

Special Issue

Endogenous PCSK9 for Patients with Type 2 Diabetes Mellitus (T2DM)

Message from the Guest Editors

PCSK9 is a serine protease produced primarily (but not exclusively) by the liver and the intestine that acts as a regulator pathway for hepatic low-density lipoprotein receptor (LDLR) degradation through an endosomal/lysosomal pathway. PCSK9 plays additional roles in atherosclerosis development and progression via a variety of nonclassical mechanisms that involve inflammatory, apoptotic, and immune pathways. Endogenous PCSK9 also plays a role in the cardiovascular complications of patients with diabetes, as highlighted by genetic studies correlating gain-of-function and loss-of-function mutations in the PCSK9 gene with coronary artery disease, as well as by the cardiovascular benefit observed with PCSK9-inhibitors. In this Special Issue of *Cells*, I invite you to contribute original research articles, reviews, or shorter perspective articles on all aspects related to the title *Endogenous PCSK9 for Patients with Type 2 Diabetes Mellitus*.

Guest Editors

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Deadline for manuscript submissions

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About the Journal

Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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manuscripts are peer-reviewed and a first decision is provided to authors approximately 16 days after submission; acceptance to publication is undertaken in 2.7 days (median values for papers published in this journal in the first half of 2025).