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EFFECT OF DIFFERENT DIETRAY LEVELS OF CADMIUM AND SELINIUM AND THEIR MIXTURE ON 2. BIOCHEMICAL CONSTITUENTS OF BROILER CHICKENS

By

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Abstract: *This investigation was carried out to study the effect of selenium (Se) and cadmium (Cd) separately and their mixture on biochemical constituents of broiler chicks. A total number of 320, one day old of commercial broiler chicks were housed in galvanized wire-floored pens. Birds were distributed randomly into 16 dietary treatments groups, each groups represented by duplicate pens (10 chicks each) until the end of the experiment (49 days of age). The 16 dietary treatments were divided into 4 major groups. First group was used as a control and fed the starter and finisher basal diet. The second treatment group was fed the basal diet supplemented with 0.5, 1.0, 1.5, 2.0 and 2.5 mg Se as a sodium selenite (Na_2SeO_3) /kg in the starter diet and 2.0, 4.0, 6.0, 8.0 and 10.0 mg Se as a sodium selenite (Na_2SeO_3) /kg in the finisher diet, respectively. The third treatment group was fed the basal diet supplemented with 0.25, 0.5, 0.75, 1.0 and 1.25 mg Cd as a Cd chloride ($CdCl_2$) /kg in the starter diet and 1.0, 2.0, 3.0, 4.0 and 5.0 mg Cd as a Cd chloride ($CdCl_2$) /kg in the finisher diet, respectively. The fourth group was fed the starter and the finisher basal diet supplemented with mixture of the different levels of Se and Cd as previously mentioned in the second and the third groups.*

Increasing Se, Cd and their mixture levels did not significantly affect serum total lipid concentrations. Total lipid concentrations in flesh showed neither linear nor quadratic responses with addition of various levels of Se, Cd and their mixtures. Increasing Se levels increased linearly hepatic total lipid concentrations. Increasing Se and Cd levels decreased linearly serum total protein concentrations, while hepatic total protein significant increased. Increasing Se and Cd mixture levels showed linear increase in serum and hepatic total soluble protein concentrations. Results indicated that increased dietary Se, Cd and their mixture concentrations decreased POD activity but this decrease was significant only with mixture levels. However, the activity of GOT and GPT was not affected by Se, Cd and their mixture. The number and intensity of the protein bands increased with low dietary Se levels. Increasing Cd levels did not remarkably change the number and the intensity of the bands compared to that of control. However, increasing Se with Cd concentration remarkably increased the number and intensity of the bands compared to that of control.

INTRODUCTION

Metals are redistributed naturally in the environment by both geologic and biological cycles. The later includes bio-concentration by plants and animals and incorporation into food cycles. These natural cycles may exceed the anthropogenic cycle (Ng and Paterson, 1981).

Se is well known to be an essential trace element with many vital functions in humans and animals. In nature, it can be found in the elemental form as well as incorporated into both inorganic and organic compounds. Se is the central structural component of specific selenoenzymes including glutathione peroxidases, iodothyronine deiodinases, thioredoxin reductases, selenophosphate synthetase and many others. To date, about 30 selenoproteins have been identified, but the precise function of most of them is not yet known. The best understood selenoenzyme are cytosolic glutathione peroxidase (cGSH-Px, EC 1.11.1.9), which works as the antioxidant by removing reactive oxygen species (Behne and Kyriakopoulos, 2001 and Surai, 2006). The GPX family of enzymes are crucial players in the integrated antioxidant system, neutralizing potential threats to the integrity of cellular macromolecules by eliminating hydrogen peroxide and detoxifying lipid hydro peroxides (Brigelius-Flohe, 1999). Adequate intake of Se is needed for immunocompetence and Se deficiency resulted in the insufficient cell and humeral immune response of humans and animals (Surai, 2006). On the other hand, super nutritional doses of Se have been found to have anticarcinogenic effects in humans (Schrauzer, 2000).

The liver is involved in the metabolism of selenium, and the content of Se in the liver likely reflects overall Se status (Daniels, 1996). Cetin *et al.* (2002) showed that organic and inorganic Se supplementation (0.3 ppm/kg diet) did not affect blood biochemical parameters and did not negatively affect health of broiler. Kucharzewski *et al.* (2003) found that blood glutathione peroxidase activity was depended on dietary intake of Se with no differences between forms of administrations. Also, Se concentration in the blood and liver showed similar manner of responses. Moreover, Habiba (2006) found that Se addition increased plasma globulin and total protein concentration while, the albumen/globulin ratio significantly decreased. Ljubic *et al.* (2006) suggested that organic Se supplementation influences cholesterol metabolism in adipose tissue by decreasing total cholesterol concentration during the fattening period and increasing free cholesterol concentration after 48-hour feed deprivation.

Kjellstrom, *et al.* (1975) recognized that Cd is a toxic metal that have environmental pollutant effect and accumulated in the lever and kidneys. Nezel *et al.* (1981) observed that Cd acetate and cysteine-bound Cd (20 and 40 mg Cd/kg diet) increased Cd in chicks tissues, while Cd sulfide (20 and 40 mg Cd/kg diet) had no effect. Czarnecki and Baker (1982) reported that Cd levels (30 or 60 mg/kg diet) increased chick's kidney Cd concentration. Gupta and Kar (1999) indicated that administration of Cd chloride (2.5 mg/kg body weight/day) to chickens daily for 15 days decreased serum (T3), hepatic 5'-monodeiodinase (5'D-I) and superoxide dismutase (SOD) activities with a concomitant increase in lipid peroxidation (LPO). So they conclude that the metal-induced inhibition in hepatic 5'D-I activity is mediated through lipid peroxidation (LPO). Erdogan *et al.* (2005) indicated that Cd increased the broiler plasma malondialdehyde (MDA) level as an indicator of lipid peroxidation and lowered the activity of blood superoxide dismutase (SOD) also; liver function

enzymes, aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and gamma glutamyl transferase (GGT) activities were not changed by Cd. They also observed that Cd mainly accumulated in the kidneys, liver, pancreas, and muscle. Berzina *et al.* (2007) reported that the additional Cd chloride ($CdCl_2$) to the diet induced the formation of lipid peroxides in chicken fed the Cd-enriched diet for 30 days; also Cd accumulation in the intestine was markedly higher in the Cd-treated animals compared to the control group.

