Mechanisms of visceral hyperalgesia: identification of molecular targets

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Visceral pain is the most frequent form of clinically relevant pain. The study of its mechanisms is therefore immediately relevant to human pain conditions but it also offers a unique insight into the generation of hyperalgesic states. All forms of visceral pain generate enhancements of pain sensitivity in locations remote from the originating injury, a process known as "referred hyperalgesia" that is equivalent to the secondary hyperalgesia that develops following a somatic injury. In some cases, referred hyperalgesia can be the only manifestation of an altered pain state in the absence of an apparent injury or dysfunction of an internal organ. Referred hyperalgesia, like secondary hyperalgesia, is the expression of an alteration of sensory processing in the CNS and analysis of the molecular targets implicated in its generation can shed light on the general mechanisms of pain hypersensitivity. In this lecture the spinal cord mechanisms implicated in the generation of visceral hyperalgesia will be discussed with reference to an animal model of referred visceral hyperalgesia and to some of the potential molecular mediators of visceral hyperalgesic states.