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Influence of Dietary Calcium on Subacute Lead Toxicity in the Rat

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Abstract: An investigation of the influence of dietary calcium (0.3% and 2.7) on the toxicity of dietary lead (50 mg L^{-1}) in the young male rats in a six week period indicated that as dietary calcium increased, the severity of lead toxicity decreased. Evidence include decreased lead concentration in kidney, liver, blood, brain and femurs, accompanied by a distribunces in essential metal (Ca, Zn, Cu, Fe and Mg) levels in kidney, liver brain, femur muscles and blood.

Key words: Lead, calcium, rats

Introduction

Calcium is an essential mineral that plays an important role in the development and maintenance of bones and teeth and additional important roles in nerve conduction, muscle contraction and blood clotting (O'Brien, 1998; Ginty *et al*., 1998).

The relationship which exist between calcium and lead are complex. Six and Goyer (1970) and Korolev and Sukhanov (1996), demonstrated that body retention of lead and the severity of clinical lead toxicity are inversely related to the level of dietary calcium. Increased dietary calcium levels appeared to decreased both the elimination and intestinal absorption of lead (Meredith *et al*., 1977; Quarterman *et al*., 1978). It is evident that the effect of dietary calcium on lead toxicity could be mediated by changes in intestinal lead absorption, tissue distribution, elimination, or any combination of these factors (Meredith *et al*., 1977).

The purpose of the present study is to clarify the effect of increased dietary calcium level in subacute lead toxicity in rats by determining the concentrations of lead as well as the concentrations of essential metals (Ca, Zn, Cu, Fe and Mg) in kidney, liver, brain, femur muscles and blood.

Materials and Methods

50 male albino Sprague-Dawely rats initially weighing 45 to 55 grams were assigned to these experiments. Rats were housed in individual stainless steel cages and were allowed to adapt to the laboratory environment for one week. A 12-h tight dark cycle was maintained. The rats were randomly assigned to one of six treatment groups, with ten rats per group. Rats of all groups were received a basal diet which were mixed in stainless steel bowls and containing 200 g wheat grains mixed with 200 g dried milk powder (Ca content = 430 mg) mixed by few drops of water to form a coarse paste mixture. Rats of group (I) were received the basal diet and had a drinking water after adding to it sodium acetate trihydrate at an equivalent concentration of acetate (2.85 mg L^{-1}) which added to the other groups in the form of lead acetate. Together with the basal diet rats of group (II) received 50 mg L^{-1} lead acetate in drinking water. Rats of group (III) were received 5 g calcium carbonate added to heir basal diet, while rats of groups (IV) were received 50 g calcium carbonate added to their basal diet paste. In groups (V, VI) rats were received 50 mg L^{-1} Pb acetate in their drinking water. The choice of these Ca and Pb concentrations was based on the results of previously reported studies of Bogden *et al*. (1991, 1992) using identical concentrations to assess the effects of Ca on Pb toxicity.

The length of the experiment was 6 weeks. Six rats from each group were killed at the end of the 6 week, blood was withdrawn, under light ether anaesthesia, by cardiac puncture and was collected into sterile heparinized closed polyethylene tubes. After blood collects rats were completely anaesthetized and sacrificed to collect selected tissues (kidney, liver, brain and femur skeletal muscles) and were stored in elastic containers at 3°C to metal analysis.

Organ concentrations of Pb, Ca, Zn, Fe, Cu and Mg we determined by previously described techniques of Slander and Cramer (1968) using flame atomic absorption spectrophotometer (Pye-Unicam A.A.S.SP-1900 model) after digestion with nitric acid and perchloric acid (5:1 ratio).

Data were analyzed by ANOVA. If ANOVA indicated that there was significant ($p < 0.05$) differences among the six treatment groups, pairwise comparison were made using Duncan's multiple range test (Wallenstein *et al*., 1980) between control and diet II, III and V and between diet II and diet IV and diet VI.

Results

Metal concentrations for kidney, liver, brain, femur muscles and blood are shown in Table 1-5.

The distribution of lead observed in this study indicate that kidney, liver and femur muscles retained the largest portion of lead followed by blood and brain. Increasing dietary calcium from 0.3 to 2.7% can alleviate many of the toxic effects of lead as indicated by the decreasing of lead levels in most of the organs studied. On the other hand excess dietary calcium with 50 mg/Pb/L caused a distribunces in most of the essential minerals studied (Fullmer and Rosen, 1990).

Discussion

The present results provide evidence that lead is accumulated in the following order: kidney > blood > liver > brain. Victery *et al*. (1979), Maitani *et al*. (1986), Nolan and Shaikh (1992) and Han *et al*. (1996), suggestsed that this element is strongly bound to macromalecules in the intracellular compartment because lead binding proteins have been isolated from the kidney, liver, blood and brain (Mistry *et al*., 1986; Guilarte *et al*., 1994; Han *et al*., 1996; Raghavan and Gonick, 1977).

