



RESEARCH ARTICLE

Clinicopathological Studies on the Ameliorative Effects of Selenium and Vitamin E against Cadmium Toxicity in Chickens

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Abstract

Cadmium (Cd) is one of the most dangerous environmental bioaccumulative heavy metals that concern a vital interest due to its injurious to animal and public health. An experimental trial was conducted for six weeks to evaluate the protective effect of selenium and /or vitamin E against the adverse effect of cadmium. One hundred and five, one day old broiler chicks were divided into five random groups. Group1 (negative control) was fed on basal diet; Group 2 was supplemented with 100 mg of cadmium /kg diet; Group 3 was supplied with 0.5 mg/kg diet of selenium; Group 4 was fed on 100 mg/kg diet of vitamin E and Group 5 was supplemented with Cd, Se and vitamin E combinations. The results revealed that cadmium group showed a highly significant increase in serum alanine amino transferase (ALT) and aspartate amino transferase (AST) (36 ± 1.1 and 220 ± 3.53 , respectively) activities, serum uric acid and creatinine (25.68 ± 1.01 and 4.38 ± 0.15 , respectively) and a significant decrease in serum total proteins, albumin and globulins values (2.34 ± 0.09 , 1.14 ± 0.05 and 1.20 ± 0.05 , respectively) particularly at the end of the sixth week in comparable to the control group. In addition, a significant increase in serum malondialdehyde (MDA) and nitric oxide (NO) (3.9 ± 0.09 and 51.33 ± 0.88 , respectively), a significant decrease in glutathione peroxidase (GSH-PX) values (0.05 ± 0.005) associated with a significant reduction in interleukin 2 (IL2) and interleukin 10 (IL10) (1.8 ± 0.30 and 7.4 ± 0.43 , respectively) values were reported specially at the end of the sixth week compared with the control group. Overall, our results showed an improvement in groups supplied with either Se or vitamin E when compared with cadmium group. In conclusion, supplementation of selenium or vitamin E alone didn't recover the depressive effect of Cd but their combination could improve its deleterious effect due to their antioxidant activity.

Keywords: Cadmium, Selenium, Vitamin E, Antioxidant, Oxidative stress.

Introduction

Cadmium (Cd) is an environmental modern toxic heavy metal which is widely distributed. Toxicity occurs due to industrial and agricultural pollution [1]. Animals can be subjected to Cd pollution through respiration, food and water intake [2]. The highest accumulation of cadmium occurs in kidney and liver and its biological half-life in these organs is long, approximately 20 to 30 years. It is absorbed poorly then transported to plasma, bound with albumin and accumulated in kidney and liver [3]. Cadmium is forming

reactive oxygen species (ROS) that increases the lipid peroxidation, depresses the antioxidants such as glutathione and protein-bound sulfhydryl groups, produces inflammatory cytokines and decreases the protective effect of nitric oxide [4,5]. The mechanism of cadmium toxicity primarily relied on increasing the production of free radicals resulting in oxidative effects in a number of tissues such as kidneys [6] and livers [7], which are the primary target of Cd toxicity.

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Selenium (Se) is a vital micronutrient and is considered one of the most efficient trace elements against Cd toxicity [8]. It could protect the renal and hepatic tissues against Cd toxicity by the antioxidant effect; hence it reduces lipid peroxidation (LPO) and increases the activities of the antioxidant enzymes in these tissues [9]. Vitamins are ideal antioxidants as they increase tissue protection from oxidative stress due to their easy, effective and safe dietary administration [10, 11]. One of the most important vitamins is vitamin E, which is a biological antioxidant that improved growth, physiological, and immunological performance in broiler chickens because of its ability to neutralize the free radicals and reduce the lipid peroxidation [12]. Both vitamin E and selenium are essential nutrients for humans and animals. They are involved in the protection of biological membranes against lipid peroxidation and preventing the free radicals damage to phospholipids` membranes and enzymes [13]. This study was undertaken to estimate the protective effect of selenium and/or vitamin E against cadmium toxicity in broilers.

Materials and Methods

Animals

One hundred and five-one day old broiler chicks were obtained from Ommate for Poultry Production Company, Dakahlia Governorate, Egypt. Chicks were housed under proper hygienic conditions, maintained on a commercial balanced starter and finisher rations and water ad-libitum throughout the period of the experiment.

