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Dietary management of heart failure: room for improvement?

Thomas Butler*

Department of Clinical Sciences and Nutrition, University of Chester, Chester CH1 4BJ, UK

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Abstract

There is growing awareness of the role of diet in both health and disease management. Much data are available on the cardioprotective diet in the primary and secondary prevention of CVD. However, there is limited information on the role of diet in the management of heart failure (HF). Animal models of HF have provided interesting insight and potential mechanisms by which dietary manipulation may improve cardiac performance and delay the progression of the disease, and small-scale human studies have highlighted beneficial diet patterns. The aim of this review is to summarise the current data available on the role of diet in the management of human HF and to demonstrate that dietary manipulation needs to progress further than the simple recommendation of salt and fluid restriction.

Key words: Heart failure: Diets: Remodelling: Cardiac function

Heart failure (HF) represents a clinically defined end point that can be the result of many different cardiac diseases that impair ventricular function. Impaired ventricular function results in clinical signs of disease such as dyspnoea, fatigue and oedema. HF can be classified based upon the time course of events, the side of the heart affected, whether systolic or diastolic function is impaired, ejection fraction (EF) and the severity of symptoms[1]. Mortality still remains high with HF, although data from the UK National Heart Failure audit show that in-hospital mortality has fallen from 11·1 to 9·5 % between 2011/2012 and 2013/2014[2,3]. However, 6·2 % of patients who survive to discharge die in the 30 d following discharge, and overall 1-year mortality stands at 27 %[3].

In the UK, the most common New York Heart Association (NYHA) classification at the time of first hospital admission is class III or IV, representing a total of 80 % of those diagnosed with HF[3]. Ischaemic heart disease (IHD) and hypertension (HTN) are observed in 46 and 54 % of HF patients, respectively[3], suggesting that both conditions are important risk factors for the development of HF. Indeed, a medical history of IHD is more likely to result in the diagnosis of left ventricular (LV) systolic dysfunction and hence reduced EF, whereas HTN or valvular disease is associated with non-systolic HF with a preserved or normal EF (HFpEF)[3]. This latter form of HF is more frequently observed in obese women with pre-existing diabetes[4], whereas male sex, smoking and prior myocardial infarction (MI) are associated more strongly with HF with reduced EF (HFrEF)[5]. Recognised comorbidities present in the HF population include anaemia, cachexia, cancer, chronic obstructive pulmonary disease (COPD), depression, diabetes, gout, hyperlipidaemia, HTN, Fe-deficiency anaemia and renal dysfunction, all of which may require careful management in addition to the condition of HF[1]. Interestingly, those patients with HFpEF tend to have a higher non-cardiac comorbidity burden when compared with patients with HFrEF[6], potentially identifying them as a unique patient group.

In addition to the known medical causes, HF has important socio-economical determinants. Individuals with HF living in the most deprived areas of the UK are more likely to present at a younger age when compared with those living in less deprived areas[3], suggesting that additional factors – rather than just medical comorbidities – may influence prognosis. Such factors may include access to care, educational level but also lifestyle choices, including dietary habits.

The evolving knowledge of substrate usage in the failing heart has prompted several investigators to re-examine the importance of dietary modification in this patient group. This manipulation has extended further than preventing uncontrolled weight loss, itself shown to be linked with greater incidence of mortality[7], to diet patterns linked with improvements in cardiac function and delayed mortality. It may be suggested that the window for nutritional intervention becomes narrower as HF progresses, with prevention of unintentional weight loss potentially more important in end-stage disease. Indeed, management of malnutrition and cachexia in HF patients is a key priority, and it has been reviewed extensively[8].

There is a substantial gap in clinical guidance for the dietetic management of patients with HF, despite widely recognised nutritional deficiencies[9]. Na restriction has been the significant

Abbreviations: DASH, Dietary Approaches to Stop Hypertension; EF, ejection fraction; FA, fatty acid; FAO, FA oxidation; HF, heart failure; HFpEF, HF with a preserved or normal EF; HFrEF, HF with reduced EF; HTN, hypertension; NYHA, New York Heart Association.

* **Corresponding author:** T. Butler, fax +44 1244 511310, email t.butler@chester.ac.uk

nutritional recommendation by the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) for the reduction of congestive symptoms[10]; however, this is not mirrored by European guidance[1], itself providing limited advice other than of fluid restriction, maintenance of healthy weight and prevention of malnutrition. Irrespective of Na, both guidelines provide little information into additional dietary changes that may be of benefit to the patient. The aim of this review is to present current developments in the understanding of nutrition in HF and to highlight the areas that need crucial development.

Ventricular remodelling

LV hypertrophy (LVH) is an important step in the development of HF. LVH may initially be beneficial in normalising wall stress and haemodynamic function[11], and several animal models have suggested that inhibiting the initial hypertrophic process is detrimental[12–14]. Pathological ventricular remodelling patterns have recently been shown to be associated with the incidence of HF and interestingly display differential risk for HF with HFpEF and HFrEF[13,14]. Specifically, individuals with eccentric remodelling have a greater than 2-fold risk of developing HFrEF, whereas those with concentric changes showed increased risk of HFpEF. These statistics are of significance given the high prevalence of HTN and IHD in HF patients[3].

Metabolic remodelling

Ventricular remodelling processes also extend to metabolism and have been extensively reviewed[15–17]. Classically, the predominance of fatty acid (FA) oxidation (FAO) in the healthy heart is replaced by glycolytic substrate usage and reduced ability to utilise FA in the failing heart[17,18], although this concept has been challenged[19]. Indeed, the conflicting changes observed in animal models may represent confounding factors such as the method used to induce HF, the strain of animal and duration of the intervention giving rise to different cardiac responses when challenged with varying diets[20]. Nonetheless, in patients with NYHA class IV HF, the mRNA and protein levels for key enzymes associated with FAO are reduced, supporting the metabolic change[21]. In addition to altered FAO, there is evidence that mitochondrial oxidation of glucose may be diminished in HF[18], leading to a scenario in which the heart cannot process sufficient FA or glucose to maintain adequate energy supply. As such there is reduced ability to synthesise ATP leading to impaired contractile function. This concept of the failing heart being energy-starved is not new, and it is why the failing heart has been likened to 'an engine out of fuel'[22]. Many groups have used this concept to suggest that manipulation of the diet to facilitate sufficient ATP production may be important in regulating function in the failing heart.

The role of lipid in heart failure

Much of the work on dietary manipulation has been performed in experimental models of LVH and/or HF, and has been reviewed extensively[23,24]. A limitation of such models is that while providing useful mechanistic insight, they do little to represent benefits in quality-of-life and reduced rates of hospital admission. However, from these mechanistic studies, there is evidence to suggest that manipulation of nutrient intake – predominantly carbohydrate and fat content – has an important role in regulating cardiac structure and function in HF[25]. The importance of fat is often overshadowed by its high energy content per gram; however, in HF patients, this same parameter may be beneficial in increasing an individual's energy intake and preventing unintentional weight loss and cachexia[8]. Several animal studies have also shown a potential beneficial role of dietary fat that extends beyond energy content, forcing us to question whether we should be encouraging a greater intake of this macronutrient in the HF population. For example, coronary artery ligation in Wistar rats has shown to reduce stroke volume and EF, although this finding can be partially attenuated by the provision of a diet containing 60 % lipid (25 % palmitic acid, 33 % stearic acid and 33 % oleic acid)[26]. This study also demonstrated that the high-fat diet had no impact upon cardiac performance in response to a dobutamine stress test, suggesting no additional impairment to contractile reserve. Equally, when failing hearts from rats fed a high-fat diet are perfused *ex vivo*, they demonstrate an improvement in cardiac FAO, which is similar to that of non-infarcted controls[27]. The authors of this study raise an important argument that following an MI, providing sufficient fuel for the non-infarcted myocardium is vitally important as the burden of function is often shifted to healthy tissue. This is further compounded by the observation that acutely limiting the availability of circulating FA in patients with cardiomyopathic HF depresses cardiac function, suggesting an important role of FA in HF[28] (Table 1).

