Scans Show Altered Brain Connectivity in Chronic Back Pain

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February 28, 2012 (Palm Springs, California) — Patients with chronic low back pain (CLBP) have altered brain connectivity on functional magnetic resonance imaging compared with healthy controls, and experience temporary changes in this pattern when their pain is exacerbated, a new study shows.

"What we are seeing is there is at least some type of plasticity in the system," explained lead author Marco Loggia, PhD, from Harvard Medical School in Boston, Massachusetts, in an interview with Medscape Medical News. "The system is somehow being disrupted by the presence of pain."

The findings "bring us closer to the development of an objective biomarker — a 'brain reading' of pain, to tell us how much pain a person is feeling," he said.

The new report was presented here at the American Academy of Pain Medicine (AAPM) 28th Annual Meeting.

Default Mode Network

The study, which was selected as one of the meeting's top 6 posters, used arterial spin labeling magnetic resonance imaging (MRI) to measure neural activity and cerebral blood flow to specific regions of the brain.

The imaging focused on a brain area called the default mode network (DMN) that includes the medial prefrontal cortex (MPFC), posterior cingulate, lateral temporal cortices, hippocampal formation, and inferior parietal lobule.

"As DMN activity has been shown to be altered in chronic pain, we hypothesized that functional connectivity within the DMN would be altered," the authors wrote.

Scans were compared from 16 subjects with discogenic low back and radicular pain (average pain, 4.8 out of 10; and average duration, 6.2 years), and 16 age-matched healthy controls.

Imaging was done both in a baseline resting state as well as after a series of physical maneuvers that were designed to be painless for the control subjects but to exacerbate chronic low back pain in the patients.

The imaging revealed that compared with controls, patients with low back pain had stronger baseline connectivity between the MPFC and DMN which weakened with pain exacerbation. The degree of connectivity between the DMN and MPFC correlated negatively with the degree of pain reported by patients.

In contrast, connectivity between the DMN and another brain region — the insula — correlated positively with pain, and as pain increased with the maneuvers, DMN-insula connectivity increased.

"In this study we show, for the first time that clinical pain severity in CLBP patients appears to be encoded in brain connectivity patterns," the authors write.

Compensatory Mechanism

"We cannot make too many inferences based on a single study but one possibility is that the increased connectivity
between the medial prefrontal cortex and DMN that we see at baseline might be some compensatory mechanism, as if maybe the patients raise their baseline connectivity in order to somehow brace for the next pain episode which, as our data suggest, will disrupt the connectivity," said Dr. Loggia.

"We don't really understand exactly what is behind all this, any interpretation is pure speculation at this point, but what we see is that the pain exacerbation induces a temporary disruption in the connectivity pattern."

While the findings suggest that the brain can compensate and adapt to pain, it is not clear whether overall brain function might be compromised in some way.

"Increased DMN-MPFC connectivity suggests increased prefrontal contributions to cognitive processing. The MPFC is a key area processing pain and mood," write the authors. "In some patients with chronic pain you can often see psychiatric comorbidities, and you can also see some cognitive impairment," said Dr. Loggia.

Similarly, altered brain connectivity has recently been reported by one of Dr. Loggia’s coauthors in patients with fibromyalgia, raising "the intriguing possibility that this is a general feature of chronic pain, which would seem to be generalizable across conditions."

The fact that the brain's response to pain can be objectively identified and quantified on imaging could lead to improved monitoring of treatments, he explained.

"My hope would be that this type of application would be useful both in the clinic and in clinical trials," he said. "Right now we're basing our judgment about the effectiveness of a treatment on self-report, but these verbal ratings are far from perfect and cannot be obtained from everybody such as preverbal children or people with severe dementia."

**Signature for Pain Severity**

Reached for comment on the study, Catherine Bushnell, PhD, president of the Canadian Pain Society and professor of anesthesia, dentistry, and neurology at McGill University, Montreal, Quebec, said, "This exciting study adds to the growing literature indicating that chronic pain alters the brain. Showing changes in connectivity between brain regions important for mood and cognitive function could help explain why pain patients frequently develop anxiety disorders and have problems with memory and decision making."

She said studies of brain function and structure in a wide variety of chronic pain conditions, including fibromyalgia, irritable bowel syndrome, arthritis, and neuropathic pain, are now showing changes in the brain "suggesting that chronic pain is more than just a symptom. Because of these types of studies, many doctors are beginning to think of chronic pain as a disease in itself."

Dr. Loggia’s study is particularly important because "it reveals a signature for the severity of chronic pain," she added, something "which could be extremely useful for evaluating patients with limited ability to report their pain verbally."

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