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Mechanism of Cortical Information Output after Exercise Fatigue Induced by D2DR

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Objective In this experiment, the Local field potential (LFPs) was observed in the substantia nigra compact and electrical activity change in corticostriatal pathway after D2DR intervention in exercise-induced fatigue rats. We analyzed the changes of DA neuron discharge and D2DR mediated corticostriatal pathway information transmission. To explore the mechanism of D2D2 mediated DA system in the information output of cortical M1 region.

Methods Wistar rats were used to establish the model of exercise-induced fatigue. The rats were divided into control group (CG), 7 days fatigue group (7FG) and 24 hour recovery group (24RG). We used in vivo multichannel recording technology to record electrical activity in the M1, striatum and substantia nigra compact of rats and observed the electrophysiological changes after D2DR intervention. We also detected the expression of TH proteins in the dorsolateral striatum before and after exercise-induced fatigue by immunohistochemistry.

Results 1) Compared with group CG, the expression of TH protein in the dorsolateral area of striatum was significantly decreased in group 7FG (P<0.05). 2) Compared with the CG group, the power spectral density of the θ , α and β band of the SNc was increased after seven days of exhaustion exercise(P < 0.05); After 24 hours of recovery, the PSD value decreased significantly compared with the 7FG group(P<0.05). 3)Compared with the CG group the power spectral density of alpha (7-13Hz) and beta (15-30Hz) bands in the M1 region and striatum was increased in 7FG after injection D2DR agonist(P < 0.05) .

Conclusions After exercise-induced fatigue, the activity of substantia nigra was increased, and the activity of M1 and striatum was lower than that of the blank control group after the D2DR agonist injection. As a key receptor for the DA signal system, D2DR regulates the electrical activity of the nigrostriatal DA pathway and affects the comprehensive information output of the cortex, which can be regarded as a target for exercise-induced fatigue (NSFC: 31401018, SKXJX: 2014014, Corresponding houlj@bnu.edu.cn).