



The short-term association of road traffic noise with cardiovascular, respiratory, and diabetes-related mortality

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ABSTRACT

Background: Road traffic noise has well-documented effects on cardiovascular, respiratory, and metabolic health. Numerous studies have reported long-term associations of urban noise with some diseases and outcomes, including death. However, to date there are no studies on the short-term association between this pollutant and a set of various specific causes of death.

Objectives: To investigate the short-term association of road traffic noise with daily cause-specific mortality.

Methods: We used a time-stratified case-crossover design with Poisson regression. Predictor variables were daytime, nighttime, and 24-h equivalent noise levels, and maximum daytime and nighttime noise levels. Outcome variables were daily death counts for various specific causes, stratifying by age. We adjusted for primary air pollutants (PM_{2.5} and NO₂) and weather conditions (mean temperature and relative humidity).

Results: In the ≥ 65 age group, increased mortality rates per 1 dBA increase in maximum nocturnal noise levels at lag 0 or 1 day were 2.9% (95% CI 1.0, 4.8%), 3.5% (95% CI 1.1, 6.1%), 2.4% (95% CI 0.1, 4.8%), 3.0% (95% CI 0.2, 5.8%), and 4.0% (95% CI 1.0, 7.0%), for ischemic heart disease, myocardial infarction, cerebrovascular disease, pneumonia, and COPD, respectively. For diabetes, 1 dBA increase in equivalent nocturnal noise levels at lag 1 was associated with an increased mortality rate of 11% (95% CI 4.0, 19%). In the < 65 age group, increased mortality rates per 1 dBA increase in equivalent nocturnal noise levels at lag 0 were 11% (95% CI 4.2, 18%) and 11% (95% CI 4.2, 19%) for ischemic heart disease and myocardial infarction, respectively.

Conclusion: Road traffic noise increases the short-term risk of death from specific diseases of the cardiovascular, respiratory, and metabolic systems.

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1. Introduction

An issue of increasing concern in urban environments is the health impact of road traffic noise, given the large exposed population and the long exposure time-periods (Tobías et al., 2015b). Some 20% of the EU population is exposed to noise levels higher than 65 dBA in the daytime, and 30% to levels higher than 55 dBA in the night-time, which are considered health protection values (WHO, 2011). For such noise levels, a number of studies have reported significant associations with cardiovascular diseases

(Banerjee et al., 2014; Argalášová-Sobotová et al. 2013; Sørensen et al., 2014), respiratory diseases (Niemann et al., 2006; Ising et al., 2003, 2004), type 2 diabetes mellitus (Sørensen et al., 2013), and more recently, severe depressive symptoms (Orban et al., 2016) and adverse birth outcomes (Díaz and Linares, 2016).

According to a recent meta-analysis (Hänninen et al., 2014), road traffic noise ranks among the four environmental risk factors with highest health impact in European countries, which means a loss of 400 – 1500 healthy life years due to ischemic heart disease per million people. The impact of urban noise on public health (8% of the environmental burden of disease) was rated medium-high, comparable to that of secondhand smoke and radon, and only behind fine particles (PM_{2.5}).

Prospective studies (Selander et al., 2013; Babisch et al., 2005; Sørensen et al., 2012) have reinforced the hypothesis of the long-term association between road traffic noise and the incidence of myocardial infarction in large cities, especially in the over-65 age

Abbreviations: COPD, Chronic obstructive pulmonary disease; Leqd, Diurnal equivalent noise level; Leqn, Nocturnal equivalent noise level; Leq24, 24-h equivalent noise level; Ldmax, Maximum diurnal noise level; Lnmax, Maximum nocturnal noise level

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group. In two cohorts (Gan et al., 2012; Sørensen et al., 2011), increased daily noise levels were associated with increased mortality from ischemic heart disease and stroke, respectively, after adjustment for air pollutants.

Unlike the case of cardiovascular disease, there are few studies dealing with respiratory outcomes, even fewer those considering objective noise as a predictor variable. Two pioneering studies focusing on children and nocturnal road traffic (Ising et al., 2003, 2004) found a dose-response relationship between traffic load and the prevalence of asthma and chronic bronchitis, suggesting an important combined effect of noise and air pollution. Yet the correlation between noise levels and NO₂ concentration was very high and thus confounding could not be ruled out. Afterwards the LARES study (Niemann et al., 2006) reported significant risks of bronchitis in children highly annoyed with road traffic noise, but no association was found for asthma. Owing to their cross-sectional design, the above studies could not demonstrate one-way causality.

The association of road traffic noise with the incidence of type 2 diabetes has recently been investigated in a cohort (Sørensen et al., 2013); the results were significant only for the over-65 age group.

