**Supplementary Figure 1. Shorter duration of dexamethasone treatment leads to hyperglycemia.**

Insulin responsiveness, via ITT, normalized to baseline (A). Insulin was given via i.p. injection at a concentration of 2.5 U/kg following five weeks of dexamethasone (NCD n=12; HFD n=12) or vehicle (NCD n=12; HFD n=12) treatment and 17 weeks of diet. ITT (B) and baseline blood glucose (C) were measured in another cohort of mice following two weeks of dexamethasone (NCD n=10; HFD n=14) or vehicle (NCD n=13; HFD n=11) treatment and 10 weeks of diet. Insulin was given via IP injection at a dose of 0.75 U/kg (NCD) or 1.5 U/kg (HFD). Fat (D) and lean mass (E) were measured weekly via EchoMRI for the duration of the study. One week following the ITTs, blood glucose levels (F), insulin clearance rates (G), and amount of glucose uptake in gastrocnemius muscle, eWAT and iWAT (H) and heart and brown adipose tissue (I) were measured during a hyperinsulinemic euglycemic clamp in the obese mice only. For clamp experiments, insulin was infused at 8 mU/kg/min following a prime continuous infusion of 40mU/kg bolus. Mice were fasted for five hours prior experiments. Crosses indicate a significant interaction between diet and treatment. Asterisks indicate a statistically significant treatment effect for the pairwise comparison.

**Supplementary Figure 2. Obesity leads to reduced HSL phosphorylation**

Western blot image (A) and quantification (B) of HSL phosphorylated and total protein from IWAT. Mice euthanized at 28 weeks of age following six weeks of dexamethasone (NCD n=8; HFD n=10) or vehicle (NCD n=8; HFD n=10) treatment.