Special Issue

Molecular and Cellular Regulation of the Skeletal System in Healthy and Pathological Conditions

Message from the Guest Editors

Molecular pathways and cellular processes regulating the skeletal system play a central role in maintaining lifelong physiological conditions. However, alterations in molecular and cellular processes regulating osteogenic or chondrogenic differentiation impair the skeletal system. In this context, the transcription factors involved in mesenchymal stem cells' commitment towards osteogenic or chondrogenic lineages can be considered as leading agents of cell signaling regulation. The aging process, as well as metabolic and degenerative diseases, may affect bone and cartilage. Mutations affecting the expression of genes involved in the regulation of skeletal development cause several disorders as well. This Special Issue provides a collection of original research and review articles related to the physiological regulation of the skeletal system and its impairment caused by molecular and cellular disruption as well as by mutations of key genes involved in osteogenesis or chondrogenesis.

Guest Editors

Dr. Maria Teresa Valenti

Department of Neurosciences, Biomedicine and Movement Sciences, University of Verona, 37128 Verona, Italy

Prof. Monica Mottes

Department of Neurosciences, Biomedicine and Movement Sciences, Biology and Genetics Section, University of Verona, Verona, Italy

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Cells
Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
cells@mdpi.com

mdpi.com/journal/cells

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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.

Editors-in-Chief

Dr. Alexander E. Kalyuzhny
Dental Basic Sciences, University of Minnesota, 308 Harvard St. SE, Minneapolis, MN 55455, USA

Prof. Dr. Cord Brakebusch
Biotech Research & Innovation Centre, The University of Copenhagen, Copenhagen, Denmark

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manuscripts are peer-reviewed and a first decision is provided to authors approximately 15.5 days after submission; acceptance to publication is undertaken in 2.8 days (median values for papers published in this journal in the second half of 2025).