Some Observations on the Intake of Inorganic Lead and Animal Growth

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Out of the heavy metals, cadmium, mercury and lead are commonly recognized as toxic contaminants of our environment. Many childhood deaths have occurred due to lead intoxication (CHISOLM, Jr. 1971). ZEILHUIS (1972) published an extensive report on blood lead (Pb B) values in the lead exposed nationals of various countries. On the other hand different sources of lead were analyzed (HAUKIN et al. 1973, JOSELOW et al. 1974, and SAYRE et al. 1974). Milk, polluted air, use of ceramic-drinking vessels, chipping paint and dust are now under consideration. However, determination of blood lead, urine lead values as well as radiological signs have been the diagnostic tools in the lead induced health hazards. We have made an attempt to establish a corelation between lead intake (through gut) and total body weight of albino rats. The hypothesis that "lead inhibits growth" have been justified discussing physiological and biochemical disorders thus caused.

Methods

A group of albino rats irrespective of sex but of the same age and weight (70 to 75 gms) were selected from the stock. They were housed in separate cages suitable to mark daily records. A standard diet containing lead nitrate was fed to them on alternate days with tap water ad libitum. The experiment continued for one month. After one month, rats were fed on a standard diet alone again for a period of 30 days. Control subjects were provided standard laboratory diet alone with free access to water. At random, five rats were selected from the lead fed group, after fifteen days of experiment. Biopsy specimen of liver and kidney were taken out and processed hitochemically to confirm the adsorption of lead (RANA 1972). The details of experimental procedure are illustrated by the Table 1.

Results and Discussion

After one month, a comparative study of both the groups was made. Rats were exposed to lead, were found to be smaller in size than the normal rats. The mean body weights in relation to the amount of lead consumed are exhibited by Fig. 1. As the internal lead load increased, the body weight decreased. Progressive increase of lead in diet resulted into the progressive decrease in the total body weight. It was surprising to note that no increase

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TABLE 1

Details of the procedure employed to feed different amounts of lead nitrate through gut to each rat.

Days	Amount of $PbNo_3$ suspended, in gms.	Total weight of diet in gms.	Amount of lead consumed in gms.	Average body weight in gms.
0	0.25	15.0	0.11	76
1	-	-	-	-
2	0.50	15.0	0.15	76
3	-	-	-	-
4	0.75	15.0	0.17	76
5	-	-	-	-
6	1.00	15.0	0.18	75
7	-	-	-	-
8	1.25	15.0	0.25	75
9	-	-	-	-
10	1.50	15.0	0.31	75
11	-	-	-	-
12	1.75	15.0	0.40	75
13	-	-	-	-
14	2.00	15.0	0.45	75
15	-	-	-	-
16	2.25	15.0	0.52	74
17	-	-	-	-
18	2.50	15.0	0.61	74
19	-	-	-	-
20	2.75	15.0	0.92	73
21	-	-	-	-
22	3.00	15.0	1.42	73
23	-	-	-	-
24	3.25	15.0	1.80	72
25	-	-	-	-
26	3.50	15.0	1.82	72
27	-	-	-	-
28	3.75	15.0	1.82	70
29	-	-	-	-
30	4.00	15.0	1.85	68
31	-	-	-	-
32	Nil	15.0	Nil	68
/				
60	"	"	"	"

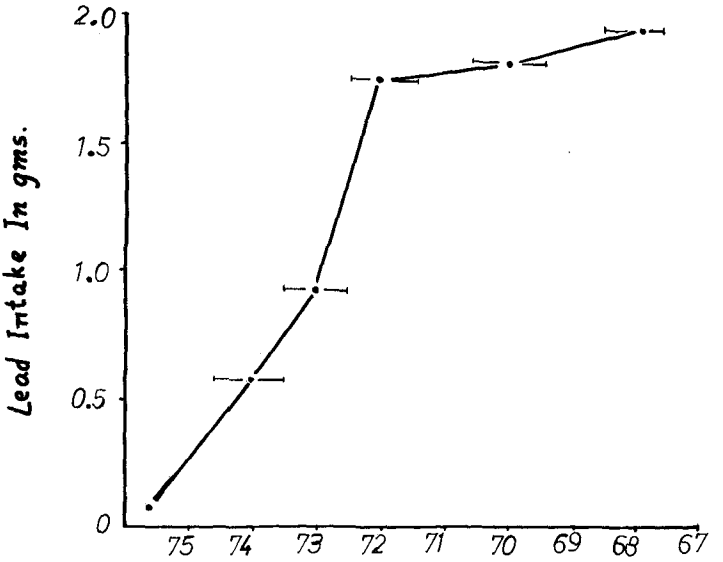

Fig. 1. The mean body weights in relation to the amount of lead consumed, are expressed by this figure. Progressive decrease in the total body weight is observed, when amount of lead is increased in the diet.

