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The Noise/Stress Concept, Risk Assessment and Research Needs

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Abstract

In principle, the noise/stress hypothesis is well understood: Noise activates the pituitary-adrenal-cortical axis and the sympathetic-adrenal-medullary axis. Changes in stress hormones including epinephrine, norepinephrine and cortisol are frequently found in acute and chronic noise experiments. The catecholamines and steroid hormones affect the organism's metabolism. Cardiovascular disorders are especially in focus for epidemiological studies on adverse noise effects. However, not all biologically notifiable effects are of clinical relevance. The relative importance and significance of health outcomes to be assessed in epidemiological noise studies follow a hierarchical order, i.e. changes in physiological stress indicators, increase in biological risk factors, increase of the prevalence or incidence of diseases, premature death. Decision-making and risk management rely on quantitative risk assessment. Epidemiological methods are the primary tool for providing the necessary information. However, the statistical evidence of findings from individual studies is often weak. Magnitude of effect, dose-response relationship, biological plausibility and consistency of findings among studies are issues of epidemiological reasoning. Noise policy largely depends on considerations about cost-effectiveness, which may vary between populations. Limit or guideline values have to be set within the range between social and physical well-being - between nuisance and health. The cardiovascular risk is a key-outcome in non-auditory noise effects' research because of the high prevalence of related diseases in our communities. Specific studies regarding critical groups, different noise-sources, day/evening/night comparisons, coping styles and other effect-modifying factors, and the role of annoyance as a mediator of effect are issues for future research in this field.