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Long term effects of traffic noise on mortality in the city of Barcelona, 2004–2007

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ABSTRACT

Numerous studies showing statistically significant associations between environmental noise and adverse health effects already exist for short-term (over one day at most) and long-term (over a year or more) noise exposure, both for morbidity and (albeit to a lesser extent) mortality. Recently, several studies have shown this association to be independent from confounders, mainly those of air pollutants. However, what has not been addressed is the problem of misalignment (i.e. the exposure data locations and health outcomes have different spatial locations). Without any explicit control of such misalignment inference is seriously compromised.

Our objective is to assess the long-term effects of traffic noise on mortality in the city of Barcelona (Spain) during 2004–2007. We take into account the control of confounding, for both air pollution and socioeconomic factors at a contextual level and, in particular, we explicitly address the problem of misalignment.

We employed a case-control design with individual data. We used deaths resulting from myocardial infarction, hypertension, or Type II diabetes mellitus in Barcelona between 2004 and 2007 as cases for the study, while for controls we used deaths (likewise in Barcelona and over the same period of time) resulting from AIDS or external causes (e.g. accidental falls, accidental poisoning by psychotropic drugs, drugs of abuse, suicide and self-harm, or injuries resulting from motor vehicle accidents). The controls were matched with the cases by sex and age.

We used the annual average equivalent A-weighted sound pressure levels for daytime (7–21 h), evening-time (21–23 h) and night-time (23–7 h), and controlled for the following confounders: i) air pollutants (NO_2, PM_{10} and benzene), ii) material deprivation (at a census tract level) and iii) land use and other spatial variables. We explicitly controlled for heterogeneity (uneven distribution of both response and environmental exposures within an area), spatial dependency (of the observations of the response variables), temporal trends (long-term behaviour of the response variables) and spatial misalignment (between response and environmental exposure locations). We used a fully Bayesian method, through the Integrated Nested Laplace Approximation (INLA). Specifically, we plugged the whole model for the exposure into the health model and obtained a linear predictor defined on the entire spatial domain. Separate analyses were carried out for men and for women.

After adjusting for confounders, we found that traffic noise was associated with myocardial infarction mortality along with Type II diabetes mellitus in men (in both cases, odds ratios (OR) were around 1.02) and mortality from hypertension in women (ORs around 1.01). Nevertheless, only in the case of hypertension in women, does the association remain statistically significant for all age groups considered (all ages, $\geq$ 65 years and $\geq$ 75 years).

1. Introduction

Environmental noise poses a very significant risk to human health and road traffic has been identified as the major source of noise exposure. It is estimated that every year in Europe there are

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at least 10,000 premature deaths due to exposure to noise (European Environmental Agency (EEA), 2015). In fact, the European Union's Environmental Noise Directive (European Union, 2002) defines a threshold of 55 decibels (dB) for day, evening and night periods (in order to reduce 'annoyance') and of 50 dB for night periods (in order to reduce sleep disturbance). However, the levels at which adverse effects occur could be even lower. According to the World Health Organization (WHO) (World Health Organization, 2009), there is a night noise guideline (NNG) threshold of 40 decibels (dB) which, when exceeded, is considered as having an adverse effect on health. The European Environmental Agency (EEA) points out that over 83 million Europeans are exposed to levels of noise greater than 50 dB at night, mainly as a consequence of road traffic (European Environmental Agency (EEA), 2014). This traffic-related noise in Western Europe has resulted in the loss of, at least, one million healthy life years every year (WHO (World Health Organization), 2011). A recent meta-analysis indicates traffic noise to be among the four environmental factors that have the greatest impact on health, causing between 400 and 1500 disability-adjusted life years (DALY) per million in Europe (Hänninen et al., 2014).

There are already numerous studies showing statistically significant associations between environmental noise and adverse health effects, both for morbidity (World Health Organization, 2009; WHO (World Health Organization), 2011; Hänninen et al., 2014; Perron et al., 2012; Ising and Braun, 2000; van Kempen et al., 2002; Schwela et al., 2005; Stansfeld et al., 2005; Niemann et al., 2006; Babisch, 2008, 2006; Selander et al., 2009a; de Kluizenaar et al., 2007; Babisch and van Kamp, 2009; Sørensen et al., 2011a, 2011b, 2014, 2013; van Kempen and Babisch, 2012; Babisch et al., 2014a, 2014b; Foraster et al., 2014; Fuks et al., 2011; Dratva et al., 2012; Ising et al., 2003, 2004a, 2004b; Linares et al., 2006; Tobias et al., 2001; Eriksson et al., 2014; Schell et al., 2006; Dadvand et al., 2014; De Roos et al., 2014; Halonen et al., 2015) and (to a lesser extent) mortality (Halonen et al., 2015; Gan et al., 2012; Kihal-Talantikite et al., 2013; Tobías et al., 2015a, 2015b, 2014). These health effects not only include sleep disturbance, (World Health Organization, 2009; WHO (World Health Organization), 2011; Perron et al., 2012) and/or psychological stress (Hänninen et al., 2014), but also other adverse health effects including cardiovascular disease (WHO (World Health Organization), 2011; van Kempen et al., 2002; Babisch, 2008, 2006; Selander et al., 2009a, 2014; Tobias et al., 2001; Sørensen et al., 2011, 2014; Halonen et al., 2015) or mortality (Halonen et al., 2015; Gan et al., 2012; Tobías et al., 2015b) as well as an increase in cardiovascular risk factors such as hypertension (de Kluizenaar et al., 2007; Babisch and van Kamp, 2009; Sørensen et al., 2011; van Kempen and Babisch, 2012; Babisch et al., 2014a, 2014b; Foraster et al., 2014; Fuks et al., 2011; Dratva et al., 2012). Furthermore, respiratory diseases (Ising et al., 2003, 2004a, 2004b; Linares et al., 2006; Tobias et al., 2001) and mortality (Tobías et al., 2014), along with Type II diabetes, morbidity (Sørensen et al., 2013; Eriksson et al., 2014) and mortality (Tobías et al., 2015a) and adverse pregnancy outcomes such as low birth weight (Schell et al., 2006; Dadvand et al., 2014).

