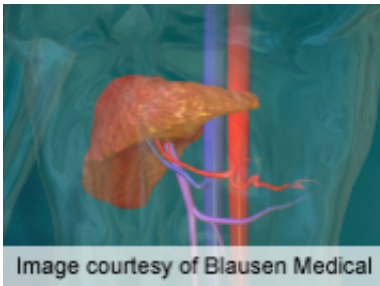


Estrogen may improve pathway-selective insulin resistance

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Estrogen treatment at the time of surgical menopause may reverse aspects of pathway-selective insulin resistance in the liver associated with a high-fat diet in mice by promoting insulin action on glucose metabolism but limiting hepatic lipid deposition, according to a study published in the February issue of *Diabetes*.

(HealthDay)—Estrogen treatment at the time of surgical menopause may reverse aspects of pathway-selective insulin resistance in the liver associated with a high-fat diet (HFD) in mice by promoting insulin action on glucose metabolism but limiting hepatic lipid deposition, according to a study published in the February issue of *Diabetes*.

Lin Zhu, from the Tennessee Valley Healthcare System in Nashville, and colleagues tested whether [estrogen treatment](#) given at the time of ovariectomy (OVX) in mice may alter the impact of HFD feeding. Insulin sensitivity, tracer incorporation into hepatic lipids, and liver triglyceride export were assessed with hyperinsulinemic-euglycemic clamps.

The researchers note that increased adiposity was seen in OVX mice, and was prevented with estradiol (E2) treatment at the time of OVX. Insulin sensitivity was increased with E2 treatment in mice with OVX and HFD. HFD feeding induced fatty liver, and insulin reduced hepatic apoB100 and liver triglyceride export in sham and OVX mice. Liver lipid deposition was reduced with E2 treatment, which also prevented the decrease in liver triglyceride export during hyperinsulinemia. In mice lacking the liver estrogen receptor α , E2 treatment after OVX reduced adiposity but did not improve insulin sensitivity, limit liver lipid deposition, or avert insulin suppression of export of liver triglycerides.

"In conclusion, estrogen treatment may reverse aspects of pathway-selective [insulin resistance](#) by promoting [insulin action](#) on [glucose metabolism](#) but limiting hepatic lipid deposition," the authors write.

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