DE GRUYTER OPEN

DOI: 10.1515/folmed-2017-0045

ORIGINAL ARTICLE, MEDICINE

Community Noise Exposure and its Effect on Blood Pressure and Renal Function in Patients with Hypertension and Cardiovascular Disease

Angel M. Dzhambov[1], Mariya P. Tokmakova[2,3], Penka D. Gatseva[1], Nikolai G. Zdravkov[2,3], Dolina G. Gencheva[2,3], Nevena G. Ivanova[4,5], Krasimir I. Karastanev[2,3], Stefka V. Vladeva[6,7], Aleksandar T. Donchev[5], Svetlan M. Dermendzhiev[8]

[1] Department of Hygiene and Ecomedicine, Faculty of Public Health, Medical University of Plovdiv, Plovdiv, Bulgaria
[2] Section of Cardiology, First Department of Internal Diseases, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria
[3] Clinic of Cardiology, St George University Hospital, Plovdiv, Bulgaria
[4] Department of Urology and General Medicine, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria
[5] St Karidad Hospital, Plovdiv, Bulgaria
[6] Medical College, Medical University of Plovdiv, Plovdiv, Bulgaria
[7] Clinic of Endocrinology and Metabolic Disorders, Kaspela University Hospital, Plovdiv, Bulgaria
[8] Section of Occupational Diseases and Toxicology, Second Department of Internal Diseases, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria

Correspondence:
Angel M. Dzhambov, Department of Hygiene and Ecomedicine, Faculty of Public Health, Medical University of Plovdiv, Bulgaria, 15A Vassil Aprilov Blvd., 4002 Plovdiv, Bulgaria
E-mail: angelleloti@gmail.com
Tel: +359 32 580 562

Received: 23 Jan 2017
Accepted: 22 Mar 2017
Published Online: 28 Apr 2017
Published: 29 Sep 2017

Key words: noise exposure, road traffic noise, noise annoyance, glomerular filtration, air pollution

Citation: Dzhambov AM, Tokmakova MP, Gatseva PD, Zdravkov NG, Gencheva DG, Ivanova NG, Karastanev KI, Vladeva SV, Donchev AT, Dermendzhiev SM. Community noise exposure and its effect on blood pressure and renal function in patients with hypertension and cardiovascular disease.

Folia Medica 2017;59(3):344-356.
doi: 10.1515/folmed-2017-0045

Background: Road traffic noise (RTN) is a risk factor for cardiovascular disease (CVD) and hypertension; however, few studies have looked into its association with blood pressure (BP) and renal function in patients with prior CVD.

Aim: This study aimed to explore the effect of residential RTN exposure on BP and renal function in patients with CVD from Plovdiv Province.

Materials and methods: We included 217 patients with ischemic heart disease and/or hypertension from three tertiary hospitals in the city of Plovdiv (March – May 2016). Patients' medical history, medical documentation, and medication regimen were reviewed, and blood pressure and anthropometric measurements were taken. Blood samples were analyzed for creatinine, total cholesterol, and blood glucose. Participants also filled a questionnaire. Glomerular filtration rate was estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. All participants were asked about their annoyance by different noise sources at home, and those living in the city of Plovdiv (n = 132) were assigned noise map L_{den} and L_{night} exposure. The effects of noise exposure on systolic blood pressure (SBP), diastolic blood pressure (DBP), and estimated glomerular filtration rate (eGFR) were explored using mixed linear models.

Results: Traffic noise annoyance was associated with higher SBP in the total sample. The other noise indicators were associated with non-significant elevation in SBP and reduction in eGFR. The effect of L_{den} was more pronounced in patients with prior ischemic heart disease/stroke, diabetes, obesity, not taking Ca-channel blockers, and using solid fuel/gas at home. L_{night} had stronger effect among those not taking statins, sleeping in a bedroom with noisy façade, having a living room with quiet façade, and spending more time at home. The increase in L_{den} was associated with a significant decrease in eGFR among men, patients with ischemic heart disease/stroke, and those exposed to lower air pollution. Regarding L_{night}, there was significant effect modification by gender, diabetes, obesity, and time spent at home. In some subgroups, the effect of RTN was statistically significant.

Conclusions: Given that generic risk factors for poor progression of cardiovascular diseases cannot be controlled sufficiently at individual level, environmental interventions to reduce residential noise exposure might result in some improvement in the management of blood pressure and kidney function in patients with CVD.

BACKGROUND

Road traffic noise (RTN) is a risk factor for cardiovascular disease (CVD)[1] due to its stress effect on the hypothalamic-pituitary-adrenal and simpatico-adrenal axes and disturbance of normal sleep architecture.[2] A meta-analysis estimated OR = 1.03 (95% CI: 1.01, 1.06) of hypertension per every 5 dB increase in RTN exposure.[3] The public health importance of these findings is due to the wide spread of both CVD and RTN exposure. Globally, 17.3 million people die of CVD each year[4], with 9.4 of those due to hypertension[5], and this trend is projected to persist in decades to come.[4] Of note, elevated BP is not merely an outcome of noise exposure, but also a risk factor for adverse cardiovascular (CV) events such as myocardial infarction and stroke.[4] CVD mortality in Bulgaria rose from 61.5% in 1990 to 65.6% in 2012[6]; the raise in hypertension-specific mortality was even greater – from 2.9% to 7.5%[7]. Conversely, 125 million Europeans are exposed to day-evening-night noise levels (L_{den}) > 55 dB and 37 million to L_{den} > 65 dB[8], and in Bulgaria the proportion of citizens exposed above these safety thresholds is much higher.[9]

In contrast to hypertension, fewer studies have looked into the association between RTN and blood pressure (BP), showing close-to-null results and lack of a clear exposure-response relationship in the general population, possibly due to exposure misclassification and effect modification by antihypertensive mediation use.[10-14] Several Bulgarian studies conducted in Sofia evidenced higher prevalence of hypertension and higher BP in people exposed to day-evening equivalent noise level ($L_{eq\ 6\text{-}22\ h}$) > 60 dB, but they are dated (2000 – 2001) and reported unadjusted results.[15-18] Also of note, previous studies focused on the general population, rather than on patients with prior diagnosis with CVD, who are arguably more susceptible to the deleterious effect of RTN. In fact, subgroup analyses have revealed that the noise-attributed increase in BP was somewhat higher in CVD and diabetic patients in comparison to the rest.[13,19] Although the decline in renal function increases the risk of CVD morbidity and mortality[20], there are no data on its relationship with RTN. One previous study, however, reported significantly lower glomerular filtration rate in stroke survivors who lived within 50 m of a major road.[21] Finally, current strategies for prevention of adverse cardiovascular outcomes fail to adequately control individual-level risk factors in patients.[22,23]

Given the above listed gaps in the literature, this study aimed to explore the effect of residential RTN exposure on BP and renal function in patients with CVD from Plovdiv Province.