Cardiac TAG and lipotoxicity

The ability to store and utilise endogenous TAG has been shown to be important for cardiac function[29], and the role of endogenous TAG is particularly important in the context of cardiac lipotoxicity. The traditional view of lipotoxicity relies upon the concept that a reduced capacity of the cardiomyocyte to oxidise FA coupled with normal or increased FA delivery leads to progressive lipid accumulation, the shuttling of FA species into the formation of biologically active intermediates such as diacylglycerol and ceramide, and ultimately cellular and organ dysfunction[30]. An excellent review on the role of FA and their derivatives as signalling molecules can be found in van Bilsen & Planavila[31].

The traditional view of lipotoxicity being a pathology solely attributable to lipid accumulation is not completely accurate, and endogenous TAG accumulation may actually protect against biologically active intermediate formation with a specific role of various FA in this process. Indeed, previous research suggested that excessive supply of palmitate leads to increased apoptosis, and that provision of oleate in addition to palmitate can attenuate this by channelling palmitate into the formation of endogenous TAG and away from ceramide synthesis[32]. Although impressive, this study was performed in a cell culture

Table 1. Summary of studies presented in this review investigating the role of fatty acids (FA) in heart failure (HF) patients and experimental models

Study	Participant characteristics	Study design	Measures and time points	Key observations
Berthiaume *et al.*[26]	Male Wistar rats Control + standard diet: *n* 9–10 Control + high SFA: *n* 9–10 Intervention + standard diet: *n* 9–10 Intervention + high SFA: *n* 9–10	Control: sham procedure followed by 8 weeks of normal diet (10 % energy from fat) or a high-SFA diet with 60 % energy from fat (25 % palmitic acid, 33 % stearic acid and 33 % oleic acid) Intervention: coronary artery ligation followed by 8 weeks of normal diet or high-SFA diet as above	Cardiac function using echocardiography and pressure-volume catheter, plasma and metabolic parameters, and genomic expression Diet intervention for 8 weeks	High-SFA diet did not exacerbate ventricular remodelling associated with coronary artery ligation High-SFA diet prevented decline in stroke volume associated with coronary artery ligation and improved function during stress tests Greater transcription of nuclear material in failing hearts from the high-SFA diet compared with respective surgical controls
Berthiaume *et al.*[27]	Male Wistar rats Control + standard diet: *n* 13–16 Control + high SFA: *n* 13–16 Intervention + standard diet: *n* 13–16 Intervention + high SFA: *n* 13–16	Control: sham procedure followed by 8 weeks of normal diet (10 % energy from fat) or a high-SFA diet with 60 % energy from fat (25 % palmitic acid, 33 % stearic acid and 33 % oleic acid) Intervention: coronary artery ligation followed by 8 weeks of normal diet or high-SFA diet as above	Cardiac function using echocardiography, pressure-volume catheter and working-heart perfusions, plasma and tissue metabolite analysis, and genomic expression Diet intervention for 8 weeks	Cardiac TAG significantly increased following high-SFA diet High-SFA diet prevented decline in EF and stroke work observed in dietary control Failing hearts from rats fed the high-SFA diet showed normalisation of glucose and oleate oxidation compared with dietary controls
Tuunanen *et al.*[28]	Total participants: *n* 24 Control group: *n* 8 Intervention group: *n* 18 Control group: 75·0 % men Intervention group: 77·7 % men Control group EF: 66·0 % Intervention group EF: 33·0 % Control group NYHA class: 0·0 Intervention group NYHA class: 2·2 Control group BMI: 26·0 kg/m^2 Intervention group BMI: 28·0 kg/m^2 Race and weight not reported It is noteworthy that all patients had idiopathic dilated cardiomyopathy	Prospective study Both groups received acipimox (250 mg orally twice daily)	Myocardial perfusion and oxidative metabolism via PET, cardiac dimensions and function and insulin sensitivity Baseline and after treatment	Comparable levels of β-oxidation at baseline between groups Acipimox reduced cardiac work and cardiac efficiency in the intervention group only
Listenberger *et al.*[32]	Chinese hamster ovary and 25RA cells, and diacylglycerol transferase 1-deficient fibroblasts	Cells cultured in knockout Dulbecco's modified Eagle's medium supplemented with 10 % FBS, 1 mmol/L-glutamine, 50 units/ml penicillin G Na and 50 units/ml streptomycin sulphate. Cell culture incubated with palmitate and/or oleate bound to BSA at 6·6:1 molar ratio Cells supplemented with FA medium for 6 h with ^{14}C-labelled palmitate	Apoptosis, uptake and accumulation of palmitate, lipid accumulation palmitate incorporation into TAG, MS for ceramide and TAG, enzyme activity* DNA laddering measured after 26 h Palmitate, neutral lipid accumulation, alterations in lipid composition and lipotoxicity measured after 6 h of incubation with different FAs	Palmitate-associated apoptosis and DNA laddering was prevented with co-incubation with oleate Oleate prevented increase in ceramide associated with palmitate Increased activity of SCD-associated TAG synthesis and resistance to palmitate-induced apoptosis Oleate promoted neutral lipid accumulation and led to greater incorporation of palmitate into TAG Failure of *Dgat1*$^{-/-}$ fibroblasts to accumulate TAG was associated with cell death

Table 1. *Continued*

Study	Participant characteristics	Study design	Measures and time points	Key observations
O'Donnell *et al.*[34]	3-week-old male Sprague–Dawley rats Control group: *n* 16 Intervention group: *n* 18	Control: sham procedure Intervention: pressure-overload model of cardiac failure via TAC	Substrate metabolism using NMR, cardiac function, lipid content and turnover Hearts excised 10–12 weeks post-banding and perfused	Oxidation of TAG was not evident in failing hearts, yet it was observable in control rats TAG turnover significantly reduced in HF compared with control hearts TAG turnover uncoupled from workload in failing hearts Reduced ability to oxidise endogenous TAG was not matched by increase in exogenous oxidation of palmitate
Lahey *et al.*[35]	3-week-old male Sprague–Dawley rats Control oleate: *n* 10–15 Control palmitate: *n* 10–15 Intervention oleate: *n* 10–15 Intervention palmitate: *n* 10–15	Control: sham procedure Intervention: pressure-overload model of cardiac failure via TAC	*Ex vivo* cardiac function and metabolism measured following ^{13}C-labelled palmitate and oleate perfusion, TAG dynamics, DAG and ceramide content, and protein expression Hearts were excised 12 weeks post-banding and perfused	TAC + oleate prevented decline in contractility seen in TAC + palmitate TAC + oleate preserved normal TAG turnover and had greater TAG enrichment TAC + palmitate hearts had lower levels of DAG and increased C16 ceramide TAC + oleate leads to preservation of PPAR-*α* target gene mRNA
Tavazzi *et al.*[36]	Randomised 7046 patients Excluded 71 Total participants: *n* 6975 Control group: *n* 3481 Intervention group: *n* 3494 Control group: 78·8 % men Intervention group: 77·8 % men Control group EF: 33·2 % Intervention group EF: 33·0 % Control group NYHA class: 63·2 % II, 34·1 % III, 2·7 % IV Intervention group NYHA class: 63·7 % II, 33·7 % III, 2·9 % IV Control group BMI: 27·0 kg/m^2 Intervention group BMI: 27·0 kg/m^2 Race and weight not reported	Randomised controlled trail Control group: placebo Intervention group: 1 g/d *n*-3 PUFA (850–882 mg EPA:DHA ratio 1:1·2) All participants were also randomly assigned to 10 mg/d oral rosuvastatin Study power of 90 %	Cardiovascular examination, vital signs, 12-lead electrocardiogram, compliance with study protocol, assessment of adverse events and blood biochemistry Primary outcome(s): time to death, and time to death or admission to hospital for cardiovascular reasons Secondary outcome(s): cardiovascular mortality or admission for any reason, sudden cardiac death, admission for cardiovascular reasons, admission for HF, MI and stroke Baseline, 1, 3, 6 and 12 months, and then every 6 months until the end of the trial Median follow-up of 3·9 years	Significantly greater all-cause mortality observed in control group Fewer deaths or hospital admissions attributable to cardiovascular reasons in intervention group Significant reduction in plasma TAG in intervention group

