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Health impact assessment of traffic noise in Madrid (Spain)

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ABSTRACT

The relationship between environmental noise and health has been examined in depth. In view of the sheer number of persons exposed, attention should be focused on road traffic noise. The city of Madrid (Spain) is a densely populated metropolitan area in which 80% of all environmental noise exposure is attributed to traffic. The aim of this study was to quantify avoidable deaths resulting from reducing the impact of equivalent diurnal noise levels (LeqD) on daily cardiovascular and respiratory mortality among people aged $\geq$ 65 years in Madrid. A health impact assessment of (average 24 h) LeqD and $PM_{2.5}$ levels was conducted by using previously reported risk estimates of mortality rates for the period 2003–2005: For cardiovascular causes: LeqD 1.048 (1.005, 1.092) and $PM_{2.5}$ 1.041(1.020, 1.062) and for respiratory causes: LeqD 1.060 (1.000, 1.123) and $PM_{2.5}$ 1.030 (1.000, 1.062). The association found between LeqD exposure and mortality for both causes suggests an important health effect. A reduction of 1 dB(A) in LeqD implies an avoidable annual mortality of 284 (31, 523) cardiovascular- and 184 (0, 190) respiratory-related deaths in the study population. The magnitude of the health impact is similar to reducing average $PM_{2.5}$ levels by 10 $\mu g/m^3$. Regardless of air pollution, exposure to traffic noise should be considered an important environmental factor having a significant impact on health.

1. Introduction

According to a recent study by the World Health Organisation Regional Office for Europe (WHO, 2011), 20% of the population of EU countries is exposed to traffic noise levels of above 65 dBA during the day and 30% is exposed to levels of over 55 dBA at night, which translates as a loss of 61,000 disability-adjusted life years (DALYs). For levels above these values, which are considered health-protective, many studies report statistically significant associations between exposure to noise and cardiovascular diseases (Niemann et al., 2006; Coghlan, 2007; Van Kempen et al., 2002; Davies et al., 2005; Schwela at al., 2005; Stansfeld et al., 2005), respiratory system diseases (Niemann et al., 2006; Ising et al., 2003, 2004) and connective tissue diseases (arthritis) (Niemann et al., 2006). According to a recent meta-analysis, traffic noise would rank among the four environmental factors having the greatest health impact and causing anywhere from 400 to 1500 DALYs per million population in Europe (Hänninen et al., 2014).

It is estimated that 3% of cases of ischaemic heart disease in large cities are attributable to road traffic noise (Babisch, 2008). There are ever more studies that point to a significant association between urban noise and severe cardiovascular events, such as myocardial infarction and stroke (Babisch, 2006; Selander et al., 2009; Sørensen et al., 2011). The risk factors which are directly related to cerebrovascular accident are hypertension, arteriosclerosis and low heart-rate variability index.

Furthermore, the degree of acceptance of environmental noise is determined by psychosocial and demographic aspects, such as time of residence, sensitivity, attitude and personality (Paunovic et al., 2009; Guski, 1999). This response is channelled by the limbic system to the hypothalamus, in an endocrine process that culminates in the adrenal cortex with the release of cortisol. Exposure to nocturnal noise produces sleep interruptions and electroencephalography (EEG) arousal, which cause a lack of deep, repairing slow-wave sleep (SWS), as well as affecting rapid eye movement (REM) sleep (Eberhardt, 1988; Belojevic et al., 1997). A reduction in the time of repairing sleep causes an increase in cortisol levels on the following day (Leproult et al., 1997; Vgontzas et al., 1999, 2003; Spiegel et al., 2003; Ising et al., 2004). All studies report a generalised long-term inability on the part of individuals to adapt to nocturnal noise, which may lead to chronification of cortisol overproduction (Maschke, 2002, 2003; Ising and Ising, 2002). Hypercortisolaemia is associated with the development or exacerbation of atherosclerosis. Indeed, cortisol activates the adipose tissue metabolism in order to increase the supply of energy to the body in response to stress. Lypolisis of triglycerides

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increases the quantity of fatty acids in the arteries, favouring the irreversible accumulation of plaques that increase the risk of cerebrovascular accident due to ischaemia or thrombosis (Samra et al., 1998, Spreng, 2000a, 2000b).

