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Histopathological Effects of Chronic White Noise Exposure on the Brain, Heart, Testis and Intestine of Wistar Albino Rats: Restorative Potential of Post-exposure Ascorbic Acid

Esin ÜNSALDI[1]

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ABSTRACT

Background: Noise is a common environmental stressor that may induce structural alterations in multiple tissues following chronic exposure. This study aimed to evaluate the histopathological changes induced by chronic exposure to 70 dB SPL broadband white noise in the brain, heart, testis and intestine of Wistar albino rats and to investigate the potential restorative effects of post-exposure ascorbic acid administration.

Methods: Twenty Wistar albino rats aged 8-10 weeks were randomly assigned to control or noise-stress groups. Rats in the noise-stress group were exposed to 70 dB SPL broadband white noise for 1 h/day for 30 days. After exposure, five animals from each group were euthanized and brain, heart, testis and intestinal tissues were collected for histopathological examination. The remaining animals received ascorbic acid in drinking water at 100 mg/kg/day for 30 days, after which the same tissues were examined histopathologically.

Result: Chronic exposure to white noise resulted in histopathological alterations of varying severity in all examined organs. Intestinal tissues showed epithelial degeneration, desquamation and inflammatory cell infiltration in the lamina propria. Brain sections demonstrated gliosis and neuronal degeneration. Testicular tissues showed degeneration and necrosis in some seminiferous tubules, while hyaline degeneration was observed in the myocardium. Following ascorbic acid administration, lesion severity was reduced compared with the noise-stress group and overall tissue architecture was better preserved.

Key words: Ascorbic acid, Histopathology, Multi-organ injury, Noise stress, White noise, Wistar rat.

INTRODUCTION

Environmental noise has emerged as one of the most pervasive environmental pollutants accompanying rapid urbanization, industrialization and the expansion of transportation networks worldwide. Beyond its well-established detrimental effects on the auditory system, chronic noise exposure is now recognized as an important environmental stressor capable of inducing widespread physiological disturbances. Experimental studies in laboratory animals have demonstrated that prolonged noise exposure can affect cardiovascular, neurological, endocrine, metabolic, reproductive and gastrointestinal systems through complex neuroendocrine, oxidative and inflammatory pathways. Consequently, environmental noise represents an important environmental health concern because of its potential effects on both human and animal health (Spreng, 2000; Demirel *et al*., 2009; Gannouni *et al*., 2014; Lasheen *et al*., 2015; Baldwin *et al*., 2006).

Chronic exposure to environmental noise initiates a cascade of biological events extending beyond the auditory system and affecting the structural integrity of multiple organs. Experimental studies in rats and rabbits have shown that prolonged noise exposure induces tissue injury through interconnected mechanisms involving neuroendocrine activation, oxidative imbalance, mitochondrial dysfunction, inflammatory responses and apoptosis, ultimately leading to cellular degeneration in susceptible tissues (Demirel *et al*., 2009; Abouee-Mehrizi *et al*., 2021; Ünsaldı and Kalınbacak, 2025). Histopathological alterations associated with noise exposure have been reported in several organs, including the liver, kidney, lung, brain and reproductive tissues. These changes include neuronal degeneration and reactive gliosis in the brain, myocardial degeneration and vascular alterations in the heart, degeneration of seminiferous tubules and impaired spermatogenesis in the testis and epithelial injury accompanied by inflammatory cell infiltration in the intestine. The severity and distribution of these lesions appear to depend on exposure intensity, duration and tissue-specific susceptibility (Xue *et al*., 2014; Lasheen *et al*., 2015; Abouee-Mehrizi *et al*., 2021;

[1]Department of Anatomy, Faculty of Veterinary Medicine, Necmettin Erbakan University, 42310 Ereğli, Konya, Türkiye.

Corresponding Author: Esin ÜNSALDI, Department of Anatomy, Faculty of Veterinary Medicine, Necmettin Erbakan University, 42310 Ereğli, Konya, Türkiye. Email: esinunsaldi@gmail.com

ORCID: 0000-0003-1656-329X

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Yang *et al*., 2024). Despite these observations, most available studies have evaluated individual organs separately and comprehensive histopathological investigations examining multiple organ systems within the same experimental model remain limited.

