

Acute effects of night-time noise exposure on blood pressure in populations living near airports

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Aims

Within the framework of the HYENA (hypertension and exposure to noise near airports) project we investigated the effect of short-term changes of transportation or indoor noise levels on blood pressure (BP) and heart rate (HR) during night-time sleep in 140 subjects living near four major European airports.

Methods and results

Non-invasive ambulatory BP measurements at 15 min intervals were performed. Noise was measured during the night sleeping period and recorded digitally for the identification of the source of a noise event. Exposure variables included equivalent noise level over 1 and 15 min and presence/absence of event (with L_{Amax} > 35 dB) before each BP measurement. Random effects models for repeated measurements were applied. An increase in BP (6.2 mmHg (0.63–12) for systolic and 7.4 mmHg (3.1, 12) for diastolic) was observed over 15 min intervals in which an aircraft event occurred. A non-significant increase in HR was also observed (by 5.4 b.p.m.). Less consistent effects were observed on HR. When the actual maximum noise level of an event was assessed there were no systematic differences in the effects according to the noise source.

Conclusion

Effects of noise exposure on elevated subsequent BP measurements were clearly shown. The effect size of the noise level appears to be independent of the noise source.

Keywords

Environmental noise • Blood pressure • Night-time sleep • Acute effects • Epidemiological study

Introduction

Noise, defined as undesirable sound, is known to be a stress stimulus that can produce acute blood pressure (BP) elevation in animals¹ and in humans in laboratory or occupational settings.²

Persons exposed to high-level noise (including recorded industrial or transportation noise) in the laboratory showed BP rises during the stimulus and for seconds to minutes after its

cessation.^{3–6} In field studies, workers exposed to high-level industrial noise and having their BP measured via ambulatory BP monitoring (ABPM) showed BP increments during exposure and for a few hours after.^{7,8} Although sound levels of transportation (mainly aircraft and road traffic) noise are usually lower, they may produce cardiovascular effects via the neuroendocrine system by causing emotional reactions and annoyance through interference with the individual's mental tasks, relaxation or

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Table 1 Descriptive characteristics of the 140 study subjects

	Athens (n = 43)	London (n = 16)	Milan (n = 50)	Stockholm (n = 31)
Gender [n; male (%)]	14 (32.6)	8 (50.0)	26 (52.0)	16 (48.5)
Age [years; mean (SD)]	53 (7.8)	58 (7.9)	56 (7.9)	56 (6.4)
Number of BP measurements per night [mean (SD)]	29 (6.2)	30 (4.4)	32 (3.8)	31 (5.2)
Systolic BP [mmHg; mean (SD)]	111 (17.3)	104 (13.2)	110 (15.1)	106 (16.3)
Diastolic BP [mmHg; mean (SD)]	66 (12.3)	62 (10.1)	66 (11.7)	63 (11.2)
Heart rate [b.p.m.; mean (SD)]	65 (9.7)	63 (9.2)	64 (10.7)	61 (8.8)
Number of aircraft events per night ^a median (25th—75th) percentile	19 (5–32)	0 (0–17)	2 (0–7)	0 (0–5)
Number of road traffic events per night ^a median (25th—75th) percentile	1 (0–9)	0 (0–38)	0 (0–1)	0 (0–6)
Number of indoor source events per night ^a median (25th—75th) percentile	14 (8–26)	5 (0–22)	14 (10–21)	9 (5–15)

^aEvent identified as present if measured LA_{max} > 35dB.

sleep, at cortical (conscious) or subcortical level.^{9–11} Indeed, systolic BP responses to moderate noise in field conditions were more consistent than those to intense noise in a laboratory on the same individuals.¹² Moreover, cardiovascular responses of the same individuals have been found greater during sleep than during wakefulness.¹³ In a sleep laboratory, BP and heart rate (HR) increments were traced after tonal acoustic stimuli or recorded transportation noise; arousal was not needed for sound to produce cardiovascular effects.^{14,15} Noise disturbance during sleep is regarded as one of the most important aspects of environmental noise exposure with possible effects on health.^{11,13,16} However, field studies on the effects of noise on BP during sleep in real life conditions are lacking.

