# *Editorial*

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**Noise and ischemic heart disease**

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# Editorial

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## Noise and ischemic heart disease

The incidence and mortality of ischemic heart disease (IHD) declined dramatically in affluent countries from the early 1980s until at least the mid 1990s (1). This change in occurrence is ascribed to improved lifestyle factors, primarily a decrease in smoking and an increase in leisure-time exercise, and better care of the coronary heart patient (2). The downward shift in incidence has taken place in spite of the escalating obesity epidemic and a high prevalence of occupational and environmental noise exposure (3, 4). IHD is still the leading cause of death, thus, as some preventive measures become fully implemented, the identification of novel risk factors becomes more important. In the field of occupational and environmental health, there are at least three widespread exposures that are not entirely recognized as causal risk factors by influential opinion-makers such as the American Heart Association (5). In addition to noise (6) and shift work (7), these also include psychosocial and organizational factors (8).

In this issue of the *Scandinavian Journal of Work, Environment and Health*, Poul Suadicani and co-authors add a new study (9) to the short list of four published follow-up studies addressing the risk of IHD in relation to occupational noise exposure (6, 10–12). They report a follow-up through 16 years of mortality due to IHD among almost 3000 men from Copenhagen and find no indication of increased risk according to self-reported noise, either before or after adjustment for classical cardiovascular risk factors. This result is consistent with an Israeli study that did not show increased noise-related risk of IHD (but rather total mortality) (12) and a Finnish census study showing fairly weak associations (11). But the result is not consistent with the increased IHD mortality observed in a large study of Canadian lumber-mill workers (10). Although exposure levels to noise in that study were far above the average exposure levels in the Danish industry (3), the increased risk was not limited to high exposure levels exceeding 90–95 dB(A). Thus the discrepant results are not explained by differences in exposure levels. The findings of the Suadicani et al study seem also contradictory to a long-term follow up of industrially employed men in Helsinki, where occupational noise was associated with an increased risk of IHD that persisted even when the workers had passed the age of retirement (6). Is residual confounding due to insufficient control for social class at stake in the Helsinki and lumber mill studies or are crude exposure assessments attenuating true effects in the Copenhagen study?

Unfortunately we do not find an answer among the mounting evidence from studies of cardiovascular risk factors related to environmental noise – mainly road and air traffic noise (4). Although community sound levels are orders of magnitude lower than industrial levels, contrary to occupational exposure, environmental exposure affects sleeping hours and resting time off work. Furthermore, the total number of exposed hours is higher, ear protection is not an option, and transportation noise causes more annoyance than industrial noise (13). The last point might be important if sound annoyance (noise) rather than sound pressure were harmful and if psychological discomfort reinforced the acute physiological stress response that noise exposure evidently elicits (14).

On the other hand, experimental as well as field studies of human exposure to noise show marked adaptation of the physiological stress response with continuous or repeated exposure – but perhaps not without ill-health effects in the long run (15). Accordingly, noise in both the workplace and environment represents different modalities, which may be associated with different risk profiles. Although several cross-sectional studies provide some evidence that road traffic and airport noise is related to small

increments in ambulant blood pressure, the few available follow-up studies of IHD do not indicate an increased risk (16, 17).

Knowledge on the extra-auditive effects of noise is of interest from a different perspective. Over decades, research on the risk of IHD in relation to job strain and other work-related psychosocial factors has not convinced the entire scientific community that observed associations are likely of a causal nature (18–20). A major methodological obstacle is the lack of independent measures of exposure (19, 21). In this context, it is of interest that some of the suggested mechanisms responsible for cardiovascular effects of psychosocial aspects of life are similar to mechanisms suggested for cardiovascular effects of noise – namely a prolonged unspecific biological stress response in terms of activation of the autonomic nervous system and neuroendocrine pathways (22, 23). In fact, the acute physiologic effect of psychosocial factors and noise are much alike (14). Thus noise can be considered an objective stressor and studies of noise may provide insight into the long-term effects of sustained activation of the physiologic stress response, if such effects exist. It should be acknowledged, however, that the empirical evidence for a long-term, low-dose activation of the autonomous nervous system and the hypothalamic-pituitary-adrenal axis by psychosocial factors and noise is limited (24–26).

Although the findings of the Suadicani et al study in this issue of the Journal are reassuring, it seems far too early to dismiss ischemic heart disease morbidity and mortality as a long-term consequence of noise even at environmental exposure levels. Several cohorts with good data on exposure, outcomes, extraneous determinants, as well as possible individual effect modifiers will hopefully provide new insight and inform the public and decision-makers in occupational and public health.

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