Interaction between parenterally administrated Cd and Se occurs in several mammalian species. Se counteracts the toxicity of Cd and Cd alters the metabolism of Se (Meyer *et al.*, 1982). Apsite *et al.* (1982) found that high Cd diet increased Cd concentration in blood, liver, kidney, pancreas and intestine, decreased the activity of glutathione peroxidase in liver, also, the excess Cd decreased liver Cu, Mn, Fe, Zn and Cr and increased that of Ni.

To provide further information concerning the toxicity of Se and Cd as well as focused light on the interaction effect of their high levels, this work was carried out to explore the effect of high dietary Se and Cd levels and their mixture on the biochemical and metabolic processes of broiler chickens.

MATERIALS AND METHODS

A total number of 320, one day old of commercial broiler chicks with average BW ranged between 35 and 40 g were randomly housed in 32 galvanized wire-floored pens (10 chicks each) until the end of the experiment (49 days of age). The pens were divided into 16 dietary treatments groups, each groups represented by duplicate pens. The 16 dietary treatments were divided into 4 major groups. First group was used as a control and fed the starter and finisher basal diet (Table 1). The second treatment group was fed the basal diet supplemented with 0.5, 1.0, 1.5, 2.0 and 2.5 mg Se as a sodium selenite (Na_2SeO_3) kg/kg in the starter diet and 2.0, 4.0, 6.0, 8.0 and 10.0 mg Se as a sodium selenite (Na_2SeO_3) kg/kg in the finisher diet, respectively. The third treatment group was fed the basal diet supplemented with 0.25, 0.5, 0.75, 1.0 and 1.25 mg Cd as a Cd chloride ($CdCl_2$) kg/kg in the starter diet and 1.0, 2.0, 3.0, 4.0and 5.0 mg Cd as a Cd chloride ($CdCl_2$) kg/kg in the finisher diet, respectively. The fourth group was fed the starter and the finisher basal diet supplemented with mixture of the different levels of Se and Cd as previously mentioned in the second and the third groups.

Feed and water were provided *ad libitum* throughout the experimental period (one day-49 days of age). Birds were illuminated with constant (24 hr) light. Vaccination and veterinary medical care were done according to common veterinary care under veterinarian supervision.

At the end of the experimental period (49 days of age), four birds from each 16 dietary treatments were randomly selected. The birds were fasted 12-16 hrs prior to slaughter, weighted and sacrificed by cervical dislocation. Blood samples were collected from each bird, serum prepared immediately and kept frozen at - 20°C until used for analyses. After scalding, feather picking and organs (heart, gizzard, spleen, liver and breast meat) were weighed and immediately kept frozen at – 20°C until analysis.

Biochemical analysis: Total soluble protein content was determined by the method of Henry *et al.* (1974), total lipids by the method of (Zoliner and Kirsch, 1962) and metals in liver determined by acid digestion procedure according to Cottenie *et al.* (1982).

Enzyme analysis in liver: Glutamic oxaloacetic transaminase activity (GOT) and glutamic pyruvate transaminase activity (GPT) (POS) catalyses activity was determined colorimetric according to Kahn (1981).

Statistical analysis:

Data was subjected to analysis of variance with completely randomized design. were determined colorimetric by the method of Reitman and Frankel (1987). Peroxidase

Orthogonal polynomials were used for treatment comparisons (Steel and Torrie, 1980). Linear, quadratic and cubic terms of treatments were calculated according to the computer software program.

RESULTS AND DISCUSSION

Biochemical studies

Total lipid concentration

Although, increasing Se, Cd and Se with Cd mixture levels did not significantly affect serum total lipid concentrations although the total lipid concentrations tended to decrease with increasing Se and Cd while increased with increasing Se with Cd mixture supplementations Tables (2 and 4). Quadratic response was observed with increased Se, Se with Cd mixture and with increasing Cd level.

Total lipid concentrations in flesh showed neither linear nor quadratic responses with addition of various levels of Se, Cd and their mixtures Tables (2 and 4). Although the responses were not significant, the total lipid concentrations tended to decrease with Se while increased with Cd and mixture levels.

Increased Se levels increased linearly hepatic total lipid concentrations and did not show quadratic response Table (2). On the other hand, increasing Cd and Se with Cd mixture levels showed neither linear nor quadratic responses on hepatic total lipid concentration Tables (2 and 4).

The results showed non significant decreases with Se and Cd, while showed non significant increase with mixture. In case of flesh the total lipid concentration showed non significant decrease with Cd and Se and Cd mixture. The hepatic total lipid concentration showed linear significant increase with increasing Se, non significant decrease with increasing Cd and non significant increase with increasing mixture Se and Cd supplementation Tables (2 and 4). This could be attributed to the imbalance between the rate of the lipid synthesis and rate of its release to the circulation (Arthur, 1988). Cetin *et al.* (2002) showed that organic and inorganic Se supplementation (0.3 ppm/kg diet) did not affect blood biochemical parameters and health of broiler. Furthermore, Habiba (2006) found that Se addition significantly decreased total lipids and triglycerides. Likewise, Ljubic *et al.* (2006) suggested that organic Se supplementation influences cholesterol metabolism in adipose tissue by decreasing total cholesterol concentration during the fattening period and increasing free cholesterol concentration after 48-hour feed deprivation.

Total soluble protein

Increasing Se and Cd levels decreased linearly serum total protein and showed no quadratic response on serum total protein Table (3). In contrary, increasing Se with Cd mixture showed both linear and quadratic responses on serum total protein Tables (2 and 4).

The total soluble protein concentrations in flesh showed neither linear nor quadratic responses according to Se and Se with Cd mixture levels Tables (3 and 4). Meanwhile, the total soluble protein concentrations in flesh showed

linear decrease and quadratic response with increasing dietary Cd levels Table (3). Hepatic total soluble protein concentrations showed neither linear nor quadratic response, however they tended to decrease with increasing dietary Se levels Table (3). On the other hand, increasing dietary Cd resulted in linear increase and quadratic response in hepatic total protein concentrations Table (3). Meanwhile, increasing Se with Cd mixture levels linearly increased hepatic total soluble protein concentrations Tables (3 and 4).

