LAYING HENS PERFORMANCE AND SOME HISTOLOGICAL CHANGES AS AFFECTED BY THE DIET POLLUTED WITH THE LEAD ADDED WITH CLAY OR VITAMIN E

By

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Abstract: The experimental work was carried out to define the effect of feed contaminated with three levels of lead acetate (Pb) (0.0, 500 and 1000 mg/kg diet) and three levels of feed additives (without clay or vitamin E, 3% natural clay and 200 mg vitamin E /kg diet) on laying hens performance and histological changes in the liver, kidney and intestine. 216 birds (189 hens and 27 cocks) Mandara laying hens local strain, 40 weeks of age were used in this study. Hens and cocks were randomly distributed into 9 treatments, (21 hens and 3 cocks) in 3 replicates, each of 7 hens and one cock.

The results indicated that feed contaminated with the lead at levels of 500 or 1000 mg/kg diet decreased gradually and significantly ($P \le 0.05$) egg production (%), egg number, egg weight (g), egg mass(g), feed intake(g), feed conversion (kg feed/kg egg), percentages of carcass, heart, fertility and hatchability during the periods from 41-44, 45-48, 49-52 and 41-52 wks of age, while, egg weight at 49-52 wks of age was insignificant. The birds fed the diet polluted with lead had insignificantly higher percentages of spleen, kidney and liver. The addition of clay or Vit. E to the diets caused significantly ($P \le 0.05$) improvement of egg production (%) and egg number at 49-52 and 41-52 wks of age, egg mass and feed conversion at all periods and fertility at 40-52 wks of age, while insignificantly improved egg number and egg production (%) at 41-44 and 45-48 wks of age, egg weight, feed intake(g), hatchability (%) at all periods, fertility (%) at (41-44, 45-48 and 49-52 wks of age), percentages of carcass, heart, spleen, kidney and liver as compared to without clay or vitamin E supplementation. Addition of clay or Vit. E to the diets without or polluted with lead caused significantly ($P \leq 0.05$) improvement egg number, egg production (%), egg mass (g), feed intake (g) feed conversion (kg feed/kg egg), fertility rate % at all periods. While, egg weight (g) at 41-44 and 41-52 wks of age, hatchability % from (45-48, 49-52 and 41-52 wks of age) and heart (%) showed significantly ($P \le 0.05$) increase as compared to without addition. On the other hand, egg weight (g) at 45-48 and 49-52 wks of age, hatchability (%) at 41-44 wks of age, percentages of carcass, spleen, kidney and liver insignificantly improved ($P \le 0.05$) as compared without addition.

The histological changes in the liver, kidney and intestine in the birds fed the diet polluted with lead at 500 or 1000 mg/kg diet, revealed focal replacement of the hepatic tissues and various degenerative changes or necrosis in the liver, nephritic changes in the tubular epithelium with leukocytic infiltration and focal interstitial aggregations of lymphocytes (kidney). Intestinal mucosa showed partial desquamation of their lining epithelium and great distortion of their upper third (intestine). Addition of clay or Vit. E with the diet polluted with lead either 500 or 1000 mg/kg diet showed slightly improvement in the hepatic cells and moderate improvement in the renal tubules of the kidney and in the intestinal mucosa of the intestine.

INTRODUCTION

The contamination of laying hen's diets or environment with heavy metals remain a problem for poultry meat industry, food safety, regulatory agencies and consumers. According to the extensive use of lead in human activities e.g. industrial processes, plant protection, paint industry and motorengine emission, the possibilities of bird's diet pollution become more evident.

Lead is considered one of the major environmental pollutants (Jeng et al., 1997). Lead is a neurotoxin and commonly generates abnormal brain and nerve functions (Hais, 1992). Lead is one of the most toxicant known to cause red blood cell hemolytic through damage to the cell membrane and neurological and reproductive disorders (Khan et al., 1999). In addition, lead and cadmium also cause many serious diseases and dysfunction of organs. The adverse effect of lead on reproductive system and efficiency has been recognized by Assennato et al., (1986) and Vodela et al., (1997). The lead pollution may also cause deleterious effects on biochemical indices (Abou-Zeid et al., 2000), inhibits enzyme active (Klauder and Petering, 1975), semen characteristics, (El-Zaiat, 2003), fertility and hatchability (Ayyat et al., 2005). Khan et al. (1993) showed alterations in serum total protein, aspartate aminotransferase, cholesterol and alkaline phosphatase levels. On the other hand, addition of Zeolite (5%) reduce aflatoxin toxicity to growing chicks by 41% as indicated by weight gain, liver weight and serum biochemical measurements (Harvey et al., 1993).

In addition, vitamin E is a crucial lipid soluble antioxidant against oxidation, Vitamin E also increase the immune response in the chicken (Franchine *et al.*, 1995). Vitamin E sometimes called antisterility. As reported by Kling and Scares (1980) in Japanese quail maintained on a low vitamin E in the diet for 35 weeks, a lowered incidence fertile eggs was shown after 20 weeks and hatchability severely depressed probably due to the inadequate deposition of vitamin E in the egg to support embryo growth. Moreover, vitamin E has a number of different biological functions. One of the most important functions its role as an intracellular antioxidant. Gore and Qureshi (1997) speculated that vitamin E prevents oxidation of unsaturated lipid materials within cells, thus protecting the cell membrane from oxidative damage.

The present study aimed to investigate the efficacy of clay or vitamin E to alleviate severity of the diet contaminated with the lead particularly their effects on the laying hens performance and economic efficiency of Mandara strain, beside histopatholigical changes in same organs of hens.

MATERIALS AND METHODS

The present work was carried out at Inshas Poultry Research Station, Sharkia Province, belonging to Animal Production Research Institue, Agricultural Research Center, Dokki, Giza, Egypt.

A total number of 216 Mandara strain at 40 weeks of age, was randomly divided into 9 treatments (21 hens and 3 cocks each). A 3 x 3 factorial design experiment was performed including three levels of lead (as lead acetate trihydrate) contamination (0, 500 and 1000 mg/kg diet) and three levels of feed additives (without clay and vitamin E, 3% natural clay or 200 mg vitamin E/kg diet). Each treatment was allocated into three replicates each of 7 hens and one cock. Tafla is desert clay analyzed as soluble cations and anions (mg/100g DM soil) were Ca⁺⁺ 0.75, Mg⁺⁺ 0.25, N^+ 0.05, K 0.10, Cl 0.55, So₄ 0.30 and HCo₃ 0.75. Exchange cable cations (mg/100g DM soil) were 2.65 and available nutrients (mg/100 g DM soil) were P, 5.0, K, 1.2, Mn, 2.4, Zn 0.74, Cu 0.30 and Fe 0.55 mg (Marai et al., 1996). Basal diet formulation and their calculated analysis are presented in Table (1). The average initial live body weight and egg production percentage were nearly similar at 40 weeks of age. Also, all laying hens were reared under the same managerial, hygienic and environmental conditions. The feed and water were supplied ad-libitum during the experimental period. Artificial light source was used, giving a total 16 hours of light per day during the whole experimental period which extended for 12 weeks from 41 to 52 weeks of age.

Eggs were collected and weighed daily till the termination of this experiment. The average egg production was calculated per replicate during the different periods (41-44 wks, 45-48 wks, 49-52wks old) and the whole period (from 41 to 52 wks old). Daily feed consumption per replicate was estimated. Egg number, egg weight (g), percentage of egg production (egg weight x egg number), feed consumption (g) feed conversion ratio (g feed/g egg/replicate) were also recorded.

At the end of the experiment (52 weeks of age), three birds from each treatment were taken around the average body weight of the treatment. Then after, the birds were slaughtered and carcass measurements were calculated as a percentage of live body weight.

For histological studies, liver, kidney and intestines were weighed and then after fixed in 10% neutral buffered formalin. The section was stained with Haematoxyline and Eosin stains for histopathological examinations and the macroscopic lesions according to Durry and Wallington (1980).

Fertility rate was estimated at the end of hatching period as a number of fertile eggs/number of eggs set. Hatchability of eggs from each treatments was also estimated as the ratio of number of chicks hatched to number of fertile eggs.

Economic efficiency for egg production was calculated from the input – output analysis according to the price of the experimental diets and eggs produced. Values of economic efficiency were calculated as the net revenue per unit of total costs. Data were subject to factorial design using General Linear Model of SAS® software statistical analysis (SAS, 2004). Significant means were separated by Duncan's New Multiple Range Test (Duncan, 1955).

RESULTS AND DISCUSSION

Lying performance:

Egg production traits:

Data presented in Table (2) showed the effect of diet contaminated with lead added with clay or Vit. E on egg number and egg production (%) determined as hen day (HD %).

