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Targeting Immune Cell Metabolism in Kidney Diseases

Niels Olsen Saraiva Camara
University of Sao Paulo, Brazil

The connection between immunometabolism and inflammation is central in several immunity-mediated inflammatory processes, such as the renal diseases. The renal cell populations possess distinguished metabolic demands, which are associated with their physiological roles. However, upon injury, this metabolic profile could be reprogrammed to fit new stresses, ultimately leading to inflammation and tissue fibrosis. Further, the metabolic alterations seen in immune cells that infiltrate the kidneys after injury can also modulate the metabolism of renal cells and change their phenotype. Recent studies suggest that the development of fibrosis is associated with changes in the glycolytic and oxidative metabolism of proximal tubular epithelial cells and podocytes. The maintenance of cell energy homeostasis involves a series of molecules, with hypoxia-induced factor 1 alpha (HIF-1α) being a key mediator of this process. HIF-1α acts as a transcriptional regulator of genes, such as genes related to glycolytic metabolism. However, disturbances of O2 homeostasis and abnormal HIF-1α activity may contribute to the pathogenesis of diseases. In this talk, we will discuss the role of HIF-1α in the modulation of renal cell metabolism and in the development and progression of renal diseases. Finally, we will exploit the metabolism of renal cells as putative targets for new adjuvant therapeutics.