Basrah Journal of Veterinary Research 2026; 25(1):48-61

Print ISSN: 1813-8497
Online ISSN: 2410-8456
https://bjvr.uobasrah.edu.iq/

Effect of Glutathione on Reproductive Efficiency in Male Rock Pigeons (*Columba livia gaddi*) Treated With di (2-ethyle hexyl) phthalate

Article Info.

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Article History

Received: Oct. 15, 2025
Accepted: Jan. 19, 2026
Published: March 31, 2026
Article type: **Research Article**

https://doi.org/10.23975/bjvr.2026.166263.1253

Abstract

The present investigation assessed the reproductive toxicity caused by di(2-ethylhexyl) phthalate (DEHP) and the modulating effect of reduced glutathione (GSH) in adult male Rock Dove pigeons (Columba livia gaddi). Sixty pigeons (>10 months old) were randomly allotted into groups of fifteen for 28 days; control groups received saline (1 mL), treatment groups received GSH (0.55 mg/kg BW), treatment groups received DEHP (400 mg/kg BW; below the LD50 value of 435.6 mg/kg BW), and the combined treatment of DEHP+GSH. DEHP is pervasive in the plastic and environmental media; therefore, exposure in birds is possible and likely. The treatment was designed to determine the effect of GSH supplementation in reducing the DEHP induced damage in the reproductive cells by assessing the functional parameter of both sperm and the testosterone levels in the pigeon model. Weight of the testicular tissue, epididymal sperm indexes, and blood testosterone were measured; testosterone was measured using a competitive ELISA. In the presence of GSH alone, there was no harm to the biological tissue measurements. There was a significant decrease in testosterone to 1.77±0.36 IU/ml (43.1% lower than control values), along with a decrease in the weight of the right/left side of the testis to 0.99±1.07 g and 1.10±0.58 g. Regarding the sperm concentration values, the results were lower in the DEHP-exposed groups (0.91±0.16×10⁶/mm^3; 86.1% lower compared to control groups), as well as the percentage of live sperm (0.18±4.42% lower compared to control groups). Co-exposure mitigated the effect to some extent on the sperm concentration (1.17×10⁶/mm^3; $p<0.01$) and live sperm (24.88% lower; $p<0.001$.

Keywords: Male, Glutathione, DEHP, Pigeon.

Publisher: University of Basrah,
College of Veterinary Medicine, Basrah, Iraq

Introduction

Endocrine-disrupting chemicals (EDCs) and environmental pollutants cause great risk to animal as well as human health (1). Di(2-ethylhexyl) phthalate (DEHP), a versatile plasticizer for use in manufacturing flexible plastics, has been greatly in the focus of attention owing to its high incidence in the environment and on account of the interference it exerts on hormone homeostasis (2). DEHP finds extensive usage in consumer articles, medical implants, packaging items و and plant protection products (3). After environmental release, it can penetrate soil, water, and air, making exposure practically unavoidable for fauna and humans. DEHP, being lipophilic, has a tendency to bioaccumulate in adipose tissues and can cross biological membranes, such as the blood-testis barrier, thus impairing testicular function and male fertility (4). Various studies have indicated that DEHP and its primary metabolite, mono-(2-ethylhexyl) phthalate (MEHP), may cause reproductive dysfunction through interference with steroidogenesis, the production of oxidative stress, and histological alterations in the testes (5).

DEHP exposure in male mammals has been linked to decreased testosterone levels, defective spermatogenesis, decreased sperm motility, and sperm morphological abnormalities (6). The mechanisms are primarily mediated by the production of reactive oxygen species (ROS), mitochondrial impairment, and disruption of the hypothalamic-pituitary-gonadal (HPG) axis (7). Avian models, such as pigeons (Columba livia), are widely utilized as model species in reproductive endocrinology to assess the toxicological effects of environmental pollutants, especially on reproductive function and endocrine disruptions (8). Testosterone, the major male sex hormone produced mainly by Leydig cells in the testes, is a key regulator of spermatogenesis, sexual behavior, secondary sexual traits, and fertility (9). Testosterone synthesis is strictly controlled by the HPG axis through the pulsatile release of gonadotropin-releasing hormone (GnRH), which stimulates the anterior pituitary to release luteinizing hormone (LH) (10).