Egg number or egg production percentage showed significantly gradually ($P \le 0.05$) decrease with increasing lead contamination in diets hen at all experimental periods. The differences in egg numbers and egg production (%) in laying hens fed the diet supplemented with clay or Vit. E were insignificantly higher at the periods of 41 to 44 wks and 45 to 48 wks, while significantly (P≤0.05) higher at 49 to 52 weeks and 41 to 52 weeks of age when compared to the control diet. These findings may be due to zeolites can adsorb toxic products of digestion and decrease accumulation of toxic substances in tissues, thus decreasing the incidence of internal disorders (Mumpton and Fishman, 1977). Moreover, there was a significant effect (P ≤ 0.05) on the egg numbers and egg production (%) due to lead contamination and feed additives interaction, where T3 recorded the highest values (18.44, 18.19, 19.20 and 55.83 or 65.86, 64.95, 68.58 and 66.46%), whereas T7 gave the lowest egg numbers or percentage (12.67, 12.44, 12.29 or 37.39 and 45.24, 44.42, 43.89 and 44.52 %) at the experimental periods from 41-44, 45-48, 49-52 and 41-52 weeks, respectively. This reduction in egg production was observed in chickens consumed lead at 200 mg/kg diet or greater (Vodela et al., 1997). These results supported by Stone and Soares (1974) who found a sharp decline in the rate of egg production in White Leghorn hens fed the diets contained 630 mg/kg diet lead oxide or more. However, Whisenhunt and Maurice (1981) reported that feeding hens on diet containing 500 mg lead/kg diet showed no significant effects on egg production. Meluzzi et al. (1996) reported that egg production and egg quality for laying hens were negatively and significantly affected by heavy metals (500, 100 and 2000 mg/kg diet for Cr as Crcl3, 100, 300 and 500 mg/kg diet for Ni as Ni So4 and 20, 30 and 100 mg/kg diet for Pb as Pbo). Voget (1992) reported that the addition of bentonite or diatomite at levels of 10, 20 and 40 g/kg decreased laying performance. Barreto et al. (1999) reported that daily egg production/hen, egg weight, egg number/ hen housed, viability, final female body weight and egg yolk percentage of broiler breeder hens were not affected by 4 and 16% P and vitamin E 25 and 250 mg/kg diet. However, greater hatching chick weight (P < 0.01) was observed in those from 32 week old broiler breeder hens fed the diet containing 16% P and vitamin E 250 mg/kg.

Egg weight and mass :

The effect of treatments on egg weight are shown in Table (3). Data showed that the differences in egg weights were significant ($P \le 0.05$) due to diet contaminated with lead effects at all the experimental periods except at 49-52 weeks of age. However, there was no significant differences between the average values of egg weight due to feed additives (clay or Vit.

E). Moreover, the effect of interaction between lead contamination and feed additives on egg weight were significant ($P \le 0.05$) at 41-44 and ($P \le 0.01$) at 41-52 weeks of age, while there were no significant difference ($P \le 0.05$) between the average values of egg weight due to lead contamination and feed additives interaction during the periods of 45-48 and 49-52 weeks of age. The average egg weight increased gradually with decreasing the lead contamination from 1000 to 0.0 mg/kg diet with supplemental vitamin E.

The effect of treatments on egg mass is presented in Table (3) There was a significant difference in egg mass among hens fed the diet with added feed additives or lead contamination. Hens receiving low lead level with added vitamin E gained the highest egg mass, however, the high level of lead without additive gained the lowest egg mass. A significant difference was observed in the average egg mass due to interaction between lead contamination and feed additives. Generally, the average egg mass increased gradually with decreasing lead levels from 1000 to 0.0 mg/kg diet supplemented with vitamin E.

The highest ($P \le 0.05$) egg weight or egg mass (53.30, 50.90, 50.27% and 51.49 (g) or 983.57, 925.76, 965.67 and 2875.74 (g) was recorded by the hens in T3, while the least (49.00, 47.30, 46.50 and 47.60 (g) or 620.76, 586.78, 572.42 and 1779.08 (g)) was obtained by the hens in T7 at 41-44, 45-48, 49-52 and 41-52 weeks of age, respectively, for the interaction effect. The increase in egg production and egg weight resulted in a larger increase in egg mass.

El-Zaiat *et al.* (2003) reported that egg weight significantly ($P \le 0.01$) decreased with increasing lead level from 0.0 to 1000 mg/kg diet at 40-44 and 48-52 weeks of age. While, Whisenhunt and Maurice (1981) reported that hens fed on the diet containing 500 mg lead/kg diet showed no significant effects on egg weight.

Feed consumption and conversion :

Data presented in Table (4) showed the effect of treatments on the amount of feed intake for different experimental birds. There was a significant ($P \le 0.05$) effect on the amount of feed consumed (g/hen/day) due to lead contamination effect. Hens receiving diet without lead consumed significantly ($P \le 0.05$) more feed than those receiving the lead contamination diets (500 and 1000 mg/kg diet). No significant differences in feed intake due to feed additives. A significant difference was observed in the average feed intake were observed due to lead contamination and feed additives interaction. Hens in T1 recorded the highest value of feed intake (92.50, 97.0, 103.0 and 97.30 g/hen/day), while those in T8 gave the least

value (82.36, 85.50, 90.32 and 86.06 g/hen/day) for different experimental periods 41-44, 45-48, 49-52 and 41-52 weeks of age, respectively. Generally, the amount of feed consumed decreased with increasing lead levels and feed additives (clay or Vit. E).

Average values of feed conversion ratio (FCR) calculated as amount of feed (kg) required to produce/kg of eggs are shown in Table (4). There were significant differences ($P \le 0.05$) among lead contamination where diets containing 1000 mg/kg diet recorded the poorest FCR. The diets without lead contamination improved FCR as compared to the diets contaminated with 500 or 1000 mg lead/kg. These results may be attributed to the different amounts of feed consumed and egg production or the supplemental lead to the diet which increased FCR. A significant difference $(P \le 0.05)$ was observed in the FCR due to feed additives. The diets containing vitamin E or clay improved FCR, as compared to the diet without additives. The interactions effects between lead contamination and feed additives on FCR was significant (P<0.05). The best FCR values (2.63, 2.91, 2.94 and 2.83) were recorded by the hens in T3, while the worst ones were obtained by hens of T7 (3.77, 4.17, 4.52 and 4.13) at the experimental periods 41-44, 45-48, 49-52 and 41-52, respectively. Generally, the best feed conversion ratio was noticed with all diets containing vitamin E or clay. Similarly, Edens and Melvin (1989) found that feed consumption decreased in laying quail fed the diet containing 500 ppm of lead. Stanchev (1989) reported also that the feed intake was increased by increasing the levels of lead acetate in broiler diets from 100 to 500 ppm of lead.

Fertility and hatchability percentages :

The effects of treatments on average values of fertility (%) and hatchability (%) are presented in Table (5). Fertility and hatchability (%) were significantly (P \leq 0.05) affected by lead contamination at all experimental periods, while no significant differences were detected between the average values of fertility and hatchability due to feed additives (clay or Vit. E) at all experimental periods, except during the period from 41-52 weeks of age. The interaction effects between lead contamination and feed additives on fertility and hatchability were significant (P \leq 0.05) at all experimental periods, except during the period from 41-52 weeks of age. The interaction effects between lead contamination and feed additives on fertility and hatchability were significant (P \leq 0.05) at all experimental periods, except during the period from 41-44 week of age. Similarly, Engelman *et al.* (1999) observed that feeding dosages of 20 000 mg alpha-tocopheryl acetate/kg diet to laying hens caused decreased (P \leq 0.05) hatching rates (30.7%) compared with the control group (74.7%) fed a diet containing 19 mg alpha tocopherol/kg.

Edens et al. (1976) reported reproductive dysfunctions increasing significantly (P<0.01) the age of sexual maturity in Japanese quail fed lead diet contained 10, 100 or 1000 mg lead. The reduction in hatchability rates may be due to that eggs laid by hens treated with lead contained high proportion of source element. The egg components are the main source nutrients for the developing embryo and the newly hatched chicks absorb in their body approximately 6% of the total egg yolk. This amount of yolk is usually consumed during the first days of life after hatching. This might mean that lead will be exit in the metabolism of the developing embryo as well as due to the early days of the hatched chick. Abaza et al., (1996) demonstrated that the lead cause dysfunctions in the reproductive and physiological systems of cockerels. It is manifested by decrease in semen volume and relative sperm motility. The obtained results are in agreement with the findings of El-Zaiat (2003) and Ayyat *et al.* (2005) who showed that, fertility and hatchability significantly (P <0.01) decreased with increasing lead levels 250 to 500, 1000 mg/kg diet in laying hens. De Gennaro (1978) reported that the lead treated embryos failed to be hatched and the embryos died before 21 days of incubation. Vodela et al. (1997) indicated also, that the broilers drinking water containing 6-7 mg lead significantly increased the embryonic mortality (68.84%) compared with the control birds (16.16%). El-Zaiat (2003) and Ayyat et al. (2005) found an increase in fertility and hatchability by the clay or vitamin E additives feed in laying hen diets toxicity by lead at levels of 250, 500 and 1000 mg/kg diet.

Generally, supplementation of birds diets with natural clay improved fertility and hatchability this may be due to activation of the immune system and production of lymphocytes which produce antibodies, in addition to retard the absorption of toxic products of digestion that reduced toxicity of lead and toxic diminish their harmful respect (Ayyat *et al.*, 1997). Its ability to reduce the harmful effects of pesticide organ phosphorus, (Youssef and Mari, 1995).

Slaughter examination :

Data presented in Table (6) showed that a significant decrease (P \leq 0.05) in carcass (%) and heart (%) in hens fed diet contaminated with lead at 500 or/1000 mg lead/kg diet compared to without lead contamination diet. However, no significant differences were observed in spleen (%), kidney (%) and liver (%)due to lead contamination. Similarly, no significant differences in the percentages of each of carcass, heart, spleen, kidney and liver were observed due to feed additives effects (clay or Vit. E). The interaction effect between lead contamination and feed additives on the percentages of each of carcass, spleen, kidney and liver were not significant, while a significant (P \leq 0.05) differences in heart (%). These results are in agreement with those of El-Zaiat (2003) who found that carcass and liver weight (%) decreased with increasing lead level in cocks diet at 52 weeks of age. Attia *et al.* (2005) reported that carcass and heart weights were significantly decreased with increasing lead levels in the diets of laying hens at 52 weeks of age. Fathi *et al.* (1999) found that the decrease in carcass yield was due to increase in level dietary lead.

