

β 2-chimaerin, a GAP for Rac1, is a novel regulator of Hepatic Insulin Signaling and Glucose Metabolism

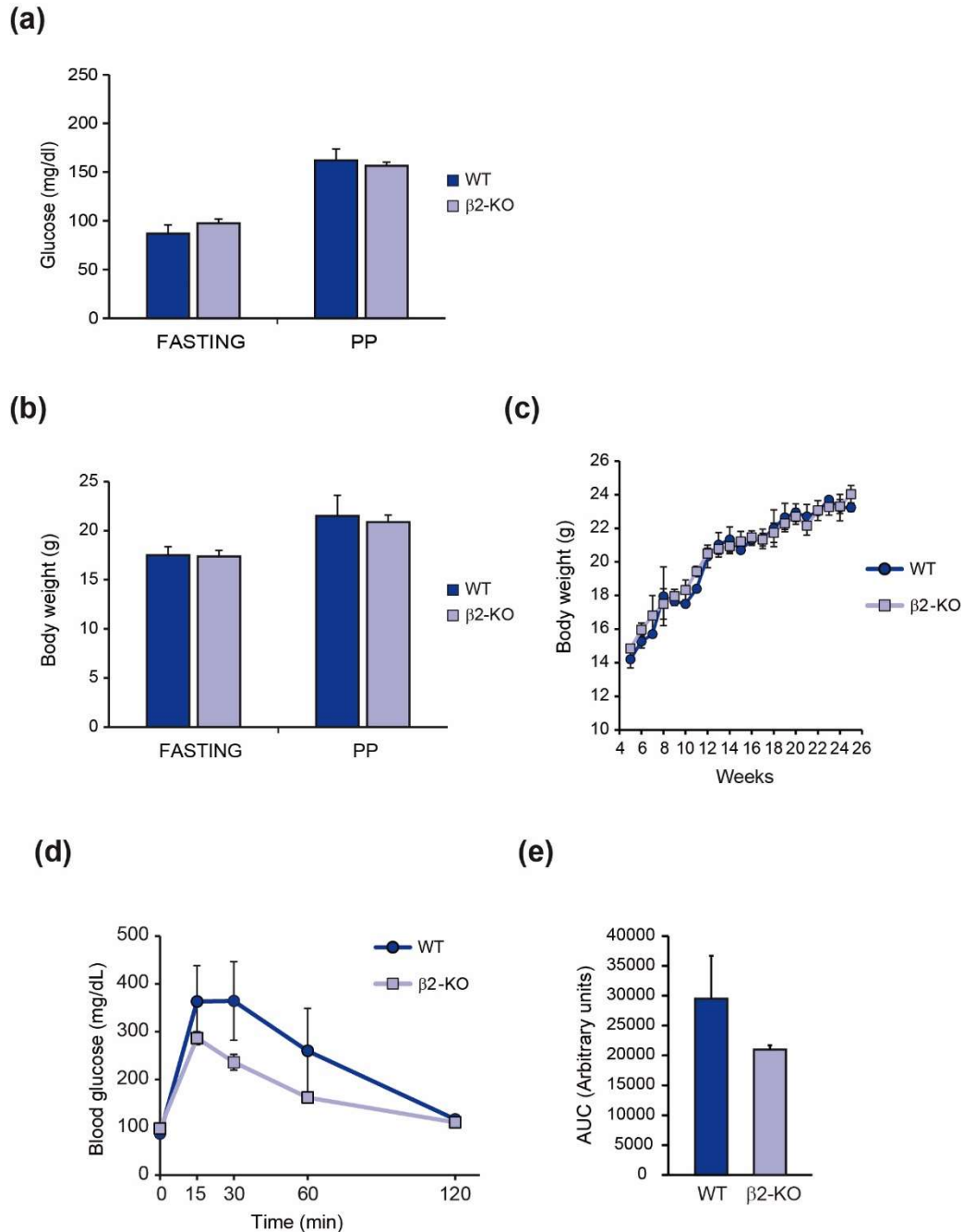
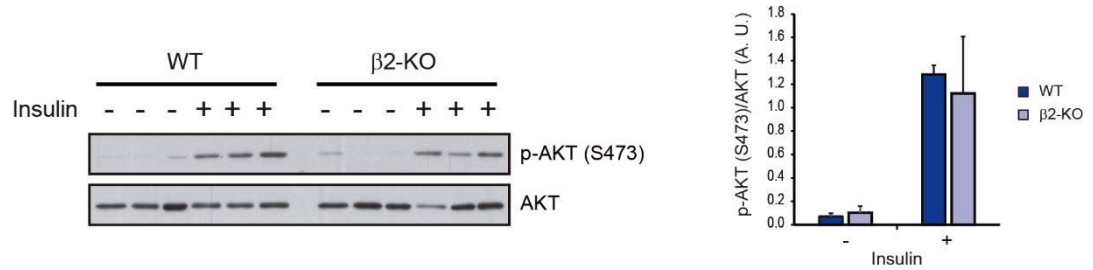


Figure S1. Metabolic features of β 2-chimaerin KO female mice: (a) Blood glucose levels and (b) Body weight of WT and β 2-chimaerin KO (β 2-KO) female mice in fasting and postprandial (PP) conditions at 2-3 months of age (n=4 WT, 8 β 2-KO); (c) Body weight evolution of WT and β 2-KO female mice (n= 4 mice per genotype). Body weight was measured weekly from age 4-26 weeks; (d) Intraperitoneal glucose tolerance test (IPGTT) of female mice at 2-3 months of age (n=4 WT, 8 β 2-KO); (e) Area under the curve (AUC) of the IPGTT. Results are expressed as mean \pm SEM. Statistical significance was assessed by two-tailed, unpaired Student's t test.

(a)



(b)

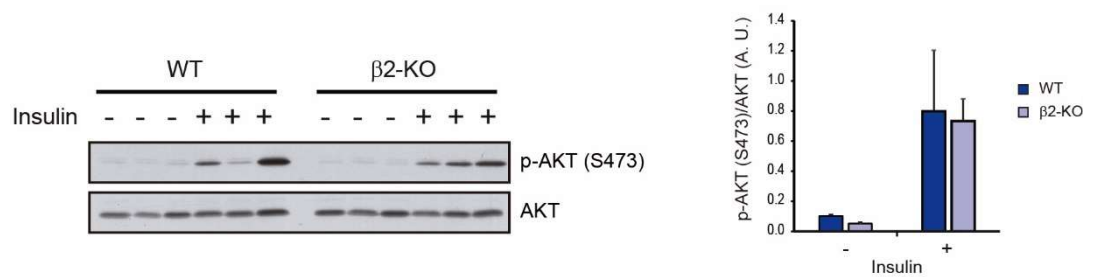


Figure S2. Effect of $\beta 2$ -chimaerin deletion on AKT phosphorylation in adipose tissue and muscle. Representative immunoblot images of basal and insulin-stimulated phosphorylation of AKT (S473) in (a) adipose tissue or (b) skeletal muscle (gastrocnemius) extracts from WT and $\beta 2$ -KO mice. Histograms display densitometric analysis of AKT phosphorylation levels. Data are presented as mean \pm SEM. Statistical significance was assessed by unpaired, Student's *t* test ($p = 0.77$ and $p = 0.89$ for insulin-stimulated p-AKT in WT vs. $\beta 2$ -KO adipose tissue and muscle, respectively). A.U, arbitrary units.