Current Cardiovascular Risk Reports (2018) 12: 17
https://doi.org/10.1007/s12170-018-0580-y

ELDERLY AND HEART DISEASE

Nutritional Assessment and Dietary Interventions in Older Patients with Heart Failure

Ran Lee[1] · Marty C. Tam[1] · Thomas M. Cascino[1] · Scott L. Hummel[1,2]

Published online: 1 May 2018

Abstract

Purpose of Review Older patients with heart failure (HF) are challenging to manage due to the high prevalence of frailty, polypharmacy, cognitive impairment, and other geriatric conditions. Malnutrition and poor dietary habits are also common and strongly predict adverse outcomes in older patients with HF. Current dietary recommendations for HF primarily advise reducing sodium intake, but randomized studies suggest that excess restriction can lead to adverse outcomes. The optimal dietary pattern, caloric intake, advice for weight management, and strategies for self-care in this population have not been determined.

Recent Findings Screening tools for malnutrition, including the Geriatric Nutritional Risk Index and Mini Nutritional Assessment, can be used to assess nutritional risk and predict mortality, hospitalization, and functional decline in older patients with HF. The ideal dietary pattern for patients with HF has not been determined, though epidemiologic and observational cohort studies suggest that the Dietary Approaches to Stop Hypertension (DASH) and Mediterranean dietary patterns are reasonable to recommend to many patients. Recent dietary intervention studies highlight the importance of individualized nutritional counseling interventions, maintaining adequate nutritional intake during sodium restriction, and the potential for caloric restriction to improve outcomes in some patients.

Summary Nutritional status and dietary intake are prognostically important and potentially modifiable in older patients with HF. Ongoing interventional and translational studies may help define the ideal sodium intake, dietary regimen, and recommendations for weight loss, but more research is needed in this important domain.

Keywords Geriatric · Diet · Malnutrition · Sodium · Frailty

Introduction

By the year 2030, more than 40% of the US population will have some form of cardiovascular disease, with patients over the age of 65 representing a disproportionate percentage of this total [1]. When compared to the younger population, older adults have higher incidence and prevalence of a wide variety of cardiovascular disease states, including coronary artery disease, stroke, and heart failure (HF) [1]. Due to the high frequency of concurrent frailty, cognitive impairment, polypharmacy, and other geriatric conditions, HF represents an archetype for the emerging field of geriatric cardiology. In patients with HF, dietary sodium "indiscretion" is believed to be a common cause of decompensation and hospitalization. Malnutrition is highly prevalent, often concurrently present with frailty, and is associated with adverse events in patients with HF. Despite the potential importance of these issues, dietary patterns and nutritional status are understudied aspects of HF in older adults.

This article is part of the Topical Collection on *Elderly and Heart Disease*

✉ Scott L. Hummel
scothumm@med.umich.edu

[1] Frankel Cardiovascular Center, Michigan Medicine, University of Michigan, 1500 E. Medical Center Drive, 2383 CVC/SPC 5853, Ann Arbor, MI 48109, USA

[2] Ann Arbor Veterans Affairs Health System, Ann Arbor, Michigan, USA

Summary of Current Recommendations and Gaps in Evidence

Dietary recommendations for HF have historically focused on sodium intake [2]. Much of this emphasis stems from the ability of sodium restriction to lower blood pressure [3] and potentially reduce the risk of incident cardiovascular disease

in persons without HF [4, 5]. Nonetheless, dietary sodium restriction remains the most commonly recommended self-care intervention for patients with HF [4]. This advice is largely based on the clinical observation that increased sodium intake can lead to volume retention and HF decompensation [6, 7]. However, sodium restriction can also cause neurohormonal activation and reduced renal perfusion, potentially worsening the underlying HF syndrome [4]. Few studies have adequately characterized the relationship between dietary sodium intake and HF outcomes, and several randomized trials raise troubling concerns [8••, 9, 10••]. Despite these limitations, sodium restriction is recommended by current US guidelines, with advice for persons at risk of developing HF to limit sodium to 1500 mg/day, and in those with prevalent HF, to limit sodium without a definitive recommendation on the amount [2].

Sodium consumption does not occur in isolation, but as part of an overall dietary pattern. Sodium and calorie intake correlate closely in the USA [11], raising the possibility that advice to restrict sodium intake could lead to insufficient nutritional intake overall. Indeed, in one small study in middle-aged patients with HF, 1 week of dietitian-guided adherence to a 2000-mg/day sodium allowance was associated with reduced intake of calories, carbohydrates, fats, calcium, thiamine, and folate [12]. Whether these dietary changes would be harmful over a longer time period is unknown, but it is important to note that malnutrition is a major risk factor for hospitalization, institutionalization, and death in older adults, including in those with HF [13•, 14]. Moreover, diuretics, commonly used in all types of HF, are highly likely to worsen existing nutritional deficits through urinary losses of electrolytes, antioxidants, and vitamins [15, 16]. Current US guidelines for HF offer no guidance on the intake of other nutrients beyond sodium [2].

