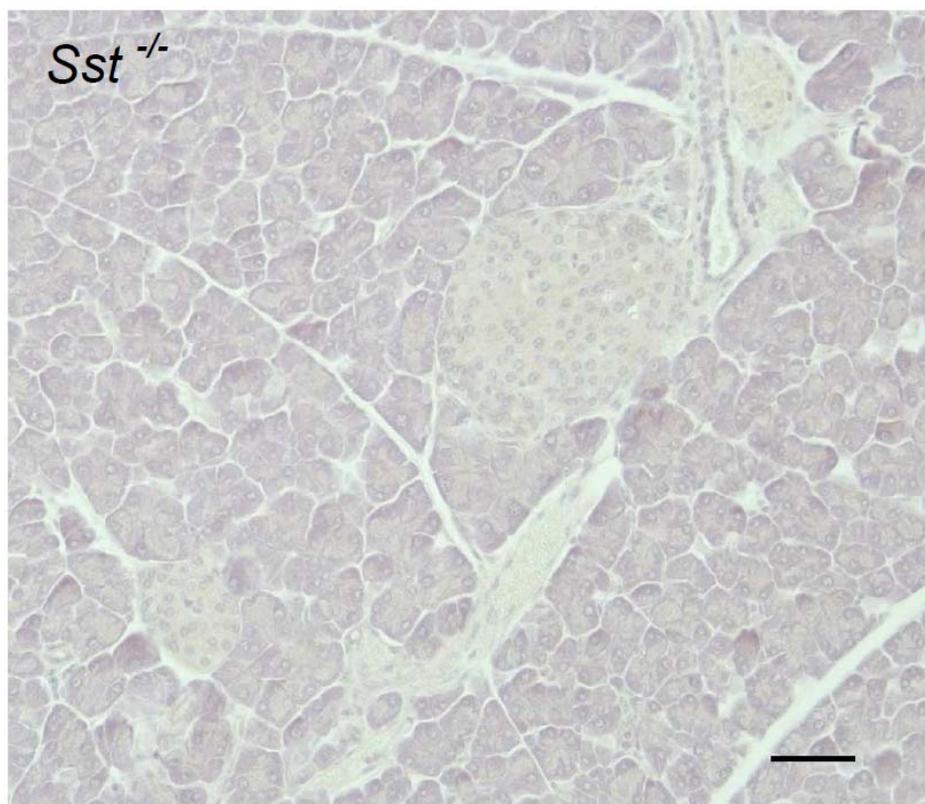
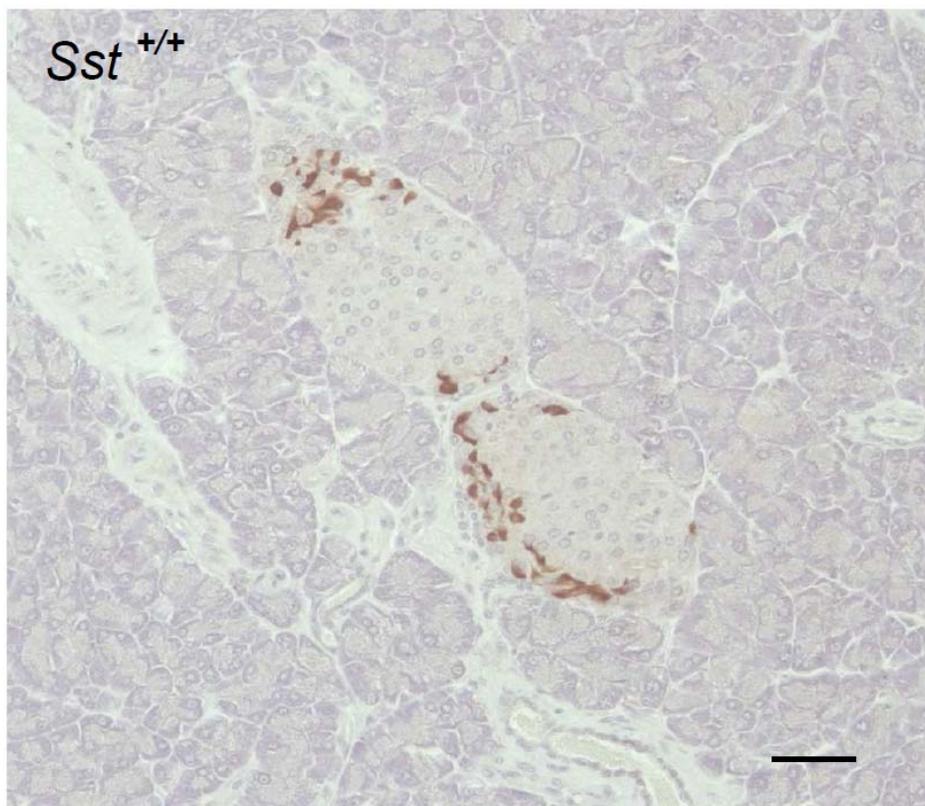