MATERIALS AND METHODS

Study population

The study population consisted of patients with CVD admitted for a follow-up in three tertiary hospitals in the city of Plovdiv in the period March – May 2016. Details on the study design and methodology are reported elsewhere.[24]

Briefly, the three hospitals were St George University Hospital (SGUH), Kaspela University Hospital (KUH), and St Karidad Hospital (SKH). They have comparable laboratory and anthropometric equipment and different catchment areas. SGUH is a government hospital and the largest tertiary hospital in Bulgaria, whereas KUH and SKH are private hospitals. Data were collected in the Cardiology Clinic of SGUH (40 staffed beds), the Endocrinology and Metabolic Diseases Clinic (40 staffed beds) of KUH, and the Cardiology Ward (26 staffed beds) of SKH.

All patients who were Bulgarian-speaking, decisionally-capacitated, able to read and write in Bulgarian, > 18 years of age, and resident in the Plovdiv Province during the preceding 12 months were eligible for the study. They had to have been diagnosed with hypertension (ICD-10: I10-I13) and/or ischemic heart disease (ICD-10: I20-I25) at least 12 months prior to their current hospitalization. We excluded patients with secondary hypertension (ICD-10: I15), secondary diabetes (ICD-10: E08, E09, E13), cancer, and pregnancy.

A cardiologist/internist took patients' medical history, reviewed their medical documentation and medication regimen, and carried out blood pressure and anthropometric measurements. A nurse collected fasting blood samples. Participants were also asked to fill a questionnaire on demographic, housing, neighborhood, and health-related factors. If they faced some problems, the questionnaire was administered during an interview.

Ethics

The study was reviewed and approved by the Ethics Review Committee at the Medical University of Plovdiv. It adhered to the Declaration of Helsinki, participants signed informed consent declarations, and their participation was voluntary and anonymous. They received no incentives.

Folia Medica

Sample size

The necessary sample size was estimated for bivariate and multiple linear regression models predicting blood pressure from RTN and noise annoyance. The input parameters for G*Power 3.1 were the desired statistical power (0.8), moderate anticipated effect size (0.15), and standard deviations of the predictor (L_{den}: ≈ 5-10 dB; noise annoyance: 2.27 – 1.43) and outcome variables (SBP: 20.4 mmHg; DBP: 12.3 mmHg). The latter were elicited from Dzhambov and Dimitrova[25] and Kotseva et al[26]. Overall, 217 patients were included: 111 from SGUH, 57 from KUH, and 49 from SKH (participation rates > 95%).

Noise exposure

Participants were asked about their annoyance by different noise sources while at home – traffic, industrial, neighborhood, building, and dwelling/apartment noises. These annoyances were measured on an 11-point Likert scale (0 – 10) and are recommended by the Bulgarian noise control regulation.[27] Noise annoyance gives account of the combined psychological and psychological stress responses to noise.[28] Two variables were derived from these questions – general noise annoyance (GNA) (averaged from all annoyances) and traffic noise annoyance (all types of motor vehicles). (See ref. 24)

For participants living in the city of Plovdiv (n = 132), we used the strategic noise map of Plovdiv prepared according to the Environmental Noise Directive (END) 2002/49/EC.[29] In 2015, field measurements were performed throughout the city by an independent consultant to the Municipality in order to update the acoustic model of Plovdiv.[30] Further details on the noise mapping are reported elsewhere.[25,31] Participants' addresses were geocoded and linked to the noise map. Because we had data on the orientation of rooms within each building, we were able to assign road traffic day-evening-night equivalent noise level (L_{den}) and night equivalent noise level (L_{night}) to the living room and bedroom façades, respectively. Noise levels were determined for each individual case using 3D Google Earth imagery, Google Street View, and in-person visits, and corrected by an expert if necessary, in order to minimize the exposure misclassification pertinent to European noise mapping.[32] We also calculated indoor L_{den} and L_{night} by subtracting different correction factors from the façade noise levels depending on the presence of soundproofing insulation and the window-opening frequency.[14] (See ref. 24)

Outcome variables

A cardiologist/internist measured patients' morning systolic and diastolic BP (SBP and DBP, respectively), according to the *American Heart Association* guidelines.[33] A calibrated aneroid sphygmomanometer with standard cuff-size (in most cases) was used. The cuff was placed at the level of the right atrium. After patients rested for 5 min in a sitting position, three consecutive readings were taken in 1-min intervals. The first reading was taken on both arms, and the rest – on the arm showing higher BP values. We averaged the results of the three readings to compute the SBP and DBP variables. We also calculated the mean arterial pressure (MAP) as (2 × DBP + SBP)/3.

Glomerular filtration rate is the "best overall measure of kidney function", but since its direct measurement is oftentimes infeasible, different equations for its estimation have been developed.[34] Herein we used estimated glomerular filtration rate (eGFR) as an indicator of renal function, calculated according to the Chronic Kidney Disease Epidemiology Collaboration equation (CKD-EPI)[35]:

$$\text{GFR mL/min/1.73 m}^2 = 141 \times \min(\text{Scr}/\kappa, 1)^{\alpha} \times \max(\text{Scr}/\kappa, 1)^{-1.209} \times 0.993^{\text{Age}} \times 1.018 \text{ [if female]} \_ 1.159 \text{ [if black]},$$

where Scr is serum creatinine, κ is 0.7 for females and 0.9 for males, α is -0.329 for females and -0.411 for males, min indicates the minimum of Scr/κ or 1, and max indicates the maximum of Scr/κ or 1.[35]

Serum creatinine (μmol/l), analyzed using enzymatic creatinine assay and biochemical analyzer (Konelab 60i, Thermo Electron Corporation), was divided by 88.4 to convert it to mg/dl for the CKD-EPI equation. The CKD-EPI equation has been recommended over alternative methods for estimating eGFR.[36] It was previously validated in Bulgaria.[37]

Covariates

Participants were queried about gender, age, ethnicity, highest educational attainment, marital status, occupation, perceived socioeconomic status, dietary intake, alcohol consumption, smoking, moderately vigorous psychical strain (for at least 10 min), perceived stress (4-point scale), noise sensitivity (0 – 10 Likert scale), sleep disturbance (0 – 10 Likert scale), hearing impairment (yes/no), energy

sources used for domestic heating/cooking, floor of the dwelling/apartment, duration of residence, and the average time/day spent at home in the past 12 months.

Diet quality was assessed with a food frequency questionnaire, taking into account common food products divided into recommended items and non-recommended items summarized on a diet quality scale in such a way, so that lower scores indicated "less frequent consumption of recommended and more frequent of non-recommended foods" and higher scores, "less frequent consumption of non-recommended and more frequent of recommended foods".[38]

Traffic-related fine particulate matter ($PM_{2.5}$) levels were elicited from the dispersion model of Plovdiv described elsewhere[25] and linked to the geocoded addresses.