Further complicating dietary recommendations for HF patients is the often debated "obesity paradox." Obesity increases the risk of incident cardiovascular disease, including HF [17]. Despite this observation, patients with prevalent HF who are obese have a more favorable long-term prognosis [18–24]. Several potential explanations have been proposed, including potentially protective neurohormonal changes and a more robust ability to tolerate physiological stressors in patients who are obese [25]. Others cite the potential for unmeasured confounders to explain this relationship through selection bias [26]. Due to this controversy, current guidelines do not make a recommendation regarding weight loss in patients with prevalent HF [2].

Nutritional Assessment in HF

The American Society for Parenteral and Enteral Nutrition recommends screening populations at risk for malnutrition, and subsequently defining further assessment for patients that screen positive to develop strategies for improve nutritional status [27]. Malnutrition is common in older adults, with rates ranging 23–60% in the acute care setting, 5–30% in the ambulatory community, and 16–70% in assisted care settings [28]. The prevalence of malnutrition within the HF population has been variably estimated at 16–90%; the risk increases with the severity and acuity of the HF syndrome [13•].

Several nutritional screening and assessment tools have been studied in the general older adult population and successfully applied in patients with HF [13•]. One simple and commonly used tool in hospitalized older patients is the Geriatric Nutritional Risk Index (GNRI), which combines the ratio of measured weight to ideal body weight with serum albumin levels as follows: GNRI = [(1.489 × serum albumin (g/L)) + (41.7 × (current body weight/ideal weight))] [29]. In hospitalized older adults, the GNRI predicts hospital length of stay [30••] and nutrition-related complications [31]. In patients presenting with acute HF, GNRI scores upon admission independently predict mortality (HR 0.92 per 1-point increase, 95% CI 0.88–0.95, $p < 0.001$) [32••]. In stable outpatients with heart failure with reduced ejection fraction (HFrEF) aged ≥ 65 years on optimal medical therapy, the GNRI predicted survival independent of standard clinical factors and improved model risk discrimination. A GNRI > 98, reflecting adequate nutrition, appeared highly protective (HR 0.39, 95% CI 0.27–0.58, $p < 0.001$) [33]. In addition, the GNRI also predicts functional dependency and death in patients with HF and preserved ejection fraction [34].

The Mini Nutritional Assessment (MNA) was designed to assess nutritional risk in older adults (http://www.mna-elderly.com/forms/MNA_english.pdf), and its more nuanced assessment may represent the best balance between practicality and information. The MNA questionnaire incorporates anthropometric measures and patient-reported assessment of food types consumed and overall intake in hospitalized patients with HF, undernutrition according to the MNA approximately quadruples long-term mortality [13•]. The MNA also predicts mortality in patients undergoing consideration for ventricular assist devices or transplantation [35] and has also been linked to quality of life and mortality in the ambulatory HF population [36].

It is important to remember that malnutrition in patients with HF may not always be accompanied by obvious physical signs, such as weight loss or overt cachexia. Among the obesity epidemic in older adults, nearly one-third of Medicare patients hospitalized for HF now have a body mass index ≥ 30 kg/m^2 [37]. Often obscured in this setting is sarcopenic obesity, the concomitant presence of excess fat mass and decreased muscle mass. Sarcopenic obesity is most commonly defined by dual-energy x-ray absorptiometry as > 60% of body fat percentage of body fat and decreased muscle mass defined by appendicular skeletal muscle index [38]. Sequelae

include pro-inflammatory states, increased oxidative stress, decreased testosterone and growth hormone, all of which can contribute to decrement in skeletal muscle mitochondrial function and phenotypic exercise intolerance in patients with HF [38].

Dietary Interventions in HF—Practical Considerations

Patients with HF receive recommendations regarding diet, exercise, medication use, and self-care as a part of standard treatment. Several studies in patients with HFpEF [39] and HFrEF [40] noted a signal for lower risk of readmission with specific dietary discharge recommendations in the former and improved self-care behaviors and lower readmission rates with comprehensive 1-h nurse educator time on issues including diet in the latter. However, several factors can challenge adherence to these recommendations, and all must be considered when attempting a dietary intervention. In addition to poor dentition and the loss of smell and taste sensation that can occur with aging, food intake in older patients with HF is often compromised by decreased hunger sensation, early satiety, fatigue, shortness of breath, and nausea [41]. Complex dietary restrictions related to comorbid illnesses, such as diabetes mellitus, chronic kidney disease, or medications such as warfarin, affect the feasibility of following recommendations.

Depression can prevent healthy behaviors and carries prognostic importance in patients with HF. Song et al. demonstrated an association between depressive symptoms and dietary micronutrient deficiency in outpatients with HF, with both additively decreasing cardiac event-free survival [42]. Social isolation, cognitive impairment, and mobility limitations may make following dietary recommendations logistically difficult in older adults. Low health literacy and lack of disease-specific knowledge remain barriers for many patients. Both contribute to increased death and rehospitalization in patients with HF [43–46]. Socioeconomic status also plays an important role in dietary choices, as healthy food options can potentially be costly or less accessible [47].

Which Dietary Strategy Should Be Recommended for Patients with HF?

