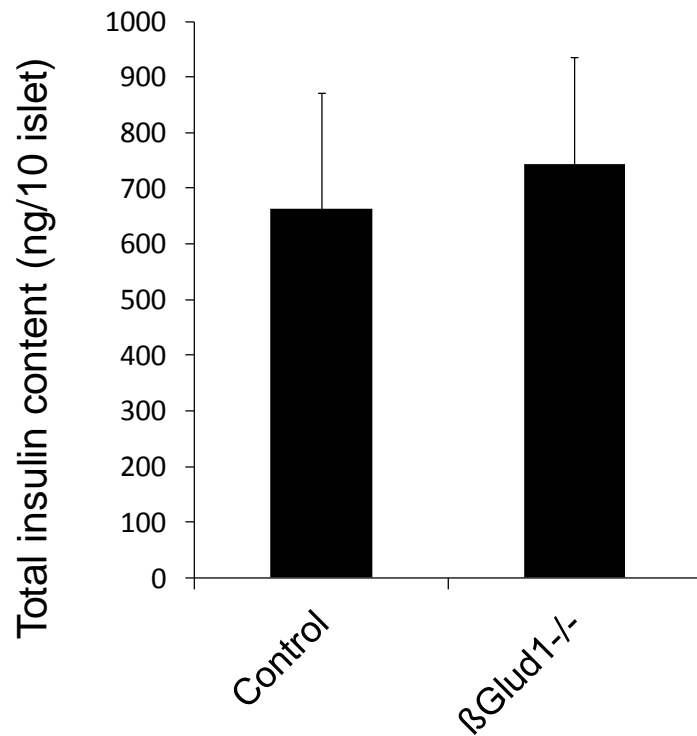


Delineation of glutamate pathways and secretory responses in pancreatic islets with beta-cell specific deletion of the glutamate dehydrogenase.

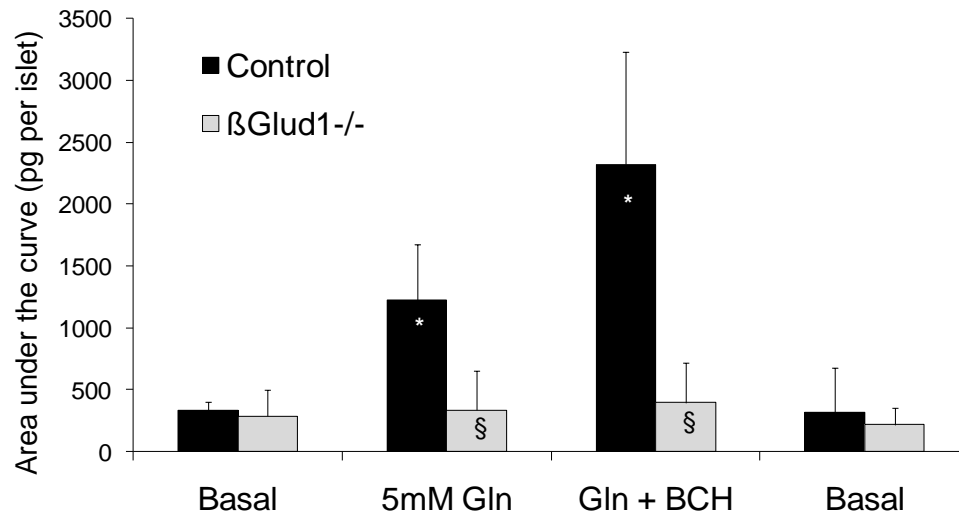
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Supplemental Material

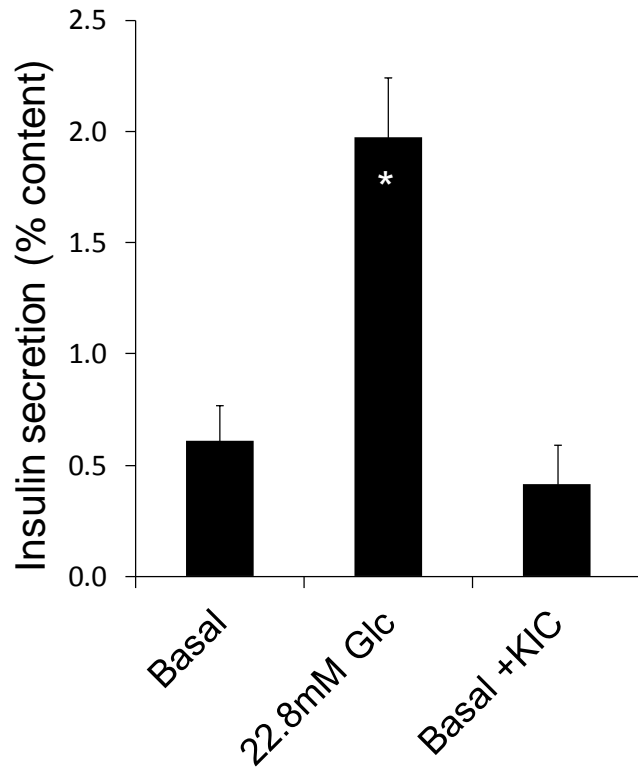
Supplementary Fig.S1. Total islet insulin contents measured on islets isolated for static insulin secretion experiments. Values are means \pm SD, n=25.



Supplementary Fig.S2. Lack of glutamine-induced insulin secretion in β Glud1^{-/-} islets rescued by GDH over-expression. Quantification of insulin secretion depicted in Figure 2A expressed as area under the curve (AUC). After an overnight culture in RPMI-1640 medium, islets isolated from control and β Glud1^{-/-} mice were hand-picked and perfused with KRBH at 2.8mM glucose (Basal). Islets were sequentially stimulated for 15min with 5mM of glutamine (Gln) and Gln plus 10mM BCH.



Supplementary Fig.S3. Secretory responses of control islets to KIC. Insulin secretion was tested in islets over a 1h incubation period at 2.8mM glucose (Basal), 22.8mM glucose (Glc), and at Basal plus 10mM α -ketoisocaproate (KIC). Values are means \pm SD, n=5-8, *p<0.01 versus Basal.



Supplementary Fig.S4. In situ pancreatic perfusion of control and β Glud1^{-/-} mice. Pancreases were perfused with KRBH at 2.8mM glucose before 15min stimulation with 5mM glutamine plus 10mM BCH. Values are means \pm SE of 3 independent mice.