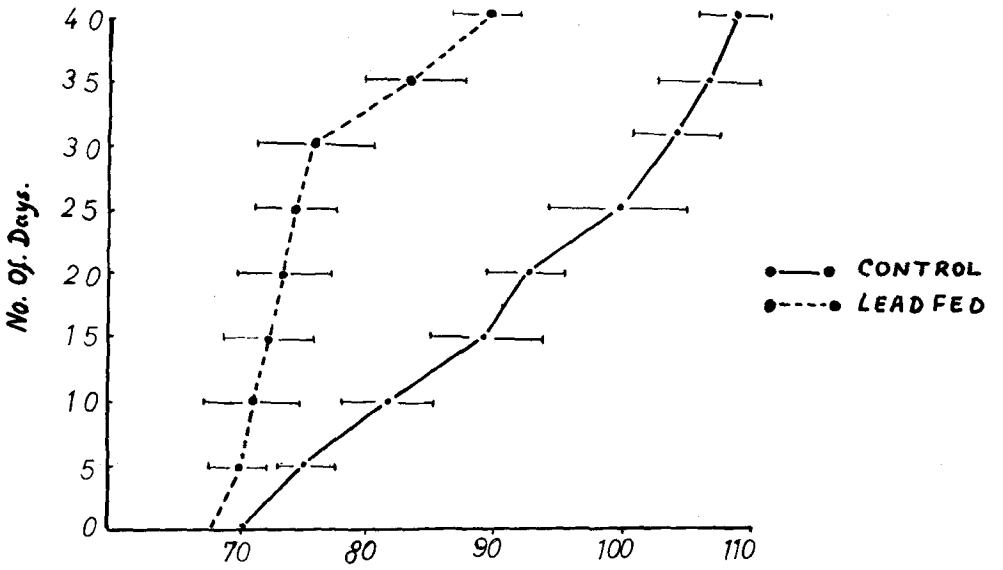

Fig. 2. Progressive increases in the body weight of rats is noticed after a quiet period of thirty days, when the same rats are provided a standard diet alone.

in the body weight was noted even after one month of standard diet feeding. We recognized this period as the quiet period. Mean body weight was calculated to be 68.0 gms. It remained constant for one month. However progressive increase in the weight again started after this period (Fig. 2). These findings with a population of 50 rats enforce us to conclude that lead has a temporary effect on animal growth.

Data from PENTSCHEW (1958) show that adult rats are unexpectedly resistant to lead poisoning. Contrary to this view, our rats were found quite sensitive to lead. MICHAELSON (1973) while performing experiments on suckling rats noted a 60% decrease in total body weight. This is the case when offsprings were taking the milk of mothers already exposed to lead. His findings are further confirmed here, though the adult rats were fed directly on lead nitrate. Moreover, a constant stop in the body weight was noted even when they were not taking lead through their diet.

The present study confirms adverse effects of lead on animal growth. However, if the subjects are provided a lead free environment, they recover after passing a quiet period that may fluctuate in different species and ages of subjects. Now the important question that triggers the mind, "what physiological changes are responsible for this check in growth", remains to be answered. Although experimental data are lacking, it is likely that lead causes greater inhibition to new cell formation. Specific effects of lead on normal metabolism of porphyrin and heme synthesis has been extensively studied by KELLIHER et al. (1973). An essential biochemical evidence is the inhibition of porphyrin synthesis at specific enzyme sites with the accumulation of -aminolevulinic acid. Further, this biochemical disorder is accompanied by excess of porphyrobilinogen and cproporphyrin. None of these three metabolites have been reported to be toxic. However a casual metabolic results into lead encephalopathy. Moreover, lead interferes with the respiratory enzymes of endothelial cells and prevents these cells in taking part in the increased metabolic demand of the developing tissues. It is interesting to note that lead shortens the life span of erythrocytes (HERNBERG 1967, PASSOW 1970). Not only this ERICH and WALLER (1967) concluded from their own investigations that the premature splenic sequestration of circulating erythrocytes in lead intoxication is a consequence of disturbed metabolism caused by lead.

When the same subjects are provided with healthy environment after passing a period of thirty days they start recovering without any therapeutic measures. It means metabolic disorders were brought to normal. At present we can also comment that lead exposure resulted into inhibition of erythrocyte mechanism for glutathione synthesis (ROEL et al. 1975). However recent studies of BONDI et al. (1975) have shown that human erythrocytes in whole blood may renew their whole glutathione content in about six days. The same process may be true with these rats. Regeneration of depleted glutathione content again regulates ALAD activity of erythrocytes.

However further experimental evidence is necessary to confirm the hypothesis. Histochemical evidence (RANA 1972) showed that lead is absorbed in liver, kidney, spleen and even thyroid of squirrels after feeding them on lead nitrate. What factors are responsible for the mobilization of absorbed lead needs much attention. It was also reported (RANA 1974) that lead causes depletion and distortion in the enzymorphology of certain tissues. We conclude, once the case of lead poisoning is confirmed by simple tests, regular observations of body weight may also serve the purpose of discriminating criterion for estimation of the degree of exposure to lead.

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