Chemicals

Cadmium: whitish red colour powder as Cadmium Chloride (CdCl_2) obtained from El-Gomhoria Company, Zagazig, Egypt, and was used at a dose of 100 mg /kg body weight (BW) as described previously [14]. Selenium as sodium selenite (Na_2SeO_3 ; Alfa Aesar, Ward Hill MA 01835, USA) is anhydrous salt, typically 99.75% mineral (metal basis) was used at a dose of 0.5 mg/kg BW as described elsewhere [15]. Vitamin E was obtained from Pharco Pharmaceutical Company, Alex., Egypt, and was used at dose of 100 mg/kg BW [16].

Experimental design

One hundred and five-one day old chicks were divided into five groups each contained 20 chicks except Group 2 which contained 25 birds. The experiment was designed as following: Group 1 was fed on a basic diet and kept as a negative control; Group 2 was supplied by a basal diet included 100 mg Cd/ kg diet; Group 3 was given 0.5 mg Selenium as sodium selenite (Na_2SeO_3) /kg plus 100 mg Cd/kg of diet; Group 4 was administered by a diet enriched with 100 mg vitamin E /kg and 100 mg Cd /kg feed, and Group 5 was fed on a combination of 100 mg Cd /kg + 0.5 mg Se /kg +100 mg vitamin E /kg diet. The previous treatments were supplemented from one day to six weeks old. Clinical signs and mortality rate were recorded through the experimental period. This study was approved by the committee of Animal Welfare and Research Ethics, Faculty of Veterinary Medicine Zagazig University.

Sampling

At the end of the 2nd, 4th and 6th weeks of the experiment, blood samples were collected from randomly selected birds (n=5) of each group. Blood samples (3mL) were obtained from the wing vein in a clean and dry plan centrifuge tube without anticoagulant, left to clot at room temperature and then centrifuged at 3000 rpm for 5 min for separation of serum and assay of serum biochemical parameters.

Biochemical assays

Calometric method was used for determination of serum ALT (alanine amino transferase) and AST (aspartate amino transferase) activities [17]. Total protein [18] and albumin levels [19] were determined, as well as serum globulins which were calculated by subtracting the obtained albumin levels from the total protein level as described previously [20]. Serum uric acid [21] and serum creatinine levels [22] were also detected.

Oxidative stress markers

Serum malondialdehyde (MDA) level was estimated according a previous published paper [23]. Nitric oxide (NO) and serum glutathione peroxidase (GSH-PX) activities were estimated according to the methods described elsewhere [24, 25].

Immunological studies

Determination of interleukin 2 and interleukin 10 (IL2 and IL10) levels were performed according the manufacture instruction in the pamphlet of CUSABIO BIOTECH CO, LTD, PRC; Cat. No. CSB-E06755CH and Kamiya Biochemical Company US; Cat. No. KT-18959, respectively.

Statistical analysis

The obtained data were statistically analyzed by F-test [26] using "MSTAT-C" computer program. Means in the same columns follows by different letters were statistically significant and the highest values represented with the letter a.

Results

Administration of cadmium (Group 2) resulted in depression, restlessness, gasping, dullness, recumbancy, ruffled feathers, increase of water consumption, anorexia and diarrhea; other cases showed imbalance gait and lameness. These signs appeared from the 2nd week of age and increased at the end of the experiment. On the other hand, chickens in

Groups 3, 4 and 5 showed milder signs as slight dullness and decrease in water consumption that improved at the end of the 4th week. The mortality rate was 32% in chicks of group 2 that supplemented with cadmium only as one chick died in the 1st week, two in the 3rd week and five by the end of 6th week. This ratio was decreased to 20% in the third group that treated with selenium as four chicks died in the 2nd and 3rd week, 15 % in group 4 fed on vitamin E as one bird died in the 1st week and two in the 2nd week, while it reduced to 5% in group 5 supplemented with cadmium, selenium and vitamin E as one bird died at the 3rd week.

As shown in Table 1, chickens orally supplemented with cadmium revealed highly significant increase in serum ALT and AST (36±1.1 and 220±3.53, respectively) activities especially at the end of the sixth week. On the other hand, there was a significant decrease in serum total proteins, albumin and globulins values in the same group all over the experimental period particularly at the end of the sixth week (2.34±0.09, 1.14±0.05 and 1.20±0.05, respectively) in comparable to the control group (Table 2).

