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Protective Effects of Vitamin E on Growth Performance and Tissue Weight Against Toxicity Induced by Hexavalent Chromium in Laboratory Chicks

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ABSTRACT

Chromium can enter in body through air, food and water. Most abundant toxic form of this metal in the environment is Cr(VI). Chromium (VI) readily enters all type of cells through a general action channel of plasma membrane and reduced into Cr(III) by various oxidants. Therefore, present study has been carried out to investigate the effects of vitamin E on growth performance and tissue weight (liver and kidney) in chicks against toxicity induced by hexavalent chromium. Developing chicks (Croilers, *Gallus gallus domesticus*, body weight 100±20 gm) were used as experimental animals. Body weight was taken once in a week for growth performance during experimental period. Tissue weight has been taken after 30 days of treatment. Oral administration of Cr(VI) adversely affects the growth performance of chicks. It is observed that during administration of chromium the animal body weight decreases and tissue weight is also found to be reduced. However, supplementation of vitamin E along with Cr(VI) show significant recovery in body weight of animals as compared to chromium treated chicks. Along with the growth, the weights of different tissues like liver and kidney are also decreases in Cr(VI) treated chicks. Administration of vitamin E in these chicks also significant increase in tissue weight was observed. Thus present study reveals that supplementation of vitamin E significantly protect from the adverse effect of Cr(VI).

KEYWORDS: Vitamin E, Croilers, Hexavalent chromium, Growth performance, Tissue weight

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INTRODUCTION

Poultry production has experienced a remarkable growth within the past thirty five years. However, there are still a lot of problems facing the industry in its bid to attain viable level of production. Deposition of heavy metals in birds is due to the feeding of contaminated feed and water as well as exposure to different manufacturing processes of factories and industries ¹. Animal management involves a combination of good housing, health and feeding. Therefore the quality and safety of feed given to animals determines their performance.

Heavy metals contamination originates from many sources such as industrial, anthropogenic and exogenous means. According to Lenntech ², Heavy metals refer to any metallic element that has a relatively high density and is toxic or poisonous even at low concentration. Heavy metals are specifically regarded with their toxicological as well as carcinogenic effects ³. Some of these elements play an essential role in physiological and biochemical processes in living cells when present in required amount ^{4,5}. When these elements are present in lethal dose, it results in deleterious effect such as reduced growth rate, malfunction of vital organs such as liver, kidney and consequently death of the animal. Supplementation of heavy metals with a large safety margin in broilers has resulted into higher mineral excretion and ends up in the environment ^{6,7}.

Chromium is a naturally occurring element found in rocks, animals, plants, soil, volcanic dust and gases. It occurs in various oxidative states from Cr(II) to Cr(VI). A significant amount of chromium enters into the environment from chrome plating, textile dyes and paint industries. Chromium can enter in body through air, food and water. It can easily penetrate and accumulate into internal organs like the kidney, lungs, liver and in some cases causes damage in the blood. Most abundant toxic form of this metal in the environment is Cr(VI). In animal body it is quickly converted into Cr(III) through Cr(V) and Cr(IV) intermediates. Chromium (VI) readily enters all type of cells through a general anion channel of plasma membrane and reduced into Cr(III) by various oxidants. This form of chromium is trapped and accumulated within the cell, as it is less permeable through cell membrane than Cr (VI).

In cells Cr(V) and Cr(IV) intermediates act as free radicals and increase oxidative stress. Natural antioxidants vitamin E, ascorbic acid, riboflavin, cytochrome P-450 reductase, glutathione reductase etc. are free radical scavengers and quickly convert the highly toxic form of Cr (VI) to Cr(III) in the tissues which does not readily leave the cell ⁸. The metal is slowly released through kidney and bile ⁹. However, during its stay, Cr(III) forms stable complexes with ligands of protein, DNA and GSH and causes all kind of metabolic, genetical, immunological, developmental and carcinogenic changes ^{8,10}, therefore, a quick removal of the metal from the body is necessary.

