Protección miocárdica

myocardial protection: Latest results from PubMed
URL: https://pubmed.ncbi.nlm.nih.gov/rss-feed/?feed_id=1lKnPBUS1ZVgVcAw3tPvoYngl6sK7K6o7ePuJpr6j_daulok04&utm_content=1lKnPBUS1ZVgVcAw3tPvoYngl6sK7K6o7ePuJpr6j_daulok04&utm_source=Other&ff=20250703115540&utm_medium=rss&v=2.18.0.post9+e462414
Actualizado: hace 10 semanas 4 días

Participation in a comprehensive cardiac rehabilitation program improves mid- and long-term prognosis in survivors of acute coronary syndrome

Jue, 07/03/2025 - 10:00

Am J Prev Cardiol. 2025 Jun 13;23:101042. doi: 10.1016/j.ajpc.2025.101042. eCollection 2025 Sep.

ABSTRACT

BACKGROUND: Cardiovascular disease is the leading cause of death in developed nations. While survival rates of myocardial infarction have improved in recent decades due to advanced treatment options, secondary prevention efforts are often neglected. This study evaluates the effects of participation in a comprehensive cardiac rehabilitation program (CCR) on survival in patients presenting with acute coronary syndrome (ACS).

METHODS: In Hungary, since January 2014, data on patients with ACS have been mandatorily entered into the National Myocardial Infarction Register. This Register now holds information on 155,000 ACS events involving over 130,000 patients. A retrospective analysis was performed on the data of said Register.

RESULTS: We examined data on 76,153 ACS cases that occurred from 2014 to 2019. For the purposes of this study, we focused on early survivors, and 66,905 patients were included in our analysis (alive 30 days after the index event). The main modifiable protective factors, analyzed by binary regression model, were percutaneous coronary intervention (PCI), direct admission to a PCI-capable hospital, and participation in a comprehensive cardiac rehabilitation (CCR) program. In Hungary, such programs include supervised physical exercise as well as patient education on smoking cessation, dietary changes, and medication compliance. Our study showed that participation in CCR programs was associated with a 42 % reduction in 1-year mortality for patients with ST-elevation myocardial infarction (STEMI) and improved long-term survival rates across various patient subgroups. Despite its efficacy, the participation rate in CCR was low, with only 21 % of eligible patients completing such programs. The lowest CCR participation rate was in non-ST-elevation myocardial infarction (NSTEMI) patients who did not undergo PCI; these patients also had the highest mortality rates. Factors predicting lower participation rates were older age, male gender, NSTEMI presentation, and lack of percutaneous coronary intervention (PCI).

CONCLUSION AND RELEVANCE: This study shows a significant survival benefit of participation in a comprehensive cardiac rehabilitation program in early survivors of ACS. Unexpectedly, this finding was contrasted by a very low participation rate in this highly effective and cost-effective intervention. Increasing awareness of CCR's benefits both amongst patients and providers, as well as increasing access to and availability of CCR should significantly improve survival rates following ACS.

PMID:40606515 | PMC:PMC12221284 | DOI:10.1016/j.ajpc.2025.101042

Categorías: Protección miocárdica

Cardioprotection Through Pharmacological Activation of Sirtuin 5 in a Murine Model of Acute Myocardial Infarction

Jue, 07/03/2025 - 10:00

Drug Des Devel Ther. 2025 Jun 27;19:5489-5505. doi: 10.2147/DDDT.S509337. eCollection 2025.

ABSTRACT

PURPOSE: Sirtuins (SIRTs) play a critical role in redox and metabolic regulation of the myocardium; however, the cardioprotective potential of SIRT5 in terms of infarct size (IS) reduction is still elusive. Herein, we employed the newly synthesized SIRT5-specific agonist, MC3215, developed by our group, to explore for the first time the pharmacological activation of SIRT5 as a target for cardioprotection.

METHODS AND RESULTS: In in vitro screening experiments, SIRT1 and SIRT5 agonists, namely, MC2606 and MC3215, at 1-20 μM were added to cardiomyoblasts (H9c2) and human endothelial cells (EA.hy-926) during 24 h hypoxia/2 h reoxygenation (H/R). SIRT1 and SIRT5 agonists mitigated H/R injury. Male C57BL/6J mice underwent 30 min ischemia (I) followed by 2 h or 24 h reperfusion (R). Mice received vehicle, the SIRT1 or SIRT5 agonists at 20 and 30 mg/kg at the 20th min of ischemia, and IS was quantified via triphenyl-tetrazolium chloride staining (n=5-7/group). MC3215-mediated SIRT5 activation reduced IS at 24 h R at 20mg/kg compared to controls (25.18±2.7% vs 38.80±4.7%). MC3215 treatment resulted in reduced protein malonylation in all experimental settings. Targeted mass-spectrometry-based metabolomics in the ischemic heart at the 10th min of R suggested increased fatty acid oxidation, as indicated by increased N3-Trimethyllysine and D-pantothenate. Concomitantly, molecular analysis indicated that the SIRT5 agonist activated AMPKα and Reperfusion Injury Salvage Kinase (RISK) pathway. Additionally, at 3 h reperfusion, MC3215 led to increased mitofusin 2 without altering apoptosis, paving towards improved mitochondrial dynamics. Co-administration of SIRT5 inhibitor, TW-37, abrogated MC3215-mediated cardioprotection.

CONCLUSION: SIRT5 pharmacological agonism emerges as a novel cardioprotective target, leading to RISK pathway activation and mitochondria-related metabolic effects, converging at salvaging ischemic myocardium from I/R injury.

PMID:40606000 | PMC:PMC12214431 | DOI:10.2147/DDDT.S509337

Categorías: Protección miocárdica

Association of Serum Thromboinflammatory Biomarkers With Atherosclerotic Plaques and Burden in a Community-Based Population

Jue, 07/03/2025 - 10:00

Arterioscler Thromb Vasc Biol. 2025 Jul 3. doi: 10.1161/ATVBAHA.125.322586. Online ahead of print.

ABSTRACT

BACKGROUND: The pathogenesis of atherosclerosis involves complex mechanisms, with inflammation playing a central role. Thromboinflammation may contribute to its development and progression. We investigated the association between circulating thromboinflammatory biomarkers and atherosclerotic plaques.

METHODS: Participants aged 50 to 75 years from the baseline survey of the PRECISE study (Polyvascular Evaluation for Cognitive Impairment and Vascular Events) were included. Serum levels of thromboinflammatory biomarkers (sGPVI [soluble glycoprotein VI]; sADAMTS13 [soluble a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13]; and sP-selectin) were assessed by ELISA and Luminex assays and categorized into quartiles based on their empirical

distribution within the study population. Eligible participants underwent imaging using computed tomography angiography and magnetic resonance imaging for coronary atherosclerosis, intracranial atherosclerosis, and extracranial atherosclerosis, respectively.

RESULTS: A total of 3019 participants (mean age, 61.20±6.68 years; 46.47% male) were analyzed. After multivariable adjustment, participants in the fourth quartile of sGPVI levels had higher odds of coronary atherosclerotic plaque burden (common odds ratio, 1.42 [95% CI, 1.14-1.76]; *P*=0.0017). Conversely, those in the highest sADAMTS13 quartile had lower odds of coronary plaques (odds ratio, 0.75 [95% CI, 0.60-0.93]; *P*=0.0094), as well as reduced coronary plaque burden, including segment involvement score (common odds ratio, 0.75 [95% CI, 0.61-0.93]; *P*=0.0087) and segment stenosis score (common odds ratio, 0.75 [95% CI, 0.61-0.93]; *P*=0.0086). No significant associations were observed between sGPVI or sADAMTS13 levels and intracranial or extracranial atherosclerosis. Likewise, after multivariable adjustment, no significant associations were observed between sP-selectin levels and coronary, intracranial, or extracranial atherosclerosis.

CONCLUSIONS: In our study, serum sADAMTS13 levels showed a negative association with both the presence and burden of coronary atherosclerosis, while sGPVI levels showed a positive association with the burden of coronary atherosclerosis. However, significant associations between the level of thromboinflammatory biomarkers, intracranial atherosclerosis, and extracranial atherosclerosis were not found.

PMID:40605745 | DOI:10.1161/ATVBAHA.125.322586

Categorías: Protección miocárdica

Association between statins use and mortality in critically ill patients with sepsis included myocardial injury: a retrospective cohort study

Jue, 07/03/2025 - 10:00

Eur J Med Res. 2025 Jul 2;30(1):543. doi: 10.1186/s40001-025-02711-3.

ABSTRACT

BACKGROUND: The treatment of patients with sepsis included myocardial injury (SIMI) remains a subject of debate. Given the pharmacological effects of statins in both sepsis and cardiovascular disease, this study aimed to investigate the association between statins use and the prognosis of SIMI.

METHODS: Patients with SIMI were primarily identified from the MIMIC-IV database. The patients were categorized into statins use and non-statins use groups, followed by propensity score matching (PSM) analysis to address baseline discrepancies between the groups. Univariate and multivariate Cox regression analyses were employed to assess the impact of statins on the prognosis of SIMI patients. The primary endpoint was 28-day all-cause mortality, with secondary outcomes including 90-day and 1-year all-cause mortality. To investigate whether the effects of certain factors vary across subgroups, subgroup analyse were performed.

RESULTS: The cohorts before and after PSM comprised 2550 and 1368 patients, respectively. In the PSM cohort, statins use was associated with a reduced 28-day all-cause mortality (hazard ratio [HR] = 0.65, 95% confidence interval [CI] 0.53-0.79, P < 0.001), 90-day all-cause mortality (HR = 0.65, 95% CI 0.54-0.77, P < 0.001), and 1-year all-cause mortality (HR = 0.72, 95% CI (0.53-0.79), P < 0.001). In addition, subgroup analyse suggested potential interactions between statins use and factors such as chronic kidney disease (CKD).

CONCLUSIONS: Statins use is associated with a reduced short-term and long-term all-cause mortality rates in patients with SIMI.

PMID:40605094 | DOI:10.1186/s40001-025-02711-3

Categorías: Protección miocárdica

Association of serum uric acid-to-high-density lipoprotein cholesterol ratio (UHR) with risk of myocardial infarction among individuals with diabetes: a cross-sectional analysis using data from NHANES 2005-2020

Jue, 07/03/2025 - 10:00

Eur J Med Res. 2025 Jul 2;30(1):554. doi: 10.1186/s40001-025-02845-4.

ABSTRACT

BACKGROUND: The serum uric acid-to-high-density lipoprotein cholesterol ratio (UHR) is a novel indicator of cardiometabolic health that has demonstrated strong predictive potential in various studies. However, the association between UHR and the occurrence of myocardial infarction (MI) among individuals with diabetes has not been well-established. This study aimed to assess the relationship between UHR and the presence of MI in diabetic individuals and to provide evidence for early identification of high-risk groups.

METHODS: This cross-sectional study included 7039 adult participants from the NHANES 2005-2020 data set. The association between UHR and MI risk was examined using UHR quartile grouping, multivariable logistic regression, and restricted cubic spline (RCS) analyses. Subgroup analyses were performed to evaluate whether the predictive value of UHR differed among population subgroups. All statistical procedures incorporated appropriate sample weights to ensure nationally representative estimates.

