Experimental pain sensitivity differs as a function of clinical pain severity in symptomatic knee osteoarthritis.

Submitted by admin on Fri, 03/14/2014 - 9:15am

Title
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Publication Type
Journal Article

Year of Publication
2013

Authors

Journal
Osteoarthritis Cartilage

Volume
21

Issue
9

Pagination
1243-52

Date Published
2013 Sep

ISSN
1522-9653

Keywords
Acute Pain, Arthralgia, Body Mass Index, Disability Evaluation, Educational Status, Female, Hot Temperature, Humans, Male, Middle Aged, Osteoarthritis, Knee, Pain Measurement, Pain Threshold, Physical Stimulation, Pressure, Severity of Illness Index

Abstract
OBJECTIVE: Pain in knee osteoarthritis (OA) has historically been attributed to peripheral pathophysiology; however, the poor correspondence between objective measures of disease severity and clinical symptoms suggests that non-local factors, such as altered central processing of painful stimuli, also contribute to clinical pain in knee OA. Consistent with this notion, recent evidence demonstrates that patients with knee OA exhibit increased sensitivity to painful stimuli at body sites unaffected by clinical pain.

DESIGN: In order to further investigate the contribution of altered pain processing to knee OA pain, the current study tested the hypothesis that symptomatic knee OA is associated with enhanced sensitivity to experimental pain stimuli at the knee and at remote body sites unaffected by clinical pain. We further anticipated that pain sensitivity would differ as a function of the OA symptom severity. Older adults with and without symptomatic knee OA completed a series of experimental pain assessments. A median split of the Western Ontario and McMaster Universities Index of Osteoarthritis (WOMAC) was used to stratify participants into low vs high OA symptom severity.

RESULTS: Compared to controls and the low symptom group, individuals in the high symptom
group were more sensitive to suprathreshold heat stimuli, blunt pressure, punctuate mechanical, and cold stimuli. Individuals in the low symptomatic OA group subgroup exhibited experimental pain responses similar to the pain-free group on most measures. No group differences in endogenous pain inhibition emerged.

**CONCLUSIONS:** These findings suggest that altered central processing of pain is particularly characteristic of individuals with moderate to severe symptomatic knee OA.