Histological changes:

Liver: (0.0 mg lead/kg diets)

The hepatic parenchyma was apparently normal Fig. (1).

Liver: (500 mg lead/kg diets)

Focal replacement of the hepatic tissues by numerous extra rested erythrocytes (focal haemorrhages) and the surrounding hepatic cells suffered from various degenerative changes or necrosis Fig (2). The portal areas showed numerous leukocyte infiltration mainly lymphocytes beside bile duce proliferation.

Liver: (1000 mg lead/kg diets)

Focal areas of coagulative necrosis accompanied with multiple leukocyte aggregation and congestions were common (Fig. 3). Portal areas showed hyalinized portal vein wall, numerous leukocytes aggregations beside scattered apoptotic bodies.

Liver: (0.0 mg lead + 3% clay/kg diet)

The hepatic cells appeared vacuolated with apparently normal portal area and hyperplasic kuffer cells (Fig. 4).

Liver: (500 mg lead + 3% clay/kg diet)

The hepatic cells suffered from various degenerative changes varied from vacuolar and hydropic degeneration with dilated hepatic sinusoids (Fig. 5).

Liver: (1000 mg lead + 3% clay/kg diet)

Focal necrosis or degeneration of the hepatic cells with hyalinized wall of hepatic arteriole could be seen (Fig. 6). Individual scattered apoptotic bodies could be seen.

Liver: (0.0 mg lead + 200 mg vit. E/kg diet)

Portal and interstitial leukocyte aggregations mainly lymphocytes with normal hepatic cells and portal areas were the common microscopic picture (Fig. 7).

Liver: (500 mg lead + 200 mg vit. E/kg diet)

The hepatic cells were slightly swollen with congestion of central vein and hepatic sinusoids side (Fig. 8).

Liver: (1000 mg lead + 200 mg vit. E/kg diet)

The hepatic parenchyma was focally replaced with round cells with degeneration in the surrounding hepatic cells beside hyperplastic kuffers cells (Fig. 9).

Kidney: (0.0 mg lead/kg diet)

The renal parenchyma was apparently normal (Fig. 10).

Kidneys: (500 mg lead/kg diet)

Nephrotic changes in the tubular epithelium with leukocyte infiltration mainly lymphocytes in the interstitial tissue and hemorrhagic areas were encountered (Fig. 11).

Kidneys: (1000 mg lead/kg diet).

Server destruction of kidney tissues with interstitial hemorrhages and fibroblastic proliferation could be seen Fig (12). The remaining renal tubules exhibited hydropic degeneration or pressure atrophy of their epitheliums.

Kidneys: (0.0 mg lead + 3% clay/kg diet).

Inter-renal capillaries were congested with interstitial hemorrhages and leukocyte aggregations mainly lymphocytes and macrophages were seen (Fig. 13).

Kidneys: (500 mg lead + 3% clay/kg diet).

The renal tubules showed nephrosis in their lining epithelium (Fig. 14).

Kidneys: (1000 mg lead + 3% clay/kg diet).

Severe congestion of renal blood vessels and interstitial leukocyte aggregations mainly lymphocytes severe degeneration is some renal tubules were common (Fig. 15).

Kidneys: (0.0 mg lead + 200 mg vit. E/kg diet).

The renal blood vessels were congested and a few cellular casts could be seen inside the Lumina of some renal tubules (Fig. 16).

Kidneys: (500 mg lead + 200 mg vit. E/kg diet).

The lining epithelial cells of some renal tubules had degenerative or necrotic changes beside contraction of some glomenlar tufts (Fig. 17).

Kidneys: (1000 mg lead + 200 mg vit. E/kg diet).

Necrosis of renal tubules with replacement with mononuclear cells mainly lymphocytes and histiocytes could be seen (Fig. 18).

Intestine: (0.0 mg lead /kg diet).

All the intestinal coats apparently normal (Fig. 19).

Intestine: (500 mg lead /kg diet).

The intestinal mucosa showed partial desquamation of their lining epithelium and great distortion of the upper third of the mucosa. Moreover, leukocyte infiltration could be seen (Fig. 20).

Intestine: (1000 mg lead /kg diet).

Partial or complete desquamations of villous epithelium beside intense leukocyte infiltration in both mucosa and submucosa and edematous muscular coat were common (Fig. 21).

Intestine: (0.0 mg lead + 3% clay /kg diet).

The intestinal villi appeared taller with partial sloughing of their villous epithelium and thickened lamina propria with lymphocytes were seen (Fig. 22). Intestinal glands showed metaplasia to goblete cells.

Intestine: (500 mg lead + 3% clay /kg diet).

Some intestinal glands in submucosa revealed partial necrosis of their lining epithelum with per glandular fibroblastic proliferation beside goblet cell metaplasia to some glands (Fig. 23).

Intestine: (1000 mg lead + 3% clay /kg diet).

Intestine replacement of some glands in submucosa with leukocytes mainly lymphocytes (Fig. 24). The epithelial lining of intestinal villi was destructed. Edema could be seen between muscle fibers in tunica muscularis.

Intestine : (0.0 mg lead + 200 mg vit. /kg diet).

The intestinal villi revealed mild thickening of lamina propria due to edema and infiltration by lymphocytes (Fig. 25).

Intestine : (500 mg lead + 200 mg vit. /kg diet).

Moderate thickening of intestinal villi with leukocytes, edema and fibroblasts were evident (Fig. 26). The surface epithelium showed partial or completed dequamation.

Intestine : (1000 mg lead + 200 mg vit. /kg diet).

Chronic chatarrhal enteritis characterized by local replacement of some glands with lymphocytes with mild pariglandular fibrosis (Fig. 27). The chronic inflammatory reaction usually seen in mucosa and submucosa and partially extended to the muscular coat.Economic efficiency :

The economic efficiency and money return per hen fed the different formulated diets are summarized in Table 7. The net revenue and economic efficiency values ranged between 2.80-8.04 and 0.272-0.699, respectively. The lowest values were recorded for the hens in T7, while the highest values were listed for the hens in T3 for the interaction effect.

In conclusion, birds fed diets contaminated with lead deleteriously affected laying hens performance and economic efficiency. Natural clay and vitamin E addition to the polluted birds diets with lead reduce the lead toxicity by reducing lead absorption in the intestinal tract and increasing fecal excretion. It could be recommended that addition of clay or vitamin E to the polluted bird diets with the lead improved the productive efficiency and histological changes in the liver, kidney and intestinal tissues.



Fig. (1) : Liver of chicken received 0.0 mg lead/kg diet showing apparently normal hepatic parenchyma (H & E x 120).



Fig. (3): Liver of chicken received 1000 mg lead/kg diet showing focal necrosis with multiple leukocyte aggregations (H & E x 120).



Fig. (5) : Liver of chicken received 500 mg lead + 3% clay/kg diet showing degenerative changes in the hepatic cells (H & E x 150).



Fig. (2) : Liver of chicken received 500 mg lead/kg diet showing focal haemorrhages in the hepatic parenchyma (H & E x 120).



Fig. (4) : Liver of chicken received 0.0 mg lead + 3% clay/kg diet showing vacuolated hepatic cells and hyperplastic Kuffer's cells (H & E x 300).



Fig. (6): Liver of chicken received 1000 mg lead + 3% clay/kg diet showing focal necrosis of hepatocytes with hyalinized wall of hepatic arterioles. (H & E x 150).



Fig. (7) : Liver of chicken received 0.0 mg lead + 200 mg vit. E/kg diet showing portal and interstitial leukocytic aggregations (H & E x 120).



Fig. (9) : Liver of chicken received 1000 mg lead + 200 mg vit. E/kg diet showing focal replacement of hepatic parenchyma with round cells. (H & E x 150).



Fig. (8) : Liver of chicken received 500 mg lead + 200 mg vit. E/kg diet showing slightly swollen hepatic cells and dialted central vein. (H & E x 80).



Fig. (10): Kidney of chicken received 0.0 mg lead/kg diet showing apparently normal renal parenchyma (H & E x 120).



Fig. (11) : Kidney of chicken received 500 mg lead/kg diet showing haemowhages and leukocytic infiltration. (H & E x 120).



Fig. (12): Kidney of chicken received 1000 mg lead/kg diet showing haemowhages and fibroblastic proliferation in renal tissues. (H & E x 300).



Fig. (13) : Kidney of chicken received 0.0 mg lead + 3% clay/kg diet showing congested capillaries with interstitial lymphocytic infiltration. (H & E x 300).



Fig. (15) : Kidney of chicken received 1000 mg lead + 3% clay/kg diet showing congestion with interstitial leukocytic aggregation. (H & E x 80).



Fig. (14) : Kidney of chicken received 500 mg lead + 3% clay/kg diet showing nephrosis in some renal tubules (H & E x 150).



Fig. (16) : Kidney of chicken received 0.0 mg lead + 200 mg vit. E showing congested renal blood vessels and a few cellular casts (H & E x 120).