RESULTS: UHR was significantly elevated among participants with diabetes who had MI ($p < 0.001$). MI prevalence rose progressively across UHR quartiles (8.20% vs. 8.55% vs. 9.01% vs. 14.87%; $p < 0.001$). In the unadjusted model, each 1-unit increase in UHR was associated with a 4.5% higher odds of MI (OR = 1.045, 95% CI 1.021-1.071, $p < 0.001$). Participants in the highest quartile (Q4) had 1.957 times higher odds of MI compared to those in the lowest quartile (Q1) (95% CI 1.399-2.735, $p < 0.001$). RCS analysis revealed no significant non-linear association, suggesting a potentially linear relationship between UHR and the odds of MI. No significant interaction was observed across subgroups, such as sex, race, and education level (P for interaction > 0.05).

CONCLUSIONS: Elevated UHR was significantly associated with higher odds of MI in individuals with diabetes, demonstrating potential predictive value. As a simple and cost-effective indicator, UHR may assist in the early identification and stratification of individuals at higher likelihood of cardiovascular disease among people with diabetes; however, prospective studies are warranted to confirm its clinical utility.

PMID:40605038 | DOI:10.1186/s40001-025-02845-4

Categorías: Protección miocárdica

Curcumin mitigates high glucose-induced cardiac oxidative stress via Notch1 pathway activation

Mié, 07/02/2025 - 10:00

Sci Rep. 2025 Jul 2;15(1):23660. doi: 10.1038/s41598-025-09105-9.

ABSTRACT

This study aims to investigate the protective effects of curcumin (CUR) in high glucose (HG)-induced oxidative stress and apoptosis of primary cardiomyocytes by activating the Notch1 signaling

pathway. CUR is a natural polyphenol isolated from turmeric rhizomes and is known for its antioxidant, anti-apoptotic, and anti-inflammatory effects, particularly relevant in diabetes.Therefore, we used neonatal rat cardiomyocytes exposed to HG conditions, followed by treatment with CUR and DAPT, respectively. We detected and assessed myocardial cells viability and antioxidant enzyme activity by CCK-8 reagent and antioxidant enzyme kit. Apoptosis was detected by flow cytometry. The production of reactive oxygen species was detected by fluorescence labeling, and the expression of related genes and proteins was detected by qRT-PCR and Western blot. HG-induced primary rat cardiomyocytes not only increased apoptosis and ROS production, but also decreased the activity of antioxidant enzymes and the expression of Notch1 and Hes1 proteins. After pre-treatment by CUR, surprisingly, we found that CUR markedly improved viability of HG-treated cardiomyocytes. The results showed that CUR could inhibit the apoptosis of rat cardiomyocytes, inhibit the production of intracellular ROS, and increase the activity of antioxidant enzymes. Further, we found that CUR can upregulate the expression of Notch1 and Hes1 proteins and related genes, suggesting that the protective effect of CUR on HG-induced damage involves the Notch1/Hes1 signaling. These results suggest that CUR protects cardiomyocytes from HG-induced oxidative stress by activating Notch1 and its downstream target genes.

PMID:40603691 | DOI:10.1038/s41598-025-09105-9

Categorías: Protección miocárdica

Advances in Cardiovascular Pharmacotherapy. IV. Sodium-Glucose Cotransporter Type 2 Inhibitors, Part 2: Mechanisms for Myocardial Protection, Adverse Effects, and Perioperative Implications

Mié, 07/02/2025 - 10:00

J Cardiothorac Vasc Anesth. 2025 Jun 8:S1053-0770(25)00473-2. doi: 10.1053/j.jvca.2025.06.015. Online ahead of print.

ABSTRACT

This second part of a two-part review on the cardiovascular pharmacology of sodium-glucose cotransporter type 2 inhibitors (SGLT2i) describes the mechanisms that have been proposed to explain how these drugs improve outcomes in heart failure and myocardial infarction and discusses their adverse effects and perioperative implications for patients with or without diabetes undergoing major surgery. The mechanism(s) by which SGLT2i exert beneficial cardiovascular actions are incompletely understood at present, but they are most likely multifactorial in origin, as no single factor has been proven definitive when considered alone. SGLT2i increase the risk of genital mycotic infections and diabetic ketoacidosis (DKA) in patients with diabetes, but the drugs do not cause severe hypoglycemia requiring intervention, urinary tract infection, hypovolemia, or acute kidney injury, among other postulated adverse outcomes. Perioperative euglycemic DKA (euDKA) is rare, but vigilance for its occurrence is required when an anion gap metabolic acidosis develops despite normal or only modestly elevated glucose concentration. Current guidelines recommend withholding SGLT2i for at least 48 hours to minimize the risk of DKA before elective major surgery in patients with but not without diabetes. The guidelines further emphasize the need to maintain a high index of suspicion for DKA and euDKA when SGLT2i therapy cannot be stopped because of urgent or emergent surgery so that appropriate treatment can be promptly initiated to prevent morbidity and mortality.

PMID:40603214 | DOI:10.1053/j.jvca.2025.06.015

Categorías: Protección miocárdica

Intermedin₁₋₅₃ improves aging-associated cardiac remodeling and dysfunction via mitochondrial SIRT3-mediated SOD2 deacetylation

Mié, 07/02/2025 - 10:00

J Mol Cell Cardiol. 2025 Jun 30:S0022-2828(25)00109-9. doi: 10.1016/j.yjmcc.2025.06.011. Online ahead of print.

