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The abnormal cannabidiol analogue O-1602 reduces nociception in a rat model of acute arthritis via the putative cannabinoid receptor GPR55.

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Abstract

Cannabinoids classically act via CB_1 and CB_2 receptors to modulate nociception; however, recent findings suggest that some cannabinoids bind to atypical receptors. One such receptor is GPR55 which is activated by the abnormal cannabidiol analogue O-1602. This study investigated whether the synthetic GPR55 agonist O-1602 can alter joint nociception in a rat model of acute joint inflammation. Acute (24 h) inflammatory joint pain was induced in male Wistar rats by intra-articular injection of 2% kaolin and 2% carrageenan. Single unit extracellular recordings were made from arthritic joint afferents in response to mechanical rotation of the knee. Peripheral administration of O-1602 significantly reduced movement-evoked firing of nociceptive C fibres and this effect was blocked by the GPR55 receptor antagonist O-1918. Co-administration of the CB_1 and CB_2 antagonists (AM281 and AM630 respectively) had no effect on O-1602 responses. This study clearly shows that atypical cannabinoid receptors are involved in joint nociception and these novel targets may be advantageous for the treatment of inflammatory pain.