Luteinizing hormone activates the testosterone synthesis of the Leydig cells through the cAMP pathway. Disrupting such a finely tuned axis as DEHP causes serious impairment of reproductive function (11). In determining toxicity for the type of endocrine disruption by chemicals such as DEHP, quantification of testosterone is a critically important goal. Antioxidant treatment has shown encouraging effectiveness among the new methods to reverse the toxic actions of DEHP and other endocrine-disrupting chemicals (EDCs) (12). Glutathione (GSH), a cysteine, glutamate, and glycine tripeptide, is one of the most efficient intracellular antioxidants (13). It is one of the most crucial molecules to neutralize reactive oxygen species, redox homeostasis, xenobiotic detoxification, and cellular integrity maintenance (14).

Glutathione is found ubiquitously in the reproductive tissues and has been revealed to be protective against testicular function by blocking lipid peroxidation and catalyzing enzymic activity demanded in steroidogenesis (15). Experimental findings indicated that supplementation with GSH was able to inhibit testicular oxidative stress-evoked injury and restore normal reproductive function in numerous animal models (16). In these situations, it is critically important to find out

whether glutathione has the capability of inhibiting the toxic effects of DEHP against the male reproductive system (17). Although mammalian models have provided extensive information about glutathione's role in protection during reproduction, there is a significant lack of research on avian models, particularly in pigeons, which are relevant due to their important roles in both ecological and physiological contexts (18). Regarding reproductive traits such as hormonal control and spermatogenic cycles, pigeons have much in common with other birds and mammals, which predisposes them to the study of the influence of environmental pollutants and possible protective medication (19). Moreover, enzyme-linked immunosorbent assay (ELISA) measurement of testosterone offers a reliable and sensitive evaluation of endocrine function. Competitive ELISA assays enable precise quantitation of serum content of testosterone based on specific antigen-antibody pairs and color development. Incorporation of testosterone assays in toxicological studies enables the examination of hormone disruption and intervention efficiency, such as glutathione supplementation (20).

The study seeks to examine the protective effect of glutathione on DEHP-induced testicular toxicity in male pigeons. In the current investigation, the effects of DEHP on testicular weight, sperm characteristics, and serum testosterone level are assessed, and the possibility of reversal of toxic effects by glutathione treatment is also investigated. This investigation adds to the growing evidence in favor of antioxidant therapy as a treatment against environmental reproductive toxicants by incorporating biochemical, histological, and hormonal evaluation.

Material and Methods

Experimental Design and Ethical Problems

The experiment was conducted according to the ethical guidelines of animal research and was approved by the Institutional Animal Care and Use Committee (IACUC) at the College of Veterinary Medicine, University of Mosul (Approval No.: UM.VET.2023.030, dated 4/10/2023). A total of sixty male Rock Dove pigeons (*Columba livia gaddi*) aged over 10 months and weighing 115–145 g were randomly divided into four groups (n = 15 per group). The experiment was conducted for 28 consecutive days.

The groups were treated in the following manner:

Group 1 (Control): Administered 1 ml of normal saline orally.

Group 2 (Glutathione): From Sigma Aldridge company and CAS Number (70-18-8), Co-treated with reduced glutathione at a dosage of 0.55 mg/kg BW orally according to co. protocol.

Group 3 (DEHP): From Sigma Aldridge company and CAS Number (117-81-7). Given Di(2-ethylhexyl) phthalate (DEHP) at a dosage of 400 mg/kg BW orally according to the co. protocol.

Group 4: Co-treated with glutathione and DEHP as used to treat Groups 2 and 3, respectively.

Table (1): Measure the Median Lethal Dose (LD-50).

The second part of the series	K: Represent the test series that begins as follows O	OO	OOO	OOOO		Standard error (LD-50)
XOOO	157 -.0	154 -.0	154 -.0	154 -.0	OXXX	**σ 0,61**
XOOX	878 -.0	861 -.0	860-.0	860 -.0	OXXO	
XOXO	701.0	737.0	741.0	741.0	OXOX	
XOXX	084.0	169.0	181.0	182,0	OXOO	
XXOO	305.0	372.0	380.0	381.0	OOXX	
XXOX	305 -.0	169 -.0	144 -.0	142 -.0	OOXO	
XXXO	288.1	500.1	544.1	549.1	OOOX	
XXXX	555.0	897.0	985.0	000,1	OOOO	
	X	XX	XXX	XXXX	**The second part of the series**	
	K: Represent the test series that begins as follows					

LD_{50} Determination

The median lethal dose (LD_{50}) of DEHP was determined by the up-and-down method according to previously used methodology. The LD_{50} value was determined to be 435.6 mg/kg BW. The sub-lethal dose of 400 mg/kg was used for the experiment to induce testicular toxicity but not death Table1.

