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STATE OF THE ART
REVIEWS

Janet Bond Brill, PhD, RD, LDN

Lifestyle Intervention Strategies for the Prevention and Treatment of Hypertension: A Review

Abstract: *Hypertension (HTN) is an extraordinarily common progressive cardiovascular syndrome in the United States, afflicting approximately one third of the adult population. HTN is a powerful and unequivocal independent risk factor for cardiovascular and renal diseases, including coronary heart disease, stroke, and renal failure. Despite major advances in the understanding and treatment of HTN over the past several decades, the disease remains the most common primary diagnosis in the United States and is a major public health concern. Adoption of healthy lifestyle modifications has proven to be highly effective in both the prevention of new-onset HTN and in the treatment of those diagnosed with HTN. In view of the continuing epidemic of HTN and blood pressure (BP)–related diseases and the invaluable role of applying nonpharmacological therapy in the prevention and management of HTN, a review of current therapeutic lifestyle strategies appears warranted. This review will define 6 well-established nonpharmacological lifestyle modifications for preventing and managing HTN in addition to 3 novel lifestyle interventions that show promise as effective adjunct strategies for lowering BP. A healthy lifestyle prescription ideally comprising a number of these BP-lowering lifestyle intervention strategies should be dispensed by all primary care physicians for both the prevention and treatment of elevated BP, an action that would have major, positive public health ramifications.*

Keywords: blood pressure; hypertension; health promotion; disease prevention; lifestyle; DASH dietary pattern; weight loss; sodium reduction

Introduction

According to the most recent report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (the JNC 7 report) released in 2003, blood pressure (BP) is classified into 4 categories: normal (<120/80 mm Hg), prehypertension (120/80-139/89 mm Hg), stage 1 hypertension (HTN; 140/90-159/99 mm Hg), and

> Therapeutic BP-lowering lifestyle strategies, ideally practiced simultaneously, are indispensable in the clinical management of individuals diagnosed with either prehypertension or HTN (as an adjunct to medication) and for the prevention of new-onset HTN.

stage 2 HTN (≥160/100 mm Hg).[1] The number of American adults with diagnosed HTN is staggering; 1 in 3 has HTN, estimated at 74.5 million individuals.[2] An additional 25% of the US population ≥20 years of age (~53.6 million men and women) has prehypertension. The economic burden for HTN in 2010 has been estimated at $76.6 billion (direct and indirect costs).[2] HTN is a critical independent risk factor for cardiovascular and renal diseases, including coronary heart disease, stroke, heart, and kidney failure.[3] Approximately 69% of people who have a first heart attack, 77% of those who have a first stroke, and 74% of those who have congestive heart failure have HTN.[2] In fact, HTN is the number 1 attributable risk for death worldwide.[4,5] Overt HTN shortens life span as revealed in the Framingham Heart Study, where 50-year-old hypertensive participants showed a significantly reduced life expectancy when compared with normotensive participants. Compared with the normotensive participants, total life expectancy was decreased in hypertensive men and women by 5.1 years and 4.9 years, respectively.[6]

Antihypertensive Pharmacotherapy

There are several major antihypertensive drug classes with excellent trial outcome data, including angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, β-blockers, calcium channel blockers, and thiazide-type diuretics.[1] A review of 24 randomized controlled trials (with an overall sample size of 58 040 patients) measuring total cardiovascular events for thiazides, ACE inhibitors, calcium channel blockers, and β-blockers found that low-dose thiazides are most effective as a first-choice drug for treatment of adult HTN.[7] The JNC 7 report indicates that thiazide-type diuretics should be used for treating uncomplicated HTN—either alone or in combination with other drug classes—whereas high-risk hypertensives would require 2-drug combinations. (High-risk is defined as HTN in addition to other compelling antihypertensive drug therapy indications, such as heart failure,

postmyocardial infarction, high coronary disease risk, diabetes, chronic kidney disease, or history of stroke.)[1] There are additive effects with different classes of drugs; the BP-lowering action from combining 2 classes of drugs is approximately 5 times greater than doubling the dose of a single drug.[8] The BP goal for most hypertensive patients is <140/90 mm Hg or <130/80 mm Hg for patients with diabetes or chronic kidney disease.[1]

Antihypertensive pharmacotherapy clearly reduces cardiovascular and renal morbidity and mortality.[9] Clinical trials have shown that antihypertensive therapy is associated with a significant reduction in the incidence of stroke (35%-40%), congestive heart failure (>50%), and coronary heart disease (20%-25%).[9] Prehypertension should also be treated aggressively because almost a third of BP-related deaths from cardiovascular disease occur in this population.[10] Despite these facts, HTN is still poorly controlled in the United States,[11] with only 53% of people with diagnosed HTN at goal BP.[12]

Treatments for all grades of HTN are classified as either lifestyle or pharmacotherapy.[12] The JNC 7 guidelines state that lifestyle modifications should be adopted for the treatment of prehypertension, and in conjunction with drug therapy for stage 1 and stage 2 HTN, and encouraged among all Americans for the prevention of HTN. Therapeutic BP-lowering lifestyle strategies, ideally practiced simultaneously, are indispensable in the clinical management of individuals diagnosed with either prehypertension or HTN (as an adjunct to medication) and for the prevention of new-onset HTN.[1]

This article will therefore present a summary of well-established lifestyle strategies in addition to several novel interventions that have shown promise in the prevention and treatment of HTN, and it will offer suggestions for their clinical application.

