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Research Article

Effect of Quail Egg on Cadmium-induced Toxicity in Rats

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Abstract

Cadmium (Cd) is a toxic environmental and occupational contaminant that affects most body systems. Quail egg consumption has been reported to remove toxins and heavy metals from the body but there is dearth of information on its effect on cadmium toxicity. This study investigated the protective effect of quail egg on haematological profile, spermatogenic parameters, renal anti-oxidant and inflammatory markers in cadmium toxic male Wistar rats. Cadmium toxicity was induced with a daily dose of Cd (0.5mg/kg) for 7days**.** Twenty-five male Wistar rats were randomly divided into five groups: Group 1(control), Group 2-cadmium untreated (CUT), Group 3-Quail egg pre-treated + cadmium (QEPC), Group 4-Quail egg and cadmium co-treated (QECT), Group 5-cadmium + Quail egg post-treated (CQEP). Experimental groups were treated orally with quail egg (5ml/kg bw) for 14 days before or after cadmium induction. Blood through retro-orbital puncture from each rat was analysed for hematological indices. Caudal epididymis and kidneys were analysed for spermatogenic parameters, renal myeloperoxidase (MPO), superoxide dismutase (SOD) activities, and nitric oxide (NO). Oral administration of quail egg significantly increased ($p<0.05$) PCV, Hb, RBC, NO, SOD, sperm motility, viability and count with a decrease ($p<0.05$) in WBC and MPO compared to control while CUT showed significant increase ($p<0.05$) in WBC, MPO and a significant decrease in PCV, Hb, RBC, NO, SOD, sperm motility, viability and count when compared to all Quail egg treated groups. Treatment with quail egg reversed the toxic effects of cadmium on haematological profile, Spermatogenic parameters, renal antioxidants and inflammation possibly via an anti-oxidative mechanism.

Keyword: *Cadmium toxicity, Quail egg, Haematology, Spermatogenic indices*

INTRODUCTION

Cadmium is a toxic heavy metal and an environmental pollutant capable of affecting human health (Jarup *et al.,* 1998: Godt *et al.,* 2006). Major sources of exposure include tobacco smoking, application of phosphate fertilizers, and combustion of fossil fuels, cement production, food and drinking water (Nobuhiko, 2009). Inhalation and ingestion of high quantities of cadmium has been shown to cause kidney failure, nephrotoxicity, renal stone formation, bone disease, nerve or brain damage, hypertension, iron deficiency anemia, hepato-toxicity, testicular damage, lung damage, intestinal damage, fragile bones and persistent proteinuria (Longe, 2005; Ige *et al*., 2011). Long term exposure to cadmium results in accumulation of the metal in the liver and kidney rendering them primary target organs for destruction (Smalinskiene *et al*., 2004; Smalinskienel *et al*., 2006). Cadmium has a very long biological half life (10-30yrs) in the body and its toxicity depends on the route, amount, and duration of exposure (Satarug *et al.,* 2003).

Cadmium exposure has been reported to induce blood disorders and immunological defects which were observed as the earliest indicators of toxic effects on tissues (Johnson *et al*., 2012). In another study, declining fertility associated with reduced sperm count and testicular function in men were observed to correlate with cadmium exposure (Siu *et al*., 2009). Exposures to cadmium have been reported to cause adverse effect on male reproductive system through degenerative changes in epididymis, testes, and seminal vesicles (Oliveira, 2012). It has been suggested that cadmium induces toxicity in various organ systems via its ability to generate free radical that exert deleterious effects on these organs and systems in the body (Bagchi *et al.,* 1996; Kowalczyk *et al.,* 2003).

Quail egg is a good source of nutrients for human health (Tunsaringkarn *et al.,* 2013). Traditionally among the Chinese, it is used for the treatment of many diseases like tuberculosis, asthma, and even diabetes (Tunsaringkarn *et al*., 2013). It has been reported to strengthen the immune system due to its antioxidant effects and the presence of minerals as well as vitamins, detoxifies blood and remove heavy metals from the blood (Tunsaringkarn *et al.,* 2013). There is however a dearth of information on the protective effect of quail egg consumption on cadmium-induced toxicity. This study therefore investigated the haematological profile, spermatogenic indices, renal inflammation and antioxidant status in cadmium toxic rats treated with quail eggs.

MATERIAL AND METHODS

Chemicals and Reagents: All chemicals and reagents used were of analytical grades. Cadmium sulphate and Adrenaline were obtained from Sigma Aldrich Chemical Company, Germany.

Animals and Experimental Design: Twenty-five male Wistar rats, weighing between 180–200g were obtained from animal house of the Department of Physiology, University of Ibadan. The animals were fed with standard rat pellet and watered *ad libitum*. The rats were housed in well ventilated cages and maintained at room temperature according to the guidelines for the care and use of laboratory animals of the

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University of Ibadan, Nigeria. The animals were randomly divided into five groups of 5 animals as follows: Group 1 served as control and was given 10ml/kg of distilled water, Group 2 (CUT) received cadmium sulphate ($CdSO_4$, 0.5mg/kg i.p) for 7days (Satarug and Moore, 2004), Group 3 (QEPC) were pre-treated with 5ml of *Quail egg* for 14days followed by $CdSO_4$ for 7days. Group 4 (QECT) received $CdSO_4$ (0.5mg/kg i.p) and quail egg (5ml) simultaneously for 7days and thereafter Quail egg only (5ml/kg) for 14days. Group 5 (CQEP) were pre-treated with $CdSO_4$ (0.5mg/kg i.p) for 7days followed by Quail egg (5ml/kg) for 14days.

Blood Collection: At the end of the treatment period (14 days), animals were weighed and blood samples were obtained from the retro-orbital sinus under mild ether anaesthesia into a 5ml heparinised EDTA tubes to prevent coagulation. Haematological indices were assessed in all blood samples collected using an automatic analyzer (Haematology auto analyzer Sysmex KX-21N). Haematological parameters analyzed include; Total and differential white blood cell count (WBC), Red Blood Cell (RBC) Count, haemoglobin concentration (Hb), and Hematocrit (PCV).

