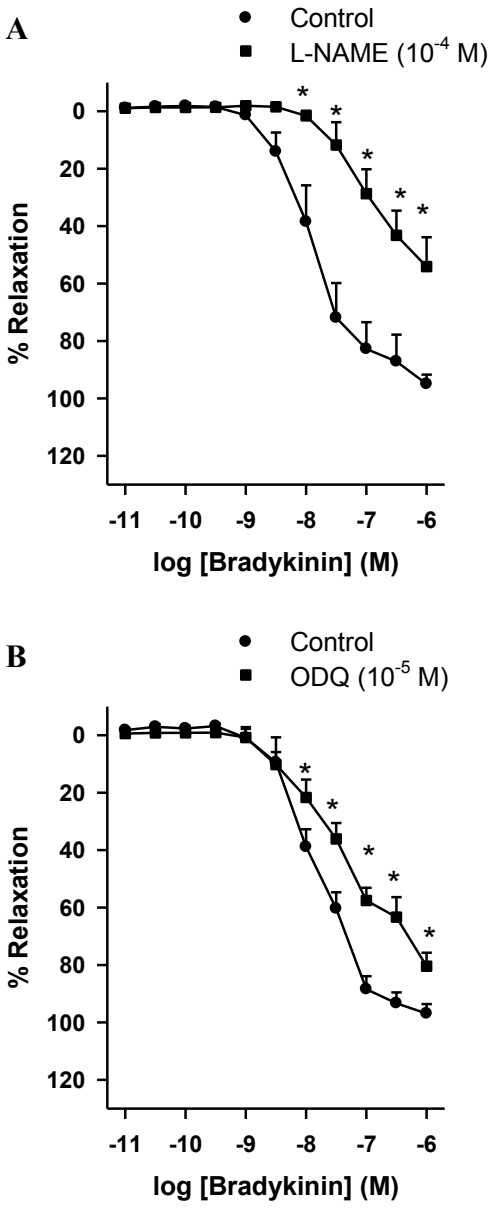
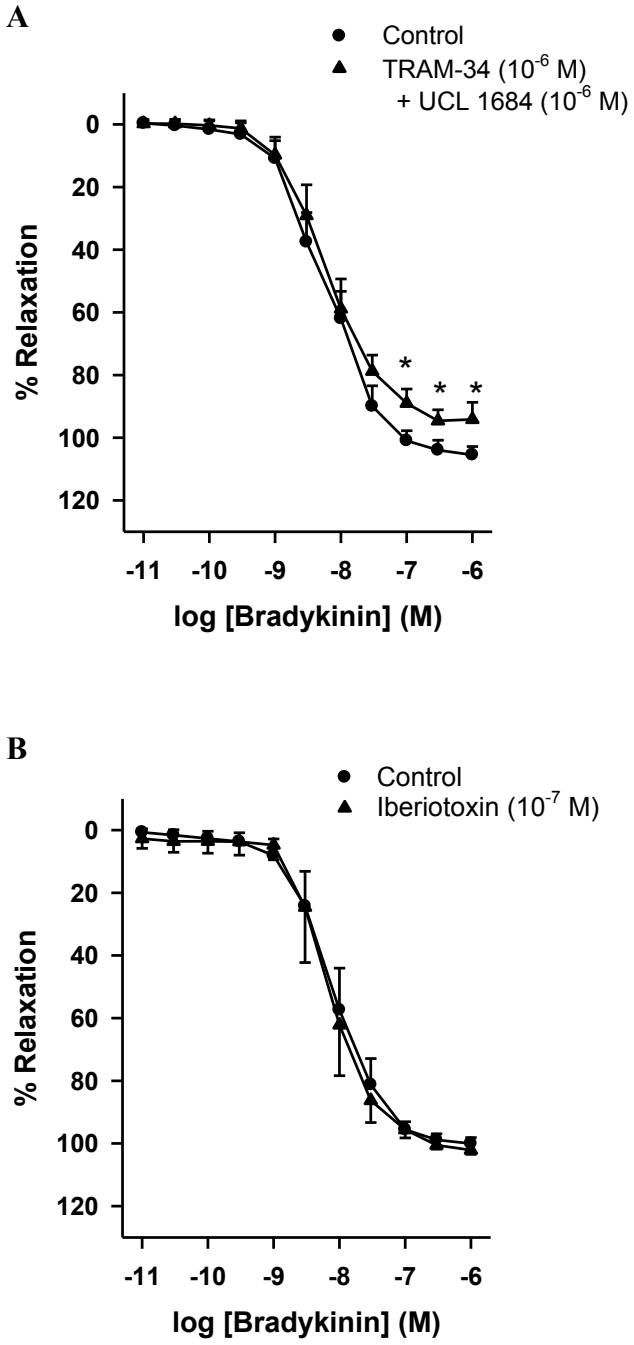