EF, ejection fraction; NYHA, New York Heart Association; PET, positron emission tomography; FBS, fetal bovine serum; BSA, bovine serum albumin; SCD, stearoyl-CoA desaturase; DAG, diacylglycerol; TAC, transverse aortic constriction; MI, myocardial infarction.

* SCD activity measured at 0, 18, 24 and 28 h.

model, and it may not reflect the chronic nature of lipid accumulation in disease or the consequences of prolonged accumulation (Table 1). Nonetheless, it reflects the complexity of lipid dynamics[33] and rasies questions over whether lipid accumulation *per se* is damaging, or wherther impairment to the dynamic nature of this energy store is more important.

In HF, endogenous TAG may be an important yet inaccessible source of substrate. The induction of HF in rats leads to a significant reduction in TAG turnover, suggesting impaired access to this energy store[34]. An inability to utilise stored TAG through decreased oxidation may lead to reduced energy provision in the setting of HF. Consequently, improving the heart's access to its own endogenous energy supply may have a significant impact upon cardiac function. In support of this theory, provision of oleate to failing hearts of Sprague–Dawley rats maintains the myocardial TAG pool and increases TAG turnover when compared with palmitate[35]. This finding was associated with improved cardiac contractility, augmentation of target genes associated with FAO and a reduction in the reactive intermediate C16 ceramide[35]. Although performed in rodents, the significance of this study is that by manipulating the exposure of the failing heart to different FA species mechanical performance can be improved (Table 1).

n-3 Intake in heart failure

n-3 Supplementation is currently listed as a class IIb recommendation and level B evidence in patients with systolic HF in European guidance[1], with similar recommendations present in ACCF/AHA guidance[10].

The Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico-Heart Failure study demonstrated the advantageous method of supplementing stage II–IV HF patients with 1 g daily of an EPA:DHA mix; however, it only produced a small yet significant reduction in hazard ratio for mortality compared with the placebo group[36] (Table 2). A recent meta-analysis[37] has also confirmed the beneficial effect of *n*-3 on cardiac health and function in HF patients. In this study, pooled results of four studies totalling 350 participants showed fish oil supplementation to significantly reduce LV end-systolic volume compared with placebo. Similarly, analysis also suggested fish oil to be associated with improved LVEF[37]. Although this meta-analysis supports the notion that fish oil supplementation may have a beneficial effect in patients with HF, it remains to be determined whether similar effects can be observed by dietary sources alone.

Two systematic reviews and meta-analyses have been included to highlight the requirement of greater dietary research in HF patients. The first meta-analysis by Rizos *et al.*[38] considered randomised controlled trials whereby *n*-3 were administered to participants by supplementation or diet, with outcomes being all-cause mortality, cardiac death, sudden death, MI and stroke. The authors found no significant relationship between *n*-3 supplementation and measured outcomes, although a substantial limitation is evident when examining the dose of *n*-3 intake used in studies. Indeed, studies using a higher dose of *n*-3 supplement tended to show benefit, yet they themselves were limited by small sample size and therefore did not carry weight in the analysis. A more recent meta-analysis[39] has also examined the relationship between *n*-3 and coronary risk as part of larger review of the relationship between all FA and coronary risk. The authors showed that *n*-3 supplementation was found not to be significantly associated with a reduced risk of coronary event in randomised controlled trials, whereas dietary *n*-3 intake was inversely associated with coronary outcomes in prospective studies. Indeed, this latter point is reinforced by the observation that a higher marine or dietary *n*-3 (EPA and DHA) intake is inversely associated with the development of HF[40]. It may be argued that if there is discrepancy in the dietary evidence base for the general population, is it safe and justifiable to offer the same advice to HF patients?

Considering all studies above regarding FAs, it is clear that the role of fat in HF is not as simple as once thought. Rather than focusing solely on the energy content of lipid, we should consider the biological and metabolic effects various FA may have, and use these to a potential therapeutic advantage. *n*-3 Supplementation may be of some benefit in HF patients, although it remains to be determined whether such benefits could be gained from increasing intake from dietary sources. At present, there are no recommendations for HF patients in terms of *n*-9 FAs, and thus it would be of use if appropriate studies were performed to examine the effects of increasing *n*-9 FA consumption in addition to *n*-3 in this patient group.

Sodium and fluid restrictions in heart failure

HF is characterised by altered renal perfusion, which itself leads to increased sympathetic activation and stimulation of the renin–angiotensin–aldosterone system (RAAS). Na and fluid are retained, leading to increased circulating volume in an attempt to preserve cardiac output. However, combined with fluid expansion, vasoconstriction caused by increased sympathetic activity raises blood pressure. Although initially beneficial, chronic activation of the RAAS and augmented Na and fluid retention increases both afterload and preload, contributing to oedema formation and congestive symptoms[41]. Reflecting the potential link between Na intake and fluid accumulation, the ACCF/AHA advise Na restriction in patients with symptomatic HF, although this class of recommendation is IIa and carries a C level of evidence[10]. Fluid restriction to 1·5–2·5 litres/d is also suggested by the ACCF/AHA in those patients with NYHA class IV[10], in particular patients with hyponatraemia, with a similar recommendation by European guidance (although the latter carries no class or recommendation or level of evidence)[1]. This is of concern given that Na and fluid restriction are viewed as a mainstay of dietary intervention in HF and is further complicated by the presence of 'salt-sensitive' phenotype, itself associated with increased mortality independent of blood pressure[42].

