

PROPHYLACTIC ROLE OF DIETARY ZINC AGAINST CADMIUM TOXICITY IN BROILER CHICKS WITH SPECIAL REFERENCE TO CUMULATIVE EFFECT OF CADMIUM ON THE LEVEL OF SOME ELEMENTS IN VARIOUS TISSUES

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ABSTRACT

The objective of the present study is to detect in appropriate way evidence of cadmium toxicity as a consequence of exposure to sublethal doses added to drinking water at the concentration level of 10 and 100 ppm singly or in combination with a low concentration of zinc (5ppm) as daily supplement to chicks for 30 days. The body gain in 10 and 100 ppm Cd-treated chicks was significantly decreased at one month. Moreover, the percent of food and water consumption were reduced. Addition of zinc (5ppm) to 100 ppm Cd-exposed chicks enhanced the gain in body weight as well as food and water consumption.

The erythrocytic count, haemoglobin content and hematocrit values were significantly decreased in all cadmium-treated groups. Zinc supplementation improved the haematological picture to slight extent. The serum of Cd-treated chicks showed a significant increase in AST, ALT, Alkaline phosphatase and total bilirubin levels. On the other hand, total proteins were decreased. The cadmium level in liver, kidneys, skeletal muscle, small intestine and bone exceeded over controls in all treated chicks. Zinc supplementation (5ppm) decreased the cadmium accumulation in the investigated tissues. Zinc levels were reduced in bone and increased in liver, kidneys, skeletal muscle and small intestine. The copper content was decreased in kidneys and increased in the skeletal muscles. Significant decrease in the iron content was observed in the liver, kidneys, small intestine and bone.

It could be concluded that dietary zinc plays a prophylactic role against cadmium toxicity.

INTRODUCTION

In recent years, the development of modern technology and growing industrialization are among the foremost factors contributing to the general pollution in Egypt. The environmental contaminants spread through a variety of channels, many of which sooner or later reach our food, drinking water and atmospheric air⁽¹⁾. Some of heavy metals as cadmium gained access to food through their use as colouring agents⁽²⁾ and pesticides. Food as coffee, tea and vegetable crops contains cadmium to less extent, whereas, the average cigarette smoke contains as much as 1 µg cadmium⁽³⁾ and heavy smokers may have an intake of 20 µg Cd/day or more.

Cadmium could be absorbed through lungs 10-50% and from the contaminated food and water through intestine 6%⁽⁴⁾. The environmental pollution with cadmium may affect the general health of human, animals and plants. High cadmium residues were detected in the kidneys, liver, gastrointestinal tract, uterus, lung, testes and lower levels were found in the skeletal muscles, bone and even milk^(5,6). Exposure of pregnant animals to high doses of cadmium may result in an increased incidence of embryotoxic effects and growth retardation⁽⁷⁾. Cadmium toxicity may lead to loss of appetite, reduced body gain, toxic changes in blood picture, proteinuria, testicular atrophy, hypertension, hepatic and renal dysfunction as well as reduced fertility in both males and females⁽⁸⁾. All chemicals are hazardous but there are many ways in which the risk can be reduced.

Some specific dietary constituents such as microminerals (Zn⁽⁹⁾ and Mn⁽¹⁰⁾ and macromineral Ca⁽¹¹⁾) have been invoked as factors affecting the toxicity of cadmium. Antagonism between cadmium and zinc were reported in rats^(12,13). Some studies showed a protective effect of zinc against inhibition of SGOT by cadmium⁽¹⁴⁾. The previous authors suggested that zinc and cadmium may compete not only at the absorption sites but also at the enzymatic level.

The present study was conducted to elucidate whether the dietary zinc plays a prophylactic role against cadmium absorption, accumulation and toxicity.

MATERIAL AND METHODS

Experimental design :

Eighty hubbard chicks, one day old, were used in this study. They were divided into four equal groups, each of twenty chicks. The treated groups received cadmium chloride (Merck, Darmstadt, Germany) singly or combined with zinc sulphate (Merck Darmstadt, Germany) for 30 days through drinking water. Groups 1, 2 and 3 received 10, 100 and 100ppm cadmium chloride respectively in addition to 5ppm zinc sulphate for group 3. Group 4 was given tap water (control). Balanced ration and tap water ad libitum was available for all chicks. The body gain, food and water consumption were calculated for each group throughout the experiment.

