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Overtraining results in abnormality of renal silt diaphragm in rats with persistent proteinuria

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Objective Silt diaphragm is the most important and bioactive membrane structure in the filtration barrier of kidney, and the root cause of proteinuria is the structural and functional abnormalities of Silt diaphragm. So far, there is little literature on the changes of silt diaphragm caused by overtraining. This research establishes a model of rats with exercise-induced proteinuria with long-term intensitive treadmill exercise, and it simulates the progressive-load training in the cycle of athletes. Histological and ultrastructural changes of kidney immediately and 24 h after exercise are observed, and it aims to analyze the change law of silt diaphragm during the occurrence of persistent proteinuria.
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. Observe the histological changes of renal glomerulus by optical microscope, and the ultrastructure of silt diaphragm by TEM. Detect urine total protein by BCA, serum and urine creatinine by Jaffe, serum testosterone and corticosterone by radioimmunoassay, serum urea by two-point dynamic method, and the expression of Nephrin 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 the exercise group. Glomerular morphology, group C: The structure of glomerulus is compact, the boundary between vascular sphere and the wall of capsule is obvious, and the distribution of erythrocytes in vessels is regular; Group EI: The thickness of glomerulus membrane is uneven, the structure of the podocyte is incomplete, part of the foot process is fused, and SD is discontinuous; Group EA: Part of the glomerular endodermis is abnormal, part of the foot process is fused, detached, and unevenly distributed, and SD is discontinuous. 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($p<0.05$). Compared with group C, Serum Testosterone/Corticosterone of rats in group EI and EA is significantly decreased, and there is a significant difference ($p<0.01$). However, there is a significant decrease in EA group but it is still significantly lower than group EI($p<0.01$). Serum corticosterone is significanty 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($p<0.01$). Serum urea and espression of Nephrin are significantly decreased in group EI and EA($p<0.01$), but there are no significant difference between group EI and EA($p>0.05$).
Conclusions The rats suffer from overtraining syndrome caused by intensitive training, and with persistent proteinuria, their renal function is disordered and cannot be effectively recovered after 24

h rest. Meanwhile, the renal morphology and ultrastructure of silt diaphragm in rats undergo "pathogenical-like" changes, which do not significantly decrease with the extension of recovery time. It is revealed that the injury of renal structure and ultrastructure of silt diaphragm caused by overtraining are the structural basis of continuous exercise-induced proteinuria.