Iraqi Journal of Veterinary Sciences

www.vetmedmosul.com

Potency of nano-zinc oxide on caspase-3 of male quail exposed to lipopolysaccharide

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Article information

Article history:
Received 06 July, 2023
Accepted 27 October, 2023
Available online 17 December, 2023

Keywords:
Apoptosis
Johnsen's score
Testosterone
Spermatogenesis

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Abstract

It is known that lipopolysaccharide-induced endotoxemia promotes male sterility. This research project intended to examine the repercussions of nano-zinc oxide on caspase-3 and sex competence in male quail lipopolysaccharide-exposed cells. The quail were distributed at randomization into four groups; the first collection of birds was allowed to receive the usual feed and was used as a control, while the second collection was injected intraperitoneally LPS twice weekly at a prescribed dosage of 1 mg/kg body weight, the third collection was given nano-zinc oxide at a prescribed dosage of 40 mg/kg fodder for six weeks. The four-group collection was treated with the same nano zinc oxide particle dose and injected LPS at the same decided dose. The result indicates that management of the LPS effect significantly drops in testosterone, live sperm, sperm count, testis morphometric, and Johnsen's scoring, accompanied by a rise in dead, abnormal sperm and caspase-3 as linked with control. The application of NZO 40 mg/kg diet exhibited a higher significance in testosterone and live sperm with testicular criteria and Johnsen's scoring with drop dead, abnormal sperms, and caspase-3. Treated with nano-zinc oxide concurrently with LPS causes a rise in testosterone and Johnsen's scoring with a decrease in caspase-3 comparable to the LPS group and returns the value of testosterone hormone to normal value. It concluded that supplementation of nano zinc oxide is necessary to overcome the contamination with LPS, which negatively affects male sex characteristics, and nano zinc repairs that effect.

DOI: 10.33899/ijvs.2023.141585.3121,

Introduction

Bacterial lipopolysaccharide (LPS) modulates acute endotoxemia, a rapid inflammatory reaction in the host. A lively constituent of the external membrane of the cell barrier of the Gram-negative bacteria because LPS-induced animal models can mimic inflammatory diseases and endotoxemia, they can be used to examine the link between mechanisms of inflammatory illnesses and infertility (1). Across the past few generations, reliable proof proposes that oxidative stress in the testis can be linked to inflammatory conditions within (2). LPS stimulates the expulsion of cytokines that trigger inflammation like interleukins (1), tumor necrotic factor, interferon, and growth factor-transforming by attaching to receptors that are responsible and located on WBCs (3). Acute period responses such as infection, anorexia (4), renal failure (5), and endocrine alterations (6) are regulated by cytokines. One of the primary inflammatory mediators formed by monocytes and macrophages in reaction to an infection is interleukin-1 (IL-1) (7). Septicemia frequently exhibits decreased serum testosterone levels and elevated plasma IL-1 concentrations (8). A former investigation set that IL-1 is a potent blocker of Leydig cells' ability to produce androgen when stimulated by luteinizing hormone/human choriogonadotropin (hCG) (9). There have been indications that testosterone suppression can cause germinate cells to be driven to programmatic cell death (10) and that the released cytokines may be due to the disruption of Leydig cell steroidogenesis (11). However, recent studies revealed disagreement on androgen receptor localization in

germ cells (12). Later-stage germ cells do not require androgen receptors, as evidenced by the fact that the ability to reproduce and sperm generation were unaffected by germ cell-specific androgenic receptor knockout to prevent androgen receptor expression during or after (13). The sensitivity of progressive-phase germ cells to ROS activation appears to be caused by a decline in the superoxide dismutase enzyme activity and their expression of Cu/Zn and Zn levels as spermatogenesis progresses (14). As a result, spermatogonia may be less vulnerable to ROS attack because of their elevation amounts of Copper/Zinc, SOD, and Zinc (14). Recent findings demonstrated that LPS from a normal isolate significantly upsurges. The overall number of animals granulate cells, white blood cells, as well the activity of phagocytes (15). When treated with nano ZO particles and vitamin E, Taha and Ismail's research in 2023 found that the testis' histological changes were improved compared to the control group (16). The Nano ZO particles are materials ranging from 1 to 100 nm (17).

The current investigation aimed to determine whether NZO had protective effects on sex criteria after LPS exposure in male quails.

Materials and methods

Ethical acceptance

This research project was granted permission by the veterinary medicine college's institutional animal care and committee, with the certificate number UM.VET.2022.073.