Total soluble protein concentration in flash showed non significant decrease with Se and Se with Cd mixture while showed a significant decrease with increasing Cd level. Hepatic total soluble protein cementations showed non significant decrease with Se level, while showed significant increase with Cd and Se with Cd mixture. These findings suggested that increasing Se level enhanced protein degradation due to the decrease in peroxidase activity lead to less hydroperoxidase reduction; this resulted in a faster oxidation of amino acids in protein (Otter *et al.*, 1989). Increasing Cd levels lead to increase its amount transported to blood, where it bound to RBC's, albumin and subsequently taken up by various organs mainly liver and kidney (Reddy and Hayes, 1989). Increasing Cd in liver stimulated the synthesis of low molecular weight protein SH-containing protein called metallothionein. The turnover of Cd metallothionein not associated with the loss of tissue Cd. Rather the majority of Cd released from degraded metallothionein appears to be immediately incorporated into newly synthesized metallothionein (Reddy and Hayes. 1989). On the other hand, increasing Se with Cd mixture levels altered the total soluble protein concentrations in the serum and increased their concentrations in the liver. This resulted from; first, the antagonism of Se metabolism by Cd (Meyer *et al.*, 1982); second, chemical reaction between the two elements; third, inhabitation or interference in metabolic pathways; forth, competition for binding sites; fifth, induction of binding proteins (Welch and House, 1980). However, Cetin *et al.* (2002) showed that organic and inorganic Se supplementation (0.3 ppm/kg diet) did not affect blood biochemical constituents and did not negatively affect health of broiler. On the other hand, Habiba (2006) found that Se addition increased plasma globulin and total protein concentration while, the albumen/globulin ratio.

Trace elements concentrations

Hepatic Cu concentration showed neither linear nor quadratic response with increasing Se, Cd and their mixture concentrations Tables (5 and 7). Hepatic Cu concentration showed non significant decrease with either Se or Cd level and non significant increase with increasing Se with Cd mixture concentrations. These results were in agreement with that reported by Meyer *et al.* (1982) who found that increasing Cd intake insignificantly decreased hepatic Cu concentration. Also, they reported that low dietary Se did not affect the concentration of Cd, Zn, Fe or Cu in liver. Casarette and Doull (1991) reported that THE toxicity to either Se or Cu was influenced by the intake of both metals.

Hepatic Zn concentration showed neither linear nor quadratic response with increasing Se, Cd and their mixture concentrations. The metal concentration tended to decrease with Se while increase with Cd and Se and Cd mixture Tables Tables (5 and 7). Our results agree with that obtained by and Apsite *et al.* (1982). In addition, the present findings agreed with that reported by Bafundo *et al.* (1984) who found that supplementary Zn ameliorate some of the adverse effects of Cd toxicity, but Cd additions tended to facilitate Zn deposition in tissues and increased synthesis of metallothionein. The

offered possible explanations of this phenomenon; first, Cd interferes in normal excretion of Zn (Gunn *et al.*, 1962); second, Zn turnover rate is lowered by Cd (Contzias *et al.*, 1962); third, Cd displaces Zn from metallothionein and increased its deposition in tissues (Mohamed *et al.*, 2005 and Casarette and Doull, 1991).

Hepatic Se concentration showed linear increased with increasing dietary Se and Se and Cd mixture concentrations with no quadratic response. On the other hand, hepatic Se concentration showed neither linear nor quadratic response with increasing dietary Cd concentrations, meanwhile Se concentrations tended to increase with increasing Cd levels Tables (5 and 7). The present results agreed with those reported by Flegal *et al.* (1982) showed that feeding Cd level (50 ppm Cd chloride for 6 wks depressed tissue retention of added inorganic dietary Se and this related to interfering with Se absorption.

Hepatic Cd concentration showed neither linear nor quadratic response with increasing Se and Cd concentrations. Hepatic Cd concentration showed non significant decrease with increasing dietary Se concentration but it showed non significant increase with increasing Cd level. On the other hand, increasing Se with Cd mixture concentrations increased linearly hepatic Cd concentrations. However, Leach *et al.* (1979) reported that all levels (3, 12 and 48 ug/g) of Cd resulted in increases the Cd content of kidney while only 12 and 48 ug/g resulted in increases in the Cd content of liver and muscle. As little as 3 ug/g Cd consistently increased the Cd content of liver and muscle but this did not prove to be statistically significant as displayed in Tables (5 and 7). On the other hand, Naoki and Chieko (1987) reported that Se did not interfere with Cd absorption and in addition, the simulative effect of large doses of Se on Cd absorption was not dietary related to metallothionein synthesis in the mucosa. Naoki and Chieko (1988) observed that the levels of Cd in serum, kidney, liver and intestine were higher in the Se with Cd group than that in the Cd group, but there were no differences in the intestinal metallothionein level between Se with Cd and Cd groups. Furthermore, Erdogan *et al.* (2005) observed that Cd mainly accumulated in the kidneys, liver, pancreas, and muscle. Berzina *et al.* (2007) reported that Cd accumulation in the intestine was markedly higher in the Cd-treated animals compared to the control group.

Enzymes activities in liver

The activities of glutamate oxaloacetate transaminase (GOT) and glutamate pyrovate transaminase (GPT) in liver

Determination the activity of hepatic enzymes released into the blood by the damaged liver is one of the most useful tools in the study of hepatotoxicity. Glutamate oxaloacetate transaminase (GOT) and glutamate pyrovate transaminase (GPT) are the most often measured for evaluation of liver damage (Hayes, 1989). GOT and GPT activity showed neither linear nor quadratic responses with increasing dietary Se, Cd and their mixture concentrations. GOT activity tend to decrease with increasing dietary Se or Cd supplementations, while GPT tend to increase with increasing dietary Se or Cd supplementations and decreased with increased dietary Se with Cd mixture supplementations Tables (6 and 7). Erdogan *et al.* (2005) indicated that liver function enzymes, aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and gamma glutamyl transferase (GGT) activities were not changed by Cd.

