



Noise Stress Impairs Social Interaction in Adult Male Mice: Role of Oxidative Stress and Neuroendocrine Markers

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Abstract

Objectives: Noise is a potential environmental stressor affecting brain function as evidenced by altered stress neurotransmitters and hormone levels and psychobehavioral changes. Here, we explored the effects of noise exposure on the social behavior of mice as well as cerebral oxidative stress and neuroendocrine markers.

Materials and Methods: In this experimental study, adult male mice were randomly allocated in; control-acute, acute-noise 90-dB, acute-noise 110-dB, control-chronic, chronic-noise 90-dB, and chronic-noise 110-dB groups. Animals in acute- and chronic-noise groups were exposed to 2 h/day of 90- or 110-dB white noise for one-day and three-month, respectively. At the end of the noise interventions, mice subjected to social interaction test. Also, serum levels of cortisol and cerebral malondialdehyde (MDA) levels, and superoxide dismutase (SOD) and glutathione peroxidase (GPx) activities as well as total antioxidant capacity were measured.

Results: Although mice in both acute noise 90- and 110-dB groups did not show any alteration in social behaviors and novel recognition performance, mice in chronic noise in 90- and 110-dB groups exhibited lower levels of mentioned behavioral paradigms. Moreover, these behavioral changes in the both chronic noise groups were accompanied by lower body weight, increased MDA levels, and decreased SOD and GPx activities, and reduced total antioxidant capacity levels. Interestingly, the acute noise increased brain MDA levels as well as enhanced SOD and GPx activities and total antioxidant capacity. Finally, we found lower serum cortisol levels in both acute and chronic groups.

Conclusions: The noise exposure at levels ≥ 90 -dB impairs sociability, associated with an imbalance of cerebral oxidative status and increased stress hormone.

Keywords: Chronic noise stress, Acute noise stress, Social interaction, Social behaviors, Antioxidant status, Cortisol

Introduction

The sound is considered as a mechanical wave that plays a cardinal role in the communication; however, undesirable levels of sound are characterized as noise. With fast industrialization and urbanization, the problem of noise pollution, particularly in big cities, has become a major public health challenge. Noise exposure is a serious environmental stressor leading to numerous disturbances in human and non-human species. The exposure to noise stress resulted in exhaustion, annoyance, and arousal response followed by sleep disturbance (1). Evidence has shown that noise exposure at levels exceeded 80 dB can damage the physiological function of auditory systems and also affects non-auditory systems including central nervous system (CNS) (2). With respect to CNS, pre-clinical studies have demonstrated detrimental effects of short- and long-term noise stress on the brain function as evidenced by altered neurotransmitters and neuroendocrine factors and subsequent psychobehavioral and cognitive changes (3,4).

From the neurobiological perspective, during the experience with noise stress, acute responses to noise stimuli can cause a CNS-mediated stress reaction leading to an alternation of several neurophysiological functions. The hypothalamic pituitary–adrenal (HPA), as a main hormone system, plays a basic role in the maintenance of homeostatic balance in response to stressful events. Evidence has shown that noise stress causes HPA axis activation and an instantaneous secretion of corticosteroids (5). The increased corticosterone level accelerates the production of free radicals, which in turns suppresses the immune function. Due to the highest percentage of unsaturated fats in the neurons, cerebral tissues are highly sensitive and vulnerable to oxidative stress damage caused by lipid peroxidation (6). Indeed, oxidative stress has been acknowledged to have a crucial role in the development of noise stress-related brain dysfunction (7). The neurophysiological reactions to noise stress are also accompanied by some temporary or lasting neurobehavioral symptoms including cognitive

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impairment, mood disturbance, as well as anxiety and stress responses (8).

Owing to the increase in the noise producing stressors in the daily life, the exploration of effect of noise stress on various aspects of mental health is clearly warranted. To the best of our knowledge, this is the first report investigating the adverse effects of noise exposure on the social interaction behavior of mouse. In this study, the neuronal oxidant and antioxidant markers as well as neuroendocrine marker namely, serum cortisol, were also measured as possible involved mechanisms in the observed behavioral deficits.

