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Effect of Milk Thistle Seed Powder Supplementation on The Performance of Growing Japanese Quail Fed Diet Polluted by Cadmium

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ABSTRACT: Two hundred and fifty Japanese quail chicks, seven days old without sexing were used in this study to investigate cadmium's harmful impacts on performance and immunological parameters, as well as their regulation by various levels of milk thistle plant seed powder (Mthsp) in growing quail. The quails were separated into five equal groups, each with 50 chicks, and each group was assigned five replicates (10 each). Group 1 served a basal diet with no supplements (T1), group 2, fed 0.10 g cadmium chloride/kg diet (T2), groups 3, 4, and 5 fed on 0.10 g cadmium chloride/kg diet supplemented with either of 4, 8 and 12g (Mthsp) /kg diet, respectively. Cd caused a significant ($P \leq 0.01$and 0.001) increase in white blood cell (WBCs) count and a decrease in hemoglobin (HGB) and a numerical decrease in packed cell volume (PCV) as compared with those of the control free of Cd supplementation. While a nonsignificant decrease in red blood cell (RBC) count was detected. Cd intoxicated quail had a nonsignificant ($P \geq 0.05$) reduction in serum catalase, and a significant ($P \leq 0.05$) decrease in total antioxidant capacity, although MDA was significantly ($P \leq 0.01$) elevated in contrast to the control group. These results mean that dietary supplemented with (Mthsp) /kg diet might be useful on performance, and hematological parameters induced by Cd and alleviated the adverse effect of Cd on antioxidative.

Keywords: Japanese quail, Cadmium, Milk thistle plant seed powder, Hematology, **Antioxidative**

INTRODUCTION

Heavy metal exposure can cause a wide range of health problems, and this is a global topic of concern (**Patra *et al.* 2011 and Adnan *et al.*, 2022**).

Environmental pollutants, in particular metals that are heavy, have been found to pose major risks to animal and human health. These threats are typically chronic, as heavy metals tend to accumulate in the body over extended periods of exposure **(Lippmann, 2009)**. **Adham et al. (2001)**, showed that there is a documented presence of metals including lead, cadmium, zinc, and mercury in the food and water supplies of animals and birds. These metals are widely utilized in farming and industrial chemical manufacturing.

One of the main hazards to the environment and workers is cadmium (Cd). Cadmium exposure in animals mostly occurs through consumption or inhalation. The recent surge in mining activities and unregulated industrialization has led to an increase in environmental cadmium contamination. Plants easily absorb cadmium from the soil, leading to its accumulation in various parts **(Bingham et al., 1975)**. Certain fish species and shellfish, including scallops, oysters, and mussels can also gather cadmium. When fish meal or oyster shell grit is added to animal feed as a calcium supplement, this can pose a serious risk to poultry and other livestock **(Alisauskas et al., 2007; Krishnakumar and Bhat, 2006)**. The respiratory, skeletal, digestive, reproductive, and cardiovascular systems, as well as important organs like the liver and kidneys, are among the systems that are harmed by cadmium's high toxicity **(Jama et al., 2013; Charkiewicz et al., 2023)**. Exposure to Cd increases the production of hydroxyl radicals, hydrogen peroxide, and reactive oxygen species (ROS), which destabilize the balance of calcium and sulfhydryl, enhance peroxidation of lipid peroxidation, and cause oxidative stress and destruction of DNA **(Sevcikova et al., 2011; Ebrahimi et al., 2023)**. These toxic effects can impair performance, alter hematology, and negatively impact serum biochemical parameters, causing damage to the kidneys, liver, and bursa of Fabricius.

The use of antioxidants as feed additives has grown in popularity because they shield cells from the harmful effects of xenobiotics, toxicants, medications, and carcinogens **(Akter et al., 2008;**

Zeweil et al., 2013; Nagori et al., 2023). These natural antioxidants are often used as preventive measures to counteract toxic substances that lead to peroxidation **(Aboubakr et al., 2014; Abd El-Hack et al., 2018, 2021, 2023; Abdel Baset et al., 2020; El-Shall et al., 2020; Hussein et al., 2020)**. However, herbs, though rich in active compounds, should be used with caution as they can cause side effects or interact with other treatments and should be administered under the guidance of a botanical medicine specialist. Herbs, spices, and various plant extracts have shown potential as alternatives to antibiotic growth promoters in animal diets.

Phytogenic plants serve as excellent sources of antioxidants and are generally safe for organisms and the environment. However, their phytogenic content can vary widely depending on factors such as processing methods, botanical origin, agricultural practices, and environmental conditions **(Windisch et al., 2008)**. **Wu et al. (2009)** noted that the milk thistle plant (**Silybum marianum**) contains various phytochemicals, including silymarin, which comprises the flavanolignans silybin, silydianin, and silychristine, with silybin being the most biologically active. Silymarin is most concentrated in the fruit but is also present in the leaves and seeds. The active component, silymarin, constitutes about 4% of the dried seeds or aerial parts of the milk thistle plant **(Pradhan and Girish, 2006; Rajiha, 2012)**. **Kshirsagar et al. (2013)** demonstrated that silymarin is an effective antioxidant, capable of scavenging reactive oxygen species (ROS) and inhibiting lipid peroxidation, thereby protecting cells from ROS-induced damage.

Given these findings, chickens raised in field conditions might be exposed to cadmium as an environmental pollutant, leading to various toxic effects and increased cadmium accumulation in their bodies, which could also pose a risk to humans. Therefore, this study aims to explore the toxic effects of cadmium exposure and assess the potential protective effects of milk thistle seed powder.

MATERIALS AND METHODS

The study was carried out experimentally from March to April 2022 at the Poultry Research Laboratory, which is part of the Animal and Fish Production Department of the Faculty of Agriculture (Saba Basha), Alexandria University.

MATERIALS. The source of cadmium chloride was Nasr Pharmaceutical Chemicals Co. in Egypt.

Dehydrated milk Thistle plant seeds were gathered from Abeis village in the El-Behera governorate, and they were processed using an electric dry mill to a fine powder. The powder is kept at room temperature (about 25°C) in tightly sealed black plastic bags until it is needed to create the trial diets.

Experimental design. Two hundred and fifty Japanese quail chicks, growing seven days old without sexing were used. The quail were weighed individually and then divided into 5 experiments, each containing fifty chicks. For each group, five replicates, each containing ten chicks, were created in a totally randomized fashion. First, a basal diet free of additives was provided to the group. In addition to the same basic diet, the second group was given 100 mg of cadmium chloride per kg of diet. The third, fourth, and fifth groups received the basic diet along with 4, 8, and 12 g of Mthsp/kg of diet, respectively. The quail were housed in open-house wire cages for the duration of the six-week testing period, which started at one week of age.

For the whole five-week study, feed and water were freely available. All birds were exposed to 23 hours of continuous light every day during the development season. The experimental period lasted for 16 weeks.

The environmental, sanitary, and administrative settings under which each experimental chick was grown were identical. Table 1 displays the experimental basal diet's composition and chemical analysis.

MEASUREMENTS

Performance evaluation

Weekly measurements of quail body weight, weight gain, and feed intake were taken. The feed conversion ratio was computed as (g feed / g gain).

Hematological and biochemical evaluation

Three birds per treatment group had individual blood samples drawn at six weeks of age in order to assess the various hematological and biochemical traits. After collection, blood samples were split into two equal halves. According to **Hawk et al. (1965)**, the first portion of the blood was drawn using heparin as an anticoagulant (0.1 ml of heparin to 1 ml of blood) in order to calculate the total leukocyte count (WBC) in accordance with **Natt and Herrick (1952)**. To measure the differential leukocyte count, blood smears were prepared and stained **(Cook, 1959)**. To separate the blood serum, the second portion of each blood sample was centrifuged for 15 minutes at 4000 rpm. Until analysis, the collected serum was stored frozen at -18 °C. Using the designated kits, serum samples were subjected to biochemical analysis.