A timely evaluation of the effects of exposure to chromium toxicity and its amelioration with vitamin E to animals will provide useful information relating to the therapy of toxicated animals. Although extensive studies have been done on the effect of heavy metals on animals, yet there is a dearth of information on chromium toxicity contamination in chicks. Therefore, present study has been undertaken to investigate protective role of vitamin E on growth performance and tissue weight (liver and kidney) in chicks intoxicated by hexavalent chromium.

EXPERIMENTAL SECTION

Animals – The experiment was carried on Domestic chicks – Croiler Chabro (*Gallus gallus domesticus*). Newly hatched chicks were purchased from the Uttarakhand Village Poultry Project (State Govt. Poultry Farm), Bin, Pithoragarh (Uttarakhand). Selected all chicks were maintained and acclimatized according to the laboratory condition. The animals were housed in battery cages under laboratory conditions at existing room temperature and relative humidity. They were fed on commercial food (Starter, Grower and Finisher) purchased from the local market and tap water *ad libitum*. Healthy male and female chicks (approximately 2-3 weeks old, body weight 100±20 g) were used in present study. The experimental protocol was approved by Institutional Animal Ethical Committee (IASC), Department of Pharmaceutical Science, Bhimtal, Kumaun University, Nainital and the member secretary, CPCSEA, Ministry of Environment, Forest and Climate Change, Government of India (Protocol No.- KUDOPS/89). The animals were kept under standard conditions throughout the experiment to reduce the error. Minimum number of animals was used to obtain reliable results.

Chemicals – Vitamin E (α -tocopherol) was purchased from Sigma Chemical Co. (St. Louis, Mo, USA). Potassium dichromate ($K_2Cr_2O_7$) was procured from Glaxo (India). All the reagents and chemicals used in this study were of analytical grade and highest purity procured from standard commercial sources in India.

Experimental Treatments - The selected chicks were divided into three groups (A, B and C) randomly, each containing at least 6 chicks. Chicks of group A were administered with sublethal dose of potassium dichromate ($K_2Cr_2O_7$) (05 mg/100 g body weight) by gavage on each alternate day for 30 days. Chicks of group B were treated with potassium dichromate ($K_2Cr_2O_7$) as chicks of group A but also administered with vitamin-E (intramuscularly) (0.5 IU/100 gm body weight) on each alternate day for 30 days. Chicks of Group C were administered with saline only to serve as purely control.

The animal weight, general health and behavior were recorded in all the groups. All the birds were monitored for clinical signs twice daily. Each chick was weighted weekly. After the final

treatment on Day 30, chicks from each group were anesthetized with light diethyl ether/chloroform and sacrificed in the morning hours. Kidney and liver were simultaneously removed, weighted and used for further investigations.

Statistical analysis - All body and tissue weight values were expressed as mean \pm SE. All data were analyzed statistically using one-way analysis of variance (ANOVA) followed by Student's *t*-test. Statistical significance was considered at $P < 0.05$.

RESULT AND DISCUSSION

All the chicks administrated with Cr showed more prominent signs of toxicity (+++) as compared to control. Clinical signs observed in the present study are decreased body weight gain and chicks were dull and depressed (table 01). Effect of vitamin E on body weight gain is presented in table 02. Live body weight decreased significantly in Cr treated chicks in comparison to the therapeutic and control group. The chicks treated with vitamin E along with Cr, showed significant increase in live body weight. The control chicks on 15th day of their treatment were weighing 190 ± 10.0 gm. Within next seven days the weight increased (240 ± 10.0 gm on 22th day), which reveals a satisfactory growth of the animals. The chicks administrated with a dose of 05 mg/100 gm body weight potassium dichromate (group A) for 15 days shows reduction in their weights (133.3 ± 7.63) as compared to control. When these animals were given Cr for next seven days (day 22), their weight was 185 ± 13.22 gm (less than their respective control), similarly their weights on day 30 also showed a significant decrease in body weights as compared to control. Weight gain was maximum in vitamin E therapeutic group i.e. group B.

