Traffic Noise Emissions and Myocardial Infarction Risk

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Abstract

We examined the possible association between road traffic noise in residential areas and myocardial infarction (MI) incidence. We conducted an ecological study among 25-64-year-old men in the general population of Kaunas city. The study comprised all first time MI cases among stable residents of Kaunas treated in hospitals in 1999-2001 (518). We measured traffic-related noise levels at the 117 electoral districts and linked these levels to residential addresses using Geographical Information System (GIS) techniques. In daytime period (10-12 hr, 10 min.) traffic-related noise emission fluctuated between 58 dB(A) to 82 dB(A) and about 18% of citizens were exposed to noise level exceeding 65 dB(A) in their residential district. The age-adjusted MI incidence per 1,000 increased by increasing noise exposure. In the total group of 25-64 years old men the incidence tended to increase from 2.07 in the 1st (lowest) exposure area to 2.57 in the 4th (highest) exposure area (Risk ratio (RR) 1.33; 95% confidence interval (CI) 0.76-2.32). In the subgroup of 55-64 years old men, the risk ratio increased by 92% (RR = 1.92; 95% CI 1.00-3.67). Our results indicate a relationship between traffic noise exposure and MI incidence among 55-64-year-old men.

Keywords: environmental noise, myocardial infarction, incidence, ecological study

Introduction

Traffic noise is one of the main environmental quality problems in economically developed countries, related to society’s increasing dissatisfaction with existing situation. In Europe about 113 million citizens (17% of the population) are exposed to ambient noise levels above Leq 65 dB(A) and about 9.7 million citizens are exposed to noise levels above 75 dB(A) [1]. Today in large cities the number of inhabitants exposed to unacceptable levels of noise is two or three times greater than national averages. Ambient noise has become an increasing concern of national authorities and the European Commission [2]. The main source of acoustic nuisances is road traffic. There is a growing amount of evidence that road traffic noise increases about 1-3 decibels a year in most cities. According to prognosis, the noise load will double in a 15-year period [3].

Excess noise has a wide range of effects on individuals, ranging from disturbance to chronic stress and damage to hearing. Therefore, the World Health Organization (WHO) has attributed noise to occupational risk factors [4]. Traffic noise causes annoyance, emotional distress, chronic stress and increases the risk of hypertension [5-7]. Long-term noise causes changes in homeostasis, which are accompanied by disorders in heart rhythm, muscle tenseness and changes in brain electrical potentials [8]. A positive association was found between noise annoyance and serum lipid levels [9], exposure to noise and fibrinogen, and plasma viscosity [10], causal risk factors of ischemic heart disease (IHD).
Exposure to noise increases physiological stress indicators - catecholamines and steroid hormones and through the neurohumoral pathway stimulates changes in the cardiovascular system, stimulates arterial hypertension, development of IHD [11, 12], and might increase myocardial infarction (MI) risk [13, 14]. The meta-analysis of occupational noise exposure studies have revealed that evidence for a relation between noise exposure and IHD is still inconclusive because of the limitation in exposure characterization and adjustment for confounders [15]. There are only a few published community studies, among them Berlin case-control study [16] and Carlphilly and Speedwell cohort studies [17].

Epidemiological studies have shown that noise-induced health effects depend on noise character and exposure time, as well as duration and other environmental factors. Individual sensitivity, age and susceptibility to noise determined noise-induced responses. Large individual differences in sensitivity to noise co-vary with expressed annoyance resulting from noise [18]. Lack of measurements of noise levels in human settlements, difficulty in exposure quantification and the small number of cases are factors of great importance why epidemiologic evidence of cardiovascular effects caused by environmental noise exposure is still limited [19]. The present population-based study was conceived with the aim of exploring the possible association between MI incidence and environmental noise exposure using geographical information system (GIS) techniques.

**Subjects and Methods**

Our study was conducted in Kaunas, the second largest city of Lithuania. With an area of 132 km² and a population of about 400,000 the city is situated in a valley and neighboring plain. The study base population comprised all 25-64-year-old men residing in 12 districts of the city. In Kaunas more than 97% of the hospitalized MI cases in the age range of 25-64 are treated in 4 cardiological departments. Specially trained staff identified the subjects for MI registration at these departments. All hospitalized patients with a first-time MI that occurred from 1999 to 2001 were eligible for the study. The association between traffic noise in the residential area and MI risk was studied using an ecological design.

