



Molecular Links between Sensory Nerves, Inflammation, and Pain 2.0

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Message from the Guest Editors

Capsaicin-sensitive peptidergic sensory nerves do not only transfer sensation and pain into the central nervous system (afferent function), but they also exert important efferent functions. They play complex regulatory roles in a broad range of inflammatory and pain conditions, such as arthritis/osteoarthritis, gastrointestinal diseases (irritable and inflammatory bowel diseases), neuropathic pain, and migraine. Several pro- and anti-inflammatory neuropeptides and other mediators (tachykinins, calcitonin gene-related peptide, pituitary adenylate cyclase-activating polypeptide, somatostatin, and purines) are released in response to their activation. Their balance and functions on immune cells and vessels determine the overall role of these nerves in different pathophysiological conditions related to unmet medical need diseases. Furthermore, inflammatory cell-derived mediators act back on these nerves to induce activation or inhibition. Exploring the molecular mechanisms of the complex sensory-immune-vascular interactions and identifying key targets can open promising novel anti-inflammatory and analgesic drug developmental perspectives.





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Message from the Editor-in-Chief

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