ASH Position Paper: Dietary Approaches to Lower Blood Pressure

Lawrence J. Appel, MD, MPH, on Behalf of the American Society of Hypertension Writing Group

A substantial body of evidence has implicated several aspects of diet in the pathogenesis of elevated blood pressure (BP). Well-established risk factors for elevated BP include excess salt intake, low potassium intake, excess weight, high alcohol consumption, and suboptimal dietary pattern. African Americans are especially sensitive to the BP-raising effects of excess salt intake, insufficient potassium intake, and suboptimal diet. In this setting, dietary changes have the potential to substantially reduce racial disparities in BP and its consequences. In view of the age-related rise in BP in both children and adults, the direct, progressive relationship of BP with cardiovascular-renal diseases throughout the usual range of BP, and the worldwide epidemic of BP-related disease, efforts to reduce BP in nonhypertensive as well as hypertensive individuals are warranted. In nonhypertensives, dietary changes can lower BP and delay, if not prevent, hypertension. In uncomplicated stage I hypertension, dietary changes serve as initial treatment before drug therapy. In hypertensive individuals already on drug therapy, lifestyle modifications can further lower BP. The current challenge is designing and implementing effective clinical and public health interventions that lead to sustained dietary changes among individuals and more broadly in the general population. J Clin Hypertens (Greenwich). *2009;11:358–368.*

From the Department of Medicine, Division of General Internal Medicine, Johns Hopkins University, Baltimore, MD
Address for correspondence: Lawrence J. Appel, MD, MPH, Johns Hopkins University, 2024 East Monument Street, Suite 2-618, Baltimore, MD 21205-2223
E-mail: lappel@jhmi.edu
Manuscript received October 31, 2008; accepted November 6, 2008

doi: 10.1111/j.1751-7176.2009.00136.x

Worldwide, elevated blood pressure (BP) is the leading cause of death, even exceeding deaths attributable to smoking and elevated cholesterol.[1] This finding reflects the fact that BP is a strong, consistent, continuous, independent, and etiologically relevant risk factor for cardiovascular disease (CVD)-renal disease.[2] Importantly, there is no evidence of a threshold—the risk of CVD increases progressively throughout the range of usual BP including the nonhypertensive and prehypertensive ranges.[3] Nearly a third of BP-related deaths from coronary heart disease occur in individuals who are not hypertensive.[4]

Elevated BP is extraordinarily common. According to the most recent national survey data in the United States (1999–2004), 32% of adult Americans have hypertension, and roughly another third have prehypertension.[5,6] Prehypertensive individuals are at high risk for developing hypertension and carry an excess risk of CVD compared with nonhypertensive individuals.[7] On average, African Americans have higher BP than non–African Americans,[8] as well as an increased risk of BP-related complications, particularly stroke[9,10] and kidney failure.[11] According to recent survey data, the prevalence of hypertension is increasing, while control rates remain low (<40%) but are improving slightly.[6]

Table I. Diet-Related Lifestyle Recommendations to Lower Blood Pressure

Lifestyle Modification	Recommendation
Weight loss	For overweight or obese persons, lose weight, ideally attaining a body mass index <25 kg/m^2 For nonoverweight persons, maintain desirable body mass index <25 kg/m^2
Reduced sodium intake	Lower sodium intake as much as possible, with a goal of no more than 2300 mg/d in the general population and no more than 1500 mg/d in blacks, middle- and older-aged persons, and individuals with hypertension, diabetes, or chronic kidney disease
DASH-style dietary pattern	Consume a diet rich in fruits and vegetables (8–10 servings/d), rich in low-fat dairy products (2–3 servings/d), and reduced in saturated fat and cholesterol
Increased potassium intake	Increase potassium intake to 4.7 gm/d, which is also the level provided in the DASH diet
Moderation of alcohol intake	For those who drink alcohol, consume $\leq$2 alcoholic drinks per d (men) and $\leq$1 alcohol drinks/d (women)[a]

Abbreviation: DASH, Dietary Approaches to Stop Hypertension. [a]One alcoholic drink is defined as 12 oz of regular beer, 5 oz of wine (12% alcohol), or 1.5 oz of 80 proof distilled spirits.[16]

A cardinal feature of the BP epidemic is the age-related rise in BP. In adults, systolic BP (SBP) rises by approximately 0.6 mm Hg per year.[12] Among children, ages 1 to 18 years, the average rise in SBP per year is considerably steeper, approximately 1.5 mm Hg per year in girls and 1.9 mm Hg per year in boys.[13] As a result of the age-related rise in BP, hypertension typically occurs in middle- and older-aged adults. Among adults, 50 years and older, the lifetime risk of becoming hypertensive is 90%.[14]

Elevated BP results from environmental and genetic factors and interactions among these factors. Available evidence indicates that dietary factors have a prominent and likely predominant role. In individuals without hypertension, dietary changes reduce BP and prevent hypertension, thereby lowering the risk of BP-related complications. Indeed, even a small reduction in BP, if applied to an entire population, could have a tremendous beneficial impact. It has been estimated that a 3 mm Hg reduction in systolic BP should lead to an 8% reduction in mortality from stroke and a 5% reduction in mortality from coronary heart disease.[15] In stage I hypertension, dietary changes can serve as initial therapy before the start of blood pressure medication. Among hypertensive individuals who are already taking medication, dietary changes can further lower BP and facilitate stepdown of drug therapy. In general, the extent of BP reduction is greater in hypertensives than in nonhypertensives.

