



## **Transportation noise and public health outcomes: biological markers and pathologies**

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### **ABSTRACT**

**In 2009 the World Health Organisation recommend that for the prevention of subclinical adverse health impacts related to night-time noise, the general population should not be exposed to noise levels greater than 40 dB(A). Contemporary scientific studies exploring the relationship between transport noise and health-related outcomes have served to reinforce the veracity of this recommendation. Indeed, a number of recent studies suggest that adverse impacts begin to occur at even lower levels - somewhere in the 30-39 dB(A) range. Within the foregoing context, this paper systematically reviews the contemporary academic literature in an attempt to delineate specific biological markers and pathologies associated with noise-health outcomes as a result of transportation noise exposure. In doing so, we highlight and categorise these markers specifically for a range of emerging health impacts. By highlighting such relationships, the goal is to allow other researchers to easily identify key health-related variables in national and international data sets. By utilising this data in conjunction with noise mapping data it may be possible to determine dose-effect and burden of disease relationships more accurately for a wider range of health issues in specific cities across Europe.**

### **1 INTRODUCTION**

Since the World Health Organisation (WHO) produced the 'Guidelines for Community Noise' in 1999<sup>1</sup> (WHO, 1999), there has been substantial progress in the study of non-auditory health-related outcomes associated with the impact of environmental noise on urban populations. The understanding of how, and the extent to which, environmental noise impacts on public health has advanced considerably over the past number of years. Pershagen et al.<sup>2</sup> among others emphasise how recent research, has made a significant contribution to understanding the negative effects of environmental noise pollution on health. In response to such developments, the WHO is currently

in the process of reviewing the results from existing research in order to update its recommendations regarding environmental noise in Europe. Such revision is urgently required since it is estimated that approximately 65 million people in Europe are believed to be exposed to levels of environmental noise that exceed the recommended levels<sup>2</sup>.

The four main sources of environmental noise disturbance in urban areas are road traffic noise, railway noise, aircraft noise, and industrial noise<sup>3</sup>. In terms of the severity of disturbance, the majority of research<sup>4</sup> indicates that aircraft noise causes the most severe disturbance, followed by road traffic noise, railway noise, and industrial noise. Due its prevalence, road traffic noise is the most researched environmental noise source relative to its impact on public health. About half the population of the European Union is estimated to be exposed road traffic noise that is considered to be negative for public health and well-being<sup>5</sup>. Indeed, road traffic noise is considered to be the second most prevalent environmental risk factor, after fine particle pollution, to human health in Europe.

## **2 PHYSIOLOGICAL RESPONSES TO ENVIRONMENTAL NOISE**

The non-auditory effects of environmental noise, as mediated by sleep disturbance and stress-related annoyance, causes a physiological response to stress. This physiological response to stress is generated, as an immediate stress response, by the sympathetic-adrenal-medullar (SAM) axis which produces catecholamines<sup>6</sup>, and as a prolonged stress response, by the hypothalamic-pituitary-adrenocortical (HPA) axis which produces glucocorticoids, including cortisol<sup>7</sup>.

Catecholamines include epinephrine (adrenaline), norepinephrine, and dopamine, and function as neurotransmitters, transmitting signals from neuron to neuron, and hormones, regulating a variety of physiological functions, such as those relating to the cardiovascular and respiratory systems. Catecholamines and cortisol produce energy resources and increase blood glucose, which is associated with the metabolic dysfunction that causes and exacerbates diabetes<sup>8</sup>. The overproduction of catecholamines also disrupts the ability of lymphocytes (i.e. white blood cells) to mobilise and attach to tissue. This can increase the risk of developing Non-Hodgkin Lymphoma (NHL) which originates from malignancies in the white blood cells<sup>9</sup> contributes to immune system dysfunction. Dysfunction of the immune system disrupts the body's ability to fight infection, disease, and destroy damaged cells, which may also increase the risk of breast cancer and produce negative effects on the respiratory system<sup>8</sup>. The overproduction or disruption in the normal functioning of catecholamines, as the process relates to the normal functioning of neurotransmitters, also increases the risk of developing or exacerbating psychiatric conditions such as depression or anxiety, as well as impairing cognitive function<sup>10-11</sup>.