Table 1: Ameliorative effects of selenium and /vitamin E against cadmium toxicity on ALT and AST activities (mean values ±S.E) in broiler chickens under experimental conditions

Groups	2 nd week		4 th week		6 th week	
	ALT	AST	ALT	AST	ALT	AST
Gp 1 (control)	14.74±0.43 ^c	104.0±0.70 ^e	17.12±0.6 ^c	107.4±0.92 ^d	18±1.7 ^c	106±1.41 ^d
Gp 2 (cadmium)	29.40±1.77 ^a	179.2±1.74 ^a	35.2±0.8 ^a	211.4±2.50 ^a	36±1.1 ^a	220±3.53 ^a
Gp 3 (Cd + Se)	23.38±0.53 ^b	161.6±1.36 ^b	25.6±0.81 ^b	147.6±4.03 ^b	25±2.3 ^b	127±1.61 ^b
Gp 4 (Cd + Vit E)	22.28±0.58 ^b	147.8±2.13 ^c	24.7±7.6 ^b	144±4.97 ^b	24.33±2.6 ^b	125±2.46 ^b
Gp 5 (Cd + Se + Vit E)	21.58±0.50 ^b	142.8±1.01 ^d	21.8±0.58 ^{bc}	127±2.09 ^c	19±0.9 ^c	107.13±1.61 ^d

Means within the same column which are significantly different are followed by different letters. P < 0.05

Gp: Group, Cd: Cadmium, Se: Selenium, Vit: Vitamin, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase. ALT and AST levels were measured by U/L

Table 2: Serum proteinogram (mean values ± S.E) in different groups in broiler chickens with cadmium toxicity and treated with vitamin E and /selenium

Groups	2 nd week			4 th week			6 th week		
	TP	Albumin	Globulins	TP	Albumin	Globulins	TP	Albumin	Globulins
Gp 1 (control)	4.51±0.02a	2.57±0.04a	1.94±0.03a	4.76±0.27a	2.62±0.08a	2.14±0.14a	4.98±0.14a	2.68±0.09a	2.30±0.09a
Gp 2 (cadmium)	3.16±0.09d	1.9±0.07c	1.26±0.04c	2.38±0.19d	1.36±0.11c	1.02±0.06c	2.34±0.09c	1.14±0.05c	1.20±0.05c
Gp 3 (Cd + Se)	3.74±0.06bc	2.16±0.09b	1.58±0.10b	3.77±0.22c	2.04±0.07b	1.73±0.08b	4.28±0.07b	2.25±0.03b	2.03±0.06b
Gp 4 (Cd + Vit E)	3.62±0.05c	2.14±0.05b	1.48±0.04b	3.84±0.17c	2.07±0.06b	1.77±0.05b	4.33±0.14b	2.24±0.01b	2.09±0.02b
Gp 5 (Cd+Se +Vit E)	3.92±0.07 ^b	2.26±0.08 ^b	1.66±0.05 ^b	4.65±0.20 ^a	2.53±0.03 ^a	2.12±0.18 ^a	4.82±0.23 ^a	2.55±0.03 ^a	2.27±0.02 ^a

Gp: Group, Cd: Cadmium, Se: Selenium, Vit: Vitamin, TP: Total protein.

Means within the same column which are significantly different are followed by different letters, P < 0.05

All tested parameters were measured by g/dL.

Oral supplementation of broilers with either Se or vitamin E in the third and fourth groups revealed also a significant increase in ALT and AST activities with a significant decrease in values of TP, albumin and globulin compared with the healthy control group all over the experimental period. In comparison with cadmium group, they showed a significant decrease in serum ALT and AST activities (19 ± 0.9 and 107.13 ± 1.61 , respectively). Moreover, a significant increase in serum total proteins, albumin and globulins was observed especially at the end of the 6th week to be non-significant in the 5th group and return to values of control at the end of 4th and 6th week (4.82 ± 0.23 , 2.55 ± 0.03 and 2.27 ± 0.02 , respectively) (Tables 1, 2).

