

OPEN ACCESS

EDITED AND REVIEWED BY
Gaetano Santulli,
Albert Einstein College of Medicine,
United States

*CORRESPONDENCE

Ki Ho Park

kiho.park@virginia.edu

Bowen Wang

■ bowenwang@northwestern.edu

RECEIVED 04 August 2025 ACCEPTED 08 August 2025 PUBLISHED 20 August 2025

CITATION

Park KH and Wang B (2025) Editorial: Calcium homeostasis and cardiovascular health: mechanisms and therapeutic insights. *Front. Endocrinol.* 16:1679610. doi: 10.3389/fendo.2025.1679610

COPYRIGHT

© 2025 Park and Wang. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.

Editorial: Calcium homeostasis and cardiovascular health: mechanisms and therapeutic insights

Ki Ho Park 1* and Bowen Wang 2*

¹University of Virginia, Charlottesville, VA, United States, ²Northwestern University, Chicago, IL, United States

KEYWORDS

calcium homeostasis, cardiovascular disease, mitochondria-associated membranes (MAMs), vascular calcification, cellular senescence, irisin, atherosclerosis, non-alcoholic fatty liver disease (NAFLD)

Editorial on the Research Topic

Calcium homeostasis and cardiovascular health: mechanisms and therapeutic insights

Calcium signaling is essential for the regulation of cardiovascular physiology, orchestrating myocardial contraction, vascular tone, gene expression, and more. Disturbances in calcium homeostasis contribute to a wide range of cardiovascular diseases, yet the molecular mechanisms linking calcium dynamics to cardiovascular pathology are not fully understood. In the current Research Topic, we highlight five valuable contributions that uncover new key mechanisms and potential therapeutic targets involved in calcium regulation in cardiovascular health and disease.

In their original research article, Li et al. demonstrated the "Cardioprotective effect of Yiqi Huoxue decoction on post-myocardial infarction injury mediated by Ca²⁺ flux through MAMs". Using both in vivo and in vitro models, the authors found that Yiqi Huoxue decoction, a traditional Chinese herbal formula, restored mitochondrial-associated membrane (MAM) structure, improved Ca²⁺ homeostasis, and regulated the expression of critical proteins including IP3R2, GRP75, VDAC1, MCU, and Sigma-1R. These findings support a protective role of MAMs in cardiomyocyte injury and open avenues for pharmacological targeting of intracellular Ca²⁺ flux in ischemic heart disease.

Li et al. reviewed the role of "Endoplasmic reticulum-mitochondria crosstalk: new mechanisms in the development of atherosclerosis". This review article comprehensively discussed how MAMs mediate key processes linked to atherogenesis, including lipid metabolism, calcium transport, oxidative stress, and apoptosis. The authors emphasized the dynamic regulation of MAM integrity and suggested that MAM-targeted therapies could be an innovative strategy for modulating plaque progression and cardiovascular risk.

Park and Wang 10.3389/fendo.2025.1679610

The review by Fang et al. titled "The functional role of cellular senescence during vascular calcification in chronic kidney disease" explored how senescent vascular cells promote vascular calcification through the release of microvesicles and senescence-associated secretory phenotype (SASP) factors. These mechanisms enhance osteogenic differentiation in vascular smooth muscle cells (VSMCs) and impair vascular regeneration, suggesting that targeting cellular senescence may represent a promising strategy to mitigate VC in chronic kidney disease (CKD) patients.

Shuangshuang Wang et al. contributed the mini-review "The emerging roles of irisin in vascular calcification", which focused on the exercise-induced myokine irisin. This review highlighted irisin's protective role in modulating vascular inflammation, endothelial function, and VSMC phenotypic switching, likely via mechanisms involving calcium signaling and oxidative stress. The authors discussed how irisin may serve both as a predictor and a potential therapeutic modulator for vascular calcification and hypertension.

Finally, the original research article by Hao et al. titled "Observational and genetic association of non-alcoholic fatty liver disease and calcific aortic valve disease" identified an association between non-alcoholic fatty liver disease (NAFLD) and increased risk of calcific aortic valve disease. =The link to ectopic calcification and systemic metabolic dysfunction provides critical context for calcium-driven cardiovascular pathologies.

Together, these articles reflect the complex, multi-layered role of calcium homeostasis in cardiovascular disease.

We thank all the contributing authors for their valuable insights and efforts. We hope this Research Topic will inspire further exploration into calcium-centered mechanisms and therapeutic innovation in cardiovascular research.

Author contributions

KP: Writing – original draft, Writing – review & editing. BW: Writing – original draft, Writing – review & editing.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The author(s) declare that no Generative AI was used in the creation of this manuscript.

Any alternative text (alt text) provided alongside figures in this article has been generated by Frontiers with the support of artificial intelligence and reasonable efforts have been made to ensure accuracy, including review by the authors wherever possible. If you identify any issues, please contact us.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.