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Nutrition, Heart Failure and Quality of Life: Beyond Dietary Sodium

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Abstract

Despite significant advancements in the past decades in the understanding of and targeted treatments for heart failure (HF), it remains a deadly disease with a high risk of hospitalizations and reduced cardiorespiratory fitness, ultimately leading to reduced quality of life (QoL). Notably, patients admitted to the hospital for acute decompensated HF present with minimal therapeutic options. Lifestyle interventions, such as diet, have shown promising data in improving HF-related outcomes; however, very little is known about their role in decompensated patients admitted for acute HF.

Keywords

heart failure; diet; nutrition; sodium

Sodium Restriction in Heart Failure

Dietary sodium restriction is perhaps the most commonly implemented nonpharmacologic strategy for patients with HF, particularly in those admitted for acute decompensated HF. However, the evidence is both limited and controversial, such that current ACCF/AHA HF guidelines do not provide strong recommendations for such an approach. The theoretical principle behind sodium restriction is that excess dietary sodium promotes fluid retention and thereby exacerbates HF. To the contrary, sodium restriction in HF has been associated with neutral or even worse prognosis compared to less stringent goals.⁽¹⁾ The latter has been hypothesized to occur through multiple mechanisms: excess contraction of intravascular volume, renal hypoperfusion and reduced cardiac output. Such effects can ultimately

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promote the activation of the renin-angiotensin-aldosterone system, which is associated with unfavorable outcomes in HF.

In addition, adherence to sodium restriction remains challenging, even when meals are provided as part of a clinical trial. Sodium reduction is associated with 1) increased perceived thirst particularly when paired with fluid restriction, and 2) unintended reduction of dietary calories, macro- and micronutrients may be, at least in part, attributable to a perceived decreased palatability of food secondary to sodium restriction

The effects of sodium restriction on QoL and clinical outcomes remain to be elucidated. Fortunately, SODIUM-HF, an ongoing multicenter dietitian-led randomized controlled trial, is currently investigating whether a sodium restriction to <1,500 mg/day improves these parameters in ambulatory patients with HF (NCT02012179). Of note, similar trials in acute decompensated HF are lacking.

Nutritional Status in Heart Failure

Importantly, patients with HF often present with poor nutritional status that can profoundly affect the progression of the disease and its related prognosis, including excess adiposity (i.e., obesity), reduced lean mass strength, amount and functionality (i.e., sarcopenia), a combination of both (i.e., sarcopenic obesity) or unintentional significant weight loss resulting in both lean and fat mass tissues loss (i.e., cachexia). The nutritional causes and potential related targeted treatments of these abnormalities extend beyond sodium intake (Table 1).(2) Almost two decades ago, it was described that patients with HF present with a hypercatabolic state and an increased resting energy expenditure (REE): using indirect calorimeter, REE was increased by approximately 250 kcal/day compared to apparently healthy sedentary control, therefore promoting a chronic negative energy balance and protein-calorie malnutrition (3). Furthermore, those with poor caloric intake tend to have worse intake of essential micronutrients. (3) The initial thought was that HF leads to insufficient caloric intake by eliciting a hypercatabolic state, making the former more of a marker rather than a mediator of the disease; however, this may not necessarily be the case. In fact, the direct effects of insufficient caloric intake on QoL and clinical outcomes in HF are largely unknown.

Insufficient Caloric Intake in Acute HF

In this issue of the *JACC: Heart Failure*, Bilgen and colleagues provide crucial data furthering our understanding of this question in patients admitted for an event of HF exacerbation (about one-third of whom presented with HF with preserved ejection fraction). (4) They performed a post-hoc analysis of the GOURMET-HF trial (NCT02148679), which previously found that a home-delivered DASH-like meal program for four weeks started at time of discharge, was associated with favorable, yet exploratory, effects on clinical status and 30-day readmission rate. In the current analysis, the authors used the well-validated Block Food Frequency Questionnaire (FFQ) to retrospectively collect dietary information in 57 patients providing an average of the dietary intake for the year prior to the HF hospitalization event. The foods collected with the FFQ were then converted into calories,

macronutrients and micronutrients. Energy requirements were estimated using validated predictive equations; the authors defined individuals as having insufficient caloric intake when the actual caloric intake fell below 90% of the estimated total energy requirements. Finally, they investigated whether being classified as having inadequate caloric intake was associated with 12-week post-discharge changes in 1) clinical status measured with the clinical summary score of the Kansas City Cardiomyopathy Questionnaire (KCCQ) and 2) all-cause hospital readmissions.