Therefore, the present study aimed to characterize the histopathological effects of chronic exposure to 70 dB SPL broadband white noise in the brain, heart, testis and intestine of Wistar albino rats. In addition, the study evaluated the histopathological changes observed following post-exposure administration of ascorbic acid, with particular emphasis on the severity and distribution of tissue lesions across multiple organ systems. By examining these organs within the same experimental model, this study sought to provide an integrated assessment of the multi-organ histopathological effects of chronic white noise exposure and the potential restorative effects of post-exposure ascorbic acid administration.

MATERIALS AND METHODS

Ethical approval

The animal experiment from which the tissue specimens used in the present study were obtained was approved by the Ankara University Animal Experiments Local Ethics Committee (Approval No. 2024-10-81). All experimental procedures were conducted in accordance with institutional guidelines and internationally accepted principles for the care and use of laboratory animals.

Animals and housing conditions

At the beginning of the experimental protocol, the animals were 8-10 weeks old and weighed 250-300 g. The original experimental study included twenty clinically healthy wistar albino rats, comprising 10 males and 10 females. Animals were obtained from the Ankara University Experimental Animal Research and Application Center, where the experimental procedures were carried out in 2025.

Following a one-week acclimatization period, the rats were housed under standard laboratory conditions with free access to standard laboratory chow and tap water *adlibitum*. Environmental conditions were maintained at 22±2°C, 50-60% relative humidity and a 12-h light/12-h dark cycle. Male and female animals were housed separately in standard polycarbonate cages. Within each experimental group, animals were housed in cages containing either three or two rats. Throughout the study, the control animals were maintained in a separate quiet room to prevent unintended exposure to the noise stimulus. During the 30-day noise-exposure period, animals in the experimental group had unrestricted access to standard laboratory chow and drinking water. Following completion of the noise-exposure period, ascorbic acid was added to the drinking water of the treatment animals. The amount of ascorbic acid added to the drinking water was calculated based on the body weight of each animal to provide a target dose of 100 mg/kg/day.

Experimental design

Following acclimatization, the animals were randomly assigned to either a control group (n = 10) or a noise-stress group (n = 10).

Animals assigned to the noise-stress group were exposed to 70 dB SPL broadband white noise for 1 h/day over 30 consecutive days, whereas the control animals were maintained under identical environmental conditions without noise exposure.

At the end of the 30-day exposure period, five animals from each group were euthanized and tissue samples from the brain, heart, testis and intestine were collected for histopathological examination.

The remaining animals were maintained for an additional 30 days, during which ascorbic acid was administered through drinking water at a dose of 100 mg/kg/day, calculated according to the body weight of each rat. Following completion of the treatment period, the remaining animals were euthanized and tissue samples from the same organs were collected for histopathological evaluation.

The present study was conducted using archived tissue specimens obtained from animals used in a doctoral thesis approved by the Ankara University Animal Experiments Local Ethics Committee (Approval No. 2024-10-81). No additional animals were used and no further experimental procedures or euthanasia were performed specifically for the present investigation. The use of these archived specimens was consistent with the principles of the 3Rs (Replacement, reduction and refinement).

Noise exposure protocol

Broadband white noise was generated using a timer-controlled digital white-noise generator connected to a 15-W loudspeaker positioned adjacent to the experimental cages. The exposure system was programmed to operate automatically at the same time each day. Animals assigned to the noise-stress group were exposed to 70 dB SPL broadband white noise for 1 h/day over 30 consecutive days. The sound level was adjusted to 70 dB SPL and verified before each exposure session using an Extech 407780A integrated sound level meter (Extech Instruments, China). During each exposure period, the control animals remained in a separate quiet room and were not exposed to the noise stimulus. Throughout the experiment, the control group animals were housed in a separate and quiet laboratory room away from the experimental group and the noise-exposure area.

Histopathological examination and evaluation

Immediately after euthanasia, the whole brain, representative myocardial tissue, representative testicular tissue and a representative segment of the small intestine were collected and fixed in 10% neutral buffered formalin for 24-48 h. Following fixation, tissues were washed under

running tap water, dehydrated through a graded ethanol series, cleared in xylene and embedded in paraffin according to routine histological procedures.