Tissue samples: Caudal epididymis was harvested from each rat for spermatogenic indices (sperm motility, viability and count) (Badkoobeh *et al*., 2013). The kidneys of each animal per group was harvested, weighed and homogenized in ice cold 10 % w/v phosphate buffer (0.1 M, pH 7.4). Homogenates were centrifuged for 15 minutes using a cold centrifuge at 7500rpm and the supernatant obtained was used for biochemical analysis.

Biochemical Analysis

Renal SOD activity was assessed as described by Misra and Fridovich (1972), renal MPO activity was determined according to the method of Smith and Castro (1978) while nitric oxide level was determined using the Griess method (Griess, 1879). Sperm motility and viability was estimated at room temperature using a bifocal microscope, while the sperm counts were determined using the Neubauer haemocytometer.

Immunohistochemistry

Immunohistochemical staining was done using iNOS immunoassay kit. This kit employed the indirect assay principle using primary and secondary antibodies that open up iNOS antigens (Linnoila and Petruz, 1984).

Statistical Analysis: Data were analyzed using One-way ANOVA while Newman-Keuls' Post-hoc test was used to establish the statistical significance at $p<0.05$. Data were computed using Graph Pad Prism statistical software (5.04).

RESULTS

Body weight

Cadmium induced and untreated group (CUT) had 9.54% increase in body weight when compared to the body weight before exposure. The final body weight recorded in Cadmium induced and untreated group was not significantly different from control. All quail egg treated groups had increase in body weight though not significant (Table 1).

Table 1:
Effect of quail egg on Body weight in cadmium-induced toxic rats

Group	Weight before exposure to cadmium (g)	Weight after 21 days of treatment (g)
Control	182.80 ± 8.10	221.80 ± 13.47
Cadmium untreated	182.00 ± 8.23	201.20 ± 10.75
Quail egg pre-treated +cadmium	183.80 ± 5.32	195.60 ± 4.50
Quail egg and cadmium co-treated	185.60 ± 7.10	201.20 ± 10.75
Cadmium + quail egg Post-treated	185.00 ± 5.90	203.20 ± 5.55

Values are expressed as mean ± SEM, n=5; p < 0.05
CUT- Cadmium untreated group
QEPC- Quail egg pre-treated+ cadmium group
QECT- Quail egg and cadmium co-treated group
CQEP - Cadmium + quail egg Post-treated group

Table 2:
Effect of quail egg on Mean kidney weight in cadmium-induced toxic rats

Treatment groups	Mean Kidney weight
Control	0.55 ± 0.02
CUT	0.47 ± 0.01*
QEPC	0.49 ± 0.03*
QECT	0.49 ± 0.02*
CQEP	0.53 ± 0.04

Values are Mean ± SEM; n=5; P <0.05 * indicates values significantly *decrease when compared with control and CQEP*
CUT- Cadmium untreated group
QEPC- Quail egg pre-treated+ cadmium group
QECT- Quail egg and cadmium co-treated group
CQEP - Cadmium + quail egg Post-treated group

Mean kidney and testicular weight

Cadmium exposed and untreated with quail egg group had 14.49% significant reduction ($p<0.05$) in mean kidney weight when compared to control. Treatment with quail egg in QEPC, QECT and CQEP increased the mean kidney weight by 4.08%, 4.08% and 12.77% respectively when compared to CUT. Increase in mean kidney weight of CQEP was observed to be significantly increased when compared to CUT, QEPC and QECT (Table 2).

The mean Testicular weight decreased significantly ($p< 0.05$) in cadmium untreated group compared to control by 32.93%**.** Quail egg administration significantly increased mean testicular weight in CQEP by 23.36% when compared to cadmium untreated, QEPC and QECT (Table 3).

Hematological changes

There was significant decrease ($p< 0.05$) in PCV, Hb and RBC in CUT group compared to control. The values for PCV, Hb and RBC obtained in the QEPC were significantly reduced compared to control, QECT and CQEP groups (Table 4).

There was a significant increase ($p<0.05$) in WBC count of CUT group when compared to control. Treatment with quail egg significantly decreased ($P<0.05$) WBC when compared with CUT (Table 4). A significant increase ($p<0.05$) in

lymphocytes and neutrophils was observed in CUT group and QEPC when compared to control, QECT and CQEP. There was a significant decrease in the monophils count of CQEP compared to QECT. There was no significant difference (*P<0.05*) in MCV, MCH and MCHC values of control and all quail egg treated groups (Table 4).

Table 3:
Effect of quail egg on Mean testicular weight in cadmium-induced toxic rats

Treatment groups	Mean Testicular weight
Control	1.09 ± 0.01
CUT	0.82 ± 0.07*
QEPC	0.57 ± 0.13*
QECT	0.84 ± 0.07*
CQEP	1.07 ± 0.04

Values are Mean ± SEM; n=5; P <0.05 * indicates values significantly *decreased when compared with control and CQEP*
CUT- Cadmium untreated group
QEPC- Quail egg pre-treated+ cadmium group
QECT- Quail egg and cadmium co-treated group
CQEP - Cadmium + quail egg Post-treated group

Myeloperoxidase (MPO) activity: There was 74.75% increase (p< *0.05*) in MPO activity of CUT group compared to control. Values obtained in the quail egg treated groups show a decrease in MPO activity (Figure 1).

Nitric Oxide level: Nitric oxide value obtained in CUT group was 41.32% reduced compared to control. Values obtained in the QECT and CQEP groups were 50.91% and 50.31% increased (*P<0.05*) compared to the CUT group (Figure 2).

Activity of Superoxide Dismutase: There was 56.29% reduction (*p<0.05*) in SOD activity of CUT group when compared to control and all quail egg treated groups. Group 5 (*QEPT*) showed significant increase (*P< 0.05*) in SOD activity compared to control, QEPC and QECT groups. This is equivalent to 68.42% increase (Figure 3).

Spermatogenic parameters

Sperm Motility: Sperm motility was significantly decreased (*p< 0.05*) in CUT group by 95.83% when compared to control and all quail egg treated groups. Values obtained in the QECT and QEPT groups were significantly reduced compared to QEPC and control (Figure 4).

Sperm viability: Sperm viability decreased significantly (*P < 0.05*) in CUT group by 92.88% when compared to control and all quail egg treated groups. Group 5 (QEPT) showed significantly lowered (*p < 0.05*) sperm viability when compared to control (Figure 5).