The purpose of this position paper is to summarize evidence on the effects of diet-related factors that lower BP and to present recommendations for health care providers. Supportive evidence includes results from animal studies, cross-cultural studies, within-population observational studies, trials with BP as an outcome variable, and more recently some trials with clinical outcomes. The recommendations in this document are based on, and therefore similar to, those expressed in policy documents issued by the American Heart Association[16] and the federal government.[17]

SCIENTIFIC BACKGROUND

Recommended Dietary Approaches that Lower BP (Table I)

Weight Loss. Weight is directly associated with BP. The importance of this relationship is evident by the high and increasing prevalence of overweight and obesity worldwide. Approximately 65% of US adults are classified as overweight or obese and more than 30% of US adults are obese.[18] Over the past decade, the prevalence of overweight in US children and adolescents has increased, as have levels of BP.[19]

With rare exception, trials have documented that weight loss lowers BP. Importantly, reductions in BP occur without attainment of a desirable body weight. In one meta-analysis of 25 trials, mean systolic/diastolic BP reductions were 4.4/3.6 mm Hg from an average weight loss of 5.1 kg.[20] Within-trial, dose-response analyses[21,22] and prospective observational studies[23] have documented that greater weight loss leads to greater BP reduction. Other trials have documented that modest weight loss, with or without sodium reduction, can prevent hypertension by approximately 20% among prehypertensive individuals [24] and can facilitate medication stepdown and drug withdrawal.[25,26] Nonetheless, the long-term effects of sustained weight loss on BP are uncertain, with some studies suggesting attenuated BP reduction over time.[22,27]

In aggregate, available data strongly support weight reduction as an effective approach to prevent and treat elevated BP. In view of the well-recognized

challenges of maintaining weight loss, public health efforts to prevent overweight and obesity are critically important.

Reduced Salt Intake. On average, as salt (sodium chloride) intake increases, so does BP. To date, more than 50 predominantly short-term trials have been conducted. In a recent meta-analysis,[28] a median reduction in urinary sodium of approximately 1.8 g/d (78 mmol/d) lowered systolic/diastolic BP (DBP) by 2.0/1.0 mm Hg in nonhypertensive and by 5.0/2.7 mm Hg in hypertensive adults. In a meta-analysis of trials conducted in children, sodium reduction lowered mean SBP/DBP by 1.2/1.3 mm Hg.[29]

In addition to lowering BP, clinical trials have documented that reduced sodium intake prevents hypertension (relative risk reduction of approximately 20% with or without concurrent weight loss),[24] lowers BP in the setting of antihypertensive medications,[30,31] and improves hypertension control.[25,26] In observational studies, reduced sodium intake is associated with a blunted age-related rise in systolic BP.[32] To date, policy recommendations have relied on evidence that sodium reduction lowers BP. Recently, two trials of behavioral interventions to lower sodium and one trial of reduced sodium/high potassium salt have reported clinical outcomes. In each instance, there was a 21% to 41% reduction in clinical CVD events (significant reduction in 2 trials[33,34] and nonsignificant trend in the third[31]) in those who received a reduced-sodium intervention. Such evidence highlights the potential for sodium reduction to prevent CVD and should dispel any residual concern that sodium reduction might be harmful.[35]

The BP response to changes in dietary sodium intake is heterogeneous.[36] In general, the effects of sodium on BP tend to be greater in blacks and middle- and older-aged individuals. Genetic and dietary factors also influence the BP response to sodium. In the setting of either the Dietary Approaches to Stop Hypertension (DASH) diet[37] or high potassium intake,[38,39] the rise in BP for a given increase in sodium intake is blunted. While it is possible to identify subgroups that benefit more from sodium reduction, there is considerable overlap within subgroups. Importantly, there are no effective means to distinguish persons who are more or less salt-sensitive. Hence, the concept of salt sensitivity, while an important biological construct, has no clear clinical or public health application.

Available evidence supports population-wide sodium reduction, as recommended by the 2005 Dietary Guidelines for Americans. Specific recommendations are an upper limit of 2300 mg/d in the general population and an upper limit of 1500 mg/d in blacks, middle- and older-aged persons, and individuals with hypertension, diabetes, or chronic kidney disease. Together these groups comprise 69% of US adults.[40] Survey data indicate that most children and adults exceed these limits. For example, median sodium intake (mg/d) in 1988–1994 was 3700 and 3100 in boys and girls, aged 9 to 13 years, respectively, and 4300 and 2900 in men and women, aged 31 to 50 years, respectively.[41]

To reduce sodium intake, consumers should choose foods low in sodium and limit the amount of added salt. However, because more than 75% of sodium comes from processed foods,[42] any effective strategy to reduce sodium intake must involve the cooperation of food manufacturers and restaurants, which should progressively reduce the sodium added to foods by 50% over the next 10 years, as recommended by the American Medical Assocation.[2,43] Public health efforts designed to reduce sodium intake are underway in several countries (eg, Great Britain, Finland).[44] Additional efforts are likely, in large part because of the World Action on Salt and Health (WASH), an international advocacy group.

Increased Potassium Intake. High potassium intake is associated with lower BP. While data from individual trials have been inconsistent, 3 meta-analyses have each documented that increased potassium intake lowers BP in nonhypertensives and hypertensives.[45–47] In one meta-analysis,[46] average SBP/DBP reductions associated with a net increase in urinary potassium excretion of 2 gm/d (50 mmol/d) were 4.4/2.5 mm Hg in hypertensives and 1.8/1.0 in nonhypertensive individuals. Potassium appears to reduce BP to a greater extent in blacks than whites.[46] Because high intake of potassium can be achieved through diet and because potassium derived from foods is also accompanied by other nutrients, particularly bicarbonate precursors, the preferred strategy to increase potassium intake is consumption of potassium-rich foods rather than pills.

The extent of BP reduction from potassium depends on concurrent levels of salt intake and vice versa. Specifically, potassium lowers BP to a greater extent in the setting of a high salt intake compared with a low salt intake. Conversely, a reduced salt intake lowers BP to a greater extent when potassium intake is low rather than high. These data are consistent with subadditive effects from reduced

salt intake and increased potassium intake. This pattern, namely, a subadditve effect when ≥2 lifestyle interventions are implemented together, appears to occur with other combinations of effective BP-lowering therapies.[24,48]

The lack of dose response trials hinders recommendations on desirable levels of potassium intake as a means to lower BP. However, an intake of at least 4.7 gm/d (120 mmol/d) is reasonable given several considerations. This level of intake corresponds to the average total potassium intake in clinical trials of potassium supplements,[46] the highest dose in the one available dose response trial,[39] the potassium content of the DASH diet intake,[49] and the adequate intake level set by an Institute of Medicine committee.[50] Few Americans achieve this level of potassium intake. In the National Health and Nutrition Examination Survey (NHANES) III, the average intake of potassium was 2.9 to 3.2 mg/d (74–82 mmol/d) in adult men and 2.1 to 2.3 g/d (54–59 mmol/d) in adult women; only 10% of men and <1% of women consumed 4.7 gm/d (120 mmol/d) or more of potassium.[50]

In healthy individuals with normal kidney function, a potassium intake >4.7 gm/d (120 mmol/d) from foods poses no risk because excess potassium is excreted in the urine. However, in persons with impaired ability to excrete potassium, an intake <4.7 g/d (120 mmol/d) is appropriate because of the potential for adverse cardiac effects (arrhythmias) from hyperkalemia. Common drugs that can substantially impair potassium excretion are angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, nonsteroidal anti-inflammatory agents, and potassium-sparing diuretics. Medical conditions that are associated with impaired potassium excretion include advanced diabetes, chronic renal insufficiency, end-stage renal disease, severe heart failure, and adrenal insufficiency.

