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*Original Article*

# Blood Lead Levels in Japanese Children: Effects of Passive Smoking

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## Abstract

**Blood lead levels (BLLs) of 188 pediatric patients were measured and their parents were queried as to the smoking style in their home. Their mean BLL was 3.16 μg/dl, which was among the lowest levels in the world, and none of them had levels of over 10 μg/dl. Preschool children (1 to 6 years of age) with parents who smoked in the same room had a significantly higher BLL (mean; 4.15 μg/dl) than those with parents who never smoked (mean; 3.06 μg/dl) (P<0.01). However, the mean BLL of school children (6 to 15 years of age) with parents who smoked in the same room was not significantly different from that of school children with parents who never smoked. Passive smoking caused an increase of the BLL only in preschool children in Japan. This is probably because preschool infants spend much more time with their parents and have much more contact with passive smoking than school children and, additionally young infants have a limited ability to excrete lead from the body.**

**Key words:** Blood lead levels, Lead, Passive smoking

## Introduction

Pediatric lead poisoning has been a public health problem in the United States and European countries [1-3]. In Japan, however, clinicians take very little notice of pediatric lead poisoning, and there are no statistical data on the blood lead levels (BLLs) of Japanese children. On the other hand, it has been reported that children of smoking parents have a higher BLL than those of non-smoking parents in European countries [4, 5]. The authors measured BLL of Japanese children and studied the effects of passive smoking on the children's BLL.

## Materials and Methods

Venous blood samples were obtained from 188 children (106 boys, 82 girls) aged 1 to 15 years (mean; 7.6 years), who visited the pediatric clinic of Shizuoka General Hospital from June to October 1993. Most patients had common diseases, for example, respiratory infections, abdominal symptoms and asthma attacks. All patients with metabolic disorders or substance poisoning were excluded from this study. The authors asked their parents about their smoking style in the home.

Whole blood lead was measured using a graphite furnace atomic absorption spectrophotometer (Perkin-Elmer Model 4000 equipped with the HGA-400 programmer) after treatment of the samples by the method of Murphy et al [6]. The graphite furnace program followed the method of Miller et al [7]. The detection limit was 0.1 μg/l. Analysis of each specimen was performed in duplicate, and the mean of the duplicate measurements was reported.

The subjects were divided into three groups according to the parents' smoking style;

Group A: Children of smoking parents who usually smoked in the same room with their children.
Group B: Children of smoking parents who usually took care not to smoke in the same room with their children.
Group C: Children of non-smoking parents.

They were classified further into preschool children and school children. As a result, there were six subgroups in all. The mean and the range (in the parenthesis) of age of each group were as follows:

Preschool-group A; 3.6 years (1.0 to 6.1 years), preschool-group B; 3.6 years (1.2 to 5.9 years), preschool-group C; 3.4 years (1.1 to 6.3 years), school-group A; 11.2 years (6.3 to 15.7 years), school-group B; 10.9 years (6.6 to 15.7 years), and school-group C; 11.1 years (6.8 to 15.3 years).

Statistical analysis of the data was performed by one-way analysis of variance (Bonferroni's test).

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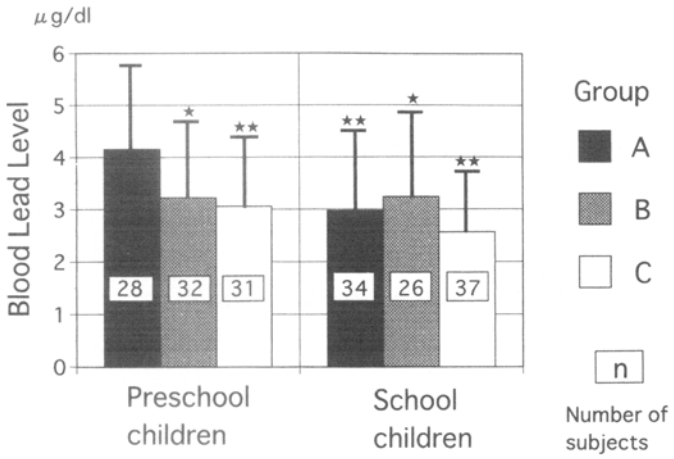


**Fig. Blood lead levels in Japanese children.**
**Group A: Children of smoking parents who usually smoke in the same room with their children.**
**Group B: Children of smoking parents who take care not to smoke in the same room with their children.**
**Group C: Children of non-smoking parents.**
**★★P<0.01, ★P<0.05, significantly lower than the BLL of group A preschool children.**

## Results

The BLL (mean ± standard deviation) of all the subjects was 3.16 ± 1.50μg/dl, and ranged between 0.80μg/dl and 9.51μg/dl. The BLL of boys was 3.17 ± 1.34μg/dl and that of girls 3.14 ± 1.69μg/dl. There was no significant difference between them.

