ISSN 1949-8462 (online)

# World Journal of *Cardiology*

*World J Cardiol* 2024 November 26; 16(11): 619-672

Published by Baishideng Publishing Group Inc

World Journal of Cardiology

Contents

Monthly Volume 16 Number 11 November 26, 2024

## Contents

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## RESPONSIBLE EDITORS FOR THIS ISSUE



**NAME OF JOURNAL**
*World Journal of Cardiology*

**ISSN**
ISSN 1949-8462 (online)

**LAUNCH DATE**
December 31, 2009

**FREQUENCY**
Monthly

**EDITORS-IN-CHIEF**
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**EDITORIAL BOARD MEMBERS**
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**PUBLICATION DATE**
November 26, 2024



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*World J Cardiol* 2024 November 26; 16(11): 632-643

DOI: 10.4330/wjc.v16.i11.632

ISSN 1949-8462 (online)

*REVIEW*

# Cardiovascular and nonalcoholic fatty liver disease: Sharing common ground through SIRT1 pathways

Kenneth Maiese

**Specialty type:** Cardiac and cardiovascular systems

**Provenance and peer review:** Invited article; Externally peer reviewed.

**Peer-review model:** Single blind

**Peer-review report's classification**
**Scientific Quality:** Grade C
**Novelty:** Grade B
**Creativity or Innovation:** Grade B
**Scientific Significance:** Grade B

**P-Reviewer:** Wang Z

**Received:** July 13, 2024
**Revised:** August 27, 2024
**Accepted:** October 10, 2024
**Published online:** November 26, 2024
**Processing time:** 109 Days and 16.6 Hours

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## Abstract

As a non-communicable disease, cardiovascular disorders have become the leading cause of death for men and women. Of additional concern is that cardiovascular disease is linked to chronic comorbidity disorders that include nonalcoholic fatty liver disease (NAFLD). NAFLD, also termed metabolic-dysfunction-associated steatotic liver disease, is the greatest cause of liver disease throughout the world, increasing in prevalence concurrently with diabetes mellitus (DM), and can progress to nonalcoholic steatohepatitis that leads to cirrhosis and liver fibrosis. Individuals with metabolic disorders, such as DM, are more than two times likely to experience cardiac disease, stroke, and liver disease that includes NAFLD when compared individuals without metabolic disorders. Interestingly, cardiovascular disorders and NAFLD share a common underlying cellular mechanism for disease pathology, namely the silent mating type information regulation 2 homolog 1 (SIRT1; *Saccharomyces cerevisiae*). SIRT1, a histone deacetylase, is linked to metabolic pathways through nicotinamide adenine dinucleotide and can offer cellular protection though multiple avenues, including trophic factors such as erythropoietin, stem cells, and AMP-activated protein kinase. Translating SIRT1 pathways into clinical care for cardiovascular and hepatic disease can offer significant hope for patients, but further insights into the complexity of SIRT1 pathways are necessary for effective treatment regimens.

**Key Words:** AMP-activated protein kinase; Cardiovascular disease; Diabetes mellitus; Erythropoietin; Metabolic-dysfunction-associated steatotic liver disease; Nicotinamide; Nicotinamide adenine dinucleotide; Nonalcoholic fatty liver disease; Silent mating type information regulation 2 homolog 1 (*Saccharomyces cerevisiae*); Stem cells

**Core Tip:** Cardiovascular disease is the principal cause of non-communicable diseases with individuals succumbing to heart disease every thirty-three seconds and has a significant comorbidity with nonalcoholic fatty liver disease (NAFLD). These two disorders impact millions of individuals across the globe, yield significant disability and death to individuals, and have a common underlying cellular pathway with silent mating type information regulation 2 homolog 1 (*Saccharomyces cerevisiae*) that may offer innovative prospects for the treatment of both cardiovascular disorders and NAFLD.