The tissue weight (liver and kidney) of different groups is presented in table 03. Tissue weight also decreased significantly in Cr toxicated group as compared to control group, while co-treatment with vitamin E increases tissue weight significantly. (table 03)

Table 01: Clinical signs and behavioral alterations in chicks of different groups administered with chromium, chromium + Vitamin E and control group

Treatment	Dullness	Depression	Sluggish	Reduced body weight
Chromium	+++	+++	+++	+++
Chromium+ Vit E	+	+	+	-
Control	-	-	-	-

No sign (-), mild (+), severe (+++)

Table 02: Mean values of live body weight (g) in chicks of different groups administered with chromium, chromium + Vitamin E and control group

Treatment	Day 1	Day 8	Day 15	Day 22	Day 30
Chromium	105.0±5.0	126.6±7.63	133.3±7.63	185.0±13.22	191.6±15.27
Chromium +Vitamin E	101.6±2.88	188.3±12.58	211.6±7.63	255.0±8.66	311.6±12.58
Control	103.3±10.4	168.0±7.63	190.0±10.0	240.0±10.0	290.0±10.0
ANOVA (p-value)	NS	**	**	NS	**

Results are expressed as mean ± SE. ** indicates significant at p< 0.05. NS = Not significant.

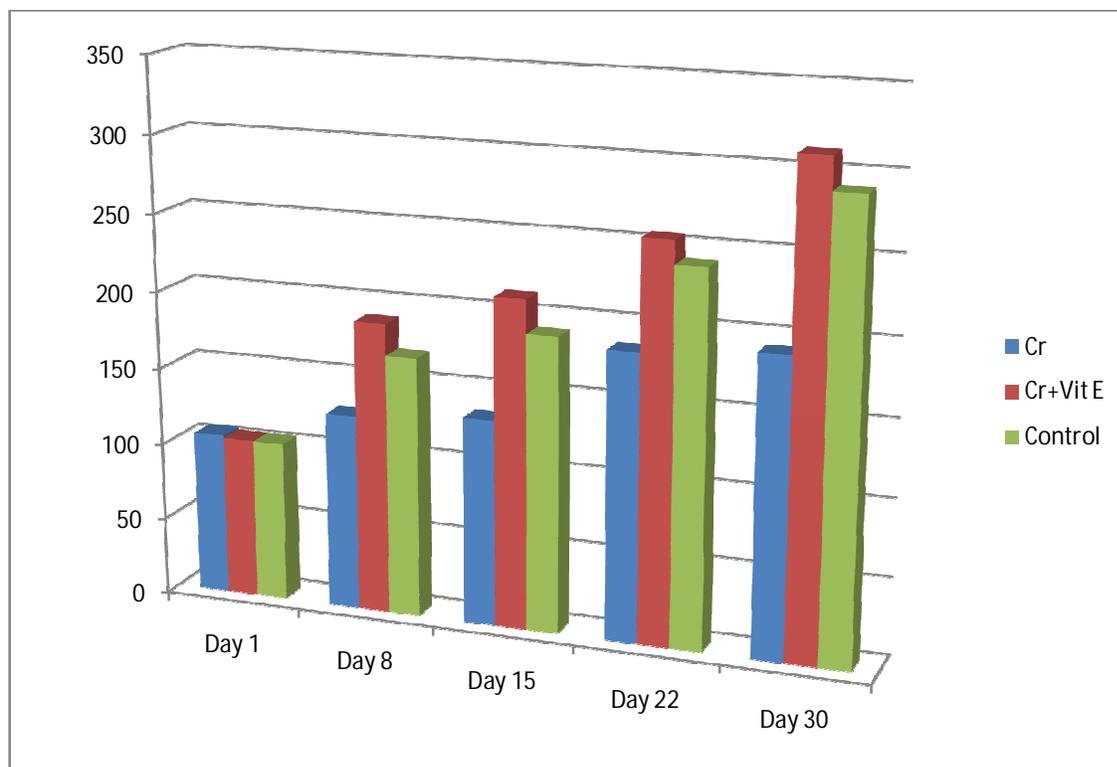


Figure 01: Influence of Vitamin E on live body weight in chromium fed chicks

Table 03: Mean values of tissue weight (g) in chicks of different groups administered with chromium, chromium + Vitamin E and control group.

