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The Role of Diet and Nutrition in Heart Failure Care

Mateusz Kasprzak, ORCID <https://orcid.org/0009-0000-0639-1055>

E-mail mb.kasprzak1@gmail.com

University Hospital in Poznań, ul. Przybyszewskiego 49, 60-355 Poznań, Poland

Kacper Godyń-Müller, ORCID <https://orcid.org/0009-0008-1907-4671>

E-mail godyn.kacper@gmail.com

University Hospital in Poznań, ul. Przybyszewskiego 49, 60-355 Poznań, Poland

Michał Grześkowiak, ORCID <https://orcid.org/0009-0006-0379-3204>

E-mail poczta.michal.grzeskowiak@gmail.com

University Hospital in Poznań, ul. Przybyszewskiego 49, 60-355 Poznań, Poland

Hanna Pelant, ORCID <https://orcid.org/0009-0001-1059-1727>

E-mail hannapelant@gmail.com

Centrum Medyczne HCP, ul. 28 Czerwca 1956 r. 194, 61-485 Poznań, Poland

Martin Zeigner, ORCID <https://orcid.org/0009-0009-0886-9927>

E-mail mt.zeigner@gmail.com

Independent Public Health Care Institution of The Ministry of the Interior and Administration
in Poznan, ul. Dojazd 4, 60-631 Poznań, Poland

Joanna Błaszczyk, ORCID <https://orcid.org/0009-0001-4467-7684>

E-mail asiajb2@gmail.com

Independent Public Health Care Institution of The Ministry of the Interior and Administration
in Poznan, ul. Dojazd 4, 60-631 Poznań, Poland

Bartosz Kulak, ORCID <https://orcid.org/0009-0005-0291-9525>

E-mail bartoszkulak5@gmail.com

Poznan University of Medical Sciences, ul. Fredry 10, 61-701 Poznań, Poland

Michał Brzeziński, ORCID <https://orcid.org/0009-0000-3894-5622>

E-mail brzezinskimichal15@gmail.com

Poznan University of Medical Sciences, ul. Fredry 10, 61-701 Poznań, Poland

Zuzanna Majchrzak, ORCID <https://orcid.org/0009-0006-1065-275X>

E-mail zuzanna.majchrzak26@gmail.com

Poznan University of Medical Sciences, ul. Fredry 10, 61-701 Poznań, Poland

Klaudia Pierzyńska, ORCID <https://orcid.org/0009-0003-8703-3381>

E-mail kpierzynska747@gmail.com

Pomeranian Medical University in Szczecin, ul. Rybacka 1, 70-204 Szczecin, Poland

Corresponding Author

Mateusz Kasprzak

E-mail mb.kasprzak1@gmail.com

Abstract

Introduction. Heart failure is a complex state of systemic metabolic dysfunction characterized by progressive myocardial energy deficiency. Traditionally managed through pharmacological and mechanical means, the role of nutritional status has emerged as a primary determinant of disease progression and clinical stability.

Methodology. A review was conducted using PubMed, Google Scholar and ResearchGate. Search terms included: heart failure, sodium restriction, Mediterranean diet, DASH diet, mitochondrial bioenergetics, gut-heart axis and nutrition. Studies and clinical guidelines published between 2015 and 2026 were reviewed.

Results. Evidence from recent trials indicates that aggressive sodium restriction does not significantly reduce mortality and may stimulate compensatory neurohormonal mechanisms. In contrast, dietary patterns like the Mediterranean and DASH diets attenuate cardiac remodeling, reduce systemic inflammation and improve left ventricular ejection fraction. Research into the gut-heart axis has identified microbial metabolites, such as trimethylamine

N-oxide and short-chain fatty acids, as key modulators of fibrosis and vascular health. Additionally, nutritional interventions targeting mitochondrial bioenergetics and protein intake have shown potential in mitigating sarcopenia and improving functional independence. However, the efficacy of these interventions is heavily dependent on patient behavioral readiness and individualized multidisciplinary support.

