

Noise levels and cardiovascular mortality: a case-crossover analysis

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Abstract

Background: The relationship between occupational noise and cardiovascular outcomes has been widely investigated. Regarding environmental noise levels, the attention is focused on road traffic noise due to the large number of exposed persons and the large periods of exposure. There are few studies assessing the short-term effects of traffic noise on cardiovascular outcomes. The aim of this study was to quantify the short-term effects of urban noise levels on age-specific cardiovascular mortality.

Methods: A case-crossover design was used. Daily mortality counts in Madrid city due to cardiovascular causes (ICD codes: 390–459) from 1 January 2003 to 31 December 2005 were obtained. Data noise levels were collected as diurnal equivalent noise (Leq_{d8–22h}), night equivalent noise (Leq_{n22–8h}), and daily equivalent noise (Leq_{24h}). Confounding variables as daily levels of air pollutants, temperature, and relative humidity data were controlled. Overdispersed Poisson regression models were adjusted to control for both seasonality and time trends. Estimated effects are reported as percentage increase in the relative risk (IRR) associated with an increase of 1 dBA.

Results: The strongest associations between all noise exposure levels and cardiovascular mortality were reported at lag 1: IRR 4.5% (95% CI 0.6, 8.7%), IRR 3.9% (95% CI 0.6, 7.3%), and IRR 6.2% (95% CI 2.1, 10.6%) for Leq_d, Leq_n, and Leq₂₄, respectively. Analysing by age-specific groups at lag 1, statistically significant associations were found for those aged ≥65: 4.5% (95% CI 0.3, 8.9%), 3.4% (95% CI 0.1, 6.9%), and 6.6% (95% CI 2.2, 11.1%) for Leq_d, Leq_n, and Leq₂₄, with no substantial changes in the effects of noise exposure levels at lag 1 after adjusting for PM_{2.5} and NO₂.

Conclusion: The association found between noise exposure levels and cardiovascular mortality suggests a joint effect of diurnal and night-time noise levels. Our results also reveal independent effects of noise exposure levels and the air pollutants analysed. This strongly suggests the need to seriously consider the high noise exposure levels reported as an important public health issue.

Keywords

Cardiovascular mortality, case-crossover, traffic noise, urban noise

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Introduction

The highest noise exposure levels have been usually associated with occupational environments. For this reason, the relationship between occupational noise and cardiovascular outcomes has been widely investigated.^{1–3} Regarding environmental noise levels, airport proximities are often considered especially noisy areas where the association between noise and cardiovascular disease has also been widely studied.^{4,5} However, in urban settings, attention is focused on road traffic noise due to the large number of exposed persons and the large periods of exposure. According to a recent WHO/Europe study,⁶ 40% of the EU population are exposed to road traffic levels above the WHO health

protection values⁷ of 55 dBA, 20% above the daytime protection value of 65 dBA, and 30% above the night-time protection value of 55 dBA. The consequence of such exposure implies a loss of 61,000 healthy life-years due to disability.⁷

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Recently, several cohort studies have investigated the long-term effects of chronic noise exposures on cardiovascular mortality,⁸ and more specifically on myocardial infarction^{9–11} and stroke.¹² Unlike the case of chemical air pollution and despite the physiopathological evidence,¹³ there are few studies assessing the short-term effects of traffic noise on cardiovascular outcomes.^{14,15} This is probably due to the lack of networks, or monitoring stations, to measure real-time traffic noise levels. In this sense, the city of Madrid offers a suitable scenario to study the short-term effects of noise levels, since the Madrid's Municipal Automatic Air Pollution Monitoring Network also measures real-time noise pollution levels, mainly produced by road traffic.

The aim of this study was to quantify the short-term effects of urban noise levels on age-specific cardiovascular mortality. Moreover, we obtained evidence of an air pollution-independent association after adjusting for primary chemical air pollutants.

