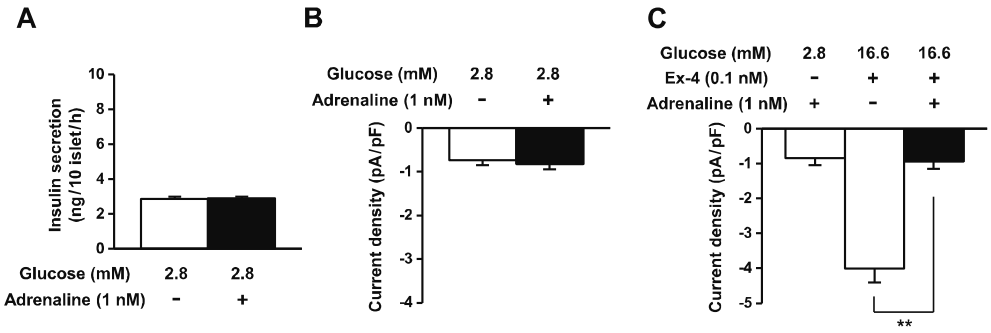
SUPPLEMENTARY DATA

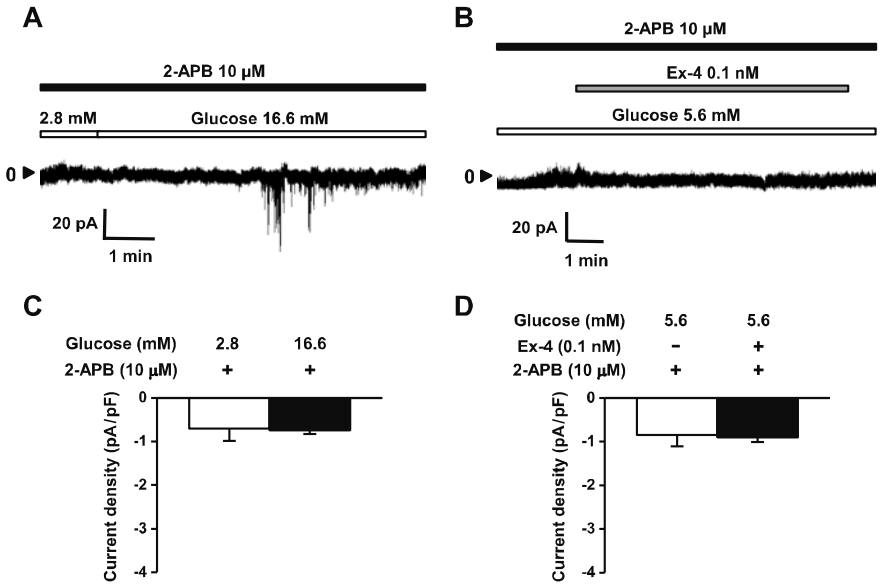
Supplementary Figure 1. Effects of low concentration of adrenaline on the basal insulin secretion in isolated islets, and NSCC current at 2.8 mmol/L glucose and 16.6 mmol/L glucose with Ex-4 in β-cells. (A) Basal insulin secretion from mouse isolated islets at 2.8 mmol/L glucose with or without 1 nmol/L adrenaline. (B) The NSCC current in mouse β-cells at 2.8 mmol/L glucose with or without 1 nmol/L adrenaline. (C) The NSCC current increase by Ex-4 (0.1 nmol/L) at 16.6 mmol/L glucose with or without 1 nmol/L adrenaline. (n = 5 cells). **P < 0.01.



SUPPLEMENTARY DATA

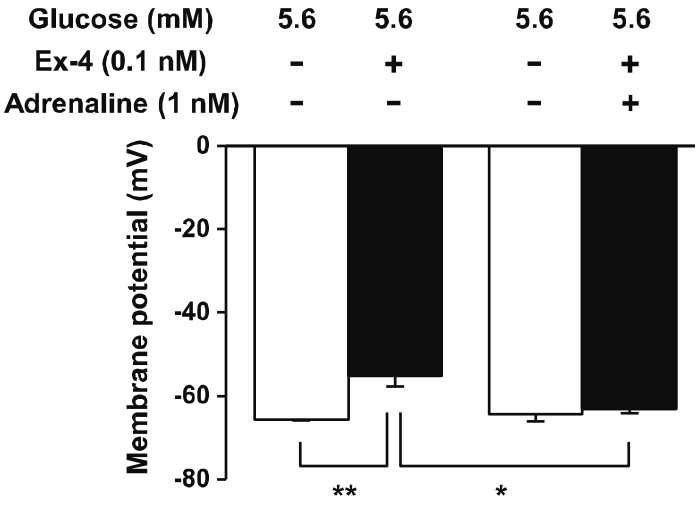
Supplementary Figure 2. TRP channel blocker (2-APB) inhibits NSCC-current increase stimulated by high glucose and Ex-4 in mouse β-cells.

In the presence of 10 μmol/L 2-APB, which is a non-selective TRP channel blocker, the NSCC current was not increased by exposure to a high glucose concentration at 16.6 mmol/L (A and C), and to 0.1 nmol/L Ex-4 (B and D). (n = 5 cells).



SUPPLEMENTARY DATA

Supplementary Figure 3. Effects of 1 nmol/L adrenaline on Ex-4-induced membrane depolarization in mouse β-cells.
The steady-state membrane potentials measured during 0.1 nmol/L Ex-4-induced action potential firings. (n = 7-8 cells). *P < 0.05 and **P < 0.01.



SUPPLEMENTARY DATA

Supplementary Figure 4. High concentrations of adrenaline induced hyperpolarization of the membrane that is depolarized by high glucose or Ex-4 in β-cells.
(A) The depolarized membrane in β-cells by exposure to 16.6 mmol/L glucose was hyperpolarized by 5 μmol/L adrenaline. (B) The depolarizing effect of 0.1 nmol/L Ex-4 on the membrane was reversed to the control level by 5 μmol/L adrenaline. (n = 5-6 cells). *P < 0.05 and **P < 0.01.