



## **Pain and Neurogenic Inflammation (Progress in Inflammation Research)**

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Pain and inflammation are inextricably linked phenomena. The observation that chemical mediators with combined pro-inflammatory, algescic and/or hyperalgesic activity occur at the site of inflammation is fundamental not only to our present understanding of the inflammatory process but also to our attempts to devise clinically useful anti-inflammatory therapies. Over a hundred years ago it was recognised that primary sensory neurones play a crucially important "dual" role in inflammation. By affecting the transfer of information from peripheral nociceptors to the spinal cord, a subpopulation of sensory nerves {"pain fibres"} initiate algescia and hyperalgesia, whose sensations are then modified and fine-tuned in the central nervous system. Equally important is the release from the peripheral terminals of sensory neurones of neuropeptides, the acute effects of which are observed as changes in microvascular tone and permeability leading to neurogenic inflammation. Over the last decade it has become increasingly clear that this view of the function of sensory nerves is somewhat over-simplified. For example, the mechanisms underlying hyperalgesia may, in certain circumstances, be mimicked in other conditions such as the hypersensitivity associated with asthma. Furthermore, it has become increasingly evident that over a longer time period the release of neuropeptides from peripheral sensory nerve endings may also have modulating effects on immune cells and that this may be relevant to chronic inflammatory disease and possibly also to inflammatory hyperalgesia.

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