INTERNATIONAL JOURNAL OF CURRENT MULTIDISCIPLINARY STUDIES
Available Online at http://www.journalijcms.com
VOL. 2, ISSUE, 6, PP.304-309, JUNE, 2016

IJCMS

REVIEW ARTICLE

TOXIC EFFECTS OF LEAD ON BIRDS - A REVIEW

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ARTICLE INFO

Received 14 th, March, 2016,
Received in revised form 27 th,
April, 2016, Accepted 13 th, May, 2016,
Published online 18th,June , 2016

Keywords:

Lead, toxicity, birds

ABSTRACT

The toxicity of heavy metals depends on various factors, the total dose absorbed, route of exposure and whether the exposure was acute or chronic. The age and sex of the organism can also influence toxicity. Young, growing birds are typically more sensitive to the toxic effects of chronic metal exposure than adults. The findings revealed that lead is virtually ubiquitous, not only being deposited and accumulated in various tissues, but found in varying concentrations in air, soil, food, and water. Exposure to lead produces various deleterious effects on the morphology, body weight, hematopoietic, renal, reproductive system and also imbalance the levels of protein,cholesterol and various enzymes and hormonal concentration.

INTRODUCTION

The tremendous increase in the use of heavy metals over the past few decades has inevitably resulted in an increased flux of metallic substances in the aquatic as well as terrestrial environments. Metals are of special concern because of their diversified effects and the range of concentration stimulated toxic ill effects in different organisms by them. Negative effects of heavy metals are particularly dangerous to birds, whose metabolism is more rapid compared to other groups of animals. They are, therefore, exposed to a greater threat of accumulating heavy metals in their bodies. Today, heavy metals are abundant in our drinking water, air and soil. They are present in virtually every area of modern consumerism-from construction materials to cosmetics, medicines to processed foods, fuel sources to agents of destruction, appliances to personal care products. It is very difficult for anyone to avoid exposure to any of the many harmful heavy metals that are so prevalent in our environment.

Lead is found in the soil, plants and grains grown on contaminated soil, and tissues of animals that eat contaminated plants and feed grains. Because of widespread environmental exposure, low levels of lead can be demonstrated in tissues of clinically normal birds and animals. Lead toxicosis occurs when an animal or a bird inhales or ingests a concentrated source of lead (Doganoc, 1996).

The environmental contamination by lead generated from human activities has become an evident problem during the last decades. Lead can penetrate to the human or animal organisms by inhalation, ingestion and by skin (Ryanand Terry, 1988; El-Feki *et al*., 2000). The adverse effects of lead on health and productivity have been studied relatively little in poultry, particularly in layers, in comparison with other farm animals. Berg *et al.*, (1980) stated that contamination of the environment with lead has reached such a level that can affect the growth, productivity, and health of poultry as the toxicity of these metals (cadmium, lead) relies on binding the metallic cations with sulphydryl, amino and carboxylic groups of enzymes thus inhibiting enzymatic activities and disturbing energy metabolism.

General Effects of Lead on Birds

The birds are vital members of ecosystem and act as important bioindicators of metal contamination. Trainer and Hunt (1965) have observed that lead poisoning works as a mortality factor of wild waterfowl as in Wisconsin (USA) since 1940, more than 1,700 Canadian geese (*Brantacanadensia*) have succumbed to lead poisoning in Wisconsin. Lead salts are only toxic to birds at a high dietary dosage (100 mg/kg or more). Exposure of quail from hatching and up to reproductive age resulted in effects on egg production at dietary lead levels of 10 mg/kg. Although a variety of effects at high dosage have been reported, most can be explained as a primary effect on food consumption.

Diarrhoea and lack of appetite, leading to anorexia and weight loss, are the primary effects of lead salts. Stone and Soares (1976) have noticed highly significant decrease in red blood cell delta-aminolevulinic acid dehydratase (RBC-ALAD), body weight; liver weight and egg production although weight of kidney increased in the Japanase quail fed lead. According to

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Revis *et al.,* (1980) number and size of atherosclerotic plaques in the aorta were increased in pigeons given drinking water containing lead and / or cadmium. Deborah *et al*., (1980) noticed that behavioral changes induced by lead in pigeons cannot be attributed to central nervous system (CNS) dysfunction alone, but may arise from starvation, or from combined CNS damage and starvation and that gastrointestinal (GI) impaction in birds resulting from lead exposure appear to be a systemic rather than a local effect.

