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KNOCKDOWN OF STROMAL INTERACTION MOLECULE 1 DOWN-REGULATES THE DIFFERENTIATION OF ENDOTHELIAL PROGENITOR CELLS AND REENDOTHELIALIZATION AFTER VASCULAR INJURY

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Objectives The study was to investigate the effect of stromal interaction molecule 1(STIM1) silencing on endothelial progenitor cells (EPCs) differentiation and reendothelialization.

Methods Following balloon injury, EPCs, which were transfected with Ad-si/rSTIM1, Ad-hSTIM1 and Ad–non silencing control (NSC), were transplanted to the rat. Evans Blue dye was performed to measured reendothelialization at 7 and 14 day after injury, the neointimal formation was evaluated by staining with hematoxylin and eosin at 14 day after injury. EPCs differentiation was examined by western blot, and rat gene expression array was detected.

Results At 7 and 14 day, the reendothelialized area in the Ad-si/rSTIM1-EPCs infected arteries was obviously less than that in NSC-infected groups and Ad-si/rSTIM1+ Ad-hSTIM1-EPCs group ($p<0.05$). A marked increase in the neointimal area and I/M ratio was shown in Ad-si/rSTIM1-EPCs group compared with NSC-transduced groups and Ad-si/rSTIM1+ Ad-hSTIM1-EPCs group at 14 day ($p<0.05$). Consistent with these findings, knockdown of STIM1 suppressed EPCs differentiation in vitro and 12 genes were downregulated by at least 100-fold during knockdown of STIM1 reduced EPCs differentiation, including C4-2, Sgcg, LOC360504, Rhoj, Krt10, Ucp1, Thrsp, Gpc3, Mgp, Hba-a2 and Igfbp6.

Conclusions Silencing of STIM1 inhibited EPCs differentiation and reendothelialization, indicating a possible new mechanism through EPCs underlying the process of vascular repair.