ABSTRACT

The aging-associated cardiac remodeling (AACR) is characterized by myocardial hypertrophy, fibrosis and cardiac dysfunction, which could be further aggravated by angiotensin II (Ang II) and pressure-overload in aged people. In this study, we aimed to investigate the roles and mechanisms of intermedin1-53 (IMD1-53), an endogenous peptide, in AACR in aged mice (18 months) with subcutaneous Ang II infusion (1000 ng/kg/min) for 2 weeks via osmotic pump or transverse abdominal aorta constriction (AAC) surgery for 4 weeks. In aged mice undergoing Ang II infusion or AAC surgery, the results showed that the mRNA and protein levels of IMD1-53 were significantly reduced, but the protein levels of its receptor complex components were increased; blood pressure (BP), myocardial hypertrophy, fibrosis, and cardiac dysfunction were notably aggravated; mitochondrial Sirtuin 3 (SIRT3) protein level, superoxide dismutase 2 (SOD2) activity and ATP production were remarkably decreased, but acetylated SOD2 (acSOD2) protein level was markedly increased when compared with the old mice. The above alterations could be effectively alleviated by the subcutaneous IMD1-53 administration (5 ng/kg/min) for 2 or 4 weeks. In Ang II-stimulated cardiomyocytes, IMD1-53 treatment improved Ang II-induced mitochondrial dysfunction and oxidative distress, up-regulated SIRT3 protein expression, and reduced acSOD2 protein level, which were notably weakened by SIRT3 knockdown. Moreover, SIRT3 deletion attenuated the protective effects of IMD1-53 on myocardial hypertrophy, fibrosis, and cardiac dysfunction in aged mice undergoing Ang II infusion. In addition, the effect of IMD1-53 on up-regulating SIRT3 expression was effectively inhibited by the antagonism of IMD1-53 receptor or blocking PI3K/Akt, cAMP/PKA and AMPK signaling pathways in vitro. Taken together, IMD1-53 alleviated AACR and cardiac dysfunction aggravated by Ang II or pressure-overload involving the improvement of mitochondrial oxidative distress through SIRT3-medaiated SOD2 deacetylation.

PMID:40602647 | DOI:10.1016/j.yjmcc.2025.06.011

Categorías: Protección miocárdica

Exploring the mechanism of TLR4/NF-kappaB signaling pathway in hypoxic myocardial injury: Implications for traditional Chinese medicine therapy

Mié, 07/02/2025 - 10:00

Fitoterapia. 2025 Jun 30:106721. doi: 10.1016/j.fitote.2025.106721. Online ahead of print.

ABSTRACT

Hypoxic myocardial injury is the core mechanism of many cardiovascular diseases and poses a serious threat to global public health. Its pathogenesis involves energy metabolism disorder, oxidative stress, inflammatory response, apoptosis regulation imbalance, and other links, resulting in myocardial dysfunction and damage. In recent years, a large number of studies have confirmed that the Toll-like receptor 4 (TLR4)/nuclear factor-κB (NF-κB) signaling network can regulate hypoxic myocardial injury by mediating HMGB1, MyD88, Caspase-3, HIF-1α, TNF-α, NLRP3, HSP70, etc. Traditional Chinese Medicine (TCM) and its active components have obvious advantages in the treatment of complex diseases such as hypoxic myocardial injury, and inhibiting the key target of TLR4/NF-κB signaling network is one of the mechanisms of myocardial protection. However, there are few systematic reviews and summaries in this field. Based on this, this review summarizes the regulatory mechanism of TLR4/NF-κB signaling pathway involved in hypoxic myocardial injury and the intervention effect of TCM in recent years, to provide a theoretical basis for basic research and new drug development of hypoxic myocardial injury.

PMID:40602631 | DOI:10.1016/j.fitote.2025.106721

Categorías: Protección miocárdica

Naringenin exerts antiarrhythmic action in septic cardiomyopathy by downregulating the CaMKⅡ/Drp1/Bcl-2 pathway

Mié, 07/02/2025 - 10:00

Phytomedicine. 2025 Jun 20;145:157006. doi: 10.1016/j.phymed.2025.157006. Online ahead of print.

ABSTRACT

BACKGROUND: Septic cardiomyopathy (SCM) is associated with sepsis and is often accompanied by progressive arrhythmia. Naringenin (Nar) is a natural dihydroflavonoid compound that plays a protective role in various cardiovascular diseases. Calcium/calmodulin-dependent kinase II (CaMKII) is a key therapeutic target in cardiac arrhythmias.

PURPOSE: This study investigated the effect of naringenin on arrhythmia and cardiac electrophysiology in SCM and explored the mechanism involved.

METHODS: Lipopolysaccharide was used to establish SCM in a mouse model and in an H9c2 cell line. The protective role of naringenin in SCM was investigated by pretreatment with naringenin, amiodarone, and a CaMKII inhibitor (KN-93). Cardiac function, susceptibility to arrhythmia, and electrophysiological changes were assessed in the mice using echocardiography, electrocardiography, and optical mapping techniques. Network pharmacology approaches, molecular docking, and molecular dynamics simulations were used to screen for pivotal targets. The mechanism(s) underlying the protective impact of naringenin on SCM were examined in vivo, ex vivo, and in vitro.

RESULTS: Naringenin protected against SCM by exerting anti-inflammatory effects, alleviating myocardial injury, improving cardiac dysfunction, reducing the susceptibility to arrhythmia, and stabilizing electrophysiology. Network pharmacology, molecular docking, and molecular dynamics simulations indicated that the key target protein of naringenin may be Bcl-2. Further studies confirmed that naringenin attenuated apoptosis, improved mitochondrial dysfunction, and downregulated the CaMKⅡ/Drp1/Bcl-2 pathway in SCM.

CONCLUSIONS: Naringenin attenuates the phosphorylation of Drp1 by inhibiting phosphorylation of CaMKⅡ, thereby ameliorating mitochondrial dysfunction, suppressing apoptosis, modulating myocardial electrophysiology, and ultimately reducing susceptibility to arrhythmia while improving cardiac function in SCM.