The trained doctors registered all patients with a first time MI treated in hospitals. An eligible case was a person with a clinical diagnosis coded I21 of the 10th revision of International Classification of Diseases (ICD-10) on the hospital registry. Criteria for MI included: (i) certain symptoms according to case history information, (ii) specified changes in blood levels of the enzymes CK and LDH, and (iii) specified ECG-changes. Cases were identified from two sources and included at the time of disease incidence. The sources were the coronary and intensive care units at the cardiovascular departments and the hospital discharge register. In total, 518 male first-time MI cases were registered. Traffic noise exposure was assessed by measurement of the traffic noise emission level at 10 m distance from the center of the street.

To create a map of daytime (10-18 hr) noise exposure in the area under study, short-term (10 min.) single measurements of A-weighted average sound pressure level were carried out. These measurements were performed in all busy streets (continuous traffic flow during daytime) and many side streets (single-event traffic). We measured noise levels near main streets in 117 Kaunas electoral districts. We used Bruell&Kjaer Precise Noise Meter Type 2203 and Noise Level Analyzer Type 4426 to assess the mean of 10 min. noise level measurements.

![Fig. 1. Myocardial infarction (MI) cases in electoral districts ascribed to different noise exposure categories.](image)
Measurements for a given street and electoral district were then linked to all subjects living in those electoral districts. The addresses were transformed into geographical coordinates using standard GIS computer software [20] in conjunction with a Kaunas geographical address database. We used the following information layers: city streets network, electoral district borders, and population density data.

The noise levels were classified according to 4 dB(A) categories: 1st – <60 dB(A), 2nd – 60-64 dB(A), 3rd – 65-69 dB(A) and 4th – ≥70 dB(A).

Subjects were also categorized into age subgroups: 25-44, 45-54, 55-64, and we calculated age-adjusted MI incidence rates in these subgroups as well as in the total group of 25-64 years old men. We used direct standardization for estimation of age-adjusted MI incidence rates per 1,000 per year and calculated relative risks (RR) and 95% confidence intervals (CI) across noise exposure categories. We used noise level in the 1st exposure category as the reference category (low exposure). We used SPSS version 10.0 for the statistical analyses and Arc View GIS software for creating maps.

Results

The daytime road traffic noise levels ranged between 58 and 82 dB(A), and about 18% of citizens in their residential districts were exposed to noise levels exceeding 65 dB(A).

The number of identified MI cases was 518. Fig. 1 presents the number of MI cases in electoral districts’ exposed to different levels of traffic noise. Fig. 2 shows age-standardized incidence rate of MI per 1,000 per year in electoral districts with different noise exposure categories.

Table 1 shows the number of subjects and number of cases by age groups in each traffic-noise exposure category. Incident rates per 1,000 per year in different age groups in the city were as follows: in 25-44 age group it was 0.48, in 45-54 age group - 3.05, in 55-64 age group - 5.4 and in 25-64 age group - 2.18.

Table 2 presents relationships between traffic noise emission and incidence rate of MI in different age groups. Incident rate tended to increase with increasing age and noise level. The incidence rate in the 25-64 year age group ranged from 2.07 in the 1st noise exposure category to 2.57 in the 4th exposure category. The corresponding risk ratios ranged from 1.07 (95% CI 0.88-1.30) to 1.33 (95% CI 0.76-2.32). The strongest association was found in the older subgroup of 55-64 year old men with risk ratios ranging from 1.05 (95% CI 0.80-1.38) to 1.93 (95% CI 1.00-3.67). However, no trend was found in the subgroup of 45-54-year-old subjects.

Discussion

This study suggested an association between the risk of MI and noise exposure, assessed by daytime traffic noise estimates in the place of residence. The clearest results were found in a group of older man. We found no evidence of a significant association between noise exposure and MI risk in 25-44 and 45-54 year age groups, which may be due to small numbers of myocardial infarction. Only 2 additional cases in the highest exposure category would change the direction of the association. The positive relationship between traffic noise and MI incidence was only found in the 55-64-year-old men who contributed the most to the total number of cases.