Sample Collection and Tissue Preparation

After the experimental duration, all birds were euthanatized humanely under deep chloroform anesthesia. Testes were excised and weighed to calculate the organ relative weight. Testes samples were examined for gross morphological changes.

The epididymal heads were treated for sperm concentration by haemocytometer after formalin-eosin staining. The epididymal tails were treated for estimation of sperm viability and morphological integrity by the eosin-nigrosin staining technique. Smears of the semen of individual birds were prepared, and a minimum of 200 spermatozoa were examined under a light microscope (×400 magnification), and percentages of live, dead, and morphologically deformed sperm.

Testosterone Assay

Serum testosterone concentrations were detected using a competitive enzyme-linked immunosorbent assay (ELISA) kit (Cat No. PT872, Solarbio, China) according to the protocol

instructions. In brief, the diluted serum samples in proportion were added into pre-coated wells of anti-testosterone antibody of the microplates. Subsequently, horseradish peroxidase-labelled testosterone conjugate was added, and the plate incubation was carried out at room temperature (25–28°C) and in darkness for 2 hours. After three washes of wells, they were incubated with tetramethylbenzidine (TMB) substrate for 15–20 minutes. The reaction was stopped using the provided stop solution, and the absorbance was read at 450 nm using a microplate reader (21).

The calibration curve was constructed with the testosterone standards presented below: 0, 0.3125, 0.625, 1.25, 2.5, 5, 10, and 20 ng/ml. The absorbance values were plotted against the known values, and the sample concentrations were derived from a four-parameter logistic curve. Precision was acquired with duplicate runs on all samples and standards (22).

Statistical Analysis

All the data was analyzed with SPSS version 26.0 (IBM Corp., Armonk, NY, USA). One-way analysis of variance (ANOVA) was used to compare mean values between groups. Duncan's multiple range test was used for post hoc analysis if differences were found to be significant (23). Data were expressed as mean ± standard error (SE), and the level of statistical significance was set at $P < 0.05$.

Results

Determination of median lethal dose (LD50) of Plasticizer di(2-ethylhexyl) phthalate:

The median lethal dose (LD_{50}) of the plasticizer di(2-ethylhexyl) phthalate (DEHP) was determined in a Rock Dove pigeon (*Columba livia*) model using the up-and-down procedure described previously. The procedure has worldwide acceptance for reducing the number of animals used while still providing statistically valid LD_{50} estimates. Five male adult Rock Doves of weights 155 to 170 grams each were selected for the experiment. The birds were maintained singly in standard laboratory conditions with free access to food and water (Table 1).

The first median lethal dose of oral DEHP was selected to be 400 mg/kg body weight based on the data generated by previous toxicological studies carried out on avian and rodent models. The DEHP was administered by gavage orally using a sterile feeding needle. Post-dosing, each bird was kept under close observation for 24 hours to monitor the emergence of clinical manifestations of acute toxicity, such as lethargy, ataxia, dyspnea, convulsions, and mortality. Based on the response of each bird (mortality or survival), follow-up doses were calculated following the up-and-down method according to Table 2. This method permitted the effective estimation of the LD_{50} value with reduced animal use and distress.

Table2. ″Median Lethal Dose (LD_{50}) of" oral di(2-ethylhexyl) phthalate by up and down method.

Variable	Results
LD_{50} mg/kg Bw	435.6
range of utilized dose of the doses utilized (mg/kg Bw)	500-350=150
first dose (mg/kg Bw)	400
final dose (mg/kg Bw)	500
Increase or decrease in the dose (mg/kg)	50
Number of birds involved	(XOOOX) 5

X: Death,
O: A Live,
The LD_{50} was calculated by the up-and-down method.