In the present data oral lead administration for 6 weeks caused a decrease in Ca level in all of the studied organs and blood. This is agreement with different animal research which indicated that lead decreases calcium absorption (Chai and Webb, 1988), decreases serum calcium level (Hsu *et al*.,

Table 1: Concentrations of minerals in the kidney of rats consuming two levels of calcium and 50 mg Pb/L for 6 weeks

Experimental groups	Pb	Ca	Zn	Cu	Fu	Mg
	g/g wet tissue weight					
Control {Basal diet)	0.08 ± 0.018	3.61 ± 0.360	5.3 ± 0.05	0.03 ± 0.003	1.94 ± 0.16	1.22 ± 0.12
Diet II (Basal diet + 50 mg Pb/L)	7.30 ± 0.7*	4.98 ± 0.050*	0.12 ± 0.01*	0.02 ± 0.002	0.82 ± 0.06*	1.71 ± 0.17
(3) Diet III (Basal diet + 0.3 % Ca supplements	0.05 ± 0.3	5.50 ± 0.32*	0.219 ± 1.19*	0.03 ± 0.29	0.89 ± 0.03*	0.05 ± 1.19*
(4) Diet IV (Diet III + 50 mg Pb/L)	2.30 ± 0.19*	3.17 ± 0.37*	0.14 ± 0.01"	0.02 ± 0.002	0.61 ± 0.07*	1.14 ± 0.11
(5) Diet V (Basal diet +	0.01 ± 0.22	6.64 ± 0.564*	0.14 ± 0.01*	0.01 ± 0.02*	0.79 ± 0.01*	0.31 ± 1.28*
(6) Diet VI (Diet + 50 Mg Pb/L)	0.59 ± 0.053*	6.64 ± 0.564*	0.15 ± 0.02*	0.02 ± 0.002	1.85 ± 0.2*	1.00 ± 0.1

Each value represents the mean of six experiments ± standard error. All experimental intervals (6 experiments) are included in ANOVA. Mean values that differ significantly ($p < 0.05$) from control or lead treated groups are indicated by astersis.

Table 2: Concentrations of minerals in the liver of rats consuming two levels of calcium and 50 mg Pb/L for 6 weeks

Experimental groups	Pb	Ca	Zn	Cu	Fu	Mg
	g/g wet tissue weight					
Control (Basal diet)	0.05 ± 0.005	0.49 ± 0.05	0.85 ± 0.07	0.07 ± 0.07	3.36 ± 0.28	3.62 ± 0.36
Diet II (Basal diet + 50 mg Pb/L)	1.45 ± 0.10*	3.95 ± 0.40*	0.16 ± 0.01*	0.03 ± 0.003*	0.08 ± 0.05*	2.11 ± 0.211
Diet III (Basal diet 3% Ca supplements)	0.04 ± 0.004	0.95 ± 0.001 *	0.186 ± 0.99*	0.02 ± 0100*	0.919 ± 10.10*	1.84 ± 0.98*
Diet IV (Diet III + 50 mg Pb/L)	0.59 ± 0.59*	3.95 ± 0.40*	0.14 ± 0.01*	0.04 ± 0.004*	0.89 ± 0.07*	1.31 ± 0.13*
Diet V (Basal diet +	0.03 ± 0.004	1.14 ± 0.09*	0.02 ± 0.51*	0.03 ± 0.21.	0.73 ± 0.3*	0.58 ± 0.77*
Diet VI (Diet V + Mg Pb/L)	0.17 ± 0.017*	4.49 ± 0.454"	0.19 ± 0.01*	0.06 ± 0.005.	1.40 ± 0.11*	

Each value represents the mean of six experiments ± standard error. All experimental intervals (6 experiments) are included in ANOVA. Mean values that differ significantly ($p < 0.05$) from control or lead treated groups are indicated by astersis.

