Environmental Research 146 (2016) 359–370

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Review article

# Road traffic noise effects on cardiovascular, respiratory, and metabolic health: An integrative model of biological mechanisms

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ARTICLE INFO

*Article history:*
Received 20 September 2015
Received in revised form
30 December 2015
Accepted 31 December 2015

*Keywords:*
Noise
Cardiovascular disease
Respiratory disease
Diabetes
Immune system
Psychosomatics

ABSTRACT

*Background:* Road traffic noise is a major public health issue, given the documented association with several diseases and the growing number of exposed persons all over the world. The effects widely investigated pertain to cardiovascular health, and to a lesser extent to respiratory and metabolic health. The epidemiological design of most studies has made it possible to ascertain long-term associations of urban noise with a number of cardiovascular, respiratory, and metabolic disorders and diseases; additionally, time series studies have reported short-term associations.
*Objectives:* To review the various biological mechanisms that may account for all long-term as well as short-term associations between road traffic noise and cardiovascular, respiratory, and metabolic health. We also aimed to review the neuroendocrine processes triggered by noise as a stressor and the role of the central nervous system in noise-induced autonomic responses.
*Methods:* Review of the literature on road traffic noise, environmental noise in general, psychosomatics, and diseases of the cardiovascular, respiratory, and metabolic systems. The search was done using PubMed databases.
*Discussion:* We present a comprehensive, integrative stress model with all known connections between the body systems, states, and processes at both the physiological and psychological levels, which allows to establish a variety of biological pathways linking environmental noise exposure with health outcomes.
*Conclusions:* The long- and short-term associations between road traffic noise and health outcomes found in latest noise research may be understood in the light of the integrative model proposed here.


## 1. Introduction

Non-auditory health effects of noise have been widely investigated over the past decades. Field studies provide strong evidence for risk associations and causal relationships. The biological plausibility of such associations is reasonably well documented, which leads to the consideration of noise pollution as a true risk factor for disease and, given its frequency in the population, a major public health issue (WHO, 2000).

Of interest in urban environments is road traffic noise, given the large exposed population and the long exposure time-periods. Some 20% and 30% of the EU population are exposed to noise levels higher than 65 dBA in the daytime and 55 dBA in the nighttime, respectively (WHO, 2011). For such noise levels, a number of studies have reported significant associations with cardiovascular diseases (Selander et al., 2009), respiratory diseases (Niemann et al., 2006), type 2 diabetes (Sørensen et al., 2013), and adverse birth outcomes (Díaz and Linares, 2015). Moreover, short-term associations with cardiovascular, respiratory, and diabetes-related outcomes including mortality have been found (Tobías et al., 2001, 2014, 2015a, 2015c; Linares et al., 2006).

Road traffic noise ranks second – only behind fine particles – among the nine 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 (Hänninen et al., 2014). In the city of Madrid (Spain) a health impact study reported a reduction of nearly 200 and 300 deaths per year due to cardiovascular and respiratory causes, respectively, for a 1 dBA decrease in diurnal noise levels, comparable to the death rate reduction obtained with an equivalent decrease in fine particle concentration (Tobías et al., 2015b).

*Abbreviations:* CNS, Central nervous system; ANS, Autonomic nervous system; SNS, Sympathetic nervous system; PNS, Parasympathetic nervous system; SAM, Sympathetic–adrenal–medullar (axis); HPA, Hypothalamic–pituitary–adrenocortical (axis); BP, Blood pressure; HRV, Heart rate variability; REM, Rapid eye movement; SWS, Slow wave sleep

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http://dx.doi.org/10.1016/j.envres.2015.12.036
0013-9351/

First we review the various biological mechanisms whereby exposure to environmental noise is likely to cause or aggravate cardiovascular, respiratory, and metabolic disorders. Second we briefly look at the neuroendocrine processes triggered by noise as a stressor, and their implications for autonomic balance. Third we present an integrative model comprising all reviewed psychological and pathophysiological mechanisms involved in the onset and development of noise-induced adverse effects on cardiovascular, respiratory, and metabolic health.

## 2. Methods

First we searched PubMed databases for articles with combinations of the following keywords in the title: “noise”, “cardiovascular”, “heart disease”, “atherosclerosis”, “infarction”, “stroke”, “hypertension”, “blood pressure”, “heart rate”, “diabetes”, “respiratory”, “annoyance”, and “sleep”, from January 2000 to March 2015. Then we manually searched for appropriate articles – i.e. those dealing with specific physiological or psychological mechanisms – included in the references within the primary articles, of any publication date. Over 300 articles were screened and finally 160 selected, of which 23 were animal studies (Table 1).