The activities of peroxidase enzyme (POD) in liver

Peroxidase enzyme which protects cells against the destructive effect of hydrogen peroxide was taken as an indicator to the effect of sub-lethal exposure of broiler chickens to Se, Cd and their mixture concentrations. Liver POD activity showed

non quadratic response, but it appeared to decrease with increasing Se and Cd concentrations. Also, liver POD showed linear decrease with increasing the mixture concentrations. The results indicated that increased dietary Se, Cd and their mixture concentrations decreased POD activity but this decrease was significant only with the mixture levels Tables (6 and 7). These findings agree with that of Black *et al.* (1979) who reported that glutathione peroxidase (GSH-PX) activity was not affected by feeding Cd to rats for either 3 or 6 wks, but at 12 wks the activity of GSH-PX in liver was lower in rats fed Cd and Se than in those fed Se alone. In addition, Meyer *et al.* (1982) found that the main effect of dietary Cd on GSH-PX activity was not significant. Furthermore, Magos and Webb (1978) proposed mechanisms for interactions between Se and Cd include: chemical reactions between the elements, inhabitation or interference in metabolic pathways, competition for binding sites and induction of binding proteins. Ganther and Baumann (1962) suggested that Cd could adversely affect the pathway for changing the oxidation state of Se involved in GSH-PX activity. Kucharzewski *et al.* (2003) found that blood glutathione peroxidase activity was depended on dietary intake of Se. Gupta and Kar (1999) indicated that administration of Cd chloride (2.5 mg/kg body weight/day) to chickens daily for 15 days decreased hepatic 5'-monodeiodinase (5'D-I) and superoxide dismutase (SOD) activities. Erdogan *et al.* (2005) indicated that Cd increased the plasma malondialdehyde (MDA) level as an indicator of lipid peroxidation and lowered the activity of blood superoxide dismutase (SOD). Berzina *et al.* (2007) reported that the additional Cd chloride ($CdCl_2$) to the diet induced the formation of lipid peroxides in chicken fed the Cd-enriched diet for 30 days.

Visualization of liver peroxidase isoenzymes by ultrathin-layer isoelectric focusing on PAG-plates

Liu (1973) reported that peroxidase (donor: H_2O_2 oxidoreductase; EC 1.1.1.7) is a heme protein commonly found in higher organism and in multiple molecular forms (isoenzymes) which are recognized by their distinct electrophoretic mobilities. Bergmeyer (1983) reported that isoelectric focusing is usually the most important electrophoretic variant of isoenzymes studies. Also, the peroxidases reduce peroxides and are readily detected because they can oxidize many substances.

Results in Figure (1) indicated differences in the number of liver peroxidase isoenzyme bands and their intensities among dietary supplemented groups compared to that of control. Furthermore, Figure (2) demonstrated the scanning of liver peroxidase isoenzymes of the control and the Se supplemented chickens. The number of peaks decreased subsequently with increased the Se levels compared to that of the control which represent five (POD_1, POD_2, POD_3, POD_4 and POD_5) isoenzymes. The results revealed that at low Se levels (2 and 4 mg Se/kg diet) the activity of POD isoenzymes increased and new isoenzymes appeared. This finding could be attributed to the replacement of sulfur atom of cysteine by Se atom to form selenocysteine amino acid in which presumably the Se-group of this residue has advantageous properties in the mechanisms of peroxidase isoenzymes activities (Hoekstra, 1975). Conversely, at high Se levels (6, 8 and 10 mg Se/kg diet) the activity of POD isoenzymes decreased with the disappearance of POD_3, POD_4 and POD_5 detected in control, this could be attributed to the increase replacement of the peroxidase isoenzymes toward substrate and also denaturation by the toxic effect of high Se levels. Vadhanavikit and Ganther (1988) reported that depression of peroxidase activity (compared with values

achieved by 0.5 mg Se/kg diet) was observed in rats fed high level of Se. They also reported that although the mechanism is not known, the degree of depression is dose dependent and it is not known whether high levels of Se have a direct effect on the biosynthesis of peroxidase enzyme, or whether Se has a more general toxic effect that indirectly reduces the function of the organ. These findings agree with that reported by many investigators about the essentiality of Se in much smaller amounts in the diet of animals and humans and the margin between the toxic level and the required dietary level is small (Fusheng *et al.*, 1988 and Vadhanavikit and Ganther, 1988). Consequently, our results revealed that at Se level 2 mg/kg diet, increased the peroxidase isoenzymes activities compared to that of the control and the other levels.

Figure (3) represented the scanning of liver peroxidase isoenzymes of the control and the Cd supplemented chickens. There were subsequent decreased in the number of peaks with increasing Cd levels compared to that of the control which represent five (POD_1, POD_2, POD_3, POD_4 and POD_5) isoenzymes. The results indicated that at low level of Cd it showed a little effect on the activities of liver POD isoenzymes, since most of the Cd in the liver bound to metallothionein which is thought to be non toxic, however, when the levels of Cd exceed the critical concentration, it becomes toxic. The factors that determine the level of Cd or Cd-metallothionein complex that is toxic are not clear, but several hypotheses have been proposed. A favored hypothesis attributes the nephrotoxicity of Cd to that fraction of Cd within cells that is not bound to metallothionein, which is when the level of Cd exceeds that of metallothionein available for binding (Goyer *et al.*, 1989). Another hypothesis is that extracellular Cd bound to metallothionein is toxic (Cherian *et al.*, 1976). Cd-metallothionein derived from Cd-induced synthesis in reticulocytes (Tanaka *et al.*, 1985) or released from liver cells is filtered by the renal glomeruli and reabsorbed by proximal tubular lining cells, where it is catabolized, releasing Cd ions, which cause renal damage (Dudley *et al.*, 1988).

Figure (4) represented the scanning of liver peroxidase isoenzymes of control chickens and those supplemented with Se and Cd mixture levels. Increasing mixture levels led to remarkable disappearance of POD isoenzymes compared to those represented in control POD_1, POD_2, POD_3, POD_4 and POD_5. These results could be attributed to (i) Cd possibly impaired Se absorption (Welch and House, 1980), (ii) Cd altered Se utilization (Magos and Webb, 1978), (ii) Cd adversely affect the pathway for changing the oxidation state of Se involved in peroxidase activity (Ganther and Baumann, 1962).Interactions between high levels of dietary Se and relatively high levels of dietary Cd apparently resulted in an antagonism of Se metabolism by Cd in some systems and partial amelioration of Cd toxicity by Se in other systems (Meyer *et al.*, 1982).

