ORIGINAL ARTICLE

Blood Pressure and Arterial Stiffness in Association With Aircraft Noise Exposure: Long-Term Observation and Potential Effect of COVID-19 Lockdown

Wiktoria Wojciechowska[ID],* Andrzej Januszewicz,* Tomasz Drożdż, Marta Rojek, Justyna Bączalska[ID], Michał Terlecki[ID], Karol Kurasz, Agnieszka Olszanecka[ID], Mikołaj Smólski, Aleksander Prejbisz, Piotr Dobrowolski[ID], Tomasz Grodzicki, Tomasz Hryniewiecki, Reinhold Kreutz[ID], Marek Rajzer[ID]

ABSTRACT: In a cross-sectional analysis of a case-control study in 2015, we revealed the association between increased arterial stiffness (pulse wave velocity) and aircraft noise exposure. In June 2020, we evaluated the long-term effects, and the impact of a sudden decline in noise exposure during the coronavirus disease 2019 (COVID-19) lockdown, on blood pressure and pulse wave velocity, comparing 74 participants exposed to long-term day-evening-night aircraft noise level >60 dB and 75 unexposed individuals. During the 5-year follow-up, the prevalence of hypertension increased in the exposed (42% versus 59%, *P*=0.048) but not in the unexposed group. The decline in noise exposure since April 2020 was accompanied with a significant decrease of noise annoyance, 24-hour systolic (121.2 versus 117.9 mm Hg; *P*=0.034) and diastolic (75.1 versus 72.0 mm Hg; *P*=0.003) blood pressure, and pulse wave velocity (10.2 versus 8.8 m/s; *P*=0.001) in the exposed group. Less profound decreases of these parameters were noticed in the unexposed group. Significant between group differences were observed for declines in office and night-time diastolic blood pressure and pulse wave velocity. Importantly, the difference in the reduction of pulse wave velocity between exposed and unexposed participants remained significant after adjustment for covariates (−1.49 versus −0.35 m/s; *P*=0.017). The observed difference in insomnia prevalence between exposed and unexposed individuals at baseline was no more significant at follow-up. Thus, long-term aircraft noise exposure may increase the prevalence of hypertension and accelerate arterial stiffening. However, even short-term noise reduction, as experienced during the COVID-19 lockdown, may reverse those unfavorable effects. (***Hypertension**. 2022;78:325–334. DOI: 10.1161/ HYPERTENSIONAHA.121.17704.) • **Data Supplement**

Key Words: aircraft ■ arterial ■ arterial stiffness ■ COVID-19 ■ hypertension

According to the newest European Environment Agency Report published in 2020, over 4 million European Union citizens are exposed to aircraft noise on a level exceeding 55 dB L_{DEN} (long-term day-evening-night noise level).[1] L_{DEN} values above 55 dB are associated with adverse health effects and are also higher than the upper limit recommended by the current World Health Organization guidelines[2] for the European region for all types of environmental noise. Arterial hypertension is well recognized as one of the negative health consequences of environmental noise.[3–7] Stress reaction to environmental noise is considered to be a primary causal link to hypertension development.[8–10] Nocturnal noise exposure is yet more relevant for cardiovascular disorders, including hypertension, than exposure during daytime.[11] Noise annoyance, along with the noise exposure level, has been shown to increase the risk of hypertension and cardiovascular disorders.[12,13] Aircraft noise is perceived as the most annoying and sleep-disturbing among all transportation noise sources.[14] In a meta-analysis performed by Babisch and van Kamp,[12] each increase in the aircraft noise level L_{DEN} by 10 dB increased the

Novelty and Significance

What is New?

- The blood pressure and arterial stiffness decline was documented as a consequence of aircraft noise reduction during the coronavirus disease 2019 (COVID-19) lockdown.

What is Relevant?

- Although long-term aircraft noise exposure increases arterial hypertension prevalence and arterial stiffness,

even short-term aircraft noise reduction may substantially reduce blood pressure and improve arterial compliance.

Summary

These findings confirm the relationship between aircraft noise exposure and development of arterial hypertension, as well as the potential reversal effect of environmental noise limitations in this regard.

Nonstandard Abbreviations and Acronyms

ABPM	ambulatory BP measurement
BMI	body mass index
BP	blood pressure
COVID-19	coronavirus disease 2019
DBP	diastolic BP
PE	parameter estimate
PWV	pulse wave velocity
SBP	systolic BP

relative risk of arterial hypertension by 13%.[12] Conversely, however, we cannot assume that a corresponding reduction of 10 dB in aircraft noise exposure can also reduce the risk of hypertension. This is based entirely on the fact that studies addressing this question have not yet been conducted. Accordingly, the potential impact of a reduction in aircraft noise exposure on arterial stiffness in people previously exposed to increased aircraft noise is also unknown.

In a previous cross-sectional case-control study in 2015, we revealed an increase in blood pressure (BP) and arterial stiffness as determined by carotid-femoral pulse wave velocity (PWV) in individuals exposed to increased aircraft noise levels.[15] The aim of this study was to evaluate the long-term effects of aircraft noise exposure after 5 years and the potential impact of an unexpected sudden decline in aircraft noise exposure. The latter resulted from the substantial decrease in aircraft traffic since the beginning of the coronavirus disease 2019 (COVID-19) pandemic.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Population

We previously conducted in 2015 a population-based case-control study, to compare BP, arterial stiffness, and cardiac organ damage in a group of individuals exposed to aircraft noise (N=101), with control subjects unexposed to increased aircraft noise (N=100).[15] Individuals living in 2 suburban areas of Krakow, Poland, were examined to obtain an equal number of participants exposed and unexposed to aircraft noise.[15] The 2 study areas were selected based on the acoustic maps, in which we identified an area with high aircraft noise exposure (exceeding 60 dB) near the Krakow John Paul II airport and an area with low exposure (below 55 dB) of L_{DEN}.[16,17] These cutoff levels were consistent with the European environmental guidelines for evaluating the health effects of noise.[17]

Data on aircraft noise exposure during follow-up were obtained from the obligatorily collected noise-monitoring data for Krakow airport, provided by the Chief Inspectorate of Environmental Protection. The detailed study protocol was previously reported.[15] Inclusion criteria were age between 40 and 65 years, which was considered optimal for assessing hypertension-mediated organ damage, and time of residence in the given area (minimum of 3 years). The exclusion criteria were coronary artery disease, stroke, heart failure, chronic kidney disease, respiratory disease, liver disease, and deafness or serious hearing loss. All participants from the original investigation in 2015 were invited for a follow-up evaluation in June 2020, for assessment by the same study protocol. Finally, 74 participants in exposed and 75 in the unexposed group were available for re-evaluation in 2020 (dropout causes are described in Figure S1 in the Data Supplement). The study was conducted according to the Helsinki declaration for Investigation of Human Subjects. The Jagiellonian University Ethics Committee approved the study protocol. All participants submitted a written informed consent.