Lymphoid organs weight:

At the conclusion of the trial, three birds were chosen at random from each treatment, fasted for a duration of 12 hours, were weighed, had their jugular veins sliced, and were subsequently scalded and defeathered. After meticulously dissecting the carcasses, they were weighed. The following organs were removed, weighed individually, and their weights were reported as a proportion of the starting weight: the spleen, bursa of Fabricius, and thymus.

Statistical analysis.

One-way ANOVA was used to statistically evaluate the differences between the treatments using the **SPSS® (2007)** statistical software package for Windows version 23.0. Using Duncan's Multiple Range Test **(Duncan, 1955)**, the important variations in treatment means were identified.

RESULTS AND DISCUSSION

Growth performance

Live body weight and body weight gain:

The effects on the live BW and BW gain of developing Japanese quail of both pure cadmium poisoning and cadmium poisoning in combination with a milk thistle plant seed powder (Mthsp) food supplement are displayed in (Table 2).

Table 2 shows that the experimental birds were randomly distributed among the treatments and that there were no significant differences in the birds' initial body weights across treatments.

Up to one weeks of age, the results indicated no significant effect of the various treatments; however, throughout the entire experimental period, body Throughout the entire experimental period, from 1-3, 3-6, and 1-6 weeks of age, there was a substantial effect on body weight gain, and there were significant ($P \leq 0.01$) variations in body weight. When T2 received supplemental dietary cadmium, their body weight decreased by 21% between the ages of 2 and 6 weeks in comparison to the control group, which did not receive any supplements.

Nutrient availability determines how much weight can be gained. Thus, the observed decrease in weight gain may be explained by a reduced feed intake or by an accelerated breakdown of lipids and proteins due to cadmium poisoning (Erdogan et al., 2005). Cadmium toxicity's effects on intestinal absorption can lead to weight loss **(Graef, 1994). Sant'Ana et al. (2005)** linked the significant reduction in body weight seen in growing Japanese quail given 100 ppm cadmium chloride for 28 days to the role of metallothionein's (MT). MTs help retain cadmium in tissues, acting as a protective mechanism, but they also contribute to cadmium-induced hepatic toxicity. The weight loss observed in our study due to cadmium exposure could result from cadmium's widespread toxic effects on the birds' bodily processes. The results that chronic cadmium exposure has been found to induce oxidative stress in the kidneys and liver **(Yiin et al., 1999)** and to change enzymes involved in energy metabolism and glycogenesis in the liver and muscles **(Tourry et al., 1985)** provide weight to this notion. Additionally, according to **Jarup et al. (1998)**, prolonged exposure to cadmium can exacerbate the advancement of leukemia, hypertension, osteoporosis, and diabetic renal problems in both humans and animals. Cadmium exposure may also cause an increase in body weight loss in those animals. Additionally, cadmium may reduce body weight by impacting nutrient absorption in the small intestine or by causing renal tubule dysfunction that disrupts nutrient metabolism **(Abaza, 1996)**. Due to competition between iron and cadmium at the intestinal iron transport system binding sites, prolonged exposure to dietary cadmium can cause iron deficiency anemia **(Schafer and Forth, 1985)**.

As reported by **Abdo** and **Abdulla (2013)**, chickens that received 10 mg/l of Cd in their drinking water grew at significantly slower rates than the control group, which resulted in significantly smaller final body weights. While supplementation did not fully restore these features to normal levels, adding Mthsp at varied amounts to the diets of quail intoxicated with cadmium enhanced BW at 6 weeks of age and BW gain over the course of the whole trial period, significantly ($P < 0.01$). Though levels did not revert to those of the control group, Comparing the group receiving only cadmium, the CdCl2-intoxicated quails treated with 12 g Mthsp significantly ($P<0.05$) increased their body weight and weight gain.

These results imply that adding Mthsp to the diet may help counteract the weight growth and decrease in body weight caused by cadmium, hence reducing its negative effects. However, there was no improvement in these negative effects observed in the group fed a diet containing 2g Mthsp/kg. In contrast, compared to other experimental feed additives employed in this study, 4, 8, and 12g Mthsp/kg diet were more successful in reducing the detrimental impacts on weight and weight gain.

Dietary *Silybum marianum* extract was found to boost both daily weight gain and final body weight under high temperatures, as verified by **Zarei et al. (2016)**. Additionally, **Chand et al. (2011)** discovered that supplementing broiler hens with milk thistle led to a considerable increase in body weight gain. In a similar vein, **Muhammad et al. (2012)** found that supplementing broiler chicks with milk thistle (10 g/kg diet) greatly increased their body weight gain. According to **Tedesco et al. (2004) and Gowda and Sastry**

(2000), introducing silymarin phytosome® at 600 mg/kg feed to lessen aflatoxin B1 toxicity caused an increase in body weight. **Chakarverty and Parsad (1991)** similarly observed increased weight gain in a group that used milk thistle supplements.

According to **Khaleghipour et al. (2019)**, from 7 to 35 days, birds fed diets supplemented with 1000 mg silymarin/kg showed a daily weight growth of 0.4g, or 6.35%, more than control quails (p<.05). **Sureshkumar et al. (2022)** have reported a linear rise in BWG with increasing dietary silymarin supplementation from 0% to 0.06% from days 7 to 21 (p = 0.013) and throughout the entire period (p = 0.043). But during the course of the experiment, there was no discernible impact on FI, FCR, or mortality rate (%).

The exact process underlying the increased body weight gain is unclear, although it's possible that Silybum marianum-treated birds boosted immune systems (IgG and IgM) are to blame.

Feed intake and feed conversion ratio:

The influence of the poisoning of cadmium and varying dosages of Mthsp supplementation on the consumption of feed and the FCR in growing Japanese quail are displayed in Table2. At 1-3, 3-6, and 1-6 weeks of age, there was a significant (P < 0.001 and 0.01) difference in feed intake depending on the level of Mthsp supplementation. Over the course of the trial, dietary cadmium supplementation increased feed intake by 1% and significantly (P < 0.001) improved FCR relative to the control group by 30.6%. During the 1–6-week of age period, quail fed 2 g of milk thistle seed powder per kg meal showed a substantial decrease in feed intake relative to the other treatment groups.

The cadmium group showed a considerable reduction in feed consumption when compared with the control cohort; nevertheless, our findings differ from those of **Cinar et al. (2011)**. Over six weeks, **Erdogan et al. (2005)** found that cadmium at a rate of 25 mg/L significantly decreased feed efficiency (FE), although feed consumption (FC) was not significantly impacted. More recently, **Olgun (2015)** found that feed intake was reduced with increasing supplementation of dietary cadmium (20, 40, and 80 mg/kg) but had no effect at doses as high as 10 mg/kg. On the other hand, certain research has demonstrated that modest dietary supplements of water (1.5 mg/L) or cadmium (3 mg/kg) improved performance metrics **(Pribilincova and Marettova, 1996; Toman et al., 2005)**.

Adding Mthsp to diets of quail intoxicated with cadmium led to a non-significant (P ≤ 0.05) increase in feed consumption at the age of six weeks and FCR for the duration of the trial. These findings are in line with those published by **Khaleghipour et al. (2019)**, who discovered that, in quails fed diets containing 2000 mg silymarin/kg for a period of 7–35 days, the FCR decreased by 0.22 units (6.6%) and daily weight growth rose by 0.4 g (6.35%).

According to **Zarei et al. (2016)**, feed conversion at high temperatures was enhanced by dietary feeding Silybum marianum extract. According to **Chand et al. (2011)**, supplementing broilers with milk thistle greatly increased their FCR. In a similar vein, **Muhammad et al. (2012)** found that supplementing with milk thistle at a rate of 10 g/kg diet greatly enhanced feed intake and enhanced feed conversion rate. Other researchers that fed milk thistle to broiler chickens indicated good effects also reported the same results. On the other hand, **Sureshkumar et al. (2022)** discovered that during the whole trial, dietary silymarin supplementation had no discernible impact on FI or FCR.

