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Glial cells surrounding the damaged nerves release cytokines and proinflammatory mediators that activate resident immune cells and recruit circulatory immune cells. Toll-like receptors on the glial cells play a crucial role in the pathogenesis of chronic pain. Animal models indicate an immune mechanism of neuropathic pain.

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Overview: This translational research program addresses pathophysiological processes related to neuropathic pain and the potential confluence of chronic pain, autoimmunity, infectious diseases, and their intersections in human patients. Chronic neuropathic pain can affect any part of the body and can occur due to a variety of insults, infections, autoimmune or metabolic disorders (e.g., diabetic peripheral neuropathy).

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chronic pain Immune mechanisms that regulate the blood-brain-interface (BBI) Immune factors that affect health psychology Sleep, exercise, immunity and health Immune system interactions that affect behavior following use of psychotropic drugs, alcohol

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Abstract. The role of central glial cells in the mechanisms underlying pain has been intensively studied in the last two decades. Most studies on glia and pain focused on the potential detrimental role of glial cells following noxious stimulus/insults manifested as an "activation" or a "reactive" state (increase in glial marker expression and production of proinflammatory/nociceptive molecules).

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