**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.

**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-κB binding sites were cloned into pGL3 vector. Luciferase assay was used to analyse VCAM-1 promoter activity in endothelial cells. NF-κB 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-κB 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-κB pathway.