At the heart of the matter: do we still underestimate noise effects on cardiovascular health?

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The Lancet Regional Health - Europe 2024;47: 101134
Published Online xxx
https://doi.org/10.1016/j.lanepe.2024.101134

In European populations who live increasingly in dense cities, transportation noise constitutes a growing environmental exposure and risk factor. For Europe, the European Environment Agency estimates that at least 1 million healthy life years are lost annually due to transportation noise, from sleep disturbances, annoyance, ischemic heart disease, and cognitive impairment in children.[1] Known pathways by which chronic noise exposure affects the cardiovascular system include the elicitation of oxidative stress and inflammation and an activation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in the secretion of stress hormones, vasoconstriction, blood pressure increases, and the elicitation of arrhythmias.[2]

Atrial fibrillation (AF) is a frequent health problem in our aging European populations, affecting about 2–3% of Europeans overall and almost 15% of those above 80 years of age.[3] AF leads to increased risks of thromboembolic events such as stroke, and can cause heart failure and death. In high income countries, about 1.2% of all deaths can be attributed to AF.[4] Treating patients with AF is expensive due to frequent hospitalizations and complications of the disease. It is therefore reasonable to shed light on potential modifiable risk factors to decrease the burden of disease from AF as much as possible.

In the November issue of *The Lancet Regional Health–Europe*, Thacher et al. investigated the potential relationship between long-term exposure to transportation noise and incident AF using data from 11 Scandinavian cohorts in the NordSOUND project.[5] They included more than 160,000 participants with available address history and free of AF at baseline and followed them over for about 20 years, during which almost 19,000 cases of incident AF developed. The authors investigated different sources of transportation noise, namely road traffic, aircraft and railway traffic, and calculated the day-evening-night (L_{den}) noise level as 1- and 5-year mean exposures at the most exposed façade of their residence for each participant. They also assigned residential long-term air pollution exposure to fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2) from a high-resolution dispersion model. As transportation noise and air pollution come at least partly from the same sources and can be correlated, adjustment for potential confounding is an important feature of this study.[6]

Applying Cox proportional hazards models, the authors found a 2–3% higher risk of incident AF for 10 dB higher 1- and 5-year L_{den} road traffic noise exposure, with slightly stronger associations in threshold models at 53 dB. The results did not change materially when adjusting for fine particles; however, there was some indication of confounding by NO_2, an air pollutant specifically related to road traffic. For aircraft noise, a higher, but less certain risk increase of 12% was found in those exposed to more than 50 dB L_{den} compared to those less than 40 dB L_{den}. Interestingly, no association was found for railway noise. Taking multiple noise sources into account (a still underdeveloped area of noise research), the risk of AF increased in those exposed to two or more noise sources above 45 dB compared to no exposure.

Several aspects related to the noise assessment seem important. The current study uses the time-weighted L_{den} that includes a respective 5 and 10 dB penalty for the latter two periods, calculated for all 24-h days in a one-year period. Due to the expected very high correlations between the noise metrics, an independent assessment of the effects of noise exposure at night was not possible in this study. Unfortunately, noise at the least exposed façade, shown to have stronger association with AF than the most exposed façade in the Danish Nationwide Cohort,[7] was not available for all NordSOUND cohorts and was not considered. Also, individual noise events, that may be especially noticeable at night when background noise is lower, are not reflected well with energy-based metrics such as the L_{den}.[8] It has been shown that the temporal discontinuity, represented by the so-called intermittency ratio, has an independent association with cardiovascular disease.[9] From a pathophysiological standpoint, it is very plausible that highly intermittent noise could activate the HPA axis and elicit a stress response through frequent night time arousals even at medium and low noise

DOI of original article: https://doi.org/10.1016/j.lanepe.2024.101091
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levels. The results in this study may therefore be conservative estimates of the true association, as also pointed out by the authors.

It remains to be seen whether accounting for noise at the least exposed façade and the discontinuous nature of noise, specifically from railways and aircraft,[8] will yield associations with AF (and further health outcomes) even at levels far below current guideline values.[10] More work needs to be done to identify and use the most relevant noise exposure characteristics for an efficient protection of health. As cities continue to densify, and more people are exposed to multiple transportation sources, noise mitigation measures should play an increasingly important role in urban planning.

Contributors

Both authors, Barbara Hoffmann and Danielle Vienneau, wrote the manuscript and critically reviewed it.

Declaration of interests

The authors declare no conflict of interest.

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