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Evidence of both systemic inflammation and neuroinflammation in fibromyalgia patients, as assessed by a multiplex protein panel applied to the cerebrospinal fluid and to plasma.

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Abstract

In addition to central hyperexcitability and impaired top-down modulation, chronic inflammation probably plays a role in the pathophysiology of fibromyalgia (FM). Indeed, on the basis of both animal experiments and human studies involving the analysis of cytokines and other inflammation-related proteins in different body fluids, neuroinflammatory mechanisms are considered to be central to the pathophysiology of many chronic pain conditions. However, concerning FM, previous human plasma/serum and/or cerebrospinal fluid (CSF) cytokine studies have looked only at a few predetermined cytokine candidates. Instead of analyzing only a few substances at a time, we used a new multiplex protein panel enabling simultaneous analysis of 92 inflammation-related proteins. Hence, we investigated the CSF and plasma inflammatory profiles of 40 FM patients compared with CSF from healthy controls (n=10) and plasma from blood donor controls (n=46). Using multivariate data analysis by projection, we found evidence of both neuroinflammation (as assessed in CSF) and chronic systemic inflammation (as assessed in plasma). Two groups of proteins (one for CSF and one for plasma) highly discriminating between patients and controls are presented. Notably, we found high levels of CSF chemokine CX3CL1 (also known as fractalkine). In addition, previous findings concerning IL-8 in FM were replicated, in both CSF and plasma. This is the first time that such an extensive inflammatory profile has been described for FM patients. Hence, FM seems to be characterized by objective biochemical alterations, and the lingering characterization of its mechanisms as essentially idiopathic or even psychogenic should be seen as definitively outdated.