Lastly, one of the most surprising effects of exposure to noise, and one that has been investigated for some years now, is the increased risk of respiratory diseases among children. While a study undertaken in 2002 in the city of Madrid concluded that noise was a risk factor for daily all-cause emergency hospital admissions (Tobias et al., 2001) across all age groups, another undertaken in 2006 reached the same conclusion for children under the age of 10 years in respect of hospital admissions due to all causes, respiratory diseases and, more specifically, pneumonia (Linares et al., 2006). In other studies, bronchitis and other respiratory symptoms, such as bronchial asthma, were associated with the sensation of noise annoyance, both severe and moderate, as well as sleep disturbances caused by nocturnal noise, particularly in children (Niemann et al., 2006; Ising et al., 2004).

Health outcomes deriving from long-term exposure to noise include both cardiovascular and respiratory events, a phenomenon that has been well documented by cohort studies undertaken in recent years (Hart et al., 2013), but short-term studies which link traffic noise to short-term mortality due to the above-described causes are practically non-existent. Recent research conducted in the city of Madrid links short-term circulatory (Tobías et al., 2014a) and respiratory mortality (Tobías et al., 2014b) in the over-65 age group to traffic noise, regardless of the effects of chemical air pollution. The above association was quantified using relative risk (RR), rather than an indicator that might give an idea of the real impact on population health, such as assessment of the direct impact on annual mortality. This study now calculates the impact on annual mortality attributed to traffic noise among subjects aged over 65 years and compares it to $PM_{2.5}$-related mortality, which is the indicator of traffic-related chemical pollution recommended by the WHO for ascertaining health impacts (WHO, 2005).

2. Material and methods

2.1. Context and study population

The city of Madrid is a densely populated metropolitan area situated in the central region of Spain. In the period 2003–2005, it had a mean population of 3,099,834, and of this total, 2,507,563 (80%) were aged under 65 years (National Statistics Institute/*Instituto Nacional de Estadística*); both groups formed the target population for this study. The vehicle fleet exceeded 2 million vehicles, which had a mean daily intensity of 2.4 million cars that peaked in May (2.5 million) and fell to a minimum in August (1.7 million), and a mean speed of 24 km/h according to the Madrid City Council's Statistical Yearbook (*Anuario Estadístico*). Road traffic is the principal source of environmental noise, accounting for 80% of exposure; other sources are industry (10%), railways (6%) and leisure activities (4%) (Díaz et al., 2003).

2.2. Mortality data

Daily mortality data for the period 2003–2005 were furnished by the Madrid Regional Revenue Authority (*Consejería de Hacienda de la Comunidad de Madrid*.http://www.madrid.org/). These data comprised two time series of 1096 entries: the one for mortality due to cardiovascular causes (International Classification of Diseases-9th Revision (ICD-9): 390-459), and the other for mortality due to respiratory causes (ICD-9: 460-519). The age group targeted was that aged 65 years and over.

2.3. Noise-level data

Eighty per cent of all urban noise comes from road traffic, and specifically from automobile internal combustion engines and vehicle friction with ground and air. Noise levels were recorded by the Permanent Acoustic Pollution Monitoring Grid (*Red Fija de Control de la Contaminación Acústica*.http://www.mambiente.munimadrid.es/). This network consists of six urban background stations background stations across the city that capture environmental noise data in real time, these being in turn transmitted to a central station for their processing. Technically, the measuring process involves the following steps: (a) an outdoor anti-bird omnidirectional microphone, provided with wind screen, captures the data; (b) the captured signal connects with a statistical noise analyser; latest stations' 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); (d) the central station is equipped with a distributor adapted to ISDN that communicates with all stations at set intervals to send the data. For study purposes, three different measures of noise were used:

LeqD: equivalent diurnal noise level (8–22 h) in dBA.

