



## Exercise Biochemistry Review

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### Effect of resveratrol on preventing iron overload in liver of mice

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**Objective** To investigate the mechanism of resveratrol improving iron overload in liver of mice and its effect on alleviating liver oxidative damage.

**Methods** 40 male Balb/Cj mice, 2 months old, were randomly divided into 4 groups, Quiet control group (group C), sucrose iron group (group I), resveratrol group (group R), resveratrol + sucrose iron group (group IR), 10 in each group. Group IC and group IR were intraperitoneally injected with sucrose iron solution (100mg/5mL) once every two days, 75uL each time; R group and IR group were intragastrically resveratrol (Res), dissolved in 1% DMSO solution, each time 30mg / Kg; Group C and IC group were intragastrically administered with 1% DMSO solution once daily for 30 mg/Kg. After the end of the intervention for 8 weeks, the mice were dissected and taken. Perls' staining was used to observe the distribution of iron in the liver of mice; biochemical kit for serum glucose, serum iron (SI), total iron binding capacity (TIBC), liver iron, liver superoxide dismutase (T-SOD), liver malondialdehyde (MDA), total liver oxidative capacity (T-AOC); enzyme-linked immunosorbent assay (ELISA) for detection of serum ferritin (SF), octahydroxydeoxyguanosine (8-OHDG); Western blot detection of FPN1 protein expression in liver; the expression of Hecpidin in liver was detected by PCR.

**Results** (1) Perls' staining results: 8 weeks of iron sucrose caused a significant increase in iron content in mouse hepatocytes, and Res decreased the amount of iron ions in hepatocytes; (2) Results of iron metabolism index: 8 weeks of sucrose iron had a significant increase in liver iron, SI, TIBC and SF ( $P<0.01$ ), and Res intervention reduced liver iron ( $P<0.05$ ) and SF ( $P<0.01$ ). The content of TIBC ( $P<0.01$ ) and SI ( $P<0.05$ ) increased, and the iron supplement and Res interacted with the effects of liver iron, SI and TIBC; (3) Results of glucose metabolism index: iron overload increased glucose level in mice ( $P<0.01$ ), Res decreased glucose level, iron overload reduced liver glycogen storage in mice ( $P<0.05$ ), iron supplement and Res, there is no interaction on the effects of glucose metabolism indicators; (4) Oxidation index results: 8 weeks of sucrose iron significantly inhibited liver T-SOD and T-AOC activity ( $P<0.01$ ), increased liver MDA and 8-OHDG activity ( $P<0.01$ ), and Res promoted liver T-AOC ( $P<0.01$ ) and T-SOD activity increased, and the liver 8-OHDG ( $P<0.01$ ) and liver MDA activity were decreased. The effects of iron supplement and Res on 8-OHDG and T-AOC were interactive ( $P<0.01$ ); (5) 8 weeks of sucrose iron inhibited the expression of FPN1 protein in the liver of mice, and the intervention of Res could enhance the expression of FPN1 protein; iron supplement promoted the expression of Hecpidin in liver ( $P<0.01$ ), Res inhibited Hecpidin, iron supplement and Res had no interaction with the expression of FPN1 and Hecpidin in liver.

**Conclusions** 8 weeks of sucrose iron caused iron overload in the liver of mice, and increased oxidative stress in the liver. Res can alleviate iron overload, reduce oxidative stress and improve glucose metabolism. The main reason is that Res inhibits Hecpidin expression in liver and promotes FPN1 expression and promotes The liver iron is released, thereby reducing the state of iron deposition.