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Commentary

Capsaicin's Effects on Temporo-mandibular Joint Arthritis in animal models

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Introduction

Female Lewis rats were given a unilateral injection of a heat-killed Mycobacterium butyricum suspension in paraffin oil into the TMJ, resulting in TMJ arthritis. The identical technique was used to administer paraffin oil to the control rats. Capsaicin or denervation of the mandibular branch of the trigeminal nerve were used to treat arthritic and control rats. Tissues were obtained for neuropeptide extraction and radioimmunoassays as well as reverse-phase high-performance liquid chromatography were used to analyse the results. The trigeminal ganglia had higher levels of substance P (SP), calcitonin gene-related peptide (CGRP), and neuropeptide Y (NPY) like immunoreactivity (LI) than the TMJs in all groups. Capsaicin reduced the levels of SP-LI in the trigeminal ganglia and TMJ in control rats, but not CGRP-LI or NPY-LI.

Capsaicin pretreatment significantly reduced the SP-LI and CGRP-LI in the trigeminal ganglia and TMJ in arthritic rats, but not the NPY-LI. Unilateral denervation of the trigeminal ganglia dramatically reduced SP-LI in normal rats and SP-LI and CGRP-LI in arthritic rats. When compared to the arthritic control rats and the contralateral side, NPY-LI, SP-LI, and CGRP-LI were considerably lower on the denervated side of the arthritic TMJ. The neuropeptide concentration in the trigeminal ganglia and the TMJ was reduced in this rat model after pretreatment with capsaicin and surgical denervation. After inducing arthritis in the rat, the data clearly show a close relationship between increased neuropeptide release from sensory and sympathetic neurones.

There are several diseases that cause TMJ pain, which frequently affects speech, chewing, and other essential everyday activities due to pain-related disability. Despite the fact that these disorders can be assessed in terms of lost productivity and human suffering, little is known about the underlying pathologic mechanism of TMD-related pain. Experimental models that allow researchers to investigate the mechanisms behind these inflammatory and pain diseases have a lot of clinical utility. Intra-articular injections of harmful chemicals are used to create rat models of TMJ inflammation. In this case, zymosan, a sugar derived from yeast cell walls, causes a severe and erosive inflammation.

This paradigm has primarily been used to examine knee inflammatory illness; however, no study has employed zymosan to produce TMJ inflammatory disease to our knowledge. Gas synthase (NOS) synthesises NO from L-arginine, and three isoforms of NOS have been identified. The epithelium (NOS1) and vegetative cell (NOS3) isoforms are both natural, but NOS2 is an inducible isoform (iNOS). The immunohistochemical localization of inducible NOS within the secretion tissue of human TMJs has been established previously, and NO's antinociceptive action within the caudal a part of the spinal cranial nerve nucleus during chronic carrageenan-induced inflammatory disease in rats TMJ has also been established. Previously, associate associatetinociceptive action of NO within the caudal a part of the spinal cranial nerve nucleus during chronic carrageenan-induced inflammatory disease in rats has been incontestable, as has the immunohistochemical localization of inducible NOS within the secretion tissue of human TMJs.

Conclusion

We also aimed to see the time course of tube-shaped structure and cellular events that occur secondary to zymosan-induced TMJ inflammatory disease in rats, as well as the reputed involvement of NO and neutrophils, because each of the mediators involved in pain mechanisms throughout

inflammatory arthropathies and also the precise role of NO in pain development are still not completely outlined.

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Conflict of Interest

We have no conflict of interests to disclose and the manuscript has been read and approved by all named authors.