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Review

A Focus on Heart Failure Management through Diet and Nutrition: A Comprehensive Review

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Abstract: There is emerging evidence to suggest that diet and dietary interventions can have an impact on heart failure (HF) outcomes. Currently, the restriction of salt intake is the only dietary advice that is consistently guideline-recommended for the management of HF despite conflicting evidence for its efficacy. Dietary components that have been investigated in people with HF include middle-chain triglyceride (MCT) oil, beta-hydroxybutyrate (BHB) salts, ketone esters and coenzyme Q10 (CoQ10). Supplementation with these components is thought to be cardioprotective possibly due to an increase in myocardial energy production. There have been research studies on the effectiveness of The Dietary Approaches to Stop Hypertension (DASH) diet and the Mediterranean Diet (MedDiet) in the treatment of HF, but with conflicting results. The ketogenic diet (KD) has come to the forefront of interest due to evidence indicating its effectiveness in addressing the metabolic shift that occurs in HF. However, there is a lack of randomised controlled trials (RCT) centred around the KD. In any dietary intervention, factors such as adherence and compliance affect the validity of the results. Malnutrition, sarcopenia and/or cardiac cachexia can be present in the more advanced stages of heart failure. Nutritional screening, assessment and support/intervention are important aspects of treatment in the advanced stages of heart failure. Furthermore, HF management through dietary intervention is further complicated by the presence of comorbidities, such as diabetes mellitus (DM) and coronary artery disease (CAD). Long-term studies on the use of dietary modifications in people with HF are warranted to ascertain their efficacy, safety and side effects.

Keywords: heart failure; ejection fraction; dietary intervention; dietary guidelines; salt restriction; ketones; comorbidities

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1. Introduction

Heart Failure (HF) is a clinical condition characterised by the heart's inability to pump a sufficient amount of blood to meet the body's metabolic needs [1]. It occurs secondary to an abnormality in the structure and function of the heart, characterised by changes in cardiac substrate metabolism, structural remodelling and impaired contractibility. HF has a global prevalence of approximately 64 million people, and in Australia, 179,000 hospitalisations in 2020–2021 were attributed to HF [2,3]. HF was the ninth leading cause of death in 2021 and affects 1–2% of the Australian population [3]. HF is a significant public health problem, with patients experiencing a decrease in life expectancy of approximately 60% [4]. Despite emerging medications and device therapy for HF, further research is needed to explore alternate treatment options beyond pharmacological therapies. One such promising therapeutic option for HF is through dietary interventions.

Diet is a cornerstone of cardiovascular disease (CVD) prevention and treatment. However, there are few randomised controlled trials (RCT) in the area of diet and nutrition

for patients with HF. Dietary patterns and components have been proposed, with only salt restriction (<2 g/day) as a guideline-recommended intervention for HF treatment (Figure 1) [5]. However, a meta-analysis of RCTs evaluating the effect of sodium restriction in patients with HF found no reduction in deaths or hospitalisations but improvements in symptoms and quality of life (QoL) [6].

