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Molecular Mechanisms of Platelet-Related Disorders

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Deadline for manuscript submissions:
closed (31 January 2024)

Message from the Guest Editors

Dear Colleagues,

Disorders in platelet count or function can have a genetic, acquired reason, etc, but in many patients with bleeding tendency, the cause of platelet disorder is still unknown. Patient and mouse knockout studies indicated that hundreds of platelet-expressed proteins are required for normal function. These proteins include multiple receptors, signaling proteins, etc. Via regulated surface exposure or activated membrane proteins or phospholipids, platelets can interact with nearby blood cells, vascular components or the coagulation system. Aberrant or pathophysiological interactions of platelets with immune or other cells may extend the consequences of platelet dysfunction to many other diseases. Platelet-related disorders are thus considered not only as a cause of bleeding but also as a factor in thrombosis, inflammation, infection or neoplasia. However, for many expressed genes and proteins the link to normal platelet function is still unclear.

The Special Issue will cover research papers and reviews on possible/new causes of quantitative or qualitative platelet disorders. Papers with molecular data will be welcomed, but clinical papers are discouraged by IJMS.

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Message from the Editor-in-Chief

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