



PERSISTENT PAIN: Important mechanisms and functions in nociceptor cell bodies

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Chronic pain and some of its common consequences, such as opioid abuse, are enormous global problems. Improved treatments that minimize abuse potential will require better understanding of the mechanisms that persistently drive pain. While the worst complaints are often about ongoing, apparently spontaneous pain rather than painful hypersensitivity to external stimuli, far more is known about mechanisms of hypersensitivity than about mechanisms of ongoing pain. Converging evidence indicates that electrophysiological and molecular mechanisms critical for continuously driving ongoing pain in rodents and humans are especially prominent in cell bodies of the primary sensory neurons (nociceptors) that are specialized for detecting harmful stimuli.

I will describe electrophysiological and cellular mechanisms found to persistently drive spontaneous electrical activity in the cell bodies of rat, mouse, and human nociceptors in peripheral and central neuropathic conditions. These involve unusual mechanisms for generating spontaneous depolarizations in membranes that transition from electrical silence to ongoing activity, as well as enhanced sensitivity to cytokines and major roles for cAMP, ERK, and calcium signaling. I will end the talk by considering the functional significance and possible therapeutic implications of hyperactivity induced in nociceptor cell bodies by diverse signals of injury, informed by an evolutionary perspective comparing nociceptor plasticity in insects, snails, squid, rats, mice, and humans.



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Time: 12:00h



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