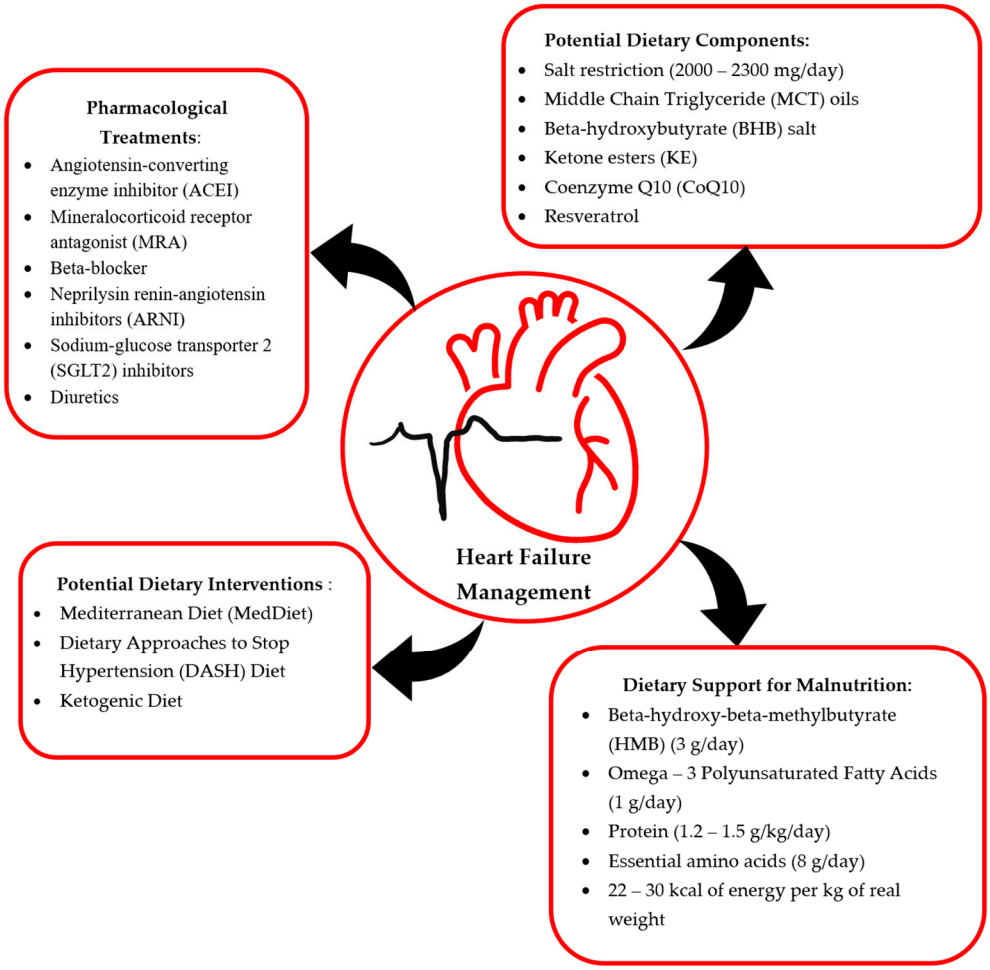

Figure 1. Current pharmacological, dietary patterns and dietary supplements that have been investigated in the management of HF.

The Mediterranean diet (MedDiet) and Dietary Approaches to Stop Hypertension (DASH) diet have been recommended for HF management [7]. However, little is known about their effect on HF outcomes. More recently, the ketogenic diet (KD) has emerged as a promising dietary intervention, thought to increase adenosine triphosphate (ATP) yield of ketone oxidation, which benefits the failing heart [8]. Supplementation with ketone body (KB) precursors or derivatives are being investigated for their potential to elevate ketone levels [9]. This review aimed to summarise the available evidence on dietary components and dietary patterns in the treatment of people with HF and identify areas for future research.

2. Definition and Current Pharmacotherapies in HF

A diagnosis of HF can be classified according to left ventricular heart failure (LVEF): heart failure with reduced ejection fraction (HFrEF) where LVEF is <50% and heart failure with preserved ejection fraction (HFpEF) where LVEF is ≥50% but diastolic dysfunction is present [5]. The standard, combination treatment for HFrEF is an angiotensin-converting enzyme inhibitor (ACEI), mineralocorticoid receptor antagonist (MRA) and a beta blocker, which has been shown to decrease mortality over 1–3 years by at least 50% [10]. More

recently, combined neprilysin renin–angiotensin inhibitors (ARNI) and sodium-glucose transporter 2 (SGLT2) inhibitors have become the standard of care based on their positive effect on reduced hospitalisations and improved QoL [11,12]. The SGLT2 inhibitors Dapagliflozin and Empagliflozin, originally used in the treatment of Type 2 Diabetes, have been shown to reduce cardiovascular (CV) death and hospitalisations in people with HFrEF [13,14]. Individual characteristics further influence HF prognosis and outcomes, such as age, gender, ethnicity and genetic predisposition for inherited cardiac diseases [5]. Notably, sex differences can affect the pharmacological outcomes where studies have shown that women reach maximum plasma concentrations 2.5 times higher than men for the same treatment regime when given similar doses, indicating higher sensitivities [15,16]. Phenotypic differences in types of HF, with women more likely to have HFpEF and men more likely to have HFrEF, might account for these differences [17].

