The antiplatelet activity of rutaecarpine, an alkaloid isolated from Evodia rutaecarpa, is mediated through inhibition of phospholipase C.

林建煌;許準榕

Sheu JR;Kan YC;Hung WC;Su CH;Lin CH;Lee YM and Yen MH

Abstract

In this study, the mechanism involved in the anti- affect nitrate production in collagen (10 mg/ml)-platelet activity of rutaecarpine in human platelet induced human platelet aggregation. On the othersuspensions was investigated. In platelet suspen- hand, various concentrations of rutaecarpine (50,sions (4.53108/ml), rutaecarpine (100 and 200 mM) 100, and 200 mM) dose-dependently inhibited did not influence the binding of FITC-triflavin to [3H]inositolmonophosphate formation stimulated platelet glycoprotein IIb/IIIa complex. Addition- by collagen (10 mg/ml) in [3H]myoinositol-loaded ally, rutaecarpine (200 mM) did not significantly platelets at different incubation times (1, 2, 3, and change the fluorescence of platelet membrane la- 5minutes). It is concluded that the antiplatelet activbeled with diphenylhexatriene (DPH). On the ity of rutaecarpine may possibly be due to the inhiother
hand, rutaecarpine (50 and 100 mM) dose- bition of phospholipase C activity, leading to redependently inhibited the increase in intracellular duce phosphoinositide breakdown, followed by the free Ca21 of Fura 2-AM loaded platelets stimulated inhibition of thromboxane A2 formation, and then by collagen. Moreover, rutaecarpine (100 and 200 inhibition of [Ca21]imobilization of platelet aggremM) did not significantly affect the thromboxane gation stimulated by agonists. n of TxA2 formation, and finally inhibition of [Ca2+]imobilization and ATP-release.