

GIP controls adipose insulin sensitivity via activation of CREB and p110 β isoform of PI3 Kinase

Sameer Mohammad¹, Lavoisier S. Ramos², Jochen Buck², Lonny R. Levin², Francesco Rubino³, and Timothy E. McGraw^{1#}

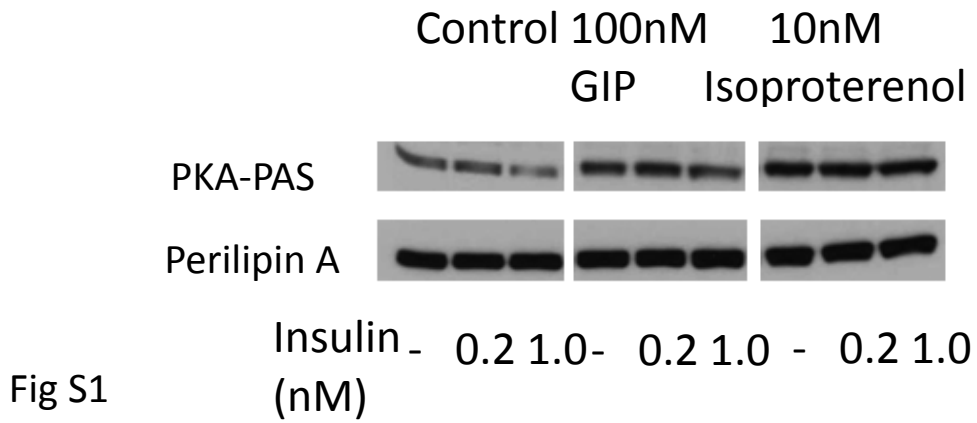
Supplemental Figure legends.

Figure S1

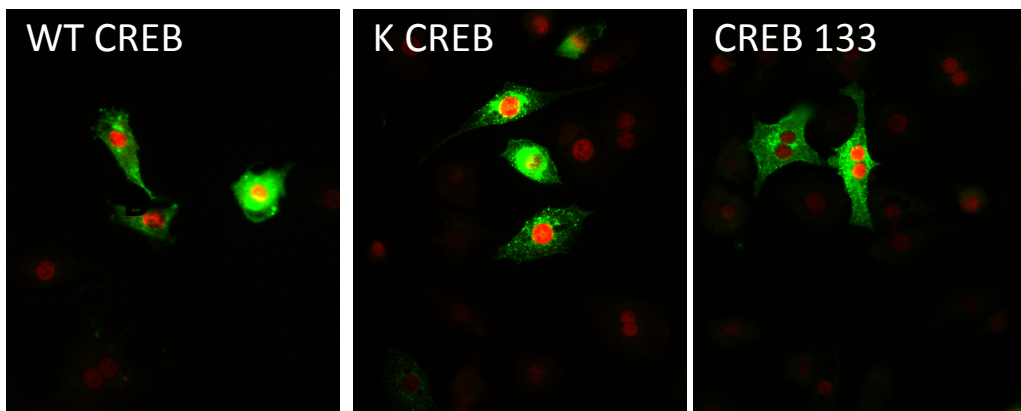
3T3-L1 adipocytes were serum starved for 2 h and incubated with indicated concentrations of GIP and isoproterenol in the presence of various concentrations of insulin for 30'. Cells were harvested in 1X sample buffer and loaded onto a 10% SDS gel. Blots were probed with anti-PKA-PAS and anti-perelipin antibodies.

Figure S2

3T3-L1 adipocytes were electroporated with HA-GLUT4-GFP alone or together with CREB constructs (WT, KCREB or CREB 133). Cells were fixed and stained with anti-CREB antibodies and imaged (A). Images were quantified to measure expression of CREB in cells positive for HA-GLUT4-GFP (B).



A



HA-GLUT4-GFP
CREB

HA-GLUT4-GFP
CREB

HA-GLUT4-GFP
CREB

B

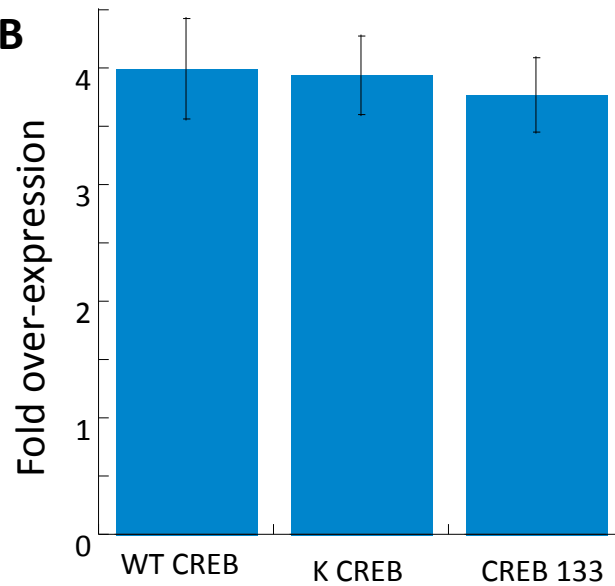


Fig S2