## 3. Noise and cardiovascular health

Over the years, evidence has grown for the hypothesis of a long-term association between road traffic noise exposure in large cities and the occurrence of ischemic heart disease and cerebrovascular disease, in the light of the results from a recent meta-analysis of cross-sectional studies (Banerjee et al., 2014) and from longitudinal studies with increasing statistical power (Babisch et al., 2005; Selander et al., 2009; Argalášová-Sobotová et al., 2013; Sørensen et al., 2011a, 2012, 2014). Among the specific health outcomes investigated are myocardial infarction and stroke.

Furthermore, time series studies have found short-term associations with both cardiovascular morbidity and mortality. Tobías et al. (2015c) reported a 6.6% increased risk of death in the elderly for a 1 dBA increase in daily noise levels, with no changes after adjusting for air pollutants.

Cardiovascular events and diseases arise as a consequence of physiological disorders of the circulatory system, which may be observed through various cardiovascular markers such as (a) blood pressure, (b) blood lipid concentration, (c) inflammatory and blood clotting factors, and (d) heart rate variability.

### 3.1. Blood pressure and hypertension

Animal experiments have revealed associations of high noise levels with significant increases in blood pressure (BP), probably as a result of structural changes in the sympathetic nervous system during the exposure (Fisher and Tucker, 1991). Recently, a laboratory study on humans using recorded road traffic noise reported significant increases in BP of 2–4 mmHg after 10 min of high level exposure (Paunovic et al., 2014); the BP increases were due to vasoconstriction and concurrent reduced cardiac flow during the exposure.

Cross-sectional studies have found significant associations of road traffic noise with BP in children and adults (Liu et al., 2014, Belojevic et al., 2008, Sørensen et al., 2011b) and with the prevalence of hypertension in adults (Chang et al., 2014). A meta-analysis concluded that the prevalence of hypertension was some 3% higher per a 5-dBA increase in diurnal noise levels (van Kempen and Babisch, 2012).

As regards incidence, some cohort studies have found significant associations between occupational noise exposure and elevated blood pressure and hypertension (Chang et al., 2013). However, the fact that the only prospective study to date dealing with road traffic noise and hypertension did not find any significant association for the incidence (Sørensen et al., 2011b) leaves the question that noise itself might cause hypertension somewhat inconclusive, yet the overall evidence points to the possibility that noise be a component cause, i.e. a cause which works in combination with other factors (whether environmental or not).

**Table 1**
Number of articles screened for each topic. The database search commands included the expression “(traffic OR transportation OR environmental OR residential OR community OR urban) AND noise” in conjunction with the terms listed below.

| Topic (keywords) | Database search | Manual search |
|---|---|---|
| “cardiovascular” OR “heart disease” OR “atherosclerosis” | 24 | 12 |
| “infarction” OR “stroke” | 13 | 5 |
| “blood pressure” OR “hypertension” | 31 | 18 |
| “heart rate” OR “cardiac” OR “autonomic” | 6 | 21 |
| “diabetes” | 3 | 7 |
| “respiratory” | 2 | 15 |
| “annoyance” | 66 | 20 |
| “sleep” | 36 | 24 |
| TOTAL | 181 | 122 |

### 3.2. Atherogenic processes and atherosclerosis

Atherosclerosis is the main pathological state responsible for cardiovascular disease. Factors associated with noise favouring the progression of subclinical atherosclerosis, or liable to trigger acute cardiovascular events because of their prothrombotic activity, are: (a) excess blood lipids, (b) inflammation of the endothelium and endothelial dysfunction, and (c) blood clotting alteration and platelet aggregation.

Atherosclerosis is a degenerative process of the vascular system characterized by thickenings of the innermost layer of arteries (the intima) as a result of a chronic inflammatory disorder. Excess blood lipids, endothelial inflammatory processes, and increased platelets and blood clotting factors interact in the development of atherosclerotic lesions (Hansson, 2005; Libby, 2013). The core of an atheroma contains lipids, activated immune cells, proinflammatory cytokines, and debris; the surrounding cap consists of connective tissue and a collagen-rich matrix, making up the atherosclerotic plaque. Proinflammatory molecules secreted by the immune cells inside the plaque increase oxidative stress and weaken its structure, which turns unstable and vulnerable. Eventually, plaque rupture takes place and causes the release of the thrombogenic core to the blood, which may lead to occlusion of the artery and acute coronary syndrome (e.g. myocardial infarction).

The mechanism by which noise might act as a proatherogenic agent has to do with cortisol overproduction – in turn associated with exposure to specific noise levels – as a result of the neuroendocrine system activation during acute or chronic stress. Stressful experiences produce changes in lipid and lipoprotein levels, as shown in human experiments (Qureshi et al., 2009). Moreover, animal studies reveal that acute psychological stress causes lipid peroxidation as a result of oxidative stress in tissues, and then such modified lipids turn into proinflammatory agents (Kovács et al., 1996; Wang et al., 2007). Studies on occupational noise – where bias due to exposure misclassification is less likely – have reported higher blood cholesterol and triglyceride concentration, as well as long-term increased risk of dyslipidemia, in workers exposed to noise levels above 80 dBA (Mehrdad et al.,