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Presentation Abstract

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Presentation Title: Effect of human Mesenchymal Stem Cells and Endothelial Progenitor Cells on rat cortical neurons injured by oxygen and glucose deprivation.

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Abstract: Ischemic events and traumatic injuries in the brain result in neuronal loss, mainly due to oxygen and glucose deprivation (OGD). Nowadays few therapeutic approaches are available for the treatment of these conditions, and often the outcome is unfavorable for the patient or at least unpredictable.
Stem cells have been proposed for the treatment of OGD injured-neurons. Mesenchymal Stem Cells (MSCs) can be isolated from bone marrow as well as from various tissues and have neuroprotective properties and poor immunogenicity. In particular MSCs have been demonstrated to alleviate ischemic brain injuries in animal models. The Endothelial Progenitor Cells (EPCs) are a more recently isolated stem cell population, present both in the bone marrow and in the peripheral blood even if at low frequencies. They are thought to play a role in the recovery of cerebrovasculature integrity after stroke. Moreover EPCs can be mobilized by the administration of drugs such as statins, representing thus a valuable therapeutic tool.
In the present study we evaluated the potential neuroprotective effect of human MSC and human EPCs on rat embryonic cortical neurons injured by OGD. OGD was induced by incubating the cortical neurons in a hypoxia incubator chamber in a 95% N2-5% CO2 atmosphere at 37°C in a medium without glucose. Different ODG periods ranging from 30 minutes and 6 hours were evaluated. After the ODG the neurons were returned in normoxic atmosphere and were 1) co-cultured with either MSCs or EPCs seeded on a cell culture insert that prevents neurons and MSCs or EPCs direct contact even if they share the same medium, or 2) cultured in a medium previously conditioned by either MSCs

or EPCs. Neuronal survival was evaluated by MTT assay and viable cells counting. Also neuronal morphology was taken into account to evaluate the potential MSCs and EPCs neuroprotective effect.
Both MSCs and EPCs increased neuronal survival after ODG. This effect was observed in absence of a direct contact between MSCs or EPCs and the injured neurons, suggesting that the release of soluble factors may be the main mechanism of action.
In conclusion both MSCs and EPCs could represent a potential therapeutic approach for the treatment of brain ischemic injury. Further studies are needed to identify the specific molecules involved in the neuroprotective effect of MSCs and EPCs.

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