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## **Serum mineral changes under the effect of carbon disulfide intoxication**

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With 1 table

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Carbon disulfide is used as a solvent in artificial silk industry (16). Since then it has been gradually known to have certain toxic effects, which become more manifested on longer duration of exposure or as a result of increasing its concentration in the surrounding atmosphere (9). Massoud et al. (12) reported that workers exposed to carbon disulfide developed general complaints including nausea, diarrhea, vomiting, pain behind breast bone, constipation, indigestion, loss of appetite, and loss of weight.

Significant histopathological changes occurred in the liver, kidney, lung heart, and spleen as a result of carbon disulfide, which are known to effect mineral metabolism (8). Different types and degree of anemia have been reported among workers exposed to carbon-disulfide poisoning (1, 2, 3). Generalized hyperplasia of the reticuloendothelium system have been observed in poisoned rabbits (4).

Cohen et al. (5) reported that carbon disulfide reacts with free groups of protein and leads to the formation of thiozolidone and thiocarbamate compounds. These compounds would chelate the polyvalent inorganic ions and because of this low dissociation would thus interfere with cellular metabolism.

The aim of the present study deals with investigations on changes in the serum levels of certain minerals due to occur as a result of accumulation of carbon disulfide in the body. The effect of dose stoppage upon the reversibility of such pathogenic alterations in mineral metabolism is also investigated.

### Materials and methods

The material of this study comprised 80 rats of both sexes weighing 120-150 g. Rats were categorized into six groups each of ten rats, and were daily intramuscularly injected with 0.05 ml carbon disulfide in 0.2 ml olive kernel oil/rat over a period of 50 days. During experimentation, rats were maintained on the laboratory stock diet and allowed to eat ad libitum (14). Every 10 days, rats of one group were killed by decapitation and blood was collected.

Twenty rats of similar weight were included, fed on the same diet, injected with 0.2 ml olive kernel oil alone to serve as controls. Four of them were killed out of each of the injected groups. Carbon-disulfide injection was stopped for group six after the 50th day of the experiment to test the extent of regression of the developed biochemical derangements.

The method of *Sinaha and Gabrielli* (17) was used for determination of serum zinc. Serum iron, potassium, and sodium were estimated by the method published in *Beckman* Analytical method by Atomic Absorption Spectrophotometer. Serum calcium and magnesium were determined using the method of *Willis* (19).

### Results and discussion

Intramuscular administration of carbon disulfide for the different periods showed that the levels of serum zinc, iron, calcium, and magnesium were found to be significantly decreased and such decreases were more marked in groups receiving multiple doses of carbon disulfide (table 1). The observed drop in the levels of serum zinc, iron, calcium, and magnesium under the effect of carbon-disulfide intoxication may be the result of restricted mineral supply from dietary sources as a result of the observed loss of appetite.

The chemical reaction of carbon disulfide with amino groups of amino acids and protein leads to the formation of thiocarbamate, which is cyclized to thiozolidone (5). The formed thiocarbamate and thiozolidone groups would chelate the polyvalent inorganic ions and because of their low dissociation would thus interfere with cellular metabolism (10). As the concentration of the dithiocarbamate and thiozolidone increase in the animal body, the amount of inorganic elements bound to protein derivatives in the cells of the intoxicated tissue should increase. Accordingly, ionic concentration of these elements must decrease within the body of the rats at which the ions are supplied to the cell.

Furthermore, the increase loss of polyvalent inorganic ions in urine (6, 18, 7, 11) as a result of either back decomposition of the dithiocarbamate due to the release of tissue, particularly renal, as a part of the generalized tissue degradation which occurs under the effect of carbon-disulfide intoxication may be another contributing factor of mineral deficiency.

