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The adverse effects of environmental noise exposure on oxidative stress and cardiovascular risk.

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Abstract

Epidemiological studies have provided evidence that traffic noise exposure is linked to cardiovascular diseases such as arterial hypertension, myocardial infarction and stroke. Noise is a non-specific stressor that activates the autonomous nervous system and endocrine signaling. According to the noise reaction model introduced by Babisch and colleagues, chronic low levels of noise can cause so called non-auditory effects, such as disturbances of activity, sleep and communication, which can trigger a number of emotional responses, including annoyance and subsequent stress. Chronic stress in turn is associated with cardiovascular risk factors, comprising increased blood pressure and dyslipidemia, increased blood viscosity and blood glucose and activation of blood clotting factors, in animal models and humans. Persistent chronic noise exposure increases the risk of cardiometabolic diseases including arterial hypertension, coronary artery disease, diabetes mellitus type 2 and stroke. Recently, we demonstrated that aircraft noise exposure during nighttime can induce endothelial dysfunction in healthy subjects and is even more pronounced in coronary artery disease patients. Importantly, impaired endothelial function was ameliorated by acute oral treatment with the antioxidant vitamin C suggesting that excessive production of reactive oxygen species (ROS) contributes to this phenomenon. More recently, we introduced a novel animal model of aircraft noise exposure characterizing the underlying molecular mechanisms leading to noise-dependent adverse oxidative stress-related effects on the vasculature. With the present review we want to provide an

overview of epidemiological, translational clinical and preclinical noise research addressing the non-auditory, adverse effects of noise exposure with focus on oxidative stress.

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