Stroke-induced neurogenesis

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Stroke is caused by occlusion of a cerebral artery, which gives rise to focal ischemia with irreversible injury in a core region and partially reversible damage in the surrounding penumbra zone.

Stroke is the leading cause of disability in adult humans in developed countries. After ischemic stroke neurons are rapidly damaged and usually die but cellular loss can occur hours and days thereafter. Stroke increases stem cell proliferation in the subventricular zone (SVZ) and the generated neuroblasts migrate to the stroke-damaged area of the brain where they become mature neurons. This process may continue for several months and produce a significant number of new, functional neurons. The evidence of increased neuroblast production after stroke has also been demonstrated in human brain. A bulk of experimental evidence supports the idea that the stroke-damaged adult brain makes an attempt to repair itself by producing new neurons also in areas where neurogenesis does not normally occur, e.g., striatum and cerebral cortex. Knowledge about mechanisms regulating the different steps of neurogenesis after stroke is rapidly increasing but still incomplete. The functional consequences of stroke-induced neurogenesis and the level of integration of the new neurons into existing neural circuitries are poorly understood. In order to have a substantial impact on the recovery after stroke, this potential mechanism for self-repair needs to be markedly enhanced, primarily by increasing the survival and differentiation of the generated neuroblasts. Moreover, for efficient repair, optimization of neurogenesis most likely needs to be combined with promotion of other endogenous neuroregenerative responses, e.g., protection and sprouting of remaining mature neurons, and transplantation of stem cell-derived neurons and glia cells.