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Editorial

Exercise-induced neuroplasticity: The central mechanism of exercise therapy for chronic low back pain

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Chronic low back pain is common in the clinical setting and its pathology is complicated. Patients usually have kinesiophobia, painful catastrophic thoughts, anxiety, depression, reduced self-efficacy, and other psychological barriers in addition to local pain and limited function, which eventually lead to social function damage, absence from work, and lower quality of life. According to new research, the neuroplasticity change of chronic low back pain patients is an important cause of their biological-psychological-social level dysfunction, which is primarily manifested in several aspects such as central sensitization, steady-state dopamine system change, thalamic cortex dysrhythmia, and sensory motor cortex reorganization. As a result, regulating the plasticity of the central nervous system of the brain with certain rehabilitation measures may be a feasible measure to improve the overall rehabilitation effect of chronic low back pain.

Neuroplasticity is the brain's ability to adapt and change in response to internal and external stimuli. Many factors both inside and outside the body can influence neuroplasticity, resulting in neuronal network reorganization, synaptic activity changes, and nervous system steady-state reconstruction. From the standpoint of therapy, neuroplasticity can be induced in two ways: top-down intervention, which includes exercise imaging, cognitive behavioral therapy, and non-invasive brain stimulation technology, and bottom-up intervention, which includes exercise, manipulation therapy, and stimulation of peripheral physical factors. Exercise-induced neuroplasticity is a key theoretical underpinning of exercise rehabilitation. Its essence is to achieve specific rehabilitation goals by recognizing benign changes in brain structure, function, and metabolism through exercise intervention.

The following are possible mechanisms of induced neuroplasticity in the treatment of chronic low back pain: 1. Activation of the endogenous pain regulation mechanism. Exercise can activate the endogenous opioid system and raise endogenous opioid peptide levels. Furthermore, changes in serotonin, catecholamine, and endocannabinoid levels caused by exercise are one of the possible mechanisms of its analgesia. 2. Increase the functional activity of sensorimotor brain areas. Changes in protein synthesis, synaptogenesis, and M1 functional reorganization in the primary motor cortex (M1) can affect the local release of GABA inhibition in M1, increase the excitability of related cortical spinal cord, and improve synaptic efficiency. By influencing M1 plasticity, the normal activation sequence of core muscles can be restored, and lumbar spine stability can be improved. 3. Increase the concentration of brain-

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derived neurotrophic factor. Exercise has been shown to increase the level of brain-derived neurotrophic factor, resulting in changes in neuroplasticity. Brain-derived neurotrophic factor has been shown in studies to drive nerve remodeling, promote nerve function maintenance and injury repair, and promote long-term enhancement to maintain related brain functions. Furthermore, BDNF can improve dopaminergic system function and increase self-generating activity of related neurons, promoting its central analgesic effect.

Exercise therapy is widely used in the clinical rehabilitation of chronic low back pain, and its curative effect is widely acknowledged. According to the action mechanism analysis, exercise therapy not only has peripheral effects like activating core stabilizing muscles, improving neuromuscular control, and rebuilding lumbar spine stability, but it also has a positive impact on the plasticity of the central nervous system, thus improving the physical and mental disorders of patients with chronic low back pain, as well as their quality of life and social participation. The latter is a physical manifestation of exercise-induced neuroplasticity.