Stimulatory effect of paeoniflorin on the release of noradrenaline from ileal synaptosomes of guinea-pig in-vitro

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Abstract

The effect of paeoniflorin (an active principle of Paeoniae Radix, commonly used in traditional Chinese medicine) on the release of noradrenaline (norepineprhine) from nerve terminals was investigated using guinea-pig isolated ileal synaptosomes. Release was determined as the amount of noradrenaline, quantified by high-performance liquid chromatography-electrochemical detection, from samples incubated with paeoniflorin or vehicle. Paeoniflorin stimulated the release of noradrenaline in a concentration-dependent manner without an effect on the level of lactate dehydrogenase in the bathing medium. Tetrodotoxin abolished the action of paeoniflorin at concentrations sufficient to block sodium channels. The depolarizing effect of paeoniflorin on the membrane potential was also illustrated by a concentration-dependent increase in the fluorescence of bisoxonol. Moreover, the effect of paeoniflorin on bisoxonol fluorescence in ileal synaptosomes seems more potent than that of 4-aminopyridine. That paeoniflorin causes influx of calcium ions via the depolarization of nerve terminals could be considered. The noradrenaline-releasing action of paeoniflorin was abolished by removal of calcium chloride from the bathing medium. This action of paeoniflorin was also attenuated by Rp-cAMP atconcentrations sufficientto inhibitthe action of cyclicAMP. Therefore, paeoniflorin could induce a calcium-dependent and cyclic-AMP-related release of noradrenaline from sympathetic nerve terminals of guinea-pig ileum. Guanethidine inhibited the noradrenaline-releasing action of paeoniflorin in a concentration-dependent manner. The effect of paeoniflorin on the increase of bisoxonol fluorescence was not modified by atropine. Release of noradrenaline by paeoniflorin from noradrenergic nerve terminals was characterized. These findings suggest that paeoniflorin can stimulate tetrodotoxin-sensitive depolarization of membranes to result in a calcium-dependent and cyclic-AMP-related release of noradrenaline from noradrenergic nerve terminals.