BLOOD LEAD LEVELS OF A POPULATION GROUP NOT OCCUPATIONALLY EXPOSED TO LEAD IN SINGAPORE

KS Neo[1], KT Goh[1] and CT Sam[2]

[1]Institute of Environmental Epidemiology, Ministry of the Environment, Singapore; [2]Industrial Health Laboratory, Institute of Science and Forensic Medicine, Ministry of Health, Singapore

Abstract. A survey was conducted between 1995 and 1997 to assess the impact of introduction of unleaded petrol and other public health measures on the blood lead level of the population. The geometric mean blood lead level of 269 government employees as determined by graphite furnace atomic absorption spectroscopy, was 66.0 μg/l, much lower than that recorded before introduction of lead-free petrol. Using multiple regression analysis, factors significantly associated with blood lead levels were: exposure to traffic, age (>50 years) and active smoking. Passive smoking, exposure to recent paint work, consumption of alcohol and traditional medicine were found not to be significantly associated with the blood lead level.

INTRODUCTION

Human exposure to lead occurs through a combination of inhalation and ingestion. Inhalational route is more important in the occupationally exposed groups while the oral route plays a bigger role in the general population. The contribution of air-borne lead comes mainly from vehicular exhaust which runs on petroleum containing alky-lead derivatives as anti-knock agent (Tri-Tugaswati *et al*, 1987; Tera *et al*, 1985; Ahmed *et al*, 1987), burning of solid waste and cigarette smoke.

Various studies have shown that chronic low level lead exposure can have impact on the health of the general population. Slower nerve conduction was demonstrated in adults at blood lead levels of 30 to 40 μg/l (Ashby, 1980; Hernberg *et al*, 1984). In children, chronic low level lead absorption has been shown to cause disturbances in cognition, behavior and attention resulting in lower intelligence quotient (Schwartz, 1994; Wigg *et al*, 1988; Fulton *et al*, 1987; Schwartz and Otto, 1987). An association between blood lead level below 100 μg/l and cognitive function of middle-age and elderly men has also been reported (Payton *et al*, 1998). Other effects from chronic exposure in humans include that on blood pressure and kidney function (Pirkle *et al*, 1985, Campbell *et al*, 1977).

Exhaust emission from vehicles using leaded petrol was identified as the major source of environmental lead exposure among the general population in Singapore. Consequently, a concerted effort was made by the Ministry of the Environment to reduce the lead content of petrol from 0.4 g/l to 0.15 g/l in 1987 and unleaded petrol was introduced in 1991. This resulted in a sharp decline of roadside lead level from 1.1 $\mu g/m^3$ in 1986 to 0.13 $\mu g/m^3$ in 1997 and a moderate decline of ambient lead from 0.5 $\mu g/m^3$ to 0.1 $\mu g/m^3$ in the same period (Ministry of the Environment, 1986-1997). The level of roadside lead in Singapore in 1997 was comparable to that in cities of developed countries such as Central London in 1998 and Los Angeles in 1996 (WHO/UNEP, 1992).

To determine the impact of introduction of lead-free petrol and other public health measures for the control of environmental lead, a survey on the blood lead levels in the general population was conducted during 1995-1997.

MATERIALS AND METHODS

The subjects were all government employees who were recruited during a healthy lifestyle program in which blood was collected for testing of glucose and cholesterol. All of them were not known to have any occupational exposure to lead other than that present in the environment. With the consent of each participant, an additional 5 ml of blood were obtained through venepuncture and stored in a heparinized tube. Personal particulars including details of specific risk factors such as transportation, exposure to traffic and tobacco smoke and paintwork, and consumption of alcohol or traditional medicines were gathered using a struc-

Correspondence: Dr KT Goh, Institute of Environmental Epidemiology, 40 Scotts Road, #22-00, ENV Building, Singapore 228231.
E-mail: goh_kee_tai@env.gov.sg

tured questionnaire with closed ended answers.

The samples were despatched to the Institute of Science and Forensic Medicine, Singapore, at the end of each collection. The blood lead level was determined by preliminary treatment with a dilute surfactant followed by automated furnace atomic absorption spectroscopy with a L' vou platform (Vahter *et al*, 1991). Quality assurance for the laboratory was through participation in two external programs with the Centre de Toxicologie du Quebec, Canada, and the Health and Safety Executive, United Kingdom.

In the statistical analysis, logarithmic transformation of the blood lead levels was performed to approximate a normal distribution. Geometric mean and its 95% confidence limits were obtained. The relationship of log-transformed blood lead levels (dependent variables) to lifestyle and environmental factors (independent variables) were determined by multiple linear regression with dummy variables. A p-value of less than 0.05 was considered statistically significant in a two-tailed test. All calculations were performed using SPSS (Statistical Package For Social Science) release 7 and Excel software.