Several studies have shown little clinical benefit in restricting Na and/or fluid, although these may be confounded by their acute setting[43–45] (Table 2). Compared with acute decompensated HF patients managed with a free-fluid regimen, acute

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Table 2. Summary dietary sodium studies in heart failure (HF) patients presented in the current review

Study	Participant characteristics	Study design	Measures and time points	Key observations
Travers *et al.*[43]	Total participants: *n* 67 Control group: *n* 33 Intervention group: *n* 34 Control group: 48·4 % men Intervention group: 58·8 % men Control group EF: 40·2 % Intervention group EF: 37·4 % All patients had diagnosis of NYHA class IV HF Control group weight: 72·1 kg Intervention group weight: 76·2 kg Number screened, race and BMI not reported	Randomised controlled trial Control group group: free fluid Intervention group: fluid restriction to 1 litres/d free fluid	Renal profile measured for the duration of the experiment. BNP assayed for first 7 d and alternative days following this until stability. Daily weight, HF status and medication review Primary end point: time in days to clinical stability Secondary end points: changes in renal parameters, BNP, duration of intravenous HF therapy and compliance with fluid restriction Followed until clinical stability	Significant reduction in fluid intake in intervention group No significant difference in average weight loss, time to clinical stability, duration of intravenous HF therapy, BNP or renal profile at the time of clinical stability between groups
Aliti *et al.*[44]	813 individuals screened 738 excluded Total participants: *n* 75 Control group: *n* 37 Intervention group: *n* 38 Control group: 64·8 % men Intervention group: 73·6 % men Control group EF: 24·6 % Intervention group EF: 27·4 % Control group NYHA class: 45·9 % III, 48·6 % IV Intervention group NYHA class: 47·3 % III, 42·1 % IV Control group weight: 82·4 kg Intervention group weight: 78·0 kg Race and BMI not reported	Randomised controlled trial Control group: 3–5 g/d Na intake, minimum fluid intake of 2·5 litres/d Intervention group: 800 mg/d Na and 800 ml/d fluid	Daily assessment of perceived thirst, weight, use of intravenous diuretics, vasodilators and inotropes and clinical congestion score Serum biochemical analysis Primary outcome: weight loss and clinical stability during hospital stay (measured at 3 d) Secondary outcomes: assessment of thirst and hospital re-admission within 30 d of discharge 30-d follow-up	No statistical difference in length of stay, weight loss clinical congestion score, intravenous medications, laboratory tests or 30 d re-admission score Rating of thirst was significantly increased in the intervention group compared with the control. The intervention group showed significantly greater congestion at 30-d follow-up
Colín-Ramirez *et al.*[45]	451 individuals screened 413 excluded Total participants: *n* 38 Control group: *n* 19 Intervention group: *n* 19 Control group: 38·9 % men Intervention group: 36·8 % men Control group EF: 46·5 % Intervention group EF: 34·5 % Control group NYHA class: 84·2 % II, 15·8 % IV Intervention group NYHA class: 94·7 % II, 5·3 % IV Control group BMI categories: 0·0 % <18·5 kg/m^2, 21·1 % 18·5–24·9 kg/m^2, 26·3 % 25·0–29·9 kg/m^2, 52·6 % ≥30 kg/m^2 Intervention group BMI categories: 0·0 % <18·5 kg/m^2, 10·5 % 18·5–24·9 kg/m^2, 26·3 % 25·0–29·9 kg/m^2, 63·2 % ≥30 kg/m^2 Population 95 % white, 3 % Afro-American and 3 % South Asian Weight not reported	Randomised controlled trial Control group: moderate-Na intake (<2300 mg/d) Intervention group: low-Na intake (<1500 mg/d) Both groups were prescribed 50–55 % dietary energy from carbohydrate, 15–20 % protein and 25–30 % lipids, and were provided with a sample of six daily menus according to their energy requirements	3-d food record during the week before clinical visit (2 weekdays + 1 weekend day) Serum biochemical analysis, BNP Quality of life using KCCQ Baseline, 3-month and 6-month follow-up	2 patients dropped-out and 1 died Both groups significantly reduced Na intake compared with baseline values At 6 months, median BNP significantly reduced in the intervention group but did not differ between groups Median quality of life scores improved significantly in the intervention group and trended to improve in the control group No change in NYHA classification between groups was observed

Table 2. *Continued*

Study	Participant characteristics	Study design	Measures and time points	Key observations
Paterna *et al.*[46]	2 phases Phase 1 4728 screened. 1927 participants met entry criteria Total participants: *n* 1927 Control group: *n* 974 Intervention group: *n* 953 Control group: 37·1 % men Intervention group: 36·9 % men Control group EF: 34·4 % Intervention group EF: 33·7 % Control group weight: 84·5 kg Intervention group weight: 82·7 kg All participants were NYHA class III at entry Phase 2 Total participants: *n* 1927 Control group: *n* 974 Intervention group: *n* 953 Control group: 36·4 % men Intervention group: 37·3 % men Control group NYHA: 83·4 % I, 16·8 % II Intervention group NYHA: 77·2 % I, 22·8 % II BMI and race not reported	Randomised controlled trial Phase 1 Control group: intravenous infusion of furosemide (250 mg) twice daily, low-Na diet (1·8 g/d) and 1000 ml/d fluid restriction Intervention group: hypertonic saline solution (150 ml of 1·4–4·6 %) twice daily, intravenous infusion of furosemide (250 mg) twice daily, moderate-Na diet (2·8 g/d), 1000 ml/d fluid restriction Phase 2 Groups from phase 1 were continued on respective Na-restricted diets as out-patients	Serum biochemical analysis, BNP, 24-h natriuresis and diuresis, clinical and pharmacological assessment and cardiac function Primary outcomes: death or first hospitalisation for worsening HF Secondary outcomes: death from cardiac cause, hospitalisation for cardiac causes and combined end point of death from cardiac cause or hospitalisation for a change for a cardiac cause and change in NYHA classification Phase 1 Baseline and discharge Phase 2 Every week for the 1st month, every month for the first 6 months and 3-monthly thereafter	Phase 1 Significant increase in diuresis observed in both group from admission to discharge, although was significantly greater in the intervention group Natriuresis was significantly greater in the intervention group Significant increase in serum Na concentration in the intervention group. No increase in control group Significantly lower BNP in the intervention group at discharge when compared with the control group Greater number of patients moving from NYHA class III to class I following intervention Phase 2 156 subjects from phase 1 did not complete phase 2, leaving 1771 subjects who completed the study (control group *n* 890; intervention group *n* 881) BNP significantly lower in the intervention group when compared with the control group Greater weight stability and diuresis in the intervention group Significant reduction in mortality and combined mortality + re-admissions in the intervention group at 57 months of follow-up
Jefferson *et al.*[48]	Total participants: *n* 18 77·7 % men EF: 28·0 % NYHA class: 22·2 % I, 61·1 % II, 16·6 % III BMI: 31·1 kg/m^2 Weight and race not reported	Prospective study All participants received a <2000 mg/d Na-restricted diet + individualised counselling from a dietitian before discharge and during the study period (1 week)	3-d food record collected before baseline and daily food record during study Baseline and 1-week follow-up	2 subjects were excluded because of missing data. Final data based on *n* 6 Significant reduction in Na and energy intake at 1 week compared with baseline values Ca, phosphate, thiamine and folate intakes were significantly reduced at 1 week

EF, ejection fraction; NYHA, New York Heart Association; BNP, brain-type natriuretic peptide; KCCQ, Kansas City Cardiomyopathy Questionnaire.

