Platelet proteomic progress and restraining mechanisms in glycoprotein VI-mediated thrombus formation

Propositions belonging to this dissertation:

Platelet proteomic progress and retraining mechanisms in glycoprotein VI-mediated thrombus formation

1. The global quantitative comparison of platelet proteome and platelet/megakaryocyte transcriptome indicates low translation level, low transcription and retainment in megakaryocyte are three restraining factors for platelet protein identification. (This thesis)
2. The inter-species comparison indicates that the correlation for the protein with estimated copy number is higher than for the quantified transcripts. (This thesis)
3. The majority of the platelet phosphoproteins have similar expression levels in patients with *GNAS* mutation and healthy individuals, except for the proteins involve in the key signaling pathways. (This thesis)
4. Integrin αIIbβ3, phosphatase SHP2 and G-coupled protein receptor GPR56 regulate the shear-dependent signaling in GPVI-mediated platelet activation and thrombus formation. (This thesis)
5. Central regulating signaling cascade of platelets is conserved in genetic, while the only differences are observed in specific mRNA and protein levels between human and mouse. (Balkenhol, BMC Genomics 2020)
6. The blocked GPVI impairs the platelet-tumor cell interaction and tumor metastasis indicates the promising antimetastatic effect of GPVI inhibition. (Mammadova-Bach, Blood 2020)
7. Phosphoproteomic quantitation of platelet not only elucidates the signaling mechanisms downstream of GPVI, but also determine effectors, biomarkers and therapeutic targets associated to platelet-related disorders. (Babur, Blood 2020)
8. The application of platelet proteomics and platelet transcriptomics in clinical trials provides a powerful tool to detect biomarkers for diagnosis and treatment.
9. Genius is 1% inspiration, 99% perspiration. (Thomas Edison)
10. The long PhD journey, from CHINA to EUROPE.