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Exercise and dietary intervention reduce myocardial oxidative stress in male rats with metabolic syndrome

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Objective Both aerobic exercise and alimentary control relieve oxidative stress (OS). However, there may be different modes. The objective of this study is to identify the effects of OS in metabolic syndrome (MS) rats and explore the mechanisms involved in aerobic exercise and alimentary control.

Methods Seventy rats were used. Eight rats were randomly chosen for the control group, while the remaining rats were subjected to MS using a high-fat diet for 18 wk. The MS model rats were subsequently divided into the MHC (model control with high-fat diet), MRC (model control with routine diet), MHE (model training with high-fat diet) and MRE (model training routine diet) groups for 12 wk. The chemotactic factor macrophage chemoattractant protein-1 (MCP-1), the adherence factor plasminogen activator inhibitor I (PAI-1), the oxidative stressor oxidized low density lipoprotein (ox-LDL), and the antioxidative factor endothelial nitric oxide synthase (eNOS) were tested in the serum; moreover, the expressions of MCP-1, PAI-1, and eNOS and the regulatory factor of OS Peroxisome proliferator-activated receptor- α (PPAR α) were detected in the myocardium.

Results OS related markers in serum had changes following the interventions of aerobic exercise and/or diet control. Compared with C, the ox-LDL, MCP-1 and PAI-1 in the MS rats exhibited significant decreases ($P < 0.05/0.01$), and the eNOS had a significant increase ($P < 0.05$). Compared with MHC, the diet intervention alone significantly increased eNOS; when interventions of aerobic exercise but not diet control (MHE) were applied, they significantly decreased ox-LDL, MCP-1 and PAI-1 ($P < 0.01$); the applied interventions of both aerobic exercise and diet control (MRE) decreased ox-LDL, MCP-1 and PAI-1 and significantly decreased eNOS ($P < 0.01$). The MRE compared to MHE exhibited significant changes in MCP-1 and eNOS ($P < 0.01$). The mRNA levels of the processing markers of OS in the myocardium. Compared with C, the MS rats had significant increases in the chemotactic factor MPC-1 ($P < 0.05$) and the adhesion factor PAI-1 ($P < 0.01$), which indicated that MS rats exhibited enhanced OS. Moreover, the MS rats had an increased antioxidant marker of eNOS, which was not significant, and the regulatory factor of PPAR α decreased ($P < 0.01$). Compared to MHC, the MRC rats exhibited decreased MPC-1 ($P > 0.05$) and PAI-1 ($P < 0.05$) and increased eNOS ($P < 0.01$) and PPAR α ($P > 0.05$); moreover, the MHE and MRE rats decreased more in MPC-1 and PAI-1 ($P < 0.01$) with increased eNOS and PPAR α ($P < 0.01$). Compared to MHE, the MRE rats had a further decrease in MPC-1 ($P < 0.01$) and PAI-1 ($P > 0.05$) with increased eNOS ($P < 0.05$) and PPAR α ($P > 0.05$).

Conclusions OS increased in MS rats. Moreover, aerobic exercise and alimentary control could decrease OS to reduce the damage in MS rats. The OS regulatory factor PPAR α , which could mediate the expression of OS-related genes, such as MCP-1, PAI-1, and eNOS in cardiovascular tissues, was only enhanced by aerobic exercise and not by diet control.