causation.

MEASURING BLOOD PRESSURE AND BLOOD LEAD: PRACTICAL DIFFICULTIES

Blood pressure is subject to extensive biological variability which may be compounded by observational errors. As previously described in detail (Hertz-Picciotto & Croft, 1993), repeated measurements and cuffs of correct size have too rarely been used in studies. If small differences in blood pressure are under investigation, sub-optimal blood pressure measurement is likely to reduce the power of studies and thereby generate inconsistent results. The treatment of subjects considered to be hypertensive has in general been less than ideal. Hypertensives have either been excluded, which reduces study power but is not likely to create much bias, or included but without logical consideration of the impact of therapy on blood pressure. For dichotomous comparisons of 'hypertensives' with 'normotensives', the former category should include those with blood pressure above a specified (albeit arbitrary) cut-off point plus those on treatment for hypertension. With one exception (Hu *et al.,* 1996), this has not been the case in any such comparisons. When blood pressure is to be investigated as a continuous variable, the inclusion of 'hypertensives' on treatment will bias any association between lead and blood pressure towards the null, unless some suitable adjustment of blood pressure is made for those on treatment. No such approach has been used in studies to date. The investigation of lead levels among those on treatment for hypertension is further complicated by the effects of some commonly used antihypertensive agents on blood lead levels (Staessen *et al.,* 1996).

The measurement of blood lead levels has been considered to be a reasonable surrogate for exposure and, given the consistent use of atomic absorption spectrometry, has been described as a strength of these investigations (Hertz-Picciotto & Croft, 1993). More recently, however, it has been suggested that the most accurate marker of chronic internal lead dose biologically relevant to hypertension is not blood, but bone concentration (Hu *et al.,* 1996).

Finally, appropriate handling of blood lead levels in analyses has been controversial. Given the skewed distribution of blood lead levels in most studies, they have usually been log transformed in analyses. But the use of this approach has not been universal (Neri *et al.,* 1988), and some authors have questioned whether it is appropriate (Sharp *et al.,* 1988).

2.7.2 BLOOD LEAD AND BLOOD PRESSURE: THE ASSOCIATION

Before a judgement on causation can be made, the following aspects of this association should be considered: strength; dose–response; independence; consistency; coherence and biological plausibility; and time sequence.

STRENGTH

In a recent review (Hertz-Picciotto & Croft, 1993), and two meta-analyses (Staessen *et al.,* 1994; Schwartz, 1995), the impact of blood lead on blood pressure was found on average to be small, but positive and statistically significant.

Of eight population based studies reported between 1980 and 1992 (Pocock *et al.,* 1984; Harlan *et al.,* 1985; Pirkle *et al.,* 1985; Elwood *et al.,* 1988; Neri *et al.,* 1988; Grandjean *et al.,* 1989; Staessen *et al.,* 1991), seven of the studies showed higher blood pressures associated with higher lead levels, although in only three studies was the result significant. A difference in blood lead levels from below 12 μg/dl to above 25 μg/dl was associated with a blood pressure difference of 1.4 to 8.0 mm Hg systolic and 1.2 to 4.0 mm Hg diastolic.

In an overview of six occupational cohorts (Kirkby & Gyntelberg, 1985; Orssaud *et al.,* 1985; Weiss *et al.,* 1986; Parkinson *et al.,* 1987; Neri *et al.*, 1988; Sharp *et al.,* 1988), five reported variably higher levels of systolic blood pressure associated with higher blood lead levels. These differences ranged between 1 and 8 mm Hg. For five of the six occupational cohorts the effects of lead on diastolic pressure were reported, and for four of these five a positive association was found, with three studies showing a larger effect on diastolic than on systolic pressure.

In Staessen's meta-analysis of 23 studies (Staessen *et al.,* 1994), a doubling of blood lead level was associated with an increase of 1 mm Hg in systolic pressure ($p = 0.002$) and an increase of 0.6 mm Hg in diastolic pressure ($p = 0.02$). In a subsequent meta-analysis (Schwartz, 1995) of 15 studies which focused on blood lead and systolic pressure among men, a doubling of blood lead from 5 to 10 μg/dl was associated with an increase of 1.25 mm Hg in systolic pressure with a range of 0.25 to 3.17 mm Hg. It is interesting that although similar effects of blood lead

were observed on systolic pressure in the two meta-analyses, Staessen concluded that such effects were inconsistent and unlikely to be causally related and had no public health implications, whereas Schwartz felt that the association demonstrated in his meta-analysis, when put in the context of other coherent data, "should be considered causal" (Schwartz, 1995).

