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NG2+ Oligodendrocyte Precursor Cells (OPCs) take part in neurovascular unit remodelling during EAE

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NG2+ Oligodendrocyte Precursor Cells (OPCs) are an abundant glial population in mammalian adult Central Nervous System (CNS) committed to oligodendrocyte differentiation and myelin repair (1). Our previous studies in a model of murine Experimental Autoimmune Encephalomyelitis (EAE) revealed demyelination of cerebral cortex subpial areas together with an increased number of OPCs and Blood-Brain Barrier (BBB) breakdown (2,3). The observation of several EAE activated OPCs in tight contact with EAE cortex microvessels prompt us to further analyse the nature of this association and ascertain if during EAE, OPCs establish a privileged relationship with BBB microvessels. Laser confocal microscopy and morphometric analyses were applied to immunohistochemically revealed cell- (OPCs, endothelial cells, pericytes, astrocytes) and vascular basement membrane- (collagen type IV and VI) specific antigens. The main vessel/OPC assessed parameters were OPC vascular coverage (OPC number/vessel length) and raw integrated density (OPC area x mean gray value)/vessel density which were calculated in brains from both healthy control and EAE affected mice. The results confirmed the increased number of EAE OPCs, their enhanced branching and frequent perivascular location. Compared with healthy mice, in EAE the OPC coverage and the integrated density, as well as the evaluated minimal vascular OPC distance were significantly increased. These data suggest that in EAE, OPCs assume a critical position with respect to the vessel wall, remaining distinct from GFAP+ astrocytes, as an additional cell component of the neurovascular unit. In this position, the increased vascular subset of EAE NG2+ OPCs may interfere with neurovascular unit cell-cell signalling in turn affecting BBB regulation and maintenance.

References:
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Key words
EAE, OPC, neurovascular unit, NG2.