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EFFECT OF YOGA ON INFLAMMATORY MARKERS IN MYOCARDIAL INFARCTION AND HEART FAILURE

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ABSTRACT
Myocardial infarction (MI) and heart failure (HF) are leading causes of cardiovascular morbidity and mortality worldwide, with inflammation recognized as a central mechanism driving disease progression, adverse cardiac remodeling, and poor clinical outcomes. Elevated circulating inflammatory biomarkers, including C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and various interleukins, have been identified as independent predictors of cardiovascular risk and mortality. In recent years, yoga has emerged as a promising, evidence-based mind–body intervention with potential immunomodulatory and cardioprotective effects. This review aimed to evaluate the impact of yoga on inflammatory markers in patients with MI and HF. A comprehensive search of PubMed, Scopus, and Cochrane databases was conducted from inception to July 21, 2025. Randomized controlled trials involving human participants that assessed yoga interventions and reported inflammatory or immune-related outcomes were included. The primary outcomes comprised CRP, TNF-α, interleukins, cytokines, and total leukocyte count. Evidence from the included studies suggests that yoga may attenuate systemic inflammation by modulating the hypothalamic–pituitary–adrenal axis, reducing sympathetic overactivity, enhancing glucocorticoid receptor sensitivity, and suppressing pro-inflammatory signaling pathways such as NF-κB. In HF populations, yoga interventions were associated with reductions in inflammatory biomarkers, particularly IL-6 and CRP. At the same time, studies in post-MI patients demonstrated favorable effects on endothelial function, oxidative stress, and selected inflammatory indices. Nevertheless, interpretation of the findings is constrained by small sample sizes, heterogeneity in patient populations, variability in biomarker assessment, and differences in yoga protocols across studies. Despite these limitations, the available evidence indicates that yoga may serve as a valuable adjunct to conventional cardiovascular care by targeting inflammation, a fundamental pathological process in both MI and HF. Future large-scale, multicenter randomized trials with standardized interventions and long-term clinical endpoints are warranted to establish the role of yoga in contemporary cardiovascular rehabilitation.

KEYWORDS: Myocardial Infarction, Heart, Inflammation, Yoga.

INTRODUCTION
Myocardial infarction (MI), or "heart attack," is a life-threatening cardiovascular disease and the most severe clinical manifestation of coronary artery issues. [1] It affects millions worldwide and incurs significant social and economic burdens. [2] Deaths related to cardiovascular disease rose from 17.8 million in 2017 to 19.1 million in 2020, reaffirming CVD as the leading global cause of death, responsible for nearly half of all cardiovascular-related fatalities. [3][4] Prior research has shown notable disparities in health outcomes, with individuals of lower socio-economic status facing a higher risk of premature death [5]. Risk factors for cardiovascular diseases include obesity, sedentary behavior, hypertriglyceridemia, and inflammation-related markers, such as high-sensitivity C-reactive protein (hs-CRP). [6] The rising incidence of MI underscores inflammation as a key emerging risk factor. The progression of heart and vascular diseases involves inflammation, with immune cells, including macrophages, neutrophils, and lymphocytes, playing central roles. These cells release cytokines that either promote (pro-inflammatory) or suppress (anti-inflammatory) inflammation. Such immune responses significantly influence disease development and progression.
Previous studies have highlighted this association. [7][8] Widespread inflammation is recognized as a strong predictor of overall mortality. [9] Several Chronic conditions, including Cardiovascular disease [10], cancer [11], and

rheumatoid arthritis [12], are characterized by persistent low-grade inflammation. It can be assessed indirectly through biomarkers such as TNF-α (tumor necrosis factor-α), interleukins (IL-6, IL-10, IL-12), and acute-phase reactants such as C-reactive protein (CRP) and total leukocyte count. [13] Elevated CRP levels, a well-known inflammatory marker, are associated with a range of cardiovascular and inflammatory conditions, particularly acute myocardial infarction (AMI). [14] Extensive research emphasizes CRP's role in cardiovascular pathology, showing its potential for diagnosis, risk assessment, and targeted therapy. [15] [16] In the context of acute MI, inflammation plays a vital pathological role by contributing to adverse left ventricular remodelling, myocardial injury, and the development of heart failure. [17] Understanding the dynamics of post-AMI CRP and its relationship with clinical outcomes is crucial for improving patient care and developing targeted treatments.