3. Dietary Components in HF

3.1. Limiting Salt Intake

Dietary sodium restriction is a common, non-pharmacological adjunct treatment for HF. The rationale for a low-sodium diet stems from the hypothesis that sodium retention causes fluid overload, raising blood pressure and increasing the risk of HF. However, there are conflicting reports as to whether a low-sodium diet reduces episodes of HF [18–20].

The largest international RCT investigating sodium restriction to date is the SODIUM-HF (Study of Dietary Intervention under 100 mmol in Heart Failure) trial [21]. The SODIUM-HF trial included 806 patients (median age of 67 years, 66% male participants), with chronic heart failure (CHF) from six countries (Australia, Canada, Colombia, Chile, Mexico and New Zealand). These participants were randomly assigned to a low-sodium diet (<1500 mg sodium/day) or "usual care" (general advice to restrict sodium) for 12 months [21]. Compared to usual care (an average of 2119 mg/day), a dietary sodium restriction of <1500 mg daily did not reduce CVD-related emergency department visits. Similarly, a 2022 meta-analysis of RCTs (n = 16 studies) in HFrEF patients (mean age range of 54–76 years, 37–84% male participants) showed that sodium restriction alone (without fluid restriction) caused an increase in hospitalisation risk and mortality [22].

There are mixed reports on how salt restriction affects QoL, most tending towards an improvement [6,23,24]. Zhu et al.'s systematic review of RCTs (n = 302 studies) showed that the pooled effect of salt restriction on QoL was insignificant and that results were largely heterogeneous [23]. The SODIUM-HF trial led to a moderate improvement in QoL, although improvement may have been constrained by the lack of blinding [21]. The PROHIBIT (Prevent Adverse Outcomes in Heart Failure by Limiting Sodium) pilot trial (n = 83 patients with HF, mean age of 62, 22% male participants) showed improvements in QoL on 1500 mg/day but not on 3000 mg/day [25]. Interestingly, Stein et al. demonstrated that the questionnaire type used can influence the outcomes of QoL assessments. The primary conclusion is that salt restriction appears to improve QoL but the evidence is limited by the lack of blinding and the observational nature of studies [22].

There is, in fact, controversy over the validity of findings drawn from sodium studies. Cappucino et al. argue that what is measured today is "usual" sodium intake, which cannot be paralleled with biologically "normal" [26]. They maintain that while sodium restriction is endorsed, it should not be hindered by unreliable research. In particular, the authors highlight the flaws in techniques used to measure dietary sodium in studies including spot urine samples with discredited conversion formulas in 24 h urine excretion calculations, resulting in misleading conclusions. However, it is to be noted that cohort studies with multiple urine measurements show a linear trajectory between sodium excretion and CV outcomes [27,28].

The variability of sodium restriction on outcomes across studies may be due to multiple factors, for example, age, ethnicity, the assessment of sodium intake, willingness to adhere to intervention and sodium level restriction. Meta-analyses show that low-sodium diets are potentially more beneficial for older individuals and non-Caucasian populations with

blood pressure in the highest 25th percentile [29,30]. Furthermore, while the gold standard for determining sodium intake remains a 24 h urine excretion, the majority of studies use food diaries as a more practical method to measure in clinical trials. Basuray et al. also showed that in the chronic HF population, adherence to low-sodium diets was low, and most individuals consumed much higher amounts than recommended [31]. Furthermore, there appears to be a restricted range over which sodium restriction is effective, where differences in clinical practice guidelines range from <1.5, 2–3 to <3 g/day, reflecting the heterogeneity of evidence [5,32–34]. Severe sodium restriction (<2000 mg/day) has led to a higher weight in HF patients, suggesting congestion, so this may not be an effective strategy [35]. Currently, it would seem prudent to follow the recent American Heart Association (AHA) guidelines and Australian Nutrient Reference Value guidelines for sodium intake of 2300 mg/day for HF patients and 2000 mg/day (suggested dietary target) until further studies are conducted [36].

