

Publication**A Transient Expression of Prospero Promotes Cell Cycle Exit of Drosophila Postembryonic Neurons through the Regulation of Dacapo****JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)****ID** 749184**Author(s)** Colonques, J.; Ceron, J.; Reichert, H.; Tejedor, F. J.**Author(s) at UniBasel** [Reichert, Heinrich](#) ;**Year** 2011**Title** A Transient Expression of Prospero Promotes Cell Cycle Exit of Drosophila Postembryonic Neurons through the Regulation of Dacapo**Journal** PLoS ONE**Volume** 6**Number** 4

Cell proliferation, specification and terminal differentiation must be precisely coordinated during brain development to ensure the correct production of different neuronal populations. Most Drosophila neuroblasts (NBs) divide asymmetrically to generate a new NB and an intermediate progenitor called ganglion mother cell (GMC) which divides only once to generate two postmitotic cells called ganglion cells (GCs) that subsequently differentiate into neurons. During the asymmetric division of NBs, the homeodomain transcription factor PROSPERO is segregated into the GMC where it plays a key role as cell fate determinant. Previous work on embryonic neurogenesis has shown that PROSPERO is not expressed in postmitotic neuronal progeny. Thus, PROSPERO is thought to function in the GMC by repressing genes required for cell-cycle progression and activating genes involved in terminal differentiation. Here we focus on postembryonic neurogenesis and show that the expression of PROSPERO is transiently upregulated in the newly born neuronal progeny generated by most of the larval NBs of the OL and CB. Moreover, we provide evidence that this expression of PROSPERO in GCs inhibits their cell cycle progression by activating the expression of the cyclin-dependent kinase inhibitor (CKI) DACAPO. These findings imply that PROSPERO, in addition to its known role as cell fate determinant in GMCs, provides a transient signal to ensure a precise timing for cell cycle exit of prospective neurons, and hence may link the mechanisms that regulate neurogenesis and those that control cell cycle progression in postembryonic brain development.

Publisher Public Library of Science**ISSN/ISBN** 1932-6203**edoc-URL** <http://edoc.unibas.ch/dok/A5844178>**Full Text on edoc** No;**Digital Object Identifier DOI** 10.1371/journal.pone.0019342**PubMed ID** <http://www.ncbi.nlm.nih.gov/pubmed/21552484>**ISI-Number** WOS:000290020700049**Document type (ISI)** Article