The Effect Of Living In Polluted Environment: A Case Study Of Albino Mice Subjected To Lead Assimilation At Three Different Sources

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Abstract

Objective: The effect of lead and other environmental pollution due to heavy metals is one of man's major problems in the health sector. This paper aims at evaluating the physiological effect of lead in albino mice as a relative measure of estimating leads non beneficial property in man.

Methodology: Albino mice were reared in cages and fed adlibitum in the laboratory for two weeks before lead sulphate was administered into their food, water, food and water and in the surrounding. Control mice were fed with normal food and water (no lead added) at the same time of treatment.

Result: Of the total number of mice subjected to treatment (n=30) with lead sulphate 12 died of lead poisoning. The percentage mortality rate recorded was 33.3%. It was also recorded that albino mice in treatments GBX, GBW and GBY had their weight reduced by -12.9g, -18.0g and -8.7g respectively. The mice in the control treatment did not suffer weight loss rather they increase in weight by 20.7g. the effect of lead by inhalation also showed mild effect even though there was slight weight increase (6.6g) and low percentage mortality (8.7%).

Conclusion: The physiological and psychological effect of lead and other environmental consequences of lead pollution are highlighted.

INTRODUCTION

The heavy metal ‘lead' is a common toxic mineral and the most abundant contaminant of our environment and our body. It is the worst and most widespread pollutant, though luckily not the most toxic. It occurs more in North America than anywhere else in the world. In the United States alone, it is estimated that approximately 1.3 million tons of lead are used yearly in the manufacture of solder, pottery pigments, gasoline, paint, and many other useful substances. Somewhere between 400,000 and 600,000 tons of lead per year go into our atmosphere, onto our earth, into our food, and into our body and tissues ([1]).

Exposure to lead occurs primarily through drinking water, food, airborne lead-containing particulates and lead-based paints. The burning of fossil fuels (especially lead-based gasoline), municipal waste and incorrect removal of lead-based paint results in airborne lead concentrations. After lead is airborne for a period of ten days it falls to the ground and becomes distributed in soils, plants and water sources (fresh and salt water surface, well water and drinking water) ([2,3,4,5]). However, the primary source of lead in drinking water is from lead-based plumbing materials ([4]).

Cigarette smoke is also a significant source of lead exposure; people whom smoke tobacco or breathe in tobacco smoke may be exposed to higher levels of lead than people whom are not exposed to cigarette smoke ([6]).

Another source of lead pollution is tetraethyl lead added to all gasoline; after combustion this lead goes directly into the atmosphere as air pollution and is inhaled by us and other living breathing entities; heavily traveled roadways show higher concentrations of lead in the air, soil, and nearby vegetation especially those grown near industrial areas or busy cities or roadways. Grains, legumes, commercial and garden fruit, and most meat products pick up some lead in higher concentrations than vegetation growing in more

secluded areas ([2]).

When lead levels become too high due to inhalation or ingestion, it can prove fatal and return our body to its origin in the earth as it circulates in the blood stream and distributes primarily in the soft tissues (kidneys, brain, lungs and muscle) and bone. About 73-95% of absorbed lead has been reported in bones of animals ([4]).

Though this is not completely clear, lead most likely interferes with functions performed by essential minerals such as calcium, iron, copper, and zinc. Lead does interrupt several red blood cell enzyme systems, including delta-aminolevulinic dehydratase and ferrochelatase. It is known to bind with the sulfhydryl bonds and inactivates the cysteine-containing enzymes, thus allowing more internal toxicity from free radicals, chemicals, and other heavy metals. Especially in brain chemistry, lead may create abnormal function by inactivating important zinc-, copper-, and iron-dependent enzymes. When body levels of these three minerals are high, there is first less absorption of lead and then more competition with lead for enzyme-binding sites. It may also diminish hemoglobin synthesis and can react with cell membranes. This may cause increased permeability of the cells and damage or even death of the cells ([7]).

Early signs of lead toxicity may be overlooked, as they are fairly vague: headache, fatigue, muscle pains, anorexia, constipation, vomiting, pallor and anemia. These can be followed by agitation, irritability, restlessness, memory loss, poor coordination and vertigo, depression, irreversible brain damage ‘encephalopathy' (a syndrome that arise from advanced chronic toxicity), seizure coma, decrease birth rate and death if not treated immediately ([4]). Neuropsychological impairment has been shown to occur in individuals exposed to moderate levels of lead ([8]). These are characterized by poor balance, confusion, hallucinations and speech and hearing problems.

The kidneys are targets of lead toxicity and prone to impairment with both acute and chronic nephropathy as major effects of lead toxicity. These effects are known to increase death rates among occupationally exposed individuals. Kidney impairment can be seen in morphological changes in the kidney epithelium, increases in the excretion rates of many different compounds, reductions in glomerular filtration rate, progressive glomerular arterial and arteriolar sclerosis and an altered plasma albumin ratio ([9], [10]). Other signs/symptoms of lead toxicity include gastrointestinal disturbances-abdominal colic-like pain, nausea and vomiting, cramps and muscle weakness, constipation, anorexia and weight loss-immunosuppression and slight liver impairment. Immunosuppression lowers host resistance to bacteria and viruses and thus, allows an increase in infection. It may also influence our cancer risk ([4]).