Established Lifestyle Modifications Proven to Lower Blood Pressure

The US government,[1,13] the American Heart Association,[3] the Canadian

government,[14] and the British Hypertension Society[15] have each defined a set of independent lifestyle measures proven to effectively lower BP either alone or in combination. Table 1 summarizes and compares these BP-lowering lifestyle modifications. Each of the 6 modifiable lifestyle factors addressed in Table 1 has substantial scientific evidence supporting its effectiveness, either alone or in combination, in promoting a significant reduction in BP. Here is a closer look at these well-established BP-lowering lifestyle strategies.

Weight Loss

Weight gain increases BP, and weight loss appears to reduce it.[16] A body mass index (BMI) above 25 is a strong risk factor for HTN and by far the strongest risk factor for incident HTN.[17] There is a positive association between overweight and BP because overweight is a significant and independent predictor of the level of BP.[18] Numerous trials have shown that weight loss is an effective lifestyle intervention for lowering BP in overweight and obese individuals.[19-22] According to JNC 7, a 10-kg weight loss promotes a 5- to 20-mm-Hg reduction in BP.[1]

Observational data. The Nurse's Health Study II,[17] the Physician's Health Study,[23] and the Framingham Heart Study[24] are ongoing landmark prospective cohort studies that clarify the association between excess body weight and incident HTN. In the Nurse's Health Study II of more than 116 000 women nurses, those who practiced several healthy lifestyle behaviors (as shown in Table 1) had an approximately 80% reduction in risk of developing HTN. BMI alone was the most powerful predictor of HTN.[17] In the Physician's Health Study of 13 563 men, results showed a positive significant association between BMI and risk of developing HTN in middle-aged and older men (4920 men developed HTN).[23] The Framingham Heart Study of approximately 10 000 men and women mirrored these findings. A 5% weight gain on follow-up was associated with a 20% to 30% increase in the risk of being hypertensive.[24]

347

Table 1.

A Comparison of Lifestyle Recommendations Proven to Effectively Lower BP

Recommendation	JNC 7[1]	NHLBI[13]	AHA[3]	CHEP[14]	BHS IV[15]
Lose weight	Maintain normal body weight (BMI 18.5-24.9)	Maintain normal body weight for adults (BMI 18.5–24.9 kg/m²)	For overweight and obese individuals, lose weight, aiming for a BMI < 25 kg/m², for nonoverweight individuals, maintain a desirable body weight	Maintain a healthy body weight (BMI 18.5 kg/m² to 24.9 kg/m²) and waist circumference (smaller than 102 cm for men and smaller than 88 cm for women)	Maintain ideal BMI (20-25 kg/m²)
Adopt a DASH-type dietary pattern	Consume a diet rich in fruits, vegetables, and low-fat dairy products, with a reduced content of saturated fat and total fat	Consume a diet rich in fruits, vegetables, and low-fat dairy products, with a reduced content of saturated and total fat (DASH eating plan)	Consume a diet rich in fruits, vegetables (8-10 servings per day), low-fat dairy products (2-3 servings per day), and reduced in saturated fat and cholesterol	Follow a diet that is reduced in saturated fat and cholesterol, one that emphasizes fruits, vegetables, and low-fat dairy products, dietary and soluble fiber, whole grains, and protein from plant sources	Consume a diet rich in fruits, vegetables, and low-fat dairy products, with reduced content of saturated and total fat
Reduce sodium intake	Reduce dietary sodium intake to no more than 2.4 g/d	Reduce dietary sodium intake to no more than 2.4 g/d	Lower salt (sodium chloride) intake as much as possible, ideally to 1.5 g/d of sodium	Restrict dietary sodium to less than 2300 mg (100 mmol) per day (and 1500–2300 mg [65-100 mmol] per day in hypertensive patients)	Reduce dietary sodium intake to <2.4 g/d
Increase potassium intake	—	Maintain adequate intake of dietary potassium (>3500 mg/d)	Increase potassium intake to 4.7 g/d, which is also the amount provided in DASH-type diets	—	—
Limit alcohol intake	Limit consumption to no more than 2 drinks per day (1 oz or 30 mL ethanol [eg, 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey] in most men and no more than 1 drink per day in women and lighter-weight persons	Limit alcohol consumption to no more than 1 oz (30 mL) ethanol (eg, 24 oz beer, 10 oz wine, or 2 oz 100-proof whiskey) per day in most men and to no more than 0.5 oz ethanol per day in women and lighter-weight persons	For those who drink, consume ≤2 alcoholic drinks per day (men) and ≤1 alcoholic drink per day (women)	Limit alcohol consumption to no more than 14 units per week in men or 9 units per week in women	Men ≤21 units per week; women ≤14 units per week ethanol; in the UK, a unit of alcohol is defined as 10 mL (or ~8 g ethanol
Engage in regular aerobic exercise	Regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)	Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)	—	Perform 30 to 60 minutes of aerobic exercise 4 to 7 days per week	Engage in regular aerobic physical activity, such as brisk walking, for at least 30 minutes most days

