Chapter 8

Studies on Effects of Aircraft Noise on Behavior of Rats, Their Plasma Norepinephrine Levels and Cell Morphology of the Temporal Lobe

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Additional information is available at the end of the chapter

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Abstract

To study the physiological effects of airport noise exposure on organisms, Sprague-Dawley (SD) rats were exposed in soundproof chambers to previously recorded aircraft-related noise for 65 d. As a comparison, unexposed control rats were also used. According to aircraft flight schedules, aircraft noise was replayed and its weighted equivalent continuous perceived noise levels (L_{WECPN}) were adjusted to 75 and 80 dB for the two experimental groups. Rat behaviors were observed through an open field test and the concentrations of plasma norepinephrine (NE) were tested by high-performance liquid chromatography-fluorimetric detection (HPLC-FLD). The morphologies of neurons and synapses in the temporal lobe were also examined by transmission electron microscopy (TEM). Our results indicated that SD rats of experiment group exposed to airport noise of 80 dB had significantly lower line crossing number ($P < 0.05$) and significantly longer center area duration ($P < 0.05$) compared with that of control group. After 29 d of airport noise exposure, the concentrations of plasma NE of experiment group were significantly higher than that of control group ($P < 0.05$). It was determined that the neuron and synapsis of the temporal lobe of experiment group exposed to 80 dB for 65 d showed signs of damage. In conclusion, exposing rats to long-term aircraft noise affects their behaviors, plasma NE levels, and cell morphology of the temporal lobe. Of course, the differences in the hearing sensitivity to different sound frequencies and circadian rhythms between rats and humans can bring variances in physiological effects under the same noise exposure. Therefore, if this study results are applied into humans, it should be further confirmed.

Keywords: Aircraft noise, open field test, norepinephrine, neuron, synapse

1. Introduction

Studies have shown that aircraft noise has a great impact on the health status of populations residing in areas near air traffic, particularly citing cardiovascular diseases and the use of sleep and cardiovascular medications [1, 2]. In addition, researchers have concluded that there is a dose-response relationship between aircraft noise levels and blood pressure of the residents of an area near a military aircraft center [3].

However, most knowledge of the physiological effects of noise is generally obtained through animal experiment. To begin with, the open field test (OFT) is commonly used as a mechanism to assess the neurobehavioral effects of noise. Katz *et al*. [4] showed that white noise of 95 dB increased motor behaviors of rats in OFT and decreased their defecation after 1 h of acute stress. Food intake in OFT is reduced when rats are exposed to white noise of 95 dB, while their defecation increases [5]. In addition, the levels of neurotransmitters or hormones in plasma or brain may reflect the neurobiological effects of noise. Typically, the concentration of norepinephrine (NE) in the brain and cochlea of rats decreases after acute noise stress [6, 7]. Corticosterone levels in mice plasma have been shown to be significantly increased when exposed to acute noise for 10 min [8]. Male rats exposed to broadband white noise of 100 dB have been shown to have a significantly increased level of NE in the brain and corticosterone in plasma [9]. Furthermore, observing the morphological changes in neuronal cells can assess the effects of noise. Outer hair cell apoptosis has been observed in the cochlea of chinchilla after intense impulse noise exposure [10]. In addition, continuous noise stress has been shown to affect both degeneration of epithelial cells and apoptosis of stromal cells in the brain of pig [11]. We have not found any studies indicating that noise exposure induces morphological damage in the temporal lobe, the lobe related to perception and memory [12].

None of the aforementioned studies studied airport noise. Broadband white noise was diffusely applied in previous experimental studies to examine the physiological effects of noise. However, it is important to note that white noise in normal environment is almost non-existent. In China, the aircraft-related weighted equivalent continuous perceived noise level (L_{WECPN}) in many residential areas around airports overstepped the 75 dB limit stipulated by the "Standard of Aircraft Noise for Environment around Airport" policy. In this study, we thus sampled actual aircraft noise and played it back to laboratory rats. We then systematically studied their behaviors, plasma NE levels, and cell morphology of the temporal lobe.

Of course, there are some differences in the hearing sensitivity to different sound frequencies and circadian rhythms between rats and humans, which can bring variances in physiological effects under the same noise exposure. Therefore, if this study results are applied into humans, it should be further confirmed.

