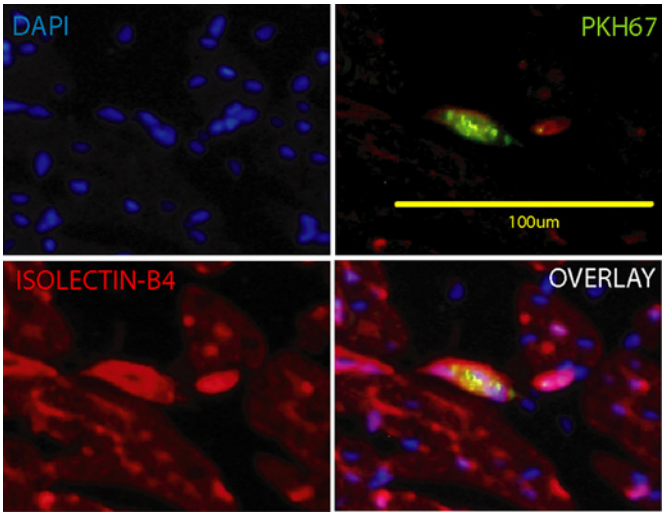
were observed to have extravasated after 60 min (Abstract 119 figure 2). Coronary flow was not affected by injection of BMMNCs, whereas MSC injection led to a transient decrease of coronary flow.



Abstract 119 Figure 2

Conclusion Our study using an original model showed retention rates of BMMNC in normal hearts were low regardless of the cell number injected, suggesting a critical limiting factor for the treatment's success. Retention rates were improved by I-R. Larger BMMNCs were retained with an increased efficiency. This information provides intriguing potential methods for increasing retention in future clinical studies, thereby enhancing the therapeutic effect.

120 BLOOD FLOW IS REQUIRED FOR ENDOTHELIAL REPAIR IN A NOVEL IN VIVO MODEL USING ZEBRAFISH EMBRYOS

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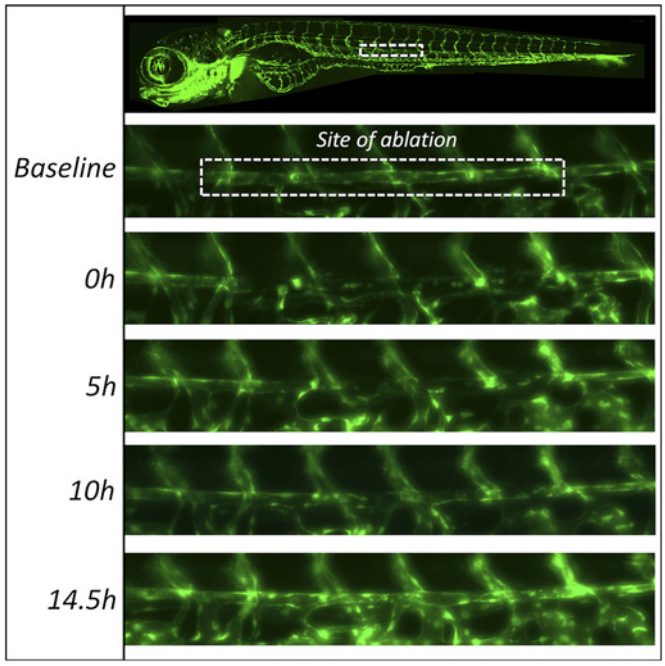
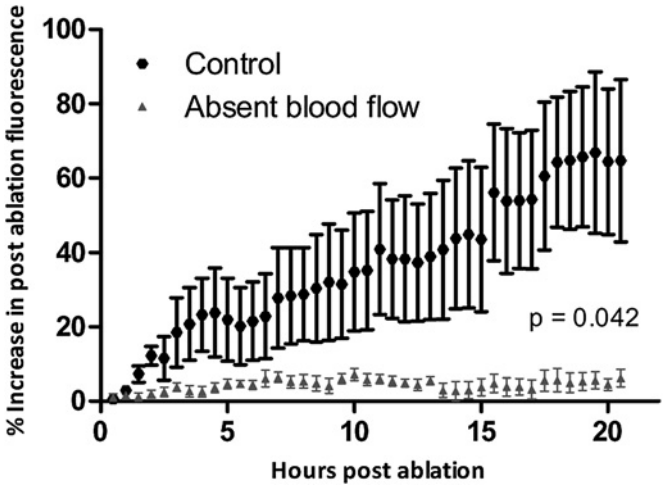
Introduction Endothelial repair is essential for vascular homeostasis but difficult to study in vivo using existing animal models. The role of blood flow in the repair response remains unclear. We therefore established a novel in vivo model of endothelial repair using transgenic zebrafish embryos, and tested whether this was affected by the absence of blood flow.

Methods 4-day old Fli1:eGFP transgenic embryos (expressing green fluorescent protein in endothelium) were anaesthetised and mounted in low melting point agarose. We used focused laser injury (Micropoint laser mounted on a Nikon inverted microscope) to ablate the endothelium of the dorsal aorta in the region between somites 15–17. Time-lapse fluorescence or confocal microscopy was then used to visualise recovery of the ablated endothelium in real time. To determine the role of blood flow in endothelial repair, embryos were treated with the anaesthetic tricaine which reversibly halts cardiac contraction and recovery from aortic ablation determined as above. This does not affect the embryos' development, which survive due to oxygen diffusion.

Results Abstract 120 figure 1 demonstrates endothelial ablation at 0 h. Over the following 14.5 h we observed migration of endothelial cells from both proximal and distal vasculature (aorta, vein, and intersegmental vessels) to repopulate the ablated region, suggesting this is a model of endothelial migration and proliferation. We did not observe recruitment of circulating cells to the site of injury. Complete recovery in all ablated embryos was achieved by 15–20 h post injury. Reversible cessation of blood flow significantly prevented endothelial repair compared with control (fluorescence at 20 h post injury: control 60%±22% of baseline vs absent blood flow 5%±2.4%, $p<0.05$ (Abstract 120 figure 2).



Abstract 120 Figure 1 Timecourse of endothelial repair following laser injury.



Abstract 120 Figure 2 Timecourse of endothelial repair in embryos with (black) and without (grey) blood flow.

Conclusions The ability to visualise endothelial damage and recovery in living zebrafish embryos offers a powerful new model for dissecting the mechanisms of endothelial repair in vivo. Endothelial repair requires blood flow even in the absence of recruitment of circulating cells.

121 BIOINFORMATIC IMAGING AND MOLECULAR INVESTIGATION FOR A ROLE OF $P22^{PHOX}$ C242T POLYMORPHISM IN INHIBITING ENDOTHELIAL ROS PRODUCTION

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The $p22^{phox}$ is a key component of the cytochrome b558 of the NADPH oxidase (Nox), which by generating reactive oxygen species