Interestingly, there is some evidence that lower-salt diets can also be achieved by salt substitutes. For example, potassium-enriched salt is 75% sodium chloride and 25% potassium chloride in comparison to regular salt, which is 100% sodium chloride [37]. These are now more widely available and have been shown to lower salt intake and blood pressure in large clinical trials [38]. There are limited studies on salt substitutes for HF patients, although preliminary reports suggest that it is safe, with some benefits toward exercise capacity [39]. However, there are concerns of possible hyperkalaemia and, thus, arrhythmias, as a result of exceeding the daily recommended intake of potassium [40].

3.2. Dietary Supplementation

3.2.1. MCT Oil

Middle-Chain Triglycerides (MCTs) are composed of short-chain saturated fatty acid (6 to 12 carbons) atoms, allowing them to be rapidly metabolised into free fatty acids [41]. This facilitates quick transportation to the liver, where they are converted into ketone bodies. Since a failing heart increases KB oxidation to meet its energy demands, MCT oil has been suggested as a potential exogenous source to promote ketosis for HF patients [9]. MCT oil is commonly found in dietary sources of coconut and palm oils, milk fat (milk, butter and yoghurt) and commercial supplements of MCT oil and Liquigen (Nutricia). MCT (C8 and C10) supplementation in healthy adults elevates plasma ketone bodies, acetoacetate and beta-hydroxybutyrate (BHB) levels by 2-fold within a 4 h period, with a rise in BHB levels after 30 min [42]. MCT oil has been therapeutically used for obesity, and a meta-analysis of RCTs (n = 7 studies) showed that MCT oils produce favourable decreases in body composition and weight without adversely affecting the lipid profile [43]. In animal models, MCT alleviates insulin resistance, which is important for individuals with Type 2 Diabetes who are susceptible to developing HF [44]. However, no studies have assessed the use of MCT oil specifically in patients with HF.

3.2.2. Beta-Hydroxybutyrate (BHB) Salt

BHB is a KB synthesised in the hepatic mitochondria and is one of the primary substrates for myocardial ATP production. In HF, the upregulation of KB oxidation occurs at the cost of fatty acid oxidation [45]. Hence, exogenous BHB supplementation (orally or through injection) has been proposed as a potential therapeutic option for HF. In animal models, continuous BHB infusion can preserve cardiac function [46]. In humans, a population-based study (n = 6134, mean age 53 years) showed that elevated plasma BHB concentrations correlated with an 83% increase in HFrEF incidence [47]. Another study (n = 45 patients with chronic HF, mean age of 57 years) found that high BHB concentrations correlated with lower LVEF [48]. An in vivo human study found that ketone uptake in the hearts of patients with HF was dependent on the levels of circulating concentration of ketones [49]. This suggests that BHB supplementation would aid in the uptake of ketones by the heart. Consistent with this notion, a study of 24 patients with HFrEF found that an exogenous infusion of BHB increased cardiac output (CO) by 40% [50]. However, a concern

identified in this study was the rapid decrease in BHB levels once the infusion was stopped, highlighting the need to maintain high BHB concentrations to sustain therapeutic levels. There is a need to determine the efficacy and safety of long-term BHB supplementation in people with chronic HF.

