In the current issue of *Circulation: Heart Failure*, 2 provocative articles address the influence of diet as therapy for heart failure (HF). Although the studies vary greatly in design and in neither case are the findings definitive, they are indeed intriguing. They require us to pause in the march toward optimal evidence-based medical and device therapy and reconsider not only the preventive but also the therapeutic benefits of diet in the management of patients with known HF.

Multiple iterations of the American College of Cardiology Foundation/American Heart Association HF clinical practice guidelines have endorsed, with a high degree of reasonable evidence, the importance of sodium restriction, particularly in American College of Cardiology Foundation/American Heart Association stage A and stage B HF.1 There is reasonable certainty that diet modification is an effective strategy to prevent HF and modify the progression from stage A to stage B. More trepidation emerges in consideration about the strength of recommendations for sodium restriction for stage C and stage D HF: Class IIa, level of evidence C, Sodium restriction is reasonable for patients with symptomatic HF to reduce congestive symptoms. This hardly definitive recommendation was driven by a surprising lack of evidence addressing sodium restriction specifically and dietary management more broadly.2 The 2012 European Society of Cardiology Heart Failure Guideline3 opted to provide no recommendations on sodium restriction in symptomatic HF precisely because of the poor evidence base. Recent data, albeit of questioned validity, have even suggested the possibility of harm in those patients with symptomatic HF who undergo significant sodium restriction.4,5

Regarding other dietary recommendations, the current American College of Cardiology Foundation/American Heart Association HF guidelines endorse the use of omega-3 fatty acids for therapeutic benefit in HF with reduced ejection fraction but offer a class of recommendation III for nutritional supplementation in HF with preserved ejection fraction.6 This uncertainty in a foundational element of care, that is, nutritional guidance, is unacceptable and represents not just an evidence gap but a jaundiced view. Diet is important. We can and should know more.

The 2 articles published by Levitan et al7 and Hummel et al8 revitalize core consideration of dietary management in HF. Levitan et al7 explored the relationship of dietary patterns consumed before diagnosis with mortality among women with HF who were participants in the Women’s Health Initiative (WHI). The authors included data from both the WHI Clinical Trial and the Observational Study, among participants all of whom contributed self-reported diet data collected at baseline and periodically thereafter. The diagnosis of HF was confirmed on the basis of a locally adjudicated HF hospitalization. Using multiple statistical models and sensitivity analyses, the authors examined associations between diet scores calculated to compare adherence with the MEDITERRANEAN Diet,9,10 the Dietary Approaches to Stop Hypertension (DASH) diet,11,12 and cardiovascular disease mortality after HF hospitalization. Of the 1385 WHI participants who experienced a HF hospitalization, those with highest DASH diet scores had a significantly lower hazard rate of mortality than those with the lowest DASH scores. Although the comparable MEDITERRANEAN Diet adherence scores were not statistically significant as associated with HF mortality, the trend was favorable. Adherence to both diets constituted higher intakes of fruit, vegetables, nuts, legumes, whole grains, low-fat dairy, fish, and lower intakes of red and processed meat and sugar sweetened beverages.7,10,12 Sodium intake was inversely associated with adherence to the DASH diet, but positively associated with adherence to the MEDITERRANEAN diet in this analysis. The authors concluded that diet recommendations along the lines of DASH and the MEDITERRANEAN Diet were modestly associated with lower mortality in women with HF.7 These results illustrate the potential preventive benefit of adherence to this type of eating pattern.

As noted, these data acquired from 1993 to 2005 reflect a treatment era that is coincident with the introduction of angiotensin-converting enzyme-inhibitor use for which uptake was remarkably slow and predated significant use of evidence-based β blockers. Neither therapy was represented in >40% of those women with HF.7 Primary prevention with implantable cardioverter defibrillator implantation or cardiac resynchronization therapy had not yet been introduced in standard treatment regimens. Moreover, we are not provided with the relative representation of HF with reduced ejection fraction or HF with preserved ejection fraction in these data. Thus, the possibility that these findings would be muted in a contemporary experience is real, and more data are required. Conversely, the 16% reduction in the risk of death after HF hospitalization...
eclipses any currently available or previously investigated therapies for hospitalized HF. In our search for the silver bullet, we have overlooked the silver plate.