Variations in testes weight and testosterone levels across different experimental groups:

Differences in testicular weights (both right and left) and plasma testosterone concentration were found between the control group and glutathione-treated group, and therefore glutathione per se does not impair male reproductive parameters. In contrast, treatment with di(2-ethylhexyl) phthalate (DEHP) resulted in a significant reduction ($P < 0.05$) in testicular weights and plasma testosterone levels compared to the glutathione alone and control groups. These findings reflect that DEHP exhibits a robust testicular toxicity, perhaps due to its endocrine-disrupting potential and oxidative stress-induced activity (Table 3).

In addition, co-administration of DEHP + glutathione (DEHP + GSH group) also showed a reduced ($P < 0.05$) weight of the testis and testosterone concentration compared to the control group. Although glutathione has antioxidant properties, it was not able to reverse the damaging effects induced by DEHP using this model. The results indicate that the protective action of glutathione failed to reverse the testicular toxicity and endocrine disruption caused by DEHP exposure, reflecting the extent of DEHP-induced damage and the possibility of further effective or combined antioxidant therapies.

Table3. Variations in testes weight and testosterone levels across different experimental groups in the study.

PARAMETERS GROUPS	"Right testis weight /g"	"Left testis weight /g"	Testosterone (IU/ml)
Control normal saline (1ml/ birds) orally	1.45 ± 0.96 a	1.52 ± 0.92 a	3.11 ± 0.33 b
Glutathione (0.55 mg/kg BW) orally	1.41 ± 1.00 ab	1.48 ± 0.75 ab	3.98 ± 0.78 a
(DEHP) (400mg/kg) orally	0.99 ± 1.07 d	1.10 ± 0.58 c	1.77 ± 0.36 c
Glutathione (0.55 mg/kg Bw) + DEHP) (400mg/kg) orally	1.10 ± 1.04 c	1.19 ± 0.61 c	1.82 ± 1.12 c

Values represent:(mean±SD) Different in letters (a,b,c, and d) in superscript "indicate significant difference between groups P<0.05.

Variations in sperm concentration, live, dead, and abnormal across different experimental groups in the study:

Sperm number and quality parameters, including live, dead, and abnormal percentages of sperm, were quite different among the experimental groups. No statistically significant difference was found in sperm number or viability between the control group and the group treated with glutathione alone (0.55 mg/kg BW). Both groups possessed elevated sperm densities (6.53 ± 0.22 and 6.02 ± 2.01 × 10^6/mm^3, respectively) and elevated live sperm rates (89.35 ± 3.11% and 90.10 ± 0.13%, respectively), and reduced dead and abnormal sperm occurrence (Table 3).

Conversely, DEHP (400 mg/kg BW) resulted in a significant ($P < 0.05$) reduction in sperm count (0.91 ± 0.16 × 10^6/mm^3) and percentage of live sperm (0.18 ± 4.42%) and an increased rate of dead and abnormal sperm, indicating severe testicular toxicity. Co-treatment of glutathione and DEHP reduced the negative effects partially, with the improved sperm count (1.17 ± 5.20 × 10^6/mm^3) and live sperm percentage (24.88 ± 2.91%), though the results were still remarkably lower than the control and glutathione-alone groups. These findings show that glutathione offers a degree of protection against DEHP-induced reproductive toxicity, but insufficient to fully normalize sperm parameters (Figure 1).

Table 4. Variation in Sperm concentration, live, dead and abnormal sperm

PARAMETERS GROUPS	Right testis weight /g	Left testis weight /g	Testosterone (IU/ml)
Control normal saline (1ml/ birds) orally	1.45 ± 0.96 a	1.52 ± 0.92 a	3.11 ± 0.33 b
Glutathione (0.55 mg/kg BW) orally	1.41 ± 1.00 ab	1.48 ± 0.75 ab	3.98 ± 0.78 a
(DEHP) (400mg/kg) orally	0.99 ± 1.07 d	1.10 ± 0.58 c	1.77 ± 0.36 c
Glutathione (0.55 mg/kg Bw) + DEHP) (400mg/kg) orally	1.10 ± 1.04 c	1.19 ± 0.61 c	1.82 ± 1.12 c

Values represent (mean ± SD or SE) and d Different letters (a, b, and c) in superscript indicate significant difference between treated groups P<0.05.