Hematological parameters Hematology profile:

The hematological parameters in animals serve as a crucial indicator of their physiological or pathophysiological state **(Khan and Zafar, 2005)**. Both people and animals' hematopoietic systems and hematological profiles may be altered or damaged as a result of heavy metal exposure **(Costa et al., 2004)**.

The findings shown in Table 3 demonstrate that, in comparison to the control group, which did not receive a cadmium supplement, cadmium exposure considerably (P < 0.001) raised white blood cell (WBC) counts and significantly (P ≤ 0.001) decreased hemoglobin (HGB) levels. Additionally, there was a nonsignificant drop in the packed cell volume (PCV) and red blood cell (RBC) counts. Except for eosinophils, which significantly (P ≤ 0.05) decreased in cadmium-intoxicated groups, Table 3 shows that the percentages of lymphocytes, heterophils, monocytes, and the H/L ratio were not significantly affected by cadmium or different levels of milk thistle seed powder compared to the control group. The additives did not influence on the eosinophils' toxicity caused by cadmium. These hematological results imply that quail exposed to cadmium developed macrocytic hyperchromic anemia, except for eosinophils, which were considerably (P < 0.05) reduced in the cadmium-intoxicated groups as compared to the control group. The various doses of milk thistle seed powder were unable to protect eosinophils from the harmful effects of cadmium. However, the hematological results of this investigation indicate that quail treated with cadmium may have acquired macrocytic hyperchromic anemia.

Al-Hamdany (2010) observed comparable results, indicating a substantial elevation in white blood cell counts in rats subjected to cadmium chloride exposure, which was ascribed to inflammation and increased production. The accumulation of metal within red blood cells is probably the cause of the notable drop in hemoglobin, which may be caused by an inhibition of the ferrochelatase enzyme, which oversees attaching iron to the globin protein. Rats given heavy metal treatments also showed elevated WBC counts, which may be the consequence of an inflammatory reaction acting as a defense mechanism, according to studies by **Ekanem et al. (2015) and Sharaf et al. (2017)**.

Following a 6-week exposure to greater cadmium concentrations (100 mg/kg), **Szilagyi et al. (1994)** detected considerably lower WBC and RBC counts in chickens. Comparably, in hens fed drinking water containing 10 mg of cadmium/L daily for 30 days, **Abdo and Abdulla (2013)** showed substantial declines ($P \leq 0.05$) in hemoglobin, hematocrit, and total erythrocyte count. According to **Wintrobe (1978)**, cadmium-treated hens may have had their hematological parameters reduced as a result of mature RBCs being destroyed and erythrocyte formation being inhibited—a process that is impacted by pollution.

According to **Khangarot and Tripathi (1991)**, hematopathology or acute hemolytic crisis may be connected to a drop in red blood cell count. This could result in severe anemia in a variety of vertebrates, including chicken species, that are exposed to diverse environmental contaminants. Moreover, **James et al. (1992)** suggested that the loss of red blood cells (RBCs) could result in severe anemia due to a decline in growth and other utilization of food measures. By reducing the quantity of iron absorbed from the digestive tract, cadmium may also inhibit the synthesis of heme, claim **El-Sharkawy and El-Nisr (2012)**.

After a month of treatment, **Abdo and Abdulla (2013)** observed substantial reductions ($P<0.05$) in the RBC count, hemoglobin content, and hematocrit value in chickens treated with cadmium when compared to controls. Higher dosages of cadmium significantly decreased WBCs, RBCs, HGB, and PCV in comparison to the control birds, according to recent research by **Andjelkovic et al. (2019)**.

Our findings align with previous research that employed distinct animal models, exposure pathways, and dosage schedules, all of which witnessed declines in RBC, HGB, and HCT **(El-Boshy et al., 2017; Abdou and Hassan, 2014; Mladenović et al., 2014; Sharma et al., 2010, 2011)**.

In this study, quail exposed to cadmium without treatment exhibited larger erythrocytes on average than healthy controls, which were normally loaded with hemoglobin. This suggests regenerative anemia due to intense hemolysis, rather than iron deficiency, which would result in smaller erythrocytes with less hemoglobin. In a similar vein, **Gross and Siegel (1983)** showed that the heterophil / lymphocyte (H/L) ratio is a valid measure of immunological activity connected to heat stress in laying hens. According to **Nicol et al. (2009) and Cravener et al. (1992)**, physical and physiological stressors cause a rise in the H/L ratio and a decrease in hematocrit levels in birds that are subjected to heat stress. These findings may also be used as a guide for hen welfare.

According to **Abdalla et al. (2018)**, meals supplemented with silymarin increased phagocytic activity, the phagocytic index, the H/L ratio, and lymphocyte numbers significantly when compared to controls. These results are consistent with a large body of clinical research and trials conducted on cattle, which has demonstrated that supplementing with silymarin diet can improve health and productive performance, especially in relation to hepatic function **(Quarantelli et al., 2004)**. Moreover, silibinin has been reported by **Das et al. (2008) and Kshirsagar et al. (2009)** to possess membrane-protective qualities and to potentially shield blood components from oxidative damage. Silymarin functions as a powerful antioxidant, scavenging free radicals (reactive oxygen species) and preventing lipid peroxidation, according to **Bhattachrya (2011) and Kshirsagar et al. (2013)**.

High concentrations of antioxidants, including silibinin, silidianin, and silichristin, have been found in the popular herbal remedy Silybum marianum, according to **Borges et al. (2018)**. In addition to being utilized to treat liver disease and cancer, this antioxidant property has been widely used to reduce the potentially dangerous in vivo effects of numerous oxidative agents. Previous research has also examined Silybum marianum's ability to prevent cadmium-induced DNA damage in human blood cells.

Antioxidative parameters

In Table 2, blood serum lipid peroxidation, total antioxidant capacity, and catalase are compared to the control group for chicks that were cadmium-intoxicated and quail that were co-administered cadmium with varied amounts of milk thistle seed powder (Mthsp). When quail were exposed to cadmium, their serum catalase levels decreased numerically, and their serum total antioxidant capacity decreased significantly ($P < 0.05$). Comparing the experimental group to the control group did not result in a significant ($P < 0.01$) increase in malondialdehyde (MDA), an indication of lipid peroxidation and oxidative stress.

Cadmium has been shown to induce oxidative stress and observable alterations of membranes in the central nervous system. Decreased activity of acetylcholinesterase , increased oxidative stress markers, and decreased levels of antioxidants such as glutathione, catalase, glutathione-S-transferase, superoxide dismutase 2, and glutathione are some of these indications **(Shagirtha et al., 2011)**. The activation of calcium/calmodulin-dependent protein kinase II may have resulted in the apparent death of cortical cells in the central nervous system as a result of these changes **(Chen et al., 2011)**. Cadmium can also block the entry of calcium through calcium channels **(Bodereau-Dubois et al., 2012)**.

Conversely, as previously reported **(Erdogan et al., 2005; Hudecova and Ginter, 1992)**, Cadmium-induced lipid peroxidation was significantly ($P < 0.01$) decreased when ascorbic acid and/or dried garlic powder were included into the diet. Serum MDA concentrations significantly decreased ($P < 0.01$) to support this. When compared to birds that had been intoxicated with cadmium, there was no significant ($P \leq 0.05$) decrease in the overall antioxidant capacity or catalase activity.

Cadmium-intoxicated quail were shown to have a significant ($P \leq 0.05$) drop in catalase but a nonsignificant decrease in serum total antioxidant capacity, according to recent observations by **Dosoky et al. (2024)**. On the other hand, MDA, a marker of oxidative stress, was much ($P < 0.01$) higher in the experimental group than in the control group.

