

Recent advances in research on non-auditory effects of community noise

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SUMMARY

Non-auditory effects of noise on humans have been intensively studied in the last four decades. The International Commission on Biological Effects of Noise has been following scientific advances in this field by organizing international congresses from the first one in 1968 in Washington, DC, to the 11th congress in Nara, Japan, in 2014. There is already a large scientific body of evidence on the effects of noise on annoyance, communication, performance and behavior, mental health, sleep, and cardiovascular functions including relationship with hypertension and ischemic heart disease. In the last five years new issues in this field have been tackled. Large epidemiological studies on community noise have reported its relationship with breast cancer, stroke, type 2 diabetes, and obesity. It seems that noise-induced sleep disturbance may be one of the mediating factors in these effects. Given a large public health importance of the above-mentioned diseases, future studies should more thoroughly address the mechanisms underlying the reported association with community noise exposure.

Keywords: noise; cancer; stroke; diabetes mellitus type 2; obesity

INTRODUCTION

Community noise is a major environmental nuisance. According to a recent WHO (World Health Organization) study on environmental burden of disease, noise is ranked the third most important environmental factor, while air pollution by particulate matter (PM_{2.5}) and secondhand smoke are ranked first and second, respectively [1]. More than one million healthy life years (disability adjusted life years, DALY) are lost annually in the European Union member states alone because of community noise exposure. Noise-induced sleep disturbance is responsible for about 900,000 DALYs, annoyance for 650,000, ischemic heart disease for 65,000, and children's cognitive impairment for 45,000 DALYs [1].

The sources of community noise are mainly in urban areas and road-traffic noise accounts for about 80% of total urban noise pollution [2]. About a quarter of European population and about a third of the population of the United States are exposed to equivalent continuous day-evening-night noise level (L_{den}) exceeding 55 dB (A) and equivalent continuous noise level for 24 h [L_{eq} (24 h)] exceeding 70 dB (A) [3, 4]. L_{den} and L_{eq} (24 h) are continuous sound pressure levels equivalent to the total sound energy over a given period of time. Noise indicator L_{den} is accepted as the most accurate one, because it takes into account bonuses of 5 dB and 10 dB for evening and night period, respectively, given that sleep disturbances are most prominent in these periods.

According to the WHO community noise guidelines L_{eq} (A) of 55 dB during the daytime and 40 dB (A) during night should not be ex-

ceeded in the outdoor residential areas in order to prevent noise annoyance during daytime and sleep disturbances at night [4, 5]. Research in Serbia on noise and health has predominantly been performed by the Belgrade Team for Biological Effects of Noise (BETBEN). The BETBEN team has so far conducted scientific research on the effects of noise on mental performance [6, 7, 8], sleep disturbances [9], noise annoyance [10, 11], hypertension [12], myocardial infarction [13], as well as on blood pressure in children [14, 15].

Objective

Important advances have occurred in the last five years in the research of non-auditory effects of noise. The aim of this review is to analytically describe the recent studies on the relation between noise and the following diseases: stroke, cancer, type 2 diabetes, and obesity.

Methods

Internet search was performed by both authors in Pub Med database for the period 2010 up to the present, using the following keyword search terms: "noise," "stroke," "cancer," "diabetes," and "obesity." Inclusion criteria for references were as follows: full original articles and reviews, the English language, and journals listed in Science Citation Index. The outcomes under study were noise exposure and the occurrence of stroke, cancer, diabetes mellitus type 2, and obesity. The reviewed studies were grouped into four sections, based on their main out-

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comes. Noise units are reported exactly as stated in original papers, either as dB (unit of sound pressure level), or as dBA (unit of A-weighted sound pressure level).

NOISE AND STROKE

Long-term community noise exposure alone or combined with high nitrogen dioxide (NO₂) concentrations in the air have been related to ischemic stroke. The most recent population-based study was performed in Denmark on a cohort of about 57,000 people, followed from 1987 to 2009, aged 50–64 years at enrollment. The ischemic stroke incidence rate ratio (IRR) of 1.16 (95% confidence interval (CI): 1.07–1.24) was related to the 10 dB increase of community noise exposure. The strongest association was found for the combination of high noise and high NO₂ (IRR = 1.28; 95% CI = 1.09–1.52) [16]. In another study on a Danish cohort the association between noise and stroke was significantly influenced by age, given that people aged over 64.5 years were significantly more prone to stroke if exposed to road-traffic noise, while those under 64.5 years were not (IRR: 1.27; 95% CI: 1.13–1.43; and 1.02; 95% CI: 0.91–1.14, respectively) [17].

