EDITORIAL COMMENTARY

Air traffic noise and hypertension in Stockholm County

On any given day, according to *The Economist*, over 4 million people take to the skies. A quarter of a million people are airborne at any moment.[1] Health impacts of noise can begin even before birth, and with high levels and an increasing distribution of exposure, any associations between aircraft noise and morbidity are clearly of relevance to public health.[2 3]

Rosenlund *et al* (page 769) assess associations between blood pressure and aircraft noise by comparing two study populations, around Arlanda airport and in another part of Stockholm County. Noise contours are constructed over the region for maximum noise level (MNL) and an energy averaged level (FBN).

Health outcomes of people living in proximity tend on average to be more alike than they are to those from other areas. Reasons may include "alikeness" in individual characteristics and factors at area level.[4] Statistical models assume independence between observations, but in a hierarchical study observations within areas are not independent. The alikeness also makes it difficult to ascribe health variation between areas to one particular exposure—other differences may be responsible for apparent associations.

These problems are inherent in investigations such as the current Stockholm study, which contain a geographical hierarchy of subjects within areas. One way to tackle them is to account for the confounding in the model. Another (or in combination) is to include the hierarchy in the model, using multilevel or other hierarchical/spatial approaches.[5–7] Analyses which ignore the hierarchy may be in serious error. In a methodological exercise within another hierarchical study—the CESAR air pollution study—1000 datasets were simulated with a zero air pollution effect.[8] In analyses ignoring hierarchy (but including four individual confounders), the mean of the 1000 pollution effect estimates was roughly zero. However, only half the 95% confidence intervals contained zero. The other half implied spurious significant effects (negative or positive) as a result of the standard errors being too small.

From what I can tell from the map and the number of participants close to the airport, it may have been impossible in the Stockholm study to construct meaningful "areas" to enter into (say) a multilevel model. The authors have controlled for four potential confounders at the individual level—whether this is adequate is unknown. Unlike CESAR, there is some "within area" variation of exposure, as the noise contours cut through some districts near to the airport—although in fact the low exposure group is overwhelmingly resident in the outermost stratum. There is an issue of multiple testing, too, as four outcomes were apparently analysed—one as the focus of this paper, the other three used to refute possibilities of recall bias. But, difficulties notwithstanding, there is suggestive evidence for an association between prevalence of hypertension and aircraft noise.

As well as the primary question, the authors compare the two exposure measures, average and maximum noise. Comparisons of deviance show little difference between models including one or the other. A separate result seems to prompt the conclusion that the highest effect occurs in areas of low average but high maximum noise. Small numbers (which are acknowledged) and large confidence intervals make the drawing of such a conclusion wholly dubious. Yet references in the scientific literature to "startle factor" and to noise acclimatisation, although inconsistent, make the hypothesis intriguing. In terms of public health policy and what is achievable, these distinctions may be of considerable importance. A larger and statistically more rigorous study is needed. Various single airport studies have been performed and, while welcoming them, we now await the multicentre studies to test the hypotheses that they have generated.

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