BP Measurement

The study was conducted in an outpatient clinic of the University Hospital in Krakow during one visit. Office and 24-hour ambulatory BP measurements (ABPM) were performed during the baseline visit and follow-up visit, as previously reported, using OMRON M5-I and SpaceLabs 90207 devices. ABPM measurements were taken every 15 minutes during the day (06:00–22:00 hours) and every 20 minutes during the night (22:00–06:00 hours). Hypertension was defined based on a

prior diagnosis, or use of antihypertensive treatment, or elevated office systolic BP (SBP) or diastolic BP (DBP) values ≥140 or ≥90 mm Hg or elevated 24-hour SBP ≥130 or DBP ≥80 mm Hg during ABPM.

PWV Measurements

We assessed carotid-femoral PWV using a SphygmoCor device (AtCor Medical Pty Ltd, West Ryde, New South Wales, Australia) running software Version 7.1 at baseline and Version XCEL at follow-up. The measurements were performed in agreement with the expert consensus document on the measurement of aortic stiffness in daily practice using PWV.[18]

Echocardiographic Measurements

Echocardiographic measurements were obtained by 2 examiners using the Vivid 7 and Vivid 9 ultrasound devices (General Electric Healthcare, Milwaukee, WI), at baseline and at follow-up, respectively. The examination protocol was in agreement with European Society of Cardiology recommendations.[19] Relevant functional and structural parameters of the left ventricle and left atrium were determined as reported previously.[15]

Other Measurements

We used a standardized questionnaire to obtain information on the medical history, including concomitant diseases, smoking and drinking habits, and use of medications.

Additionally, we evaluated the subjective noise annoyance and sleep quality. Noise annoyance was evaluated using a 3-point scale from 0 to 2, where 0 indicated lack of annoyance and 2 indicated maximal annoyance. The sleep quality assessment included the following self-reported parameters: difficulties with falling asleep, awakening during the night and fatigue during the day as described by Thorpy.[20] In participants with presence of all of the above-mentioned parameters, insomnia was defined according to the classification of sleep disorders.[20] Additionally, the presence of snoring and diagnosed sleep apnea, and the duration of overnight sleep, were obtained in the questionnaire.

To explore the potential influence of COVID-19 lockdown on studied parameters, we introduced additional items into the questionnaire by asking about lifestyle changes caused by the lockdown in the opinion of the participants (analyzed parameters listed in Table S1).

Statistical Methods

For database management and statistical analysis, we used SAS software, Version 9.2 (SAS Institute, Cary, NC). For comparison of means and proportions, we applied a *t* test for paired observations and the χ^2-statistic, respectively, or Wilcoxon Signed Rank test, for nonparametric data. For independent group comparison, a 2-sample *t* test or Wilcoxon Rank-sum test for parametric and nonparametric types of test were used. The differences between groups exposed and unexposed to aircraft noise in BP changes were assessed using ANOVA for repeated measurements in a PROC GLM procedure with adjustment for age, gender, changes in body mass index (BMI), and antihypertensive treatment in one model; an additional model was used with the same covariates in which baseline BP values were also implemented. The analysis of differences between exposed and unexposed to aircraft noise groups in PWV decrease were adjusted for age, gender, changes in BMI, 24-hour SBP and DBP, antihypertensive treatment, and baseline PWV. To explore the potential influence of a lockdown on BP values, we assessed the cross-sectional association between BP and age among participants without antihypertensive treatment at baseline. Based on obtained parameter estimates (PE; PE) and time of follow-up (5.5 years), the expected BP of each subject (without antihypertensive treatment at the follow-up observation) was calculated as baseline DBP+5.5×PE. We tested the null hypothesis of no differences between the regression slopes of expected and measured values at the follow-up visit BP on age, using multivariable ANOVA in the PROC GLM procedure of the SAS package. In all analyses, statistical significance was a *P* value of 0.05 or less on 2-sided tests.

RESULTS

During the follow-up period between 2015 and March 2020, the exposure to aircraft noise remained constant in the exposed group. In contrast, a marked decrease in the average aircraft noise level occurred in April 2020, resulting in a reduction from 61.7 to 47 dB during the day and from 55.4 to 43.4 dB during the night, as compared with April 2019 (detailed data are summarized in Figure S2). Thus, starting from April 2020, the formerly exposed group was exposed to aircraft noise levels similar to the control group. In the latter, the environmental noise exposure conditions did not change in the corresponding residential area and remained below <55 dB throughout the entire follow-up period between 2015 and 2020 (mean of 5.5 years).

Characteristics of Participants

Table 1 shows the characteristics of participants for both the exposed (N=75) and unexposed (N=74) groups at baseline and at the follow-up visit. The data for the overall cohort (N=100 and N=101, respectively) at the baseline visit are also shown for comparison.

During follow-up, a significant increase in BMI, the prevalence of hypertension and the percentage of patients treated with antihypertensive medications was observed in the group exposed to aircraft noise (Table 1).

The incidence of arterial hypertension in the exposed group tended to be higher. The average number of drugs used by the patient and the particular drug class use did not change significantly during follow-up. The exposed participants at follow-up visit reported significantly lower noise annoyance (*P*=0.006) in comparison to the baseline visit, but still had higher levels than the unexposed group (*P*=0.001, Table 1). During follow-up, no significant changes were revealed in the unexposed group in comparison to the baseline visit (Table 1).

