EFFECT OF ISOFLAVONES ON THE VASCULAR ACTIONS OF PHOSPHODIESTERASE INHIBITORS

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Isoflavones are present in high concentrations in soy products, the intake of which is associated with lower incidence of cardiovascular diseases. They can enhance contraction and inhibit relaxation through cyclic adenosine monophosphate (cAMP)-dependent pathway. The present study aimed to examine whether or not the major soy isoflavones, genistein, daidzein and glycetein, interfere with the action of phosphodiesterase (PDE) inhibitors on the vasculature. Porcine coronary arteries were isolated and suspended in organ chambers for the measurement of isometric tension. Both rolipram (cAMP-selective PDE4 inhibitor) and zaprinast (cyclic guanosine monophosphate (cGMP)-selective PDE5 inhibitor) concentration-dependently relaxed porcine coronary artery rings that were contracted with U46619 (stable thromboxane A2 analogue, 30 nM). L-NAME (nitric oxide synthase inhibitor, 300 μM), but not indomethacin (cyclooxygenase inhibitor, 10 μM), inhibited rolipram- and zaprinast-induced relaxations. Genistein (3 μM), daidzein (3 μM) and glycetein 10 μM) did not affect zaprinast-induced relaxations in the absence or presence of LNAME or indomethacin. Relaxations to rolipram were also not affected by genistein and daidzein, while glycetein significantly inhibited rolipram-induced relaxation. This inhibitory effect of glycetein was not affected by L-NAME or indomethacin. The present findings suggest that the basal release of nitric oxide contributes significantly to rolipram- and zaprinast-induced relaxation. While genistein and daidzein do not affect the vascular actions of these PDE inhibitors, glycetein inhibits relaxation to rolipram through a pathway that is independent of nitric oxide synthase activity.