Management of quail

This trial employed 60 male quail at three weeks of age sourced from Mosul hatchability and bred in the Animals housed at Mosul University's Veterinary College. All across the experiment, in May and June of 2022, the quails were reared in an air-conditioned chamber with a circadian rhythm of twelve hours of daylight and dark intervals and a temperate of 25 C˚±3C˚. They were divided into four collections, each with 15 birds housed in a wooden chip-lined floor cage. Prior to any treatment, the birds were permitted seven days to adapt and were given access to drinking fresh water and regular food, according to NRC (18).

The arrangement of experiment

After the quail reached the age of four weeks at a weighted range of 136–144 g, they were allocated at randomization. For six weeks, the first collection of birds was allowed to receive ordinary feed and twice weekly intraperitoneal injections of saline, which was used as a control, while the second collection was injected intraperitoneally with lipopolysaccharide from *Escherichia coli* (LPS) (Solarbio®, China, purified by phenol extraction) twice weekly (at 1 mg per kilogram of body weight) (19), the third collection given nano-zinc oxide made with a size of 20-30 nanometers (US Research Nanomaterials, Inc®., USA, purity 99.8) at a prescribed dosage of 40 mg/kg fodder (20) for six weeks. The final collection was treated with the same dose of nano zinc oxide particles, followed by injecting LPS at the same decided dose.

Gathering and processing of samples

After the test, the bird is slain, and its blood is collected. Serum must be obtained for testosterone hormone testing. After that, the gonads were dissected using a longitudinal abdominal incision, and one of the testes was immersed in buffer formalin for histological investigation. This was done following fixing, dehydrating, clearing, impregnation, and paraffin incorporation. The tissues were then cut into slices, and Harris Hematoxylin and Eosin staining (H&E), and the glass slices were analyzed to determine a seminiferous tubule's distance, the germinal epithelium's thickness layer at 100X magnification, gotten through an eyepiece and at 400X magnification in twenty tubular structures of every specimen, and another testis was placed in aluminum foil for detection caspase-3 by ELISA kit (Solarbio, China) by colorimetric assay at a wavelength to 405 nm. the mean number of Leydig and cells of Sertoli were counted as a mean of 5 measurements/field (60.08μm^2/400X) for five fields for every animal in the group by using the software camara (USB 2.0 digital image camara "Omax ToupVeiw 9.0-Megapexil China") calibration by uses the lenses of Microscopic-Olympus-CX31 through assistance 0.01mm stage micrometer (ESM-11/Japan). The epididymis was then dissected to collect semen, which was subsequently diluted with natural saline, and spermatogenic metrics, including the number of sperm, were determined using the Neubauer chamber and the equation as follows no. of sperm in 80 small square × 10000 = cell/mm^3 However, the sperm abnormality, live and dead percentages can be calculated by spreading one drop of eosin mixed with one drop of nigrosine on a glass slide to make a thin smear, then calculating 200 sperm from each slid and calculating the percentage of live, dead, and abnormal sperm (21). Furthermore, Johnsen's scores managed to graduate from a score of 10, which is a significant peak spermatogenic function, to a score of 1, which implies the absolute lack of sperm cells. As an illustration, this score is in table 1 (22).

Probability and statistic of data

The data was documented using an Excel program when it was collected. The data was investigated utilizing SPSS software One-way ANOVA- Duncan multiple range analysis and a result is regarded as significant if $P \leq 0.05$.

Results

According to the statistical information in the second table, giving lipopolysaccharide intraperitoneally to quail significantly decreased ($P \leq 0.05$) the hormone testosterone

while dramatically raising caspase-3 compared to the control value. The application of nano oxide 40 mg/kg diet exhibited a higher significance (P≤0.05) in testosterone hormone, whereas the apoptotic enzyme indicator (caspase-3) significantly decreased when compared to the standard control value. When birds were treated with nano-zinc oxide and concomitantly injected with LPS, it caused a rise in the hormone and a decrease in caspase-3 comparable to the LPS group and returned the value of testosterone hormone to normal control value (Table 2).

Table 1: Johnsen score standard for histological grading of seminiferous tubules

Score	Grad	Description
10	Preserved spermatogenesis	Zone of spermiation and many mature spermatids
9	Slightly lowered spermatogenesis	Fewer zones of spermiation and fewer mature spermatids
8	Distinct spermatogenesis reduction	Few mature spermatids, no spermiation
7	Significantly decreased spermatogenesis	No spermiation, only immature spermatids, and no mature spermatids
6	Drastically lowered spermatogenesis	Limited numbers of immature spermatids and a shorter germinal epithelium
5	Spermatogenesis stops once the main spermatocytes are formed	The seminiferous tubules' lumen is lined with many spermatocytes.
4	Stopping spermatogenesis at the primary spermatocyte stage	A few primary spermatocytes are present
3	Inhibition at the spermatogonia stage	Spermatogenesis cells of a type called spermatogonia multiply but mature
2	Only Sertoli cells (OSC)	There are only Sertoli cells present, no germ cells.
1	Neither Sertoli nor germ cells exist.	Connective tissue ground material takes the place of the seminiferous tubules

