Transient and persistent aspects of human platelet activation

Propositions belonging to the dissertation

Transient and persistent aspects of human platelet activation
By Jinmi Zou

1. The reversibility of αIIbβ3 integrin activation in human platelets, as induced by receptor agonists, is steered by protein kinase C isoforms. *(This thesis)*

2. Autocrine released ADP as well as signaling via protein kinase C isoforms contribute to persistent integrin αIIbβ3 activation and hence to stabilized platelet aggregation. *(This thesis)*

3. Regulation of the long-term calcium entry in platelets, stimulated via collagen or thrombin receptors, is achieved by the channel protein Orai1 and by Na^+/Ca^{2+} exchangers in the plasma membrane. *(This thesis)*

4. Protein phosphorylation via conventional and novel protein kinase C isoforms acts as a strong negative regulator of the calcium entry in platelets, thereby suppressing platelet-dependent procoagulant activity and coagulation. (*This thesis)*

5. In whole blood thrombin generation, red blood cells play an initial role, whilst activated platelets support this process at later stage. *(This thesis)*

6. Previously activated platelets that have returned to a resting state can still contribute to the formation of a thrombus. *(De Simone, J Thromb Haemost 2023)*

7. Bridging integrator 2 is a central regulator of platelet activation in thrombosis and thrombo-inflammatory disease settings. *(Volz, J Clin Investig 2020)*

8. An ideal antiplatelet drug selectively inhibits thrombosis, preserving essential hemostatic mechanisms, with research focused on signaling molecules downstream of receptor-mediated platelet activation. *(Gawaz, Nat Rev Cardiol, 2023)*

9. Engagement in scientific research is a fascinating experience, as unexpected experimental outcomes serve as catalysts for uncovering novel mechanisms.

10. Those who work will succeed, and those who walk will arrive at their destination. 为者常成，行者常至——《晏子春秋》 *(Yanzi Chunqiu)*

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