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Lead exposure in Saudi Arabia and its relationship to smoking

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Lead was determined in whole blood samples obtained from 202 Saudi male volunteers. The influence of smoking on lead exposure was investigated. Blood lead was significantly higher in current smokers than in non-smokers and previous smokers ($P < 0.05$). The distribution of blood lead data in the screened subjects suggested the eixstence of two mixed populations and a cut-off of 12 $\mu g\ dl^{-1}$ was found where the two populations separate. Of the exposed population, 80% with blood lead concentrations above 12 $\mu g\ dl^{-1}$ were smokers.

Keywords: lead, smoking

Introduction

Smoking is thought to increase lead exposure. Tobacco, like other plants, contains some lead absorbed from the soil and may also have lead deposited on the surface of the leaves. The widespread use of lead arsenate as a pesticide on tobacco crops has contaminated tobacco products with both lead and arsenic in the past. Since the late 1940s, in the USA, lead arsenate has gradually been replaced by organic pesticides and by 1975 no production or use of this compound was reported. However, residual lead in the soil from historic applications and from atmospheric deposition remains a potential source of lead in domestic tobacco products (NAS 1980). Several studies have shown that smoking does significantly contribute to blood lead concentrations (McLaughlin & Stopps 1973, Zielhuis *et al.* 1977, Grandjean *et al.* 1981, McIntosh *et al.* 1982, Shaper *et al.* 1982, Elinder *et al.* 1983, Pocock *et al.* 1983, Chiba & Masironi 1992, Hense *et al.* 1992, Willers *et al.* 1992). The actual concentration of lead in tobacco itself is extremely variable having been reported as between 21 and 84 μg per cigarette. It has been estimated that the direct inhalation intake of lead from smoking 20 cigarettes a day would lie between 1 and 5 μg (WHO 1977). Tobacco imports in Saudi Arabia increased by more than 80% in less than 10 years. This can be viewed as an indicator of the increase of smoking prevalence. Of the few surveys on the prevalence of smoking that have been carried out among Saudi male students in Riyadh, more than 50% of the male university students in Riyadh were found to be smokers (Bener 1987).

In view of these earlier observations, a pilot study was conducted to examine to what extent smoking among Saudi males influences blood lead concentrations.

Materials and methods

The study population of 202 males aged 16–57 years old living in Riyadh city, the capital of Saudi Arabia, was drawn from two sources: 136 smokers, 11 previous smokers and 55 non-smokers were Saudi volunteers from blood bank donors attending either the Blood Bank of the King Faisal Specialist Hospital and Research Centre or the Anti-smoking clinic. In conjunction with the collection of blood samples, each participant was individually interviewed and a questionnaire was completed. The interviews were administered by trained technicians. The questionnaire included questions about smoking habits, age and health conditions. Smoking habits was classified as follows: non-smoking, previous smoking and current smoking. Current smokers were classified by their total reported daily number of manufactured cigarettes. Light smokers were defined as those consuming less than the equivalent of 10 cigarettes of these smoking materials per day. Moderate smokers had from 10 to 19 cigarettes per day. Heavy smokers had the equivalent of at least 20 cigarettes. All subjects studied had no history of illnesses.

Lead concentrations in whole blood were determined by a Varian AA-40 atomic absorption spectrophotometer with a lead hollow cathode lamp and a deuterium lamp for background correction, coupled to a GTA-96 electrothermal

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atomizer and a programmable sample dispenser (Varian Techtron, Australia). Details of the analytical procedures have been reported previously (Al-Saleh 1990). The detection limit was approximately 0.4 $\mu g\,dl^{-1}$.

During this study, two types of quality control samples were incorporated to measure and maintain accuracy and reproducibility of the procedure. Internal quality control samples were prepared by spiking pooled fresh human blood, which had a very low background concentration of lead, using a method by Taylor (1988). Three sets of blood controls with known lead concentrations determined by a reference laboratory were obtained from the University of Surrey (Guildford, UK).

