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Brain-immune system and its maintenance of brain pain memory in fibromyalgia

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Although some medicines to suppress the pain symptoms in fibromyalgia have been recently developed and used in clinic, the treatments remain to be fully satisfied. One of reasons may be attributed to the long-term use, which may cause side effects. Based on this point of view, we are studying the systems pathology on chronic pain. We have proposed the hypothesis that feed-forward amplification of pain mechanisms plays key roles in neuropathic pain. One of evidence is that self-amplification of lysophosphatidic acid (LPA) production develops and maintains the pain memory in the peripheral neuropathic pain, which may be reinforced by neuro-inflammatory activation. On the analogy of the hypothesis in neuropathic pain, we have been studying the new mechanisms of central pain memory and its reinforcement system in fibromyalgia-like models. Fibromyalgia-like wide-spread centralized pain is developed by twice muscular injection with acidic saline, intermittent cold stress (autonomic) and intermittent psychological stress. All these wide-spread chronic pain diseases were completely cured by repeated brain injections of pregabalin or LPA receptor antagonist. In the present study we will report that peripheral immune system may reinforce the pain memory and it is also regulated by brain pain memory vice versa.