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Small Peptide, P1P, Derived from Prominin-1/CD133 Improves Heart Function Following Myocardial Infarction in Rats

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Background and Aims:

Acute myocardial infarction (AMI) is the most common reason for death in western world. Stimulation of angiogenesis can restore perfusion of the ischemic tissue and therefore lead to better healing of the myocardium after AMI. In previous experiments we have shown that a novel 12 amino acid peptide (P1P) derived from the stem cell marker Prominin-1/CD133 binds vascular endothelial growth factor (VEGF), stabilizes VEGF dimmers, and increases VEGF binding to the VEGF receptors. The aim of the current study is to characterize this unique pro-angiogenic peptide following myocardial infarction in rats.

Methods:

P1P or vehicle were injected I.P to rats 2,5,7,9 and 12 days post ligation of the left coronary artery. Left ventricular function was evaluated prior to ligation and at 2 and 14 days after the ligation using echocardiography.

Results and Discussion:

Administration of P1P significantly improved cardiac function at 14 days post MI: fractional shortening was augmented from 23.3±4.7 to 29.7±5 % and area fractional change was increased from 39.4±8.9 to 52.5±11 %, Vehicle and P1P, respectively ($p<0.05$, n=7-10). VEGF expression in the ischemic area was increased, while scar area and collagen deposition (measured in Masson tri chrome stained sections) were reduced.

Conclusions:

The pro-angiogenic peptide P1P improves heart function following MI and may offer a novel approach to treat myocardial infarction.