

Effects of Repeated Environmental Noise Exposure on Ovarian Histomorphology, Reproductive Hormones and Antioxidant Enzyme Activities in Adult Female Wistar Rats

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ABSTRACT

Environmental noise is a prevalent environmental stressor that may influence physiological function through neuroendocrine and redox pathways. The present study evaluated the effects of repeated exposure to four recorded environmental noise sources on body weight, reproductive hormones, ovarian antioxidant enzymes activities and ovarian histomorphology in adult female Wistar rats. Twenty-five rats were randomly allocated to five groups (n=5/group): An unexposed control group and groups exposed to a car horn (90 dB), siren (80 dB), gunshots (100 dB), or generator (100 dB) noise for 3 h daily for 21 days. Body weight was recorded before and after exposure. Serum oestrogen and luteinizing hormone (LH) were measured by ELISA, while ovarian superoxide dismutase (SOD) and glutathione peroxidase (GPx) activities were determined spectrophotometrically. Ovarian sections were examined following haematoxylin and eosin staining. Gunshot exposed rats showed the greatest percentage reduction in body weight ($-10.07 \pm 3.18\%$), significantly different from controls ($p = 0.011$). Differences in serum oestrogen and LH concentrations were not statistically significantly. Histology revealed morphological alterations in exposed groups, including stromal loosening, reduced follicular representation, stromal degeneration and, in the generator group, evidence of luteal regression and vascular congestion. These findings provide preliminary evidence of ovarian morphological alterations associated with repeated noise exposure in adult female Wistar rats. However, because noise source, sound intensity and acoustic pattern were not independently manipulated, the individual contribution of these factors cannot be established. Larger studies incorporating oestrous cycle staging, quantitative histomorphometry, direct markers of oxidative damage and neuroendocrine stress are warranted.

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INTRODUCTION

Environmental noise is increasingly recognized as an environmental stressor with effects that extend beyond the auditory system (Sohag *et al.*, 2014). Repeated exposure to unwanted or excessive sound has been associated with cardiovascular, neurological, psychological and other systemic responses (WHO, 2018). Experimental models have similarly demonstrated that noise can activate stress related neuroendocrine pathways and alter physiological function (Ising & Kruppa, 2004; Basner *et al.*, 2014).

Stress responses are coordinated principally through the hypothalamic- pituitary- adrenal (HPA) axis. Activation of hypothalamic corticotropin-releasing hormone stimulates adrenocorticotrophic hormone secretion and subsequent glucocorticoid release from the adrenal cortex (McEwen, 2007). The HPA axis interacts with the hypothalamic-pituitary- gonadal (HPG) axis and sustained stress may influence gonadotropin -releasing hormone pulsatility and downstream reproductive endocrine function (Chrousos, 2009; Yuen & Mobbs, 2009). Because ovarian activity depends on coordinating endocrine signalling, prolonged physiological stress may influence follicular development, steroidogenesis and luteal function

Experimental studies have reported reproductive and metabolic responses to environmental or laboratory noise, although the magnitude and direction of these responses vary with exposure characteristics, including intensity, duration

and acoustic pattern. Noise may also affect animal behaviour and reproductive interactions (Kight & Swaddle, 2011). Evidence relating repeated noise exposure specifically to ovarian morphology remains less extensive than evidence concerning general stress responses. Histological examination is therefore useful for identifying structural changes that may not be reflected by circulating hormone concentrations alone.

The present study was designed to examine body -weight change, selected reproductive hormones, ovarian antioxidant enzyme activities and ovarian histomorphology following repeated exposure to four acoustically distinct recorded noise sources. The study was exploratory in nature and was intended to provide integrated physiological biochemical and morphological observations. Because the exposure groups differed simultaneously in sound and nominal sound intensity, the study does not permit independent estimation of the effects of intensity versus acoustic source. It was hypothesized that repeated exposure to environmental noise would alter ovarian histomorphology and may affect body weight, reproductive hormone concentrations and ovarian antioxidant enzyme activities in adult female Wistar rats.

