Glutamatereceptors in chronic pain-induced neuroplasticity

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Chronic pain represents a major challenge to clinical practice and basic science. Excitatory neurotransmission in somatosensory nociceptive pathways is predominantly mediated by glutamatergic synapses. A key feature of these synapses is their ability to adapt synaptic strength in an activity-dependent manner. Such disease-induced synaptic plasticity is paramount to alterations in synaptic function and structure. Recent work has recognized that synaptic plasticity at both excitatory and inhibitory synapses can function as a prime mechanism underlying pathological pain. In this presentation, cellular and molecular mechanisms underlying synaptic plasticity in nociceptive pathways will be reviewed and discussed. New insights derived from these advances are expected to expedite development of novel interventional approaches for treatment of pathological pain.