

Figure S1. Effect of K channel inhibition with TEA on hypoxia- and adenosine-induced dilatation in pig coronary artery.

(A) Concentration-response curves for O₂ lowering in coronary arterial segments without endothelium contracted with $PGF_{2\alpha}$ (10 µM) in the absence and presence of the K channel inhibitor TEA (1 mM and 10 mM) (n=7, *P=0.046, ***P<0.001 by two way ANOVA). (B) Concentration-response curves for adenosine in coronary arterial segments without endothelium contracted with $PGF_{2\alpha}$ (10 µM) in the absence and presence of the K channel inhibitor TEA (1 mM and 10 mM) (n=7, ***P<0.001 by two way ANOVA).



Figure S2. Effect of K channel inhibition on hypoxia-induced dilatation in pig coronary artery.

Concentration-response curves for O₂ lowering in coronary arterial segments without endothelium contracted with PGF_{2α} (10 μ M) in the absence and presence of **(A)**: 4-AP 0.5 mM (n=12, * P<0.001 by two way ANOVA), **(B)**: IbTX 100 nM (n=6, * P<0.001 by two way ANOVA) or **(C)**: glibenclamide 3 μ M (n=7, * P<0.05 by two way ANOVA).



Figure S3. Effect of K channel inhibition on hypoxia-induced dilatation in pig coronary artery.

Concentration-response curves for O₂ lowering in coronary arterial segments without endothelium contracted with PGF_{2α} (10 μ M) in the absence and presence of (A): 4-AP 0.5 mM and IbTX 100 nM (n=6, * P<0.01 by two way ANOVA), (B): Glibenclamide 3 μ M and 4-AP 0.5 mM (n=6, * P<0.001 by two way ANOVA)



Figure S4.

Effect of K channel inhibition on hypoxia-induced dilatation in pig coronary artery.

Concentration-response curves for O_2 lowering in coronary arterial segments without endothelium contracted with PGF_{2α} (10 μ M) in the absence and presence of XE991 10 μ M, 4-AP 0.5 mM and IbTX 100 nM (n=7-8, *** P<0.001 by two way ANOVA),



Figure S5. Effect of K_v7.1 channel inhibition on hypoxia- and adenosine-induced dilatation in pig coronary artery.

(A) Concentration-response curves for O₂ lowering in coronary arterial segments without endothelium contracted with PGF_{2α} (10 μ M) in the absence and presence of the K_V7.1 channel inhibitor Chromanol 293B (10 μ M) (n=5, P=0.22 by two way ANOVA). (B) Concentration-response curves for adenosine in coronary arterial segments without endothelium contracted with PGF_{2α} (10 μ M) in the absence and presence of the K_V7.1 channel inhibitor Chromanol 293B (10 μ M) in the absence of the K_V7.1 channel inhibitor Chromanol 293B (10 μ M) in the absence and presence of the K_V7.1 channel inhibitor Chromanol 293B (10 μ M) in the absence and presence of the K_V7.1 channel inhibitor Chromanol 293B (10 μ M) (n=5, P=0.45 by two way ANOVA).



Figure S6. Detection of BK_{C} and $K_{V}7$ K channels by immunoblotting.

Immunoblot with samples from a normoxic artery in lane 1 and from a hypoxic artery in lane 2 and showing the presence of (A) $BK_{Ca}\alpha$ located at 100 kDa (B) $BK_{Ca}\beta$ located at 30 kDa (C) $K_V7.4$ located at 77 kDa and (D) $K_V7.5$ located at 100 kDa.(E) incubated with secondary antibody goat anti-rabbit IgG conjugated to HRP.