Several established dietary regimens can mitigate cardiovascular risk factors, such as hypertension, dyslipidemia, and diabetes, as well as potentially prevent progression to incident HF. Systemic hypertension is one of the strongest risk factors for developing HF. The Dietary Approaches to Stop Hypertension (DASH) eating pattern, higher in fruits, vegetables, legumes, and fiber and lower in added sugars and saturated fat than the standard Western diet, has its strongest blood pressure-lowering effect in older adults [48]. In large observational cohorts of postmenopausal women and middle-age-to-older men, those who are most adherent to the DASH eating pattern had less incident HF after adjustment for other risk factors [49, 50]. In women with existing HF, the DASH diet and the Mediterranean eating pattern have both been associated with lower long-term mortality [51]. In a large Spanish study that obtained dietary questionnaires at the time of HF hospitalization, patients who were following the Mediterranean diet had fewer rehospitalizations over the subsequent year [52••]. No long-term dietary intervention studies have yet been completed in older patients with HF. At this juncture, either the DASH or Mediterranean pattern is reasonable to recommend to most patients, although in the case of DASH, care must be taken regarding potassium intake in patients with chronic kidney disease [53].

On the other hand, Mozaffarian et al. sought to determine the relative contribution of lifestyle factors on HF development in more than 5000 adults ≥ 65 years of age enrolled in the Cardiovascular Health Study cohort [54]. When four separate dietary patterns, including DASH, were evaluated along with physical activity, alcohol, smoking, and obesity, no dietary pattern was found to be associated with prevention of HF ($p > 0.05$) [54]. However, patients with the highest quintile of sodium intake had a higher risk of incident HF [54]. Other epidemiologic studies, such as the first National Health and Nutrition Examination Survey (NHANES) study, also suggest that chronically high sodium intake is associated with HF development in overweight and obese individuals [55]. Some observational studies have shown greater risk for episodes of acute decompensation in patients with HF who consume higher amounts of sodium, including one by Arcand et al. demonstrating a daily intake of 3.8 ± 0.8 g of sodium leading to an adjusted HR of 2.55 (95% CI: 1.61, 4.04; $p < 0.001$) [7].

The inconsistencies across epidemiologic studies may relate to challenges in the accurate assessment of dietary intake. Cobb et al. assessed 26 cohort studies relating sodium intake to cardiovascular diseases and found an average of 3–4 methodological issues per study that could have resulted in inverse associations and null associations between sodium and outcomes [56]. A suggestion to reduce systematic error in sodium assessment was to standardize several 24-h urine collections, though the authors recognized limitations in level of burden on study subjects [56]. The intake of other nutrients can be even more difficult to assess, as most do not have a readily available physiological measurement that approximates consumption.

Randomized Controlled Dietary Interventions in Patients with HF

Paterna et al. have conducted several trials investigating the impact of dietary sodium restriction in patients with HF. In the

largest study, individuals with HFrEF with New York Heart Association (NYHA) class II symptoms at hospital discharge were randomized to a 1.8 g/day sodium diet vs. a 2.8 g/day sodium diet and followed out to 6 months, with both groups maintaining the same daily fluid intake [9]. The rate of hospital readmissions was reduced in the normal-sodium (2.8 g/day) group compared to the low-sodium group (1.8 g/day) (7.63 vs. 26.32%, respectively; $p < 0.05$) as was the composite endpoint of mortality plus readmissions (12.71 vs. 39.47%, $p < 0.001$) [9]. Serum creatinine, blood urea nitrogen, renin, aldosterone, and natriuretic peptides all increased in the low-sodium diet arm, suggesting plausible mechanisms for the observed increase in adverse events [9]. However, this study is challenging to interpret: both groups maintained high doses of loop diuretics regardless of clinical status, and many patients were not treated with contemporary regimens of neurohormonal blocking agents.

Doukky et al. performed a secondary analysis of the Heart Failure Adherence and Retention Trial (HART), a randomized behavioral trial of self-management counseling vs. education alone. Outcomes analyzed included death, cardiac death, and HF hospitalization in symptomatic patients (NYHA II-III) with either HFrEF or HFpEF. Sodium intake was estimated via a modified Food Frequency Questionnaire. Patients were dichotomized at an estimated sodium intake of 2.5 g/day, and cohorts were matched via propensity score. Patients from the sodium-restricted group demonstrated a higher risk of death or HF hospitalization (42.3 vs. 26.2%, HR 1.83, 95% CI 1.21–2.94, $p = 0.004$) driven mainly by HF hospitalization (32.3 vs. 20.0%, HR 1.82, CI 1.11–2.96, $p = 0.015$) [10••]. These data suggest the potential for negative effects of sodium restriction in HF. However, several potential confounders exist: the sodium questionnaire used had not previously been validated against other instruments, and the study results were not adjusted for caloric intake or overall nutritional status.

Recognizing the limitations in these prior trials, Colin-Ramirez et al. recently completed the Study of Dietary Intervention Under 100 mmol in Heart Failure (SODIUM-HF). This pilot trial randomized 38 patients with HF to dietary counseling to promote a low-sodium (1.5 g/day) vs. a moderate-sodium (2.3 g/day) intake while adjusting HF medical therapy based on clinical status and treatment guidelines [57••]. Over a follow-up of 6 months, natriuretic peptide levels fell in the low-sodium group (216 to 71 pg/ml, $p = 0.006$) and Kansas City Cardiomyopathy Questionnaire (KCCQ) score, a measure of disease-specific quality of life, rose in both groups (63 to 75, $p = 0.006$ in low; 66 to 73, $p = 0.07$ in moderate) [57••]. Questionnaire assessments suggest that the dietary counseling used in this study was able to maintain overall dietary quality while successfully reducing sodium intake [58].