In 1996 Staessen published the results of a small prospective study (Staessen *et al.,* 1996) which took place in Belgium during a five year period when mean blood lead levels fell by 32%. The authors summarised the results as showing no consistent association between blood lead and blood pressure, which was carefully measured 15 times on each subject under standardised conditions and by 24 hour ambulatory machines. However, after full adjustment of these data, 24 hour and conventional diastolic pressures in women were significantly associated with blood lead ($p = 0.05$ and $p = 0.009$ respectively).

Three other cohort studies have also shown a significant association between blood pressure and blood lead, although the effect was greatly attenuated after controlling for covariates in one of the studies (Grandjean *et al.,* 1989), was associated with systolic and not diastolic pressure in another (Weiss *et al.,* 1986) and was more closely associated with diastolic in the third (Neri *et al.,* 1988).

Although these data are inconsistent as to whether diastolic or systolic pressure is affected, they are consistent with the overall impression of a small positive association.

DOSE–RESPONSE

That most studies have shown positive significant regression coefficients in linear models of blood lead and blood pressure affords some evidence of a dose–response relationship.

A recent study of 507 Austrian law enforcement officers (Wolf *et al.,* 1995) suggested that an increasing trend in blood pressure, albeit a nonlinear one, was apparent with increasing blood lead. The low levels of blood lead examined in several studies suggest that there is no lower threshold for the effect of lead on blood pressure and, in two studies, formal investigation produced no evidence for a threshold (Pirkle *et al.,* 1985; Schwartz, 1988). However, some studies (Pocock *et al.,* 1984; Orssaud *et al.,* 1985; Schwartz, 1988) have suggested a moderation of the effect of lead at higher levels.

In summary, some evidence for a dose–response effect of blood lead on blood pressure is available but this evidence is not definitive. Given the size of the likely overall effect, this is not surprising.

INDEPENDENCE

Few would disagree that an association between blood lead and blood pressure exists, but the most contentious issue is whether it is independent. In the NHANES II analyses (Harlan *et al.,* 1985), 15 significant correlates of blood lead were reported and, because many variables including age, BMI, socioeconomic status, ethnicity, alcohol intake and other aspects of nutrition are determinants of both blood pressure and blood lead, establishing the independent effects of lead on blood pressure is extremely difficult.

The adjustment of data for covariates has been variable in different studies and frequently lacks apparent justification. Most studies have, either by analyses or design, adjusted data for age, sex and ethnicity, but thereafter similarities in approach cease. Unlike most studies, that by Beevers (Beevers *et al.,* 1976), which appeared to be the stimulus for the spate of subsequent research, did not adjust data for alcohol intake or smoking habit. Although alcohol increases blood pressure and is an important source of ingested lead, it has been argued that since lead may be a mechanism by which alcohol increases blood pressure and alcohol also influences lead absorption and metabolism, adjustment for alcohol may result in overcorrection of the data (Hertz-Picciotto & Croft, 1993). One recent study of 1319 Roman males (Menditto *et al.,* 1994) only found an association between blood lead and blood pressure among the majority who drank alcohol, but within this group, data were not adjusted for alcohol intake. Perhaps more contentious has been whether adjustment for haemoglobin is appropriate (de Silva, 1984; EPA, 1986). Erythrocytes bind 95% of blood lead and hence in conditions causing anaemia or haemoconcentration (which diuretics tend to induce) blood lead levels reflect lead exposure less accurately.

Even more fundamental is whether data should be adjusted for age. This question was overcome in two studies (Pocock *et al.,* 1984; Grandjean *et al.,* 1989) in which a narrow age band of subjects was observed. However, if blood pressure changes with age are mediated in part by blood lead, which rises with age, then adjustment for age is at least in part inappropriate. Indeed in one recent study, which evaluated bone and blood lead content, the impact of age on blood pressure was not independent once lead was included in the model (Hu *et al.,* 1996).