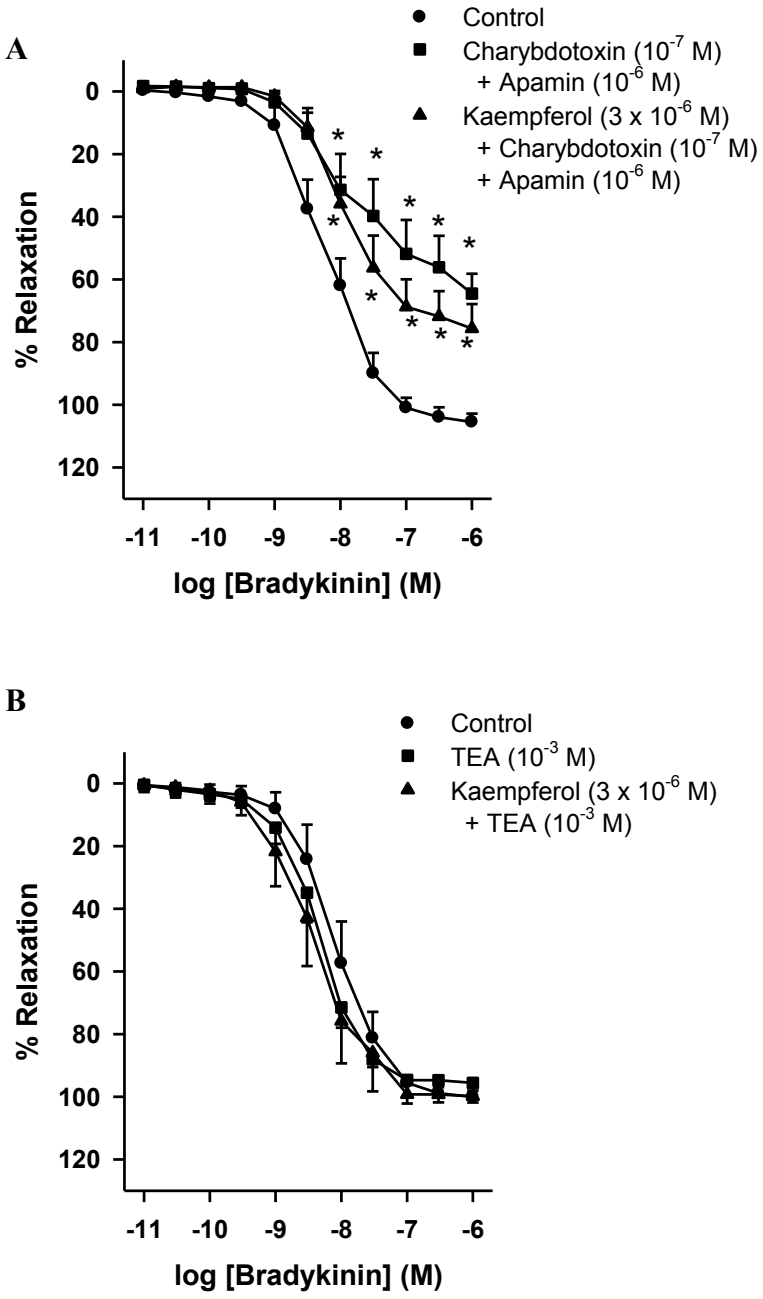
Supplementary Figure 1