Instead of focusing on sodium, the recent PICNIC (Nutritional Intervention Program in Hospitalised Patients with Heart Failure who are Malnourished) study aimed to improve overall dietary quality [59••]. Patients hospitalized with HF were stratified by nutritional status using the MNA and randomized to usual care vs. a 6-month intervention conducted by a dietitian team lead by a physician who specialized in nutrition. The intervention began during the index hospital stay and continued through monthly post-discharge outpatient visits and included diet optimization, tailored recommendations, and supplement prescriptions relevant to comorbid states [59••, 60]. The intervention also addressed symptoms affecting food intake including nausea, dyspepsia, anorexia, and even transit disorders [60]. Over the year post-discharge, patients in the intervention arm had a lower rate of all-cause death compared to the control arm (20.3 vs 47.5%, respectively; HR 0.37, 95% CI, 0.19–0.72, $p = 0.003$) and a lower rate of HF readmission (10.2 vs. 36.1%, respectively, $p = 0.001$) [59••]. While resource-intensive, this intervention highlights the potential for individualized nutritional counseling in HF.

Perspective on Future Research Goals for Diet and Nutrition in HF

Current dietary guidelines in patients with HF are not strongly evidence-based, which relates to the relative paucity of randomized trials as well as conflicting results from epidemiological studies. This is perhaps best illustrated by current HF guidelines advising sodium intake of 2–3 g/day [2, 61], a higher level than the 1.5 g/day the American Heart Association recommends for the general population [62]. Indeed, given the historical focus on this topic and concerns raised by prior randomized trials, the appropriate amount of sodium intake for patients with HF is one of the most pressing issues to address. A large multicenter trial based on the SODIUM-HF pilot referenced above is currently in recruitment; the trial will evaluate the effects of a 1.5- vs. 2.3-g/day sodium restriction on all-cause mortality, cardiovascular hospitalization, and cardiovascular emergency department visits (NCT02012179).

Additional information is needed about the advisability of weight loss in obese patients with HF. In the Swedish National Patient Registry, obese individuals who underwent bariatric surgery had a reduced risk for incident HF compared with nonsurgical obese patients (HR 0.37, 95% CI 0.30-0.46) [63]. A US study found qualitatively similar results in obese patients with HF who underwent bariatric surgery, who then had fewer HF hospitalizations beginning 13 months post-operatively [64]. Bariatric surgery patients are highly selected, and unmeasured confounders may mediate some of this effect.

Few trials have studied the effects of diet-induced weight loss in HF. Kitzman et al. conducted the Effect of Caloric Restriction or Aerobic Exercise Training on Peak Oxygen Consumption and Quality of Life in Obese Older Patients with HFpEF trial. In this study, 100 patients with chronic stable

HFpEF and a mean body mass index of 39.3 were enrolled to 20 weeks of exercise, diet, a combination of both, or standard control [65••]. Caloric restriction (1.3 ml/kg body mass/min, 95% CI 0.8–1.8, $p < 0.001$) or aerobic exercise (1.2 ml/kg body mass/min, 95% CI 0.7–1.7, $p < 0.001$) both increased peak oxygen consumption, and the combination was additive [65••]. It is not yet clear whether the benefits of weight loss would extend to all obese patients with HF, particularly those with sarcopenic obesity.

It seems likely that the nutritional status of a patient would modulate the benefits of exercise and weight loss, but this has not yet been studied. Older patients are at high risk for malnutrition and concomitant frailty [66], which often worsens during and immediately after hospitalization [67, 68]. The transition of acute inpatient HF care to outpatient HF management may provide the strongest opportunity for dietary interventions. Hummel et al. recently completed a pilot study in which patients with HF were randomized to preprepared home-delivered DASH-compliant meals or usual dietary advice for 4 weeks after hospital discharge [69•]. Patients receiving meals tended to have fewer HF symptoms and functional limitations, and all-cause and HF hospitalizations trended lower in comparison to the usual care group. A larger multicenter trial to extend and confirm these findings is in the planning stages.

Areas of translational study involve the relationships between diet and metabolism in patients with HF. As one example, the gut microbiota is responsible for the metabolism of dietary lecithin, choline, and carnitine to trimethylamine, which subsequently will be metabolized by the liver to trimethylamine N-oxide (TMAO). Elevated serum levels of TMAO provide prognostic value for cardiovascular disease in general [70–74] and for HF specifically [72, 75, 76]. The physiologic changes of chronic HF, such as gut wall edema, are linked to bacterial translocation [77, 78]. This can lead to bacterial overgrowth and pathogenic shift [79, 80]; in turn, the composition of the gut microbiome affects the degree of TMAO production [81].