Table 3: Concentrations of minerals in the brain of rats consuming two levels of calcium and 50 mg Pb/L for 6 weeks

Experimental groups	Pb	Ca	Zn	Cu	Fu	Mg
	g/g wet tissue weight					
Control (Basal diet)	0.23 ± 0.023	0.96 ± 0.10	2.2 ± 0.2	0.05 ± 0.005	1.19 ± 0.11	2.09 ± 0.21
Diet II (Basal diet 50 mgPb/U	1.80 ± 0.10*	0.79 ± 0.082*	0.14 ± 0.01*	0.01 ± 0.001*	0.36 ± 0.03*	0.58* ± 0.06
Diet III (Basal diet + 3% Ca supplements	0.19 ± 0.08	1.19 ± 0.01*	0.19 ± 2.73*	0.03 ± 1.00*	0.88 ± 0.10*	0.74 ± 0.06*
Diet IV (Diet III + 50 mg Pb/L)	0.41 ± 0.041*	0.75 ± 0.05	0.09 ± 0.01*	0.02 ± 0.002*	0.42 ± 0.04*	0.83 ± 0.090*
Diet V (Basal diet + 2.7% Ca supplements)	0.18 ± 043	1.99 ± 0.01*	0.22 ± 0.43*	0.04 ± 0.99*	0.72 ± 0.2	0.56 ± 0.07*
Diet VI (Diet V + 50 Mg Pb/L)	0.25 ± 0.025*	0.80 ± 0.08	0.12 ± 0.01*	0.01 ± 0.001	0.65 ± 0.06*	1.92 ± 0.19*

Each value represents the mean of six experiments ± standard error. All experimental intervals (6 experiments) are included in ANOVA. Mean values that differ significantly ($p < 0.05$) from control or lead treated groups are indicated by astersis.

Table 4: Concentrations of minerals in the femur of rats consuming two levels of calcium and 50 mg Pb/L for 6 weeks

Experimental groups	Pb	Ca	Zn	Cu	Fu	Mg
	g/g wet tissue weight					
Control (Basal diet)	1.31 ± 0.13	0.47 ± 0004	0.93 ± 0.09	2.70 ± 0.24	0.05 ± 0.05	0.365 ± 0.34
Diet II (Basal diet + 50 mg Pb/L)	5.59 ± 0.56*	0.52 ± 0.05*	0.14 ± 0.01*	0.75 ± 0.07*	0.02 ± 0.002*	0.26 ± 0.000*
Diet III (Basal diet + 3% Ca supplements	1.20 ± 0.42	1.09 ± 0.20*	0.123 ± 0.01*	1.05 ± 0.10*	0.04 ± 1.10*	1.05 ± 0.10*
Diet IV (Diet III + 50 mg Pb/L)	4.10 ± 0.41*	1.20 ± 0.12*	0.07 ± 0.008*	0.70 ± 0.06*	0.03 ± 0.003*	0.71 ± 0.07*
Diet V (Basal diet + 2.7% Ca supplements	1.71 ± 0.23	1.20 ± 0.14*	0.42 ± 0.43*	0.324 ± 0.20*	0.04 ± 1.10*	0.32 ± 0.20*
Diet VI (Diet V + 50 Mg Pb/L)	1.03 ± 0.10*	2.25 ± 0.23*	0.18 ± 0.02*	2.99 ± 0.1 *	0.04 ± 0.004*	2.60 ± 0.26*

Each value represents the mean of six experiments ± standard error. All experimental intervals (6 experiments) are included in ANOVA. Mean values that differ significantly ($p < 0.05$) from control or lead treated groups are indicated by astersis.

1975), increases urinary calcium excretion (Victery *et al*., 1986), inhibits mitochondria uptake of calcium in the brain (Goldstein, 1977) displaces, calcium in mitochondria and interferes with calcium messenger system (Bressler and Goldstein, 1991).

A marked decreasese in the tissue icron contents were reported among rats treated with lead. A depletion in iron observed that this study came in accordance with the reports of Kochen and Greener (1975) and Victery *et al*. (1986) who indicated that lead competes with iron for f erritin binding sites end increased

Table 5: Concentrations of minerals in the blood of rats consuming two levels of calcium and 50 mg Pb/L tor 6 weeks

Experimental groups	Pb	Ca	Zn	Cu	Fu	Mg
	o/g wet tissue weight					
Control (Basal diet)	0.23 + 0.123	0.72 + 0.07	0.018 + 0.002	0.05 + 0.005	0.63 + 0.05	0.165 + 0.016
Diet II (Basal diet + 50 mg Pb/L)	3.16 + 0.30*	0.25 + 0.25*	0.014 + 0.001	0.001 + 0.0001*	0.24 + 0.02*	0.680 + 0.070*
Diet III (Basal diet + 3% Ca supplements	0.25 + 0.22	3.612 + 0.23*	0.001 + 0.001 *	0.001 + 0.002*	0.26 + 0.02*	0.09 + 0.09*
Diet IV (Diet III + 50 mg Pb/L)	0.22 + 0.022*	5.29 + 0.53*	0.010 + 0.001	0.03 ± 0.003*	0.28 + 0.02*	0.46 + 0.05*
Diet V (Basal diet + 2.7% Ca supplements	1.31 ± 0.041	3.34 ± 0.41*	0.011 ± .0.002	0.001 + 0.001*	0.16 + 0.05*	0.106 + 0.04*
Diet VI (Diet V + m Pb/l)	0.16 + 0.016*	4.29 + 0.43*	0.015 + 0.002	0.02 + 0.002*	0.60 + 0.05*	0.30 + 0.03*

Each value represents the mean of six experiments ± standard error. All experimental intervals (6 experiments) are included in ANOVA. Mean values that differ significantly ($p < 0.05$) from control or lead treated groups are indicated by astersis.

urinary iron excretion.

