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High-intensitive training leads to increasing apoptosis of podocyte in kidneys of rats

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Objective Athletes often choose high-intensitive training load to improve athletic ability in their training cycle, its purpose is to pursue the super-compensation after training, but athletes and coaches frequently ignore the balance between load and reasonable recovery, which produce a sustained decline in athletic ability and physical function, and cause the viscera function disorder. High-intensitive training damages the structure of renal filtration barrier in rats, especially the destruction and fracture of Silt diaphragm ultrastructure, as well as the partial fusion of foot processes, etc., so that the large amount of macromolecular proteins in the blood leak out far beyond the threshold of renal tubular reabsorption and form exercise-induced proteinuria. Podocyte is one of the most important. This research establishes a model of rats which simulates the progressive-load training in the cycle of athletes. Observe the apoptosis of renal cells in rats by bioimaging. Determine whether long-term intensive training causes apoptosis in kidney of rats or podocytes, and detect the expression and distribution of Bcl-2 and Bax, and the protein expression of Caspase-3, which is the final regulatory factor of apoptosis pathway. Study the mechanism of the abnormal podocyte due to intensive training from the characterization of kidney to the molecular level, providing experimental basis for explaining the relationship between intensive-training and exercise-induced proteinuria to guide of scientific training.

Methods This study selects 36 Sprague-Dawley rats, which are randomly divided into 3 groups: a control group (group C, 12), a group drawn immediately after exercise(group EI, 12), a group drawn 24 h after exercise(group EA, 12). Group C does not train. The rats in group EI and EA train on the treadmill with an increasing load for 6 weeks(10% grade, 6 d/w): in the first week, the rats run for 10 min at 10 m/min. Starting from the second week, the running speed increases by 5m/min/w, and the training time increases by 30min/w. In the last week the rats run to exhaustion if they could not maintain the target intensity. Record the exhausting time of rats, then group EI and group EA are respectively drawn immediately and 24 hours after exercise. Detect the apoptosis of renal cell apoptosis in rats by TUNEL, observe the ultrastructure of podocytes by TEM, detect urine total protein by BCA, serum and urine creatinine by Jaffe, serum urea by two-point dynamic method, the expression and distribution of Bcl-2 and Bax by immunohistochemistry, and the expression of Caspase-3 by western-blot.

Results The rats in group EI and EA gradually lose weight at the first weekend of training, and their weight drop significantly from the third weekend to the end, it shows a significant difference compared with group C($p<0.01$). There is no significant difference between group EI and EA. Total protein/ creatinine in urine of rats 30 min and 24 h after exercise is significantly higher than that of group C($p<0.01$), and group EA is slightly retuned and lower than group EI($p<0.05$). The H-SCORE of group EI and EA is significantly higher than group C($p<0.05$), while that of group EA is higher than group EI($p<0.05$). It shows by fluorescence microscopy that the positive cells in the group EI are close to the glomerulus and more than group EA. The results of TEM show that group C is normal, and the podocytes own normal chromatin and regular nucleus; Group EI: there are podocytes with apoptosis characteristics, cell chromatin aggregation in nucleus, cell volume reduction, concentration of cytoplasm, cell membrane integrity, but foot processes significantly disappear. Group EA: there are podocytes with apoptosis characteristics, the chromatin of the podocyte nucleus ruptures into several

fragments, the cell volume decreases, the cytoplasm concentres, the pseudopod decreases, the cell membrane is intact, and the vacuole in the cytoplasm increases obviously. Compared with group C, serum corticosterone is significantly decreased in group EI and EA, and there is a significant difference ($p<0.01$), while group EA is significantly decreased but still significantly lower than that in group EI($p<0.01$). The Bcl-2 histological mean optical density in group EI and EA is lower than that of group C, and there is a very significant difference ($p<0.05$), while there is no significant difference between the two groups. But Bax in group EI and EA has no significant difference with group C. The distribution of Bax is significantly different: group C is distributed in the entire field of vision, and little in glomerulus, while the Bax distribution in glomerulus of group EI and EA became extremely rich, and the aggregation trend of group EI is the most obvious. Only the ratio of kidney Bax/ bcl-2 in group EA shows a significant difference compared with group C ($p<0.01$). The expression of Caspase-3 in group EI has no significant difference with group C($p>0.05$), while group EA is higher than group C and EI($p<0.05$).

Conclusions Persistent proteinuria is observed in rats after intensitive training, their renal function is disordered and cannot be effectively recovered after 24 h rest, and renal cell apoptosis increases. High-intensitive training reduces the expression of Bcl-2, and the Bax/Bcl-2 ratio increases with the prolongation of recovery time. Caspase-3 shows the same trend. It is suggested that the change in the expression and ratio of Bax and Bcl-2 in renal under excessive training stress is an important regulatory mechanism for the occurrence of apoptosis in renal cells. Meanwhile the apoptosis of renal cells increases in rats after training, and the apoptosis characteristics of podocyte in the glomerulus are obvious at 30 min and 24 h after exercise, and the structure and function of Slit diaphragm are damaged.