Serum uric acid and creatinine were performed to evaluate the effect of cadmium nephrotoxicity on broilers. The highly significant elevation was observed in serum uric acid and creatinine levels all over the experimental period in Cd oral supplementation only (Table 3). However, including of either Se or vitamin E in diet resulted in a significant increase of these parameters at the second week with an improvement at 6th week compared with

cadmium group to be non-significant at the fourth and sixth weeks in chickens of group 5 compared with the control group (Table 3).

As shown in Table 4, a significant increase in serum MDA and NO values and a significant decrease in serum GSH-PX activities were observed in broilers orally supplemented with Cd all over the study compared to the control Group. Administration of selenium and /or vitamin E resulted in a significant decrease in oxidative stress parameters with a significant increase in serum GSH-PX activities compared with cadmium Group. However, the 5th Group showed a significant change in the previous parameters at the end of 2nd week with non-significance at the end of 4th and 6th weeks to return to the values of the control group.

As described at Table 5, Cd toxicity had an adverse effect on the immune status of the birds manifested by decreasing serum IL2 and IL10 levels in cadmium treated chicken (Group 2). On the other hand, Groups 3 and 4 showed a significant increase in serum IL2 and IL10 values compared with the cadmium group. However, complete improvement was observed in serum IL2 and IL10 values in the 5th group compared with the control group.

Table 3: Some renal function tests (mean values \pm S.E) in different groups of broiler chickens with cadmium toxicity and treated with vitamin E and /selenium

Groups	2 nd week		4 th week		6 th week	
	Uric acid	Creatinine	Uric acid	Creatinine	Uric acid	Creatinine
Gp 1 (control)	5.5 ± 0.40^c	1.26 ± 0.07^c	5.83 ± 0.5^c	1.35 ± 0.02^c	5.52 ± 0.11^c	1.28 ± 0.02^c
Gp 2 (cadmium)	12.5 ± 1.32^a	2.88 ± 0.18^a	19.4 ± 2.6^a	3.23 ± 0.28^a	25.68 ± 1.01^a	4.38 ± 0.15^a
Gp 3 (Cd + Se)	9.53 ± 0.40^b	1.84 ± 0.08^b	9.0 ± 0.5^b	1.9 ± 0.12^b	7.38 ± 0.23^b	1.75 ± 0.05^b
Gp 4 (Cd + Vit E)	9.23 ± 0.49^b	2 ± 0.23^b	8.2 ± 0.54^b	1.78 ± 0.11^b	7.18 ± 0.12^b	1.63 ± 0.08^b
Gp 5 (Cd + Se + Vit E)	8.2 ± 0.40^b	1.9 ± 0.03^b	6.2 ± 0.23^c	1.44 ± 0.57^c	5.68 ± 0.16^c	1.30 ± 0.02^c

Gp: Group, Cd: Cadmium, Se: Selenium, Vit: Vitamin

Means within the same column which are significantly different are followed by different letters, $P < 0.05$

All tested parameters were measured by mg/dL

Table 4: Serum markers for oxidative stress and antioxidant activities (mean values \pm S.E) in different groups of broiler chickens with cadmium toxicity and treated with vitamin E and /selenium

Groups	2 nd week			4 th week			6 th week		
	MDA nmol/mL	NO nmol/mL	GPX mg/dL	MDA nmol/mL	NO nmol/mL	GPX mg/dL	MDA nmol/mL	NO nmol/mL	GPX mg/dL
Gp 1 (control)	1.3 \pm 0.05 ^c	18 \pm 0.58 ^d	0.51 \pm 0.01 ^a	1.4 \pm 0.05 ^d	18.6 \pm 0.52 ^d	0.45 \pm 0.02 ^a	1.6 \pm 0.10 ^c	19.2 \pm 0.53 ^c	0.48 \pm 0.017 ^a
Gp 2 (cadmium)	2.9 \pm 0.23 ^a	43.67 \pm 0.88 ^a	0.19 \pm 0.02 ^d	3.5 \pm 0.34 ^a	47 \pm 1.15 ^a	0.09 \pm 0.005 ^c	3.9 \pm 0.09 ^a	51.33 \pm 0.88 ^a	0.05 \pm 0.005 ^d
Gp 3 (Cd + Se)	2.2 \pm 0.11 ^b	36.33 \pm 0.88 ^b	0.23 \pm 0.01 ^c	2.4 \pm 0.10 ^b	31.67 \pm 0.88 ^b	0.25 \pm 0.017 ^b	2.5 \pm 0.05 ^b	28.33 \pm 0.87 ^b	0.42 \pm 0.011 ^c
Gp 4 (Cd + Vit E)	2 \pm 0.11 ^b	34.33 \pm 0.87 ^b	0.28 \pm 0.02 ^c	2.2 \pm 0.13 ^{bc}	29.67 \pm 0.87 ^b	0.27 \pm 0.005 ^b	2.3 \pm 0.08 ^b	27 \pm 1.14 ^b	0.43 \pm 0.005 ^{bc}
Gp 5 (Cd+Se +Vit E)	1.9 \pm 0.17 ^b	28 \pm 1.15 ^c	0.40 \pm 0.02 ^b	1.7 \pm 0.12 ^{cd}	20.33 \pm 0.57 ^d	0.37 \pm 0.17 ^a	1.7 \pm 0.11 ^c	21.3 \pm 0.8 ^c	0.46 \pm 0.005 ^{ab}