LeqN: equivalent nocturnal noise level (22–8 h) in dBA.

Leq24: equivalent daily noise level (0–24 h) in dBA.

Measurements made by automatic measurement stations from Monitoring Network Air Quality Madrid City Council. Each monitor's daily level (LeqD, LeqN and Leq24) was recorded and then a citywide average was calculated from all monitors for a given day during the study period.

2.4. Calculation of impact on mortality

The health impact of noise -in terms of the number of avoidable deaths- is estimated by *risk of death attributable to noise (atribuible al ruido/RA)* for an increase q in the average noise level:

$$RA = \frac{Me - Mo}{Me} = 1 - \frac{Mo}{Me}$$

where Me and Mo are the mortality rates in periods with and without exposure to an increase q in the noise level respectively. The Me/Mo ratio can be replaced by $(RR)^q$, the value of the relative risk (RR) shown in the Poisson regression in previous publications (Tobías et al., 2014a, 2014b) for any increase in the noise level (q), such that:

$$RA = 1 - \frac{1}{RR^a}$$

Furthermore, $RA \times Me = Me - Mo$ is the mortality rate due to an increase q in the noise level. Given that noise enters the Poisson regression as a continuous variable, every day in the study period is a day with risk; thus, Me can be replaced by M, the annual mortality rate. After multiplying by the total population, P, the expression $RA \times M \times P$ is as follows:

$$\textit{Annual deaths due to an increase q in the noise level} = \left(1 - \frac{1}{RR^q}\right)MP$$

where M is the crude mortality rate among persons aged ≥ 65 years due to cardiovascular (ICD-9: 390-459) and respiratory (ICD-9: 460-519) causes, and P is the population at risk, with both M and P being sourced from municipal registry data.

To compare the results for noise- and air pollution-related mortality, the impact was calculated for comparable noise and $PM_{2.5}$ values. The RR values for $PM_{2.5}$ and for noise were obtained from previous studies undertaken in the city of Madrid for the same target period (Tobías et al., 2014a, 2014b). In the cited papers, we fitted over-dispersed 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 (Lu et al., 2008). Models were also adjusted for temperature and humidity, using one temperature average to control for the immediate effects dominated by heat (the average on the exposure day and the day before) and a second temperature average to control for effects of lower temperatures at longer lags (the average on the second to fourth days before the exposure) by using, for both, natural cubic splines with 3 degrees of freedom, and a five-day average humidity (day of exposure and four 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.

3. Results

Table 1 shows the descriptive statistics for the variables considered in the study and Fig. 1 depicts the histograms and box plot.

WHO guideline values (WHO, 2000) were exceeded on 52% of days and 100% of nights across the period analysed.

In order to ensure that the impacts of noise and $PM_{2.5}$ were comparable, an increase of $q_{LeqD}=1$ dBA was considered for the average diurnal noise level. For the purposes of comparison, annual deaths attributable to an increase of $q_{PM2.5}=10\ \mu g/m^3$ in $PM_{2.5}$ concentrations were also calculated. These increases were chosen such that the proportion q_{LeqD}: $q_{PM2.5}$ was comparable to the proportion of interquartile ranges, RI_{LeqD}: $RI_{PM2.5}$, which is approximately 1:10, thus ensuring comparability of results.

In the period 2003–2005, the mean population aged ≥ 65 years totalled $P=592{,}271$. In the Table 2 and taking the above-described methodology into account, if the mean annual LeqD value were to decline by 1 dB(A), 284 (31, 523) annual deaths due to cardiovascular causes and 184 (0, 356) annual deaths due to respiratory causes would be avoided in the over-65 age group, with the effect on cardiovascular mortality being greater than that on avoidable respiratory mortality. The number of avoidable deaths attributable to $PM_{2.5}$ is a little bit smaller for cardiovascular causes and somewhat larger for respiratory causes (within comparable confidence intervals).