Materials and Methods

Animals and Experimental Design

Sixty adult male BALB/c mice (21-23 g) were obtained from the animal center of Tabriz University of Medical Sciences, Iran. Mice were housed in cages (5 mice/cage) for at least 10 days to adapt to the new environment before the experiment. All mice were kept in a room at $25 \pm 1^\circ\text{C}$ temperature and 12 h light/dark cycle, with free access to standard laboratory chow and tapwater ad libitum. After the acclimatization period, animals were randomly divided into six groups ($n=10$): (I) control-acute noise, (II) acute-noise 90-dB, (III) acute-noise 110-dB, (IV) control-chronic noise, (V) chronic-noise 90-dB, and (VI) chronic-noise 110-dB. Animals in the I and IV groups were kept in their home cages under silent conditions (≤ 40 dB SPL). Mice in the II and III groups were subjected to acute-noise 90-dB and 110-dB, respectively. While the groups of V and VI were subjected to chronic-noise 90-dB and 110-dB, respectively. During the experiment, all animals in the chronic groups were weighed weekly.

Noise Stress Induction

The noise stress procedure was carried out according to the method described previously (9). Briefly, animals were exposed to 90 or 110 dB sound pressure level (SPL) white noise (2 h/day; 11:00 AM– 1:00 PM). The white noise with a frequency range of 20–20000 Hz was produced by a computer software (NCH Tone Generator 3.26) and delivered to a loudspeaker, which located at 30 cm from the animal's cage. The noise intensity was monitored by a sound level meter (Smart Tools Co., Ltd., Japan). The acute and chronic noise stress were induced for one-day and three-month, respectively.

Social Interaction Test

The social interaction test was performed as described previously by Kaidanovich-Beilin et al (10). The apparatus was comprised of a rectangular open arena (L,W, H: $57 \times 45 \times 30$ cm), including a three-chamber box with bedding of grey gravel. Each chamber was 19×45 cm and the dividing walls were made from transparent Plexiglas, with an open middle section (10×28 cm) allowing access into each chamber. The task consisted of habituation

(adaptation) session and sociability (phase-I), and social novelty (phase-II) sessions. In the habituation session, the subject mouse was located at the center of the middle chamber and allowed to explore it for 5 minutes, while the dividing Plexiglas was put on either side of the walls. Then, one control mouse (designated as a stranger 1 mouse) with the same background of gender, age, and weight was placed inside a wire containment cup that was located on one of the side chambers. The social affiliation aspect of the task immediately began with removing the walls between the compartments allowing free access for the subject mouse to explore each of the three chambers for 10 minutes. Then, to perform social novelty session, a second control mouse was placed inside a wire containment cup in the opposite side chamber, as a stranger 2 mouse, and subject mouse was allowed to freely explore the chambers for next 10 minutes. At the end of each trial, the mouse was removed from the apparatus, any boluses were removed and the floor and walls were cleaned with 70% ethanol. The behavior of each mouse was recorded by a video camera that was placed above the arena. Social interaction between subject mouse and strange 1 mouse was expressed as a ratio of time spending around the housing to non-housing containment cup. Direct contact between the subject mouse and the containment cup was counted as an active contact. Moreover, the ratio of the number of entries to a chamber with strange 1 mouse versus empty chamber was determined, as a phase I-entrance index. For the phase II, the same parameters described in phase I were determined, whereas behaviors between the subject mouse in the presence of stranger 1 compared with stranger 2 were considered.

Sampling

After behavioral tests, mice were deeply anaesthetized with an intraperitoneal injection of ketamine (90 mg/kg) and xylazine (10 mg/kg) (6), and then blood samples were collected by cardiac puncture and centrifuged at 4000 rpm for 15 minutes at room temperature and serum samples were stored at -70°C . Then animals were sacrificed by decapitation and the whole brain were quickly removed and frozen in liquid N_2 . Tissue samples were homogenized in ice-cold 1.15 % KCl solution and centrifuged at 112 g for 10 minutes at 4°C , and the supernatant was used to determine malondialdehyde (MDA) and total antioxidant capacity (TAC) levels, as well as enzymatic activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx).

Lipid Peroxidation and Antioxidant Assays

The MDA levels, a marker of lipid peroxidation, were measured using the thiobarbituric acid reactive substances (TBARS) method as previously described (11). To determine MDA levels, the absorbance of pink colour of TBARS producing by the reaction of MDA with TBA was spectroscopically measured and expressed as MDA

per mg of protein.

