Inhibitory mechanisms of naloxone on human platelets.

許準榕
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Abstract

1. In the present study, naloxone was tested for its antiplatelet activities in human platelet-rich plasma (PRP). In human PRP, naloxone (0.1-0.5 mmol/L) inhibited aggregation stimulated by a variety of agonists (i.e. collagen, adenosine diphosphate (ADP), U46619 and adrenaline). 2. Naloxone (0.1-0.5 mmol/L) did not significantly affect cyclic adenosine monophosphate and cGMP levels in human washed platelets, whereas naloxone (0.5 mmol/L) significantly inhibited thromboxane B2 formation stimulated by collagen (5 micrograms/mL) in human washed platelets. 3. Naloxone (0.5 mmol/L) significantly inhibited [3H]-inositol monophosphate formation of [3H]-myoinositol-loaded platelets stimulated by collagen and U46619. Moreover, naloxone did not influence the binding of 125I-triflavin to platelet membranes. Triflavin is an Arg-Gly-Asp-containing specific fibrinogen receptor antagonist. 4. Addition of naloxone (0.5 mmol/L) to platelet preparations tagged with diphenylhexatriene (DPH) resulted in a considerable decrease in relative fluorescence intensity. 5. It is suggested that the anti-platelet effects of naloxone may be caused, at least partly, by the induction of conformational changes in the platelet membrane initially, followed by the inhibition of thromboxane A2 formation and phosphoinositide breakdown of platelets stimulated by agonists.