Traffic Noise Exposure Increases Gastric Acid Secretion in Rat

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Abstract- Noise is considered as one of the most severe sources of environmental and work place constraints. Noise effects on immune function, hormonal levels, cardiovascular and respiratory systems are well known. The aim of the study is to evaluate the effects of traffic noise on basal and stimulated gastric acid secretion. 48 healthy rats were divided into five traffic noise exposures (1, 7, 14, 21, 28 days) and a control groups. Pentagastrin was used IP for stimulation of gastric acid secretion. The gastric contents were collected by the wash-out technique and then titrated. Histological studies were performed on gastric epithelial layer. In the 1, 7, 14 and 21 days traffic noise exposure, basal and pentagastrin-stimulated gastric acid secretion increased compared to the control group (P<0.001), but a significant decrease was seen in hyperacidity in 28th days, in the both basal and stimulated states(P<0.05). Histological study showed that mucosal layer thickness of stomach increased, while the number of oxyntic glands and cell nuclei decreased. It seems that 1,7,14 and 21 days traffic noise increase gastric acid secretion, while 28 days traffic noise can induce adaptation.

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Key words: Noise, transportation; gastric acid; rats, inbred strains; pentagastrin

Introduction

Our ears are constantly at work, ready to receive information from environment. Noise pollution is considered as one of the most severe sources of environmental and work place constraints and pollution and now is recognized as a serious health hazard in our modern societies. There are many considerations regarding noise effects on immune function, hormonal levels, mental illness, sleep disturbances, hypertension, cardiovascular and respiratory systems (1-6). Traffic noise is an increasingly prominent feature of the noise in urbanized environments.

The central nervous system role in controlling the gastrointestinal tract has been studied in the last decade (7). An 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.

In addition, the noise, particularly explosive noise effects on gastric secretion and ulcer formation have been studied and have revealed increased gastric acid secretion (8), edema, erosion of gastric mucosal layer and ulcer formation (9). Since, academic study regarding traffic noise effects on gastric acid secretion was not found; this study was designed to investigate the effects of short term and long term traffic noise on basal and stimulated gastric acid secretion in rats.

Materials and Methods

48 Wistar male rats weighing 200–250 g were used. Animals were housed in groups of five rats per cage and maintained in a temperature controlled room with a 12/12 light/dark cycle (lights on at 07:00 am). The animals had free access to food and water, except for the 24 hours before the experiments when they were deprived of food had free access to water up to the beginning of the experiments (10). The procedure was in accordance with the guidelines for the care and use of laboratory animal of Tehran University of Medical Sciences. The animals were divided into six groups with 8 animals in

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each group. The control group was not exposed to 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.

To provide the traffic noise, traffic sound of various places in different squares of the city was recorded. Sound level meter (SLM) instrument was used to adjust the level of the output intensity and was set at 86 dB intensity (11). The noise room was an acoustic one, with two speakers. The animal cages were put 30 centimeter away from the speakers. To keep the sound level constant and to be sure of output level a dosimeter was used. Within 30 min after the end of noise exposure, the animals were anesthetized by an IP injection of 50 mg/kg sodium thiopental (12), and then tracheotomy was performed. To prevent the gastric reflux into oral cavity, after tracheotomy cervical esophagus was ligated. Laparatomy was done and a polyethylene canola with 2.5 millimeter external diameter and 10 centimeter length was placed into stomach via duodenal transverse incision. Residual gastric secretion was removed by lavage several times with 1-2 milliliters of normal saline at 37ºC, and then was allowed 30 min for recovery (13). For basal secretion measurement, 1 milliliter normal saline was introduced into the stomach. After 15 min secreted acid was collected by the washout technique and titrated by 0.001 normal, NaOH (14). Then pentagastrin (25µg/kg, IP) was used to stimulate gastric acid secretion (15) and 15 min later secreted acid was collected. Finally, the stomach of all the animals were removed and fixed in the formalin 10% for histological study.

Statistical analysis

Data showed as Mean ± SE. Differences between the two groups were compared using the paired and unpaired t-test; and more than three groups were compared with one way ANOVA and followed by Tukey's post hoc test. $P<0.05$ was considered to be statistically significant.