Paraffin blocks were sectioned at 4 µm using a rotary microtome. The sections were mounted on glass slides and stained with hematoxylin and eosin (H and E) using mayer's hematoxylin according to standard histological techniques.

Histopathological examination was performed using a Leica DM2500 light microscope (Leica Microsystems, Wetzlar, Germany) by a board-certified veterinary pathologist who was blinded to the experimental groups. Representative photomicrographs were captured using a digital imaging system attached to the microscope.

All tissue sections were assessed for general histopathological alterations, including degenerative, necrotic, inflammatory, vascular and structural changes. Lesions were evaluated semi-quantitatively according to their severity and distribution using a four-grade scoring system: 0 = no lesion, 1 = mild lesion, 2 = moderate lesion and 3 = severe lesion.

Histopathological scores were assigned based on the overall severity of lesions observed in each tissue section and were used for descriptive comparison of lesion severity among the experimental groups.

Statistical analysis

Histopathological scores were evaluated descriptively using the semi-quantitative four-grade scoring system. Because the primary aim of the study was to characterize the distribution and severity of histopathological lesions, the scoring results are presented as descriptive ordinal data.

RESULTS AND DISCUSSION

Histopathological examination revealed no remarkable lesions in the control animals and the normal tissue architecture of the intestine, brain, heart and testis was preserved. In contrast, chronic exposure to 70 dB SPL broadband white noise induced distinct histopathological alterations of varying severity in all examined organs. The observed lesions included epithelial degeneration and desquamation accompanied by inflammatory cell infiltration in the intestine, neuronal degeneration and gliosis in the brain, myocardial hyaline degeneration in the heart and seminiferous tubular degeneration with necrosis in the testis. Following post-exposure ascorbic acid administration, the severity of the lesions appeared to be reduced and overall tissue architecture was better preserved than in the untreated noise-exposed animals.

The semi-quantitative histopathological lesion scores are presented in Table 1.

The semi-quantitative histopathological scores presented in Table 1 are consistent with the microscopic observations and demonstrate that chronic white noise exposure produced organ-dependent lesion severity. The intestine exhibited the highest lesion score, followed by the testis, whereas brain and heart tissues showed moderate changes. No histopathological lesions were observed in either the control or control + ascorbic acid groups. Following post-exposure ascorbic acid administration, lesion scores decreased to mild levels in all examined organs, supporting the microscopic evidence that ascorbic acid was associated with reduced lesion severity and partial preservation of tissue architecture. The pathological significance of these findings is discussed separately for each organ below.

In the intestine, chronic white noise exposure resulted in evident mucosal injury. The main histopathological findings were degeneration and desquamation of the lamina epithelialis, accompanied by inflammatory cell infiltration within the lamina propria. The inflammatory infiltrate was predominantly composed of mononuclear cells, with occasional eosinophilic granulocytes (Fig 1). These findings indicate that chronic noise exposure may impair intestinal mucosal integrity and induce a local inflammatory response. Following post-exposure ascorbic acid administration, epithelial degeneration, desquamation and inflammatory cell infiltration were less prominent, suggesting partial preservation of the intestinal mucosa.

The intestinal findings of the present study are consistent with Baldwin *et al*. (2006), who reported morphological alterations in the intestinal mucosa of noise-exposed rats, including increased eosinophils and mast cell degranulation in the intestinal villi. The presence of occasional eosinophilic granulocytes in the lamina propria in the present study is particularly compatible with their interpretation that noise-related stress may activate mucosal immune responses. Similarly, Zhang *et al*. (2023) demonstrated that white noise exposure adversely affected gut microbiota, antioxidant activity and immune-related gene expression in mice, together with increased lipid

Table 1: Semi-quantitative histopathological lesion scores in the examined organs.