The ducklings fed silver, copper, cobalt, tellurium, cadmium and zinc frequently developed lesions characteristic of Se-deficiency; such as necrosis of skeletal and cardiac muscles and smooth muscles of gizzard and intestine have been observed by Van *et al.,* (1981). Edens and Garlich (1983) have found that lead influences egg production in leghorn and Japanese quail hens, and plasma calcium in quail hens was reduced significantly by 10 mg/ kg diet. He also found that lead induces histopathological changes in different organs. Nicholson and Osborn, (1984) detected necrosis of renal proximal tubule cells in free-living seabirds from Britain and experimentally in Starlings (*Sturnus vulgaris*) at kidney concentrations of 10 to 70 mg of Cd/kg (f.w.) (converted from dry weight).

Anwer *et al*., (1987) have noted thatlead (50micro g) injected on seventh day of incubation into the yolk sac of developing chick embryo caused stunted growth in chick embryos with several deformities such as defective beaks and legs, hydrocephalus, micropthalmia and anopthalmia. However the combined treatment of lead and calcium showed a remarkable protective aspect characterized by development of chick embryos with low incidence of deformities. Captive mallard ducks exposed to cadmium in their diet exhibited moderate to severe tubular degeneration over a renal cadmium concentration range of 88 to 134 mg of Cd/kg. (Whitehead *et al*., 1988).

Burger and Gochfeld (1988) have reported that 0.1 or 0.2 mg lead/g caused significant differences in bill length, tarsus length and wing bone length in young herring gulls after 8 days of exposure. Rao *et al.*, (1989) reported that renal corpuscles of the ducks treated with methylmercury (MeHg) lead and cadmium either alone or in two way combination exhibited minor ultrastructural changes, crystallization of granules in the juxt aglomerular cell was observed in lead and cadmium treated birds and lead exposed birds had a large number of secondary lysosomes and swollen mitochondria in proximal tubule cells. Knowles and Donaldson (1996) showed that dietary lead increases tissue peroxide levels in chick, peroxidation of lipids in hepatic microsomal membranes from birds intoxicated with lead by either route of administration was more than double that of untreated controls. Scheuhammer, (1996) has reported that low dietary calcium enhanced hepatic and renal accumulation of cadmium in Zebra finch and ring doves.

According to Benkoel *et al*., (2000) the plasma membrane is the first structure encountered by a toxic agent upon reaching a cell, so after alterations in plasma membrane mitochondrial alterations also observed. Growth and feed efficiency were reduced significantly by lead plasma and telencephalon tryptophans were also lower in lead treated chicks. Joanna and Gochfeld (2000) have shown that low-level lead affected growth, locomotion, balance, food begging, feeding, thermoregulation, depth perception and individual recognition in laboratory as well as in wild birds.

Errede *et al.,* (2001) have observed that lead concentration was higher in chorioallontoic membrane (CAM) and in metanephros (MN) when chick embryo chorioallontoic membrane was treated with lead acetate solution at the days of 10 and 11incubation. Darrell *et al.,* (2003) havereported that lead accumulation was highest in kidneys (1,360 ppb), than livers (500 ppb), lead content of the egg yolk has been strongly correlated with lead levels.

Teraoka *et al*.andBurger and Eichhorst (2007) suggest that cadmium showed age-dependent but not sex-dependent accumulation in the liver and kidney in Japanese cranes. Wayland *et al.,* (2007) noted year to year correlations among individual White-Winged Scoters and King Eiders in levels of blood cadmium, lead, mercury and selenium. Positive correlations were found in six, five and two of seven correlations for cadmium, lead, selenium, and mercury. Uric acid concentration and gamma-glutamyl transferase activity were increased in Trumpeter swans and phosphorus concentration was decreased in Canadian geese in association with high blood lead concentration ($P < 0.05$) have been observed by Katavolos *et al.,* (2007).