The BLLs of groups A, B and C in preschool children were 4.15 ± 1.56μg/dl (n=28), 3.22 ± 1.46μg/dl (n=32) and 3.06 ± 1.31μg/dl (n=31), respectively. The BLLs of groups A, B and C in school children were 2.97±1.50μg/dl (n=34), 3.24±1.66μg/dl (n=26) and 2.56 ± 1.19μg/dl (n=37), respectively.

The BLLs of the six subgroups were compared, and the results are shown in the Figure. The mean BLL of group A preschool children was significantly higher than those of the other five subgroups, and there were no significant differences among the other five subgroups.

## Discussion

There have been no statistical data on the BLL in Japanese children. The authors presented recent data on the BLL of pediatric patients seen in an outpatient clinic. Since the patients selected had common diseases and all patients with metabolic disorders or substance poisoning were excluded, the BLL was unlikely to be influenced by their diseases.

Hayes et al. studied the BLL of children aged 6 months to 5 years (mean; 2.7 years) in Chicago and showed that the mean value was 12 μg/dl in 1988[8]. Norman et al. reported that the percentage of children with elevated BLL (≥10μg/dl) was 25.2% in 2-year-olds and 13.7% in 5-year-olds in North Carolina in 1994[9]. Jin et al. reported that the mean BLL was 6.0μg/dl (range; 1.24 to 17.6μg/dl) in children aged 24 to 36 months in Vancouver in 1995[10]. Sherlock et al. measured the BLL of Caucasian and Asian children (age range 2.5 to 5 years, age and sex-matched) living in London, and reported that the mean value was 9.7μg/dl in Caucasian children and 8.1 μg/dl in Asian children in 1985[11]. Cambra and Alonso reported that the mean BLL of children aged 2 to 3 years in Basque country in Spain was 5.7 μg/dl and that 14% of the children had levels that exceeded 10 μg/dl in 1995[12]. Andren et al. reported that the mean BLL of Swedish children (8 to 13 years of age) of non-smoking parents was 2.95μg/dl in 1988[4].

In this study, the authors demonstrated that the mean BLL of Japanese children was among the lowest levels in the industrialized world[4,8-12]. The reasons proposed are that leaded gasoline use was prohibited in Japan more than two decades ago and Japanese people generally do not often do house-painting, which has been considered to be a major cause of lead poisoning in children in the United States and European countries[13].

It has been reported that smokers have a higher BLL than non-smokers[14,15], and children who are exposed to tobacco smoke have a higher BLL than children who are not[4,5]. Andren et al. showed that parental smoking, not other environmental or dietary factors was related to the BLL in Swedish children (8 to 13 years of age, the mean 11 years); in children of non-smoking parents the mean BLL was 2.95μg/dl and in those whose parents both smoked it was 4.70μg/dl[4].

This study clearly revealed that preschool children with parents who smoked in close proximity to their children (group A) had a higher BLL than preschool children with parents who never smoked (group C). The lead content of Japanese cigarettes is reported to be 1.28 ± 0.31μg/cigarette (mean ± standard deviation), ranging between 0.96 and 2.00μg/cigarette[15]. The proportion of smokers in the Japanese population was reported to be 61.2% for men and 14.2% for women in 1991[16]. The figure for smoking in the male adult population is one of the highest in industrialized countries. Since the harmful effects of passive smoking have been documented recently in the general population[17], increasing numbers of smoking parents take care not to smoke in close proximity to their children. The authors found that about half of smoking parents, however, still smoked in the same room with their children in Japan[18]. It is noteworthy that preschool children of smoking parents who take special care not to smoke in the same room with their children (group B) had a low BLL similar to the case of non-smoking parents. The mean BLLs of all three subgroups of school children were lower than that of group A preschool children. About the reasons for the significantly higher BLL of only group A preschool children, the authors speculate that preschool infants may spend much more time with their parents and may have much more contact with passive smoking than school children and, additionally young infants have a limited ability to excrete lead from the body because of the immaturity of renal function[19]. Okada studied intake and excretion of lead in Japanese rural populations of various ages, and reported that daily urinary lead excretion was less in infants aged one to two years (7.9 to 14.0μg/day, mean; 10.2μg/day, n=5) than in children aged seven to fourteen years (8.0 to 190.6μg/day, mean; 34.6μg/day, n=35), and that daily lead storage was much higher in the infants (mean; 43μg/day) than in the children (mean; 12μg/day)[20].

