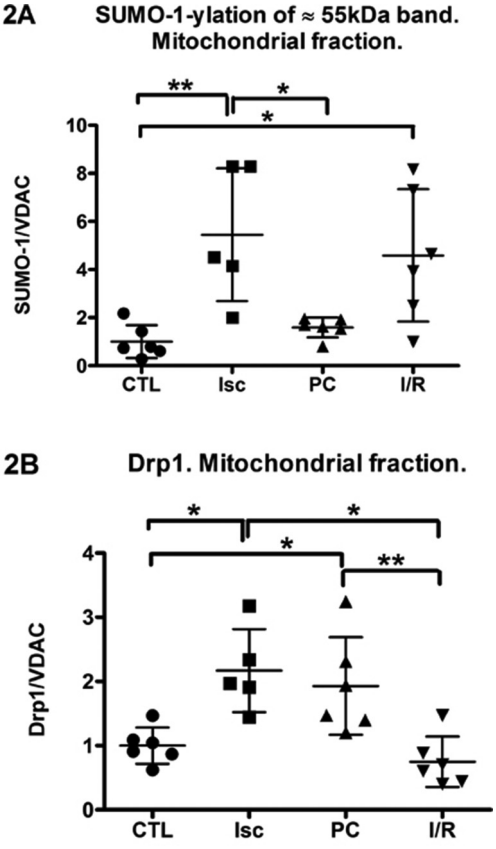
compared to both the control and preconditioning groups (Fig 2A). Intriguingly, in contrast to our finding in neurons, where mitochondrial partitioning of Drp1 decreases during ischaemia, in cardiac tissue we observed recruitment of Drp1 to mitochondria, with no change in total protein levels. Furthermore, Drp1 recruitment to mitochondria was increased by preconditioning. In the I/R group, in which cells are undergoing apoptosis, levels of Drp1 at the mitochondria are similar to controls (Fig 2B).



Abstract 229 Figure 2

Conclusion Taken together our data suggest a delicate balance between SUMOylation and deSUMOylation that regulates the recruitment of Drp1 to mitochondria. This pathway plays an important role in the vulnerability of cardiomyocytes to ischaemic damage and myocardial reperfusion injury. Interestingly, the interplay between the relevant proteins appears to differ between heart and brain cells.

230 TRACKING LATE OUTGROWTH ENDOTHELIAL CELLS IN AN ACUTE ARTERIAL INJURY MODEL

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Aim Late outgrowth endothelial cells (EOC) are strong contenders to be the true circulating endothelial progenitor cells since they are capable of clonogenic expansion, exhibit a mature endothelial phenotype, and contribute to angiogenesis *in vivo*. These cells may play a crucial role in the process of vascular repair, but whether they are able to accumulate at sites of vascular damage *in vivo* is not clear. We hypothesise that EOC, delivered locally or systemically, accumulate at, and incorporate into, a site of arterial injury.

Methods Experimental groups comprised systemic administration of the glucose analogue radiotracer ^{18}F-Flurodeoxyglucose (FDG) (Sys-Free) or FDG-labelled EOC (Sys-EOC), or local administration of free FDG (Local-Free) or FDG-labelled EOC (Local-EOC). EOC were isolated from peripheral blood from patients with coronary heart disease (n=3). Left femoral artery injury was achieved in male Sprague Dawley rats (300–350g) under general anaesthesia by inserting micro-renathane tubing via the popliteal artery. EOC were labelled with FDG (25 MBq/ml, 30 min, 37°C) and 1 million cells were administered either locally into the femoral artery or systemically via the tail vein (0.3–1.3 MBq, n=3 per group). Following injection of radiolabelled cells or free FDG, rats underwent dynamic PET scanning over 4 hours (Mediso nanoPET/CT scanner, Hungary). AuroVist and Fenestra (MediLumine Inc, Canada) were used as computed tomography vascular contrast agents. Images were analysed with PMOD software (PMOD, Switzerland) and standardised uptake values were calculated.

Results FDG radioactivity was successfully visualised by micro-PET/CT. The activity was distributed in the bladder, kidneys, heart, brain, lungs, spleen and liver (descending order). Radioactivity in the lungs was significantly higher (80 and 120 min) following systemic EOC administration compared with the other three groups (two-way ANOVA with Bonferroni post-test, $p<0.001$), and peak activity in the injured artery (55 min after administration) was significantly higher than in the non-injured right artery (two-way ANOVA with Bonferroni post-test, $p<0.001$). Following local EOC administration, radioactivity in the injured artery was significantly higher than in the non-injured right artery, or in the injured artery following local free FDG administration (two-way ANOVA with Bonferroni post-test, $p<0.001$). Radioactivity in the injured artery following local administration was considerably higher (~10 fold) than following systemic administration of either free FDG or labelled EOC.

Conclusion Preliminary analysis shows that EOC are able to target sites of vascular injury following systemic and local administration. These observations suggest that late outgrowth endothelial cells have the potential to contribute to vascular repair and regeneration.

231 SUPER RESOLUTION IMAGING UNVEILING THE DYADIC ULTRASTRUCTURE IN ATRIAL AND VENTRICULAR CARDIOMYOCYTES

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Calcium-induced calcium release drives contraction in cardiomyocytes. Located on the sarcoplasmic reticulum, ryanodine receptors (RyR) are responsible for the release of intracellular calcium stores. It has recently become apparent that the size and shape of RyR cluster may affect the functionality of that cluster, along with the relative distance to neighbouring clusters. Here, we compared the distribution of RyRs in the atria and ventricle.