Patients' medical history and documentation were reviewed for family history with ischemic heart disease (ICD-10: Z82.4), stroke (ICD-10: Z82.3), or diabetes (ICD-10: Z83.3), and for co-morbidity with ischemic heart disease (ICD-10: I20-I25), stroke (ICD-10: I61, I63-I64), hypertension (ICD-10: I10-I13), diabetes (ICD-10: E10-E12), and chronic kidney disease (CKD) (ICD-10: N18). The regular intake in the past year of antihypertensive and lipid-lowering medication was also assessed (yes/no).

Morning venous blood samples were drawn by a trained nurse after an overnight fast. Blood glucose, lipids, creatinine, and uric acid were analyzed at the central clinical laboratory of each hospital using enzymatic methods and automatic biochemical analyzers (Konelab 60i, Thermo Electron Corporation). For this study, we used only data on glucose, total cholesterol, and creatinine.

Body weight was measured in light clothing on a calibrated digital scale. Height was measured standing without shoes. Body mass index was calculated as weight in kg divided by height squared in cm. Waist circumference was measured at the midpoint between the lower margin of the least palpable rib and the top of the iliac crest, using a stretch-resistant tape. Measurements were performed twice at the end of a normal expiration.[39]

Data analysis

The dataset was first screened for missing values (replaced using the expectation-maximization algorithm) and outliers (winsorized). The distributional normality of the dependent variables was tested with the D'Agostino-Pearson K^2 test and histograms. Likert-scale variables were treated as continuous.[40]

The bivariate associations between different variables were explored with the Welch's t-test/ANOVA, the Pearson's chi-squared test/Fisher's exact test/Fisher-Freeman-Halton test, and the Spearman correlation.

The main effects of L_{den}, L_{night}, GNA, and traffic noise annoyance on SBP, DBP, and eGFR were explored in separate mixed linear models with restricted maximum likelihood estimator and random intercept to account for clustering on hospitals (one model for each exposure – outcome pair). Each of the outcome variables (SBP, DBP, and eGFR) was regressed on the exposure indicator of interest (L_{den}, L_{night}, GNA) and additionally adjusted for relevant confounders. The covariate set in each model was selected using directed acyclic graphs and DAGitty v. 2.3, based on the expertise of the authors in the field and the literature. We report several types of adjusted models depending on the exposure – outcome pair. Because only 132 participants had objective noise exposure data, the models for the indicators L_{den} and L_{night} were run on that sample of 132 participants, whereas the models for noise annoyance, on all 217 participants.

Potential modifiers of the effect of outdoor L_{den} and L_{night} were tested at the relaxed $p < 0.2$ level in order to have more power.[24] Other results were considered statistically significant at the $p < 0.05$ (two-tailed) level.

RESULTS

Sample characteristics

The sample included 217 participants, 132 of whom were living in the city of Plovdiv. Participants' characteristics are shown in **Table 1**.

The univariate Spearman correlations showed that SBP was associated with male gender ($\rho = -0.20$, $p = 0.004$), alcohol consumption ($\rho = 0.20$, $p = 0.003$), lower diet quality ($\rho = -0.23$, $p = 0.001$), waist circumference ($\rho = 0.15$, $p = 0.033$), noise sensitivity ($\rho = 0.25$, $p < 0.001$), sleep disturbance ($\rho = 0.21$, $p = 0.002$), traffic noise annoyance ($\rho = 0.22$, $p = 0.001$), hearing impairment ($\rho = 0.19$, $p = 0.005$), higher cholesterol ($\rho = 0.18$, $p < 0.001$), and β-blocker use ($\rho = 0.22$, $p = 0.001$). DBP was associated with less smoking ($\rho = -0.15$, $p = 0.029$), more physical strain ($\rho = 0.19$, $p = 0.006$), noise sensitivity ($\rho = 0.34$, $p < 0.001$), sleep disturbance ($\rho = 0.27$, $p < 0.001$), stress ($\rho = 0.18$, $p = 0.010$), traffic noise annoyance ($\rho = 0.24$, $p < 0.001$), air pollution ($\rho = 0.17$, $p = 0.047$), having CVD (ischemic heart disease/stroke)

Folia Medica

Table 1. Descriptive characteristics of the patients (n = 217)

Characteristics	Total sample
Age (n, %)	
< 53	64 (29.5)
53 – 63	45 (20.7)
63 – 72	55 (25.3)
> 72	53 (24.4)
Men (n, %)	104 (47.9)
Bulgarian (n, %)	200 (92.2)
Education (n, %)	
primary/junior high school or less	56 (25.8)
secondary	95 (43.8)
higher	66 (30.4)
Lower socioeconomic status (n, %)	66 (30.4)
Diet quality (mean, SD)	42.81 (6.22)
Alcohol drinking (n, %)	
lifetime abstainer/former drinker	89 (41.0)
current light drinker	57 (26.3)
current moderate/heavy drinker	71 (32.7)
Smoking (n, %)	
never smoker	115 (53.0)
former smoker	48 (22.1)
current smoker	54 (24.9)
Physical strain (n, %)	
inactive	42 (19.4)
low activity	80 (36.9)
active	95 (43.8)
Noise sensitivity (mean, SD)	4.58 (2.24)
Stress (mean, SD)	2.35 (1.03)
GNA (median, IQR)	3.20 (2.87)
Traffic noise annoyance (median, IQR)	3.50 (3.00)
Traffic $PM_{2.5}$ > 2.0 μg/m^3 (n, %)	72 (33.2)
Sleep disturbance (mean, SD)	3.62 (2.31)
CVD (ischemic heart disease/stroke) (n, %)	106 (48.8)
Hypertension (n, %)	214 (98.6)
Diabetes (n, %)	99 (45.6)
CKD (n, %)	16 (7.4)
Hearing loss (n, %)	88 (40.6)
ACE-inhibitors (n, %)	129 (59.4)
Diuretics (n, %)	125 (57.6)
Ca-channel blockers (n, %)	76 (35.0)
β-blockers (n, %)	135 (62.2)
Statins (n, %)	67 (30.9)
SBP (mean mmHg, SD)	131.84 (12.38)
DBP (mean mmHg, SD)	80.26 (8.13)
MAP (mean mmHg, SD)	97.45 (8.80)
eGFR (mean mL/min, SD)	65.03 (20.81)
Total cholesterol (mean mmol/l, SD)	5.47 (1.22)
Blood glucose (mean mmol/l, SD)	6.68 (1.66)
Creatinine (mean μmol/l, SD)	101.04 (31.02)
Waist circumference (mean cm, SD)	94.80 (19.07)
Body mass index (mean kg/m^2, SD)	29.65 (5.54)

Note. SD – standard deviation, GNA – global noise annoyance, CKD – chronic kidney disease, SBP – systolic blood pressure, DBP – diastolic blood pressure, MAP – mean arterial pressure, eGFR – estimated glomerular filtration rate, CaA – calcium, CVD – cardiovascular disease, ACE – angiotensin-converting enzyme, IQR – interquartile range, n – number, $PM_{2.5}$ – fine particulate matter.