Sperm count: Sperm count decreased significantly (*P < 0.05*) in CUT rats when compared to control and all quail egg treated groups. Group 3 (QEPC) and group 4 (QECT) showed significantly lowered (*p < 0.05*) sperm viability when compared to control and QEPT group (Figure 6).

Immunohistochemistry(kidney):
Inducible NOS was not expressed in control Kidney (plate 1), but there were abundant aggregates and specific iNOS immunoreactivity (indicated with black arrow) in CUT kidney (plate 2), expression of iNOS at the recruiting phase (indicated with red arrow) in QEPC (plate 3) and fewer expression of iNOS immunoreactivity (indicated with blue arrow) in QECT (plate 4). The kidney of QEPT shows scanty aggregates and specific iNOS immunoreactivity (yellow arrow).

Table 4:
Haematological changes in control and treated rats

	PCV (%)	Hb (g/dL)	RBC (10^6/ mm^3)	WBC (mm^3)	Lymp (%)	Mono (%)	Neut (%)	MCV (fl)	MCH (pg)	MCHC (g/dl)
Control	39.60 ± 1.03	13.32 ± 0.35	6.65 ± 0.15	6510 ± 861.70	59.20 ± 3.25	2.80 ± 0.37	36.20 ± 2.58	59.57 ± 0.36	29.73 ± 0.17	3.36 ± 0.02
CUT	28.60 ± 0.81*	9.10 ± 0.21*	4.20 ± 0.22*	11300 ± 1734*	69.00 ± 2.12*	2.60 ± 0.51	26.00 ± 2.35*[b]	69.15 ± 5.13	31.54 ± 1.46	3.20 ± 0.14
QEPC	35.60 ± 0.75#	11.88 ± 0.24#	5.81 ± 0.18#	6862 ± 1115	59.20 ± 1.10	3.60 ± 0.40	33.80 ± 1.53	61.40 ± 0.79	29.97 ± 0.19	3.34 ± 0.02
QECT	39.60 ± 0.40	13.36 ± 0.18	6.35 ± 0.28	5188 ± 955.20	59.20 ± 1.10	3.60 ± 0.40	33.80 ± 1.53	62.76 ± 2.45	29.65 ± 0.17	3.37 ± 0.02
CQEP	39.20 ± 0.49	13.24 ± 0.15	6.54 ± 0.06	6446 ± 680.70	61.60 ± 0.51	1.80 ± 0.37[a]	30.60 ± 0.75	59.94 ± 0.53	29.61 ± 0.29	3.38 ± 0.03

Values are Mean ± SEM; n=5; P <0.05 * Indicates values significantly *decreased when compared with control, QEPC, QECT and CQEP*
Indicates values significantly decreased when compared with control, QECT and CQEP
[a]*indicates values significantly decreased when compared QECT*
[b]*indicates values significantly decreased when compared to control*
[c]*indicates values significantly decrease when compared to control and QECT*
CUT- Cadmium induced and untreated with Quail egg group
QEPC- Quail egg pre-treated+ cadmium induced group
QECT- Quail egg and cadmium co-treated group
CQEP - Cadmium + quail egg Post-treated group:MCV- Mean Cell Volume (measured in femtolitres)
MCH- Mean Cell Haemoglobin (measured in picogrammes): MCHC- Mean Cell Haemoglobin Concentration (measured in g/dL)

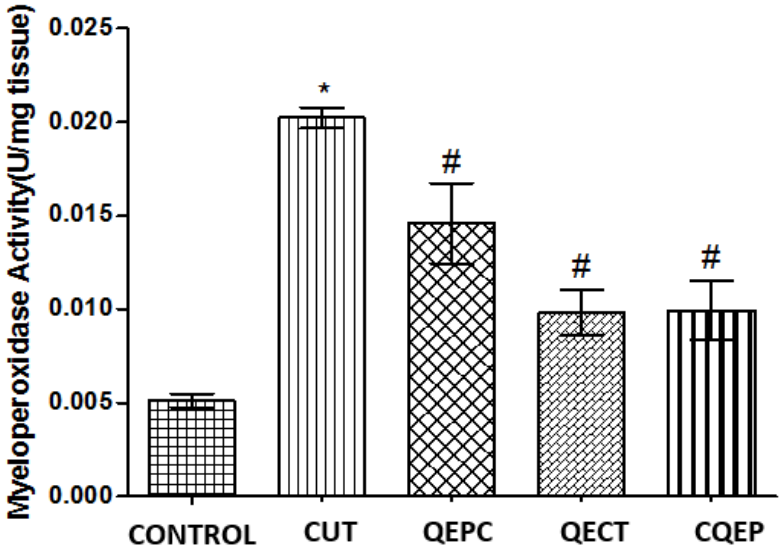

Figure 1: Effect of quail egg on myeloperoxidase activity in cadmium-induced toxic rats *Values are Mean ± SEM; n=5; P <0.05* * Significant (c.f. *control, QEPC, QECT and CQEP)* # Significant *(c.f.* control)

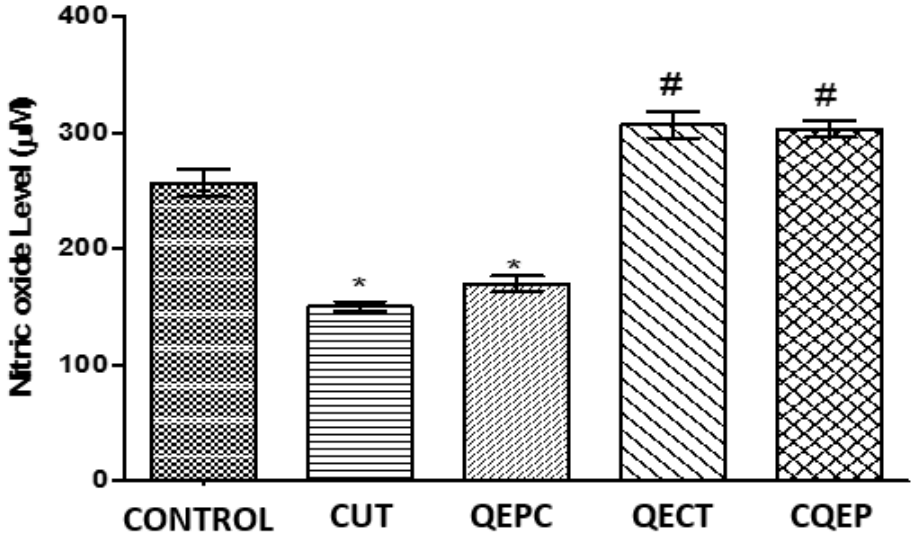

Figure 2: Effect of quail egg on nitric-oxide level in the kidney of cadmium-induced toxic rats . *Values are Mean ± SEM; n=5; P <0.05* * * Significant (c.f. *control, QEPC, QECT and CQEP)* # Significant *(c.f.* control)