Haematological studies :

Blood samples were collected in 10ml bottles containing 5mg EDTA from chickens at sacrifice after one month from the beginning of the experiment. The blood samples were examined for determination of haemoglobin⁽¹⁵⁾, erythrocytic and leucocytic counts⁽¹⁶⁾, and packed cell volume (PCV)⁽¹⁷⁾.

Biochemical studies :

Another blood samples were collected in centrifuge tubes, at slaughtering without anticoagulant and placed in slant position till the serum was separated from the clot. The separated fluid was centrifuged at 3000 r.p.m. for 20 minutes to separate the clear serum. The serum samples were analysed for determination of Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT)⁽¹⁸⁾, alkaline phosphatase (ALP)⁽¹⁹⁾, total bilirubin⁽²⁰⁾ and total proteins⁽²¹⁾.

Tissue residues :

Cd, Cu, Zn and Fe were estimated in various tissues (liver, kidneys, small intestine, skeletal muscle and bone) following nitric acid digestion using atomic absorption spectrometer (Perkin-Elmer 5000)⁽²²⁾ and levels are reported as µg/g of tissues.

Statistical analysis :

The obtained results were subjected to Student's (t) test to reveal the significance of differences between treatment and control groups⁽²³⁾.

RESULTS

Body gain, food and water consumption :

The treated birds were apparently healthy throughout the experiment. The body gain was significantly decreased in 10 and 100ppm cadmium-treated broilers. Addition of Zn to the drinking water of

cadmium-fed chicks increased the body gain, food and water consumption (Table 1).

Haematological findings :

The RBCs count, PCV% and haemoglobin content were significantly decreased at one month of treatment in 10 & 100ppm Cd-exposed chicks. The erythrocytic count was nonsignificantly changed at one month of treatment with 100ppm cadmium chloride and 5ppm zinc. On the other hand, WBCs count was significantly increased at one month in all-treated chicks (Table 2).

Biochemical results :

AST and ALT as well as ALP activities were significantly increased at one month in all-treated groups. The total serum proteins were significantly decreased in all-treated chickens at one month, whereas, the total serum bilirubin was nonsignificantly changed (Table 3).

Tissue residues :

The Cd, Cu, Zn and Fe levels were detected in broiler tissues (liver, kidneys, small intestine, skeletal muscle and bone) at the end of the experiment. The cadmium concentration was significantly increased in all cadmium-treated groups. The highest cadmium level was detected in the kidneys followed by the liver, small intestine, skeletal muscle and then bone (Table 4). The zinc concentration in the treated chicks was significantly increased in the liver, kidneys as well as small intestine and decreased in the bone (Table 5). The copper content was significantly decreased in kidneys in all treated groups however its level was nonsignificantly changed in the other investigated tissues (Table 6). The iron content was significantly decreased in the liver, kidneys, small intestine and bone in all-treated chicks (Table 7).

Table (1): The body gain, food intake and water consumption after one month in chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean ± S.E) (n = 10).

Parameter	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
Body gain (g %)	463 ± 11.37 (100%)	389 ± 9.12** (84%)	301 ± 10.18*** (65%)	426 ± 12.04 (92%)
Food intake (%)	100%	92%	89%	91%
Water consumption (%)	100%	80%	62%	93%

** P < 0.001

*** P < 0.0001.

Table (2): Blood picture of chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean \pm S.E) (n = 10).

Parameter	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
RBCs ($10^6/\mu\text{l}$)	3.39 \pm 0.04	3.14 \pm 0.03**	2.87 \pm 0.03***	3.26 \pm 0.04
WBCs ($10^3/\mu\text{l}$)	12.80 \pm 0.11	18.70 \pm 0.19***	21.40 \pm 0.29***	19.30 \pm 0.18***
Hb (g/L)	10.68 \pm 0.19	9.96 \pm 0.21*	9.28 \pm 0.25**	10.27 \pm 0.31
PCV (%)	35.95 \pm 0.61	33.15 \pm 0.47*	32.22 \pm 0.58*	33.89 \pm 0.54

* P < 0.01 ** P < 0.001 *** P < 0.0001.

Table (3): Effect of cadmium single or combined with zinc through drinking water for 30 days on some serum biochemical variables (Mean \pm S.E) (n = 10)

Parameter	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
AST μL	29.3 \pm 0.41	32.5 \pm 0.43**	38.4 \pm 0.56***	31.6 \pm 0.38*
ALT μL	21.7 \pm 0.22	23.8 \pm 0.29**	29.2 \pm 0.44***	22.9 \pm 0.21*
ALP mmu	38.5 \pm 0.46	69.4 \pm 0.74***	78.3 \pm 0.83***	64.8 \pm 0.69***
Total protein g/100ml	4.6 \pm 0.09	4.23 \pm 0.05*	3.85 \pm 0.07***	4.16 \pm 0.08*
Total bilirubin μml	1.34 \pm 0.06	1.36 \pm 0.05	1.38 \pm 0.04	1.37 \pm 0.04

* P < 0.01 ** P < 0.001 *** P < 0.0001.

