Neuronal control of inflammation

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The neuronal control of inflammation can be evaluated from at least three different points of view: the historical prospective of neurogenic inflammation, which involves tachykinin neuropeptides, mainly Substance P (SP), the neuroprotective effect afforded by peroxisome proliferators activated receptors (PPARs) agonism and the inflammatory response which is observed in the CNS during the neurodegenerative diseases.

Our group has contributed especially to the first and second of the three research fields depicted above. SP receptors have been identified on pro-inflammatory cells and their agonism can evoke a large array of inflammatory responses. Most of the data on the neurogenic inflammation are referred to the peripheral nervous system, but neurogenic inflammation has been involved in the pathogenesis of migraine in the past and, more recently, a few studies have demonstrated that it plays a role in acute brain injury. The activation of the PPAR isoforms and has been demonstrated by several experimental data, including those obtained in our laboratory, to prevent post-ischemic inflammation and neuronal damage, negatively regulating the expression of genes induced by acute brain injury, including ischemic stroke. Ongoing data point to a similar patho-physiological role of PPAR /.

Furthermore, PPAR agonism has been demonstrated by our group to promote neuronal differentiation and neurite outgrowth, thus suggesting a possible role of these receptors in the formation and conservation of a proper neuronal connectivity within neuronal networks.