Moderation of Alcohol Intake. Observational studies have documented a direct dose-dependent relationship between alcohol intake and BP, particularly above approximately 2 drinks per day.[51,52] One alcoholic drink is defined as 12 oz of regular beer, 5 oz of wine (12% alcohol), or 1.5 oz of 80 proof distilled spirits. Although some studies suggest that the alcohol-BP relationship also extends into the light drinking range (≤2 drinks per day), this is the range in which alcohol may reduce the risk of coronary heart disease. In a recent meta-analysis of 15 trials,[52] decreased consumption of alcohol (median reduction in self-reported alcohol consumption of 76%, range of 16%–100%) lowered SBP/DBP by 3.3/2.0 mm Hg. BP reductions were similar in nonhypertensives and hypertensives.

In aggregate, available data support moderation of alcohol intake, among those who drink, as an effective approach to reduce BP. Alcohol consumption should be limited to no more than 1 alcoholic drink per day in women and lighter-weight persons and to no more than 2 alcoholic drinks per day in most men.

Dietary Patterns

Vegetarian Diets. Vegetarian diets are associated with reduced BP. In industrialized countries, vegetarians have markedly lower BP than nonvegetarians.[53,54] In observational studies, vegetarians also experience a blunted rise in BP with age.[55] Several aspects of a vegetarian lifestyle might lower BP, including nondietary factors, established dietary risk factors, and other aspects of a vegetarian diet (eg, high fiber, no meat). Some trial evidence suggests that established dietary risk factors and nondietary factors are not fully responsible for the BP-lowering effects of vegetarian diets and that other aspects of vegetarian diets lower BP. Specifically, in 2 trials, one in nonhypertensives[65] and another in hypertensives,[56] lactoovovegetarian diets reduced SBP by approximately 5 mm Hg but had equivocal effects on DBP.

DASH-Style Dietary Patterns. The effects of modifying whole dietary patterns have been investigated in several randomized feeding studies that tested either the original DASH diet or variants. The DASH diet emphasizes fruits, vegetables, and low-fat dairy products; includes whole grains, poultry, fish, and nuts; and is reduced in fats, red meat, sweets, and sugar-containing beverages. Accordingly, it is rich in potassium, magnesium, calcium, and fiber, and reduced in total fat, saturated fat, and cholesterol; it is also slightly increased in protein.[57] It is likely that several aspects of the DASH diet, rather than just one nutrient or food, reduced BP. Among all participants, the DASH diet significantly lowered mean SBP/DBP by 5.5/3.0 mm Hg. A second diet, that emphasized just fruits and vegetables, also lowered BP but to a lesser extent, about half of the effect of the DASH diet.

The DASH diet lowered BP in all major subgroups (men, women, African Americans, non–African Americans, hypertensives, and nonhypertensives).[58] However, the effects of the DASH diet in African Americans (SBP/DBP reductions of 6.9/3.7 mm Hg) were significantly greater than

corresponding effects in non–African Americans (3.3/2.4 mm Hg). The effects in hypertensives (SBP/DBP reductions of 11.6/5.3 mm Hg) were substantial and significantly greater than corresponding effects in nonhypertensives (3.5/2.2 mm Hg). In a subsequent trial,[37] the DASH diet significantly lowered BP at each of 3 sodium levels. A third trial, OmniHeart, compared 3 variants of the DASH diets (a diet rich in carbohydrate [58% of total calories], a second rich in protein [about half from plant sources], and a third diet rich in unsaturated fat [predominantly monounsaturated fat]).[59] In several respects, each diet was similar to the DASH diet—each was reduced in saturated fat, cholesterol, and sodium, and rich in fruit, vegetables, fiber, and potassium at recommended levels. While each diet lowered SBP, substituting some of the carbohydrate (approximately 10% of total kcal) with either protein (about half from plant sources) or with unsaturated fat (mostly monounsaturated fat) further lowered BP.

The original DASH diet, as well as the diets studied in the OmniHeart trial, are safe and broadly applicable to the general population. However, because of their high potassium, phosphorus, and protein content, these diets are not recommended in persons with chronic kidney disease.

Dietary Approaches Without Sufficient Evidence for Recommendations

Fish Oil Supplementation. Several predominantly small trials and meta-analyses of these trials[60–63] have documented that high-dose omega-3 polyunsaturated fatty acid (commonly termed "fish oil") supplements lower BP in hypertensive individuals but not in nonhypertensive individuals. The effect of fish oil appears to be dose-dependent, with BP reductions occurring at doses of 3 gm/d or more. In hypertensive individuals, average SBP and DBP reductions were 4.0 mm Hg and 2.5 mm Hg, respectively.[62] In view of side effects and the high dose required to lower BP, fish oil supplements cannot be routinely recommended as a means to lower BP.

Fiber. Dietary fiber consists of indigestible components of food from plants. Evidence from observational studies and several trials suggests that increased fiber intake may reduce BP.[64] A meta-analysis of these trials,[65] restricted to the 20 trials that increased just fiber intake, documented that supplemental fiber (average increase of 14 g/d) was associated with net SBP and DBP reductions of 1.6 and 2.0, respectively. Subsequently, in a recent large trial,[66] supplemental fiber did not lower BP. Overall, data are insufficient to recommend an increased intake of fiber alone as a means to lower BP.