Also, statistical analysis of our finding histological study, was done by ANOVA and Tukey's post hoc test.

Results

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

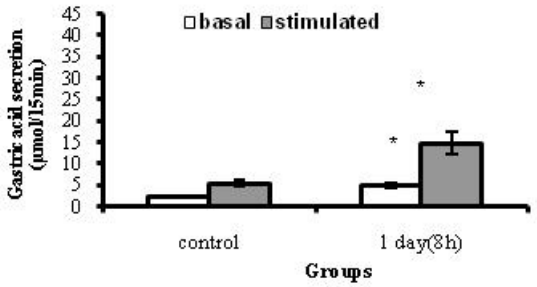

Figure 1. Comparison of the acid secretion between the control and short term (8h) noise exposure groups (n = 8 in each group), Data expressed as Mean + SE, * $P< 0.001$.

Effect of short term traffic noise exposure on basal acid secretion

Basal acid secretion was increased significantly in response to short term traffic noise exposure in comparison with control group (4.99 ± 0.43 vs 2.28 ± 0.26µmol/15min, $P< 0.001$) (Figure 1).

Effect of short term traffic noise on stimulated acid secretion

In short term group, pentagastrin-stimulated acid secretion was increased in comparison to the control group (14.93 ± 2.62 vs 5.55 ± 0.87 µmol/15min, $P<0.001$), (Figure 1).

Effects of long term traffic noise on basal acid secretion

Basal acid secretion after 7, 14 and 21 days (7.01 ± 0.33, 13.82 ± 0.7, 14.55 ± 1.11µmol/15min, respectively), showed a significant increase in comparison with the control group (2.28±0.26 µmol/15min) ($P<0.001$). Longer exposure (28 days) decreased basal acid secretion (4.12 ± 0.73 µmol/15min). There was no significant difference between 28 days exposure with the control groups (Figure 2).

Effects of long term traffic noise exposure on stimulated acid secretion

Pentagastrin-stimulated acid secretion after 7, 14 and 21 days of noise exposure showed a significant increase in comparison to the control group (19.25±2.33, 29.4 ± 5.09, 35.47 ± 5.15, 5.55±0.87µmol/15min, respectively, $P<0.001$). Longer traffic noise exposure (28 days) caused much less increase in hyperacidity after stimulated acid secretion (15.15 ± 1.55µ mol/15min) than above groups (Figure 2).

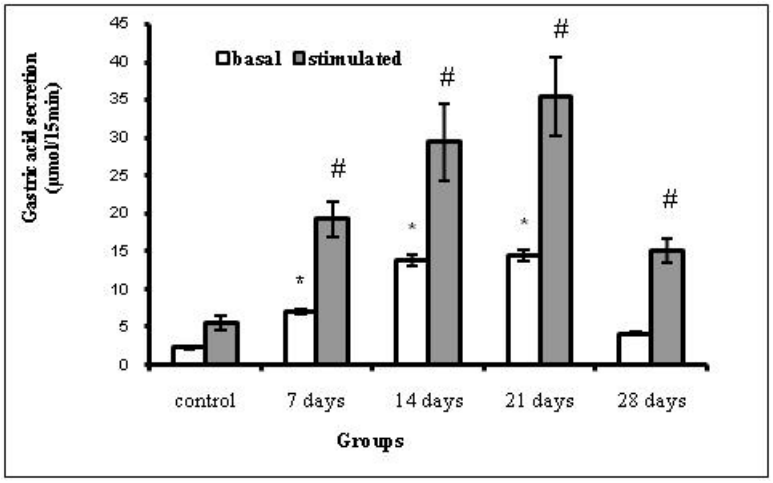

Figure 2. Comparison of the basal and stimulated acid secretion between the control and long term noise exposure groups (n = 8 in each group), Data expressed as Mean + SE

***** Basal acid secretion between the control and long term noise exposure groups, $P<0.001$

Stimulated acid secretion between the control and long term noise exposure groups, $P<0.001$

Comparison between basal and stimulated acid secretion in all groups

Our findings showed that pentagastrin- stimulated acid secretion in the control group was significantly more than the basal state (5.55±0.87, 2.28±0.26µ mol/ 15min, $P<0.01$) (Figure 2). Also, in all traffic noise exposure groups, our results showed that pentagastrin-stimulated acid secretions were significantly more than the basal state [(1day 14.93 ± 2.62, 4.99 ± 0.43 µmol/15min)] (Figure 1), 7days (19.25 ± 2.33, 7.01 ± 0.33 µmol/15min), 14 days (29.4±5.09, 13.82±0.70 µmol/15min), 21 days (35.47 ± 5.15, 14.55 ± 1.11 µmol/15min) and 28 days (15.15 ± 1.55, 4.12 ± 0.73 µmol/15min) (Figure 2).

