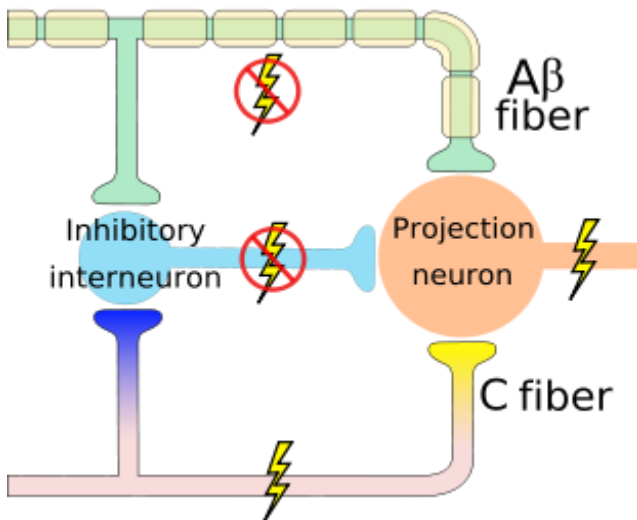


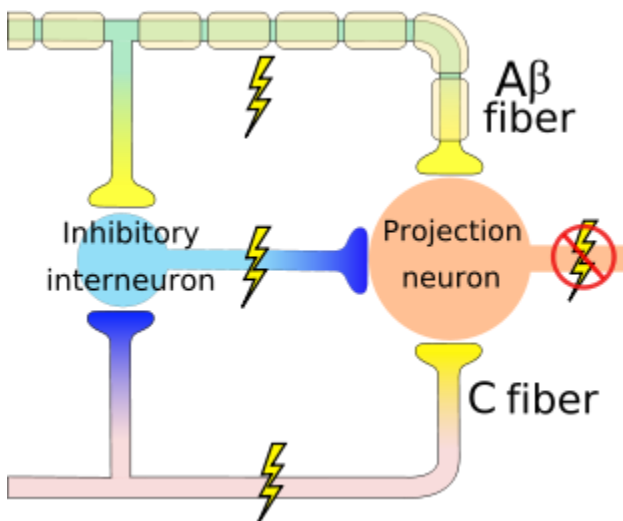
Gate Control Theory of Pain

The **gate control theory of pain**, put forward by [Ronald Melzack](#) (a Canadian psychologist) and [Patrick David Wall](#) (a British physician) in 1962,^[1] and again in 1965,^[2] is the idea that the perception of physical [pain](#) is not a direct result of activation of [nociceptors](#), but instead is modulated by interaction between different [neurons](#), both pain-transmitting and non-pain-transmitting. The theory asserts that activation of nerves that do not transmit pain signals can interfere with signals from pain fibers and inhibit an individual's perception of pain.

Physiology



The firing of the projection neuron determines pain. The inhibitory interneuron decreases the chances that the projection neuron will fire. Firing of C fibers inhibits the inhibitory interneuron (indirectly), increasing the chances that the projection neuron will fire.^[3] Inhibition is represented in blue, and excitation in yellow. A lightning bolt signifies increased neuron activation, while a crossed-out bolt signifies weakened or reduced activation.



Firing of the [Aβ](#) fibers activates the inhibitory interneuron, reducing the chances that the projection neuron will fire, even in the presence of a firing nociceptive fiber.^[3]

Gate control theory asserts that activation of nerves which do not transmit pain signals, called nonnociceptive fibers, can interfere with signals from pain fibers, thereby inhibiting pain. [Afferent](#) pain-receptive nerves, those that bring signals to the brain, comprise at least two kinds of fibers - a fast, relatively thick, [myelinated "Aδ" fiber](#) that carries messages quickly with intense pain, and a small, unmyelinated, slow ["C" fiber](#) that carries the longer-term throbbing and [chronic pain](#). Large-diameter Aβ fibers are nonnociceptive (do not transmit pain stimuli) and inhibit the effects of firing by Aδ and C fibers.

The [peripheral nervous system](#) has centers at which pain stimuli can be regulated. Some areas in the [dorsal horn](#) of the [spinal cord](#) that are involved in receiving pain stimuli from Aδ and C fibers, called [laminae](#), also receive input from Aβ fibers.^[3] The nonnociceptive fibers indirectly inhibit the effects of the pain fibers, 'closing a gate' to the transmission of their stimuli.^[3] In other parts of the laminae, pain fibers also inhibit the effects of nonnociceptive fibers, 'opening the gate'.^[3]

An inhibitory connection may exist with Aβ and C fibers, which may form a [synapse](#) on the same [projection neuron](#). The same neurons may also form synapses with an [inhibitory interneuron](#) that also synapses on the projection neuron, reducing the chance that the latter will fire and transmit pain stimuli to the [brain](#) (image on the right). The inhibitory interneuron fires spontaneously.^[3] The C fiber's synapse would inhibit the inhibitory interneuron, indirectly increasing the projection neuron's chance of firing. The Aβ fiber, on the other hand, forms an *excitatory* connection with the inhibitory interneuron, thus *decreasing* the projection neuron's chance of firing (like the C fiber, the Aβ fiber also has an excitatory connection on the projection neuron itself). Thus, depending on the relative rates of firing of C and Aβ fibers, the firing of the nonnociceptive fiber may inhibit the firing of the projection neuron and the transmission of pain stimuli.^[3]

Gate control theory thus explains how stimulus that activates only nonnociceptive nerves can inhibit pain. The pain seems to be lessened when the area is rubbed because activation of nonnociceptive fibers inhibits the firing of nociceptive ones in the laminae.^[3] In [transcutaneous electrical stimulation](#) (TENS), nonnociceptive fibers are selectively stimulated with [electrodes](#) in order to produce this effect and thereby lessen pain.^[3]

One area of the brain involved in reduction of pain sensation is the [periaqueductal gray matter](#) that surrounds the [third ventricle](#) and the [cerebral aqueduct](#) of the [ventricular system](#). Stimulation of this area produces [analgesia](#) (but not total numbing) by activating descending pathways that directly and indirectly inhibit nociceptors in the laminae of the spinal cord.^[3] It also activates [opioid](#) receptor-containing parts of the spinal cord.

Afferent pathways interfere with each other constructively, so that the brain can control the degree of pain that is perceived, based on which pain stimuli are to be ignored to pursue potential gains. The brain determines which stimuli are profitable to ignore over time. Thus, the brain controls the perception of pain quite directly, and can be "trained" to turn off forms of pain that are not "useful". This understanding led Melzack to assert that *pain is in the brain*.

Comparison to other theories

Prior theories of the [neurochemistry](#) of pain had not taken the brain into account - pain was thought to be simply a direct response to a stimulus, a one-way "alarm system" like that proposed by [René Descartes](#). This did not, for instance, explain why a carpenter can hit his thumb and not feel much pain, while a novice is in agony, nor did it explain [phantom limb pain](#), when the signal is in fact impossible to receive, since the wiring for it is gone.^[citation needed]