Organ	Lesion evaluated	Control	Noise stress	Control + AA	Noise-stress + AA
Intestine	Epithelial degeneration, desquamation and inflammatory cell infiltration in the lamina propria	0	3	0	1
Brain	Neuronal degeneration and gliosis	0	2	0	1
Heart	Myocardial hyaline degeneration and structural alteration	0	2	0	1
Testis	Seminiferous tubular degeneration and necrosis	0	2-3	0	1

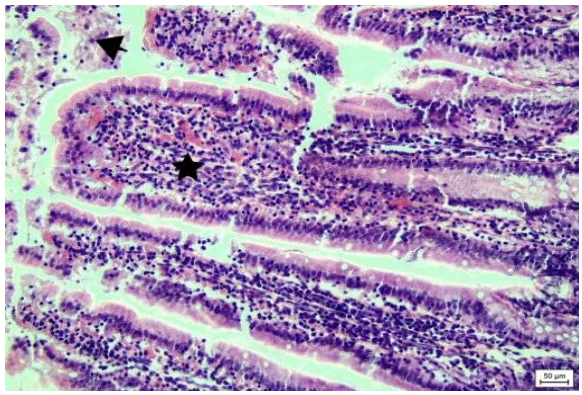
Scoring scale: 0= No lesion; 1= Mild lesion; 2= Moderate lesion; 3= Severe lesion, AA: Ascorbic acid.

peroxidation. In this context, the epithelial degeneration and inflammatory infiltration observed in the present study support the view that chronic noise exposure may disturb intestinal homeostasis through stress-related inflammatory and oxidative mechanisms. However, since the present study was based on routine H and E histopathology and did not evaluate mast cells, epithelial barrier proteins, microbiota, or oxidative stress markers, these mechanisms should be considered as plausible explanations rather than directly proven pathways.

Brain sections from the noise-exposed group showed neuronal degeneration accompanied by gliosis (Fig 2). Degenerative changes were observed in some neurons, while the glial reaction indicated a reactive tissue response to neural injury. These findings suggest that chronic white noise exposure may affect central nervous system tissue and induce both neuronal and glial alterations. In the ascorbic acid-treated animals, neuronal degeneration and gliosis appeared less evident than in the untreated noise-exposed group, indicating a milder degree of neural tissue damage. Similar evidence of stress-related neuronal and inflammatory histopathological alterations, together with partial improvement following an intervention, has been reported in stressed rats (Kranthi *et al*., 2022).

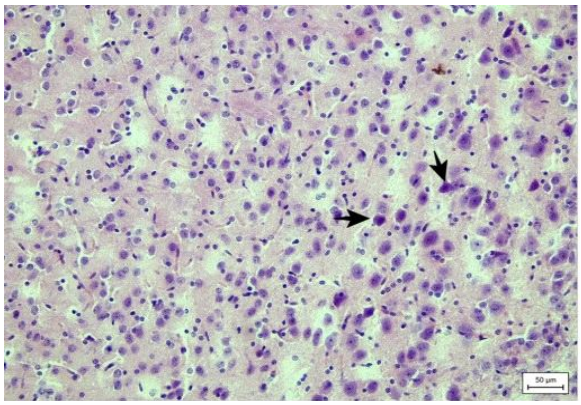
The cerebral lesions observed in the present study are consistent with previous experimental studies indicating that chronic noise exposure can affect the brain tissue beyond the auditory pathways. Gai *et al*. (2017) reported that chronic white noise exposure altered the corticotropin-releasing factor system in the rat hippocampus and was associated with Alzheimer's disease-like tau hyperphosphorylation. More recently, Ma *et al*. (2024) demonstrated that chronic noise exposure induced Alzheimer's disease-like neuropathological changes and cognitive impairment in rats through ferroptosis-related mechanisms in the hippocampus. Although the present study did not investigate specific brain regions or molecular markers such as tau phosphorylation, amyloid-β accumulation, ferroptosis, or oxidative stress, the observation of neuronal degeneration and gliosis supports the concept that repeated noise exposure may trigger neurodegenerative and reactive glial changes. An important difference was that several previous studies used higher noise intensities or focused on molecular alterations in the hippocampus, whereas the present study demonstrated detectable histopathological changes after exposure to 70 dB SPL broadband white noise for 1 hour/day over 30 days. This suggests that even moderate-intensity chronic white noise exposure may be sufficient to produce structural changes in brain tissue, although the severity and distribution of such lesions may depend on noise intensity, duration, frequency characteristics and the brain region examined.