2. Materials and methods

When airplanes took off or landed at Xiaoshan International Airport (Hangzhou, China), aircraft noises on the roof of a residential building standing 100 m from the edge of airport

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were sampled using an LDS four-channel dynamic signal analyzer (Photon II, Royston, England). Based on the 24-h flight schedule of airport and airplane type, aircraft noises were played through a non-directional dodecahedron sound source (Nor270, Norsonic, Lierskogen, Norway) and the intensity of noise was adjusted by a power amplifier (Nor280, Norsonic, Lierskogen, Norway). The sound absorber and insulation device were optimally assembled so that the L_{WECPN} values of the experimental groups I (EG-I) and II (EG-II) were (75 ± 1.0) dB (L_{Aeq} = 65.3 dB) and (80 ± 1.0) dB (L_{Aeq} =70.3 dB), respectively. In addition, the laboratory was customized to better control acoustics, as the doors were sound-proofed and the vents were installed with mufflers so that background noise was no more than 40 dBA, the highest sound intensity heard by our control group (CG). We measured the intensity of noise exposure with a sound level meter (AWA6291, Hangzhou, China), which was sound-calibrated by a loud-speaker before measurement.

Fifty male Sprague-Dawley rats (6 weeks old, weighing (150 ± 20) g) were purchased from the Experimental Animal Center of Zhejiang University, and were randomly divided into three groups: CG (n = 10), EG-I (n = 20), and EG-II (n = 20). Rats were housed five per cage and maintained in temperature- controlled ((21 ± 3) °C) rooms with cycles of 12 h of light and 12 h of dark (light on at 8:00 a.m. daily), and allowed free access to water and food. Rats were marked on their fur with picric acid to distinguish individuals. Before experiments were carried out, SD rats were bred for 3 days to adapt to the laboratory environment. After this, SD rats in groups EG-I and EG-II were exposed to aircraft noises, while the CG was not exposed. OFT and blood collection for neurotransmitter determination were carried out at 17:00 on Days 1, 8, 15, 22, 29, and 36 after noise exposure (blood collection excluded on Day 36). All data including OFT and neurotransmitter determination were collected on the same rats in CG (n = 5), EG-I (n = 10), and EG-II (n = 10). For investigating long-term effects of airport noise, after 65 d of continuous (excepting time for OFT/blood collection) noise exposure, four rats each were randomly selected from CG and EG-II, respectively, for additional neuronal morphology studies. Animal breeding and experiments were performed in line with the "Quality Management Approach to Laboratory Animals," and all efforts were made to minimize the number of animals used and their suffering.

The size of open field was 100 × 100 × 50 cm, and its bottom divided into 25 grids (20 × 20 cm) by white lines. We termed the nine grids located in the center of the open field as "center area." The open field was located in a 2.0 × 2.0 m audiometric cabin and lit by a 15-watt red lamp for background lighting. We handled the rats by the base of their tails, carried them to the center of the open field, and allowed them to explore the apparatus for 5 min. The behaviors of the rats were tracked and recorded by the camera fixed above the apparatus. The behaviors measured included line crossing number and center area duration.

In order to test the concentration of neurotransmitters in each group, venous blood (1.0 ml) was sampled from the orbital vein. Blood was transferred into 1.5 ml boil-proof microtubes (Axygen, United States) and was kept quiescence for 10 min, and then centrifuged at 4000 r/min for 10 min at 4°C. Next, 200 μl of supernatant was extracted from each sample and 200 μl of 5% perchloric acid was added to it. Their mixture was shaken, left at room temperature for 20 min to fully precipitate the plasma proteins, and centrifuged at 10,000 r/min for 15 min.

Finally, supernatants were filtered with 0.45 μm membrane filters, and high-performance liquid chromatography-fluorimetric detection (HPLC-FLD) was used to measure the concentration of NE. The instrument parameters used for HPLC-FLD were as follows: column, Agilent Zorbax SB-C_{18} column (Agilent, US); mobile phase, methanol-buffer (buffer: 0.07 mol NaH_2PO_4, 10 mmol sodium octanesulfonate, pH 3.5). The gradient procedure of the mobile phase was as follows: at 0 min, 10% methanol and 90% buffer; at 5 min, 10% methanol and 90% buffer; at 30 min, 60% methanol and 40% buffer (1.0 ml/min of flow rate, 20 μl of injection volume, 35.0°C of column temperature, 280 nm of fluorescence excitation wavelength, and 315 nm of emission wavelength). Under these conditions, various substances in plasma were completely separated so that no interference to determination of the targets is experienced.