Toxicological Effects of Lead on the General Morphology /Body Weight etc. of birds

There are few reports available on the best of our knowledge which indicate that lead reduced the body weight of birds, of them Latta and Donaldson (1986) have noticed that lead significantly reduced body weight gain, whereas added methionine increased weight gain, he also found that the weight gain to feed consumption ratios were lower for chicks supplemented methionine, both in the presence and absence of dietary lead, indication greater efficiency of lead feed utilization. Bakalli *et al.*,(1995) have observed that chickens are more susceptible to lead intoxication he found that even 1.0 mg/kg lead in the diet can caused significant depression in the growth of broiler chickens. Vodela *et al.*,(1997) have reported that drinking water contained a mixture of arsenic, benzene, cadmium, lead and trichloroethylene at low concentrations (0.80, 1.3, 5.0, 6.7 and 0.65 ppm) and high concentrations (8.6, 13, 50, 67 and 6.5 ppm) significantly decreased water and feed intake, decreased weight gain and also suppressed natural, humoral and cell mediated immune response after 49 days intoxication of broiler chickens. Lead (1000 mg/kg) addition to the diet of layer caused decreased the egg weight, strength and thickness of egg shell, content of calcium, iron and vitamin E in blood as well as a decreased in solidity of the tibiae of the hens have been reported by Saly *et al.*,(2004). Studies of Erdogan*et al.*,(2005) showed that lead acetate added to the diet at a level of 200 mg/kg for 42 days reduced the body weight gain of the broiler chicks. Some contradictory reports are indicated that lead did not inhibit the body weight of birds. Hermayer *et al.*,(1977) have observed that lead exposure of 100 mg /kg B wt for 56 days did not exert an adverse effect on laying hens. According to Jeng *et al.,* (1997) body weights of Tsaiya ducks

was not influenced by lead ingestion (via gelatin capsule) in a dose of 10 or 20 mg/kg B wt/d for 3 months. Although these two levels of lead consistently increased lead in blood, kidney, liver and gizzard, whereas only 20 mg lead/kg B wt per day of dose additionally increased the lead acetate in femoral muscles.

Toxicological Effects of Lead on the Haematology of birds

Haematological observations allow the most rapid detection of changes in all organisms; disrupted haematoligical patterns appear very quickly and precede changes in chicken behavior and visible lesions. The rapidity of toxic effects, exerted by heavy metals is related to the blood's transport function, with the blood distributing the metals to all the body parts. Evaluation of toxic effects of the metals is facilitated by results of the basic haematological assays i.e. leucocytes, erythrocytes, haemoglobin and haematocrit.

Depending on duration and severity of exposure, the effects range from non-specific gastrointestinal symptoms (anorexia, constipation, nausea and vomiting) to central-nervous symptoms (headache, nervousness, tremor, lethargy or coma) and death. All clinical symptoms result from the toxic effects of lead, which are manifested mainly in the blood (anemia), nervous system (encephalopathy and neuropathy), and kidneys (renal dysfunction), the three major sites of lead intoxication.
The anemia results from diminished haemoglobin synthesis, haemolysis of immature erythrocytes, and direct haemolysis of mature erythrocytes with a shortened life span. The reduced haemoglobin synthesis following lead intoxication may be attributed primarily to an inhibition of heme synthesis. Strukie (1965) has observeddeclines in (WBC) white blood cells and spleen plaque-forming cell in male mallards, exposed to lead by either natural ingestion or intubation, whereas haematocrit value or PCV which is a measure of erythrocyte number, size or both and is a useful measure of physiological status of the birds with respect to oxygen carrying capacity and metabolic efficiency has also been insight. Ohi *et al.,* (1980) had suggested that lead acetate caused dose related mortality and decreased weight, heamatocrit and delta-aminoleulinic acid dehydratase activity (ALA-D) in *Columba livia*. Ekperigin and Vohra (1981) reported that excess dietary intake of methionine significantly depressed ($P<0.01$) body weight gain, PCV and Hb % in broiler chicks. Whereas Hoffman *et al.*,(1981) reported that Eagles when dosed by force-feeding with 10 lead shot significantly decreased (20-25%) their haemoglobin percentage and packed cell volume at the end of 1 week.