RESULTS

A total of 269 adults (226 males and 43 females) aged between 22 and 67 years were tested. The geometric mean blood lead level was 66.0 μg/l. Although there was high variability, none had values above 200 μg/l.

Stratification of the blood lead levels by age-group showed that the mean blood lead level increased with age. The mean blood lead level (was 57.2 μg/l) in the 20-29 year age group 58.2 μg/l in the 30-39 year age group, 59.1 μg/l in the 40-49 year age group and 71.3 μg/l in the 50-59 year age group. The blood lead level declined to 69.7 μg/l in the age group over 60 years but was still higher than that of the 40-49 age group (Table 1).

Analysis of the mean blood lead level of the different ethnic groups showed that Malays had the highest mean blood lead level of 65.8 μg/l, followed by Indians with a mean blood lead level of 64.7 μg/l, and Chinese with a mean blood lead level of 56.8 μg/l.

Among active smokers, the blood lead level was related to the number of cigarettes smoked per day. The levels were 56.8 μg/l, 67.9μg/l and 69.7

Table 1
Relationship between mean blood lead level and age group in the sample population.

Age group (yrs)	Sample size	Mean blood lead level (μg/l)
20-29	34	57.2
30-39	96	58.2
40-49	70	59.1
50-59	40	71.3
60+	29	69.7

Table 2
Relationship between mean blood lead level and exposure to tobacco smoke in the sample population.

	No.	Mean blood lead level (μg/l)	95%CI
Passive smoking			
Non exposed	61	58.9	53.4-65.0
1-10 cigarettes daily	71	57.7	52.9-62.9
>10 cigarettes daily	32	51.3	44.6-59.1
Active smoking			
Non smokers	164	56.8	53.6-60.3
1-10 cigarettes daily	50	67.9	61.4-74.9
>10 cigarettes daily	55	69.7	63.2-77.0

Table 3
Relationship between mean blood lead level and exposure to traffic.

	No.	Mean blood lead level (μg/l)	95%CI
Mode of daily transportation			
Motorcycle/bicycle/walk	135	64.9	61.8-70.7
Car/bus/train	134	57.7	53.9-61.0
Job-related exposure to traffic			
Office workers (air-conditioned premises)	30	52.0	44.7-60.5
Office workers (non-air-conditioned premises)	83	58.9	54.1-64.2
Outdoor workers	106	64.5	60.9-68.3
No. of hours spent on outdoor work			
1-4 hours daily	82	55.5	50.6-60.9
5-8 hours daily	138	64.5	60.7-68.5
Dwelling-related exposure to traffic			
Up to fifth storey	115	63.8	59.3-68.7
6^{th} to 10^{th} storey	108	60.2	55.9-64.7
Above 10^{th} storey	46	57.6	52.1-63.6

Table 4
Relationship between blood lead levels and specific exposure factors.

	No.	Mean blood lead levels (μg/l)	95%CI
Exposure to paintwork			
Non exposed	155	62.9	59.4-66.7
Exposed ≥3 months ago	41	57.4	50.8-64.9
Exposed <3 months ago	73	60.0	54.4-66.2
Consumption of traditional medicine			
Non consumers	188	61.4	58.0-64.8
Less than once a week	23	61.2	53.7-69.7
At least once a week	58	60.9	54.7-67.9
Consumption of alcohol			
Non drinkers	182	61.0	57.6-64.6
Occasional drinkers (less than once a week)	63	62.1	56.6-68.1
Regular drinkers (at least once a week)	24	60.8	52.0-70.9

μg/l for non-smokers, light smokers (1-10 cigarettes daily) and moderate/heavy smokers (>10 cigarettes daily), respectively (Table 2). No such relationship was found for passive smoking.

The effect on blood lead levels due to exposure to traffic was determined by four parameters: mode of daily transport, job-related exposure to traffic, length of time spent outdoor and dwelling-related exposure. It was found that blood lead levels in workers who walked and travelled by motorcycles/bicycles were higher that those who travelled by airconditioned cars/buses and trains. Blood lead levels were also related to nature of the job. The mean blood lead level of outdoor workers was higher than that of office workers. Among office workers, the mean blood lead level of those who worked in non air-conditioned environment was higher than that of those working in air-conditioned environment. The mean blood lead level

Table 5
Structured equation selected by multiple regression analysis for blood lead levels.

