Pharmacologically active microcarriers influence VEGF-A effects on mesenchymal stem cell survival

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Résumé en anglais	Resistance of transplanted mesenchymal stem cells (MSCs) in post-ischemic heart is limited by their poor vitality. Vascular-endothelial-growth-factor-A (VEGF-A) as such or slowly released by fibronectin-coated pharmacologically-active-microcarriers (FN-PAM-VEGF) could differently affect survival kinases and anti-apoptotic mediator (e.g. Bcl-2). Therefore VEGF-A or FN-PAM-VEGF could differently enhance cell proliferation, and/or resistance to hypoxia/reoxygenation (H/R) of MSCs. To test these hypotheses MSCs were incubated for 6-days with VEGF-A alone or with FN-PAM-VEGF. In addition, MSCs pre-treated for 24-hrs with VEGF-A or FN-PAM-VEGF were subsequently exposed to H/R (72-hrs 3% O(2) and 3-hrs of reoxygenation). Cell-proliferation and post-hypoxic vitality were determined. Kinases were studied at 30-min., 1- and 3-days of treatment. Cell-proliferation increased about twofold ($P < 0.01$) 6-days after VEGF-A treatment, but by a lesser extent (55% increase) with FN-PAM-VEGF ($P < 0.05$). While MSC pre-treatment with VEGF-A confirmed cell-proliferation, pre-treatment with FN-PAM-VEGF protected MSCs against H/R. In the early phase of treatments, VEGF-A increased phospho-Akt, phospho-ERK-1/2 and phospho-PKCε compared to the untreated cells or FN-PAM-VEGF. Afterword, kinase phosphorylations were higher with VGEF, except for ERK-1/2, which was similarly increased by both treatments at 3 days. Only FN-PAM-VEGF significantly increased Bcl-2 levels. After H/R, lactate dehydrogenase release and cleaved Caspase-3 levels were mainly reduced by FN-PAM-VEGF. While VEGF-A enhances MSC proliferation in normoxia, FN-PAM-VEGF mainly hampers post-hypoxic MSC death. These different effects underscore the necessity of approaches suited to the various conditions. The use of FN-PAM-VEGF could be considered as a novel approach for enhancing MSC survival and regeneration in hostile environment of post-ischemic tissues.
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