BMC Cardiovascular Disorders

EDITORIAL Open Access

Omics approaches to understand cardiovascular disease

Zeeshan Ahmed[1,2,3*] and Goo Jun[4]

Abstract
Omics approaches have emerged as indispensable tools in unravelling the intricate molecular landscape of cardiovascular disease (CVD) by providing comprehensive insights into the underlying mechanisms driving CVD pathogenesis, progression, and response to therapy. Integrative omics approaches further enhance our understanding by integrating multi-omics data to explain complex molecular networks and identify novel disease pathways. In this collection of *BMC Cardiovascular Disorders*, we invited submissions on omics approaches to investigate and understand CVD. Successfully achieving the goals of this collection, we were able to publish some interesting and impactful research articles after a rigorous peer review process.

Keywords Cardiovascular disease, Omics, Bioinformatics, Biomarkers, Machine learning

Editorial

Cardiovascular diseases (CVDs) are among the causes leading to preventable deaths in the US and around the world [1]. Despite significant developments in CVD diagnostics, prevention, and treatment, approximately half of the affected patients reportedly die within five years of receiving a diagnosis [2]. This leads to the development of timely, essential, and innovative diagnostic and therapeutic strategies for improving CVD patient's survival and quality of life. CVDs encompass a diverse range of conditions, each influenced by complex and highly individualized risk factors [3]. Evidence from the Framingham Heart Study suggests that CVD has a complex multifactorial etiology including a genetic component [4, 5]. Omics approaches have emerged as indispensable tools in unravelling the intricate molecular landscape of CVDs by providing comprehensive insights into the underlying mechanisms driving CVD pathogenesis, progression, and response to therapy. These are bolstering the field of personalized medicine by enabling translational researchers and clinicians to identify individuals at heightened CVD risk and tailor treatments according to each patient's unique genetic profile [6].

Omics approaches represent a transformative paradigm in cardiovascular research, and clinical practice, offering unprecedented opportunities to decipher the molecular intricacies of CVD and revolutionize patient care. The application of omics technologies holds immense promise in advancing precision medicine approaches for CVD management. Integrative omics approaches further enhance our understanding by integrating multi-omics data to explain complex molecular networks and identify novel disease pathways [7]. By leveraging omics data, clinicians can stratify patients based on their molecular profiles, enabling tailored treatment strategies and

*Correspondence:
Zeeshan Ahmed
zahmed@ifh.rutgers.edu
[1]Department of Medicine, Division of Cardiovascular Diseases and Hypertension, Robert Wood Johnson Medical School, Rutgers Health, 125 Paterson St, New Brunswick, NJ 08901, USA
[2]Institute for Health, Health Care Policy and Aging Research, Rutgers Health, 112 Paterson St, New Brunswick, NJ 08901, USA
[3]UConn School of Medicine, UConn Health, 263 Farmington Ave, Farmington, CT, USA
[4]Human Genetics Center, School of Public Health, University of Texas Health Science Center Houston, Houston, TX, USA

improving therapeutic outcomes [7]. Omics-based biomarkers offer non-invasive tools for early disease detection, risk prediction, and monitoring disease progression [6].

BMC Cardiovascular Disorders invited research to its collection "Omics approaches to understand cardiovascular disease", seeking contributions that address a spectrum of topics e.g., *genomics*, *epigenomics*, *transcriptomics*, *phenomics*, *metagenomics*, *metabolomics*, *lipidomics*, *proteomics*, *integrative omics analyses*, *omics-based biomarker discovery*, *molecular pathways in cardiovascular disease*, *personalized medicine approaches*, *therapeutic targets and drug development*, *and clinical applications of omics technologies in cardiovascular medicine* (Fig. 1). In response, we received a high number of submissions. A few high quality research articles have been selected after a rigorous peer review process and their key findings include shortage of adequate studies on the effectiveness and safety of using Antiarrhythmic Drugs (AADs) following Circumferential Pulmonary Vein Isolation (CPVI) [8]; new therapeutic strategies for CryAB (R120G)-related cardiomyopathy by targeting specific pathways and hub genes to mitigate cardiac dysfunction [9]; and identification of plasma proteomic biomarkers that are differentially expressed between physically frail and non-physically frail adults with heart failure (HF) for better understanding of the underlying pathophysiology [10]. Successfully achieving the goals of this collection, we were able to publish some interesting and impactful research articles.