SOD activity was determined using a RANSOD kit (Randox Laboratories Ltd, Crumlin, UK) based on the method previously described. The absorbance was measured spectrophotometrically at $\lambda = 505$ nm at 37°C (6) and results were expressed as U per mg protein.

GPx activity in the brain supernatant was determined using RANSEL (Randox Laboratories Ltd, Crumlin, UK) assay kit. The decrease in absorbance of NADPH was measured at 340 nm (37°C) using a spectrophotometer. GPx activity was expressed as unit (U) per mg protein (6).

The brain TAC levels were colorimetrically assessed using a Randox total antioxidant status kit (Randox Laboratories Ltd, Crumlin, UK) according to the manufacturer's instructions and the results were expressed as nmol/L.

Serum Cortisol Measurement

Serum cortisol concentration was determined using a specific enzyme-linked immunosorbent assay (ELISA) kit for cortisol (IBL International, Germany) following the manufacturer's instructions. To avoid fluctuations on cortisol concentrations due to circadian rhythms, blood samples were collected between the hours of 10:00 and 12:00 noon.

Statistical Analysis

Statistical analyses were performed using GraphPad Prism 6.01 (Graph Pad Software Inc., La Jolla, CA, USA) and data expressed as mean \pm standard error mean (SEM). Differences between groups for all behavioral and biochemical data were carried out by one-way ANOVA followed by Tukey post hoc test. The one-way ANOVA was also used for comparison of data related to the body-weight gain in the chronic groups during the three-month of noise induction. Differences were considered as

significant if $P < 0.05$ (in all versus corresponding control).

Results

Social Interaction Test

Phase-I (Sociability Performance)

As shown in Figure 1A (left), Tukey post hoc comparison showed a non-significant decrease in the social interaction index after a 2-hour exposure to 90 dB and 110 dB noise (for both, $P > 0.05$). However, chronic exposure to noise stress revealed a remarkable decrease in the social interaction index for both 90 dB and 110 dB noise groups as compared to control group (for both, $P < 0.001$) (Figure 1A, right). Data from entrance index for phase-I showed a decrease in both 90 dB and 110 dB acute groups; however, these decreases were not significant by ANOVA (for both, $P > 0.05$) (Figure 1B, left). However, chronic exposure to noise stress showed a significant decrease in the entrance index for both 90 dB and 110 dB noise groups when compared to control group (for both, $P < 0.01$) (Figure 1B, right).

Phase-II (Social Novelty Performance)

As shown in Figure 1C (left), Tukey post hoc comparison showed a non-significant decrease in the novel recognition index after a single exposure to 90 dB and 110 dB noise (for both, $P > 0.05$). However, chronic exposure to noise stress resulted in a remarkable decrease in the novel recognition index for both 90 dB and 110 dB noise groups as compared to control group (for both, $P < 0.01$) (Figure 1C, right). Data from entrance index for phase-II exhibited a non-significant decrease in both 90 dB and 110 dB acute groups (for both, $P > 0.05$) (Figure 1D, left). However, chronic exposure to noise stress showed noticeable decrease in the entrance index for both 90 dB and 110 dB noise groups when compared to control group (for both, $P > 0.001$) (Figure 1D, right).

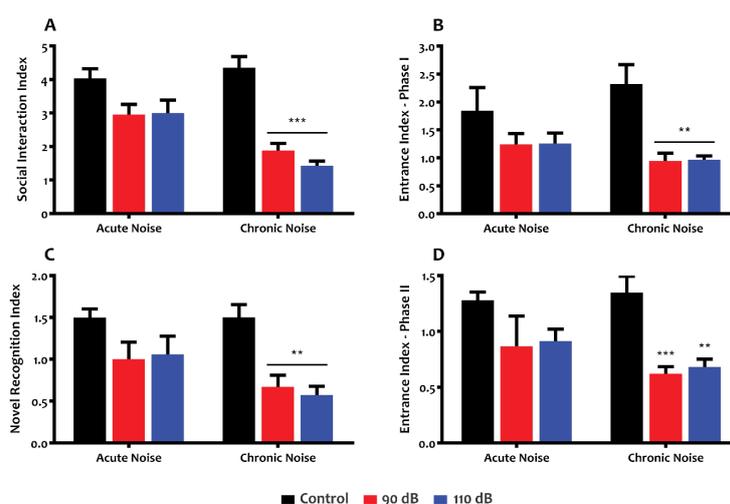


Figure 1. The effects of acute (one-day) and chronic (three-month) noise exposure at 90 dB and 110 dB on (A) social interaction index, (B) entrance index – phase I, (C) novel recognition index, and (D) entrance index – phase II. Data represent mean \pm SEM. ** $P < 0.01$ and *** $P < 0.001$ vs. corresponding control groups.