After being exposed to aircraft noise for 65 d, four rats were randomly selected from the CG and EG-II groups (two per group). We then examined the neuronal and synaptic morphologies of the temporal lobe by transmission electron microscopy (TEM). Rats were anesthetized by administration of an overdose of sodium pentobarbital and then perfused with glutaraldehyde transcardially. The temporal lobe was localized by digital brain stereotaxic instrument (ZH-LanXing B/S, Huaibei, China) with a soft-type cranial drill. After perfusion for about 1 h, we decapitated rats and stripped the whole brain rapidly fixed in glutaraldehyde. After fixed for 24 h, the temporal lobes were removed, cut into thin slices, and further fixed in glutaraldehyde for 3 d. Based on this, slices were washed using PBS, fixed using 1% osmium tetroxide, stained using 2% aqueous solution of uranyl acetate, dehydrated using different concentrations of alcohol and acetone gradient, penetrated and embedded using embedding medium, aggregated in oven, and finally cut into ultra-thin slices stained using 4% uranyl acetate and citrate. Cell structure in these ultra-thin slices was observed by TEM (Philips Tecnai 10, The Netherlands).

3. Results

All data from OFT and HPLC-FLD are expressed as mean ± standard error of the mean (SEM). Differences between means for unpaired samples were tested by one-way analysis of variance (ANOVA) using SPSS Version 16 software. The criterion for significance was $P < 0.05$.

3.1. Open field test

Line crossing number and center area duration were obtained by three individuals working independently, and their mean values were adopted as the final results of OFT.

The line crossing number in OFT is shown in **Figure 1a**, with no significant difference between CG and EG-I over the duration of the experiment. Nevertheless, the line crossing number of EG-II after 8 d of airport noise exposure was significantly less than that of CG ($P < 0.05$). From **Figure 1a**, two conclusions can be drawn: long-term exposure to aircraft noise below L_{WECPN} of 75 dB has no significant impact on the line crossing number of rats, while L_{WECPN} of aircraft noise reaching 80 dB is likely to have an impact on line crossing number in rats.

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The result of center area duration in OFT showed that the center area durations of CG and EG-I in OFT are almost unchanged, but the center area duration of EG-II is significantly longer than that of CG ($P < 0.05$) after the 8 d of noise exposure (**Figure 1b**). On other days, center area duration among the three groups showed no significant difference ($P > 0.05$).

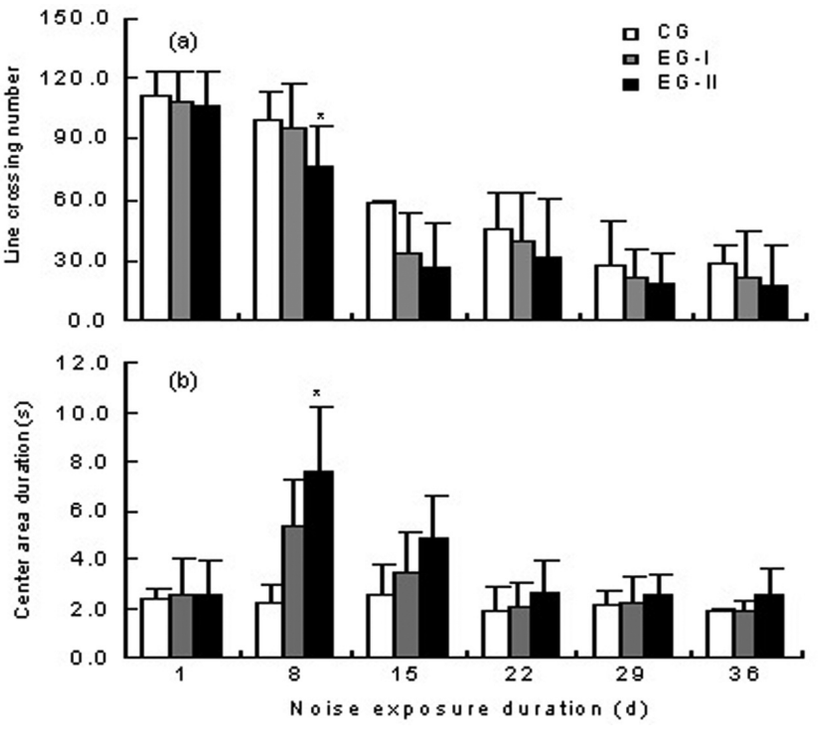

Figure 1. Line crossing number (a) and center area duration (b) in OFT.

There were ten rats in each of EG-I and EG-II and five rats in CG. Data are expressed as mean ± SEM. *$P < 0.05$, compared with the CG (ANOVA).

