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**MENSTRUAL BLOOD STEM CELLS RESCUE MYOCARDIOCYTES IN RAT MYOCARDIAL INFARCTION BY REGULATING REDOX AND DECREASING ROS GENERATION THROUGH PARACRINE ACTION**

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**Objective** In the study, we hypothesised that menstrual blood stem cells (MBSCs) protect ischemic myocardium through paracrine effect.

**Methods & Result** During in vitro experiments, neonatal rat ventricle myocytes (NRVMs) were cocultured with MBSCs and then underwent hypoxia/reoxygenation procedure. The apoptosis ratio of NRVMs in coculture group was lower than the control group ( $10.26\pm 3.9\%$  vs  $17.38\pm 4.4\%$ ,  $p<0.05$ ). The medium of normoxia and hypoxia cultured MBSCs was collected to detect the secreted EGF, VEGF, TGF $\beta$  ( $559\pm 21$ ,  $0$ ,  $635\pm 59$  ng/ml in normoxia culture, respectively,  $582\pm 19$ ,  $198\pm 6$ ,  $664\pm 102$  mg/ml in hypoxia culture, respectively). The RNA was also extracted and reverse transcriptional PCR showed that MBSCs expressed multiple cytokines.

The MBSCs were injected into five sites around the infarct region after ligation of the left anterior descending (LAD) in rats. 4 weeks later, 18-fluorodeoxyglucose (18-FDG) microPET were performed and the images indicated more viable myocytes in the scar area of transplant group than of saline group ( $0.357\pm 0.067$  vs  $0.275\pm 0.053$ ,  $p<0.01$  in transverse section,  $0.333\pm 0.046$  vs  $0.267\pm 0.045$ ,  $p<0.01$  in coronal section). The infarct size of transplant group was less than saline group in Masson trichrome stain ( $33.6\pm 2.9\%$  vs  $41.6\pm 4.9\%$ ,  $p<0.05$ ). Gene chip cluster analysis suggested that the expression level of *Akr1c12*, *Alox12l*, *Fmo2* and *Aldh5a1*, which belong to the REDOX family, were downregulated in saline group, but upregulated in transplant group. Quantitative PCR confirmed partly the results of gene chip analysis. During the in vitro study, the generation of reactive oxygen species (ROS) in NRVMs were reduced by cocultured with MBSCs after H/R treatment.

**Conclusion** MBSCs paracrined multiple cytokines to protect myocytes from apoptosis by upregulating several oxidoreductase expression and decreasing ROS generation.