Methods VCAM-1 RNA expression was analysed by real time RT-PCR. The endothelial surface expression of VCAM-1 was measured by flow cytometry. VCAM-1 promoter with NF-κ B binding sites were cloned into pGL3 vector. Luciferase assay was used to analyse VCAM-1 promoter activity in endothelial cells. NF-κ B translocation was observed by immunocytochemistry.

Results Treatment with Ang II resulted in an increase of VCAM-1 expression on endothelial cells, whereas Ang-(1–7) alone had no effects. However, preincubation with Ang-(1–7) inhibited Ang II induced VCAM-1 expression, which is demonstrated by flow cytometry and real time RT-PCR. In addition, Ang-(1–7) inhibited Ang II induced VCAM-1 promoter activity. Immunocytochemistry showed that Ang-(1–7) blocked Ang II induced translocation of NF-κ B from cytoplasm into nucleus, and the effects of Ang-(1–7) were abolished in the presence of MAS receptor antagonist A779.

Conclusions These results suggest that Ang-(1–7) inhibited VCAM-1 expression, at least in part, through negative modulation of Ang II induced NF-κ B pathway.

GW23-e2141 **DIFFERENT EFFECTS OF ANGIOTENSIN II AND ANGIOTENSIN-(1–7) ON ENDOTHELIAL VCAM-1 EXPRESSION DURING ATHEROSCLEROSIS**

doi:10.1136/heartjnl-2012-302920a.181

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Objectives Angiotensin (Ang) II and Ang-(1–7) are two of the bioactive peptides of the renin-angiotensin system. Ang II is involved in the development of cardiovascular disease, such as hypertension and atherosclerosis, while Ang-(1–7) shows cardiovascular protection in contrast to Ang II. We studied effects of Ang II and Ang-(1–7) on endothelial VCAM-1 expression, which are critical in the formation of atherosclerotic lesion.