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Titel des Beitrags: The dimeric platelet collagen receptor GPVI-Fc reduces platelet adhesion to activated endothelium and preserves myocardial function after transient ischemia in mice.

Abstract: Platelets play a critical role in the pathophysiology of reperfusion, sepsis, and cardiovascular diseases. In a multiple step process, they adhere to activated endothelium and release proinflammatory cytokines thereby promoting the inflammatory process. Glycoprotein VI (GPVI) is the major collagen receptor on the platelet surface and triggers platelet activation and primary hemostasis. Activation of GPVI leads to stable platelet adhesion and degranulation of platelet granules. However, GPVI is critically involved in platelet adhesion to activated endothelium without exposure of subendothelial matrix. Earlier studies show that the soluble GPVI-Fc binds to collagen and protects mice from atherosclerosis and decreases neointima proliferation after arterial injury. Here, we show for the first time that recombinant GPVI-Fc binds to activated endothelium mainly via vitronectin and prevents platelet/endothelial interaction. Administration of GPVI-Fc reduced infarct size and preserved cardiac function in a mouse model of myocardial infarction. This process was associated with reduced GPVI-induced platelet degranulation and release of proinflammatory cytokines in vitro and in vivo. Taken together, administration of GPVI-Fc offers a novel strategy to control
platelet-mediated inflammation and to preserve myocardial function following myocardial infarction.