Dissertation Abstract

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Pain is a critical survival mechanism that signals potential or actual damage, but can become pathological when it persists beyond the injury. Chronic pain is a major health issue that affects 10-20% of the adult population, and is found disproportionately in women. There are numerous, interacting mechanisms underlying this phenomenon. This study used female rats to investigate the impact of estrogens on sensory signaling by substance P, a neuropeptide that contributes to the development of chronic pain, and the PI3K-Akt-mTOR pathway, an intracellular mechanism of nervous system plasticity.