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on mechanical allodynia of carrageenan in f ammation

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Introduction: Descending serotonergic projections may facilitate or inhibit nociceptive processing in the spinal cord depending on several factors. Unlike other pain states, spinal 5-hydroxytryptamine 3 receptors (5-HT3R) were shown to play a limited role in nociceptive transmission of carrageenan-induced in ammatory pain. Instead, a facilitatory role of 5-HT1AR and 5-HT1BR in spinal nociceptive processing was observed during early-phase of carrageenan model. Although, the maximum release of 5-HT in spinal cord reaches the maximum 2-3 hours a er carrageenan injection (early-phase), its release returns to baseline 8 hours.

Aim: To identify the role of Spinal 5-HT1A receptor in directing serotonergic modulation toward inhibition on mechanical allodynia of carrageenan in ammation.

Methods: E ects of intrathecal (i.t.) nonspeci c 5-HTR agonist, subtype agonist or antagonists (5-HT1AR, 5-HT1BR, 5-HT3R), and 5,7-dihydroxytryptamine (5,7-DHT, a serotonergic neurotoxin) on mechanical allodynia were tested for early- and late-phase allodynia.

Results: Lesioning spinal serotonergic projections with 5,7-DHT induced a significant increase in the intensity of mechanical allodynia at both early and late-phase. is increase was attenuated by i.t. 5-HT. Also, i.t. 5-HT itself produced a significant antiallodynic e ect in late-phase, but not in early-phase. Similarly, i.t. 5-HT1AR agonist (8-OH-DPAT) attenuated the intensity of late-phase allodynia in a dose-dependent manner which was antagonized by 5-HT1AR antagonist (WAY-100635), but produced no e ect on the early-phase allodynia. However, other agonists or antagonists of 5-HT1BR and 5-HT3R did not produce any anti or pro-allodynic e ects.

Conclusion: Descending serotonergic modulation plays a vital role in inhibition of nociceptive processing during late-phase allodynia, which involves spinal 5-HT1A, but not 5-HT1B or 5-HT3 receptors in carrageenan-induced inflammation. However, the de ned role of 5-HT1A and serotonergic inhibition during early-phase remains undetermined.

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In the present study, we hypothesized that listening to music would modulate the e ects of allodynia, hyperalgesia and fatigue in patients with bromyalgia (FM). Due to its emotional e ect, we expected that listening to music would have a greater moderating e ect on the perception of pain and fatigue than listening to non-musical sounds. To investigate this hypothesis, we carried out a study in which people with FM were given a listening device for four weeks enabling them to listen to either music or environmental sounds when they experienced pain, in either an active (while carrying out a physical activity) or passive