The overproduction of cortisol, which takes place as a result of long-term stress response from prolonged exposure to environmental noise and/or disruption in recuperative sleep, leads to accumulated levels of cortisol. This process is known as hypercortisolaemia and ultimately results in atherosclerosis which is the main pathology associated with a number of cardiovascular complications including high blood pressure, hypertension, ischaemic heart disease, and stroke. Cortisol overproduction also increases the retention of visceral fat in adipose depots resulting in an increased risk for obesity, which in turn, increases the risk for diabetes<sup>12</sup>. As previously outlined, cortisol also increases blood glucose, but the HPA axis is also responsible for insulin repression<sup>8</sup>, which again increases the risk for diabetes.

## **3 EMERGING HEALTH IMPACTS OF ENVIRONMENTAL NOISE**

### **3.1 Breast and Colorectal Cancer**

Recent studies have begun to investigate the potential link between exposure to environmental noise and the increased risk for breast cancer. Such research is at an early stage and is relatively limited. Interest in developing this hypothesis has been prompted by the fact that increased rates of breast cancer are observed in highly industrialised regions<sup>13</sup>. This suggests that characteristics peculiar to urban living, and certain environmental exposures, including noise exposure, may account for this increased risk.

Sleep disturbance is one pathway in which environmental noise from transportation in metropolitan areas has potential to increase the risk for breast cancer. This is because sleep disturbance inhibits the production of melatonin which is believed to potentially reduce breast carcinogenesis in a number of ways including through the suppression of antioxidant processes and angiogenesis<sup>14</sup>. A disruption in recuperative sleep also impacts negatively on immune system repair processes, inhibiting the generation of endocrine cytokine white blood cell production, whereby the immune system's ability to destroy damaged cells is compromised<sup>15</sup>. Previous studies associating sleep disturbance with breast cancer have encouraged the hypothesis that environmental noise may increase the risk for breast cancer. Phipps et al. and Palesh et al.<sup>16-17</sup> found that sleep disturbance was associated with increased mortality rates amongst breast cancer patients. Although sleep disturbance is a side-effect of chemotherapy, breast cancer patients also report sleep disturbance before treatment and years after treatment has finished<sup>17-18</sup>.

Stress is another pathway in which environmental noise has potential to increase the risk for breast cancer. The overproduction of the glucocorticoid cortisol may increase the risk for breast cancer development and/or exacerbate previous conditions due to immune system dysfunction which incorporates a reduction in the ability to locate and repair cancer cells, disruption in DNA repair, and a disruption of BRCA1, a tumour suppressor gene responsible for repairing DNA. In a study by Pan et al.<sup>19</sup>, it was found that in a cohort of 1,378 early stage breast cancer patients, glucocorticoid production was associated with an increased risk for mortality amongst cancer patients.

In the analysis of breast cancer risk, it is appropriate to separate the disease into estrogen receptor positive (ER+) or estrogen receptor negative (ER-) tumour types. This is because the aetiology associated with each tumour type is very different. In a study examining the potential risk for exposure to road traffic and railway noise with postmenopausal breast cancer, Sørensen et al.<sup>20</sup> found that both sources of environmental noise had the potential to increase the risk for ER- tumour types. In a recent study on a cohort of 57,053 Danish participants, which included 1,759 breast cancer patients, Roswall et al.<sup>21</sup> found no association between road traffic noise and mortality, either overall, or in relation to ER+ or ER- tumour types. However, Hegewald et al.<sup>22</sup> found that exposure to aircraft noise was associated with an increased risk for ER negative breast cancer, with environmental noise from road and rail sources less evident.

### **3.2 Diabetes and Obesity**

Incidents of diabetes, including type 2 diabetes and gestational diabetes mellitus (GDM) are rising rapidly across the globe. Recent studies have begun to focus on the potential negative impact that exposure to environmental noise has on the growth of diabetes in metropolitan areas. Such research is based on the hypothesis that, since environmental noise has the potential to lead to stress and sleep disturbance, and since the overproduction of stress hormones and sleep disturbance

has the potential to lead to a higher risk for diabetes, it seems logical that exposure to environmental noise may be associated with diabetes incidence.

