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Lead Reduction of Petrol and Blood Lead Concentrations of Athletes

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ABSTRACT. In 1984, it was determined that the blood of long-distance runners in South Africa contained unacceptably high concentrations of lead. Subsequently, the petrol lead level in South Africa was reduced from 0.8 g/l to 0.4 g/l. In view of this reduction, a follow-up investigation of its effect on the blood lead concentration of South African runners was undertaken. Blood lead samples were analyzed by graphite furnace atomic absorption spectrophotometry. The mean values of blood lead concentrations dropped from 52 to 13 μg/dl and from 20 to 8.5 μg/dl for the urban and rural trainers, respectively. A highly significant decrease in blood lead levels was found and was mainly attributable to the reduction in the petrol lead levels. The blood lead results for rural trainers indicated that there still exists a certain degree of lead pollution in athletes from nonremote areas.

IN COUNTRIES where petroleum that contains high lead levels is still used, atmospheric lead pollution from vehicular exhaust aerosols is a major source of lead pollution.[1-3] Lead enters the circulation mainly after inhalation[2] and also via gastrointestinal absorption. Many variables in the absorption, storage, and excretion of lead modify blood lead concentration and, therefore, its effects.

Lead has long been known to be neurotoxic[1,2]; in fact, the clinical features of lead poisoning were fully described in 1839.[4] In a recent study, the long-term effects of exposure to low doses of lead were investigated, and the authors concluded that exposure to lead in childhood is associated with deficient central nervous system functioning that persists into young adulthood. In 1987,[6] the American Academy of Pediatrics Committee on Environmental Hazards, in an effort to prevent lead exposure in children, urged that all lead be immediately and completely removed from gasoline. The Academy also vigorously encouraged public agencies to develop safe and effective methods for the removal and proper disposal of all lead-based paint from public and private housing. Currently, it is generally believed that the hazardous effects of lead are more perceptible in children than in adults. In recent years, consensus on the toxic levels of lead has changed. The Agency for Toxic Substances and Disease Registry[7] reviewed studies published up to 1987, and it defined the

threshold for neurobehavioral toxicity to be 10–15 μg/dl, even though concentrations of less than 25 μg/dl whole blood were viewed as an acceptable norm.

An analysis of a chronological trend in blood lead levels[8] indicated that from February 1976 to February 1980, average whole blood levels in the United States dropped from 14.6 to 9.2 μg/dl (approximately 37%). The lead content of gasoline in the United States was reduced significantly (approximately 55%) during this period, which is the most likely explanation for the decrease in blood levels.

In Hong Kong, the effect of petrol lead reduction on the lead content of curbside dust was studied[9]; lead content in dust samples obtained in 1977 and 1987 was compared. The petrol lead content averaged 0.84 g/l in 1977 and was followed by a stepwise reduction to 0.25 g/l by early 1987 (i.e., 67% reduction in lead contamination of the roadside environment in Hong Kong). Use of petrol lead in Wales decreased during a 3-y period, the effect of which was a 52–61% reduction in atmospheric lead. However, a study[11] in which the lead content of petrol and petrol sales remained unchanged reported that the blood lead levels in a New Zealand population decreased about 43%; this reduction was ascribed to changes in dietary intake and the elimination of lead in domestic and industrial environments. Lead-free petrol provides a margin of safety for children before known toxic levels are reached[12]; however, the control of existing sources of lead (in paint) in and around housing (dust) is a more intractable problem[13] of lead pollution than is the control of new inputs.