Conclusions. Nutritional management offers broad therapeutic potential in heart failure through a shift from restrictive paradigms toward precision nutrition. Nevertheless, the integration of structured behavioral models and stage-matched educational support is necessary to improve long-term adherence and clinical outcomes.

Keywords. heart failure, sodium restriction, Mediterranean diet, DASH diet, mitochondrial bioenergetics, gut-heart axis, nutrition, endothelial dysfunction, TMAO.

1. Introduction

Heart failure represents a profound and escalating global health crisis with a prevalence exceeding 64 million individuals (Savarese et al., 2023). The rising prevalence of heart failure is, paradoxically, a product of clinical success. As more patients survive acute ischemic events, the aging global population faces a growing trajectory of chronic cardiac dysfunction (Groenewegen et al., 2020). Pharmacological neurohormonal blockade and mechanical circulatory support remain the cornerstones of heart failure management. However the nutritional status is starting to be recognized not merely as a comorbidity, but as an important factor in disease trajectory (McDonagh et al., 2021). This state creates a systemic metabolic derangement that affects peripheral skeletal muscle, driving sarcopenia and cardiac cachexia (Habaybeh et al., 2021). Physical frailty correlates with mortality and the rate of rehospitalization, yet they are often exacerbated by the very dietary restrictions conventionally recommended in clinical practice (Alqurain et al., 2026). For decades, sodium and fluid restriction have served as the foundational non-pharmacological strategies for reducing preload. However, the efficacy of aggressive restriction (<1500 mg/day) is increasingly under investigation. Data from the SODIUM-HF trial indicates that while such measures may improve patient quality of life, they do not significantly reduce mortality or the

rate of hospital admissions (Ezekowitz et al., 2022). Excessive restriction may be counterproductive, potentially worsening the state of neurohormonal activation via the renin-angiotensin-aldosterone and sympathetic nervous systems (Mullens et al., 2024). Moreover, these patients are at risk of reducing total caloric and protein intake and accelerating malnutrition (Jefferson et al., 2015). Beyond simple restriction, focus is now placed on nutrient-dense patterns like the Mediterranean and DASH diets. By providing high levels of fiber and antioxidants, these diets help maintain healthy myocardial structure and may delay the progression of heart failure (Levitan et al., 2016). Additionally, the gut-heart axis has emerged as a key element of maintaining cardiac function with microbial metabolites like TMAO promoting inflammation and fiber-derived short-chain fatty acids exerting protective effects (Mylavarapu et al., 2026). Because long-term efficacy depends on behavioral sustainability, structured models like the Transtheoretical Model have proven more effective at improving clinical outcomes than standard dietary advice (Yuan et al., 2025). This review provides a multidisciplinary synthesis of current evidence regarding nutritional management in heart failure. It moves beyond the narrow focus on sodium restriction to explore how precision nutrition and individualized support can improve clinical stability and physical performance.

2. Methodology

We analyzed articles available in PubMed, Google Scholar and ResearchGate databases to perform a comprehensive review of the nutritional management and metabolic pathophysiology of heart failure. The following keywords were used in the search: heart failure, sodium restriction, Mediterranean diet, DASH diet, mitochondrial bioenergetics, gut-heart axis and nutrition. The search prioritized high-impact clinical trials, systematic reviews and meta-analyses published between 2015 and 2026.

3. Dietary Sodium in Heart Failure

The lack of clinical benefit from aggressive sodium restriction is often attributed to the complex neurohormonal response as in heart failure. The effective circulating volume is often reduced and the body attempts to compensate through the activation of the renin-angiotensin-aldosterone system (RAAS). Sodium depletion can trigger a paradoxical intensification of this

response, resulting in increased sympathetic nerve activity, inflammation and impaired endothelial function (Mullens et al., 2024; Zhu et al., 2022). This hormonal feedback loop may contribute to cardiorenal syndrome, exacerbating renal hypoperfusion and leading to hemodynamic decompensation (Chrysohoou et al., 2022; Mullens et al., 2024).