3.2.3. Ketone Esters

Ketone esters (KEs) can be synthesised from BHB. When ingested orally, KEs elevate circulating BHB levels and can increase CO by 15% after 1 h in healthy individuals [51]. Since continuously infusing BHB can be problematic, repeated ingestions of KEs provide an alternative approach. In a mouse model of HF, KEs in drinking water increased ketone levels and ejection fraction, without negatively impacting glucose, fatty acid or insulin levels [52]. Oral administration of a KE drink in humans with HF also significantly increased BHB levels by a factor of ten correlating to myocardial remodelling [53]. In 2024, an RCT was conducted in 24 patients with stable HFrEF (mean age of 65 years, 71% male participants) who received a 14-day oral KE drink with 4 daily doses [54]. The authors found that resting CO and LVEF were higher after KE treatment with beneficial effects on markers of left ventricle unloading and reverse remodelling. These findings indicate that two-week treatments are safe and provide short-term benefits for patients with HFrEF; however, the long-term efficacy and safety of chronic supplementation are unknown.

3.2.4. Coenzyme Q10

Coenzyme Q10 (CoQ10) is a lipid-soluble molecule that plays a crucial role in the mitochondrial electron transport chain for ATP production, making it a powerful antioxidant [55]. CoQ10 has the potential to improve endothelial function and protect against myocardial ischemia via increased energy production [56,57]. In patients with HF, lower levels of myocardial CoQ10 correlate with an increased risk of mortality [58]. Furthermore, CoQ10 levels decline with age, so supplementation particularly for the elderly may be beneficial [59]. The only large RCT (Q-SYMBIO) took place over 106 weeks, where 429 participants (mean age of 62 years, 71% male participants) were randomised to 100 mg of CoQ10 3 times daily to achieve a serum level of 2 μg/mL or placebo tablets [60]. They found an approximately 40% reduction in CV death and all-cause mortality. These findings were mirrored in the *post hoc* analysis of a European subgroup of Q-SYMBIO RCT, where improved LVEF was seen [61].

3.2.5. Resveratrol

Resveratrol (3,5,4-trihydroxystibene) is a natural polyphenolic compound found in plants including nuts, grapes and berries in response to environmental stress [62]. Given its potential antioxidant properties, it has been suggested as a possible supplement for those with heart failure. Various animal studies have demonstrated how Resveratrol reverses or prevents CV functional deficits in hypertension, ischaemic heart disease and cardiomyopathy [62]. However, there are few clinical trials in humans, and most trials thus far are limited by participant numbers. In relation to HF, a small RCT in 2020 (n = 60, mean age of 66 years) conducted on patients with HFrEF receiving 100 mg of Resveratrol for 3 months found a decrease in erythrocyte aggregation, inflammatory cytokines and cardiac markers including NT-proBNP [63,64]. These improvements were correlated with findings of improved LVF and exercise capacity, suggesting that Resveratrol has a positive effect on coronary and peripheral blood flow by modulating inflammatory processes. Similarly, an older RCT on patients with coronary artery disease (CAD) (n = 40, mean age of 66 years) also showed that Resveratrol improved LVF, as well as improvements in endothelial function and LDL cholesterol [65].

4. Dietary Pattern Alterations in Heart Failure Management

4.1. Dietary Approaches to Stop Hypertension (DASH)

The DASH diet has been well-established in the treatment of hypertension, a key modifiable risk factor for HF. It is characterised as low in salt (maximum of 2.3 g/day), rich in fruits, vegetables and plant proteins from legumes and nuts, moderate in low-fat dairy products and low in red and processed meats and fats [66]. It has been shown to lower levels of cardiac troponin-1, a diagnostic marker of cardiac injury, as well as NT-proBNP (N-terminal pro B-type natriuretic peptide), in individuals with untreated hypertension [67]. The high concentration of antioxidants decreases inflammatory cytokines and biomarkers of oxidative stress, thereby maintaining endothelial function [68].

