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Glaucoma: From Pathophysiology to Novel Therapies

Guest Editor:

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Deadline for manuscript submissions:

closed (30 June 2022)

Message from the Guest Editor

Dear Colleagues,

Primary open-angle glaucoma (POAG) is the leading cause of irreversible blindness affecting over 57 million people worldwide. Progressive loss of retinal ganglion cells (RGCs) and degeneration of optic nerve axons constitute the pathological hallmark of glaucoma. Elevated intraocular pressure (IOP) due to dysfunction of trabecular meshwork (TM) is the most significant and the only known modifiable risk factor for glaucoma. Although glaucomatous TM damage is known to be mainly responsible for IOP elevation, which can lead to optic neuropathy and vision loss, none of the current treatments target TM pathology this is partly due to the lack of understanding of pathological mechanisms of TM damage. In this Special Issue, we invite original manuscripts and review articles that focus on understanding the pathological mechanisms of glaucomatous TM damage and novel therapies targeting these pathologies.













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Editor-in-Chief

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Message from the Editor-in-Chief

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