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decompensated HF patients managed with fluid restriction showed no improvement in time to clinical stability or time spent receiving intravenous HF therapy[43]. An important limitation of this study is the difference in achieved fluid intake in both groups. In the free-fluid group, total daily fluid intake was 1466·6 ml compared with 1074·3 ml in the fluid-restricted group. Although it is statistically significant, clinically a greater restriction may have led to potential improvements; however, as the authors note, this may have increased thirst and reduced compliance. Similarly, a restriction of Na (800 mg/d) and fluid intake (800 ml/d) in acute decompensated HF patients increased thirst and led to no improvement in 30-d hospital re-admission rates when compared with a control group receiving no such restriction[44]. Furthermore, levels of brain-type natriuretic peptide (BNP) were significantly higher in the restricted group at the end of the study. A very real confounding factor in these trials examining Na restriction is their acute setting. Indeed, a low-Na (1500 mg/d) diet proved to be more effective at reducing BNP in ambulatory HF patients with NYHA II/III when compared with a moderate-Na (2300 mg/d) diet[45]. An important aspect of this study is the use of ambulatory HF patients as opposed to acute decompensated patients[40,41].

To further complicate the issue of Na restriction in HF patients, a moderate in-hospital Na restriction (2800 mg/d) combined with hypertonic saline solution, 250 mg twice daily intravenous furosemide and 1000 ml fluid restriction in patients with HFrEF produced a greater improvement in diuresis and natriuresis when compared with a group of HF patients receiving a greater Na restriction (1800 mg/d) and no hypertonic saline solution. These patients were discharged on their in-hospital Na and fluid restrictions in addition to 50–125 mg twice daily furosemide. Those who maintained the moderate-Na intake showed reduction in the occurrence of the combined end point of mortality and hospital re-admission in comparison with the restricted group[46]. The authors of this study speculate that the greater Na intake during the hospital admission and discharge may improve serum Na levels, chronically reduce neuro-hormonal activation and improve delivery of diuretics to the loop of Henle, thus increasing their action of diuresis (Table 2).

It is also relevant to consider in the context of Na restriction that salt taste diminishes with age[47], and that restricting Na in hospitalised HF patients may lead to an increased desire to satisfy the salt taste on discharge, further compounding difficulties of adhering to a low-Na diet. This concept would support the observations of Aliti *et al.*[44]. As such, consideration needs to be given to the different HF populations (ambulatory or hospitalised) in addition to the support required for patients to adhere to such a diet upon discharge. Without support, we are expecting a great deal from the elderly HF population, which may be an additional reason why low-Na diets are so difficult to follow. It would also be prudent to note that restricting Na intake in HF patients has been shown to be associated with reduced intake of other important nutrients such as Ca, phosphate, thiamine and folate[48], and therefore it would be advisable that patients discharged from hospital with low-Na advice receive regular follow-up to ensure compliance and also so that dietary adequacy can be reviewed (Table 2).

A recent Cochrane meta-analysis[49] has suggested that Na restriction leads to increased plasma renin, aldosterone, adrenaline and noradrenaline, irrespective of whether the individual is hypertensive or not, and as such may aggravate features of decompensated HF and explain the outcomes in previously mentioned studies. Furthermore, elevated levels of plasma renin activity have been linked with increased mortality in patients with stable symptomatic HF NYHA class III–IV, irrespective of pharmacotherapy[50]. In the analysis by Graudal *et al.*[49], the authors report that restriction of Na to a sub-normal level resulted in a 1 and 3·5 % decrease in systolic blood pressure (SBP) in normotensive and hypertensive individuals, respectively. They also suggested that in normotensives a greater duration of Na restriction produced a larger reduction in SBP (estimated mean difference of 0·4 mmHg); however, the reduction in SBP following Na restriction in hypertensive individuals did not appear to be time-dependent. It may be inferred from these observations that Na restriction may have a greater impact upon afterload in those HF patients with co-existing HTN who are salt-sensitive. Although HTN is more common in those individuals with HFpEF, it is not exclusive to this group, and therefore examining the specific benefits of low-Na diets in both hypertensive and non-hypertensive HFrEF and HFpEF populations would be of use.

Considering different responses to Na restriction between acute decompensated and compensated HF patients, in addition to those who may be more salt-sensitive, a well-designed clinical trial comparing short and long-term effects of Na restriction is required not solely on the outcome of mortality but on additional clinically relevant factors such as quality of life and hospital re-admission. A key recommendation should be that any Na and fluid Na restrictions need be individualised based on the severity of HF, dose of diuretic, degree of fluid accumulation and the clinical setting.

Dietary patterns and disease progression in heart failure

Discussion of the dietary management of each individual comorbidity experienced by HF patients is beyond the scope of this review. However, is the author's opinion that through appropriate nutritional education there is no reason why dietary patterns such as the Mediterranean or Dietary Approaches to Stop Hypertension (DASH) diet cannot be modified to account for comorbidities such as diabetes, COPD or gout, and act as an adjunct to traditional pharmacotherapy for these conditions in HF patients.

Dietary Approaches to Stop Hypertension and Mediterranean diet

Cohort studies have identified several dietary patterns as being cardioprotective. Famous examples include the Mediterranean and DASH diets[51]. A dietary pattern approach is important, as it acknowledges the synergistic effects of different foods, rather than focusing on a single nutrient, and recently studies have examined diet patterns in relation to specific outcomes in HF[52]. Higher intakes of salty foods are associated with a shortened

time to transplantation in patients with advanced HF, and increasing the intake of foods rich in MUFA and PUFA from 'occasionally' to 'several times a week' was associated with approximately 50 % reduction in risk of death/deterioration[52]. Other interesting results from this study include the association between different food groups. SFA was significantly associated with increased consumption of salty food, and inversely associated with MUFA and PUFA. Similarly, both MUFA and PUFA also positively correlated with fruits/vegetables/legume intake, thus suggesting that the consumption of one nutrient may predict other dietary components. This observation may be important for the clinician or dietitian when taking a diet history, and it may allow a more rapid determination of diet quality. However, although interesting, this study is limited by the use of the food FFQ and does not provide information on the amount of such nutrients consumed by the participants.

The DASH diet has a recognised beneficial effect in delaying the incidence of HF[53], and it should be examined for use in HF patients. Such a diet is typically low in SFA, with increased consumption of low-fat dairy products, complex carbohydrate, fish and vegetables[51]. This dietary pattern is in contrast to that of the UK population, which typically consumes a diet higher in refined carbohydrate and SFA and lower in vegetables[54]. If individuals with HF are required to change their diet, support and guidance to the most appropriate way of achieving an optimal nutrient intake should be provided.

Hummel *et al.*[55] demonstrated a significant improvement in ventricular diastolic function in thirteen patients with HFpEF when these patients were provided with a Na-restricted DASH diet (50 mmol/8786 kJ (2100 kcal)). Specifically, adherence to this dietary pattern improved EF by 8 % and increased stroke volume by approximately 11 %. Although impressive, the relatively small sample size and feeding protocol (controlled feeding with prepared meals) mean that such a finding may not be observed in free-living individuals with HF. In addition, the nature of the population studied means that this finding may also be only linked to those with HTN and HFpEF (Table 3). The Geriatric Out of Hospital Randomised Meal Trial in Heart Failure is one such study that will address whether such findings can be reproduced using a home-delivered low-Na meal, examining quality of life and cardiac functional parameters, although this study itself is still limited by the provision of meals[56].

