



The parvalbumin positive neuron involved the regulation of motor cortex excitability in the exercise-induced fatigue

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Objective Objective: Cortical parvalbumin-expressing inhibitory neurons (PV) control the activity of excitatory neurons and regulate their spike output. The present experiment is to determine the role of PV neuron in the regulation of excitability of primary motor cortex (M1) during the exercise-induced fatigue and possible molecular mechanism.

Methods Methods: Male Wistar rats randomly divided into control group (C), exhaustive exercise group (E) and repeated exhaustive exercise group (RE). The gradually increasing load treadmill exercise-induced fatigue model was employed in the Group E and RE. The in vivo multi-channel recording methods was used for recording the neuronal electrophysiological activities of primary motor cortex. To observe the neuron firing rate changes during the rest state, immediately after exhausted exercise and after repeated exhaustive exercise. We also detected the expression of PV positive neurons in the primary motor cortex by the immunofluorescence method. The western blot method was used to determine the expression of calmodulin-dependent protein kinase II (CaMKII), phosphorylated calmodulin-dependent protein kinase II (pCaMKII) and extracellular signal regulated kinase (ERK) in the primary motor cortex.

Results Results: The electrophysiological results indicated that the neuron firing rate after repeated exhausted exercise the neuron firing rate significantly decreased compared with the rest state ($P < 0.05$), but have no significant changes as compared with exhausted exercise; The expression of PV positive neurons in the group of E and RE significantly increased compared with the group C ($P < 0.01$); The western blot results indicated that the protein expression of ERK in group RE significantly decreased compared with group C, the pCaMKII expression of group RE decreased, but have no statistical difference.

Conclusions Conclusion: After exercise-induced fatigue, the increase of PV positive neuron maybe one reason for the excitability changes in primary motor cortex. The alterations in the electrical signal may be participate in the regulation of exercise-induced fatigue. pCaMKII and ERK signal pathway may involved in the molecular mechanism of exercise-induced fatigue.