MATERIALS AND METHODS

Experimental Animals and Ethical Approval

Twenty-five adult female Wistar rats weighing between 140-220 g were obtained and housed in the Animal House of the

Faculty of Basic Medical Science, Baze University, Abuja, Nigeria. Animals were acclimatized for one week before commencement of the exposure protocol and maintained at $25 \pm 1^\circ\text{C}$ under a 12 h light / 12 h dark cycle in plastic cages. Standard commercial rodent chow (Vital Feeds Ltd. Jos, Nigeria) and clean tap water were provided *ad libitum*. Experimental protocols adhered to the National Institute of Health (NIH) *Guide for the care and Use of Laboratory Animals* and received institutional ethical approval (BU/URES/ANA/1013).

Oestrous Cycle Staging

To account for cycle-dependent hormonal fluctuations, daily vaginal cytology was performed on all animals during the 7-day acclimatization period and throughout the 21-day exposure phase between 08:00 am 9:00 am. Vaginal lavage was performed using 50 μL of sterile normal saline (0.9 % NaCl), smeared onto clean glass slides, stained with 0.1 % crystal violet, and examined under a light microscope at $\times 100$ and $\times 400$ magnification. Oestrous cycle phases (proestrus, oestrous, metestrus, dioestrus) were identified based on the relative proportions of epithelial cells, and leukocytes.

Noise Exposure

Four recorded environmental noise stimuli representing common regional environmental noise (Car horn, Emergency

siren, Gunshot sound, and heavy-duty power generator sound) were used. Nominal sound intensities were 90 dB for car horn, 80 dB for siren, 100 dB for gunshot and 100 dB for generator exposure. Sound exposure was delivered dedicated sound-attenuated exposure chambers (1.2 m x 0.8 m x 0.8 m) lined with high -density acoustic absorption foam. Cages were placed directly beneath overhead omnidirectional acoustics transducers suspended 50 cm above animal head level. Acoustic intensity was continuous/intermittent depending on the stimulus characteristics and delivered for 3 h/day (09:00 to 12:00 h) over 21 consecutive days.

Sound Calibration and Metrics: Acoustic sound pressure levels (SPL) were continuously monitored and standardized using a calibrated Class 1 Sound Level Meter (Type 2250, Gruel & Kjaer, Naerum, Denmark) fitted with a $\frac{1}{2}$ inch free field microphone, alongside a secondary smartphone -based acoustic analyser (Decibel X Pro v8.2, SkyFox Labs) running on an Apple iPhone 13. The measurement system was calibrated before each session using an acoustic calibrator (Type 4231, Gruel and Kjaer) delivering 94.0dB SPL at 1000Hz.

Acoustic Weighting: A-frequency weighting (dB(A)) with slow temporal response (1000 ms) was utilized to mimic mammalian auditory response curves.

Experimental Design and Grouping

Table 1: Animals were Randomly Allocated to Five Groups (n=5/group)

Group	Exposure	Nominal level	Exposure duration
1	Control; no experimental noise	—	—
2	Car horn	90 dB (A) L_{Aeq}	3 h/day for 21 days
3	Siren	80 dB (A) L_{Aeq}	3 h/day for 21 days
4	Gunshot	100 dB (A) L_{Aeq}	3 h/day for 21 days
5	Generator	100 dB (A) L_{Aeq}	3 h/day for 21 days

All groups received standard commercial rat feed and drinking water *ad libitum*.

Body-Weight Measurement:

Individual animal body weights were recorded before the exposure phase (Day 0) and at the end of the (21- day) exposure period using a digital precision balance (*Kern PCB 3500-2*, Balingen Germany). Percentage change was calculated from the individual initial and final body weights.

Sample Collection

Following 12 hours of overnight fasting (to standardize baseline metabolic status), and exactly 18 hours after the final noise exposure on Day 21, all animals were confirmed to be in the dioestrus phase via vaginal lavage and humanely euthanized via inhalation of mild isoflurane anaesthesia followed by cervical dislocation.

Blood was collected via cardiac puncture immediately following euthanasia into plain non-anticoagulant gel- barrier tubes. Samples were allowed to clot at 25°C for 30 minutes and centrifuged at $3,000 \times g$ for 15 minutes at 4°C . Serum was separated, aliquoted into microcentrifuge tubes, and stored at -80°C until endocrine analysis. Pair-ovaries were excised via a lower abdominal midline incision, trimmed of adhering adipose tissue and weighed.

Left ovary: Immediately immersion fixed in 10 neutral-buffered formalin (NBF) for 48 hours for histological processing.