Gp: Group, Cd: Cadmium, Se: Selenium, Vit: Vitamin, MDA: Malondialdehyde, NO: Nitric oxide, GPX: Glutathione peroxidase

Means within the same column which are significantly different are followed by different letters, P <0.05

Table 5: Interleukin 2 and interleukin 10 levels (mean values \pm S.E) in different groups of broiler chickens with cadmium toxicity and treated with vitamin E and /selenium

Groups	2 nd week		4 th week		6 th week	
	IL2	IL10	IL2	IL10	IL2	IL10
Gp 1 (control)	6 \pm 0.57 ^a	16 \pm 0.28 ^a	6.6 \pm 0.28 ^a	16.7 \pm 0.26 ^a	6.6 \pm 0.50 ^a	16.5 \pm 0.40 ^a
Gp 2 (cadmium)	2.5 \pm 0.28 ^c	9.2 \pm 1.2 ^c	1.8 \pm 0.17 ^d	8.5 \pm 0.86 ^c	1.8 \pm 0.30 ^c	7.4 \pm 0.43 ^c
Gp 3 (Cd + Se)	2.8 \pm 0.11 ^c	10.8 \pm 0.62 ^{bc}	3.8 \pm 0.20 ^c	11.26 \pm 0.69 ^b	5.6 \pm 0.10 ^b	14.2 \pm 0.40 ^b
Gp 4 (Cd + Vit E)	3 \pm 0.17 ^{bc}	11.1 \pm 0.78 ^{bc}	4.1 \pm 0.43 ^c	11.9 \pm 0.51 ^b	5.4 \pm 0.40 ^b	14.5 \pm 0.28 ^b
Gp 5 (Cd + Se + Vit E)	3.8 \pm 0.14 ^b	12.2 \pm 0.46 ^b	6.1 \pm 0.14 ^a	15.23 \pm 0.56 ^a	6.3 \pm 0.35 ^a	15.9 \pm 0.38 ^a

Gp: Group, Cd: Cadmium, Se: Selenium, Vit: Vitamin, IL: Interleukin.

Means within the same column which are significantly different are followed by different letters, P <0.05

Values were measured by Pg/mL

Discussion

Cadmium is an environmental toxic heavy metal; a potential pollutant resulted from industrial and agricultural sources [27]. The long biological half-life of cadmium makes Cd as a cumulative toxin, [28], which is primarily distributed to the liver and then redistributed to the kidney [29]. Selenium supplementation in poultry is associated with energy metabolism, increased feed efficiency, improved reproduction, and improved immune responses. Poultry are exposed to environmental stressors that require the antioxidant protection of selenium supplementation. Organic forms of selenium have been shown to be generally safer and better absorbed [30]. Vitamin E has many biological functions, the antioxidant function being the most important due to its easy, effective and safe dietary administration [31].