Due to the defective heme formation, impaired synthesis of haemoglobin must be expected also in the case of enzymes possessing heme as the prosthetic group such as, the activity of the cytochrome P-450 complexes has been shown to be reduced, especially in acute lead poisoning, while there was only a mild inhibition in chronic cases have been shown by DeMichele, (1984). Rocke and Samuel (1991) reported significant declines in WBC and spleen plaque-forming cell in male mallards exposed to lead by either ingestion or intubation, but it was not noticed in females. Redig *et al.,* (1991) have found that 0.082 mg Pb/kg B wt for 11 weeks decreased packed cell volume and haemoglobin levels in red tailed hawks. According to Khan *et al.,* (1993) lead administration to broiler chickens produced more deleterious effects characterized by adverse changes in haematological parameter. Concurrent administration of lead and monensin caused a severe depression of haematological profiles have been observed by Khan *et al.,* (1994). Murase *et al.,* (1993) suggested that the ALA-D activity ration is a very useful and sensitive indicator for the diagnosis and evaluation of therapeutic effects after lead poisoning. A highly positive correlation was observed between the concentration of blood lead and the ALA-D activity ratio, the ALA-D ratio rapidly increased after the administration of lead pellets.

Ermentrout *et al.,* (2006) have suggested that lead and cadmium heavy metal toxins cause many pathophysiological effects including anemia. These metals are able to replace coordinated Zn atoms in the zinc fingers of transcription factors and that can alter the structure and DNA binding characteristics of these proteins. Lead is able to replace in the zinc fingers of the hematopoietic transcription factor GATA -1. Katavolos *et al.*, (2007) observed that in both species (Trumpeter swans and Canadian geese) PCV, Hb concentration and MCHC decreased significantly ($P<0.05$) with increasing blood lead concentrations.

Hiraga *et al.,* (2008) have found thatlead shot orally administered to young chickens increased the number of RBCs with pycnotic nuclei, reticulocytes and enucleated RBCs increased concurrently. He also suggested that lead shot changes nuclear morphology in the peripheral blood of domestic fowl. Romero *et al.*, (2009) have reported that lead and cadmium decreased the number of erythrocytes in mallard ducks, he also observed that the percentage of apoptotic cells increased as the concentration of metals increased.Very Recently Karim *et. al.,* (2014) observed that lead caused decrease in level of blood parameters whereas chromium and cadmium increased level of blood parameters. Red Blood Corpuscles (RBCs) decreased due to cadmium and lead whereas White Blood Corpuscles (WBCs) decreased with exposure to lead and chromium. Lead also decreased the life span of RBCs of *Gallus domesticus*.

Toxicological Effects of Lead on Protein, Cholesterol and Enzymes (ACP, ALP, GOT and GPT)

Biochemical and enzymological estimation are very sensitive parameters, which can show conformity of tissue lysions, even cellular disintegration by increased or decreased values of different enzymes, protein and cholesterol, which show disorders of metabolic processes found in particular organ. In spite of much survey work, not good amount of literature could be collected in avian species with regard to the protein, cholesterol and selected enzymes. It is well known fact that enzymes play an important role in controlling the metabolic activities. Enzymes have been used as a tool, on the diagnosis of many diseases. Acid phosphatase (ACP), alkaline phosphatase (ALP), glutamatepyruvatetransaminase (GPT) or alanine transaminase (ALT) and glutamate oxaloacetate transaminase (GOT) or aspartate transaminase (AST) are very important enzymes and play vital role in metabolism these enzyme estimations in tissue extract are good bioindicators for metabolic disturbance and cellular disintegrations. Acid and alkaline phosphatases are the most important enzyme which have been studied by many workers his to chemically and

biochemically in chickens and other avian species after exposure to toxicants in different doses and exposure periods (Mooge, 1946; Mohan *et al.,* 1992**;** Benkoel *et al.,* 2000, Venkateswara, 2006).

