Abstract:

BACKGROUND: The platelet glycoprotein (GP) IIb/IIIa integrin binds to fibrinogen and thereby mediates platelet aggregation. Here, we addressed the role of GP IIb for platelet adhesion and determined the relevance of platelet GP IIb for the processes of atherosclerosis and cerebral ischemia-reperfusion (I/R) injury. METHODS AND RESULTS: GP IIb(-/-) mice were generated and bred with ApoE(-/-) animals to create GP IIb(-/-)ApoE(-/-) mice. Platelet adhesion to the mechanically injured or atherosclerotic vessel wall was monitored by in vivo video fluorescence microscopy. In the presence of GP IIb, vascular injury and early atherosclerosis induced platelet adhesion in the carotid artery (CA). In contrast, platelet adhesion was significantly reduced in the absence of GP IIb integrin (P<0.05). To address the contribution of platelet GP IIb to atheroprogression, we determined atherosclerotic lesion formation in the CA and aortic arch (AA) of GP IIb(+/+)+ApoE(-/-) or GP IIb(-/-)+ApoE(-/-) mice. Interestingly, the absence of GP IIb attenuated lesion formation in CA and AA, indicating that platelets, via GP IIb, contribute substantially to atherosclerosis. Next, we assessed the implication of GP IIb for cerebral I/R injury. We observed...
that after occlusion of the middle cerebral artery, the cerebral infarct size was drastically reduced in
mice lacking GP IIb compared with wild-types. CONCLUSIONS: These findings show for the first time
in vivo that GP IIb not only mediates platelet aggregation but also triggers platelet adhesion to
exposed extracellular matrices and dysfunctional endothelial cells. In a process strictly involving GP
IIb, platelets, which are among the first blood cells to arrive at the scene of endothelial dysfunction,
contribute essentially to atherosclerosis and cerebral I/R injury.

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