MP84-16 Foxc2 Overexpression in Endothelial Progenitor Cells Enhances Re-endothelialization Following cavernous Arterial Injury

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Introduction

Corpora cavernosa vascular endothelium injury is an important cause of erectile dysfunction (ED). Targeted repair of penile vascular endothelium is a vital way for ED treatment. Endothelial progenitor cells (EPCs) can facilitate endothelial repair through direct differentiation into endothelial cells and/or via the paracrine mechanisms. Foxc2 plays an important role in the combinatorial regulation of endothelial gene expression and vasculogenesis.

Aims

The aim is to investigate that if Foxc2 can promote EPCs' homing and differentiation, which repair the cavernous vascular endothelium injury eventually.

Materials & methods

Rat cavernous vascular injury was established using a flexible guide wire. The rat subjected to vascular injury were randomly assigned into experimental groups. EPCs were isolated and cultured from wild type rat. EPCs were transfected with Foxc2 expression vector (Foxc2-EPCs) or with empty control vector (Ctrl-EPCs). The rat subjected to cavernous vascular injury received PBS, Ctrl-EPCs or Foxc2-EPCs by tail vein injection. Fluorescent microscopy was used to examine the homing capacity of EPCs to the injury sites. For assessment of re-endothelialization animals were perfused with Evans blue dye at 7 days after EPCs delivery.

Results

At 3 days after EPCs delivery, cells were strictly restricted to the injury sites. The number of recruited cells was significantly higher in the rat transfused with Foxc2-EPCs compared with Ctrl-EPCs (about 3-fold of Ctrl-EPCs). At 7 days after EPCs delivery, Foxc2-EPCs delivery significantly increased the degree of re-endothelialization relative to Ctrl-EPCs (84.3±1.2%vs. 50.1±1.1%, P< 0.05). At 28 days, the inhibitory effect of Foxc2-EPCs delivery was lower than Ctrl-EPCs (0.32±0.01vs. 0.78±0.05, P< 0.05).

Conclusion

Foxc2 overexpression may increase EPCs homing and recruitment to the sites of cavernous vascular injury, and thereby enhance the therapeutic benefit of EPCs for facilitating re-endothelialization.

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