# Stretch, inflammation and hypertension

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Monocytes are known to play an important role in hypertension, although the exact mechanism remains unclear. It is hypothesized that a potential source of monocyte activation originates from its interaction with the vascular endothelium.

In a study of circulating human monocytes and monocytes cultured with human aortic endothelial cells, David Harrison, MD, and colleagues

explored the influence of increased vascular endothelial stretch on monocyte phenotype and function during hypertension.

The study published in *Cardiovascular Research* sheds light on how changes in mechanical forces in the vessel enhance immune cell activation thus promoting hypertension.

The authors demonstrate that upon interaction with mechanically stretched endothelial cells, human monocytes undergo differentiation into intermediate-phenotype and to cells bearing dendritic cell properties. Furthermore, STAT3, a transcription factor, IL-6, a pro-inflammatory cytokine, and hydrogen peroxide were shown to play roles in monocyte differentiation.

Interventions to enhance bioavailable nitric oxide and other methods to inhibit STAT3 or to reduce hydrogen peroxide production may have anti-inflammatory roles in hypertension and related conditions.

**More information:** Roxana Loperena et al. Hypertension and increased endothelial mechanical stretch promote monocyte differentiation and activation: roles of STAT3, interleukin 6 and hydrogen peroxide, *Cardiovascular Research* (2018). DOI: 10.1093/cvr/cvy112

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