Table 2: The outcomes of nano-zinc oxide on testosterone hormone concentration and the caspase-3 in quail males exposed to lipopolysaccharide

Groups	Testosterone (IU/ml)	Caspase-3 (µMol/L)
Control	2.87± 0.92 b	1.46± 0.96 b
LPS	1.09± 0.12 c	2.10± 1.00 a
Nn-Zn	3.12± 1.17 a	1.29± 0.74 c
LPS + Nn-Zn	2.75± 0.76 b	1.21± 0.53 c

The average and standard error are applied to depict the values. The distinction between treatments is significant at P≤0.05. It is indicated in the pillar with small letters.

Administration of lipopolysaccharide 1 mg/kg BW intraperitoneally to the quail male caused a significant decline in live sperm and sperm count accompanied by a rise in dead and abnormal sperm compared with control. Giving zinc oxide a 40 mg/kg diet caused a significant increase in live sperm, together with drop dead and abnormal sperms, but the sperm concentration did not change as compared with control groups. Donate Lipopolysaccharide 1 mg/kg BW intraperitoneally simultaneously with nano-zinc oxide 40 mg/kg diet significantly enhanced live sperm; however, it did not alter the sperm count match with the LPS group. The data of dead and abnormal sperms reveal significant fall contrast with LPS, which appeared in table 3.

Testis morphometric parameters were calculated and listed in table 4 and revealed that the employer of LPS to the birds triggered significantly lesser in the seminiferous tubules' distance, the width of the epithelium of germ cells sheet, numbers of Sertoli cells, and the number of Leydig cells when matched up to control group. It was handled with zinc oxide, initiated significantly enhanced distance of seminiferous tubules, numbers of Sertoli cells, and the sum of Leydig cells in contrast to the control group. Management of Lipopolysaccharide 1 mg/kg BW intraperitoneally concurrently with nano-zinc oxide 40 mg/kg diet significantly increased the above testicular criteria compared to the LPS group.

Table 3: The outcomes of nano-zinc oxide on sperm criteria in quail males exposed to lipopolysaccharide

Treatments	Live sperm (%)	Dead sperm (%)	Abnormal sperm (%)	Sperm count (10^6/$mm^{3)}$
Control	84.72± 4.12 c	21.84± 3.09 b	5.31± 1.72 b	2.01±0.28 a
LPS	57.32±2.49 d	32.52± 4.11 a	11.34±2.19 a	1.12±0.18 b
Nn-Zn	89.69± 5.84 a	17.62± 2.45 d	3.49± 1.04 c	2.12±0.27 a
LPS + Nn-Zn	87.75± 5.39 b	19.53± 2.74 c	5.22± 1.81 b	2.06±0.10 ab

The average and standard error are applied to depict the values. The distinction between treatments is significant at P≤0.05. It is indicated in the pillar with small letters. The sperm count was in.

Table 4: The outcomes of nano-zinc oxide on histological indicators of testis in LPS-exposed male quail

Treatments	Ds	TGE	SE	LC
Control	322.80± 74.14 b	131.40±9.82 a	18.09±0.70 b	3.80± 0.58 b
LPS	295.81±50.53 c	79.00± 9.39 c	15.40±0.50 d	2.01± 0.31 c
Nn-Zn	334.00±60.19 a	132.60± 6.81 a	20.12±1.13 a	8.84± 1.06 a
LPS + Nn-Zn	329.20±34.21 b	129.40±17.18 b	17.80±0.37 c	3.80± 0.37 b

DS= Diameters of seminiferous tubules (µm), TGE= Thickness of germinal epithelium layer (µm), SC= Numbers of Sertoli cells (60.08µm2/400X)/ 5 field/ animal/ group), LC= Numbers of Leydig cells (60.08µm2/400X)/ 5 field/ animal/ group). The average and standard error are applied to depict the values. The distinction between treatments is significant at P≤0.05. It is indicated in the pillar with small letters.

This experiment employed Johnsen's score for evaluating the testis activity to examine the impact of using nano-zinc oxide alone or in combination with LPS on gametogenesis. In this technique of practical reality, we were using the Johnsen guidelines that evaluate the development of gametocytes in the seminiferous tubules, supporting the evaluation of semen for reproductive success. `which included a ten-mark grading scale for assessing sperm production based on the characteristics of cells present anywhere along the seminiferous. The result revealed in Figure 1 shows the lowest significance (P≤0.05) in Johnsen's scoring in the grouping of LPS over to control. The administration of zinc oxide nanoparticles exposed superlative scoring that was edited by Johnsen after statistical exploration (P≤0.05) association with controller collection. Handout nanoparticles of zinc oxide after intraperitoneal injection of LPS enhanced significantly (P≤0.05) the value of Johnsen's score and returned to normal control value, which can be seen in figure 1.