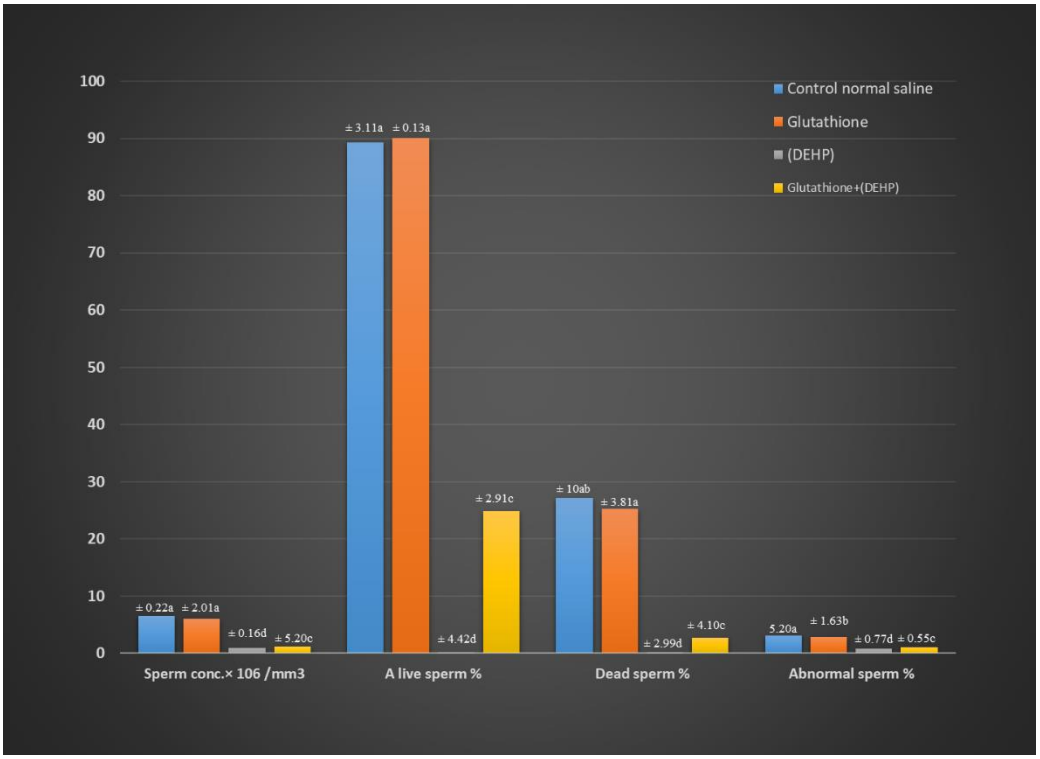

Figure1. The influence of different treatments on sperm concentration (×10⁶/mm³) and percentages of live, dead, and abnormal sperms in adult male Columba livia (Rock Dove). Groups are: Control (normal saline), Glutathione (0.55 mg/kg BW), DEHP (400 mg/kg BW), and Glutathione + DEHP. Values represented as mean ± standard deviation (SD). Different letters above bars indicate statistically significant differences between groups (P < 0.05).

Serum Testosterone Levels Assayed by ELISA

Serum testosterone levels were determined by competitive enzyme-linked immunosorbent assay (ELISA). A calibration curve was prepared using serial dilutions of testosterone standard between 0, 0.3125, 0.625, 1.25, 2.5, 5, 10, and 20 ng/ml. 450 nm absorbance was measured and modeled to a four-parameter logistic (4PL) model. The resulting standard curve had a very good inverse relationship between testosterone concentration and absorbance with an R^2 value of greater than 0.98, demonstrating the sensitivity and reliability of the assay (Figure 2).