Effects of traffic noise exposure on the histology of stomach

Effects of traffic noise exposure on mucosal layer thickness, number of oxyntic glands and number of epithelial cell nuclei were studied. Mucosal layer thickness was increased significantly in response to long term traffic noise exposure ($P< 0.01$). However, the number of oxyntic glands was significantly decreased in response to long term traffic noise exposure ($P<0.01$). Also, the number of epithelial cell nuclei in the long term groups was significantly decreased comparison with the control group ($P<0.001$) (Table 1).

On the other hand, complementary observations showed that in traffic noise groups, parietal cells look larger than those of the control.

Table 1. Comparison of mucosal layer thickness, number of glands, and number of epithelial cell nuclei between the control and noise exposure groups (n=8). Data expressed as Mean ± SE

Groups	Mucosal layer thickness (µm)	Number of glands / 0.25cm^2	Number of epithelial cell nuclei / 0.25cm^2
control	9.5 ± 0.44	2.83 ± 0.27	30.2 ± 1.5
1 day	10.6 ± 0.24	2.29 ± 0.12	29.7 ± 0.7
7days	12.4 ±0.4*	1.97 ± 0.02*	19.7 ± 0.7*
14days	11.8 ± 0.3*	2.1 ± 0.14*	20.7 ± 1.15*
21days	11.8 ± 0.2*	2 ±0.03*	21.55 ± 0.7*
28days	11.4 ± 0.5*	2 ± 0.04*	19.15 ± 0.51*