Similar to prior reports,(3) more than 50% of patients admitted for HF reported a caloric intake less than 90% of estimated energy requirements. Surprisingly, individuals with insufficient caloric intake were more likely to be obese, although they also presented with a numerically greater number of comorbidities. Twelve weeks following discharge, patients with and without insufficient caloric intake experienced a significant improvement in clinical status measured with the KCCQ clinical summary score; however, improvements were significantly smaller in those not meeting their estimated energy requirements. Even after adjustments for key nutritional risk screeners, inadequate caloric intake was associated with increases in both total length of rehospitalizations in days and number of patients with rehospitalizations: 124 days versus 18 days and 52% versus 17%, respectively. Importantly, individuals with insufficient caloric intake were also more likely to have reduced sodium, protein, and micronutrient intake; however, sodium intake did not predict changes in QoL and clinical outcomes. This finding raises the possibility of worse outcomes observed in patients consuming lower sodium intake in prior studies being potentially mediated by insufficient caloric intake.(1) Thus, in clinical trials using dietary interventions (i.e., sodium restriction), investigators should aim for maintaining isocaloric participant diets (or at least preventing negative energy balance) throughout the trial to minimize the risk of confounders.

The results of this study, although limited by the small sample size and the exploratory nature of the analysis, complement a prior study published in the *JACC: Heart Failure* indicating that at time of admission in patients with acute HF, a greater adherence to a Mediterranean diet, a dietary pattern rich in unsaturated fatty acids and antioxidants, was associated with reduced risk of HF rehospitalization up to 1 year.(5) Furthermore, in chronic stable HF, dietary intervention aimed at increasing unsaturated fatty acids consumption or improving adherence to DASH, among others, have proven to be feasible and associated with favorable changes in exercise and functional capacity.(2) Of note, both the Mediterranean diet and DASH are primarily composed of plant-based foods, suggesting that a prudent plant-based dietary pattern might be beneficial in HF.

In conclusion, the authors are commended for providing novel evidence, which will guide future randomized controlled trials investigating the effects of different caloric intake strategies in patients admitted for acute decompensated HF on clinical outcomes and QoL. Finally, dedicated trials investigating the distinction between patients with HF with reduced ejection and HF with preserved ejection fraction separately and using indirect calorimetry for a more accurate estimate of energy requirements are encouraged to determine whether the effects discussed herein are efficacious in both forms of HF.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

ACCF	American College of Cardiology Foundation
AHA	American Heart Association
DASH	Dietary Approaches to Stop Hypertension
FFQ	food frequency questionnaire
HF	heart failure
QoL	quality of life

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Table 1.

Clinical Studies Investigating the Effects of Diet in Heart Failure

Study	Trial Type	EF (%)	N	Dietary Intervention		Outcomes
Sodium Restriction						
Online Reference (1)	Randomized controlled trial	40±15.6 (Intervention) 42.3±15.5 (Control)	65	2,000–2,400 mg/day sodium vs. control for 6 months		Increase in physical activity and QoL assessed by a sum of KCCQ and MLHFQ scores and improvement in NYHA class in intervention group vs. control.
Online Reference (2)	Randomized trial	EF <35%	232	2,760 mg/day vs. 1,840 mg/day sodium in recently decompensated patients (30 days post-discharge)		2,760 mg/day sodium group had lower readmissions at 180 days (primary endpoint) and combined readmission and mortality at 90 days.
Online Reference (3)	Randomized single-blind controlled trial	26.0±8.7	75	Inpatient, admitted for decompensated HF 800 mg/day sodium restriction vs. control until 7 th day of hospitalization or discharge		No differences in 3-day change in weight and clinical congestion score in intervention vs. control (primary endpoint) and 30-day readmissions in intervention vs. control.
Online Reference (4)	Multicenter, randomized controlled trial	34±11 (Intervention) 37±15 (Control)	97	2,000–3,000 mg/day sodium restriction vs. control for 12 weeks		The intervention group demonstrated signs of clinical improvement vs. control (composite primary outcome), driven mostly by improvement in NYHA class and edema.
Online Reference (5)	Open-label, randomized controlled pilot trial	42.0 (25.0–50.5)	38	1,500 mg/day (low) vs. 2,300 mg/day (moderate, control) sodium restriction for 6 months		BNP decreased from baseline in the low sodium group alone but there were no significant differences between groups; KCCQ clinical score increased in both groups with no difference between groups.
Dietary Patterns						
Online Reference (6)	Open-label pilot trial	69±6	13	21 days of home delivered DASH/sodium restricted diet (1,150 mg sodium/day)		DASH diet lowered clinic and 24-hour ambulatory blood pressure and BNP. Cardiac systolic and diastolic function, 6MWT and NYHA class were improved.
Online Reference (7)	Randomized controlled trial	41±13 (DASH) 40±15 (Control)	48	3 months of DASH diet intervention vs. control		Endothelial function was not better in the DASH group (primary endpoint) but quality of life assessed by MLHFQ and 6MWT was improved vs. control.
Online Reference (8)	Multicenter, randomized single-blind controlled trial	39±18	66	Home delivered DASH/sodium restricted diet (1,500 mg sodium/day) vs. control for 30 days post-hospital discharge for HF exacerbation		No significant difference between groups in change of KCCQ summary score (primary endpoint). There was a trend towards lower HF rehospitalization at 30 days in DASH vs. control.
Online Reference (9)	Multicenter, prospective, observational study	51±14	991	Adherence to a Mediterranean diet assessed by patient questionnaire in those seen in the emergency department for acute HF		After a mean follow up of 2.1±1.3 years, adherence to the Mediterranean diet was not associated with a decrease in all-cause mortality (primary endpoint) but was associated with a decrease in rehospitalizations for HF.
Online Reference (10)	Cross sectional analysis	39±20	372	Analysis of adherence to Mediterranean dietary pattern via food frequency questionnaire and MedDietScore		A Mediterranean dietary pattern was associated with improvements in filling pressure (via log E/A ratio).

Study	Trial Type	EF (%)	N	Dietary Intervention	Outcomes
Online Reference (11)	Multicenter, prospective cohort study	21.5 (15.3–28.9)	318	Analysis of diet via food frequency questionnaire in candidates waitlisted for a heart transplant	After median follow-up of approximately a year, High sodium diets (correlated with higher fluid and saturated fat intake) were associated with high urgency of transplantation; intake of poly- and monounsaturated fatty acid was associated with a decreased risk of deterioration/mortality.
Online Reference (12)	Prospective cohort study	33.9 ± 14.0	212	Analysis of average lycopene (a carotenoid found in red, orange and yellow produce) intake via four days of food diaries	Greater intake of lycopene was associated with improvements in cardiac event-free survival (related cardiac death and time to first hospitalization for HF), independent of intake of sodium.
Fatty Acids					
Online Reference (13)	Multicenter randomized, double-blind controlled trial	33.0±8.5 (N-3 PUFA) 33.2±8.5 (placebo)	6975	N3 PUFA 1g daily vs. placebo	After a median follow-up of 3.9 years, the N3 PUFA group demonstrated lower all-cause mortality, as well as the combined endpoint of lower all-cause mortality and hospital readmission (primary endpoint).
Online Reference (14)	Randomized, double-blind controlled trial	36±7 (N-3 PUFA) 37±6 (placebo)	133	N3 PUFA 1g daily vs. placebo for 12 months	Increase in EF in N3 PUFA vs. placebo (primary endpoint), as well as improvement in diastolic function, decrease in inflammatory biomarkers and lower rate of HF admission.
Online Reference (15)	Randomized double-blind controlled trial	28±1	31	Supplementation of L-alanyl-L-glutamine (8 g/day) and N3 PUFA (6.5 g/day) vs. placebo for 3 months	No change in peak VO ₂ , 6MWT, handgrip or leg/arm skeletal muscle function in intervention vs. placebo (primary endpoints).
Online Reference (16)	Cross sectional analysis	45 (30–60)	118	Analysis of averaged 3-day baseline dietary fatty acid consumption for patients enrolled in a sodium restriction trial	As a percent of total kcal, increased PUFA and saturated fatty acids were associated with decreased and increased all-cause mortality, respectively.
Online Reference (17)	Cross sectional analysis	60.4 (57.1–63.0)	23	Analysis of baseline 24-hour dietary recall in patients enrolled in a trial for anti-inflammatory therapy	Dietary UFA were positively associated with peak VO ₂ , greater fat-free mass and more favorable diastolic function.
Online Reference (18)	Single-arm pilot trial	58±4	9	In patients with comorbid obesity, 12 weeks of daily supplementation with 1 serving of food rich in UFA. Preferred recommendations for intake were extra-virgin olive oil (54 g), canola oil (54 g), lightly salted mixed tree nuts (walnuts, hazelnuts, almonds and pecans) or peanuts (28 g)	There was an increase in dietary UFA and plasma UFA (primary endpoints). An increase in exercise time and oxygen pulse, as well as a trend towards an increase in peak VO ₂ was observed.
Online Reference (19)	Prospective cohort study	32±14	42	Analysis of average fatty acid consumption via food diaries from four days	Saturated and trans fat intakes were associated with higher levels of TNF- α ; omega-3 and PUFA intake were associated with lower sTNF-R1 and sTNF-R2 levels. Both sTNF-R1 and TNF- α were associated with decreased event-free survival (emergency department visit or hospitalizations for HF, as well as cardiac mortality).
Caloric Restriction					
Online Reference (20)	Randomized controlled trial	26±7.3	14	12 weeks of a calorically restricted diet (500 to 800 kcal deficit), one group with high-protein (30% kcal),	Greater weight loss, reduction in waist circumference, fat mass and increase in 6MWT and peak VO ₂ in the high-protein group vs. the other two groups

Study	Trial Type	EF (%)	N	Dietary Intervention	Outcomes
Online Reference (21)	Randomized controlled trial	61±6	100	20 weeks of a 2x2 factorial study to assess the effects of exercise vs. diet 4 groups – exercise (supervised aerobic exercise 3x week), caloric restriction alone (400 kcal deficit), exercise and caloric restriction combined and control	The independent effects of diet from baseline included increased peak VO ₂ , but no effect on MLHF total score (primary endpoints). Exercise time, peak workload, 6MWT, reduction in weight, fat mass, inflammatory biomarkers and total KCCQ score also improved with diet.
Protein and Amino Acid Supplementation					
Online Reference (22)	Randomized double-blind controlled study	34±9 (Intervention) 32±6 (placebo)	95	30 days of mixed amino acid supplementation (4 g BID) vs. placebo	Peak VO ₂ , workload and oxygen pulse improved in the intervention group but not placebo.
Online Reference (23)	Randomized, double-blind controlled pilot trial	25±10 (Intervention) 24±5 (placebo)	29	In patients with clinically significant weight loss, 6 weeks of a high caloric (600 kcal) high protein (20 g) oral nutrition supplement vs. placebo (12 kcal/day)	From baseline, intervention increased weight gain and overall MLHFQ score (primary endpoints), as well as 6MWT. Peak VO ₂ was not increased.
Online Reference (24)	Randomized controlled trial	Not described	66	3 months of resistance training plus branch-chain amino acid supplement (10 g/day) vs. resistance training alone	There were no effects of branch chain amino acids on muscle strength or peak VO ₂
Online Reference (25)	Open-label, randomized controlled trial	32.4±6.3 (Intervention) 32.4±7.3 (Control)	50	500 mg orodispersible L-carnosine vs. control for 6 months	In intervention vs. control, there was an increase in 6MWT, peak VO ₂ , workload, and quality of life by visual analog score.
Oral Micronutrient Supplementation					
Online Reference (26)	Randomized, double-blind controlled trial	26.1±6.7	32	Daily multiple micronutrient supplement vs. placebo for 9 months	Increase in LVEF in the micronutrient group vs. placebo (primary endpoint), as well as increase in quality of life scores assessed by questionnaire. 6MWT, NYHA class and inflammatory biomarkers remained unchanged in both groups.
Online Reference (27)	Randomized, double-blind, controlled trial	38.3±11.4 (Intervention) 45.1±9.0 (Control)	74	Daily multiple micronutrient supplement vs. placebo for 12 months	No significant difference in EF between intervention and placebo (primary endpoint) or in MLHFQ questionnaire score, 6MWT, NTproBNP and inflammatory biomarkers.
Online Reference (28)	Randomized, double-blind control crossover trial	29.5±2.5	9	28 days of treatments with 300 mg/day thiamine vs. placebo	Significant improvement in EF in thiamine group vs. placebo (primary endpoint); trend towards improvement in 6MWT.
Online Reference (29)	Multicenter, randomized double-blind controlled trial	31±10	420	2 years of treatment with CoQ10 100 mg 3x daily vs. placebo	Reduction in major adverse cardiac events in intervention vs. placebo (primary endpoint), as well as a reduction in rates of hospital stays for HF and in all-cause and cardiovascular mortality.
Online Reference (30)	Randomized, double-blind randomized crossover trial	32±2	13	9 days of 70 ml beet root juice BID that was concentrated vs. depleted in nitrates	No significant difference in time to exercise intolerance between nitrate and placebo (primary endpoint) or peak VO ₂ .