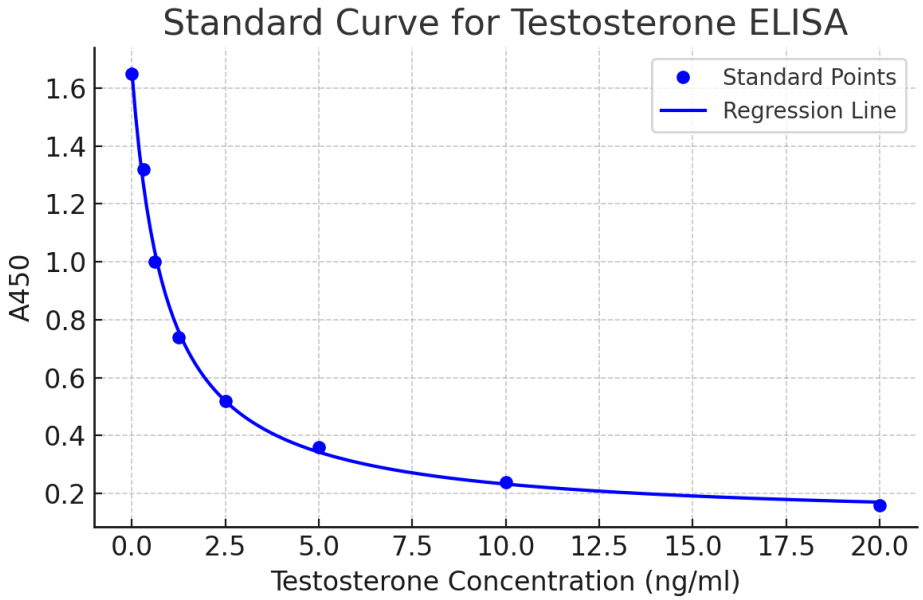

Figure2. Standard curve for testosterone as standard start concentrations among (0–20 ng/ml) are indicated by blue circles.

Discussion

This study examined the reproductive toxicity of di(2-ethylhexyl) phthalate (DEHP) in male pigeons and determined the protective effect of glutathione (GSH) on DEHP-induced toxicity. The findings revealed that DEHP exposure caused profound impacts on testicular morphology, quality of sperm, and testosterone content in the serum, but glutathione co-administration could overcome these toxic effects (24). These findings align with other research on the toxicities of DEHP and support the effectiveness of antioxidants against reproductive harm generated by environmental chemicals (12, 24).

DEHP is a ubiquitous plasticizer with endocrine-disrupting activity, and most notably, it is anti-androgenic (25,26). DEHP treatment in the present work led to dose-dependent weight loss in the testes as well as gross histological evidence of atrophy, such as pale and desiccated testes (27).

These results were consistent with previous observations of histopathological changes reported in testicular tissue following phthalate exposure that include seminiferous tubule degeneration and loss of germ cells (28). Reduced testicular weight reported in the current study is most likely to reflect structural disruption as well as functional suppression of spermatogenesis (6).

Sperm analysis further highlighted the harmful effects of DEHP on male fertility. Birds exposed to DEHP showed a clear and troubling decline in sperm count, motility, and overall quality, along with a noticeable increase in abnormal sperm shapes (29). These results support previous research indicating that DEHP interferes with the normal process of sperm production and maturation in the epididymis, largely through mechanisms involving oxidative stress and disruptions to hormonal signaling (30). Because DEHP is fat-soluble, it tends to accumulate in reproductive tissues, where it can cause mitochondrial dysfunction, upset the balance of cellular processes, and trigger cell death in developing sperm (17).

Importantly, the study also revealed a significant drop in serum testosterone levels in the DEHP-treated group (32). The most noted effect of phthalate exposure is hormonal suppression and is believed to be caused by interference with key enzymes in testosterone synthesis by the Leydig cells (33). In contrast, testosterone is important for the maintenance of spermogenesis, testicular structure, as well as secondary male sexual features; its suppression would most likely be accountable for the reproductive effects seen in this study (34).

Encouragingly, the birds treated with glutathione and DEHP both improved. These birds had higher weights in their testes, improved sperm profiles, and much higher levels of testosterone than birds treated with DEHP only. Glutathione is an endogenous antioxidant found in cells that can neutralize harmful reactive oxygen species and protect cells from oxidative stress. Its ability to ease the reproductive harm caused by DEHP highlights how oxidative stress is a central factor in phthalate toxicity (12). These results are consistent with earlier studies in rodents, where antioxidants have been shown to help prevent reproductive damage caused by environmental toxins like DEHP (35).

However, there was absolutely no difference in any of the tested parameters between the glutathione-alone groups and the controls, which points to glutathione supplementation having no way whatsoever of encroaching upon normal reproductive function and also being safe at this dose. That only makes it an even stronger candidate for an agent of prophylaxis or therapy against environmentally generated neutralizing reproductive toxins.

This study describes strong evidence for DEHP causing damage to the reproductive health of males in pigeons belonging to its endocrine-disrupting activity category. Mechanism of glutathione acts as a cellular homeostasis preservation. These findings have wildlife implications and potentially for human health, where phthalate exposure is not specific.