Table 1. Characteristics of Participants by Study Phase and Noise Exposure Area

Parameters	All participants at baseline			Participants with follow-up					
				Unexposed			Exposed		
	Unexposed	Exposed	P value	Baseline	Follow-up	P value*	Baseline	Follow-up	P value*
Number of participants	100	101		75	75		74	74	
Age, y	53.6 (8.5)	53.4 (8.1)	0.91	53.4 (8.2)	58.6 (8.3)	NA	53.3 (7.2)	58.4 (7.1)	NA
Female, n (%)	71 (71)	65 (64.4)	0.39	52 (69.3)			50 (67.6)		
Body mass index, kg/m²	27.7 (4.7)	27.2 (4.3)	0.41	28.2 (4.9)	28.7 (4.4)	0.06	27.0 (4.4)	28.2 (6.0)	0.01
Current smoking	10 (10)	14 (13.9)	0.53	4 (5.3)	5 (6.7)	0.73	5 (6.7)	6 (8.1)	0.62
Alcohol intake	23 (23)	31 (30.7)	0.28	17 (22.7)	18 (24)	0.84	21 (28.4)	23 (31)	0.85
Regular physical activity, times a week	3 (0.4)	3.8 (0.42)	0.61	2.7 (0.53)	3.19 (0.42)	0.39	2.7 (0.5)	3.8 (0.42)	0.80
Hypercholesterolemia	28 (28)	34 (33.7)	0.47	24 (32)	32 (43)	0.11	25 (33.9)	30 (40.5)	0.49
Hypertension	50 (50)	53 (52.5)	0.83	30 (40)	37 (49)	0.32	31 (42)	44 (59)	0.048
Diabetes	8 (8)	8 (7.9)	0.99	5 (6.7)	10 (13.3)	0.27	5 (6.7)	5 (6.7)	1.0
Antihypertensive medication use, n (%)	42 (42)	39 (38.6)	0.73	27 (36.0)	34 (45.6)	0.32	23 (31.1)	36 (48.6)	0.039
Number of antihypertensive drugs	1.9 (1.5)	2.1 (0.9)	0.39	2.03 (0.8)	2.0 (0.8)	0.48	2.0 (0.8)	1.9 (0.9)	0.36
Time of residence in the selected area, y	34.8 (19.9)	35.7 (17.3)	0.74	35.7 (18.0)	37.3 (20.4)	0.10	36.6 (16.9)	38.8 (16.3)	0.62
Time spent at home within 24 h	17 (5.0)	16.3 (4.8)	0.31	17.5 (5.0)	16.4 (5.7)	0.62	16.3 (4.9)	16.4 (6.3)	0.87
Aircraft noise annoyance	0.01 (0.01)	1.37 (0.06)	<0.001	0.016 (0.02)	0.063 (0.03)	0.37	1.36 (0.8)†	1.06 (0.09)‡	0.006

Values are mean (SD), numbers of subjects (%), aircraft noise annoyance and regular physical activity as mean (SE). One alcohol dose (50 mL vodka/cognac/liqueur or 150 mL wine or 250 mL beer) per week. Regular physical activity means 40 minutes once a week. P value denotes significance of between group differences for all participants at baseline. NA - not applicable.

*P value: significance of changes from baseline to follow-up within unexposed or exposed group in participants with follow-up.

†P<0.05 for differences between unexposed and exposed participants at baseline.

‡P<0.05 for differences between unexposed and exposed participants at follow-up visit.

Sleep Characteristics

Table 2 summarizes the results obtained with the sleep quality questionnaire. The average sleep duration in both groups was similar, with about 7 hours both at baseline and during follow-up. Difficulties in falling asleep were more prevalent in the group exposed to aircraft noise compared with the control group at baseline (P=0.02). A significant increase in difficulty falling asleep at the follow-up visit was observed in both studied groups (Table 2). At baseline, >40% of the subjects in the exposed group reported awakenings at night, compared with only 24% in the unexposed group. Of note, this difference between groups in awakening during the night was no longer significant at the follow-up visit during the lockdown period. Similarly, the significantly higher prevalence of reported fatigue during the day observed in the exposed group at baseline was not maintained at the follow-up visit (Table 2).

The prevalence of insomnia was higher at baseline in the exposed group compared with participants

Table 2. Sleep Quality Parameters and Insomnia in Subjects Exposed or Unexposed to Aircraft Noise and Changes During 5-Years of Follow-Up

Parameters	Unexposed, N=75			Exposed, N=74		
	Baseline	Follow-up	P value	Baseline	Follow-up	P value
Difficulty falling asleep	9 (12.0)	18 (24.0)	0.038	20 (27.0)*	27 (36.5)†	0.008
Awakening during the night	18 (24.0)	30 (40.0)	0.06	32 (43.2)*	44 (59.4)	0.11
Fatigue during the day	11 (14.7)	37 (49.3)	0.61	32 (43.2)*	42 (56.7)	0.66
Insomnia	5 (6.7)	16 (21.3)	0.04	12 (16.2)*	18 (24.3)	0.21
Snoring	20 (26.7)	36 (48.0)	0.009	26 (35.1)	31 (41.9)	0.026
Sleep apnea	2 (2.7)	3 (4.0)	0.99	3 (4.0)	5 (6.8)	0.9
Average sleep duration, h	7.0 (0.8)	7.0 (1.0)	0.88	7.0 (0.8)	7.1 (1.2)	0.43

Values are expressed as number (%), total score categories mean (SE), average sleep duration mean (SD). P value denotes significance of changes from baseline to follow-up in unexposed or exposed group in participants with follow-up.

*P<0.05 for differences between unexposed and exposed participants at baseline.

†P<0.05 for differences between unexposed and exposed participants at follow-up visit.

unexposed to aircraft noise, but this difference disappeared at the follow-up visit because the unexposed participants experienced a significant increase in insomnia frequency ($P=0.04$; Table 2).

Lockdown Lifestyle Changes

Self-reported lifestyle parameters and working habits during the COVID-19 lockdown period did not change significantly as compared with the period before the lockdown in 2020, in either group (Table S1).

BP Phenotypes and Changes During Follow-Up

The baseline and follow-up BP measurements are shown in Table 3. During the baseline visit, participants in the exposed group had higher office and night-time DBP, as well as higher central SBP and DBP, compared with the unexposed group. At follow-up, significant group differences in BP phenotypes were only detected for central SBP. At the follow-up visit, a substantial drop in BP was observed in participants from both groups. In the unexposed group, 24-hour SBP, daytime SBP, and daytime DBP were lower at follow-up visit than during the initial measurements ($P\leq0.013$). In participants exposed to aircraft noise, a significant decrease of DBP was observed for all measurement methods ($P\leq0.022$), while a decrease of SBP was significant in averaged 24-hour values and during daytime measurements ($P\leq0.034$; Table 3).