In "*Effects of low-dose metoprolol succinate sustained-release tablets on patients with paroxysmal atrial fibrillation undergoing circumferential pulmonary vein isolation: a retrospective cohort study*", Ding et al. investigated the impact of low-dose sustained-release metoprolol succinate tablets on Heart rate variability (HRV) and arrhythmia in patients with paroxysmal atrial fibrillation (PAF) post-circumferential pulmonary vein isolation (CPVI). They used a retrospective study design with 67 patients divided into a medication group ($n = 33$) with 23,75 mg/day of metoprolol succinate and a non-medication group ($n = 34$). Patients underwent 24-hour Holter monitoring before the procedure and at 1–3, 6, 12, and 18 months post-procedure. Next, they performed statistical analysis to compare various measurements between the medication and non-medication group, which concluded with no significant differences in the HRV parameters and arrhythmia data between the two groups. The overall findings signaled that low-dose metoprolol succinate may not have significantly affected autonomic function or arrhythmic events post-CPVI, most probably due to subtherapeutic dosing. Authors recognized limitations of the study due to the small sample size possible bias by retrospective design and recommended prospective design with larger sample size for further validation.

In "*Comprehensive bioinformatic analysis identifies potential therapeutic drugs for CryAB (R120G)-related cardiomyopathy*", Zheng et al. utilized a multi-step bioinformatic approach to identify potential therapeutic drugs for cardiomyopathy caused by the R120G mutation in the Alpha-B Crystallin (CryAB). CryAB prevents aggregation of misfolded proteins and provides protection against ischemic damage and heart failure, but mutations including R120G disrupts its function and may cause myocardial injury and heart failure. Authors engineered a CryAB(R120G) transgenic (TG) mouse model and generated RNA-seq data from heart tissues. These mice exhibited increased cardiac biomarkers (ANP, BNT, MYH7), significant myocardial fibrosis, and aggregation of insoluble proteins. Differentially expressed genes (DEGs) were analyzed by Gene Ontology (GO) and KEGG pathways. Drug compounds that could potentially reverse these gene expressions were searched using the Connectivity Map (CMaP) database and were further evaluated using molecular docking simulation. Reported findings by the author included overexpression of CryAB(R120G) and heart failure phenotypic alterations in CryAB (R120G) TG mouse, nine key regulatory nodes, potential candidates and targets, constructive binding affinity between nortriptyline and all nine proteins encoded by hub genes.

In "*Plasma proteomic biomarkers of physical frailty in heart failure: a propensity score matched discovery-based pilot study*", Denfeld et al. focused on the identification of plasma proteomic biomarkers that are differentially expressed between physically frail and non-physically frail adults with HF. They performed a secondary analysis of data and plasma samples on 20 matched pairs of physically frail and non-frail patients with chronic stable HF. Measurements included sociodemographic and clinical information, physical frailty assessment, and proteomics data. Mass-spectrometry based proteomics analysis identified 2,684 proteins and 67 of them were significantly different between two patient groups. After further significance and consistency-based filtering, authors identified seven proteins (*MMP14*, *CPNE1*, *FCG3A*, *FCG3B*, *KVD24*, *GSTM1*, and *ARLY*) fully different between the two groups. The most prominent markers were related to the immune system, suggesting a link between physical frailty and immune dysfunction. The findings from this pilot study suggests possible future follow-up studies for the study of physical frailty in HF.

The potential implications of this collection will increase our understanding of CVDs with the analysis of omics data using bioinformatics approaches.