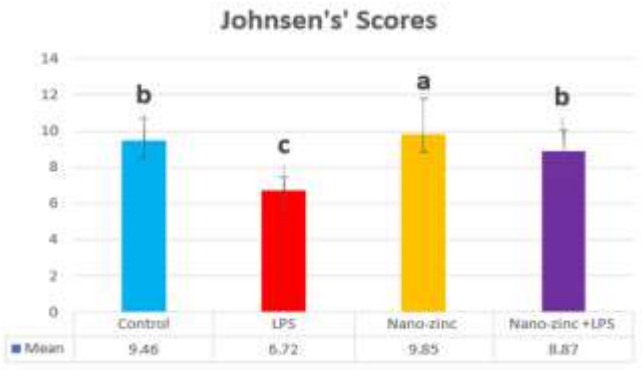

Figure 1: A histogram of the effect of Nano-zinc oxide on Johnsen's scores of testicular histological sections in LPS-exposed male quail.

The average and standard error are applied to depict the values. The distinction between treatments is significant at (P≤0.05). It is indicated in the pillar with small letters. The histopathological studies of untreated quail testes exhibit all of the sperm cells of the serial in a column manner, as well as normal widths and thicknesses of the seminiferous tubules with the impression of intact Sertoli and Leydig cells (Figures 2 and 3).

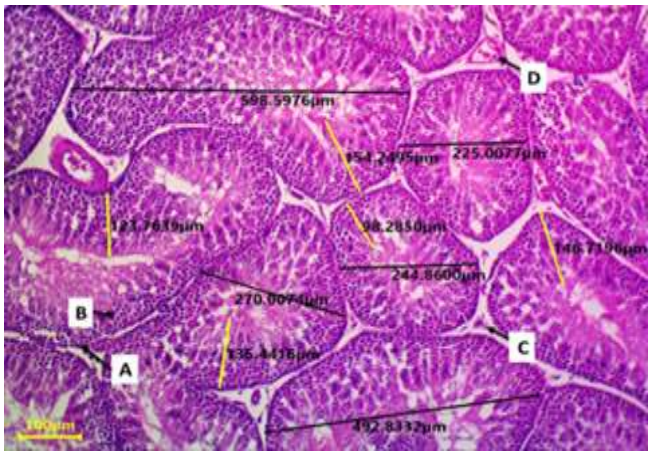

Figure 2: Quail testis photomicrograph slice of control group showing seminiferous tubules (A) with spermatogenesis cells (B), interstitial tissue (C), and blood vessels (D), with the measurements of the diameter of seminiferous tubules and thickness of the epithelium. H&E stain, 100X.

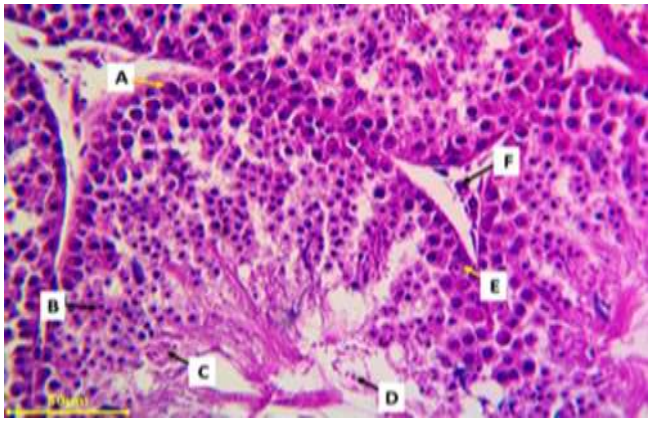

Figure 3: Quail testis photomicrograph slice of control group showing seminiferous tubule with spermatogonia (A), spermatocytes (B), spermatids (C), spermatozoa (D), Sertoli cell (E) and Leydig cell (F). H&E stain, 400X.

When fed nano-zinc particles, the diameter but not the thickness of seminiferous tubules improved photogrammetrically and statistically, as did the population of Leydig and Sertoli cells (Figures 4 and 5). The tissue sections of male quail testes infected with bacterial wall fragments (LPS) revealed disorder and thin thickness of seminiferous tubules, necrosis and deterioration of sperm cells during genesis and congested blood vessels with fewer nutritive (Sertoli) and hormonal (Leydig) cells (Figures 6 and 7).