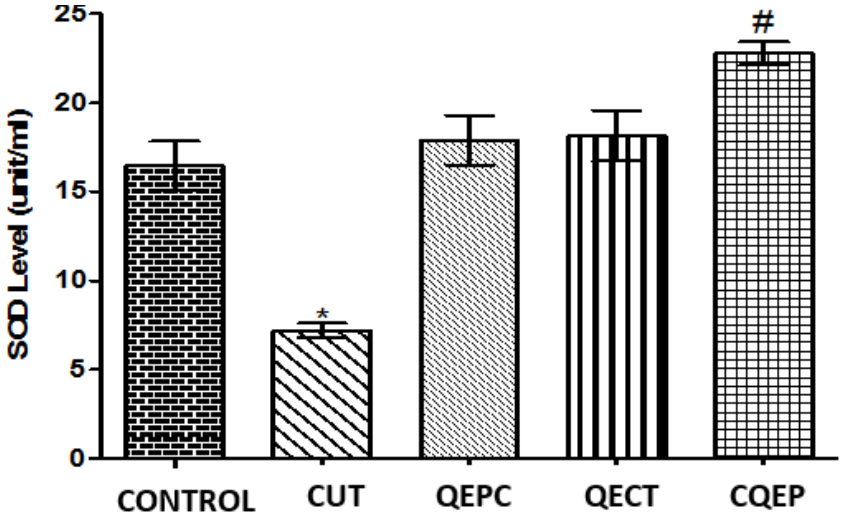

Figure 3: Effect of quail egg on SOD activity in cadmium-induced toxic rats *Values are Mean ± SEM; n=5; P <0.05* * Significant (c.f. *control, QEPC, QECT and CQEP)* # Significant *(c.f.* control, QEPC, QECT)

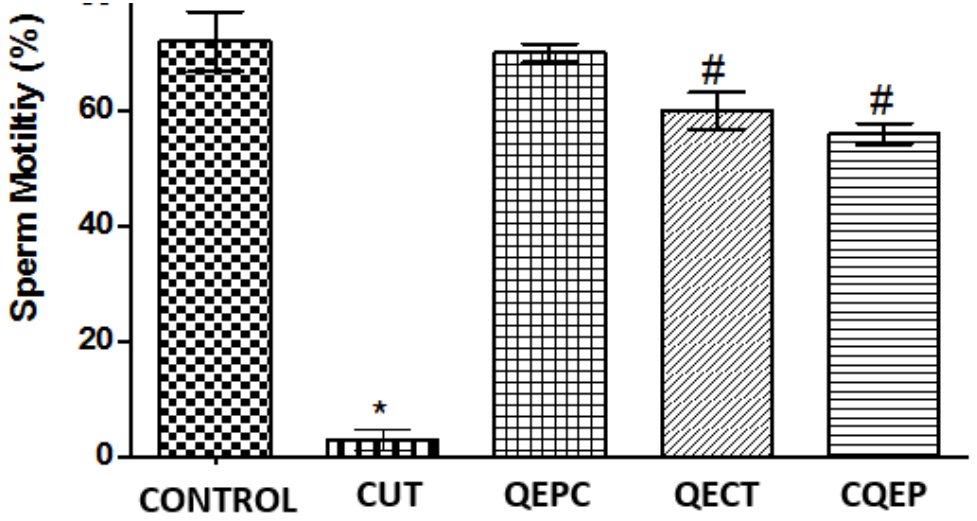

Figure 4: Effect of quail egg on sperm motility in cadmium-induced toxic rats. *Values are Mean ± SEM; n=5; P <0.05* * Significant (c.f. *control, QEPC, QECT and CQEP)* # Significant *(c.f.* control and QEPC)

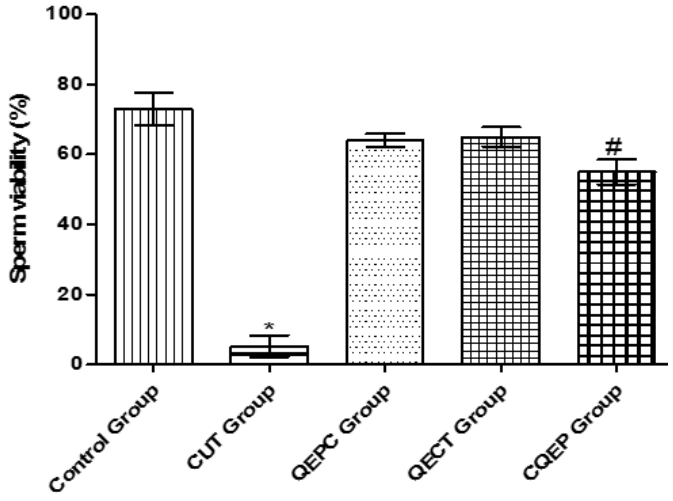

Figure 5: Effect of quail egg on sperm viability in cadmium-induced toxic rats. *Values are Mean ± SEM; n=5; P <0.05* * Significant (c.f. *control, QEPC, QECT and CQEP).* # Significant *(c.f.* control)