Right ovary: Snap-frozen in liquid nitrogen and stored at -80°C for biochemical antioxidant profiling.

Histological Processing

The fixed left ovarian tissues were dehydrated through an ascending ethanol series (70%, 80%, 90% 95%, 100%) cleared in xylene and embedded in paraffin wax. Serial sections were cut at 5 μm thickness using a rotary microtome (Leica RM2125rt, Wetzlar, Germany) every 10^{th} section was selected to prevent double-counting follicles and stained with Hematoxylin and Eosin (H&E). Sections were evaluated under a light microscope (Leica DM750) equipped with a digital camera (Amscope MU1803 MP USB3.0Color CMOS C-Mount Microscope Camera, United Scope, LLC).

Hormonal Assays

Serum hormone concentrations were measured using quantitative enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions. Oestrogen was quantified using a competitive ELISA kit (Cat No: EIA-2693, DRG Instruments GmbH, Marburg, Germany). Analytical sensitivity: 9.71 pg/mL; intra-assay CV: 4.8%; inter-assay CV: 6.7%. While Luteinizing Hormone (LH) was quantified using commercial rat ELISA kits (Elabscience Biotechnology Co, Ltd Wuhan, China) according to manufacturer instructions. Analytical sensitivity: 0.056 mIU/mL; intra-assay CV: 5.2% inter-assay CV: 7.9%. absorbance for both assays was read at 450 nm using a microplate reader ELISA reader (*Stat Fax 2100*; Awareness Technology Inc., Palm City FL USA)/ Hormonal concentration were interpolated against standard curves.

Ovarian Antioxidant Enzyme Assays

The right ovarian tissues were thawed weighed, and homogenized (1:10 w/v) in ice-cold 0.05 M phosphate buffer (pH 7.4) containing 1 mM EDTA using a glass-Teflon homogenizer. The homogenate was centrifuged at 10,000 x g for 20 minutes at 4 °C and the clear supernatant was collected. Total protein content was quantified using the Bradford assay (Bio-Rad Laboratories, Hercules, CA, USA) with bovine serum albumin (BSA) as standard. Superoxide Dismutase (SOD) was assayed using a commercial colorimetric kit (Cat No: SD125; Randox Laboratories; Crumlin; UK) based on the inhibition of nitroblue tetrazolium (NBT) reduction mediated by xanthine/xanthine oxidase. Absorbance was read at 560 nm. One unit (U) of SOD activity was defined as the amount of enzyme producing 50 % inhibition of NBT reduction. Activity was expressed as U/mg protein. Glutathione Peroxidase (GPx) was measured using a coupled enzymatic kit (Cat No: RS505; Randox Laboratories; Crumlin; UK) following the Paglia and Valentine method. GPx catalyses the oxidation of glutathione by cumene hydroperoxide in the presence of glutathione reductase and NADPH. Oxidation of NADPH to NADP⁺ was monitored by decreasing absorbance

at 340 nm over 3 minutes. Activity was expressed as U/mg protein.

Statistical Analysis

Data were analysed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk; NY; USA). Continuous variables were tested for normality using the Shapiro-Wilk test and for homogeneity of variance using Levene's test. To control for significant differences in baseline body weights across groups, an Analysis of Covariance (ANCOVA) was performed on final body weights using initial body weight as the covariate; followed by Bonferroni post-hoc pairwise comparisons. For the Biochemical, hormonal and Morphometric Data, differences among groups were analysed using One-Way Analysis of Variance (ANOVA) followed by Dunnett's post-hoc pairwise comparisons. For the Biochemical, hormonal, and Morphometric Data, differences among groups were analysed using One-Way Analysis of Variance (ANOVA) followed by Dunnett's post-hoc test comparing each noise exposure group against the unexposed Control group. Data are presented as Mean $\pm$ Standard Deviation (SD). Statistical significance was defined as $p < 0.05$.