In summary, although it is by no means clear whether adjustment should be made for certain covariates, and despite the fact that the size of the association between lead and blood pressure tends to be reduced by adjustment, most studies do demonstrate a small positive association after adjustment. This may of course be the result of confounding, but over correction of the data is also possible.

CONSISTENCY

As described above, the data are by no means consistent. This inconsistency drove Staessen to exclude causation as a likely explanation for the association (Staessen *et al.,* 1994), but other reviewers have taken the opposite view whilst acknowledging the inconsistency of the data (Hertz-Picciotto & Croft, 1993; Schwartz, 1995).

Although data supporting the lead–blood pressure association in man arise from cross-sectional, case–control and cohort studies of both general populations and occupational groups, there is clear heterogeneity regarding age, sex, the impact of alcohol intake and whether the impact is greater (or even present) for systolic or diastolic pressures.

However, given the problems involved in measuring blood pressure (and to a lesser extent lead) and the handling of confounding described above, and given that the effect on blood pressure is likely to be small, such inconsistencies are not surprising and should not lead one to reject causation.

COHERENCE AND BIOLOGICAL PLAUSIBILITY

Over 150 years ago, it was recognised that high level lead exposure could be linked to various cardiovascular and renal diseases (Tanquerel des Planches, 1839). In 1935 the prevalence of systolic hypertension was observed to be twice as high among lead workers as among other workers (Vigdortchik, 1935). Data from several occupational cohorts with heavy exposure to lead confirm excess death rates and morbidity from chronic renal disease, cerebral haemorrhage and hypertensive vascular disease (Lilis *et al.,* 1968; Malcolm & Barnett, 1982; McMichael & Johnson, 1982; Cooper *et al.,* 1985; Selevan *et al.,* 1985; EPA, 1986). Two case–control studies (Khera *et al.,* 1980; Voors *et al.,* 1982) showed higher levels of lead in the blood and aorta, respectively, in patients admitted with or dying of cardiovascular diseases than in patients with non-cardiovascular diseases.

A population based case–control study in Glasgow (Beevers *et al.,* 1976) demonstrated higher blood lead levels (albeit unadjusted for alcohol and smoking) among hypertensives than among normotensives. This study was of particular interest because it did not relate to an occupational cohort with heavy lead exposure, and it paved the way for dozens of other studies on human and animal models investigating the impact of low-level lead exposure on blood pressure.

Controlled studies of animal models, including various rat strains, pigeons and dogs, show that the ingestion of low to moderate doses of lead in the diet is associated with an increase in blood pressure (Fouts & Page, 1942; Perry & Erlanger, 1978; Bogden *et al.,* 1991; Nakhoul & Brandbar, 1992).

How lead increases blood pressure is not clear. Several possible mechanisms have been suggested, including clinical or subclinical nephrotoxicity (Batuman *et al.,* 1983), increased pressor responses to catecholamines (Chai & Webb, 1988), and a direct impact on arteriolar smooth muscle, in part mediated by lead inducing raised intracellular calcium and hence increasing contractility (Sharp *et al.,* 1987).

Lead also appears to affect the renin–angiotensin system, although in human subjects the data are confusing; whereas short-term, low dose exposure was associated with increased renin activity in one study, other studies have shown low renin levels among those with a history of long-term high exposure to lead (Sharp *et al.,* 1987).

Until the mechanism whereby lead increases blood pressure (if indeed it does) has been clearly identified, the optimal measure of internal lead exposure will remain debatable.

TIME SEQUENCE

This important criterion for evaluating causation cannot easily be established from the cross-sectional or case–control studies which form the majority of studies currently available. However, the experimental data on animals described above do allow the time sequence between lead exposure and blood pressure to be established, and the four cohort studies (Weiss *et al.,* 1986; Neri *et al.,* 1988; Grandjean *et al.,* 1989; Staessen *et al.,* 1996), all of which showed a small if inconsistent positive association, also indicate that the higher lead levels preceded increased blood pressure and not *vice versa*.

SUMMARY

- The association between blood lead levels and blood pressure is not aetiologically strong, although most studies demonstrate some suggestion of a dose–response effect. It is important to acknowledge that small improvements in blood pressure in the population as a whole are likely to product important benefits in terms of reduced cardiovascular morbidity and mortality.