In physiological terms, diabetes is first of all caused by inducement of the hypothalamus-pituitary-adrenal axis (HPA axis) activity resulting in metabolic dysfunction, including cortisol overproduction and insulin suppression<sup>8</sup>. Diabetes associated metabolic dysfunction includes hyperglycemia and ketoacidosis, which is caused by noise induced increases in catecholamine and cortisol which activates energy resources and increases blood glucose levels<sup>8</sup>. Hyperosmolar syndrome, a complication of type 2 diabetes, occurs in response to the biological need to expel the excess blood glucose levels produced by the process, which is discharged through urination. The process can result in acute dehydration and ultimately death. The physiological response to sleep disturbance is considered paramount in understanding the physiological response potentially leading to the increased risk for diabetes. As well as cortisol suppression and depressed sympathetic nervous system function, slow wave sleep (SWS) is also important for controlling glucose production<sup>23</sup>. A reduction in SWS caused by environmental noise results in impaired glucose tolerance and produces poor insulin sensitivity<sup>24</sup>. Sleep disturbance also lowers leptin hormone levels and increases ghrelin hormone levels, which increase the risk for obesity and diabetes<sup>25</sup>.

Controlling for air pollution, Sørensen et al. found that exposure to road traffic noise was associated with type 2 diabetes, with higher noise levels, and the longer the period of time exposed, associated with higher risk<sup>26</sup>. In a nationwide study, Heidemann et al. found that cohorts living in the vicinity of roads with a very high volume of traffic, reported a twofold increase in the risk for type 2 diabetes, relative to cohorts living in the vicinity of low volume traffic<sup>27</sup>. Roswall et al. found a significant association between road traffic noise and increased risk for diabetes, but not in relation to rail traffic noise, which may again be related to general findings that railway noise is less annoying than other transportation sources<sup>28</sup>. Research by Tobías et al. found that, for populations over 65 years, for every 1 dB increase in night-time road traffic noise, the risk for diabetes related mortality rose by 9.4%<sup>29</sup>. While Dzhambov and Dimitrova found that exposure to road traffic noise was associated with an increased risk for gestational diabetes mellitus, and in the context of pregnancy, Ashin et al. found that road traffic noise was also related to an increase risk for gestational diabetes mellitus, defined here as a glucose intolerance occurring during the onset of pregnancy<sup>30-31</sup>.

Another avenue of research developing in relation to the association between environmental noise and metabolic complication is that associated with an increased risk for obesity. In physiological terms, the risk for obesity caused by exposure to environmental noise is associated with cortisol overproduction resulting in increased visceral fat retention in adipose depots, consisting of mesenteric, epididymal white adipose tissue, and perirenal depots<sup>12</sup>. A study by Roswall et al. found that exposure to road traffic noise was associated with physical lethargy), which increases the risk for obesity as well as diabetes<sup>28</sup>.

Obesity is a serious risk factor for the development of cardiovascular incidence and diabetes. Although Oftedal et al. found no association between road traffic noise and obesity in a general population from the city of Oslo, positive associations were found in a cohort of women who were highly sensitive to noise<sup>32</sup>. This suggests that such cohorts may be more prone to the negative metabolic complications caused by environmental noise. As discussed previously, this contrasts with the gender differentials suggesting that males are more likely to experience cardiovascular incidents in relation to noise.

In a study of 57,053 middle-aged participants, Christensen et al. found an association between road traffic noise and obesity<sup>33</sup>. However, the relationship was primarily found amongst pre-existing cases of obesity, which corresponds with analysis of pre-existing cardiovascular

conditions. In the context of railway noise, only sound levels greater than 60dBs showed a significant association. In another study analysing exposure to road traffic, railway, and aircraft noise, Pyko et al. found that transportation noise exposure was correlated with an increased risk for obesity from all three sources, with aircraft noise the strongest predictor, followed by road traffic noise, and railway noise, with correlations independent of socio-economic factors and air pollution<sup>12</sup>. Such findings are generally consistent with previous research analysing exposure to transportation noise with annoyance and sleep disturbance<sup>34</sup>. Combined source models were also found to increase the risk of obesity as they related to an increase in metabolic complication<sup>12</sup>.

In an analysis of aircraft noise, Eriksson et al. found a significant correlation between long-term exposure and an increase in waist circumference, possibly as the result of chronic stress caused by hyperactivation of the hypothalamic-pituitary-adrenal axis and impaired control of glucocorticoid receptors<sup>35</sup>. Finally, controlling for socio-economic factors, age, gender, and other transportation sources (i.e. rail and aircraft), Nicole found that all measurements of obesity were correlated with road traffic noise<sup>36</sup>. As such, every 10 dB increase in road traffic noise over a 5 year period was correlated with an increase of .35 cm in waist circumference and an increase of .18 in Body Mass Index (BMI). This association with obesity was increased if road traffic noise exposure was combined with railway noise exposure of over 60 dB.

