Effect of butylidenephthalide on calcium mobilization in isolated rat aorta.

許準榕
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Abstract

Butylidenephthalide (Bdph), an antispasmodic compound originally isolated from the rhizome of Ligusticum chuaxiong, has a selective anti-anginal effect without changing blood pressure. Experiments have been performed to determine the mechanism of this action. Synthetic Z-butylidenephthalide concentration-dependently relaxed phenylephrine (1 microM)- or KCl (60 mM)-induced precontractions of intact and denuded rat aorta rings. The relaxation induced by Bdph was endothelium-independent. Bdph (30-300 microM) concentration-dependently reduced cumulative phenylephrine- and KCl-induced contractions of intact rat aortic rings and non-competitively inhibited their log concentration-response curves. The pD2' values of Bdph for phenylephrine- and KCl-induced contraction were 3.66+/-0.13 (n = 8) and 3.71+/-0.07 (n = 8), respectively, which were not significantly different from each other. Bdph also concentration-dependently reduced cumulative Ca2+-induced contractions of intact rat aortic rings in high-KCl (60 mM) Ca2+-free physiological salt solution and non-competitively inhibited its log concentration-response curve. The pD2' value of Bdph for the Ca2+-induced contractions was 3.21+/-0.01 (n = 7) which was significantly different from the pD2' value obtained from the cumulative KCl-induced contractions. These results suggest that Bdph inhibits calcium release from calcium stores more selectively than calcium influx from extracellular space via voltage-dependent calcium channels. The inhibition by Bdph of calcium release from KCl-sensitive calcium stores might be similar to its inhibition of calcium release from phenylephrine-sensitive calcium stores. However, because phenylephrine generates inositol-1,4,5-trisphosphate (IP3) whereas KCl does not, the inhibitory effect of Bdph might not be related to IP3 production.