Likewise, **Hashem et al. (2019)** discovered that quail exposed to cadmium poisoning had a considerable rise in MDA level (3.9±0.09 and 51.33±0.88, respectively), but there was also a significant drop in serum GPX activity (0.05±0.005) when compared to the control group (0.48±0.017).

This imbalance between oxidant and antioxidant actions may be due to cadmium's effects or the release of ROS, NO, and H2O **(Li et al., 2013; Liu et al., 2015; Eriyamremu et al., 2008)**. Cadmium exposure has also been connected to decreased GPX levels and increased renal lipid peroxidation, as seen in hens **(Yang et al., 2012)**. According to **Kant et al. (2011)**, reduced blood glutathione levels is possibly brought on by more oxidative stress and the depletion of GSH reserves, which are important defenses against cadmium toxicity.

Our results are consistent with those of **Pari et al. (2007)**, who observed a significant decline in SOD and CAT activity in groups exposed to cadmium. This is probably because cadmium directly inhibits these enzymes **(Patra et al., 1999; Casalino et al., 2002)**. Due to its increased utilization in combating ROS and lipid peroxidative products, GPx may be depleted **(Singhal et al., 1987)**. Comparably, **Jurczuk et al. (2004)** found that drinking water with 50 mg Cd/l caused a drop in the liver's SOD activity and a rise in the kidney's SOD activity. The drinking water also caused CAT activities to decrease in both the liver and the kidney.

All doses of Mthsp supplementation were associated with higher blood activities of total antioxidant capacity (TAC) and catalase, indicating improved antioxidant status in developing Japanese quail fed diets polluted with cadmium. The groups receiving 12g of powdered milk thistle seed/kg diet had the best TAC values, followed by 8g and 4g, in that order. As a measure of oxidative stress and lipid peroxidation, malondialdehyde (MDA) was nonsignificantly reduced in all treatments when compared to T2 (Table 3). The group supplemented with 12g Mthsp/kg diet had the lowest MDA levels, most likely because silymarin has potent antioxidant effects.

In agreement with this, **Abdalla et al. (2018)** discovered that MDA levels were much lower (between 27.0% and 27.6%) in comparison to the control group, and TAC was significantly raised in a diet supplemented with 25g milk thistle/kg compared to other supplemented groups. Silymarin is a strong antioxidant that lowers blood cholesterol, stimulates the regeneration of liver cells, and helps prevent cancer, according to **Soto et al. (2004)**. According to various clinical research and experiments conducted on cattle, silymarin supplementation can improve health and productive performance, especially with regard to hepatic function **(Quarantelli et al., 2004)**. Our results are in line with these findings. Furthermore, silibinin may protect blood components from oxidative damage and has membrane-protective qualities, according to studies by **Das et al. (2008)** and **Kshirsagar et al. (2009)**. Silymarin functions as a powerful antioxidant, scavenging free radicals, preventing lipid peroxidation, and enhancing the non-enzymatic and enzymatic antioxidant defense systems of cells, such as reduced glutathione, superoxide dismutase, and catalase **(Bhattachrya, 2011; Kshirsagar et al., 2013)**.

Lymphoid organ weights

Table (4) provides a summary of the impact of various treatments on the characteristics of the Lymphoid organ. The findings indicate that there were substantial ($P < 0.001$ or 0.01) alterations in the relative weights of the bursa and spleen in the cadmium-intoxicated group. In contrast to the control group, there was no discernible difference in the relative weights of the bursa, thymus, and spleen. Additionally, the results showed that adding various levels of Mthsp to the cadmium-intoxicated diet led to significant ($P \leq 0.01$) enhancements in most of these parameters. This suggests that dietary supplementation with different levels of Mthsp may help reverse the reductions in absolute and relative lymphoid organ weights induced by cadmium, alleviating the negative effects on these organs of cadmium. Additionally, the different levels of Mthsp tended to reverse the depressive effects of cadmium on the relative weights of the thymus and spleen, making these organs comparable to those in the control group.

Researchers **Abdalla et al. (2018)** demonstrated that groups fed various treatments had considerably higher relative weights of the spleen and bursa when compared to the control group. Among the experimental groups, the group given a meal containing 25g MTh/kg had the highest relative weight of the bursa. But the relative weights of the thymus were unaffected by supplementing with various substances in a meaningful way. **Fani et al. (2013)** observed that supplementing with SMS had no significant effect on spleen weight. According to a recent study by **Khaleghipour et al. (2019)**, birds given diets containing silymarin had greater proportional testis weights and lower percentages of the liver and spleen ($P<0.05$).

Table (1): Composition and calculated analysis of the basal experimental diet.

Ingredients	**%**
Yellow corn	**57.44**
Soybean meal (48 %)	**39.85**
Mono-calcium phosphate	**0.820**
Limestone	**0.880**
Sunflower oil	**0.100**
Vit. and min. mix. *	**0.400**
Salt (NaCl)	**0.400**
L-Lysine HCl	**0.020**
DL-Methionine	**0.090**
Total	**100**
Calculated analyses[1]:	
Crude protein, %	**24.05**
ME (Kcal/ Kg diet)	**2907.10**
Ether extract, %	**2.44**
Crude fiber, %	**3.63**
Methionine, %	**0.76**
Methionine + cystine, %	**0.88**
Lysine, %	**1.42**
Calcium, %	**1.11**
Av. Phosphorus, %	**0.39**

* Each kg of vitamin and minerals mixture contained: Vit. A, 4,000,000 IU; Vit. D_3, 500,000 IU; Vit, E, 16.7 g., Vit. K, 0.67 g., Vit. B1, 0.67 g., Vit. B2, 2 g., Vit. B 6, .67 g., Vit. B12, 0.004 g., Nicotinic acid, 16.7 g., Pantothenic acid, 6.67 g., Biotin, 0.07 g., Folic acid, 1.67 g., Choline chloride, 400 g., Zn, 23.3 g., Mn, 10 g., Fe, 25 g., Cu,1.67 g., I, 0.25 g.,Se, 0.033 g. and,Mg, 133.4 g.

[1] According to NRC (1994).

Table (2): Effect of milk thistle plant seed powder (Mthsp) on growth performance of Japanese quail exposed to cadmium toxicity during the period from 1 to 6 weeks of age

		Dietary treatments*					Items
***P* value**	**MSE**	**T5**	**T4**	**T3**	**T2**	**T1**	
				Live body weight (g/bird/period)			
1.000	**0.200**	**32.57**	**32.58**	**32.60**	**32.64**	**32.63**	**7 days**
0.0001	**0.919**	**88.73^{bc}**	**87.01^{bc}**	**85.76^{c}**	**91.71^{b}**	**107.90^{a}**	**21days**
0.0001	**1.549**	**180.13^{b}**	**170.05^{c}**	**161.26^{d}**	**164.40^{cd}**	**208.18^{a}**	**42days**
				Body weight gain (g/bird/period)			
0.0001	**0.886**	**56.16b^{c}**	**54.42b^{c}**	**53.15^{c}**	**59.07^{b}**	**75.27^{a}**	**1-3 Weeks**
0.0001	**1.311**	**91.02^{b}**	**83.04^{c}**	**75.51^{d}**	**72.69^{d}**	**100.28^{a}**	**3-6 Weeks**
0.0001	**1.520**	**147.18^{b}**	**137.47^{c}**	**128.66^{d}**	**131.76^{cd}**	**175.55^{a}**	**1-6 Weeks**
				Feed consumption (g/bird/ period)			
0.0001	**2.314**	**162.40^{b}**	**163.06^{b}**	**158.84b**	**184.04^{a}**	**155.60^{b}**	**1-3 Weeks**
0.003	**3.885**	**295.60^{a}**	**285.74^{ab}**	**259.02^{c}**	**273.04b^{c}**	**295.90^{a}**	**3-6 Weeks**
0.008	**4.263**	**458.00^{a}**	**448.80^{a}**	**417.86^{b}**	**457.08^{a}**	**451.50^{a}**	**1-6 Weeks**
				Feed conversion ratio			
0.0001	**0.084**	**2.90^{a}**	**3.02^{a}**	**3.00^{a}**	**3.15^{a}**	**2.07^{b}**	**1-3 Weeks**
0.025	**0.088**	**3.26^{ab}**	**3.46^{ab}**	**3.51^{a}**	**3.80^{a}**	**2.95^{b}**	**3-6 Weeks**
0.0001	**0.071**	**3.18^{a}**	**3.32^{a}**	**3.33^{a}**	**3.33^{a}**	**2.55^{b}**	**1-6 Weeks**

*Cd: Cadmium chloride, Mthsp: milk thistle plant seed powder, T1: Control, T2: Control+100 mg Cd /kg diet, T3: Control+ 100 mg Cd /kg diet + 4g Mthsp /kg diet, T4: Control+ 100 mg Cd /kg diet + 8g Mthsp /kg diet, T5: Control+ 100 mg Cd /kg diet+ 12g Mthsp /kg diet.
Different lower case letters indicate significant differences ($P<0.05$).