Parametric statistical analyses were used to analyze the data. The analysis of the data was accomplished with Statgraphics programs (Stratgraphics 1987). A confidence level of 95% was chosen to evaluate the calculated P value.

Results

The cumulative distribution plot of blood lead concentrations in the participating subjects ($n=202$) did not follow a Gaussion distribution and the bend of the curve occurs at about 12 $\mu g\,dl^{-1}$ but is positively skewed (1.59), see Figure 1. Participants with blood lead below 12 $\mu g\,dl^{-1}$ follow a normal distribution curve (Figure 2). Therefore

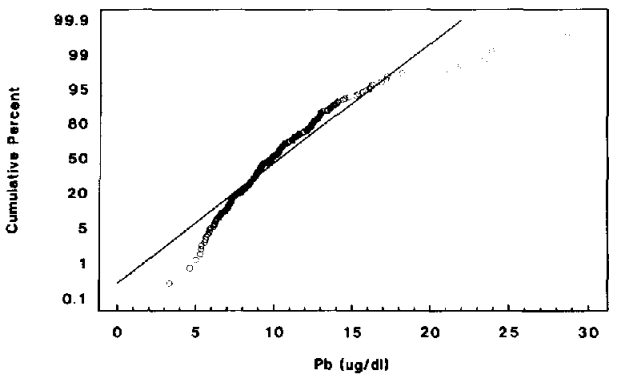

Figure 1. Cumulative frequency distribution plot of lead.

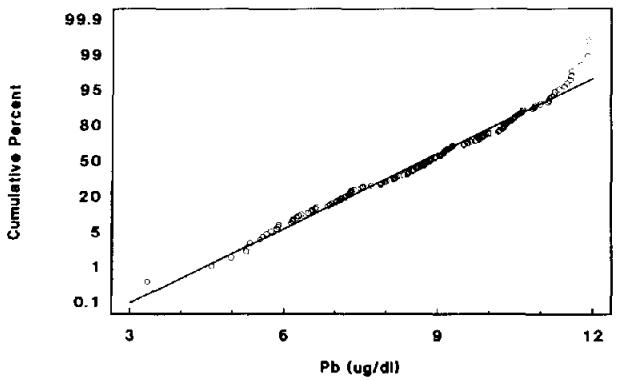

Figure 2. Cumulative frequency distribution plot for blood lead data less than 12 $\mu g\,dl^{-1}$.

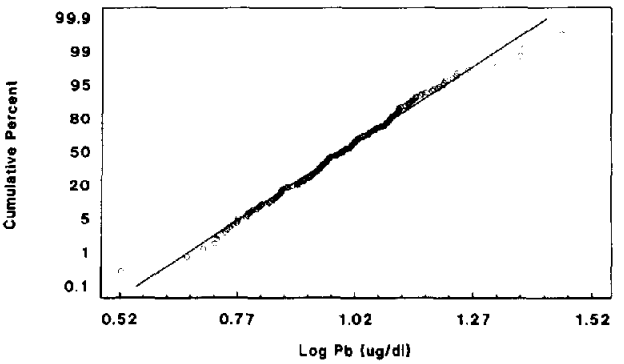

Figure 3. Cumulative frequency distribution plot of log-transformed lead.

Table 1. Mean blood lead concentrations ($\mu g\,dl^{-1}$) in different smoking groups

Group	n	Mean ± SD	Range
Non-smokers	55	9.51 ± 2.51	4.97–16.84
Previous smokers	11	8.53 ± 2.55	3.33–13.55
Current smokers	136	10.76 ± 3.87	4.59–28.65

Table 2. One-way analysis of variance: influence of smoking on blood lead concentrations

Source of variation	Sum squares	d.f.	Mean squares	F	P
Between groups	0.149	2	0.074	3.95	0.02
Within groups	3.751	199	0.019		
Total	3.90	201			

all blood lead measurements were subjected to log transformation to obtain approximate normality of their distribution (skewness = 0.135), see Figure 3.

