

Reduced Hippocampal Function in Fibromyalgia

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Research funded by the American Fibromyalgia Syndrome Association (AFSA) brings scientists closer to understanding what is happening in the brains of people with fibromyalgia. Lead investigator Patrick Wood, M.D., found an essential brain substance was significantly lower in the hippocampus region in a group of fibromyalgia patients, compared to age-matched healthy controls.¹

The hippocampus plays an essential role in memory and learning, pain regulation, and toning down the body's response to stress. Given that fibromyalgia patients have cognitive difficulties, severe pain, and a hyperactive stress response, Wood hypothesized that he would find abnormalities in the hippocampus using magnetic resonance spectroscopy (MRS) imaging.

MRS is a technology that enables researchers to measure the concentrations of various brain chemicals or metabolites being produced. One such metabolite, N-acetylaspartate (NAA), is the substance that Wood found to be significantly low in the hippocampus of people with fibromyalgia. NAA is only produced by neurons (not other types of cells), and a reduced level may represent loss of neurons or metabolic dysfunction of the neurons in the hippocampus. Either way, it implies a reduced hippocampal function.

While many abnormalities have been reported in fibromyalgia, only those that can be tied to the symptoms or disease severity are likely to provide treatment insight. Wood found a link that correlates lower NAA values and the person's level of impairment based on the core features of physical functioning, work status, depression, anxiety, sleep, pain, stiffness, fatigue, and well being. As such, he says his findings lend credence to the idea that a functional or structural defect in the hippocampus contributes to the development of fibromyalgia.

In a previously published study by Wood, he found that fibromyalgia patients did not release dopamine in response to a painful stimulus.² The healthy control group of subjects released plenty of dopamine and experienced much less pain than the group of fibromyalgia patients. The current finding that the hippocampus is not operating properly offers an explanation because it plays a critical role in controlling the amount of dopamine released by other centers of the brain in response to pain. In addition, it further highlights why medications that increase dopamine in the central nervous system are being evaluated for the treatment of fibromyalgia.

What's next? A 2007 report by M. Catherine Bushnell, Ph.D., of McGill University in Montreal, showed an accelerated gray matter loss in the hippocampus and many other brain structures involved in pain processing.³ In August of this year, Bushnell was funded by AFSA to replicate these findings using a larger group of subjects and to determine if the results correlate with a variety of measures pertaining to cognitive function and pain. AFSA is a 501 (c)3, all-volunteer, nonprofit organization dedicated to funding research on the causes and treatments of fibromyalgia. For more information, visit AFSA's website.

1. Wood PB, et al. J Pain [Epub ahead of print] Sept. 2, 2008.
2. Wood PB, et al. Eur J Neurosci 25:3576-3582, 2007.
3. Kuchinad A, et al. J Neurosci 27:4004-4007, 2007.