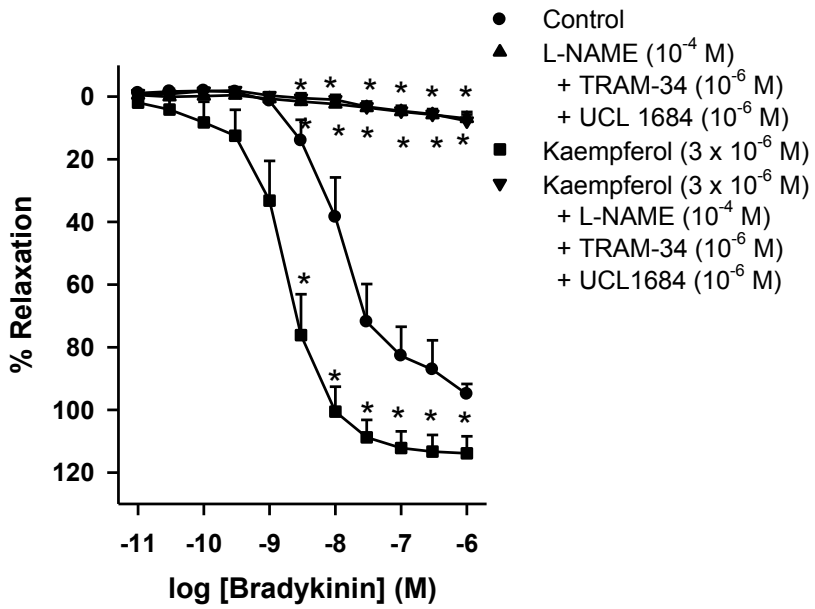
Supplementary Figure 2



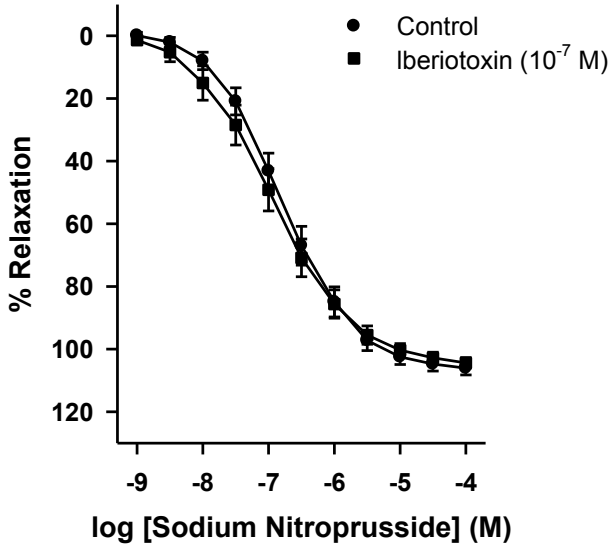
Supplementary Figure 3



Supplementary Figure 4



Supplementary Figure 5



Supplementary Figure 1. Effect of (A) nitric oxide synthase inhibitor or (B) guanylyl cyclase inhibitor on bradykinin-induced relaxations. Rings from porcine coronary arteries were incubated with either L-NAME (10^{-4} M) or ODQ (10^{-5} M) for 30 minutes. Rings were then contracted with U46619 (3×10^{-8} M) before bradykinin was cumulatively added. n = 6-7. *P<0.05 when compared to the control group.

Supplementary Figure 2. Effect of (A) intermediate plus small conductance calcium-activated potassium channels inhibitors or (B) large conductance calcium-activated potassium channel inhibitor on bradykinin-induced relaxations. Rings from porcine coronary arteries were incubated with TRAM-34 (10^{-6} M) plus UCL 1684 (10^{-6} M) or iberiotoxin (10^{-7} M) for 30 minutes. Rings were then contracted with U46619 (3×10^{-8} M) before bradykinin was cumulatively added. n = 7-8. *P<0.05 when compared to the control group.

Supplementary Figure 3. Effect of (A) large and intermediate plus small conductance calcium-activated potassium channels inhibitors and (B) non-selective calcium-activated potassium channel inhibitor on bradykinin-induced relaxations in the absence or presence of kaempferol. Rings from porcine coronary arteries were incubated with charybdotoxin (10^{-7} M) plus apamin (10^{-6} M), TEA (10^{-3} M), with or without kaempferol (3×10^{-6} M) for 30 minutes. Rings were then contracted with U46619 (3×10^{-8} M) before bradykinin was cumulatively added. n = 7-8. *P<0.05 when compared to the control group.

Supplementary Figure 4. Effect of the combination of nitric oxide synthase and intermediate plus small conductance calcium-activated potassium channels inhibitors on bradykinin-induced relaxations in the absence or presence of kaempferol. Rings from porcine coronary arteries were incubated with L-NAME (10^{-4} M), TRAM-34 (10^{-6} M) plus UCL 1684 (10^{-6} M) and/or kaempferol (3×10^{-6} M) for 30 minutes. Rings were then contracted with U46619 (3×10^{-8} M) before bradykinin was cumulatively added. n = 7-8. *$P<0.05$ when compared to the control group.

Supplementary Figure 5. Effect of large conductance calcium-activated potassium channel inhibitor on sodium nitroprusside-induced relaxations. Rings from porcine coronary arteries were incubated with or without iberiotoxin (10^{-7} M) for 30 minutes. Rings were then contracted with U46619 (3×10^{-8} M) before sodium nitroprusside was cumulatively added. n = 6-7.

