

POSTER PRESENTATION

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# The role of VASP in cGMP-mediated vascular smooth muscle relaxation

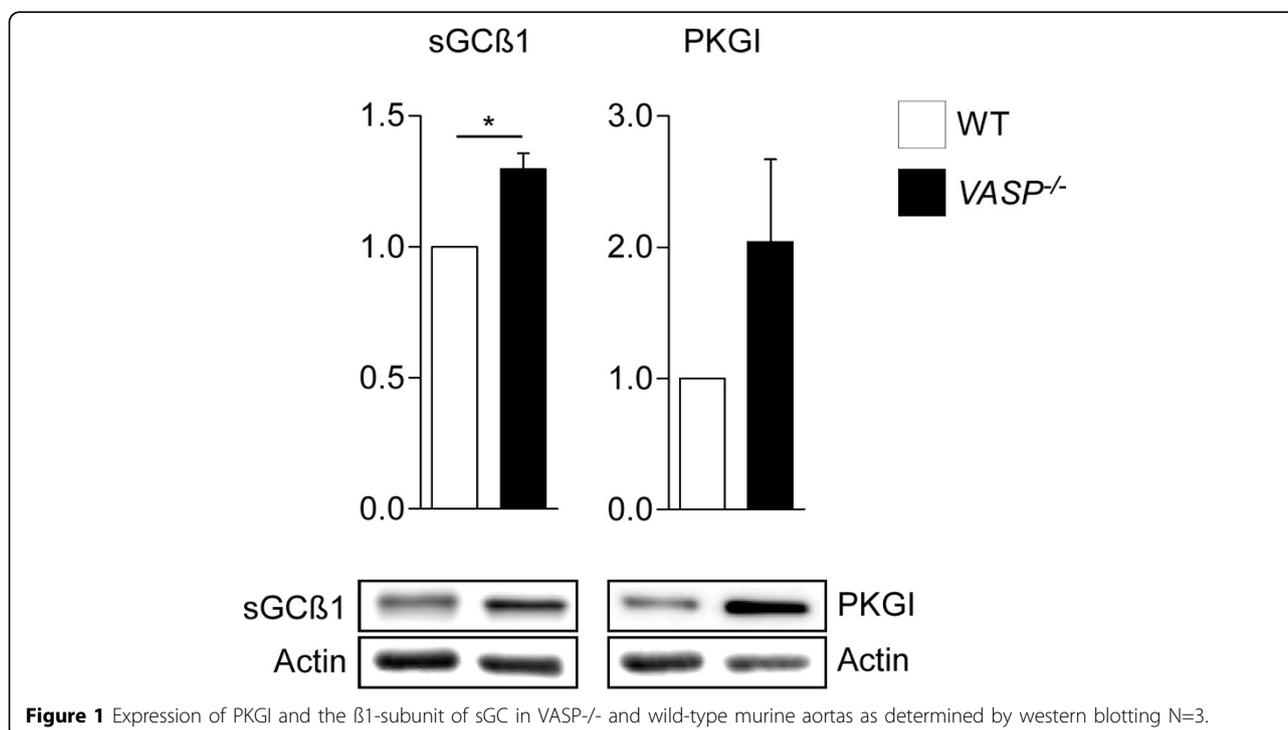
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## Background

Cyclic GMP (cGMP) is a major mediator of relaxation in the vascular system. cGMP is produced by the enzyme soluble Guanylyl Cyclase (sGC) in response to nitric oxide (NO) released from neighbouring endothelial cells. cGMP activates Protein Kinase G (PKG), which in turn

mediates vascular relaxation through phosphorylation of various targets. One of the major substrates of PKG is the Vasodilator-Stimulated Phosphoprotein (VASP). The role of VASP in vascular smooth muscle relaxation is currently unknown. However, recent studies show that VASP-deficient brown adipocytes have an increased



**Figure 1** Expression of PKGI and the  $\beta$ 1-subunit of sGC in VASP<sup>-/-</sup> and wild-type murine aortas as determined by western blotting N=3.

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activity of the small GTPase Rac1 and elevated levels of sGC [1]. These data suggest a regulatory role for VASP in cGMP-mediated processes.

## Results

Preliminary data acquired from the analysis of VASP-deficient (VASP<sup>-/-</sup>) mice provide evidence for the importance of VASP in the cGMP mediated relaxation pathway: VASP<sup>-/-</sup> aortas show higher levels of PKG and sGC compared to wild type (see Figure 1), and increased sensitivity to NO-induced relaxation. Additionally, cultured vascular smooth muscle cells (VSMCs) transduced with a constitutively active Rac1 mutant (RacL61) show elevated sGC and PKG expression as well as increased PKG activity. Cultured VSMCs from VASP<sup>-/-</sup> aortas also demonstrate decreased proliferation rates compared to wild-type cells in preliminary experiments.

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