

Commentary

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Commentary on: Dualism, Materialism, and the relationship between the brain and the mind in experiencing pain

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My paper addresses a fundamental issue in understanding pain, namely, where do we experience the suffering from an injury? A simple question, yet obtaining the answer has been complicated with important implications for pain management. The consensus is that pain is experienced in consciousness; but then how does nociceptive information from the site of an injury or inflammation get to consciousness? According to the traditional Physicalist/materialist view, consciousness is inseparable from the activities of the brain¹. In other words, the consciousness of pain results via the propagation of action potentials (APs) along neural networks, the transmission across synapses, and the biochemical reactions that follow. Given this perspective, pain can be controlled by blocking essential events in these networks and this pharmacological approach has had some success with the development of opiates. Unfortunately, like most drugs, there are unwanted side effects and overuse of opiates often leads to addiction.

In opposition to this traditional view was the proposal by the French philosopher and scientist Rene' Descartes (1596-1650) that the brain and consciousness, i.e. the mind, are functionally and spatially distinct. What I presented in my paper was evidence that dualism is correct and that the separation between the brain and consciousness is important because it offers a promising new way to treat pain. My approach was to follow the flow of nociceptive information as it courses from the site of injury throughout the nervous system and what follows is a summary of my findings^{2,3}. A crucial source of data came from imaging techniques that identified areas in the brain that are activated after an injury and these could be depicted as an integrated pain network (IPN: Figure 1).

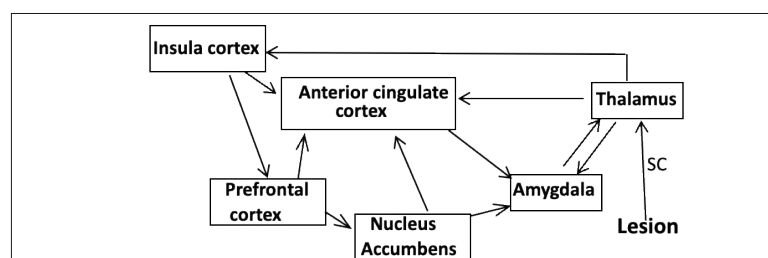


Figure 1: Basic integrated pain network (IPN) depicting the flow of nociceptive information from a lesion. Information from the site of the lesion is encoded in APs that propagate across synapses and along a tract in the spinal cord (SC) to the thalamus. After processing, the information is distributed to circuits in neuronal centers that can influence the ultimate intensity of the pain. The insula cortex and anterior cingulate cortex (ACC) focus attention on the lesion. The ACC also contains a circuit with a population of pyramidal neurons whose activity is necessary for experiencing pain. Activating circuits in the amygdala or nucleus accumbens enhances or reduces pain, respectively. Areas in the prefrontal cortex organize the information and formulate the response. Modified from².

Pain is a complex sensation because the perceived intensity of pain is subjective and several of the centers in this network contribute to modulating the intensity of the pain. Activation of neurons in the amygdala, for example, exacerbates pain, whereas activation of those in the nucleus ambiguous (NucA) reduces pain. Most significant, however, was the finding that the activation of a population of pyramidal neurons in the anterior cingulate cortex (ACC) is essential for experiencing pain². Consequently, my initial thought was that characterizing the electro-physiological and molecular events that occur in these neurons in response to an injury would provide new targets for drug development and might also provide clues as to how these events result in the consciousness of pain.