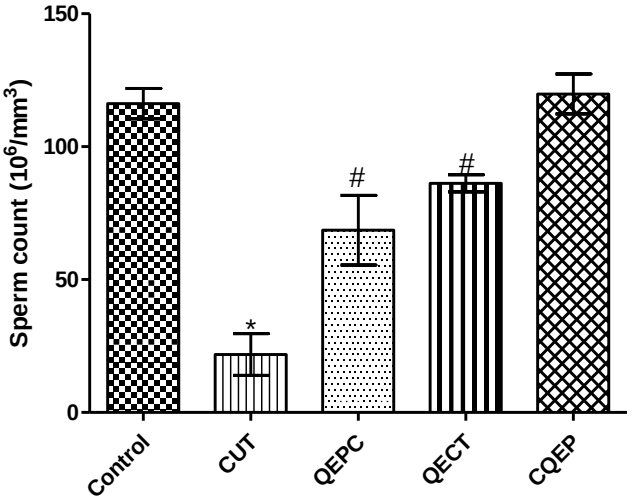

Figure 6:
Effect of quail egg on sperm count in cadmium-induced toxic rats. *Values are Mean ± SEM; n=5; P <0.05* * Significant (c.f. *control, QEPC, QECT and CQEP).* # Significant *(c.f.* control and QEPT)

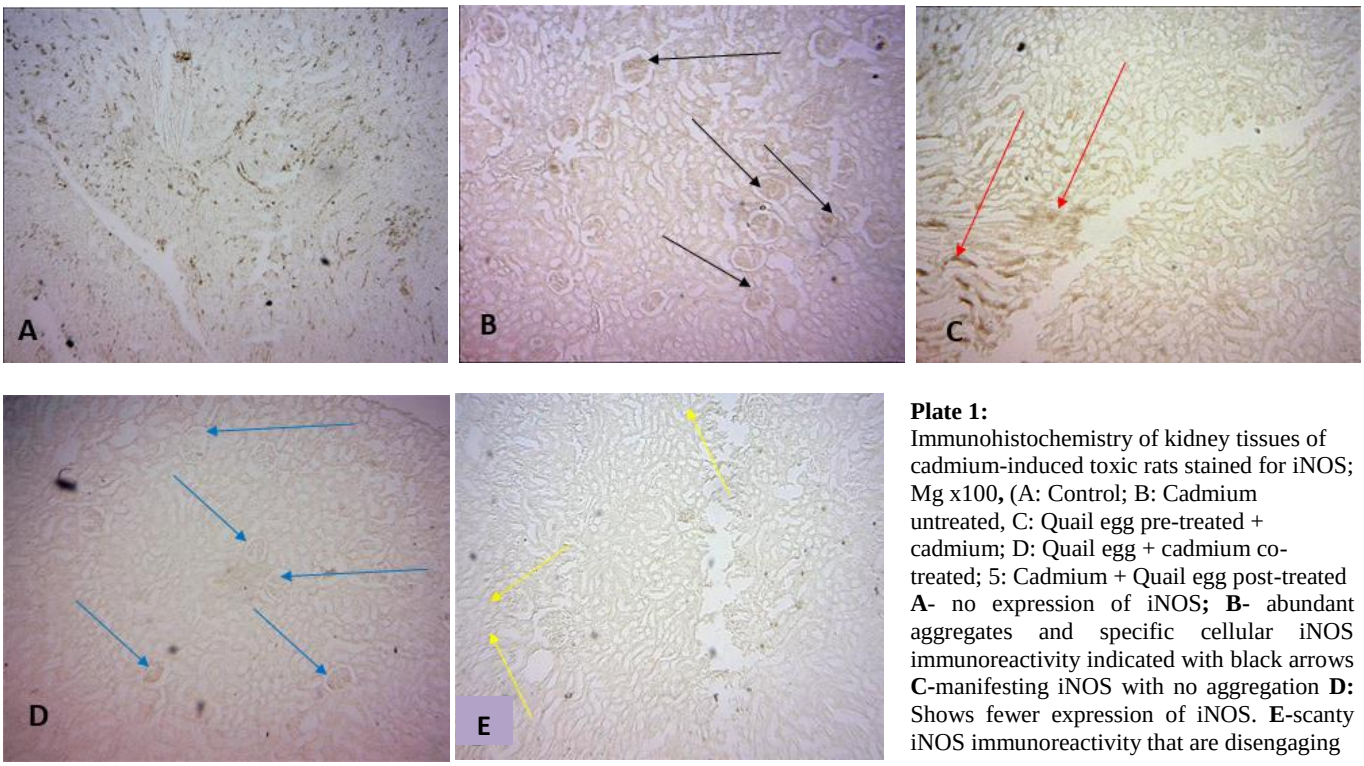

Plate 1:
Immunohistochemistry of kidney tissues of cadmium-induced toxic rats stained for iNOS; Mg x100**,** (A: Control; B: Cadmium untreated, C: Quail egg pre-treated + cadmium; D: Quail egg + cadmium co-treated; 5: Cadmium + Quail egg post-treated **A-** no expression of iNOS**; B-** abundant aggregates and specific cellular iNOS immunoreactivity indicated with black arrows **C**-manifesting iNOS with no aggregation **D:** Shows fewer expression of iNOS. **E**-scanty iNOS immunoreactivity that are disengaging

DISCUSSION

Exposure to environmental chemicals including heavy metals has been reported to pose a great health challenge to both animals and humans (Diamanti-Kandarakis *et al*., 2009). Cadmium, an environmental pollutant that gets introduced into the environment either naturally or through other sources has no known physiological importance. It has however been reported to interfere with normal homeostatic control even at a micro-molar concentration (Walkes, 2003; Takiguchi and Yoshihara, 2006; Tomaz *et al*., 2012). In this study, a significant decrease in kidney and testicular weights were observed in CUT when compared to control. The observed decrease in kidney weight may be due to degeneration of renal epithelial cell (Ola-Mudathir *et al*., 2008; Mahran *et al*., 2011). The decrease in the mean testicular weight of cadmium untreated rats observed in this study could be due to derangement in some testicular structures such as seminiferous tubules and leydig cells (Setchell, 1998; Rekha *et al*., 2011). Treatments with quail egg significantly increased the kidney and testicular weight suggesting possible prevention of degenerative processes that would have caused a decrease in organ weight.