Conclusion

In the current investigation, the findings clearly supported the fact that Di(2-ethylhexyl) phthalate (DEHP) causes severe reproductive toxicity in male pigeons, which is apparent in the reduced weight and motility score of sperm, along with reduced serum testosterone. These toxic effects are closely related to the generation of oxidative stress and the endocrine disruption effects. The protective effect provided by the simultaneous administration of the potent intracellular antioxidant, glutathione, reduced the DEHP-induced toxic effects by partially modifying the serum testosterone.

Conflicts of interest

The authors declare that there is no conflict of interest.

Ethical Clearance

This work is approved by The Research Ethical Committee.

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تأثير الجلوتاثيون على الكفاءة التناسلية لدى ذكور حمام الصخور (كولومبا ليفيا غادي) المعالجة بثنائي (2-إيثيل هيكسيل) فثالات

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الخلاصة

هدفت الدراسة الحالية إلى تقييم السمية التناسلية الناتجة عن ثنائي (2-إيثيل هكسيل) الفثالات (DEHP) وتأثير الجلوتاثيون المختزل (GSH) المُعدِّل لهذه السمية في ذكور حمام الصخور البالغة *(Columba livia gaddi)*. شملت الدراسة ستين حمامة يزيد عمرها على 10 أشهر، وُزِّعت عشوائيًا إلى أربع مجموعات (15 طائرًا لكل مجموعة) لمدة 28 يومًا؛ حيث تلقت مجموعة السيطرة محلولًا ملحيًا (1 مل)، وتلقت مجموعة المعالجة GSH بجرعة 0.55 ملغم/كغم من وزن الجسم، فيما تلقت مجموعة أخرى DEHP بجرعة 400 ملغم/كغم من وزن الجسم) أقل من قيمة الجرعة المميتة الوسطية LD_{50} البالغة 435.6 ملغم/كغم(، كما خضعت مجموعة رابعة للمعالجة المشتركة بـ DEHP + GSH. ونظرًا لانتشار DEHP على نطاق واسع في المواد البلاستيكية والوسط البيئي، فإن تعرّض الطيور له يُعد ممكنًا ومرجحًا. صُمِّمت المعالجة بهدف تحديد دور مكملات GSH في الحد من الأضرار التي يُحدثها DEHP في الخلايا التناسلية، وذلك من خلال تقييم المؤشرات الوظيفية لكلٍّ من الحيوانات المنوية ومستويات هرمون التستوستيرون في نموذج الحمام. جرى قياس وزن نسيج الخصية، ومؤشرات الحيوانات المنوية في البربخ، ومستوى التستوستيرون في الدم، حيث قيس التستوستيرون باستخدام اختبار الممتز المناعي المرتبط بالإنزيم (ELISA) التنافسي. أظهرت نتائج مجموعة GSH وحده عدم حدوث أي تأثيرات ضارة على القياسات النسيجية الحيوية. في المقابل، سُجِّل انخفاض معنوي في مستوى التستوستيرون ليبلغ 0.36±1.77 وحدة دولية/مل (بانخفاض قدره 43.1% مقارنةً بقيم مجموعة السيطرة)، ترافق مع انخفاض وزن الخصية اليمنى واليسرى إلى 0.99±1.07 غم و 0.58±1.10غم، على التوالي. وفيما يخص تركيز الحيوانات المنوية، فقد انخفضت القيم في المجموعات المعرضة لـ DEHP إلى/10^6×0.16±0.91 مم^3 (بانخفاض قدره 86.1% مقارنةً بمجموعة السيطرة)، وكذلك انخفضت نسبة الحيوانات المنوية الحية (بانخفاض قدره 44.18% مقارنةً بالسيطرة). أما التعرض المشترك لـ DEHP و GSHفقد خفّف من هذه التأثيرات إلى حدٍّ ما، حيث تحسّن تركيز الحيوانات المنوية إلى /10^6×1.17مم^3 ($p<0.01$) وارتفعت نسبة الحيوانات المنوية الحية (بانخفاض أقل بلغ 24.88%؛ ($p<0.001$) .

الكلمات المفتاحية: ذكر, جلوتاثيون, DEHP , الحمام.