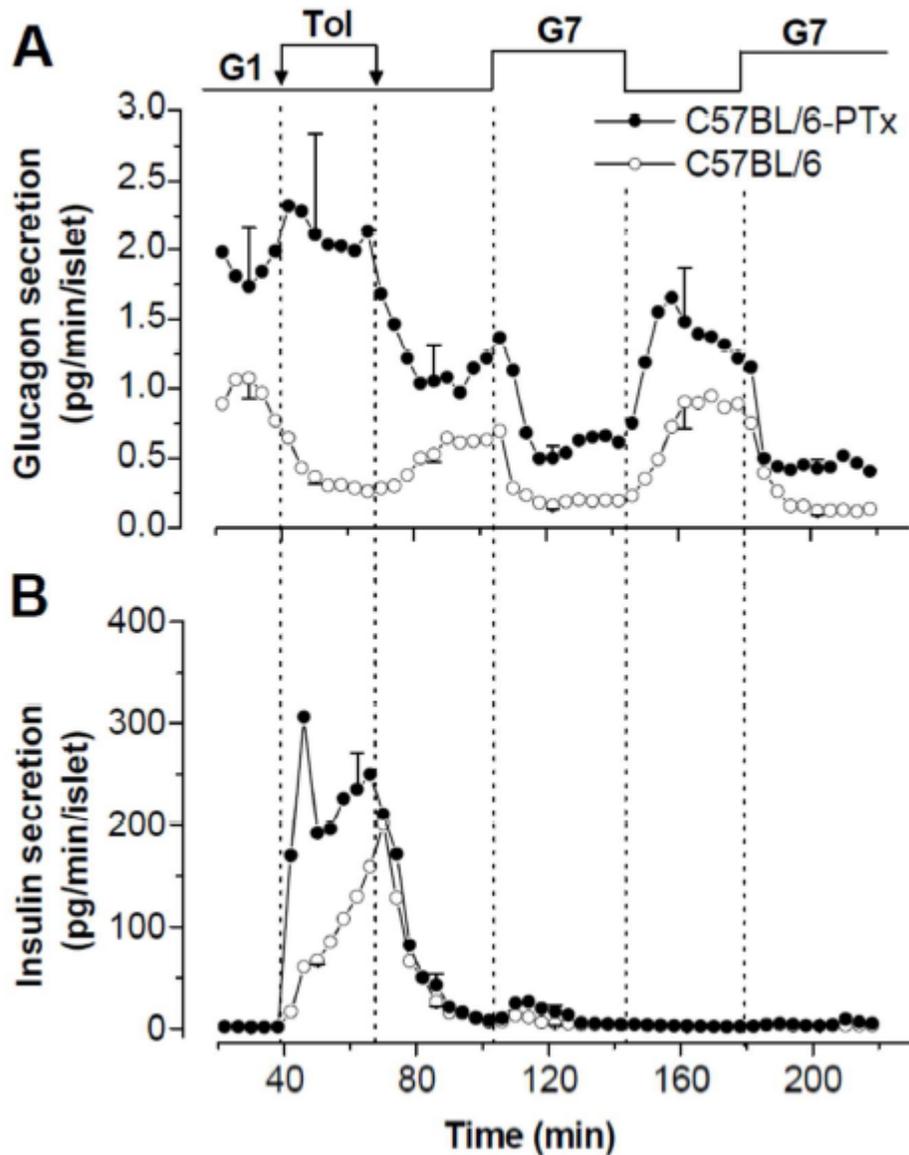
SUPPLEMENTARY DATA

Supplementary Figure 1. Lack of somatostatin in islets from *Sst*^{-/-} mice. Immunocytochemical detection of somatostatin in pancreatic sections of *Sst*^{+/+} or *Sst*^{-/-} mice. Scale bars: 50 μ m.



