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Genetic Susceptibility to Chemotherapy-Induced Heart Failure: A Systematic Review

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INTRODUCTION & AIM

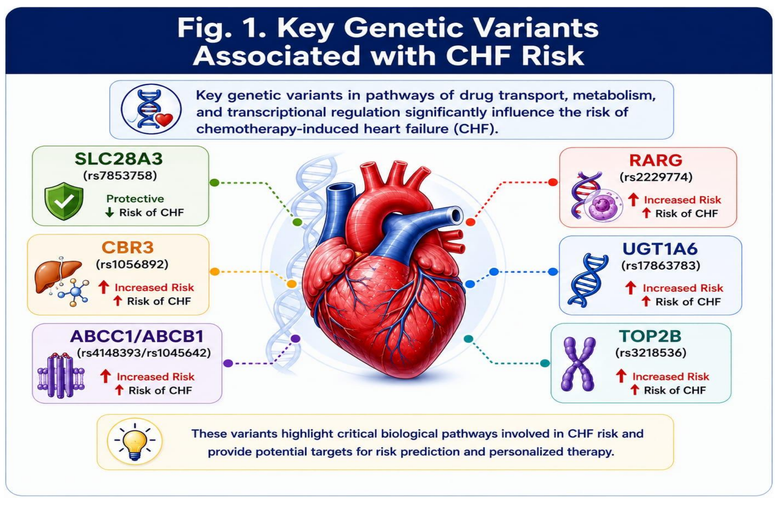
Chemotherapy-induced heart failure (CHF) is a serious adverse effect that can limit treatment efficacy and compromise patient survival. Individual susceptibility varies significantly, and genetic factors are thought to play a crucial role. This systematic review synthesizes the current evidence on genetic variants associated with an increased risk of CHF, focusing on their biological pathways, clinical relevance, and potential for risk stratification.

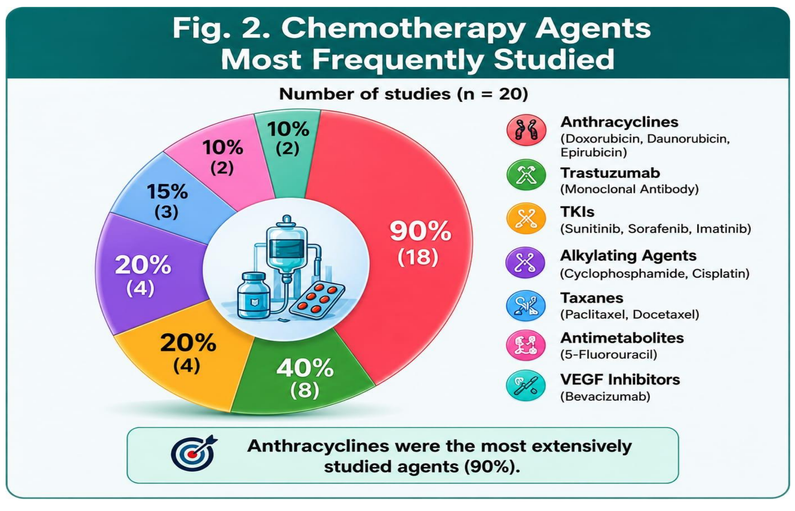
METHOD

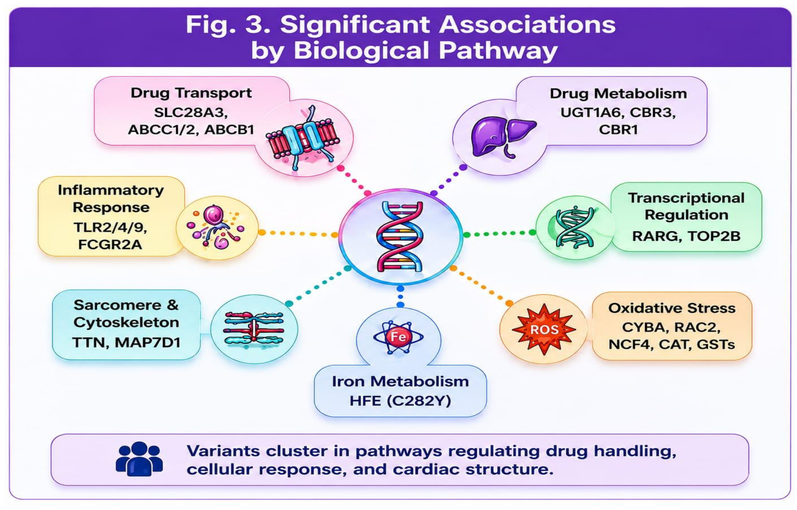
We conducted a systematic review of studies published from database inception to January, 2026. Our search was performed exclusively in the PubMed electronic database for English-language studies. The review encompassed various study designs, including cohort studies, case-control studies, genome-wide association studies (GWAS), and systematic reviews, to comprehensively capture the genetic evidence on this condition. A total of 20 studies were selected for final inclusion from an initial 1,405 identified records after a rigorous, multi-stage screening process.