Treatment	Liver	Kidney
Chromium	8.89±0.44	3.36±0.55
Chromium+Vitamin E	13.01±0.29	4.48±0.32
Control	12.25±0.55	4.12±0.07
ANOVA (p-value)	**	**

Results are expressed as mean ± SE. ** indicates significant at p< 0.05

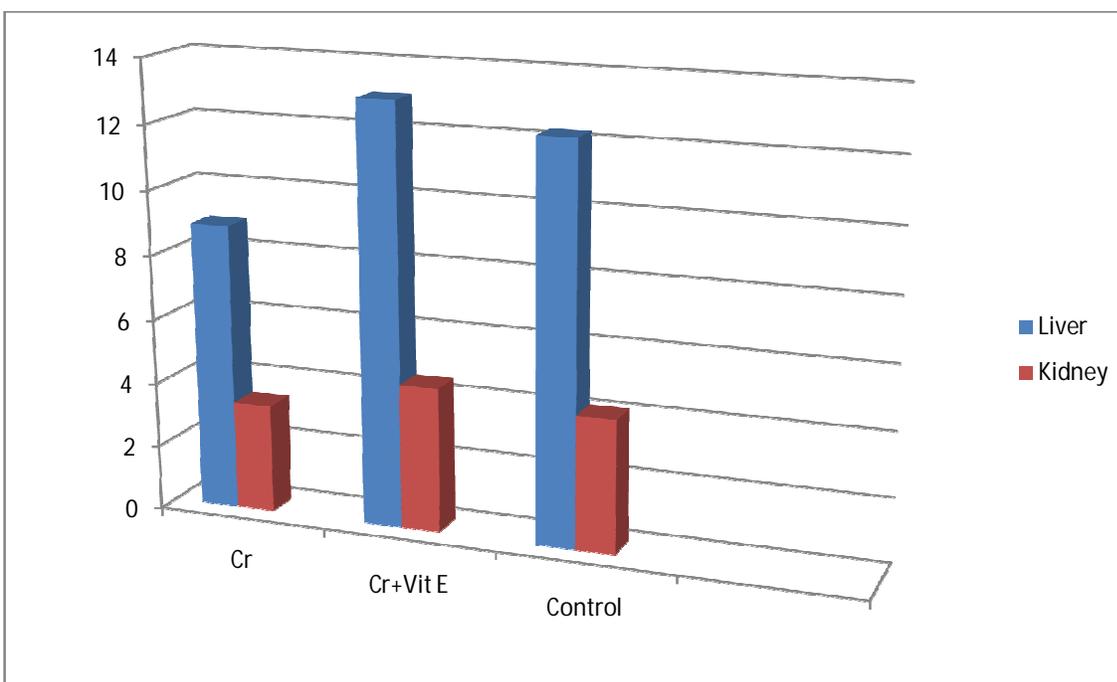


Figure 02: Influence of Vitamin E on tissue weight in chromium fed chicks

Studies reporting body weight effects in humans exposed to Cr(VI) were not identified. Significant decreases in body weight have been reported in several intermediate-duration oral Cr(VI) studies in animals¹¹⁻¹⁷. Previous studies carried out in broiler chicks¹⁸ and rats¹⁹ noted a significant reduction in body weight. Decrease in body weight could be due to decrease internal activity which damage health leading to impaired growth and weight gain²⁰. Another reason of decreased body weight could be due to irregularities in metabolism mediated by Cr-induced liver damage²¹.

The first symptom of toxic effect of chromium, observed in chicks, is the decreased live body weight. The indication of toxic effect is visible on the 8th day of intoxication when the animal become sluggish, lost interest in routine activities and stands idle in the corner of cage. The process continued and after fifteen days of intoxication the body weight further reduced significantly. There were significant differences in values obtained in the final weight of chicks across the treatments; this could be due to biotoxic effect of chromium to the body when consumed in a higher quantity. However, it should be noted that high concentrations of chromium in drinking water decrease palatability of water, resulting in decreased water consumption; thus, decreased body weight may, in part, be due to decreased water consumption, in addition to other causes. In male rats exposed to 73 mg Cr(VI) /kg/day as potassium dichromate in drinking water for 30 days, body weight was decreased by 11.6%¹⁶. A 19% decrease in body weight gain was observed in male rats exposed to 42 mg Cr(VI) /kg/day¹¹ and a 10% decrease was reported in male mice exposed to 6 mg Cr(VI) /kg/day¹³ as potassium dichromate in drinking water for 12 weeks.

