Disclosures: M.J. McDonough: None. **M.S. Saha:** None.

Poster

231. Cell Fate

Location: Hall F-J

Time: Sunday, October 14, 2012, 1:00 PM - 5:00 PM

Program#/Poster#: 231.15/ A41

Topic: A.02. Neurogenesis and Gliogenesis

Support: National Science Council Grant NSC-98-2320-B-039-038

China Medical University Grant CMU98-N1-32

Title: Spatiotemporal expression of murine Nolz-1/Zfp503 implicates a potential role in developmental regulation of ventral thalamus in the mouse brain

Authors: Y.-C. LIU[1], S.-Y. CHEN[2], F.-C. LIU[2], *L.-Y. CHANG[1]
[1]Grad. Inst. of Mol. Systems Biomedicine, China Med. Univ., Taichung, Taiwan; [2]Inst. of Neuroscience, Natl. Yang-Ming Univ., Taipei, Taiwan

Abstract: *Nolz-1* (also known as *Zfp503*), a murine member of the NET zinc finger genes family, is related to the embryonic development of the central nervous system. Specific expression of *Nolz-1* mRNA in distinct domains and neuronal cells of developing striatum, hindbrain and spinal cord has recently been documented. However, the spatiotemporal expression pattern of Nolz-1 in other brain regions is still unrevealed. In the present study, we analyzed the expression of Nolz-1 protein in developing diencephalon at embryonic day (E) 11.5 to E16.5 by immunohistochemistry analysis. Expression of Nolz-1 was detected in the roof plate of diencephalon, ventral part of hypothalamus and distinct domains of ventral thalamus, but was not detected in dorsal thalamus. The ventral thalamus is composed of three major nuclei, reticular (RT), zona incerta (ZI) and ventral lateral geniculate (vLG). During development, the post-mitotic vLG neurons migrate laterally alone the zona limitans intrathalamica (ZLI), the transverse border of ventral and dorsal thalamus, to their final destination. By comparing with the expression patterns of Pax6, Isl1, Lhx1/5 transcription factors and reelin secretary protein, whose expressions mark distinct ventral thalamic nuclei, we found that Nolz-1 was not expressed in RT nucleus, but was preferentially expressed in vLG nucleus and rostral, ventral and dorsal ZI (ZIr, ZIv and ZId, respectively) nuclei. Differential spatiotemporal expression patterns of Nolz-1 and other markers within these nuclei were observed by double immunostaining. The results showed that Nolz-1 was only expressed in subdivision neurons of vLG and ZI nuclei. In summary, our results suggest that Nolz-1 may play a regulatory role in developmental control of ventral thalamic nuclei, *i.e.*, regulating specification and differentiation of specific cell types with a nuclei subdivision-specific manner. Supported by: NSC97-2320-B-039-038 and CMU98-N1-32.

Disclosures: Y. Liu: None. **S. Chen:** None. **F. Liu:** None. **L. Chang:** None.

Poster

231. Cell Fate

Location: Hall F-J

Time: Sunday, October 14, 2012, 1:00 PM - 5:00 PM

Program#/Poster#: 231.16/ A42

Topic: A.02. Neurogenesis and Gliogenesis

Support: Estonian Ministry of Education and Research Grant 0140143

Title: Neuralized-1 is an e3 ubiquitin ligase for cgmp-specific phosphodiesterase 9a.

Authors: *K. TAAL, G. RULLINKOV, M. PIIRSOO, M. SEPP, T. NEUMAN, R. TAMME, T. TIMMUSK
Dept. of Gene Technol., Tallinn Univ. of Technol., Tallinn, Estonia

Abstract: Neuralized (Neur) functions as an important positive regulator in the Delta-Notch pathway in flies by promoting ubiquitination of Notch ligands Delta and Serrate, which results in their efficient endocytosis and signalling. However, removal of the mouse homologs of Drosophila neuralized, Neur1 or Neur2, does not result in any obvious Notch-related defects. Surprisingly, mice homozygous for Neur1 or Neur2 null mutation are fully viable and without any developmental defects, indicating that this gene is not essential for development and survival in mammals. Therefore, it is possible that Neur has some yet unidentified functions outside the Notch pathway. In a yeast two-hybrid based screen we have identified a cGMP-hydrolyzing phosphodiesterase, PDE9A, as an novel interactor for mouse Neur1. We confirmed this interaction using both co-immunoprecipitation and co-localization experiments. We show that both Neuralized Homology Repeat (NHR) domains of Neur1 can interact with PDE9A and that this interaction results in re-localization of PDE9A from the cell membranes to the cytosol. We also show that Neur1 can promote ubiquitination of PDE9A, and direct PDE9A to proteasome-mediated degradation. Neur2 can also interact with PDE9A, however, with lower efficiency and is not able to re-localize PDE9A to the cytosol. On the contrary to Neur1, PDE9A does not seem to be a substrate for Neur2 since it is not able to ubiquitinate PDE9A. In conclusion, our results suggest that Neur1 may act as a novel regulator of PDE9A.

Disclosures: K. Taal: None. **G. Rullinkov:** None. **M. Piirsoo:** None. **M. Sepp:** None. **T. Neuman:** None. **R. Tamme:** None. **T. Timmusk:** None.

Poster

231. Cell Fate

Location: Hall F-J