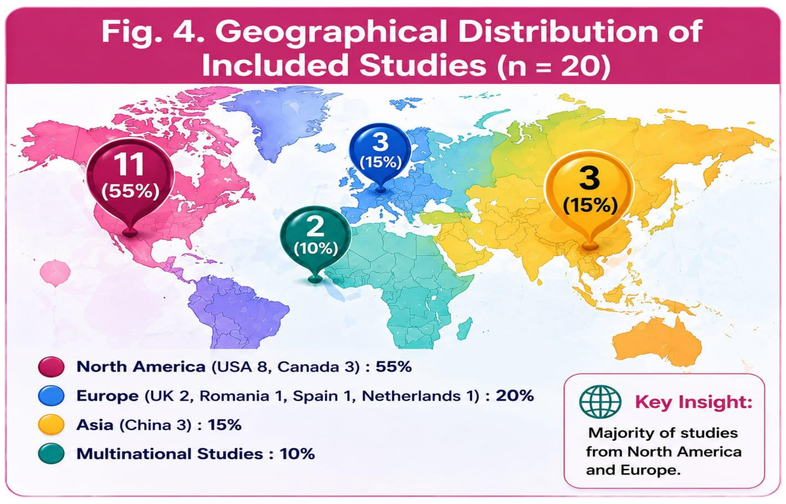
RESULTS & DISCUSSION

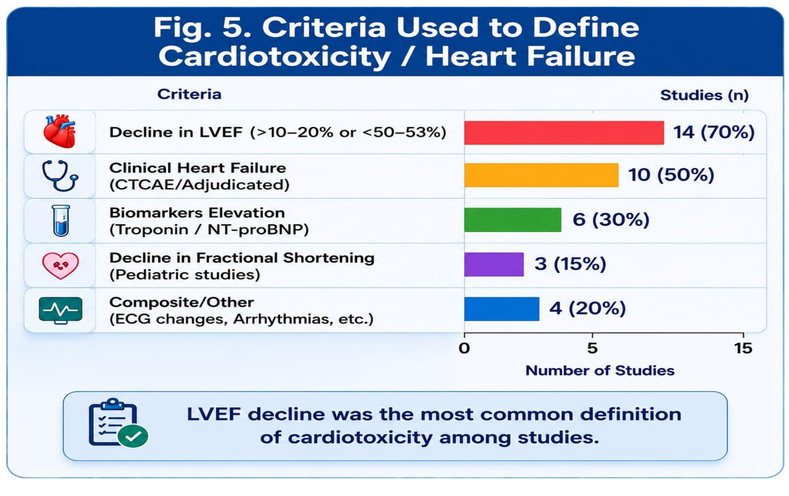
20 studies were included. The research landscape was dominated by North America and Europe, with a significant focus on anthracyclines. Key genetic variants were identified in pathways governing drug transport (e.g., SLC28A3 rs7853758, protective), drug metabolism (e.g., CBR3 rs1056892, risk; UGT1A6 rs17863783, risk), and transcriptional regulation (e.g., RARG rs2229774, risk). The most robust evidence supports the consideration of genetic testing for RARG, SLC28A3, and UGT1A6 in pediatric patients before anthracycline therapy. Common limitations across studies included small sample sizes, heterogeneous definitions of cardiotoxicity, and a lack of ethnic diversity.

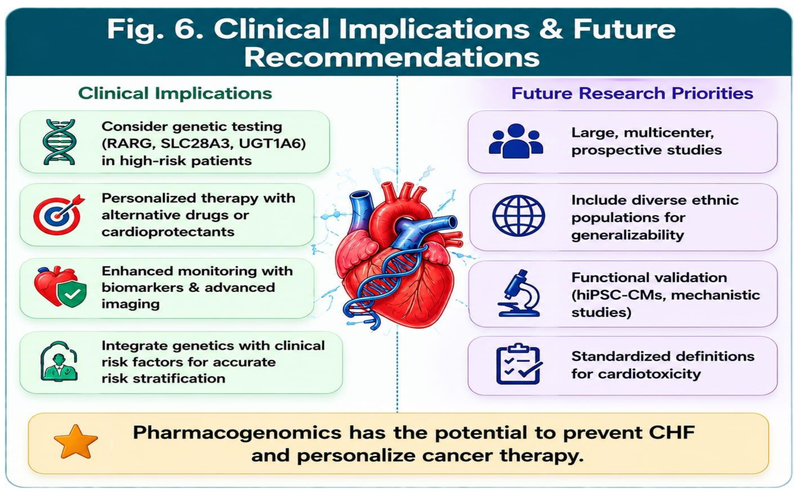












CONCLUSION

Specific genetic variants significantly modulate the risk of CHF. The implementation of pharmacogenomic testing, particularly in high-risk populations, holds promise for personalizing cancer therapy and preventing cardiotoxicity. Future research must focus on large, multi-ethnic prospective studies, standardized endpoints, and functional validation of genetic associations to translate these findings into routine clinical practice.