Calcium and Magnesium. Evidence that dietary calcium affects BP comes from a variety of sources including animal studies, observational studies, trials, and meta-analyses. While meta-analyses suggest modest reductions in SBP of 0.9 to 1.4 mm Hg and in DBP of 0.2 to 0.8 mm Hg from calcium supplementation (400–2000 mg/d),[67–69] a trial that tested a combination of 1000 mg of elemental calcium plus 400 IU of vitamin D3 in 36,282 postmenopausal women documented no effect on BP in the whole population and in subgroups.[70] The body of evidence implicating magnesium as a major determinant of BP is likewise inconsistent. In a meta-analysis of 20 trials, there was no clear effect of magnesium intake on BP.[71] Overall, evidence is insufficient to recommend either supplemental calcium or magnesium as a dietary approach to lower BP.

Carbohydrate. A complex body of evidence suggests that both amount and type of carbohydrate affect BP.[72] Worldwide, many populations that eat carbohydrate-rich, low-fat diets have low BP levels compared with western countries.[53] Still, the results of observational studies have been inconsistent.[73–75] In early trials, albeit small, increasing carbohydrate by reducing total fat typically did not reduce BP.[76] In contrast, the recently completed OmniHeart feeding trial documented that partial substitution of carbohydrate with either protein (about half from plant sources) or monounsaturated fat lowers BP.

A few trials have also tested the effects of acute consumption of sugars on BP. In several[77,78] but not all studies,[79] increased sugar consumption raised BP. Consistent with these studies are results from a weight loss trial in which a low glycemic index diet lowered BP to a greater extent than a high glycemic index diet.[80] Overall, additional research is warranted before making recommendations about the amount and type of carbohydrate as a means to lower BP.

Intake of Fats Other than Omega-3 Polyunsaturated Fatty Acid (Fish Oil). Total fat includes saturated fat, omega-3 polyunsaturated fatty acids, omega-6 polyunsaturated fatty acids, and monounsaturated fat. Evidence on the effects of total fat, saturated fat, and omega-6 polyunsaturated fatty acids on BP is inconsistent with most studies documenting no significant effect. For monounsaturated fat, earliest trials did not support a relationship between monounsaturated fat and

BP.[76] However, subsequent trials have shown that diets rich in monounsaturated fats lower BP.[81,82] Overall, increased monounsaturated fat appears to modestly lower BP. However, an increase in monounsaturated fat is commonly linked with a reduction in the amount of carbohydrate consumed and potentially confounded by changes in the type of carbohydrate consumed.[83] Hence, the effects of monounsaturated fat intake, per se, are uncertain.

Protein Intake. An extensive, and generally consistent, body of evidence from observational studies has documented that higher protein intake is associated with lower BP.[65,84] Two major observational studies have documented this inverse relationship.[75,85] In these studies, protein from plants was associated with lower BP, while protein from animal sources had no significant effect. Some trials have also examined the effects on BP of increased protein intake. Most of these trials tested soy-based interventions that concomitantly reduced carbohydrate. In some but not all of these trials, the soy interventions reduced BP.[86,87] In one large trial conducted in China, increased protein intake from soy supplements lowered BP.[88] In the OmniHeart study, partial substitution of carbohydrate with protein (about half from plant sources) also lowered BP.[59] In aggregate, data from clinical trials, in conjunction with evidence from observational studies, support the hypothesis that substitution of carbohydrate with increased intake of protein, particularly from plants, lowers BP. However, it remains uncertain whether the effects result from reduced carbohydrate or increased protein.

Cholesterol. Few studies have studied the effect of dietary cholesterol intake on BP. In one major cohort study, there were significant, direct relationships between dietary cholesterol intake and both SBP and DBP.[73] In another cohort study, there were significant direct relationships of change in SBP over 8 years with both dietary cholesterol intake and the Keys score.[75] Still, despite these reports, the paucity of evidence precludes any conclusion about a relationship between dietary cholesterol intake and BP.

Vitamin C. A variety of evidence including laboratory studies, depletion-repletion studies, and observational studies suggest that increased vitamin C intake or status is associated with lower BP. In a systematic review by Ness and colleagues,[89] 10 of 14 cross-sectional studies reported an inverse association between blood vitamin C levels and BP, and 3 of 4 studies reported an inverse association with vitamin C intake. The 2 nonrandomized and 4 randomized trials were all small, and results were inconsistent. In a subsequent trial, 500 mg of vitamin C had no effect on BP over the course of 5 years.[90] In summary, the effects of increased vitamin C intake on BP are uncertain.

Special Populations

Children. Elevated BP begins early in childhood, during the first 2 decades of life and perhaps in utero.[91] Survey data have documented a steep age-related rise in BP in children, ages 1 to 18 years.[13] It is well-documented that BP tracks with age from childhood into the adult years.[92–94] Hence, efforts to reduce BP and prevent the age-related rise in BP in childhood are warranted. The importance of these efforts is highlighted by evidence that as levels of childhood obesity rise, so do BP levels in children and adolescents, ages 8 to 17 years.[19]

The potential impact of dietary factors on BP in children has been reviewed by Simons-Morton and Obarzanek.[95] Unfortunately, most studies had methodologic limitations. A recent meta-analysis of sodium reduction trials in children has documented that reduced sodium intake significantly lowers SBP and DBP, each by approximately 1 mm Hg.[29]

Older-Aged Persons. Because of the high prevalence of BP-related CVD, dietary interventions should be especially beneficial as adults age. Although most diet-BP trials were conducted in middle-aged individuals, several were conducted in older-aged persons.[26,96,97] Other trials presented age-stratified results. Several important patterns emerge. First, older-aged persons can make and sustain dietary changes, specifically dietary sodium reduction and weight loss.[26,98] Second, greater BP reductions occur as individuals get older.[99,100] Third, because of high-attributable risk associated with elevated BP, the beneficial effects of dietary changes in the elderly should translate into substantial reductions in CVD.[101]

African Americans. In the United States, African Americans have higher BP[8] and are at greater risk of BP-related complications[9–11] than non–African Americans. As noted previously, in efficacy studies, African Americans compared with non–African Americans achieve greater BP reduction from several dietary therapies, sodium reduction, increased potassium intake, and the DASH diet. The potential benefits of these dietary therapies are amplified because survey data indicate that African Americans consume high levels of sodium while their

