

## Special Issue

# The Role of Inflammation in Diabetic Retinopathy

### Message from the Guest Editor

Recent studies have revealed the principal role of immune cells and cytokines in the development and progression of diabetic retinopathy (DR). Traditionally, immune cells are characterized into M1 and M2 phenotypes, which are considered to target the regulation of polarization and function of macrophages/microglia. The new therapies directed toward attenuating the progression of DR could be involved in modulation.

For the Special Issue, we accept both reviews and articles. Reviews should provide a state-of-the-art overview of retinal or eye disorders during diabetes in the context of inflammation and immunomodulation, and may also highlight new approaches to elucidate the different roles of immune cells during DR or other eye pathologies associated with DM development, progression and treatment. Articles should address the pathophysiology of DR in experimental cell-culture, animal models, and clinical practice, including new findings on biomarkers, signaling pathways, and new therapeutic strategies. Our aim for this Special Issue is to shine a light on multidisciplinary research examines the immune cells in diabetic retinopathy from a different perspective.

### Guest Editor

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### Deadline for manuscript submissions

closed (31 October 2023)



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### Message from the Editor-in-Chief

The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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### Editor-in-Chief

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