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

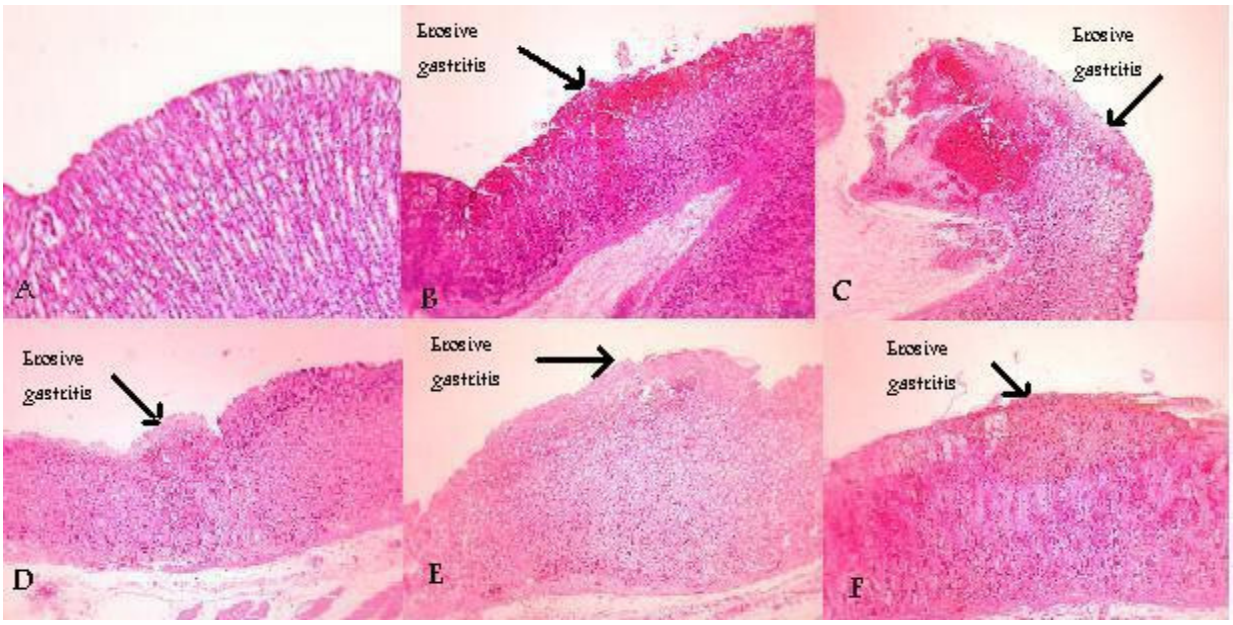

Figure 3. Gross specimens of mucosal layer: **A**, the control group, **B**: 1 day group, **C**: 7 days group, **D**: 14 days group, **E**: 21 days group, **F**: 28 days group

In addition, erosive gastritis was seen in all groups and the parietal cell cytoplasm showed prominent granularity in1, 7 and 14 days groups (Figure 3).

Discussion

In the present study, both short and long term traffic noise exposure increased basal and stimulated gastric acid secretion, except for 28th day. Traffic noise exposure increased basal gastric acid secretion up to day 21 with a mild slope, but a significant decrease was seen in hyperacidity on 28th day. Although in this group (28 day) stimulated acid secretion was significantly higher than control, but a significant decrease was seen on 28th day by comparison with 21 day group. Rate of acid secretion in response to pentagastrin in all groups were higher than basal state.

Our histological results showed that mucosal layer thickness increased in traffic noise exposure and parietal cells were larger than control group. Erosive gastritis was seen in all groups.

We found that traffic noise exposure increases gastric acid secretion. This effect may be caused via activation of the vagus nerve in medulla. It has been shown that noise exposure activates hypothalamic-pituitary-adrenal (HPA) axis (6, 16, and 17). Therefore, it is thought that hypothalamus has an important role in noise pathophysiological effects and probably, is activated via medial geniculate nuclei (MGN) in the thalamus and other areas in the brain. Shiraishi T *et al.* have shown that gastric acid secretion (GAS)-related neurons are located in the lateral hypothalamic areas (LHA), and the preventricular nucleus (PVN) was found to affect gastric acid secretion. Their effects are exerted on oxynitic cells via medulla oblongata and the vagus (7, 18), but more investigations are needed to elucidate this relation.

Also, in this experiment, traffic noise increased pentagastrin-stimulated acid secretion. Several researches have shown that environmental noise affects hormonal systems (2). In 2006 it was shown that after stimulation by explosive noise, concentration of motilin, substance P (Sp) and somatostatin were increased (19). Liu GS *et al.* reported that concentrations of serum gastrin and endothelin were apparently high after explosive noise exposure (9).

Although the pervious studies have assessed explosive but not traffic noise, it could be assumed that gastrin has been increased in our study too. Therefore, increment of gastric acid secretion after traffic noise exposure result from the effect of gastrin on parietal cells and vagus nuclei in medulla respectively. The use of pentagastrin, in stimulated state, produces further increases in gastric acid secretion compared to the basal state.

To our knowledge, gastrin has a trophic effect on gastric epithelial layer. According to our histological

findings probably gastrin-induced parietal cell hypertrophy resulted in the increase mucosal layer thickness and increase gastric acid secretion.

Surprisingly however, on the 28th days, hyperacidity decreased in both basal and stimulated states. It seems that excessive acid secretion results in cell exhaustion and fatigue of gastric acid secretion processes and/or change of parietal cell function as down regulation of gastrin or acetylcholine receptors, and therefore decreases gastric acid secretion in long term, although more studies should be conducted to evaluate this phenomenon. Also, its probable, increase of somatostatin secretion decreased acid secretion after 28 days. Auditory damage may be another reason for the decrease in gastric acid secretion. Bohne BA *et al.*. reported that exposure to an octave band of noise with a sound pressure level of 80 or 86 dB SPL resulted in cell loss (20). It was also shown that exposure to a traumatic sound inducing up to 80 dB causes hearing loss, and damage outer and inner hair cells (21). Therefore, it seems that long term traffic noise exposure (28 days) causes damage to the cells in auditory system and so decreases stimulatory impulses to CNS, and decrease in vagus stimulation decrease gastric acid secretion on the 28th day.

In conclusion, short term traffic noise increases gastric acid 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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