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Skin-Related Neurogenic Inflammation and Chronic Pain

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Abstract

It is risky to treat any pain with oral medications; acupuncture or topical medications should be used instead. The best and safest way to manage pain, which is felt in the skin, is using topical medications. The pain chemokine cycle makes chronic pain worse in the skin. Pain felt in the skin causes neurogenic inflammation, which spreads inflammation throughout the body. Pain and inflammation are reduced by blocking the pain receptors on skin sensory neurons.

Keywords: Chronic pain • Neurogenic inflammation • Pain • Pain chemokine cycle

Introduction

Even pain from fractured bones, cancer, kidney stones and other interior sources is mostly felt in the skin. This is due to communication between internal pain-transmitting neurons and pain-sensing skin neurons that reach the brainstem. Numerous interconnected processes in the skin and other places are involved in chronic pain. Transient Receptor Potential (TRP) cation channels are among the many receptors on skin sensory neurons that sense pain and prolong it. On skin sensory neurons, a variety of TRP channels are present. On certain neurons, populations of particular TRP channels can be detected. Chronic pain is prolonged and made worse by the pain chemokine cycle in the skin.

Description

Chemokines are released when a sensory neuron in the skin is injured or activated, drawing neutrophils and macrophages into the skin. Leukotrienes released by neutrophils cause pain by activating TRP channels on epidermal sensory neurons. Leukotrienes have lengthy half-lives, which may enable them to cause pain over an extended period of time in a large number of nearby neurons. Cyclooxygenase 2 (COX2), an enzyme found in macrophages, secretes prostaglandins, which when they bind to prostaglandin receptors, produce pain. By transactivating TRP channels, prostaglandins can intensify and prolong pain. Skin-resident T cells in the skin release IL-17 in response to chemokines. In response to IL-17, sensory neurons produce more chemokines. The majority of chronic pain is caused by a self-maintaining pain chemokine cycle that transforms the skin into an organ of pain 1[].

Male and female skin clearly differs from one another in terms of thickness, hormonal reaction and food intake. The identified disparities in acute pain perception and reactions between men and women may be partially explained by these differences. But there hasn't been enough research done on how skin-related characteristics affect chronic pain differently in men and women.

TRP channel activation results in the release of inflammatory proteins from sensory skin neurons, including substance P, high mobility group box 1 protein, neurokinin A and calcitonin gene-related peptide (CGRP). The root cause of neurogenic inflammation is this. In inflammatory illnesses like arthritis and asthma, the skin is crucial [2].

High quantities of CGRP, which is secreted by skin sensory neurons, are discovered in arthritis sufferers' synovial fluids. It is evident that CGRP has a role in the inflammatory response to arthritis. Inhibiting skin TRP channels with monoterpenoids may lower CGRP levels in synovial fluids and have anti-inflammatory effects. In addition to inducing pain, CGRP also plays a role in the pain that arthritis sufferers feel as well as chronic pain, which is brought on by the pain chemokine cycle.

A G protein coupled receptor known as the calcitonin receptor-like receptor is where CGRP binds (CLR,14). The interaction of the receptor with RAMP (14), which contributes to the pain and inflammation brought on by CGRP, can dramatically increase CLR activity.

Activated macrophages, such the monocytes and macrophages drawn to the skin by chemokines during the pain chemokine cycle, produce HMGB1. Toll-like receptors (TLR2 and 4), Receptor for Advanced Glycation End Products (RAGE) and HMGB1 interact with one another to cause the release of inflammatory cytokines. Adipokine/cytokine tumour necrosis factor is implicated in the promotion of inflammation in rheumatoid arthritis. TNF- is released more often from macrophages as a result of HMGB1. Tumor necrosis factor can spread to distant places and cause inflammation there, as well as in synovial tissues [3, 4].

Skin sensory neurons emit neurokinin A, which intensifies pain and inflammation. There are many neurokinin receptors, including the G protein-coupled NK1R, NK2R and NK3R. Another kinin that is secreted by skin sensory neurons is substance P. To cause pain and inflammation, substance P also interacts with the NK1R, NK2R and NK3R receptors. It is evident that substances produced from the skin, such as substance Pare and neurokinin A, travel to distant locations and have a role in inflammatory diseases such as arthritis [5].

Conclusion

The mechanism of neurogenic inflammation links chronic pain with chronic inflammation. Inflammatory proteins are released from skin sensory neurons as a result of an early inflammation, which might make the condition worse. Most likely, persistent inflammation causes both chronic pain and more inflammation. The release of inflammatory proteins from skin sensory neurons in response to an initial discomfort, on the other hand, might result in the induction of inflammation in the body. Inflammatory proteins that amplify pain in other sensory neurons and may result in chronic inflammation are released over time as a result of chronic pain.

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Conflict of Interest

No potential conflict of interest was reported by the authors.

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