Explanatory variables	Regression coefficients	*t*-statistics
Intercept	1.646	-
Female	-0.042	-0.699
Age: 30-39 years	0.044	0.476
Age: 40-49 years	0.065	0.731
Age: 50-59 years	0.218	2.699[a]
Age: 60+ years	0.150	2.000[b]
Light smoker	0.153	2.488[a]
Moderate/heavy smoker	0.205	3.325[a]
Transport: motorcycle/bicycle/walk	0.146	2.443[b]
Residence: high-rise apartment (above 10 storeys)	-0.069	-1.186
Exposure to recent paintwork (< 3 months)	-0.060	-1.026
Consumer of traditional medicine (at least once a week)	-0.046	-0.768
Regular alcohol drinker (at least once a week)	-0.002	-0.037

[a]p<0.01
[b]p<0.05

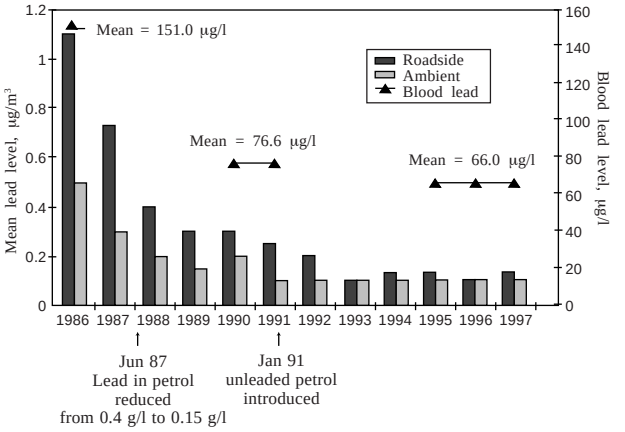

Fig 1–Roadside, ambient and blood lead levels in Singapore, 1986-1997.

Data for roadside and ambient lead levels were obtained from the Pollution Control Department, Ministry of the Environment.

also increased with the amount of working time spent outdoor; 64.5 µg/l in those working 5-8 hours outdoor daily compared to 55.5 µg/l in those working 1-4 hours daily. Mean blood lead levels were also affected by the height of the residential premises. Those workers staying above 10 storeys had the lowest blood lead level (Table 3).

The mean blood lead level was found not to be related to recent exposure to paintwork (Table 4). Consumption of alcohol and traditional medicine also did not appear to affect the mean blood lead levels among the study population.

Multiple linear regression analysis showed that older age group (>50 years), smoking and exposure to vehicular exhaust were significant factors that contributed to elevated blood lead levels (Table 5). Exposure to recent paintwork, height of residential premises and consumption of traditional medicine and alcohol were found not to be significantly associated with elevated blood lead levels.

DISCUSSION

We confirmed that the introduction of unleaded petrol in 1991 has had a definite impact on the blood lead level of the population surveyed. Reduction of blood lead level as a consequence of the reduction of gasoline lead has been noted in other countries (Pirkle *et al*, 1994; Grobler *et al*, 1992; Quinn *et al*, 1989; Annest *et al*, 1983). The decline in blood lead levels from 151.0 µg/l in 1986 (Goh *et al*, 1989) to 76.6 µg/l in 1990/1991 (Ooi *et al*, 1991) and to 66.0 µg/l in 1995/1997 was consistent with the decrease in ambient lead concentrations monitored during this period (Fig 1). Nevertheless, contribution of lead from vehicular exhaust continued to remain a significant determinant of blood lead levels in our study.

We also confirmed the findings of other studies (Nygard *et al*, 1977; McLaughlin and Stopps 1973; Zielhuis *et al*, 1977; Tola and Nordman, 1977) that age and smoking are significant factors associated with increasing blood lead levels. To prevent exposure of the population from tobacco smoke, public health measures against smoking are being further intensified.

The lack of correlation between blood lead levels and exposure to paintwork could be due to the introduction of a labelling scheme to limit the content of lead in paint in February 1993. Under this scheme, all paints containing lead compounds in excess of 0.25% of lead by weight of the paint formulation had to be labelled with either a Type 1 or Type 2 label depending on the lead content of the paint. (Type 1 label for red lead oxide >1% and other lead compounds >5%, Type 2 label for red lead oxide and other lead compounds between 0.66% and 1%). The effectiveness of the stringent regulatory measures taken to eliminate lead contamination in beverages and traditional medicine is demonstrated by the lack of association between blood lead levels and intake of these products.

While further research is being continued to define what constitutes an entirely "safe" level for environmental lead (Matte, 1999), the Ministry of the Environment is closely monitoring the lead concentration in air, water, food and consumer products to ensure that exposure of the general population to this toxic chemical is reduced to the minimum.