Conclusions

Current guideline-based dietary recommendations for older patients with HF are sparse and not based on strong evidence. The historical advice to restrict sodium can produce unintended consequences of neurohormonal activation and worsening malnutrition if careful attention is not paid to medical management and overall dietary quality. Older patients with HF often have substantial barriers to healthy eating patterns that can relate to symptoms, geriatric domains, comorbid illnesses, and/or socioeconomic characteristics. These factors must be addressed to realize the benefits of individualized nutritional counseling. The ideal diet for patients with HF is not fully defined—ongoing and planned interventional studies and translational research initiatives should provide important information in the near future.

Compliance with Ethical Standards

Conflict of Interest Dr. Hummel has received funding from PurFoods, LLC to conduct dietary intervention studies in patients with HF. None of the other authors have relevant disclosures.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subject performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. Circulation. 2011;123:933–44.
2. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. Circulation. 2013;128:e240–327.
3. He FJ, Li J, Macgregor GA. Effect of longer term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomised trials. BMJ. 2013;346:f1325.
4. Gupta D, Georgiopoulou VV, Kalogeropoulos AP, et al. Dietary sodium intake in heart failure. Circulation. 2012;126:479–85.
5. Strazzullo P, D'Elia L, Kandala N-B, Cappuccio FP. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. BMJ. 2009;339:b4567.
6. Michalsen A, König G, Thimme W. Preventable causative factors leading to hospital admission with decompensated heart failure. Heart Br Card Soc. 1998;80:437–41.
7. Arcand J, Ivanov J, Sasson A, et al. A high-sodium diet is associated with acute decompensated heart failure in ambulatory heart failure patients: a prospective follow-up study. Am J Clin Nutr. 2011;93:332–7.
8. •• Paterna S, Fasullo S, Parrinello G, et al. Short-term effects of hypertonic saline solution in acute heart failure and long-term effects of a moderate sodium restriction in patients with compensated heart failure with New York Heart Association class III (Class C) (SMAC-HF Study). Am J Med Sci. 2011;342:27–37. **This large trial found that patients with heart failure randomized to low-sodium intake at hospital discharge had higher risk of death and readmission. The study is challenging to interpret because diuretic doses were not adjusted based on post-discharge clinical status.**
9. Paterna S, Gaspare P, Fasullo S, Sarullo FM, Di Pasquale P. Normal-sodium diet compared with low-sodium diet in compensated congestive heart failure: is sodium an old enemy or a new friend? Clin Sci (Lond). 2008;114:221–30.

10.•• Doukky R, Avery E, Mangla A, et al. Impact of Dietary Sodium Restriction on Heart Failure Outcomes. JACC Heart Fail. 2016;4: 24–35. **This was a secondary analysis of the Heart Failure Adherence and Retention Trial (HART), which tested self-management counseling vs. education alone in sodium restriction and dietary self-care. Patients from the sodium-restricted group had a higher risk of death or readmission**.

11. Strom BL, Anderson CAM, Ix JH. Sodium reduction in populations: insights from the Institute of Medicine committee. JAMA. 2013;310:31–2.

12. Jefferson K, Ahmed M, Choleva M, et al. Effect of a Sodium-Restricted Diet on Intake of Other Nutrients in Heart Failure: Implications for Research and Clinical Practice. J Card Fail. 2015;21:959–62.

13.• Lin H, Zhang H, Lin Z, Li X, Kong X, Sun G. Review of nutritional screening and assessment tools and clinical outcomes in heart failure. Heart Fail Rev. 2016;21:549–65. **This systematic review of nutritional tools and their use in predicting HF outcomes showed that prevalence of malnutrition within the HF population varies but worsens with severity and acuity of HF**

14. Song EK, Kang S-M. Micronutrient Deficiency Independently Predicts Adverse Health Outcomes in Patients With Heart Failure. J Cardiovasc Nurs. 2017;32:47–53.

15. Keith ME, Walsh NA, Darling PB, et al. B-vitamin deficiency in hospitalized patients with heart failure. J Am Diet Assoc. 2009;109: 1406–10.

16. Nicholls MG. Interaction of diuretics and electrolytes in congestive heart failure. Am J Cardiol. 1990;65:17E–21E. discussion 22E–23E

17. Kenchaiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. N Engl J Med. 2002;347:305–13.

18. Horwich TB, Fonarow GC, Hamilton MA, MacLellan WR, Woo MA, Tillisch JH. The relationship between obesity and mortality in patients with heart failure. J Am Coll Cardiol. 2001;38:789–95.

19. Lavie CJ, Osman AF, Milani RV, Mehra MR. Body composition and prognosis in chronic systolic heart failure: the obesity paradox. Am J Cardiol. 2003;91:891–4.

20. Bozkurt B, Deswal A. Obesity as a prognostic factor in chronic symptomatic heart failure. Am Heart J. 2005;150:1233–9.

21. Fonarow GC, Srikanthan P, Costanzo MR, Cintron GB, Lopatin M. ADHERE Scientific Advisory Committee and Investigators. An obesity paradox in acute heart failure: analysis of body mass index and inhospital mortality for 108,927 patients in the Acute Decompensated Heart Failure National Registry. Am Heart J. 2007;153:74–81.