In December 1985, after the British government decided to further reduce the maximum lead content of petrol from 0.40 g/l to 0.15 g/l, the Department of the Environment elected to monitor the effects from 1974 to 1987. Although the air levels at roadside sites had decreased by 55% in response to a 60% reduction in lead emissions from motor cars, there was no effect on blood lead concentrations of adults, and a minimal decrease of 1 μg/dl in the blood of children was noted. The reduction of petrol lead in Italy—from 0.6 to 0.4 g/l—also effected a significant reduction in air lead concentration,[15] but the effect on blood lead levels was not evaluated. In contrast, a reduction (i.e., from 0.4 to 0.22 g/l) of the petrol lead content in Athens[16] during a 3-y period resulted in a decrease in blood lead levels of about 18% in men and 30% in women. In 1978, and for the succeeding 11 y, the Belgian population's blood lead levels were monitored regularly. The median values of blood lead concentrations dropped from 17 to 7.5 μg/dl, in response to the stepwise reduction in gasoline lead from 0.75 g/l to 0.15 g/l.

We determined whole blood lead concentration in South African long-distance runners[18] who had participated in the 1984 Comrades Marathon, which is the premier ultra-marathon held annually in South Africa. The petrol lead content prior to and at that time was 0.8 g/l. The mean blood lead levels in 1984 were 20.1 μg/dl and 51.9 μg/dl for the rural and urban trainers, respectively. Thereafter, petrol lead was lowered to 0.6 g/l and, finally, in January 1989, it was reduced to 0.4 g/l,. In view of this reduction, we decided to conduct a follow-up investigation of the effect of the reduction in petrol lead, if any, on blood lead concentrations of South African long-distance runners.

Materials and methods

Blood lead levels of the Comrades Marathon (distance = 90 km) athletes who participated in the May 1990 event were determined. The sample, which was randomly representative of two groups of runners, was the same as in our previous study (1984 competition[18]). These groups, however, were completely separate from the 1984 runners. One group (urban trainers, n = 57, all males; mean age = 35.7 y; standard deviation = 7.6) consisted of city dwellers who made no effort to avoid heavily trafficked roads during their training. The other group (rural trainers, n = 30, all males; mean age = 33.1 y; standard deviation = 8.7) included athletes whose training was conducted in a rural environment; therefore, they had minimal exposure to vehicular traffic exhaust fumes, except during competitions in city areas. The control group was selected from adult urban male nonrunners (n = 31) who, in their daily routine, were not exposed to unusual atmospheric lead pollution. Persons who were occupationally exposed to lead or whose usual activities suggested that they were in contact with known sources of lead pollution (motor mechanics and lead workers, i.e., melting, battery plant, shipyard workers) were excluded (n = 10).

The specifications for the apparatus, procedure, glassware, water, and reagents were similar to those used by Subramanian and Meranger in their study; however, a few alterations were made, which are outlined in our previous article.[18] Briefly, 100 μl of whole blood, obtained by finger prick, was heparinized, to which ammonium phosphate Triton-x matrix modifier was added. The sample was then analyzed for lead by graphite furnace atomic absorption spectrophotometry.

Results

Three groups of subjects were included in this study, i.e., 1984 group,[18] 1990 group, and "controls." The 1984 and 1990 groups were further subdivided with respect to training and residence in urban or rural areas (Table 1).

This type of study design can be analyzed by a two-way analysis of variance, if most of the distributional assumptions and requirements are satisfied. The graphical display of blood lead levels evidenced that gross differences existed with respect to location (mean levels) and distribution shape among the six groups.

The structural heteroscedasticity (i.e., the spread associated with the different subsamples increased as the mean [location] increased) led us to apply a logarithmic transformation to the blood lead observations. Statistical comparisons of the mean blood levels of the different groups were conducted on the transformed data. All of the differences were statistically significant at a 1% level.

A highly significant difference was found between the 1984 and 1990 rural trainers and between the urban

Table 1.—Whole Blood Lead Levels (μg/dl Pb/dl) of Controls and Rural and Urban Area Trainers for the 1984 and 1990 Comrades Events

	1984 remote rural control (n = 30)	1990 urban control (n = 25)	1984		1990	
			Rural Tr (n = 29)	Urban Tr (n = 51)	Rural Tr (n = 30)	Urban Tr (n = 57)
Mean	3.4	9.7	20.1	51.9	8.5	13.0
Median	3.3	10.0	17.7	55.5	7.0	8.5
SD	1.5	4.1	10.6	16.7	5.0	6.2
IQR	2.1	4.3	18.4	26.4	4.0	5.7
Maximum	7.5	16.0	42.8	77.7	24.5	31.1
Minimum	0.5	3.0	4.2	19.9	0.6	2.2