PMID:40602293 | DOI:10.1016/j.phymed.2025.157006

Categorías: Protección miocárdica

The protective mechanism of Hydroxysafflor yellow A for the treatment of stroke - heart - syndrome via activating the ZBP1-NLRP3 signaling pathway

Mié, 07/02/2025 - 10:00

Phytomedicine. 2025 Jun 18;145:157011. doi: 10.1016/j.phymed.2025.157011. Online ahead of print.

ABSTRACT

BACKGROUND: Hydroxysafflor yellow A (HSYA), the primary active constituent of Safflower, a traditional Chinese medicine, has demonstrated promising therapeutic potential in the treatment of cardiovascular and cerebrovascular injuries. However, the impact of HSYA on stroke-induced cardiac

syndrome and the underlying mechanisms remain to be elucidated.

METHODS: Laser super-resolution microscopy and transmission electron microscopy were employed to examine cerebral ischemic injury. Echocardiography and immunofluorescence techniques were utilized to assess cardiac function and inflammatory damage. Western blot analysis was conducted to measure the expression levels of apoptosis-related proteins in heart tissue.

RESULTS: HE revealed that SHS induced inflammatory infiltration in the myocardium. Echocardiographic findings indicated that SHS impaired cardiac function. ELISA results demonstrated that SHS led to elevated levels of norepinephrine and epinephrine. Transmission electron microscopy (TEM) observations confirmed that SHS resulted in mitochondrial damage within cardiac cells. Immunofluorescence analysis further showed that SHS facilitated the recruitment of cardiac macrophages, upregulated the expression of ZBP1 and NLRP3, and increased the production of inflammatory cytokines and inflammasomes. Co-immunoprecipitation experiments demonstrated that ZBP1 interacts with NLRP3. Inhibiting sympathetic overactivation exerts a protective effect on the heart. Furthermore, HSYA not only reversed the aforementioned conditions but also exerted protective effects on both cardiac and cerebral tissues. Immunofluorescence analysis revealed that HSYA inhibited the formation of the ZBP1 and NLRP3 complexes, as well as the inflammasome complex. Molecular docking studies indicated that HSYA and ZBP1 share the LYS-166 binding site, and protein docking results demonstrated that ZBP1 and NLRP3 also share this binding site. Mutations at this site diminished the protective efficacy of HSYA against SHS.

CONCLUSIONS: HSYA mitigates macrophage recruitment through the inhibition of the ZBP1-NLRP3 signaling pathway, thereby improving sympathetic nerve function, suppressing panoptosis, and alleviating SHS injury by competitively binding to the LYS-166 site of ZBP1 with NLRP3.

PMID:40602292 | DOI:10.1016/j.phymed.2025.157011

Categorías: Protección miocárdica

Interaction Between Aldosterone and Mineralocorticoid Receptor Antagonist: Findings From the EPHESUS Trial

Mié, 07/02/2025 - 10:00

JACC Heart Fail. 2025 Jul 1;13(8):102479. doi: 10.1016/j.jchf.2025.02.025. Online ahead of print.

ABSTRACT

BACKGROUND: Mineralocorticoid receptor antagonists (MRAs) block the activation of mineralocorticoid receptors by aldosterone, thereby mitigating cardiovascular risks. However, data on whether impact of aldosterone on outcomes differs with MRA use remain limited.

OBJECTIVES: The study aims to explore the associations between baseline aldosterone, its changes and outcomes, and their interaction with eplerenone.

METHODS: In a subset of the EPHESUS (Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study) trial, associations between baseline serum aldosterone concentrations, their changes from baseline to month 1, and outcomes were separately assessed in the eplerenone and placebo groups. The primary outcome was a composite of cardiovascular death or heart failure (HF) hospitalization.

RESULTS: Among 453 patients (mean age: 62 ± 11 years; 75% male), baseline median serum aldosterone was 5.6 ng/dL (Q1-Q3: 3.1-9.2 ng/dL). Higher baseline serum aldosterone was associated with primary outcome in the placebo group (HR per 1 ng/dL: 1.04 ng/dL [95% CI: 1.02-1.07 ng/dL]; P = 0.002), but not in the eplerenone group (HR per 1 ng/dL: 0.99 ng/dL [95% CI: 0.93-1.05 ng/dL]; P = 0.64; P for interaction = 0.048), and these associations persisted after covariate adjustment (ie, prior HF history and renal function). At month 1, eplerenone increased serum aldosterone more than

placebo (P < 0.001). High serum aldosterone changes (≥ median value) were associated with increased risk of primary outcome in the placebo group but not in the eplerenone group (HR: 3.48 [95% CI: 1.35-8.99]; P = 0.01 in placebo; HR: 0.81 [95% CI: 0.36-1.82]; P = 0.60 in eplerenone; P for interaction = 0.046), and these associations persisted after covariate adjustment.

CONCLUSIONS: In patients with left ventricular systolic dysfunction and/or HF after myocardial infarction, higher baseline or rising aldosterone levels were associated with increased risk of HF events. However, eplerenone mitigated aldosterone-associated risks.

PMID:40602178 | DOI:10.1016/j.jchf.2025.02.025

Categorías: Protección miocárdica

Network pharmacology and experimental verification: Rosmarinic acid alleviates doxorubicin-induced cardiomyocyte apoptosis by regulating BCL2L1

Mié, 07/02/2025 - 10:00

Hum Exp Toxicol. 2025 Jan-Dec;44:9603271251354890. doi: 10.1177/09603271251354890. Epub 2025 Jul 2.