With the growing prevalence of lifestyle-related disorders, there is a global shift toward evidence-based complementary and alternative therapies. Among these, yoga has emerged as a scientifically supported mind-body intervention. A growing body of empirical research suggests that yogic practices exert modulatory effects on systemic inflammation [18][19], contributing to the regulation of chronic low-grade inflammation in metabolic syndrome [17], cardiovascular diseases[21], autoimmune disorders [22], and even certain cancers.[23] [24] These findings highlight the potential of yoga as a non-pharmacological adjunct therapy, calling for further clinical and translational research to address the increasing issue of inflammation in myocardial infarction and heart failure. Ongoing investigation in this area is crucial for understanding the underlying mechanisms and refining yoga-based protocols for specific clinical conditions.

METHODS

We conducted this review with the help of medical librarians from PubMed, Scopus, and Cochrane. For all articles published in each database from its inception through 21 July 2025, we considered those for inclusion. The following keywords and their Boolean combinations were used: "yoga and myocardial infarction," "yoga and inflammation," "Heart attack and Inflammation, yoga and heart failure," and "yoga and inflammation in myocardial infarction." Additional manual searches were performed by screening the reference lists of relevant articles. Randomized controlled trials (RCTs) that evaluated the effects of yoga as an intervention on the immune system in myocardial infarction were included in this review. The RCTs involving human participants, regardless of age, gender, or underlying clinical condition, were included. Those RCTs comparing the effects of yoga as an intervention for at least one parameter of the immune system versus no yoga or usual standard of care were also included. The outcomes assessed were various immunological markers such as Interleukins (IL-4, IL-6, IL-10), CRP, TNF-α, cytokines, total leukocyte count, and markers of genetic expression. Systematic reviews, meta-analyses, cohort studies, case reports, and non-human studies were excluded.

Inflammation in Cardiovascular Disease

Inflammation plays a central role in the development and progression of cardiovascular disease, including MI (Myocardial Infarction) and HF (Heart Failure), from endothelial dysfunction to clinical syndrome [25] [26]. Recent studies have underlined the critical role of CVDs, demonstrating that lowering the inflammatory burden can lead to a reduction of future cardiovascular events. [27] Consistent with these findings, evidence from various clinical trials has shown that higher levels of circulating inflammatory markers, such as high-sensitivity C-reactive protein and interleukin-6 (IL-6), are independent predictors of atherosclerotic cardiovascular diseases. [28] [29] [30] Inflammatory cytokines have a dual role: initially protective, but when excessive or chronic, they become pathological. They have been used to predict cardiovascular disease based on traditional risk factors. [31] Understanding the inflammatory mechanisms in Cardiovascular disease provides a foundation for exploring alternative therapies.

Yoga as an Inflammatory Intervention

Rooted in the Vedas, yoga can be integrated into daily life to enhance holistic health and well-being. [32] Initially, yoga was practiced in ancient times as a means to attain Samadhi (higher states of consciousness), and today it is used as a powerful tool to promote health and wellness. It is now the most widely adopted mind–body therapy in the US, accounting for about 80% of participation across the three primary mind–body therapies: tai chi, yoga, and qigong. [33] Yoga is a discipline that combines muscular activity with inward-focused awareness of bodily sensations, breath, and flow of prana (energy). [34] Systematic reviews of yoga have demonstrated that it is effective, and in some cases superior, to exercise in improving various health-related outcomes. [35] Moreover, it has shown comparable benefits to traditional aerobic physical activity in reducing various risk factors of cardiovascular disease. [36] Yoga intervention may also positively impact inflammatory markers through multiple interconnected mechanisms. [37]