Concerning the association between aircraft noise and stroke, the important results were obtained by the Hypertension and Environmental Noise near Airports study (HYENA) comprising people living near airports in six European countries [18]. The study showed a positive association between night-time average aircraft noise and heart disease and stroke in those who had lived in the same place for ≥ 20 years (odds ratio [OR]: 1.25 [95% CI: 1.03–1.51] per 10 dB [A]). However, the authors suggested a possible confounding effect of air pollution.

In another population based study around Heathrow airport in London, the relative risk (RR) of hospital admissions for stroke was 1.24 (95% CI: 1.08–1.43) for people exposed to daytime aircraft noise levels L_{eq} 16h exceeding 63 dB compared to those exposed to aircraft noise levels below 51 dB [19]. Corresponding relative risks for mortality were of similar magnitude, although with wider confidence limits (RR: 1.21 [95% CI: 0.98–1.49]).

The proposed explanations for the association between noise exposure and the occurrence of stroke are related to mediating effect of hypertension and the changes in arterial function. It has been suggested that endothelial dysfunction may play a mediating role in the effects of noise on stroke. The authors reported that the flow-mediated dilation of a brachial artery, was significantly reduced (from 9.6 ± 4.3 to $7.9 \pm 3.7\%$; $p < 0.001$) in 60 noisy nights ($L_{eq} = 46.9 \pm 2.0$ dB [A]) compared to quiet control nights ($L_{eq} = 39.2 \pm 3.1$ dB [A]) [20].

NOISE AND CANCER

A special interest of the international scientific community has been provoked by a recently published article by Sørensen et al. [21] on the relation between exposure

to traffic noise and postmenopausal breast cancer. In a population-based Danish cohort of about 30,000 women aged 50–64 years at enrolment in 1993–1997 and during follow-up through 2010 the authors reported an increase of risks of estrogen receptor-negative breast cancer by 28% (95% CI: 1.04–1.56), by 23% (95% CI: 1.00–1.51) and by 20% (95% CI: 0.97–1.48) associated with a 10-dB higher level of road traffic noise during the previous one, five and 10 years, respectively. There was no association between noise exposure and estrogen receptor-positive breast cancer. The authors hypothesized that noise induced sleep disturbance may be related to a higher risk of breast cancer by lowering melatonin levels in blood and melatonin might protect from cancer by suppressing tumor angiogenesis [22]. Given the fact that this is the first study on the association between traffic noise and breast cancer, the conclusions may not be easily generalized and further studies are therefore needed to elaborate this hypothesis and to provide some explanations.

NOISE AND TYPE 2 DIABETES

Several studies on the association between noise and type 2 diabetes have been published recently. The strongest evidence is proposed by a meta-analysis that comprised six relevant studies on the association between residential noise exposure and the occurrence of diabetes [23]. Faced with methodological differences between the studies, the author transformed all noise metrics into L_{den} in order to quantify the tested association. The analysis showed that people who were exposed to $L_{den} > 60$ dB had 22% higher risk for type 2 diabetes in comparison to people exposed to $L_{den} < 60$ dB (RR: 1.22; 95% CI: 1.09–1.37) [23]. However, the results of this meta-analysis are mainly based on the research of Sørensen et al. [24]. In this study a population-based Danish cohort of about 57,000 people aged 50–64 years at enrollment was followed for 9.6 years. The cases of diabetes were obtained from a National Registry Database. Exposure to road traffic noise at the time of diabetes diagnosis and during the five years preceding diabetes diagnosis was obtained from noise maps and was linked to participants' home addresses. The study showed an increase of incidence rate ratio of type 2 diabetes by 11% (IRR: 1.11; 95% CI: 1.03–1.19) per 10 dB increase of road traffic noise at the time of diagnosis, as well an increase of incidence rate ratio by 14% (IRR: 1.14; 95% CI: 1.06–1.22) per 10 dB increase of road traffic noise during the five years preceding the diagnosis. The authors controlled potential confounders such as age, body mass index, waist circumference, education level, some lifestyle characteristics (smoking, eating habits, alcohol consumption, and physical activity), as well as air pollution (concentrations of nitrogen oxides (NO_x) at the time of diagnosis and during the five years before the diagnosis). They were not able, however, to consider other noise sources (noise from neighbors and indoor sources), bedroom location (orientation toward the street or away from it) and other factors that may influence noise exposure. The study promotes the

idea that socio-economic, educational, and residential factors may not play a large mediating role in the relationship between noise exposure and diabetes [24].

