

Traffic Noise Exposure Increases Gastric Pepsin Secretion in Rat

Azam Moslehi¹, Fatemeh Nabavizadeh², Mansoor Keshavarz², Nematollah Rouhbakhsh³,
Masoud Sotudeh⁴, Ehsan Salimi⁵, and Amir Barzegar Behrooz²

¹ Department of Physiology & Pharmacology, School of Medicine, Qom University of Medical Sciences, Qom, Iran

² Department of Physiology, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

³ Department of Audiology, Tehran University of Medical Sciences, Tehran, Iran

⁴ Department of Pathology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

⁵ Department of Pharmacy, Tehran University of Medical Sciences, Tehran, Iran

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Abstract- Noise is considered as one of the most severe sources of environmental and workplace constraints. Many noise effects are well known on immune function, hormonal levels, cardiovascular and respiratory systems. In this study, our aim is to evaluate the effects of traffic noise exposure on basal and stimulated gastric pepsin secretion. 48 male rats were exposed to traffic noise (86 dB) for a short term of (8h/day for 1 day) and a long term of (8h/day for 7, 14, 21 and 28 days) as well as a control group. The gastric contents were collected by the wash-out technique. Pepsin secretion was measured by employing the Anson method. Histological studies were carried out on the epithelial layer. The corticosteroid hormone was measured in the serum for the stress augmentation. The present finding indicated no changes in pepsin secretion content in the short term, but in the 14 and 21 days traffic noise exposure, basal gastric pepsin secretion increased markedly compared to the control group. Histological results showed that the number of oxyntic glands and cell nuclei decreased in comparison with the control group while the thickness of the epithelial layer increases. In addition, the corticosterone levels increase in all groups in comparison with the control. It seems that the increase of gastric pepsin secretion is due to the description and translation processes in the peptic cells and needs enough time for completion.

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Introduction

Noise (unwanted sound) is considered as one of the most severe sources of environmental pollution at workplaces. Many noise effects are well known on hearing loss, disturbed sleep, immune function, hormonal levels, and mental illness, respiratory and cardiovascular systems (1-7). Traffic noise, as one of the noise types, is a widespread and an increasingly distinguished feature of the urban environments, especially in developing countries.

Interaction between noise and gastrointestinal function would seem likely, given the abundant neural connections between the human auditory system, the autonomic nervous system and the gastrointestinal tract (8,9). It is shown that the noise effects on the plasma levels of motilin, somatostatin (SS), Substance P (Sp),

gastrin, endothelin (ET) and Nitric oxide (NO) in the gastrointestinal tract (10,1). It has been demonstrated that low-frequency noise destroys intestinal microvilli and leads to duodenal lesions. In addition, noise especially the explosive noise increases gastric acid secretion and gastrointestinal transit (11,12). In the previous study, we reported that the traffic noise increased basal and stimulated gastric acid secretion (13).

Pepsinogens, precursors of pepsins, are aspartic proteinases which are mainly secreted by peptic cells. It has been demonstrated that pepsinogen secretion is affected by many factors, such as neural, hormonal and environmental factors. Previously, the role of pepsin is well known in the pathogenesis of peptic ulcer. Clinical studies have also been reported the diagnostic value of human pepsinogens in various gastroduodenal diseases,

Corresponding Author: F. Nabavizadeh

Department of Physiology, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
Tel: +98 913 3410451, Fax: +98 21 88989485, E-mail address: nabavizadeh@tums.ac.ir

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such as; peptic ulcer, gastritis, duodenal ulcer and gastric cancer (3-8).

Since, no academic study was found regarding traffic noise effects on gastric pepsin secretion; therefore, this study was designed to aim the evaluation of traffic noise effects on basal and stimulated pepsin secretion.

Materials and Methods

Wistar male rats, weighing 200–250 g were used in this study. Animals were kept at a controlled room temperature and 12: 12, light/ dark cycle with free access to standard laboratory chow and water. They were treated according to the ethical committee of Tehran University of Medical Sciences (TUMS). The animals were randomly divided into 6 equal groups (N=6). The control group was not exposed to traffic noise, while the animals in the other five groups were subjected to 8h (from 23:30 to 07:30) noise exposure for 1, 7, 14, 21 and 28 days.

Traffic sound of various places in different squares of the Tehran city was recorded to provide the traffic noise. The sound level meter (SLM) instrument was used to adjust the level of the output intensity and set at an intensity of 86 dB (14). The noise room was an acoustic one, equipped with two speakers. The animal cages were placed at a distance of 30 centimeters from the speakers. A dosimeter was used to keep the sound level constant and to be sure of output level. Within 30 min after the end of noise exposure, the animals were anesthetized by an IP injection of 50 mg/kg sodium thiopental (15), and a tracheotomy was performed after that. To prevent the gastric reflux into the oral cavity, the cervical esophagus was ligated after tracheotomy. Laparotomy was done, and a polyethylene canola with 2.5-millimeter external diameter and 10-centimeter

length was placed into the stomach via the duodenal transverse incision. Residual gastric secretion was removed by lavage several times with 1-2 milliliters of normal saline at 37°C and then allowed 30 min for recovery (16). For pepsin basal secretion measurement, 1 milliliter normal saline was introduced into the stomach. After 15 min, secretion was collected by the washout technique and measured by Anson method. In this method, pepsin acted on the substrate (hemoglobin) and the final product of the reaction was measured by UV spectrophotometer (JENWAY 6105 UV/Vis, UK, $\lambda = 280$ nm) (18,17). Then, the pentagastrin (25 $\mu\text{g}/\text{kg}$, IP) was used to stimulate gastric pepsin secretion (8).

