## Immunology, Immunity, Inflammation & Immunotherapies

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## Platelets: The new innate immune cell on the block

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Platelets are abundant small anucleate cells in the blood classically known for their essential role in hemostasis and thrombosis. However, the versatility of platelets is becoming increasingly recognized and it is now clear their function extends far beyond hemostasis. Particularly, their ability to significantly modulate an immune response has established a new appreciation of platelets as an innate immune cell. In addition to microbial phagocytic functions, platelets contain large stores of cytokines and chemokines. Platelets also express surface receptors including CD40L, P-selectin, adhesion receptors, MHC-1 etc. and through direct contact can antigen present, support leukocyte rolling, extravasation and immunoglobulin classswitch. Typically, platelet activation leads to a supportive pro-inflammatory function. We previously reported in autoimmune thrombocytopenia (ITP), the immune response against platelet surface receptors GPIba and GPIIbIIIa are fundamentally different. Antibodies targeting GPIba predominantly leads to platelet activation, desialylation, and Fc-independent platelet clearance. Furthermore, an anti-GPIba immune response, as we recently observed, leads to the lower generation of protective CD8+ Tregs. Thus, anti-GPIba mediated ITP is more resistant to standard immunosuppressive/immunomodulatory therapies including steroids and IVIG, a finding which has been substantiated in human ITP studies. Although most reports have focused on the pro-inflammatory roles of platelets, our recent data reveal an emerging concept of the immunosuppressive role of platelets. Whereby clearance of resting desialylated platelets in the liver may lead to systemic immune-suppression against platelet-associated antigens, possibly through the large stores of TGF $\beta$  contained within the platelet. Thus, platelets play dynamic roles in immunomodulation and elucidation of these mechanisms may have far-reaching implications in various diseases including cancer, infections, sepsis, and others.

## **Biography**

Heyu Ni received his MD (from China), PhD (Immunology; from Canada), and Postdoctoral fellow (Pathology; Harvard, USA). He is a Scientist of the Canadian Blood Services Centre for Innovation, the Platform Director for Hematology, Cancer and Immunological Diseases at St. Michael's Hospital. Since 2013, he has been a Full Professor in the Departments of Laboratory Medicine and Pathobiology, Physiology, and Medicine at the University of Toronto. His laboratory currently investigates the roles of adhesion molecules involved in thrombosis/hemostasis, inflammation/immune response, and tumorigenesis. They study these processes using a confocal intravital microscopy suite, proteomics, and gene-targeted mice. By directly monitoring molecular/cellular events in vivo, they hope to uncover some novel mechanisms of platelet aggregation, platelet-leukocyte/cancer cell interactions to enrich our knowledge in cardiovascular diseases, inflammation/ immune response, and tumor metastasis. The laboratory also studies also- and autoimmune diseases, such as Autoimmune Thrombocytopenia (ITP), Fetal and Neonatal Alloimmune Thrombocytopenia (FNAIT).

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