Unadjusted comparison of changes in BP parameters from baseline to follow-up are summarized in Table 3. The magnitude of the DBP decreases at follow-up visit in office, central and night-time values, as compared with the baseline, was significantly greater in the exposed group also after adjustment for age, gender, BMI, and antihypertensive treatment (Figure S3). Introduction of baseline BP values in the adjusted analysis ameliorated this difference resulting in no significant group differences. There was no significant relationship between BP changes and the decline in noise annoyance. The heart rate decreased in the comparison between baseline and follow-up in both groups during office BP measurements (unexposed group: 71.4 versus 66.8 beats per minute, $P=0.001$; exposed group: 71.5 versus 66.1 beats per minute, $P=0.001$). However, ABPM heart rates were comparable (unexposed group: 62.8 versus 62.2 beats per minute, $P=0.66$; exposed group 63.7 versus 62.7 beats per minute, $P=0.16$).

Explorative Analysis of Differences Between Expected and Measured BP in Untreated Participants

From regression modeling of BP phenotypes with age at baseline, the presumable yearly increase of BP in untreated participants was obtained, and the estimated BP values at 5.5 years follow-up were calculated. The latter were compared with the actually measured BP values. This analysis revealed that, in the exposed participants without antihypertensive treatment at follow-up (n=38), the observed office DBP was significantly lower than the expected DBP (DBP [least squares means]: expected 87.6 mm Hg, observed 82.7 mm Hg; $P=0.048$). In contrast, in untreated participants of the unexposed group (n=41), there was no difference between the expected and observed DBP values (DBP [least squares means]: expected 78.7 mm Hg, observed 77.8 mm Hg; $P=0.61$, Figure S4). No differences were detected for other BP phenotypes.

Pulse Wave Velocity

Table 4 shows the mean values of PWV in studied groups at baseline and follow-up visits with changes observed in PWV during follow-up. At baseline, PWV was significantly higher in the exposed group as compared with the unexposed group (Table 4), with a mean value exceeding the normal range of PWV; that is, 10 m/s. At the follow-up visit, PWV was similar with 8.8 m/s in both groups due to a significant decrease in the exposed group. The differences in the reduction of PWV remained significant after adjustment for age, gender, changes in BMI, averaged 24-hour SBP and DBP, antihypertensive treatment, and baseline PWV (estimate: −1.49 versus −0.35 m/s; $P=0.017$).

The univariate linear regression analyses between PWV and age among untreated participants are shown in Figure. Of interest, in the group with exposure to aircraft noise at baseline no significant association was detected (PE=0.01 m/s/y; $P=0.71$), while in the unexposed group a strong, association was found (PE=0.13 m/s/y; $P<0.001$). At the follow-up visit, the significant association between PWV and age became evident (PE=0.15 m/s/y; $P<0.001$) in the exposed group and was comparable to the unexposed group (PE=0.12 m/s/y; $P=0.003$). However, the mean PWV in untreated participants remained still higher in the exposed than in the unexposed group (8.5±1.69 m/s versus 7.8±1.51 m/s; $P=0.03$, Figure).

Echocardiographic Parameters

Table 4 summarizes the data obtained by echocardiographic assessment. There were no between group differences in left ventricular mass index at baseline and follow-up visit.

In the exposed group, a significant, albeit small, decrease in the ratio of early and late diastolic mitral peak velocity (E/A) was observed at the follow-up visit. The average values of left atrium volume index at follow-up were significantly higher as compared with the

Table 3. Office, Central and Ambulatory Blood Pressure in Subjects Exposed or Unexposed to Aircraft Noise and Changes During 5-Years of Follow-Up

mm Hg	Unexposed, N=75				Exposed, N=74			
	Baseline	Follow-up	*P* value	Δ	Baseline	Follow-up	*P* value	Δ
Office BP								
Systolic pressure	138.0 (15.8)	136.4 (16.8)	0.43	−1.6	142.3 (18.5)	140.5 (18.4)	0.44	−1.8
Diastolic pressure	80.1 (8.0)	80.1 (9.7)	0.97	0.0	87.5 (11.6)*	82.2 (8.1)	0.001	−5.3‡
Pulse pressure	58.0 (11.9)	56.3 (12.5)	0.28	−1.7	54.8 (12.4)	58.2 (15.3)	0.023	3.4‡
Central BP								
Systolic pressure	124.1 (15.5)	122.7 (18.8)	0.55	−1.4	130.4 (17.5)*	128.7 (15.7)†	0.46	−1.7
Diastolic pressure	79.9 (8.0)	81.1 (9.9)	0.32	1.2	86.8 (11.4)*	83.2 (8.4)	0.022	−3.6‡
Pulse pressure	44.1 (11.4)	43.2 (11.5)	0.49	−0.9	43.6 (12.2)	45.5 (12.2)	0.19	1.9
ABPM 24-hour								
Systolic pressure	120.9 (9.5)	117.5 (10.4)	0.011	−3.4	121.2 (14.6)	117.9 (12.1)	0.034	−3.3
Diastolic pressure	72.8 (6.4)	71.7 (7.4)	0.14	−1.1	75.1 (8.9)	72.0 (7.6)	0.003	−3.1
Pulse pressure	48.1 (6.5)	45.8 (7.8)	0.01	−2.3	46.1 (10.0)	45.8 (9.5)	0.81	−0.3
ABPM daytime								
Systolic pressure	127.1 (10.3)	121.9 (11.0)	0.012	−5.2	126.9 (15.2)	122.3 (12.6)	0.008	−4.6
Diastolic pressure	78.9 (9.3)	75.7 (7.8)	0.013	−3.2	79.3 (8.9)	75.9 (8.6)	0.002	−3.4
Pulse pressure	48.3 (9.9)	46.3 (7.8)	0.12	−1.4	47.6 (10.1)	46.4 (9.5)	0.17	−1.2
ABPM night-time								
Systolic pressure	107.9 (9.3)	107.6 (11.3)	0.61	−0.3	110.4 (15.8)	108.1 (12.7)	0.23	−2.3
Diastolic pressure	62.7 (6.5)	63.2 (6.9)	0.72	0.5	66.2 (9.9)*	63.8 (6.9)	0.014	−2.4‡
Pulse pressure	45.1 (7.2)	44.5 (8.1)	0.34	−0.6	44.2 (10.2)	44.3 (10.5)	0.43	0.1

Values are mean (SD). *P* value denotes significance of changes from baseline to follow-up within unexposed or exposed group in participants with follow-up; and Δ, changes of BP parameters calculated as follow-up BP minus baseline BP. ABPM indicates ambulatory blood pressure measurement; and BP, blood pressure.

