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Co-exposure to particulate matter and aircraft noise adversely impacts the cerebro-pulmonary-cardiovascular axis in mice

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ABSTRACT

Worldwide, up to 8.8 million excess deaths/year have been attributed to air pollution, mainly due to the exposure to fine particulate matter (PM). Traffic-related noise is an additional contributor to global mortality and morbidity. Both health risk factors substantially contribute to cardiovascular, metabolic and neuropsychiatric sequelae. Studies on the combined exposure are rare and urgently needed because of frequent co-occurrence of both risk factors in urban and industrial settings. To study the synergistic effects of PM and noise, we used an exposure system equipped with aerosol generator and loud-speakers, where C57BL/6 mice were acutely exposed for 3d to either ambient PM (NIST particles) and/or noise (aircraft landing and take-off events). The combination of both stressors caused endothelial dysfunction, increased blood pressure, oxidative stress and inflammation. An additive impairment of endothelial function was observed in isolated aortic rings and even more pronounced in cerebral and retinal arterioles. The increase in oxidative stress and inflammation markers together with RNA sequencing data indicate that noise particularly affects the brain and PM particularly affects the lungs. Noise also increased levels of circulating stress hormones adrenaline and noradrenaline, while PM increased levels of circulating cytokines CD68 and MCP-1. We demonstrate an additive upregulation of ACE-2 in the lung, suggesting that there may be an increased vulnerability to COVID-19 infection. The data warrant further mechanistic studies to characterize the propagation of primary target tissue damage (lung, brain) to remote organs such as aorta and heart by combined noise and PM exposure.

Keywords (3-6): Environmental risk factors; traffic noise exposure; air pollution; oxidative stress; inflammation; cardiovascular risk.