ABSTRACT

PurposeThis study investigated the mechanism by which Rosmarinic acid (RA) may alleviate doxorubicin (DOX)- induced cardiomyocyte apoptosis.MethodsThe target genes of RA, DOX-related differentially expressed genes, and GEO database related genes were retrieved by bioinformatics analyses. The results of these analyses were further intersected to identify candidate genes. The protein-protein interaction network was constructed to develop the pharmacophore model. The molecular docking was simulated to determine the core target B-cell lymphoma 2-like 1 (BCL2L1) for subsequent molecular mechanism investigation *in vitro*. The effects of DOX and RA on the apoptosis of H9c2 cells were assessed using the CCK8 assay. The present study investigated the effect of RA on DOX-induced oxidative stress in cardiomyocytes. This investigation was conducted using an ELISA test and a DCFH-DA probe. The JC-1 probe was utilized to assess the effect of RA on DOX-induced cardiomyocyte mitochondrial membrane permeability. A Western blot assay was conducted to ascertain the activation of multiple signaling molecules, including those belonging to the BCL-2 and caspase-3 families, within the apoptosis pathway.ResultsA total of 17 differentially expressed genes (DEGs) were screened, and five genes were selected as hub DEGs. A subsequent KEGG enrichment analysis revealed that these DEGs were significantly enriched in various biological processes and pathways, including the MAPK signaling pathway, autophagy, apoptosis, and the TNF signaling pathway. The pharmacophore model and molecular docking of five candidate targets with RA were successfully established. It is noteworthy that DOX treatment led to a suppression of SOD and GSH levels, an exacerbation of oxidative stress, and a promotion of cardiomyocyte apoptosis. Furthermore, it has been demonstrated to suppress mitochondrial membrane permeability. Subsequent RT-qPCR analysis of the hub genes revealed that only *BCL2L1* exhibited significant alterations. Treatment with DOX altered the expression levels of apoptosis-associated proteins, BCL-2 family members, and caspase-3 family members. However, the administration of RA mitigated the deleterious effects of DOX on cardiomyocytes.ConclusionsThe protective effects of RA may against myocardial cell apoptosis are likely mediated through its activation of BCL2L1 and inhibition of caspase cascade protein expression in myocardial cells.

PMID:40600628 | DOI:10.1177/09603271251354890

Categorías: Protección miocárdica

Protective mechanism of polysaccharides from Phellinus linteus on H9c2 cardiomyocyte injury induced by hypoxia/reoxygenation

Mié, 07/02/2025 - 10:00

BMC Res Notes. 2025 Jul 1;18(1):263. doi: 10.1186/s13104-025-07308-x.

ABSTRACT

OBJECTIVE: This study aimed to explore the protective mechanism of Phellinus linteus polysaccharides (Phps) against hypoxia/reoxygenation (H/R)-induced injury in H9c2 cardiomyocytes, focusing on oxidative stress, apoptosis, and PI3K-AKT pathway regulation.

RESULTS: H9c2 cardiomyocytes were divided into control, H/R model, and Phps-treated groups (low/medium/high doses). The H/R model (established by exposing cells to hypoxia for 10 h followed by 4 h of reoxygenation.) induced significant injury: cell viability decreased, SOD activity reduced by 45%, and Bcl-2 expression declined at both mRNA and protein levels, while LDH activity increased by 66%, MDA content surged by 99%, and Bax expression (mRNA/protein) and p-PI3K and p-AKT levels were upregulated, with statistical significance ($P < 0.05$ vs. control). Compared to the H/R model group, the Phps treatment (low, medium, high) groups showed a significant increase in H9c2 cardiomyocytes viability, SOD activity, and mRNA and protein expression levels of Bcl-2. The LDH activity, MDA content, mRNA levels of Bax, and protein expression levels of Bax, p-PI3K and p-AKT significantly decreased, with statistical significance ($P < 0.05$). These results suggest that Phps may improve H/R induced damage in H9c2 cardiomyocytes by downregulating the ratio of Bax/Bcl-2 through the PI3K-AKT pathway.

PMID:40598329 | PMC:PMC12220461 | DOI:10.1186/s13104-025-07308-x

Categorías: Protección miocárdica

Protective effect of scutellarin on myocardial cells treated with high glucose

Mié, 07/02/2025 - 10:00

Biomed Rep. 2025 Jun 19;23(3):143. doi: 10.3892/br.2025.2021. eCollection 2025 Sep.

ABSTRACT

Diabetic cardiomyopathy (DCM) is an important cause of death in patients with diabetes. DCM can be simulated by cardiomyocyte injury induced by high glucose (HG) *in vitro*. Scutellarin (Scu) is a flavonoid extracted from *Erigeron breviscapus*. The H9c2 cell line was used as an *in vitro* model in the present study to investigate the mechanism by which Scu reduces HG-induced cardiomyocyte injury. Moreover, the present study aimed to provide scientific evidence on the mechanism by which Scu prevents DCM. The following groups were used: Control, model, Scu (50/100/200/400 µM) and curcumin. Following H9c2 cell adherence, the control and model groups were treated with normal medium; the Scu group cells were treated with different concentrations of Scu, whereas the curcumin group cells were treated with 4 µM curcumin for 4 h. Subsequently, the normal group was cultured in normal medium, and the other groups were treated with medium containing 100 mM HG for 48 h. The results indicated that Scu improved the morphology of H9c2 cells treated by HG, enhanced cell proliferative activity, reduced the production of reactive oxygen species and the induction of apoptosis. Moreover, Scu promoted the expression of Bcl-2 and inhibited the expression levels of caspase-3, cleaved caspase-3, caspase-9, cleaved caspase-9, caspase-12, Bax, NADPH oxidase (Nox)2 and Nox4. The findings indicated that Scu could inhibit oxidative stress and reduce the induction of apoptosis in cardiomyocytes, thereby alleviating HG-induced myocardial injury.