*$P<0.05$ for differences between unexposed and exposed participants at baseline.

†$P<0.05$ for differences between unexposed and exposed participants at follow-up.

‡$P<0.05$ for differences between the unexposed and exposed groups in BP phenotypes changes (Δ) during follow-up.

baseline in both groups. Moreover, the magnitude of changes in the examined parameters over time did not differ between groups.

DISCUSSION

Transportation noise, particularly aircraft noise, is increasingly recognized as an important risk factor for arterial hypertension, cardiovascular organ damage, stroke, and coronary artery disease.[21,22] An increase in arterial stiffness, as reflected by an elevated PWV after adjustments for other covariates in subjects exposed to aircraft noise was an important finding in the baseline cross-sectional analysis of our case-control study conducted in 2015.[15] In the current follow-up analysis, we investigated whether a short-term reduction in aircraft noise exposure caused by the COVID-19 lockdown may have beneficial effects on the increases in BP and arterial stiffness, as previously reported.[23] First of all, we demonstrated that long-term exposure for 5 years to increased (>60 dB) aircraft noise levels does indeed increase the prevalence of hypertension in our cohort. However, at the time of re-examination during the follow-up visit, participants in the exposed group had been unloaded from increased

aircraft noise for about 4 months due to the significant air traffic reduction due to COVID-19. Instead of progression in arterial stiffness, that is, a further increase in PWV, as a result of aging and the 5 years aircraft noise exposure,[24] we detected a normalization of the elevated at baseline PWV. In addition, the formerly seen decoupling of the association between age and PWV at baseline was also restored at the follow-up visit in untreated participants of the exposed group, in parallel with the normalization of PWV. Along with the reduction in aircraft noise in the exposed group a significant reduction in aircraft-noise-induced annoyance was observed in the participants, which provides a possible mechanistic link.[23] Hence, the study by Van Gerven et al[25] assessed the relation between age and self-reported annoyance from environmental noise (including aircraft noise). The results revealed an inverted U-shaped pattern, where the largest number of highly annoyed individuals was found in the middle-aged segment of the sample (peaking at around 45 years).[25] These results can be extrapolated to our group (mean age of 53 years) and explain the high annoyance level and subsequently higher PWV in this group observed at baseline. Consequently, the aircraft noise decrease and decline in annoyance at follow-up

Table 4. Pulse Wave Velocity and Echocardiographic Parameters in Subjects Exposed or Unexposed to Aircraft Noise and Changes During 5-Years of Follow-Up

Parameters	Unexposed, N=75				Exposed, N=74			
	Baseline	Follow-up	P value	Δ	Baseline	Follow-up	P value	Δ
PWV, m/s	9.3 (1.5)	8.8 (1.8)	0.002	−0.5	10.2 (1.8)*	8.8 (1.7)	0.001	−1.4‡
Echocardiographic parameters								
LVMI, g/m²	100.9 (22.5)	98.7 (23.9)	0.09	−2.2	104.9 (24.6)	100.4 (22.2)	0.36	−4.5
RWT, mm	0.41 (0.07)	0.42 (0.06)	0.12	0.01	0.40 (0.06)	0.43 (0.06)	0.09	0.03
LAVI, mL/m²	24.7 (7.3)	31.9 (6.5)	0.001	7.2	25.6 (7.5)	31.7 (5.2)	0.001	6.1
E′, cm/s	9.1 (3.4)	9.2 (2.9)	0.85	0.1	8.7 (3.0)	9.1 (2.8)	0.20	0.4
E/E′	8.6 (2.6)	8.4 (2.8)	0.88	−0.2	8.6 (2.9)	8.3 (2.6)	0.33	−0.3
E/A	1.07 (0.3)	1.19 (0.31)	0.056	0.12	1.06 (0.35)	1.00 (0.32)	0.032	−0.06

Values are mean (SD). A indicates late diastolic mitral peak flow velocity; E, early diastolic mitral peak flow velocity; E/A, ratio of E to A; E/E′, ratio of E to E′; E′, early diastolic mitral annulus mean (of septal and lateral) velocity; LAVI, left atrial volume index; LVMI, left ventricle mass index; PWV, carotid–femoral pulse wave velocity; and RWT, relative wall thickness. Δ denotes changes in PWV and echocardiographic parameters calculated as follow-up values minus baseline values; and P value, significance of changes from baseline to follow-up within unexposed or exposed group in participants with follow-up.

*$P<0.05$ for differences between unexposed and exposed participants at baseline.

†$P<0.05$ for differences between the unexposed and exposed groups for changes (Δ) in PWV and echocardiographic parameters during follow-up.

may, at least in part, explain the PWV reduction and the restoration of the positive physiological[26] correlation between PWV and age.[27]

Increased PWV as a consequence of aortic stiffening is associated mainly with older age and elevated BP.[27,28] Carotid-femoral PWV as an index of arterial stiffening is a BP-dependent parameter,[26] so changes in PWV should be considered with the changes in BP observed at the follow-up visit. Lower BP values were detected in the exposed group but also in the unexposed group at the follow-up visit. In particular, the magnitude of the decrease in office, central, and ABPM night-time DBP were significantly more pronounced in the group exposed to aircraft noise in comparison to the unexposed group, even after accounting for age, gender, and antihypertensive treatment. Acute hemodynamic changes result in substantial changes in PWV.[29] Thus, lower BP in the exposed group may have contributed to the detected lower PWV at the follow-up visit. Taken together, both