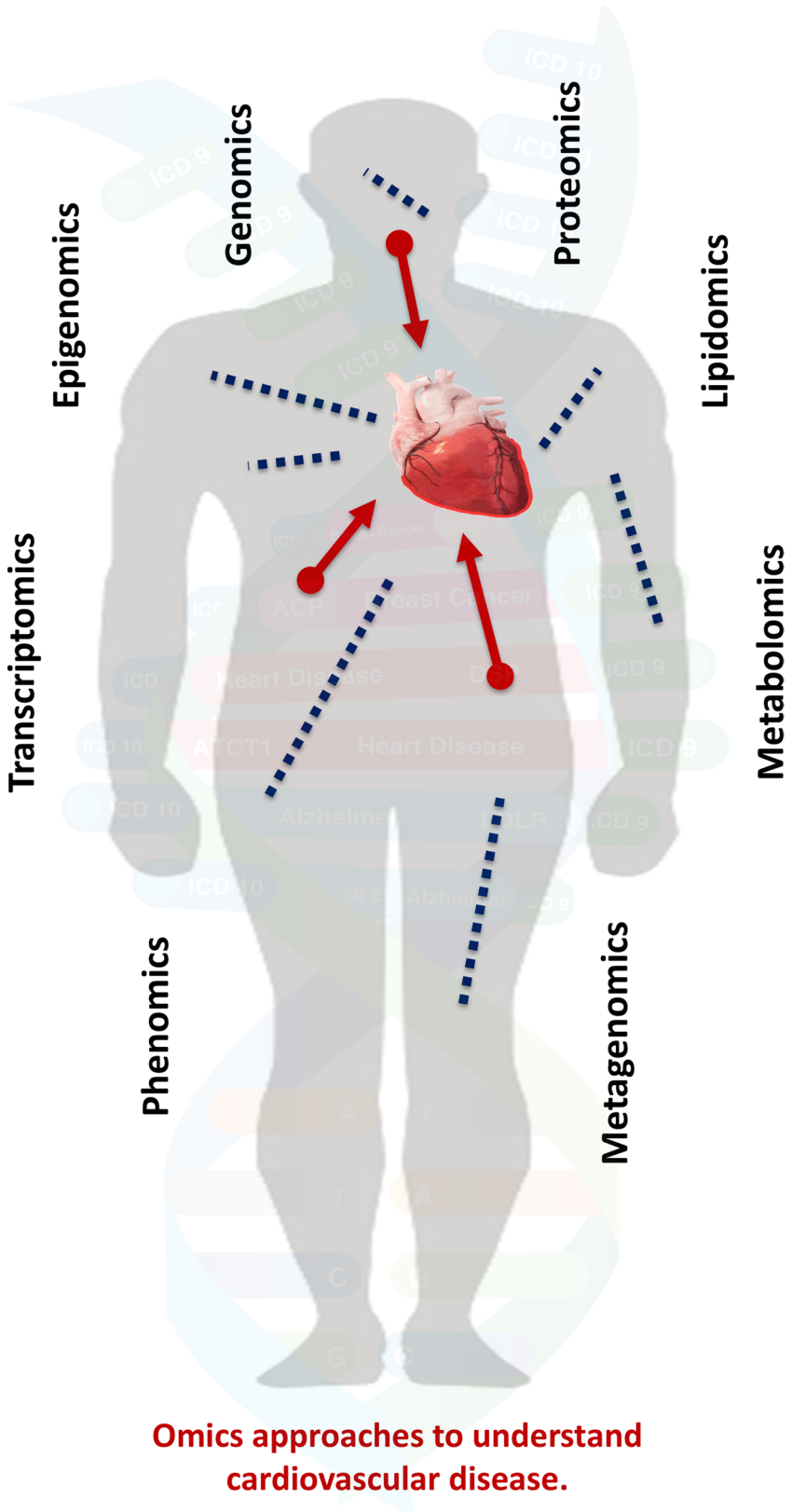

Fig. 1 Omics approaches to understand cardiovascular disease

Abbreviations

AADs	Antiarrhythmic Drugs
CryAB	Alpha-B Crystallin
CVD	Cardiovascular disease
CPVI	Circumferential Pulmonary Vein Isolation
HF	Heart Failure
TG	Transgenic

Acknowledgements
We are grateful to BMC Cardiovascular Disorders, and editorial staff for their endless support in the preparation, collection, peer-review, editing, press, and publication processes. We thank the honorable reviewers for their time and constructive suggestions to the authors for the possible quality and scientific improvements to their studies. We appreciate efforts and dedication of the authors who contributed to this collection.

Clinical trial number
Not applicable.

Authors' contributions
ZA drafted the manuscript. ZA and GJ both contributed to the conception and writing of the article. Both authors read and approved the final manuscript.

Funding
Not applicable.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 27 November 2025 / Accepted: 1 December 2025
Published online: 18 December 2025

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Publisher's Note

Zeeshan Ahmed is an Assistant Professor at the Department of Medicine, Division of Cardiovascular Diseases and Hypertension, Robert Wood Johnson Medical School, and a Core Faculty Member at the Rutgers Institute for Health, Health Care Policy, and Aging Research, Rutgers Health. Furthermore, ZA is the Adjunct Assistant Professor at the School of Medicine, UConn Health, CT

Goo Jun is an Associate Professor of Epidemiology at the UTHealth Houston School of Public Health.