(ρ = 0.26, p < 0.001), hearing impairment (ρ = 0.15, p = 0.033), total cholesterol (ρ = 0.30, p < 0.001), use of diuretics (ρ = 0.18, p = 0.009), β-blockers (ρ = 0.20, p = 0.003), and statins (ρ = 0.23, p = 0.001).

eGFR, on the other hand, was lower in women (ρ = -0.24, p < 0.001), older people (ρ = -0.54, p < 0.001), Bulgarians (ρ = -0.19, p = 0.006), those with lower education (ρ = 0.19, p = 0.005) and socioeconomic status (ρ = 0.17, p = 0.015), smoking (ρ = 0.28, p < 0.001) and consuming alcohol less (ρ = 0.17, p = 0.012), lower physical strain (ρ = 0.17, p = 0.014), having CVD (ρ = -0.18, p = 0.008) and CKD (ρ = -0.39, p < 0.001), higher glucose (ρ = -0.16, p = 0.017) and creatinine (ρ = -0.82, p < 0.001), use of diuretics (ρ = -0.23, p

Table 2. Associations of global noise annoyance (per one interquartile range), traffic noise annoyance (per 1 unit), road traffic noise (per 5 dB), with blood pressure and eGFR (mixed linear models)

	Change (95% CI)		
Exposure indicator (sample size)	**SBP (mmHg)**	**DBP (mmHg)**	**eGFR (mL/min/1.73 m²)**
Global noise annoyance (n = 217)			
Basic model (gender and age)	2.14 (-0.30, 4.59)	1.44 (-0.05, 2.92)	1.66 (-1.69, 5.02)
Main model[a]	0.22 (-2.82, 3.27)	0.33 (-1.56, 2.21)	0.20 (-3.97, 4.36)
Main model + mediators[c]	.	.	
Traffic noise annoyance (n = 217)			
Basic model (gender and age)	**1.11 (0.44, 1.78)***	**0.62 (0.21, 1.03)***	-0.10 (-1.04, 0.83)
Main model[a]	**0.94 (0.11, 1.77)***	0.49 (-0.02, 1.00)	-0.54 (-1.67, 0.59)
Main model + mediators[c]			-0.49 (-1.69, 0.72)
Outdoor L_{den} (n = 132)			
Basic model (gender and age)	0.24 (-1.54, 2.01)	-0.19 (-1.27, 0.90)	-0.29 (-2.65, 2.07)
Main model[a]	-0.20 (-1.96, 1.56)	-0.45 (-1.56, 0.67)	-0.48 (-2.84, 1.88)
Main model + air pollution[b]	-0.14 (-1.93, 1.64)	-0.25 (-1.35, 0.86)	-0.73 (-3.10, 1.65)
Main model + mediators[c]	-0.13 (-1.90, 1.64)	-0.52 (-1.67, 0.62)	-0.66 (-3.01, 1.69)
Indoor L_{den} (n = 132)			
Basic model (gender and age)	0.32 (-1.14, 1.79)	-0.24 (1.15, 0.66)	0.19 (-1.74, 2.12)
Main model[a]	-0.03 (-1.56, 1.49)	-0.52 (-1.48, 0.45)	0.42 (-1.62, 2.46)
Main model + air pollution[b]	0.03 (-1.53, 1.58)	-0.31 (-1.27, 0.65)	0.17 (-1.90, 2.24)
Main model + mediators[c]	0.09 (-1.44, 1.62)	-0.53 (-1.52, 0.45)	-0.10 (-2.15, 1.95)
Outdoor L_{night} (n = 132)			
Basic model (gender and age)	**1.97 (0.27, 3.67)***	0.20 (-0.87, 1.29)	-0.71 (-2.96, 1.54)
Main model[a]	0.90 (-0.87, 2.66)	-0.23 (-1.39, 0.94)	-0.87 (-3.22, 1.48)
Main model + air pollution[b]	0.88 (-0.88, 2.66)	-0.24 (-1.37, 0.89)	-0.84 (-3.18, 1.50)
Main model + mediators[c]	0.80 (-0.98, 2.58)	-0.38 (-1.57, 0.81)	-0.62 (-3.03, 1.80)
Indoor L_{night} (n = 132)			
Basic model (gender and age)	0.48 (-0.72, 1.68)	-0.32 (-1.06, 0.41)	-0.41 (-1.88, 1.06)
Main model[a]	-0.01 (-1.22, 1.20)	-0.44 (-1.21, 0.33)	0.06 (-1.49, 1.60)
Main model + air pollution[b]	0.39 (-0.81, 1.59)	-0.20 (-0.98, 0.58)	-0.12 (-1.71, 1.47)
Main model + mediators[c]	0.08 (-1.12, 1.28)	-0.42 (-1.20, 0.37)	-0.06 (-1.61, 1.49)

Note. L_{den} – day-evening-night equivalent noise level, L_{night} – night equivalent noise level. $PM_{2.5}$ –fine particulate matter, eGFR – estimated glomerular filtration rate, SBP – systolic blood pressure, DBP – diastolic blood pressure. Interquartile range for global noise annoyance is 2.80. *significant at p < 0.05. [a]Adjusted for gender, age + ethnicity, socioeconomic status, education, diet quality, smoking, alcohol drinking, physical strain, waist circumference, and noise sensitivity (the models with SBP, DBP, and eGFR), and creatinine (only the models with SBP and DBP). [b]Main adjustments + fine particulate matter. [c]Main adjustments + stress, sleep disturbance, and traffic noise annoyance (the models with SBP and DBP) or stress, sleep disturbance, glucose, total cholesterol, and mean arterial blood pressure (the model with eGFR).

= 0.001), and β-blockers (ρ = -0.18, p = 0.008).

Objective noise exposure in the city of Plovdiv (N = 132)

Table 2 shows the multivariate effects of the objective noise indicators on SBP, DBP, and eGFR. SBP increased non-significantly with the increase in noise exposure in most models. L_{night} at the bedroom façade was a better predictor of SBP than L_{den} at the living room façade, but its effect was only significant in the Basic model. As regards eGFR, outdoor L_{den} and L_{night} were associated with non-significantly lower eGFR.