In toxicated animals, the tissues weight is also reduced significantly. Thus, chromium causes deficient food intake that influence the development of the organs leading to reduce body weight. The decrease of body weight during chromium intoxication is also reported in mice, rat and human²²⁻²⁴. Identical results are obtained in mice²⁵, and chick^{26, 27} during mercury and methylmercury intoxication under similar experimental conditions.

Chromium intoxication also causes behavior alteration leading to dizziness, indigestion, diarrhea, abdominal pain, vomiting, gastritis and weakness⁸, that may stop the animals to take food, which results in reduced body weight. Decrease in protein content in all the tissues in intoxicated group of animals may be an important factor for weight loss in tissues and animal as a whole. Kim and Na²⁸ also found decrease in total amino acid within two hours of chromium injection. Chromium (III & VI) binds to active site of proteins and/or DNA to form complexes that results DNA-protein and DNA-amino acid cross links, DNA replication, DNA damage, gene mutation and gene expression²⁹⁻³².

Weight gain was maximum in vitamin E supplemented group and minimum in chromium intoxicated group. Overall performance revealed better weight gain in vitamin E supplemented group as compared to control group. Thus it is clear from the study that supplementation of vitamin E increases the food intake and not only recovered the animal's tissue and body weights but also changed the animal behavior making them active. Such changes can be clearly seen in growing birds. The present study reveals simultaneously recovery of animal tissue and body weights during vitamin E therapy. Thus, application of vitamin E not only eliminates toxicity of Cr and restores their reduced level but also restore tissue and animal weight leading to normal growth of the animal. This is also evident from body weight and size of therapeutic animals, which is more than compared to control on 15th and 30th day of their treatment.

CONCLUSION

From the above study it could be concluded that the inclusion of chromium have deleterious effects on chicks which could affect the growth performance, tissue weight and health status of the chicks. However, supplementation of vitamin E has the potential to provide protection in chicks against chromium induced toxicity in growth performance (body weight) and tissue weight respectively.

REFERENCES

1. Gabol K. Tabassum R. Khan MZ. Induced effect of Cadmium chloride on rock pigeon (*Columba livia*). *J. Nat. Hist. Wild.* 2003; 2(1): 39-43.
2. Lenntech. *Water Treatment and Air Purification* Published by Lenntech Rotterdamseweg, Netherlands. 2004.

3. Shaheen T. Akhtar T. Assesment of Chromium in *Cyprinus carpio* through hematological and bio-chemical blood markers. Turk. J. Zool. 2012; 36(5): 682-690.
4. Nolan K. Copper toxicity Syndrome, J. Orthomol, Psychiatry. 2003; 12(4): 270-282.
5. Young RA. Toxicity Profiles; Toxicity Summary for Cadmium Risk Assessment Information System, RAIS, University of Tennessee. 2005.
6. Abdul-Jameel A. Sirajudeen J. Abdul-vahith R. Studies on heavy metal Pollution of ground water sources between Tamilnadu and Pondicherry, India. Advances in Applied Science Research. 2012; 3(1): 424-429.
7. Demirezen D. Urac K. Comparative study of trace elements in certain fish, meat and meat products. Meat Sci. 2006; 74: 255-260.
8. ATSDR: Toxicological profile for chromium (update). U.S. Department of Health and Human Service, Public Health Services. 1999.
9. Yamaguchi S. Sano K. Shimojo N. On the biological half-time of Hexavalent chromium in rats, Ind. Health. 1983; 21: 25-34.
10. Singh J. Carlisle DL. Pritchard DE. et al. Chromium-induced genotoxicity and apoptosis; relationship to chromium carcinogenesis. Oncol. Rep. 1998; 5(6): 1307-1318.
11. Bataineh H, Al-Hamood MH, Elbetieha A, et al. Effect of long-term ingestion of chromium compounds on aggression, sex behavior and fertility in adult male rat. Drug Chem Toxicol. 1997; 20(3): 133–149.
12. Chowdhury AR, Mitra C. Spermatogenic and steroidogenic impairment after chromium treatment in rats. Indian J Exp Biol. 1995; 33: 480–484.
13. Elbetieha A, Al-Hamood MH. Long-term exposure of male and female mice to trivalent and hexavalent chromium compounds: Effect on fertility. Toxicology. 1997; 116: 39–47.
14. NTP. Final report on the reproductive toxicity of potassium dichromate (hexavalent) (CAS No. 7778-50-9) administered in diet to SD rats. National Institute of Environmental Health Sciences, National Toxicology Program. 1996a.
15. NTP. Final report on the reproductive toxicity of potassium dichromate (hexavalent) (CAS No. 7778-50-9) administered in diet to BALB/c mice. National Institute of Environmental Health Sciences, National Toxicology Program. 1996b.
16. Quinteros FA, Poliandri AHB, Machiavelli LI, et al. *In vivo* and *in vitro* effects of chromium VI on anterior pituitary hormone release and cell viability. Toxicol Appl Pharmacol. 2007; 218: 79–87.