22. Oreopoulos A, Padwal R, Kalantar-Zadeh K, Fonarow GC, Norris CM, McAlister FA. Body mass index and mortality in heart failure: a meta-analysis. Am Heart J. 2008;156:13–22.

23. Clark AL, Chyu J, Horwich TB. The obesity paradox in men versus women with systolic heart failure. Am J Cardiol. 2012;110:77–82.

24. Shah R, Gayat E, Januzzi JL, et al. Body mass index and mortality in acutely decompensated heart failure across the world: a global obesity paradox. J Am Coll Cardiol. 2014;63:778–85.

25. Wang TJ. The obesity paradox in heart failure: weighing the evidence. J Am Coll Cardiol. 2014;64:2750–2.

26. Banack HR, Kaufman JS. Does selection bias explain the obesity paradox among individuals with cardiovascular disease? Ann Epidemiol. 2015;25:342–9.

27. Mueller C, Compher C, Ellen DM. American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Board of Directors. A.S.P.E.N. clinical guidelines: Nutrition screening, assessment, and intervention in adults. JPEN J Parenter Enteral Nutr. 2011;35:16–24.

28. Agarwal E, Miller M, Yaxley A, Isenring E. Malnutrition in the elderly: a narrative review. Maturitas. 2013;76:296–302.

29. Bouillanne O, Morineau G, Dupont C, et al. Geriatric Nutritional Risk Index: a new index for evaluating at-risk elderly medical patients. Am J Clin Nutr. 2005;82:777–83.

30.•• Cereda E, Klersy C, Pedrolli C, et al. The Geriatric Nutritional Risk Index predicts hospital length of stay and in-hospital weight loss in elderly patients. Clin. Nutr. Edinb. Scotl. 2015;34:74–8. **This is a prospective multicenter cohort study assessing the use of the GNRI to predict hospital length of stay and nutrition-related complications in older hospitalized patients.**

31. Abd-El-Gawad WM, Abou-Hashem RM, El Maraghy MO, Amin GE. The validity of Geriatric Nutrition Risk Index: simple tool for prediction of nutritional-related complication of hospitalized elderly patients. Comparison with Mini Nutritional Assessment. Clin Nutr Edinb Scotl. 2014;33:1108–16.

32.•• Honda Y, Nagai T, Iwakami N, et al. Usefulness of Geriatric Nutritional Risk Index for Assessing Nutritional Status and Its Prognostic Impact in Patients Aged ≥65 Years With Acute Heart Failure. Am J Cardiol. 2016;118:550–5. **This is a prospective cohort study evaluating the use of the GNRI on admission in older patients hospitalized with acute HF. These scores were found to independently predict mortality.**

33. Sargento L, Vicente Simões A, Rodrigues J, Longo S, Lousada N, Palma Dos Reis R. Geriatric nutritional risk index as a nutritional and survival risk assessment tool in stable outpatients with systolic heart failure. Nutr Metab Cardiovasc Dis NMCD. 2017;27:430–7.

34. Kinugasa Y, Kato M, Sugihara S, et al. Geriatric nutritional risk index predicts functional dependency and mortality in patients with heart failure with preserved ejection fraction. Circ J Off J Jpn Circ Soc. 2013;77:705–11.

35. Aggarwal A, Kumar A, Gregory MP, et al. Nutrition assessment in advanced heart failure patients evaluated for ventricular assist devices or cardiac transplantation. Nutr Clin Pract Off Publ Am Soc Parenter Enter Nutr. 2013;28:112–9.

36. Sargento L, Satendra M, Almeida I, et al. Nutritional status of geriatric outpatients with systolic heart failure and its prognostic value regarding death or hospitalization, biomarkers and quality of life. J Nutr Health Aging. 2013;17:300–4.

37. Powell-Wiley TM, Ngwa J, Kebede S, et al. Impact of Body Mass Index on Heart Failure by Race/Ethnicity From the Get With The Guidelines-Heart Failure (GWTG-HF) Registry. JACC Heart Fail. 2018;6:233–42.

38. Upadhya B, Haykowsky MJ, Eggebeen J, Kitzman DW. Sarcopenic obesity and the pathogenesis of exercise intolerance in heart failure with preserved ejection fraction. Curr Heart Fail Rep. 2015;12:205–14.

39. Hummel SL, DeFranco AC, Skorcz S, Montoye CK, Koelling TM. Recommendation of low-salt diet and short-term outcomes in heart failure with preserved systolic function. Am J Med. 2009;122:1029–36.

40. Koelling TM, Johnson ML, Cody RJ, Aaronson KD. Discharge education improves clinical outcomes in patients with chronic heart failure. Circulation. 2005;111:179–85.

41. Lennie TA, Moser DK, Heo S, Chung ML, Zambroski CH. Factors influencing food intake in patients with heart failure: a comparison with healthy elders. J. Cardiovasc. Nurs. 2006;21:123–9.

42. Song EK, Moser DK, Kang S-M, Lennie TA. Association of Depressive Symptoms and Micronutrient Deficiency With Cardiac Event-Free Survival in Patients With Heart Failure. J Card Fail. 2015;21:945–51.