The drop in serum iron based on previously mentioned factors together with the finding of hyperplastic state of the bone marrow (2) may suggest the pattern of iron metabolism under conditions of carbon-disulfide intoxication to be as follows. A deficiency of iron intake leading to a state of hypoferriemia revealed by low level of serum iron. The latter state may be manifested by chelating action and the formed thiocarbamate and thiozolidone even to that iron released due to hemolysis of red blood

Table 1. Serum mineral levels in control and carbon-disulfide intoxicated rats.

Item		Control	Carbon-disulfide injections					Stopp. inj. for 20 days
			10 inj.	20 inj.	30 inj.	40 inj.	50 inj.	
Zinc	$\mu\text{g}/100\text{ ml}$	Mean S. E. $\pm$ P >	129.30 3.69 0.15	124.10 3.09 0.15	111.70 3.59 0.005	94.50 4.18 0.005	84.40 3.35 0.005	101.40 5.49 0.025
Iron	$\mu\text{g}/100\text{ ml}$	Mean S. E. $\pm$ P >	178.0 6.14 0.15	172.0 6.23 0.025	152.0 5.17 0.005	137.0 5.17 0.005	127.0 4.87 0.005	150.0 7.23 0.025
Calcium	$\text{mg}/100\text{ ml}$	Mean S. E. $\pm$ P >	7.21 0.094 0.15	6.79 0.176 0.025	6.13 0.213 0.005	4.84 0.316 0.005	3.64 0.282 0.005	5.29 0.286 0.025
Magnesium	$\text{mg}/100\text{ ml}$	Mean S. E. $\pm$ P >	1.80 0.141 0.15	1.70 0.182 0.025	1.65 0.105 0.025	1.38 0.148 0.005	1.23 0.096 0.005	1.58 0.172 0.15
Potassium	$\text{mg}/100\text{ ml}$	Mean S. E. $\pm$ P >	25.22 0.866 0.15	27.55 0.479 0.025	29.73 0.781 0.025	31.93 1.104 0.005	34.92 1.013 0.005	29.86 1.245 0.025
Sodium	$\text{mg}/100\text{ ml}$	Mean S. E. $\pm$ P >	232.38 4.472 0.15	236.96 2.602 0.15	234.41 3.019 0.15	225.55 2.803 0.155	217.20 2.309 0.025	224.51 4.449 0.15

corpuscle together with the hyperplastic state of the bone marrow may account for the hyperchromic type of anemia met with under such conditions (15).

In our study, an observed increase in the level of serum potassium under the effect of carbon disulfide intoxication, this hyperkalemia was found in rats, particularly receiving aggressive doses of carbon disulfide in groups 4 and 5 (table 1). This state may be due to tissue destruction irrespective to carbon-disulfide intoxication (13). These findings together with the demonstration of hemolytic action of carbon disulfide may be another contributing factor of a state of hyperkalemia. *Brieger* (1) found that carbon disulfide has no hemolytic action, while *Cappellini* (3) found a hemolytic anemia among workers exposed to carbon-disulfide poisoning.

On the other hand, under the effect of carbon-disulfide intoxication, the serum sodium level was not significantly affected. No definite correlation could be drawn between sodium level in control and carbon-disulfide intoxicated rats.

In addition, stoppage of dosage brought about return to normal pattern of these mineral levels, whereby the animals started to restore their appetite. The improvement in mineral metabolism may be due to diminution in the concentration of chelating agent. Such findings suggest that workers exposed to carbon disulfide have to be leaved at intervals or shifted from polluted areas to prevent development of undesirable mineral derangement or to alleviate the condition if present.

### Summary

Under the effect of carbon-disulfide intoxication on serum levels of zinc, iron, calcium, magnesium, potassium, and sodium of albino rats, five groups of rats were injected with daily doses of carbon disulfide over a period of 50 days. The extent of regression of the developed biochemical derangement was also studied. A significant decrease was observed in the levels of serum zinc, iron, calcium, and magnesium, while a slight elevation in the level of serum potassium. No definite correlation could be found between the level of serum sodium in control and carbon-disulfide intoxicated rats. By stoppage of dosage at 20 days, most of these derangements started to be normal.

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