In the heart, myocardial tissue from noise-exposed rats showed hyaline degeneration (Fig 3). This lesion reflects degenerative alteration of myocardial fibers and indicates that chronic white noise exposure may adversely affect cardiac tissue. After ascorbic acid administration, myocardial hyaline degeneration appeared less pronounced and the



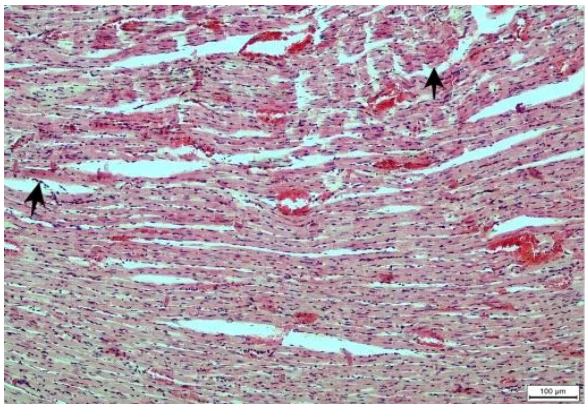
H and E; Scale bar = 50 µm.

Fig 1: Degeneration and desquamation in the lamina epithelialis (arrowhead), mononuclear cell infiltrates (asterisk) in the lamina propria in the intestine.



Arrows indicate degenerated neurons. H and E; Scale bar = 50 µm.

Fig 2: Gliosis and degeneration of some neurons in the brain.



H and E; Scale bar = 100 µm.

Fig 3: Hyaline degeneration in the heart (arrows).

myocardial architecture was better preserved compared with the untreated noise-exposed group.

The cardiac findings of this study support literature suggesting that environmental noise can act as a cardiovascular stress factor. Münzel *et al*. (2018) emphasized that environmental noise can promote cardiovascular injury through neurohormonal activation, oxidative stress, inflammation, endothelial dysfunction and vascular impairment. In an experimental model, Gannouni *et al*. (2014) exposed adult rats to 70 dB(A) noise and reported morphological alterations in the heart, including dilated veins, endothelial damage, inflammatory reactions, myocardial disorganization and necrosis, particularly after longer exposure durations. The myocardial hyaline degeneration observed in the present study is consistent with these reports and supports the possibility that chronic noise exposure may induce degenerative myocardial injury.

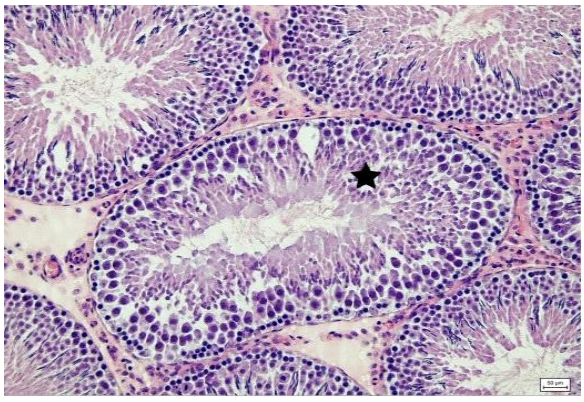
However, not all experimental studies have reported cardiac lesions following noise exposure. Abouee-Mehrizi *et al*. (2021) observed that exposure to 85 dB white noise for 5 consecutive days did not significantly alter heart and lung tissues in rabbits, although liver and kidney tissues were affected. This difference does not necessarily contradict the present findings, because the experimental conditions were markedly different. Species differences, exposure duration, daily exposure schedule, type of noise, tissue susceptibility and the histopathological endpoints examined may all influence the presence and severity of cardiac lesions. In particular, the 5-day noise-exposure model in rabbits used by Abouee-Mehrizi *et al*. (2021) is not directly comparable with the 30-day repeated-exposure protocol used in the present study. Therefore, the present findings suggest that the myocardial effects of noise exposure may be strongly dependent on the experimental model and duration of exposure.

