ABSTRACTS

acts as a key functional regulator of CDCs. The aims of this study were to confirm that CDCs significantly improve cardiac function and augment neovascularisation following myocardial infarction (MI) and to test the effect of endoglin depletion.

CDCs were derived from Engfl/fl;Rosa26-CreERT2 neonatal mice and were treated with or without 4hydroxy-tamoxifen in vitro to deplete endoglin. Acute MI was induced in adult C57Bl/6 mice by permanent ligation of the left anterior descending coronary artery. Wild-type (WT) or endoglin knock-out (Eng-KO) CDCs were delivered by intramyocardial injection into the infarct border zone. At 28 days cardiac MRI revealed that both WT and Eng-KO CDC recipient groups showed an increase in ejection fraction compared with PBS-treated controls and left ventricular volume indices were also reduced suggesting attenuated adverse remodelling. Injection of WT-CDCs led to an increased angiogenic response, which was significantly reduced when Eng-KO cells were transplanted.

Initial data suggests that endoglin expression is dispensable for CDC-mediated augmentation of cardiac function in injured hearts; however it is essential for stimulating an enhanced angiogenic response.

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CARDIOSPHERE-DERIVED CELL TRANSPLANTATION RESCUES CARDIAC FUNCTION POST-MI INDEPENDENTLY OF ENDOGLIN EXPRESSION

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Cardiosphere-derived cells (CDCs) have been shown to promote cardiac repair in vivo, however the mechanisms involved are poorly understood. Endoglin is a pro-angiogenic TGFβ co-receptor highly expressed by CDCs. Endoglin heterozygous human mononuclear cells (MNCs) demonstrate reduced efficacy in promoting cardiac repair in a mouse model. Therefore we hypothesise that endoglin