Title	1,25-Dihydroxyvitamin D3 acutely reduces acetylcholine-induced endothelium-dependent contraction in hypertensive rat aorta through activation of protein kinase C
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1,25-DIHYDROXYVITAMIN D3 ACUTELY REDUCES ACETYLCHOLINE-INDUCED ENDOTHELIUM-DEPENDENT CONTRACTION IN HYPERTENSIVE RAT AORTA THROUGH ACTIVATION OF PROTEIN KINASE C

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Vitamin D derivatives affect the regulation of cardiovascular system. Recent study from our lab indicated that 1,25-dihydroxyvitamin D3, the major metabolite of vitamin D, acutely reduced endothelium-dependent contraction to acetylcholine in spontaneously hypertensive rats (SHR) aorta. This reduction was associated with reduced cytosolic-free calcium in endothelial cells. In the other cell types, vitamin D caused calcium depletion from intracellular calcium store and inhibited membrane calcium channel activity via protein kinase C (PKC) activation. The present experiment examined whether or not PKC was involved in the inhibition of endothelium-dependent contraction by 1,25-dihydroxyvitamin D3. Aortic rings from male SHR aged 36-40 weeks were mounted on organ chamber for isometric force measurements. All experiments were performed in the presence of Nω-nitro-L-arginine methyl ester (L-NAME; 100 μM) for the study of endothelium-dependent contraction. Both 1,25-dihydroxyvitamin D3 (100 nM) and PKC inhibitor, GF 109203X (5 μM), remarkably inhibited acetylcholine-induced endothelium-dependent contraction. In the presence of GF 109203X, the inhibitory effect of 1,25-dihydroxyvitamin D3 was not observed. Our finding suggests that 1,25-dihydroxyvitamin D3 activated PKC to suppress endothelium-dependent contraction. The acute inhibitory effect by 30 minutes incubation of 1,25-dihydroxyvitamin D3 unlikely modulate gene expression that is typical of a classic nuclear vitamin D receptor. Hence further experiments are planned to investigate whether or not 1,25-dihydroxyvitamin D3 induces the translocation of vitamin D receptor from nucleus to plasma membrane for its acute vascular effects