Table II. Effects of Dietary Factors and Dietary Patterns on Blood Pressure: A Summary of the Evidence

	Hypothesized Effect	Evidence
Weight	Direct	++
Sodium chloride (salt)	Direct	++
Potassium	Inverse	++
Magnesium	Inverse	+/−
Calcium	Inverse	+/−
Alcohol	Direct	++
Fat		
Saturated	Direct	+/−
Omega-3 polyunsaturated	Inverse	++
Omega-6 polyunsaturated	Inverse	+/−
Monounsaturated	Inverse	+
Protein		
Total	Uncertain	+
Vegetable	Inverse	+
Animal	Uncertain	+/−
Carbohydrate	Direct	+
Fiber	Inverse	+
Cholesterol	Direct	+/−
Dietary patterns		
Vegetarian diets	Inverse	++
DASH-type dietary patterns	Inverse	++

Abbreviation: DASH, Dietary Approaches to Stop Hypertension. Key to evidence: +/− = limited or equivocal evidence. + = suggestive evidence, typically from observational studies and some clinical trials. ++ = persuasive evidence, typically from clinical trials.[16]

potassium intake is low compared with non–African Americans.[50] Such data highlight the potential of dietary change as a means to reduce racial disparities in BP and its complications.[102]

Practical Considerations and Recommendations

Individual-Based Approaches: Behavioral Interventions. A large number of behavioral intervention trials have tested the effects of dietary change on BP. A variety of theories and models have informed the design of these trials (social cognitive theory, self-applied behavior modification techniques "behavioral self-management," the relapse prevention model, and the transtheoretical or stages-of-change model). Application of these models and theories often leads to a common intervention approach that emphasizes behavioral skills training, self-monitoring, and self-regulation, along with motivational interviewing.[103] Typically, these trials enrolled motivated individuals, who expressed readiness to change, at least at the start of the trial. Further, these studies relied on skilled interventionists, often health educators or dietitians, who met frequently with participants. Characteristic findings are successful behavior change over the short-term, typically 6 months during the height of intervention intensity, and then subsequent recidivism when intervention intensity diminishes.

The limited long-term success of intensive behavioral intervention programs highlights the importance of environmental changes that facilitate adoption of desirable lifestyle changes in broad populations. Indeed, even motivated individuals find it difficult to sustain behavior change given powerful cultural forces, societal norms, and commercial interests that encourage a sedentary lifestyle, a suboptimal diet, and overconsumption of calories. Despite these impediments, available evidence from efficacy studies is sufficiently robust and persuasive to advocate dietary change as a means to lower BP and thereby prevent BP-related CVD.

Individual-Based Approaches: Clinic-Based Approaches. By example and through advice, physicians have a powerful influence on their patients' willingness to make dietary lifestyle changes.[104] Although frequent and intensive behavioral counseling is beyond the scope of typical office practices, simple assessments (eg, measurement of body mass index) and provision of basic advice (eg, "eat less, move more") is feasible. The success of physician-directed attempts to achieve lifestyle changes is dependent on several factors including the organizational structure of the office, the skills of the physician and staff, and the availability of management algorithms that incorporate locally available resources.

Physician-directed lifestyle advice should be based on the patient's willingness to make lifestyle changes. Motivated patients should be referred to a health educator, skilled dietitian, or a behavioral change program, in large part, because success typically requires frequent contacts and visits. However, even without such programs, physicians should routinely encourage lifestyle modification.

Public Health Strategies. Ultimately, people select the types and volume of food they eat and the amount of physical activity they perform. Still, as noted by other policy-making bodies, the environment (cultural forces, societal norms, and commercial interests) has a powerful influence on whether people consume excess calories, follow a healthy diet, and are physically active.[17] In view of the tremendous adverse impact of the environment and the vast scope of the BP epidemic, an effective public health strategy must be implemented concurrent

with individual-based medical care. Government, the food industry, and employers each have a vital role.

A comprehensive public health strategy must be multifactorial—no one strategy applies to each of the known dietary factors that affect BP. For weight reduction, environmental changes include prominent calorie labeling at point of purchase in both restaurants and stores, and government initiatives that encourage rather than discourage physical activity. For sodium reduction, food manufacturers and restaurants should gradually reduce the salt content of processed foods. Promotion of an overall healthy dietary pattern will require individual behavior change but also government and employer incentives that promote greater access and consumption of healthy food.

CONCLUSIONS

Multiple dietary factors affect BP. Dietary modifications that lower BP are weight loss, reduced salt intake, increased potassium intake, moderation of alcohol consumption (among those who drink), and consumption of an overall healthy dietary pattern, similar to a DASH-style diet. Other aspects of diet may also affect BP, but the effects are small and/or the evidence is uncertain (Table II).

In view of the age-related rise in BP in both children and adults, the direct progressive relationship of BP with cardiovascular-renal diseases throughout the usual range of BP, and the worldwide epidemic of BP-related disease, efforts to reduce BP in both nonhypertensive and hypertensive individuals are warranted. Such efforts will require individuals to change behavior and society to make substantial environmental changes. The current challenge is designing and implementing effective clinical and public health interventions that lead to sustained dietary changes among individuals and more broadly in the general population.

Acknowledgments: This article was reviewed by ***Richard H. Grimm, MD, PhD,*** *and* ***Thomas G. Pickering, MD, DPhil.*** *An earlier draft on this topic was prepared by* ***Norman K. Hollenberg, MD, PhD,*** *and* ***Norman M. Kaplan, MD.*** *The American Society of Hypertension Writing Group Steering Committee:* ***Thomas D. Giles, MD, Chair; Henry R. Black, MD; Joseph L. Izzo, Jr, MD; Barry J. Materson, MD, MBA; Suzanne Oparil, MD,*** *and* ***Michael A. Weber, MD.***

REFERENCES

1 Lopez AD, Mathers CD, Ezzati M, et al. Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data. *Lancet*. 2006;367:1747–1757.

2 Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension*. 2003;42:1206–1252.

