

Resting brain activity associated with spontaneous fibromyalgia pain

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A recent study from researchers at Massachusetts General Hospital and University of Michigan provides the first direct evidence of linkage between elevated intrinsic (resting-state) brain connectivity and spontaneous pain intensity in patients with fibromyalgia (FM). This research shows an interaction of multiple brain networks, offering greater understanding of how pain arises. Details of the study appear online and in the August issue of *Arthritis & Rheumatism*.

Chronic pain syndromes such as FM can cause widespread pain that varies in intensity and fluctuates over time. In addition to pain, FM patients may experience other symptoms such as fatigue, sleep disturbances, memory problems, and temperature sensitivity. The National Institute of [Arthritis](#) and Musculoskeletal and Skin Diseases estimates that FM affects 5 million American 18 years of age or older, occurring more often in women (80%-90%).

In the current study, Vitaly Napadow, Ph.D. and colleagues enrolled 36 female subjects —18 FM patients and 18 healthy control subjects with a mean age of 38.9 and 36.1 years of age, respectively. FM study subjects had a disease-duration of at least 1 year, self-reported pain for more than 50% of each day, and were willing to limit introduction of new medications or treatment strategies to control FM symptoms.

As a part of the study, 6 minutes of resting-state functional magnetic resonance imaging (fMRI) data from study subjects were collected. Data were analyzed using dual-regression independent components

analysis—a data-driven approach for the identification of independent brain networks. Intrinsic connectivity was evaluated in multiple brain networks: the default mode network (DMN), the executive attention network (EAN), and the medial visual network (MVN), with the MVN serving as a negative control.

Prior to undergoing the MRI scan, participants were asked to rate the intensity of their FM pain on a scale of 0–10, where 0 is equivalent to "no pain present" and 10 is equivalent to "the worst pain they could imagine." The pain scores ranged widely, from 0 to 8.1.

"Our results clearly show that individuals with FM have greater connectivity between multiple [brain networks](#) and the insular cortex, which is a brain region previously linked with evoked pain processing and hyperexcitability in FM," said Dr. Napadow. The research team found that patients with FM had greater intrinsic connectivity within the right EAN, and between the DMN and the insular cortex—a brain region linked to evoked pain processing. "In patients with FM, our findings strongly implicate the insular cortex as being a key node in the elevated intrinsic connectivity," added Dr. Napadow. "Patients demonstrated greater DMN connectivity to the left anterior, middle, and posterior insula." In the right EAN, FM patients demonstrated greater intra-network connectivity within the right intraparietal sulcus (iPS). Researchers found no differences between the FM and healthy control groups for the left EAN or the MVN.

The current findings provide better understanding of the underlying brain mechanisms of clinical pain in FM and may potentially lead to markers of disease progression. Broader implications for explaining how subjective experiences such as pain arise from a complex interplay among multiple [brain](#) networks can also be derived from this study. "Our approach represents a novel step forward in finding the neural correlates of spontaneous clinical pain," concluded Dr. Napadow. "However, our

results were derived strictly from patients with FM and may not be generalized to other chronic pain states, an area we are currently evaluating for further research."

More information: "Intrinsic Brain Connectivity in Fibromyalgia Is Associated With Chronic Pain Intensity." Vitaly Napadow, Lauren LaCount, Kyungmo Park, Suzie As-Sanie, Daniel J. Clauw, and Richard E. Harris. *Arthritis & Rheumatism*; Published Online: April 6, 2010 ([DOI: 10.1002/art.27497](https://doi.org/10.1002/art.27497)); Print Issue Date: August 2010.

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