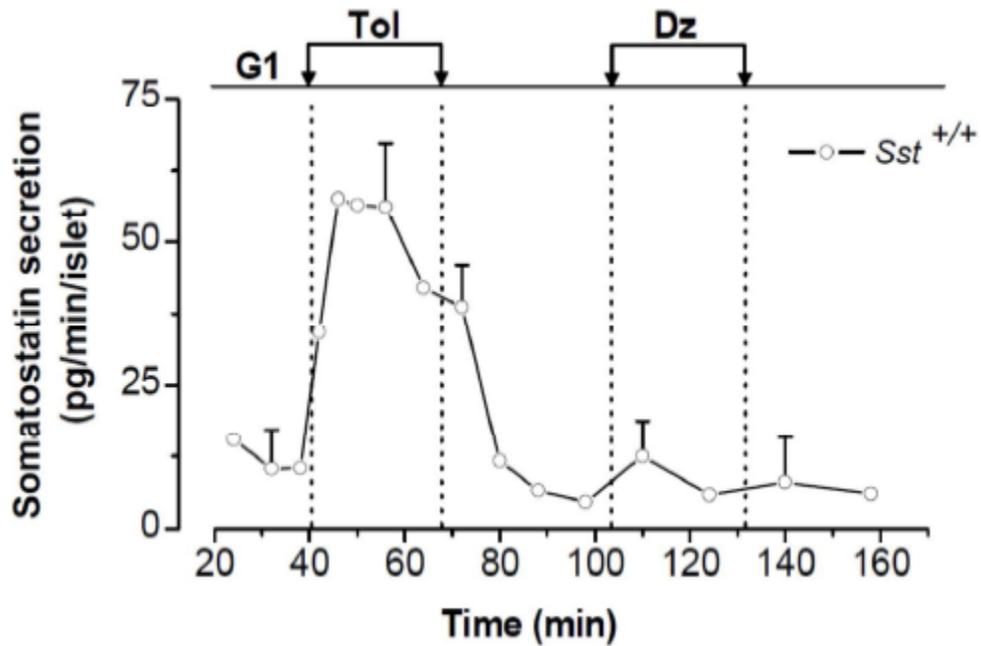
SUPPLEMENTARY DATA

Supplementary Figure 2. Removal of the SST paracrine influence by pretreatment with PTX transforms the inhibitory effect of tolbutamide into a stimulatory one but does not prevent the glucagonostatic effect of glucose. Islets from C57BL/6 mice were pretreated or not for 18h during the culture with 200 ng PTX. They were then perfused with a medium containing alanine, glutamine, arginine (2 mmol/l each, Mix AA). The glucose (G) concentration of the medium was changed between 1 (G1) and 7 mmol/l (G7), and 500 μ mol/l tolbutamide (To) was applied when indicated. Traces are means \pm SE for 3-4 experiments with islets from different preparations.



SUPPLEMENTARY DATA

Supplementary Figure 3. Tolbutamide stimulates somatostatin release from islets perfused in an amino acid-free medium containing 1 mmol/l glucose (G). 500 μ mol/l tolbutamide (Tol) or 250 μ mol/l diazoxide (Dz) were applied when indicated. Traces are means \pm SE for 3 experiments with islets from different preparations.



SUPPLEMENTARY DATA

Supplementary Figure 4. Zn^{2+} is not the paracrine factor responsible for glucose-induced suppression of glucagon secretion. Batches of 7 islets from $ZnT8^{+/+}$ and $ZnT8^{-/-}$ mice were preincubated for 1h at 37° C with 7 mmol/l glucose and then incubated for 1h in the presence of 10 mmol/l glucose (G10) or in the absence of the sugar (G0), and in the presence of 20 mmol/l arginine. Values are means \pm SE for 17-20 batches of islets. **P < 0.01 vs G0 in the same strain of mice.

