New pathogenic paths in Multiple Sclerosis: better diagnosis and new therapies?

Multiple Sclerosis (MS) is the paradigm of demyelinated diseases, in which the myelin-forming cells of the CNS, the oligodendrocytes, die. MS has been traditionally considered as an autoimmune disease and current treatments are immunomodulators. In the last years, intensive work in the neurodegenerative facets of demyelination opened new perspectives for neuroprotective and neurorepair therapies. Combining the use of animal and human samples, our group explores novel aspects which have substantially modified aspects of MS pathogenesis and could ameliorate the diagnosis of the disease, like i) the presence and detection of the FGF-2/anosmin-1 system, ii) the role of FGF-2 and megalin in blood-brain-barrier functioning and iii) the role of myeloid-derived suppressor cells in demyelination. Among other research lines pursued in our lab, these exemplify the implication of developmental neurobiology in pathogenesis, CNS reaction to demyelinating injury, the relevance of endogenous oligodendrocyte precursor cells and spontaneous remyelination and the potential of the latter to design future therapeutic approaches complementing current MS treatment.

Selected publications: