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Nutrition intervention in heart failure: should consumption of the DASH eating pattern be recommended to improve outcomes?

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Abstract
Heart failure (HF) is a chronic, systemic disorder that presents a serious and growing public health problem due to its high prevalence, mortality, and cost of care. Due to the aging of the population and medical advances that prolong the life of HF patients, more effective, widespread strategies for improved HF management in this rapidly growing patient population are needed. While the prevalence of malnutrition in HF patients has been well characterized, evidence is limited regarding the effects of specific macro- and micronutrient deficiencies on HF outcomes and their interaction with other aspects of HF management, including pharmacotherapy. There is a mounting appreciation for the effects of nutritional intervention on pathophysiology, treatment, and outcomes in patients with HF. Heart-healthy dietary patterns, such as the Dietary Approaches to Stop Hypertension (DASH), carry importance for the prevention and treatment of hypertension. While preliminary evidence looks promising regarding effects of DASH eating pattern consumption on ventricular function and 30-day hospitalizations in HF patients, more research is needed to confirm its effects on short-term and long-term HF outcomes while better understand underlying mechanisms in the context of HF pharmacotherapy.

Keywords Heart failure · Nutrition · Intervention · DASH · Malnutrition

The burden of heart failure

Heart failure (HF) is a systemic disorder that presents a serious and growing public health problem due to its high prevalence, mortality, and cost of care. Approximately 6.5 million Americans aged 20+ years lived with a diagnosis of HF based on the National Health and Nutrition Examination Survey 2012–2014, and projections show that this number will increase to over 8 million by the year 2030. Total medical cost attributable to HF was $30.7 billion in 2012, about 80% of which were related to hospitalizations [1]. Older Americans are more often hospitalized for HF than for any other condition [2]. Most (83%) of HF patients are hospitalized at least once, and 45% are hospitalized four times or more post diagnosis [1]. With 29.6% of Medicare beneficiaries not surviving the first year after a HF hospitalization, mortality in this population remains high [3]. Given that rehospitalization, death, and high cost of care are most likely in those previously hospitalized, emphasis has been placed on strategies to reduce the rate of HF hospital readmissions through policy imposing financial penalties for hospitals with excessive 30-day readmission rates, efforts to increase implementation of evidence-based prevention and treatment approaches in ambulatory and hospital settings, and new preventative measures and therapies, including the use of implantable hemodynamic measurement devices [4]. Due to the aging of the population and medical advances that prolong the life of HF patients, more effective, widespread strategies for improved HF management in this rapidly growing patient population are needed.

Dietary sodium restriction in HF management

Arguably the most heavily debated HF treatment strategy has been dietary sodium restriction [5, 6]. Assumed to be a

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cornerstone of optimized HF self-management, various levels of sodium restriction have been endorsed by clinical guidelines despite low levels of evidence (e.g., expert opinion, level of evidence C). Intake recommendations vary widely from 3000 to 4000 mg/day ("moderate"), as recommended by the American College of Cardiology/American Heart Association, to less than 2000 mg/day in moderate to severe HF, as recommended by the HF Society of America. In contrast, the 2016 European Society of Cardiology HF Guidelines omits sodium intake recommendations entirely due to the weak evidence base [7]. Moreover, the American Heart Association recommends a more stringent sodium intake restriction of 1500 mg/day for the general population than for HF patients, which appears as a paradox [5]. Heart failure pathophysiology is characterized by systolic and diastolic abnormalities of the myocardium that result in reduced cardiac output (CO) and reduced renal perfusion. These trigger compensatory mechanisms, including left ventricular dilatation and activation of the sympathetic and renin-angiotensin-aldosterone systems (RAAS) which lead to a vicious cycle of systemic vasoconstriction, increased blood pressure and heart rate, and sodium and water retention despite existing volume overload. Physiological and mechanistic studies have shown that dietary sodium restriction leads to further neurohormonal activation, is not associated with an improvement in glomerular filtration rate or natriuresis, and may result in increased plasma renin activity (PRA), worsened cardiac remodeling, and increased HF symptoms even in the presence of pharmacotherapeutic blockage of the RAAS. High PRA is an independent predictor of mortality in high-risk HF patients [8, 9]. Furthermore, evidence from few randomized controlled trials suggests either no benefit of sodium restriction on clinical improvement, diuretic therapy, and hospital readmission rates [10, 11] or lower mortality and hospital readmission rates for patients receiving a moderate (2800 mg/day) vs. low-sodium (1800 mg/day) diet [12, 13]. A meta-analyses of nine randomized controlled studies demonstrated a lack of high-quality evidence (e.g., small sample sizes) and inconclusive results regarding the improvement of clinical outcomes in both inpatient and outpatient HF patients with dietary sodium restriction [14]. The need for more robust evidence has been emphasized in a joint executive summary from the US National Heart, Lung, and Blood Institute and the National Institutes of Health Office of Dietary Supplements [14]. Support to restrict dietary sodium stems mostly from observational studies that have shown greater symptom burden, increased HF hospitalizations, increased need for heart transplantation, and increased mortality in patients with greater sodium intake over an extended period of time, using the gold standard 24-h urine sodium excretion method to estimate dietary sodium intake [15–19]. Understanding the true independent effect of sodium consumption on HF outcomes appears to be a difficult undertaking. Such efforts would include addressing limitations of previous studies by using adequately designed and powered RCT to assess the effect of sodium consumption in the context of pharmacotherapy, fluid and nutrient intake on important HF outcomes, using validated measures, and long-term follow-up.

Malnutrition in HF patients

The etiology of malnutrition in HF patients is likely multifactorial and intertwined (Fig. 1). Anorexia, characterized as both inadequate food intake and suppressed appetite, can be attributed to the underlying pathophysiology of their disease, defined by a chronic inflammatory state and fluid overload, which leads to nausea, loss of appetite and early satiety from gastrointestinal edema, and hepatic congestion [20, 21]. The development of appetite suppression, early satiety, and insulin resistance is further exacerbated by neurohormonal etiologies of anorexia [1]. For example, leptin-induced anorexia has been characterized in patients with cardiac cachexia, as evident with significant decreases in leptin levels [20]. Reduced energy intake and negative energy balance have been associated with lower intake of all macronutrients, including total and saturated fat, protein, and carbohydrate intake [10–12]. Unfortunately, sodium restrictive diets, often recommended to HF patients, could potentiate decreased food intake due to decreased palpability of foods which is problematic in the context of chemosensory impairments commonly present in older adults [22]. As a consequence, sodium-restricted diets may contribute to anorexia and poor nutritional status in this population. With increasing HF progression, malnutrition is further exacerbated by increased energy requirements from higher resting metabolic rates in patients with severe HF [23]. Patients with HF are in a hypercatabolic state with increased levels of catecholamines, including epinephrine, norepinephrine, and cortisol, and decreased anabolic hormones, such as insulin-like growth factor [24]. Increased catecholamine levels likely contribute to malnutrition in HF patients through the development of intestinal ischemia with decreased splanchnic circulation leading to increased permeability and increased inflammatory cytokine release and can attribute to malabsorption of lipids on the one, and excessive protein loss on the other hand [4]. Therefore, HF disease pathophysiology predisposes patients to a disadvantage in nutritional status, as patients with HF are experiencing malnutrition from an energy imbalance arising from increased energy requirements and decreased energy intake.

Malnutrition in patients with HF has been associated with cardiac cachexia, a serious complication of HF, characterized by loss of lean muscle, fat, and bone tissue, and a risk factor for HF prognosis and mortality that is independent of HF severity, left ventricular ejection fraction, exercise capacity, and patient age [25]. Cardiac cachexia is defined as

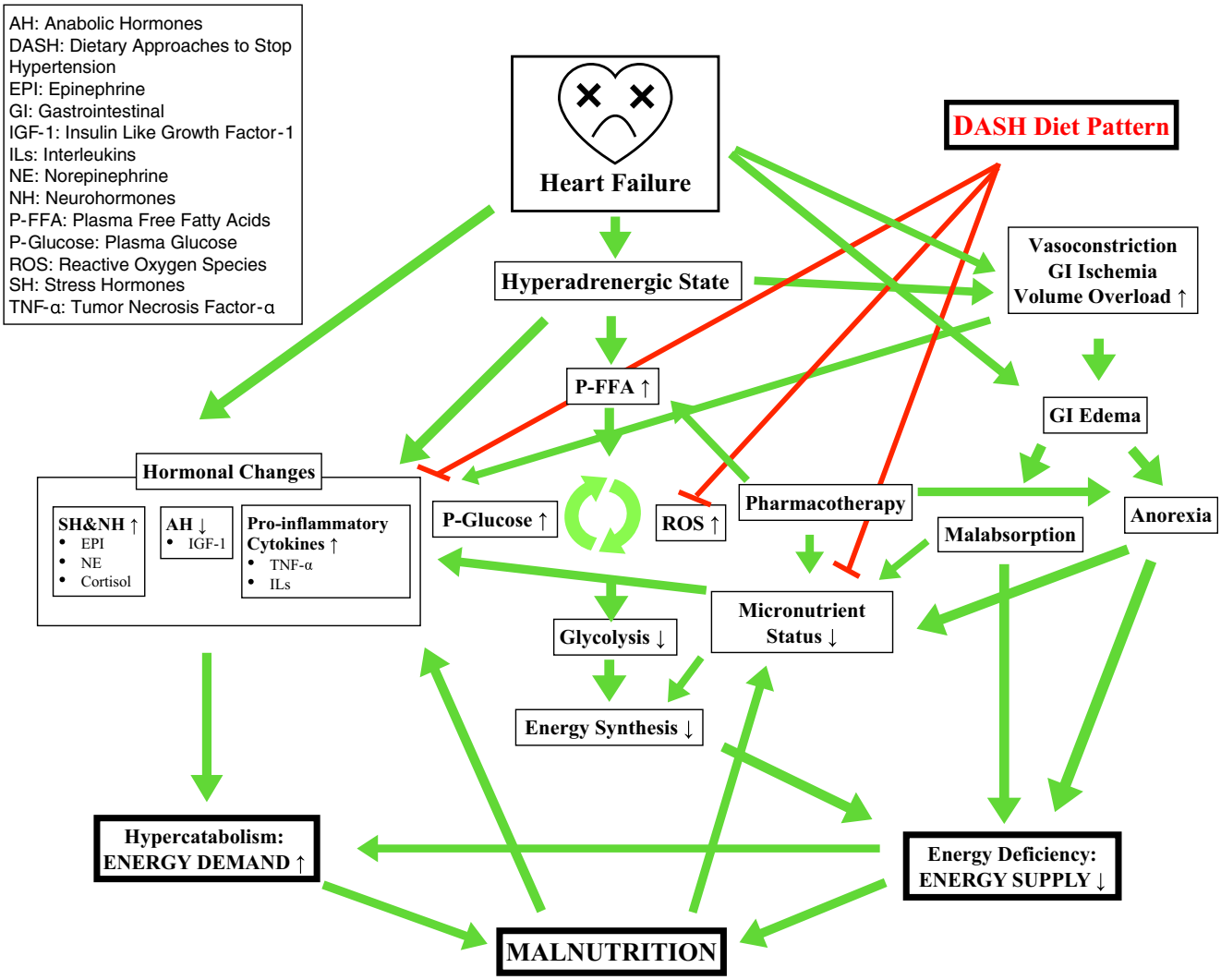

Fig. 1 Suggested influence of the Dietary Approaches to Stop Hypertension (DASH) Diet on heart failure pathophysiology and nutritional status: a conceptual framework

unintended, edema treatment-independent weight loss equal to at least 6% of body weight within 6–12 months and is a result of pathophysiological manifestation and progression of HF [25]. Half of patients with HF have some degree of malnutrition, with 15% being in a state of cardiac cachexia [24, 26]. The progression of cardiac cachexia is strongly associated with increasing levels of functional decline, as demonstrated by NYHA classification [27]. In addition, mortality rates are increased in patients with cardiac cachexia, with rates up to 50% as compared with 17% in non-cachectic patients [24]. Abnormal metabolism and a chronic inflammatory state, characterized by excessive release of tumor necrosis factor (TNF)-alpha, previously termed cachexin, represent the underlying etiologies of cardiac cachexia. The effect of TNF-alpha on muscle wasting can be explained by its direct effect on the endothelium and by mitigating cell apoptosis and activating protein degeneration through which lean tissue mass is weakened in both structure and function [26, 28]. This chronic systemic inflammatory state is further worsened by the release of oxidative species and intestinal microorganism and/or endotoxin translocation, due to the intestinal edema that results from fluid overload due to HF [25, 26, 28, 29]. A vicious cycle ensues, as cardiac dysfunction manifests (e.g., reduced cardiac output) which in turn leads to cascading downstream effects including intestinal hypoperfusion, peripheral edema, and mucosal permeability. These factors affect normal processes of nutritional intake and absorption [24–26, 29]. In addition, patients with cardiac cachexia show heightened circulating levels of the metabolic hormone adiponectin, which is associated with lean muscle wasting and represents an independent risk factor for mortality [30, 31].