Next, we stratified the Main model, regressing SBP on outdoor L_{den}/L_{night} by participants' characteristics. Subgroups with a statistically significant (p < 0.05) increase in SBP or significant interac-

Table 3. Stratified analysis for identifying potential effect modification of the association between outdoor road traffic noise (per 5 dB) and systolic blood pressure (mixed linear models)

	Change (95% CI) in SBP (mmHg)			
	Outdoor L_{den}	**$p_{interaction}$**	**Outdoor L_{night}**	**$p_{interaction}$**
Cardiovascular disease		**0.018**		0.168
no	-2.26 (-5.17, 0.64)		-0.27 (-3.34, 2.79)	
yes	2.56 (-0.19, 5.31)		***2.55 (-0.02, 5.12)****	
Diabetes		**0.036**		0.749
no	-2.37 (-5.49, 0.75)		0.45 (-2.89, 3.79)	
yes	1.79 (-0.52, 4.10)		1.11 (-1.07, 3.28)	
Body mass index		**0.154**		0.542
< 30 kg/m^2	-1.05 (-3.54, 1.45)		0.14 (-2.36, 2.65)	
≥ 30 kg/m^2	1.85 (-1.26, 4.95)		1.39 (-1.77, 4.55)	
Ca-channel blockers		**0.193**		0.857
no	0.09 (-2.01, 2.19)		1.67 (-0.37, 3.71)	
yes	-2.45 (-5.64, 0.76)		1.29 (-2.31, 4.89)	
Statins		0.267		**0.027**
no	0.65 (-1.47, 2.76)		1.72 (-0.55, 3.99)	
yes	-1.93 (-5.97, 2.11)		-3.30 (-7.13, 0.54)	
Bedroom orientation		0.256		**0.146**
quiet façade	1.82 (-0.32, 3.97)		1.43 (-1.11, 3.96)	
noisy façade	-0.79 (-4.75, 3.17)		**6.64 (0.09, 13.18)****	
Living room orientation		0.857		**0.179**
quiet façade	-1.02 (-3.54, 1.50)		2.43 (-0.34, 5.20)	
noisy façade	-1.61 (-7.49, 4.28)		-0.21 (-2.88, 2.47)	
Heating/cooking energy		**0.064**		0.214
electricity/steam radiator only	-0.34 (-2.51, 1.82)		0.66 (-1.47, 2.78)	
wood/coal/gas used	**3.58 (0.05, 7.11)****		3.78 (-0.66, 8.22)	
Time at home/day		0.295		**0.085**
< 12 hours	-1.24 (-4.48, 1.99)		-2.36 (-6.12, 1.40)	
≥ 12 hours	0.85 (-1.34, 3.04)		1.40 (-0.64, 3.43)	
$PM_{2.5}$		0.224		0.246
< 2.0 μg/m^3	-1.56 (-5.25, 2.12)		**3.84 (0.49, 7.19)****	
> 2.0 μg/m^3	1.04 (-0.96, 3.04)		1.51 (-0.56, 3.58)	

Note. L_{den} – day-evening-night equivalent noise level, L_{night} – night equivalent noise level, $PM_{2.5}$ – fine particulate matter. *significant at p < 0.1, **p < 0.05. Models are adjusted for gender, age, ethnicity, socioeconomic status, education, diet quality, smoking, alcohol drinking, physical strain, total cholesterol, creatinine, waist circumference, and noise sensitivity. (When the model was stratified by the respective factor, the latter was removed from the covariate set.)

tion terms ($p < 0.2$) are shown in **Table 3**. The effect of L_{den} was more pronounced in patients with prior CVD, diabetes, obesity, not taking Ca-channel blockers, and using solid fuel/gas at home. The estimate itself was significant only among people using solid fuel/gas. Outdoor L_{night} had stronger effect among those not taking statins, sleeping in a bedroom with noisy façade, having a living room with quiet façade, and spending > 12 h/day at home. The increase in SBP was significant among people with CVD, noisy bedroom, and low exposure to $PM_{2.5}$.

From **Table 4**, the increase in outdoor L_{den} was associated with a significant ($p < 0.05$) decrease in eGFR among men, patients with CVD, and those exposed to lower $PM_{2.5}$. The same factors were also significant effect modifiers ($p < 0.2$). Outdoor L_{night} was associated with a significantly ($p < 0.05$) lower eGFR in men; there was also a significant effect modification ($p < 0.2$) by gender, diabetes, obesity, and time spent at home.

Noise annoyance in the Plovdiv province (n = 217)

From **Table 2**, higher traffic noise annoyance was associated with a significant increase in SBP and DBP (Basic model). None of the estimates for global noise annoyance was statistically significant. Noise annoyance was not associated with eGFR either.

DISCUSSION

Findings on noise and blood pressure

Overall, results showed that higher traffic noise annoyance at home was associated with an increase in SBP, which agrees with the higher risk of hypertension reported by the meta-analysis of Ndrepepa and Twardella.[41] Regarding RTN, the effect on SBP in the whole sample was non-significant, and there was no effect on DBP. This is also in line with the literature. In their cohort study, Sørensen et al. found that in the subsample of people with prior diagno-

Table 4. Stratified analysis for identifying potential effect modification of the association between outdoor road traffic noise (per 5 dB) and renal function (mixed linear models)

	Change (95% CI) in eGFR (mL/min/1.73 m²)			
	Outdoor L_{den}	$p_{interaction}$	Outdoor L_{night}	$p_{interaction}$
Gender		**0.154**		**0.116**
men	***-3.14 (-6.28, 0.01)****		**-3.24 (-6.17, -0.32)****	
women	0.55 (-3.43, 4.53)		0.75 (-3.28, 4.78)	
Diabetes		0.757		**0.139**
no	-0.86 (-3.60, 1.88)		0.90 (-2.02, 3.82)	
yes	0.01 (-4.75, 4.77)		-2.99 (-7.25, 1.26)	
Body mass index		0.984		**0.200**
< 30 kg/m²	-0.31 (-3.62, 3.01)		-2.06 (-5.32, 1.19)	
≥ 30 kg/m²	-0.25 (-4.03, 3.54)		1.21 (-2.57, 5.00)	
Family history with CVD		**0.077**		0.826
no	0.72 (-2.88, 4.33)		-0.16 (-3.80, 3.48)	
yes	**-3.96 (-7.67, -0.25)****		-0.75 (-4.38, 2.88)	
Time at home/day		0.616		**0.102**
< 12 hours	-1.71 (-6.19, 2.76)		-3.96 (-8.67, 0.75)	
≥ 12 hours	-0.27 (-3.69, 3.15)		0.77 (-2.37, 3.92)	
$PM_{2.5}$		**0.010**		0.525
< 2.0 μg/m³	**-5.36 (-9.65, -1.07)****		-2.90 (-7.38, 1.59)	
> 2.0 μg/m³	1.29 (-1.43, 4.02)		-1.18 (-4.01, 1.65)	

Note. L_{den} – day-evening-night equivalent noise level, L_{night} – night equivalent noise level, $PM_{2.5}$ – fine particulate matter. *significant at $p < 0.1$, **$p < 0.05$. Models are adjusted for gender, age, ethnicity, socioeconomic status, education, diet quality, smoking, alcohol drinking, physical activity, waist circumference, and noise sensitivity. (When the model was stratified by the respective factor, the latter was removed from the covariate set.)