- The data from currently available studies are not consistent but, given the likely size of the effect of lead on blood pressure and the difficulties of measurement and analyses, this is not surprising.

- The data on low-level lead exposure in humans are coherent with the acknowledged adverse effects of high lead exposure and experimental data in animals. Furthermore, plausible mechanisms whereby lead increases blood pressure have been demonstrated.

- Most recent studies evaluating the association between lead and blood pressure are either cross-sectional or case–control in design. However, four recent cohort studies and the animal experimental studies establish that lead exposure precedes elevation of blood pressure. It would be useful to investigate the effect on blood pressure of lowering blood lead levels, to establish reversibility.

2.7.3 CONCLUSION

Although the adverse impact of blood lead levels on blood pressure is small and may be due to confounding or other types of bias, it is likely, on current evidence, that the effect is causative. As no harmful effects of reducing levels of lead in the environment are known, current efforts to reduce environmental lead levels should be encouraged.

REFERENCES

Batuman V, Landy E & Maesaka JK (1983) Contribution of lead to hypertension with renal impairment. *New Engl J Med, 309*, 17–21

Beevers DG, Erskine E & Robertson M (1976) Blood-lead and hypertension. *Lancet*, *2*, 1–3

Bogden JD, Gertner SB & Kemp FW (1991) Dietary lead and calcium: effects on blood pressure and renal neoplasia in Wistar rats. *J Nutr, 121,* 718–728

Chai S & Webb RC (1988) Effects of lead on vascular reactivity. *Environ Health Perspect, 78,* 85–89

Cooper WC, Wong O & Kheifets L (1985) Mortality among employees of lead battery plants and lead-producing plants. 1948–1980. *Scand J Work Environ Health, 11,* 331–345

de Silva PE (1984) Blood lead levels and the haematocrit correction. *Ann Occup Hyg, 28,* 417–428

Elwood PC, Yarnell JWG & Oldham PD (1988) Blood pressure and blood lead in surveys in Wales. *Am J Epidemiol, 27*, 942–945

EPA (1986) *Air Quality Criteria for Lead* (EPA-600/8-83/028df), Research Triangle Park NC, USA, US Environmental Protection Agency

Fouts PJ & Page IH (1942) The effect of chronic lead poisoning on arterial blood pressure in dogs. *Am Heart J, 24,* 329–331

Grandjean P, Hollnagel H, Hedegaard L, Christensen JM & Larsen S (1989) Blood lead–blood pressure relations: alcohol intake and hemoglobin as confounders. *Am J Epidemiol, 129,* 732–739

Harlan WR, Landis JR, Schmouder RL, Goldstein NG & Harlan LC (1985) Blood lead and blood pressure. *J Am Med Assoc, 2543,* 530–534

Hertz-Picciotto I & Croft J (1993) Review of the relation between blood lead and blood pressure. *Epidemiol Rev, 15,* 352–373

Hu H, Aro A, Payton M, Korrick S, Sparrow D, Weiss ST & Rotnizky A (1996) The relationship of bone and blood lead to hypertension: the normative aging study. *J Am Med Assoc,* 275, 1171–1176

Khera AK, Wibberley EG & Edwards KW (1980) Cadmium and lead levels in blood and urine in a series of cardiovascular and normotensive patients. *Inter J Environ Stud, 14,* 309–312

Kirkby H & Gyntelberg F (1985) Blood pressure and other cardiovascular risk factors of long-term exposure to lead. *Scand J Work Environ Health*, *11,* 15–19

Lilis R, Gavrilescu N, Nestorescu B, Dumitriu C & Roventa A (1968) Nephropathy in chronic lead poisoning. *Brit J Ind Med, 25,* 196–202

Malcolm D & Barnett HAR (1982) A mortality study of lead workers, 1925–76. *Brit J Ind Med, 39,* 404–410

McMichael AJ & Johnson HM (1982) Long-term mortality profile of heavily-exposed lead smelter workers. *J Occup Med, 24,* 375–378

Menditto A, Morisi G, Spagnolo A, Menotti A, Cigna G, Giampaoli S, Lanti M, Mariotti S, Meli P, Scafoto E, Scagna M, Chessa A, Dicarlo G, Dima F, Farchi F, Giuli B, Pasquali M, Parriachi V, Santaquilani A & Seccareccia F (1994) Association of blood lead and blood pressure on men aged 55 to 75 years: effect of selected social and biochemical confounders. *Environ Health Perspect, 102,* 107–111