Due to the pathophysiological link between malnutrition and HF, several studies have investigated the prognostic impact of nutrition on HF outcomes, using nutritional assessment indices based on biochemical and clinical markers, including serum albumin, total cholesterol levels, and total lymphocyte

count [4, 5]. Scores that represented poorer nutritional status were associated with longer hospital stays, in-hospital mortality, progressive HF requiring readmission, and cardiovascular events [32–34]. While there are limitations to these indices due to an underlying pathophysiological state of inflammation in HF per se, they may detect both poor malnutrition and cardiac cachexia. For example, prealbumin, a clinical marker of nutritional status, could be indicative of protein malnutrition or altered due to disproportionate mobilization of muscle and degradation of protein [35].

Unlike non-cardiac patients, there is no significant association between dietary energy intake and body mass index (BMI) in persons with HF, as the nutritional status of HF patients is greatly influenced by variables outside of dietary energy consumption alone [10]. Therefore, use of BMI as a sole factor in nutritional assessment as a means of risk stratification is not recommended, particularly as it masks malnutrition in the presence of edema which complicates accurate weight measurements [36, 37]. Furthermore, malnutrition is present even in overweight and obese HF patients, as is illustrated in a study that found malnutrition in 22% of overweight HF patients in their sample [38]. While overweight and obesity have been identified as risk factors for HF development, patients with existing HF and higher than normal BMIs have shown to have improved outcomes and rates of survival, a phenomenon appropriately termed the obesity paradox [24]. Such paradox has been explained by (1) neutralization of TNF-alpha and lipopolysaccharides through excess adipose tissue and by (2) the fact that obesity among HF patients is associated with younger age, increased albumin levels, and increased lean muscle mass, strength, and exercise tolerance [39]. Generally, overweight and obese HF patients tend to have better nutritional and functional status than their normal and underweight counterparts.

Micronutrient deficiencies in HF

Micronutrient deficiencies commonly observed in HF patients may be attributed to malnutrition and underlying pathophysiologic mechanisms of HF. Most of the literature describes decreased micronutrient intake in patients with HF compared with the recommended reference nutrient intake; however, the ability of patients with HF to meet the daily recommended intake for both macro- and micronutrients is uncertain. In addition, while malnourished patients with HF experience an increased risk of cardiac cachexia and mortality, it is not clear from experimental evidence whether underlying micronutrient deficiencies are in the causal pathway [20]. Reduced energy intake in HF patients has been associated with lower intake of micronutrients, including magnesium, iron, phosphorous, potassium, selenium, zinc, sodium, B vitamins, and vitamin E [10–12]. Depleted micronutrient status, in turn, has been associated with an increase in mortality in patients with HF, most notably with serum potassium less than 4 mmol/L [8]. Moreover, a larger number of micronutrient deficiencies have been shown to be associated with significantly shorter event-free survival, therefore underlining the importance of micronutrient status and high-quality dietary intake [40].

Despite such important evidence, dietary management of HF in clinical practice has historically emphasized the reduction of a single micronutrient (i.e., sodium) and fluid management, which oversimplifies the complexity between multiple dietary inadequacies, including micronutrient deficiencies, and HF pathophysiology in these patients. Such information is particularly important within the context of an association between sodium restriction and decreased intake of other micronutrients, including calcium, phosphate, thiamine, and folate, in addition to reduced intake of energy and carbohydrates [2]. Various micronutrients are essential co-factors in energy metabolism and influencing cardiac and metabolic remodeling [41]. While deficiencies in HF patients have been described for micronutrients that are involved in carbohydrate and fatty acid metabolism, calcium homeostasis, renin production, and antioxidant properties, their effects on energy utilization, cardiac contractility, endothelial function, and reactive oxidative stress have not been fully described in patients with HF [20, 26].

