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Transcriptomics analysis revealed an indirect effect of aqueous cigarette smoke extract in promoting the adhesion of monocytic cells to endothelial cells

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Background

The adhesion of monocytic cells to the 'dysfunctional' endothelium constitutes a critical step in the initiation of atherosclerosis.

Cigarette smoke (CS) has been shown to contribute to the monocyte-endothelial adhesion process. However, the complex underlying molecular mechanisms remain to be unraveled.

Materials and methods

To investigate the impact of CS on the adhesion of monocytic cells to the endothelium, we developed a conditioned-medium experiment combined with an *in vitro* adhesion assay intended to mimic the situation found in the systemic compartment. Using a transcriptomics approach followed by confirmation experiments, we were able to identify a key mechanism by which aqueous CS extract in the form of smoke-bubbled phosphate buffered saline (sbPBS) promotes the adhesion of monocytic mono mac 6 (MM6) cells to human umbilical vein endothelial cells (HUVECs).

Results

While soluble CS constituents elicit a strong oxidative stress response in both cell types, the induced expression of E-selectin, vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) responsible for the binding of MM6 cells to HUVECs occurs through a pro-inflammatory paracrine effect. Our results show that this effect is largely driven by tumor necrosis factor α (TNF α) produced by MM6 cells exposed to sbPBS.

Conclusions

Our findings demonstrate that the adhesion of monocytic cells to endothelial cells is promoted through an indirect effect of sbPBS, mainly involving a key soluble factor TNF α and open new avenues for translational research in the comprehension of atherosclerosis development.

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