Table (3): Effect of Effect of milk thistle plant seed powder (Mthsp) on hematological and antioxidative parameters of Japanese quail exposed to cadmium toxicity at 6 weeks of age

		Dietary treatments*					Items
***P* value**	**MSE**	**T5**	**T4**	**T3**	**T2**	**T1**	
				Hematological parameters			
0.002	**0.222**	**20.90^{ab}**	**20.13^{bc}**	**20.21^{bc}**	**21.89^{a}**	**19.43^{c}**	**White blood cells (WBCs10^{3}/m^{3})**
0.092	**0.124**	**3.02**	**2.97**	**2.88**	**2.86**	**3.24**	**Red blood cells (RBCs10^{6}/mm^{3})**
0.001	**0.124**	**10.38^{b}**	**10.28^{b}**	**10.22^{b}**	**10.08^{b}**	**11.44^{a}**	**Hemoglobin (Hb g/dl)**
0.927	**0.124**	**42.74**	**42.22**	**43.5**	**42.46**	**44.18**	**Packed cell volume (PCV %)**
1.000	**0.894**	**62.6**	**63.6**	**63.4**	**66.2**	**59.2**	**Lymphocytes%**
0.816	**0.865**	**31.00**	**29.40**	**29.60**	**28.00**	**31.00**	**Heterophils%**
0.787	**0.021**	**0.498**	**0.475**	**0.468**	**0.448**	**0.530**	**H/L ratio**
0.122	**0.244**	**4.40**	**4.60**	**4.60**	**3.80**	**5.80**	**Monocytes%**
0.046	**0.247**	**2.00^{b}**	**2.40^{b}**	**2.40^{b}**	**2.00^{b}**	**4.00^{a}**	**Eosinophils%**
				Antioxidative parameters			
0.001	**0.001**	**0.426^{ab}**	**0.425^{ab}**	**0.422^{bc}**	**0.417^{c}**	**0.431^{a}**	**Total antioxidant capacity (mg/dl)**
0.126	**4.47**	**391.56**	**397.17**	**374.41**	**373.77**	**402.71**	**Catalase (mg/dl)**
0.203	**0.134**	**10.35**	**10.43**	**10.52**	**11.08**	**10.10**	**Malondialdehyde MDA (nmol/ml)**

*Cd: Cadmium chloride, Mthsp: milk thistle plant seed powder, T1: Control, T2: Control+100 mg Cd /kg diet, T3: Control+ 100 mg Cd /kg diet + 4g Mthsp /kg diet, T4: Control+ 100 mg Cd /kg diet + 8g Mthsp /kg diet, T5: Control+ 100 mg Cd /kg diet+ 12g Mthsp /kg diet.
Different lower case letters indicate significant differences (P<0.05).

Table (4): Effect of milk thistle plant seed powder (Mthsp) on lymphoid organ weights%of Japanese quail exposed to cadmium toxicity at 6 weeks of age

Items	Dietary treatments*						
	T1	**T2**	**T3**	**T4**	**T5**	**MSE**	***P* value**
Spleen	**0.060^{b}**	**0.310^{a}**	**0.070^{b}**	**0.070^{b}**	**0.110^{b}**	**0.028**	**0.006**
Bursa	**0.210**	**0.190**	**0.140**	**0.160**	**0.140**	**0.019**	**0.766**
Thymus	**0.150^{c}**	**0.300^{a}**	**0.210^{b}**	**0.210^{b}**	**0.230^{b}**	**0.012**	**0.001**

*Cd: Cadmium chloride, Mthsp: milk thistle plant seed powder, T1: Control, T2: Control+100 mg Cd /kg diet, T3: Control+ 100 mg Cd /kg diet + 4g Mthsp /kg diet, T4: Control+ 100 mg Cd /kg diet + 8g Mthsp /kg diet, T5: Control+ 100 mg Cd /kg diet+ 12g Mthsp /kg diet.
Different lower case letters indicate significant differences (P<0.05

REFERENCES

Abaza, M. (1996). Cadmium toxicity in broiler chickens. Egyptian Poultry Science., 16 (2): 347-364.

Abd El-Hack, M.E., B.A. Alaidaroos, R.M. Farsi, D.E. Abou-Kassem, M.T. El-Saadony, A.M.Saad, M.E. Shafi, N.M. Albaqami, A.E. Taha, E.A. Ashour. (2021). Impacts of supplementing broiler diets with biological curcumin, zinc nanoparticles and bacillus licheniformis on growth, carcass traits, blood indices, meat quality and cecal microbial load. Animals, 11(7), 1878.

Abd El-Hack, M.E., D.H. Samak, A.E. Noreldin, K. El-Naggar, M. Abdo. (2018). Probiotics and plant-derived compounds as eco-friendly agents to inhibit microbial toxins in poultry feed: a comprehensive review. Environ. Sci. Poll. Res. 9 25(32): 31971-31986.

Abd El-Hack, M.E., M. Kamal, A.H. Alqhtani, R.M. Alreemi, R.S. Alazragi, H. Khojah, A.A. Swelum, A.F. Khafaga, S. Świątkiewicz. (2023). Detoxification impacts of dietary probiotic and prebiotic supplements against aflatoxins: an updated knowledge. Ann. Anim. Sci., 23(4): 1049 – 1060.

Abdalla, A. A., B. M. Abou-Shehema, Rawia, S. Hamed, and M. R. Elden (2018). Effect of silymarin supplementation on the performance of developed chickens under summer conditions. Egypt.poult.sci.vol (38) (i): (305-329).

Abdel Baset, S., E.A. Ashour, M.E. Abd El-Hack, M.M. El-Mekkawy. (2020). Effect of different levels of pomegranate peel powder and probiotic supplementation on growth, carcass traits, blood indices, antioxidant status and meat quality of broilers. Anim. Biotech., 33(4), 690-700.

Abdo, K .S . and Huda, A . Abdulla (2013). Effect of cadmium in drinking water on growth, some haematological and biochemical parameters of chicken. uropean Journal of Experimental Biology, 2013, 3(5):287-291.

Abdou, H.M., and M.A. Hassan (2014). Protective role of omega-3 polyunsaturated fatty acid against lead acetate-induced toxicity in liver and kidney of female rats. BioMed Res. Int, 2014, 435857.

Aboubakr, M., M. EL-Badawy, A. Soliman, and M. El-Hewaity. (2014). Embryotoxic and teratogenic effects of norfloxacin in pregnant female albino rats. Adv Pharmacol Sci. Article ID 924706, 6 pages.

Adham, K.G., I.M. Farousia, B. Mahmoud, A. Hassan, E. Wafaa and S.M. Salwa (2001). Interaction between lead poisoning and dietary calcium in rat. Alex. J. Vet. Sci, 17(1): 183-199.