**Citation:** Maiese K. Cardiovascular and nonalcoholic fatty liver disease: Sharing common ground through SIRT1 pathways. *World J Cardiol* 2024; 16(11): 632-643
**URL:** https://www.wjgnet.com/1949-8462/full/v16/i11/632.htm
**DOI:** https://dx.doi.org/10.4330/wjc.v16.i11.632

## INTRODUCTION

Non-communicable diseases (NCDs) lead to disability and death in a significant spectrum of individuals worldwide and encompass disorders that include cardiac disease, cancer, trauma, respiratory disease, stroke, Alzheimer's disease, diabetes mellitus (DM), influenza and pneumonia, kidney disease, and suicide[1-3]. Cardiac and cardiovascular disorders are the most prominent of NCDs as the leading cause of death for both women and men and greater than one million individuals suffer a heart attack every year[4-7]. Several therapeutic pathways can lessen the severity of cardiovascular disease by addressing tobacco exposure, inadequate nutrition, hypertension, serum cholesterol values, obesity, infection, and the existence of DM[8-11].

In fact, DM not only is a risk factor for cardiovascular disease, but also for liver disease and the development of nonalcoholic fatty liver disease (NAFLD), a significant comorbidity of cardiovascular disorders[12-14]. NAFLD, also known as metabolic-dysfunction-associated steatotic liver disease, is increasing in presence throughout the world and this has occurred with concurrent the rise in DM globally[15-19]. NAFLD is a chronic disorder of the liver with excessive fat accumulation and is associated with at least one metabolic risk factor, such as obesity, DM, hypertension, elevated serum triglycerides, low serum high-density lipoprotein (HDL) cholesterol, and advanced age[13,20-23]. A recent paper by the authors Batta and Hatwal[24] brings to light the clinical link between cardiovascular disease and NAFLD and that in combination these disorders can lead to an increased risk of major impairment in cardiovascular function as well as cerebral function, such as stroke.

## THE METABOLIC AND PROTECTIVE PATHWAYS FOR SILENT MATING TYPE INFORMATION REGULATION 2 HOMOLOG 1 (*SACCHAROMYCES CEREVISIAE*)

Given that metabolic disorders, such as DM, are common risk factors for the development of cardiovascular disease and NAFLD, it is significant to note that a common underlying cellular pathway that can oversee both of these disorders involves the silent mating type information regulation 2 homolog 1 (SIRT1; *Saccharomyces cerevisiae*)[25-27]. SIRT1 is present in the heart, skeletal muscle, pancreas, liver, brain, spleen, and adipose tissue[13,25,28-30]. SIRT1 is a member of the sirtuin family (sirtuin 1) and is a histone deacetylase that promotes transcription of DNA through the transfer of acetyl groups from e-N-acetyl lysine amino acids to DNA histones[31-33]. Nicotinamide adenine dinucleotide ($NAD^+$), a coenzyme, functions as a SIRT1 substrate[22,28,34,35]. SIRT1 can control metabolic homeostasis[36,37] and functions closely with $NAD^+$ and the vitamin nicotinamide[22,23,34,35]. As the amide form of the vitamin B3 (niacin), nicotinamide is the precursor for $NAD^+$[31,38-40]. SIRT1 oversees nicotinamide phosphoribosyl-transferase that is required for $NAD^+$ production and is tied to circadian clock gene rhythms[41]. It is important to note that sufficient levels of $NAD^+$ are required to prevent vascular disease, dementia, and mitochondrial function[21,42,43]. Pools of cellular $NAD^+$ are susceptible to fluctuation with aging and circadian clock gene function[9,44-46]. SIRT1 activation that leads to increased levels of $NAD^+$ have been reported to lessen cardiac injury, maintain metabolic homeostasis, and reduce cellular inflammation[47-49].

In addition to maintaining metabolic homeostasis, SIRT1 also controls growth factor function through the activity of $NAD^+$[50-53]. Erythropoietin (EPO) employs SIRT1 to preserve synaptic connections for memory function[54,55], enhance survival of cardiovascular cells[56,57], and protect against toxic events with liver cells[52,58]. At the cellular level, EPO oversees SIRT1 activity to prevent mitochondrial membrane depolarization, activation of BCL2 associated agonist of cell death (Bad), and caspase pathway activity[59-61].