RESULTS AND DISCUSSION**Body Weight****Table 1: Effects of 21-day Repeated Environmental Noise Exposure on Body Weight in Adult Wistar Rats (n=5/group)**

Group (n=5)	Initial body weight (g)	Final body weight (g)	Body-weight change (%)	p-value vs control
1 control	149.00 $\pm$ 23.01	141.40 $\pm$ 21.61	-4.80 $\pm$ 4.38	—
2 car horn 90 dB	142.60 $\pm$ 19.70	137.40 $\pm$ 20.11	-3.69 $\pm$ 3.94	0.462
3 Siren; 80 dB	121.00 $\pm$ 21.53	113.40 $\pm$ 19.84	-5.78 $\pm$ 4.66	0.998
4 Gunshot; 100 dB	214.80 $\pm$ 14.72	193.20 $\pm$ 14.38	-10.07 $\pm$ 3.18*	0.011
5 Generator; 100 dB	208.40 $\pm$ 9.71	205.80 $\pm$ 23.35	-1.38 $\pm$ 7.94	0.571

Values are expressed as Mean $\pm$ SD. * $p < 0.05$ indicates statistically significant difference compared to control (ANCOVA with Bonferroni post hoc test)

Post-hoc Dunnett comparisons indicated that rats exposed to Gunshot noise (100 dB(A)) experienced a significantly higher mean reduction in body weight (-10.07 $\pm$ 3.18%) compared to the Control group (-4.80 $\pm$ 4.38%, $p = 0.011$). Body weight

alterations in the Car Horn ($p = 0.462$), Siren ($p = 0.998$), and Generator ($p = 0.571$) groups did not differ significantly from Control.

Serum Oestrogen**Table 2: Oestrogen Hormone Profile following 21 days of Environmental Noise Exposure (n = 5/group)**

Group (n=5)	Oestrogen (pg/mL)	p-value vs control
1 control	40.53 $\pm$ 16.65	—
2 Car horn 90 dB	50.16 $\pm$ 8.18	0.339
3 Siren 80 dB	35.81 $\pm$ 20.99	0.737
4 Gunshot 100 dB	53.76 $\pm$ 11.16	0.235
5 Generator 100 dB	38.08 $\pm$ 7.25	0.796

Values expressed as mean $\pm$ SD. Statistical test: One way ANOVA followed by Dunnett's post-hoc test.

Mean concentrations ranged from 35.81 $\pm$ 20.99 pg/mL (Siren) to 53.76 $\pm$ 11.16 pg/mL (gunshot) compared to Control (40.53 $\pm$ 16.65 pg/mL). One-Way ANOVA showed

no overall statistically significant difference among groups (F (4; 20) = 1.04, $p = 0.412$)

Serum luteinizing Hormone**Table 3: Serum Luteinizing Hormone Profile following 21 days of Environmental Noise Exposure (n = 5/group)**

Group (n=5)	LH (mIU/mL)	p-value vs control
1 control	1.32 $\pm$ 0.41	----
2 Car horn 90 dB	1.63 $\pm$ 0.29	0.261
3 Siren 80 dB	0.94 $\pm$ 0.69	0.386
4 Gunshot 100 dB	1.28 $\pm$ 0.14	0.878
5 Generator 100 dB	0.91 $\pm$ 0.29	0.160

Values expressed as mean $\pm$ SD. Statistical test: One way ANOVA followed by Dunnett's post-hoc test.

Mean concentration ranged from 0.91 ± 0.29 mIU/mL. Generator to 1.63 ± 0.29 mIU/mL (Car horn) compared to Control (1.32 ± 0.41 mIU/mL). One-way ANOVA showed no statistically significant difference among groups. ($F(4, 20) = 1.10$, $p=0.381$).

Ovarian GPx activity

Table 4: Ovarian Glutathione Peroxide (GPx) Activity (n = 5 / groups)

Group (n=5)	GPx (U/mg protein)	p-value vs control
1 Control	14.68 ± 4.34	----
2 Car horn 90 dB	14.46 ± 2.68	0.936
3 Siren 80 dB	13.80 ± 3.67	0.769
4 Gunshot 100 dB	14.07 ± 4.99	0.861
5 Generator 100 dB	15.97 ± 3.91	0.674

Values expressed as mean $\pm$ SD. Statistical test: One-way ANOVA followed by Dunnett's post-hoc test.

Ovarian GPx activities range between 13.80 ± 3.67 U/mg protein, Siren – 15.97 ± 3.91 U/mg protein, (Generator) compared to control (14.68 ± 4.34 U/mg protein), Differences were not statistically significant ($F(4,20)$, $p=0.892$).