In the present study the effect of environmental noise on BP and HR during night-time sleep of persons living in the vicinity of four major European airports was investigated within the wider framework of the HYENA (hypertension and exposure to noise near airports) project.¹⁷

Methods

Sampling

The sample for the present study was selected from the main sample of the HYENA project¹⁷ and consisted of subjects living around four European airports with night flights: Athens (Greece), Malpensa (Italy), Arlanda (Sweden) and London Heathrow (UK). The initial sample for the HYENA study¹⁷ was 6000 persons living in the vicinity of the study airports. A total of 4861 persons (2404 men and 2457 women) between 45 and 70 years old at the time of interview participated in the study. The samples were representative from the populations exposed to various levels of aircraft and traffic noise around airports based on noise contours. Participation rates differed between the countries, from circa 30% in Italy and the UK, to 56% in Greece and 78% in Sweden. More details may be found in Jarup *et al.*¹⁸ We selected subjects from various aircraft noise exposure categories, as assessed by the A-weighted annual equivalent noise level

LA_{eq}24h based on their residence, in order to obtain a larger variability in noise exposure situations.

The following exclusion criteria were applied: (1) antihypertensive medication, (2) diagnosis of diabetes mellitus, (3) diagnosis of obstructive sleep apnoea syndrome, (4) diagnosis of secondary hypertension, (5) working in night shift, (6) using sleeping pills and sedatives, (7) diagnosis of hearing impairment, (8) regular use of earplugs, (9) diagnosis of atrial fibrillation. Criteria 1–6 were applied as they affect the night-time BP; criteria 7 and 8 as they modify noise exposure; and criterion 9 as it hinders ABPM. Twenty-one subjects were excluded due to technical problems with the monitoring equipment. The final sample consisted of 140 subjects (Table 1). Approval for the study was granted by each centre's Ethical Committee.

Measurements and data management

Continuous noise measurement with the type I 'CESVA SC310' noise-meter¹⁹ (time constant 'fast' 125 ms) as well as noise recording with an MP3 recorder connected to the noise-meter's high-quality microphone were done during the study night in each participant's bedroom. Each participant was followed up for one night. The noise level equivalents for every second, for every 1 min before and for every 15 min period between BP measurements were calculated as follows:

$$LA_{eq} = 10 \times \log \left(\sum_{i=1}^t 10^{LA_{eq1sec}/10} \right) - 10 \times \log(t)$$

where t is the 1 min or 15 min period in seconds.

Using playback and visualization of sound recordings on a computer, the source of each event was identified and synchronized with the sound measurements with a program written for this purpose. An event was defined as present if its indoor LA_{max} exceeded 35 dB. Noise events were classified into four categories according to source: indoor, aircraft, road traffic, and other outdoor. Other outdoor events were very rare and thus excluded from the analysis.

Non-invasive 24 h ABPM, with HR measurements, was performed at 15 min intervals with the validated 'Mobilograph' device,^{20,21} including the study night. The 15 min frequency has been implemented before⁷ and was chosen as optimal for frequent measurements without excessive sleep disturbance. The three instruments (noise meter, noise

recorder, and ABPM device) and the participants' alarm clock were synchronized at 1 min precision.

Specially trained nurses installed the noise equipment, placed the ABPM device on the participants and gave them written instructions, during a home visit at least 3 h before normal sleeping time. Each participant was instructed not to engage in unusually heavy activity during the measurements' period and filled in a sleep log indicating actual sleeping times.