4. Discussion

Despite having the same source, noise levels and air pollutants in Madrid have similar and independent effects on health (Tobías et al., 2014a, 2014b). As Table 2 showed, if avoidable mortality attributable to noise is compared to avoidable $PM_{2.5}$-related mortality, it will be observed that both figures are very similar in the case of cardiovascular cause mortality and that the effect of noise on respiratory mortality was practically double that attributable to $PM_{2.5}$, though these differences did not prove statistically significant at $p<0.05$.

This contrasts with the results reported by Hänninen et al. (2014), in which the estimates for noise levels were markedly lower than those for particulate matter. This could be due to the different methods of assessing health effects, using long periods of exposure instead of short-term effects (Tobías et al. 2014a, 2014b). In addition, noise levels in Madrid are far higher than those reported by Hänninen et al. (2014). The average diurnal noise level during the study period was 65 dBA (range 62.1–67.3), exceeding WHO health protection values on 52% of days and 100% of nights. In the case of air pollution, the WHO $PM_{2.5}$ guideline value set at a daily mean of 25 μg/m3 (WHO, 2006), was exceeded on 20% of days, whereas in the case of noise, the WHO LeqD guideline value was exceeded on 55% of occasions over the course of the study period.

Furthermore, the results obtained in this study tend to question the WHO health protection threshold values. Although the mean LeqD value obtained in this study, 65.1 dB(A)), was very similar to the value of 65 dB(A) set by the WHO (WHO, 2000), there was nevertheless noise-attributable mortality due to both circulatory and respiratory causes. Other studies undertaken on the short-term impact of noise on hospital admissions in the city of Madrid, have shown that there is a linear relationship with emergency admissions, subject to no threshold, in both the childhood (Linares et al., 2006) and general populations (Tobias et al. 2001) for levels under 65 dB(A). These results are consistent with those found in a systematic review (Babisch, 2006, 2008). In the association between diurnal noise (LeqD) and myocardial infarction, the value of 60 dBA is classified as NOAEL (No Observed Adverse Effect Level), yet in view of the above evidence of a health impact for LeqD values below the those set by WHO, we feel that these values should be updated by lowering the health protection values.

The results reported by Hänninen et al. (2014), read in conjunction with our recent findings in Madrid (Tobías et al. 2014a, 2014b) on the short-term effects of traffic noise on daily mortality, show that environmental noise should be considered a major pollutant. Hence, control of traffic noise should be a top priority in public health policy and environmental decision-making. Some preventive measures would be fairly easy to implement, e.g., a decrease of 0.5 dBA could be achieved if 12% of the fleet of cars were electric vehicles (Warburg et al., 2014). Additionally, we feel that, in order to obtain the real impact of traffic noise on health, both short- andlong-term effects should be considered (as in the case of $PM_{2.5}$).

Lastly, it should be noted that the main limitation of this study lies in its being ecological, inasmuch as it was based on spatially aggregated data, so that a given exposure to a noise level in a specific place cannot be associated with a given individual who was situated in that same place and died one or two days afterwards due to cardiovascular or respiratory causes. Nevertheless, part of this limitation is attenuated by the fact that the data were disaggregated in time, i.e., daily data were used, not only for noise levels, but also for deaths due to both causes, thereby avoiding the well-known ecological fallacy inherent in this type of design (the correlation between individual variables is deduced from the correlation of the variables collected for the group to which those individuals belong).In case-crossover design, the levels of individual exposures are not linked directly with the frequency of the disease at individual level. The daily occurrence of health

Table 1
Summary statistics for cardiovascular and respiratory mortality, noise exposure levels and $PM_{2.5}$ concentrations in Madrid 2003–2005.

