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Aircraft noise exposure induces inflammation and impairment of vascular / cardiac function after myocardial infarction

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ABSTRACT

Aims: Traffic noise may play an important role in the development and deterioration of ischemic heart disease. Thus, we sought to determine the mechanisms of cardiovascular dysfunction and inflammation induced by aircraft noise in a mouse model of myocardial infarction (MI) and in humans with incident MI. ***Methods and Results:*** C57BL/6J mice were exposed to noise alone (average sound pressure level 72dB; peak level 85dB) up to 4d, resulting in pro-inflammatory aortic gene expression in the myeloid cell adhesion/diapedesis pathways. Noise alone promoted adhesion and infiltration of inflammatory myeloid cells in vascular/cardiac tissue, paralleled by an increased percentage of leukocytes with a pro-inflammatory, reactive oxygen species (ROS)-producing phenotype and augmented expression of Nox-2/phospho-NFκB in peripheral blood. Ligation of the LAD resulted in worsening of cardiac function, pronounced cardiac infiltration of $CD11b^{+}$ myeloid cells and $Ly6C^{high}$ monocytes and induction of interleukin (IL) 6, IL-1β, CCL-2 and Nox-2, being aggravated by noise exposure prior to MI. MI induced stronger endothelial dysfunction and more pronounced increases in vascular ROS in animals preconditioned with noise. Participants of the population-based Gutenberg Health Cohort Study (median follow-up:11.4y) with incident MI revealed elevated CRP at baseline and worse LVEF after MI in case of a history of noise exposure and subsequent annoyance development. ***Conclusion:*** Aircraft noise exposure before MI substantially amplifies subsequent cardiovascular inflammation and aggravates ischemic heart failure, facilitated by a pro-inflammatory vascular conditioning. Our translational results suggest, that measures to reduce environmental noise exposure will be helpful in improving clinical outcome of subjects with MI.

Keywords: aircraft noise, myocardial infarction, inflammation, myeloid cells, endothelial dysfunction