Testicular sections from noise-exposed rats revealed degeneration and necrosis in some seminiferous tubules, accompanied by disruption of the normal seminiferous tubular architecture and degenerative changes affecting the germinal epithelium (Fig 4). These findings indicate that the seminiferous epithelium may be vulnerable to chronic noise-induced stress. Following post-exposure ascorbic acid administration, seminiferous tubular degeneration and necrosis appeared milder and the tubular architecture was relatively better preserved. Edema and hyperemia were observed in the interstitial space.

The testicular findings of the present study are consistent with previous reports indicating that chronic noise stress may impair male reproductive tissue. Lasheen *et al*. (2015) reported that chronic noise exposure caused distortion of seminiferous tubules, loss of normal spermatogenic epithelial organization, degenerative changes and increased apoptotic germ cells in rat testes. Similarly, Swami *et al*. (2007) reported adverse effects of chronic noise exposure on testicular histology and spermatogenesis. The degeneration and necrosis observed in some seminiferous tubules in the present study support these previous findings and suggest that chronic white noise exposure may compromise the structural integrity of the germinal epithelium. Functional studies also support this interpretation. Jalali *et al*. (2012) reported decreased reproductive organ weights and altered sperm parameters in rats exposed to noise pollution. Although the present study did not evaluate sperm count, sperm motility, testosterone concentration, or fertility outcomes, the observed seminiferous tubular lesions may have potential reproductive significance. Therefore, functional reproductive impairment cannot be directly concluded from the present data, but the histopathological findings provide structural evidence that chronic noise exposure may adversely affect testicular tissue.

The reduction in lesion severity following post-exposure ascorbic acid administration is biologically plausible considering the antioxidant and anti-inflammatory properties of ascorbic acid. Previous experimental studies in rats have likewise demonstrated an association between oxidative stress, altered antioxidant defenses and histopathological tissue degeneration (Lohiya *et al*., 2017). In the present study, intestinal epithelial injury and inflammatory infiltration, neuronal degeneration and gliosis, myocardial hyaline degeneration and seminiferous tubular degeneration were all less severe after ascorbic acid administration. These findings suggest that ascorbic acid may contribute to partial tissue recovery or preservation after chronic noise-induced injury.

Previous studies support the protective potential of antioxidant treatment in noise-related tissue damage. Loukzadeh *et al*. (2015) reported that ascorbic acid reduced noise-induced hearing loss in rats, while Le Prell *et al*. (2007) emphasized that oxidative stress is an important mechanism in noise-induced injury and that antioxidant strategies may have protective value. In addition, Carr and Maggini (2017) and Gęgotek and Skrzydlewska (2022) described the free radical scavenging, antioxidant and anti-



H and E; Scale bar = 50 µm.

Fig 4: Degeneration and necrosis of seminiferous tubules in the testis (asterisk).

inflammatory roles of ascorbic acid. The present findings extend this concept by suggesting that post-exposure ascorbic acid administration may be associated with milder histopathological alterations not only in auditory-related injury models but also in systemic organs affected by chronic noise exposure.

An important aspect of the present study is that ascorbic acid was administered after the completion of the noise exposure period rather than before or during exposure. Therefore, the study design is more closely related to post-exposure tissue recovery than to prophylactic protection. The milder lesions observed in the ascorbic acid-treated animals may reflect attenuation of ongoing oxidative and inflammatory tissue responses after cessation of noise exposure. Nevertheless, this interpretation should be made cautiously, because spontaneous recovery after termination of the noise stimulus may also have contributed to the improved histological appearance. The absence of a separate noise-exposed recovery group without ascorbic acid limits the ability to clearly distinguish the restorative effect of ascorbic acid from spontaneous tissue repair.

Overall, the simultaneous occurrence of intestinal, cerebral, myocardial and testicular lesions indicates that chronic white noise exposure may act not only as an auditory stimulus but also as a systemic biological stressor. This interpretation is consistent with Spreng (2000), who suggested that noise exposure may activate stress-related endocrine responses, particularly through cortisol-related pathways. Demirel *et al*. (2009) also demonstrated that noise exposure increased oxidative stress parameters in rats and suggested that noise-induced oxidative imbalance may affect not only the ear but also the whole body. Similarly, Münzel *et al*. (2018) emphasized that environmental noise can induce stress responses, oxidative stress, inflammation, vascular dysfunction and systemic cardiovascular effects. In the present study, the presence of degenerative and inflammatory lesions in multiple organs supports the concept of systemic tissue involvement after repeated noise exposure. However, since oxidative stress markers, inflammatory cytokines, apoptotic pathways and stress hormones were not evaluated, the involvement of these mechanisms remains inferential.