A decrease in zinc levels were observed between rats treated with lead. The results of Michaelson and Sauerthoff (1973), El-Gazzar *et al*. (1978) and Miller *et al*. (1984) demonstrated that high lead intake decreases zinc level in blood, liver, brain and kidney.

The present data showed lower copper levels in all studied tissues than those of control after oral lead administration. Limited data propose that lead interferes with copper utilization (Van Compen, 1971). It has been reported to increase urinary copper excretion in rats (Victery *et al*., 1986) and decrease brain levels of copper in suckling rats (Michaelson and Sauerthoff, 1973).

Whole blood analysis revealed an increase in magnesium level after oral lead administration. In renal failure, magnesium content tends to rise in the blood (Harper *et al*., 1979); Bogden *et al*., 1992). On the other hand a decrease in liver, brain and femur Mg content were observed between rats treated with lead, these results are consistent with prior studies of Bogden *et al*. (1991).

Our results show that there was a marked trend of decreasing organ lead concentrations with increasing dietary calcium for each of the studied organs and for whole blood. Moreover, a further decrease in organ lead concentration in rats fed the 2.7% Ca diet in comparison to those fed the 0.3% Ca diet. Nevertheless, the lower organ lead concentrations in rats fed the higher calcium supplemental diet may reduce the toxic effects of lead in all of these organs.

On the other hand, Ca supplementation increase the Ca levels in the kidney, liver, blood and femur. The very high kidney calcium concentrations found in rats fed 2.7% Ca diet may be due to the tendency of this diet to produce nephrocalcinosis. Hoek *et al*. (1988) have shown that increases in diet calcium increased the calcium and phosphorus content of the kidney and the extent of nephrocalcinosis in female rats.

In the present study dietary calcium alone interacts with numerous minerals (Mg, Fe, Cu, Zn and others), several investigations indicated that the mechanism by which calcium interacts with other minerals are extremely varied (Miller and Groziak, 1997). Mainly, Ca decreases the absorption of these metals, for example, calcium reduces the solubility of Ca phytate zinc complex and hence zinc absorption (Goyer, 1995). Moreover, calcium supplementation has been shown to modify the absorption of zinc and magnesium and iron (Willoughby and Thawely, 1975; Dursun and Aydogan, 1994), it reduces copper absorption by raising the pH in the intestine causing the precipitation of copper hydroxide.

In the present study, Ca supplementation did not influence organ concentrations in lead treated animals of the other four divalent metals studied (Fe, An, Cu and Mg). Exceptions of this generalization are the effect of the 2.7% dietary calcium on iron concentration in all tissues studied, specially in the kidney and the blood which returned to their levels as the control. Also, the concentration of 2.7% Ca, influenced the Cu concentration in the liver and the femur and also the liver, brain and femur Mg concentrations, which indicated the treatment effect of this calcium concentration, However, the effects of dietary calcium, specially 2.7%, on the metal concentrations in the studied organs were modest in comparison to its striking effects on organ lead concentrations. In other words, the effects of dietary Ca on organ lead accumulation seem to be specific.

Research studies conducted on animals as well as human indicated that calcium and lead interact in a negative (antagonistic) manner. The exact mechanisms by which calcium interacts with lead are not all known. Laboratory animal research indicates that calcium decreases lead absorption, (Barton *et al*., 1978) promotes urinary excretion of lead, (Sukhanov and Korolev, 1990), increases the release of lead from bones in culture media, (Rosen and Wexler, 1990) and reverses lead's inhibition of aceylcholine release from ganglia (Kostial and Vouk, 1957). Some researches indicated that calcium intake rather than calcium status decreases lead absorption (Bogden *et al*., 1992). Barton *et al*. (1978) proposed that calcium may inhibit lead absorption via physical competition between calcium and lead for common binding sites on intestinal binding proteins for absorption.

Our results demonstrate that oral calcium supplemetations can limit lead absorption in rats. This decrease has been shown by the significant reduction of lead in soft tissue as well as of the blood. In addition high dietary calcium itself reduce tissue concentrations of some essential metals which suggest caution in the use of calcium supplements and also suggest the need for addition studies on the treatment of lead toxicity by dietary calcium.

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