

Special Issue

Nephrogenetics and Kidney Genomics—the Future Is Now?

Message from the Guest Editors

Monogenic and oligogenic forms of kidney disease are an important cause of kidney disease in adults and children with prevalence estimates emerging recently. The pace of molecular characterization and discovery in the space of heritable and genetic forms of kidney disease has been significant recently. Directed therapies are also beginning to emerge for several genetic kidney disorders across the broad spectrum of preclinical studies, clinical trials and also implementation into practice. With our increasing understanding, acceleration of the discovery and clinical translation in a multidisciplinary and patient centric manner is critical to realizing global benefit delivered by clinicians, researchers, industry and health systems.

In this Special Issue, we welcome reviews and original articles in regard to all facets of the study of genetic forms of kidney disease. These include, but are not limited to, genetic and genomic characterization (including epidemiology, case series and novel case reports), molecular mechanisms of disease, clinical genomics, research genomics, functional genomics, preclinical research, clinical studies and trials. We look forward to your contributions.

Guest Editors

Prof. Dr. Andrew Mallett

Prof. Dr. Zornitza Stark

Dr. Amali Mallawaarachchi

Deadline for manuscript submissions

closed (15 February 2024)

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About the Journal

Message from the Editor-in-Chief

Genes is central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fast-moving field. There is a need for good quality, open access journals in this area, and the *Genes* team aims to provide expert manuscript handling, serious peer review, and rapid publication across the whole discipline of genetics. Starting in 2010, the journal is now well established and recognised. Why not consider *Genes* for your next genetics paper?

Editor-in-Chief

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