Tyrosine-protein phosphatase non-receptor type 11 (Shp2) is a widely expressed intracellular enzyme with multiple cellular functions. Previous studies have revealed the critical role of Shp2 in a variety of neural cell types; however, further investigation into the function of Shp2 within radial glia is required. In the present study, a conditional knockout mouse was generated using a human glial fibrillary acidic protein (hGFAP)-Cre driver, in which the Shp2 genes were deleted within radial glia. Loss of Shp2 within radial glia was associated with developmental retardation, postnatal lethality, reduced brain size and thinner cerebral cortices in newborn mice. Deletion of Shp2 also led to an increase in gliogenesis, a reduction in neural genesis and extracellular signal-regulated kinase signaling within the cerebral cortex. Furthermore, glial cell defects within the cerebellum of Shp2 mutants were observed, with abnormal granular cell retention and glial cell alignment in the external granular layer. In addition, Shp2 mutants exhibited impaired sensory-motor development. The results of the present study suggested that Shp2 may have an important role within radial glia, and regulate cerebral cortical and cerebellar development in newborn mice [1)].

1)
Zhu Y, Shen J, Sun T, Jiang H, Xu K, Samuthrat T, Xie Y, Weng Y, Li Y, Xie Q, Zhan R. Loss of Shp2 within radial glia is associated with cerebral cortical dysplasia, glial defects of cerebellum and impaired sensory-motor development in newborn mice. Mol Med Rep. 2017 Dec 11. doi: 10.3892/mmr.2017.8236. [Epub ahead of print] PubMed PMID: 29257282.