Ovarian SOD activity

Table 5: Ovarian Superoxide Dismutase (SOD) Activity: (n = 5/groups)

Group (n)	SOD (U/mg protein)	p-value vs control
1 Control (5)	39.10 ± 9.67	----
2 Car horn, 90 dB (5)	33.20 ± 15.12	0.536
3 Siren 80 dB (5)	38.80 ± 9.00	0.966
4 Gunshot 100 dB (5)	41.67 ± 6.89	0.680
5 Generator 100 dB (5)	42.93 ± 7.12	0.547

Values expressed as Mean SD: Statistical test: One-way ANOVA followed by Dunnett's post-hoc test.

Ovarian SOD activities range from 33.20 ± 15.12 U/mg protein; (Car horn) to 42.93 ± 7.12 U/mg protein, (Generator) across groups were not statistically significant ($F(4, 20)$, $p=0.745$). relative to control (39.10 ± 9.67 U/mg protein). Differences

Ovarian Histomorphology

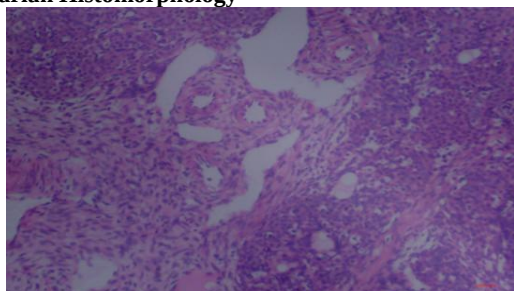


Figure 1: Photomicrograph of Group I showing: well preserved Corpus Luteum (CL), Follicle (F), and Stroma (S) (H&E $\times 40$)

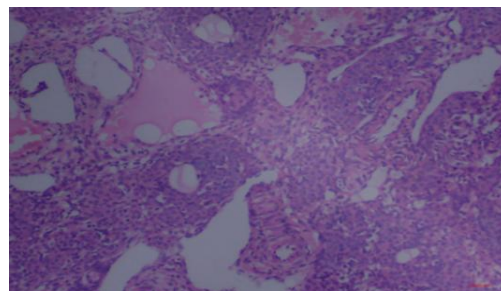


Figure 2: Photomicrograph of Group II showing: Corpus Luteum (CL), Follicle (F), and loosened Stroma (S) with Blood Vessels (BV) (H&E $\times 40$)

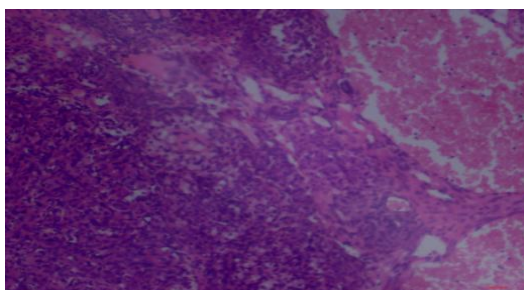


Figure 3: Photomicrograph of Group III showing: Corpus iLuteum (CL), mildly shrunk Follicles (F), and less compact Stroma (S) (H&E $\times 40$)

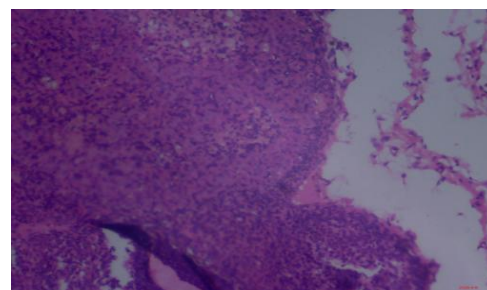


Figure 4: Photomicrograph of Group IV showing: poorly defined Corpus Luteum (CL), reduced Follicles (F), and disrupted Stroma (S) (H&E $\times 40$)