Statistical analysis

Linear mixed models which included random intercept and random coefficients for the various noise indicators were applied for each centre separately in order to assess acute effects of noise on BP and HR during night-time sleep. The number of repeated measurements per subject corresponds to the number of systolic and diastolic BP measurements (mmHg), as well as HR measurements (beats per minute—b.p.m.) during the self-reported sleeping period. The A-weighted indoor noise level equivalents of 1 min (LAeq1min) and of 15 min (LAeq15min) before BP measurements were used as short-term noise exposure variables. In this type of model, where an individual serves as his own 'control', there is no need to adjust for individual confounding factors. In order to account for the possible confounding effect of misreporting of sleeping and waking times (potentially associated with both BP and noise levels), the above noise exposure variables were also adjusted for the sequence of BP measurements, using 2 linear terms. The first linear term denoted the sequence of the BP measurements (1,2,...,k) from the start to the middle of each persons sleep period and the second denoted the sequence of the BP measurements (1,2,...,k) from the middle to the end of each persons sleep period. Other noise exposure variables were the presence or absence of a source-specific noise event during the 15 min periods and each source-specific event's LAmax. If more than one event were present in the 15 min interval, the higher LAmax was used. Since a noise stimulus may not have the same effect in the presence of other noise, in all models assessing the source-specific noise, we adjusted for the 10th percentile of the noise level equivalent (L90) in all the 15 min intervals. After obtaining the four centre-specific effects using random effect models, we then combined the centre-specific results using either fixed or random effects meta-analysis depending on the absence or presence of heterogeneity. All reported *P*-values are based on two-sided hypotheses and the significance level used was 5%.

Results

Table 1 shows the descriptive characteristics of the samples. The mean number of repeated BP measurements per night was similar in all centres (range 29–32). BP and HR displayed a normal distribution (data not shown). The number of aircraft events during the night-time sleep was higher in Athens compared with the other centres. The presence of subjects with no or few night aircraft events was explained by the sampling procedure through which a number of subjects were selected from the main sub-sample of low exposure to aircraft noise. In Milan and Stockholm, the number of indoor events was much larger than aircraft or traffic events. The median equivalent noise levels (LAeq15min, LAeq1min) for all centres were comparable. Also the source-specific LAmax (aircraft, road or indoor) was similar in the four samples (Figure 1).

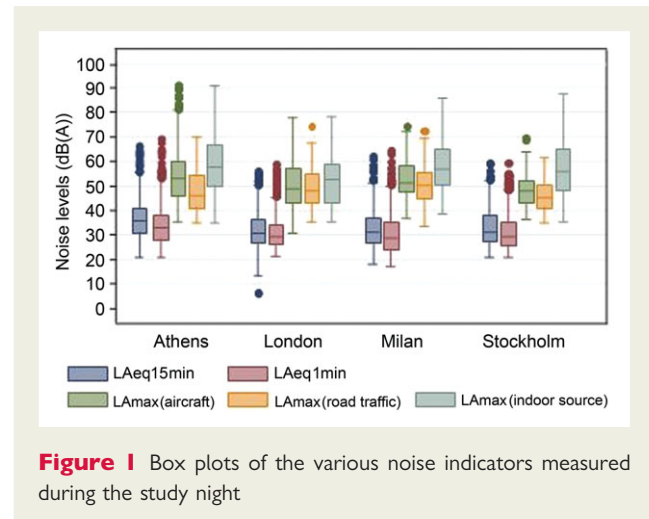


Figure 1 Box plots of the various noise indicators measured during the study night

Table 2 shows the pooled effect estimates of the noise exposure indicators on BP and HR. The measured noise 1 and 15 min before each BP measurement was associated with higher systolic and diastolic BP and with higher HR. For example, a 5 dB increment in LAeq15min was associated with a 0.63 mmHg increase in diastolic BP. The magnitude of the effect on BP was somewhat lower when the LAeq1min was considered but remains statistically significant. The effect remains similar when adjustment was applied for the sequence of measurements during sleep time. An increase in BP (6.2 mmHg for systolic, 7.4 mmHg for diastolic) and HR (by 5.4 b.p.m.) was observed over 15 min intervals in which an aircraft event occurs but for HR this was not statistically significant. A similar magnitude of increase in BP was observed during time periods with traffic or indoor source event. In contrast, the effect on HR was smaller during periods with indoor events and was not observed over periods with traffic events. When the actual noise level assessed by LAmax was taken into account (adjusting for the presence of an event from a specific source and for L90), a positive association was found between noise from all the three sources and BP, which was statistically significant and similar to the effect of measured noise from all sources. The corresponding effects on HR were lower than those from all sources and reached statistical significance only for indoor source noise.

