



Time-sequential Changes of Myocardial AMPK and PGC-1 α Expression during Detraining between High-intensity Interval Training and Moderate-intensity Continuous Training on Wistar Rats

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Objective Without appropriate training stimulus, the athlete experiences a loss of the physiological adaptations brought about by exercise. In most of highly trained athletes, short of training induces a rapid decline in VO_{2max} , but it remains above control values. However, there is no specific information for normal people about the effects of detraining during certain period aerobic training. Therefore, the purpose of this study was to examine the effects of detraining between high-intensity interval training (HIIT) and moderate-intensity continuous training (MICT) on myocardial AMPK and PGC-1 α expression characterization time-sequential changes in Wistar rats, as well as the changing characteristics between AMPK and PGC-1 α expression characterization and cardiac respiratory fitness (CRF). Moreover, the potential mechanism for exercise arrangement was also investigated.

Methods In this study, 27 four-month-old male Wistar rats were randomly divided into the sedentary control group (C), MICT group (M) and HIIT group (H). Animals in the training groups ran on a treadmill 5 days/week during 6 weeks. HIIT group (70%-90%-50% VO_{2max}) and MICT (50% VO_{2max}) group was ran for 50min exercise every training day. All the rats free to gather the food and drinking water. All rats were measured the VO_{2max} after a week adaptive training and then the M group and H group began to exercise intervention. After 6 weeks of training, rats were randomly selected from each group at the 24h, the 3rd day, the 7th day and the 10th day. The Maximal oxygen uptake test was carried out before the samples were taken, and the abdominal aortic blood, myocardium and other tissues were collected after anesthesia. The expression characterization of AMPK and PGC-1 α was tested by Western blotting analysis. All statistical analyses were performed using SPSS 17.0 and GraphPad Prism 5.01 for Windows. Data was presented as mean and standard deviation (SD), unless otherwise stated. The two-way ANOVA (intervention \times time) with repeated measures were used to analyze differences of HIIT and MICT with time-sequential. One-way ANOVA was used to compare the difference between time-sequential among the groups for each variable. The relationship between variables was assessed using the Pearson correlation coefficient. The expression characterization of the detraining effect was also assessed using Cohen's d effect sizes (ES) and thresholds (<0.5=small; 0.5~0.79=moderate; \geq 0.8=large). The level of significance was set at $P < 0.05$ and the confidence intervals at 95%.

Results VO_{2max} showed a gradual downward trend in both H and M groups throughout the 10 days detraining periods. Detraining in the 10th day, training cessation resulted in the VO_{2max} of H and M group were significantly lower than detraining 24h. ($P < 0.05$). Detraining in 3rd day, myocardial AMPK and PGC-1 α increased in H group, it was significantly higher than the C group ($P < 0.05$), but there is no differences in the other detraining days ($P > 0.05$). Furthermore, detraining in the 7th day myocardial PGC-1 α decreased in H group, this value was significantly lower than detraining 24h ($P < 0.05$). Detraining in 7th day, myocardial AMPK and PGC-1 α started decreasing, but it was not significant than C group or other detraining days ($P > 0.05$).

Conclusions (1) The present data suggest that 6 weeks HIIT and MICT can increase the expression of myocardial AMPK and PGC-1 α , the VO_{2max} training effects disappeared after 10 days detraining.

(2)Detraining during the 3rd day and the 7th day was the critical time point for retraining, endurance training intervention should be arranging among these days. (3)The VO_{2max} time-sequential changes was partially consistent with AMPK and PGC-1 α expression characterization, but AMPK and PGC-1 α expression characterization was more sensitive than VO_{2max}.