This excessive reduction can clinically manifest as increased fatigue, which limits physical activity and social participation (Zhu et al., 2022). Physiological findings postulate that the failing myocardium demands a careful electrolyte balance to maintain cardiac efficiency and that universal, rigid sodium caps may interfere with the body's adaptive mechanisms (Bianchi, 2020).

For decades, the reduction in dietary sodium to levels below 1500 mg per day was considered a non-negotiable standard of care for all patients with heart failure. However, current large-scale clinical trials and comprehensive meta-analyses have challenged the efficacy of this aggressive approach. The SODIUM-HF trial demonstrated that a dietary intervention targeting less than 1500 mg of sodium daily did not result in a significant reduction in all-cause mortality, cardiovascular-related hospitalizations, or emergency department visits over a 12-month period (Ezekowitz et al., 2022). Specifically, cardiovascular hospitalization rates were comparable between the low-sodium group and the usual care group (Ezekowitz et al., 2022; Colin-Ramirez et al., 2024).

The neutral findings of individual trials are reinforced by systematic reviews of the broader evidence base. The effect on all-cause mortality alone remained statistically neutral in most analyses, however, a non-significant trend toward harm was observed in patients with established heart failure when levels were pushed to restrictive extremes (Alqurain et al., 2026; Urban et al., 2024). Furthermore, observational data consistently suggest that both excessively high and low intakes are associated with increased risk (Alqurain et al., 2026; Mullens et al., 2024).

While the impact on mortality is recognized as neutral, the evidence regarding quality of life (QoL) and functional status is more nuanced. Research demonstrates that patients in the low-sodium group experienced modest but significant improvements in their Kansas City Cardiomyopathy Questionnaire scores and New York Heart Association functional class (Ezekowitz et al., 2022). This suggests that some patients may derive symptomatic benefit from sodium management, potentially through the reduction of congestive symptoms and edema (Billingsley et al., 2020).

However, these benefits are not universally observed across the literature. A systematic review of randomized controlled trials specifically evaluating quality of life outcomes found that dietary sodium restriction did not improve long-term QoL for the majority of heart failure survivors (Zhu et al., 2022). In fact, many patients report distress associated with restrictive diets, including increased perceived thirst, xerostomia and an overall reduction in the palatability of nutrient-dense foods (Mullens et al., 2024). This often leads to reduction of their total food consumption to avoid salt, causing a decline in essential caloric and protein intake (Jefferson et al., 2015).

The shifting evidence has led to a recent downgrading of sodium restriction recommendations in major clinical guidelines. The 2021 European Society of Cardiology (ESC) guidelines now advise only against dietary sodium excess (more than 5 g of salt or 2 g of sodium per day), while the American Heart Association (AHA) and Heart Failure Society of America (HFSA) acknowledge that the evidence for specific sodium targets is limited (McDonagh et al., 2021; Heidenreich et al., 2022). Current consensus emphasizes that a universal approach is inappropriate and that sodium management should be personalized according to the patient's clinical profile, medication use and overall nutritional status (Driggin et al., 2022). Avoiding excessive salt remains a sensible clinical goal, however the evidence does not support aggressive restriction as a primary means of reducing mortality or hospitalization in heart failure survivors.

4. Dietary Patterns and Myocardial Health

While the Mediterranean diet is a well-known tool for fighting inflammation due to its focus on plant-based foods and healthy fats, its role in preventing the initial onset of heart failure is less definitive. According to Papadaki et al. (2017), clinical data suggest that even if the diet doesn't strictly prevent heart failure from developing, it significantly improves internal health markers. Specifically, the researchers noted a decrease in NT-proBNP and oxidized LDL. In patients with established chronic heart failure, higher adherence to the Mediterranean diet improves clinical stability. Retrospective cohort data indicate that patients with high adherence experience a trend toward fewer acute decompensation episodes and lower plasma concentrations of NT-proBNP and CA125 (Billingsley et al., 2020; Jiménez-Torres et al., 2024). Although the Mediterranean pattern may not provide a clear survival benefit in terms

of all-cause mortality, patients who comply with it have a lower risk of heart failure-related rehospitalization (Jiménez-Torres et al., 2024).