Bloor (1943) had observed that cholesterol plays an important part in body metabolism and also characteristics constituents of all animal tissues and takes part in blood coagulation. Navikoff (1961) showedin his studies that acid phosphatase is a lysosomal enzyme which hydrolyses the ester linkage phosphate esters and helps in autolysis of the cell after its death. According to Goldfisher *et al.,* (1964) alkaline phosphatase enzyme is brush border enzyme which splits various phosphate esters at an alkaline pH and is a mediator for membrane transport. Rahman *et al.,* (1990) observedreduction in protein after oral administration of Isoprocarb at different concentration daily for 21 days in *Gallus gallusdomesticus*.

Lead administration to broiler chickens produced more deleterious effects characterized by adverse changes in haematological parameter and serve alterations in serum total protein, aspartate amino transferase (AST), cholesterol and alkaline phosphatase levels. Seddek *et al.,* (1997) reported that 1000, 1500 ppm sodium fluoride alter the activities of alkaline phosphatase and triglycride. Metal levels in eggs can often be used as an indicator of exposure and of potential effects, when eggs were exposed to cadmium, lead and flurothene (alone) before incubation by applying injection into the air cell significantly decreased alkaline phosphatase activity, while none of the compounds caused significant reduction in ALP activity in immersion group have been reported by Kertesz and Hlubik (2002).

Teresa *et al.,* (2004) observed that lead and cadmium decreased the activity of ACP and ALP in brain. The general decrease of the activity of these enzymes observed after treatment with cadmium acetate (10 mg/l) and lead acetate (300 mg/l) suggests that these metals are able to reach the central nervous system and impairs its function by neurochemical changes.Very few workers have observed enzymological changes in birds, exposed to cadmium, lead and other heavy metals. Yuan *et al.,* (2013) find out that addition of lead at 60 mg/kg resulted in increased levels of serum urea nitrogen, urea acid, and glutamic oxalacetic transaminase activity in 40 week old Hy-Line Brown hens.

CONCLUSION

It was observed from the review that intoxication of lead in birds including chicken produces various deleterious effects on body weight, general morphology, protein, cholesterol, acid phosphatase, alkaline phosphatase, glutamate pyruvate transaminase, glutamate oxaloacetate transaminase, thyroid hormone and different blood parameters.

References

A. B. Navikoff (1961). The cell (Eds. J. Brachet and A. P. Mirsky), New York, Academic Press. : 423.

A. Deborah, Robert H. Cory-Slechta, Garman and S. David (1980). Lead-induced crop dysfunction in the pigeon. *Toxicol. Appl. Pharma*.52 (3) : 462-467.

A. El-Feki, F. Ghorbel, M. Smaoul, F. Makni-Ayadiand and A. Kammoun (2000). Effets du plombd origine automobile sur la croissance generale et lactivite sexuelle du rat. *Gynecol. Obstet. Fertil.* 1: 51-59.

A. M. Scheuhammer (1996). Influence of reduced dietary calcium on the accumulation and effects of lead, cadmium and aluminium in birds.*Environ. Poll.* 94 (3) : 337-343.

A. Sh. Seddek, Th. A. Ibrahim, A. H. Ahlam and M. Abd. El-Nasser, (1997). Hematoloigcal and biochemical studies of fluorine poisoning in chickens, a trial for treatment. *Assiut. Vet. Med. J.* 36 (72) : 175-192.

A. M. Teresa, C. Laura and L. M. Luisa (2004). Biochemical changes in the kidneys after perinatal intoxication with lead and or/ cadmium and their antagonistic effects when coad ministered. *Ecotoxicol. Environ. Safety.* 57 (2) : 184-189.

Anwer. S. Ali, and N. K. Mehrotra (1987).Protective role of calcium in lead toxicity during development of chick embryo. *Drug Chem. Toxicol.* 10 (3-4) : 357-367.