Levitan *et al.*[57] studied women enrolled in the Women's Health Initiative who were admitted to hospital with HF to identify whether adherence to a Mediterranean or DASH diet pattern influenced CVD mortality. After a median of 4·6 years of follow-up, there were 1385/3215 deaths following HF hospitalisation. When stratified into quartiles, greater adherence to either the Mediterranean or DASH diet was associated with a substantial reduction in the hazard rate (HR) associated with mortality. Specifically, the HR for death was 16 and 15 % lower in the DASH and Mediterranean diet group, respectively, although only reaching significance in the DASH group. Further analysis of the dietary intake of either Mediterranean or DASH patients revealed that greater adherence to each diet was associated with increased consumption of fruit and vegetables, nuts, legumes, whole grains and fish, and reduced intake of sweetened beverages and red and processed meat. However, important limitations of this study were acknowledged by the authors, including difficulty in recording Na, fluid and olive oil intakes, in addition to the group comprising those diagnosed with HFpEF. Although the results may be promising for the DASH diet, they do not support the advocacy for the Mediterranean-style diet, despite a favourable trend. However, previous cross-sectional data have shown that adherence to a Mediterranean Diet is associated with improved diastolic function in individuals with congestive HF[58] (Table 3), and subsequent studies have shown the Mediterranean diet to reduce HF biomarkers in individuals at high-risk CVD[59]. Therefore, at present, the role of the Mediterranean diet in the management of HF remains to be fully examined. There is a clear need for large, randomised trials investigating whether the improvement in mortality rate observed in the DASH group is driven by the restriction in Na or a rather combined effect of diet and Na restriction, and whether the Mediterranean diet has a role in the management of HF.

Low carbohydrate and high protein

There are several interesting reports regarding the use of low-carbohydrate diets in humans with HF. However, an important limitation of some of these studies cited is that they are almost exclusively conference abstracts, and thus caution should be exercised when interpreting them. Nonetheless, in patients with HF and right-ventricular dysfunction, a diet classified as low in carbohydrate (40 % carbohydrate, 40 % fat, 20 % protein) has been shown to be effective at increasing weight loss and improving O_2 saturation when compared with a conventional diet containing 50 % of energy as carbohydrate[60]. In addition, the authors report an improvement in HF functional class. Similar to many HF trials, the study suffered from a relatively small sample size and short duration, including twenty-one individuals studied for a duration of 2 months. Therefore, the long-term consequences of such a pattern remain unknown in HF patients. Importantly, this study highlights a key issue facing nutritional interventions: how diets are defined; 40 % energy as carbohydrate may be regarded by many as not being 'low carbohydrate' and is consistent with that achieved in the Prevención con Dieta Mediterránea (PREDIMED) study[61] (widely defined as a Mediterranean diet). It would be appropriate for the The National Heart, Lung, and Blood Institute and National Institutes of Health Office of Dietary Supplements Working Group[62] to also consider a standard protocol for reporting the nutritional composition of experimental diets in HF studies to facilitate greater comparison of dietary interventions, in addition to their other current recommendations (Table 3).

Modifying protein intake has been shown to be effective in reducing weight in obese patients (mean BMI 37·3 kg/m^2) with NYHA class II–III HF. Evangelista *et al.*[63] compared a 12-week hypoenergetic diet (5021–6276 kJ/d (1200–1500 kcal/d)) containing (as percentage of energy) 30 % protein, 40 % carbohydrate and 30 % fat with a standard protein, hypoenergetic diet (55 % total energy from carbohydrates, 15 % from protein and 30 % from fat) or the recommendations by the AHA. The authors noted that the high protein hypoenergetic diet led to a

Table 3. Summary dietary studies in heart failure (HF) patients presented in the current review

Study	Participant characteristics	Study design	Measures and time points	Key observations
Spaderna *et al.*[52]	380 participants met inclusion criteria 340 consented. 22 did not complete questionnaire Total participants: *n* 318 72·8 % men EF: 21·5 % NYHA class(*n* 316): 39·6 % II, II–III, III, 36·1 % III–IV, 24·4 % IV BMI: 25·9 kg/m^2 Race and weight, not reported	Prospective study Participants recruited from The Waiting for a New Heart Study were mailed a FFQ	FFQ and fluid intake Resting heart rate, EF, mean blood pressure, peak VO_2, serum Na, interventricular conduction delay, ischaemic diagnosis (used to calculate Heart Failure Survival Score) Death on waiting list, high-urgency transplantation, elective transplantation, delisting because of clinical deterioration or improvement Baseline and occurrence of outcome listed above (mean follow-up of 462·8 d)	6 participants were lost to follow-up Fluid intake >2 litres/d associated with hyponatraemia Greater intake of salty food significantly associated with shortened time to transplantation Consumption of foods high in MUFA + PUFA associated with reduced hazard ratio for death: deterioration
Hummel *et al.*[55]	Screened 22 participants Total participants: *n* 14 7·1 % men EF: 66·0 % NYHA class: 14·3 % II, 85·7 % III Weight: 94·0 kg BMI: 35·5 kg/m^2 Race not reported Total population classed as displaying HFpEF	Prospective study Participants randomised to a DASH diet with a goal of 1150 mg Na/ 8786 kJ (2100 kcal)	3-d food diary, 24-h urinary Na and K, blood pressure and cardiac function Day 1 (blood pressure) and 2 (cardiac function), and 25 d of follow-up (21 d of diet)	1 participant was withdrawn because of hyperkalaemia Significant decrease in systolic blood pressure following diet Arterial elastance, stroke volume and EF all improved significantly following dietary intervention
Levitan *et al.*[57]	Identified 4043 participants Excluded 828 Total participants: *n* 3215 0·0 % men BMI: 30·5 kg/m^2 85·4 % white not of Hispanic origin, 10·5 % black, 1·7 % Hispanic, 1·0 % Asian/Pacific Islander, 0·5 % American Indian/Alaskan Native No measures of cardiac function or NYHA classification or weight	Prospective study Participants were taken from the Women's Health Initiative dietary modification and observational study and were followed up from HF hospitalisation to date of death or last contact with participant before August 2009	Modified block FFQ, Mediterranean and DASH diet scores Median follow-up of 4·6 years	1385 deaths occurred, of which 694 were attributable to CVD Women who died were older, more likely to smoke, were less active and had a lower BMI Highest quartile* of Mediterranean and DASH scores had greater intake of fruit and vegetables, nuts, legumes, whole grains, low-fat dairy products, fish and lower intakes of red and processed meat, in addition to sugar-sweetened beverages Higher DASH score associated with significantly lower hazard rate of death Non-significant trend for lower hazard rate for death following Mediterranean diet Vegetables, nuts, nuts and legumes and whole grain were inversely associated with mortality post hospitalisation from HF