At the neuroendocrine level, negative emotions and stress can activate the hypothalamic-pituitary-adrenal (HPA) axis, increasing sympathetic discharge of hormones that trigger the fight-or-flight response. This rise in somatic sympathetic responses is associated with numerous pathological effects, including vascular inflammation leading to

CVDs, dangerous arrhythmias, and sometimes sudden cardiac death. [38] [39] Atrial fibrillation and ventricular tachycardia, linked to significant morbidity and mortality, are triggered and sustained by an activated sympathetic nervous system. [40] [41]

Prolonged stress results in sustained elevation of glucocorticoids, activating the nuclear factor B (NF-κB) pathway, which is a key regulator of the inflammatory response. [42] Studies have demonstrated that yoga influences the HPA axis by decreasing glucocorticoids and enhancing glucocorticoid receptor sensitivity. This regulatory effect can reduce or prevent the consequences of cardiovascular dysfunction and aging.[43] When NF-κB activity increases due to stress via activation of the sympathetic nervous system, it promotes the expression of genes that code for inflammatory cytokines such as IL-6, TNF-α, and IL-1β. [44] Mind–body practices like yoga directly inhibit the ability to suppress NF-κB, thereby decreasing the production of inflammatory genes and offering protective effects against stress-related inflammatory diseases. [45]

Inflammatory Response in Myocardial Infarction

After an acute myocardial infarction, the immune system initiates an inflammatory response to clear dead myocardial tissue and begin repair. [46] This is a highly complex process that involves a carefully coordinated interaction between various immune cells and inflammatory molecules. While this inflammation is essential, if it becomes excessive, it can cause myocardial damage. [47] Neutrophils are among the first responders, helping to eliminate necrotic tissue. They release ROS (reactive oxygen species) and proteolytic enzymes that aid in clearing necrotic tissue. [48] [49] In the subsequent phase, macrophages play a key role in the inflammatory response. The pro-inflammatory macrophages (M1) secrete TNF-α, interleukin (IL)-1β, and IL-6, which promote inflammation, while later, anti-inflammatory macrophages (M2) release cytokines like IL-10 and transforming growth factor-beta (TGF-β), aiding in resolution and tissue repair. [50] [51] Other immune cells, such as T regulatory cells and myeloid-derived suppressor cells, also help suppress excessive inflammatory responses. [52] Here, the balance between pro-inflammatory and anti-inflammatory cytokines is crucial for optimal healing, because excessive pro-inflammatory activity can increase fibrosis and cause extensive tissue damage, leading to structural changes in the heart and heart failure. [53] Conversely, insufficient inflammation may impair the clearance of necrotic tissue and delay healing. [54] [55] [56] Oxidative stress in myocardial infarction involves reactive oxygen species (ROS), which damage cellular components, exacerbate inflammation, and cause myocardial injury. ROS also promote the release of CRP in the liver, which not only acts as an inflammatory marker but also activates the NLRP3 inflammasome, increasing the inflammatory cascade through the production of IL-1β and IL-18. [57] Persistent inflammation after myocardial damage has been linked to poorer prognosis and an increased risk of heart failure.

Inflammation in Heart Failure

Inflammation is a part of the physiological response that plays an important role in the pathophysiology of Heart Failure. [58] [59] Inflammatory response in Heart failure can arise from various sources, including myocardial infarction and atherosclerosis, and systemic conditions like diabetes and obesity or obesity, or chronic infections. When the heart is damaged from any of these sources, the body releases danger signals like pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs), which activate immune receptors known as TLR4 and NLRP3. This activation triggers a complex network of immune responses that leads to the elevation of pro-inflammatory cytokines, such as TNF-α, IL-1, IL-6, IL-8, and IL-18, as well as acute-phase proteins like CRP (C-reactive protein). [60] [61]These cytokines contribute to worsening heart function by promoting myocardial fibrosis (stiffness of heart muscles), endothelial dysfunction, apoptosis, and weakening of the heart's ability to pump.[62] Research studies demonstrated that higher biomarker levels contribute to cardiac remodelling, affect multiple organs, and increase mortality in HF patients.[63][64] Higher levels of inflammatory markers show worse patient symptoms and a higher risk of hospitalization and death. [65]

Review of Studies

Despite increasing recognition of inflammation as a key contributor to the development and progression of myocardial infarction and heart failure, there remains a lack of clinical research exploring the anti-inflammatory potential in this area. To date, only four randomized controlled trials have directly evaluated yoga as an intervention focusing on modulating inflammatory markers in this population.