Methods

Setting

The city of Madrid constitutes a dense metropolitan area located in the central region of Spain, with over 2 million motor vehicles. The city has a daily mean traffic volume of 2.4 million vehicles, reaching the maximum in May (2.5 million) and the minimum in August (1.7 million). The mean speed of automobiles in the whole city is nearly 24 km/h. The main outdoor noise source is road traffic, being attributed 80% of the overall noise exposure.¹⁶ Other sources that contribute to outdoor noise levels are industry (10%), rail traffic (6%), and leisure activities (4%).¹⁶

Mortality data

Daily mortality counts due to cardiovascular causes (International Classification of Diseases, 9th revision: 390–459) were obtained from the Madrid Regional Inland Revenue Department, which is the department responsible for mortality registry, from 1 January 2003 to 31 December 2005.

Noise exposure levels

Equivalent noise levels were collected from the Noise Pollution Monitoring Grid. This network consists of six urban background stations, specifically located to become representative of the noise levels across the city, that capture environmental noise data and in turn transmit to a central station for their processing. Technically, the measuring process involves

the following steps: (a) an outdoor antibird omnidirectional microphone, provided with wind screen, captures the data; (b) the captured signal connects with a statistical noise analyser: the latest analysers also allow audio recording and frequency analysis (1/1- and 1/3-octaves); (c) the information stored in the analyser is transferred to a central station via a high-speed telephony modem (ISDN); and (d) the central station is equipped with a distributor adapted to ISDN that communicates with all stations at set intervals to send the data. Data were collected as daily mean levels of diurnal equivalent noise levels for the 8–22 h period (Leqd), night-time equivalent noise levels for the 22–8 h period (Leqn), and daily equivalent noise levels for the 0–24 h period (Leq24).

Other covariates

Daily mean levels of primary chemical air pollutants – particulate matter with aerodynamic diameter <2.5 µm (PM_{2.5}) and nitrogen dioxide (NO₂) – were supplied by the Madrid's Municipal Automatic Air Pollution Monitoring Network. Mean temperature and relative humidity data were obtained from the Madrid-Retiro Observatory and their were supplied by the State Meteorology Agency (Agencia Estatal de Meteorología (AEMET)).

Design and statistical analysis

The association of noise levels with daily cardiovascular mortality was investigated using a time-stratified case-crossover design¹⁷ in which the noise exposure of the index day of the cardiovascular event (case day) is compared with the noise exposure during one or more control periods on which the cardiovascular event does not occur (control days). We followed a time-stratified approach by dividing the study period into monthly strata and selecting control days to be all days falling on the same day of the week within the same stratum as the case day.¹⁷ This is the common approach using in environmental epidemiology studies,^{18,19} which allows minimizing bias from both time trends in the exposure series and other short-term time-varying confounders.¹⁷

We fitted overdispersed Poisson regression models adjusted for a three-way interaction term between day of the week, month, and year to control for both seasonality and time trends. This choice was motivated by the need to replicate the adjustment made by the case-crossover design with the time-stratified approach for the selection of control days.²⁰ Models were also adjusted for temperature and humidity, using one temperature mean to control for the immediate effects dominated by heat (mean on the exposure day and the day before) and a second temperature mean to control for effects of lower temperatures at longer lags

(mean on the second to fourth days before the exposure) by using, for both, natural cubic splines with 3 degrees of freedom, and a 5-day mean humidity (day of exposure and 5 days before). Finally dummy variables for bank holidays and heat-wave days were also included. To take into account possible overdispersion of daily death rates, we used quasi-likelihood estimation.

The effects of noise exposure were examined for the same day (lag 0) to 4 days after the exposure (lag 4). Estimated effects are reported as percentage increase in the relative risk (IRR), calculated as $(RR - 1) \times 100$ per 1 dBA increase. In order to examine differences in this association among different age groups, we analysed the effects of pollution on cardiovascular mortality for subjects aged $<$ and ≥ 65 . We also studied an air pollution-independent association by adjusting for primary chemical air pollutants (PM_{2.5} and NO₂) at lag 1, based in previous studies conducted in Madrid.²¹ All analyses were carried out using Stata statistical software version 12 (Stata-Corp, College Station, TX, USA).