## A COMMON CELLULAR PATHWAY INVOLVING SIRT1 FOR CARDIOVASCULAR DISEASE AND NAFLD

SIRT1 offers a clinical target for both cardiovascular disease and hepatic disease that can lead to NAFLD (Figure 1). In regard to cardiovascular disease, SIRT1 can foster the function of stem cells[62-64] and enhance cardiac function and repair[65,66]. SIRT1 can improve the function of endothelial cells[42,67,68], reduce coronary artery disease[69,70], inhibit

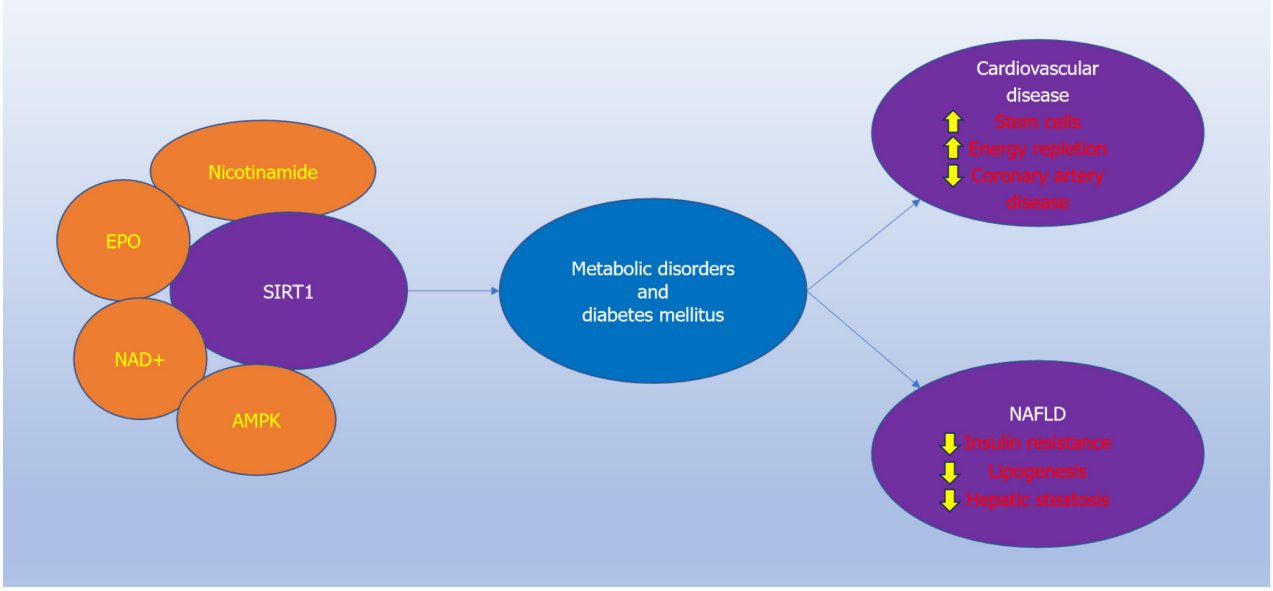


**Figure 1 Silent mating type information regulation 2 homolog 1 is an integral pathway for metabolic disorders such as diabetes mellitus and the clinical outcomes for cardiovascular disease and nonalcoholic fatty liver disease.** The silent mating type information regulation 2 homolog 1 (SIRT1; *Saccharomyces cerevisiae*) and the complementary pathways of SIRT1 that include nicotinamide, erythropoietin, nicotinamide adenine dinucleotide, and AMP-activated protein kinase function to maintain glucose homeostasis during metabolic disorders such as diabetes mellitus, Ultimately, the pathways of SIRT1 impact cardiovascular disease by promoting stem cell function, enhancing cellular energy repletion, and preventing coronary artery disease and influence nonalcoholic fatty liver disease by inhibiting insulin resistance, lipogenesis, and hepatic steatosis. SIRT1: Silent mating type information regulation 2 homolog 1; $NAD^+$: Nicotinamide adenine dinucleotide; EPO: Erythropoietin; AMPK: AMP-activated protein kinase; NAFLD: Nonalcoholic fatty liver disease.