Table (4): Cd concentration ($\mu\text{g/g}$ wet weight) in liver, kidneys, small intestine, skeletal muscle and bone of chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean \pm S.E) (n = 5).

Tissue	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
Liver	0.84 \pm 0.07	17.8 \pm 0.39***	185.4 \pm 3.25***	119.6 \pm 2.43***
Kidneys	2.95 \pm 0.14	87.3 \pm 1.66***	962.2 \pm 5.57***	549.4 \pm 3.81***
Small intestine	0.49 \pm 0.05	5.85 \pm 0.71**	68.4 \pm 2.39***	27.6 \pm 0.64***
muscle	0.31 \pm 0.04	4.36 \pm 0.47**	15.9 \pm 0.86***	9.4 \pm 0.53***
Bone	0.29 \pm 0.04	3.8 \pm 0.36**	11.5 \pm 0.67***	6.2 \pm 0.52***

** P < 0.001 *** P < 0.0001.

Table (5): Zn concentration ($\mu\text{g/g}$ wet weight) in liver, kidneys, small intestine, skeletal muscle and bone of chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean \pm S.E) (n = 5).

Tissue	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
Liver	45.71 \pm 0.65	46.83 \pm 0.47	49.68 \pm 0.52*	51.42 \pm 0.43**
Kidneys	38.90 \pm 0.29	39.56 \pm 0.36	41.90 \pm 0.41*	42.30 \pm 0.26**
Small intestine	20.90 \pm 0.48	21.30 \pm 0.38	23.70 \pm 0.34*	22.10 \pm 0.47
Muscle	33.86 \pm 0.27	34.61 \pm 0.49	34.14 \pm 0.53	34.79 \pm 0.31
Bone	129.8 \pm 1.53	120.1 \pm 1.84*	114.3 \pm 1.62**	123.6 \pm 1.37

* P < 0.05

** P < 0.001

Table (6): Cu concentration ($\mu\text{g/g}$ wet weight) in liver, kidneys, small intestine, skeletal muscle and bone of chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean \pm S.E) (n = 5).

Tissue	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
Liver	4.83 \pm 0.34	4.85 \pm 0.23	4.81 \pm 0.26	4.82 \pm 0.29
Kidneys	14.6 \pm 0.17	13.51 \pm 0.21*	12.28 \pm 0.19**	13.05 \pm 0.18*
Small intestine	1.74 \pm 0.05	1.70 \pm 0.09	1.69 \pm 0.04	1.73 \pm 0.04
Muscle	1.95 \pm 0.06	1.91 \pm 0.03	1.89 \pm 0.04	19.3 \pm 0.03
Bone	0.869 \pm 0.03	0.857 \pm 0.02	0.861 \pm 0.03	0.864 \pm 0.05

* P < 0.01

** P < 0.001

Table (7): Fe concentration ($\mu\text{g/g}$ wet weight) in liver, kidneys, small intestine, skeletal muscle and bone of chicks treated daily with cadmium single or combined with zinc through drinking water for 30 days (Mean \pm S.E) (n = 5).

Tissue	Control 0 ppm Cd	Treated groups		
		10 ppm Cd	100 ppm Cd	100 ppm Cd + 5ppm Zn
Liver	135.38 \pm 1.26	120.63 \pm 0.99**	105.8 \pm 1.23***	117.4 \pm 1.18**
Kidneys	86.19 \pm 1.13	74.5 \pm 1.22**	68.7 \pm 1.14**	79.6 \pm 1.25*
Small intestine	33.7 \pm 0.58	24.1 \pm 0.43***	18.7 \pm 0.67***	27.5 \pm 0.39**
Muscle	63.7 \pm 1.18	60.4 \pm 1.12	59.8 \pm 1.24	61.2 \pm 0.96
Bone	39.8 \pm 0.54	34.7 \pm 0.38**	29.4 \pm 0.35***	34.6 \pm 0.28**

* P < 0.01

** P < 0.001

*** P < 0.0001.

DISCUSSION

The pollution of our environment with one or more of heavy metals is still one of the major problems that face us in Arab Republic of Egypt owing to the development of industry. The consumption of the contaminated food, water or inhalation of polluted atmosphere with cadmium may affect the general health of human, animals and plants.

