Platelet glycoprotein Ib-IX is a regulator of systemic inflammation

The platelet glycoprotein (GP)Ib-IX receptor is an adhesion receptor supporting hemostasis and thrombosis via interactions with von Willebrand factor (VWF). Here, we report studies examining the GPIb-IX/VWF axis in polymicrobial sepsis, as modeled by cecal ligation and puncture (CLP). Genetic absence of the major GPIb-IX ligand, VWF, prolongs survival following CLP, but genetic absence of the receptor, GP Ib-IX, does not. Since absence of VWF or GP Ib-IX significantly impairs hemostasis and thrombosis we sought to define additional GP Ib-IX-dependent pathways that would impact survival in the CLP model. We document the absence of GPIb-IX leads to reduced platelet-neutrophil and platelet-monocyte interactions resulting in a robust increase in major proinflammatory cytokines and Mac-1 expression following CLP. These findings suggest a previously unreported GP Ib-IX-dependent anti-inflammatory function. Thus in sharing the leucine rich motifs with toll-like receptors, platelet GPIb-IX can be considered a multi-functional participant in hemostasis, thrombosis, and the inflammatory cascade.

Lunch provided for students with the seminar speaker from 12:00 – 1:15 pm in Laney Hall Rm 105.
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