Adnan, M., B. Xiao, P. Xiao, P. Zhao, and S. Bibi. (2022). Heavy metal, waste, COVID-19, and rapid industrialization in this modern era—fit for sustainable future. Sustainability, 14(8), 4746.

Akhere, A., F.O. Omonkhua and P. Obi (2008). Evaluation of the effects of vitamin C in rats exposed to sub-chronic low doses of cadmium, Int. J. Toxicol., pp: 1559-3916.

Al-Hamdany, A.S. (2010). The effect of lead acetate on histological structure of liver, kidney, spleen and some blood parameters in the white rats Rattus rattus. Msc Degree. Dept. biology, Univ. of Babylon. Iraq.

Alisauskas, R. T., D. Kellett, J. Traylor, C. Swoboda, and E. Neugebauer. (2007). Year-to-year correlations in blood metal levels among individuals of two species of North American sea ducks. Environ Pollut. 150:329 337.

Andjelkovic, M., A. B. Djordjevic, E. Antonijevic, B. Antonijevic, M. Stanic, J. Kotur-Stevuljevic , V.S. Kalimanovska, M. Jovanovic, N. Boricic, D. Wallace and Z. Bulat (2019). Toxic Effect of Acute Cadmium and Lead Exposure in Rat Blood, Liver, and Kidney. Int. J. Environ. Res. Public Health 2019, 16, 274; doi:10.3390/ijerph16020274.

Bhatti, N., Z. Hussain, M. Mukhtar, A. Ali, I. Muhammad, R Asim, M. Sohail, and R. Saad (2016). Effects of Vitamins E and C Supplementation on the Immune Response of Broiler Chicks.J .Antivir. 8(4): 151-154.

Bhattacharya, S. (2011). Milk thistle (*Silybum marianum* L. Gaert.) seeds in health. In: Preedy V.R., Watson R.R., Patel V. Editors. Nuts and Seeds in Health and Disease Prevention. 1st Ed., London, Burlington, San Diego: Academic Press (an imprint of Elsevier).

Bingham, F. T., Page, A. L., Mahler, R. J. and Ganje, T. J. (1975). Growth and cadmium accumulation of plants grown on a soil treated with cadmium-enriched sewage sludge. J. Environ. Qual. 4: 207–211.

Bodereau-Dubois, B., O. List, D. Calas-List , D. Marques , O. Communal , P.Y. Thany, and B . Lapied (2012). Transmembrane potential polarization, calcium influx, and receptor conformational state modulate the sensitivity of the imidacloprid-insensitive neuronal insect nicotinic acetylcholine receptor to neonicotinoid insecticides, Journal of Pharmacology and Experimental Therapeutics, Vol. 341 (2): 326–339.

Borges, F.F.V.; e Silva, C.R.; Goes,W.M.; Godoy, F.R.; Franco, F.C.; Véras, J.H.; Bailão, E.F.L.C.; e Silva, D.d.M.; Cardoso, C.G.; da Cruz, A.D(2018). Protective effects of silymarin and silibinin against DNA damage in human blood cells. BioMed Res. Int. 2018, 2018, 6056948. [CrossRef]].

Casalino, E., G. Calzaretti, C. Sblano, and C. Landriscina (2002). "Molecular inhibitory mechanism of antioxidant enzymes in rat liver and kidney by cadmium." Toxicology, 179(1-2): 37-50.

Chakarverty, A. and J. Parsad (1991). Study on the effect of Neem leaf extract and Neem and cake extract on the performance of broiler chicks. Indian Poult. Advisor. 24(9), 37-38.

Chand, N., F. R. Durrani, Q. M. S. ureshi, D. Muhammad, and Z. Rehman (2011). Protective effects of milk thistle (Silybum marianum) against aflatoxin B1 in broiler chicks. Asian- Australas. J. Anim. Sci. 24(7), 1011-1018.

Chen, S., Y. Xu, B. Xu, and M. Guo (2011). CaMKII is involved in cadmium activation of MAPK and mTOR pathways leading to neuronal cell death, Journal of Neurochemistry, Vol. 119 (5): 1108–1118.

Charkiewicz, A. E., W. J. Omeljaniuk, K. Nowak, M. Garley, and J. Nikliński. (2023). Cadmium toxicity and health effects-a brief summary. Molecules, 28(18), 6620.

Cinar, M., A. A. Yigit, I. Yalcinkaya, E. Oruc, O. Duru and M. Arslan (2011). Cadmium Induced Changes on Growth Performance, Some Biochemical Parameters and Tissue in Broilers: Effects of Vitamin C and Vitamin E. Asian Journal of Animal and Veterinary Advances, 6: 923-934.

Cook, F.W. (1959). Staining fixed preparation of chicken blood cells with combinations of May-Gru¨nwald-Wright-Phyloxine B stain. Avian Disease, 3: 272–290.

Costa, L.G., M. Aschner, A. Vitalone, T. Syversen and O.P. Soldin (2004). Developmental Neuropathology of Environmental Animals. Annu. Rev. Pharmacol. Toxicol. 44: 87-110.

Cravener, T.L., W.B. Roush, and M.M., Mashaly (1992). Broiler production under varying population densities. Poult. Sci, 71: 427—433.

Das, S.K., S. Mukherjee, and D.M. Vasudevan (2008). Medicinal properties of milk thistle with special reference to silymarin: An overview. Nat. Prod. Rad.; 7: 182-192.

Dosoky, W.M., Farag, A. Soha, A. A. Almuraee , I. M. Youssef, O. F.A. Awlya, Abusudah, F. Wafaa, A. Qadhi , A. F. Arbaeen , M. Moustafa , H.Hassan , and G. Tellez-Isaias (2024). Vitamin C and/or garlic can antagonize the toxic effects of cadmium on growth performance, hematological, and immunological parameters of growing Japanese quail, *Poultry Science* (2024), Doi: https://doi.org/10.1016/j.psj.2024.103457.

Duncan, D.B. (1955). Multiple Range and Multiple F-test. Biometrics., 11: 1-42.

Ebrahimi, R., M. Ebrahimi, and M. Shakeri. (2023). Mitigating the adverse effects of lead and cadmium heavy metals-induced oxidative stress by phytogenic compounds in poultry. Poult. Sci., 2(2), 235-251.

Ekanem, A.U., H.D. Kwari, S.H. Garba and H.A. Salami (2015) . Effect of lead acetate on spleen and blood parameters in albino rats. Journal of Dental and Medical Sciences, 14(3): 43-49.

El-Boshy, M., A. Ashshi, M. Gaith, N. Qusty, T. Bokhary, N. AlTaweel, and M. Abdelhady (2017). Studies on the protective effect of the artichoke (Cynara scolymus) leaf extract against cadmium toxicity-induced oxidative stress, hepatorenal damage, and immunosuppressive and hematological disorders in rats. Environ. Sci. Pollut. Res., 24, 12372–12383.

El-Shall, N., A. Awad, M.E. Abd El-Hack, M. Naiel, S.I. Othman, A.A. Allam, M. Sedeik. (2020). The simultaneous administration of a probiotic and prebiotic with live salmonella vaccine improves growth performance and reduces faecal shedding of the bacterium in salmonella challenged broilers. Animals, 10(1), 70.

El-Sharkawy, E. E. and B. N. A. El-Nisr (2012). Lactational cadmium exposure induced alterations in the hematological indices and oxidative status in brain, liver and testes of rat pups. Scientific Journal of Veterinary Advances., 1 (3): 70-81.

Erdogan, Z., S. Erdogan, S. Celik and A. Unlu (2005). Effects of ascorbic acid on cadmium-induced oxidative stress and performance of broilers. Biol. Trace Elem. Res., 104: 19-31.

Eriyamremu, G.E., S.E. Ojimogho, S.O. Asagba, and V.E. Osagie (2008) . Palm oil induced changes in ocular tissue lipid peroxidation, antioxidant enzymes and ATPases of rabbi ts in cadmium toxici ty. Food Chem Toxicol, 46(9): 3155-3158.

