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Increased Activation of the TRESK K<sup>+</sup> Channel Mediates Vago-vagal Reflex Malfunction in Diabetic Rats

**Short Title:** Malfunction of vago-vagal reflex in diabetes

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**Abbreviations used in this paper:** 2PK<sup>+</sup>: two-pore-domain potassium channel; BB/W, BioBreeding/Worcester rat strain; [Ca<sup>2+</sup>]<sub>i</sub>, intracellular calcium ion concentration; cAMP, cyclic-3',5'-adenosine monophosphate; BK, large conductance, voltage, and calcium-activated potassium channel; CCK, cholecystokinin; CCKAR, CCK-A receptor; DRG, dorsal root ganglia; GFP, green fluorescent protein; NFAT, nuclear factor of activated T-cells; NG, nodose ganglia; R<sub>in</sub>, neuronal input resistance; sAHP, slow afterhyperpolarization; shRNA, short hairpin RNA; siRNA, small interfering RNA; SK, small-conductance, voltage, and calcium-activated potassium channel; STZ, streptozotocin; TALK, TWIK-related alkaline pH-activated potassium channel; TASK, acid-sensitive potassium channel; THIK, TWIK-related halothane-inhibited K<sup>+</sup> channel; TREK, TWIK-related potassium channel; TRESK, TWIK-related spinal cord potassium channel; TWIK, two-pore-domain weak inward-rectifying potassium channel; V<sub>m</sub>, resting membrane potential; VR1, vanilloid receptor 1

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**Conflicts of Interest:**

The authors declare no conflicts of interest.

**Author Contributions:**

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