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Poster

390. Oligodendrocytes and Schwann Cells: Development, Neuron-Interaction, and Myelination

Location: Halls B-H

Time: Monday, November 14, 2016, 1:00 PM - 5:00 PM

Program#/Poster#: 390.01/A1

Topic: A.01. Neurogenesis and Gliogenesis

Support: Wellcome Trust

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Title: Developmental increase in cortical myelination and internodal length variability in the mouse neocortex

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Abstract: Myelin is essential for cognitive functioning and its loss or damage is associated with a number of neurological disorders and mental illnesses [1]. Alternations in myelination are increasingly being implicated as a mechanism for learning. Despite the importance of myelin, age-related myelination changes are poorly understood. Increases in white matter volume have been observed across several brain regions in childhood and adolescence, both in humans and other species[2,3,4]. However, while many studies investigate myelination in rodent models, the exact nature of developmental myelination changes in mice is still unclear. Importantly, differential myelination patterns have been found in distinct cortical layers within the mouse brain[5]. In addition to the overall myelination pattern, various other structural features such as the amount and length of internodes along axons may potentially affect conduction speed and change over time. In the peripheral nervous system, a functional relationship between internodal distance and conduction speed has been demonstrated and internodal length increases throughout development[6]. The question arises whether the same pattern occurs in the central nervous system (CNS). Therefore, we investigated myelin distribution and structure in the mouse brain neocortex at various time points during development (*p0-p118*). In addition to overall myelination we studied specific myelin features such as internodal length. We used immunohistochemistry to stain for myelin changes and by using high-resolution confocal imaging we were able to quantify cortical myelination and internodal distances. Our preliminary results indicate an increase in cortical myelination over time: a gradual increase in all cortical layers was observed, with no myelination present in the first week. While we indeed observed higher myelin density in deeper layers, our results suggest that the variability of internodal length (instead of overall internodal

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Title: Human Schwann cell senescence is not prevented by ectopic expression of human telomerase reverse transcriptase

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Abstract: Isolated human Schwann cells (hSCs) typically become senescent and unresponsive to mitogenic factors with continued in vitro expansion. Data from our lab has shown that adult nerve-derived hSCs, in contrast to rodent (rat) SCs, inevitably stop proliferating and acquire a senescent phenotype characterized by high levels of senescence-associated (SA)-β galactosidase activity and morphological changes that include cell enlargement and appearance of multi-nucleated cells. As opposed to rodent SCs, hSC cultures consist of mixed populations of proliferating cells, senescent cells and cells at different stages of differentiation regardless of the nerve of origin and other donor-specific factors. RNA-seq analysis of representative cultures of hSCs did not reveal the presence of telomerase reverse transcriptase (TERT) mRNA while other TERT-related genes (e.g. TERF1, telomeric repeat binding factor, and TEP1, telomerase-associated protein) were well-represented in the hSC transcriptome. In an attempt to overcome senescence, we used retroviral vectors and antibiotic selection to generate hSC lines ectopically expressing human (h)-TERT. For these experiments, highly proliferative, non-senescent, early passage hSC cultures were stably transduced with the retroviruses h-TERT-hygro or h-TERT-puro, each encoding the h-TERT gene along with hygromycin or puromycin resistance genes, respectively. Transduced hSC cultures from three different donors were selected and subjected to three rounds of expansion in medium containing chemical mitogens. Subsequently, the cultures were analyzed for their rate of proliferation by means of EdU incorporation assays and the acquisition of senescence by means of SA-β galactosidase activity assays in each round. We

found that whereas ectopic h-TERT expression extended the lifespan of cultured hSCs when compared to non-infected or GFP-expressing cells, it was not sufficient to confer immortalization and overcome senescence. In sum, our results suggest that progression of the hSCs to a senescent state likely is stress-induced rather than dependent on replication-associated telomere shortening.

Disclosures: **N.D. Andersen:** None. **B. Kuo:** None. **G. Piñero:** None. **K. Ravelo:** None. **P. Rai:** None. **P. Monje:** None.

Poster

390. Oligodendrocytes and Schwann Cells: Development, Neuron-Interaction, and Myelination

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Shriners Hospitals for Children

Title: Role of neural stem factor sox2 in postnatal oligodendrocyte development

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Abstract: Sox2, a SoxB1 transcription factor, plays an important role in maintaining the stemness of neural stem cells (NSCs) in both embryonic and adult ages in the central nervous system (CNS). It is generally thought that Sox2 is downregulatedor absent in the progenies derived from NSCs. Our previous study reports that Sox2 expression is maintained in the adult quiescent astrocytes (Guo et al., 2011 J Neurosci). Here, we showed a dynamic expression pattern of Sox2 in the oligodendroglial lineage cells.Sox2 is expressed at low level in virtually all oligodendroglial progenitor cells (OPCs) at both early postnatal and adult CNS, significantly upregulated in premyelinating oligodendrocytes (OLs) and downregulated in postmyelinating OLs after the completion of CNS myelination. Based on these observation, we hypothesize that Sox2 may play stage-dependent roles (**CNS developmental stages and oligodendrocyte developmental stages**) during postnatal oligodendrocyte development. Using inducible Cre-LoxP conditional knockout (cKO) system to ablate Sox2 in early postnatal OPCs, we