Neuroblast

A neuroblast is a dividing cell that will develop into a neuron often after a migration phase.

Neuroblasts differentiate from neural stem cells and are committed to the neuronal fate.

The main difference between a neuroblast and a neuron is the ability to divide; neuroblasts can still undergo mitosis, whereas neurons are postmitotic.

Neuroblasts are mainly present as precursors of neurons during embryonic development, however, they also constitute one of the cell types involved in adult neurogenesis. Adult neurogenesis is characterized by neural stem cell differentiation and integration in the mature adult mammalian brain. This process occurs in the dentate gyrus of the hippocampus and in the subventricular zones of the adult mammalian brain. Neuroblasts are formed when a neural stem cell, which can differentiate into any type of mature neural cell (i.e. neurons, oligodendrocytes, astrocytes, etc.), divides and becomes a transit amplifying cell. Transit amplifying cells are slightly more differentiated than neural stem cells and can divide asymmetrically to produce postmitotic neuroblasts or glioblasts, as well as other transit amplifying cells. A neuroblast, a daughter cell of a transit amplifying cell, is initially a neural stem cell that has reached the “point of no return.” A neuroblast has differentiated such that it will mature into a neuron and not any other neural cell type.

Neuroblasts are being studied extensively as they have the potential to be used therapeutically to combat cell loss due to injury or disease in the brain, although, their potential effectiveness is debated.

Neuroblasts migration

In humans, neuroblasts produced by stem cells in the adult subventricular zone migrate into damaged areas after brain injuries. However, they are restricted to the subtype of small interneuron-like cells, and it is unlikely that they contribute to functional recovery of striatial circuits.

Neurogenesis has provided therapeutic options for treating TBI. Brain derived neurotrophic factor (BDNF) plays a key role in neuroblasts migration.

Wu et al. aimed to investigate to the key regulating principle of BDNF in endogenous neuroblasts migration in a mouse TBI model.

In this study, controlled cortical impact (CCI) mice (C57BL/6J) model was established to mimic TBI. The sham mice served as control. Immunofluorescence staining and enzyme-linked immunosorbent assay were performed on the CCI groups (day 1, 3, 7, 14 and 21 after CCI) and the sham group. All the data were analyzed with Student's t-test or one-way or two-way analysis of variance followed by Tukey's post hoc test.

The results revealed that neuroblasts migration initiated as early as day 1, peaking at day 7, and persisted till day 21. The spatiotemporal profile of BDNF expression was similar to that of neuroblasts migration, and BDNF level following CCI was consistently higher in injured cortex than in

subventricular zone (SVZ). Reactive astrocytes account for the major resource of BDNF along the migrating path, localized with neuroblasts in proximity. Moreover, injection of exogenous CC chemokine ligand 2 (CCL2), also known as monocyte chemoattractant protein-1, at random sites promoted neuroblasts migration and astrocytic BDNF expression in both normal and CCI mice (day 28). These provoked neuroblasts can also differentiate into mature neurons. CC chemokine ligand receptor 2 antagonist can restrain the neuroblasts migration after TBI.

Neuroblasts migrated along the activated astrocytic tunnel, directed by BDNF gradient between SVZ and injured cortex after TBI. CCL2 might be a key regulator in the above endogenous neuroblasts migration. Moreover, delayed CCL2 administration may provide a promising therapeutic strategy for late neurogenesis post-trauma [1].

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Wu N, Sun X, Zhou C, Yan J, Cheng C. Neuroblasts migration under control of reactive astrocyte-derived BDNF: a promising therapy in late neurogenesis after traumatic brain injury. Stem Cell Res Ther. 2023 Jan 5;14(1):2. doi: 10.1186/s13287-022-03232-0. PMID: 36600294.