MDA Levels

As shown in Figure 2A (left), acute noise stress at both 90 dB and 110 dB considerably increased brain MDA levels (for both, $P > 0.001$). Mice in chronic noise groups also showed a significant increase in the cerebral MDA levels as compared to control group (for both sound levels, $P > 0.05$); however this increase was somewhat less than those in the acute noise groups (Figure 2A, right).

Antioxidant Status

Our results (Figure 2B, left) showed that mice in acute noise groups exhibited a significant elevation in the brain SOD activity (for both sound levels, $P < 0.01$). On the other hand, as shown in Figure 2B (right), the SOD activity in the brain was markedly decreased by a three-month exposure to noise stress compared to untreated stress group (for both sound levels, $P < 0.01$).

As shown in Figure 2C (left), following a 2-hour exposure to 90 dB and 110 dB noise, a significant increase in GPx enzyme activity levels was also observed in the brain of animals when compared with those in the control animals (for both sound levels, $P < 0.001$). On the other hand, although chronic exposure to noise stress for three-month resulted in a decrease in the brain GPx activity, this decrease was only significant at 110 dB sound level with respect to control-chronic noise group ($P < 0.05$) (Figure 2C, right).

As shown in Figure 2D (left), the TAC levels were non-significantly decreased in the mice brain subjected to acute noise stress as compared to control-acute noise group (for both sound levels, $P > 0.05$). However, a significant decline in brain TAC levels was observed in the chronic noise groups when compared to control-chronic noise group (for both sound levels, $P < 0.001$) (Figure 2D, right).

Serum Cortisol Levels

Serum cortisol levels were measured at the end of experiment. As shown in Figure 3 (left), Tukey post-hoc comparison showed a significant increase in the serum cortisol levels following a 2-hour exposure to 90 dB and 110 dB noise (for both, $P < 0.001$). In addition, mice in chronic noise groups exhibited a significant elevation in the serum cortisol levels (for both sound levels, $P < 0.05$) (Figure 3, right).

Body Weight Gain

As shown in Figure 4, with respect to chronic groups, baseline body weights of control and 90 dB and 110 dB noise groups was not significantly different ($P > 0.05$). The trend of weight gain in both 90 dB and 110 dB noise groups were more gradual than in the control group over the 3-month. Tukey post hoc comparison revealed that body weight of mice in the chronic 90 dB and 110 dB noise groups were significantly lower than in the control group during the 6th and 12th weeks of noise exposure (for 90 dB: $P < 0.05, 0.05, 0.05, 0.05, 0.05$, and 0.01 ; for 110 dB: $P < 0.05, 0.05, 0.05, 0.01, 0.05$, and 0.01 , respectively).

Discussion

The main feature of the present study is a determination of the sociability status following exposure to loud noise. This is a novel issue in the field of psychiatry and mental health. The present work showed that chronic noise stress led to social interaction deficit as confirmed by decreased ratio of time spending around the housing to non-housing containment cup, decreased novel recognition and the number of entries to the chamber with strange mice compared to the control. Moreover, these behavioral changes in chronic groups were accompanied by