Folia Medica

sis of CVD SBP increased with 1.15 mmHg (95% CI: -0.54, 2.85) per 10 dB, but there was no effect on DBP.[13] Another cross-sectional study reported 2.10 mmHg (95% CI: -0.59, 4.79) increase in SBP and 1.22 mmHg (95% CI: -0.25, 2.69) increase in DBP per 10 dB in people with CVD.[19] Foraster et al. found that nighttime noise affected SBP more than DBP – there was 0.36 mmHg (95% CI: -0.06, 0.77) increase in SBP per 5 dB outdoor L_{night} and 0.72 mmHg (95% CI: 0.29, 1.15) per 5 dB indoor L_{night}.[14] Several population-based studies observed null[12,11] or even inverse relationship between RTN and BP[42]. All these results are most likely due to exposure misclassification or other methodological limitations. Nevertheless, the effects on SBP are quite important because just 1 mmHg population-wide decrease in SBP would result in 13.3/100 000 person-years reduction in heart failure events.[43]

SBP increased significantly with the increment in outdoor L_{den} among people using solid fuel/gas at home, and in L_{night} among those with prior CVD, sleeping in a bedroom with a noisy façade, and exposed to lower $PM_{2.5}$. The greatest increase in SBP was related to the orientation of the bedroom, which is important since elevated SBP at night (when it is supposed to dip) is a predictor of cardiovascular morbidity and mortality independently from daytime SBP.[44] Like us, Dratva et al. reported stronger effect among diabetics.[19] Indoor air pollution, a risk factor for CVD per se[45], emerged as an important factor aggravating the effect of noise. Taking BP-lowering medication seemed to buffer the effect of RTN in our study, while Foraster et al. did not evidence a significant interaction.[14] Unexpectedly, RTN had stronger effect among people exposed to lower traffic-related air pollution, which we explain by unmodelled geospatial confounding due to the urban fabric of Plovdiv.

Findings on noise and eGFR

We found a significant reduction in eGFR in men, those with a family history of CVD, and exposed to lower $PM_{2.5}$. Several mechanisms are surmised to explain this effect. On one hand, elevated BP may result from noise exposure and thereby lead to impaired kidney function and CKD.[46] The lack of a correlation between BP and eGFR in our sample can be attributed to the cross-sectional design of the study. However, as participants lived at the same address for many years, it is possible that RTN affected their eGFR through elevated BP. Another mechanism could be sleep disturbance due to nighttime noise, which has been linked to aduction in eGFR.[47] The stronger effect of L_{night} compared to L_{den} is in line with this hypothesis.

Although direct comparison with previous studies is not possible, a recent study of 1103 post-stroke patients reported that those living within 50 m of a major road had -3.9 mL/min/1.73 m^2 (95% CI: -1.0, -6.7; p=0.007) lower eGFR compared to those living more than 1000 m away.[21] A clinical trial, that is not yet completed, exposes men to 30-minute recordings of aircraft noise and monitors the changes in renal hemodynamics.[48]

Strengths and limitations

This study focused on a sample of patients with CVD, whereas previous research on residential noise and BP draws on population-based samples. The relationship between RTN and eGFR we found is also novel and needs further exploration. Other strengths include the rich set of potential mediators, covariates, and effect modifiers we tested. Going further, we had information on the location of rooms within each building, soundproofing insulation, and window-opening frequency, which allowed us to assign L_{den} and L_{night} levels to different facades and to correct them to indoor noise levels.

A few limitations need to be acknowledged. First, the study was cross-sectional, precluding any causal interpretation of the association between self-reported noise annoyance and the outcomes. Conversely, for RTN there is little bias in this regard, because most participants were non-movers for many years, and also it is highly unlikely for those with higher BP and lower eGFR to have moved to noisier streets on purpose.[25] Future studies should preferably adopt longitudinal designs. Moreover, (quasi-)experimental studies are needed to investigate the association between noise stress and renal function and the underlying pathophysiological mechanisms. Second, although we used validated noise annoyance questions and the latter are well-suited for explorative purposes and hypothesis testing, a wide range of personality differences impact on noise perception, which could potentially lead to exposure misclassification. Third, some subsamples (e.g., people living in the city of Plovdiv, those with CKD) were smallish, leading to lower power in the subgroup analyses. Fourth, despite the corrections we did to minimize exposure misclassification, the END noise maps have their limitations[49]; therefore, our results are probably biased towards the null.

CONCLUSIONS

Traffic noise annoyance was associated with higher blood pressure in the total sample, but no other indicator was associated with blood pressure or eGFR. Nevertheless, there were potentially susceptible subgroups in which road traffic noise had an adverse effect on blood pressure and eGFR. Given that generic risk factors for poor progression of cardiovascular diseases cannot be controlled sufficiently at individual level, environmental interventions to reduce residential noise exposure might result in some improvement in the management of blood pressure and kidney function in patients with CVD.

ACKNOWLEDGEMENTS

The authors would like to thank all patients who participated in the study, the administrations of the involved hospitals, and the staff members who facilitated data collection.

CONFLICT OF INTEREST STATEMENT

The authors declare that they do not have any relationships that could be construed as potential conflict of interest. The work was not supported financially by external sources.