Notes: Tr = trainers, *SD* = standard deviation, and IQR = interquartile range.

trainers. No significant difference was found among the 1990 urban trainers, rural trainers, and city controls. However, a significant difference was demonstrated between (a) 1990 rural trainers and remote rural controls,[19] (b) 1990 urban trainers and remote rural controls,[19] and (c) urban controls and remote rural controls.[19]

Discussion

To the authors' knowledge, this is the first controlled follow-up study to report any decrease in the blood lead levels of competitive runners since the petrol lead content was lowered.

The criteria employed in our previous study design[18] were strictly observed in this follow-up study to ensure their comparability. Only male nonsmokers who were not occupationally exposed to lead contamination and who did not take any medication were selected, a restriction that accords with the procedures used in our previous study.[18] All athletes had to qualify for this marathon event, and it was found that for several months prior to the event, they had trained an average of 7 h/wk.

One major factor that could have accounted partially for the magnitude of the blood lead decrease since 1984 was the increased public awareness of lead sources and toxicity. Other potentially confounding variables, e.g., lead in food, water, ceramic glazes, crystalware, and house paint, could not be excluded totally. However, the large decrease in blood lead levels in our city trainer group, which included athletes who made no attempt to avoid traffic during training, could not be explained by these confounders. Furthermore, during 1984 to 1990, South African authorities did not make any special effort to reduce lead in foodstuffs or in industrial or domestic environments. The only obvious factor that could have resulted in the reduction of blood lead levels was the lowering of gasoline lead content from 0.8 g/l to 0.4 g/l during the period under review.

The blood lead levels of urban trainers dropped remarkably and significantly (52 to 13 μg/dl [Table 1]). In Athens,[16] a 50% reduction of gasoline lead content (0.4 to 0.22 g/l) was associated with a 24% reduction in blood lead levels of its inhabitants. It could be argued that a more pronounced influence would be observed in runners because, during strenuous exercise, respiratory exchange rates are high and accompanied by an increase in all homeostatic variables.[20] For example, the rate of oxygen uptake by blood per minute may be increased to 4 000 ml, compared with a resting value of 250 ml.[21] In the United States, a decrease in gasoline lead content of approximately 55% resulted in a 37% reduction in blood lead levels (14.6 to 9.2 μg/dl) of its inhabitants.[8]

If the results obtained in 1984 and 1990 are compared, both rural and urban trainers showed a highly significant decrease ($p < .001$) in blood lead levels (Table 1). The decrease in blood lead levels of urban trainers was more dramatic than that of their rural counterparts. This probably reflects that rural trainers were only exposed to higher air lead levels when they participated in events held in the urban areas.

The mean blood lead levels of rural and urban trainers and urban controls did not differ significantly, once the petrol lead concentrations had been lowered (Table 1). However, the mean blood lead levels of all the groups before and after the reduction of petrol lead content differed significantly from those in the remote rural area (Table 1). This indicates the existence of a certain degree of air lead pollution in the respective areas from which the samples were chosen.

The release of bone lead deposits occurs very slowly. However, as this process continues, the blood lead levels should decline even further. In 1976, Rabinowitz et al.[22] studied lead turnover kinetics in the human body. They proffered a three-compartment model, which was later refined by Batschelet et al.,[23] who reported lead half-lives of 15.5, 34.7, and 600–3 000 d for blood, soft tissue, and bone, respectively. The blood of our 1990 groups was sampled 510 days subsequent to the official reduction in petroleum lead con-

tent. One could, therefore, expect a further decrease in the blood lead levels as lead is removed from deeper compartments.

In conclusion, the sharp decline in the blood lead levels of athletes was associated with a decrease in petrol lead levels. Whether a further reduction in petrol lead would result in any significant reduction in blood lead levels of athletes still remains uncertain.

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