Children are susceptible to the most damaging effects of lead toxicity. Prenatal and postnatal developments are compromised significantly by the presence of lead in the body ([11]).

MATERIALS AND METHODS

Eight weeks old albino rats (n=30) were reared in cages, fed adlibitum and allow to acclimatize in the laboratory, during which they were observed for latent physiological disorders due to micro organisms for possible treatment before the commencement of the experiment. The rats were divided into two treatment blocks (GA and GB). GA had 6 control rats while GB had 4 replicate treatments each containing 6 rats. All the rats were weight and initial readings recorded.

The GA rats were fed with normal food supplement while the GB rats had 50mg/kg body weight of lead administered into their fed for a period of five weeks. The lead was administered using four different routes (in feed, in drinking water, in feed and drinking and by inhalation). The weight of rats was determined at interval of seven days for a period of five weeks and the average weight was recorded as mean weight.

A rat was selected at random from each of the treatment and control and sacrificed (dissected). The liver, lungs and kidney were collected using standard procedures ([12]) and fixed in bouins fixative for 24 hrs to arrest tissues metabolism and autolysis. The tissues were passed through 70% alcohol for 24 hours and there after into 90% alcohol for 12 hours for the cells to dehydrate. The preparation was allowed to remain over night for complete dehydration. Excess alcohol was removed from the tissue with xylene.

The tissues were then harden with Paraffin wax and allowed to solidify for 24 hours before sectioning. The sections were passed on a hot plate for 10 minutes to melt and the preparation washed with xylene and 90% alcohol. Water was use to rinsed the excess alcohol and the preparation was stained with eosin for 3 minutes before observation was

carried out under the X10 and X40 objective lens([12]). The result was statistically analyzed using chi-square test to determine the effect of lead in living tissues.

RESULTS

The four treatments (table 1) showed considerable decrease in weight with increase period of exposure. The control experiment showed an enhanced weight increase of 204.5g for the five weeks of treatment. The average weight gain of 200.4g was more (20.7g) than 183.8g recorded at the onset of the experiment, while GBW, GBX, and GBY recorded tremendous decrease in weight of -12.9g, -18.0g, and -8.7g respectively.

Figure 1

Table 1: effect of lead on the average body weight of experimental rats

	Average weight								
Group	at onset	Week 1	Week 2	Week 3	Week 4	Week 5	Mean	Weight Difference	SEM
GA	183.8	186.4	187.6	190.9	226.5	231.2	204.5	20.7	9.1
GBW	131.1	130.7	126.8	119.9	110.3	108.2	119.2	-12.9	4.4
GBX	160.9	155.2	148.2	140.2	132.6	128.5	140.9	-18.0	5.3
GBY	165.1	137.5	129.5	128.6	119.5	140.3	131.1	-8.7	5.3
GBZ	147.6	150.1	151.1	152.1	155.4	157.3	153.2	6.6	6.4

Foot note: GA = control
GBW= lead in water
GBX= lead in feed
GBY= lead in water and feed
GBZ= lead in cage (inhalation)

Among the GB rats, GBX showed high decrease in weight (-18.0g) than the other groups. It was observed that GBZ rats did not suffer weight loss (153.2) like the GBW, GBX and GBY which had their mean weights decreased to 119.2, 140.9 and 156.4 respectively. The relative loss in weight for the GBW GBX and GBY was -12.9g, -18.0g, and -8.7 respectively. The difference in weight of the control and treatment variables are presented in figure 1 where all the GB treatments weigh less than the GA with the exception of GBZ which had a slight increase of 6.6g.

The effect of lead recorded in table 2 shows the percentage mortality of rats. GBX and GBY had the highest mortality rates of 33.3% each and GBW and GBZ recorded 25% and 8.3% mortality rates respectively. The control was observed to have a zero percent mortality.