3 Lewington S, Clarke R, Qizilbash N, et al. Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: A meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*. 2002;360:1903–1913.

4 Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks. US population data. *Arch Intern Med*. 1993;153:598–615.

5 Wang Y, Wang QJ. The prevalence of prehypertension and hypertension among US adults according to the new joint national committee guidelines: New challenges of the old problem. *Arch Intern Med*. 2004; 164:2126–2134.

6 Cutler JA, Sorlie PD, Wolz M, et al. Trends in hypertension prevalence, awareness, treatment, and control rates in united states adults between 1988-1994 and 1999-2004. *Hypertension*. 2008;52:818–827.

7 Vasan RS, Larson MG, Leip EP, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *N Engl J Med*. 2001;345:1291–1297.

8 Fields LE, Burt VL, Cutler JA, et al. The burden of adult hypertension in the united states 1999 to 2000: A rising tide. *Hypertension*. 2004;44:398–404.

9 Giles WH, Kittner SJ, Hebel JR, et al. Determinants of black-white differences in the risk of cerebral infarction. the national health and nutrition examination survey epidemiologic follow-up study. *Arch Intern Med*. 1995;155:1319–1324.

10 Ayala C, Greenlund KJ, Croft JB, et al. Racial/ethnic disparities in mortality by stroke subtype in the united states, 1995-1998. *Am J Epidemiol*. 2001;154:1057–1063.

11 Klag MJ, Whelton PK, Randall BL, et al. Blood pressure and end-stage renal disease in men. *N Engl J Med*. 1996;334:13–18.

12 Burt VL, Whelton P, Roccella EJ, et al. Prevalence of hypertension in the US adult population. results from the third national health and nutrition examination survey, 1988-1991. *Hypertension*. 1995;25:305–313.

13 Appel LJ. At the tipping point: Accomplishing population-wide sodium reduction in the united states. *J Clin Hypertens (Greenwich)*. 2008;10:7–11.

14 Vasan RS, Beiser A, Seshadri S, et al. Residual lifetime risk for developing hypertension in middle-aged women and men: The framingham heart study. *JAMA*. 2002;287:1003–1010.

15 Stamler R. Implications of the INTERSALT study. *Hypertension*. 1991;17:I16–I20.

16 Appel LJ, Brands MW, Daniels SR, et al. Dietary approaches to prevent and treat hypertension: A scientific statement from the american heart association. *Hypertension*. 2006;47:296–308.

17 Dietary Guidelines Advisory Committee. *2005 Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans*. Springfield, VA: US Department of Agriculture, Agricultural Research Service; 2005.

18 Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999-2000. *JAMA*. 2002;288:1723–1727.

19 Muntner P, He J, Cutler JA, et al. Trends in blood pressure among children and adolescents. *JAMA*. 2004; 291:2107–2113.

20 Neter JE, Stam BE, Kok FJ, et al. Influence of weight reduction on blood pressure: A meta-analysis of randomized controlled trials. *Hypertension*. 2003; 42:878–884.