43. Kommuri NVA, Johnson ML, Koelling TM. Relationship between improvements in heart failure patient disease specific knowledge and clinical events as part of a randomized controlled trial. Patient Educ Couns. 2012;86:233–8.

44. Chen AMH, Yehle KS, Albert NM, et al. Health Literacy Influences Heart Failure Knowledge Attainment but Not Self-Efficacy for Self-Care or Adherence to Self-Care over Time. Nurs Res Pract. 2013;2013:353290.

45. Wu J-R, Holmes GM, DeWalt DA, et al. Low literacy is associated with increased risk of hospitalization and death among individuals with heart failure. J Gen Intern Med. 2013;28:1174–80.

46. Evangelista LS, Rasmusson KD, Laramee AS, et al. Health literacy and the patient with heart failure–implications for patient care and research: a consensus statement of the Heart Failure Society of America. J Card Fail. 2010;16:9–16.
47. Dubowitz T, Ghosh-Dastidar M, Eibner C, et al. The Women's Health Initiative: The food environment, neighborhood socioeconomic status, BMI, and blood pressure. Obes Silver Spring Md. 2012;20:862–71.
48. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med. 2001;344:3–10.
49. Levitan EB, Wolk A, Mittleman MA. Consistency with the DASH diet and incidence of heart failure. Arch Intern Med. 2009;169:851–7.
50. Levitan EB, Wolk A, Mittleman MA. Relation of consistency with the dietary approaches to stop hypertension diet and incidence of heart failure in men aged 45 to 79 years. Am J Cardiol. 2009;104:1416–20.
51. Levitan EB, Lewis CE, Tinker LF, et al. Mediterranean and DASH diet scores and mortality in women with heart failure: The Women's Health Initiative. Circ Heart Fail. 2013;6:1116–23.
52.•• Miró Ò, Estruch R, Martín-Sánchez FJ, et al. Adherence to Mediterranean Diet and All-Cause Mortality After an Episode of Acute Heart Failure: Results of the MEDIT-AHF Study. JACC Heart Fail. 2018;6:52–62. **This large Spanish prospective study followed nearly a thousand patients after an episode of decompensated heart failure. Those adherent to the Mediterranean Diet had lower rates of rehospitalizations over the next year.**
53. Kidney Disease Outcomes Quality Initiative (K/DOQI). K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. Am J Kidney Dis Off J Natl Kidney Found. 2004;43:S1–290.
54. Del Gobbo LC, Kalantarian S, Imamura F, et al. Contribution of Major Lifestyle Risk Factors for Incident Heart Failure in Older Adults: The Cardiovascular Health Study. JACC Heart Fail. 2015;3:520–8.
55. He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, Whelton PK. Dietary sodium intake and incidence of congestive heart failure in overweight US men and women: first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Arch Intern Med. 2002;162:1619–24.
56. Cobb LK, Anderson CAM, Elliott P, et al. Methodological issues in cohort studies that relate sodium intake to cardiovascular disease outcomes: a science advisory from the American Heart Association. Circulation. 2014;129:1173–86.
57.•• Colin-Ramirez E, McAlister FA, Zheng Y, Sharma S, Armstrong PW, Ezekowitz JA. The long-term effects of dietary sodium restriction on clinical outcomes in patients with heart failure. The SODIUM-HF (Study of Dietary Intervention Under 100 mmol in Heart Failure): a pilot study. Am Heart J. 2015;169:274–281.e1. **This study randomized patients to low (1.5 g/day) vs. moderate (2.3 g/day) sodium intake while adjusting HF medical therapy based on guidelines and patient's clinical status. Patients in the low sodium group had lower natriuretic peptide levels, while quality of life scores improved in both groups.**
58. Colin-Ramirez E, McAlister FA, Zheng Y, Sharma S, Ezekowitz JA. Changes in dietary intake and nutritional status associated with a significant reduction in sodium intake in patients with heart failure. A sub-analysis of the SODIUM-HF pilot study. Clin Nutr ESPEN. 2016;11:e26–32.
59.•• Bonilla-Palomas JL, Gámez-López AL, Castillo-Domínguez JC, et al. Nutritional Intervention in Malnourished Hospitalized Patients with Heart Failure. Arch Med Res. 2016;47:535–40. **The PICNIC study was a randomized controlled trial comparing usual care vs. a dietitian/physician intervention which included tailored recommendations, monthly post-discharge visits, and diet optimization through counseling and questionnaires. Patients in the intervention group had a lower rate of death and HF readmission.**
60. Gámez-López AL, Bonilla-Palomas JL, Anguita-Sánchez M, et al. Rationale and design of PICNIC study: nutritional intervention program in hospitalized patients with heart failure who are malnourished. Rev Espanola Cardiol Engl Ed. 2014;67:277–82.
61. Heart Failure Society of America, Lindenfeld J, Albert NM, et al. HFSA 2010 Comprehensive Heart Failure Practice Guideline. J Card Fail. 2010;16:e1–194.
62. Appel LJ, Frohlich ED, Hall JE, et al. The importance of population-wide sodium reduction as a means to prevent cardiovascular disease and stroke: a call to action from the American Heart Association. Circulation. 2011;123:1138–43.
63. Persson CE, Björck L, Lagergren J, Lappas G, Giang KW, Rosengren A. Risk of Heart Failure in Obese Patients With and Without Bariatric Surgery in Sweden-A Registry-Based Study. J Card Fail. 2017;23:530–7.
64. Shimada YJ, Tsugawa Y, Brown DFM, Hasegawa K. Bariatric Surgery and Emergency Department Visits and Hospitalizations for Heart Failure Exacerbation: Population-Based, Self-Controlled Series. J Am Coll Cardiol. 2016;67:895–903.
65.•• Kitzman DW, Brubaker P, Morgan T, et al. Effect of Caloric Restriction or Aerobic Exercise Training on Peak Oxygen Consumption and Quality of Life in Obese Older Patients With Heart Failure With Preserved Ejection Fraction: A Randomized Clinical Trial. JAMA. 2016;315:36–46. **This study is one of a few that have studied the effects of diet and exercise on weight loss in obese patients with HFpEF. Caloric restriction or aerobic exercise led to increased peak oxygen consumption, and the combination was additive.**
66. Dorner TE, Luger E, Tschinderle J, et al. Association between nutritional status (MNA®-SF) and frailty (SHARE-FI) in acute hospitalised elderly patients. J Nutr Health Aging. 2014;18:264–9.
67. Krumholz HM. Post-hospital syndrome–an acquired, transient condition of generalized risk. N Engl J Med. 2013;368:100–2.
68. Chaudhry SI, McAvay G, Chen S, et al. Risk factors for hospital admission among older persons with newly diagnosed heart failure: findings from the Cardiovascular Health Study. J Am Coll Cardiol. 2013;61:635–42.
69.•• Wessler JD, Maurer MS, Hummel SL. Evaluating the safety and efficacy of sodium-restricted/Dietary Approaches to Stop Hypertension diet after acute decompensated heart failure hospitalization: design and rationale for the Geriatric OUt of hospital Randomized MEal Trial in Heart Failure (GOURMET-HF). Am Heart J. 2015;169:342–348.e4. **This is a study design paper for a randomized controlled intervention testing the efficacy and safety of follow-up DASH diets after acute heart failure hospitalizations. Few dietary interventions exist in a randomized controlled manner, and this study aims to test an at-risk population in between transition from the inpatient to the outpatient setting**.
70. Zhu W, Gregory JC, Org E, et al. Gut Microbial Metabolite TMAO Enhances Platelet Hyperreactivity and Thrombosis Risk. Cell. 2016;165:111–24.
71. Wang Z, Tang WHW, Buffa JA, et al. Prognostic value of choline and betaine depends on intestinal microbiota-generated metabolite trimethylamine-N-oxide. Eur Heart J. 2014;35:904–10.
72. Stubbs JR, House JA, Ocque AJ, et al. Serum Trimethylamine-N-Oxide is Elevated in CKD and Correlates with Coronary Atherosclerosis Burden. J Am Soc Nephrol JASN. 2016;27:305–13.
73. Senthong V, Li XS, Hudec T, et al. Plasma Trimethylamine N-Oxide, a Gut Microbe-Generated Phosphatidylcholine Metabolite, Is Associated With Atherosclerotic Burden. J Am Coll Cardiol. 2016;67:2620–8.