There is increasing evidence of a significant association between ambient noise and serious cardiovascular events such as ischaemic heart disease (World Health Organization, 2009; Stansfeld et al., 2005; Babisch, 2008; Selander et al., 2009a; Gan et al., 2012) and stroke (Sørensen et al., 2011, 2014; Halonen et al., 2015). Furthermore, recent meta-analyses suggest a very likely causal relationship between hypertension and aircraft (de Kluizenaar et al., 2007; Babisch and van Kamp, 2009) and/or road traffic noise (van Kempen and Babisch, 2012).

The pathophysiological mechanisms explaining such associations could well be related to the response to environmental noise. This response is forwarded to the hypothalamus through the limbic system in an endocrine process culminating in the release of adrenaline, norepinephrine and, more importantly, cortisol in the adrenal cortex. Exposure to noise causes disruptions to night sleep and awakens electroencephalogram effects, causing a shortage of deep and restful SWS (slow-wave sleep) sleep and disrupting REM (rapid eye movement) sleep (Belojevic et al., 1997). Decreased restful sleep time results in an increase in cortisol levels the following day (Belojevic et al., 1997; Vgontzas et al., 1999; Ising et al., 2004a). All of the studies observed individuals' general maladjustment to long-term night noise, which may lead to chronicity in an overproduction of cortisol (Ising and Ising, 2002; Maschke et al., 2002, 2003).

Hypercortisolemia is associated with the development of atherosclerosis. In response to stress, cortisol activates the metabolism of adipose tissue in order to increase the available energy in the body. Lipolysis of triglycerides increases the amount of fatty acids in the arteries, favouring the irreversible accumulation of plaques that, in turn, increase the risk of a cardiovascular event through ischaemia or thrombosis (Samra et al., 1998; Spreng, 2000a, 2000b). It is also well known that hyperglycaemia resulting from the overproduction of cortisol can lead to insulin resistance and thus increase the risk of developing Type II diabetes (Sørensen et al., 2013; Eriksson et al., 2014; Tobías et al., 2015a). High noise levels activate the body's sympathetic nervous system, increasing blood pressure, blood viscosity and vasoconstriction, all of which leads to increased heart rate and blood lipids (Haralabidis et al., 2008; DKV, 2012). Furthermore, it has been reported that, in children, an increase of cortisol concentration activates the hypothalamus–pituitary–adrenal axis, leading, in the long term, to an aggravation of respiratory diseases (Ising et al., 2003, 2004a, 2004b),.

For almost all of these adverse health events, close to 50% of the studies made have focused on the effects of short-term (essentially exposure over a day maximum) and the other fifty centred on the long-term (exposure over a year or more) effects (Niemann et al., 2006; Selander et al., 2009a; Sørensen et al., 2011a, 2011b, 2014, 2013; Babisch et al., 2014a, 2014b; Foraster et al., 2014; Fuks et al., 2011; Dratva et al., 2012; Ising et al., 2003, 2004a, 2004b; Eriksson et al., 2014; Schell et al., 2006; Halonen et al., 2015; Gan et al., 2012; Kihal-Talantikite et al., 2013) of high noise levels.

However, the independent association between traffic noise and adverse health effects has been questioned by the occurrence of confounding, mainly by air pollutants (Beelen et al., 2009; Hart et al., 2013; Schwela et al., 2005). Traffic not only contributes to 80% of the environmental noise in a large city, but it is also the main source of air pollution (Díaz et al., 2003). In Babisch et al. (2014a, 2014b), for instance, mutual adjustment for $PM_{2.5}$ and traffic noise led to associations (with hypertension) which, although positive, could no longer be considered statistically significant. Likewise, Foraster et al. (2014) found inconsistent associations (with hypertension and high blood pressure) when considering outdoor traffic noise, which is probably due to a high degree of collinearity with NO_2. Sørensen et al. (2014) found indications of a combined effect of both road traffic and air pollution (NO_2) on the risk of stroke. Nevertheless, several recent studies have shown an association between adverse health events, including mortality, and environmental noise (Ising and Braun, 2000), independent of the effect of the air pollutants that are routinely measured. Niemann et al. (2006), for instance, argued that the risk of respiratory diseases in children could actually be caused by environmental noise rather than air pollution. Adverse health events include cardiovascular events such as hypertension (de Kluizenaar et al., 2007; Sørensen et al., 2011; Babisch et al., 2014a, 2014b), high blood pressure (Fuks et al., 2011; Dratva et al., 2012) and stroke (Halonen et al., 2015), respiratory diseases (Niemann et al., 2006), as well as cause-specific mortality such as cardiovascular disease (Halonen et al., 2015).