Beyond its proven capacity to reduce blood pressure, the DASH diet has been shown to improve structural and functional cardiac metrics. Epidemiological evidence indicates that adherence is inversely correlated with the incidence of heart failure, particularly within cohorts under 75 years of age (Goyal et al., 2021). Specifically, clinical trials have demonstrated that DASH adherence results in a significant reduction in high-sensitivity troponin and NT-proBNP, suggesting a direct reduction in myocardial damage and wall tension (Juraschek et al., 2021). In contrast to the conventional sodium restriction models, the DASH diet emphasizes high potassium, magnesium and calcium intake, which may collectively attenuate cardiac remodeling by suppressing systemic inflammation measured by markers such as C-reactive protein, thereby limiting the pro-fibrotic environment that leads to ventricular stiffness (Frangogiannis, 2021; Levitan et al., 2016; Soltani et al., 2020).

Clinical trials in the secondary prevention setting have also highlighted the functional benefits of this pattern. In the GOURMET-HF trial, heart failure survivors who received home-delivered, sodium-restricted DASH meals for four weeks following hospital discharge showed substantial symptomatic improvement and a statistically underpowered trend toward fewer rehospitalizations compared to those receiving standard care (Hummel et al., 2018).

Emerging research has introduced the 'High Exogenous Antioxidant Restorative Treatment' (HEART) as a concept specialized in secondary cardiovascular prevention. This nutritional intervention is characterized by an exceptionally high concentration of polyphenols and flavonoids, which is hypothesized to promote the myocardial recovery. Through the targeted attenuation of oxidative stress and the stabilization of mitochondrial dynamics, such restorative diets act as a primary barrier against the molecular drivers of initial cardiac decline (Singh et al., 2022).

Innovative dietary strategies such as time-restricted eating (TRE) are currently under investigation for their role in heart failure management. It is hypothesized that combining sustainable dietary patterns with fixed fasting windows may facilitate a metabolic switch, enabling the heart to utilize ketone bodies more efficiently and reducing metabolic stress and risk of major cardiovascular events (Small et al., 2023). These findings suggest that the management of heart failure must move beyond simple sodium-restricted paradigms to

embrace comprehensive, nutrient-dense dietary patterns that support cardiac structure and bioenergetic efficiency.

5. Mitochondrial Bioenergetics and Functional Capacity

The hallmark of chronic heart failure is a decline in physical functional capacity, primarily driven by a perpetual energy deficit of the myocardium. Recent research has shifted the understanding of heart failure from a purely mechanical pump deficiency to a state of systemic metabolic failure. In this context, mitochondrial dysfunction serves as the common pathophysiological pathway where nutritional status directly determines exercise tolerance and clinical stability.

The healthy myocardium maintains cardiac adenosine triphosphate (ATP) production through the efficient oxidation of glucose and lipids. However, in a state of heart failure it undergoes a metabolic shift toward an energy-depleted state. This transition is driven by impaired substrate flexibility and a progressive decline in mitochondrial performance (Lopaschuk et al., 2021). This bioenergetic crisis results in a progressive depletion of ATP, leading to impaired contractility and relaxation (Brown et al., 2017). Chronic micronutrient deficiencies exacerbate this state by depriving the mitochondrial electron transport chain of essential co-factors required for cellular respiration (Bomer et al., 2022).

