Guest Editorial

Noise Pollution and Arterial Hypertension: Can we Remain Mute Anymore?

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Citation: *European Cardiology Review* 2017;**12**(1):24–5. DOI: 10.15420/ecr.2017.12.1.GE4

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Although the gains of the industrial revolution have transformed human life, they have also left an adverse impact on our health and environment.¹ Therefore, factors such as environmental pollution (and pollutants) have lately been the subject of intense scientific investigation.

Traffic noise represents a major environmental burden, which has been suggested to lead to serious cardiovascular diseases.^{1–3} Studies in the field of environmental pollution indicate that only airborne particulate matter affects more people in the industrialized world than environmental noise.² The WHO has identified a threshold of >55 dB as a level of noise that is associated with health problems³ and very large numbers of individuals across the whole age spectrum are affected by traffic noise in cities in the developed world. Noise pollution above the WHO identified threshold is considered to cause more than 500 additional cases of myocardial infarction² and approximately 800 cases of stroke.⁴ Exposure to noise can increase heart rate, blood pressure, concentrations of cortisol and noradrenaline and also promote low grade inflammation, which is likely to be a key pathogenic process in the development of atherosclerosis and other major diseases.^{5,6} Noise pollution can affect sleep patterns and the latter are known to be associated with increased cardiovascular morbi-mortality.⁷ Indeed, epidemiological studies have shown that individuals who are exposed to traffic noise at night for long periods of time are more likely to suffer from atherosclerosis than subjects who live in an environment with low levels of traffic noise.⁸

Some investigators have reported an association between noise pollution (mainly derived from road traffic) and mortality from cardiovascular diseases. A recent study by Halonen et al. involving over 8 million people in London, assessed the effects of long-term exposure to road traffic noise on both all-cause and cardiovascular mortality and

on hospital admissions for cardiovascular disease.⁹ The study, which looked at both younger and older subjects, showed that long-term exposure to road traffic noise was associated with an increased risk of all-cause mortality and cardiovascular mortality and morbidity in the general population. Among this increased risk, the prevalence of stroke was particularly high in the elderly.

In this issue of *European Cardiology Review*, Münzel and Sørensen review the role of noise pollution in enhancing the risk of arterial hypertension in a scholarly and timely fashion.¹⁰ The authors showcase a multitude of individual studies and meta-analyses that suggest a positive association between noise pollution and hypertension. The authors further speculate that the association could be mediated by direct and indirect pathways (of relevance being the biological effects on autonomic nervous system, sleep physiology and endothelial function).

However, there are several limitations to available evidence about this association. First, as the authors themselves have acknowledged, the clear majority of studies cited were cross-sectional and therefore limited in their ability to inform whether the association is indeed real. Second, where prospective studies were conducted, the associations were either inconsistent or became non-significant upon adjustments for potential confounding factors. Finally, outdoor (especially traffic-related) noise pollution is intertwined heavily with air pollution and other environmental factors such as temperature and human activity, which have not been adequately and consistently accounted for, even among the largest of the epidemiological studies to date. Hence, it is yet unclear whether the observed associations are real or artefactual.

Should we therefore remain mute, both towards the science as well as the implications of the scientific findings so far? Perhaps

not – in fact, certainly not! While the existing body of evidence on the association between noise pollution and arterial hypertension is not yet strong, it is becoming increasingly obvious through parallel investigation (e.g. by the inverse association between air pollution and HDL cholesterol levels)¹¹ that adverse environmental factors likely have a major impact on human health and wellbeing. However, it is unlikely – using simplistic methodological approaches – that debates regarding causality will be resolved successfully, especially in complex exposures such as environmental risk factors.

There is an urgent need to establish and undertake large-scale, adequately powered prospective studies having the unabashed ability to borrow cross-cutting methods (e.g. principal components analysis, which is widely used in metabolomic research) to enhance the scientific veracity of findings related to complex environmental exposures. This will not only help address the causality debate, but will also provide a robust scientific framework to support key policy decisions worldwide. To this end, a recent announcement by the British Heart Foundation¹² pledging support for environmental research in relation to cardiovascular diseases, is an important step forward. ■

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