In another study by Heidemann et al. [25] about 3,600 German non-diabetic adults aged 18–79 years were followed-up during 12 years. Residential traffic exposure was assessed by the questionnaire rating the busyness of the road where participants' homes were located. The streets were classified into five groups, including those with rare or no traffic, moderately busy side streets, considerably busy side streets, heavily busy main or through roads and extremely busy through roads. The study showed that people residing on extremely busy roads had doubled risk of developing diabetes during the follow-up (OR: 1.97; 95% CI: 1.07–3.64) in comparison to people living in streets with no traffic. The odds for other types of streets did not reach statistical significance after adjusting for age, sex, active and passive smoking, type of heating, education, body mass index, waist circumference, sport activity, and family history of diabetes. Unfortunately, this study provided no evidence whether the observed association between traffic intensity and diabetes risk was attributable to traffic noise or to air pollution. The authors suggested that some inflammatory responses and noise annoyance may account for the observed association to a certain degree [25].

The effect of noise on diabetes may not be completely separated from the effect of air pollution. Several systematic reviews and meta-analyses constantly report of a significant relationship between air pollution and type 2 diabetes. Generally, two air pollutants are considered in those studies, such as particulate matter of diameter below 2.5 μm ($\text{PM}_{2.5}$) and NO_2 . For example, a recent meta-analysis of 10 cohort studies states that the risk of diabetes increases substantially in persons exposed to higher concentrations of $\text{PM}_{2.5}$ (RR: 1.39; 95% CI: 1.14–1.68 per 10 $\mu\text{g}/\text{m}^3$), and in those exposed to NO_2 (RR: 1.11; 95% CI: 1.07–1.16 per 10 $\mu\text{g}/\text{m}^3$) [26]. Similarly, another meta-analysis including five cross-sectional and five cohort studies reports a significant risk of exposure to NO_2 (hazard ratio [HR]: 1.13; 95% CI: 1.01–1.22), and of exposure to $\text{PM}_{2.5}$ (HR: 1.11; 95% CI: 1.03–1.20) on the occurrence of diabetes [27]. It should be noted that both meta-analyses imply a similar size of the association between air pollution parameters and type 2 diabetes.

A contradictory finding was reported in a large prospective study by Park et al. [28]. The authors followed about 6,800 persons aged 45–84 years from six sites in the United States for nine years. Although they confirmed a positive association between parameters of air pollution ($\text{PM}_{2.5}$ and NO_x) with the prevalence of diabetes at baseline, they were not able to identify such an association with the incidence of diabetes during the follow-up period. A possible explanation was related to a large within-site and between-site variations in $\text{PM}_{2.5}$ and NO_x concentrations over the investigated period [28].

The mechanisms underlying the association between noise and diabetes are currently under investigation. On the other side, there is a large body of evidence about the

direct and indirect effects of particulate matter on the development of type 2 diabetes. The possible pathways of action of $\text{PM}_{2.5}$ include endothelial dysfunction and subsequent insulin resistance, inflammation in several peripheral tissues, mitochondrial dysfunction, and alterations in visceral and brown adipose tissue; some changes in the pancreatic function, glucose-regulatory hormones, and insulin action have also been considered [29].

