arborization, outgrowth and synaptic formation. The signaling pathway of the neurotrophic effect of tianeptine on the structural plasticity was studied by evaluation of the possible kinases phosphorylation after treatment. Also specific kinase antagonists was pre-treated to confirm the signaling pathway.
Results: We found that tianeptine (10 μg/ml) enhanced the dendritic outgrowth, arborization and synaptic formation, as effective as nerve growth factor β. At the same time, tianeptine up regulated the expression of cytoskeletal protein MAP 2, Neuro filament L and synaptophysin. Tianeptine at 10μg/ml dose significantly phosphorylated CaMK II, and pretreating with KN93 (CaMK II antagonist) decreased the neutrophic effect of tianeptine.
Conclusions: Tianeptine enhanced hippocampal neurite outgrowth and synaptic formation by activation of CaMK II phosphorylation.

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Ovariectomy affects activation of ERK and pain hypersensitivity in acid injection-induced widespread muscle pain

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Background: Musculoskeletal pain is a major clinical disorder. Fibromyalgia syndrome (FMS) is a type of chronic widespread muscle pain (CWP) and is characterized with generalized pain, diffuse tender points, morning stiffness, disturbed sleep, and day-time fatigue. In particular, there is a higher ratio in female population than in male to have FMS. Several pathophysiological hypotheses have been proposed underlying CWP, however, few studies had aim to elucidate the gender effect on the development of CWP. Meanwhile, though ERK MAPK plays a pivot role in signaling cascade of pain processes, how activated ERK (p-ERK) is involved in CWP has never been discussed.
Methods: In a repeated acid injection-induced muscle pain (AIMP) model, nociceptive thresholds in response to von Frey stimulation and radiant heat were measured. The adult female rats were allocated into 2 groups: the ovariectomized female (OVX) and the control female (Sham surgery). Three weeks after ovariectomy or sham surgery, acid solution was repeatedly injected to left gastrocnemius to produce pain. Behavioral changes and pERK amount in the spinal dorsal horn was measured at different time points. Besides, intrathecal injection of U0126, an ERK inhibitor, was conducted to evaluate the role of pERK in AIMP.