# Figure Legends

**Figure 1: Metabolic characteristics of Cushing’s disease patients in our study.** A) Morphometric data from control (non-secreting adeoma) and Cushing’s disease subjects. A.C indicates abdominal circumference. B) HOMA-IR score, fasting insulin and fasting blood glucose from subjects. C) Liver enzymes from subjects D) Glycerol release from isolated subcutaneous adipose tissue. Asterisks indicates p<0.05.

**Figure 2:** **Dexamethasone treatment results in decreased lean mass and increased fat mass in mice.** Weekly body weight (A), lean mass (B), fat mass (C) and percent fat (D) from control (black) and dexamethasone (red) treated mice. E) Average food consumption per mouse per day. F) Insulin tolerance test. Following a 6 hour fast, insulin (1 mU/g) was administered via IP injection and blood glucose was measured at baseline and the indicated time post injection. G) Inguinal (IWAT) and epididymal (EWAT) fat pad weights, for right fat pads only. Asterisks indicate p<0.05.

**Figure 3: Differentially expressed transcripts in subcutaneous adipose tissue from Cushing’s disease subjects.** A) Heatmap of genes with significant differential expression. The bar on the top indicates control subjects (non-secreting adenoma; black) and Cushing’s subjects (red). B) Genes involved in cortisol signaling. C) Leptin and Adiponectin mRNA levels. Asterisks indicate q<0.05.

**Figure 4: Elevated glucocorticoids result in elevated fatty acid and triglyceride synthesis genes.** A) Fatty acid synthesis genes in Cushing’s disease and control patients. B) Fatty acid desaturases in Cushing’s disease patients. C) Triglyceride synthesis genes. D) Lipolysis genes. E) Steroid biogenesis genes. F) Evaluation of lipogenic genes in mouse subcutaneous adipose tissue. Asterisks indicate q<0.05.

**Figure 5: Glycolysis and glucose oxidation genes are upregulated with elevated glucocorticoids.** Schematic of A) glycolysis and B) the TCA cycle, colored by gene expression changes in subcutaneous adipose tissue from Cushing’s disease subjects. C) qPCR analysis of selected glucose oxidation genes from mouse subcutaneous adipose tissue after 12 weeks of dexamethasone treatment. Asterisks indicate q<0.05.

**Figure 6:** **Increased glucocorticoids are associated with increased protein degradation and decreased strength.** A) Mouse grip strength (N) assessed at baseline, 4, 8 and 12 weeks of dexamethasone treatment. Muscle atrogene (B) and proteasomal transcript expression changes in gastrocnemius muscles from mice following 1 week of dexamethasone treatment. C) Proteosomal mRNA levels from subcutaneous adipose tissue of mice treated with dexamethasone for 12 weeks. Proteasomal (D) and protein catabolism (E) transcript expression changes in subcutaneous adipose tissue from Cushing’s disease and control subjects. F) Heatmap of differentially expressed ribosomal transcripts in Cushing’s disease and control subjects.

**Figure 7: Expression of insulin signaling transcripts, ceramides and inflammatory transcripts in control vs. Cushing’s disease subjects.** A) Insulin signaling transcript expression levels. B) Ceramide levels. C) MHC complex transcript expression levels.

**Figure 8: Transcript expression changes in Cushing’s disease are less robust after adjusting for obesity.**  *FASN* (A), *PSMD8* (B), *IDH1* (C), and lysosomal (D) transcripts in non-obese and obese Cushing’s subjects.