Separation of liver protein by ultra-thin layer isoelectric focusing on PAG-plates

Figure (5) represented liver protein pattern separated from 7 wks age broiler chickens supplemented with different Se, Cd and their mixture using ultra-thin layer isoelectric focusing on PAG-plates. It was observed that the number and intensity of the band increased with low dietary Se levels while they decreased with increasing its levels. This indicates that low Se levels induce protein synthesis but high levels induce its degradation (Otter *et al.*, 1989). Increased Cd levels did not remarkably change the number and the intensity of the bands compared to that of control. On the other hand, it was observed that increasing mixture levels remarkably increased the number and intensity of the bands compared to that of control reflecting the induction of protein synthesis. This could

be attributed to the antagonism between Se and Cd in which Cd could ameliorate the destructive effect of Se on liver proteins, noticed in Se supplemented animals (Hayes, 1989).

Table (1). Composition of the experimental diets.

Ingredient	Starter diet %	Finisher diet %
Yellow Corn	65	75
Soybean meal (44%)	25	15
Concentrate(52%CP)*	10	10
Total	100	100
Calculated analysis		
Crude protein (%)	21.79	18.25
ME (kcal/kg)	2966	3088
C/P ratio	136	170
Ether extract %	2.50	3.26
Fiber%	3.05	2.70
Calcuim (%)	0.94	0.91
Av. Phosphorus (%)	0.45	0.47
Lysine%	1.20	1.0
Selenium (mg/kg)	0.1	0.1

*Concentrate analysis: - 52%CP, 2440 ME kcal/kg, 2.0% EE, 3.0%CF, 7.5%, Ca 3.5% P and salt 5%. Vit+Min mixture provides per kilogram of diet: vitamin A, 12000 IU; vitamin E, 20 IU; menadione, 1.3 mg; Vit. D_3, 2500 ICU; riboflavin, 5.5 mg; Ca pantothenate, 12 mg; nicotinic acid, 50 mg; choline chloride, 600 mg;
vitamin B_{12}, 10 g; vitamin B_6, 3 mg; thiamine, 3 mg; folic acid, 1.0 mg; d-biotin, 50 g. Trace mineral (milligrams per kilogram of diet): Mn, 80; Zn, 60; Fe, 35; Cu, 8; Se, 0.10.

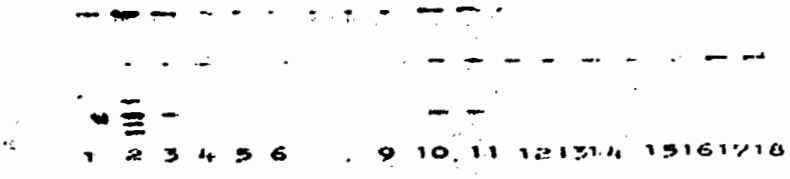

Figure (1): Direct visualization of chicken liver peroxidase isoenzyme by ultrathin-layer isoelectric focusing on PAG-plate: Lane (1) Control.
Lanes :(2), (3), (4), (5) and (6): Se levels.
Lanes:(9), (10), (11), (12) and (13): Cd levels.
Lanes:(14), (15), (16), (17) and (18): Se+ Cd mixture levels.

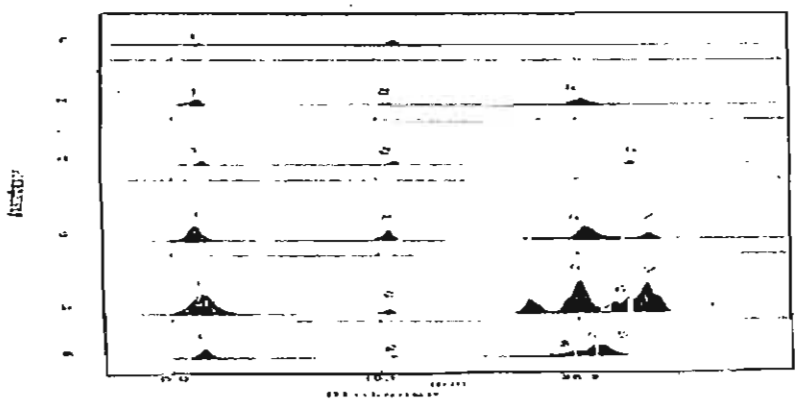

Figure (2):Densitometry scans of visualized chicken liver peroxidase isoenzyme of control and Se supplemented samples. (a) Control. (b) 2 mg/kg. (c) 4mg/kg. (d) 6 mg/kg.
(e) 8 mg/kg. (f) 10 mg/kg.

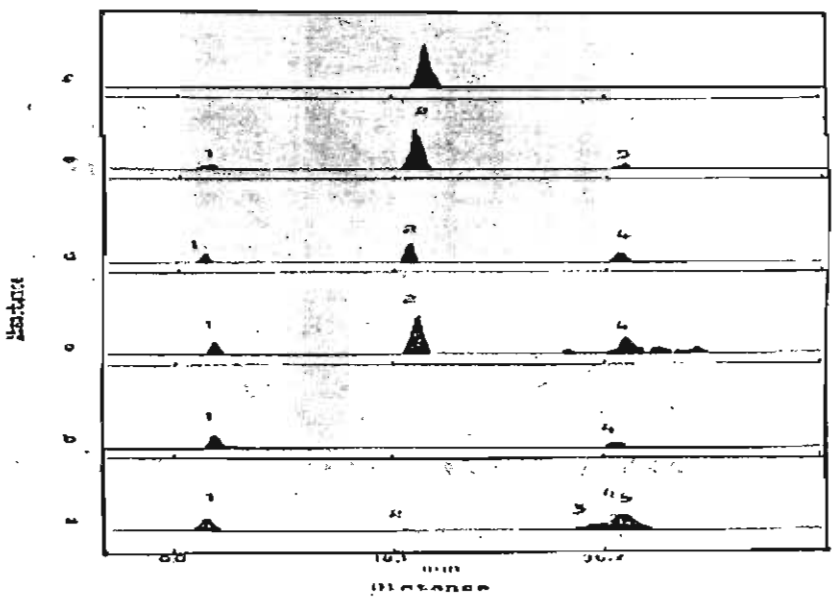

Figure (3):Densitometry scans of visualized chicken liver peroxidase isoenzyme of control and Cd supplemented samples. (a) Control. (b) 1 mg/kg. (c) 2mg/kg. (d) 3 mg/kg.
(e) 4 mg/kg. (f) 5 mg/kg.

