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

Gintautas Grabauskas, Xiaoyin Wu, Il Song, Shi-Yi Zhou, Thomas Lanigan, Chung Owyang

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

Short Title: Malfunction of vago-vagal reflex in diabetes

Gintautas Grabauskas,[1] Xiaoyin Wu,[1] Il Song,[1] Shi-Yi Zhou,[1] Thomas Lanigan,[2] Chung Owyang[1]

[1]Division of Gastroenterology, Department of Internal Medicine
3912 Taubman Center, SPC 5362
1500 East Medical Center Drive, Ann Arbor, MI 48019

[2]Center for Gene Therapy, Department of Internal Medicine
3554 Medical Sciences Research Building II, SPC 5688
1150 West Medical Center Drive, Ann Arbor, MI 48109

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Abbreviations used in this paper: $2PK^+$: two-pore-domain potassium channel; BB/W, BioBreeding/Worcester rat strain; $[Ca^{2+}]_i$, 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_{in}, 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^+ channel; TREK, TWIK-related potassium channel; TRESK, TWIK-related spinal cord potassium channel; TWIK, two-pore-domain weak inward-rectifying potassium channel; V_m, resting membrane potential; VR1, vanilloid receptor 1

Correspondence:
Chung Owyang, MD
3912 Taubman Center, SPC 5362
1500 East Medical Center Drive
University of Michigan Health System
Ann Arbor, Michigan, 48109
E-mail: cowyang@med.umich.edu
Telephone: 734-936-4785
Fax: 734-936-7392

Conflicts of Interest:
The authors declare no conflicts of interest.

AuthorContributions: