

Impact of Particulate Matter Exposure on Human Umbilical Vein Endothelial Cells: Insights into Oxidative Stress, Inflammation, and Endothelial Dysfunction

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This paper shows a literature review of the effects of PM_{2.5} in Human umbilical vein endothelial cells (HUVEC). The purpose is a) to inform the reader about the up-to-date literature of the mechanisms by which PM particulates cause endothelial damage and b) to raise collective awareness on the effect of air pollution in the disease linked to the cardiovascular system. PM_{2.5} presents a pollutant with a high potential of causing cardiovascular and pregnancy-linked complications, where the most common one is preeclampsia[1]. Results from *in-vitro* studies emphasize a connection between PM_{2.5} concentration and their cytotoxic effect in endothelial cells [2]. HUVEC represents one of the most suitable *in vitro* models to analyze the changes in the endothelium post-exposed to the cytotoxic action of PM_{2.5} due to the fact that a) they are cells of human origin, b) they are easy to isolate and culture, c) standard protocol for their cultivation eases experiment result reproduction d) they are cost-effective and e) they are the most commonly used cell cultures thus making the experiment results more understandable to the scientific community [3,4]. The consequences of short time (24,48,72hr) and long time (≥ two weeks) exposure show an elevation in pro-inflammatory proteins (e.g. IL-6 and TNF-α), elevation of oxidative stress markers, as well as emphasis on apoptosis through activation of the caspase-8 pathway [4]. The following table summarizes the important events of the action of PM_{2.5} in HUVEC, based on the relevant field literature of the last ten years, highlighting the need of excessive research in this field.

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References

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Study (Author, Year)	Inflammatory Markers	Oxidative Stress Markers	Apoptotic and Genotoxic Markers	Dosage	Exposure Duration	Health Outcome Studied	Experimental Model
Garcia et al. (2023)	TNF-α, IL-6	ROS	N/A	25 µg/m ³	Chronic	Preeclampsia, endothelial dysfunction	HUVEC
Kim et al. (2022)	TNF-α	ROS	Caspase-8, p53, DNA damage	80 µg/m ³	24 hours	Apoptosis, endothelial dysfunction	HUVEC
Wang et al. (2020)	N/A	ROS, SOD	N/A	100 µg/m ³	72 hours	Endothelial barrier dysfunction	Endothelial cells
Scherzad et al. (2017)	TNF-α	ROS, MDA	DNA damage, γ-H2AX	15 µg/m ³	48 hours	Oxidative stress, endothelial damage	HUVEC

Figure 1: Summary of PM_{2.5} impact results in different *in vitro* models