The activation of the pyramidal neurons begins when algogenic agents released at the site of an injury evoke APs that encode information about the severity of the lesion. The APs propagate to the thalamus and, after processing, the information is communicated via thalamic axons to synapses on the extensive network of pyramidal dendrites. Activation of the thalamo-pyramidal synapses in response to the injury causes the release of the excitatory neurotransmitter glutamate, thereby evoking the synchronous activation of thousands of postsynaptic dendrites⁴. Synchronicity is important for the processing of pain information. The activation of the synapses has two consequences. First, it creates Electromagnetic (EM) waves that oscillate in synchrony with the synaptic activity around the pyramidal neurons^{5,6}. Second, it induces a long-term potentiation (LTP) in the dendrites that is necessary for experiencing pain⁷. The LTP sensitizes the transmission across the synapses that is manifest as allodynia so that even a few APs evoked by a gentle touch to the injured area will elicit pain hours or days after the injury. The duration of the allodynia is determined by the events associated with the LTP, which involves the activation of NMDA receptors and the enzymes adenylate cyclase (AC) and protein kinase A (PKA), among others. Inhibiting the LTP, or blocking the activity of AC, prevents visceral⁸ and neuropathic pain⁹ in mice, but does not affect acute pain¹⁰. Thus, the LTP is responsible only for the pain that persists after the injury. PKA is important because it enters the nucleus to activate the transcription factor CREB, resulting in the synthesis of proteins that alter the phenotype of the pyramidal neurons and maintain the sensitization and generation of the EM waves¹¹. Phenotypic changes are not readily reversed and the LTP can, in theory, last indefinitely. It follows, therefore that characterizing the processes responsible for inactivating the LTP is important for understanding how pain persists beyond the point of healing and becomes chronic.

Identifying the enzymes that are necessary for experiencing pain was significant, but ultimately

disappointing because AC and PKA are found in many areas of the cortex and are therefore not suitable targets for drug development. Moreover, none of the events involved in the development of the synaptic sensitization could explain the suffering from an injury because LTP is induced in many other cortical circuits not related to pain. What this meant was that the traditional view that the electrical and biochemical activities of circuits in the brain can explain suffering was not correct and the focus then shifted to the role of the synchronized oscillating EM waves.

The photons in EM waves travel at the speed of light in the vacuum of space and contain information about their source that is encoded in their frequency, amplitude, and phase. The photons are also packets (quanta) of energy that can influence the activity of neuronal circuits as they traverse the brain. S. Pockett¹² and J McFadden¹³ had proposed that EM waves communicate with consciousness and, taken together with the findings above, this resulted in a theory that the information about pain that was originally encoded in APs is transformed at the pyramidal neurons into EM waves which then convey the information to consciousness where the pain is experienced^{2,3}.

The initial theory did not explain how the subjectivity of pain arises from the activity of the centers in the IPN, but it turns out that in addition to the EM waves from the pyramidal neurons in the ACC, pain-associated oscillating EM waves can be detected from the insula cortex, NucA, and amygdala^{14,15}. The waves from each of these centers contain information that can influence the intensity of the pain that is ultimately experienced. The intensity is determined by the amplitude of each wave and when two waves interact, the amplitude of the resulting wave is the algebraic sum their amplitudes. For example, fear occurs due to the activation of circuits in the amygdala and fear exacerbates pain. When EM waves from the amygdala interact with waves from the pyramidal neurons, the result is a wave with greater amplitude and the intensity of the pain increases. Similarly, interaction of waves from the pyramidal neurons with those from the NucA will attenuate the pain¹⁵.

The outcome from these findings was consistent with an earlier proposal¹⁴ that EM waves comprise a system that integrates information from the various centers in the ICN and that the waves that result are communicated to consciousness where the suffering occurs¹⁵. Moreover, since the waves are intermediaries between the brain and consciousness, disrupting the waves should prevent pain from being experienced and this can be accomplished using transcranial magnetic stimulation (TMS) protocols. TMS is a non-invasive procedure with no significant side-effects that introduces a transient electric current into a coil on the scalp above a target area of the cerebral cortex¹⁶. The current generates a magnetic field in the underlying cortex that can block pain by directly cancelling the

endogenous waves¹² or by inhibiting the activity of NMDA receptors, thereby blocking the LTP that is necessary for the creation of the waves¹⁷. TMS is being used to treat depression¹⁸ and has already had some success in alleviating pain¹⁹. Since there is an almost infinite number of EM waves, future studies should focus on identifying the most efficacious frequency to apply, as well as to ascertain the best location of the TMS source on the scalp. In conclusion, my findings that EM waves are carriers of information about pain to consciousness indicate that preventing the transmission of this information using TMS can be an important alternative to pharmacological approaches for managing pain. This might be especially important for managing chronic pain.

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