3.2. Levels of plasma NE

Figure 2 shows the relationship between the time rats were exposed to different intensities of aircraft noise and the average concentration of plasma NE measured. We found that there was no significant difference in NE levels between EG-I and CG over the period of the noise exposure.

Data are expressed as mean ± SEM. *$P < 0.05$, compared with the CG (ANOVA).

Nevertheless, on the 29th day of noise exposure, the levels of plasma NE between CG and EG-II showed significant difference ($P < 0.05$). By analyzing these results, we found that aircraft noise below L_{WECPN} of 75 dB has no significant impact on the plasma NE of rats. Besides, L_{WECPN} of aircraft noise reaching 80 dB is likely to have a negative effect on NE levels under long-term exposure. Therefore, we have known that the intensity of aircraft noise and the duration of aircraft noise exposure are the controlling factors to the level of NE in plasma of rats.

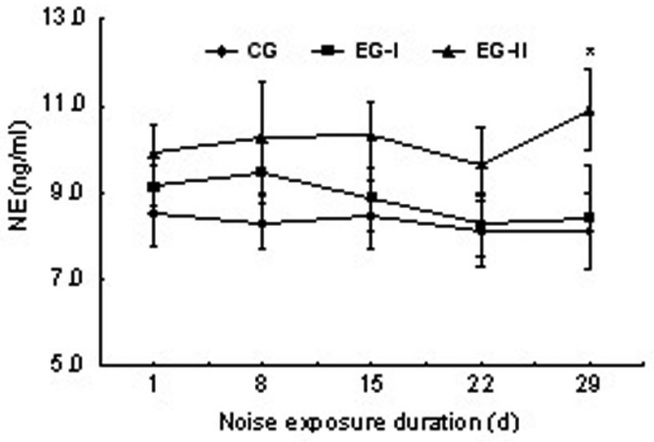

Figure 2. Relationship between NE concentration and noise exposure duration.

3.3. Temporal lobe cell morphology

We observed the neuronal and synaptic morphologies of the temporal lobes from TEM and the representative pictures are shown in **Figure 3**. In our experiments, neuronal and synaptic damages were observed in the temporal lobe of EG-II rats, but no damage was seen in the CG.

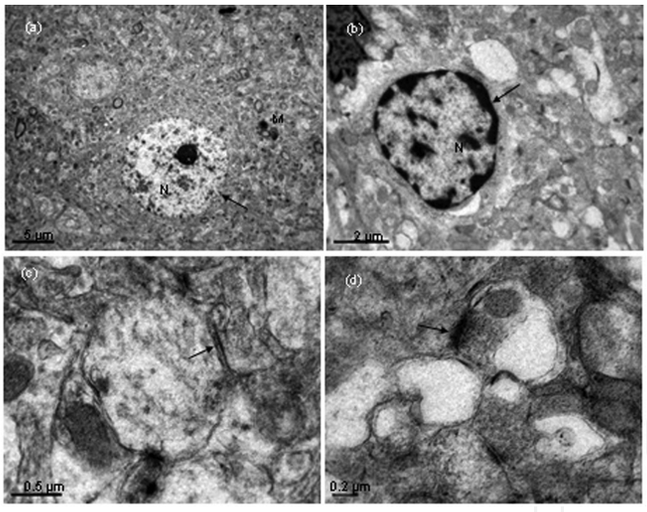

Figure 3. Neuronal and synaptic morphologies of the temporal lobe of rats.

(a) The nuclei (N) in the neurons of the temporal lobe of the CG were oval and their membrane structure was clear, yet mitochondria (M), rough endoplasmic reticula, and other organelles could be seen in cytoplasm, and their distributions were uniform and morphologically normal. (b) The nuclei (N) of neurons in the temporal lobe of EG-II were irregular-shaped, the nuclear membrane was deep-stained and its structure vague, chromatin accumulated along the edge, and cytoplasm was condensed. (c) The synaptic cleft of the temporal lobe area of the CG was clear, and mitochondria and synaptic vesicles.