B. Joanna and M. Gochfeld (2000). Metals in Albatross Feathers from Midway Atoll: Influence of Species, Age, and Nest Location. *Environ. Research*.82 (3) : 207-221.

C. J. White head, D. N. Prashad and R. O. Blackburn (1988).Cadmium-induced changes in avian renal morphology .*Experientia.* 44 (3) : 193-8.

C. L. Stone and J. H. Jr. Soares (1976).The effect of dietary selenium level on lead toxicity in the Japanese quail.*Poult. Sci.* 55 (1) : 341-349.

C.Yuan, H. H. Song, Y. J. Jiang, M. M. M. Azzam, S. Zhu and X. T. Zou (2013) Effects of lead contamination in feed on laying performance, lead retention of organs and eggs, protein metabolism, and hormone levels of laying hens. *The Journal of Applied Poultry Research*.Volume 22(4): 878-884.

D. J. Hoffman, O. H. Pattee, S. N. Wiemeyer and B. Mulhern (1981).Effects of lead shot ingestion on delta-aminolevulinic acid dehydratase activity, hemoglobin concentration, and serum chemistry in bald eagles. *J. Wildl. Diseases.*17 (3) : 423-430.

D. M. Latta and W. E. Donaldson (1986). Lead toxicity in chickens: Interactions with dietary methionine and choline. *J. Nutr.* 116 (8) : 1561-1568.

D. Romero, H. A. Garcia, C. A.Tagliati, E. M. Lopez and A. J. Garcia-Fernandez (2009). Cadmium and lead induced apoptosis in mallard erythrocytes (*Anasplatyrhynchos*). *Ecotoxi. Environ. Safety* 22 (1) : 37-44.

D. Z. Doganoc (1996).Distribution of lead, cadmium and zinc in tissues of hens and chickens from Slovenia. *Bull. Environ. Contam.Toxicol.*57 : 932-937.

F. Mooge, (1946). The physiological significance of phosphomonoesterases. *Biol. Rev.*21 : 41-49.

F. W. Edens and T. D. Garlich (1983). Lead induced egg production in leghorn and Japanese quail hens. *Poult. Sci.*62 : 1757-1763.

G. Ohi, H. Seki, K. Minowa, I. Mizoguchi and F.Sugimori (1980).Acute lead poisoning of the pigeon induced by single intraperitoneal administration of lead acetate. *Arch. Toxicol.* 46 : 265-272.

H. Teraoka, Y. Kumagai, H. Iwai, K. Haraguchi, and T. Ohba (2007).Heavy metal contamination status of Japanese cranes (*Grusjaponensis*) in east Hokkaido, Japan-

extensive mercury pollution. *Environ. Toxicol. Chem.* 26 (2) : 307-12.

H.E. Ekperigin and P. Vohra, (1981). Histopathological and biochemical effects of feeding excess dietary methionine to broiler chicks. *Avian Dis.*25 (1) : 82-95.

J. J. Burger and B. Eichhorst (2007). Heavy metals and selenium in grebe feathers from Agassiz National Wildlife Refuge in northern Minnesota. *Arch Environ. Contam.Toxicol.* 53 (3) : 442-449.

J. Burger and M. Gochfeld **(1988).** Effects of lead on growth in young herring gulls (*Larusargentatus*).*J. Toxicol. Environ. Hlth.* **25 (2) : 227-36.**

J. F. Van Vleet, G. D. Boon and V. J. Ferrans (1981).Induction of lesions of selenium-vitamin E deficiency in ducklings fed silver, copper, cobalt, tellurium, cadmium, or zinc: Protection by selenium or vitamin E supplements. *Am. J. Vet. Res.* 42 (7) : 1206-1217.

J. K. Nicholson, and D.Osborn (1984). Kidney lesions in juvenile starlings *Sturnus vulgaris* fed on a mercury contaminated synthetic diet. Environ. Poll. 33:195–206

J. K. Vodela, J. A. Renden, S. D. Lenz, W. H. Mcelhenney and B. W. Kemppainen (1997). Drinking water contaminants (Arsenic, Cadmium, Lead, Benzene, and Trichloroethylene). 1. Interaction of contaminants with nutritional status on general performance and immune function in Broiler Chickens. *Poult. Sci.*76 : 1474-1492.