Anaemia is an important manifestation of cadmium toxicity (ATSDR, 1999). Cadmium-induced anaemia has been attributed to impairment in the synthesis of erythropoietin, a

hormone which promotes formation of red blood cell (Horiguchi *et al.,* 1999). Haematological parameters (PCV, Hb and RBCs) evaluated in this study were significantly decreased in cadmium untreated rats when compared to control which may be due to alterations in iron metabolism (Menke *et al*., 2012).This result is consistent with the report of Gluhcheva *et al.* (2011) on PCV, HB and RBCs in cadmium toxicity. Treatment with quail egg significantly increased PCV, Hb and RBC count in all quail egg treated groups when compared to cadmium untreated rats. This may be due to iron constituent of quail egg which has been shown to be vital for haematopoiesis (Tunsaringkarn *et al.,* 2013).

It has been reported that the immune system is modulated in response to environmental contaminants such as cadmium (Fowler, 2009). Cadmium toxicity has also been reported to cause a decrease in immune response due to the detrimental effects of cadmium on the kidneys, liver, and T lymphocyte production (Dan et al., 2000). In this study, the total WBC count of cadmium untreated rats was significantly increased and this is consistent with the report of El-Demerdash *et al*., (2004) and Onwuka *et al*., (2010) who also reported an increase in WBC count in cadmium toxicity and suggested that this may be due to increased lymphocytes and neutrophils production. Treatment with quail egg significantly reduced WBC count in all quail egg treated groups when compared to control which suggests that quail egg consumption may possess the ability to reduce the bioaccumulation of cadmium (Tunsaringkarn *et al.,* 2013) and thus prevent the observed cadmium induced production of WBC in the cadmium untreated group.

Cadmium toxicity has been reported to cause a systemic inflammatory response that is characterized by numerical and functional changes in the granulocyte compartment, and increased levels of inflammation-related cytokine activity in the circulation (Kataranovski *et al.,* 1998; Haase *et al.,* 2010). Cadmium in low doses has been reported to inhibit the humoral and cellular immune response while at higher doses, an opposite effect is observed (Lafuente *et al.,* 2004). In this study, an increase in lymphocyte count was observed in the cadmium untreated group and this is in accordance with the reports of Lafuente *et al.,* (2004). This increase in lymphocyte level could be attributed to the activation of the immune system as a result of high cadmium titre of circulating blood. Lymphocyte values obtained in the treatment group were comparable to control values thus suggesting an attenuation of cadmium toxic effects on the immune system in the quail egg treatment groups.

Cadmium has also been reported to exert direct and indirect cytotoxic effects on various cell types, including mononuclear cells and macrophages (Theron *et al.,* 2012). In neutrophils, cadmium has been reported to trigger pro-oxidative and pro-inflammatory mechanisms which have been observed to predispose them to reactive oxygen species mediated cell death (Theron *et al.,* 2012). This study shows a decrease in monocyte and neutrophil count which may be due to cadmium induced apoptosis of neutrophils as reported by Theron *et al.,* (2012). Neutrophil values obtained in the quail egg treatment groups suggests an attenuation of cadmium toxic effects on neutrophils as values obtained in these treatment groups were comparable to controls. Monocyte values in the cadmium untreated group suggest some level of cytotoxicity may exist as values obtained were reduced compared to control animals. The quail egg pretreated and quail egg co-treatment groups on the other hand showed an increase in monocyte level compared to the cadmium untreated group which may suggest an attenuation of cytotoxicity and an increased immune response to the presence of cadmium, a potentially toxic substance, in the blood. Interestingly, monocyte values obtained in the quail egg post treated group were reduced compared to control and all other treatment groups. At present the reason for this is unknown however, this observation will form the basis of subsequent investigations in our laboratory.

It has been reported that myeloperoxidase (MPO) is mainly released by activated neutrophils (but also macrophages) at the site of inflammation (Kataranovski *et al.,* 2009). Myeloperoxidase possesses powerful pro-oxidative and pro-inflammatory properties and its activity is often taken as a measure of inflammation (Zhao *et al*., 2006). This study observed elevated MPO activity in cadmium exposed and untreated rats and this was ameliorated in all quail egg treated groups. Findings of this study are consistence with the report of Stoic, *et al*. (2010) who also reported stimulation of neutrophils activity in cadmium exposure. The ability of quail egg to remove heavy metals might have accounted for the observed reduced cadmium-induced activity of MPO in all quail egg treatment group (Tunsaringkarn *et al.,* 2013).

Nitric oxide deficiency causes systemic and glomerular hypertension, tubular interstitial injury and proteinuria as a result of impaired L-arginine biosynthesis into endothelial cells which is the main substrate for nitric oxide synthesis (Baylil, 2008).This result observed a significant decrease in tissue nitric oxide level (NO) of cadmium untreated rats when compared to control. Treatment with quail egg however increased the activity of NO in QECT and QEPT. The increase in NO activity stimulated by quail egg may be due to its ability to mop up heavy metals (Tunsaringkarn *et al.,* 2013).
Cadmium toxicity has been reported to increase the formation of free radicals whose effects are deleterious to various body systems. Antioxidants have been suggested and used in the amelioration of cadmium toxicity (Shirashi *et al*., 1993; Shaikh *et al*., 1999; Choi and Rhee, 2003). This study reported a significant decrease in SOD activity of CUT rats when compared to control which may be due to generation of reactive oxygen species overwhelming the antioxidant defense system of the body. The up-regulation of renal SOD activity in the quail egg treated groups may be due to the presence of nutritional constituents such as vitamin A, E and zinc which are known to exert antioxidant effects.

The present study showed significant decrease in sperm count, sperm motility and viability of cadmium induced untreated rats when compared to control. This result supports previous studies of Teiichiro *et al*. (2002) who reported decrease in sperm count in cadmium toxicity. The disruption of spermatogenesis in the testes due to cadmium toxicity may be attributed to competition of cadmium with zinc in zinc-containing enzymes and decreased activity of testis-specific enzymes (Caslino *et al*., 1997) thus impairing spermatogenic parameters. Treatment with quail egg significantly increased sperm count, motility and viability when compared with cadmium induced untreated rats which may be due to the presence of zinc as part of its constituents. Zinc, an essential micro-element has been implicated in DNA repair, maintenance and proliferative activity (Chasapis *et al.,* 2012). Inducible nitric oxide synthase (iNOS) is expressed in activated machrophages and cells of macrophage/monocyte

lineage as an immune response during inflammation and tumor development (Liu and Huang, 2008). This study observed an aggregation and specific iNOS expression in the Cadmium-untreated kidneys. Administration of quail egg resulted in reduced-expression of iNOS in post-treated rats and delayed cellular aggregation of iNOS in quail egg pre-treated rats. The ability of quail egg to reverse hematological alterations and protect against toxicity might have helped to potentiate the delayed expression and reduced-expression of iNOS.

