

B2 rC-Phycocyanin, a Very Potent and Novel Platelet Aggregation Inhibitor from *Spirulina platensis*.

林建煌;許準榕

Hsiao G;Chou PH;Shen MY;Chou DS;Lin CH;Sheu JR

Abstract

The aim of this study was to systematically examine the inhibitory mechanisms of C-phycocyanin (C-PC), one of the major phycobiliproteins of *Spirulina platensis* (a blue-green alga), in platelet activation. In this study, C-PC concentration-dependently (0.5-10 nM) inhibited platelet aggregation stimulated by agonists. C-PC (4 and 8 nM) inhibited intracellular Ca^{2+} mobilization and thromboxane A_2 formation but not phosphoinositide breakdown stimulated by collagen (1 μ g/mL) in human platelets. In addition, C-PC (4 and 8 nM) markedly increased levels of cyclic GMP and cyclic GMP-induced vasodilator-stimulated phosphoprotein (VASP) Ser₁₅₇ phosphorylation. Rapid phosphorylation of a platelet protein of M_w 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 C-PC (4 and 8 nM). In addition, C-PC (4 and 8 nM) markedly reduced the electron spin resonance (ESR) signal intensity of hydroxyl radicals in collagen (1 μ g/mL)-activated platelets. The present study reports on a novel and very potent (in nanomolar concentrations) antiplatelet agent, C-PC, which is involved in the following inhibitory pathways: (1) C-phycocyanin increases cyclic GMP/VASP Ser₁₅₇ phosphorylation and subsequently inhibits protein kinase C activity, resulting in inhibition of both P47 phosphorylation and intracellular Ca^{2+} mobilization, and (2) C-PC may inhibit free radicals (such as hydroxyl radicals) released from activated platelets, which ultimately inhibits platelet aggregation. These results strongly indicate that C-PC appears to represent a novel and potential antiplatelet agent for treatment of arterial thromboembolism.