Mechanism of inhibition of platelet aggregation by rutaecarpine, an alkaloid isolated from evodia rutaecarpa. Eur.

許準榕
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Abstract

In this study, rutaecarpine was tested for its antiplatelet activities in human platelet-rich plasma. In human platelet-rich plasma, rutaecarpine (40-200 microM) inhibited aggregation stimulated by a variety of agonists (i.e., collagen, ADP, adrenaline and arachidonic acid). The antiplatelet activity of rutaecarpine (120 microM) was not significantly attenuated by pretreatment with the nitric oxide synthase inhibitor N(G)-mono-methyl-L-arginine (L-NMMA) (100 microM) or N(G)-nitro-L-arginine methyl ester (L-NAME) (200 microM) and with the guanylyl cyclase inhibitor methylene blue (100 microM). In addition, rutaecarpine (40-200 microM) did not significantly affect cyclic AMP and cyclic GMP levels in human washed platelets, whereas it significantly inhibited thromboxane B2 formation stimulated by collagen (10 microg/ml) and thrombin (0.1 U/ml). Furthermore, rutaecarpine (40-200 microM) inhibited [3H]inositol monophosphate formation stimulated by collagen and thrombin in [3H]myoinositol-loaded platelets. It is concluded that the antiplatelet effects of rutaecarpine are due to inhibition of thromboxane formation and phosphoinositide breakdown.