REFERENCES

1. Münzel T, Gori T, Babisch W, et al. Cardiovascular effects of environmental noise exposure. Eur Heart J 2014;35(13):829-36.
2. Recio A, Linares C, Banegas JR, et al. Road traffic noise effects on cardiovascular, respiratory, and metabolic health: An integrative model of biological mechanisms. Environ Res 2016;146:359-70.
3. van Kempen E, Babisch W. The quantitative relationship between road traffic noise and hypertension: a meta-analysis. J Hypertens 2012;30:1075-86.
4. Mendis S, Puska P, Norrving B, WHO, World Heart Federation, World Stroke Organization, editors. Global atlas on cardiovascular disease prevention and control. Geneva: WHO; 2011.
5. WHO. A global brief on hypertension: silent killer, global public health crisis. Geneva: WHO; 2013.
6. Dikova K, Vichev M, Panayotov K. [Nosologic structure of mortality in the Republic of Bulgaria between 1990 and 2012.] Social Medicine 2014;1(1):7-9 (Bulgarian).
7. Panayotov K, Dikova K, Vichev M. [Cardiovascular diseases mortality in Bulgaria between 1990 and 2012.] Social Medicine 2014;1(2):20-1 (Bulgarian).
8. European Environment Agency. Noise in Europe 2014. Report No 10/2014. Luxembourg: Publications Office of the European Union, 2014.
9. Noise observation and information service for Europe (NOISE). Copenhagen, European Environment Agency. Available at http://noise.eionet.europa.eu.
10. van Kempen EE, Kruize H, Boshuizen HC, et al. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspect 2002;110(3):307-17.
11. Méline J, Van Hulst A, Thomas F, et al. Road, rail, and air transportation noise in residential and workplace neighborhoods and blood pressure (RECORD Study). Noise Health 2015;17(78):308-19.
12. Halonen JI, Dehbi HM, Hansell AL, et al. Associations of night-time road traffic noise with carotid intima-media thickness and blood pressure: The Whitehall II and SABRE study cohorts. Environ Int 2017;98:54-61.
13. Sørensen M, Hvidberg M, Hoffmann B, et al. Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: a cohort study. Environ Health 2011;10:92.
14. Foraster M, Künzli N, Aguilera I, et al. High blood pressure and long-term exposure to indoor noise and air pollution from road traffic. Environ Health Perspect 2014;122(11):1193-200.
15. Tzenova B, Radneva R, Tzvetkov D, et al. [Characteristics of noise exposure in modern living environment and their health effects.] Hygiene and Public Health 2000;5-6:29-33 (Bulgarian).
16. Radneva R, Tzenova B, Tzvetkov D, et al. [A prospective study of traffic noise blood pressure effects – phase 1.] Hygiene and Public Health 2001;44(1):11-4 (Bulgarian).
17. Radneva R, Torbova S, Tzenova B, et al. [Acoustic characteristics of living environment and epidemiology of arterial hypertension.] Hygiene and Public Health 2001;44(1):14-19 (Bulgarian).
18. Radneva R, Tzenova B, Torbova S, et al. [24-hour blood pressure monitoring and heart pulse rate of residents of noisy residential areas.] Hygiene and Public Health 2001;44(1):19-22 (Bulgarian).
19. Dratva J, Phuleria HC, Foraster M, et al. Transportation noise and blood pressure in a population-based sample of adults. Environ Health Perspect 2012;120(1):50-5.
20. Herzog CA, Asinger RW, Berger AK, et al. Cardiovascular disease in chronic kidney disease. A clinical update from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int 2011;80(6):572-86.
21. Lue SH, Wellenius GA, Wilker EH, et al. Residential proximity to major roadways and renal function. J Epidemiol Community Health 2013;67(8):629-34.
22. Gocheva N, Postadzhian A, Raev D, et al. [Undertreatment of hypercholesterolemia in Bulgaria in pri-

mary and secondary prevention - the CEPHEUS study.] Science Cardiology 2012;71(1):26-7 (Bulgarian).
23. Kotseva K. EUROASPIRE IV: Where do we stand after EUROASPIRE I, II and III? European Society of Cardiology. Available at: http://www.escardio.org/static_file/Escardio/education/live-events/courses/education-resource/Sat-25-EUROASPIRE-Kotseva.pdf.
24. Dzhambov AM, Tokmakova MP, Gatseva PD, et al. Is community noise associated with metabolic control in patients with cardiovascular disease? Acoustics Australia 2017;45(1):61-75.
25. Dzhambov AM, Dimitrova DD. Exposures to road traffic, noise, and air pollution as risk factors for type 2 diabetes: A feasibility study in Bulgaria. Noise Health 2016;18:133-42.
26. Kotseva K, Wood D, De Backer G, et al; EUROASPIRE Study Group. EUROASPIRE III: a survey on the lifestyle, risk factors and use of cardioprotective drug therapies in coronary patients from 22 European countries. Eur J Cardiovasc Prev Rehabil 2009;16(2):121-37.
27. Ordinance No. 6 of June 26/2006 of the Ministry of Health and the Ministry of Environment and Water (Updated State Gazette 58/18.2006 (Bulgarian).
28. Fernández-Somoano A, Llop S, Aguilera I, et al. Annoyance caused by noise and air pollution during pregnancy: associated factors and correlation with outdoor NO2 and benzene estimations. Int J Environ Res Public Health 2015;12:7044-58.
29. Directive 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise. Off J Eur Commun 2002;L189:12-25.
30. SPECTRI. [Acoustic monitoring and specific traffic counting at locations used for the development of "Strategic noise map of Plovdiv agglomeration";] 2015 (in Bulgarian).
31. Dzhambov AM, Dimitrova DD, Turnovska TH. Improving traffic noise simulations using space syntax: preliminary results from two roadway systems. Arh Hig Rada Toksikol 2014;65(3):259-72.
32. Aguilera I, Foraster M, Basagaña X, et al. Application of land use regression modelling to assess the spatial distribution of road traffic noise in three European cities. J Expo Sci Environ Epidemiol 2015;25(1):97-105.
33. Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. Circulation 2005;111(5):697-716.
34. Stevens LA, Levey AS. Measurement of kidney function. Med Clin North Am 2005;89:457-473.
35. Levey AS, Stevens LA, Schmid CH, et al. CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration). A new equation to estimate glomerular filtration rate. Ann Intern Med 2009;150(9):604-12.
36. Matsushita K, Selvin E, Bash LD, et al. Risk implications of the new CKD Epidemiology Collaboration (CKD-EPI) equation compared with the MDRD Study equation for estimated GFR: the atherosclerosis risk in communities (ARIC) Study. Am J Kidney Dis 2010;55(4):648-59.
37. Mihaylov RD. Cystatin C and albuminuria in chronic kidney diseases. [PhD dissertation]. Sofia: Medical University of Sofia; 2014.
38. Eriksson C, Hilding A, Pyko A, et al. Long-term aircraft noise exposure and body mass index, waist circumference, and type 2 diabetes: a prospective study. Environ Health Perspect 2014;122(7):687-94.
39. WHO. Waist circumference and waist-hip ratio: report of a WHO expert consultation. Geneva: World Health Organization; 2008.
40. Norman G. Likert scales, levels of measurement and the 'laws' of statistics. Adv Health Sci Educ Theory Pract 2010;15(5):625-32.
41. Ndrepepa A, Twardella D. Relationship between noise annoyance from road traffic noise and cardiovascular diseases: a meta-analysis. Noise Health 2011;13:251-9.
42. Zijlema W, Cai Y, Doiron D, et al. Road traffic noise, blood pressure and heart rate: pooled analyses of harmonized data from 88,336 participants. Environ Res 2016;151:804-13.
43. Hardy ST, Loehr LR, Butler KR, et al. Reducing the blood pressure-related burden of cardiovascular disease: impact of achievable improvements in blood pressure prevention and control. J Am Heart Assoc 2015;4(10):e002276.
44. Hermida RC, Moya A, Crespo JJ, et al. Asleep blood pressure is an independent predictor of cardiovascular events: the Hygia project. J Heart Disease 2014;11:36.
45. Fatmi Z, Coggon D. Coronary heart disease and household air pollution from use of solid fuel: a systematic review. Br Med Bull 2016;118(1):91-109.
46. Haroun MK, Jaar BG, Hoffman SC, et al. Risk factors for chronic kidney disease: a prospective study of 23,534 men and women in Washington County, Maryland. J Am Soc Nephrol 2003;14(11):2934-41.
47. McMullan CJ, Curhan GC, Forman JP. Association of short sleep duration and rapid decline in renal function. Kidney Int 2016;89(6):1324-30.