In the context of Acute Myocardial Infarction, a study by Patil et al. [66] examined the effects of a yoga care program on endothelial function, oxidative stress, and inflammatory markers. The study found significant improvements within the yoga group in endothelial dysfunction markers such as endothelin-1 (ET-1), asymmetric dimethylarginine (ADMA), and intercellular adhesion molecule-1 (ICAM-1) compared to the control group. Additionally, tumor necrosis factor-alpha and high-sensitivity C-reactive protein were assessed as key inflammatory markers in myocardial infarction patients. Results showed a marginal reduction in TNF-α in the yoga group compared to standard care (0.7±0.7 vs 0.8±0.7), but this difference was not statistically significant (p=0.52). Likewise, hs-CRP levels

showed a favorable trend (from 10.5 ± 22.1 to 4.0 ± 11.5), but the change was not statistically significant (p=0.29). These trends suggest that yoga may have anti-inflammatory effects on these markers. Still, the lack of statistical significance suggests that the intervention, study power, or sample size may need improvement to detect definitive effects.

In the study on heart failure, Pullen [67] observed inflammatory markers among the yoga group. Specifically, IL-6 (interleukin-6) levels showed a marked reduction from a baseline of 17.5 ± 5.9 mg/dL to 13.6 ± 4.5 mg/dL ($P < .001$), and CRP (C-reactive protein) levels declined from 2.18 ± 0.34 mg/dL to 1.75 ± 0.39 mg/dL ($P = .002$). Additionally, EC-SOD (extracellular superoxide dismutase) increased significantly from 540 ± 37 U/mL to 640 ± 67 U/mL ($P = .002$). On comparing the pre-post values of the yoga intervention group with the control group, significant differences were found in all three markers: CRP ($P = .002$), EC-SOD ($P < .001$), and IL-6 ($P<.001$), indicating a clear anti-inflammatory and antioxidative influence of yoga practice.

In a subsequent trial [68] conducted by the same research group among African American patients with heart failure, were observed statistically significant improvement in inflammatory markers was observed among participants in the yoga group. High-sensitivity C-reactive protein (hs-CRP) levels decreased from 2.4 ± 0.58 to 1.9 ± 0.4 mg/dL ($P < 0.001$) while Interleukin-6 (IL-6) concentrations declined from 19.6 ± 2.5 to 16.0 ± 2.1 mg/dL ($P < 0.001$).

Consistent with earlier Randomized Controlled Trials demonstrating the anti-inflammatory potential of yoga in heart failure, an Indian based study [69] examined the impact of a yogic lifestyle on CRP levels in heart failure patients. It demonstrated a reduction in CRP levels in both groups over the 12-week intervention period; however, the magnitude of the reduction was greater among patients receiving the yoga intervention. C-reactive protein (CRP) levels decreased by 49% in the yoga group, declining from a baseline value of 5.36 to 2.73 ($P < 0.001$), compared with the control group, which demonstrated a 36% reduction from 5.39 to 3.45 over the same period ($P < 0.01$). Overall, the findings suggest the incorporation of a yogic lifestyle as an adjunctive tool to provide additional anti-inflammatory benefits beyond standard medical management in heart failure patients.