Statistical analysis

Data were expressed as Mean \pm SE. Statistical analysis was performed by paired and unpaired t-test for the two groups, and more than three groups were compared with one-way ANOVA and followed by Turkey's post hoc test. $P < 0.05$ was considered to be statistically significant.

Results

The effects of traffic noise on basal and stimulated pepsin secretion were studied in two different conditions, short term (8h) and long term (8h/day for 7, 14, 21 and 28 days) noise exposure.

Effect of short term traffic noise exposure on basal pepsin secretion

Basal pepsin secretion did not change in response to short term traffic noise exposure in comparison with control group (0.45 ± 0.18 vs. $0.49 \pm 0.1 \mu\text{g}/\text{ml}/15\text{min}$) (Figure 1).

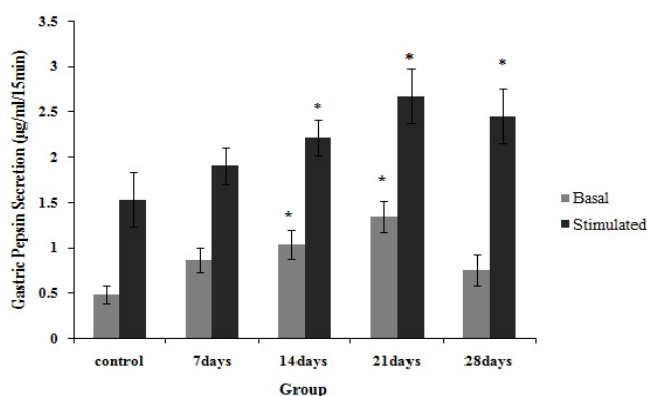


Figure 1. Comparison of the pepsin secretion between the control and short-term noise exposure groups (n = 8 in each group), Data expressed as Mean \pm SE,

Effect of short-term traffic noise on stimulated pepsin secretion

In short term group, pentagastrin-stimulated pepsin secretion did not change in comparison to the control group (Figure 1).

Effects of long-term traffic noise on basal pepsin secretion

Basal pepsin secretion after 14 and 21 days

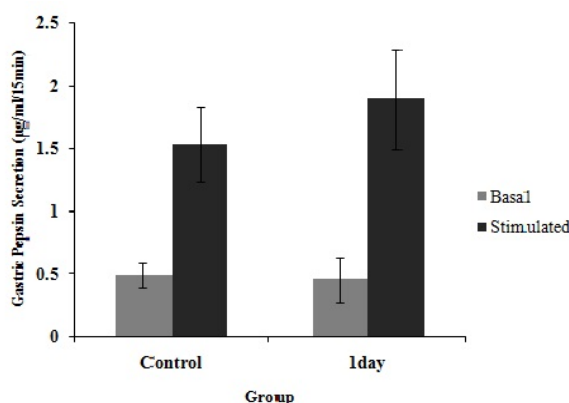


Figure 2. Comparison of the basal and stimulated pepsin secretion between the control and long-term noise exposure groups (n = 8 in each group), Data expressed as Mean ± SE

Effects of long-term traffic noise exposure on stimulated pepsin secretion

In long-term traffic noise exposure, Pentagastrin-stimulated pepsin secretion did not change in all groups and was similar to control group (Figure 2).

Comparison between basal and stimulated pepsin secretion in all groups

Our findings showed that pentagastrin- stimulated pepsin secretion in the control group was significantly more than the basal state (1.54 ± 0.4 vs. 0.49 ± 0.1 mg/ml/15min, $P < 0.01$) (Figure 2). Also, in all traffic noise exposure groups, our results showed that pentagastrin-stimulated pepsin secretions were significantly more than the basal state [(1day 1.9 ± 0.5 vs. 0.45 ± 0.18 µg/ml/15min)] (Figure 1), 7days (2.22 ± 0.3 vs. 0.87 ± 0.14 µg/ml/15min), 14 days (1.91 ± 0.3 vs. 1.04 ± 0.16 µg/ml/15min), 21 days (2.68 ± 0.4 vs. 1.35 ± 0.17 µg/ml/15min) and 28 days (2.46 ± 0.5 vs. 0.76 ± 0.17 µg/ml/15min) ($P < 0.03$) (table1).