Fig. (17) : Kidney of chicken received 500 mg lead + 200 mg vit. E showing degenerative or necrotic changes in some renal tubules (H & E x 80).



Fig. (18) : Kidney of chicken received 1000 mg lead + 200 mg vit. E showing necrosis and replacement with mononuclear cells (H & E x 150).



Fig. (19): Intestine of chicken received 0.0 mg lead/kg diet showing apparently normal intestinal coats (H & E x 120).



Fig. (21) : Intestine of chicken received 1000 mg lead/kg diet showing partial desquamation of villous epithelium. (H & E x 120).



Fig. (20) : Intestine of chicken received 500 mg lead/kg diet showing partial desquamation with distorded mucosa. (H & E x 120).



Fig. (22) : Intestine of chicken received 0.0 mg lead + 3% clay/kg diet showing sloughing of villous epithelium and thickened lamina propria. (H & E x 300).



Fig. (23): Intestine of chicken received 500 mg lead + 3% clay/kg diet showing partial necrosis of some intestinal glands or metaplasia to goblet cells. (H & E x 120).



Fig. (24): Intestine of chicken received 1000 mg lead + 3% clay/kg diet showing replacement of some glands with leukocytes. (H & E x 120).



Fig. (25): Intestine of chicken received 0.0 mg lead + 200 mg vit. E/kg diet showing mild thickening of lamina propria. (H & E x 80).



Fig. (26) : Intestine of chicken received 500 mg lead + 200 mg vit. E/kg diet showing moderate thickening of intestinal villi by leukocytes (H & E x 120



Fig. (27): Intestine of chicken received 1000 mg lead + 200 mg vit. E/kg diet showing chronic catarrhal enteritis (H & E x 120).

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Ingredients	%
Yellow corn	66.55
Extracted soybean meal,	18.00
44%	
Wheat bran	5.80
Limestone	6.76
Bone meal	2.20
Premix*	0.30
Sodium chloride	0.30
Methionine	0.09
Total	100.00
Calculated nutrients content a	ccording to NRC (1994)
ME, Kcal/ kg	2709
СР	14.54
Methionine <u>+</u> Cystine	0.58
Lysine	0.69
Calcium	3.30
Available phosphorus	0.33
Total phosphorus	0.63

 Table (1): Compositions and calculated analysis of the experimental diet

*Vitamins and minerals premix at 0.3% of the diet supplies the following per kg of the diets Vit. A : 12000 IU ; Vit. D : 2000 IU; Vit. E : 40 mg; Vit. K₃ : 4 mg ; Vit. B₁ : 3 mg ; Vit. B₂ : 6 mg Vit. B₆ : 4 m; Vit. B₁₂ : 0.3 mg; Niacin : 30 mg ; Ca pantothenate : 12 mg, Folic acid : 1.5 ; Biotin : 0.08 mg ; Choline chloride : 50%, 700 mg ; Mn : 10 mg ; Cu : 10 mg; Fe : 40 mg ; Zn : 70 mg ; Se : 0.2 mg ; I : 1.5 mg ; Co : 0.25 .

атненога	ion at differ	ent experime	inai period	S.				
		Egg nu	mber			Egg pro	duction (%)	
Ireatment	41-44 wks	45-48 wks	49-52 wks	41-52 wks	41-44 wks	45-48 wks	49-52 wks	41-52 wks
Lead level (mg/kg_diet)	*	*	*	*	*	*	*	*
0	18.10 ± 0.33^{a}	18.02 ± 0.27^{a}	18.67 ± 0.28^{a}	54.80 ± 0.58^{a}	64.65 ± 1.18^{a}	64.37 ± 0.97^{a}	66.69 ± 1.00^{a}	65.24 ± 0.69^{a}
500	15.30 ± 0.34^{b}	15.36 ± 0.40^{b}	15.31 ± 0.56^{b}	45.97 ± 1.12^{b}	54.66 ± 1.23^{b}	54.85 ± 1.43^{b}	54.69 ± 1.98^{b}	54.73 ± 1.33^{b}
1000	$13.35 \pm 0.24^{\circ}$	$13.29 \pm 0.30^{\circ}$	13.27 <u>+</u> 0.29 ^c	$39.92\pm0.74^{\circ}$	47.69 <u>+</u> 0.86 ^e	$47.47 \pm 1.08^{\circ}$	$47.41 \pm 1.02^{\circ}$	$47.52 \pm 0.88^{\circ}$
Feed additives	NS	SN	*	*	NS	NS	*	*
Without	15.01 <u>+</u> 0.81	14.95 <u>+</u> 0.86	14.76 <u>+</u> 0.94 ^b	44.73 ± 2.58^{b}	53.62 <u>+</u> 2.88	53.40 <u>+</u> 3.06	52.72 ± 3.37^{b}	53.25 ± 3.07^{b}
Clay (3%)/diet	15.72 <u>+</u> 0.69	15.74 <u>+</u> 0.66	16.09 ± 0.76^{a}	47.55 ± 1.97^{a}	56.13 <u>+</u> 2.45	56.23 <u>+</u> 2.37	57.47 ± 2.72^{a}	56.61 ± 2.35^{a}
Vitamin E (200mg/kg diet)	16.03 ± 0.72	15.98 ± 0.70	16.41 ± 0.82^{a}	48.41 ± 2.16^{a}	57.25 <u>+</u> 2.58	57.06 <u>+</u> 2.50	58.60 ± 2.92^{a}	57.63+2.57 ^a
Interaction	*	*	*	*	*	*	*	*
T1 0.0	17.97 ± 0.19^{a}	18.04 ± 0.59^{a}	18.36 <u>+</u> 0.47 ^a	54.37 ± 1.12^{a}	64.17 ± 0.66^{a}	64.44 ± 2.09^{a}	65.57 ± 1.66^{a}	64.73 ± 1.34^{a}
T2 0.0+ Clay	17.90 ± 0.85^{a}	17.84 <u>+</u> 0.55 ^a	18.46 <u>+</u> 0.68 ^a	54.20 ± 0.49^{a}	63.93 ± 3.05^{a}	63.71 ± 1.96^{a}	65.93 ± 2.42^{a}	64.52 ± 0.58^{a}
T3 0.0+vit. E	18.44 ± 0.67^{a}	18.19 ± 0.41^{bc}	19.20 ± 0.23^{a}	55.83 ± 1.31^{a}	65.86 ± 2.40^{a}	64.95 ± 1.67^{a}	68.58 ± 0.82^{a}	66.46 ± 1.56^{a}
T4 500 mg lead	14.41 ± 0.62^{bc}	14.38 ± 0.41^{bc}	13.63 <u>+</u> 0.47°	$42.42 \pm 1.33^{\circ}$	51.46 ± 2.22^{bc}	$51.35{\underline{+}}1.48^{bc}$	$48.69 \pm 1.67^{\circ}$	$50.50 \pm 1.58^{\circ}$
T5 500+ Clay	15.60 ± 0.53^{b}	15.70 ± 0.68^{b}	16.15 <u>+</u> 0.77 ^b	47.44 ± 1.52^{b}	55.71 <u>+</u> 1.89 ^b	56.06 <u>+</u> 2.43 ^b	57.67 <u>+</u> 2.75 ^b	56.48 ± 1.81^{b}
T6 500+vit. E	15.90 ± 0.36^{b}	16.00 ± 0.74^{b}	16.16 <u>+</u> 0.88 ^b	48.06 ± 1.16^{b}	56.80 ± 1.29^{b}	57.14 <u>+</u> 2.63 ^b	57.70 ± 3.15^{b}	57.21 ± 1.38^{b}
T7 1000 mg lead	12.67 ± 0.31^{d}	12.44 ± 0.41^{d}	$12.29\pm0.26^{\circ}$	37.39 ± 0.70^{d}	45.24 ± 1.09^{d}	44.42 ± 1.48^{d}	$43.89 \pm 0.94^{\circ}$	44.52 ± 0.82^{d}
T8 1000+ clay	13.65 ± 0.32^{cd}	13.70 ± 0.46^{cd}	13.67 <u>+</u> 0.42 ^c	41.01 ± 0.85^{c}	48.74 ± 1.14^{cd}	48.92 ± 1.62^{cd}	$48.81 \pm 1.45^{\circ}$	$48.82 \pm 1.01^{\circ}$
T9 1000+ vit. E	13.74 ± 0.39^{cd}	13.74 ± 0.42^{cd}	13.86 <u>+</u> 0.13 ^c	$41.35\pm0.74^{\circ}$	49.08 ± 1.40^{cd}	49.08 ± 1.51^{cd}	$49.51 \pm 0.46^{\circ}$	$49.23 \pm 0.88^{\circ}$
a, b, and d= means be:	aring differen	t letter in each o	classification,	are significar	ntly (P<0.05)	different		