It has been shown that exposure of chicks to 10ppm or 100ppm cadmium in the drinking water for 30 days significantly reduced the body gain. The observed decrease in the body gain may be due to the decreased food and water consumption evidenced in this study. The addition of zinc (5ppm) to 100ppm Cd-treated chicks improved the appetite, body gain, food and water consumption but not to control levels. The obtained results are in coordination with those recorded in rats^(24,25).

On the assumption that only 6% of cadmium ingested with food is absorbed through gastrointestinal tract, so the tolerable weekly intake of 400-500ug of cadmium per person is equivalent to 70ug per person/day⁽²⁶⁾. Any increase in the amount of cadmium derived from drinking water or inhaled from the atmosphere will reduce the amount that can be tolerated in the food. The absorbed cadmium will be distributed to erythrocytes, plasma, kidneys, liver, lungs and other tissues. The decreased erythrocytic count, haemoglobin content and packed cell volume at one month in Cd-exposed chicks could be due to the shortened life span of red cells. Similar results were previously reported^(27,28). They recorded that exposure to Cd²⁺ increased the red cells destruction. In addition, the recorded decrease in the erythrocytic count is in agreement with the reported of anemia in Cd-exposed fish⁽²⁹⁾. They also added that the observed anemia was probably due to the decrease in synthesis or release of erythrocytes into the circulation. Moreover, the induced anemia after oral ingestion of cadmium could be associated with the defect of iron metabolism as a result of its deficiency in the intestinal absorption⁽³⁰⁾. This clarify the reduced haemoglobin content, haematocrit value and erythrocytic count in Cd-exposed chicks.

The addition of zinc (5ppm) in the drinking water of cadmium-treated chicks antagonised the cadmium absorption, probably through competition with cadmium for its absorptive sites⁽¹⁴⁾ which consequently reduced its toxicity.

The long term cadmium toxicity induced elevations in the serum AST, ALT and ALP activities as well as total bilirubin level, whereas, the total proteins were decreased. The cause of this elevation

could be due to extensive liver destruction by cadmium, consequently these enzymes are liberated into the serum. Our obtained results are in coordination with those recorded by some investigators^(31,32). The latter reported that cadmium administration causes an elevation in plasma AST and ALT activities. The present investigation has shown that zinc supplementation decreased the hepatotoxicity produced by cadmium.

The cadmium concentration was high in kidneys followed by liver, small intestine, skeletal muscle, and then bone of the investigated tissues of birds. Exposure to different concentrations of cadmium (10 or 100ppm) raised its level more than one fold i.e. the increase in the cadmium concentration (through broiler drinking water) over the tolerable daily intake (70ug Cd/ person) resulted in more cadmium accumulation in the different organs and tissues. These findings are in agreement with the previous work^(33,34,35). Zinc supplementation (5ppm) to cadmium treated chicks reduced the cadmium level in various tissues & its side effects were improved and corrected^(36,37). It was obvious that there was an antagonism between cadmium and zinc due to their similarities in their physiochemical properties where Cd interacts competitively with zinc metabolism^(10,13). Previous reports have suggested that the protective effect of zinc supplementation in cadmium toxicity has been attributed to the increased synthesis of metallothionein (MT), a low molecular weight protein with high affinity for metals^(38,39). MT has a role in the metabolism of essential metals such as zinc as well as copper and in detoxification of heavy metals as cadmium and mercury.

The influence of cadmium single or combined with zinc on the metabolism of some essential metals (Zn, Cu and Fe) were investigated. It was found that there is redistribution of these essential elements in different parts of the body as a result of exposure to cadmium. The zinc content was increased in all investigated organs (liver, kidneys and small intestine) except bone. The increased zinc content could be attributed to the synthesis of metallothionein in these tissues, beside decreased elimination or increased absorption of zinc. MT induction & increased zinc concentration in liver and kidneys were observed after oral administration of cadmium⁽⁴⁰⁾ or injection of cadmium⁽⁴¹⁾. The decreased zinc content in bone may be due to no metallothionein synthesis in the bone⁽⁴²⁾. The copper content of kidneys was decreased in the treated groups. This effect could be due to the increased elimination or reduced absorption of copper during long term cadmium toxicity. The iron content of the investigated tissues (kidneys, liver, small intestine and

level sharply decreased which could be due to the interference with iron absorption and antagonism between iron and cadmium at the absorption sites in the small intestine⁽⁴³⁾. Our present data are in agreement with those previously obtained⁽⁴⁴⁾, who reported that cadmium ingestion greatly depleted iron stores in the liver and kidneys by inhibition of both ferritin synthesis and iron incorporation into this protein. On the other hand, previous report recorded that there was no significant change in the iron content of tissues of rats injected with cadmium⁽⁴²⁾.