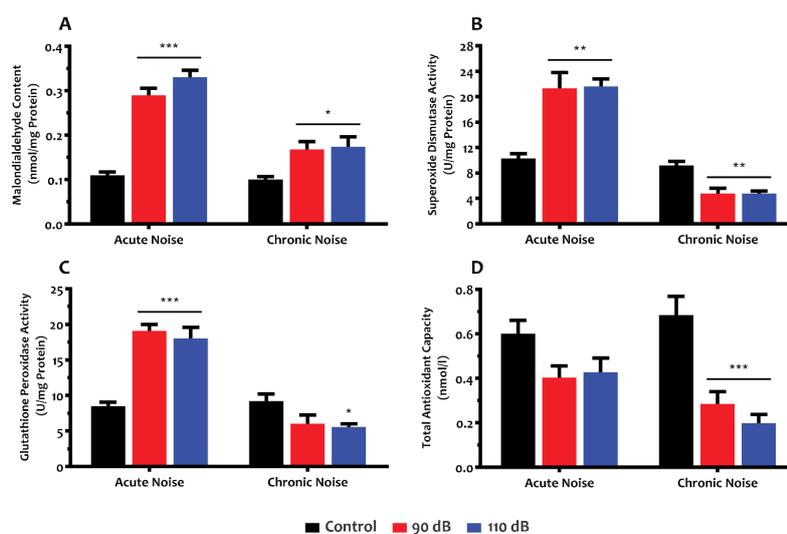


Figure 2. The effects of acute (one-day) and chronic (three-month) noise exposure at 90 dB and 110 dB on the brain (A) MDA levels, (B) SOD activity, (C) GPx activity, and (D) TAC levels. Data represent mean \pm SEM. * $P < 0.05$, ** $P < 0.01$, and *** $P < 0.001$ vs. corresponding control groups. (GPx, glutathione peroxidase; MDA, malondialdehyde; SOD, superoxide dismutase; TAC, total antioxidant capacity).

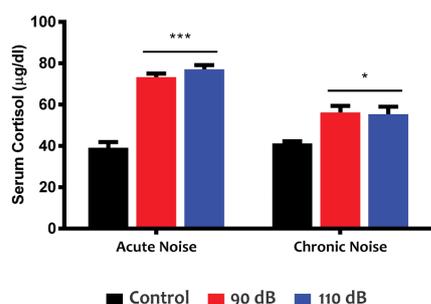


Figure 3. The effects of acute (one-day) and chronic (3-month) noise exposure at 90 dB and 110 dB on (A) serum cortisol levels. Data represent mean \pm SEM. * P <0.05 and *** P <0.001 vs. corresponding control groups.

attenuated weight gain, increased MDA levels, as an index of lipid peroxidation, and decreased SOD and GPx enzymatic activities, and attenuation of TAC levels. Interestingly, acute noise stress increased MDA levels as well as enhanced antioxidant enzyme activity (SOD and GPx) and TAC. In addition, both acute and chronic noise stress increased serum concentration of stress hormone, namely cortisol.

Noise exposure is a potent psychological stressor, which may result in hearing impairment or physiological and behavioral deficits. Although social interaction is essential for mental and physical health, the effect of noise on the social behavior of animals is mainly mysterious and mechanisms by which noise stress affects social behavior and cognition are quite complex. Social interaction test in rodents have been widely used to assess pharmacological effects on anxiety-like behaviors in which increase social interaction indicates an anxiolytic effect (12). In this study, although acute noise stress produced no response in the social interaction test, chronic 90 and 110 dB noise exposures markedly decreased time spend in the social interaction with unfamiliar partner and the number of entries to the interaction chamber containing a strange mouse in phase I and II of the test. It has been shown that decreased social interaction is a sign of an anxiogenic effect (13). Similar to our findings, File et al have demonstrated decreased social interaction in rats housed under noisy conditions for 24 days compared to quiet conditions (14). Otten et al have reported that prolonged or intermittent 90 dB noise exposure decreased social interactions in pigs (15). Studies in the various stress models have also reported social interaction deficits in rodents. Maternal separation has also been shown to decrease social interaction and enhanced plasma adrenocorticotrophic hormone (ACTH) levels (16).

Stress can increase the risk of psychiatric disorders, including mood and anxiety disorders (17). Stress also causes structural and functional disturbances in many brain areas involved in cognitive functioning, such as the prefrontal cortex and hippocampus (18). Impaired social functioning is a hallmark of psychiatric disorders

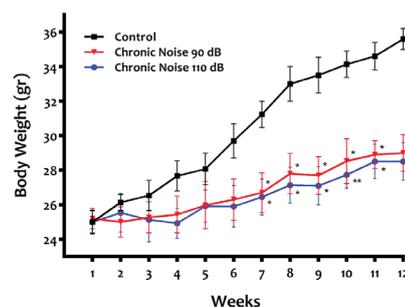


Figure 4. The effects of acute (one-day) and chronic (3-month) noise exposure at 90 dB and 110 dB on body weight gain. Data represent mean \pm SEM. * P <0.05 and ** P <0.01 vs. corresponding control group.

and highly associated with anxious and depressive moods which is possibly due to impaired signaling of reward system in the brain (19). On the other hand, psychiatric disorders such as depression and anxiety have been shown to comorbid with disturbances in social behavior, which commonly emerge before these disorders (20). Hence, these data highlight the importance of studying social interactions deficit for understanding the development of the stress response.