	Table
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amelion	ation at diff	ferent exper	imental peric	ods.				
l		Egg we	eight (g)			Egg n	nass (g)	
Treatment	41-44 wks	45-48 wks	49-52 wks	41-52 wks	41-44 wks	45-48 wks	49-52 wks	41-52 wks
Lead level (mg/kg_diet)	*	*	NS	*	*	*	*	*
0	52.86 <u>+</u> 0.39 ^a	50.76 ± 0.27^{a}	49.72 <u>+</u> 0.51	51.11 ± 0.27^{a}	957.07 ± 20.66^{a}	914.75 <u>+</u> 14.11 ^a	928.65 <u>+</u> 17.81 ^a	2801.42 <u>+</u> 38.29 ^a
500	51.40 <u>+</u> 0.50 ^b	49.30 ± 0.67^{ab}	48.17 <u>+</u> 0.63	49.62 ± 0.30^{b}	786.62 ± 19.16^{b}	757.38 <u>+</u> 22.97 ^b	738.69 <u>+</u> 32.27 ^b	2283.08 ± 64.88^{b}
1000	50.03 ± 0.48^{b}	48.13 <u>+</u> 0.73 ^b	47.27 <u>+</u> 0.83	48.48 <u>+</u> 0.48 ^c	$668.33 \pm 15.19^{\circ}$	640.21 <u>+</u> 19.54 ^e	627.82 <u>+</u> 18.95 [°]	1936.46 <u>+</u> 48.04 ^c
Feed additives	SN	SN	SN	NS	*	*	*	*
Without	50.29 <u>+</u> 0.53	48.79 <u>+</u> 0.83	47.63 <u>+</u> 0.77	49.00 <u>+</u> 0.56	762.40 ± 47.95^{b}	732.40 <u>+</u> 49.71 ^b	705.66 <u>+</u> 51.04 ^b	2200.46 ± 146.96^{b}
Clay (3%)/diet	51.77 <u>+</u> 0.51	49.63 <u>+</u> 0.57	48.67 <u>+</u> 0.77	50.02 <u>+</u> 0.46	815.12 <u>+</u> 40.48 ^a	783.68 <u>+</u> 39.64 ^{ab}	784.82 <u>+</u> 43.31 ^a	2384.05+115.65 ^a
Vitamin E (200mg/kg diet)	51.93 ± 0.68	49.77 <u>+</u> 0.63	48.86 <u>+</u> 0.66	50.19 ± 0.45	834.49 <u>+</u> 44.79 ^a	796.26 ± 39.04^{a}	804.67 <u>+</u> 48.35 ^a	2436.46+128.63 ^a
Interaction	*	SN	SN	*	*	*	*	*
T1 0.0	52.57 <u>+</u> 0.27 ^{ab}	50.57 <u>+</u> 0.26	48.80 <u>+</u> 1.20	50.55 ± 0.35^{ab}	939.13 <u>+</u> 13.65 ^a	912.39 ± 28.90^{a}	895.82 ± 30.02^{a}	2748.28 ± 62.93^{a}
T2 0.0+ Clay	53.00 ± 0.20^{ab}	50.80 ± 0.80	50.10 ± 0.40	51.30 ± 0.28^{a}	948.50+43.35 ^a	906.10 ± 28.19^{a}	924.46 <u>+</u> 29.00 ^a	2780.24 ± 15.10^{a}
T3 0.0+vit. E	53.30 ± 1.21^{a}	50.90 <u>+</u> 0.40	50.27 <u>+</u> 0.93	51.49 <u>+</u> 0.63 ^a	983.57 <u>+</u> 50.06 ^a	925.76 <u>+</u> 25.66 ^a	965.67 <u>+</u> 28.82 ^a	2875.74 ± 94.92^{a}
T4 500 mg lead	50.50±0.76 ^{bc}	48.50 ± 1.32	47.60 <u>+</u> 0.96	48.87±0.51 ^{bc}	727.32 ± 29.25^{cd}	698.04 ± 36.36^{cd}	648.75 <u>+</u> 23.31°	$2074.02 \pm 84.14^{\circ}$
T5 500+ Clay	51.80 ± 0.79^{ab}	49.60 <u>+</u> 0.80	48.30 <u>+</u> 1.38	49.90 ± 0.24^{ab}	807.25 ± 15.62^{bc}	779.61 <u>+</u> 46.51 ^{bc}	780.24 <u>+</u> 48.23 ^b	2366.85 ± 67.51^{b}
T6 500+vit. E	51.90 ± 1.11^{ab}	49.80 ± 1.57	48.60 <u>+</u> 1.15	50.10 ± 0.58^{ab}	825.28+24.27 ^b	794.49 ± 12.20^{b}	787.07 <u>+</u> 59.59 ^b	2408.38±75.26 ^b
T7 1000 mg lead	$49.00\pm0.20^{\circ}$	47.30 <u>+</u> 1.95	46.50 <u>+</u> 1.86	47.60 <u>+</u> 1.11 ^c	620.76 <u>+</u> 16.88 ^e	$586.78 \pm 10.10^{\circ}$	572.42 <u>+</u> 35.13°	1779.08 ± 35.89^{d}
T8 1000+ clay	50.50 <u>+</u> 0.93 ^{bc}	48.50 <u>+</u> 1.15	47.60 <u>+</u> 1.76	48.87 <u>+</u> 0.93 ^{bc}	689.60+27.12 ^{de}	665.33 <u>+</u> 37.28 ^{de}	649.75 <u>+</u> 21.45°	$2005.05 \pm 72.60^{\circ}$
T9 1000+ vit. E	50.60 ± 1.02^{abc}	48.60 <u>+</u> 0.87	47.70 ± 1.12	48.97±0.33 ^{bc}	694.63 <u>+</u> 6.56 ^{de}	668.53±30.76 ^{de}	661.28 <u>+</u> 16.75°	$2025.25 \pm 49.52^{\circ}$
a, b, and d= means be	aring different	t letter in each	classification,	are significal	ntly (P<0.05)	different		

Table (3): Egg weight (g) and egg mass (g) of Mandara hens as affected by lead contamination and their

NS : Not significant

ameliora	tion at differ	ent experime	ntal periods.					
1		Feed in	ntake (g)			Feed cor	iversion	
Treatment	41-44 wks	45-48 wks	49-52 wks	41-52 wks	41-44 wks	45-48 wks	49-52 wks	41-52 wks
ead level (mg/kg_diet)	*	*	*	*	*	*	*	*
	91.93 ± 1.49^{a}	96.19 ± 0.76^{a}	101.69 ± 0.85^{a}	96.60 ± 0.62^{a}	$2.70\pm0.09^{\circ}$	$2.95\pm0.05^{\circ}$	$3.07 \pm 0.06^{\circ}$	$2.90\pm0.05^{\circ}$
00	84.41 <u>+</u> 0.56 ^b	88.23 ± 0.95^{b}	92.79 ± 0.53^{b}	88.47 <u>+</u> 0.45 ^b	3.02 ± 0.09^{b}	3.29 ± 0.12^{b}	3.57 ± 0.16^{b}	3.28 ± 0.10^{b}
000	82.98 <u>+</u> 0.56 ^b	86.37 ± 0.76^{b}	90.98 ± 0.74^{b}	86.77 <u>+</u> 0.57 ^b	3.49 ± 0.09^{a}	3.81 ± 0.12^{a}	4.09 ± 0.14^{a}	3.78 ± 0.10^{a}
eed additives	NS	NS	NS	NS	*	*	*	*
/ithout	87.08 <u>+</u> 1.89	91.16 <u>+</u> 1.65	96.17 <u>+</u> 1.95	91.47 <u>+</u> 1.64	3.28 ± 0.16^{a}	3.58 ± 0.19^{a}	3.93 ± 0.21^{a}	3.58 ± 0.18^{a}
lay (3%)/diet	85.81 <u>+</u> 1.65	89.38 <u>+</u> 1.75	94.27 <u>+</u> 1.67	89.82 <u>+</u> 1.58	2.99 ± 0.12^{b}	3.24 ± 0.13^{b}	3.43 ± 0.14^{b}	3.21 ± 0.12^{b}
itamin E (200mg/kg diet)	86.42 <u>+</u> 1.50	90.25 ± 1.70	95.01 ± 1.72	90.56 <u>+</u> 1.57	2.95 ± 0.12^{b}	3.22 ± 0.12^{b}	3.38 ± 0.16^{b}	3.17 ± 0.12^{b}
iteraction	*	*	*	*	*	*	*	*
1 0.0	92.50 ± 4.33^{a}	97.0 ± 1.72^{a}	103.0 ± 2.09^{a}	97.50 <u>+</u> 1.37 ^a	$2.76\pm0.16^{\circ}$	$2.98\pm0.12^{\circ}$	$3.22\pm0.05^{\circ}$	$2.99\pm0.11^{\circ}$
2 0.0+ Clay	91.39 ± 2.41^{a}	95.43 ± 1.72^{a}	100.68 ± 0.62^{a}	95.83 ± 1.41^{a}	$2.71\pm0.16^{\circ}$	$2.95\pm0.04^{\circ}$	$3.06 \pm 0.11^{\circ}$	$2.90\pm0.05^{\circ}$
3 0.0+vit. E	91.89 ± 1.38^{a}	96.14 <u>+</u> 0.67 ^a	101.39 ± 1.60^{a}	96.48 ± 0.26^{a}	$2.63\pm0.17^{\circ}$	$2.91 \pm 0.10^{\circ}$	$2.94 \pm 0.04^{\circ}$	$2.83 \pm 0.10^{\circ}$
4 500 mg lead	85.21 <u>+</u> 0.66 ^b	89.18 ± 0.43^{b}	93.86 ± 1.04^{b}	89.42 ± 0.34^{b}	3.29 ± 0.14^{b}	3.60 ± 0.20^{b}	4.06 ± 0.10^{ab}	3.63 ± 0.13^{b}
5 500+ Clay	83.68 <u>+</u> 1.03 ^b	87.21 <u>+</u> 2.25 ^b	91.82 ± 1.05^{b}	87.57 <u>+</u> 0.37 ^{bc}	$2.91 \pm 0.09^{\circ}$	3.16 ± 0.25^{bc}	$3.32 \pm 0.21^{\circ}$	$3.11 \pm 0.09^{\circ}$
6 500+vit. E	84.32 <u>+</u> 1.31 ^b	88.29 <u>+</u> 2.13 ^b	92.68 ± 0.32^{b}	88.43 <u>+</u> 1.16 ^{bc}	$2.86\pm0.06^{\circ}$	$3.11 \pm 0.03^{\circ}$	$3.34 \pm 0.28^{\circ}$	$3.09\pm0.10^{\circ}$
7 1000 mg lead	83.54 <u>+</u> 0.92 ^b	87.29 <u>+</u> 1.75 ^b	91.64 ± 2.01^{b}	87.49 <u>+</u> 1.49 ^{bc}	3.77 ± 0.06^{a}	4.17 ± 0.13^{a}	4.52 ± 0.30^{a}	4.13 ± 0.11^{a}
8 1000+ clay	82.36 <u>+</u> 1.41 ^b	85.50 ± 0.79^{b}	90.32 ± 0.83^{b}	$86.06 \pm 0.20^{\circ}$	3.36 ± 0.16^{b}	3.62 ± 0.18^{b}	3.90 ± 0.09^{b}	3.62 ± 0.13^{b}
9 1000+ vit. E	83.04 <u>+</u> 0.72 ^b	86.32 ± 1.57^{b}	90.96 ± 1.18^{b}	86.77 ± 1.05^{bc}	3.35 ± 0.01^{b}	3.63 ± 0.18^{b}	3.85 <u>+</u> 0.49 ^b	3.60 ± 0.07^{b}
, b, and d= means be	aring different	letter in each c	classification, ar	e significantly (P<0.05) differe	ent		