Hence, it could be concluded from the present study that dietary zinc plays a prophylactic role against cadmium toxicity.

REFERENCES

- Lucas, J. : Our polluted food. London and Tonbridge. Charles Knight and Company LTD (1975).
- Barnard, J.A.B. : Arch. Toxicol. Suppl. 1: 47-54 (1978).
- Fox, M.R.S. : A review J. Food Sci. 37: 321 (1974).
- Eastler, C.I. : Lehrbuch der Allgemeinen und systematischen pharmakologie und toxicologie. F.K. Schattauer Verlag Stuttgart-Newyork (1983).
- Watanabe, M.; Shimizu, K.; Nishino, H.; Shimura, T.; Mizuno, H.; Shoji, T.; Naito, Y. and Kagaminori, S. : Environmental Res. 40: 25-46 (1988).
- Massanyi, P., Tomasi, R., Ulteri, V., Remon, P. : Italian J. of Food Sci. 7(3) : 311-316 (1995).
- Webster, W.S. : Arch. Environ. Health 35: 36 (1978).
- Müller, R.K. : Die Toxikologische chemische analyse. Verlag Chemie, Weinheim-Newyork (1976).
- Reddy, C.S. : Toxicol. Letters 31: 49 (1986).
- Yu, H.S and Chan, S.T.H : Toxicology 45: 261-272 (1988).
- Bauman, V., Valmetse, M.; Baburykin, D. : Interaction of vit. D3, calcium and heavy metals in chicks. Natural Science 1: 70-72 (1994).
- Spon, A.; Dima, I.; Stoiculescu, L. and Cristea, A. : Nahrung 13: 461-469 (1969).
- Waaliker, M.P. and Poirier, L.A. : In Vitro cadmium - DNA interactions : Toxicol Appl. Pharmacol. 75: 539 (1984).
- Goering, P.L. and Klassen, C.D. Toxicol. Appl. Pharmacol. 74: 299-307 (1984).
- Dokes, H.H. and Schwarte, L.H. Ann. J. Physio. 96: 72-89 (1930).
- Nutt, M.R. and Herrich, G.R. : Poul. Sci. 31: 735-740 (1952).
- Winnicko, M.M. : Ann. J. Med. Sci. 155: 58-75 (1933).
- Reitman, S. and Frankel, S. : J. Clin. Path. 29: 58 (1955).
- Kind, P.R. N. and Kling, E.G. : J. Clin. Path. 13: 188-199 (1965).
- Indrusick, L. : Biochem. Z. 297: 51 (1955).
- Looney, O.L.; Rosenbrough, N.J.; Parr, A.L. and Reunert, K. J. : J. Bio. Chem. 193: 265-275 (1951).
- Weitz, B. : Atom-Absorption-Spektroskopie. Z. Aufl. Verlag Chemie, Weinheim (1975).
- Snedecor, G.W. : Statistical Methods. Iowa State College Press, Ames, Iowa 4th ed., p. 583 (1971).
- Kotsonis, F.N. and Klassen, C.D. : Toxicol. Appl. Pharmacol. 46: 39-54 (1978).
- Zuruki, Y. and Yoshikawa, H. : J. Toxicol. and Environmental Health, 5: 479-487 (1981).
- Classen, H.G.; Elias, P.S. and Hommes, W.B. : Toxikologische-hygienische beurteilung von Lebensmittelinhalts und zusatzstoffen sowie bedenklicher Verunreinigungen. 1g. ed. Verlag Paul Parey, Berlin and Hamburg (1987).
- Berlin, M. and Friberg, L. : Arch. Environ. Health 1: 475-486 (1960).
- Webb, M. : Clinical chemistry and chemical toxicology of metals. Elsevier/North. Holland Biomedical press, Amsterdam-Newyork-Oxford (1977).
- Sjoberck, M.L.; Haax, H.; Larsson, A. and Lithner G. : Ecotox. Environ. Saf. 8: 303-312 (1984).
- Richardson, M. E; Fox, M.R.S. and Fry, B.E. : J. Nutr. 104: 323-338 (1974).
- Cook, J.A.; Marconi, E.A. and Di Lurio, N.R. : Toxicol. Appl. Pharmacol. 25: 292-302 (1974).
- Webb, M. : The chemistry, biochemistry and biology of cadmium. Elsevier/North-Holland Biomedical press. Amsterdam-Newyork - Oxford (1979).
- Kapoor, N.K.; Agarwala, S.C. and Kar, A.B. : Ann. Biochem. Exp. Med. 21: 51-55 (1961).
- Buhler, D.R.; Wright, D.C.; Smith, K.L.; Tinsley, J.J. : J. Toxicol. Environ. Health 8: 185-197 (1981).
- Bokori, J.; Fekete, S.; Glavits, R.; Kadar, I.; Koncz, J. and Kovari, L. : Acta Veterinaria Hungarica 44 (1) : 57-74 (1996).
- Webb, M. : Biochem. Pharmacol. 21: 2769 (1972).
- Stacey, N.H. and Klassen, C.D. : J. Toxicol. Environ. Health 7: 149-158 (1981).
- Kagi, J.H.R. and Nordberg, M. : Metallothionein. Birkhauser Verlag, Basel (1979).