J. Mohan, R. P. Moudgal, and J. N. Panda (1992). Effects of cadmium salt on phosphomonoesterases activity and fertilizing ability of fowl spermatozoa. Indian. *J. Exp. Biol.* 30 (3) : 241-243.

J. Saly, D. Baranova, L. Pesek, Z. Sevcikove, D. Kocik, V. Sutiak, J. Neuschl and J. Kremen (2004) Effect of lead on health and productivity of layers. *Bull. Vet. Inst. Pulawy*.48 : 75-80.

K. L. Hermayer, P. E. Stake and R. L. Shippe (1977). Evaluation of dietary zinc, cadmium, tin, lead, bismuth, and arsenic toxicity in hens. *Poultry Sci.*56 : 1721.

Karim Gabol, M. Zaheer Khan, M. Umair, A. Khan, Peer. Khan, Farina. Fatima, Saima Siddiqui, Tanveer Jabeen, Nadeem Baig, M. AsifIqbal, M. Usman, A. Hashmi and Muhammad Tabish (2014). Induced Effects of Lead, Chromium and Cadmium on *Gallus domesticus*.*Canadian Journal of pure and Applied Science.*Vol. 8(3): 3035-3042.

L. Benkoel, F. Dodero, E. Roussel, J. C.Baudin, R. Lambert, A. Chamlian, and H. Augier (2000). Hepatotoxic effect of metallic pollutants on enzyme histochemical activities of yellow-legged gull *Laruscachinnansmichahellis* liver.*Cell Mol. Biol.* (Noisy-le-grand) 46 (7): 1183-1189.

L. R. Berg, J. O. Nordstro, and L. E.Ousterhout (1980). The prevention of chick growth depression due to dietary lead by increased dietary calcium and phosphorus levels.*Poult. Sci.*59 : 1860-1863.

M. Errede,G.Elia, M.Bertossi, M. L. Mulas, A. Riva, D.Virgintino, V.Benagiano, and L.Ambrosi, (2001). Developmental effects of lead acetate on the chick embryo metanephros. *J. SubmicroscCytol. Pathol.* 33 (3): 261-273.

M. F. Rahman, M. K. Siddiqui, M. Mahboob, and M. Mustafa (1990). Haematological and hepatotoxic effects of isoprocarb in chicken. *J. Appl. Toxicol.* 10 (3) : 187-192.

M. Wayland, R. T. Alisauskas, D. Kellett and K. Mehl (2007).Year-to-year correlations in blood metal levels among individuals of two species of North American sea ducks. *Environ. Poll.* 150 (3) : 329-337.

M. Z. Khan, J. Szarek, D.A. Krasnodebska, and A. Koncicki (1993). Effects of concurrent administration of lead and selenium on some haematological and biochemical parameters of broiler chickens. *Acta Vet Hung*. 41 (1-2) :123-37.

M. Z. Khan, J.Szarek, A. Koncicki, and A. Krasnodebska-Depta (1994). Oral administration of monensin and lead to broiler chicks: effects on haematological and biochemical parameters. *Acta Vet. Hung.*42 : 111-120.

O. Trainer, Daniel Hunt and A. Richard (1965).Lead poisoning of waterfowl in Wisconsin. *J. Wildl. Mgt.* 29 (1) : 95-103.

P. D. Strukie (1965). Avian physiology 2nd edn.Comstock Publishing Associates. I thaca, New York : 5-6.

P. Kaemar, and A. Samo (1976). Dynamics of changes in the activity of plasma lactate dehydrogenase, leucylarylamidase and GOT and GPT aminotransferase in poultry following damage to the organism caused by cadmium and tetrachloromethane. *Vet Med (Praha).* 21 (1) : 19-26.

P. T. Redig, E. M. Lawler, S. Schwartz, J. L. Dunnette, B. Stephenson and G. E. Duke (1991). Effects of chronic exposure to sub-lethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks. *Arch. Environ. Contam.Toxicol.* 21 : 72–77.