The estimated effects of noise exposure on BP were consistent in each sample. The estimated effects on diastolic BP of measured noise by sample are shown in Figure 2. The estimates were practically identical for the three samples, whilst for London they were higher but associated with wider confidence intervals. In Figure 3 a consistent pattern may be seen for the effects of source-specific noise in the four samples. The corresponding results for systolic BP were similar, with the exception of significant heterogeneity in the effects of indoor source noise events where the effect was highest in London and lowest in Stockholm, the only non-statistically significant effect. The effects for HR were less consistent between centres. However, the only model which displayed statistically significant heterogeneity was the one assessing the presence of aircraft events.

Table 2 Pooled effect estimates of various noise indicators on blood pressure (BP) and heart rate (HR) measurements. Results from fixed effects models (except where noted)

Model		Increase in systolic BP (mmHg) (95% CI)	Increase in diastolic BP (mmHg) (95% CI)	Increase in heart rate (b.p.m.) (95% CI)
1	LAeq(15 min) ^a (5 dB)	0.74 (0.40, 1.08)	0.63 (0.34, 0.91)	0.26 (0.07, 0.44)
2	LAeq(1 min) ^b (5 dB)	0.69 (0.36, 1.02)	0.55 (0.26, 0.84)	0.30 (0.12, 0.49)
3	LAeq(15 min) ^c (5 dB)	0.82 (0.48, 1.16)	0.62 (0.36, 0.88)	0.23 (0.07, 0.40)
4	LAeq(1 min) ^d (5 dB)	0.88 (0.54, 1.22)	0.50 (0.23, 0.78)	0.35 (0.17, 0.53)
5	Aircraft events ^e (yes = 1)	6.20 (0.63, 11.77)	7.39 (3.09, 11.69)	5.42 ^g (-2.01, 12.85)
6	LAm _{max} aircraft events ^f (5 dB)	0.66 (0.33, 0.98)	0.64 (0.37, 0.90)	0.18 (-0.04, 0.40)
7	Road traffic events ^e (yes = 1)	4.81 (-2.45, 12.06)	3.34 (-7.37, 14.04)	-2.76 (-7.30, 1.77)
8	LAm _{max} road traffic events ^f (5 dB)	0.81 (0.46, 1.16)	0.55 (0.26, 0.83)	0.01 (-0.41, 0.42)
9	Indoor source events ^e (yes = 1)	7.39 (3.76, 11.02)	4.19 (0.65, 7.72)	3.00 (0.87, 5.13)
10	LAm _{max} indoor source events ^f (5 dB)	0.87 ^g (0.17, 1.57)	0.68 (0.43, 0.92)	0.21 (0.01, 0.41)

^aEquivalent noise level of the 15 min before BP measurement.

^bEquivalent noise level of the 1 min before BP measurement.

^cEquivalent noise level of the 15 min before BP measurement, adjusted for the sequence of BP measurements from the start of the sleeping period to the middle of the night-time sleep, and from the middle of the night-time sleep to the wake-up period.

^dEquivalent noise level of the 1 min before BP measurement, adjusted for the sequence of BP measurements from the start of the sleeping period to the middle of the night-time sleep, and from the middle of the night-time sleep to the wake-up period.

^eYes: event with indoor LAm_{max} > 35 dB present.

^fIndoor LAm_{max} of source-specific event adjusted for presence of event (yes: event with LAm_{max} indoor > 35 dB present) and for L90 in all 15 min time periods without the source-specific event.

^gResults from random effects models in presence of significant heterogeneity.