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Table (4): Feed intake (g) and feed conversion of Mandara hens as affected by lead contamination and their

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		in experime	mar perious					
		Fertili	(ty (%)			Hatchabili	ty (%)	
Treatment	41-44 wks	45-48 wks	49-52 wks	41-52 wks	41-44 wks	45-48 wks	49-52 wks	41-52 wks
Lead level (mg/kg_diet)	*	*	*	*	*	*	*	*
0	81.88 ± 1.18^{a}	83.03 ± 0.45^{a}	83.50 ± 0.51^{a}	82.80 ± 0.47^{a}	67.51 ± 2.40^{a}	66.95 ± 1.34^{a}	70.19 ± 1.20^{a}	68.22 ± 1.22^{a}
500	76.40 <u>+</u> 1.59 ^b	75.15 ± 1.16^{b}	71.15 ± 2.00^{b}	74.23 ± 1.04^{b}	58.33+2.35 ^b	56.20 ± 1.86^{b}	54.40+1.25 ^b	56.31 ± 0.97^{b}
1000	72.62 ± 1.51^{b}	64.43 ± 1.03	$64.82 \pm 1.39^{\circ}$	$73.32 \pm 2.64^{\circ}$	54.71 <u>+</u> 2.79 ^b	$42.22 \pm 1.67^{\circ}$	$40.19 \pm 1.95^{\circ}$	45.71 <u>+</u> 1.46 ^e
Feed additive	NS	NS	SN	*	NS	NS	NS	NS
Without	75.65 <u>+</u> 1.84	73.91 <u>+</u> 2.79	70.40 <u>+</u> 3.85	73.32 <u>+</u> 2.64 ^b	58.99 <u>+</u> 3.76	52.66 <u>+</u> 4.66	52.58 <u>+</u> 5.30	5474 <u>+</u> 4.07
Clay (3%)/diet	77.53 ± 2.12	75.26 ± 2.40	74.35+2.64	75.71 ± 2.14^{a}	60.65 <u>+</u> 2.75	55.74 <u>+</u> 3.23	56.09 <u>+</u> 4.25	57.49 ± 3.08
Vitamin E (200mg/kg diet)	77.71 <u>+</u> 1.84	76.45 <u>+</u> 1.97	74.71+2.37	76.29 ± 1.83^{a}	60.91 <u>+</u> 2.82	56.37 <u>+</u> 3.62	56.10 <u>+</u> 3.99	57.99 <u>+</u> 3.08
Interaction	*	*	*	×	NS	*	*	*
T1 0.0	81.72 ± 0.96^{ab}	83.33 ± 1.24^{a}	83.82 ± 0.81^{a}	82.95 ± 0.61^{a}	67.26 <u>+</u> 6.17	67.62 ± 1.00^{a}	70.93 ± 0.29^{a}	68.6 ± 2.16^{a}
T2 0.0+ Clay	82.43 ± 3.39^{a}	82.86 ± 0.39^{a}	83.40 ± 1.46^{a}	82.89 ± 1.10^{a}	67.76 <u>+</u> 5.07	65.72 <u>+</u> 0.77 ^{ab}	70.10 ± 1.81^{a}	67.86 ± 1.68^{a}
T3 0.0+vit. E	81.48 <u>+</u> 1.99 ^b	82.91 ± 0.85^{a}	83.28 ± 0.69^{a}	82.56 ± 1.06^{a}	67.52 <u>+</u> 2.26	67.50 <u>+</u> 4.33 ^a	69.54 ± 3.65^{a}	68.19 ± 3.20^{a}
T4 500 mg lead	74.19+2.17 ^{abc}	73.92 <u>+</u> 1.44 ^{bc}	66.31+5.29 ^{cd}	$71.47 \pm 2.04^{\circ}$	56.74 <u>+</u> 5.28	53.92 ± 3.64^{cd}	52.11+2.59 ^b	54.26+1.65 ^{bc}
T5 500+ Clay	77.22±3.12 ^{abc}	75.42 <u>+</u> 2.79 ^b	73.39 ± 1.62^{bc}	75.34 ± 1.32^{b}	59.04 ± 4.10	57.37 <u>+</u> 2.54 ^{bc}	55.46 <u>+</u> 2.81 ^b	57.28 ± 1.86^{b}
T6 500+vit. E	77.78 <u>+</u> 3.46 ^{abc}	76.11 <u>+</u> 2.25 ^b	73.75 ± 0.10^{b}	75.88 ± 1.09^{b}	59.21 <u>+</u> 4.40	57.36 <u>+</u> 4.25 ^{bc}	55.62 ± 0.52^{b}	57.39 ± 1.41^{b}
T7 1000 mg lead	$71.04\pm2.02^{\circ}$	$64.47 \pm 0.38^{\circ}$	61.08 ± 2.67^{d}	65.53 ± 0.61^{cd}	52.98 <u>+</u> 7.02	36.44 <u>+</u> 1.47f	34.70 ± 1.41^{d}	41.37 <u>+</u> 2.35 ^e
T8 1000+ clay	72.94 ± 3.19^{bc}	67.50 ± 1.44^{dc}	66.25+2.17 ^{cd}	68.90 <u>+</u> 1.75 ^{cd}	55.16 <u>+</u> 2.60	44.17 ± 0.48^{f}	42.71 <u>+</u> 4.21 ^e	47.34 ± 1.42^{d}
T9 1000+ vit. E	73.88 ± 3.19^{bc}	70.32 ± 1.10^{cd}	67.12 <u>+</u> 0.81 ^{cd}	$70.44 \pm 1.00^{\circ}$	56.00 <u>+</u> 5.94	46.06 <u>+</u> 2.28 ^{de}	$43.15 \pm 1.82^{\circ}$	48.40 ± 1.90^{cd}
a, b, and d= means bear	ing different l	etter in each c	lassification,	are significantly	y (P<0.05) diffe	erent		

Table (5): Fertility (%) and hatchability (%) of Mandara hens as affected by lead contamination and their amelioration at different experimental periods.

NS : Not significant 0° j

Table	(6): Carcass cha	racteri	stics o	of Mandara he	ens a	s aff	ected	by	lead
	contamination	and	their	amelioration	at	the	end	of	the
	experiment.								