This study was performed to evaluate the effect of selenium, vitamin E or their combination to recover the hazard effect of cadmium toxicity. The observed clinical signs of chickens treated with cadmium showed feed refusal, loss of appetite, dullness, restlessness, depression, gasping, difficult breathing, ruffled feather, inability to stand and move, weakness, recumbancy, imbalance gait which may progress to lameness and diarrhea. In some cases which may be due to Cd toxic immunosuppressive effect, increased the susceptibility of the birds to stress and disease was observed [32, 33]. Mortality rate of groups 2, 3, 4 and 5 were 32, 20, 15 and 5%, respectively. In a previous study [34], the mortality rate in guinea pigs feeding a diet supplemented with 2.5 mg Cd/Kg BW was 39.4 %. This may be due to the morphological and functional damage in liver and kidney [35]. These clinical signs became milder in groups 3, 4 and 5, as selenium and vitamin E

enhance the immune responses and prevent of clinical signs of disease or stress [36, 37].

The study revealed also a significant increase in liver enzymes ALT and AST especially at the end of the sixth week (6 ± 1.1 and 220 ± 3.53) in cadmium group which may be due to the outflow of these enzymes from the liver to the blood due to hepatotoxic effect of Cd. In several reports, administration of antioxidants like vitamin E to diet with Cd significantly decreased the activities of serum AST, ALT to return normal values in group 5 showed no significant change (19 ± 0.9 and 107.13 ± 1.61) compared to control group as these antioxidants may stabilize the hepatic cellular membrane and protect the hepatocytes against toxic effects of Cd, which may decrease the leakage of the enzymes into blood stream. Moreover, the Se limitation of the deleterious effect on liver enzymes might be due to an interaction of Se with Cd forming biologically inactive cadmium solenoid complex [38-41]. Explanation of our results in the 4th group is assumed as free radicals scavengers and antioxidant such as vitamin E have an important role in protection against Cd [41].

Regarding to proteinogram of birds exposed to cadmium toxicity in the 2nd Group, a significant decrease in the values of serum total proteins, albumin and globulins was observed. It could be attributed to hepatic and renal tubular dysfunction, change in protein synthesis and/or metabolism or decrease in feed consumption [33, 43-46]. Meanwhile, the protective mechanism of Se, vitamin E and/or both in supplemented chickens may be attributed to their ability to scavenge ROS and enhancement of antioxidant system in Cd induced tissue damage as the interactions between Se and high level of Cd resulted in partial amelioration of Cd toxicity in different system [42].

Kidney function tests showed an increase in serum creatinine and uric acid levels (225.68 ± 1.01 and 4.38 ± 0.15 , respectively) in chicken of Cd group compared to control group. Such results may be due to the

nephrotoxic effect of cadmium on renal tubules and glomerular filtration [47, 48]. Supplementation of Se and vitamin E or both with Cd led to significant reduction in the values of creatinine and uric acid (5.68 ± 0.16 and 1.30 ± 0.02 , respectively) compared with Cd group that may be due to antioxidant effect, free radicals scavenging action and protection of protein thiols from deleterious effect of Cd in kidney [49].

Concerning the oxidative stress and antioxidant markers evaluation in the present study, cadmium toxicity induced a significant increase in MDA and NO values (3.9 ± 0.09 and 51.33 ± 0.88 , respectively), whereas serum GPX activity showed a significant decrease (0.05 ± 0.005) in comparison with the control group (0.48 ± 0.017). This can be explained by imbalance between the oxidant / antioxidant action induced by cadmium [50, 51] or due to the release of ROS NO H₂O [52]. Increased renal lipid peroxidation associated with reduction of GPX or due to Cd exposure disrupted the oxidative stress in hens according to a previous study [53]. Moreover, Kant *et al.* [54] reported that blood glutathione is important in protecting the cell against Cd toxicity and its decrease may be due to exhaustion of GSH stores and the increase in oxidative stress.

Administration Se and vitamin E and their combination revealed a significant decrease in serum MDA and NO (2.3 ± 0.08 and 27 ± 1.14 , respectively) values with a significant increase in serum GPX activity (0.43 ± 0.005) in group 5 compared to cadmium group directed toward control values in the fifth group at the end of fourth and sixth weeks. This improvement could be due to (i) the antioxidant action of Se and vitamin E as Se enhances the natural antioxidant body system and prevents the formation of free radicals and the process of lipid peroxidation or (ii) Se acts as substance for various enzymes such as GPX and it is important in sulphur amino acid metabolism that protect the body against several diseases [55]. Vitamin E is a potent lipid soluble chain breaking antioxidant, which protects cellular

membrane and lipoprotein surface from lipid peroxidation and its protective role has been reported against heavy metals [56]. Synergistic effect of antioxidant such as Se and vitamin E is the most powerful in reducing storage and toxicity of ROS [57].