39. Webb, M. and Cain, K.; Biochem. Pharmacol. 31 : 137-142 (1982).
40. Flora, S.J.S.; Behari, J.R., Ashquin, M. and Tandon, S.K.; Chem-Biol. Interactions 42 : 345-351 (1982).
41. Stuard, M.D. and Webb, M.; Chem. Biol. Interactions 15 : 369-389 (1976).
42. Roth, H.P. and Kirchgessner, M.; Zbl. Vet. Med. A 24 : 177-188 (1977).
43. Schafer, S.G. and Forth, W.; Ecotox. Environ. Safety 7 : 87-95 (1983).
44. Bonner, F.W.; Kling, L.J. and Parke, D.V.; Toxicology 19 : 247-254 (1981).

الدور الوقائي للزنك ضد التسمم بالكادميوم في الكتاكيت مع الإشارة لتأثير الكادميوم التراكمي على بعض المعادن في الأنسجة المختلفة

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الهدف من هذه الدراسة كشف النقاب عن التسمم بالكادميوم نتيجة التعرض لجرعات مختلفة أضيفت إلى ماء الشرب في الكتاكيت يوميا لمدة ٣٠ يوماً، بمعدل ١٠، ١٠٠ جزء لكل مليون مفردة أو مضاف إليها الزنك بتركيز قليل ٥ جزء لكل مليون.

لقد وجد أن هناك انخفاض في أوزان الكتاكيت ونقص في استهلاك مياه الشرب والعليقة للكتاكيت المعطاة كادميوم بنسبة ١٠، ١٠٠ جزء لكل مليون مفردة. والمجموعة المضاف إليها الزنك حدث تحسن ملحوظ حيث زاد استهلاك مياه الشرب والعليقة وكذلك زادت أوزان الكتاكيت ولكن لم تصل لمستوى مجموعة الضوابط.

ولقد تبين أن هناك نقص في عدد كرات الدم الحمراء ونسبة الهيموجلوبين وسرعة الترسيب في كل المجموعات المعطاة كادميوم بينما وجدت زيادة في كرات الدم البيضاء. إضافة الزنك أحدث تحسن بسيط في صورة الدم.

أيضاً اتضح أن هناك ارتفاع في نسبة انزيم الأسبرتيت أمينو ترانس فيريز وانزيم آلانين أمينو ترانس فيريز وانزيم الفوسفاتيز القاعدى في مصل الكتاكيت بينما حدث نقص في نسبة البروتين الكلى.

لقد زاد معدل تراكم الكادميوم في الكبد والكليتين والإمعاء والعضلات والعظام أكثر من مرة عن مجموعة الضوابط. لكن إضافة الزنك خفضت من تراكم الكادميوم في الأنسجة المختلفة. ولقد زاد معدل الزنك في الكبد والكليتين والإمعاء بينما انخفض في العظام. ولقد لوحظ أيضاً إنخفاض في نسبة النحاس في الكليتين. ولقد وجد أن هناك نقص شديد في نسبة الحديد في الكبد والكليتين والإمعاء والعظام. إضافة الزنك خفضت من تراكم الكادميوم في الأنسجة المختلفة وبالتالي حدث تحسن ملحوظ في معدل بعض المعادن.

يستخلص من هذه الدراسة أن للزنك دور هام في الوقاية من التسمم بالكادميوم.