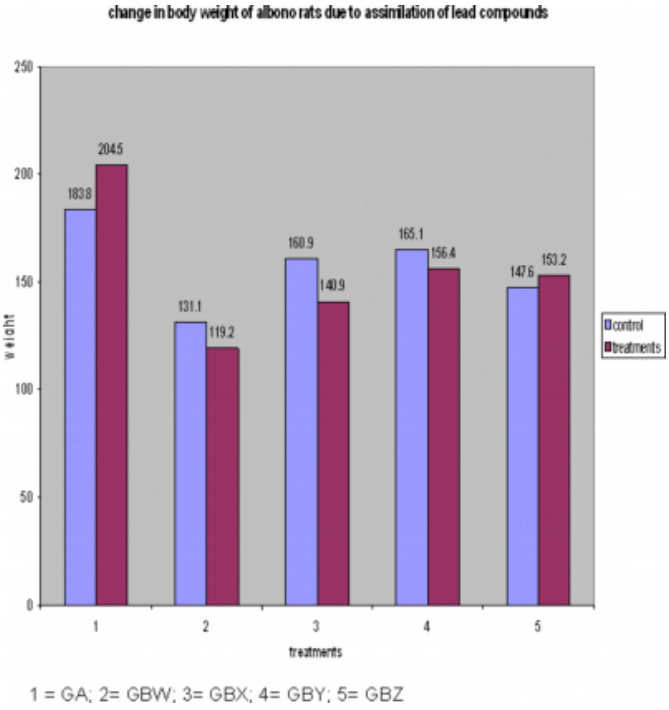
Figure 2

Table 2: rate of mortality recorded due to lead assimilation

Group	Number of rats	Number that died	% mortality
GA	6	0	0.0
GBW	6	3	25.0
GBX	6	4	33.3
GBY	6	4	33.3
GBZ	6	1	8.1
TOTAL	36	12	33.3

Foot note: GA = control
GBW= lead in water
GBX= lead in feed
GBY= lead in water and feed
GBZ= lead in cage (inhalation)

Figure 3



1 = GA; 2= GBW; 3= GBX; 4= GBY; 5= GBZ

DISCUSSION

The high decrease in weight recorded by the GBW, GBX, and GBY shows the most common routs of lead pollution. Further more, the high rate of mortality recorded (33.3%) by the GBX and GBY substantiates this fact. The death of rats confirms the high toxic effect of lead, and it is therefore, believed that the death might not be unconnected to acute lead encephalopathy, gastrointestinal disorder and immunosuppression of the defense system as reported ([4,7,13,14,15,16]).

Lead is very toxic (non beneficial to living organism) even at

low concentrations after prolong exposure ([3], [16]). It inhibits the catalytic function of most enzymes in the body. Lead is shown through X-rays to form layers in bones and other tissues of the body. This subsequently leaks out into the body thereby disrupting major physiological functions or causing physiological disorders and even death.

Because of leads high use in commercial quantity as an additive for motor fuel and leaded batteries for energy generation, the increase and continues use of lead in most countries is unquestionable. It is known to increase the octane number of fuels which reduce engine knocking tendencies and permits high engine compression capacity. But the environmental effect resulting from leads volatile compounds discharged out with exhaust fumes has serious consequences on animal life ([7], [15],)

Man is exposed to lead through mining, welding work, abrasive blasting, paints, manufacturing and disposal of car batteries, maintenance and repairs of bridges. In a nutshell, the industrial revolution has exposed large number of workers to lead poisoning. Few of the many lead effect to life are sterility, stillbirth, congenital problem, convulsion and mental retardations ([16]).

The accumulation of lead in vulnerable tissues causes toxic manifestations and the low elimination rate associated with extensive binding to ions is responsible for the cause of accumulation ([17]). Kidney could get damage when exposed to about 40µg/dl of lead, while neurodamage and anemia, could occur at high lead levels of 60µg/dl. Also, long term exposure to lead is linked to high blood pressure and stroke ([18]). High lead levels (150µg/dl) and above could course acute lead encephalopathy, headaches, constipation and abdominal pains, diarrhea and loss of appetite. Convulsion and paralysis are not exception and these may lapse into death.

The replacement of vital minerals such as calcium, potassium e.t.c and binding with the blood is believed to reduce oxygen carrying capacity of the cells. This is what makes red blood cells to become brittle and destroyed more rapidly thereby producing anemia and impairing the synthesis of hemoglobin in haemopeotic tissues ([14]).

More positive things we can do to reduce lead problems in our body include eating a wholesome diet with plenty of fresh fruits, vegetables, and whole grains to obtain adequate minerals, avoiding refined foods, and possibly taking a mineral supplement so as to competitively reduce lead absorption. Calcium and magnesium do this well, so a good level of these minerals in our diet, as well as supplements, can reduce lead contamination. Other preventive measures aim to restrict lead exposure involves awareness of increased lead contamination potential such as:

- Do not exercise along freeways or in heavy traffic.
- Do not allow children to play near busy streets.
- Do not store food in pottery.
- Avoid soldered cans, which are mostly the tin cans.

CONCLUSION

In the 1920s, tetraethyl lead was added to gasoline as an antiknock, and higher-octane additive. This has probably been the most widespread and pervasive source of environmental contamination from lead to date. Other common uses for lead are as seals for tin cans, in ceramics and pottery glazes, in insecticides, and more. Attempt to decrease this environmental contamination should be carried out by enacting laws to stop the usage of leaded substances. Cars should use unleaded gasoline. This does not, of course, eliminate the problems of carbon monoxide and burned hydrocarbons, but it will help to decrease lead exposure in the future. Another issue to be handled is to band cigarette smoking in public places and the use of pesticides containing lead should be band. It is recommended that occupationally exposed individuals should undergo the oral chelating therapy by using EDTA (a synthetic amino acid).

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