Treatments	Carcass (%)	Heart (%)	Spleen (%)	Kidney (%)	Liver (%)
Lead level (mg/kg diet)	*	*	NS	NS	NS
0	68.35 ± 0.34^{a}	0.40 ± 0.02^{b}	0.12 <u>+</u> 0.01	0.58 <u>+</u> 0.04	2.36 <u>+</u> 0.09
500	66.74 <u>+</u> 0.64 ^{ab}	0.42 ± 0.03^{b}	0.12 <u>+</u> 0.01	0.67 <u>+</u> 0.03	2.65 <u>+</u> 0.17
1000	66.17 ± 0.84^{b}	0.48 ± 0.02^{a}	0.15 <u>+</u> 0.01	0.67 <u>+</u> 0.05	2.67 <u>+</u> 0.15
Feed additives	NS	NS	NS	NS	NS
Without	66.19 <u>+</u> 0.88	0.44 <u>+</u> 0.02	0.12 <u>+</u> 0.01	0.67 <u>+</u> 0.04	2.66 <u>+</u> 0.14
Clay (3%)/diet	68.05 <u>+</u> 0.46	0.40 <u>+</u> 0.02	0.12 <u>+</u> 0.01	0.65 <u>+</u> 0.04	2.54 <u>+</u> 0.19
Vitamin E (200mg/kg diet)	67.02 <u>+</u> 0.59	0.45 <u>+</u> 0.02	0.14 <u>+</u> 0.01	0.65 <u>+</u> 0.04	2.48 <u>+</u> 0.10
Interaction	NS	*	NS	NS	NS
T1 0.0	68.58 <u>+</u> 0.19	0.42 ± 0.02^{ab}	0.11 <u>+</u> 0.02	0.56 <u>+</u> 0.10	2.21 <u>+</u> 0.18
T2 0.0+ Clay	68.70 <u>+</u> 0.76	0.39 <u>+</u> 0.05 ^{ab}	0.11 <u>+</u> 0.01	0.51 <u>+</u> 0.04	2.38 <u>+</u> 0.18
T3 0.0+vit. E	67.75 <u>+</u> 0.69	0.39 <u>+</u> 0.03 ^{ab}	0.14 <u>+</u> 0.02	0.67 <u>+</u> 0.05	2.48 <u>+</u> 0.07
T4 500 mg lead	66.00 <u>+</u> 0.84	0.41 ± 0.06^{ab}	0.14 <u>+</u> 0.01	0.67 <u>+</u> 0.06	2.82 <u>+</u> 0.15
T5 500+ Clay	68.05 <u>+</u> 0.61	0.34 <u>+</u> 0.02 ^b	0.09 <u>+</u> 0.02	0.62 <u>+</u> 0.04	2.58 <u>+</u> 0.45
T6 500+vit. E	66.19 <u>+</u> 1.59	0.50 <u>+</u> 0.03 ^a	0.13 <u>+</u> 0.03	0.72 <u>+</u> 0.07	2.55 <u>+</u> 0.31
T7 1000 mg lead	63.99 <u>+</u> 1.79	0.49 ± 0.04^{a}	0.13 <u>+</u> 0.03	0.78 <u>+</u> 0.08	2.94 <u>+</u> 0.12
T8 1000+ clay	67.39 <u>+</u> 1.07	0.46 ± 0.02^{a}	0.16 <u>+</u> 0.01	0.68 <u>+</u> 0.10	2.66 <u>+</u> 0.40
T9 1000+ vit. E	67.13 <u>+</u> 0.76	0.48 ± 0.02^{a}	0.15 <u>+</u> 0.01	0.54 <u>+</u> 0.06	2.41 <u>+</u> 0.09

a, b, .. and d= means bearing different letter in each classification, are significantly (P<0.05) different NS : Not significant

		m Perro													
Items	Lead lev	els (mg/kg	(diet)		Feed additiv	es				I	nteraction				
	0.0	005	1000	Without	Clay	Vit.E	T1	T 2	T 3	T 4	T5	T6	T 7	T8 (1000	T9
					(3% diet)	200	(0.0 mg)	(0.0 + clay)	(0.0	(500 mg	(500mg	(500 mg	(1000mg	mg lead	(1000mg
						mg/kg diet			+vit.E)	Lead)	lead +	lead	lead)	+clay)	lead
											clay)	+vit.E)			+vit.E)
Total feed intake	8.114	7.431	7.29	7.68	7.54	7.61	8.19	8.05	8.10	7.51	7.36	7.43	7.35	7.23	7.29
/ hen															
Price /kg fed	1.40	1.40	1.40	1.40	1.41	1.42	1.40	1.41	1.42	1.40	1.41	1.42	1.40	1.41	1.42
Total feed	11.36	10.40	10.20	10.75	10.64	10.80	11.47	11.35	11.50	10.5	10.38	10.55	10.29	10.19	10.35
cost (LE)															
Total number of	54.80	45.97	39.92	44.73	47.55	48.41	54.37	54.20	55.83	42.42	47.44	48.06	37.39	41.01	41.35
eggs / hen															
Total price of	19.18	16.09	13.97	15.66	16.64	16.91	19.03	18.97	19.54	14.85	16.60	16.82	13.09	14.35	14.47
eggs / hen (LE) ¹															
Net revenue hen	7.82	5.69	3.77	4.91	6.00	6.14	7.56	7.62	8.04	4.34	6.22	6.27	2.80	4.16	4.12
(LE)															
Economic	0.686	0.547	0.37	0.457	0.564	0.569	0.659	0.671	0.699	0.413	0.599	0.594	0.272	0.408	0.398
efficiency (EEF)															
Relative (EE) ³ (%)	100	79.51	53.78	100	123.41	124.51	100	101.82	106.07	62.67	90.90	90.14	40.97	62.06	60.39
1 The price of	one egg =	: 35P.T													
Not more	nor mait o	f total faa	d poort												

	Table
ex	3
perimental pe	: Economic
riods.	efficiency
	\mathbf{of}
	Mandara
	hens
	as
	affected
	by
	lead
	contamination
	and
	their
	amelioration
	at
	different

1 The price of one egg = 35P.T 2 Net revenue per unit of total feed cost 3 relative economic efficiency % of the control

REFERENCES

- Abaza, M.; Azza, El-Sebai and Szalay, L. (1996). Reproductive traits and serum parameters of cockerels exposed to heavy metals. Egyptian Journal of Poultry Science, 16 (111): 689-702.
- Abou-Zeid, A.E.; Sorour, J. and El-Haback, M.M. (2000). Magnitude of lead toxicity in White Pekine duckling . Egypt J. Polut. Sci., 20: 789-815.
- Assennato, G.; C. Paci, M. E. Baser; R. Molinini; R.G. Candela; B.M. Altmura and R. Giorgino (1986). Sperm count suppression without endoeirrne dysfunction in lead- exposed men. Arch. Environ. Health. 41: 387-390.
- Attia, A.I.; Ayyat, M.S.A.; Bakir, A.A. and El-Zaiat, A.A. (2005). The role of clay or vitamin E in silver Montazah layer hens fed diets contaminated by lead at various levels. 2nd paper: Carcass characteristics, blood components and lead residues in the tissues and eggs. Allattenyesztes takarmanyozas, 54. 179 – 190.
- Ayyat, M.S. and Marai, I.F.M. (1997). Use of natural clays in animal production. Proc. Int. Conf. Anim., Poult. and Rabbit Prod. and Health, Cairo, Egypt, 91-111.
- **Ayyat, M.S.A. ; Bakir, A.A. ; Attia , A.I and El-Zaiat A. A. (2005).** The role of clay or vitamin E in silver Montazah layer hens fed on diets contaminated by lead at various levels. 1st paper : performance and egg components. Allattenyesztes es takar manyozas, 54. 1. 81-92.
- Barreto, S. C. T.; Ferreira, W. M. and Goncalves, T. M. (1999): Protein and vitamin E levels for broiler breed hens. 1. Effects of broiler breed performance, egg composition and performance of their progeny, Arquivo- Brasileire- de-Medicinea-Veterinaria -e-Zootecina, 5: 183-192.
- **De Gennaro, L.D. (1978).** The effects of lead nitrate on the central nervous system of the chicks embry.1. Observations of light and electron microscopy. Growth, 24:141-155.
- Duncan, D.B. (1955). Multiple range and multiple F-test. Biometrics, 11: 1-42.
- **Dury, A. A. and Wallington , E. A. (1980).** Carlton 's histological technique 4th Edition , P.129 -189 , Oxford University , Press, London, Now York , Toronto.

- Edens, F. W.; Emily, B.; Morgan, G. W.; Bursian, S. J. and Thaxton, P. (1976). Effect of dietary lead on reproductive performance in Japanese quail. Tox. Appl. Pharmac., 38: 307-314.
- Edens, F. W. and Melvin, V. K. (1989). Lead influences on physiological and growth responses in conturinx Japonica selected for large body weights. Environment Research, 50 : 140-156.
- El-Zaiat, A.A. (2003). Effect of ration poluted with some poisonous materials on productive performance of lying Hens. Ph.D. Thesis, Faculty of Agriculture, Zagazi University, Egypt.
- Englemann, D.; Halle, I.; Flachosky, G. and Sallmann, H. P. (1999). Effects of high vitamin E intake in laying hens on thyroid hormones in the plasma of chickens embryos. Vitamine and zusatsstofffe in der ernahrung von mensch und Tier : 7 Symposium Jena thuringen, Germany, 22 and 23 September, 160-165.
- Fathi, M.M.; El- Hommosany, Y.M. ;Ali , U.M.; Hemid, A.A. and Khidr,
 R.E. (1999). Performance of broiler chicks fed a diet polluted with cadmium or lead. Egyptian Journal of Poultry Science, 19:813–829.
- Franchine, A.S.; Bertuzz, G.; Tosarelli, C. and Manferda, G. (1995). Vitamin E in Viral inactivated vaccines. Poult. Sci., 74. 666-671.
- Gore, A. B. and Qureshi, M. E. (1997). Enhancement of humoral and cellular immunity by vitamin E after embryonic exposure. Poultry Science, 76 : 984-991.
- Hais, E. (1992). Staying healthy with nutrition. The complete guide to diet and nutritional medicine. 10th printing, colestial arts (Berkeley, CA), 116 p. Healthy, World, Health Bookstore, ISBN 0890874816.
- Harvey, R. B., Kubena, L.F., Elissalde, M.H. and Phillips, T.D. (1993). Efficacy of zeolitic ore compounds on the toxicity of aflatoxin to growing broiler chickens. Avian – Diseases, USA, 37 (1): 67-73.
- Jeng, S. L.; Eec, S.J.; Eiu, Y.F.; Yang, S. C. and Eiou, P.P. (1997). Effect of lead ingestion on concentrations of lead in tissues and eggs of laying Tsaiya ducks in Taiwan. Poult. Sci., 76, (1): 13-16.
- Khan, M. Z.; Szarek, J.; Krasnodebska Depta, A. and Koncicki, A. (1993). Effect of concurrent administration of lead and selenium on some haemotological and biochemical parameters of broiler chickens. Acta Vet. Hungarica, Olsztyn, Poland, 41: (1-2): 123-137.