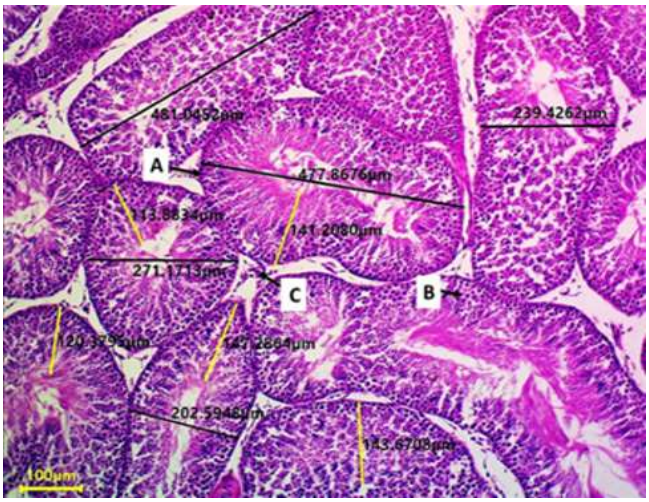

Figure 4: Quail testis photomicrograph slice after being handled with Nano-Zinc oxide group showing seminiferous tubules (A) with spermatogenesis cells (B), interstitial tissue (C), with the measurements of diameter of seminiferous tubules and thickness of epithelium. H&E stain, 100X.

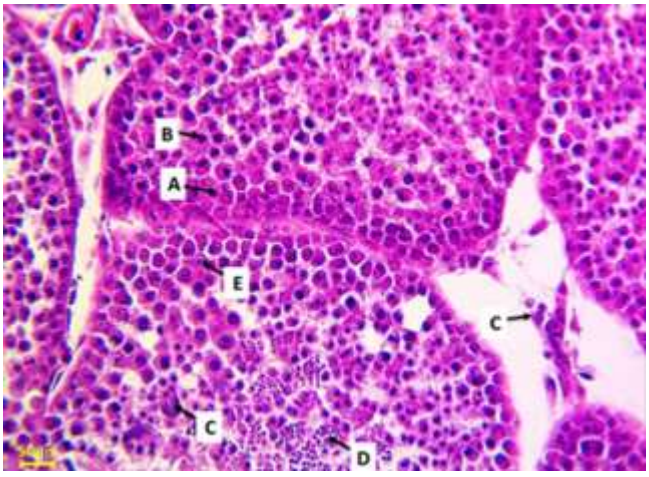

Figure 5: Quail testis photomicrograph slice after being handled with Nano-Zinc oxide group showing seminiferous tubule with spermatogonia (A), spermatocytes (B), spermatids (C), spermatozoa (D), Sertoli cell (E) and Leydig cell (F). H&E stain, 400X.

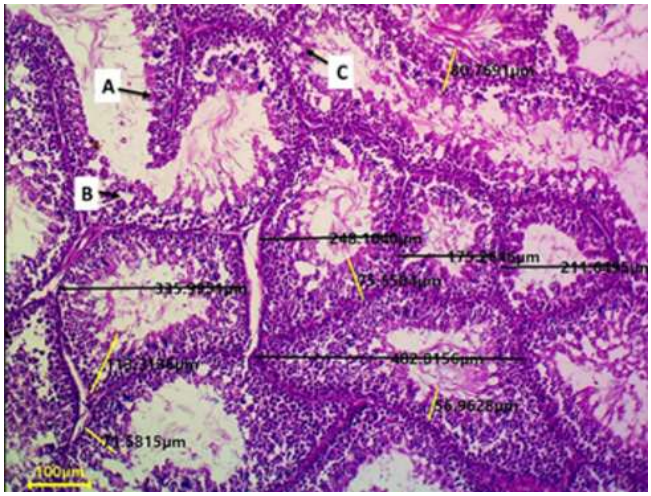

Figure 6: Quail testis photomicrograph slice after being handled with LPS group showing disorganization of seminiferous tubules (A) with necrosis and degeneration of spermatogenesis cells with thin thickness (B) and hypo - spermatogenesis (C), with the measurements of diameter of seminiferous tubules and thickness of epithelium. H&E stain, 100X.

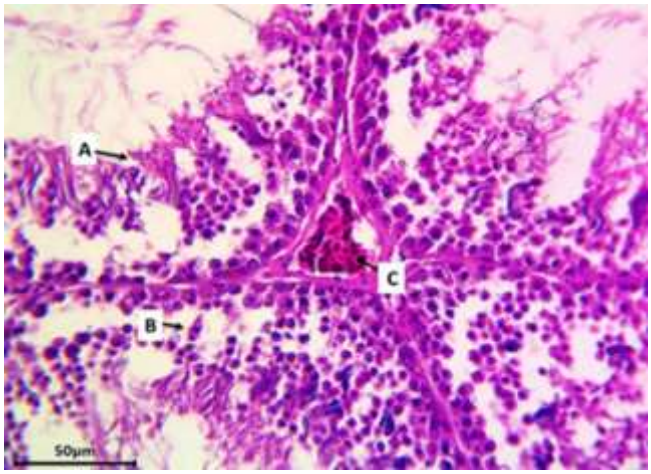

Figure 7: Quail testis photomicrograph slice after being handled with LPS group showing hypo -spermatogenesis (A), necrosis and degeneration of spermatogenesis cells (B), and congestion of blood vessels (C). H&E stain, 400X.

