

# The Clinical Implications of the Anatomy of Pain and Suffering in the Brain

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## Introduction

Pain is a distressing sensory and emotional sensation caused by actual or potential tissue damage. Chronic pain, which affects 20-30% of people worldwide, is the leading cause of human suffering because effective, specific, and safe therapies have yet to be developed. It is unequally distributed between the sexes, with women suffering more. Chronic pain can be divided into three distinct but interacting pathways: a lateral 'painfulness' pathway, a medial 'suffering' pathway, and a descending pain inhibitory pathway. One can experience pain without also experiencing suffering. Anger, fear, frustration, anxiety, and depression are all manifestations of pain sensation, which leads to suffering via cognitive, emotional, and autonomic processing.

Pain is defined as an unpleasant sensory and emotional experience that is associated with or described in terms of actual or potential tissue damage. Chronic pain, defined as pain that persists after the initial insult or injury has healed and thus lacks the acute warning function of physiological nociception, is not simply a temporal extension of acute pain but involves distinct mechanisms. Whereas acute pain can be considered a symptom of a larger problem, the International Association for the Study of Pain and the International Classification of Diseases 11 now define chronic pain as pain that lasts more than six months.

A stimulus has an effect on the various sensory receptors, which is transmitted to the sensory cortex and causes sensation. Other brain networks, such as the default mode, salience network, and frontoparietal control network, then process this sensory stimulation to generate an internal representation of the outer and inner worlds known as percept. Thus, perception can be defined as the act of interpreting and organising sensory stimuli in order to produce a meaningful experience of the world and oneself. When a person says he or she is "in pain", what the person actually says is "I have a certain amount of painfulness associated with a certain amount of suffering during a certain amount of time". These three pain components are phenomenological manifestations of three distinct pain-processing pathways [1].

## Description

Central sensitization can result in pain syndromes like fibromyalgia, temporomandibular joint disorders, chronic pelvic pain, and migraine. Centralization is mediated by infraslow oscillations in the ascending pain pathway, which are caused by altered neural astrocyte coupling. These pain syndromes are distinguished by more widespread or poorly localised pain, fatigue, mood and sleep disturbances, and a low quality of life. They

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are also frequently associated with other centralised pain syndromes and irritable bowel syndrome. These centralised pain syndromes are frequently linked to childhood adversity, such as sexual abuse and non-violent or violent childhood traumas. Although the terms "centralised pain" and "central sensitization" have been used interchangeably, central pain may be better defined as pain caused by lesions anywhere along the spino-thalamo-cortical pathway [2].

However, in addition to the genetic and epigenetic influences involved in the neuroinflammatory component of central sensitization, a third neuroinflammatory modulator may be relevant for the transition from acute to chronic pain: the microbiome. Numerous signalling molecules, such as metabolites, neurotransmitters, and neuromodulators, are produced by microbiota and act on nociceptors to regulate peripheral and central sensitization, thereby mediating the development of chronic pain. Furthermore, signal molecules modulate brain activity and connectivity both directly and indirectly via spinal pathways and the vagal nerve. The signal molecules derived from microbiota also modulate immune cells and can either increase or decrease the neuroinflammatory response, thereby modulating peripheral and central sensitization [3].

To summarise, genetic polymorphisms associated with immune responses and neurotransmission have a direct influence on peripheral and central sensitization via peripheral inflammation and neuroinflammation. Furthermore, psychological factors such as childhood adversity, as well as transgenerational or physical traumas, whether toxic or dietary, could modulate neuroinflammation indirectly via epigenetic modification of DNA expression. Genetic and environmental factors both influence the microbiome, which can modulate the neuroinflammatory response directly through the immune system and indirectly through epigenetics. Peripheral sensitization is triggered by neuroinflammation, which can progress to central sensitization. Chronic pain can result when central sensitization is combined with negative psychological characteristics. This heuristic multistep/multifactorial pathophysiological model is clearly in its early stages, but it is consistent with other neurological and psychiatric disorders [4].

Cognitive coping mechanisms. The combination of perceived unpleasantness and catastrophizing results in suffering, which can manifest as a variety of behaviours such as anger, fear, frustration, anxiety, depression, and functional disability. Suffering can thus be defined as an unpleasant experience that has a negative cognitive, emotional, and autonomic impact, resulting in behavioural changes and functional disability. In 188 countries, the Global Burden of Disease Study 2013 assessed "years lived with disability" for a wide range of diseases and injuries. Chronic low back pain was the leading cause of YLDs worldwide, followed by major depressive disorder, i.e. one manifestation of suffering. Chronic neck pain, migraine, osteoarthritis, other musculoskeletal disorders, and medication overuse headache are all common causes of YLDs [5].

## Conclusion

Pain is processed by three distinct but interconnected networks, each of which encodes a different pain characteristic. The lateral pathway, with the somatosensory cortex as its main hub, is primarily responsible for pain. The suffering component is involved in the medial pathway, with the rdACC and insula as main hubs, and the descending pain inhibitory pathway is

possibly related to the percentage of time that the pain is present. One can experience pain without also experiencing suffering. Chronic pain is most likely caused by an imbalance between ascending pain-inducing pathways and descending pain-inhibitory pathways. This balance concept suggests that a combination therapy of different approaches may be the best way to treat chronic pain, provided that the pathway for each treatment is known.

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## Conflict of Interest

There are no conflicts of interest by author.

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