In conclusion, quail egg consumption reduces the toxic effects of cadmium exposure on haematological profile, spermatogenic indices, renal antioxidants, and inflammatory level. This protective effect may be mediated via an anti-oxidative mechanism.

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REFERENCES

Agency for Toxic Substances and Disease Registry. Toxicological Profile of Cadmium (update). Atlanta, Georgia, 1999, pp.1-397.

Badkoobeh, P., Parmark, Kalantar S. M., Hosseini S. D. And Salabar A. 2013. Effect of nano-zinc oxide on doxoribino-induced oxidative stress and sperm disoders in adult male Wistar rats. *Iran J. Repro. Med.* 11 (9): 355-364.

Bagchi, D., Bagchi, M., Hassoun, E. A. and Stohs, S. J. 1996. Cadmium induced excretion of urinary lipid metabolites, DNA damage, glutathione depletion and hepatic lipid peroxidation in sprage-dawley rats. Biol Trace Elem Res, 52(2) 143.

Baylil, C. 2008. Nitric oxide deficiency in chronic kidney disease. Gottschalk Lecture 2007. AJP Renal. 29: F1–F9.

Caslino, E., Sblano, C. and Landriscina, C. 1997. Enzyme activity alteration by cadmium administration to rats: the possibility of iron involvement in lipid peroxidation. Arch. Biochem. Biophys; 346:171-179.

Chasapis, C.T., Loutsidou, A.C., Spiliopoulou, C.A., and Stefanidou, M.E. 2012: Zinc and human health: an update. Archives of Toxicology; 86(4), 521-534.

Choi, J.H. and Rhee, S.J. 2003. Effects of Vit. E on renal dysfunction in chronic Cd poisoned rats. J. Of Med.Food 6(3)209-215.

Dan G, Lall SB, Rao DN.2000. Humoral and cell mediated immune response to cadmium in mice. Drug Chem Toxicol., 23(2):349-60.

Diamanti-Kandarakis, E., Bourguignon, J., Giudice, L. C., Hauser, R., Prins, G. S., Soto, A.M., Thomas Zoeller, R. and Andrea, C. G. 2009. Endocrine-Disrupting Chemicals: An Endocrine Society Scientific Statement. *Endocrine Reviews* 30(4):293-342.

El-Demerdash, F.M., Yousef, M.I., Kedwany, F.S. and Baghdadi, H.H. 2004. Cadmium-induced changes in lipid peroxidation, blood hematology, biochemical parameters and semen quality of male rats: protective role of vitamin E and beta-carotene. Food Chem. Toxicol. 42(10): 1563-1571.

Fowler B.A. 2009 Monitoring of human populations for early markers of cadmium toxicity: a review. Toxicology and Applied Pharmacology, 238:294-300

Gluhcheva, Y., Ivanov, I. and Atanasov, V.2011. Hematological changes in case of chronic cadmium intoxication and monensin detoxication. Relationship with rheological variables, Clinical Hemorheology and Microcirculation. 49, 417-422.

Godt, J., Scheidig, F., Grosse-Siestrup, C., Esche, V., Brandenburg, P., Re-ich ,A. and Groneberg, D.A. 2006. The toxicity of cadmium and resulting hazards for human health. J Occup Med Toxicol 1: 22.

Griess P., 1987. Analysis of nitrate and nitrite in body fluids. Berichte der deutshen chemischen 12 (2):426-428

Haase H, Ober-Blöbaum JL, Engelhardt G, Hebel S, Rink L. 2010. Cadmium ions induce monocytic production of tumor necrosis factor-alpha by inhibiting mitogen activated protein kinase dephosphorylation.Toxicol Lett.;198(2):152-8.

Horiguchi, M. Sato, N Konno, M. Fukushima, 1996. Long-term cadmium exposure induces anemia in rats through hypoinduction of erythropoietin in the kidneys (1996). Arch Toxicol. 71(1-2) 11-19

Ige, S. F., Akhigbe, R. E., Edeogho, O., Ajao, F.O., Owolabi, O.Q., Oyekunle, O. S. and Ajayi, A.F. 2011. Hepatoprotective Activities of Allium Cepa in Cadmium-Treated Rats. *Int J Pharm Pharm Sci*, **3**: (5) 60-63

Järup, L., Berglund, M., Elinder C.G., Nordberg, G. and Vahter, M. 1998. Health effects of cadmium exposure- a review of the literature and a risk estimate. Scand J. Work Environ. Health 24 (Suppl 1), 1-5.

Johnson, R.C., Hounkpatin, A.S.Y., Guédénon, P., Domingo, E., Alimba, C.G., Boko, M. and Edorh, P.A. 2012.Protective Effects of Vitamin C on Haematological Parameters in Intoxicated Wistar Rats with Cadmium, Mercury and Combined Cadmium and Mercury International Research *Journal* of Biological Sciences Vol. 1(8), 76-81.

Kataranovski M, Kataranovski D, Savic D, Jovcic G, Bogdanovic Z, Jovanovic T. Granulocyte and plasma cytokine activity in acute cadmium intoxication in rats. Physiol Res. 1998;47(6):453-61.

Kataranovski M., Mirkov I., Belij S., Nikolic M., Zolotarevski L., Ciric D., Kataranovski D. 2009. Lungs: remote inflammatory target of systemic cadmium administration in rats. *Environ Toxicol Pharmacol* **28**: 225–231.

Kowalczyk, E., Kopff, A., Fijalkowski, P., Kopff, M., Niewarok, J., Blaszezyk, J., Kedziora, J. and Tyslerowicz, P. 2003. Effect of anthocyanins on selected biochemical parameters in rats exposed to cadmium. Acta Biochim Pol; 50(2):543–8.

Lafuente A, González-Carracedo A, Esquifino A.I. 2004 Differential effects of cadmium on blood lymphocyte subsets. Biometals 17(4): 451 - 456.

