Supplementary Information to the article:

Do Kv7.1 channels contribute to control of arterial vascular tone?

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Figure S1

Figure S1: Pretreatment of (-) fat murine (panel A) and (-) fat rat (panel B) mesenteric arteries with 10 μ M chromanol 293B [(+) chromanol 293B] and subsequent exposure to R-L3. (-) Chromanol 293B; control, non-treated vessels. n.s., $P \ge 0.05$. n=5 per group.



Figure S2

Figure S2: Relaxation of (-) fat murine renal artery rings by R-L3. Vessels were isolated from wild-type (*Kcnq1* +/+) mice. Arteries were precontracted by 1 μ M phenylephrine (PE) or KCl 60 mM. Tension is expressed as a percentage of KCl or PE induced contractions. *, *P* < 0.05. n=5 per group.



Figure S3: Relaxation of endothelium-denuded murine (panel A) and rat (panel B) mesenteric artery rings by R-L3. (-) Endothelium: (-) fat, endothelium-denuded vessels; (+) Endothelium: (-) fat, endothelium-intact vessels. Arteries were precontracted by 1 μ M phenylephrine (PE). Murine mesenteric arteries were isolated from $Kcnq1^{+/+}$ mice. Tension is expressed as a percentage of PE induced contractions. n.s., $P \ge 0.05$. n=5 per group.



Figure S4: Original traces showing relaxation induced by $K_v7.1$ opener ML277 in (-) fat murine mesenteric artery rings isolated from $Kcnq1^{+/+}$ (**panel A**) and $Kcnq1^{-/-}$ mice (**panel B**) preconstricted with 1 µM phenylephrine.



Figure S5

Figure S5: Dose response curves and EC₅₀ values for phenylephrine (PE)-induced contractions of (-) fat and (+) fat murine mesenteric artery rings isolated from $Kcnq1^{+/+}$ and $Kcnq1^{-/-}$ mice. Nonlinear regression model of dose-response curves to PE in the presence or absence of HMR1556 with or without fat (n=5 per group) (**panel A**) and their corresponding EC₅₀ values (**panel B**). Nonlinear regression model of dose-response curves to PE in $Kcnq1^{+/+}$ ((+) fat; (-) fat, n=7 per group) and $Kcnq1^{-/-}$ arteries ((+) fat; (-) fat, n=7 per group) (**panel C**) and their corresponding EC₅₀ values (**panel D**). *, P < 0.05.



Figure S6: Relative expression of Kcnq3-5 channels at RNA levels in (-) fat mesenteric arteries from $Kcnq1^{+/+}$ and $Kcnq1^{-/-}$ mice normalized to 18s. Relative mRNA levels for Kcnq3 (panel A) (n=6 for $Kcnq1^{+/+}$; n=5 for $Kcnq1^{-/-}$), Kcnq4 (panel B) (n=6 for $Kcnq1^{+/+}$; n=6 for $Kcnq1^{-/-}$) and Kcnq5 expression (panel C) (n=5 for $Kcnq1^{+/+}$; n=5 for $Kcnq1^{-/-}$). n.s., $P \ge 0.05$, unpaired t-test.



Figure S7

Figure S7. PCR genotyping of $Kcnq1^{+/+}$ and $Kcnq1^{-/-}$ mice. Amplification by using the forward and reverse primers gives a 240-bp product specific to the wild-type (+/+) allele. Amplification by using the Neo forward and reverse primers gives a 370-bp product specific to the null allele (-/-) (Casimiro et al., 2001).

Video S8. Demonstration of shaker-waltzer phenotype (hyperactivity, head shaking, and/or circling), due to abnormality of the vestibular apparatus in $Kcnq1^{-/-}$ mice. The video shows the typical behavior of lack of K_V7.1 function in the mouse.

Reference:

Casimiro, M.C., Knollmann, B.C., Ebert, S.N., Vary, J.C., Greene, A.E., Franz, M.R., et al. (2001). Targeted disruption of the Kcnq1 gene produces a mouse model of Jervell and Lange-Nielsen Syndrome. Proc. Natl. Acad. Sci. U. S. A. *98*: 2526–31.