	Mean	(SD)	Min.	Max.	Range	Kurtosis
Cardiovascular mortality (ICD9: 390-459)						
Overall	18.7	(5.4)	5	40	5–40	0.5/3.3
≥65 years	17.1	(5.1)	5	36	5–36	0.5/3.1
Respiratory mortality (ICD9:460-519) Overall	8	(4.4)	0	32	0–32	1.3/6.3
≥65 years	8.9	(4.2)	0	31	0–31	1.3/6.1
Noise levels (in dB(A))						
Diurnal (leqd)	65.1	(0.8)	62.1	67.3	62.1–67.3	−0.5/3.2
Night-time (leqn)	60.5	(0.9)	58.7	71.0	58.7–71.0	2.7/27
Daily (leq24)	64.1	(0.7)	61.5	66.4	61.5–66.4	−0.3/3.5
Air pollutants (in μg/m^3)						
$PM_{2.5}$	19.1	(8.6)	5.0	71.0	5–71	1.3/6.3

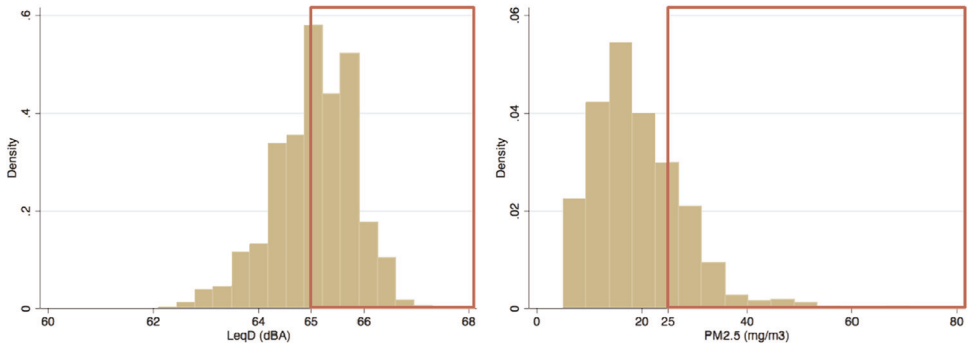

Fig. 1. Distribution of LeqD and $PM_{2.5}$ levels during the study period. The red box shows the WHO thresholds for public health protection.

Table 2
Health impact assessment of noise exposure levels and $PM_{2.5}$ concentrations in number of annual avoidable deaths for cardiovascular and respiratory causes in people aged ≥ 65 (period 2003–2005).

	M	q	RR^q(IC 95%)	Number of annual avoidable deaths (IC 95%)
Cardiovascular Mortality (ICD9: 390-459) ≥ 65 years	0.0105			
Leqd		1 dBA	1.048 (1.005, 1.092)	284 (31, 523)
$PM_{2.5}$		10 μg/m^3	1.041 (1.020, 1.062)	245 (122, 363)
Respiratory mortality (ICD9:460-519) ≥ 65 years	0.0055			
Leqd		1 dBA	1.060 (1.000, 1.123)	184 (0, 356)
$PM_{2.5}$		10 μg/m^3	1.030 (1.000, 1.062)	95 (0, 190)

RR^q (IC 95%): Relative risk for an increment *q* of daily diurnal noise level exposure and $PM_{2.5}$ daily mean concentrations; *M*: Annual crude mortality rate of people aged ≥ 65.

events, in this case mortality, reflects acute exposures with daily averages concentrations of noise and air pollutants. The variables that act as potential confounders among individuals, but not in time, can not confuse the relationship

The results obtained in this study highlight the importance posed by traffic noise to the health of citizens in large cities. The associations found with short-term mortality due to both cardiovascular (Tobías et al., 2014a) and respiratory causes (Tobías et al., 2014b), as seen from their attributable RRs, become more relevant if these results are translated into health impact by reference to the number of annual noise-related deaths. The comparison to mortality attributable to chemical air pollution due to $PM_{2.5}$ brings the health impact of both pollutants produced by traffic into perspective, and serves to highlight the imperious need to implement noise-abatement measures in major centres. The fact that it is the over-65 age group on which the impact is felt lends even greater relevance to the problem, in the light of the trend towards population ageing observed in large cities.

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