Mechanisms of spinal neuroinflammation and hyperexcitability in models of joint inflammation

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In our human body nociceptive neurons (spinal neurons) are responsible for the pain regulation. These neurons can differentiate between noxious (harmful) and innocuous (non-harmful) input, and play an important role to protect our body from further injury. Nociceptive processes, especially in chronic pain states are accompanied by spinal hyperexcitability and central sensitization. Due to this phenomenon, spinal neurons increase their activity to noxious and innocuous stimulation and expand their receptive field sizes to areas which are not affected by the peripheral insult (e.g. inflammation). The spinal nociceptive neurons interact densely with spinal glial cells and immune mediators released by neurons and glia to generate and maintain spinal hyperexcitability.

My main objective in this project as ESR 7 of TOBeATPAIN is to explore different cytokines involved in this spinal hyperexcitability and their origin of secretion and to know the exact mechanism to ease the complexity in diagnose the spinal hyperexcitability for the appropriate treatment.

To achieve the target, I am using in vivo extracellular electrophysiological recordings from spinal cord neuron that communicate the electrical impulses with carbon fiber electrodes and in addition, I am using molecular techniques (e.g. Western Blot) in vitro to differentiate between responses of neurons and glia (microglia and astroglia) to cytokines within different time courses.

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