D-myo-Inositol 1,2,6-triphosphate blocks neuropeptide Y-induced facilitation of noradrenaline-evoked vasoconstriction of the mesenteric bed

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Abstract

Perfusion of the rat mesenteric bed with 0.1 or 10 nM neuropeptide Y potentiated the noradrenaline-induced increase in mesenteric pressure; the peptide did not modify basal perfusion pressure. While perfusion with 0.1 nM neuropeptide Y significantly increased the maximal noradrenaline-evoked vasoconstriction without modifying its EC_{50}, 10 nM neuropeptide Y potentiated the maximal noradrenaline effect and significantly shifted its concentration-response curve to the left. Perfusion with 1–10 μM D-myo-inositol 1,2,6-trisphosphate (α-trinositol) reduced, in a concentration-dependent fashion, the neuropeptide Y-induced potentiation of the noradrenaline-evoked vasoconstriction without altering the potency or maximal response evoked by the catecholamine alone. Perfusion with 0.1 nM neuropeptide Y plus 1 μM α-trinositol completely abolished the neuropeptide Y-induced facilitation of the noradrenaline effect. α-Trinositol 1 μM in the presence of 10 nM neuropeptide Y caused a nonparallel rightward shift of the noradrenaline concentration-response curve as compared to that obtained in the presence of 10 nM neuropeptide Y alone. The α-trinositol blockade of the facilitatory action of neuropeptide Y was reversible.