Fani, M.O., N. Afzali, and A. Omidi (2013). Effect of Different Levels of Silymarin (*Silybum marianum*) on Growth Rate, Carcass Variables and Liver Morphology of Broiler Chickens Contaminated with Aflatoxin B1. Poultry Science Journal 1: 105-116.

Gowda, S.K. and V. R. B. Sastry (2000). Neem (*Azadirachta indica*) seed cake in animal feeding-scope and limitation: a review. Asian-Australas. J. Anim. Sci. 13, 720-728.

Graef, J. W. (1994). In: Isselbacher (ed.), “Cadmium” in Harrison’s Principles of Internal Medicine, 13th ed., McGraw Hill, NY, pp. 2462–2463.

Gross, W.B., and H.S. Siegel (1983). Evaluation of the heterophil/lymphocyte ratio as a measure of stress in chickens. Avian Dis 27:972–979.

Hashem, M. A., M.I. Gamal El-Din, and Shimaa, N.A. Eltahawy (2019). Clinicopathological Studies on the Ameliorative Effects of Selenium and Vitamin E against Cadmium Toxicity in Chickens. Zag Vet J, Volume 47, Number 3, 277-287.

Hawk, P. B., B. L. Oscar and W. Summerson (1965). Hawk's [physiological chemistry]. London J., and A. Churchill Ltd. 14th Ed.

Hudecova, A., and E. Ginter (1992). The influence of ascorbic acid on lipid peroxidation in guinea pigs intoxicated with cadmium. Food Chem. Toxicol., 30, 1011-1013.

Hussein, E.O.S., S. Ahmed, A.M. Abudabos, G.M. Suliman, M.E. Abd El Hack, A. Swelum, A.N. Alowaimer. (2020). Ameliorative effects of probiotic-supplemented diets on the performance, intestinal health, carcass traits, and meat quality of Clostridium perfringens infected broilers. Animals, 10(4), 669.

Jama, A. M., M. Dragana, and A. Kolarević. (2013). Protective effect of probiotic bacteria against cadmium-induced genotoxicity in rat hepatocytes in vivo and *in vitro*. Arch. Biol. Sci. Belgrade, 64 (3): 1197-1206.

James, R., K. Sampath, V. Jancy Pattu, and J. A. Devakiamma (1992). Utilization of Eichhorina crassipes for the reduction mercury toxicity on food transformation in Heteropneustes fossilis. J. Aqua. Trop., 7 :189- 196.

Jarup, L., M. Berglund, C.G. Elinder, G. Nordberg, and M. Vahter (1998). Health effects of cadmium exposure – A review of the literature and a risk estimate. Scandinavian Journal of Work Environment and Health, 24: 240.

Jurczuk, M., M. M. Brzoska, J. Moniuszko-Jakoniuk, M. Galazyn-Idorczuk and E. Kulikowska-Karpinska (2004). Antioxidant enzymes activity and lipid peroxidation in liver and kidney of rats exposed to cadmıum and ethanol. Food Chem. Toxicol. 42: 429-438.

Kant, V., M. Mehta, C. Varshneya, and S. Chauhan (2011). Induction of oxidative stress by subacute oral exposure of cadmium sulphate in adult poultry .Braz J Vet Pathol, 4(2): 117- 121.

Khaleghipour. B, H. Khosravinia, M. Toghiyani, and A. Azarfar (2019). Effects of silymarin on productive performance, liver function and serum biochemical profile in broiler Japanese quail challenged with dietary aflatoxins, Italian Journal of Animal Science, DOI: 10.1080/1828051X.2018.1548310**.**

Khan, A. T. and F. Zafar (2005). Hematological study in response of varying doses of estrogen in broiler chicken, International Journal of Poultry Science, Vol. 4: 748–751.

Khangarot, B. S. and D. M. Tripathi (1991). Changes in humoral and cell- mediated immune responses and in skin and respiratory surfaces of cat fish Saccobranchus fossillis, following copper exposure. Ecot. Envir. Safety., 22 (3): 291-308.

Krishnakumar, P. K., and G. S. Bhat. (2006). Monitoring trace metal contaminants in green mussel, *Perna viridis* from the coastal waters of Karnataka, southwest coast of India. Arch Environ Contam Toxicol. 51:206–214.

Kshirsagar, A., D. Ingawale, P. Ashok and N. Vyawahare (2009). Silymarin: A comprehensive review. Phcog. Rev. 3: 116-124.

Kshirsagar, M., V. Mahash, P. Srinivas and L. Mangala (2013). Evaluation of the protective effect of silymarene on doxorubicin induced chronic testicular toxicity in Rats. International Journal of Pharmacy and Biological Sciences, 4: 473 – 484.

Li, J.L., C.Y.Jiang, S.Li, and S.W. Xu (2013). Cadmium induced hepatotoxicity in chickens (Gallusdomesticus) and ameliorative effect by selenium. Ecotoxicol Environ Saf, 96: 103-109.

Lippmann, M. (2009). Environmental toxicants: human exposure and their health effect, 3rd Ed. 865-913.

Liu, L., B. Yang , Y. Cheng, and H. Lin (2015). Ameliorative Effects of Selenium on cadmium-Induced Oxidative Stress and Endoplasmic Reticulum Stress in the Chicken Kidney. Biol Trace Elem Res, 167(2): 308-319.

Mladenovi´c, J. B. Ognjanovi´c, N. Dordevi´c, M. Mati´c, V. Kneževi´c, A. Štajn, and Z. Saiˇci´c (2014). Protective effects of oestradiol against cadmium-induced changes in blood parameters and oxidative damage in rats. Arh. Hig. Rada Toksikol. 2014, 65, 37–46.

Muhammad, D. N. Chand S. Khan, A. Sultan, M. Mushtaq and Rafiullah., (2012). Hepatoprotective Role of Milk Thistle (Silybum marianum) in Meat Type Chicken Fed Aflatoxin B1 Contaminated Feed. Pak Vet Jour. February 09,2012.

Nagori, K., K. T. Nakhate, K. Yadav, Ajazuddin, and M. Pradhan. (2023). Unlocking the Therapeutic Potential of Medicinal Plants for Alzheimer's Disease: Preclinical to Clinical Trial Insights. Future pharmacol. 3(4), 877-907.

Natt, M. P. and C. A. Herrick (1952). A new blood diluent for counting erythrocytes and leucocytes of the chicken. Poultry Science, 31, 735–738.

Nicol, C.J., G. Caplen, J; Edgar, and W.J. Browne (2009). Associations between welfare indicators and environmental choice in laying hens. Anim Behav 78:413–424.

Olgun, O. (2015). The effect of dietary cadmium supplementation on performance, egg quality, tibia biomechanical properties and eggshell and bone mineralisation in laying quails, Animal 9 (8): 1298–1303.

Pari, L., P. Murugavel, S.L. Sitasawad, and K. Kumar (2007)."Cytoprotective and antioxidant role of diallyl tetrasulfide on cadmium induced renal injury: An in vivo and in vitro study." Life Sciences, 80(7): 650–658.

Patra, R.C., A.K. Rautray, and D. Swarup (2011). Oxidative stress in lead and cadmium toxicity and its amelioration. Vet. Med. Int.

Patra, R.C., D. Swarup, and S. K.Senapati (1999). Effects of cadmium on lipid peroxides and superoxide dismutase in hepatic, renal and testicular tissue of rats.” Vet Hum Toxicol., 41(2):65-77.

Pradhan, S. C. and C. Girish (2006). Hepatoprotective herbal drug, silymarin from experimental pharmacology to clinical medicine. Indian J. Med. Res. 124 (5): 491- 504.

Pribilincova, J. and E. Marettova (1996). The effect of cadmium on reproductive performance of laying hens and egg quality. Zivocisna Vyroba 41: 57–62.

