Modulation of nociceptive synaptic transmission in the spinal cord dorsal horn is a significant mechanism in the development and maintenance of different pathological pain states. Accumulating evidence indicates that the TRPV1 (transient receptor potential vanilloid 1) receptor and chemokine CCL2 (C-C motif ligand 2) may play a critical role in this process. The aim of this diploma thesis was to investigate the CCL2 induced modulation of nociceptive synaptic transmission in the dorsal horn of spinal cord and the role of the TRPV1 receptors. To investigate this aim patch-clamp recordings of spontaneous and miniature excitatory postsynaptic currents (sEPSC, mEPSC) from superficial dorsal horn neurons in acute rat lumbar spinal cord slices were used. After acute application of CCL2 on the slice preparation from naïve animals, a frequency increase of both sEPSC and mEPSC was present. This CCL2 induced increase in both sEPSC and mEPSC frequency was prevented by the TRPV1 receptor antagonist SB366791 application. No changes were observed in the amplitudes of sEPSC or mEPSC after application of the CCL2, SB366791, or co-application of CCL2 and SB366791. This suggests that the observed changes were mediated predominantly by presynaptic mechanisms. The preliminary results indicate that after chronic constriction injury (CCI) CCL2 modulates the synaptic transmission in dorsal horn by similar mechanism. The CCL2 induced increase of mEPSC frequency recorded from slices after the CCI induced neuropathy was also prevented by the SB366791 pretreatment. These data suggest that activation of presynaptic TRPV1 receptors plays an important role in the modulation of nociceptive signalling induced by the CCL2 application. Cooperation between the CCL2 activated CCR2 receptors and TRPV1 receptors on the central branches of primary afferent fibers could play important role especially during different pathological pain states and need to be further investigated.