Results

Mean daily deaths due to cardiovascular causes were 18.7, ranging from 5 to 40, with more than 90% aged ≥ 65 years (Table 1). The distribution of noise exposure (Leqd, Leqn, and Leq24) is shown in Figure 1. The mean daytime noise level in Madrid was 65.1 dBA, ranging from 62.1 to 67.3 dBA, exceeding the WHO

health protection values for 54% of the days (Table 1). The mean night-time noise level was 60.5 dBA, exceeding the WHO guideline value for 100% of the nights. Day and night-time noise levels were highly correlated ($r=0.45$). The mean daily noise level was 64.1 dBA, being highly correlated with the diurnal level ($r=0.95$) rather than with the night-time level ($r=0.54$). Primary chemical air pollutants showed mean daily levels of 19.1 and 59.9 $\mu\text{g}/\text{m}^3$ for PM_{2.5} and NO₂, respectively, which were highly correlated ($r=0.70$).

Figure 2 shows that the associations between all noise exposure levels and cardiovascular mortality were strongest at lag 1: IRR 4.5% (95% confidence interval, CI 0.6, 8.7%, $p=0.025$), IRR 3.9% (95% CI 0.6, 7.3%, $p=0.019$), and IRR 6.2% (95% CI 2.1, 10.6%, $p=0.003$) for a 1 dBA increase in Leqd, Leqn, and Leq24, respectively, although the effects of Leqn were comparable between lag 1 and lag 2 (IRR 3.9%, 95% CI 0.4, 7.4%, $p=0.026$). When analysing by age-specific groups at lag 1 (Table 2), effects were stronger for those aged ≥ 65 years (IRR 4.5%, 95% CI 0.3, 8.9, $p=0.034$; IRR 3.4%, 95% CI 0.1, 6.9%, $p=0.045$; and IRR 6.6%, 95% CI 2.2, 11.1%, $p=0.003$, for Leqd, Leqn, and Leq24, respectively). However, effects for those aged <65 years were smaller and nonsignificant.

For those aged ≥ 65 years, the effects of noise exposure levels at lag 1 did not change substantially after adjusting for PM_{2.5} and for NO₂, both at lag 1 (Table 3). In these multiple exposure models, a noise-independent effect was found for PM_{2.5} at lag 1 (IRR 1.9%, -0.3, 4.1%, $p=0.088$; IRR 2.2%, -0.1, 4.4%, $p=0.052$; IRR 2.1%, -0.1, 4.3%, $p=0.063$, for an IQR increase of 10 $\mu\text{g}/\text{m}^3$ when adjusting for Leqd, Leqn, and Leq24, respectively) and for NO₂ at lag 1 (IRR 2.2%, 0.0, 4.5%, $p=0.047$; IRR 2.4%, 0.2, 4.7%, $p=0.031$; IRR 2.1%, 0.1, 4.5%, $p=0.041$, for an IQR increase of 5 $\mu\text{g}/\text{m}^3$ when adjusting for Leqd, Leqn, and Leq24, respectively).

Discussion

Spain is the second country worldwide, after Japan, that has the largest population exposed to high noise levels.²² Nearly 9 million citizens suffer noise levels above 65 dBA, and 66.7% of those living in the major cities (over 250,000 inhabitants) are exposed to levels above 55 dBA.²³ The current high noise exposure levels in Madrid show an important public health issue, since these are similar to those reported since 1995 in previous studies.^{14,24} The main source of noise exposure levels produced in Madrid was related to road traffic,¹⁶ and there was a moderate correlation with NO₂, since both come from the same source. However, the

Table 1. Summary statistics for cardiovascular mortality, noise exposure levels, primary chemical air pollutants, and weather variables in Madrid for the study period 2003–2005.

	Mean (SD)	Range
Mortality (n) ^a		
All ages	18.7 (5.4)	5 to 40
<65 years	1.6 (1.3)	0 to 7
≥ 65 years	17.1 (5.1)	5 to 36
Equivalent noise levels (dBA)		
Diurnal	65.1 (0.8)	62.1 to 67.3
Night time	60.5 (0.9)	58.7 to 71.0
Daily	64.1 (0.7)	61.5 to 66.4
Air pollutants ($\mu\text{g}/\text{m}^3$)		
PM _{2.5}	19.1 (8.6)	5.0 to 71.0
NO ₂	59.9 (17.7)	19.0 to 133.0
Weather		
Temperature ($^{\circ}\text{C}$)	15.4 (7.9)	-1.2 to 31.5
Relative humidity (%)	54.3 (20.4)	13.0 to 96.0

^aICD9 390–459.