Using a different approach, Hummel et al.8 reported findings from a clinical trial conducted among 13 patients with treated hypertension and HF with preserved ejection fraction. Citing evidence in animal models that sodium restriction improves ventricular and vascular stiffness and function, these authors fed a sodium-restricted version (sodium intake of 50 mmol or 1150 mg/2100 kcal) of the DASH diet (DASH/sodium restricted diet [SRD]) with an anticipated outcome of improved left ventricular diastolic function.9 After 21 days of consuming the DASH/SRD diet, prepared and served under observation by dietitians in a metabolic kitchen, participants demonstrated improved left ventricular diastolic function, reduced arterial elastance, and shifted V–A coupling to reflect more efficient transfer of blood between the heart and arteries. Both dietary and urinary measures corroborated adherence with highly correlated observed versus predicted measures of sodium restriction (~56%) and potassium increase (+28%). Additionally, left ventricular ejection fraction increased from 66% to 74%. The Parametrized Diastolic Filling formalism used by these investigators to evaluate diastolic function has been validated is rigorous and provides a reasonable explanation for improvements in compliance and contractility that are deemed to be load independent. The apparent improvements in diastolic function and systolic function occurring for just a 21-day period of DASH/SRD infer a remarkable treatment effect. As a small mechanistic study, assessing clinical outcomes was not the goal but directional changes in biomarker surveys and functional capacity would have helped to corroborate these findings. It is reasonable to suggest that these physiological changes reflect the effect of marked reduction in loading conditions. Regardless, this constitutes a provocative but not definitive signal that a sodium-restricted DASH diet exerts favorable influences over the short term, at least on ventricular function.

These studies, taken together, offer several potentially important considerations: (1) diet as therapy for HF is a compelling strategy that merits further investigation, especially the signal of benefit status after HF hospitalization; (2) dietary influences on ventricular function might be important and potentially offer a salutary benefit akin to traditional medical therapy; and (3) the DASH-type diet that is fundamentally similar to the MEDITERRANEAN diet, for example, high in fruits, vegetables, plant-based foods, and especially when similar to the MEDITERRANEAN diet, for example, high nutrient-dense, plant-based foods that by their nature are typically reduced in processed food intake and thereby lower in sodium. Yet, remaining questions are vital. What are the more favorable attributes of either the MEDITERRANEAN or DASH diets specifically on HF? The data reported here are either exclusively in women or largely in women.8 Are the same observations valid in men? What is the sustainability of diet modification? What are the relative costs of both the MEDITERRANEAN and DASH diets? Has cost-effectiveness been established? Are these data sufficient to endorse more fervently not just sodium restriction but a more global modification of diet recommendations in HF? Most importantly, are these findings real? As noted, both studies have a sufficient number of design complexities that may negate either set of observations.

It is precisely these questions and this overarching topic of diet as therapy for HF that prompted the National Heart, Lung and Blood Institute and the National Institutes of Health’s Office of Dietary Supplements to convene a working group in June 2013 on Designing Clinical Studies to Evaluate the Role of Nutrition and Diet in HF Management.15 Experts in cardiology, renal disease, nutrition, epidemiology and pharmacology, among others met to review existing evidence in these areas, deliberate over the current recommendations, identify gaps in knowledge and sound treatment approaches, and develop a strategic plan for how best to prioritize and address these limitations within a constrained research budget. A thorough review of the diet and nutrition research in this arena produced few well-conducted studies with adequate sample sizes, validated diet assessment methods, and longitudinal follow-up data needed to evaluate long-term benefits. The working group concluded that more studies are needed and urgently so to draw meaningful conclusions. Important questions on not just sodium but coenzyme Q10, protein wasting, sarcopenic obesity, and the microbiome implore immediacy in our pursuit of answers to these questions.

The studies presented in this issue of Circulation: Heart Failure are important steps forward, but there is a necessity to now follow-up through with definitive mechanistic and therapeutic studies that resolve the question of diet as therapy for HF. It is regrettable that in 2013, we remain so imprecise and ill-informed about a cornerstone in patient care.

Disclosures

None.

References

Van Horn and Yancy  
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