A significant reduction in the IL2 and IL10 were observed in this study as Cd affect negatively on the immune status of the bird. However, the reduction of IL2 could be attributed to general decline in immune function and immune regulation [58]. IL2 decreases could result in detrimental effects on the immune system because it is essential for lymphocyte proliferation. On the other hand, the reduction in IL-10 values in chickens of cadmium group could be due to the hyperoxiditive state that release ROS which lead to organ damage [59]. However, administration of Se and vitamin E or both elevated the decrease of IL2 and IL10 and return them to their normal value in group 5 at the end of the six weeks, Alkhedaide *et al.* [60] found that the grape seed (potent antioxidant) ameliorate the Cd toxicity on IL10 in albino rats through alleviation the oxidative stress.

Conclusion

Cadmium had extremely adverse harmful effects that negatively affect the hepato-renal function, antioxidants and oxidative stress markers associated with the depression of the bird immunological status. Administration vitamin E or selenium alone with cadmium didn't alleviate its negative effects but their combinations could overcome its adverse effects.

Conflict of Interest

The authors stated that there are no conflicts of interest.

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المخلص العربي

دراسات باثولوجية اكلينيكية على التأثير المحسن للسيلينيوم وفيتامين هـ ضد التأثير السمي للكادميوم فى الدجاج

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يعد الكادميوم واحدا من اخطر المعادن الثقيلة المتراكمه بالبيئه و التى احتلت اهتماما كبيرا لما لها من تاثير سلبي على الحيوان والصحة العامه. قد اجريت تجربة اصطناعية على مدار ستة اسابيع لقياس التأثير الوقائى لمادتي السيلينيوم مع/ او فيتامين هـ ضد الاثر السلبي لمادة الكادميوم. تم تقسيم عدد ١٠٥ كتكوت عمر يوم الى خمس مجموعات عشوائيه. المجموعه الاولى (المجموعه الضابطه) تم تغذيتها على عليقه عاديه، المجموعه الثانيه تم امدادها ب ١٠٠ ملغ جرام من الكادميوم/ كجم من العليقه، المجموعه الثالثه تم اعطائها ٠.٥ ملغ جرام من السيلينيوم/ كجم من العليقه، المجموعه الرابعه تم اعطائها ١٠٠ ملغ جرام من فيتامين هـ/ كجم من العليقه و المجموعه الخامسه تم امدادهم بالكادميوم و السيلينيوم و فيتامين هـ سويا. اوضحت النتائج لمجموعه الكادميوم زياده معنويه لنشاط انزيمات الاسبارتيت امينو ترانسفيريز، الالانين امينو ترانسفيريز (36 ± 1.1 و 220 ± 3.53) على التوالي، حمض اليوريك و الكرياتينين (25.68 ± 1.01 و 4.38 ± 0.15) على التوالي و انخفاض معنويا لمعدل البروتين الكلى و الالبومين و الجلوبيولين فى الدم (2.34 ± 0.09 و 1.14 ± 0.05 و 1.20 ± 0.05) على التوالي وخاصة فى نهاية الاسبوع السادس مقارنة بالمجموعه الضابطه. علاوة على ذلك زياده معنويه فى معدل المولودايالهيد و اكسيد النيتريك (3.9 ± 0.09 و 51.33 ± 0.88) ونقص معنوى فى معدل الجلوتاثيون المؤكسد (0.05 ± 0.005) و خاصة فى نهاية الاسبوع السادس بالاضافه الى انخفاض معنوى فى الانترلوكين ٢ و ١٠ (1.8 ± 0.30 و 7.4 ± 0.43) على الترتيب و خاصة فى نهاية الاسبوع السادس مقارنة بالمجموعه الضابطه. كل النتائج اظهرت تحسنا ملحوظا فى المجموعات التى تم امدادها بالسيلينيوم او فيتامين هـ عند مقارنتها بمجموعه الكادميوم. مما سبق نستنتج ان اضافه السيلينيوم او فيتامين هـ منفردا لاتعطى التأثير السلبي للكادميوم ولكن عند اضافتهما معا تحسن التأثير الضار للكادميوم بسبب فاعليتهم المضاد للاكسده