the reduction in aircraft noise–induced annoyance and the drop in BP are likely to be responsible for the decrease in PWV and may share a common pathogenic background. On the contrary, the effect size of mechanisms, for example, as mediated by endothelial dysfunction and neurohumoral activation, on BP and PWV could be different. Earlier, Schmidt et al[9,10] showed that aircraft noise exposure dose-dependently impairs endothelial function and increases adrenaline release, which might have a greater impact on PWV as compared with BP. In addition, Osborne et al[30] showed that noise exposure is related to cardiovascular diseases by a mechanism that begins with increased stress-associated limbic activity and includes arterial inflammation which has been also associated with a higher risk of major adverse cardiovascular events. Activation of amygdala, which is responsible for emotional stress and fear, is also responsible for the anger/annoyance reactions.[30] Neurobiological resilience defined as lower amygdalar activity despite
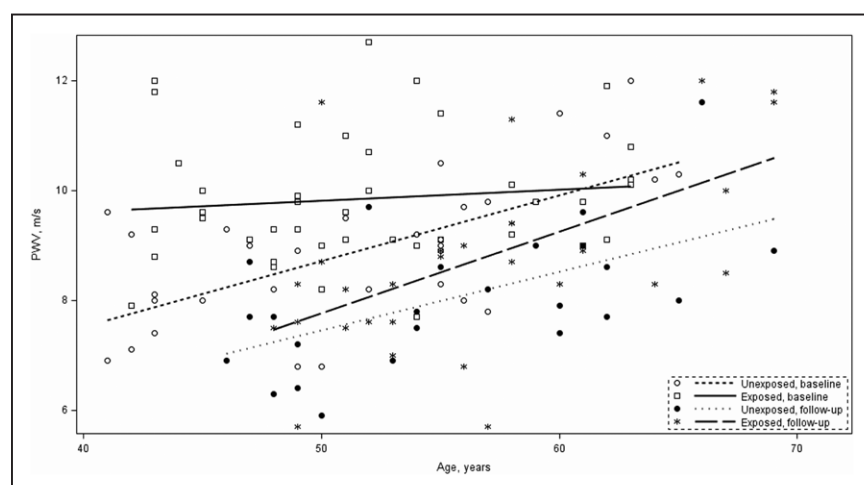

Figure. Unadjusted associations of pulse wave velocity (PWV) with age at baseline, and follow-up in untreated unexposed (Nbaseline=48; Nfollow-up=41) and exposed (Nbaseline =51; Nfollow-up=38) participants.

stress exposure including transportation noise may protect against major adverse cardiac events.[31]

Available human data are consistent with animal experiments demonstrating an increased release of stress hormones (catecholamines and cortisol), endothelial dysfunction, and oxidative stress in aircraft noise-exposed mice.[32] Of note, a recent experimental study in mice indicated that aircraft noise exposure during the sleep phase but not awake phase caused increased BP, endothelial dysfunction, increased markers of vascular/systemic oxidative stress, and inflammation.[33] Mechanistically, this was linked to increased cerebral oxidative stress and downregulation of neuronal nitric oxide synthase in response to noise exposure.[33]

Previous studies showed positive associations between aircraft noise, BP, and arterial hypertension.[5,6,12,34–37] The authors concluded that environmental night-time noise is more likely to cause cardiovascular disease than daytime noise. This is emphasized by the World Health Organization night-time noise guidelines.[38]

The meaningful effect of night-time aircraft noise on arterial hypertension was observed in the cross-sectional multicenter HYENA (hypertension and exposure to noise near airports) study[37] and in the prospective observation of the subset of individuals from that study.[21] In a longitudinal observation of 420 participants, higher aircraft noise exposure during the night significantly associated with the incidence of hypertension.[21]

The above-described data showed a similarity to results obtained in our study; that is, night-time DBP at baseline was significantly higher in participants exposed to noise as compared with unexposed participants. At follow-up in the group of previously exposed participants, DBP decreased substantially mainly due to higher baseline BP values and became similar to DBP in the unexposed group. This could be a consequence of the aircraft noise decline during the COVID-19 lockdown. It appears possible that in participants previously prehypertensive, the development of sustained hypertension and subsequent introduction of antihypertensive treatment could have in fact influence on drop in BP compared with baseline in these individuals. However, our sensitivity analysis in untreated participants showed also a similar tendency towards lower BP levels at follow-up (Table S2 and Figure S4).

Besides the BP reduction in the group previously exposed to aircraft noise, we found (in unadjusted analysis) an increase in the prevalence of arterial hypertension during follow-up. This is in agreement with previous reports showing the long-lasting effect of aircraft noise exposure on hypertension prevalence.[5,36,37]

Among the echocardiographic parameters assessed in our study, left atrium volume index showed a gradual increase during follow-up, while no changes in left ventricular mass index were observed. This lack of progression in left ventricular mass index could be related to overall normal or only slightly elevated BP on both visits in studied groups. The increase in left atrium volume index might be explained by aging.[39] This notion is supported by a recent study showing a significant correlation of the left atrium size with age ($r=0.48$, $P<0.0001$).[39] Moreover, as a potential result of the increased prevalence of hypertension during the 5-year follow-up in the exposed group, the significant decrease of E/A ratio could be noted as an indicator for early LV diastolic dysfunction.

At baseline, participants exposed to aircraft noise had a higher prevalence of insomnia as compared with the unexposed group. At the follow-up visit, the prevalence of insomnia became equal in both groups. This may be partially explained by the observed significant increase of insomnia in the unexposed group as a potential adverse effect of the COVID-19 lockdown.[40]

In this regard, our results are in agreement with work by Voitsidis et al,[41] who described increased prevalence of insomnia in a large Greek population as a result of the COVID-19 pandemic. Therefore, this effect should be expected in both groups of our study. However, in the exposed group the increase in the prevalence of insomnia was not significant, which may be attributable, at least in part, to the observed reduction in aircraft noise exposure.

A few limitations of our study should be taken into consideration. First, we presented data pertaining to a relatively small group of individuals exposed to aircraft noise; however, their BP and cardiovascular parameters were carefully phenotyped. Another shortcoming of our study is the lack of a follow-up investigation with maintained aircraft noise exposure in the exposed group that preceded the current investigation as closely as possible. However, due to the unforeseen lockdown caused by the COVID-19 pandemic this was not feasible. Looking into the future, it remains to be seen when and whether a future follow-up study with similar aircraft noise exposure levels in the exposed group will be conducted by us, since the return to the previous normal air traffic intensity in the post-COVID-19 area appears to be questionable.[40]

In conclusion, long-term aircraft noise exposure increases the prevalence of arterial hypertension. The unprecedented situation with an aircraft noise decline due to the COVID-19 lockdown indirectly confirmed this observation by reversing unfavorable changes of BP and arterial stiffness. Our study supports the importance of noise mitigation strategies for cardiovascular health.