74. Shafi T, Powe NR, Meyer TW, et al. Trimethylamine N-Oxide and Cardiovascular Events in Hemodialysis Patients. J. Am. Soc. Nephrol. JASN. 2017;28:321–31.
75. Organ CL, Otsuka H, Bhushan S, et al. Choline Diet and Its Gut Microbe-Derived Metabolite, Trimethylamine N-Oxide, Exacerbate Pressure Overload-Induced Heart Failure. Circ. Heart Fail. 2016;9:e002314.
76. Tang WHW, Wang Z, Fan Y, et al. Prognostic value of elevated levels of intestinal microbe-generated metabolite trimethylamine-N-oxide in patients with heart failure: refining the gut hypothesis. J Am Coll Cardiol. 2014;64:1908–14.
77. Wang F, Li Q, He Q, et al. Temporal variations of the ileal microbiota in intestinal ischemia and reperfusion. Shock Augusta Ga. 2013;39:96–103.
78. Llamas M-A, Aller M-A, Marquina D, Nava M-P, Arias J. Bacterial translocation to mesenteric lymph nodes increases in chronic portal hypertensive rats. Dig Dis Sci. 2010;55:2244–54.
79. Sandek A, Swidsinski A, Schroedl W, et al. Intestinal blood flow in patients with chronic heart failure: a link with bacterial growth, gastrointestinal symptoms, and cachexia. J Am Coll Cardiol. 2014;64:1092–102.
80. Pasini E, Aquilani R, Testa C, et al. Pathogenic Gut Flora in Patients With Chronic Heart Failure. JACC Heart Fail. 2016;4:220–7.
81. Cho CE, Taesuwan S, Malysheva OV, et al. Trimethylamine-N-oxide (TMAO) response to animal source foods varies among healthy young men and is influenced by their gut microbiota composition: A randomized controlled trial. Mol Nutr Food Res. 2017;61