PMID:40599620 | PMC:PMC12209994 | DOI:10.3892/br.2025.2021

Categorías: Protección miocárdica

Harnessing mRNA technology for ischemic heart disease: a review of regenerative

and protective therapies

Mié, 07/02/2025 - 10:00

Cardiol Plus. 2025 Apr-Jun;10(2):117-128. doi: 10.1097/CP9.0000000000000118. Epub 2025 Jun 30.

ABSTRACT

As ischemic heart disease (IHD) remains the leading cause of mortality worldwide, there is an urgent need for innovative therapies that go beyond symptom management. The irreversible damage to cardiac tissue following myocardial infarction (MI) and the limited regenerative and proliferative capacity of adult cardiomyocytes (CMs) present significant challenges to the development of treatments capable of restoring cardiac function. This review focuses on emerging modified and non-modified messenger ribonucleic acid (mRNA)-based therapies, which offer targeted and transient protein expression. The studies reviewed here address three major therapeutic strategies: cardiac regeneration, aimed at inducing CM proliferation to restore lost cardiac muscle; cardiac protection, centered on anti-apoptotic and anti-inflammatory methods to mitigate further tissue damage; and cardiovascular regeneration, focused on promoting angiogenesis and restoring vascular integrity after injury. By examining mRNA and modified mRNA (modRNA) therapies across these three approaches, this review showcases mRNA's promising role in advancing muscular and vascular regenerative and protective therapeutics for IHD.

PMID:40599555 | PMC:PMC12208384 | DOI:10.1097/CP9.0000000000000118

Categorías: Protección miocárdica

Lactobacillus ameliorates myocardial ischemia reperfusion injury by attenuating apoptosis, inflammation, oxidative stress, and ferroptosis

Mié, 07/02/2025 - 10:00

BMC Med. 2025 Jul 1;23(1):377. doi: 10.1186/s12916-025-04203-x.

ABSTRACT

BACKGROUND: Myocardial ischemia/reperfusion (I/R) injury is a significant complication following acute myocardial infarction (AMI) and lacks effective therapies. The involvement of gut microbiota in regulating ferroptosis during myocardial I/R injury has not been thoroughly explored. This study aimed to investigate the effect of Lactobacillus on myocardial I/R injury and explore its potential mechanisms.

METHODS: One hundred fifty eight patients with ST-elevation myocardial infarction (STEMI) were enrolled in our prospective observational study. The correlations between Lactobacillus levels and myocardial injury markers, inflammatory factors, oxidative stress, and ferroptosis were evaluated. Furthermore, 30 rats were treated with Lactobacillus or vehicle control for 4 weeks, followed by myocardial I/R surgery. The protective effects of Lactobacillus against I/R injury were assessed by quantifying myocardial apoptosis, inflammation, oxidative stress, and ferroptosis. In addition, the above results were verified in vitro. The signaling pathways were investigated through the knockdown and overexpression of sirtuin 1 (Sirt1) and nuclear factor erythroid 2-related factor 2 (Nrf2).

RESULTS: In clinical study, Lactobacillus levels were significantly negatively correlated with myocardial injury markers, inflammatory factors, and malondialdehyde (MDA), but positively correlated with glutathione (GSH). In rats, Lactobacillus decreased the levels of myocardial injury markers, reduced the size of the myocardial infarction area, ameliorated the disordered myocardial cell arrangement, and improved cardiac function. In both in vivo and in vitro studies, Lactobacillus inhibited cardiomyocyte apoptosis by upregulated B-cell lymphoma-2 (Bcl-2), downregulated Bcl-2 associated X (Bax), and caspase-3. Furthermore, Lactobacillus decreased inflammatory factors, MDA,

reactive oxygen species (ROS) levels, and increased superoxide dismutase (SOD) activity. For ferroptosis, Lactobacillus upregulated the expression of glutathione peroxidase 4 (GPX4) and downregulated the expressions of acyl-CoA synthetase long-chain family member 4 (ACSL4) and transferrin receptor protein 1 (TfR1). Finally, knockdown and overexpression of Sirt1 and Nrf2 in vitro demonstrated that Lactobacillus exerted the effect by upregulating the Sirt1/Nrf2/HO-1 pathway.

CONCLUSIONS: Our findings reveals that Lactobacillus protects against myocardial I/R injury by inhibiting apoptosis, inflammation, oxidative stress, and ferroptosis through the Sirt1/Nrf2/HO-1 signaling axis, suggesting a novel probiotic-based therapeutic potential for I/R injury.

PMID:40598393 | PMC:PMC12218948 | DOI:10.1186/s12916-025-04203-x

Categorías: Protección miocárdica

Cardiovascular risks associated with adjuvant endocrine therapy in women with breast cancer: a population-based cohort study

Mié, 07/02/2025 - 10:00

BMC Cancer. 2025 Jul 1;25(1):1063. doi: 10.1186/s12885-025-14280-z.

ABSTRACT

BACKGROUND: Endocrine therapies, including tamoxifen (TMX) and aromatase inhibitors (AIs), are widely used in breast cancer treatment. This study aims to evaluate the cardiovascular risks associated with these therapies in different age groups of non-metastatic breast cancer patients.

METHODS: We conducted a cohort study using data from patients newly diagnosed with non-metastatic breast cancer. Patients were categorized into two ages (< 45 years and > 55 years) and then divided into groups based on whether they were newly receiving either TMX or AI. Follow-up continued until the first occurrence of a study outcome, death, or the last date of data collection (December 31, 2022). Primary outcomes were coronary artery disease, myocardial infarction (MI), ischemic stroke, hospitalization for heart failure (HHF), atrial fibrillation (AF), cardiovascular mortality, all-cause mortality, and major adverse cardiovascular events (MACE).

