**Glucose metabolism and vascular progenitors: Is mechanosensing the missing link?**

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Endothelial cells lining the innermost layer of blood vessels play an indispensable role in vascular homeostasis. They sense and respond to changes in blood flow and associated shear forces through a well woven mechanism of cell signaling and nitric oxide production. Most of these responses are anti-inflammatory and anti-thrombotic in nature. Hence, any damage to the endothelium, triggers the onset of cardio-vascular complications be it hypertension, atherosclerosis or ischemic heart disease. Occasional endothelial damages are repaired by a repertoire of circulating cells such as endothelial progenitors, haemangioblasts and monocytes, which continuously experience blood flow. These cells home into the site of damage and upon binding to the exposed extracellular matrix proteins, differentiate into endothelial cells for repair. Intriguingly the reparative capacity of these cells is heavily compromised in metabolic syndrome and diabetes. Furthermore, nothing is known about the influence of impaired glucose metabolism on mechanosensing abilities of these cells. We performed two independent case-control based studies with healthy and pre-diabetic men and women, in order to determine the influence of impaired glucose metabolism on the vasculogenic property of circulating mono-nuclear cells. Cells obtained from pre-diabetic subjects exhibited poor adherence to fibronectin as well as decreased migration towards chemotactic gradient of SDF1- α. This in turn affected their ability to home into the tubular structures formed by the matured endothelial cells. No differences were observed with regard to endothelial differentiation upon 7-day culture in endothelial growth medium however, the ability of these cells to produce nitric oxide was significantly attenuated. The talk will summarize some of our current understanding on these aspects of endothelial biology.