NOISE AND OBESITY

Recently, some interesting studies have been published on the association between noise exposure and obesity. A prospective cohort study in Sweden followed about 5,100 non-diabetic participants for up to 10 years. Using a geographic information system, authors linked all residential addresses with aircraft noise levels (expressed as L_{den}) modeled by airport services. Among the studied participants, an increase of aircraft noise level by 5 dBA was associated with an increase in waist circumference by 1.51 cm (RR: 1.51; 95% CI: 1.13–1.89) [30]. This risk was independently attributable to noise after adjustment for possible confounders. In general, the risk of increasing waist circumference from baseline to follow-up was more pronounced in men (RR: 2.26; 95% CI: 1.18–2.69) than in women (RR: 1.58; 95% CI: 1.13–2.03). The authors, however, observed no association between noise exposure and changes in body mass index or the occurrence of diabetes. They proposed that several individual characteristics may modify the observed relationship, such as high stress level (job strain), low physical activity, as well as no change in home address. Contrary to what may have been expected, they reported that sleep disturbance could not be considered to be a mediator in the association between noise exposure and metabolic outcomes [30]. The newest research on road traffic noise – obesity relation has been conducted in Norway [31]. In this study, about 8,400 middle-aged participants were followed for 10 years. Each participant's home address was linked with modeled levels of road traffic noise (expressed as L_{den}). Overall, there was no association between road traffic noise and obesity markers (body mass index, waist circumference or waist-hip ratio). However, when noise sensitivity was taken into consideration, the findings changed. Among highly noise-sensitive women (but not men), a two percent increase in body mass index (OR: 1.02; 95% CI: 1.01–1.03), a one percent increase in waist circumference (OR: 1.01; 95% CI: 1.00–1.02), and a 24% increase in waist-hip ratio over 0.85 (OR: 1.24; 95% CI: 1.01–1.53) correlated with an increase of road traffic noise by 10 dB. The authors found some modifying role of bedroom orientation toward the street and long-term exposure, but not that of noise annoyance or sleep disturbances [31].

These two studies contradict a large meta-analysis by Wu et al. [32] stating that short sleep duration is significantly associated with higher incidence of obesity in adults (defined as body mass index exceeding 30 kg/m^2 in Eu-

ropean and American studies, and as body mass index exceeding 25 kg/m² in Japanese studies). Analyzing twelve studies on this subject, the authors showed that short sleep duration (less than five hours) increased the odds for obesity by 25% (OR: 1.25; 95% CI: 1.14–1.38) in comparison to the normal sleep duration. The association was similar among men and women, and among different populations [32]. So far, the proposed biological mechanisms that may explain this relationship include alterations in levels of several hormones, including cortisol, leptin, ghrelin, as well as other endocrine alterations leading to decreased glucose tolerance and insulin sensitivity [33].

Future studies on the association between noise exposure and metabolic diseases should cover a wide range of potential risk factors, including individual factors, lifestyle habits, neighborhood quality, as well as broad range of environmental factors, including radiological or chemical pollution. Further research may provide important knowledge on how to design public health measures to prevent diabetes and obesity, how to design neighborhoods that

promote good health in several aspects, and how to improve the quality of environment in general.

CONCLUSION

In addition to the well-known non-auditory effects of community noise, recent advances in research have pointed out a possible relationship between traffic noise and breast cancer, stroke, type 2 diabetes, and obesity. Further studies are needed in this direction as this may significantly raise the public health importance of community noise.

ACKNOWLEDGMENTS

This work has been financially supported by the Serbian Ministry of Education, Science and Technological Development of the Republic of Serbia, Project No. 175078, 2010–2015.

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Скорашњи напредак у истраживању неаудитивних ефеката комуналне буке

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КРАТАК САДРЖАЈ

Неаудитивни ефекти буке на људе веома се помно истражују у последње четири деценије. Међународна комисија за биолошке ефекте буке прати научни напредак у овој области организујући међународне конгресе, почевши од оног у Вашингтону, у САД, 1968. године, до једанаестог конгреса 2014. у Нари, у Јапану. Сада већ постоји знатна количина научних доказа о ефектима буке на узнемиравање људи, споразумевање, рад и понашање, душевно здравље, спавање и функције срца и крвних судова, укључујући и повезаност с хипертензијом и исхемијском болешћу срца. У последњих

пет година обрађене су нове теме у овој области. Објављене су велике епидемиолошке студије о повезаности комуналне буке с раком дојке, можданим ударом, дијабетесом тип 2 и гојазношћу. Изгледа да ометање спавања буком може бити један од посредујућих чинилаца у овим ефектима. Узимајући у обзир велики народноздравствени значај горепомених болести, будућа истраживања треба да се усредсреде на механизме који би били у основи њихове могуће повезаности с комуналном буком.

Кључне речи: бука; канцер; мождани удар; дијабетес мелитус тип 2; гојазност

Примљен • Received: 04/05/2015

Прихваћен • Accepted: 07/12/2015