Study	Trial Type	EF (%)	N	Dietary Intervention	Outcomes
Online Reference (31)	Randomized, double-blind, controlled trial	Included only 50%	20	1 week of nitrate concentrated vs. depleted beetroot juice	Increase in time to exhaustion during submaximal exercise at in intervention group (primary endpoint), but not after a single acute dose.
Online Reference (32)	Randomized, double-blind controlled crossover trial	34±2	8	Patients ingested 140 ml of a beetroot juice that was nitrate concentrated (intervention) vs. depleted (control) and performed exercise testing 2 hours after	There were no changes in ventilatory responses, but peak VO ₂ improved after intervention vs. placebo (primary endpoints).
Online Reference (33)	Randomized double-blind controlled trial	37.6±13.9	64	6 months of weekly 50,000 IU Vitamin D3 vs. placebo	There was no difference in changes in peak VO ₂ in the intervention group vs. control (primary endpoint) nor in 6MWT.
Online Reference (34)	Randomized double-blind controlled trial	26.1±10.68	229	100 ug daily Vitamin D3 vs. placebo for 1 year	Vitamin D3 did not increase 6MWD vs. placebo (primary outcome), but did increase EF and improve remodeling.
Online Reference (35)	Randomized, double-blind controlled trial	28 (23–34) (Intervention) 26 (24–35) (placebo)	892	4,000 IU Vitamin D3 daily vs. placebo for 3 years	Vitamin D3 did not reduce all-cause mortality vs. placebo (primary endpoints); there was an increased implantation of mechanical circulatory support in the Vitamin D3 group.
Online Reference (36)	Open-label randomized controlled trial	35±8	101	6 weeks of 2,000 IU vitamin D3 daily vs. control	Vitamin D3 decreased plasma renin activity (primary endpoint) and decreased plasma renin, but did not change NTproBNP.

6MWT, 6 Minute Walk Test; BID, twice a day; BNP, brain natriuretic peptide; DASH, Dietary Approaches to Stop Hypertension; EF, ejection fraction; HF, heart failure; KCCQ, Kansas City Cardiomyopathy Questionnaire; kcal, kilocalories; MedDiet Score, Mediterranean Diet Score; MLHFQ, Minnesota Living with Heart Failure Questionnaire; N, number of participant; N-3 PUFA, N-3 Polyunsaturated Fatty Acids; Na, sodium; NYHA, New York Heart Association; peak VO₂, peak oxygen consumption; QoL, quality of life; UFA, Unsaturated Fatty Acids