Table 3. *Continued*

Study	Participant characteristics	Study design	Measures and time points	Key observations
Chrysohoou *et al.*[58]	Total participants: *n* 372 84·4 % men BMI: 28·0 kg/m^2 All participants were of HFrEF (EF <40 %) Race, NYHA class and EF were not reported	Cross-sectional Statistical power of 87 %	Semi-quantitative FFQ and Mediterranean diet score, cardiac function	Greater adherence to Mediterranean diet associated with a significant improvement in diastolic function and flow propagation Greater intake of fish, olive oil and vegetables associated with improvements in diastolic indices
Olvera *et al.*[60]	Total participants: *n* 39 Control group: *n* 18 Intervention group: *n* 21 Number randomised, sex, race, BMI, EF and NYHA class not reported This study was performed in patients with HF and right-ventricular dysfunction	Randomised controlled trial Control group: standard diet with 50 % energy from carbohydrate, 30 % from fat and 20 % from protein Intervention group: 40 % energy from carbohydrate, 40 % from fat and 20 % from protein Na and fluid intake not available	Bioelectrical impedance and anthropometry, stress test and laboratory assessments Baseline and 2-month follow-up	Significant reduction in weight in the intervention group Significantly greater number of individuals with improved symptoms in the intervention group compared with the control group Improvement in O_2 saturation following intervention
Evangelista *et al.*[63]	Total participants: *n* 14 Control group: *n* 4 Intervention group 1: *n* 5 Intervention group 2: *n* 5 Control group: 75·0 % men Intervention group 1: 80·0 % men Intervention group 2: 80·0 % men Control group EF: 26·6 % Intervention group 1 EF: 27·8 % Intervention group 2 EF: 23·8 % Control group NYHA class: 25·0 % II, 75·0 % III Intervention group 1 NYHA class: 40·0 % II, 60·0 % III Intervention group 2 NYHA class: 40·0 % II, 60·0 % III Control group weight: 109·8 kg Intervention group 1 weight: 110·8 kg Intervention group 2 weight: 99·5 kg Control group BMI: 40·7 kg/m^2 Intervention group 1 BMI: 37·3 kg/m^2 Intervention group 2 BMI: 35·9 kg/m^2 kg/m^2 Control group LHFQ: 70·9 Intervention group 1 LHFQ: 68·5 Intervention group 2 LHFQ: 73·0 Control group peak VO_2: 10 9 ml/kg per min Intervention group 1 peak VO_2: 13·5 ml/kg per min Intervention group 2 peak VO_2: 12·7 ml/kg per min Race not reported	Randomised controlled trial Control group: AHA recommendations for healthy adults. No energy restrictions Intervention group 1: high-protein hypoenergetic diet (40 % total energy from carbohydrates, 30 % from fat and 30 % from protein) Intervention group 2: standard protein, hypoenergetic (55 % total energy from carbohydrates, 30 % from fat and 15 % from protein) Both intervention groups participated in intensive 12-week supervised weight-loss intervention Meal plans designed to incorporate 2092–3347 kJ/d (500–800 kcal/d) deficit	Anthropometry, functional status, biochemical measurements, LHFQ and 3-d food diary Baseline and 12-week follow-up	Significantly greater weight loss in intervention group 1 compared with intervention group 2 and control group Trend towards increased lean mass in the intervention group 1 Greater improvement in LHFQ in intervention group 1 than in intervention group 2 Significant improvement in VO_2 peak in intervention group 1

EF, ejection fraction; NYHA, New York Heart Association; HFpEF, HF with a preserved or normal EF; DASH, dietary approaches to stop hypertension; HFrEF, HF with reduced EF; LHFQ, Minnesota Living with Heart Failure Questionnaire; AHA, American Heart Association.
* Tertitles for sugar-sweetened beverages because of a limited range of intake.

Table 4. Summary of nutritional education studies in heart failure (HF) patients presented in the current review

Study	Participant characteristics	Study design	Measures and time points	Key observations
Sethares & Elliott[70]	Recruited 88 participants 8 withdrew and 10 died before follow-up Total participants: *n* 67 Control group: *n* 37 Intervention group: *n* 33 Control group: 43·2 % men Intervention group: 51·5 % men Control group: 89·2 % white Intervention group: 93·9 % white Control group EF: 38·8 % Intervention group EF: 41·5 % Control group NYHA class: 3 Intervention group NYHA class: 3 BMI and weight not reported	Randomised controlled trial Control group: received usual care Intervention: received tailored message during hospitalisation, 1 week and 1 month post discharge	Health belief scales, LHFQ, medication and hospital re-admission rates LHFQ determined at 1 month post discharge Change in benefit and barriers towards medications, diet and self-monitoring at 1 week and 1 month Re-admission rate at 3 months	No significant change in hospital re-admissions between groups No change in quality-of-life scores Intervention led to a significant improvement in understanding benefits and barriers towards diet and self-monitoring No change to perceived benefit of medication between groups
Arcand *et al.*[71]	Recruited 50 patients 3 excluded Total participants: *n* 47 Control group: *n* 23 Intervention group: *n* 24 Control group: 73·9 % men Intervention group: 75·0 % men Control group EF: 23·0 % Intervention group EF: 22·0 % Control group mean furosemide: 82 mg/d Intervention group mean furosemide: 90 mg/d Weight, BMI, race or NYHA class not reported	Randomised controlled trial Control group: Prescribed 2 g/d Na diet and provided with self-help low-Na literature Intervention: prescribed 2 g/d Na diet, low-Na literature plus two education sessions with a dietitian	3-d food record (including 2 weekdays + 1 weekend) Primary outcome: change in Na intake Secondary outcomes: weight, medication fluid Baseline and 3-month follow-up	Significant reduction in dietary Na intake following the intervention No change in dietary macronutrients between groups
Kollipara *et al.*[72]	Recruited 105 patients 7 excluded Total participants: *n* 97 Very low dietary Na knowledge: *n* 40 Not very low dietary Na knowledge: *n* 57 Very low dietary Na knowledge: 63·0 % men Not very low dietary Na knowledge: 70·0 % men Very low dietary Na knowledge: 78·0 % African-American Not very low dietary Na knowledge: 82·0 % African-American BMI, weight, EF and NYHA class	Prospective Participants grouped based on dietary Na score ≤3: very low dietary Na knowledge ≥4: not very low dietary Na knowledge	Na knowledge assessed by Parkland Dietary Sodium Knowledge Test, TOFHLA 90-d hospital re-admission	90-d hospital re-admission is inversely associated with Na knowledge Significant association between TOFHLA and dietary Na knowledge following intervention
Colín-Ramirez *et al.*[73]	Randomised 65 patients 8 excluded or lost to follow-up Total participants: *n* 58 Control group: *n* 31 Intervention group: *n* 27 Control group: 61·3 % men Intervention group: 33·3 % men Control group EF: 42·3 % Intervention group EF: 40·0 % Control group NYHA class: 56·7 % I, 30·0 % II, 13·3 % III Intervention group NYHA class: 59·3 % I, 22·2 % II, 18·5 % III Control group BMI: 27·3 kg/m^2 Intervention group BMI: 27·5 kg/m^2 Control group weight: 67·6 kg Intervention group weight: 63·9 kg Race not reported	Randomised controlled trial Control group: traditional dietary advice regarding Na and fluid intake Intervention: prescribed 2–2·4 g/d Na, 50–55 % dietary energy from carbohydrate, 15 % protein and 30–35 % lipids. Fluids limited to 1·5 litres/d. Received written and oral advice from a dietitian	Serum biochemical analysis, adapted KCCQ score and LHFQ, physical activity and 3-d food questionnaire (2 weekdays + 1 weekend) Baseline and 6-month follow-up	Significant reduction total fat and SFA following intervention Intervention led to a significant reduction in Na and fluid Significant reduction in the number of NYHA class II and II and increase in class I in the intervention group

EF, ejection fraction; NYHA, New York Heart Association; LHFQ, Minnesota Living with Heart Failure Questionnaire; TOFHLA, Test of Functional Health Literacy in Adults; KCCQ, Kansas City Cardiomyopathy Questionnaire.

greater reduction in percentage of body fat and improved the patient's quality of life (assessed by the Minnesota Living with Heart Failure Questionnaire). However, this study was performed in five individuals, and it is therefore severely limited by the small sample size (Table 3). At present, there are no available large-scale dietary trails investigating protein intake and cardiac structure and function, functional status and quality of life in HF patients, although these are in development[64].