17. Yousef MI, El-Demerdash FM, Kamil KI, et al. Ameliorating effect of folic acid on chromium(VI)-induced changes in reproductive performance and seminal plasma biochemistry in male rabbits. *Reprod Toxicol.* 2006; 21(3): 322–328.
18. Mohammed HH. El-Sayed BM. Abd El-Razik WM. et al. The influence of chromium sources on growth performance, economic efficiency, some maintenance behaviour, blood metabolites and carcass traits in broiler chickens. *Global Vet.* 2014; 12: 599–605.
19. Scibior A. Zaporowska H. Ostrowski J. et al. Combined effect of vanadium (V) and chromium (III) on lipid peroxidation in liver and kidney of rats. *Chemico-Biol. Interact.* 2006; 159: 213–222.
20. Mishra AK. Mohanty B. Chronic exposure to sublethal hexavalent chromium affects organ histopathology and serum cortisol profile of a teleost, *Channa punctatus* (Bloch). *Sci. Total Environ.* 2009; 407: 5031–5038.
21. Saxena D. Tripathi M. Hexavalent chromium induces biochemical alterations in air breathing fish, *Channa punctatus*. *J. Ecophys. Occup. Health.* 2007; 7: 171–175.
22. Glaser U, Hochrainer D, Steinhoff D. Investigation of irritating properties of inhaled CrVI with possible influence on its carcinogenic action. *Environ Hyg.* 1990; 2: 235–245.
23. Chowdhury AR, Mitram C. Spermatogenic and steroidogenic impairment after chromium treatment in rats. *Indian J. Exp. Biol.* 1995; 33: 480–484.
24. Junaid M, Murthy RC, Saxena DK. Embryo and fetotoxicity of chromium in pregestationally exposed mice. *Bull. Environ. Contam. Toxicol.* 1996; 57: 327–334.
25. Vinay SD. Raghu KG. Sood PP. An assessment of methylmercury evoked behavioral changes in rat and its chelation therapy. In: *Man Science and Environment* (Prakash, R. ed.), Ashish Pub. House, New Delhi, 1993; 123-138.
26. Patney V. Chundawat RS. Sood PP. *Poll. Res.* 2002; 21: 483-493.
27. Sood PP. Sinha N. Ansari NH. *Res. Com. Pharmacol. Toxicol.* 2002; 7: 3-22.
28. Kim E. Na KJ. Acute toxic effect of sodium dichromate on metabolism. *Arch. Toxicol.* 1990; 64: 644-649.
29. De Flora S. Banasco M. Serra D. et al. Genotoxicity of Chromium compounds: A Review. *Mutat. Res.* 1990; 238: 99-172.
30. Leonard A. Mechanism in metal genotoxicity: The significance of in vitro approaches, *Mutat. Res.* 1988; 198: 321-326.
31. Manning FCR. Xu J. Patierno SR. Transcription inhibition by carcinogenesis chromate: relationship to DNA damage. *Mol. Carcinog.* 1992; 6: 270-279.

32. Manning FCR, Blankenship LJ, Wise JP. et al., Induction of internucleosomal DNA fragmentation by carcinogenic chromate: Relationship to DNA damage, genotoxicity, and inhibition of macromolecular synthesis. *Environ. Health Perspect.* 1994; 102: 159-167.