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Personalized Treatment and Hereditary Causes of Nephrotic Syndrome

Guest Editor:

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Deadline for manuscript submissions:
closed (10 July 2021)

Message from the Guest Editor

Nephrotic Syndrome (NS) is a major cause of chronic and end-stage kidney disease worldwide. Advances in renal genetics over the past three decades have highlighted glomerular visceral epithelial cells (i.e., podocytes) as the principal cell type affected in disease pathogenesis. It is now clear that podocyte injury or loss is a necessary precursor of glomerular dysfunction in NS and studies of familial forms of the disease have identified a variety of molecular targets involved in podocyte actin cytoskeletal dynamics. In this Special Issue, we will highlight: Emerging technologies for characterizing podocyte biology; Novel insights into the mechanisms of podocyte injury; Novel model systems for modeling podocyte injury; Novel methods or approaches for high throughput screening of candidate compounds for podocytopathies; Exploration of podocyte transcriptomics and proteomics in health and disease to identify novel therapeutic targets.

We will be accepting submissions in these areas to assemble a Special Issue focused on the translation of findings in renal genetics into rational therapies for nephrotic syndrome.

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Message from the Editor-in-Chief

Journal of Personalized Medicine (*JPM*; ISSN 2075-4426) is an international, open access journal aimed at bringing all aspects of personalized medicine to one platform. *JPM* publishes cutting edge, innovative preclinical and translational scientific research and technologies related to personalized medicine (e.g., precision medicine, pharmacogenomics/proteomics, systems biology, 'omics association analysis). *JPM* is covered in Scopus, the Science Citation Index Expanded (SCIE), PubMed, PMC, Embase, and other databases.

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