21 Stevens VJ, Corrigan SA, Obarzanek E, et al. Weight loss intervention in phase 1 of the trials of hypertension prevention. the TOHP collaborative research group. *Arch Intern Med*. 1993;153:849–858.
22 Stevens VJ, Obarzanek E, Cook NR, et al. Long-term weight loss and changes in blood pressure: Results of the trials of hypertension prevention, phase II. *Ann Intern Med*. 2001;134:1–11.
23 Huang Z, Willett WC, Manson JE, et al. Body weight, weight change, and risk for hypertension in women. *Ann Intern Med*. 1998;128:81–88.
24 Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The trials of hypertension prevention, phase II. The trials of hypertension prevention collaborative research group. *Arch Intern Med*. 1997;157:657–667.
25 Langford HG, Blaufox MD, Oberman A, et al. Dietary therapy slows the return of hypertension after stopping prolonged medication. *JAMA*. 1985;253:657–664.
26 Whelton PK, Appel LJ, Espeland MA, et al. Sodium reduction and weight loss in the treatment of hypertension in older persons: A randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). TONE collaborative research group. *JAMA*. 1998;279:839–846. Available from: PM:9515998.
27 Sjostrom CD, Peltonen M, Wedel H, et al. Differentiated long-term effects of intentional weight loss on diabetes and hypertension. *Hypertension*. 2000;36:20–25.
28 He FJ, MacGregor GA. Effect of modest salt reduction on blood pressure: A meta-analysis of randomized trials. implications for public health. *J Hum Hypertens*. 2002;16:761–770.
29 He FJ, MacGregor GA. Importance of salt in determining blood pressure in children: Meta-analysis of controlled trials. *Hypertension*. 2006;48:861–869.
30 Weir MR, Hall PS, Behrens MT, et al. Salt and blood pressure responses to calcium antagonism in hypertensive patients. *Hypertension*. 1997;30:422–427.
31 Appel LJ, Espeland MA, Easter L, et al. Effects of reduced sodium intake on hypertension control in older individuals: Results from the trial of nonpharmacologic interventions in the elderly (TONE). *Arch Intern Med*. 2001;161:685–693.
32 Intersalt: An international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. Intersalt Cooperative Research Group. *BMJ*. 1988;297:319–328.
33 Chang HY, Hu YW, Yue CS, et al. Effect of potassium-enriched salt on cardiovascular mortality and medical expenses of elderly men. *Am J Clin Nutr*. 2006;83:1289–1296.
34 Cook NR, Cutler JA, Obarzanek E, et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: Observational follow-up of the trials of hypertension prevention (TOHP). *BMJ*. 2007;334:885.
35 Alderman MH, Ogihara T. Global challenge for overcoming high blood pressure: Fukuoka statement, 19 october 2006. *J Hypertens*. 2007;25:727.
36 Weinberger MH, Miller JZ, Luft FC, et al. Definitions and characteristics of sodium sensitivity and blood pressure resistance. *Hypertension*. 1986;8:II127–II134.
37 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.
38 Luft FC, Rankin LI, Bloch R, et al. Cardiovascular and humoral responses to extremes of sodium intake in normal black and white men. *Circulation*. 1979;60:697–706.
39 Morris RC Jr, Sebastian A, Forman A, et al. Normotensive salt sensitivity: Effects of race and dietary potassium. *Hypertension*. 1999;33:18–23.
40 Centers for Disease Control and Prevention (CDC). Application of lower sodium intake recommendations to adults–united states, 1999-2006. *MMWR Morb Mortal Wkly Rep*. 2009;58:281–283.
41 Food and Nutrition Board, Institute of Medicine. *Dietary Reference Intakes for Water, Potssium, Sodium, Chloride, and Sulfate*. Washington, DC: National Academies Press; 2005.
42 Mattes RD, Donnelly D. Relative contributions of dietary sodium sources. *J Am Coll Nutr*. 1991;10:383–393.
43 Havas S, Roccella EJ, Lenfant C. Reducing the public health burden from elevated blood pressure levels in the united states by lowering intake of dietary sodium. *Am J Public Health*. 2004;94:19–22.
44 He FJ, Macgregor GA. A comprehensive review on salt and health and current experience of worldwide salt reduction programmes. *J Hum Hypertens*. 2009;23:363–384.
45 Cappuccio FP, MacGregor GA. Does potassium supplementation lower blood pressure? A meta-analysis of published trials *J Hypertens*. 1991;9:465–473.
46 Whelton PK, He J, Cutler JA, et al. Effects of oral potassium on blood pressure. meta-analysis of randomized controlled clinical trials. *JAMA*. 1997;277:1624–1632.
47 Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: A metaregression analysis of randomised trials. *J Hum Hypertens*. 2003;17:471–480.
48 Appel LJ, Champagne CM, Harsha DW, et al. Effects of comprehensive lifestyle modification on blood pressure control: Main results of the PREMIER clinical trial. *JAMA*. 2003;289:2083–2093.
49 Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH collaborative research group. *N Engl J Med*. 1997;336:1117–1124. Available from: PM:9099655.
50 Institute of Medicine. *Dietary Reference Intakes: Water, Potassium, Sodium Chloride, and Sulfate*, 1st ed. Washington, DC: National Academy Press; 2004.
51 Klatsky AL, Friedman GD, Siegelaub AB, et al. Alcohol consumption and blood pressure kaiser-permanente multiphasic health examination data. *N Engl J Med*. 1977;296:1194–1200.
52 Xin X, He J, Frontini MG, et al. Effects of alcohol reduction on blood pressure: A meta-analysis of randomized controlled trials. *Hypertension*. 2001;38:1112–1117.
53 Sacks FM, Rosner B, Kass EH. Blood pressure in vegetarians. *Am J Epidemiol*. 1974;100:390–398.
54 Armstrong B, van Merwyk AJ, Coates H. Blood pressure in seventh-day adventist vegetarians. *Am J Epidemiol*. 1977;105:444–449.
55 Sacks FM, Kass EH. Low blood pressure in vegetarians: Effects of specific foods and nutrients. *Am J Clin Nutr*. 1988;48:795–800.
56 Margetts BM, Beilin LJ, Vandongen R, et al. Vegetarian diet in mild hypertension: A randomised controlled trial. *Br Med J (Clin Res Ed)*. 1986;293:1468–1471.
57 Karanja NM, Obarzanek E, Lin PH, et al. Descriptive characteristics of the dietary patterns used in the dietary approaches to stop hypertension trial. DASH collaborative research group. *J Am Diet Assoc*. 1999;99:S19–S27.
58 Svetkey LP, Simons-Morton D, Vollmer WM, et al. Effects of dietary patterns on blood pressure: Subgroup