Finally, after being managed with the nano-zinc oxide and LPS groups, quail testis photomicrograph slides show the opposite overall impact of LPS on diameters and Leydig cells as compared to the standard and Nano-zinc oxide group, but Sertoli cell and epithelium thickness improve from the LPS harm but not as the nano-zinc oxide and control groups (Figures 8 and 9).

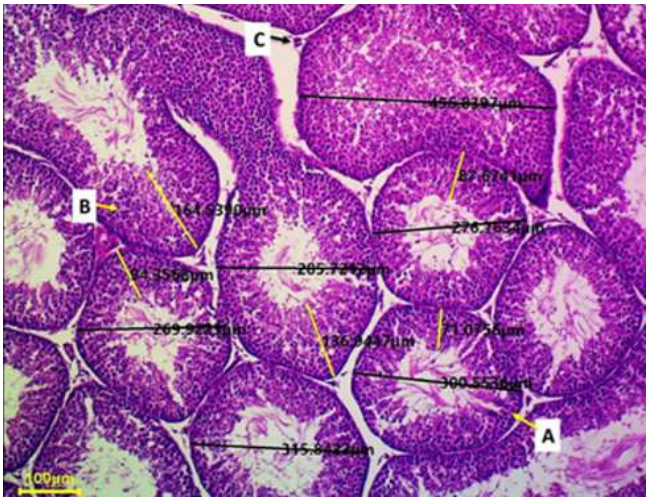

Figure 8: Quail testis photomicrograph slice after being handled with Nano-zinc oxide + LPS group showing seminiferous tubules (A) with spermatogenesis cells (B), interstitial tissue (C), with the measurements of diameter of seminiferous tubules and thickness of epithelium. H&E stain, 100X.

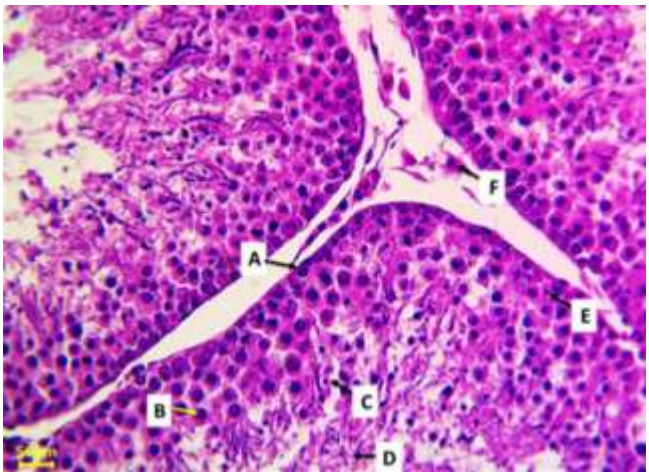

Figure 9: Quail testis photomicrograph slice after being handled with Nano-zinc oxide + LPS group showing seminiferous tubule with spermatogonia (A), spermatocytes (B), spermatids (C), spermatozoa (D), Sertoli cell (E) and Leydig cell (F). H&E stain, 400X.

Discussion

Lipopolysaccharide was given to the quail, and this action began a decline in testosterone concentration. The results of the current investigation corroborated those of Reddy *et al*. (23), who observed that intraperitoneal treatment of LPS initiated a decline in serum testosterone. The responsible for the current outcome may be the endotoxic part of gram-negative bacteria's cell walls, lipopolysaccharide, because it can trigger an acute inflammatory reaction called acute endotoxemia (24), and the inflammatory response by injecting LPS to impair testicular steroidogenesis and interrupt spermatogenesis (25). According to previous studies (26), LPS treatment suppressed Leydig cell steroidogenesis by lowering the generation of the protein steroidogenic acute regulatory (StAR) and other enzymes that contribute to the formation of steroid hormones, decreased expression of the StAR, which is essential for the mitochondria to absorb cholesterol. StAR contributes a significant process in steroidogenesis by binding and transferring cholesterol to the mitochondrial matrix (27); a whole mitochondrial electrochemical gradient is necessary to carry and process active StAR in the mitochondria and steroidogenesis. ROS-induced disturbance of the mitochondrial electrochemical gradient may inhibit StAR mitochondria (28). Excessive ROS production by LPS in the testicles may disrupt the mitochondrial electrochemical gradient, resulting in diminished hydrolysis of the peptide of StAR and reduced absorbed cholesterol for testosterone creation. Furthermore, Wang *et al*. (29) discovered that LPS changed steroidogenesis-related enzymes in testicular cell lines, implying that LPS may induce peroxisome proliferator-activated receptor (PPAR) transcribing mechanisms that affect the expression of the estrogen/androgen receptors, steroidogenesis, and ROS metabolism in testicular cells.