Limitations of the Evidence

Despite growing interest in yoga as an alternative therapy for reducing inflammation in patients with heart failure and myocardial infarction, the current findings are constrained by several important methodological and contextual limitations that limit the strength and generalizability of the conclusion. The Primary limitation lies in the lack of standardization of yogic interventions given across trials, ranging from a structured yoga care program [66] to a broader yoga lifestyle [69], with considerable heterogeneity across variation in intensity, studies, differences in duration, intensity, and other yogic components (asana, pranayama, or meditation). This lack of homogeneity makes it difficult to determine dose-response assessment and also to observe whether the effect results from asanas or pranayama, or from confounding variables such as lifestyle changes and dietary modifications. A Notable discrepancy also exists among the four included studies in which only one trial assessed patients following acute myocardial infarction, while the remaining focused on heart failure Patients. This uneven distribution restricts the generalizability of evidence, particularly for acute coronary syndromes, where inflammatory responses differ substantially from chronic heart failure.

The trials included in this study were conducted with relatively small cohorts ranging from modest to moderate enrolment, which results in limited statistical power to detect changes in inflammatory biomarkers. This limitation can be evident in the study by Patil et al [66] in the Acute MI, where a reduction in TNF-α and hs-CRP was observed, but these changes did not achieve statistical significance. Insufficient sample size increases the risk of Type II error and may lead to underestimation of the true anti-inflammatory response of yoga.

The generalizability of the current findings is also restricted by the inflammatory markers studied across the trials. Heart failure trials consistently assessed IL-6, hs-CRP, whereas Acute MI trials measured TNF-α and CRP only. This absence of a standardized biomarker panel limits cross-study comparisons and prevents meta-analytic conclusions. Moreover, Potential confounders across all the studies, such as medication, lifestyle changes, dietary modifications, and other psychosocial factors, were not uniformly reported or controlled.

Additionally, the study population is geographically and demographically polarized, focusing predominantly on single centres in India or specific African American patients, which restricts the applicability of findings to broader, clinically representative cardiovascular populations. As baseline levels of Inflammatory markers, particularly hs-CRP, vary across genetic and socioeconomic factors, the current absence of large multicentre, multi-ethnic control trials remains a limitation to incorporating yoga into global clinical rehabilitation guidelines.

CONCLUSION

The analysis of these recent trials shows that yoga's impact on inflammatory markers is limited, but emerging randomized evidence may be promising for modulating inflammatory biomarkers in patients with heart failure and acute myocardial infarction. Across the four randomized controlled trials, yogic interventions showed consistent signs of benefit in reducing inflammation, especially in heart failure, as indicated by the significant decrease in key

biomarkers like high-sensitivity C-reactive protein and interleukin-6. Although the randomized data in the post-MI population remain inconclusive, with results suggesting that inflammatory markers did not reach statistical significance, this may be due to insufficient intervention duration and limited sample size.

Collectively, yoga-based interventions for acute myocardial infarction and chronic heart failure support the biological plausibility and are clinically relevant as a non-pharmacological adjunct capable of guiding medical therapy through autonomic, neuroendocrine, and stress-related pathways. However, these convergent findings remain moderate at best, as the strength of the current evidence is constrained by a small study population, a single centre, heterogeneity in protocols, short follow-up duration, and variability in the selection of biomarkers, importantly none of the studies measured whether improvents in biomarkers translate into meaningful clinical outcomes such as mortality, rehospitalization, recurrent myocardial infarction or major adverse cardiovascular events (MACE). Within this framework, yoga should not be viewed as a replacement for conventional pharmacological treatment, but rather as a culturally adaptable, cost-effective, and safe intervention, particularly valuable in populations with limited access to standard rehabilitation programs.

Despite these limitations, the available evidence supports a potential adjunctive role for yoga to complement standard medical protocols by addressing inflammation, a fundamental pathophysiological proceess in both heart afailure and acute myocardial infarction. Moving forward, the next generation of yoga research in cardiology must prioritize large multicentre randomized controlled trials using standardized yoga interventions and long-term clinical endpoints to inform global clinical guidelines. Until then, yoga should be addressed as a safe mind-body therapy that addresses psychoneuroimmunological improvements in prognosis and routine cardiovascular care.

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