Effects of exercise-induced fatigue on autonomic activity and dopamine metabolism in rats after D2DR modulation

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Objective: After injection of D2DR antagonist and agonist, the autonomic activity and striatal neurons electrical activity of rats with exercise-induced fatigue were recorded to explore the role of DA receptors in the central mechanism of exercise-induced fatigue.

Methods: Used male Wistar rats, randomly divided into 7 groups: control group (CG), one-time exhaustive exercise group (1FG), 3D repetitive exhaust group (3FG), and 7D repetitive exhaustion group (7FG), 7D repeated exhaustive 24h recovery group (24RG) and 7D repeated exhaustive 48h recovery group (48RG). After 1 week adaptive training in rats, rats attend 7D exhaustive treadmill exercise. Subsequently, the autonomic activity changes of each group with D2DR antagonists and agonists were observed in open filed. Used glass microelectrode extracellular recording technique to observe the dorsolateral striatum neurons change of the rats injected with D2DR antagonist spiperone. Real-time PCR (RT-PCR) molecular biology methods were used to measure the expression of D1DR and D2DR in the striatum after exercise-induced fatigue. To investigate the role of DA neurotransmitter and receptor on central mechanism of exercise-induced fatigue.

Results: (1) With the increase of treadmill exercise load, the total distance of each group became shorter, and the recovery phase gradually recovered to a quiet level. The maximum exercise speed of rats in 7FG was significantly higher than 1FG (P<0.05). The average exercise speed of rats in each group was significantly lower than CG (P<0.05). The average speed of 7FG and 24RG were significantly lower than 1FG (P<0.05). The average movement speed of the 48RG was higher than 7FG; (2) As D2DR antagonist injection, the exhaustive time of rats was significantly lower than CG(P<0.01), while the exhaustive time of D2DR agonist intervention was significantly increased (P<0.01). The active areas of the rats in the open field were concentrated in the corners and margins. The distance of normal rats in 60 min was about 159 m. The activity of rats decreased after D2DR antagonist intervention, the movement distance of rats in CG, 1FG and 48RG were significant reduced; (3) After injection of D2DR agonist, The excitability of dorsolateral striatum neurons were affected by 56.10%, 9.76% (4/41) increased excitability, and 46.34% (19/41) decreased, the inhibitory effect of D2DR agonist was higher than excitatory effects (P<0.05); (4) RT-PCR data showed that there was no significant change in the expression of D1DR in the striatum after exercise-induced fatigue, and D2DR was significantly higher than the CG (P<0.01).

Conclusions: (1) With the increase of fatigue in rats, the total distance of exercise in each group gradually decreased; (2) Exercise-induced fatigue affects the expression of DA receptors in the striatum; (3) D2DR antagonists and agonists can affect the locomotor ability of rats; (4) D2DR antagonist can inhibit striatal neurons in rats with exercise-induced fatigue, suggesting that D2DR may be one of the drug intervention targets of exercise-induced fatigue. (NSFC: 31401018, SKXJX: 2014014).