In the current study, injection of lipopolysaccharide reduced the number of live sperm and sperm numbers, along with a rise in sperm that are dead or have defects. This result is accurate to Hassan *et al*. discovery that testosterone levels in LPS reduced dramatically (30). Furthermore, the percentage of viable sperm in LPS reduced dramatically. The testicular mitochondrial portion of LPS exhibited a significant rise in Fe^{2+} induced lipid peroxidation accompanied by high hydrogen peroxide level, which could explain why sperm number and motility were dramatically reduced in LPS (31). Furthermore, LPS-induced oxidative stress impairs mitochondrial enzymatic activity, resulting in functional impairment in testicular mitochondria (31). Additionally, understanding the exact mechanism by which LPS produces testicular damage revealed that oxidative damage is the primary manifestation male reproductive failure (23), and oxidative damage is known as one of the putative biological pathways implicated in Sertoli cell dysfunction (32), Free radicals are produced in the testis, predominantly in the mitochondria, and are then neutralized by the mechanisms of antioxidant defense according to typical physiological circumstances, however, this equilibrium readily achievable upset by substances resembling LPS, which throw off the prooxidant-antioxidant equipoise and trigger problems in cells of Sertoli (33). Furthermore, because this part of the cell is high in fatty acids that are polyunsaturated and low in antioxidants, the mitochondrial bilayer membrane is more vulnerable to lipid

peroxidation (LPO). Furthermore, mitochondrial membrane lipid peroxidation causes irreparable harm to mitochondrial activities such as respiration, oxidative phosphorylation, and ion movement (34). According to the most significant current research findings, LPS lowered sperm intracellular cAMP *in vitro*, negatively affecting sperm viability by increasing the percentage of dead sperms (35). Our findings show that administering LPS to quail increased caspase-3 activity; the current study is similar to previous findings by Chin *et al*. (36), who discovered that LPS increased apoptosis, nuclear disintegration, and caspase-3 activation. Since LPS can cause apoptosis, perhaps through the mitochondrial way, by increasing the level of expression of the Bax and caspase three proteins, this result may be explained by the fact that LPS enhanced the transcription of pro-caspase-3 and Bax proteins that promote apoptosis (36). The apoptotic process has been extrinsic, and intrinsic apoptotic mechanisms have been identified (37). Caspases are classified as part of the cell's cysteine protease enzyme family, essential in the onset of controlled cell death provoked by diverse stimuli (38). Members of the Bcl-2 are crucial in controlling alterations to the permeation of the outer membrane of mitochondria by releasing mitochondrial apoptotic proteins. Bcl-2, Bcl-XL, and Mcl-1 are on the rise of the Bcl-2 relatives of anti-apoptotic proteins, which have been proven in studies to safeguard the integrity of the mitochondria by affixing them to mitochondrial pores. When the Bax and Bak proteins that are activated before apoptosis adhere to the mitochondrial external part of the membrane, alterations in its permeability begin (39). The current investigation found that giving LPS to birds reduced the diameter of seminiferous tubules, the thickness gauge of the germinal epithelial layer, the number of Sertoli cells, and the number of interstitial cells significantly, this conclusion is in line with what Hassan *et al*. (30) who discovered that microscopic inspection of seminiferous tubules revealed degenerative changes in LPS and that deleterious effects of LPS on the testis revealed severe necroptosis of epithelial cells, as well as degeneration of primary along with secondary spermatocytes. Furthermore, Sertoli's cell vacuolation splits the basement membrane from the underlying tissue. Furthermore, there was necrosis of interstitial cells as well as interstitial edema. (30). The outcomes show that Johnsen's scoring is much lower in the LPS group. Jafari *et al*. (40) discovered that sperm creation, Johnsen's grade, meiotic indices, and elevation of the epithelial cells had been considerably impacted (40).