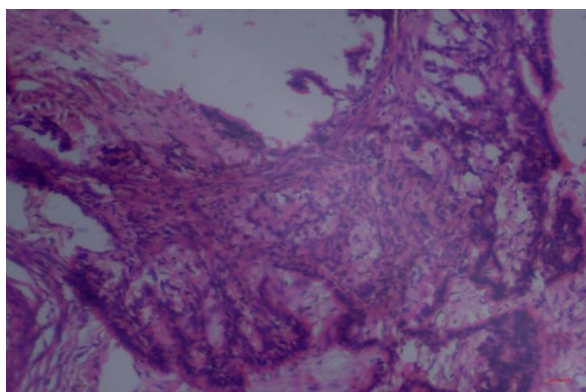


Figure 5: Photomicrograph of Group V showing: poorly defined Corpus Luteum (CL), Corpus Albicans, reduced Follicles (F), and degenerating Stroma (S) (H&E $\times 40$)

Control ovaries showed preserved ovarian architecture, with identifiable follicles at different stages of development, a well-preserved corpus luteum and an organised stromal compartment. No conspicuous vascular congestion or stromal disorganization was described. Car horn exposure was associated with mild qualitative changes, including stromal loosening, mild vascular dilation and reduced granulosa cell compactness in some follicles. In the siren group, follicles and corpus luteum remained identifiable, but reduce follicular representation; mild follicular shrinkage and reduced stromal compactness were described. The gunshot group showed more conspicuous structural changes, including a poorly defined corpus luteum, fewer visible follicles and stromal disruption with areas described as vacuolated. The generator group showed a poorly defined corpus luteum together with corpus albicans, stromal oedema, mild congestion and fewer distinct follicles with degenerative features.

Discussion

The present study examined whether repeated exposure to different recorded environmental noise conditions was associated with changes in body weight, selected reproductive hormones, ovarian antioxidant enzyme activities and ovarian morphology in adult female Wistar rats. The principal observations were a significant reduction in body weight in the gunshot group relative to control based on the analysis, absence of statistically significant differences in the measured serum hormones and antioxidant enzyme activities, and qualitative ovarian morphological alterations in exposed animals.

The reduction in body weight observed in the noise-exposed groups, particularly the gunshot group may reflect a physiological response to repeated acoustic stress. Previous studies in rats have reported reduced body-weight gain following chronic noise exposure, with some studies also showing reduced food intake. Previous studies have reported alterations in food intake and body-weight gain in Wistar rats exposed to chronic noise (Liu LiJie *et al.*, 2018); While another study found that 21days of intermittent noise significantly reduced body weight despite no significant change in ACTH or corticosterone, suggesting that weight changes may occur even in the absence of sustained alteration in these HPA-axis hormones (Konkle *et al.*, 2017). Similarly, exposure to 95dB noise have been shown to reduce food intake and eating duration in rats, supporting the possibility that stress-related changes in feeding behaviour contributed to the reduced body weight observed in the present study (González-Torres & Dos Santos, 2019; Lee *et al.*, 2024).

The greater reduction observed in the gunshot group may also reflect differences in the temporal characteristics of the noise, such as its sudden and intermittent nature, rather than intensity alone. However, food intake, energy expenditure and stress hormones were not measured in the present study; therefore, the precise cause of the weight reduction cannot be established. It is also important to note that noise does not always reduce body weight. Studies in which noise primarily disrupted sleep have reported increased food intake and body-weight gain in female Wistar rats, indicating that the effect of noise on body weight may depend on the intensity, pattern and timing of exposure (Coborn *et al.*, 2019; Houser *et al.*, 2023). Thus, the reduced body-weight observed in the present study is best interpreted as a possible consequence of repeated acoustic stress, rather than evidence of a specific metabolic mechanism.

The changes in serum oestrogen and Luteinizing hormone (LH), observed in the present study were not statistically significant and therefore do not provide sufficient evidence that noise exposure caused significant endocrine disruption. However, the findings are biologically plausible because acoustic stress may affect the hypothalamic-pituitary-gonadal (HPG) axis. In a comparable study, chronic scream sound for 21 days significantly reduced serum oestradiol in female rats; While follicular stimulating hormone (FSH) remained unchanged and LH showed non-significant downward trend; these changes were associated with reduced ovarian reserve and impaired follicular integrity (Xi *et al.*, 2022). Differences between studies may be related to the intensity, pattern and duration of noise exposure and the timing of hormone measurement. Noise can activate the stress response, and changes in HPA axis may influence reproductive hormone secretion, although this response can vary with the type and duration of stress (Babb *et al.*, 2013, Joseph and Whirlledge, 2017; Phumsatitpong *et al.*, 2021).