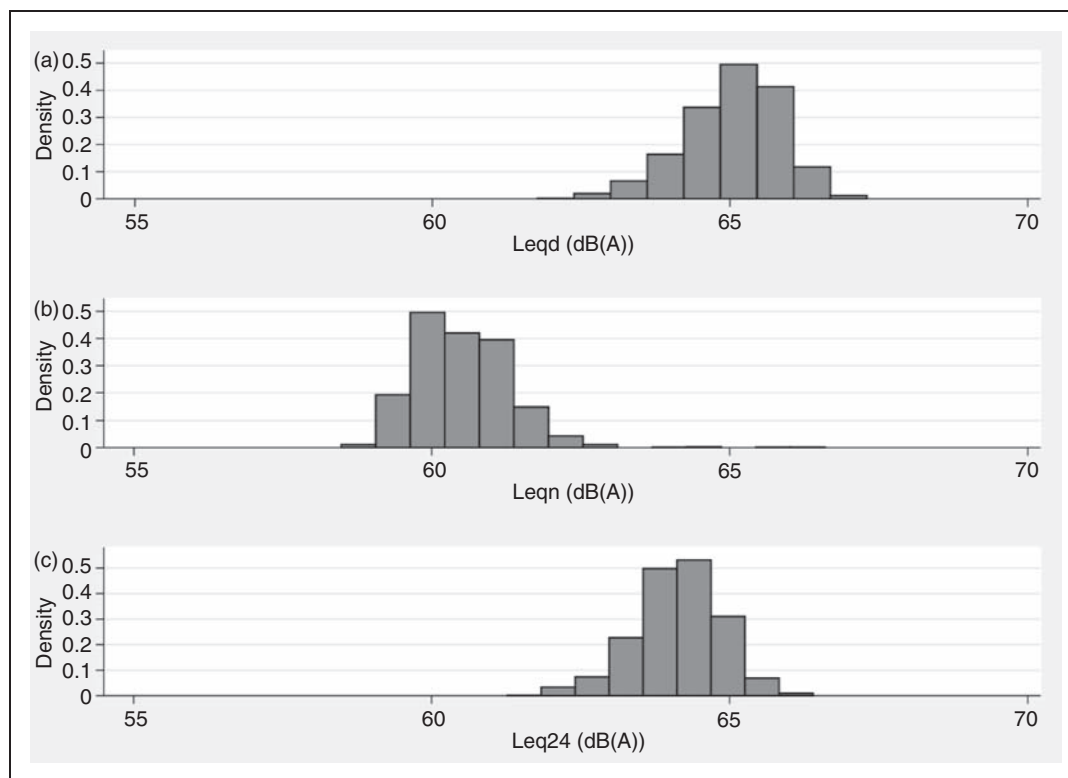


Figure 1. Distribution of diurnal (a), night-time (b), and daily equivalent noise (c) in the city of Madrid for the period 2003–2005. (a) For the 8–22 h period: Leqd, diurnal equivalent noise; (b) for the 22–8 h period: Leqn, night-time equivalent noise; (c) for the 0–24 h period: Leq24, daily equivalent noise.

correlation with $PM_{2.5}$ was lower probably due to resuspension and transport phenomena of particulate matter,²⁵ preventing emissions from being immediately captured by the sensors and thus computed as immission values. Furthermore, the natural-type component of PM has to be added to the anthropogenic component so that the actual traffic-related contribution to $PM_{2.5}$ ranges from 35 to 50%.²⁶

The association between noise exposure levels and cardiovascular mortality found in this study suggests a joint effect of diurnal and night-time noise levels (Leq24). This was also reported in a previous studies conducted in Madrid, analysing the impact of road traffic noise on hospital admission rates in children²⁴ as well as in the general population.¹⁴ Such joint effect could be primarily due to the fact that areas with high diurnal noise levels are the same as those with elevated noise levels at night. The greater impact of diurnal compared to night-time noise levels agrees with a recent WHO report.²⁷ Although there are a substantial number of studies, some of them with controversial results,²⁸ linking traffic noise with cardiovascular diseases, they generally consist of cohort^{29,30} or case-control studies³¹ related to long-term exposures. But, to our knowledge, there are no previous studies examining the short-term effects of

traffic noise by using time-series or case-crossover designs or even controlling for the effect of primary chemical air pollutants.