Mean blood lead concentrations in different smoking groups are given in Table 1. One-way analysis of variance was used on log-transformed lead as shown in Table 2. However, this analysis is only valid if the variances are equal. Bartlett's test was used to ascertain the validity of this assumption. The test showed that the variances were equal ($P>0.05$). However, this test did not indicate which groups were different. To answer this, the least significant difference multiple comparison method was applied to log-transformed lead data which were used to compare all pairs of means by the t-test. At 95% confidence level, there was no significant difference between the non-smokers and previous smokers, whereas current smokers were significantly different from the non-smokers and previous smokers.

Age did not influence the mean blood lead concentrations and when correlations between blood lead concentrations and age among different smoking categories were calculated, no significant differences ($P>0.1$) were obtained. The mean blood lead concentrations among different smoking categories according to age groups are shown in Table 3.

Table 3. Blood lead concentrations by age group

Age group (years)	Mean	Blood lead concentration		
		current smokers	non-smokers	previous smokers
16–19	11.28 ($n=24$)	12.37 ($n=15$)	8.92 ($n=7$)	11.40 ($n=2$)
20–29	10.35 ($n=120$)	10.75 ($n=77$)	9.82 ($n=38$)	8.19 ($n=5$)
30–39	9.81 ($n=46$)	10.13 ($n=35$)	8.76 ($n=8$)	8.92 ($n=3$)
40 and over	9.63 ($n=12$)	10.54 ($n=9$)	8.70 ($n=2$)	3.33 ($n=1$)

Discussion

In this study, the blood lead data distribution in the screened subjects suggests the existence of two mixed populations among the participating subjects. This raised an important question—whether this related to the existence of two different types of lead exposure or due to other factors? A cut-off point of 12 $\mu g\,dl^{-1}$ was found where the two populations separate. This revealed that 73.3% of the participants had blood lead levels below 12 $\mu g\,dl^{-1}$, which represents the first population within the studied subjects (reference population). Subjects with blood lead above 12 $\mu g\,dl^{-1}$ (26.7% of the entire studied subjects) and in the range of 12.09–28.65 $\mu g\,dl^{-1}$ are considered as the exposed population.

The present study clearly demonstrates the existence of a significant difference in blood lead concentrations between smokers and non-smokers, confirming the results of previous studies (McLaughlin & Stopps 1973, Zielhuis *et al.* 1977, Grandjean *et al.* 1981, McIntosh *et al.* 1982, Shaper *et al.* 1982, Elinder *et al.* 1983, Pocock *et al.* 1983, Hense *et al.* 1992, Willers *et al.* 1992). Of the exposed population ($n=54$), 80% with blood lead concentrations above 12 $\mu g\,dl^{-1}$ were smokers. This could suggest that smoking constitutes the main source of lead exposure among the screened population. However, other sources such as air, water or food should not be ignored since they may have acted synergistically with tobacco smoke. Al-Saleh & Taylor (1994) found that lead concentrations in the air and soil of Riyadh city were significantly higher in heavy traffic areas since the amount of lead added to Saudi Petrol is higher (0.6 $mg\,l^{-1}$) than the maximum permissible levels in many industrialized countries.

In the non-smoking subjects ($n=55$), 18.2% had blood lead above 12 $\mu g\,dl^{-1}$. On the other hand, in the previous smokers group, only one subject had blood lead above 12 $\mu g\,dl^{-1}$. As in other countries (Willers *et al.* 1992), it seems that exposure to lead from tobacco smoke in Saudi Arabia is low, relative to other sources (e.g. food, traffic and industrial emissions). This probably explains the non-significant difference in the blood lead concentrations between the non-smokers and previous smokers groups.

Previous studies (Al-Saleh 1990) showed that lead concentrations rise substantially during childhood, reaching a peak between 3 and 5 years of age and then declining with increasing age to reach a minimum at about the age of 16 years. Consistent with another study by Mahaffey *et al.* (1982), this study showed a decrease in the mean blood lead concentration with increasing age.

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