PERSPECTIVES

In this study, we investigated whether the significant noise reduction caused by the COVID-19 lockdown could reverse an unfavorable long-term aircraft noise effect on BP and arterial stiffness. In summary, we revealed that long-lasting exposure to aircraft noise is associated with higher prevalence of arterial hypertension, while during a short-term reduction in noise

exposure due to the COVID-19 lockdown a significant decline in PWV was observed. Moreover, the natural relation of PWV with age previously blunted by the noise influence was restored. Our findings emphasize the importance of noise pollution on BP and arterial stiffening. Whether the noise reduction due to COVID-19 will have an effect on arterial hypertension epidemiology or not remains uncertain, and needs future research. The consecutive waves of the COVID-19 pandemic continue to cause a considerable increase in morbidity and mortality, having a profound effect on numerous aspects of society health and environment worldwide. Thus, the cumulative effect of the COVID-19 pandemic on arterial hypertension is rather unpredictable, and the consequences of the overall traffic and particularly the decrease in aircraft noise are contributing to this complex scenario. Nevertheless, the potential reversal of aircraft noise−induced health side effects during the lockdown could stimulate the discussion with authorities to put more emphasis on the implementation of World Health Organization noise limits. In addition, the same applies to the inclusion of corresponding recommendations into cardiovascular prevention programs.

ARTICLE INFORMATION

REFERENCES

1. Environmental noise in Europe − 2020. European Environment Agency (EEA) Report 22/2019. ISSN 1977-8449. doi: 10.2800/686249
2. *Environmental Noise Guidelines for the European Region*; 2018. https://www.euro.who.int/__data/assets/pdf_file/0008/383921/noise-guidelines-eng.pdf
3. Hansell AL, Blangiardo M, Fortunato L, Floud S, de Hoogh K, Fecht D, Ghosh RE, Laszlo HE, Pearson C, Beale L, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ*. 2013;347:f5432. doi: 10.1136/bmj.f5432
4. Dratva J, Phuleria HC, Foraster M, Gaspoz JM, Keidel D, Künzli N, Liu LJ, Pons M, Zemp E, Gerbase MW, et al. Transportation noise and blood pressure in a population-based sample of adults. *Environ Health Perspect*. 2012;120:50−55. doi: 10.1289/ehp.1103448
5. Rosenlund M, Berglind N, Pershagen G, Järup L, Bluhm G. Increased prevalence of hypertension in a population exposed to aircraft noise. *Occup Environ Med*. 2001;58:769−773. doi: 10.1136/oem.58.12.769
6. Seidler A, Wagner M, Schubert M, Dröge P, Römer K, Pons-Kühnemann J, Swart E, Zeeb H, Hegewald J. Aircraft, road and railway traffic noise as risk factors for heart failure and hypertensive heart disease-A case-control study based on secondary data. *Int J Hyg Environ Health*. 2016;219:749−758. doi: 10.1016/j.ijheh.2016.09.012
7. Sørensen M, Hvidberg M, Hoffmann B, Andersen ZJ, Nordsborg RB, Lillelund KG, Jakobsen J, Tjønneland A, Overvad K, Raaschou-Nielsen O. Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: a cohort study. *Environ Health*. 2011;10:92. doi: 10.1186/1476-069X-10-92
8. Münzel T, Sørensen M, Gori T, Schmidt FP, Rao X, Brook J, Chen LC, Brook RD, Rajagopalan S. Environmental stressors and cardio-metabolic disease: part I-epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies. *Eur Heart J*. 2017;38:550−556. doi: 10.1093/eurheartj/ehw269
9. Schmidt F, Kolle K, Kreuder K, Schnorbus B, Wild P, Hechtner M, Binder H, Gori T, Münzel T. Nighttime aircraft noise impairs endothelial function and increases blood pressure in patients with or at high risk for coronary artery disease. *Clin Res Cardiol*. 2015;104:23−30. doi: 10.1007/s00392-014-0751-x
10. Schmidt FP, Basner M, Kröger G, Weck S, Schnorbus B, Muttray A, Sariyar M, Binder H, Gori T, Warnholtz A, et al. Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults. *Eur Heart J*. 2013;34:3508−314a. doi: 10.1093/eurheartj/eht269
11. Basner M, Müller U, Elmenhorst EM. Single and combined effects of air, road, and rail traffic noise on sleep and recuperation. *Sleep*. 2011;34:11−23. doi: 10.1093/sleep/34.1.11
12. Babisch W, Kamp Iv. Exposure-response relationship of the association between aircraft noise and the risk of hypertension. *Noise Health*. 2009;11:161−168. doi: 10.4103/1463-1741.53363
13. Babisch W, Pershagen G, Selander J, Houthuijs D, Breugelmans O, Cadum E, Vigna-Taglianti F, Katsouyanni K, Haralabidis AS, Dimakopoulou K, et al. Noise annoyance−a modifier of the association between noise level and cardiovascular health? *Sci Total Environ*. 2013;452-453:50−57. doi: 10.1016/j.scitotenv.2013.02.034
14. Miedema HM, Oudshoorn CG. Annoyance from transportation noise: relationships with exposure metrics DNL and DENL and their confidence intervals. *Environ Health Perspect*. 2001;109:409−416. doi: 10.1289/ehp.01109409
15. Rojek M, Rajzer MW, Wojciechowska W, Drożdż T, Skalski P, Pizoń T, Januszewicz A, Czarnecka D. Relationship among long-term aircraft noise exposure, blood pressure profile, and arterial stiffness. *J Hypertens*. 2019;37:1350−1358. doi: 10.1097/HJH.0000000000002060
16. 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 Communities*. 2002; L 189/12. https://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2002:189:0012:0025:EN:PDF
17. The European Parliament and the Council of the European Union. Decision No 1386/2013/EU of the European Parliament and of the Council of 20 November 2013 on a General Union Environment Action Programme to 2020 'Living well, within the limits of our plant'. *Off J Eur Union L 354/17*. Accessed 1 June 2017. https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32013D1386&rid=1
18. Van Bortel LM, Laurent S, Boutouyrie P, Chowienczyk P, Cruickshank JK, De Backer T, Filipovsky J, Huybrechts S, Mattace-Raso FU, Protogerou AD, et al; Artery Society; European Society of Hypertension Working Group on Vascular Structure and Function; European Network for Noninvasive Investigation of Large Arteries. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J Hypertens*. 2012;30:445−448. doi: 10.1097/HJH.0b013e32834fa8b0
19. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;28:1−39.e14. doi: 10.1016/j.echo.2014.10.003
20. Thorpy MJ. Classification of sleep disorders. *Neurotherapeutics*. 2012;9:687−701. doi: 10.1007/s13311-012-0145-6
21. Dimakopoulou K, Koutentakis K, Papageorgiou I, Kasdagli MI, Haralabidis AS, Sourtzi P, Samoli E, Houthuijs D, Swart W, Hansell AL, et al. Is aircraft

noise exposure associated with cardiovascular disease and hypertension? Results from a cohort study in Athens, Greece. *Occup Environ Med.* 2017;74:830–837. doi: 10.1136/oemed-2016-104180