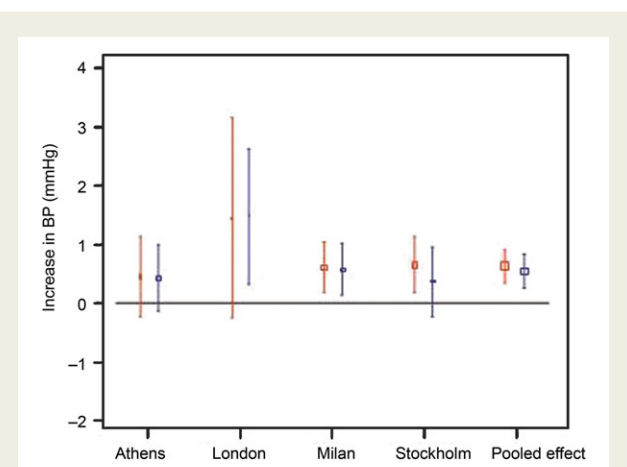


Figure 2 Centre-specific and pooled effect estimates of a 5 dB(A) increment in the equivalent noise level of 15 min (red) and 1 min (blue) before blood pressure (BP) measurement on diastolic BP

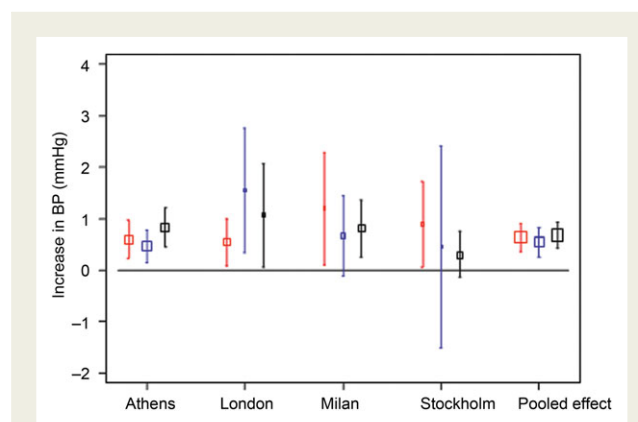


Figure 3 Centre-specific and pooled effect estimates on diastolic blood pressure (BP) and its 95% confidence Interval (CI) associated with an increase of 5 dB in LAm_{max} of aircraft event (red), of road traffic event (blue) and of indoor event (black) during night-time sleep (source-specific event identified as present if indoor measured LAm_{max} > 35 dB)

Discussion

We studied the effect of noise on BP during sleep, a state of reduced sympathetic and increased parasympathetic autonomous nervous system (ANS) tone, leading to a decrease of BP and HR.²²

We found that both systolic and diastolic BP levels as well as HR increased with higher noise levels during the preceding minutes,

independently of the noise source and of the sequence of the measurement during sleep time, which indicates absence of habituation during the study night, a matter of controversy in studies on humans or on experimental animals.^{13,23} These results are consistent with those reported by Carter *et al.*¹⁵ in a laboratory where both BP and HR increased after noise stimuli. There are major differences between laboratory and real life conditions. In a laboratory, background noise is steady whilst in real life conditions the

analysis has to adjust for variable noise sources. Davies *et al.*¹⁴ studied five subjects and found an increase in diastolic BP from 4 to 6 mmHg associated with arousal according to sleep stage. We found an increase of 6–7 mmHg in diastolic BP according to the source of the noise event, not necessarily associated with awakening.

We also found significant increases in BP and, less consistently, HR when the source of the noise was taken into account. The effects of the source-specific noise were comparable for aircraft, traffic, and indoor events and were similar to those of the total measured noise.

In our study, one common source of indoor noise was snoring. Since in some cases the study subject is expected to be the snorer too (we could not assess this information) and his BP elevation could be due to disordered breathing,^{24,25} the effect of indoor noise on BP that we report could be overestimated.

