• 系統編號	RB8301-1131
• 計畫中文名稱	Butylidenephthalide 對大白鼠主動脈鈣移動的作用
• 計畫英文名稱	The Effects of Butylidenephthalide on Calcium Mobilization in Isolated Rat Aorta.
• 主管機關	行政院國家科學委員會
• 計畫編號	NSC82-0412-B038-004
• 執行機構	台北醫學院藥理學科
• 本期期間	8108 ~ 8207
• 報告頁數	0 頁
• 使用語言	中文；英文
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• 中文關鍵字	亞丁基鄰苯二甲內酯；大白鼠主動脈；鈣移動
• 英文關鍵字	Butylidenephthalide；Rat aorta；Calcium mobilization
• 中文摘要	Butylidenephthalide(Bdph)是中國川芎的精油主成分,目前已被證實具有非專一性抗痙攣作用。在正常含鈣溶液,Bdph 對於外加 KCl 或 phenylephrine(Phe)引起大白鼠離體主動脈環的累積性收縮,呈非競爭性的抑制作用。Bdph 抑制由 KCl 或 Phe 引起的瞬間收縮,求得 pD'/sub 2/ 值分別爲 3.71.plmin.0.07(n=8)及 3.66.plmin.0.13(n=8),兩組數據比較,在統計上沒有意義的差別。另外,Bdph 對於由 KCl(60mM)或 Phe(1.mu.M)引起的持續性收縮,所產生的鬆弛作用與血管內皮細胞無關,其對抗 KCl 或 Phe 之-logIC/sub 50/值分別爲 3.90.plmin.0.09(n=8)及 3.66.plmin.0.14(n=8),二組數據在統計上沒有意義的差別。說明 Bdph 可能爲非選擇性的抑制依賴電位及受體之鈣離子通道。 同樣的,我們將 Bdph 抑制 KCl 或 Phe 引起的瞬間收縮及持續性收縮而得到的 pD'/sub 2/值及-logIC/sub 50/值,兩相比較,在統計上沒有意義的差別。Bdph 對於由 KCl 或 Phe 所引起收縮的反應,除了降低其張力以外,同時也減少肌細胞內鈣離子量。在高鉀(60mM)無鈣溶液,Bdph 爲非競爭性的抑制外加鈣離子引起累積性收縮,以上結果說明 Bdph 非競爭性的抑制鈣離子主、被動流入。 不過,Bdph(300.mu.M)及 Verapamil(10.mu.M)並不影響 Caffeine(30mM)引起血管的瞬間收縮和肌細胞內鈣離子的增加。此外, Bdph(100 or 300.mu.M)在無鈣溶液,對於 Phe(1.mu.M)引起血管的瞬間收縮也沒有影響。說明 Bdph 不影響肌細胞內鈣貯藏器之鈣離子釋放。
• 英文摘要	Butylidenephthalide (Bdph) has been proved to occur in Ligusticum Wallichii FRANCH., and to be a non-specific non-competitively inhibited contractions of isolated rat aortic rings induced by cumulatively adding KCl or phenylephrine (Phe). The pD'/sub 2/ value of Bdph to phasic contraction (PC) by KCl or Phe was 3.71.plmin.0.07 (n=8) or 3.66.plmin.0.13 (n=8), respectively. The relaxant effect of Bdph on tonic contraction (TC) by KCl (60mM) or Phe (1.mu.M) was endothelium independent, and the -logIC/sub 50/ value was 3.90.plmin.0.09 (n=8) or 3.66.plmin.0.14

(n=8), respectively. There was no significant difference between these two pD'/sub 2/values, nor two-logIC/sub 50/ values. It suggests that Bdph non-selectively blocks voltage dependent nor receptor operated calcium channel. There was no significant difference between pD'/sub 2/ and -logIC/sub 50/values to KCl, nor to Phe. The intracellular cytoplasmic calcium concentration ([Ca/sup 2+/]/sub i/) as well as TC to KCl or Phe, was also reduced by Bdph simultaneously. In high K/sup +/ (60mM) calcium free medium, Bdph non-competitively inhibited cumulative exogenous Ca/sup 2+/-induced contractions. It suggests that Bdph non-selectively inhibits active nor passive calcium influx. However, Bdph (300.mu.M) as well as verapamil (10.mu.M) did not affect caffeine (30mM)-induced transient increase of [Ca/sup 2+/]/sub i/ and tension. In addition, Bdph (100-300.mu.M) did not affect Phe (1.mu.M)-induced transient contraction in Ca/sup 2+/-free medium containing EGTA. It suggests that Bdph does not affect the Ca/sup 2+/ release from sarcoplasmic reticulum or other calcium stores.