those with known cardiovascular risk factors or disease or those found to have hypertension, renal dysfunction, or echocardiographic abnormalities. This we feel would leave very few, if any, subjects with important silent CAD. Furthermore, whether such subjects, if present, would have had raised NTpBNP levels is unclear, with no prior study analysing NTpBNP levels in subjects with asymptomatic CAD. Indeed, the paper by Kragelund et al. from which they quote found that in subjects with symptoms or signs of CAD, NTpBNP levels were raised the most in those with left ventricular systolic dysfunction, prior heart attack, diabetes mellitus, clinically significant CAD and renal dysfunction, conditions all excluded in our normal range assessment, and increased age, something stratified for in our study. Furthermore, there was virtually no difference in NTpBNP levels between those with or without CAD on angiography if left ventricular ejection fraction was normal, despite a higher risk profile in those with CAD. Thus, the merits and indeed the ethics of performing coronary angiography on asymptomatic subjects, unlikely to gain prognostic benefit and unlikely to alter the study’s findings, are unclear, especially with estimated morbidity and mortality rates of 1.8 and 0.08%, respectively. We thus stand by our conclusions and our developed normal range and would be happy for other authors to prospectively test them in other population groups.

References


Noise burden and the risk of myocardial infarction: false interpretation of results due to inadequate treatment of data: reply

Babisch et al. raise concerns regarding the part of the NaRoMi study dealing with the assessment of environmental sound levels. The authors of the publication¹ and the present response planned, designed, and coordinated the study, and collected and analysed all data on subjective environmental/work noise exposure as well as on medical and sociodemographic characteristics. In addition, acoustic engineers assessed sound pressure levels for street and work noise. In this latter capacity, Babisch was responsible for assessing levels of street traffic noise using the Berlin noise map.

Following data collection, we held regular meetings to discuss our approach to the multi-variable analyses. In particular, we need to account for the fact that the Berlin noise map does not provide data for smaller streets. In our investigation, subjects living in these areas actually comprised two separate groups: (i) a smaller group for which sound levels were reviewed and, if there were any doubts (e.g. due to the presence of a main street nearby), confirmed to be ≤60 dB(A); and (ii) a larger group of around 60% of the study population for which there were no assessments (i.e. with missing values), but in which sound levels were assumed to be ≤60 dB(A). Unexpectedly, these two groups differed markedly regarding their risk of myocardial infarction: the large group showed a significantly higher risk of myocardial infarction than the small group (for women two-fold). Interpreting these differences is difficult, because the noise map is based primarily on traffic counts in major streets and is not validated for supposedly ‘quiet’ ones.

In essence, Babisch’s suggestion to combine both the ≤60 dB(A) and the missing value groups into one reference group would have diluted the observed effects and masked major differences. Because pooling such heterogeneous reference groups does not seem acceptable from a statistical and epidemiological perspective, we decided to report the two groups separately, both in the official report to the funding institutions and in our publication¹ (Table 3). We consider the smaller group as the only valid reference group, as the larger group consists of individuals without measurement of exposure.

Without the participation or co-authorship of the NaRoMi study’s principal investigator (Wegscheider), scientific study coordinator (Keil), or statistician-in-charge (Wegscheider), Babisch et al. pursued subsequent analyses and publications. By combining the two reference groups mentioned earlier, they dismissed the increased risk for subjects exposed to environmental noise. We consider their results as optimistically biased.

It is of note that most authors of the letter by Babish et al. are employees of the German Federal Environmental Agency, a sponsor of the NaRoMi study. We do not accept their claim that the funding institutions have the privilege to determine the way data should be analysed. Apart from an obvious conflict of interest, their definition of the reference group and interpretation of data appears to downplay the cardiovascular risks of noise particularly for women. As independent scientists, we do not share their view given that the findings of our study suggest that environmental noise may lead to numerous potentially avoidable cases of myocardial infarction and subsequent death.

References


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