Special Issue

Immuno-Metabolism: Resisting Resistance to Immuno-Checkpoint Therapy

Message from the Guest Editor

Inhibitory immune checkpoints such as PD□1/PD□L1 normally maintain peripheral tolerance by disabling the activation and effector functions of autoreactive T□cells and avoiding uncontrolled myeloid cell activation, have been found to be co□opted in cancer.

Metabolic reprogramming of T and myeloid cells in patients with cancer and autoimmune diseases has been increasingly investigated. Of interest are the functional activities through which myeloid cells instruct T cells. Monocytes and macrophages serve as antigen-presenting cells and fine tune T cell responses. By expressing PD-1/PD-L1, they can also suppress T cell immunity, a functional aspect of importance in tumors with PD-1 or PD-L1hi tumor–associated monocytes and macrophages.

In this Special Issue, dedicated to Immuno-metabolism, we present a collection of works that provide insight into this diverse spectrum of metabolic reprogramming of myeloid and T cell activities within the host immune and tissue repair responses, and the contribution of chronic inflammation and metabolic syndrome to resistance and toxicity pathways to immuno-checkpoint therapy.

Guest Editor

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