RESULTS: Among patients < 45 years old, 2837 were newly treated with TMX (n = 2370) or AI (n = 467). During a median follow-up of 8.4 years, the incidence rates of coronary artery disease (5.6 vs. 6.6 per 1000 person-years), myocardial infarction (1.0 vs. 1.7 per 1000 person-years), ischemic stroke (1.5 vs. 1.5 per 1000 person-years), and cardiovascular mortality (1.4 vs. 1.5 per 1000 person-years) were similar between TMX and AI users, with no significant differences in hazard ratios or cumulative incidence curves. However, AI users had a higher risk of HHF (Weighted HR, 3.08 [95% CI, 1.54-6.13], P = 0.001) and AF (P = 0.039) compared to TMX users. For MACE, there was a non-significant elevated risk (Weighted HR, 1.59 [95% CI, 0.90-2.81]), suggesting a trend toward increased risk. Among patients > 55 years old, 11,846 were newly treated with TMX (n = 6577) or AI (n = 5269). During a median follow-up of 5.0 years, AI users had a significantly increased risk of primary cardiovascular outcomes, including CAD, MI, ischemic stroke, HHF, AF, cardiovascular mortality, and MACE (all P < 0.01).

CONCLUSION: The study indicates that AIs are linked to a higher risk of cardiovascular events in post-menopausal patients compared to TMX. In younger patients, TMX's protective effect on cardiovascular outcomes may be less pronounced. Further large-scale studies are required to corroborate and address limitations related to menopausal status and residual confounding.

PMID:40597894 | DOI:10.1186/s12885-025-14280-z

Categorías: Protección miocárdica

The effect of carvacrol on kidney injury caused by isopreterenol-induced myocardial infarction

Mié, 07/02/2025 - 10:00

BMC Nephrol. 2025 Jul 1;26(1):295. doi: 10.1186/s12882-025-04245-6.

ABSTRACT

BACKGROUND: Myocardial infarction is a major cause of morbidity and mortality, often leading to heart and kidney dysfunction. Despite advancements in treatment, the link between heart and kidney damage is poorly understood. This study aims to evaluate the potential protective effect of Carvacrol, a natural bioactive compound, on kidney injury induced by myocardial infarction.

METHODS: In this experimental study, 32 male Wistar rats were divided into four groups: Control, Carvacrol (50 mg/kg), Myocardial Infarction (85 mg/kg isoproterenol), and Myocardial Infarction + Carvacrol (50 mg/kg Carvacrol + 85 mg/kg isoproterenol). Carvacrol was administered for six weeks, and myocardial infarction was induced with isoproterenol. Blood pressure, biochemical parametres (creatinin kinase, lactate dehydrogenase, urea, creatinine, GDF-15, IL-6), and kidney tissue histopathology were evaluated.

RESULTS: Biochemical analysis showed increased Creatinin Kinase and Lactate Dehydrogenase levels in the Myocardial Infarction group compared to controls($p = 0.023$, $p = 0.020$), with carvacrol reducing these markers. IL-6 and GDF-15 levels were elevated in both the Myocardial Infarction and Myocardial Infarction + Carvacrol groups ($p = 0.009$, $p < 0.001$). Blood pressure was significantly reduced in the Myocardial Infarction group. Histopathological examination revealed severe kidney damage in the Myocardial Infarction group, while Carvacrol treatment showed less kidney damage, with only mild tubular dilation and rare necrosis.

CONCLUSION: Carvacrol appears to have protective effects against kidney injury in myocardial infarction. It reduced myocardial injury markers and kidney damage, suggesting its potential therapeutic use in cardiorenal syndrome. Further studies are needed to understand its mechanisms and clinical applications in cardiovascular and renal diseases.

PMID:40597757 | DOI:10.1186/s12882-025-04245-6

Categorías: Protección miocárdica

Effects of ranolazine on angiogenesis and oxidant-antioxidant balance: an in vivo experimental model study

Mié, 07/02/2025 - 10:00

Sci Rep. 2025 Jul 1;15(1):21563. doi: 10.1038/s41598-025-08099-8.

ABSTRACT

Ranolazine is known for its antiarrhythmic, antianginal, anti-ischemic properties, as well as its favorable effects on glycemic control. This study aimed to evaluate the effects of ranolazine on oxidative-antioxidative balance and angiogenesis using an in vivo experimental model. A total of 40 Ross 308 chick embryos were used and randomly divided into four groups ($n = 10$ per group). On the eighth day of incubation, vascular density was assessed. Following vascular evaluation, 4-5 mL of albumen was aspirated using a syringe to measure oxidative stress markers. The groups were as follows: Control, Bevacizumab (BC), Ranolazine 10-4, and Ranolazine 10-5. Total antioxidant capacity (TAC) levels were significantly higher in the bevacizumab group compared to the control group ($p < 0.05$). Similarly, oxidative stress index (OSI) levels were also significantly elevated in the bevacizumab group ($p < 0.05$). Both Ranolazine 10-4 and 10-5 groups demonstrated significantly increased TAC levels compared to the control group ($p < 0.05$). In terms of angiogenesis scores,

bevacizumab exhibited a marked anti-angiogenic effect compared to control. However, no statistically significant difference was observed between the ranolazine groups and the control group regarding angiogenesis scores ($p > 0.05$). This study provides the first in vivo evidence that Ranolazine enhances total antioxidant capacity but does not influence angiogenesis in the CAM model. Future research should explore the molecular mechanisms underlying this effect.

PMID:40596615 | PMC:PMC12215869 | DOI:10.1038/s41598-025-08099-8

Categorías: Protección miocárdica

- 1
- 2
- 3
- 4
- 5
- 6
- 7
- siguiente ›
- última »