Nighttime aircraft noise exposure: flying towards arterial disease

Marietta Charakida and John E. Deanfield*

National Centre for Cardiovascular Prevention and Outcomes, Institute of Cardiovascular Sciences, UCL, London, UK

Online publish-ahead-of-print 28 August 2013

This editorial refers to ‘Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults†’, by F.P. Schmidt et al., on page 3508

Over the last four decades there has been a steady increase in air travel worldwide. The global air transport network has contributed to the growth of economies by providing better access to national and international resources, increasing tourism, encouraging investment and innovation, and improving business operations and efficiency. Air transport also offers a vital lifeline to remote communities and rapid worldwide support during emergency situations. However, along with these benefits, detrimental effects linked to the expanding air network have been recognized.

There are increasing data to suggest that air pollution and noise from airplanes have significant environmental and cardiovascular implications. Air pollutants have been associated with climate changes and increased hospitalization and mortality due to cardiovascular disease in high risk populations.\(^1\) In parallel, auditory impairment as a result of the direct effects of sound energy has been well recognized. As a result, protective legislation requiring hearing protectors as a result of the direct effects of sound energy has been well recognized.\(^2\)\(^,\)\(^3\) At night, cerebral function recovers from the rigours of daytime activities and key brain processes are restored. Aircraft noise has been associated with sleep fragmentation and an increase in stress hormone levels, and concerns have been raised regarding cognitive development of children who are brought up in areas of environmental noise.\(^4\)\(^,\)\(^5\) There are also data to suggest that aircraft noise exposure increases cardiovascular risk in individuals.

Studies examining the effects of aircraft noise on cardiovascular disease have used a broad range of outcomes ranging from self-report measures of hypertension and use of antihypertensive medications to more objective measures of blood pressure, prevalence of ischaemic heart disease, and incidence of clinical events such as myocardial infarction. The European Union-funded Hypertension and Exposure to Noise Near Airports (HYENA) study demonstrated in 4,681 middle-aged people who had lived for at least 5 years near one of the seven major European airports, a significant exposure–response relationship between night-time aircraft noise exposure and risk of hypertension (defined as blood pressure \(\geq 140/90\) mmHg or a diagnosis of hypertension by a physician in conjunction with use of antihypertensive medications). A 10 dB increase in night-time aircraft noise exposure was associated with a 14% increase in odds for hypertension.\(^6\) However, when use of antihypertensive medication was used as an outcome, the results varied in different countries.\(^7\) For instance, in the UK and the Netherlands, a 10 dB increase in nocturnal aircraft noise was associated with a 34% increased odds of taking antihypertensives, but no association was found in other European countries.\(^7\)

Apart from hypertension, associations between residential exposure to aircraft noise and mortality from myocardial infarction have also been reported.\(^8\) In 4.6 million people \(>30\) years of age who were followed up as part of the Swiss National Cohort for 5 years, the risk of fatal myocardial infarction increased by 1.3 when aircraft noise exposure was \(>60\) dB compared with \(<45\) dB.\(^9\) Length of exposure was also an important determinant, as the strongest associations were found for people who had lived in the same highly exposed location for at least 15 years.\(^8\)

The recent study by Schmidt et al. provides further evidence and potential pathophysiological mechanisms for the adverse effects of nocturnal aircraft noise exposure on the cardiovascular system.\(^9\) In a blinded field study which included 75 young healthy volunteers, they demonstrated worsening sleep quality and a trend towards reduced endothelial function responses and increased arterial stiffness, as measured by pulse transit time, with increasing duration of nocturnal aircraft noise exposure. They also demonstrated a priming effect of noise, as blunting of flow-mediated dilatation (FMD) responses was particularly evident when participants were exposed first to 30 and then to 60 noise events.

† doi:10.1093/eurheartj/ehs339

The opinions expressed in this article are not necessarily those of the Editors of the European Heart Journal or of the European Society of Cardiology.


Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2013. For permissions please email: journals.permissions@oup.com
The findings of this study are intriguing as they demonstrate, for the first time, a small direct vascular effect of nocturnal aircraft noise exposure in a low cardiovascular risk population. It is, however, important to be cautious when considering the relevance of the findings for long-term clinical outcomes as well as for understanding causal pathways to cardiovascular disease and complications. The vascular effects which were demonstrated were very small, and the FMD values and pulse wave transit times remained within what is generally considered as the normal range for all noise exposures. It will be important to examine the effects of similar levels of noise on patients with more established cardiovascular disease in future studies.

The mechanisms by which noise induces changes in arterial function and structure remain uncertain. The authors suggest that a stress response is important as circulating adrenaline levels were increased in parallel with the vascular endothelial function changes. No causal relationships were, however, established and it is noteworthy that heart rate and blood pressure levels did not rise in the manner in which has been previously described in higher risk populations. Schmidt and colleagues went on to explore potential mechanisms by which the stress response might be linked to vascular function (Figure 1). This remains a controversial area and, in particular, the direct impact of catecholamines released by stimulation of the sympathetic nervous system is unknown. It is generally accepted that catecholamines can antagonize the vasodilatory effects of endothelial-derived nitric oxide, promoting a chronic stress condition. Noise-induced endothelial responses were reversed at least in the short term by oral administration of vitamin C, implicating oxidative stress as a mediator of vascular induced changes. Reactive oxygen species can promote atherosclerosis both by direct effects on the vascular wall and by triggering a number of redox-sensitive transcriptional pathways. Many previous small studies have demonstrated that vitamin C can improve endothelial dysfunction in diseases with increased oxidative stress such as hypercholesterolaemia, long-term smoking, congestive heart failure, hypertension, and diabetes mellitus. It is noteworthy, however, that the vascular benefits from antioxidant treatment have not been confirmed in large randomized outcome clinical trials.

A number of risk factors are likely to be on the causal pathway for development of atherosclerosis and its complications. This study, however, supports the increasing awareness that our environment may also play an important role either directly or by setting up an unfavourable internal milieu for development of vascular disease. The
findings of Schmidt and colleagues are provocative in that they demonstrate unfavourable effects of noise pollution on the arterial wall in apparently healthy young subjects. This could have important public health implications. The results will need to be replicated in larger studies with longer periods of observations, as well as in higher risk populations. The World Health Organization (WHO), however, set a desirable threshold of chronic aircraft noise exposure at 40 dB. The observations of Schmidt and colleagues emphasize how the cardiovascular community needs to broaden its remit to study and manage the environment in which we live and not just care for patients with established disease if the growing worldwide burden of arterial disease is to be reversed.

Conflict of interest: none declared.

References