### **3.3 Fertility including fetal, infant, and child development**

The relationship between exposure to road traffic noise and stress, as well as sleep disturbance, is well established<sup>34</sup>. The fact that both stress and sleep disturbance are known to negatively impact on fertility, promotes the hypothesis that environmental noise may also have a negative impact on fertility. Schliep et al. found a correlation between self-reported stress and decreased levels of procreative hormones, ovary function, and general fertility in females, while Lynch et al. found no such relationships<sup>37-38</sup>. However, Janevic et al. found that self-reported stress amongst males was correlated with reduced semen quality, while research by Louis et al. found significant correlations between stress and long-term pregnancy<sup>39-40</sup>. Christensen et al. found a relationship between exposure to road traffic noise and long term pregnancy of between 6 and 12 months, but not in relation to terms greater than 12 months<sup>41</sup>.

There is also an emerging literature on the relationship between environmental noise and low birth weight (LBW), small for gestational age (SGA), and preterm birth (PTB), though the extent of research is still very limited. In a meta-analysis of 29 studies, Dzhambov et al. found that pregnant women exposed to noise levels greater than 80dB were at significantly higher risk for having SGA, gestational hypertension, and babies with congenital malformations<sup>42</sup>. In an analysis of 70,000 birth records in Vancouver, Canada, Gehring et al. found that road traffic noise exposure significantly increased the risk for LBW, after controlling for socio-economic indicators and air pollution<sup>43</sup>. In the context of aircraft noise, a study of 160,460 births in Japan, found significant correlations between aircraft noise and LBW amongst the highest exposed cohorts in the town of Kadena<sup>44</sup>. On the other hand, in a study of 75,166 births in Denmark, Hjorteborg et al. found that exposure to traffic noise did not affect a new-born baby's size or weight, while in a study of 6,438 births in Barcelona, Spain, Dadvand et al. also found no significant associations<sup>45-46</sup>.

In the context of sleep disturbance and childhood development, it is intuitive that environmental noise induced sleep disturbance may be particularly problematic for children, since children require continuous recuperative sleep cycles in order to maximise physical growth, as well as cognitive development<sup>47</sup>. Skrzypek et al. found that exposure to road traffic noise was associated with an increased risk for sleep disturbance and attention disorder amongst children

aged 7 to 14 years, and Weyde et al. found that exposure to road traffic noise was correlated with inattention for children aged 3 to 8 years<sup>48-49</sup>. Hence, it seems that exposure to road traffic noise has a negative effect on the daily functions of the child<sup>49</sup>. In a cohort of children aged 7 years, Hjorteborg et al. found that exposure to road traffic noise was related to behavioural difficulties, and in particular, hyperactivity and inattention<sup>45</sup>. In a study of children between 7 and 8 years in schools within the vicinity of Frankfurt airport, an increase in aircraft noise exposure was found to be correlated with a decrease in quality of life scores, an increase in noise-related annoyance, and a decrease in reading scores<sup>50</sup>. Furthermore, Klatte et al. estimated that a 10dB increase in aircraft noise correlated with a 20% decline in reading scores, tantamount to a two month deficiency in reading speed<sup>50</sup>.

## 4 CONCLUSION

Despite the continuing necessity for extensive cross-disciplinary research, it is clear is that the negative impact of environmental noise on public health is serious, and urgently needs to be addressed, particularly in relation to the fact that the world's urban population is set to double from 3.10 billion in 2014 to 6.4 billion in 2050 (United Nations, 2014). It is well-established in the literature that there is a link between environmental noise exposure and annoyance, sleep disturbance and related health effects such as cardiovascular disease, hypertension, tinnitus among other impacts. This paper has provided an outline of the recent literature highlighting emerging public health risks and linkages from environmental noise exposure. Although the evidence base is not consistent, emerging studies do suggest tentative links between environmental noise and various cancers including breast cancer, colorectal cancer, and non-Hodgkin lymphoma. Much of this research is new and in its early stages and future work will be required to add weight to its emergence as a concrete evidence base.

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