Transcutaneous Electrical Nerve Stimulation: Mechanisms of Action

What is the neurophysiological trigger for analgesia?
Electrical stimulation of sensory nerve fibers (Aβ), primarily the deep tissue afferents, triggers the biochemical cascade resulting in analgesia.

What neural structures are involved?
Pain signals are blocked by inhibition of nociceptive neurons in the spinal cord dorsal horn.¹ This process is facilitated by descending signals from the periaqueductal gray (PAG) and the rostroventral medial medulla (RVM).¹ There is also evidence that pain signals are interrupted in the peripheral nervous system.¹

What is the key molecular mechanism?
Stimulation causes release of endogenous opioids that inhibit pain through activation of δ-opioid receptors.² These receptors are located throughout the nervous system, including the dorsal horn of the spinal cord.¹ Opioid receptors are G-protein coupled receptors whose activation decreases neuronal activity, such as through ion channel regulation.³

Do traditional opiates such as morphine act through δ-opioid receptors?
No, morphine and similar opiates operate through μ-opioid receptors. Although activation of both μ- and δ-opioid receptors induces analgesia, the two receptor types do not have the same neuroanatomical distribution, biological effects, or abuse potential.³

Are other neurotransmitters involved in analgesia?
Nerve stimulation increases the extracellular concentration of the inhibitory neurotransmitter GABA and decreases the concentration of the excitatory neurotransmitters glutamate and aspartate in the spinal cord dorsal horn.¹

References