analysis of the dietary approaches to stop hypertension (DASH) randomized clinical trial. *Arch Intern Med*. 1999;159:285–293.
59 Appel LJ, Sacks FM, Carey VJ, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: Results of the OmniHeart randomized trial. *JAMA*. 2005;294:2455–2464.
60 Appel LJ, Miller ER III, Seidler AJ, et al. Does supplementation of diet with 'fish oil' reduce blood pressure? A meta-analysis of controlled clinical trials. *Arch Intern Med*. 1993;153:1429–1438.
61 Morris MC, Sacks F, Rosner B. Does fish oil lower blood pressure? A meta-analysis of controlled trials. *Circulation*. 1993;88:523–533.
62 Geleijnse JM, Giltay EJ, Grobbee DE, et al. Blood pressure response to fish oil supplementation: Metaregression analysis of randomized trials. *J Hypertens*. 2002; 20:1493–1499.
63 Appel LJ, Miller ER. Bbs and bullets: The impact of dietary factors on blood pressure. *Hypertension*. 2001;37:268–269.
64 Whelton SP, Hyre AD, Pedersen B, et al. Effect of dietary fiber intake on blood pressure: A meta-analysis of randomized, controlled clinical trials. *J Hypertens*. 2005;23:475–481.
65 He J, Whelton PK. Effect of dietary fiber and protein intake on blood pressure: A review of epidemiologic evidence. *Clin Exp Hypertens*. 1999;21:785–796.
66 He J, Streiffer RH, Muntner P, et al. Effect of dietary fiber intake on blood pressure: A randomized, double-blind, placebo-controlled trial. *J Hypertens*. 2004; 22:73–80.
67 Allender PS, Cutler JA, Follmann D, et al. Dietary calcium and blood pressure: A meta-analysis of randomized clinical trials. *Ann Intern Med*. 1996; 124:825–831.
68 Bucher HC, Cook RJ, Guyatt GH, et al. Effects of dietary calcium supplementation on blood pressure. A meta-analysis of randomized controlled trials. *JAMA*. 1996;275:1016–1022.
69 Griffith LE, Guyatt GH, Cook RJ, et al. The influence of dietary and nondietary calcium supplementation on blood pressure: An updated metaanalysis of randomized controlled trials. *Am J Hypertens*. 1999;12:84–92.
70 Margolis KL, Ray RM, Van Horn L, et al. Effect of calcium and vitamin D supplementation on blood pressure: The women's health initiative randomized trial. *Hypertension*. 2008;52:847–855.
71 Jee SH, Miller ER III, Guallar E, et al. The effect of magnesium supplementation on blood pressure: A meta-analysis of randomized clinical trials. *Am J Hypertens*. 2002;15:691–696.
72 Hodges RE, Rebello T. Carbohydrates and blood pressure. *Ann Intern Med*. 1983;98:838–841.
73 Stamler J, Caggiula A, Grandits GA, et al. Relationship to blood pressure of combinations of dietary macronutrients. findings of the multiple risk factor intervention trial (MRFIT). *Circulation*. 1996;94:2417–2423.
74 Reed D, McGee D, Yano K, et al. Diet, blood pressure, and multicollinearity. *Hypertension*. 1985;7: 405–410.
75 Stamler J, Liu K, Ruth KJ, et al. Eight-year blood pressure change in middle-aged men: Relationship to multiple nutrients. *Hypertension*. 2002;39:1000–1006.
76 Morris MC. Dietary fats and blood pressure. *J Cardiovasc Risk*. 1994;1:21–30.
77 Rebello T, Hodges RE, Smith JL. Short-term effects of various sugars on antinatriuresis and blood pressure changes in normotensive young men. *Am J Clin Nutr*. 1983;38:84–94.
78 Israel KD, Michaelis OE IV, Reiser S, et al. Serum uric acid, inorganic phosphorus, and glutamic-oxalacetic transaminase and blood pressure in carbohydrate-sensitive adults consuming three different levels of sucrose. *Ann Nutr Metab*. 1983;27:425–435.
79 Visvanathan R, Chen R, Horowitz M, et al. Blood pressure responses in healthy older people to 50 g carbohydrate drinks with differing glycaemic effects. *Br J Nutr*. 2004;92:335–340.
80 Pereira MA, Swain J, Goldfine AB, et al. Effects of a low-glycemic load diet on resting energy expenditure and heart disease risk factors during weight loss. *JAMA*. 2004;292:2482–2490.
81 Rasmussen OW, Thomsen C, Hansen KW, et al. Effects on blood pressure, glucose, and lipid levels of a high-monounsaturated fat diet compared with a high-carbohydrate diet in NIDDM subjects. *Diabetes Care*. 1993;16:1565–1571.
82 Ferrara LA, Raimondi AS, d'Episcopo L, et al. Olive oil and reduced need for antihypertensive medications. *Arch Intern Med*. 2000;160:837–842.
83 Shah M, Adams-Huet B, Garg A. Effect of high-carbohydrate or high-cis-monounsaturated fat diets on blood pressure: A meta-analysis of intervention trials. *Am J Clin Nutr*. 2007;85:1251–1256.
84 Obarzanek E, Velletri PA, Cutler JA. Dietary protein and blood pressure. *JAMA*. 1996;275:1598–1603.
85 Elliott P, Kesteloot H, Appel LJ, et al. Dietary phosphorus and blood pressure: International study of macro- and micro-nutrients and blood pressure. *Hypertension*. 2008;51:669–675.
86 Teede HJ, Dalais FS, Kotsopoulos D, et al. Dietary soy has both beneficial and potentially adverse cardiovascular effects: A placebo-controlled study in men and postmenopausal women. *J Clin Endocrinol Metab*. 2001; 86:3053–3060.
87 Burke V, Hodgson JM, Beilin LJ, et al. Dietary protein and soluble fiber reduce ambulatory blood pressure in treated hypertensives. *Hypertension*. 2001;38: 821–826.
88 He J, Gu D, Wu X, et al. Effect of soybean protein on blood pressure: A randomized, controlled trial. *Ann Intern Med*. 2005;143:1–9.
89 Ness AR, Chee D, Elliott P. Vitamin C and blood pressure–an overview. *J Hum Hypertens*. 1997;11: 343–350.
90 Kim MK, Sasaki S, Sasazuki S, et al. Lack of long-term effect of vitamin C supplementation on blood pressure. *Hypertension*. 2002;40:797–803.
91 Barker DJ, Osmond C, Golding J, et al. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *BMJ*. 1989;298: 564–567.
92 Gillman MW, Cook NR, Rosner B, et al. Identifying children at high risk for the development of essential hypertension. *J Pediatr*. 1993;122:837–846.
93 Bao W, Threefoot SA, Srinivasan SR, et al. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: The Bogalusa heart study. *Am J Hypertens*. 1995;8:657–665.
94 Dekkers JC, Snieder H, Van Den Oord EJ, et al. Moderators of blood pressure development from childhood to adulthood: A 10-year longitudinal study. *J Pediatr*. 2002;141:770–779.
95 Simons-Morton DG, Obarzanek E. Diet and blood pressure in children and adolescents. *Pediatr Nephrol*. 1997;11:244–249.
96 Applegate WB, Miller ST, Elam JT, et al. Nonpharmacologic intervention to reduce blood pressure in older patients with mild hypertension. *Arch Intern Med*. 1992;152:1162–1166.

97 Cappuccio FP, Markandu ND, Carney C, et al. Double-blind randomised trial of modest salt restriction in older people. *Lancet*. 1997;350:850–854.
98 Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. 2002; 346:393–403.
99 Vollmer WM, Sacks FM, Ard J, et al. Effects of diet and sodium intake on blood pressure: Subgroup analysis of the DASH-sodium trial. *Ann Intern Med*. 2001;135:1019–1028.
100 Bray GA, Vollmer WM, Sacks FM, et al. A further subgroup analysis of the effects of the DASH diet and three dietary sodium levels on blood pressure: Results of the DASH-sodium trial. *Am J Cardiol*. 2004;94:222–227.
101 Klag MJ, Whelton PK, Appel LJ. Effect of age on the efficacy of blood pressure treatment strategies. *Hypertension*. 1990;16:700–705.
102 Erlinger TP, Vollmer WM, Svetkey LP, et al. The potential impact of nonpharmacologic population-wide blood pressure reduction on coronary heart disease events: Pronounced benefits in african-americans and hypertensives. *Prev Med*. 2003;37:327–333.
103 Miller WR, Rollnick S. *Motivational Interviewing: Preparing People to Change Addictive Behavior*, 1st ed. New York: Guilford Press; 1991.
104 Miller ER Jr, Erlinger TP, Young DR, et al. Lifestyle changes that reduce blood pressure: Implementation in clinical practice. *J Clin Hypertens (Greenwich)*. 1999; 1:191–198.