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Perspective

Functions of Neural Stem Cells in Treatment of Ischemic Brain Repair Processes

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DESCRIPTION

Infant impairments are most commonly brought on by hypoxic/ischemic damage, whereas stroke continues to be the largest cause of morbidity in both children and adults worldwide. Because the wounded brain has a limited ability to heal, there is only a slight increase in neurological function after the injury. In rats, adult neural stem cells in the ventricular-subventricular zone of the lateral ventricle make new neurons throughout the course of the animal's life, while embryonic neural stem cells in the ventricular zone create cortical neurons throughout development. Additionally to producing new neurons, neural stem cells also aid in the process of oligodendrogenesis.

For the wounded brain to heal, neurogenesis and oligodendrogenesis are crucial. The understanding of the cellular and molecular mechanisms that regulate and coordinate neurogenesis and oligodendrogenesis in perinatal hypoxic/ischemic injury and the adult ischemic brain has advanced significantly in preclinical investigations. This article will examine these findings with an emphasis on the neurogenic niche in the ventricular-subventricular zone and discuss potential uses to promote endogenous neurogenesis and consequently enhance neurological function following hypoxic-ischemic injury and stroke in perinatal patients. The most significant factor contributing to brain damage from birth problems those results in long-term neurological abnormalities is Hypoxic/Ischemic (H/I) injury. Half of preterm infants who survive perinatal H/I injury occur every year, or 1-2 per 1000 term births. Many of these infants have long-term problems, such as cerebral palsy, epilepsy, learning difficulties, and mental retardation. Around the world, stroke continues to be a major cause of morbidity. The only FDA-approved treatment for patients with an ischemic stroke that began within 4.5 hours is tissue Plasminogen Activator (tPA).

Endovascular thrombectomy with or without tPA is effective for ischemic stroke patients within 12 hours of stroke onset, according to successful randomised clinical trials. This finding suggests that prompt recanalization and reestablishing Cerebral Blood Flow (CBF) can preserve vascular integrity and minimise brain haemorrhage and parenchymal cell death. However, because the ischemic brain has a limited potential for repair, the majority of patients will experience neurological abnormalities during stroke recovery, even with efficient thrombolysis. For the development of the brain and the healing of damaged brain tissue, neurogenesis is crucial. Cortical neurons are produced by embryonic neural stem cells in the Ventricular Zone (VZ). There are at least two neurogenic areas in the adult mammalian brain: the Sub Granular Zone (SGZ) of the dentate gyrus and the Ventricular-SubVentricular Zone (V/SVZ) of the lateral ventricle. Injury to the foetal H/I promotes acute neurogenesis. Adult rodents with focal cerebral ischemia had increased neurogenesis, especially in the V/SVZ, as well as neuroblast migration to the ischemic boundary. After a stroke, newly formed neuroblasts play a role in functional recovery. The adult human brain has also shown signs of stroke-induced neurogenesis. The understanding of the cellular and molecular mechanisms that regulate and coordinate neurogenesis following perinatal H/I injury and in the adult ischemic brain has advanced significantly. We will address potential uses to encourage endogenous neurogenesis and thereby enhance neurological function following perinatal H/I injury and stroke as we go over these findings with an emphasis on the V/SVZ neurogenic niche. Radial glial cells in the VZ are neural stem cells during the embryonic stage.

While a population of dormant embryonic neural stem cells makes up the bulk (73%) of adult neural stem cells, actively proliferating embryonic neural stem cells in the VZ support cortical neurogenesis. By extending their apical processes, which are anchored at the ependymal layer of the ventricular surface, Glial Fibrillary Acidic Protein (GFAP) positive neural stem cells in the SVZ directly contact the Cerebro Spinal Fluid (CSF), according to *in vivo* studies using whole-mount tissue preparations of adult rodent brains. The stem cells also project their long basal processes to reach blood vessels in the SVZ that are located just below the epen. Thus, despite the adult brain's

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ependymal layer replacing the embryonic VZ, these findings show that adult neural stem cells exist in the V/SVZ. Furthermore, *in vivo* investigations employing genetic techniques show that quiescent and activated GFAP-positive neural stem cells coexist in the V/SVZ, expressing, respectively, phenotypes of GFAP/CD133 and GFAP/CD133/Epidermal Growth Factor Receptor (EGFR).