According to previous reports, exposure to stress activates autonomic sympathetic nervous system and HPA axis. The activation of these systems increased the release of the catecholamines and glucocorticoids namely cortisol, respectively (21). Indeed, the release of glucocorticoids from the HPA axis in response to stress is a coping response contributing to physiological adjustment to the stressful conditions (22). Results of this study also showed that both acute and chronic noise stress elevated cortisol levels compare to control animals indicating activation of HPA. Noise that exceeds 90 dB is stressful and activates the HPA axis (4). Samson et al have demonstrated that acute, sub-acute, and chronic exposure to broadband white noise (100 dB) increased the plasma levels of stress hormones including corticosterone and norepinephrine (NE) in adult male rats (3). Wang et al also reported that 3 weeks noise exposure increases corticosterone levels in mice (23).

Additionally, clinical and experimental researches have reported that administration of glucocorticoid (cortisol in humans and corticosterone in rodents) alone is able to increase ROS levels and down-regulates different antioxidant enzymes (24). Therefore, hyperactivation of HPA axis by stress may eventually increase oxidative stress in the brain.

Oxidative stress is a state of the imbalance between oxidants and antioxidants due to enhanced production of reactive oxygen and nitrogen species (25). Moreover, elevated oxidative stress plays a critical role in the pathophysiology of several psychiatric disorders (26). Furthermore, evidence shows that chronic stress affects the expression of various genes involved in regulating

of antioxidant systems, SOD, GPx, catalase, glutathione reductase (GSH), and NADPH oxidase (27). Animal studies using different intensity of noise confirm that exposure to noise stress is associated with excess levels of ROS and free radicals in the brain resulting in cellular components damage and functional abnormalities (28). In this study, elevated levels of MDA levels and reduced activity of SOD and GPx, as well as reduced TAC levels were observed in the brain of chronic noise stress subjected animals, which support the opinion that the noise results in the oxidative stress. Consistent with our findings, studies have demonstrated that acute as well as chronic noise stress produce excessive free radicals in different brain regions (7). In support to this finding, Yildirim et al also reported that noise exposure increases MDA levels and decrease in SOD and catalase in the blood samples of textile workers exposed to 105 dB noise (29). Likewise, Chen et al have demonstrated that oxidative stress increases in different brain regions following 6 weeks of noise exposure (80 dB) in rats (30). Experimental studies have also found that exposure to noise increased superoxide and hydroxyl radicals in cochlea (31). Interestingly, one-day exposure to noise stress increased MDA levels and the activity of SOD and GPx. Ohlemiller et al showed that acute (1 hour) exposure to 110 dB noise increase ROS levels in the cochlea (31). SOD and GPx are antioxidant enzymes, which have protective effects against oxidative stress damage through removing free oxygen radicals. It is possible that increases in antioxidant activities, in this study, are compensatory mechanisms to scavenge and counteract oxidative stress damage.

In this study, control animals of chronic groups showed a significant weight gain, whereas no such weight gain was observed in animals subjected to noise stress. This result is in accordance with previously published studies, which show a similar effect. Taban et al have recently reported that 30 days exposure to 90 dB noise stress reduced body weight in mice (32). Jalali et al have also shown reduced body weight in rats subjected to chronic (50 days) noise with 90-120 dB intensity (33).

Conclusions

Noise stress induces a negative effect on the social interaction, associated with increased generation of MDA and increased stress hormone. Therefore, decreasing noise exposure is critical for avoiding noise-induced abnormalities. Results from our translational study indicate that daily exposure to the noise stimulus at levels exceeded 90 dB can considerably affect social functioning in mice. Modernity and emerging of novel technologies, as well as industrialisation have completely changed the human life. In this respect, daily exposure to noise stress not only affects the physical health but also causes various adverse psychological complications. Therefore, the evaluation of the possible impacts of the noisy environment on the human sociability seems to

be a great importance. We should state that a limitation of our study is the lack of the neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) for mapping of the alterations in the affected cerebral structures following noise stress..

Conflict of Interests

Authors have no conflict of interests.

Ethical Issues

The study protocol was approved by the ethics committee of AJA University of Medical Sciences, Iran (code number: IR.AJAUMS.REC.1397.013).

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