Nakhoul Fm Kynw LH & Brautbar N (1992) Rapid hypertensiongenic effect of lead: studies in the spontaneously hypertensive rat. *Toxicol Ind Health, 8,* 89–102

National Research Council (1993) *Measuring Lead Exposure in Critical Populations*, Washington DC, USA, US National Academy Press

Neri LC, Hewitt D & Orser B (1988) Blood lead and blood pressure: analysis of cross-sectional and longitudinal data from Canada. *Environ Health Perspect, 78*, 123–126

Orssaud G, Claude JR & Moreau T (1985) Blood lead concentration and blood pressure. *Brit Med J, 290,* 244

Parkinson DK, Hodgson MJ & Bromet EJ (1987) Occupational lead exposure and blood pressure. *Brit J Ind Med, 44,* 744–748

Perry HM Jr & Erlanger MW (1978) Pressor effects of chronically feeding calcium and lead together. In: Hemphill DD, ed, *Trace Substances in Environmental Health-XII: Proceedings of the University of Missouri's 12th Annual Conference on Trace Substances in Environmental Health*, Columbia MO, USA, University of Missouri, pp 268–275

Pirkle JL, Schwartz J & Landis JR (1985) The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *Am J Epidemiol, 121,* 246–58

Pocock SJ, Shaper AG, Ashby D, Delves HT & Whitehead TP (1984) Blood lead concentration, blood pressure, and renal function. *Brit Med J, 289*, 872–874

Schwartz J (1988) The relationship between blood lead and blood pressure in the NHANES II survey. *Environ Health Perspect, 78*, 15–22

Schwartz J (1995) Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health, 50,* 31–37

Selevan SG, Landrigan PJ & Stern FB (1985) Mortality of lead smelter workers. *Am J Epidemiol, 122*, 673–683

Sharp DS, Becker CE & Smith AH (1987) Chronic low-level lead exposure: its role in the pathogenesis of hypertension. *Med Toxicol, 2,* 210–232

Sharp DS, Osterloh J & Becker CE (1988) Blood pressure and blood lead concentration in bus drivers. *Environ Health Perspect, 78,* 131–137

Staessen J, Sartor F & Roels H (1991) The association between blood pressure, calcium and other divalent cations: a population study. *J Human Hypertension, 5,* 485–494

Staessen JA, Bulpitt CJ, Fagard R, Lauwerys RR, Roels H, Thijs L & Amery A (1994) Hypertension caused by low-level lead exposure: myth or fact? *J Cardiovasc Risk, 1,* 87–97

Staessen JA, Roels H & Fagard R (1996) Lead exposure and conventional and ambulatory blood pressure: a prospective population study. *J Am Med Assoc, 275,* 1564–1570

Tanquerel des Planches L (1839) *Traite des maladies de plomb on saturnines*, Paris, France, pp 550

Victery W, Tyroler HA & Volpe R (1988) Summary of discussions. In: Victery W, ed, Symposium on lead–blood pressure relationships, April 1987, Chapel Hill NC. *Environ Health Perspect, 78,* 139–155

Vigdortchik NA (1935) Lead intoxication in the etiology of hypertonia. *J Ind Hyg, 17,* 1–6

Voors AW, Johnson WD & Shuman MS (1982) Addictive statistical effects of cadmium and lead on heart-related disease in a North Carolina autopsy series. *Arch Environ Health, 37,* 98–102

Weiss ST, Muñoz A & Stein A (1986) The relationship of blood lead to blood pressure in a longitudinal study of working men. *Am J Epidemiol, 123,* 800–808

Wolf C, Wallnöfer A, Waldhör T, Vutuc C, Meisenger V & Rüdiger H (1995) Effect of blood lead on blood pressure in occupationally nonexposed men. *Am J Ind Med, 27,* 897–903

3 Discussion

The Health Survey for England and the ALSPAC survey of children in the Avon area have shown that blood lead levels in the general population have continued to fall since the 1984–87 DoE surveys. The extent of the fall and the current levels are similar to those reported in other parts of the world and in the 1993 study in Glasgow (Watt *et al.,* 1996). The vast majority of blood lead measurements in the general population (Health Survey for England: mean 3.7 µg/dl), in children in the Avon area (3.4 µg/dl) and in Glasgow mothers (3.3 µg/dl) were below the upper limit of 10 µg/dl, accepted in 1997*.