Recent studies have explored the benefits of the DASH diet on HF. In a 2020 cohort study of Swedish men (n = 41,118, aged 45–83), long-term adherence to the DASH diet was associated with a 25% decreased risk of HF [69]. The replacement of 1 serving/day of red and processed meat with fruits, vegetables, nuts and legumes, low-fat dairy or whole grains resulted in an 8–12% HF risk reduction. This study was limited by having all male participants, with a lack of racial diversity. The REGARDS (Reasons for Geographic and Racial Differences in Stroke) cohort consisting of 18,856 participants (aged 45–98, with 32.5% black adults and 44.1% male participants) demonstrated a beneficial effect of the DASH diet [70].

The efficacy of the DASH diet was investigated in the 4-week GOURMET-HF (Geriatric Out-of-hospital Randomized Meal Trial in Heart Failure) trial where three daily meals were delivered to recently hospitalised HF patients who had been discharged and followed for 12 weeks (n = 66) [71]. This small RCT (mean age of 71 years, 70% male participants) demonstrated a 69% decrease in re-hospitalisations, with minor adverse effects that did not require hospitalisation.

4.2. Mediterranean Diet

The MedDiet consists of a high intake of whole-grain cereals, vegetables, fruits, nuts and olive oil, a moderate intake of fish and poultry, a low to moderate dairy intake, and a low red meat intake [72]. The MedDiet is widely recognised for its cardio-protective effects in the prevention of major CV events, for example, reducing inflammatory markers, lowering LDL (low-density lipoprotein) cholesterol, decreasing oxidative stress and, hence, improving endothelial function [73].

A large RCT investigating the impact of the MedDiet on HF biomarkers, including Nt-proBNP and oxidised LDL, was a sub-study of the PREDIMED (Prevention with MedDiet) trial [74]. A cohort of participants (n = 7447, 45.3% male participants) with no CVD on enrolment but at high risk of CVD (55–85 years of age) were assigned to one of three diets: a MedDiet supplemented with (i) mixed nuts or (ii) extra-virgin olive oil or the control (iii) low-fat diet. At 1-year follow-up, classical risk factors for HF—blood pressure, triglycerides and total cholesterol—had decreased for both MedDiet groups. The MedDiet-induced alteration in HF biomarkers was also found by Herrerra-Martinez et al., who demonstrated an increase in LVEF and QoL after this diet [75]. A secondary analysis of the PREDIMED trial further looked at HF incidence, with a non-significant association found across the two MedDiets and the control (HR (Hazard Ratio) of 0.68, 95% CI 0.41–1.13. $p = 0.139$ and HR 0.92, 95% CI 0.56–1.49, respectively, $p = 0.725$) [76]. However, these findings were limited by the small number of heart failure events (n = 94, 1.3% of participants), and larger randomised trials are needed to determine the effect of the MedDiet on HF outcomes.

4.3. Ketogenic Diet

In HF, dysfunctional myocardial cellular metabolism and reduced energy production can be increased via ketone metabolism [77]. An increase in ketone metabolism is characteristic of the metabolic shift that occurs in HF, where the heart relies on other substrates, particularly ketone bodies, which release more ATP compared to glucose [78,79]. Due to this alteration in nutrient metabolism, the KD has emerged as a potential dietary inter-

vention for the treatment of HF. A KD is one where a carbohydrate-reduced diet leads to the increased production of ketone bodies and induces a state of ketosis in the body ([BHB] $\geq$ 0.5 mmol/L) [80]. The diet typically consists of 5–10% carbohydrates, 10–20% protein and 70–85% fat.

In mice, KD improves cardiac function as aging occurs, as well as endothelial cell proliferation [79,81]. Contrasting studies found that continuous feeding of a ketogenic diet in a mouse model of HF for 2 months was not cardioprotective and presented impaired hepatic ketogenesis [82]. Similarly, in another study, Ho et al. also found a decrease in cardiac metabolic efficiency in HF mice fed a KD continuously [83]. Adverse effects, which were reported in both studies, include disturbances in lipid and glucose profiles. Alterations in glucose metabolism with decreased insulin levels and increased cortisol and glucagon have been noted previously [84]. Conversely, alternate-day feeding appeared to be highly beneficial in preserving hepatic ketogenesis and decreasing cardiac fibrosis while improving systolic and diastolic function [82].

