

*LETTER TO THE EDITOR*

**EFFECT OF ENVIRONMENTAL LEAD POLLUTION ON HAEMOGLOBIN AND ERYTHROCYTE ALAD ACTIVITY**

Sir,

( Received on May 21, 1999 )

There has been a great concern in recent years regarding environmental pollution of lead mainly because of its use in petrol and steep increase in vehicular traffic (1). Environmental pollution by toxic metals, such as lead, is a much more dangerous problem than is pollution by organic substances, such as pesticides, because most organic substances are degradable by natural processes, while no metal is degradable. The metallic and elemental pollutants are going to stay in the environment for long time (2).

Lead is known to affect a number of enzymes and physiological systems which results in a wide variety of changes in humans, but those affecting the haematopoietic system are much more well known. Although, blood lead levels are used as a measure of body burden (3), alterations in porphyrin metabolism have provided a useful means of detecting and assessing the severity of lead exposure and poisoning (4).

Therefore, in the present study an attempt has been made to determine the effect of lead pollution in the ambient air of Jaipur metropolis directly by measuring the accumulated lead in blood of traffic policemen (who are exposed much more) and indirectly by gauging its inhibitory effect on erythrocyte ALAD (5-aminolevulinatase) activity.

The traffic police persons on the field duty for last 2 to 10 years (mean - 4 years) were the subjects of this study (lead-exposed group). Their age ranged from 22 to 41 years (mean - 29 years) and they were not suffering from any organic disease. In contrast, the subjects of unexposed group for comparison were obtained from the remote areas of Jaipur which is expected to be less polluted with regard to lead in the air. They were also all male subjects, varying in age from 24 to 42 years (mean - 30 years).

The blood for this study was obtained in the morning in the fasting state. Blood hemoglobin, total red blood cell count and ALAD assay (5) were performed immediately on collection. A small aliquot of blood was diluted with nitric acid - Triton X-100 solution and analysed by atomic absorption spectrophotometry (6).

The result of study are presented in Table I. The Hb, TRBC and ALAD were significantly less in lead-exposed people than unexposed persons, while blood lead was significantly increased. Thus average lead was 1½ times higher and average ALAD was only two-third (2/3) of normal.

Since lead pollution of air occurs mainly due to exhausts of motor vehicles using lead containing petrol, and use of unleaded petrol

TABLE I : Blood lead and ALAD values in lead-exposed and unexposed people

Group	Hb (g%)	× TRBC (10 <sup>6</sup> cu. mm.)	ALAD (U/ml)	Pb (ng/ml)
Unexposed (27)	13.5±0.7	4.33±0.65	125.0±32.7	275.9±53.4
Lead exposed (30)	12.1±1.5	3.72±0.61	89.9±56.2	508.3±230.6

Values represent Mean ± S.D.

All values are significantly different statistically (P<0.001).

in Jaipur city is negligible, hence, the blood lead burden and ALAD activity inhibition can be ascribed to this source of contamination only. W.H.O. (1) considers blood lead below 50 µg/100ml as safe and above 80 µg/100 ml as toxic. In our study 50% of traffic policemen had blood lead above safe limit, and 10% around toxic level or more. The increased lead may explain the observed symptoms of burning of eyes, itching of skin, gastric discomfort, respiratory problems and cough in some of the traffic policemen.

Lead exposure is known to cause anemia (1). Although, hemoglobin in lead exposed group was significantly less (P<0.001) than unexposed people, it cannot be termed

anemia. It is possible that anemia may develop in some of them in due course of time. That the reduction in hemoglobin was caused by lead accumulated in blood is supported by significant negative correlation between Hb and lead exposed group ( $r=-0.387$ ,  $P<0.05$ ), but not in non-exposed group ( $r=-0.220$ ,  $P>0.1$ ). It is further substantiated by significant negative correlation of blood lead with TRBC ( $r=-0.381$ ,  $P<0.05$ ) and a highly significant negative correlation between blood lead and ALAD activity in lead exposed group ( $r=-0.62$ ,  $P<0.001$ ). Therefore, we suggest that all people at risk of exposure to lead exhausts should be monitored for their blood ALAD activity which is a very easy and simple technique and a reliable parameter of exposure.

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#### REFERENCES

1. W.H.O. Lead - Environmental aspects. Environmental Health Criteria No. 85, Geneva, W.H.O. 1989.
2. Sharma DC. Essentiality and toxicity of certain trace elements : A strategy. In "Environmental strategies", (Ed.) R.M. Lodha, Udaipur, 1995, Himanshu Publications, p. 17-27.
3. W.H.O. Inorganic lead. Environmental Health Criteria No. 165, Geneva, W.H.O. 1995.
4. Sakai T, Morita Y. Delta-aminolevulinic acid in plasma or whole blood as a sensitive indicator of lead effects, and its relation to other heme-related parameters. *Int Arch Occup Environ Health* 1996;68: 126-132.
5. Nandi DL, Baker-Cohen KF, Shemin D. 5-aminolevulinic acid dehydratase of *Rhodopseudomonas spheroides*. I. Isolation and properties. *J Biol Chem* 1968;213:1224-1230.
6. Bishop ML, Duben-Engelkirk JL, Fody EP. Clinical Biochemistry, Principles, Procedures, Correlations. Philadelphia, Lippincott, 1992, p. 609-610.

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