



Hemostasis, Thrombosis, and Immunothrombosis

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Message from the Guest Editors

The prime drivers of mortality from cardiovascular diseases are platelets and the coagulation cascade. Moreover, the global pandemic caused by SARS-CoV-2 highlighted the concept of immunothrombosis, whereby microthrombi are formed due to platelet and coagulation cascade activation as a result of inflammation. Similarly, immunothrombosis is integral to the development of thrombotic shock in septic patients. The above conditions all result from a complex interplay between platelets, coagulation, innate immunity, and the endothelium.

We are most interested in reviews or original reports that delineate the novel mechanisms of pathological thrombosis, physiological hemostasis, or immunothrombosis. Potential topics include, but are not limited to:

- Platelets in thrombosis or hemostasis;
- platelet–leukocyte interactions during immune responses;
- coagulation responses during sepsis;
- coagulation responses during SARS-CoV-2 infection;
- platelet function during sepsis;
- platelet function during SARS-CoV-2 infection;
- endothelial cell function in systemic inflammation.





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Message from the Editor-in-Chief

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