In HF patients, a short-term KD has been shown to improve haemodynamic and metabolic parameters, which remained stable after 1 year [85]. However, for patients with ischaemic-related HF, there is a caution against a KD due to potential increases in LDL cholesterol, known to promote atherosclerosis [86]. Prioritising unsaturated fats over saturated fats in a KD might address these concerns. Care would need to be taken to address ketoacidosis-inducing dehydration and electrolyte imbalances in patients also taking SGLT2 inhibitors [87]. Given the mixed outcomes from KDs, coupled with a lack of clinical trials, further investigation into the efficacy of a KD in HF is necessary to determine its therapeutic effect.

5. Comorbidity Influence on Dietary Modifications in Heart Failure

It is common for HF patients to have co-existing comorbidities, which include diabetes mellitus (DM), obesity, coronary artery disease (CAD) and chronic kidney disease (CKD) [88–90]. A 2020 systematic review of 118 clinical trials found that the reporting of comorbidities was higher in those with HFrEF compared with HFpEF and that these comorbidities did not improve over time [91]. This is even more relevant in older patients who have a high risk of HF and chronic conditions. Given the complexity of a multi-disease state, dietary interventions need to be carefully considered in the management of HF patients with comorbidities (Table 1).

Table 1. Potential dietary recommendations for patients with HF and common comorbid conditions: recent evidence from the past 5 years.

Comorbidity	Proportion of HF Patients (%) [92]	Relationship to HF	Dietary Recommendation
DM	45	• Impaired beta cell function and insulin signalling [93].	• High protein (30% vs. 15%) and energy restriction (1200–1500 kcal/day) over 3 months improved glycaemic control [88]. • A short-term (16 week) low-carbohydrate diet (50–130 g/day) resulted in weight loss and decreased systolic and diastolic pressure in patients with diabetic cardiomyopathy [94].
Obesity	29	• Excessive adipose tissue accumulation results in an increase in CO, LV diastolic dysfunction and other changes [95].	• High protein [30% (110 g/day), 40% carbohydrates (150 g/day), 30% fat (50 g/day)] reduced weight, waist circumference, blood pressure, total cholesterol and triglycerides [88]. • Hypocaloric diet improved body weight, glucose control and cardiac structure and function [96,97]. • Essential amino acid supplementation (4–7.5 g/day) reduced triglycerides and increased maximum oxygen update and exercise capacity in CHF patients [98,99].

Table 1. *Cont.*

Comorbidity	Proportion of HF Patients (%) [92]	Relationship to HF	Dietary Recommendation
CAD	48	• Build-up of atherosclerotic plaques in the coronary arteries leading to ischaemia [100].	• Consumption of: ○ whole grains (30 g/day) [101] ○ fruits (<250 g/day) and vegetables (80 g/day) [90] ○ legumes (<100 g/day) [90] ○ fish (250 g/day) with long-chain omega-3 polyunsaturated fatty acids (PUFA) [90] • Co-administration of L-arginine, PUFA, albumin, folic acid, vitamins B_6, B_{12} and C and magnesium, with statins and aspirin stabilised CAD and HF [102].
CKD	60	• Reduced renal blood flow, impaired haemodynamics contributing to ischemic injury [103].	• A low protein diet (LPD) of 0.6 g/kg/day delays the need for dialysis [89]. • A KD slowed the decline of renal function by 57% in CKD patients compared to a LPD [104].

Abbreviations: DM, Diabetes Mellitus; CAD, Coronary Artery Disease; CKD, Chronic Kidney Disease.

