Functional and structural plasticity of central synapses in pain pathways is responsible for the allodynia, hyperalgesia and pain memory caused by different insults like inflammation, neuropathy or trauma. Unfortunately molecular mechanism underlying plasticity at different levels of pain transmission are not yet clearly understood. mTOR is serine-threonine kinase playing a significant role in Long Term Potentiation (LTP) and in local protein translation in Hippocampus and Rac1 (Rho-GTPase) is the key regulator of actin cytoskeleton. Therefore we addressed the potential role of mTOR and Rac1 in activity-dependent plasticity in the spinal cord in the context of chronic pain. Using the western blot analysis, we examined the temporal expression of these proteins in L4+L5 spinal segments using inflammatory pain model. In case of Rac1 we found that the expression is unchanged. In case of mTOR we showed that the change in the expression is not significant but there is a clear trend of increase. To confirm these findings more expesiments are needed to be added. If this is confirmed there is a potential role of mTOR in pain processing.