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Commentary

The Role of Brain Functional Network in Exercise Effect on Pain

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DESCRIPTION

Exercise habits promote psychological well-being as well as improve physical function; various mechanisms are possible for exercise effects on pain. Given that exercise habits raise pain thresholds ignoring pain location, involvement of the central nervous system should be considered as an important effect. A systematic review of MRI studies by Baliki, et al. [1] supports that exercise, particularly mind-body exercise, influences the structural and functional organization of the brain. Another systematic review further emphasizes the involvement of the cortico-limbic, default-mode, and dorsolateral prefrontal cortex in the pain-relieving effects of physical activity. Thus, neuroimaging studies have shown that exercise can alter neural activity and functional connectivity in pain-related brain regions, including the somatosensory cortex, insular cortex, Anterior Cingulate Cortex (ACC), and prefrontal cortex [2].

We reported a brain functional network study for exercise effects on pain using the resting-state functional magnetic resonance imaging (rs-fMRI) in 45 people with subacute back pain. In this study, the blood oxygen level-dependent signals were extracted in 2383 voxels on the brain gray matter, Functional Connectivity (FC) between voxels was calculated, and the number of FC in the top 10% was counted as degree of hubness for each voxel. Mediation analysis revealed that people with exercise habits had lower pain intensity associated with decreased hubness in the left thalamus and right amygdala and increased one in the medial prefrontal cortex [3]. These brain regions are part of the pain matrix, which is involved in pain processing, suggesting exercise habits may alter brain functional network in such a way as to build resistance to pain.

On the other hand, chronic pain patients who were not included in our study are known to have complex pathologies, including abnormalities in brain functional network, and are likely to be ineffective with voluntary exercise. Instead, supervised exercise therapy, in which intensity, duration, and frequency of the exercise are optimized to each patient by experienced physiotherapists, may be effective for people with chronic pain. In a study of 15-week supervised physical exercise intervention, fibromyalgia patients showed significant less effect of the Exercise Induced Hypoalgesia (EIH), although pain intensity and health status were significantly improved. The functional MRI data during painful stimulation identified a significant exercise-induced effect on pain-related brain activation in the left dorsolateral prefrontal cortex and caudate [4]. Moreover, functional connectivity between the caudate and the occipital lobe increased during the stimulation after the exercise intervention within fibromyalgia patients.

Animal studies may provide valuable insights into the neural mechanisms underlying the pain-relieving effects of exercise. EIH, a temporary decrease in sensitivity to painful stimuli typically lasting up to 30 minutes after a single bout of exercise, may be attributed to epigenetic changes in activated microglia and sustained GABA synthesis in the spinal dorsal horn. Voluntary exercise activates dopamine neurons in the Ventral Tegmental Area (VTA), promoting dopamine release in the Nucleus Accumbens (NAc) and subsequently activating the mesolimbic reward system. This activation of the reward system leads to analgesic effects, contributing to pain relief [5]. A rodent study of chronic inflammatory pain revealed that daily voluntary wheel running for 30 minutes over a period of 15 days effectively relieved pain and concomitant anxiety. The study also found that regular aerobic exercise increased serotonin release and modulated synaptic plasticity in the ACC.

The key to successful exercise therapy lies in the acquisition of the EIH, in which the reward system plays an important role. Patients' motivation is also required to maximize outcomes in general rehabilitation therapy, but any program that undermines therapeutic motivation is not appropriate. On the other hand, symptomatic pain patients are at risk of experiencing increased pain due to exercise, and appropriate intervention methods must be set up to achieve therapeutic effects [6]. However, previous neuroscience studies identified that pain stimuli suppressed activity of dopaminergic neurons in the VTA, suggesting that the reward system has less function in patients with chronic pain. In fact, brain activity in the NAc induced by a removal of pain stimuli is significantly reduced in chronic low back pain patients compared to healthy controls [7]. Although the suppressed

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condition will make exercise therapy challenging in chronic pain patients, it is important to maintain the activity of daily living, considering that sedentary behavior is a risk factor for chronicity and intractability of pain. Therefore, an appropriate goal setting to keep participants' motivation is required to exercise therapy for chronic pain patents. In the future, the development of a brain activity monitoring system is expected to provide feedback on exercise therapy based on neuroscience evidence.

In a fMRI study with multiple exercise programs, baseline FC between the Dorsolateral Prefrontal Cortex (DLPFC) and Supplementary Motor Area (SMA) can predict the effectiveness of mind-body exercise in improving pain in people with knee osteoarthritis. The FC between the DLPFC and SMA decreased after the exercise programs, while the FC between the DLPFC and ACC increased. In addition, the study found a significant increase in grey matter volume in the SMA among the Tai Chi, stationary cycling, and Baduanjin groups compared to a control group, in which participants just underwent a health education [8].

Thus, in future pain medicine, exercise therapy may be proposed with the improvement of brain function. If the effectiveness of exercise therapy can be predicted by some objective biomarkers, then cost-effectiveness of the therapy can be maximized by an appropriate prescription for the right patient at the right time.

CONCLUSION

Furthermore, once appropriate indices are established, long-term follow-up can be facilitated, and the indices can assist patients in maintaining motivation for self-directed follow-up exercises, resulting in improvement of long-term prognosis and prevention of recurrent painful conditions. Both animal and human studies have demonstrated the analgesic effects of exercise, leading to alterations in brain activity and connectivity within pain-related regions. By gaining a better understanding of the brain function involved in exercise-induced pain relief, we can develop more precise and effective exercise interventions for people with pain.

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