Endothelium-dependent vasodilatory effect of (kaempferol 7-O-α-L-rhamnopyranoside) via NO-cgmp-PKG signaling

C.O. Tettey1, Yang In-Jun2 and Shin Heung-Mook2,3

1Department of Biomedical Sciences, University of Health and Allied Sciences, PMB 31. Ho, Ghana; 2Department of Physiology, College of Korean Medicine, Dongguk University, Gyeongju, South Korea; 3National Development Institute of Korean Medicine, Gyeongsan, Gyeongbuk 712-210, South Korea.

This study investigated the pharmacological effect of KR on vascular relaxation and its underlying biochemical mechanisms in endothelial cells and rat dorsal aorta. Treatment of phenylephrine (PE)-pre-contracted aortic strips with KR induced endothelium-dependent relaxation, which was attenuated by NG-nitro-L-arginine methyl ester (L-NAME), (nitric oxide synthase (NOS) inhibitor). Further studies using HUVECs indicated that eNOS phosphorylation was increased after exposure to KR. Pre-treatment of aortic strips with the cyclic GMP (cGMP) inhibitors; methylene blue (MB; 10-5 M) and 1-H-[1,2,4]-oxadiazole-[4,3-α]-quinoxalin-10-one, (ODQ; 10-6 M) suppressed the KR-induced vasodilation. Furthermore, KR also increased protein kinase G (PKG) levels whereas it suppressed levels of phosphorylated myosin light chain (MLC) and protein kinase C (PKC) in aortic strips. These results suggest that KR induces endothelium-dependent vasorelaxation via the NO-cGMP-PKG pathway.