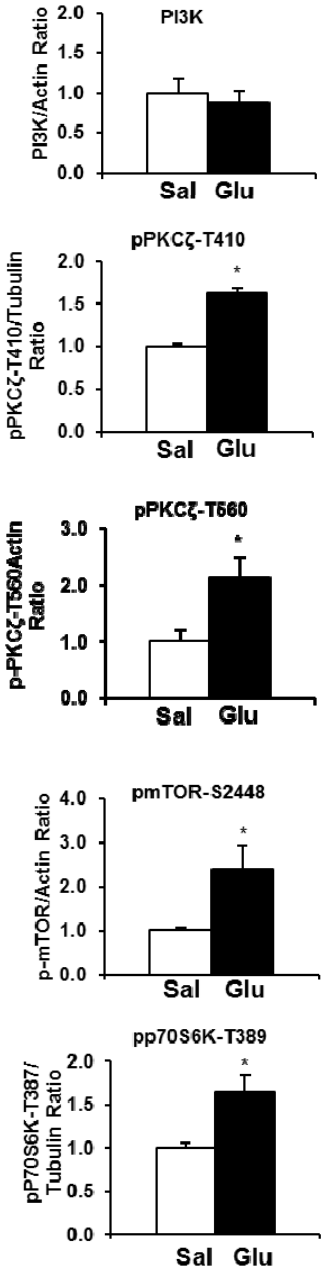
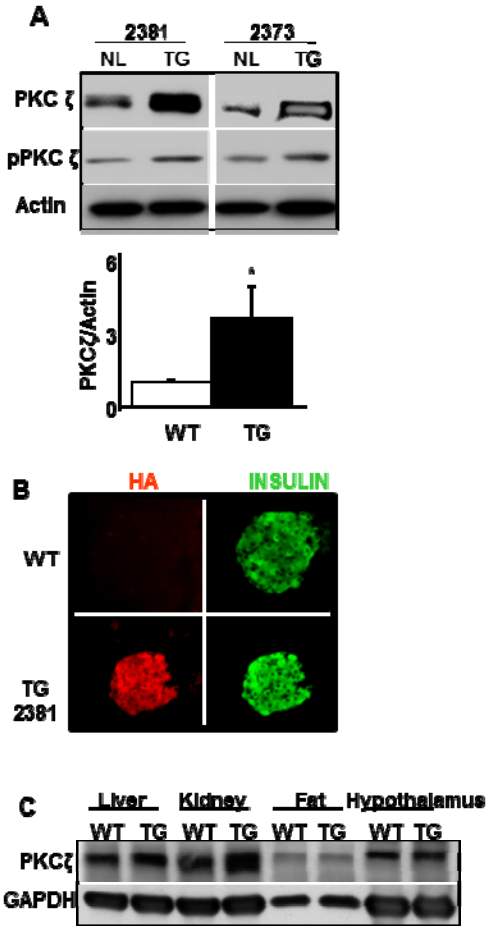
SUPPLEMENTARY DATA

Supplementary Figure 1. Glucose activates PKCζ in mouse islets in vivo. Densitometric quantitation of western blots showing levels and phosphorylation of different components of the PI3K/PKCζ/mTOR signaling pathway in islets isolated from four-day glucose- and saline-infused mice (n=4 mice per condition). Results are means±SEM and *P<0.05 vs saline-infused mice.



SUPPLEMENTARY DATA

Supplementary Figure 2. Generation of RIP-KD-PKCζ transgenic mice. (A) Representative images of western blots showing the expression of PKCζ and phospho-PKCζ in islet protein extracts from wild-type (WT) and two lines of RIP-KD-PKCζ transgenic (TG) mice and the densitometric quantitation in n=3 mice per line. Results are means±SEM and *P<0.05 vs WT. **(B)** Representative images of pancreatic sections from WT and RIP-KD-PKCζ TG mice (line 2381) stained for HA and insulin showing expression of HA only in beta cells. **(C)** Representative image of a western blot showing the expression of PKCζ in different tissues from WT and RIP-KD-PKCζ TG mice.



SUPPLEMENTARY DATA

Supplementary Figure 3. Glucose and beta cell homeostasis in RIP-KD-PKCζ transgenic mice. **(A)** Blood glucose, **(B)** plasma insulin, **(C)** glucose tolerance, and **(D)** insulin tolerance in wild type (WT) (n=8) and RIP-KD-PKCζ transgenic (TG) (n=8) mice at 8-9 weeks of age. Results are means±SEM. **(E)** Pancreas weight and **(F)** beta cell mass in the same mice in which glucose homeostasis was evaluated. Results are means±SEM.