INVOLVEMENT OF THE ENDOCANNABINOID SYSTEM IN CHRONIC PAIN

Joint pain is a common clinical problem for which both inflammatory and degenerative joint diseases are major causes. We have investigated the role of CB1 and CB2 cannabinoid receptors in the behavioral, histological, and neurochemical alterations associated with joint pain. The murine model of monosodium iodoacetate (MIA) was used to induce joint pain in knockout mice for CB1 (CB1KO) and CB2 cannabinoid receptors (CB2KO) and transgenic mice overexpressing CB2 receptors (CB2xP). In addition, we evaluated the changes induced by MIA in gene expression of CB1 and CB2 cannabinoid receptors and µ-, δ- and κ-opioid receptors in the lumbar spinal cord of these mice. Wild-type mice, as well as CB1KO, CB2KO, and CB2xP mice, developed mechanical allodynia in the ipsilateral paw after MIA intra-articular injection. CB1KO and CB2KO demonstrated similar levels of mechanical allodynia of that observed in wild-type mice in the ipsilateral paw, whereas allodynia was significantly attenuated in CB2xP. Interestingly, CB2KO displayed a contralateral mirror image of pain developing mechanical allodynia also in the contralateral paw. All mouse lines developed similar histological changes after MIA intra-articular injection. Nevertheless, MIA intra-articular injection produced specific changes in the expression of cannabinoid and opioid receptor genes in lumbar spinal cord sections that were further modulated by the genetic alteration of the cannabinoid receptor system. These results revealed that CB2 receptor plays a predominant role in the control of joint pain man.

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