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Título	Macrophages exposed to fine particulate matter mediate endothelial dysfunction in vitro
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The exposure to fine particulate matter ($PM_{2.5}$), an atmospheric pollutant, is a risk factor for cardiovascular diseases. Macrophages are the first defense against inhaled particles, and mediate endothelial inflammatory response. Once injured, there is an increase in the endothelial permeability and generation of oxygen reactive species (ROS), and a reduction in the nitric oxide (NO) bioavailability. The loss of nitric oxide favors endothelial dysfunction and the development of atherosclerosis. In this study, we aimed to evaluate if macrophages could mediate the endothelial dysfunction induced by $PM_{2.5}$. $PM_{2.5}$ retained in glass fiber filters was partially extracted with PBS and further centrifuged at 1000g for 15min. This solution (1g filter/125 mL PBS) was diluted in DMEM 10% FBS ten times. We exposed a murine macrophages cell line (RAW264.7) to $PM_{2.5}$ for 48h and used PBS as a Control. Further, we exposed a hemangioendothelioma cell line (EOMA) to the conditioned medium (10% of the 48h-exposed RAW medium in fresh medium), using fresh medium as a Control, for 48h. Cell viability was verified through MTT assay and nitrite levels by Griess method, both spectrophotometrically. ROS production was verified by DCF fluorescence. Exposure to $PM_{2.5}$ increased ROS production and nitric oxide in macrophages, indicating a pro-oxidative medium. Then, to investigate whether such alterations in the macrophage medium could be mediating endothelial dysfunction, we exposed vascular endothelial cells to the conditioned medium and evaluated some parameters indicative of endothelium integrity. We found decreased production of ROS and decreased bioavailability of nitric oxide, after exposure to the conditioned medium of macrophages exposed to the pollutant. We also assessed the cell viability, and it was decreased when compared to the fresh medium, indicating endothelial damage. Taken together, these results indicate that macrophages mediate endothelial dysfunction caused by the $PM_{2.5}$ leading to cardiovascular damage.