Involvement of altered HPA-axis reactivity and of stress in neuropathy-related pain sensitivity and in the regulation of spinal biochemical mediators

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At the clinical level, an altered hypothalamo-pituitary adrenal (HPA) axis reactivity is associated with a differential perception of pain. This thesis focused on neuropathic pain mechanisms using a chronic constriction injury (CCI) model investigated in different manipulations of the HPA axis.

In a first study, three strains of rats displaying different adrenocortical reactivity, Fischer rats with an enhanced level of circulating glucocorticoids (GC), Lewis rats with a lower one and Wistar rats as controls, were used. No specific correlation was demonstrated between spinal biomarkers on neuropathic pain and different levels of GC in these strains. This could be due to genetic modifications altering other systems.

In a second set of experiments, rats were injected daily with an agonist (dexamethasone) or an antagonist (RU 486) of the glucocorticoid receptors (GR). In this context, the excitatory mechanisms of the glutamatergic system exceeded the anti-inflammatory effects of the GC.

Finally, to my knowledge, no investigation associated a model of chronic stress with a neuropathic pain one. Usually this kind of study deals with inflammatory pain models. Using a CCI model of neuropathic pain associated with a model of chronic social stress, an exacerbated pain perception was perceived. Surprisingly alterations in spinal indicators expression related to neuropathic pain, including glial cells, pro-inflammatory cytokines, the glutamatergic system and neurotrophic factors, seemed to depend more on chronic stress mechanisms than on pain ones.

Hence, this thesis permitted a further comprehension of the relationship between the HPA axis reactivity and spinal mechanisms of neuropathic pain.

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