The absence of significant hormonal changes in the present study does not necessarily exclude local effects on the ovary. Previous studies have shown that high-sound exposure can cause ovarian structural and biochemical changes while chronic acoustic stress has also been associated with reduced ovarian reserve and increased granulosa-cell apoptosis (Kanbur and Sagir 2021; Xi *et al.*, 2022). In addition, ovarian hormones vary considerably during the rat oestrous cycle, and a single terminal measurement may therefore increase biological variability. Future studies should include repeated oestrous-cycle staging, larger sample sizes and additional reproductive and stress related markers to better determine the endocrine effects of repeated noise exposure.

The non-significant changes in ovarian SOD and GPx activities observed in the present study suggest that repeated noise exposure did not produce a clear alteration in the ovarian antioxidant defence system. However, the numerical differences may indicate some response to acoustic stress. Previous experimental work in female Wistar rats showed that high sound exposure caused ovarian structural and biochemical changes, including increased lipid peroxidation and reduced glutathione, SOD and catalase activities (Kanbur and Sagir, 2021). Other studies have also reported changes in antioxidant responses following noise exposure, although the direction and magnitude of these changes vary with tissue exposure duration and timing of measurement (Akefe *et al.*, 2020, Sorensen *et al.*, 2024, Rahmati *et al.*, 2025).

The absence of significant changes in SOD and GPx in the present study may be related to compensatory antioxidant responses, the timing of tissues collection, or the small sample size ($n=5$ per group) (Zhou *et al.*, 2023). Importantly, SOD and GPx reflect antioxidant defence and are not direct measures of oxidative damage. Therefore, the present findings do not provide sufficient evidence to conclude that noise exposure caused ovarian oxidative stress. Nevertheless, when considered together with the observed ovarian histological changes the numerical variations in SOD and GPx may suggest a possible disturbance of ovarian redox balance. Future studies should include larger samples sizes and direct markers of oxidative damage such as MDA, 4-HNE, protein carbonyls and ROS to clarify this relationship. The ovarian histological changes observed in the noise-exposed groups suggest that repeated acoustic exposure may affect ovarian tissue architecture. These changes included altered follicular morphology, stromal loosening or degenerating, vascular congestion and changes in the corpus luteum, with more noticeable changes in some higher-intensity or impulsive-noise groups. Similar findings have been reported by Kanbur and Sagir (2021), that observed follicular degeneration, vascular congestion oedematous changes and other ovarian structural alterations in female Wistar rats exposed to 102 dB sound for 28 days. Xi *et al.* (2022) also reported reduced primordial and preantral follicles, increased follicular atresia and granulosa-cell apoptosis following three weeks of chronic scream-sound exposure in female rats. These findings support the possibility that repeated acoustic stress can adversely affect ovarian morphology and follicular integrity.

The observed changes may be related to stress-induced neuroendocrine, vascular or oxidative responses. However, these mechanisms were not directly assessed in the present study and should therefore be considered possible rather than established explanations. In addition, the histological assessment was mainly qualitative without quantitative follicle counts stereological analysis or apoptosis-specific staining. Nevertheless, their similarity to previous experimental studies supports the possibility that repeated noise exposure can alter ovarian tissue architecture. Future studies should include quantitative histomorphometry, follicle counting and assessment of oxidative and apoptotic markers to better define the extent and mechanism of noise related ovarian injury.

CONCLUSION

Repeated exposure to environmental noise was associated with reduced body-weight change and qualitative alterations in ovarian histoarchitecture in adult female Wistar rats, particularly in groups exposed to the more intense or impulsive acoustic stimuli. Although numerical variations were observed in serum oestrogen, LH, SOD, and GPx

activities these changes were not statistically significant, indicating that the study did not demonstrate a significant systemic hormonal disturbance or definitive oxidative stress. The observed ovarian structural changes may nevertheless reflect the effects of repeated acoustic stress, potentially involving neuroendocrine, vascular and redox-related pathways. However, these mechanisms were not directly assessed in the present study and should therefore be regarded as plausible rather than established. Further studies with larger sample sizes, oestrous-cycle staging, quantitative ovarian histomorphometry, and direct assessment of oxidative, apoptotic and neuroendocrine markers are warranted to clarify the mechanisms by which repeated environmental noise may affect ovarian integrity and reproductive function.

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