Supplementary Table 1. Effects of acute treatment with different flavonoids on the contraction to U46619 (3×10^{-8} M) before relaxations to bradykinin (10^{-11} to 10^{-6} M) in porcine coronary arteries.

Treatment	Contraction (g)
Control	6.8 ± 0.4
Ethanol (0.1 %)	6.2 ± 0.5
Kaempferol (3×10^{-6} M)	6.8 ± 0.8
Kaempferol (10^{-5} M)	4.8 ± 0.3
Apigenin (10^{-5} M)	5.2 ± 0.5
Myricetin (10^{-5} M)	8.9 ± 0.8
Quercetin (10^{-5} M)	5.9 ± 1.0
Rutin (10^{-5} M)	7.7 ± 0.7

n=4-8 in each group.

Supplementary Table 2. Effects of different pharmacological treatments, in the absence or presence of kaempferol (3 x 10^{-6} M), on the contraction to U46619 (3 x 10^{-8} M) before relaxations to bradykinin (10^{-11} to 10^{-6} M) in porcine coronary arteries.

	Contraction (g)	
Treatment	Vehicle (Ethanol, 0.1%)	Kaempferol (3 x 10^{-6} M)
Control	6.5 ± 0.7	7.0 ± 0.9
L-NAME (10^{-4} M)	8.7 ± 0.3	8.6 ± 0.7
ODQ (10^{-5} M)	8.0 ± 0.3	7.8 ± 0.3
TRAM-34 (10^{-6} M) + UCL 1684 (10^{-6} M)	5.6 ± 0.2	5.4 ± 0.5
Iberiotoxin (10^{-7} M)	7.6 ± 1.0	6.6 ± 1.0
Charybdotoxin (10^{-7} M) + Apamin (10^{-6} M)	10.2 ± 0.7	6.4 ± 0.4
TEA (10^{-3} M)	10.1 ± 1.1	8.2 ± 1.0

n=6–8 in each group.

Apamin, inhibitor of small-conductance calcium-activated potassium channels ($K_{Ca}2.3$); charybdotoxin, inhibitor of large- ($K_{Ca}1.1$) and intermediate-conductance ($K_{Ca}3.1$) calcium-activated potassium channels; iberiotoxin, inhibitor of $K_{Ca}1.1$; L-NAME, inhibitor of nitric oxide synthase; ODQ, inhibitor of soluble gunaylyl cyclase; TEA, non-selective inhibitor of calcium-activated potassium channels; TRAM-34, inhibitor of $K_{Ca}3.1$; UCL 1684, inhibitor of $K_{Ca}2.3$

Supplementary Table 3. Effects of different potassium channel blockers, in the absence or presence of kaempferol (3×10^{-6} M), on the EC_{50} and E_{max} values of concentration–relaxation curves of bradykinin (10^{-11} to 10^{-6} M) in porcine coronary arteries contracted by U46619 (3×10^{-8} M).

	EC_{50} (log M)		E_{max} (%)	
Treatment	Vehicle (Ethanol, 0.1%)	Kaempferol (3×10^{-6} M)	Vehicle (Ethanol, 0.1%)	Kaempferol (3×10^{-6} M)
Control	-8.3 ± 0.1	-8.9 ± 0.1*	105 ± 3.0	106 ± 2.2
Charybdotoxin (10^{-7} M) + Apamin (10^{-6} M)	-7.3 ± 0.5	-7.8 ± 0.2	76 ± 8.0*	85 ± 4.1*
TEA (10^{-3} M)	-8.3 ± 0.1	-8.4 ± 0.2	95 ± 1.4	101 ± 2.5

n=7–8 in each group. * $p<0.05$ vs. control group

Apamin, inhibitor of small-conductance calcium-activated potassium channels; **c**harybdotoxin, inhibitor of large- and intermediate-conductance calcium-activated potassium channels; TEA, non-selective inhibitor of calcium-activated potassium channels

Supplementary Table 4. Effects of large-conductance calcium-activated potassium channel blocker, in the absence or presence of kaempferol (3×10^{-6} M), on the contraction to U46619 (3×10^{-8} M) before relaxations to sodium nitroprusside (10^{-9} to 10^{-4} M) in porcine coronary arteries.

	Contraction (g)	
Treatment	Vehicle (Ethanol, 0.1%)	Kaempferol (3×10^{-6} M)
Control	8.1 ± 0.2	6.2 ± 0.5
Iberiotoxin (10^{-7} M)	8.0 ± 1.2	7.7 ± 1.0

n=5–6 in each group.

Iberiotoxin, inhibitor of large-conductance calcium-activated potassium channels