In addition, little is known about micronutrient supplementation as a viable option to address micronutrient deficiencies in HF patients due to largely inconclusive results of intervention studies examining the effect of micronutrient supplementation on HF progression, morbidity, and mortality [20, 26]. Finally, not one single component of nutritional status has been independently associated with changes in HF outcomes. Given that dietary intake and nutritional status are multifactorial concepts and research is generally limited in examining the independent effects of single foods and individual nutritional components on disease outcomes, assessment of dietary quality and patterns and nutritional status on HF outcomes is needed for improved understanding of the impact of micronutrient status on HF outcomes [20, 26, 41–43].

Impact of pharmacotherapy on nutritional status in HF

A cornerstone of HF management is pharmacotherapy. While essential to comprehensive HF treatment, medications and nutrition interact through complex pharmacodynamic and pharmacokinetic relationships, such as by altering physiologic absorption, metabolism, and excretion of medications and nutrients within existing HF pathophysiology. In this context, recommended levels of macro- and micronutrients may not reflect true nutritional needs while accounting for underlying interactions [44]. Heart failure pathophysiology, poor quantity

and quality of dietary intake, and concomitant pharmacotherapy have all been associated with malnutrition in HF patients [45]. By understanding the role of pharmacotherapy in the cause and exacerbation of malnutrition and its effects on underlying HF pathophysiology, appropriate modifications to both dietary and pharmacological management of these patients can be implemented. Loop diuretics, often used for symptomatic management of HF exacerbations, are notorious at increasing the urinary excretion of water-soluble micronutrients and thus contributing to micronutrient deficiencies, such as deficiencies of B vitamins and minerals, including potassium, calcium, magnesium, and selenium [24]. Angiotensin-converting enzyme inhibitors (ACEi), which are considered a cornerstone of chronic pharmacological management of HF due to their benefit on morbidity and mortality, have been shown to lead to zinc deficiency, taste distortions, and reduced appetite [20, 24, 26]. Digoxin, a medication often used in more severe HF progression, may lead to iatrogenic anorexia in patients with HF [26, 46]. Consequently, HF patients exhibit decreased food intake as a result of factors involved in the natural aging process and pathophysiological manifestations of their disease, which are potentiated by adverse medication side effects. Moreover, the combination of ACEi, digoxin, and diuretic medications has shown to increase subcutaneous fat and decrease muscle strength in patients with HF [24]. ACEi, angiotensin receptor blockers (ARBs), aldosterone antagonists, and thiazide diuretics alter the homeostasis of micronutrients, namely by increasing potassium levels by affecting the RAAS. Beta-blockers affect potassium levels by redistributing intracellular potassium into the serum as opposed to minimizing its excretion [24]. It is important to understand the individual effects of each medication class on nutritional status as deficiencies can easily be prevented, such as through supplementation of potassium to prevent hypokalemia. While studies have examined the supplementation of other micronutrients, including calcium, vitamin D, B vitamins (thiamine), vitamin C, vitamin E, coenzyme Q, selenium, zinc, copper, omega-fatty acids/fish oil, the data is largely inconsistent with no recommendations of routine supplementation unless deficiency is identified along with severe pathophysiology and/or drug-induced etiology [24]. Further investigation regarding the quantity and quality of dietary intake and within the context of pharmacological management of the underlying pathophysiology is vital to optimal medical management of patients with HF.