Linnoila I. and Petrusz P. 1984. Immunohistochemical staining techniques and their Applications in Histopathology of the Respiratory System. *Environ. Health Perspectives* 56: 131-148.

Liu V. W. T., Huang P. L. 2008. Cardiovascular roles of nitric oxide: A review of insights from nitric oxide synthase gene disrupted mice, *Cardiovascular Research*; 77:19 – 29

Longe, L. 2005. Cadmium Poisoning. The Gale *Encyclopaedia of Alternative Medicine.* Vol. 1, 2nd edition.

Mahran, A.I., Husam, E. H. O., Ahmed, M.A. A. and Adel, M. A. 2011. Protective Effect of zinc (zn) on the histology and histochemistry of liver and kidney of Albino rat treated with cadmium. *J Cytol Histol*, **2**:4.

Menke A., Plaza-Tellez M., Acien – Navas A. 2012. Cadmium Exposure and All-Cause and Cardiovascular Mortality in the U.S General Population. Environmental Health Perspectives.120, 1017 -1022.

Misra, H., Fridovich, I. 1972. The role of superoxide anion in the auto-oxidation of epinephrine anion and a simple assay of superoxide dismutase. *J. Biol. Chem.*; 247:3170-3175

Nobuhiko, M. 2009. Individual Susceptibility to Cadmium Toxicity and Metallothionein Gene Polymorphisms: with References to Current Status of Occupational Cadmium Exposure. *Industrial. Health*. 47:487–494.

Ola-Mudathir, K.F., Suru, S.M, Fafunso, M.A., Obioha, U.E. and Faremi, T.Y 2008. Protective roles of onion and garlic extrac*ts on cadmium induced change*s in sperm characteristics and testicular oxidative damage in rats. Food Chem. Toxicol. 46(12): 3604-3611.

Oliveira, H., Lopes, T., Almeida, T., Pereira Mde, L. and Santos, C. 2012. Cadmium-induced genetic instability in mice testis. Hum Exp Toxicol.; 31(12):1228-36.

Onwuka, F.C., erhabo, O., Etengs, M. U. and Umoh, I. B. 2010. Ameliorative effect of cabbage extract on cadmium-induced changes on hematology and biochemical parameters of albino rats. *J. Toxicol. Environ. Health Sci.* 2(2): 11-16

Rekha, D.K., Tripathi, Y., Raghuveer, C.V., Sheila, R.P., Ramaswamy, C. and Priya, K. 2011. Role of vitamin c as an antioxidant in cadmium chloride Induced testicular damage. Int J. App.Biol. & Pharm Tech. 2(3):484-488.

Satarug, S., Baker, J. R., Urbenjapo, S., Haswell-Elkins, M., Reilly, P. E., Williams, D.J. and Moore, M. R., 2003. A global perspective on Cadmium pollution and toxicity in non-occupationally exposed population, Toxicol. Lett, 13765.

Setchell, B.P. and Brooks, D.E. Anatomy, vasculature, innervations and fluids of the male reproductive tract. 1998 In: Knobil, E., and Neil, J.D., eds The physiology of Reproduction. New York, NY; Raven. 34; 753–836.

Shaikh, Z.A., Thanhtan, T.Vu and Zaman, K. 1999. Oxidative stress as a mechanism of chronic Cd-induced hepatotoxicity and renal toxicity and protection by antioxidant. Toxicol. Appl. Pharmacol 123:89-96.

Shirash,i N., Uno, H. and Waalkes, M.P. 1993. Effect of L-ascorbic acid pretreatment on cadmium toxicity in the male Fischer rat. Toxicol., 85 (2-3): 85-100.

Siu, E.R., Mruk,, D.D., Porto, C.S. and Cheng, C.Y. 2009. Cadmium-induced testicular injury. Toxicol. Appl. Pharmacol. 238(3): 240-249.

Smalinskiene, A., Ryselis, S., Abdrakhmanov, O., Kregzdyte, R., Sadauskiene, I. and Ivanon, L. 2004.Investigation of cadmium concentration after acute intoxication, biologija ,2(1) 113.

Smalinskienel, A., Lesauskaitel, V., Ryselisl, S., Abdrakhmanov, O. and Kregzdyte, R. 2006. Effects of six-week intoxication on cadmium and zinc distribution in internal organs and blood and on the mitotic activity of liver cells. *Biologija*, **1**: 76-79.

Smith J.W., Castro G.A. 1978. Relation of peroxidase activity in gut mucosa to inflammation. Am. J. Physiol. 234: R72–R79.

Stosic, J., Mirkov, I., Belij S., Nikolic, M., Popov, A., Kataranovski, D. and Kataranovski, M. 2010. Gender differences in pulmonary inflammation following systemic cadmium administration in rats. *Biomed Environ Sci* **23**: 293–299.

Takiguchi, M. and Yoshihara, S. 2006. New Aspect of cadmium as an Endocrine disruptor. *Environmental Sci*., 13(2): 107-116.

Teiichiro, A., Hirmichi, I., keisuke, M., Kunihiro, H., and Makoto, H. 2002. Cadmium induced testicular damage in a rat model of subchronic intoxication. Reprod Med. Biol., 1: 59-63.

Theron AJ, Tintinger GR, Anderson R Harmful Interactions of Non-Essential Heavy Metals with Cells of the Innate Immune System. J Clinic Toxicol S3:005. doi: 10.4172/2161-0495.S3-005

Tunsaringkarn T., Tungjaroenchai W., Siriwong W. 2013. Nutrient Benefits of Quail (Coturnix Coturnix Japonica) Eggs. International Journal of Scientific and Research Publications, 3(5) 1 - 8. ISSN 2250-3153

Olszowski Tomasz, Baranowska-Bosiacka Irena, Gutowska Izabela and Chlubek. Dariusz 2012. Pro-inflammatory properties of cadmium. Acta Biochim Pol. 59(4) 2012 475-82.

Waalkes, M. 2003. Cadmium carcinogenesis. Mutation Research; 533:107-120.

Zhao, Z., Hyun, J.S., Satsu, H., Kakuta, S. and Shimizu, M. 2006. Oral exposure to cadmium chloride triggers an acute inflammatory response in the intestines of mice, initiated by the over-expression of tissue macrophage inflammatory protein-2 mRNA.*Toxicol.Lett* 164: 144