The obesity paradox

Studies by Chrysohoou *et al.*[58] and Estruch *et al.*[61] suggest a beneficial effect of weight loss in HF patients; however, it is important to recognise that uncontrolled weight loss in HF is linked with increased incidence of mortality[3]. The importance of weight in HF patients has frequently been examined as part of the obesity paradox. The obesity paradox refers to observations that link the presence of obesity (and in some instances overweight) in HF patients with improved survival in comparison with lean counterparts. Horwich *et al.*[65] was one of the first groups to demonstrate the inverse relationship between weight and mortality in patients with HF. In this study, the majority of participants were of NYHA class IV and had an EF of 22 %, with obese patients more likely to have diabetes and HTN. Following multivariate analysis, overweight and obesity were found to be associated with a significant survival benefit at 2 years, with the worst prognosis seen in those who were underweight, followed by those who were classified as recommended weight. Importantly, although this study is used to draw evidence to the protective nature of obesity, the survival benefit was not evident at the 5-year follow-up. In addition, categorisation of patients as underweight at baseline may not have accounted for unintentional weight loss before the study. Importantly, this study was only performed in individuals with HFrEF, and therefore it may not apply to those with HFpEF. Despite this, subsequently larger meta-analysis studies have further reinforced this observation. Oreopoulos *et al.*[66] analysed a total of nine observational studies demonstrating that both overweight and obesity were associated with a reduced relative risk of all-cause and cardiovascular mortality when compared with patients with normal BMI levels. Regrettably, the authors of this study did not extract data on EF; however, a more recent a meta-analysis examined whether HF subtype (HFrEF *v.* HFpEF) affected the obesity paradox. Using individual patient data, Padwal *et al.*[67] demonstrated the existence of a U-shaped relationship between BMI and all-cause death in both HFrEF and HFpEF patients. In patients with HFrEF or HFpEF, the lowest hazard ratio for all-cause mortality was observed when comparing those individuals with a BMI between 30 and 34·9 kg/m^2 against the reference BMI range of 22·5–24·9 kg/m^2. In both subtypes, a BMI <22·5 kg/m^2 was associated with a higher risk of all-cause death.

There may be several mechanisms behind the proposed obesity paradox in HF. It is well known that advanced HF is associated with cachexia[8], and in this regard greater adiposity may simply reflect greater body energy stores and hence greater resistance to the metabolic changes associated with the cachexic state. As shown by Padwal *et al.*[67], individuals who were obese were also more likely to be receiving cardiovascular medication, potentially suggesting greater clinical input and therefore greater clinical management of their condition. However, it should be noted that this was adjusted for in their study with no effect upon their findings. Also, the use of BMI as a marker of fatness in HF has been questioned, with more accurate measurements of body composition being proposed[68]. The presence of the obesity paradox means that we may need to re-examine advice to achieve a healthy weight in HF patients, and it raises important questions regarding the role of weight loss[60,63] on the outcome of mortality. There may be a point at which excess weight is not associated with any additional benefit but conversely increases risk. Indeed, in morbidly obese (BMI ≥40 kg/m^2) HF patients, the obesity paradox is absent[69]. Therefore, one may conclude that in those individuals with morbid obesity intentional weight loss may be beneficial in terms of reducing mortality rate; however, this should be carefully monitored and controlled. In lower-BMI categories, a reduction in weight may improve clinical symptoms and disease classification, but it may have a negative impact on long-term survival. It would be useful for future studies examining the relationship between body weight and HF mortality to assess adipose tissues deposits (both visceral and subcutaneous) and lean mass, in addition to cardiorespiratory fitness following weight loss.

Nutritional messages: the role of the dietitian

A key aspect of implementing a dietary strategy is addressing pre-conceived ideas and beliefs regarding nutrition. A tailored nutritional message to patients with HF is sufficient to alter patients' views and attitudes towards medications, adherence to a Na-restricted diet and self-monitoring[70]. Further support for the importance of nutritional input can be derived from Arcand *et al.*[71]. In this 3-month study, HF patients randomised to a dietitian-led education group showed greater improvements in salt reduction in comparison with usual care (self-help literature). Although such a frequent dietetic input may be unlikely in the current health-care setting, clinicians reviewing their patients may wish to follow-up nutritional advice and reinforce nutritional messages at every opportunity. Indeed, frequent nutritional counselling with HF patients may improve knowledge surrounding foods and reduce admissions. In HF patients, a low level of Na knowledge has been shown to be associated with a significantly greater OR for hospital re-admission for HF[72]. Using the Test of Functional Health Literacy in Adults tool, Na knowledge was associated with a low health literacy score. When nutritional interventions are combined with appropriate educational session, substantial improvement in quality of life and disease score can be seen. For example, a nutritional intervention consisting of 2000–2400 mg/d Na, 50–55 % (as percentage of energy) carbohydrate, 15 % protein, <10 % SFA, 15 % MUFA and 10 % PUFA coupled with written and oral instructions from a dietitian led to a significant improvement in HF classification and quality of life when compared with a control group receiving general

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nutritional advice[73]. Indeed, the improvement in HF classification was reflected by a significant reduction in the number of individuals with NYHA class II and III and an increase in the number of those with class I by the end of the study (Table 4).

As such, this would suggest that by using appropriate methods of patient education and trained individuals, it is never too late to make important and significant dietary changes that may improve quality of life.

Discussion and conclusions

HF remains a chronic and debilitating condition. Although the value of dietary manipulation is well known in the primary, secondary and tertiary prevention of CVD, it is undervalued in patients with HF and is reflected by the paucity of data in guidelines. Despite a large body of experimental data produced from animal models of HF examining the effect of different diet compositions, this has not translated into human trials. From animal trials, it is clear that the traditional demonisation of fat may not be justified in HF, and human studies should be designed to evaluate the therapeutic effectiveness of cardioprotective fats in HF. Within this, consideration should be given to the underlying HF aetiology in addition to other comorbidities. Indeed, by manipulating dietary nutrient composition, it is possible for those individuals with other comorbidities to benefit from the potential therapeutic nature of food.

Studies that have been published in this field – albeit largely observational – now suggest that diet advice in this area may need to be re-examined, with the traditional cardioprotective diets such as the Mediterranean and DASH potentially being of benefit. Such diet patterns have been shown to increase the consumption of cardioprotective food items such as fruit and vegetables, nuts, legumes, whole grains and fish and are likely to have additional health effects beyond HF.

It is simple to decide what foods an individual should consume, yet much more difficult to actually achieve this. Regular nutritional education has been shown to lead to better adoption of a prescribed diet and may lead to improved overall nutritional status. In some studies, this has also translated to improvements in quality of life and reduced severity of symptoms when delivered by nutritionally trained individuals. The feasibility of such a means of improving nutritional knowledge is clearly in need of evaluation, given the potential cost such a service may incur.

Although the studies presented in this review are promising, many are limited by small sample sizes, short duration and observational study design. It is therefore a requirement that in order to progress towards better evidence-based dietary advice for patients with HF, larger, longer, randomised clinical trials are needed. Such studies should account for differences in HF subtype (HFrEF *v.* HFpEF) and have clearly defined clinical end points. In addition, there is a requirement for standardisation of dietary reporting. The studies highlighted in this review provide a potential starting point for the development of future trials, and fundamentally demonstrate that, in addition to fluid and Na, consideration should be given to other dietary components.

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