

## عنوان مقاله:

Changes of Neurogenesis Markers in Corpus Callosum Induced by Paracrine Effect of Human Embryonic Stem Cell-Derived Mesenchymal Stem Cells in Ischemic Rat

## محل انتشار:

هشتمین کنگره علوم اعصاب و پایه و بالینی (سال: 1398)

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## خلاصه مقاله:

**Background and Aim :** Mesenchymal stem cells (MSCs) are important candidates for stem cell therapy for neurological disorders. Recent studies have showed that the use of stem cells conditioned medium (CM) are an alternative for regenerative cellular therapy, in large part through production of several molecules including cytokines, chemokines, growth factors, extracellular matrix proteins. The purpose of the present study was to assess the effect of the CM of human embryonic stem cell-derived MSCs on neurogenesis in ischemic stroke model rats. **Methods :** In male Wistar rats, ischemic stroke was induced by 90-min right middle cerebral artery occlusion (MCAO). The concentrated CM from human MSCs or DMEM (5 $\mu$ l) respectively were infused through a brain guide cannula into the left lateral ventricle of treatment and control animals three times at 1, 24 and 48 hours after MCAO induction. Furthermore, the MSC-CM was administered a single dose, one hour after reperfusion in another treatment group. The expression of nestin, Ki-67, and doublecortin (DCX) proteins in corpus callosum region were assessed by immunolabeling at 7 days after MCAO. **Results :** Our results indicated that following ischemic stroke, levels of neurogenesis markers increased in the corpus callosum. The number of nestin-, ki67-, DCX-positive neuroblasts in the corpus callosum significantly increased by CM treatments. It seems the immunopositive cells migrate from the neurogenic niche, subventricular zone (SVZ) of the lateral ventricles, toward the peri-infarct areas along the corpus callosum. **Conclusion :** Our data suggest that CM of human embryonic stem cell-derived MSCs contribute to neuroprotection following cerebral ischemia insult by promoting neurogenesis.

## کلمات کلیدی:

**لینک ثابت مقاله در پایگاه سیویلیکا:**

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