

## Special Issue

# Autophagy and AMPK/mTOR Signaling Regulate Stress Responses, Metabolism, and Immunity

### Message from the Guest Editors

Autophagy is upregulated in response to diverse types of stressful stimuli, functioning as a cell survival mechanism that removes damaged proteins/organelles and provides new building blocks and energy sources during metabolic, hypoxic, oxidative, proteotoxic, and drug-induced stress. In addition to regulating cellular metabolism and stress responses in both normal and cancer cells, autophagy shapes immunity through pathogen clearance, modulation of cytokine production, antigen presentation, and lymphocyte selection, homeostasis, and survival. The main intracellular signals for autophagy induction are the activation of the energy sensor AMP-activated protein kinase (AMPK) and subsequent inhibition of the mechanistic target of rapamycin (mTOR), the major repressor of catabolic processes, including autophagy. This Special Issue welcomes submissions on autophagy and AMPK/mTOR signaling in regulation of stress responses, metabolism, and immune cell function, with the purpose of outlining their roles and interaction in cellular and organismal homeostatic functions as well as in diverse pathologic settings, including metabolic and immune-system-related diseases.

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### Guest Editors

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### Deadline for manuscript submissions

closed (20 November 2022)



## Cells

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### 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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### Editors-in-Chief

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