Quarantelli, A., F. Righi, A. Bonomi, M. Renzi, R. Bruni and A. Schiavone (2004). Impiego della silimarina nell'alimentazione del pollo da carne. Atti. S.I.S.Vet. 58:235-236.

Rajiha, A. (2012). The therapeutic effect of Silybum marianum on the lead acetate induced - reproductive toxicity in both gender laboratory rats. Journal Science and Medicine 5 (1): 144 – 15.

Sant'Ana, M.G., R. Moraes and M.M. Bernardi (2005). Toxicity of cadmium in Japanese quail: Evaluation of body weight, hepatic and renal function and cellular immune response. Environ. Res., 99: 273-277.

Schafer, S.G. and W. Forth (1985). The interaction between cadmium and iron: a review of literature. Trace elements in medicine, 2 (4): 158-162.

Sevcikova, L., A. Pechova, and L. Pavlata. (2011). The effect of various forms of selenium supplied to pregnant goats on the levels of selenium in the body of their kids at the time of weaning. Biol. Trace Elem. Res. 143: 882–892.

Shagirtha, K., M. Muthumani, And S. Miltonprabu (2011). Melatonin abrogates cadmium induced oxidative stress related neurotoxicity in rats. European Review for Medical and Pharmacological Sciences art. 1.1971.

Sharaf, A. M. M., A. H. Farrag and H. M. Fahmy (2017). Protective Effects of Vitamin C on Hematological and Biochemical Parameters of Intoxicated Male Albino Rats with Lead and Cadmium, Middle East Journal of Applied Sciences Vol. 7: 57-67.

Sharma, V., A. Sharma and L. Kansal (2010). The effect of oral administration of Allium sativum extracts on lead nitrate induced toxicity in male mice. Food Chem. Toxicol., 48, 928–936.

Sharma, V., L. Kansal, A. Sharma, S. Lodi, S. Sharma and S. Ameliorating (2011). Effect of Coriandrum sativum extracts on hematological and immunological variables in an animal model of lead intoxication. J. Pharm. Allied Health Serv. 2011, 1, 16–29.

Singhal, R. K., M.E. Anderson, and A. Meister (1987). Glutathione, a first line of defense against cadmium toxicity. FASEB J. 1: 220–223.

Soto, C., R. Mena, J. Luna, M. Cerbon, E. Larrieta, P. Vital, E. Uria, M. Sanchez, R. Recoba, H. Barron, L. Favari, and A. Lara (2004). Silymarin induces recovery of pancreatic function after alloxan damage in rats. Life Sci., 75:2167-2180.

SPSS Statistical, Packages for the Social Sciences (2007). Statistical software for windows version 11.0 Microsoft. SPSS ® , Chicago, IL, USA.

Sureshkumar, Sh., J.H. Park, CH. Sungbo, and I.N. Kim (2022). Silymarin seed extract supplementation enhances the growth performance, meat quality, and nutrients digestibility, and reduces gas emission in broilers. Anim Biosci 35:1215-1222.

Szilagyi, M., J. Bokori, S. Fekete, F. Vetesi, M. Albert and I. Kadar (1994). Effects of long-term aluminum exposure on certain serum constituents in broiler chickens. European Journal of Clinical Chemistry and Clinical Biochemistry, 32:485-486.

Tedesco, D., S. Steidler, S. Galletti, M. Tameni, O. Sonzogni and L. Ravarotto (2004). Efficacy of Silymarin phospholipid complex in reducing the toxicity of aflatoxin B1 in broiler chicks. Poult. Sci. 83(11), 1839-1843.

Toman, R., P. Massanyi, N. Lukac, L. Ducsay and J. Golian (2005). Fertility and content of cadmium in pheasant (*Phasianus colchicus*) following cadmium intake in drinking water. Ecotoxicology and Environmental Safety, 62: 112-117.

Tourry, R., E. Boissonneau, N. Stelly, Y. Dupuis, A. Berville and R.Perasso (1985). Mitochondrial alterations in Cd2 treated rats: general regression of inner membrane criatae and electron transport. Biology of the Cell, 56: 71-86.

Windisch, W., K. Schedle, C. Plitzner and A. Kroismayr (2008). Use of phytogenic products as feed additives for swine and poultry. Journal of Animal Science, 86: 140-148.

Wintrobe, M.M. (1965). Clinical hematology.Lead and Febiger ,Philadelphia ,USA.

Wu, J. W., L. C. Lin and T. H. Tsai (2009). Drug-drug interactions of silymarein on the perspective of pharmacokinetics. J. Ethnopharmacol Ethno Pharm., 121:181- 193.

Yang, S., Z. Zhang, J. He, J. Li, J. Zhang, H. Xing, and S. Xu (2012). Ovarian toxicity induced by dietary cadmium in hen. Biol Trace Elem Res,148(1): 53-60.

Yiin, S.J., C.L. Chern, J.Y. Sheu, W.C. Tseng, and T.H. Lin (1999). Cadmium-induced renal lipid peroxidation in rats and protection by selenium. Journal of Toxicology and Environmental Health, 57:403-413.

Zarei, A. M. Morovat, M. Chamani, and A. A. Swadeshi (2016). Effect of in ovo Feeding and Dietary Feeding of Silybum marianum Extract on Performance, Immunity and Blood Cation-Anion Balance of Broiler Chickens Exposed to High Temperatures. Iranian Journal of Applied Animal Science (2016) 6(3), 697-705.

Zeweil, H.S., S. El-Nagar, S. M. Zahran, M. H. Ahmed and Y. El-Gindy (2013). Pomegranate Peel as a Natural Antioxidant Boosts Bucks' Fertility under Egyptian Summer Conditions. World Rabbit Sci. 2013, 21: 33-39.

الملخص العربى

تأثير إضافة بذور شوك اللبن المجفف علي أداء السمان اليابانى النامى المغذى على عليقة ملوثة بالكادميوم

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أجريت هذه التجربة بإستخدام 250 كتكوت ياباني عمر اسبوع غير مجنس وذلك لبيان تأثير اضافة بذورشوك اللبن المجفف لتقليل تاثير العلائق الملوثة بالكادميوم على الاداء الانتاجى وبعض الصفات الهيماتولوجيه. تم تقسيم السمان إلى خمسه مجموعات تجريبية بكل مجموعة 50 كتكوت تم تقسيمهم في خمسة مكررات بكل مكرره 10 كتاكيت في تصميم عشوائي تام . المجموعة الأولى غذيت على عليقة أساسية بدون أي إضافات وإستخدمت كمجموعة كنترول، المجموعة الثانية غذيت على عليقة أساسية مضافا إليها 100 مللجرام كلوريد الكادميوم / كجم علف، المجموعة الثالثة والرابعة والخامسة غذيت على عليقة أساسية مضافا إليها 12,8,4جرام من بذور شوك اللبن على التوالي. وأوضحت النتائج أن الكادميوم تسبب في انخفاض معنوي ($P \leq 0.001$) في الهيموجلوبين (HGB) وزيادة معنوية ($P \leq 0.001$) في عدد خلايا الدم البيضاء (WBCs) مقارنة بمجموعة الكنترول. بينما لوحظ انخفاض غير معنوي في عدد خلايا الدم الحمراء (RBC)وحجم الخلايا المعبأة. تلوث عليقة السمان بالكادميوم احدث انخفاضا غير معنوي ($P \geq 0.05$) فى نشاط انزيم الكتاليز واحدث انخفاضا معنوي في القدرة الكلية لمضادات الأكسدة في السيرم ، فى حين أن MDA كان مرتفعاً بشكل غير معنوي ($P \leq 0.01$) بالمقارنة بالكنترول. هذه النتائج توضح أن اضافة بذور شوك اللبن الى علائق السمان الملوثة بالكادميوم قد يكون مفيداً في تحسين الأداء، ومقاييس الدم الهيماتولوجية وتخفيف التأثير الضار للكادميوم على مضادات الأكسدة.