Platelets play a central role in the hemostatic process and consequently are similarly involved in the pathological counterpart, thrombosis. They adhere to various subendothelial proteins, exposed either by injury or disease, and subsequently become activated by the thrombogenic surface or locally produced agonists. These activated platelets aggregate to form a platelet plug, release agonists which recruit more platelets to the growing thrombus, and provide a catalytic surface for thrombin generation and fibrin formation. These platelet–rich thrombi are responsible for the acute occlusion of stenotic vessels and ischemic injury to heart and brain. A range of anti–platelet drugs are currently used, both prophylactically and therapeutically, in regimens to manage thrombo–embolic disorders. These include inhibitors of the generation, or effects, of locally produced agonists; several large clinical trials have supported roles for cyclooxygenase inhibitors, which prevent thromboxane generation, and thienopyridine derivatives, which antagonize ADP receptors. Similarly intravenous aIIbb3 antagonists have been shown to be effective anti–thrombotics, albeit in highly selective situations; in contrast, to date studies with their oral counterparts have been disappointing. Recent advances in understanding of platelet physiology have suggested several novel, if yet untested, targets for anti–platelet therapy. These include the thrombin receptor, the serotonin handling system, and the leptin receptor.

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