Impact of the Dietary Approaches to Stop Hypertension (DASH) diet on HF outcomes

The DASH diet emphasizes the intake of fruits and vegetables; lean protein such as poultry, fish, and nuts; fiber and whole grains; and low-fat dairy products in order to sustain adequate levels of micronutrients deemed to lower blood pressure, including potassium, calcium, and magnesium [47]. While the DASH diet has been proven to effectively reduce blood pressure, the benefit of the DASH diet in patients with HF has been inconsistently investigated [1, 19, 48–51]. However, due to the high percentage of HF patients with hypertension and the strong pathophysiological influence of long-standing hypertension in the development of HF [1, 52], beneficial effects of the DASH diet in HF patients are plausible. According to the World Health Organization, 75% of deaths from cardiovascular disease have been attributed to preventable risk factors, including dietary and lifestyle modifications. While the DASH diet and Mediterranean diets have shown to have a protective effect on the prevention of HF, there is limited literature suggesting a benefit on the slowing of disease progression, morbidity, and mortality in patients with existing HF [21, 53]. Diets consistent with DASH guidelines have been associated with lower incidence rates of HF in adults without baseline cardiac insufficiencies or disease, and to reduce blood pressure and LDL cholesterol levels [21, 47, 54]. The decrease in blood cholesterol levels can be partially attributed to a decrease in circulating fatty acids and glucose and an increase in dietary fiber content, which has been theorized to decrease the progression of cardiac hypertrophy and fibrosis, and overall decreased cardiac function as seen in HF [21, 55]. Based on the Framingham Heart Study, the decrease in systolic blood pressure is estimated to reduce the incidence of HF by about 12% [21]. Due to the known benefits of the DASH diet on cardiovascular disease, there have been preliminary studies assessing its effects on HF outcomes in patients with existing disease. In HF patients with preserved ejection fraction, DASH diet intake has shown to improve arterial elasticity, ventricular diastolic function, ventricular-arterial elastance coupling, and overall arterial stiffness [47, 54–57]. Clinically, a reduction in both blood pressure and 24-hour urinary sodium levels and an increase in 6-min walk distance and quality of life in patients with HF have been observed after consumption of a DASH diet [47, 54–58]. Mechanistically, the perceived benefit has been hypothesized to be associated with the prevention of the development and progression of endothelial dysfunction. Endothelial dysfunction, which is a surrogate for HF disease progression, is embodied by the interplay between impairments in the nitric oxide–mediated vasodilation, inflammatory cytokine cascade, and free radical production [47, 54]. Therefore, the positive effect on arterial elasticity and ventricular diastolic function from the DASH diet on patients with HF indicates a potential improvement in disease progression [47, 54]. Finally, a recent randomized controlled trial showed efficacy of home-delivered DASH meal consumption for 4 weeks post discharge from a HF hospitalization to reduce symptoms and functional status, and trended towards a reduction in 30-day hospital readmissions and days rehospitalized [58]. Thus, plausible explanations for the benefits of a DASH diet in HF are emerging to undergird further study.

Table 1 Summary of suggested questions to be addressed by future heart failure research

Topic	Research questions
Sodium	• Are sodium intake recommendations for the healthy population applicable to HF patients?
	• Should there be individualized recommendations for different ACC/AHA stages or NYHA functional classes?
	• Should there be individualized sodium restriction levels depending on sodium sensitivity?
	• Is the effect of sodium intake different for patients with HFrEF vs. HFpEF?
Malnutrition and micronutrient status	• What are the underlying mechanisms explaining the increased risk of developing cardiac cachexia among HF patients with inadequate micronutrient status?
	• What is the prognostic impact of malnutrition and/or micronutrient status on HF progression?
	• What is the effect of obesity on HF progression in HF patients with adequate vs. inadequate nutritional status?
Pharmacotherapy	• What is the effect of sodium restriction in the context of pharmacotherapeutic RAAS suppression and fluid restriction?
Dietary intake	• What is the effect of dietary patterns (e.g., DASH diet) on HF outcomes in the context of HF pathophysiology and medical HF management?

ACC, American College of Cardiology; *AHA*, American Heart Association; *DASH*, Dietary Approaches to Stop Hypertension; *HF*, heart failure; *HFpEF*, heart failure with preserved ejection fraction; *HFrEF*, heart failure with reduced ejection fraction; *NYHA*, New York Heart Association; *RAAS*, renin-angiotensin-aldosterone system

Where do we go from here?

In conclusion, there is a mounting appreciation for the effects of nutritional status on the pathophysiology, treatment, and outcomes for patients with HF. There are well-established recommendations for the DASH diet in patients with hypertension [19, 48–51]. With growing research on the effect of the DASH diet and nutritional status on outcomes of cardiovascular diseases, and specifically HF, more detailed evidence on the effect of diet within the context of HF pathophysiology and medical HF management is needed (Table 1). At the least, consideration should be given to dietary intake and nutritional status beyond dietary sodium consumption to improve outcomes in HF patients.

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Compliance with ethical standards

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