The effect of the measured noise level as well as the source-specific noise was weaker and less consistent on HR than on BP. This finding is in accordance with results from a sleep laboratory study¹⁵ where a noise threshold was detected for the effects on HR but not on BP and with previously reported mechanisms of BP rise following noise exposure, which refer mainly to vasoconstriction.^{2,26} Moreover, HR has a stronger circadian component which may mask other effects.²⁷

The remarkable consistency of the estimated effects between the four centres strengthens the evidence for causality. The effects of noise exposure on BP per specific increase in noise levels were found similar in the four samples in spite of the fact that there were differences in the profiles of noise according to the source during the night. However, the indoor L_{Amax} levels in the presence of an event were comparable in all the four centres.

In sleep laboratories, noise stimuli of levels comparable with those of real life produced cardiovascular responses for seconds.^{13,14} However, in occupational or laboratory settings, BP elevations have been found to last for minutes or even hours^{6–8} during wakefulness. Emotional responses such as anger or fear may magnify and prolong the effects, during night-time exposure to aircraft noise in real life conditions.⁹ One drawback of our study is that BP was assessed every 15 min although the noise event could have happened anytime within this interval. As expected, the distribution of noise events over the 15 min intervals between BP measurements is uniform. Only 5–11.5% of events (the range reflects different sources and samples) occurred during the minute of measurement (6.7% expected). The design of our study might have led to the 'loss' of the effect on BP or HR if the noise event happened to occur during the first minutes of intervals between measurements and the effect was of short duration. However, the fact that the effects of noise during the preceding 1 and 15 min were of similar magnitude indicates more prolonged effects.

In this study, the indoor L_{Amax} threshold for characterizing an event as 'present' was set at 35 dB. Awakening reactions are usually observed at L_{Amax} values over 40–45 dB in the bedroom but recently lower thresholds have been also suggested.^{11,28} However, according to sleep laboratory studies, haemodynamic changes can also occur at lower noise levels than the ones that cause EEG changes, although the effects are stronger when

arousal coexists.^{13,14} Indeed, autonomic responses like BP elevation have been used as a sensitive marker of sleep disturbance.^{29,30} The finding that consciousness is not needed for sound to produce its cardiovascular effects is also supported by experiments in which anaesthetized animals demonstrated BP increment when exposed to intense noise³¹ or BP reduction when exposed to music.^{32,33} These outcomes are attributed to the subcortical connections of the auditory pathway with the ANS (amygdala, hippocampus, hypothalamus)¹⁰ and justify the use of relatively low noise thresholds in the research of noise effects on BP during sleep.

To assess nocturnal BP, we used ABPM, which has been validated and used extensively for this purpose.^{19,20} ABPM has been reported to affect sleep and night-time BP³⁴ during the cuff's inflation similarly to noise arousal stimuli,¹⁴ although it is also supported, by means of intra-arterial recordings, that ABPM does not attenuate night-time BP reduction.³⁵ In any case, it can be argued that there is synergy and that the effects of noise would not be the same in the absence of ABPM during the study night. Because of this possibility, the frequency of BP measurements was kept at four per hour, although up to six measurements per hour have been used before.³⁶ Moreover, the body position during sleep can affect the ABPM measurements.³⁷ All measurements however in our study, irrespective of noise exposure level, were done with ABPM and there is no reason to assume that body position during sleep is related to noise exposure. The use of portable, non-invasive BP recorders that register BP continuously and correct automatically hydrostatic effects,³⁸ can be considered in future studies investigating the effect of noise on night-time BP in real life conditions.

Within the HYENA project we found effects of long-term noise exposure on the prevalence of hypertension¹⁸ and the acute effects reported here. Absence of short-term habituation to the cardiovascular effects of noise, especially those during sleep, found here and also reported before,^{13,16,39} as well as evidence from studies on sleep-disorder which indicate that repeated arousals are associated with a sustained increase in daytime BP,⁴⁰ support a link between acute and long-term effects of noise exposure on hypertension^{41,42} and cardiovascular disease,⁴³ in line with the general stress theory.⁴⁴

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