Antiplatelet activity of caffeic acid phenethyl ester is mediated through a cyclic GMP-dependent pathway in human platelets.

陳廷貴

Chen TG;Lee JJ;Lin KH;Shen CH;Chou DS;Sheu JR

摘要

Abstract

The aim of this study was to examine the inhibitory mechanisms of caffeic acid phenethyl ester (CAPE), which is derived from the propolis of honeybee, in platelet activation. In this study, CAPE (15 and 25 microM) markedly inhibited platelet aggregation stimulated by collagen (2 microg/ml). CAPE (15 and 25 microM) increased cyclic GMP level, and cyclic GMP-induced vasodilator-stimulated phosphoprotein (VASP) Ser157 phosphorylation, but did not increase cyclic AMP in washed human platelets. Rapid phosphorylation of a platelet protein of Mw. 47,000 (P47), a marker of protein kinase C activation, was triggered by phorbol-12, 13-dibutyrate (150 nM). This phosphorylation was markedly inhibited by CAPE (15 and 25 microM). The present study reports a novel and potent antiplatelet agent, CAPE, which involved in the following inhibitory pathways: CAPE increases cyclic GMP/VASP Ser157 phosphorylation, and subsequently inhibits protein kinase C activity, resulting in inhibition of P47 phosphorylation, which ultimately inhibits platelet aggregation. These results strongly indicate that CAPE appears to represent a novel and potent antiplatelet agent for treatment of arterial thromboembolism.