- Klauder, D. S. and Petering, G. F. (1975). Protective value of dietary copper and iron against some toxic effects of lead in rats. Environmental Health Prospect, 12 : 77.
- Kling, L.J. and Scares, J. H. (1980). Vitamin E deficiency in the Japanese quail. Poult. Sci., 59: 2352-2354.
- Marai, I.F.M.; Ayyat, M.S.; Gaber, H. A. and Abdel-Monem, U. M. (1996). Effects of concurrent administration of lead and selenium on some hematological and biochemical parameters of broiler chickens. Acta Vet. Hung., 41. (1-2): 123-137.
- Meluzzi, A.; Simonicin, F.; Sirri, F.; Vadi, L. and Giordauin, G. (1996). Feeding hens diets supplemented with heavy metals (chromium nickel and lead). Archi- Fur Gefling elkunde, 60 : 119-125.
- Mumpton, F. A. and Fishman, P. H. (1977). The application of natural zeolites in animal science and aquaculture. J. Anim. Sci., 45: 1188-1203.
- **NRC** (1994). Nutrient Requirements of Poultry. 9th Revised Edition, National Academy Press, Washington, D.C., USA.
- SAS Institute (2004).SAS, DSTAT Users Guide. SAS Institute Inc. Cary, Ne.
- Stanchev, C. (1989). Investigations about the effects of increasing lead doses on the performance of broiler and carry over of lead in whole body and in several tissues. I. Performance of broilers. 6th International Trace Element Symposium, 5: 1567-1573.
- Stone, C. L. and Soares, J. H. (1974). Studies on the metabolism of lead in the Japanese qual. Poultry Science, 54 : 182.
- Vodela, J. K.; Lenz, S.D.; Rendene, J. A.; Mcelhenney, W. H. and Kemppainen B. W. (1997). Drinking water contaminated (arsenic cadmium, lead, benzene and trichlore ethylene) 2. Effects on reproductive performance, egg quality and embryo toxicity in broiler breeders. Poultry Science 76 : 1493-1500.
- **Voget, H. (1992).** Bentonite mediatomite in layer diets. Londbauforschung Volkeniode, 43 : 89-94.
- Whisenhunt, J. E. and Maurice, D. V. (1981). The presuppose of egg shell quality to dietary manganese and lead. Poultry Science, 60 : 160.
- Youssef, S.A.H.; El-Minawy, H.M.F.; Soliman, G.A.; Sanousi, A.A. and Brawy, A.M. (1995). Some toxicological and pathological studies on the effect of subchronic lead poisoning in broiler with reference to immune system. Egypt. J. Comp. Path. Clin Path., 8: 93 – 104.

الملخص العربى

أداء الدجاج البياض وبعض التغيرات التشريحية الناتجة عن تلوث العليقه بالرصاص والمضاف إليها الطفلة وفيتامين E

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أجريت هذه التجربة لمعرفة تأثير الغذاء الملوث بثلاث مستويات من أستيات الرصاص (Pb) (صفر، 500 ، 1000 ملجم كجم / عليقه) وثلاث مستويات من الإضافات الغذائية (بدون طفلة أو فيتامين E ، 3 % طفلة و 200ملجم فيتامين E / كجم عليقه) على علائق الدجاج البياض والتغيرات التشريحية في الكبد والكليتين والأمعاء وتم استخدام 216 طائر (189 دجاجة و 27 ديك) من سلالة المندرة المحلية عند عمر 40 أسبوع في هذه التجربة 0 قسمت كل من الدجاج والديوك في هذه التجربة عشوائية إلى 9 معاملات (21 دجاجة و 3 ديوك في كل معاملة) في ثلاث مكررات لكل منها 7 دجاجة وديك واحد.

أوضحت النتائج أن الغذاء الملوث بالرصاص عند مستوى 500 أو 1000 ملجم /كجم عليقه أدى إلى انخفاضا معنوياً (على مستوى 500) فى أنتاج البيض "جم"، عدد البيض، ووزن البيض (جم) ، كمية البيض ، الغذاء المأكول ، ومعدل تحويل الغذاء ، النسبة المئوية لكل من الذبحية ، القلب، الخصوبة ، ومعدل الإخصاب ومعدل الفقس وذلك خلال الفترة من 40-44 ، 40-25 ، 41-52 أسبوع بينما كان وزن البيض غير معنوي وذلك خلال الفترة من 40-55 أسبوع . 10-55 أسبوع بينما كان وزن البيض غير معنوي وذلك خلال الفترة من 40-55 أسبوع . الدادت النسبة المئوية لكل من الطحال والكليتين والكبد بدرجة غير معنوية من 40-55 أسبوع . ازدادت النسبة المئوية لكل من الطحال والكليتين والكبد بدرجة غير معنوية الطيور المغذاة على العليقه الملوثة بالرصاص. أدى إضافة الطفلة أو فيتامين E إلى العليقه إلى تحسن معنوي(على مستوى 60.5) فى إنتاج البيض (%) وعدد البيض خلال الفترة من 52، 14-25 أسبوع وكمية البيض ومعدل تحويل الغذاء طوال فترة التجربة ومعدل الإخصاب عند الفترة من 40-55 أسبوع بينما كان هناك تحسن غير معنوي في عدد البيض وإنتاج البيض عند الفترة من 40-55 أسبوع وكمية البيض ومعدل تحويل الغذاء عند الفترة من 40-55 أسبوع وكمية المبوع ومعدل الوخصاب عند الفترة من 40-55 أسبوع وينما كان هناك تحسن غير معنوي في عدد البيض وإنتاج البيض عند الفترة من 40-55 أسبوع ولمينا كان هناك نحسن غير معنوي في عدد البيض وإنتاج البيض عند الفترة من 40-55 أسبوع وينما كان هناك تحسن غير معنوي في عدد البيض وإنتاج البيض عند الفترة من 40-54 أسبوع وكمية البيو معدل تحويل الغذاء طوال فترة التجربة ومعدل الإخصاب عند الفترة من 40-44 ، 45-48 أسبوع ، ووزن البيض والغذاء المأكول ومعدل الفقس طوال لكن من الذبيحة والقلب والطحال والكليتين والكبد مقارنة بالعليقة الخالية من الطفلة وفيتامين E لكل من الذبيحة والقلب والحال والكليتين والكبد مقارنة بالعليقة الخالية من المؤلية المئوية الكل من الخلية والغلية والكليتين والكبد مقارنة بالعليقة الخالية من الطفلة وفيتامين E لكل من الذبيحة والقاب والطحال والكليتين والكبد مقارنة بالعليقه الخالية من الطفلة وفيتامين E لكل من الخلية الخالية مالولي أدى إضافة الطفلة وفيتامين E إلى العليقة الملوثة أو غير الملوثة بالرصاص إلى تحسن معنوي (على مستوى 0.05) فى عدد البيض وإنتاج البيض (%) وكمية البيض والغذاء المأكول ومعدل تحويل الغذاء ومعدل الإخصاب طوال فترة التجربة . بينما أزداد وزن البيض عند عمر والنسبة المئوية للقلب بدون معنوية مقارنة بالعليقة الخالية من تلك الإضافات0 وعلى النقيض من ذلك حدث تحسن بدرجة غير معنوية فى وزن البيض عند عمر 54–48 ، 20 ومعدل الفقس عند عمر 14–44 أسبوع والنسبة المئوية لكل من الذبيحة، الطحال، والكليتين، والكبد مقارنة بالتعذية على العليقة الخالية من تلك الإضافات0 وعلى النقيض من

أوضحت التغيرات التشريحية فى كل من الكبد والكليتين والأمعاء فى الطيور المغذاة على العليقه الملوثة بالرصاص عند مستوى 500 أو 1000 ملجم /كجم عليقه أن هناك احتقان في الأنسجة الكبدية وتضخم وحدوث تكرز في الخلايا الكبدية (الكبد) كما أن هناك تغيرات فى الأنابيب والكريات الكلوية وخاصة في النسيج الطلائي المبطن لهذه الأنابيب والكريات الكلوية مع ظهور وتحلل وارتشاح فى الخلايا الكلوية والدموية كما أن هناك تضخم فى الخلايا البينية وارتشاح للخلايا الليمفاوية (الكليتين). أما فى الأمعاء فقد ظهر أن هناك هدم وتحلل فى الخلايا الطلائية المخاطية الداخلية المبطنة للأمعاء كذلك حدث تشوهات كبيرة فى الجزء العلوي من الأمعاء. أما عند إضافة الطفلة أو فيتامين E إلى العليقه الملوثة بالرصاص سواء عند مستوى 500 أو 1000 ملجم /كجم عليقه ظهر أن هناك تت حسن في الكبديا المحاطية المناه الطفلة أو فيتامين اليا العليقه الملوثة بالرصاص سواء عند مستوى وتحسن واضح إلى حد ما في الخلايا والأنابيب الكلوية فى الكليتين وكذلك ت