(1.04 ± 0.16 and 1.35 ± 0.17 µg/ml/15min, respectively), showed a significant increase in comparison with the control group (0.49 ± 0.1 µg/ml/15min) ($P < 0.05$). Longer exposure (28 days) decreased basal pepsin secretion there was no significant difference between 7 and 28 days groups (0.87 ± 0.14 and 0.76 ± 0.17 µg/ml/15min, respectively) exposure with the control groups (Figure 2).

Table 1. Comparison of the basal and stimulated pepsin secretion between the control and traffic noise exposure groups (n = 8 in each group), Data expressed as Mean ± SE

Groups	Basal	stimulated
control	0.49 ± 0.1	$1.54 \pm 0.4^*$
1 day	0.45 ± 0.18	$1.9 \pm 0.5^*$
7days	0.87 ± 0.14	$2.22 \pm 0.3^*$
14days	1.04 ± 0.16	$1.91 \pm 0.3^*$
21days	1.35 ± 0.17	$2.68 \pm 0.4^*$
28days	2.46 ± 0.5	$0.76 \pm 0.17^*$

*Significant differences between the basal and stimulated traffic noise exposure groups ($P < 0.05$)

Effects of traffic noise exposure on the histology of stomach

Effects of traffic noise exposure on mucosal layer thickness were studied. The mucosal layer thickness was increased significantly in response to long-term traffic noise exposure ($P < 0.001$).

On the other hand, complementary observations showed that in traffic noise groups, erosive gastritis was seen in all groups (Figure 3).

Traffic noise Pepsin secretion

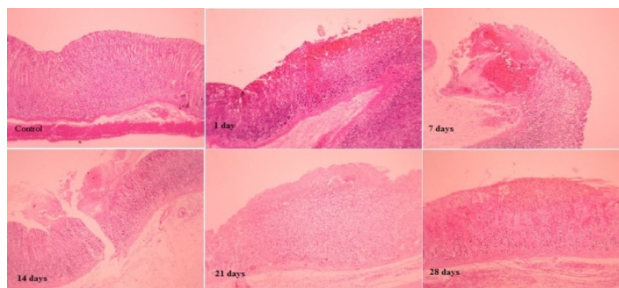


Figure 3. Gross specimens of mucosal layer: **A** the control group, **B**, 1 day group, **C**, 7 days group, **D**, 14 days group, **E**, 21 days group, **F**, 28 days group

Effects of traffic noise exposure on the levels of corticosterone

This study showed that serum corticosterone

increased in all groups of traffic noise exposure in comparison to control group (Figure 4).

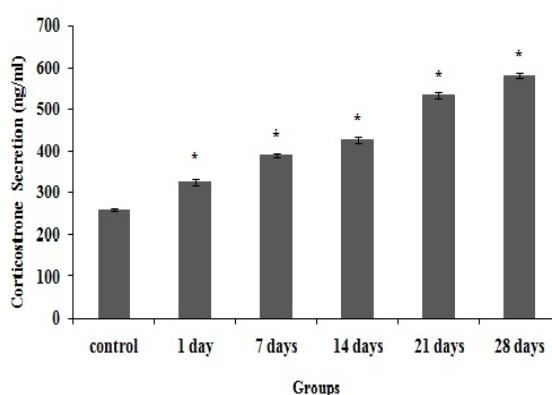


Figure 4. Comparison of corticosterone secretion between the control and noise exposure groups (n = 8 in each group), Data expressed as Mean \pm SE

Table 2. Comparison of mucosal layer thickness between the control and noise exposure groups (n=8). Data expressed as Mean \pm SE

Groups	Mucosal layer thickness (μ m)
control	9.5 \pm 0.44
1 day	10.6 \pm 0.24
7days	12.4 \pm 0.4*
14days	11.8 \pm 0.3*
21days	11.8 \pm 0.2*
28days	11.4 \pm 0.5*

*Significant differences with the control ($P < 0.001$).

Discussion

In the present study, traffic noise did not change pepsin secretion in basal and stimulated states in 1 day groups, but increased between 14 and 21 days groups in the basal states. Our histological results showed that mucosal layer thickness increased in traffic noise

exposure. Erosive gastritis was seen in all groups.

Pepsinogen is a proteolytic enzyme, and it needs description and translation processes of DNA in the cytoplasm and Golgi apparatus for synthesizing and secreting. As much as, these processes need enough time. Therefore it is expected that traffic noise has not been affected by pepsin secretion in short term groups in both states.

According to our result, traffic noise exposure increased gastric pepsin secretion in 14 and 21 days groups. This effect may be caused via activation of the vague nerve in the medulla. It has been shown that noise exposure activates hypothalamic-pituitary-adrenal (HPA) axis (6,19,20) and it is thought that hypothalamus has an important role in noise pathophysiological effects. Therefore, it seems that, in 14 and 21 days groups, there is enough time to complete description and translation processes.

On the other hand, our results showed a significant

increase of epithelial layer thickness that confirms our hypothesis. Although more investigations are needed to elucidate this relation.

In conclusion, short-term traffic noise did not gastric pepsin secretion; however, long-term traffic noise can induce adaptation. Complex structural and functional mechanisms may be involved in this phenomenon which needs further elucidation

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