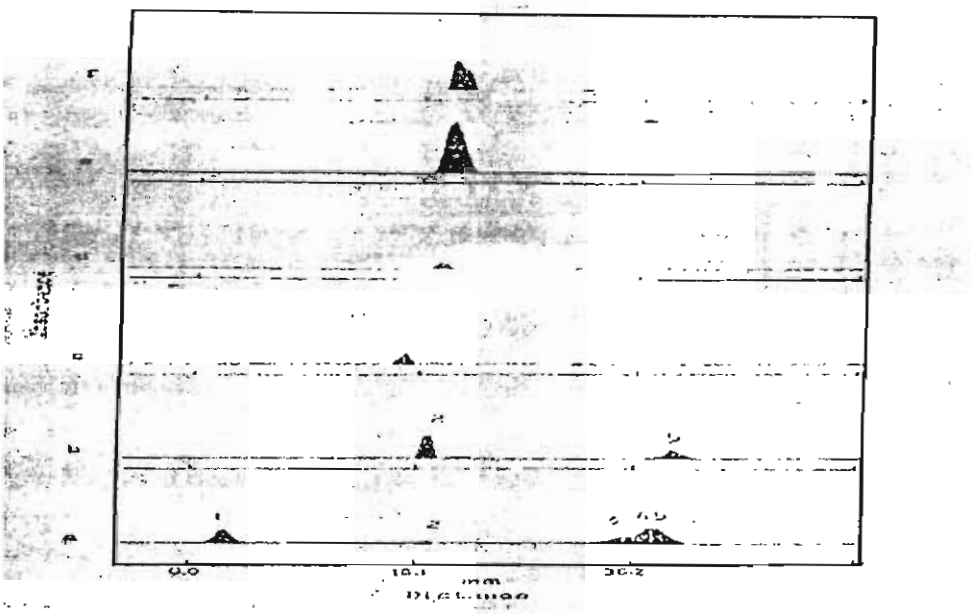

Figure (4):Densitometry scans of visualized chicken liver peroxidase isoenzyme of control and Se and Cd mixture supplemented samples.
(a) Control. (b) 2 mg Se+ 1mg Cd/kg. (c) 4 mg Se+ 2mg Cd/kg.
(d) 6 mg Se+ 3mg Cd/kg. (e) 8 mg Se+ 4mg Cd/kg. f) 10 mg Se+ 5mg Cd/kg.

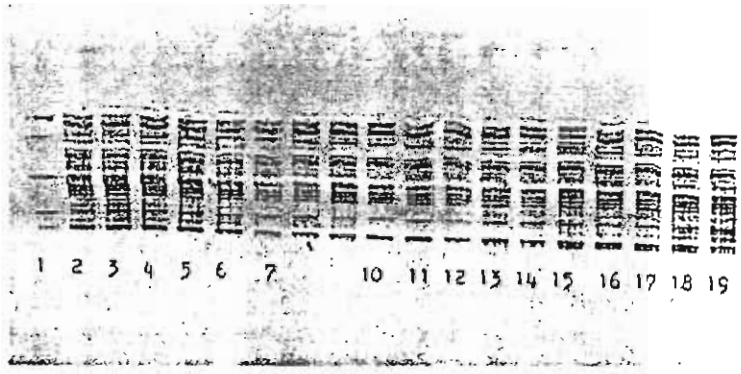

Figure (5): Isoelectric focusing pattern of supernatant protein from control and treated chicken livers:
Lane (1) Control.
Lanes :(2), (3), (4), (5) and (6): Se levels.
Lanes:(9), (10), (11), (12) and (13): Cd levels.
Lanes:(14), (15), (16), (17) and (18): Se+ Cd mixture levels.

Table (2). Total lipids concentrations in serum, flesh and liver of broiler chickens fed various dietary levels of selenium and cadmium in basal diet for 7 weeks .

Se additions mg/kg diet	Total lipids			Cd.ddltions mg/kg diet	Total lipids		
	Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)		Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)
0.0	422.65±3.35	1.613±0.138	6.41±0.83	0.0	422.65±3.35	1.613±0.138	6.41±0.83
2.0	572.75±4.25	1.069±0.010	7.78±0.80	1.0	379.30±9.69	1.440±0.078	8.05±1.60
4.0	594.70±12.79	1.387±0.325	7.99±0.18	2.0	383.40±5.60	1.502±0.349	9.65±0.33
6.0	804.90±71.09	1.277±0.427	11.51±3.62	3.0	439.50±7.49	1.248±0.133	5.99±0.17
8.0	767.45±25.55	0.925±0.122	10.73±0.95	4.0	335.75±4.15	1.499±0.016	5.72±0.32
10.0	492.50±97.50	1.206±0.238	14.48±2.98	5.0	418.25±10.75	1.782±0.314	6.07±0.76
Quadr. Comp.	**	NS	NS	Quadr. Comp.	*	NS	NS
Liner Comp.	NS	NS	*	Liner Comp.	NS	NS	NS
Regr. Coef.	-0.5011	-0.0147	0.3013*	Regr. Coef	-0.5517	0.00441	-0.0501

*Significant component at p< 0.05, **Significant component at p< 0.01, NS= Not significant.

Table (3). Total soluble protein concentrations in serum, flesh and liver of broiler chickens fed various dietary levels of selenium and cadmium in basal diet for 7 weeks .

