

NEUROSCIENCE NEWS

Studies of interest to practicing psychiatrists

Does chronic pain shrink the brain?

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Chronic pain—particularly lower back pain—is frustrating to both patient and clinician. Because most cases lack an obvious physical explanation, the doctor may wonder if the patient is faking or exaggerating—that the pain is “in the patient’s head.” Studies suggest this cerebral component may exist—but not in ways you might expect.

HOW THE BRAIN PROCESSES PAIN

According to traditional belief, the brain passively receives noxious signals from injured tissue (nociceptive) or damaged nerve (neuropathic). Extensive—some would say excessive—tests are often conducted in search of a bone or muscle injury that might explain the pain.

Functional imaging studies across 15 years have shown activity in various brain regions when subjects feel pain. In addition to the somatosensory cortex, pain also activates brain areas involved with mood, attention, and anxiety. More important, the brain does not passively receive signals from the periphery but can inhibit ascending signals with endogenous opioids, such as endorphins and enkephalins.

Apkarian et al¹ found that CNS changes may

explain persistence of pain and lack of peripheral findings. The researchers compared gray matter density in 26 patients with chronic back pain and 26 pain-free controls. Patients in the pain group met International Association for the Study of Pain criteria for chronic back pain and had unremitting pain for >1 year, primarily in the lumbosacral region.

Structural brain MRIs showed patients with chronic back pain had 5% to 11% less cortical gray matter volume than did pain-free controls (*Figures 1 and 2, page 98*). The authors noted that it would take an average pain-free person 10 to 20 years to lose this much gray matter as a function of aging. Further computer analysis of the images by brain region showed reduced bilateral dorso-lateral prefrontal cortex density. Together, these results show significant brain atrophy associated with chronic back pain.

These findings document for the first time that a heterogeneous group of patients with unre-

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lenting back pain had changes in the cerebral cortex—particularly the prefrontal cortex. These changes may help perpetuate chronic pain because the dorsolateral prefrontal cortex is believed to control pain perception from the top down.² In other words, diminished cortical control reduces the “brakes” on pain signals ascending from the periphery.

POSSIBLE CAUSES

But what reduces gray matter? Several causes have been proposed.

Genetics. Some persons who were born with less gray matter than normal may be predisposed to chronic back pain. Researchers also suggest that a decreased hippocampus may be an independent risk factor for posttraumatic stress disorder. (See “Posttraumatic stress disorder: Nature *and* nurture?” *CURRENT PSYCHIATRY*, May 2004, at www.currentpsychiatry.com.)

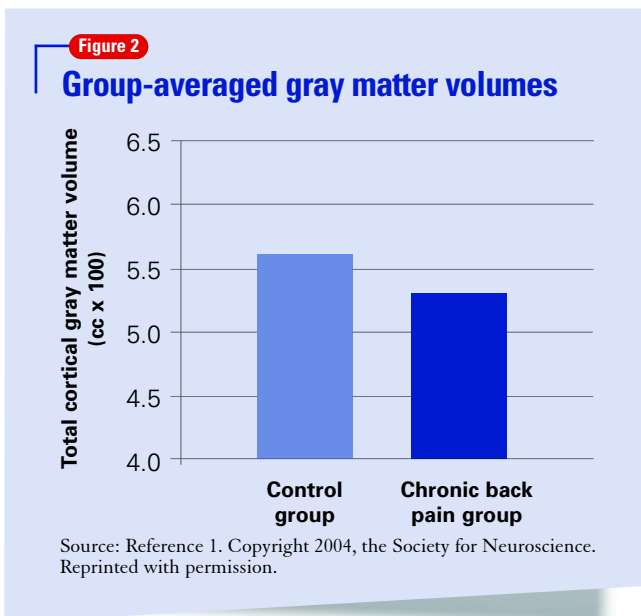
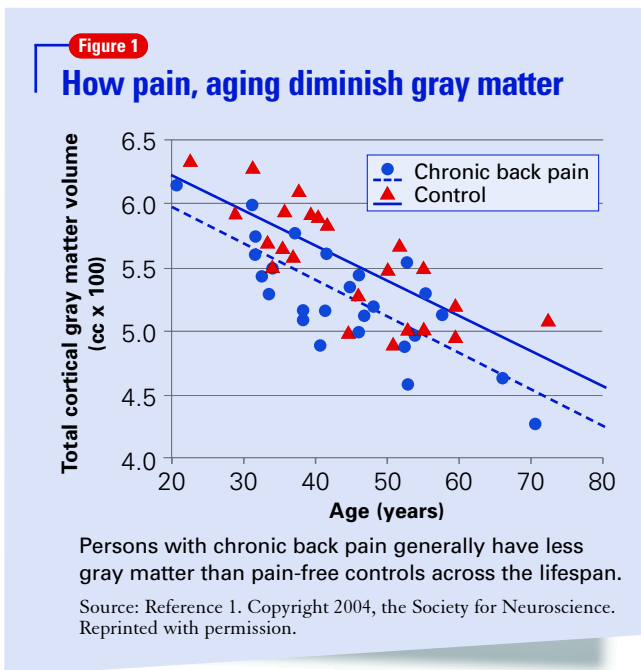
Drugs. Medications and other substances taken to alleviate pain might also reduce gray matter. Excessive alcohol and opioid use have long-term adverse effects on the CNS.³ Is treatment or self-medication mildly toxic to the brain?

Overuse atrophy. Apkarian et al¹ propose that cortical loss may be secondary to overuse. They suggest that persistent pain perception—and the resultant negative affect and stress—causes an excitotoxic and inflammatory state that wears out portions of the brain circuitry. If this is true, then chronic pain itself causes cerebral atrophy.

Whatever the explanation, this study indicates that chronic lower back pain pathology extends beyond the lower back.

Related resources

- ▶ Deyo RA, Weinstein JN. Low back pain. *N Engl J Med* 2001;344:363-70.
- ▶ International Association for the Study of Pain. www.iasp-pain.org.



References

1. Apkarian AV, Sosa Y, Sonty S, et al. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *J Neurosci* 2004;24:10410-5.
2. Lorenz J, Minoshima S, Casey KL. Keeping pain out of mind: the role of the dorsolateral prefrontal cortex in pain modulation. *Brain* 2003;126:1079-91.
3. Goldman D, Barr CS. Restoring the addicted brain. *N Engl J Med* 2002;347:843-5.