We used standardized case register technique to cover all MI events in the city population. The application of
common diagnostic criteria for MI should contribute to even and high quality of diagnosis through the whole case registration period. The method of case finding in the present study and case ascertainment was comparable to that of MI registries set up in accordance with principles adopted in the WHO MONICA program. However, there were several sources of errors that might have caused misclassification of the clinical diagnosis. These errors include diagnostic errors in silent cases, coding errors or other clerical errors in data recording. However, these errors were probably not related to residential exposure, and thus they did not bias the risk ratio estimates in the noise exposure categories. In our study, an age-adjusted MI incidence rate per 1,000 per year was calculated using all hospitalized cases.

Systematic classification bias of measured noise levels in the residential districts was not likely, since residential data on street addresses were unlikely to be affected by differential reporting bias. The most important source of random error and possible bias in our study (as was the case in most studies in which exposure was based on place of residence) was the possible misclassification of individual exposure. We estimated exposure to noise based on average measurements for the entire electoral district, which might cause an underestimation of the effects of noise exposure. Another source of possible bias was the limitation in adjustment for potential confounding factors such as smoking, body mass index, occupational exposure and others, as we in this study contracted only two main factors - gender and age. The MI risk factors, which we have not adjusted for might confound the observed association.

The data presented in this study revealed increased risk ratios for the incidence of MI in 25-64-year-old men to 1.33 (95% CI 0.76-2.32). The results were not significant because of smaller numbers of men in the highest exposure category. The Berlin case-control study that examined 41-70-year-old persons showed similar results. Risk ratios for MI among men exposed to 71-80 dB(A) were found to be 1.3 (95% CI 0.9-2.0) to compare to men exposed to noise levels of 51-60 dB(A) [16].

Our data do not support the statement that there is a relationship between road traffic noise exposure and MI incidence. The association was stronger among older men. However, we cannot exclude the possibility that these findings may be attributable to other environmental and lifestyle factors.

### Table 1. Number of subjects (N) and myocardial infarction cases (NMI) by age groups exposed to different levels of traffic noise.

<table>
<thead>
<tr>
<th>Noise exposure categories</th>
<th>25-44 years</th>
<th>45-54 years</th>
<th>55-64 years</th>
<th>In total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>NMI</td>
<td>%</td>
</tr>
<tr>
<td>1st&lt;60 dB(A)</td>
<td>61314</td>
<td>39.9</td>
<td>27</td>
<td>39.1</td>
</tr>
<tr>
<td>2nd 60-64 dB(A)</td>
<td>67824</td>
<td>44.2</td>
<td>30</td>
<td>43.5</td>
</tr>
<tr>
<td>3rd 65-69 dB(A)</td>
<td>21285</td>
<td>13.9</td>
<td>12</td>
<td>17.4</td>
</tr>
<tr>
<td>4th ≥ 70 dB(A)</td>
<td>2997</td>
<td>2.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>In total</td>
<td>153420</td>
<td>100</td>
<td>69</td>
<td>100</td>
</tr>
</tbody>
</table>

### Table 2. Myocardial infarction incidence rate per 1,000 per year (I), risk ratios (RR) and 95% confidence intervals (CI) by age groups exposed to different levels of traffic noise.

<table>
<thead>
<tr>
<th>Noise level dB(A)</th>
<th>25-44 years</th>
<th>45-54 years</th>
<th>55-64 years</th>
<th>25-64 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I RR 95% CI</td>
<td>I RR 95% CI</td>
<td>I RR 95% CI</td>
<td>I RR 95% CI</td>
</tr>
<tr>
<td>&lt;60</td>
<td>0.44 1.0</td>
<td>3.02 1.0</td>
<td>5.02 1.0</td>
<td>2.07 1.0</td>
</tr>
<tr>
<td>60-64</td>
<td>0.48 1.00</td>
<td>0.58-1.74</td>
<td>3.15 1.07</td>
<td>0.76-1.50</td>
</tr>
<tr>
<td>65-69</td>
<td>0.68 1.28</td>
<td>0.65-2.53</td>
<td>3.03 1.04</td>
<td>0.68-1.59</td>
</tr>
<tr>
<td>≥70</td>
<td>0.00 0.0</td>
<td>2.15 0.75</td>
<td>0.24-2.36</td>
<td>9.67 1.93</td>
</tr>
</tbody>
</table>
References