P. V. Rao, S. A. Jordan and M. K. Bhatnagar (1989).Ultrastructure of kidney of ducks exposed to methylmercury, lead and cadmium in combination. *J. Environ. Pathol.Toxicol.Oncol.* 9 (1) : 19-44.

R. J. Venkateswara, (2006). Sublethal effects of an organophosphorus insecticide (RPR-II) on biochemical parameters of tilapia, *Oreochromismossambicus*.*Comp. Biochem. Physiol. C Toxicol.Pharmacol.* 143 (4) : 492-498.

R. P. Ryan and C. E. Terry (1988). Toxicology desk reference: The toxic exposure and medical monitoring index, 1997-1998. Vol. 1, 4th ed. Washington D. C.: Taylor & Francis.

R.I. Bakalli, G.M. Pesti and W.L. Ragland (**1995).**The magnitude of lead toxicity in broiler chickens. *Vet. Hum. Toxicol*. **37 (1) : 15-19.**

R.M. Ermentrout, M. E. Layon, C. J. Ackley, P. Venkatesan and C. H. Lowrey(2006). The effects of lead and cadmium on GATA-1 regulated erythroid gene expression. *Blood Cells Mol. Dis.* 37 (3) : 164-172.

S. Goldfisher, E. Esser, and A. B. Novikoff (1964) Histochem. Cytochem.12 : 72-95. In: Hinton, D.E., Kendall, M.W. and Silver, B. B. Use of histological and histochemical assessment in the prognosis of the effects of aquatic pollutants. *Amer. Soc. Test. and Mat.* 528 : 194-208.

S. J. DeMichele (1984).Nutrition of lead. *Comp. Biochem. Physiol.* 3 : 401- 408.

S. L. Jeng, S. J. Lee, Y. F. Liu, S. C. Yang and P. P. Liou (1997). Effect of lead ingestion on concentrations of lead

in tissues and eggs of laying Tsaiya ducks in Taiwan. *Poult. Sci.*76 : 13-16.

S. O. Knowles and W.E. Donaldson (1996). Dietary lead alters fatty acid composition and membrane peroxidation in chick liver microsomes. *Poult. Sci.* **75 (12) : 1498-1500.**

T. E. Rocke and M. D. Samuel (1991). Effects of lead shot ingestion on selected cells of the mallard immune system. *J. Wild Disea.* 27 (1) : 1-9.

T. Hiraga, K. Ohyama, A. Hashigaya, V. Ishikawa, W. Muramoto, H. Kitagawa, N. Mizuno and H.Teraoka (2008). Lead exposure induces pycnosis and enucleation of peripheral erythrocytes in the domestic fowl. *The Veter. J.* 178 (1) : 109-114.

T. Murase, N. Horiba, I. Goto, O.Yamato, T. Ikeda, K. Sato, K. Jin, M. Inaba, and Y. Maede (1993). Erythrocyte ALA-d activity in experimentally lead-poisoned ducks and its change during treatment with disodium calcium EDTA.*Res Vet Sci.* 55 (2) : 252-257.

V. Kertesz, and I. Hlubik (2002). Plasma ALP activity and blood PCV value changes in chick fetuses due to exposure of the egg to different xenobiotics. *Environ. Poll.*117 (2) : 323-327.

W. T. Darrell, M. I. Paula, L. C.Thomas, A. K. Julie and M. E. Steve (2003). Lead contamination of chicken eggs and tissues from a small farm flock. *J. Vet. Diagn.Tnvest.*15 : 418-422.

W.A. Bloor (1943). Biochemistry of the fatty acid and their compounds, the lipids. Reinhold Publ. Corp., N.Y.

Z. Erdogan, H. Turkey, S. Erdogan, S. T. Aksu and E. Baytok (2005). The effects of dietary lead exposure and ascorbic acid on performance, lipid peroxidation status and biochemical parameters of broilers. *Turk. J. Vet. Anim. Sci.*29 : 1053-1059.

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