Moreover, extensive research supports the physiopathological mechanisms for the relationship between noise and cardiovascular outcomes. The underlying biochemical process is based on the assumption that the auditory system is one of the main physiological warning systems against possible hazards from the outside background.³² Noise activates the reticular system³³ leading to the release of adrenaline, norepinephrine, and cortisol, regarded to as stress hormones.³⁴ It has been estimated that 3% of occurrences of ischaemic heart disease in large cities are attributable to road traffic noise.³⁵ There is also increasing evidence suggesting significant associations between urban noise and severe cardiovascular endpoints such as myocardial infarction and stroke.^{11,12} These can be especially severe in people with a pre-existing underlying disease, mainly in those aged ≥ 65 years where these types of pathology is certainly more frequent. This indicates that noise effects on the cardiovascular system are likely to be greater with increasing age.³⁶ The 3.8% increased risk of daily cardiovascular mortality for a 1 dBA increase in those aged ≥ 65 years in Madrid is similar to that recently reported by Hart et al.,¹⁵

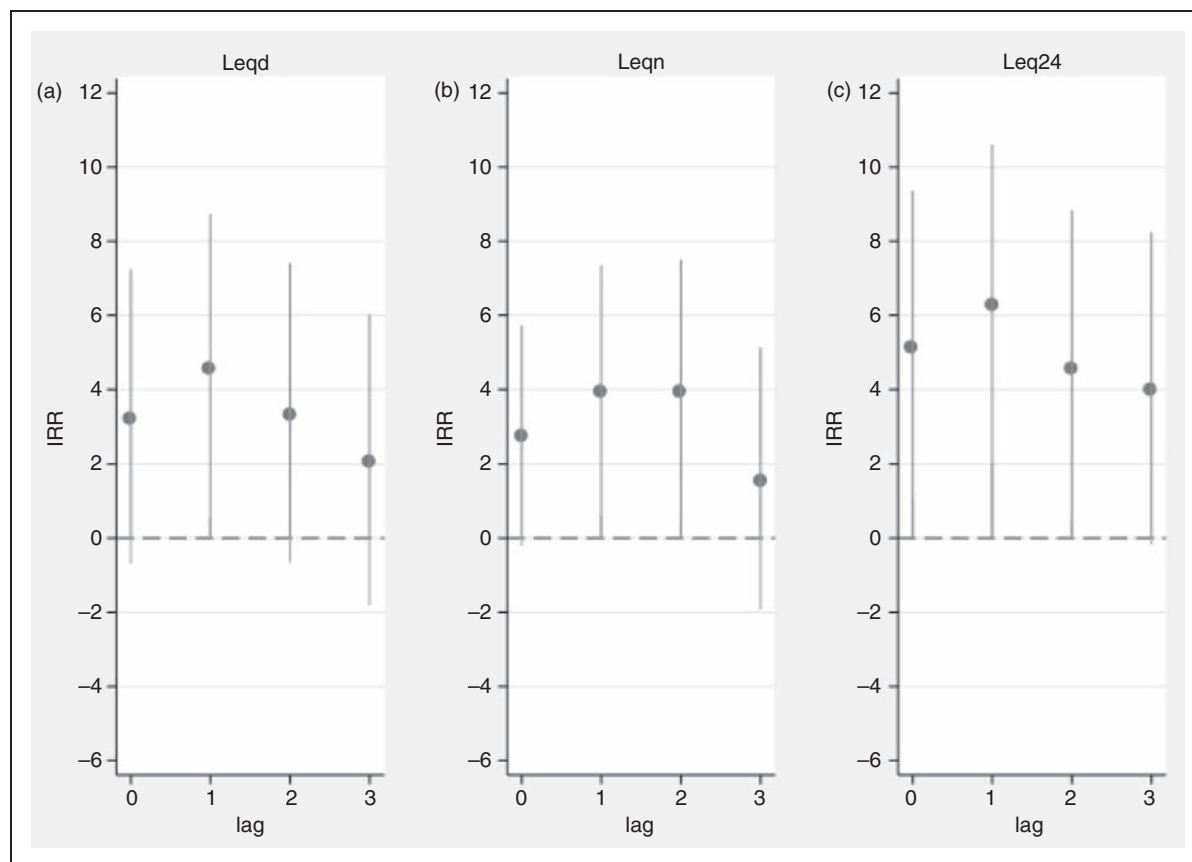


Figure 2. Increase in relative risk of cardiovascular mortality for a 1 dBA increase in diurnal (a), night-time (b), and daily equivalent noise (c) in Madrid for the period 2003–2005.