ACKNOWLEDGEMENTS

We wish to express our appreciation to Nursing Officers AG Teo, and LT Chua and the Environmental Health Officers of the Ministry of the Environment for their assistance in conducting the survey. Special thanks to Mrs BT Ang of the Institute of Science and Forensic Medicine for the laboratory analyses of blood lead concentrations.

REFERENCES

Ahmed NS, El-Gendy KS, El- Refaie AKH, *et al*. Assessment of lead toxicity in traffic controllers of Alexandria, Egypt, road intersections. *Arch Environ Health* 1987; 42: 92-5.

Annest JL, Pirkle JL, Makuc D, Neese JW, Bayes DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med* 1983; 308: 1373-7.

Ashby JAS. Biochemical changes and neurophysical findings in lead poisoning. *Br J Indust Med* 1980; 37: 80-4.

Campbell BC, Beattie AD, Moore MR, Goldberg A, Reid AG. Renal insufficiency associated with excessive lead exposure. *Br Med J* 1977; I: 482-5.

Fulton M, Thomson G, Hunter R, Raab G, Laxen D, Hepburn E. Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* 1987; I: 1221-6.

Goh KT, Ling MK, Kwok SF, Rajan U. Blood lead levels of four selected population groups in Singapore. *ASEAN J Clin Sci* 1989; 9: 75-9.

Grobler SR, Maresky LS, Kotze TJ. Lead reduction of petrol and blood lead concentration of athletes. *Arch Environ Health* 1992; 47: 139-42.

Hernberg S, Seppålåinen AM, Mantere P. A prospective study on early neurotoxic effects of lead. *Ann Acad Med Singapore* 1984; 13: 378-82.

Matte TD. Reducing blood lead levels: benefits and strategies. *JAMA* 1999; 281: 2340-2.

McLaughlin M, Stopps GI. Smoking and lead. *Arch Environ Health* 1973; 26: 131-6.

Ministry of the Environment, Singapore. Pollution Control Department, Annual reports 1986-1997.

Nygard SP, Ottosen J, Hansen JC. Whole blood lead concentration in Danes, relation to age and environment. *Dan Med Bull* 1977; 24: 49-51.

Ooi PL, Goh KT, Heng BH, Sam CT, Kong KH, Rajan U. Biological monitoring of human exposure to environmental lead in Singapore. *Rev Environ Health* 1991; 9: 207-13.

Payton M, Riggs KM, Spiro A, Weiss T, Hu H. Relations on bone and blood lead to cognitive function in the VA Normative Aging Study. *Neurotoxicol Teratol* 1998; 20: 19-27.

Pirkle JL, Schwartz J, Landis JR, Harlan WR. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *Am J Epidemiol* 1985; 121: 246-58.

Pirkle JL, Brody DJ, Gunter EW, *et al*. The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *JAMA* 1994; 272: 284-91.

Quinn MJ, Delves HT. The UK blood lead monitoring programme 1984-1987: results for 1986. *Hum Toxicol* 1989; 8: 205-20.

Schwartz J, Otto D. Blood lead, hearing thresholds, and neurobehavioural development in children and youth. *Arch Environ Health* 1987; 42: 153-60.

Schwartz J. Low-level lead exposure and children's IQ:

a meta-analysis and search for a threshold. *Environ Res* 1994; 65: 42-55.

Tera O, Schwartzman DW, Watkins TR. Identification of gasoline lead in children's blood using isotopic analysis. *Arch Environ Health* 1985; 40: 120-3.

Tri-Tugaswati A, Suzuki S, Koyama H, Kawada T. Health effects of air pollution due to automotive lead in Jakarta. *Asia- Pacific J Public Health* 1987; 1: 23-7.

Tola S, Nordman CH. Smoking and blood lead concentrations in lead exposed workers and an unexposed population. *Environ Res* 1977; 13: 250-5.

Vahter M, Berglund M, Lind B, Jorhem L, Storach S, Friborg L. Personal monitoring of lead and cadmium exposure- a Swedish study with special reference to methodological aspects. *Scand J Work Environ Health* 1991: 17: 65-74.

WHO/UNEP. Urban air pollution in megacities of the world, World Health Organisation, United Nations Environment Programme. Oxford: Blackwell, 1992.

Wigg NR, Vimpani GV, McMichael A J, *et al*. Port Pirie cohort study: childhood blood lead and neuropsychological development at age two years. *J Epidemiol Community Health* 1988; 42: 213-9.

Zielhuis RL, Stuik EJ, Herber RFM, *et al*. Smoking habits and levels of lead and cadmium in blood in urban women. *Int Arch Occup Environ Health* 1977; 39: 53-8.