6. Malnutrition in Advanced Heart Failure

Malnutrition in advanced HF can occur due to chronic inflammation and metabolic disturbances. Severe malnutrition occurs in 7.5% of HF patients and moderate malnutrition occurs in 57% of HF patients [105]. This can cause an increase in mortality of 2–10 times when compared to HF patients without malnutrition [106]. The mechanisms by which malnutrition/nutritional deficiencies occur include gut malabsorption, high energy demand, cytokine-induced anorexia and hypercatabolism, fatigue and breathlessness, early satiety, taste changes and difficulties in food preparation [107,108]. This untenable situation can result in substantial weight loss (BMI < 20 kg/m^2 or $\geq$5–6% involuntary weight loss over at least 6 months), known as cachexia [109]. Cachexia is characterised by increased protein catabolism resulting in muscle loss and a reduction in fat mass and has a significant association with both morbidity and mortality [109]. Cachectic HF patients have mortality rates 2–3 times higher than non-cachectic HF patients, with mortality reaching 50% after 18 months [110].

Atrophy of the muscles can occur earlier than cachexia, especially when patients are elderly. The wasting of skeletal muscle associated with age, which causes a loss of physical strength, is known as sarcopenia [111]. It has a prevalence of 20% to 50% in HF patients [112,113]. In HF, decreased CO results in reduced blood flow to skeletal muscle, exacerbating the loss of muscle mass and strength.

HF patients with sarcopenia have poor nutritional status, and those with cachexia are prone to anorexia [109]. Due to its prevalence and impact on prognosis, it is important to conduct nutritional screening and nutritional assessment to identify HF patients with malnutrition [114]. The loss of muscle mass and weight may be hidden by oedema in HF patients, so anthropometry and body composition are important tools to use in nutritional assessments. Such tools include handgrip strength, mid-arm muscle circumference, tricep skinfolds and calf circumference. When using bioelectric impedance analysis (BIA), it is important to use bioelectrical impedance vector analysis, which compensates for altered body hydration in HF patients [115]. Muscle ultrasound is a technique being developed to measure muscle mass, which will need to be standardised and validated in HF patients [116].

Nutrition counselling is necessary to prevent malnutrition. Individualised nutrition intervention for malnourished HF patients both in the PICNIC trial and the EFFORT trial (subgroup HF inpatients) showed that nutritional support of HF patients can improve

clinical outcomes and survival [117,118]. Six-week supplementation of a high-protein, high-energy oral nutritional supplement led to a significant increase in fat mass and lean body mass in patients with HF-related cachexia [119]. A metabolite of the amino acid leucine, called Beta-hydroxy-beta-methylbutyrate (HMB), has been found to increase muscle mass in older adults. A recent study of an oral protein supplement, with 3 g/day of HMB given to older adults with acute and chronic conditions such as heart failure or pulmonary disease, indicated that there was an improvement in the nutrition status post-discharge at day 90 and a significant decrease in 90-day mortality (4.8% vs. 9.7%, RR = 0.49, C.I. = 0.27–0.90, $p = 0.018$) [120]. Figure 1 shows other nutritional components proposed to prevent malnutrition in HF patients. Nutritional support includes supplementation with protein, omega-3 polyunsaturated fatty acid and essential amino acids [114,118,120–124]. However, the precise details of supplementation remain unclear due to the small size and/or unblinded nature of studies, as well as the varying comorbidities and conditions of HF patients [119,125–128].

7. Conclusions and Future Directions in Heart Failure Dietary Management

Future directives for the treatment of HF aim to prevent hospitalisations, improve QoL and reduce all-cause mortality. It is evident that dietary interventions have cardioprotective and anti-inflammatory roles and are essential for the treatment of HF as well as the prevention of cachexia and sarcopenia, which negatively affect HF prognosis. However, there is a clear need for longer-term interventional studies to determine the exact quantities of dietary components necessary and to ascertain efficacy and safety. There also needs to be consistency in the methods used to conduct dietary interventions such as nutritional counselling techniques, adherence determination and long-term follow-up. Additionally, whether specific diets would be more suitable for different heart failure subtypes and associated comorbidities remains uncertain. There is a clear gap in the evidence on dietary patterns in HF outcomes, and large-scale randomised nutritional interventions are needed to improve outcomes for the millions of people globally with HF.

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