enriched in high shear regions of healthy arteries and significantly reduced in plaque (Figure 1B).

**Conclusions** Endothelial cell responses to high shear are different in healthy and diseased arteries. Some shear stress related genes are different between healthy arteries and plaques could explain these differences. Future studies will focus on these shear stress related genes to identify their functions and pathways.

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**BS22 MATRIX STIFFNESS DRIVES INCREASED VASCULAR SMOOTH MUSCLE CELL VOLUME RESPONSE VIA AQUAPORIN MEDIATED WATER INFUX**

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Decreased aortic compliance is a major risk factor for the development of cardiovascular diseases including hypertension and atherosclerosis. Healthy aortae are compliant and can change shape in response changes in blood pressure. This ability arises because of the balance of collagen-I, that provides tensile strength, and elastic extracellular matrix (ECM) components in the medial layer of the aortic wall. Vascular smooth muscle cells (VSMC) are the predominant cell type in the aortic wall and their contraction decreases aortic compliance. Ageing triggers increased deposition of collagen and degradation of the elastic components. This drives stiffening of the aortic ECM. VSMCs are mechanosensitive and respond to this stiffening by generating increased actomyosin generated forces. These are known to contribute to the decreased aortic compliance associated with ageing and hypertension. However, the mechanisms driving the VSMC response remain unknown. In this study, we use polycrylamide hydrogels of physiological and pathological stiffness. Angiotensin II stimulation of quiescent VSMCs on hydrogels resulted in decreased VSMC area but VSMC volume remained unaltered. In contrast, angiotensin II treatment resulted in increased VSMC area and volume on hydrogels of pathological stiffness. Aquaporins are a family of ubiquitous transmembrane proteins involved in the transport of water across membranes. They have been shown to play important roles in the regulation of cell volume in a range of tissues. Aquaporins 1 and 4 are both expressed in VSMCs, therefore, we hypothesised that aquaporins 1 and 4 permitted increased water influx when VSMCs are exposed to enhanced matrix stiffness. To test this, we utilised the aquaporin 1 inhibitor TCAQP1 and the aquaporin 4 inhibitor TGN020. Importantly, pretreatment of with either TCAQP1 or TGN020 blocked the increased VSMC area response of angiotensin II stimulated VSMCs on hydrogels of pathological stiffness. We next plan to investigate VSMC volume response. Our data suggests that aquaporin 1 and 4 mediated water influx increases volume of angiotensin II stimulated VSMCs in environments of pathological stiffness.