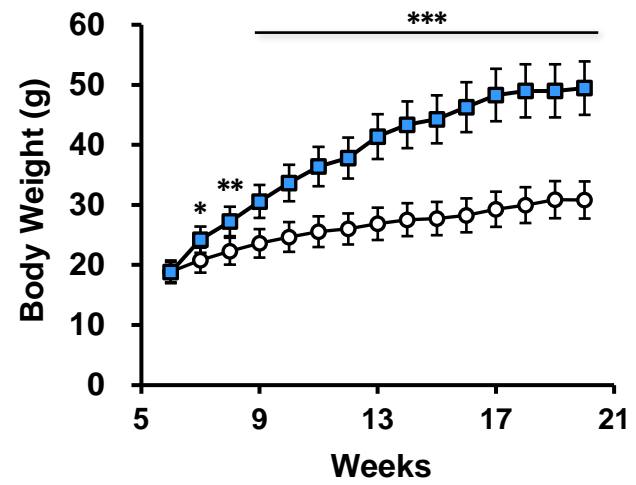
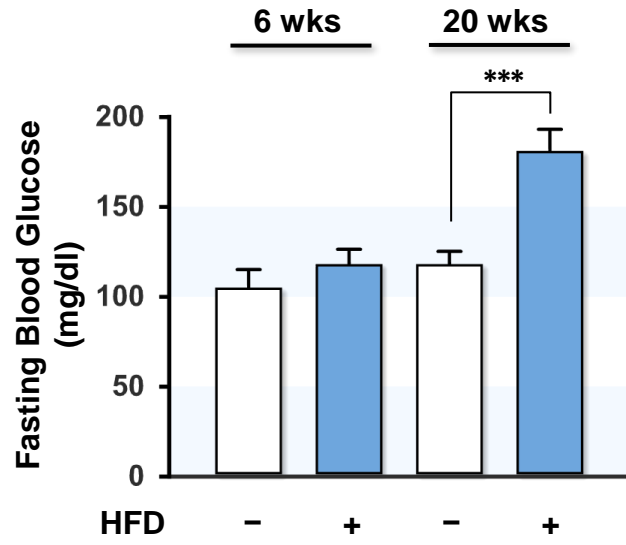


Supplementary Figure S1

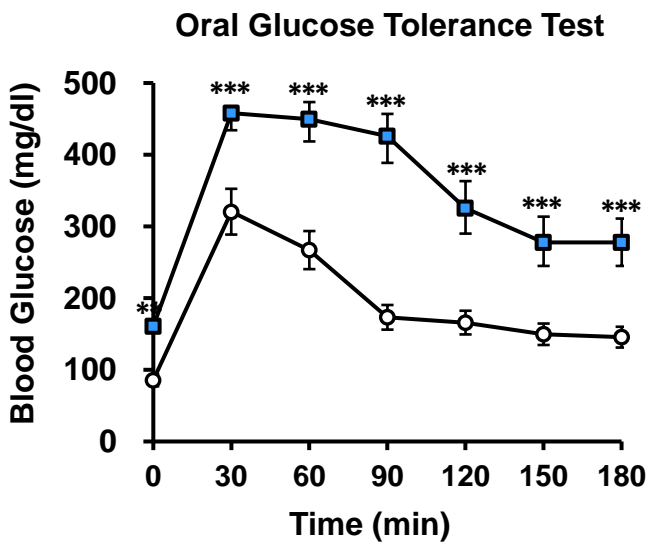
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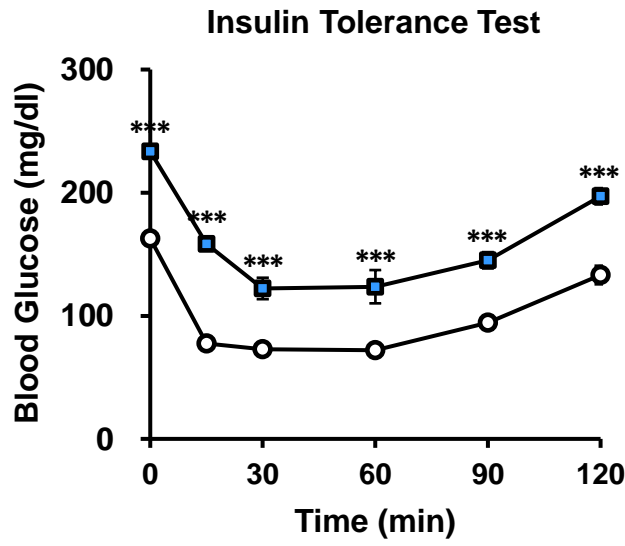
B



C



D



Supplementary Figure S1. HFD induced hyperglycemia, impaired glucose tolerance, and impaired insulin tolerance in mice.

For the diet-induced obese mice model, C57BL/6N male mice were fed either NFD or HFD at the age of 6 wk for 14 wks, as described in the Method section.

(A) The body weight was measured every week. HFD increased body weights significantly after 1 wk of feeding. (B) After 14 wks of HFD feeding (at the age of 21 wk), fasting blood glucose level was significantly increased in mice, indicating that HFD provoked hyperglycemia in mice. (C) For the oral glucose tolerance test (OGTT), mice were fasted overnight, and 20 % glucose solution (10 ml/kg of body weight) was administered orally with an 18-gauge gavage needle. Blood was drawn from a tail vein at 0, 30, 60, 120, and 180 min, and plasma glucose concentrations were measured with the Blood Glucose Monitoring System (SD Biosensor, Seoul, Korea). (D) For the insulin tolerance test (ITT), the mice were fasted 3 h, and insulin (1 U/kg of body weight) was injected intraperitoneally. Blood was drawn from a tail vein at 0, 15, 30, 60, 90, and 120 min, and the blood glucose concentrations were measured.

The whole-body disposal of blood glucose was significantly delayed in the HFD-fed mice compared to NFD-fed mice, suggesting that HFD caused hyperglycemia, IGT, and ITT in mice.

Data are expressed as mean \pm SEM. from 8 mice, for NFD control (open circles or open column) and HFD (blue rectangular or blue column); * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$ vs. NFD control.