INPLASY

INPLASY202540024

doi: 10.37766/inplasy2025.4.0024

Received: 7 April 2025

Published: 7 April 2025

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Multi-Omics in Sudden Cardiac Arrest: A Systematic Review

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ADMINISTRATIVE INFORMATION

Support - This study was supported by the National Key R & D Program of China (2020YFC1512700, 2020YFC1512705, 2020YFC1512703), National S&T Fundamental Resources Investigation Project (2018FY100600, 2018FY100602), Taishan Pandeng Scholar Program of Shandong Province (tspd20181220), Taishan Young Scholar Program of Shandong Province (tsqn20161065, tsqn201812129), National Science Fund for Distinguished Young Scholars (82325031), Youth Top-Talent Project of National Ten Thousand Talents Plan and Qilu Young Scholar Program.

Review Stage at time of this submission - Formal screening of search results against eligibility criteria.

Conflicts of interest - None declared.

INPLASY registration number: INPLASY202540024

Amendments - This protocol was registered with the International Platform of Registered Systematic Review and Meta-Analysis Protocols (INPLASY) on 7 April 2025 and was last updated on 7 April 2025.

INTRODUCTION

Review question / Objective Review question: What is the current evidence from omics studies (genomics, transcriptomics, proteomics, metabolomics, and multi-omics) regarding the pathogenesis, biomarker discovery, and potential therapeutic targets for sudden cardiac arrest (SCA)?
Objectives: To systematically review and synthesize the findings of omics-based studies in the field of sudden cardiac arrest (SCA), aiming to uncover molecular mechanisms, identify novel biomarkers, and explore precision medicine strategies.

Rationale Sudden cardiac arrest (SCA) is a major public health issue with high mortality and limited effective treatments. The underlying mechanisms are complex and not fully understood. Omics technologies—such as genomics, transcriptomics, proteomics, and metabolomics—have opened new opportunities to explore the molecular basis of SCA. Although many omics studies have been published, their findings are scattered and lack integration. This systematic review aims to summarize and synthesize current evidence from omics-based studies on SCA, in order to identify key mechanisms, biomarkers, and potential therapeutic targets.

Condition being studied Sudden cardiac arrest (SCA) is a critical medical emergency defined by the abrupt loss of heart function, often resulting in sudden cardiac death (SCD) if not treated immediately. It can be caused by various cardiac conditions such as coronary artery disease, cardiomyopathies, or primary arrhythmias. SCA is a major cause of mortality worldwide, affecting both adults and young individuals. This review focuses on the molecular mechanisms and biomarkers associated with SCA as explored through multi-omics studies.

METHODS

Search strategy Databases: PubMed, Cochrane Library, Web of Science, ScienceDirect
Keywords: ("Sudden Cardiac Death" OR "Cardiac Arrest" OR "SCD" OR "CA") AND ("Genomics" OR "Transcriptomics" OR "Proteomics" OR "Metabolomics" OR "Multi-omics")
Date range: From inception to December 2024
Screening: Two independent reviewers screened and selected studies per PRISMA flow.

Participant or population Population: Human subjects (living or deceased with biospecimens) and animal models with SCA or related cardiac arrest events.
Study design: Original research articles using omics technologies (genomics, transcriptomics, proteomics, metabolomics, or multi-omics).
Outcomes: Mechanistic insights, biomarker discovery, or treatment targets for SCA.

Intervention This review does not evaluate a specific clinical intervention. Instead, it focuses on studies that apply omics technologies—such as genomics, transcriptomics, proteomics, metabolomics, or integrated multi-omics approaches—to investigate the pathophysiology, biomarkers, and potential therapeutic targets of sudden cardiac arrest (SCA).

Comparator No clinical comparative intervention is applied. The review compares findings across various omics approaches and study types related to SCA.

Study designs to be included Eligible study designs include: experimental studies in animal models, observational studies involving human biospecimens, multi-omics integration studies.

Eligibility criteria Inclusion criteria:
1. Population: Human subjects (living or deceased with biospecimens) and animal models with SCA or related cardiac arrest events.
2. Study design: Original research articles using omics technologies (genomics, transcriptomics, proteomics, metabolomics, or multi-omics).
3. Outcomes: Mechanistic insights, biomarker discovery, or treatment targets for SCA.
Exclusion criteria
1. Non-original articles (e.g., reviews, commentaries, letters)
2. Studies unrelated to omics or lacking association with SCA mechanisms
3. Abstract-only publications or conference posters without full text.

Information sources The following electronic databases will be searched: PubMed, Web of Science, Cochrane Library, and ScienceDirect, from inception to December 2024. Reference lists of included articles will also be manually screened to identify additional relevant studies.

Main outcome(s)
The primary outcomes of this review will include:
1.Identified biomarkers of sudden cardiac arrest (SCA) or sudden cardiac death (SCD) from genomics, transcriptomics, proteomics, metabolomics, or multi-omics studies.
2.Molecular mechanisms and biological pathways implicated in the pathogenesis of SCA, as revealed by omics analyses.
3.Technological platforms and omics strategies applied, including sample types, detection techniques, and validation methods.

Additional outcome(s)
Additional outcomes include:
1. Cross-species findings, comparing data from animal models and human biospecimens.
2. Integrated multi-omics findings, such as co-expression networks or shared pathway alterations.

Data management All records identified through database searches will be imported into EndNote reference management software for de-duplication. After removing duplicates, two independent reviewers will screen the titles and abstracts for eligibility. Full texts of potentially relevant studies will be retrieved and assessed based on predefined inclusion and exclusion criteria. Data from included studies will be extracted using a standardized data extraction form developed in Microsoft Excel.

Quality assessment / Risk of bias analysis The quality of included studies will be assessed independently by two reviewers. For animal studies, the SYRCLE's Risk of Bias tool will be used. This tool is adapted from the Cochrane risk

of bias tool and is specifically designed to evaluate methodological quality in animal experiments. For human biospecimen studies, the JBI Critical Appraisal Tools will be applied according to the specific study design (e.g., cohort, case-control, or cross-sectional studies).
Any disagreements between reviewers will be resolved through discussion, and if consensus cannot be reached, a third reviewer will be consulted.

Strategy of data synthesis A narrative synthesis will be conducted to summarize the biomarkers identified in included studies using different omics approaches (genomics, transcriptomics, proteomics, metabolomics, or multi-omics). Studies will be grouped by omics type, and key details such as the biomarker name, sample source, study model (human or animal), and reported function or validation will be extracted and presented in descriptive tables. No statistical pooling or integrative analysis will be performed due to the heterogeneity of study designs.

Subgroup analysis
Subgroup analyses will be performed descriptively to compare biomarker findings across:
1. Different omics technologies (e.g., genomics vs. proteomics)
2. Study types (e.g., human biospecimen studies vs. animal models)
3. Sample sources (e.g., plasma, myocardium, brain tissue)
These subgroup summaries aim to provide insights into consistency or variability of biomarker discovery across study contexts. No statistical comparisons will be made.

Sensitivity analysis No sensitivity analysis is planned, as this review is descriptive in nature and does not involve quantitative data synthesis or meta-analysis.

Language restriction No language limits will be imposed. Articles published in any language will be considered for inclusion.

Country(ies) involved China / Shandong University Qilu Hospital.

Other relevant information No supplementary information is provided for this protocol.

Keywords Sudden Cardiac Death; Omics; Biomarkers; integrative analysis; Precision Medicine.

Dissemination plans The results will be submitted for publication in a peer-reviewed journal.

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