There are few ecological, aggregated-data studies in current environmental epidemiology. Nonetheless, when data are geographically disaggregated such studies provide support for long-term associations between urban pollutants and health outcomes (Halonen et al., 2015). On the other hand, when data are time-disaggregated, as in the case of time series, short-term associations can be investigated. Two studies of this type assessed the short-term association between road traffic noise and cardiovascular and respiratory morbidity; among the respiratory outcomes, a significant association was found for pneumonia, but not for bronchitis (Tobías et al., 2001; Linares et al., 2006). As regards cardiovascular, respiratory and diabetes-related mortality, to date only three time-series studies have investigated their short-term association with road traffic noise (Tobías et al. 2015c, 2014, 2015a), yielding significant results only for the over-65 age group.

Biological plausibility for the association of noise with cardiovascular, respiratory, and metabolic health outcomes has recently been documented in a review and summarized in an integrative model (Recio et al., 2016). Stress caused by noise may give rise to a variety of physiological reactions intended to preserve the homeostasis. When stress is considerably high and maintained, allostatic overload may lead to inefficient body responses due to overactivation of the sympathetic-adrenal-medullary and hypothalamic-pituitary-adrenocortical axes, affecting blood pressure, heart rate variability, the immune system, and the connective tissue, and promoting fat accumulation in the arteries, blood clotting, endothelial dysfunction, systemic inflammation, destabilization of atherosclerotic plaques, and insulin resistance. Some mechanisms may operate in the long- as well as the short-term, resulting in chronic or acute health outcomes, or even a concurrence of the two. Long ago, Maclure (1991) posed the possibility of acute health outcomes as a result of point exposure to an environmental stressor, considering induction time-periods of a few minutes up to a few days from the acute exposure until the adverse outcome. Modern environmental epidemiology has long used time-series analysis as a suitable strategy for the study of short-term effects of urban pollutants, allowing for different induction time-periods or lags (Maté et al. 2010).

Madrid is one of the few cities in the world provided with a monitoring network that stores real-time daily sound levels, which enables accurate examination of the relation of noise to morbidity and mortality in the short term. Conceived as an extension of the studies already published on the association between daily road-traffic noise levels in Madrid and the risk of

death from cardiovascular, respiratory, and diabetes-related outcomes (Tobías et al., 2014, 2015a, 2015c), this study extends the period of follow-up – seven years instead of three –, the number of predictor variables – with maximum noise levels added –, and the number of dependent variables, in order to assess the differences in the short-term effects of road traffic noise on the following specific causes of death: ischemic heart disease, myocardial infarction, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), pneumonia, asthma, and diabetes. Age stratification at the cutoff point of 65 years is especially relevant in this study, since many cardiovascular, respiratory, and metabolic endpoints may occur as a consequence of interaction between long-term health decline and short-term exposure to a variety of risk factors such as environmental noise.

2. Methods

2.1. Setting

Madrid is a dense metropolitan area with a mean population of 3,154,033 in the period 2003–2009, 19% over 65 years of age. The average daily traffic intensity was 2.4 million motor vehicles, reaching the maximum in May (2.5 million) and the minimum in August (1.7 million), with a mean speed of nearly 24 km/h. The main outdoor noise source is road traffic (80% of the overall noise exposure); other sources are industry (10%), rail traffic (6%), and leisure activities (4%) (Díaz et al., 2003).

2.2. Mortality data

Daily mortality records for the period 1 January 2003 – 31 December 2009 were obtained from the Madrid Regional Inland Revenue Department. Then time-series for the following causes of death were constructed: cardiovascular disease (International Classification of Diseases 10: I00–I99), respiratory disease (ICD 10: J00–J99), ischemic heart disease (ICD 10: I20–I25), myocardial infarction (ICD 10: I21), cerebrovascular disease (ICD 10: I60–I69), pneumonia (ICD 10: J12–J18), COPD (ICD 10: J40–J44, J47), asthma (ICD 10: J45–J46), and diabetes (ICD 10: E10–E14). In order to conduct age-stratified analysis, we constructed two time-series for every cause of death: one for the under-65 age group and another one for the over-65 age group. This choice was motivated by the fact that those over 65 years are especially vulnerable to the diseases and adverse outcomes studied (Nichols et al., 2014); also, setting the cut point at 65 years enables comparability with other studies.

2.3. Noise exposure

Hourly equivalent noise levels were obtained from the Madrid Noise Pollution Monitoring Grid. This network consisted of 26 urban background stations in 2003–2009, 4 m above ground level in compliance with the Directive 2002/49/EC (2002) of the European Parliament and strategically allocated to be representative of the noise levels across the city (Supplemental Fig.1). Technically, the measuring process involves the following steps: (a) an outdoor omnidirectional microphone, provided with wind screen, captures the data; (b) the captured signal connects with a statistical noise analyser which allows 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.

In order for the hourly noise levels to be representative of the

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