22. Münzel T, Sørensen M, Daiber A. Transportation noise pollution and cardiovascular disease. *Nat Rev Cardiol.* 2021;18:619–636. doi: 10.1038/s41569-021-00532-5

23. Münzel T, Miller MR, Sørensen M, Lelieveld J, Daiber A, Rajagopalan S. Reduction of environmental pollutants for prevention of cardiovascular disease: it's time to act. *Eur Heart J.* 2020;41:3989–3997. doi: 10.1093/eurheartj/ehaa745

24. Münzel T, Schmidt FP, Steven S, Herzog J, Daiber A, Sørensen M. Environmental noise and the cardiovascular system. *J Am Coll Cardiol.* 2018;71:688–697. doi: 10.1016/j.jacc.2017.12.015

25. Van Gerven PW, Vos H, Van Boxtel MP, Janssen SA, Miedema HM. Annoyance from environmental noise across the lifespan. *J Acoust Soc Am.* 2009;126:187–194. doi: 10.1121/1.3147510

26. Parikh JD, Hollingsworth KG, Kunadian V, Blamire A, MacGowan GA. Measurement of pulse wave velocity in normal ageing: comparison of Vicorder and magnetic resonance phase contrast imaging. *BMC Cardiovasc Disord.* 2016;16:50. doi: 10.1186/s12872-016-0224-4

27. Vishram-Nielsen JKK, Laurent S, Nilsson PM, Linneberg A, Sehested TSG, Greve SV, Pareek M, Palmieri L, Giampaoli S, Donfrancesco C, et al; MORGAM Project. Does estimated pulse wave velocity add prognostic information?: MORGAM Prospective Cohort Project. *Hypertension.* 2020;75:1420–1428. doi: 10.1161/HYPERTENSIONAHA.119.14088

28. Baier D, Teren A, Wirkner K, Loeffler M, Scholz M. Parameters of pulse wave velocity: determinants and reference values assessed in the population-based study LIFE-Adult. *Clin Res Cardiol.* 2018;107:1050–1061. doi: 10.1007/s00392-018-1278-3

29. Obata Y, Mizogami M, Singh S, Nyhan D, Berkowitz DE, Steppan J, Barodka V. The effects of hemodynamic changes on pulse wave velocity in cardiothoracic surgical patients. *Biomed Res Int.* 2016;2016:9640457. doi: 10.1155/2016/9640457

30. Osborne MT, Radfar A, Hassan MZO, Abohashem S, Oberfeld B, Patrich T, Tung B, Wang Y, Ishai A, Scott JA, et al. A neurobiological mechanism linking transportation noise to cardiovascular disease in humans. *Eur Heart J.* 2020;41:772–782. doi: 10.1093/eurheartj/ehz820

31. Dar T, Osborne MT, Abohashem S, Abbasi T, Choi KW, Ghoneem A, Naddaf N, Smoller JW, Pitman RK, Denninger JW, et al. Greater neurobiological resilience to chronic socioeconomic or environmental stressors

associates with lower risk for cardiovascular disease events. *Circ Cardiovasc Imaging.* 2020;13:e010337. doi: 10.1161/CIRCIMAGING.119.010337

32. Münzel T, Daiber A, Steven S, Tran LP, Ullmann E, Kossmann S, Schmidt FP, Oelze M, Xia N, Li H, et al. Effects of noise on vascular function, oxidative stress, and inflammation: mechanistic insight from studies in mice. *Eur Heart J.* 2017;38:2838–2849. doi: 10.1093/eurheartj/ehx081

33. Kröller-Schön S, Daiber A, Steven S, Oelze M, Frenis K, Kalinovic S, Heimann A, Schmidt FP, Pinto A, Kvandova M, et al. Crucial role for Nox2 and sleep deprivation in aircraft noise-induced vascular and cerebral oxidative stress, inflammation, and gene regulation. *Eur Heart J.* 2018;39:3528–3539. doi: 10.1093/eurheartj/ehy333

34. Matsui T, Uehara T, Miyakita T, Hiramatsu K, Osada Y, Yamamoto T. The Okinawa study: Effects of chronic aircraft noise on blood pressure and some other physiological indices. *J Sound Vib.* 2004;277: 469–470. doi: 10.1016/j.jsv.2004.03.007

35. Ancona C, Badaloni C, Fano V, Fabozzi T, Forastiere F, Perucci C. Aircraft noise and blood pressure in the populations living near the Ciampino Airport in Rome. *Epidemiology.* 2009;20:S125–S126. doi: 10.1097/01.ede.0000362434.82215.9d

36. Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson CG, Bluhm G. Aircraft noise and incidence of hypertension. *Epidemiology.* 2007;18:716–721. doi: 10.1097/EDE.0b013e3181567e77

37. Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, Dudley ML, Savigny P, Seiffert I, Swart W, et al; HYENA study team. Hypertension and exposure to noise near airports: the HYENA study. *Environ Health Perspect.* 2008;116:329–333. doi: 10.1289/ehp.10775

38. Kim R, Berg Mv. Summary of night noise guidelines for Europe. *Noise Health.* 2010;12:61–63. doi: 10.4103/1463-1741.63204

39. D'Andrea A, Riegler L, Rucco MA, Cocchia R, Scarafile R, Salerno G, Martone F, Vriz O, Caso P, Calabrò R, et al. Left atrial volume index in healthy subjects: clinical and echocardiographic correlates. *Echocardiography.* 2013;30:1001–1007. doi: 10.1111/echo.12217

40. Kreutz R, Dobrowolski P, Prejbisz A, Algharably EAE, Bilo G, Creutzig F, Grassi G, Kotsis V, Lovic D, Lurbe E, et al. Lifestyle, psychological, socioeconomic and environmental factors and their impact on hypertension during the coronavirus disease 2019 pandemic. *J Hypertens.* 2020;39:1077–1089. doi: 10.1097/HJH.0000000000002770

41. Voitsidis P, Gliatas I, Bairachtari V, Papadopoulou K, Papageorgiou G, Parlapani E, Syngelakis M, Holeva V, Diakogiannis I. Insomnia during the COVID-19 pandemic in a Greek population. *Psychiatry Res.* 2020;289:113076. doi: 10.1016/j.psychres.2020.113076