Se additions mg/kg diet	Total soluble protein			Cd.ddltions mg/kg diet	Total soluble protein		
	Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)		Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)
0.0							
2.0	14008.0±158.00	19.25±3.09	33.28±4.53	0.0	14008.0±158.0	19.25±3.09	33.28±4.53
4.0	13924.5±208.50	15.69±0.47	32.08±2.25	1.0	15099.5±233.50	14.69±0.82	34.92±2.83
6.0	12183.0±567.00	17.85±4.38	32.50±0.00	2.0	13916.5±166.00	12.30±0.41	46.42±2.58
8.0	12033.0±417.00	19.54±1.69	44.37±6.79	3.0	13283.0±467.00	11.08±0.93	47.46±2.58
10.0	11833.0±100.00	15.14±2.07	29.75±5.92	4.0	11991.5±308.00	11.92±1.84	39.50±0.83
Quadr.	7574.5±1691.49	12.89±1.11	31.96±1.38	5.0	12076.5±506.50	12.69±0.44	42.50±2.50
Comp.	*	NS	NS	Quadr. Comp.	NS	*	*
Liner	**	NS	NS	Liner Comp.	**	*	*
Comp.	-220.53**	-0.18148	-0.0098	Regr. Coef	-112.0857**	-0.24190*	0.34785*
Regr. Coef.							

*Significant component at p< 0.05, **Significant component at p< 0.01, NS= Not significant.

Table (4). The effect of dietary mixture levels of selenium and cadmium on total lipids and total soluble protein concentrations in serum, flesh and liver of broiler chickens.

Se. and Cd. mixture mg/kg diet Se Cd	Total lipids			Total soluble protein		
	Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)	Serum (mg/100ml)	Flesh (mg/100ml)	Liver (mg/100ml)
0.0+0.0	422.65±3.35	1.613±0.138	6.41±0.83	14008.00±158.00	19.25±3.09	33.28±4.53
2.0+1.0	544.30±23.30	1.245±0.119	5.69±0.71	9349.50±183.49	12.72±1.98	31.79±4.13
4.0+2.0	648.25±4.75	1.464±0.214	9.98±3.03	11158.00±2658.00	13.65±0.99	35.99±5.08
6.0+3.0	767.25±31.35	1.767±0.689	6.17±0.34	11308.00±25.00	13.29±0.47	37.17±2.75
8.0+4.0	437.20±29.70	1.431±0.031	6.43±0.40	13241.00±625.00	12.42±1.34	46.21±0.21
10.0+5.0	590.70±85.79	1.455±0.245	6.84±0.04	17691.00±124.90	13.21±0.79	45.37±1.21
Quadr. Comp.	**	NS	NS	**	NS	NS
Liner Comp.	NS	NS	NS	*	NS	**
Regr. Coef.	3.6544	0.0004057	0.0032	172.797*	-0.1797	0.59981**

*Significant component at p< 0.05, **Significant component at p< 0.01, NS= Not significant.

Table (5). Mean of copper, zinc, selenium and cadmium concentrations in liver of broiler chicken fed diets supplemented with different dietary levels of selenium or cadmium for 7 weeks of age.

Se.additions mg/kg diet	Copper ppm	Zinc ppm	Selenium ppm	Cadmium ppm	Cd.additions mg/kg diet	Copper Ppm	Zinc ppm	Selenium ppm	Cadmium ppm
0.0	0.360±0.10	1.847±1.11	4.00±1.00	0.088±0.01	0.0	0.360±0.10	1.847±1.11	4.00±1.00	0.088±0.01
2.0	0.285±0.11	1.879±1.11	4.00±1.00	0.063±0.01	1.0	0.241±0.11	1.897±1.11	5.00±1.00	0.077±0.01
4.0	0.351±0.11	1.362±1.11	2.00±1.00	0.107±0.01	2.0	0.249±0.11	3.093±1.11	6.00±1.00	0.095±0.01
6.0	0.183±0.11	3.330±1.11	7.00±1.00	0.067±0.11	3.0	0.389±0.11	2.511±1.11	6.00±1.00	0.174±0.01
8.0	0.163±0.11	1.811±1.11	8.00±1.00	0.065±0.11	4.0	0.204±0.11	2.130±1.11	5.00±1.00	0.165±0.01
10.0	0.113±0.11	0.138±1.11	6.00±1.00	0.057±0.10	5.0	0.265±0.11	2.602±1.11	7.00±1.00	0.180±0.01
QuadrComp	NS	NS	NS	NS	Quadr.Com	NS	NS	NS	NS
LinerComp.	NS	NS	*	NS	LinerComp.	NS	NS	NS	NS
Regr.Coef	-0.01010	-0.0387	0.15428*	-0.00108	Regr.Coef.	-0.0025485	0.02224	0.0857	0.004588

*Significant component at p< 0.05, **Significant component at p< 0.01, NS= Not significant.

Table (6): Effect of different dietary selenium and cadmium levels on the activities of glutamates oxaloacetate transaminase (GOT), glutamate pyrovate transferase (GPT) and peroxidase (POD) in the liver of broiler chicken at 7 weeks of age

Se additions mg/kg diet	GOT Unit/mg protein	GPT Unit/mg protein	POD Unit/mg protein	Cd.ddditions mg/kg diet	GOT Unit/mg protein	GPT Unit/mg protein	POD Unit/mg protein
0.0	0.00559±0.0015	0.00097±0.0002	111.05±22.95	0.0	0.00559±0.0014	0.00097±0.0002	111.05±22.95
2.0	0.00574±0.0005	0.00102±0.0005	128.35±04.425	1.0	0.00553±0.0070	0.00104±0.0001	141.20±22.80
4.0	0.00601±0.0000	0.00106±0.0007	113.85±15.85	2.0	0.00432±0.0000	0.00082±0.0001	92.25±06.55
6.0	0.00527±0.0013	0.00087±0.0009	84.70±03.90	3.0	0.00475±0.0007	0.00086±0.0002	90.05±06.65
8.0	0.00652±0.0008	0.00104±0.0009	132.75±13.25	4.0	0.00503±0.0004	0.00102±0.0000	135.75±14.25
10.0	0.00339±0.0033	0.00149±0.0005	65.18±05.12	5.0	0.00504±0.0002	0.00102±0.0001	105.85±12.45
Quadr. Comp.	NS	NS	NS	Quadr. Comp.	NS	NS	NS
Liner Comp.	NS	NS	NS	Liner Comp.	NS	NS	NS
Regr. Coef.	-0.000054	0.00001382	-1.4000	Regr. Coef	-0.00002211	0.00000134	-0.250000

*Significant component at p< 0.05, **Significant component at p< 0.01, NS= Not significant.

