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THE ROLE OF NEOROTRANSMITTERS IN PAIN AND ITS MANAGEMENT IN THE BODY

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Annotation
Pain is a complex experience. It is comprised of dynamic interactions between physical, cognitive, spiritual, emotional, and environmental factors and cannot be characterized as only a response to injury. The International Association for the Study of Pain and the American Pain Society defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." Pain modulation involves many different mechanisms that increase or decrease the transmission of pain signals throughout the nervous system. Depending on the mechanism, modulation can occur before, during, or after pain is perceived.

Keywords: pain, nociceptors, (prostaglandins, histamine, bradykinin, endogenous opioid, enkephalins, endocannabinoids, endorphin.

A wide variety of neurotransmitters act to modulate control over transmission of pain impulses in the periphery, spinal cord, and brain. The peripheral triggering mechanisms that initiate release of excitatory neurotransmitters include tissue injury (prostaglandins, histamine, bradykinin) and chronic inflammatory lesions (lymphokines). Glutamate, aspartate, substance P, and calcitonin are common excitatory neurotransmitters in the brain and spinal cord. These substances sensitize nociceptors by reducing the activation threshold, leading to increased responsiveness of nociceptors.Inhibitory neurotransmitters in the CNS include gamma-aminobutyric acid (GABA) and glycine. Norepinephrine and 5-hydroxytryptamine (serotonin) contribute to pain inhibition in the CNS but can excite peripheral nerves.

Endogenous opioids are a family of morphine-like neuropeptides that inhibit transmission of pain impulses in the periphery, spinal cord, and brain by binding with specific opioid receptors (mu [μ], kappa [κ], and delta [δ]) on neurons. They inhibit ion channels, preventing the release of excitatory neurotransmitters, such as substance P and glutamate, in the dorsal horn. In the midbrain they influence descending inhibitory pathways. In peripheral inflamed tissue, opioids are produced and released from immune cells and activate opioid receptors on sensory nerve terminals. Opioid receptors are widely distributed throughout the body and are responsible for general sensations of well-being and modulation of many physiologic processes, including control of respiratory and cardiovascular functions, stress and immune responses, gastrointestinal function, reproduction, and neuroendocrine control.

Enkephalins are the most prevalent of the natural opioids and bind to δ opioid receptors. Endorphins (endogenous morphine) are produced in the brain. The best studied endorphin is β-endorphin, which binds to μ receptors and is purported to produce the greatest sense of exhilaration as well as substantial natural pain relief. Dynorphins are the most potent of the endogenous opioids, binding strongly with κ receptors to impede pain signals. 781 Paradoxically, they play a role in neuropathic pain and in mood disorders and drug addiction. Endomorphins bind with μ receptors and have potent analgesic effects. Nociceptin/orphanin FQ is an opioid that induces pain or hyperalgesia but does not interact with opioid receptors. The nociceptin receptor is widely distributed throughout the PNS and CNS and is also associated with inflammation, immune regulation, mood, and emotion.

Synthetic and natural opiates have pharmacologic actions similar to morphine and bind as direct agonists to the opioid receptors. Morphine has a 50 times higher affinity for μ receptors in comparison with other opioids. Naloxone is the only clinically used opioid receptor antagonist, with a higher affinity for the μ receptors than for the other receptors.

Endocannabinoids are synthesized from phospholipids and are classified as eicosanoids. They activate cannabinoid CB1 (primarily in the CNS) and CB2 receptors (primarily in immune tissue [e.g., the spleen]) to modulate pain and other functions, including memory, appetite, immune function, sleep, stress response,

thermoregulation, and addiction. CB1 receptors decrease pain transmission by inhibiting release of excitatory neurotransmitters in the spinal dorsal horn, periaqueductal gray (PAG; the gray matter surrounding the cerebral aqueduct), thalamus, rostral ventromedial medulla (RVM), and amygdala. Cannabis (marijuana) produces a resin containing cannabinoids. Cannabinoids are analgesic in humans, but their use is limited by their psychoactive and addictive properties. Work is in progress to develop cannabinoid receptor agonists that do not have addictive side effects.

All in all, pain (nociception) is a complex, sensory experience involving emotion, cognition, and motivation. Acute pain is protective, promoting withdrawal from painful stimuli. Pain transmission is the conduction of pain impulses along the nociceptors into the spinal cord and eventually to the brain. Pain modulation increases or decreases the transmission of pain signals throughout the nervous system. Neuromodulators of pain include substances that stimulate pain nociceptors (e.g., prostaglandins, bradykinins, lymphokines, substance P, glutamate) and suppress pain (e.g., GABA, endogenous opioids, endocannabinoids). Some substances excite peripheral nerves but inhibit central nerves (e.g., serotonin, norepinephrine). Endogenous opioids inhibit pain transmission and include enkephalins, endorphins, dynorphins, and endomorphins. They are produced in the central nervous system and by immune cells.

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