The present study has some limitations. The sample size was limited and the histopathological evaluation was based mainly on routine HandE staining. Biochemical markers of oxidative stress, inflammatory cytokines, apoptotic markers, organ-specific functional parameters and immunohistochemical indicators were not assessed. In addition, detailed regional evaluation of the brain and functional reproductive assessment of the testis were not performed. Since ascorbic acid was administered through drinking water, individual intake may also have varied among animals. Furthermore, although both male and female rats were included in the experimental design, sex-specific histopathological responses were not evaluated. Future studies with larger sample sizes, controlled dosing strategies, biochemical assays, immunohistochemical markers, organ function tests, sex-specific analysis and a separate untreated recovery group are needed to clarify the precise mechanisms involved and to confirm the restorative role of ascorbic acid.

CONCLUSION

In conclusion, the present study showed that chronic exposure to 70 dB SPL broadband white noise was associated with degenerative and inflammatory histopathological alterations in the intestine, brain, heart and testis of rats. The milder lesions observed after post-exposure ascorbic acid administration suggest that ascorbic acid may contribute to the preservation of tissue architecture after chronic noise-induced injury. However, further mechanistic and functional studies are needed to confirm the pathways involved and to clarify the extent of the restorative effect.

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Disclaimers

The views and conclusions expressed in this article are solely those of the author and do not necessarily represent the views of the affiliated institution. The author is responsible for the accuracy and completeness of the information provided but does not accept any liability for any direct or indirect loss resulting from the use of this content.

Informed consent

The original animal experiment from which the archived tissue specimens were obtained was approved by the Ankara University Animal Experiments Local Ethics Committee (Approval No. 2024-10-81). All experimental procedures were conducted in accordance with institutional guidelines and internationally accepted principles for the care and use of laboratory animals.

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Conflict of interest

The author declares that there is no conflict of interest regarding the publication of this article.

REFERENCES

Abouee-Mehrizi, A., Rasoulzadeh, Y., Moucheshi, S. and Mehdipour, A. (2021). Histopathological changes caused by noise exposure in lung, heart, kidney and liver tissues in New Zealand white rabbits. *Journal of Air Pollution and Health.* **6(4):** 257-264. doi: 10.18502/japh.v6i4.8584.

Baldwin, A.L., Primeau, R.L. and Johnson, W.E. (2006). Effect of noise on the morphology of the intestinal mucosa in laboratory rats. *Journal of the American Association for Laboratory Animal Science.* **45(1):** 74-82.

Carr, A.C. and Maggini, S. (2017). Vitamin C and immune function. *Nutrients.* **9(11):** 1211. doi: 10.3390/nu9111211.

Demirel, R., Mollaoğlu, H., Yeşilyurt, H., Üçok, K., Ayçiçek, A., Akkaya, M., Genç, A., Uygur, R. and Doğan, M. (2009). Noise induces oxidative stress in rat. *European Journal of General Medicine.* **6(1):** 20-24. doi: 10.29333/ejgm/82631.

Gai, Z., Su, D., Wang, Y., Li, W., Cui, B., Li, K., She, X. and Wang, R. (2017). Effects of chronic noise on the corticotropin-releasing factor system in the rat hippocampus: Relevance to alzheimer's disease-like tau hyperphosphorylation. *Environmental Health and Preventive Medicine.* **22(1):** 79. doi: 10.1186/s12199-017-0686-8.

Gannouni, N., Mhamdi, A., El May, M., Tebourbi, O. and Rhouma, K.B. (2014). Morphological changes of adrenal gland and heart tissue after varying duration of noise exposure in adult rat. *Noise and Health.* **16(73):** 416-421. doi: 10.4103/1463-1741.144424.

Gęgotek, A. and Skrzydlewska, E. (2022). Antioxidative and anti-inflammatory activity of ascorbic acid. *Antioxidants.* **11(10):** 1993. doi: 10.3390/antiox11101993.