Skeletal muscle mitochondrial health is a primary determinant of systemic exercise tolerance. Heart failure patients are often suffering from sarcopenia, a loss of muscle mass and function that strongly correlates with poor prognosis (Lavine et al., 2017; Zhang et al., 2021). The data suggest that a higher body mass index may be associated with improved survival, however this benefit is largely restricted to patients with preserved lean body mass (Bianchi, 2020). The presence of sarcopenic obesity is a critical risk factor in heart failure as the combination of high fat and low muscle mass is particularly detrimental to physical functional performance (Carbone et al., 2020). Clinical evidence indicates that nutritional interventions aimed at increasing protein and amino acid intake can help manage sarcopenia, as studies utilizing nutritional supplements rich in protein and calories have displayed significant gains in 6-minute walk test distance in patients with heart failure and malnutrition (Habaybeh et al., 2021).

Omega-3 polyunsaturated fatty acids also contribute to mitochondrial health by modifying the phospholipid composition of the mitochondrial membrane as it reduces myocardial oxygen consumption and provides protection against cell apoptosis (Sakamoto et al., 2019). Systematic reviews indicate that supplementation with these fatty acids leads to improved ejection fraction and functional capacity through its anti-inflammatory properties (Nomali et al., 2024). Higher circulating levels of eicosapentaenoic acid are associated with a reduced risk of heart failure, highlighting the importance of lipid quality in maintaining cardiac health (Block et al., 2019). Mitochondrial bioenergetics can be optimized through targeted nutritional supplementation required for cellular respiration, moving the clinical focus away from dietary restriction.

6. The Gut-Heart Axis and Microbial Metabolism

Microbial metabolism of specific dietary components produces bioactive molecules that directly impact cardiac structure. A primary example is trimethylamine N-oxide (TMAO), a pro-atherogenic metabolite generated when gut bacteria process precursors like choline, phosphatidylcholine and L-carnitine (Singh et al., 2022; Wang et al., 2021). As a cornerstone of the modern Western dietary pattern, high red meat consumption provides a substantial intake of L-carnitine, further driving the cardiac decline (Koeth et al., 2019). Clinical evidence demonstrates that elevated levels of TMAO are strongly correlated with heart failure severity, higher concentrations of B-type natriuretic peptide and increased mortality risk (Li et al., 2022; Anderson et al., 2022). TMAO promotes cardiovascular pathology by inducing myocardial fibrosis, increasing platelet reactivity and exacerbating atherosclerosis (Mylavarapu et al., 2026). However, fermentation of dietary fiber by commensal gut bacteria, specifically *Faecalibacterium* and *Roseburia*, produces protective short-chain fatty acids (SCFAs) such as butyrate, which have shown anti-inflammatory properties (Adak et al., 2019). Research has established a link between the quality of dietary carbohydrates and cardiac structure as superior carbohydrate quality, characterized by a low carbohydrate-to-fiber ratio, is correlated with a reduced left ventricular mass index, suggesting a protective role against pathological cardiac hypertrophy (Yi et al., 2025). A cohort study utilizing the NHANES database found that heart failure survivors in the highest quartile of fiber intake experienced a reduction in both all-cause mortality and in cardiovascular-related mortality suggesting that high-fiber diets appear to have protective features (Wang et al., 2025).

The potential to modulate the gut-heart axis through live microorganisms has led to several clinical investigations into probiotic therapy. Study involving the *Lactobacillus* suggests that it can enhance vascular endothelial function and attenuate systemic inflammation (Hofeld et al., 2021). In a randomized, triple-blind controlled trial, heart failure patients who consumed probiotic yogurt containing *Lactobacillus acidophilus* and *Bifidobacterium lactis* demonstrated significantly decreased oxidized low-density lipoprotein levels compared to those consuming ordinary yogurt. Although this intervention did not significantly alter brain natriuretic peptide levels, it provided evidence that specific probiotic strains can reduce the oxidative stress burden in chronic heart failure (Pourrajab et al., 2020).

7. Optimizing Adherence and Patient Support

Within the field of nutrition therapy, various forms of psychological management are utilized and one of the most significant of these models is the Transtheoretical Model. This model allows to individually treat each patient according to their level of motivation for behavior change.