Table (7). The effect of dietary mixture levels of selenium and cadmium on minerals and liver enzymes concentrations in serum, flesh and liver of broiler chickens.

Se. and Cd. mixture mg/kg diet Se Cd	Minerals				Liver Enzymes		
	Copper ppm	Zinc ppm	Selenium ppm	Cadmium ppm	GOT Unit/mg protein	GPT Unit/mg protein	POD Unit/mg protein
0.0+0.0	0.360±0.10	1.847±1.11	4.00±1.00	0.088±0.01	0.00559±0.0014	0.00097±0.002	111.05±22.95
2.0+1.0	0.273±0.11	2.355±1.11	6.00±1.00	0.080±0.01	0.00604±0.0003	0.00113±0.001	118.35±08.85
4.0+2.0	0.322±0.11	2.535±1.11	6.00±1.00	0.096±0.01	0.00681±0.0006	0.00131±0.000	110.30±07.29
6.0+3.0	0.189±0.11	2.140±1.11	6.00±1.00	0.116±0.11	0.00580±0.0000	0.00106±0.000	84.62±08.18
8.0+4.0	0.787±0.11	4.700±1.11	7.00±1.00	0.449±0.11	0.00440±0.0001	0.00082±0.000	65.00±14.39
10.0+5.0	0.184±0.11	1.748±1.11	8.00±1.00	0.310±0.10	0.00490±0.0006	0.00078±0.000	87.39±01.31
Quadr. Comp.	NS	NS	NS	NS	NS	NS	NS
Liner Comp.	NS	NS	*	*	NS	NS	*
Regr. Coef.	0.003022	0.0351	0.1314*	0.01278*	-0.0000538	-0.0000012077	-1.07400*

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selenium, cadmium, biochemical constituents, broiler.

تأثير مستويات مختلفة من السيلنيوم والكادميوم والخليط منهما على

٢- العمليات البيوكيميائية و التمثلية لكتاكيت اللحم

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تم أجراء هذا البحث بهدف دراسة تأثير كل من السيلينيوم و الكادميوم بصورة منفردة او المخلوط بينهما على المعايير البيوكيميائية لكتاكيت اللحم. تم تقسيم عدد ٣٢٠ كتكوت لحم عمر يوم الى ١٦ معاملة غذائية، وكل معاملة ممثلة بمكررتان وكل مكررة تحتوى على عدد١٠ كتاكيت وذلك حتى نهاية فترة التجربة (٤٩ يوم). قسمت الـ ١٦ معاملة الى ٤ مجموعات رئيسية. المجموعة الرئيسية الأولى تعتبر المجموعة المقارنة وغذيت على العليقة الأساسية خلال مرحلتى البادئ والناهى بدون اى اضافات. المجموعة الرئيسية الثانية غذيت على العليقة الأساسية والتى تم امدادها بمستويات ٠.٥ و ١.٠ و ١.٥ و ٢.٠ و ٢.٥ مجم Se فى صورة سلينات الصوديوم/كجم علف بادئ و بمستويات ٢.٠ و ٤.٠ و ٦.٠ و ٨.٠ و ١٠.٠ مجم /كجم علف ناهى ، على التوالى. المجموعة الرئيسية الثالثة غذيت على العليقة الأساسية وتم امدادها بمستويات ٠.٢٥ و ٠.٥٠ و ٠.٧٥ و ١.٠٠ و ١.٢٥ مجم Cd فى صورة كلوريد الكادميوم /كجم علف بادئ و بمستويات ١.٠ و ٢.٠ و ٣.٠ و ٤.٠ و ٥.٠ مجم /كجم علف ناهى ، على التوالى. المجموعة الرئيسية الرابعة غذيت على العليقة الأساسية خلال مرحلة البادئ والناهى مضاف اليها مستوات مختلفة من مخلوط كلا من السلينيوم و الكادميوم بنفس المستويات الموضحة سابقابكلا من المجموعة الثانية والثالثة.

زيادة كلا من السلينيوم او الكادميوم او المخلوط بينهما لم تؤثر معنويا عل تركيزات الدهون الكلية بالسيرم. تركيز الدهون الكلية بالأنسجة لم تظهر اى تأثير خطى او كودراتيك نتيجة لأستخدام المستويات المختلفة من السلينيوم او الكادميوم او المخلوط منهما. زيادة مستويات السلينيوم يذيد خطيا من تركيز الدهون الكلية بالكبد. زيادة كلا من السلينيوم او الكادميوم يخفض بصورة خطية تركيزات البروتينات الكلية بالسيرم. بينما يذداد تركيز البروتينات الكلية بالكبد. زيادة مستويات المخلوط من السلينيوم والكادميوم توضح زيادة خطية لتركيزات البروتينات الكلية الذائبة بالكبد. تركيز عنصر السلينيوم بالكبد يزيد زيادة خطية بزيادة مستويات السلينيوم و الخليط بين السلينيوم و الكادميوم .

أوضحت النتائج ان زيادة السلينيوم و الكادميوم والخليط منهما يؤدى الى انخفاض فى نشاط انزيم البيروكسيدزPOD لكن هذا الأنخفاض كان معنويا مع استخدام الخليط بين السلينيوم والكادميوم فقط . وبصفة عامة فأن نشاط انزيمات GOT وGPT لم يتأثرا بمستويات السلينيوم او الكالدميوم او الخليط منهما.عدد و كثافة حزم البروتين تزداد مع استخدام المستويات المنخفضة من السلينيوم. زيادة مستويات الكادميوم لم يكن لها تأثير عل عدد وكثافة حزم البروتين مقارنة بالمجموعة المقارنة. وبصفة علمة زيادة المستويات من الخيط بين السلينيوم والكادميوم يذيد عدد وتركيز حزم البروتين مقارنة بالمجموعة المقارنة.