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4. Discussion

4.1. Open field test

First line indent crossing is defined as that rats breach the lattices at the bottom of the open field and the number of line crossing reflects the animals' horizontal mobility, exploration, and anxiety [13]. First and foremost, previous studies have shown that motor behaviors of rats in OFT increased after 1 h of acute stress by white noise of 95 dB [4]. However, in our test, rats of EG-I and EG-II showed no significant difference in line crossing compared with CG after 1 d of noise exposure, which means acute effects of noise exposure were not evident in our experiment. It may be that noise intensity below L_{WECPN} of 80 dB is "moderate" to rats. In addition, after suffering airport noise exposure for 8 d, rats of EG-II showed that the line crossing number decreased, which means horizontal mobility and exploration ability of the rat decreased and some anxiety appeared [13]. However, Pan *et al*. found that two weeks' noise stress (2 h/d, 85 dB) increased square crossing and vertical movement, which is in contrast to our results [14]. There are a number of possible explanations. First, the intensity of noise is an important factor. Second, the duration of daily noise exposure cannot be ignored because, in our test aircraft, noise was exposed throughout the day. Third, the neurobehavioral effect of continuous white noise is different from that of intermittent aircraft noise [15]. Last but not least, from **Figure 1a**, we also know that line crossing of rats was not significantly different between CG and EG-II, except on the 8th day. The reason is that the behaviors of rats manifest itself differently depending on the duration of stress [16]. Conrad *et al*. have also shown that behaviors of rats turned from an excited state to inhibitory state under prolonged stress [17]. Another reason may be that the mechanisms of resistance and/or adaptability are generated after longer term noise stress.

In our study, center area duration is defined as latency of the rats before leaving the center area, which is measured by anxiety-like behavior. As is known, high center area duration indicates high anxiety levels [12]. Thus, the emotions of rats in EG-II altered to anxiety after 8 d of noise exposure. The results of center area duration on other days suggest that the effect of aircraft noise on anxiety is not permanent. This conclusion is also in line with the line crossing results.

4.2. Levels of plasma NE

Epinephrine, NE, and cortisol are stress hormones that are used as indicators of body stress upon noise exposure [6, 18, 19]. NE plays an important role as a stress hormone in conducting and adapting to stress [20, 21]. Therefore, plasma NE level of EG-II increased after long-term noise exposure (29 d) in our experiment, possibly due to the cumulative effect of high-intensity noise. Unfortunately, we do not know the plasma NE level of rats after 29 d of noise exposure, because it is difficult to collect blood from more fierce rats.

Plasma NE is primarily secreted by the sympathetic nerve endings of the heart, blood vessels, and adrenal medulla, and is controlled by the sympathetic nervous system [22]. Plasma NE levels reflect the excitability of the peripheral sympathetic system [23, 24]. Due to aircraft noise

exposure in our experiments, the levels of NE in rat plasma increased, indicating that aircraft noise stimulates sympathetic excitement of the adrenal medulla system.

Several studies have shown that trait anxiety is significantly associated with increased NE concentration in blood plasma. Individuals with higher plasma NE concentrations also have more severe anxiety symptoms and the concentrations of plasma NE of patients with anxiety disorders are drastically higher than those of healthy individuals [25]. We conclude that high-intensity aircraft noise exposure may similarly induce anxiety symptoms in rats. Further, patients with hypertension have also been found to have higher plasma NE concentrations [26]. In addition, epidemiological investigations have pointed out that the incidences of heart disease and hypertension are directly related to aircraft noise exposure [1, 27]. Our results partly provide pathological evidence supporting this epidemiological research.

4.3. Temporal lobe cell morphology

Previous studies have shown that necrosis and apoptosis of neurons occur when the body is subjected to physical, chemical, or severe pathological stimulation [28]. For this reason, we consider that the long-term noise stress in rats resulted in the lesions in temporal lobe neurons and synapses of EG-II.

First line indent lobe areas are closely related to perception and memory [29]. Therefore, when neurons of the temporal lobe are damaged, a variety of mental disorders are likely to occur, such as cognitive decline, memory reduction, or subjective emotional instability [30]. When synaptic morphology changes, the related functions of the brain and CNS change accordingly, further leading to changes in behavior [31]. The results of our experiments are consistent with changes in rat behavior due to long-term exposure of aircraft noise.

5. Conclusions

In conclusion, exposing rats to long-term aircraft noise affects their behavior, specifically in the form of inhibiting mobility and increasing anxiety. Our data indicate that scrum NE levels of rats increase as a result of aircraft noise exposure. Furthermore, our findings indicate that aircraft noise exposure leads to damage of neuronal and synaptic structures of the temporal lobe in rats. Nevertheless, additional studies are necessary to further investigate the mechanisms involved.

Of course, there are some differences in the hearing sensitivity to different sound frequencies and circadian rhythms between rats and humans, which can bring variances in physiological effects under the same noise exposure. Therefore, it should be further confirmed if these study results are applied into humans.

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