The fall in blood lead levels seen in the UK and elsewhere has been ascribed to the reduction of lead in petrol and paint, and the removal of leaded solder from food cans and of lead plumbing from the home. The actual proportion of this decrease due to each measure is not clear, although it is likely that the major reduction seen since 1986 has been the result of reduced emissions of petrol lead. The apportionment of body lead burden to specific lead sources has been greatly facilitated by developments in stable isotope analysis.

Although the average blood lead levels reported in these studies are low, there are, occasionally, individuals with significantly raised blood lead levels that cannot be ascribed to occupational exposure of the individual (or of any family member). A review of the possible sources that might account for raised blood lead suggests that lead in water, derived from lead service pipes and household plumbing in areas with water of high plumbosolvency, may be important. Lead in old paint may be an occasional source associated with redecorating in older properties.

* Declaration of the Environment Leaders of The Eight on Children's Environmental Health: from a summit held in Miami, USA on 5–6 May 1997, available [at 5 December 1997] from http://www.library.utoronto.ca/www/g7/environment/envchi97.htm

The local occasional occurrence of high concentrations of lead in drinking water gives special concern for bottle fed infants. Nonetheless, the view presented herein is that, even at the higher blood lead levels found in the Health Survey for England and ALSPAC surveys, there is little likelihood of detectable developmental (intellectual) damage in the fetus or young child. Developmental measurements to be made later on during the ALSPAC survey will be compared with the blood lead measurements made at 31 months of age, but the low blood lead levels recorded and the size of the population make it unlikely that any effect will be observable. More does needed to be known about blood lead levels in bottle fed children in those parts of the country where there are local occurrences of high lead levels in domestic drinking water.

Earlier studies of occupationally exposed and other populations with higher blood lead levels had demonstrated relationships between blood lead and blood pressure, although there were some methodological difficulties. Although the range of blood lead measurements in the Health Survey for England study was relatively small, a consistent positive relationship between blood lead and diastolic blood pressure was seen in men. The data showed a dose–response relationship and no lower threshold. This is of importance in public health terms and, although the effect was small, small reductions in blood pressure at the population level are likely to be associated with important benefits in terms of reduced cardiovascular morbidity and mortality. Thus this on its own would justify efforts to reduce further the population's lead exposure.

OVERALL CONCLUSIONS

Blood lead levels in the UK have fallen significantly (approximately threefold) since the 1984–87 DoE surveys. The issues remaining concern the effects of lead on fetal and infant development and on blood pressure. There is concern about bottle fed infants in the few remaining areas with high concentrations of lead in domestic drinking water. Nonetheless, for the majority of infants with blood lead levels at the current levels observed in the ALSPAC study, there was no evidence that developmental harm would occur. However, reductions in prevailing blood lead levels could have a significant and important beneficial effect on cardiovascular morbidity and mortality through a reduction in blood pressure.

Based on the reviews and discussions presented in this report a number of further studies are recommended.

- Local areas with high lead content in drinking water should be identified. The effects of these high levels on the content of lead in blood and teeth should be investigated and comparisons should be made of blood and teeth levels in bottle *versus* breast fed children.

- Past studies and current data sets should be reviewed, and new studies developed to investigate the development of intelligence (IQ) of bottle *versus* breast fed children in areas with high levels of lead in water.

- Newer analytical methods and the use of isotope ratio techniques should be assessed to facilitate better identification of sources of lead exposure.

- It would be worthwhile to extend studies of the levels of lead in household dust and its bioavailability compared with lead from other sources.

REFERENCES

Watt GCM, Britton A, Gilmour WH, Moore MR, Murray GD, Robertson SJ & Wormersley J (1996) Is lead in tap water still a public health problem? An observational study in Glasgow. *Brit Med J, 313,* 979–981

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Acknowledgements

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Report R1 published 1994

Air Pollution and Respiratory Disease: UK Research Priorities
Report R2 published 1994 (out of print)

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