Our finding shows that applying nano oxide causes higher testosterone live sperm and enhances the diameter of the seminal tubule in the testes, the number of Sertoli cells, the number of interstitial cells, and the Johnsen score. In contrast, the apoptotic enzyme indicator (caspase-3), dead and abnormal sperms decreased, the possibility of explanation as Zn is considered an essential component for more than 80 molecules of protein that participate in enzyme synthesis and DNA the transcription process, which constitutes an essential phase in the formation of germ cells, also zinc may be vital to reproduction (41). Furthermore, zinc exhibits anti-apoptotic abilities. While inadequate zinc levels may trigger numerous cell types to undergo programmed cell death, zinc supplementation can shield cells from pro-apoptotic compounds, preventing apoptosis (42). The researchers Badkoobeh *et al*. (43) confirmed that giving nano zinc oxide caused a rise in the count of sperms and the percentage of mass mobilization and decreased the number of dying sperms and deformed sperms. It has been created that zinc loss leads to degeneration of the seminal tubules and difficulties with sperm generation in rats. Zn additionally has opposed apoptotic aspects., Zn production is a crucial task in the biology of spermatozoon and sperm formation; it has a vital character in spermatogenesis, sperm survival, avoiding sperm destruction, and stabilization of sperm membranes (43). Nano-zinc oxide may increase antioxidant activity, boost antioxidant effects, and lower free radical levels. Nano zinc oxide can increase the effectiveness of antioxidants, boost their performance, and turn down oxygen radical levels (40).

When nano-zinc particles, the diameter of seminiferous tubules improved photogrammetrically, as did the population of interstitial cells besides Sertoli cells, because of the high quantities of zinc in the male reproductive organs and seminal fluid. Zinc is a requirement for the physiology of the spermatozoon and the development of sperm. According to research, zinc deficiency in rats leads to seminiferous tubule atrophy and spermatogenesis dysfunction (44).

The administration of zinc oxide nanoparticles exposed superlative scoring that Johnsen edited after statistical analysis. The researchers confirmed that giving zinc oxide nanoparticles with melatonin did not affect the scoring system proposed by Johnsen, seminiferous tubules diameter, and finally, the tallness of the germinal epithelium (45).

Conclusion

Nano zinc oxide must be added to the male quail diet to overcome the LPS contamination that harms male sex features and to reverse that impact.

Conflict of interest

According to the researcher, there are no conflicts with any interests related to the publication of the current investigation.

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فعالية أوكسيد الزنك النانوية على كاسبيز- ٣ لذكور السمان المعرضة للسكريات الدهنية المتعددة

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الخلاصة

من المعروف أن سمية الدم الداخلي الناجم عن السكريات الدهنية المتعددة يسبب العقم عند الذكور. تم تصميم البحث للتقصي عن دور أكسيد الزنك النانوية على كاسباس -٣ والكفاءة الجنسية في ذكور السمان المعرضة للسكريات الدهنية المتعددة. تم توزيع السمان عشوائياً على أربع مجاميع، تلقت المجموعة الأولى من الطيور العليقة المعتادة، وتم استخدامها كسيطرة، بينما تم حقن المجموعة الثانية داخل الخلب بالسكريات الدهنية المتعددة مرتين أسبوعياً بجرعة ١ ملغم / كغم من وزن الجسم، المجموعة الثالثة أعطيت أكسيد الزنك النانوية بجرعة ٤٠ ملغم / كغم علف لمدة ستة أسابيع. تمت معاملة المجموعة الرابعة بنفس الجرعة من جزيئات أكسيد الزنك النانوية وتبعها حقن بالسكريات الدهنية المتعددة بنفس الجرعة المحددة. تشير النتائج إلى أن تأثير السكريات الدهنية المتعددة خفضت من مستوى هرمون التستوستيرون والنطف الحية وعدد النطف وقياس شكل الخصية ومرتبة جونسن وصاحبه ارتفاع في النطف الميتة والمشوهة وكاسباس ٣ مقارنة بالسيطرة. أظهرت النتائج ان إضافة أكسيد الزنك النانوية ٤٠ ملغم / كغم سبب ارتفاع معنوي لهرمون التستوستيرون والنطف الحية معًا مع معايير الخصية ومراتب جونسن وانخفاض معنوي بالنطف الميتة والغير الطبيعية وكاسباس ٣. أدت المعاملة بأكسيد الزنك النانوية بالتزامن مع السكريات الدهنية المتعددة إلى ارتفاع مستوى هرمون التستوستيرون ومرتبة جونسن مع انخفاض في كاسباس بالمقارنة بمجموعة السكريات الدهنية المتعددة وإرجاع قيمة هرمون التستوستيرون إلى قيم الطبيعية. واستنتج البحث إلى أن إضافة نانو أكسيد الزنك إلى عليقة ذكور السمان ضروري للتغلب على التلوث المحدث بالسكريات الدهنية المتعددة والتي تؤثر سلبًا على الخصائص الجنسية للذكور وإن أوكسيد الزنك النانوية أصلح من تلك التأثيرات.

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