48. Schmieder RE. The influence of aircraft noise exposure on renal hemodynamic in healthy individuals (LÄRM). University of Erlangen-Nürnberg Medical School. Available at https://clinicaltrials.gov/ct2/show/record/NCT02783456.
49. Morley DW, Gulliver J. Methods to improve traffic flow and noise exposure estimation on minor roads. Environ Pollut 2016;216:746-54.

Экспозиция шума и его воздействие на кровяное давление и функцию почек у пациентов с гипертонией и сердечно-сосудистыми заболеваниями

Ангел М. Джамбов[1], Мария П. Токмакова[2,3], Пенка Д. Гацева[1], Николай Г. Здравков[2,3], Долина Г. Генчева[3], Невена Г. Иванова[4,5], Красимир И. Карастанев[2,3], Стефка В. Владева[6,7], Александр Т. Дончев[5], Светлан М. Дерменджиев[8]

[1] Кафедра гигиены и экомедицины, Факультет общественного здоровья, Медицинский университет - Пловдив, Пловдив, Болгария
[2] Секция кардиологии, Первая кафедра внутренних болезней, Факультет медицины, Медицинский университет- Пловдив, Пловдив, Болгария
[3] Клиника кардиологии, Университетская больница „Св. Георги", Пловдив, Болгария
[4] Кафедра урологии и общей медицины, Факультет медицины, Медицинский университет-Пловдив, Пловдив, Болгария
[5] МБАЛ „Св. Каридад", Пловдив, Болгария
[6] Медицинский колледж, Медицинский университет - Пловдив, Пловдив, Болгария
[7] Клиника эндокринологии и метаболических нарушений, Университетская больница „Каспела", Пловдив, Болгария
[8] Секция профессиональных заболеваний и токсикологии, Вторая кафедра внутренних болезней, Факультет медицины, Медицинский университет - Пловдив, Пловдив, Болгария

Адрес для корреспонденции: Ангел М. Джамбов, Кафедра гигиены и экомедицины, Факультет общественного здоровья, Медицинский университет-Пловдив, бул. „Васил Априлов" № 15А, Пловдив, 4002, Болгария
E-mail: angelleloti@gmail.com
Тел: +359 32 580 562

Дата получения: 23 января 2017
Дата приемки: 22 марта 2017
Дата онлайн публикации: 28 апреля 2017
Дата публикации: 29 сентября 2017

Ключевые слова: экспозиция шума, шум дорожного движения, дискомфорт от шума, клубочковая фильтрация, загрязнение воздуха

Введение: Шум дорожного движения (ШДД) является фактором риска для сердечно-сосудистых заболеваний и гипертонии; вопреки тому, в сравнительно небольшой части исследований прослеживается связь между ним, кровяным давлением (КД) и функцией почек у пациентов с предшествующими ССЗ.

Цель: Целью данного исследования является установление воздействия экспозиции ШДД в жилых помещениях на КД и функцию почек среди пациентов с ССЗ из Пловдивского региона.

Методы: В исследовании приняли участие 127 пациентов с ишемической болезнью сердца и/ или гипертонией из трёх заведений специализированной медицинской помощи в городе Пловдиве (март-май 2016). Были рассмотрены медицинский анамнез пациентов, медицинская документация и медицинский режим и были исследованы кровяное давление и антропометрические данные. Анализы крови были исследованы на содержание креатинина, общего холестирина и глюкозы в крови. Участники также заполнили анкету. Скорость клубочковой фильтрации была измерена с использованием формулы эпидемиологии хронической болезни почек (Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI)). Всех участников опросили на предмет ощущения дискомфорта, вызванного различными источниками шума в жилых помещениях, а проживающие в городе Пловдиве (n = 132) были локализованы на карте экспозиции шума индикаторами Lдень и Lночь. Было исследовано влияние экспозиции шума на систолическое кровяное давление (СКД), диастолическое кровяное давление (ДКД) и была установлена расчётная скорость клубочковой фильтрации (eGFR) с использованием смешанных линейных моделей.

Результаты: Дискомфорт от шума дорожного движения ассоциируется с более высоким СКД в общей выборке. Другие индикаторы шума связаны с незначительным повышением СКД и понижением eGFR. Воздействие Lдень

Folia Medica

Образец цитирования:
Dzhambov AM, Tokmakova MP, Gatseva PD, Zdravkov NG, Gencheva DG, Ivanova NG, Karastanev KI, Vladeva SV, Donchev AT, Dermendzhiev SM. Community noise exposure and its effect on blood pressure and renal function in patients with hypertension and cardiovascular disease.
Folia Medica 2017;59(3):344-356.
doi: 10.1515/folmed-2017-0045

было более значительным среди пациентов с предшествующими ишемической болезнью сердца/ инфарктом, диабетом, ожирением, прекративших приём блокаторов кальциевых каналов и использующих твёрдое топливо/ газ в домах. Lночь имел более сильный эффект среди тех, кто не принимает статины и среди тех, кто спят в спальных помещениях с шумным фасадом, обитают жилые помещения с тихим фасадом и проводят большую часть времени дома. Повышение при Lдень было ассоциировано со значительным понижением eGFR среди мужчин, пациентов с ишемической болезнью сердца/ инфарктом и проживающих в местах с более низким уровнем загрязнения воздуха. В отношении Lночь, наблюдалось значительное изменение эффекта в соответствии с индикаторами пола, диабета, ожирения и времени, проведённого дома. В некоторых подгруппах эффекты ШДД являлись статистически значимыми.

Заключения: Учитывая, что общие факторы риска прогрессирования сердечно-сосудистых заболеваний не могут контролироваться в достаточной степени на индивидуальном уровне, изменение в экологической среде с целью понижения экспозиции шума в жилых помещениях может привести к более успешному контролированию кровяного давления и функции почек среди пациентов с ССЗ.