The authors believe that children should be protected from passive smoking for the purpose of avoiding the risk of increased BLL.

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## References

1) Sargent JD, Brown MJ, Freeman JL, Bailey A, Goodman D, Freeman DH. Childhood lead poisoning in Massachusetts communities: Its association with sociodemographic and housing characteristics. Am J Publ Health 1995; **85**: 528-34.
2) Pocock SJ, Smith M, Baghurst P. Environmental lead and children's intelligence: A systematic review of the epidemiological evidence. Brit Med J 1994; **309**: 1189-97.
3) Stromberg U, Schutz A, Skerfving S. Substantial decrease of blood lead in Swedish children, 1978-94, associated with petrol lead. Occup Environ Med 1995; **52**: 764-9.
4) Andren P, Schutz A, Vahter M, Attewell R, Johansson L, Willers S, Skerfving S. Environmental exposure to lead and arsenic among children living near a glassworks. Sci Total Environ 1988; **77**: 25-34.
5) Willers S, Schutz A, Attewell R, Skerfving S. Relation between lead and cadmium in blood and the involuntary smoking of children. Scand J Work Environ Health 1988; **14**: 385-9.
6) Murphy TF, Nomoto S, Sunderman FW. Measurements of blood lead by atomic absorption spectrometry. Annal Clin Lab Sci 1971; **1**: 57-63.
7) Miller DT, Paschal DC, Gunter EW, Stroud PE, D'Angelo J. Determination of lead in blood using electrothermal atomisation atomic absorption spectrometry with a L'vov platform and matrix modifier. Analyst 1987; **112**: 1701-4.
8) Hayes EB, McElvaine MD, Orbach HG, Fernandez AM, Lyne S, Matte TD. Long-term trends in blood lead levels among children in Chicago: Relationship to air lead levels. Pediatrics 1994; **93**: 195-200.
9) Norman EH, Bordley WC, Hertz-Picciotto I, Newton DA. Rural-urban blood lead differences in North Carolina children. Pediatrics 1994; **94**: 59-64.
10) Jin A, Hertzman C, Peck SHS, Lockitch G. Blood lead levels in children aged 24 to 36 months in Vancouver. Can Med Assoc J 1995; **152**: 1077-86.
11) Sherlock JC, Barltrop D, Evans WH, Quinn MJ, Smart GA, Strehlow C. Blood lead concentrations and lead intake in children of different ethnic origin. Human Toxicol 1985; **4**: 513-9.
12) Cambra K, Alonso E. Blood lead levels in 2- to 3-year-old children in the Greater Bilbao Area (Basque Country, Spain): Relation to dust and water lead levels. Arch Environ Health 1995; **50**: 362-6.
13) Wietlisbach V, Rickenbach M, Berode M, Guillemin M. Time trend and determinants of blood lead levels in a Swiss population over a transition period (1984-1993) from leaded to unleaded gasoline use. Environ Res 1995; **68**: 82-90.
14) Chiba M, Masironi R. Toxic and trace elements in tobacco and tobacco smoke. Bullet WHO 1992; **70**: 269-75.
15) Watanabe T, Fujita H, Koizumi A, Chiba K, Miyasaka M, Ikeda M. Baseline level of blood lead concentration among Japanese farmers. Arch Environ Health 1985; **40**: 170-6.
16) Japan Tobacco Incorporated. The annual report on smoking rates in Japan (in Japanese). Japan Tobacco Incorporated, Tokyo, 1992.
17) Hirayama T. Prevention of health hazards in children by active and passive smoking (in Japanese). Pharma Medica 1991; **9(11)**: 57-61.
18) Kaji M, Gotoh M, Takagi Y, Masuda H. A study on the smoking styles of smokers in the homes of childhood outpatients (in Japanese). Shizuoka J Med 1995; **11**: 5-9.
19) Nyhan WL, Sawyer M, Kearney T, Spector S, Hilton S. Lead intoxication in children. West J Med 1985; **143**: 357-64.
20) Okada A. A study on the contents of lead in the blood, urine and feces of the healthy Japanese rural population (in Japanese, abstract in English). Osaka City Med J 1957; **6**: 992-1021.

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