Pain Med. 2014 Aug;15(8):1346-58. doi: 10.1111/pme.12460. Epub 2014 Jul 4.

Changes in clinical pain in fibromyalgia patients correlate with changes in brain activation in the cingulate cortex in a response inhibition task.

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Abstract

OBJECTIVE:

The primary symptom of fibromyalgia is chronic, widespread pain; however, patients report additional symptoms including decreased concentration and memory. Performance-based deficits are seen mainly in tests of working memory and executive functioning. It has been hypothesized that pain interferes with cognitive performance; however, the neural correlates of this interference are still a matter of debate. In a previous, cross-sectional study, we reported that fibromyalgia patients (as compared with healthy controls) showed a decreased blood oxygen level dependent (BOLD) response related to response inhibition (in a simple Go/No-Go task) in the anterior/mid cingulate cortex, supplementary motor area, and right premotor cortex.

METHODS:

Here in this longitudinal study, neural activation elicited by response inhibition was assessed again in the same cohort of fibromyalgia patients and healthy controls using the same Go/No-Go paradigm.

RESULTS:

A decrease in percentage of body pain distribution was associated with an increase in BOLD signal in the anterior/mid cingulate cortex and the supplementary motor area, regions that have previously been shown to be "hyporeactive" in this cohort.

CONCLUSIONS:

Our results suggest that the clinical distribution of pain is associated with the BOLD response elicited by a cognitive task. The cingulate cortex and the supplementary motor area are critically involved in both the pain system as well as the response inhibition network. We hypothesize that increases in the spatial distribution of pain might engage greater neural resources, thereby reducing their availability for other networks. Our data also point to the potential for, at least partial, reversibility of these changes.