Research evaluating a model-based nutritional program found that patients who received stage-matched support experienced substantially decreased brain natriuretic peptide levels and enhancements in left ventricular ejection fraction compared to patients that underwent conventional educational management (Yuan et al., 2025).

The shift from prescribing diets to patients in general to prescribing diets according to their personalized care plan has had a significant impact upon their survival. The EFFORT trial, which provided diets according to individual needs to hospitalized patients with heart failure and high nutritional risk, demonstrated that there was a significant reduction in mortality and cardiovascular events within 30 days (Hersberger et al., 2021). These benefits were achieved through the involvement of trained dieticians in the effort to provide diets according to specific patient's requirements. The success of this treatment indicates the necessity of the multidisciplinary team to provide such care to the patient, including the nurses, dieticians and cardiologists (McDonagh et al., 2021; Driggin et al., 2022).

Adherence to diet presents significant obstacles for individuals with heart failure, ranging from socioeconomic limitations to the unpalatability of diets that are required of those with the condition. The need to limit sodium and fluid intake can lead to thirst and decreased

enjoyment of food, leading to inadequate intake of the calories that are necessary to maintain health and prevent the development of malnutrition (Ezekowitz et al., 2022; Mullens et al., 2024). Therefore, educational efforts should focus upon providing knowledge to heart failure patients of the nutritional components of healthy diets. Evidence from the GOURMET-HF trial indicates the success of meal delivery programs in delivering diets that are both healthy and palatable to patients (Hummel et al., 2018).

8. Conclusions

The nutritional management of heart failure is undergoing a fundamental paradigm shift, moving away from a fixation on dietary restriction towards metabolic restoration and physiological support. Aggressive sodium restriction, as the traditional cornerstone of HF therapy, lacks a robust evidence base for reducing mortality or cardiovascular hospitalizations. Current clinical trials show that, while a moderate level of sodium intake is sensible for management of congestive symptoms, aggressive restriction can be counterproductive, contributing to activation of the neurohormonal response and decreasing the palatability of food and resulting in malnutrition. Adherence to the Mediterranean and DASH diets has demonstrated potential to attenuate systemic inflammation and improve functional capacity. The growing understanding of the gut-heart axis presents a novel therapeutic target within the modulation of microbial metabolites. The management of sarcopenia remains critical with regard to long-term survival of patients suffering from heart failure. Nutritional interventions should focus on protein and micronutrient intake to preserve lean body mass and mitochondrial function as factors beyond caloric counts that contribute to the energy crisis present with heart failure. A universal approach is no longer justifiable within a population displaying diverse needs and readiness to adopt recommended behaviors. Usage of psychological systems like the Transtheoretical Model and leaning upon multidisciplinary teams is providing individualized support to these patients. A transition away from a restrictive paradigm towards nutritional optimization offers the best means of meeting the complex metabolic demands of the failing heart and improving quality of life and outcomes of patients suffering from heart failure.

Disclosure

Author Contributions

Conceptualization: Mateusz Kasprzak, Kacper Godyń-Müller, Michał Grześkowiak

Methodology: Martin Zeigner, Hanna Pelant, Joanna Błaszczyk

Formal analysis: Bartosz Kulak, Michał Brzeziński, Klaudia Pierzyńska, Zuzanna Majchrzak

Supervision: Mateusz Kasprzak, Kacper Godyń-Müller, Michał Grześkowiak

Investigation: Martin Zeigner, Hanna Pelant, Joanna Błaszczyk, Michał Brzeziński

Data curation: Zuzanna Majchrzak, Klaudia Pierzyńska, Michał Brzeziński

Writing-rough preparation: Mateusz Kasprzak, Kacper Godyń-Müller, Michał Grześkowiak

Writing review and editing: Martin Zeigner, Hanna Pelant, Joanna Błaszczyk, Bartosz Kulak

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