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Research Note

Pain rudiments: spins around Pain fibers

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Abstract

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (Merskey and Bogduk, 1994). Pain is felt as a result of the brain's response to electrical (neural) and chemical (hormonal) changes in the body as a result of damage, disease or injury. The propagation of pain is initiated with the activation of physiological receptors, called nociceptors, which are free nerve endings and represent the most distal part of a first-order afferent neuron consisting of small-diameter fibers, with little or unmyelinated, of A-delta or C-type, respectively (Almeida *et al*., 2004). Nociception consists of four processes: transduction, transmission, perception, and modulation. Transduction is the cellular process by which noxious stimuli are changed into the electrical energy necessary to transmit pain (Fine and Ashburn, 1998). Transmission of the pain impulse along the nociceptor fibers (nerve axons) begins when transduction is complete. During the first segment of transmission, the impulse is carried along nociceptor fibers in an ascending fashion to the dorsal horn of the spinal cord. This is followed by transmission from the spinal cord to the brain stem and thalamus. The thalamus, acting as a relay station, sends the impulse to the cortex where it can be processed (Pasero *et al*., 1999). Modulation (inhibition and alteration) of pain transmission occurs at several locations within the central nervous system (CNS) (Fine and Ashburn, 1998). The neuronal pathways involved in modulation are often referred to as the descending pain system because they originate in the brain stem and descend to the dorsal horn of the spinal cord (Portenoy and Kanner, 1996). These descending pathways release substances, such as endogenous opioids, serotonin (5HT), and norepinephrine (NE), which can inhibit the transmission of noxious stimuli and produce analgesia (Pasero *et al*., 1999, 2004). In the endorphinergic system, analgesia is mediated by the binding of endogenous opioid compounds to special subsets of receptors: mu, delta, and kappa.

Pain fibres:

Information about noxious events in the periphery is signaled to the spinal cord in unmyelinated (C-fiber) and myelinated (A-fiber) nociceptor afferents. A- and C-fiber nociceptors convey different qualities of the pain signal and they play different roles in the development and maintenance of chronic pain states (Fuchs *et al*., 2000; Magerl *et al*., 2001; Simpson *et al*., 2007). Sensory axons can be classified according to diameter and conduction velocity. The largest and fastest axons are called Aα, and include some of the proprioceptive neurons, such as the stretch receptor. The second largest group is called Aδ, which includes all of the discriminative touch receptors. Pain and temperature include the third and fourth groups, Aδ and C fibers. There are two subtypes of pain. "Fast pain", carried by the Aδ fibers, is the sharp, piercing and instantaneous

pain. "Slow pain" is carried by C fibers. C fibers are not only small, they are unmyelinated (the only sensory axons without myelin), so their conduction velocity is quite slow (Schnitzler and Ploner, 2000). Slow pain is distressing, dull and aching, and it does not trigger withdrawal reflexes like the fast pain. Slow pain is primarily mediated by tissue-damage peptides like substance P, histamine and prostaglandins (Almeida *et al*., 2004).

Pain is divided into two different groups: physiological pain and pathological pain. Physiological pain is an important physiological function for survival. Unlike physiological pain, pathological pain only happen after injury (e.g., tissue or nerve injury), and is not the result of repetitive application of physiological pain. Long-term changes are likely to occur after injury, both peripherally and centrally (Melzack *et al.,* 2001). Consequently, the injury and injury-related areas undergo long-term plastic changes, and pain sensation is significantly enhanced (hyperalgesia) or non-noxious stimuli cause pain (allodynia) (Fuchs *et al*., 2000). Practically, pain can be classified into five different types, i.e., visceral, somatic, referred, neuropathic and psychogenic, according to their origins of pain signal generation. In acute pain (predominantly nociceptive), visceral, somatic and referred mechanisms play important roles in the pain perception. In chronic pain (frequently non-nociceptive), neuropathic and psychogenic mechanisms prevail, resulting in protracted suffering and disability both physically and mentally (Cheng and Ji, 2008; Polomano *et al*., 2008).

Conflict of Interest: None

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