cardiac injury during DM and metabolic disorders[37,71-73], and assist with cellular energy repletion[74,75]. Through the prevention of cellular senescence[76] to allow progenitor cell differentiation with SIRT1, cardiovascular cells are afforded the ability for heightened resistance to injury[43,67,77,78]. In the broader cardiovascular systems, SIRT1 can oversee programmed cell death with apoptosis and autophagy, control cardiac remodeling through increased mitochondrial biogenesis, limit myocardial injury, reduce insulin resistance, and prevent cardiac hypertrophy[4,30,79-83].

Liver function is dependent upon both cellular metabolism and SIRT1 pathways[13,45,84,85]. Insulin sensitivity and the maintenance of mitochondrial function require SIRT1 activation[86-89]. Activation of SIRT1 can control hepatocyte processing of lipids and glucose level maintenance to lessen the risk of the onset of metabolic syndrome dysfunction[30,31,90]. If SIRT1 activity is limited in the pancreas and liver, insulin resistance can ensue especially during high fat consumption[13,91-93]. SIRT1 also can control de novo lipogenesis and resolve hepatic steatosis that may lead to NAFLD and require activation of related pathways of AMP-activated protein kinase (AMPK)[92,94-96]. The AMPK pathway is closely linked to SIRT1 and nicotinamide in overseeing cellular metabolic homeostasis[9,72,97-99]. In recent clinical studies, treatment with oleoylethanolamide, an endogenous peroxisome proliferator-activated receptor alpha agonist, in patients with NAFLD led to increased mRNA expression levels of SIRT1 with increases in HDL cholesterol and decreases in triglyceride levels, suggesting that SIRT1 is a therapeutic target for NAFLD[100]. In addition, increased exercise in patients with NAFLD may affect lipophagy, lipolytic pathways, and reduction in oxidative stress through SIRT1 activity [101]. Through SIRT1 pathways, exercise also affects cardiac fatty acid oxidation, tissue regeneration, improved metabolic status, dietary interventions for weight management, and reduction in age-related decline of cellular metabolic pathways [45,71,75].

## CONCLUSION

Cardiovascular disorders and NAFLD impact a significant number of individuals throughout the globe and share common aspects of underlying disease pathology related to cellular metabolic dysfunction and the intricate pathways of SIRT1 involving $NAD^+$, nicotinamide, trophic factors such as EPO, and AMPK. SIRT1 is an exciting clinical target for both cardiovascular disease and hepatic disorders, since SIRT1 activity can maintain cellular metabolic homeostasis, enhance stem cell function and differentiation, foster the survival of vascular endothelial cells, limit cardiac injury, control hepatocyte lipid production and insulin resistance, and limit hepatic steatosis that can result in NAFLD. Yet, the pathways of SIRT1 are complex and require intact cellular feedback pathways with nicotinamide, $NAD^+$, growth factors, and AMPK since lack of close biological control can lead to unwanted clinical outcomes such as tumorigenesis[4,26,87,102,103]. In addition, multiple pathways intersect with SIRT1 that involve cellular metabolic disease[21,35,97,104-109], apoptosis and autophagy[26,110,111], oxidative stress, inflammation, and mitochondrial impairment[25,76,111-121], and clock genes with Wnt proteins impairment[105,111,117,118,122-124]. SIRT1 offers exciting possibilities for the advancement of clinical care, but further elucidation of the protective pathways of SIRT1 for complex disorders such as cardio-

vascular disease, liver disorders, and metabolic dysfunction is necessary for the development of effective and safe clinical treatment strategies.

## FOOTNOTES

**Author contributions:** Maiese K conceived, designed, and wrote this article.

**Supported by** American Diabetes Association; American Heart Association; NIH NIEHS; NIH NIA; NIH NINDS; NS053956; and NIH ARRA.

**Conflict-of-interest statement:** The author reports no conflicts of interest for this article.



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**S-Editor:** Lin C
**L-Editor:** A
**P-Editor:** Zheng XM

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