Impact of traffic noise and pink noise masking on sleep fragmentation and metabolic function

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Background: Epidemiological studies show associations between chronic noise exposure and disease, but the biological pathways remain poorly understood. Here we aim to investigate the mechanisms that may link sleep disruption by environmental noise with the development of disease, and the efficacy of a non-pharmacological intervention to mitigate these effects.

Methods: This laboratory pilot study used a within-subjects crossover design. N=12 young, healthy individuals (5 female) slept for five consecutive nights in acoustically isolated bedrooms. The first night was for habituation and familiarisation with study procedures. Subsequent nights included the following, in a randomised order: one quiet baseline night; one night with nocturnal traffic noise of different types (road, rail and air) and noise levels (45-65 dB $L_{AS,max}$); one night with continuous 45 dB broadband pink noise; and one night with both traffic noise and pink noise. Sleep was measured with polysomnography and assessed using the Odds Ratio Product (ORP), a novel measure of continuous sleep depth and stability. Perceived sleep quality and recouperation were measured with morning questionnaires. Blood samples were collected each morning for metabolomics analysis. Cognitive performance across multiple domains was measured every morning and evening. Statistical analyses were implemented in linear mixed models with random subject effects, adjusted for sex, age and time in study.

Results: Discrete traffic noise events induced elevations of ORP (z-score +0.19; p<.001 relative to baseline, averaged over all traffic types and levels), indicating acute sleep fragmentation, even while total sleep time and overall sleep macrostructure were preserved. The traffic noise night was further associated with significant elevations in concentrations of leucine, lactic acid, and acetone relative to quiet control. Sleep and metabolic disturbances by traffic noise were attenuated when pink noise was played continuously throughout the night (averaged ORP z-score +0.06; p=.11 re: baseline). Cognitive performance was maintained across all exposure conditions.

Conclusions: Noise-induced sleep fragmentation induced changes in metabolic processes that in the long-term may be precursors for cardiometabolic disorders. Masking of traffic noise by continuous, neutral sound can mitigate acute physiological sleep disturbance and downstream metabolic effects. These results need to be interpreted cautiously however, given the limited sample size and subject homogeneity.