Jalali, M., Saki, G., Sarkaki, A.R., Karami, K. and Nasri, S. (2012). Effect of noise stress on count, progressive and non-progressive sperm motility, body and genital organ weights of adult male rats. *Journal of Human Reproductive Sciences.* **5(1):** 48-51. doi: 10.4103/0974-1208.97801.

Kranthi, T.R., Archana, R. and Senthilkumar, S. (2022). Vestibular stimulation as an interventional approach for cold water stress induced immunological and histopathological changes in rats. *Indian Journal of Animal Research.* **56(7):** 830-836. doi: 10.18805/IJAR.B-4491.

Lasheen, S.S., Refaat, S. H., El-Nefiawy, N.E., Abd-Elgawad, R.A., Othman, A.I. and Olama, N.K. (2015). Developmental characteristics of rat testicular tissue and the impact of chronic noise stress exposure in the prenatal and postnatal periods. *Anatomy and Physiology.* **S4:** 004. doi: 10.4172/2161-0940.S4-004.

Le Prell, C.G., Yamashita, D., Minami, S.B., Yamasoba, T. and Miller J.M. (2007). Mechanisms of noise-induced hearing loss indicate multiple methods of prevention. *Hearing Research.* **226(1-2):** 22-43. doi: 10.1016/j.heares.2006.10.006.

Lohiya, A., Kumar, V. and Punia, J.S. (2017). Imidacloprid induced oxidative stress and histopathological changes in liver of rats. *Indian Journal of Animal Research.* **51(3):** 531-536. doi: 10.18805/ijar.v0iOF.7805.

Loukzadeh, Z., Hakimi, A., Esmailidehaj, M. and Mehrparvar, A.H. (2015). Effect of ascorbic acid on noise induced hearing loss in rats. *Iranian Journal of Otorhinolaryngology.* **27(4):** 267-272. doi: 10.22038/ijorl.2015.4515.

Ma, J., Zhang, J., Ou, Z., Ren, Y., Wu, K., Zhang, Y., Chen, S. and Wang, Z. (2024). Chronic noise exposure induces alzheimer's disease-like neuropathology and cognitive impairment via ferroptosis in rat hippocampus. *Environmental Health and Preventive Medicine.* **29:** 50. doi: 10.1265/ehpm.24-00126.

Münzel, T., Schmidt, F.P., Steven, S., Herzog, J., Daiber, A. and Sørensen, M. (2018). Environmental noise and the cardiovascular system. *Journal of the American College of Cardiology.* **71(6):** 688-697. doi: 10.1016/j.jacc.2017.12.015.

Spreng, M. (2000). Possible health effects of noise induced cortisol increase. *Noise and Health.* **2(7):** 59-64.

Swami, C.G., Ramanathan, J. and Jeganath, C.C. (2007). Noise exposure effect on testicular histology, morphology and on male steroidogenic hormone. *The Malaysian Journal of Medical Sciences.* **14(2):** 28-35.

Xue, L., Zhang, D., Xiaokaiti-Yibulayin, Wang, T. and Shou, X. (2014). Effects of high frequency noise on female rat's multi-organ histology. *Noise and Health.* **16(71):** 213-217. doi: 10.4103/1463-1741.137048.

Ünsaldı, E. and Kalınbacak, A. (2025). Effect of exposure to noise stress on liver tissue histopathology in rats. *Indian Journal of Animal Research.* **59(10):** 1773-1777. doi: 10.18805/IJAR.BF-2048.

Yang, Z.J., Zhao, C.L., Liang, W.Q., Chen, Z.R., Du, Z.D. and Gong, S.S. (2024). ROS-induced oxidative stress and mitochondrial dysfunction: A possible mechanism responsible for noise-induced ribbon synaptic damage. *American Journal of Translational Research.* **16(1):** 272-284. doi: 10.62347/EVDE9449.

Zhang, Z., Wu, Y., Zhou, S., Fu, P. and Yan, H. (2023). Effects of music and white noise exposure on the gut microbiota, oxidative stress and immune-related gene expression of mice. *Microorganisms.* **11(9):** 2272. doi: 10.3390/microorganisms11092272.