(a) For the 8–22 h period: Leqd, diurnal equivalent noise; (b) for the 22–8 h period: Leqn, night-time equivalent noise; (c) for the 0–24 h period: Leq24, daily equivalent noise. IRR, Increase in relative risk.

Table 2. Associations between diurnal, night-time, and daily equivalent noise and cardiovascular mortality by age groups, at lag 1, in Madrid for the period 2003–2005.

Equivalent noise level	<65 years	≥65 years
Diurnal	2.3 (−10.3, 16.5)	4.5 (0.3, 8.9)
Night-time	2.7 (−7.5, 18.8)	3.4 (0.1, 6.9)
Daily	2.9 (−10.3, 18.0)	6.6 (2.2, 11.1)

Values are IRR (95% CI). IRR, percentage increase in relative risk for a 1 dBA increase.

although the risk is much larger (up to 3-times) for more specific outcomes like myocardial infarction.

A main limitation is that we only have available a pooled outcome for all cardiovascular diseases. Unfortunately, more specific outcomes were not available. In contrast, noise levels are reported in great detail, which allowed this study to distinguish between diurnal, night-time, and daily equivalent noise levels. Also the extensive number of monitoring stations

allowing for noise data in real time are specifically located to be representative of the noise levels across the city. Furthermore, an obvious limitation comes from the ecological design used in our study. Although individual data are geographically aggregated at city level, noise exposures and cardiovascular events remain disaggregated on a daily basis, preventing to some extent the ecological fallacy inherent to ecological studies. The evaluation of exposure to noise levels is not an easy task, like the case of urban air pollution, due to the complex mixture of sources and spatial variability. As a consequence, it will be likely to have substantial measurement error in noise exposure assessment, although this error is expected to be unrelated to the assessed cardiovascular events (non-differential errors). Thus, the estimated effects may underestimate the true underlying effects of noise levels on cardiovascular mortality rates. Another issue is the appropriate control of confounding. For this reason, primary chemical air pollutants and main weather variables were accounted for in our analysis.

Table 3. Associations between diurnal, night-time, and daily equivalent noise and cardiovascular mortality for subjects aged ≥ 65 years, at lag 1, adjusted for primary chemical air pollutants at lag 1.

Equivalent noise level	Noise level	PM _{2.5}	NO ₂
Diurnal	4.5 (0.3, 8.9)		
+PM _{2.5}	4.9 (0.5, 9.4)	1.9 (−0.3, 4.1)	
+NO ₂	4.3 (0.2, 8.7)		2.2 (0.0, 4.5)
Night-time	3.4 (0.1, 6.9)		
+PM _{2.5}	3.7 (0.2, 7.3)	2.2 (−0.1, 4.4)	
+NO ₂	3.7 (0.2, 7.3)		2.4 (0.2, 4.7)
Daily	6.6 (2.2, 11.1)		
+PM _{2.5}	6.8 (2.3, 11.5)	2.1 (−0.1, 4.3)	
+NO ₂	6.6 (2.2, 11.2)		2.3 (0.1, 4.5)

Values are IRR (95% CI). IRR, percentage increase in relative risk for a 1 dBA increase.

Our results also suggest independent effects of noise exposure levels and primary chemical air pollutants. This agrees with others concluding that there is not much confounding between traffic noise and air pollution,^{9,37,38} since the increase in risk of mortality remains nearly constant after adjustment for primary chemical air pollutants. Furthermore, the impact of noise exposure levels is greater than that of the primary chemical air pollutants usually found in urban environments. This strongly suggests the need to seriously consider urban noise as a major pollutant related to road traffic in large European cities.

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Conflict of interest

The authors declare that there is no conflict of interest.

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