Abbreviations: BP, blood pressure; JNC 7 , Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (the JNC 7 report); NHLBI, National High Blood Pressure Education Program (NHBPEP) Coordinating Committee's Primary Prevention of Hypertension: Clinical and Public Health Advisory from the National High Blood Pressure Education Program; AHA, Dietary Approaches to Prevent and Treat Hypertension: A Scientific Statement from the American Heart Association; CHEP, 2009 Canadian Hypertension Education Program recommendations; BHS IV, 2004 report of the British Hypertension Society; BMI, body mass index; DASH, Dietary Approaches to Stop Hypertension.

Meta-analyses. Two meta-analyses of clinical trials on the effects of weight reduction on lowering BP support the efficacy of weight loss intervention for both the prevention and treatment of HTN, particularly in the short term.[25,26] The Aucott et al[25] analysis reviewed 8 clinical trials and 8 cohort studies to determine the impact of weight loss on long-term (≥2 years) BP control. Results showed that for adults with a mean baseline BMI of <35 kg/m², predicting the impact of weight loss on diastolic blood pressure (DBP) was not possible; however, for systolic blood pressure (SBP), an individual with a 5-kg weight loss may expect an average 5.6 mm Hg reduction.

The other meta-analysis examining the effect of weight reduction on HTN was published in 2003 in the journal *Hypertension*, where 25 randomized controlled clinical trials were evaluated. In this analysis, half of the 4874 participants were hypertensive (defined as a BP of ≥140/90), 24% were taking antihypertensive medication, and an undefined percentage was classified as "mildly hypertensive." The results indicated that a 1-kg weight loss was associated with an approximate 1 mm Hg reduction in both SBP and DBP. Larger BP reductions were observed in populations taking antihypertensive medications. The authors thus recommended that lifestyle modifications promoting weight loss become a major component in both the treatment and prevention of HTN.[26]

It should be noted, though, that several of the studies showed that weight loss was rarely sustained over the long term and that BP tended to revert back to initial levels with weight regain. Evidence exists that the suppression of sympathetic nervous system activity that accompanies a reduced calorie intake—and not the weight loss per se—is responsible for the reduction in BP and subsequent BP recidivism once dietary restrictions are lifted.[27]

Longer term clinical trials. The Hypertension Prevention Trial (HPT) was a randomized, controlled, primary prevention trial of 841 healthy men and women conducted over 9 months in 1983 and followed up for 3 years.[28] The study was designed to assess the effect of 2 different dietary interventions (sodium reduction with calorie control and calorie counseling alone) on the primary prevention of HTN. Participants were randomly assigned to 1 of 4 dietary treatment groups: reduced calories, reduced sodium, reduced sodium and calories, and reduced sodium and increased potassium. At 3 years, both SBP and DBP, relative to baseline, decreased in all treatment groups, but the authors found that sodium reduction with calorie control was a less effective intervention than calorie counseling alone because weight loss from calorie control gave better BP reduction.

The Trials of Hypertension Prevention (TOHP) was a long-term, large-scale study conducted during the 1990s, designed to assess the feasibility and efficacy of 7 nonpharmacological interventions (weight loss, sodium reduction, and stress management as well as nutritional supplement interventions of calcium, magnesium, potassium, and fish oil) in the primary prevention of HTN among individuals with high-normal BP (prehypertension).

The initial phase (TOHP I) examined the 3 lifestyle interventions (weight loss, dietary sodium reduction, and stress management) for 18 months in 2182 men and women with high-normal DBP.[29] At 6 months, the mean weight loss was 5.7 kg with a corresponding BP reduction of −3.8/−2.5 mm Hg. At 18 months, both the weight loss and sodium reduction groups showed significant reductions in BP, albeit less than at 6 months. The sodium reduction group exhibited a BP reduction of −1.7/−0.9 mm Hg; the weight reduction group regained some weight, with a corresponding lessening of the BP decline to −2.9/−2.3 mm Hg. Thus, the authors' conclusion was that weight reduction is the most effective of the 3 BP-lowering lifestyle strategies tested among individuals with prehypertension.

The second phase of TOHP (TOHP II) tested 3 similar interventions (weight loss, sodium reduction, and a combination of the two) over a longer time,—a 3- to 4-year follow-up period—in 2382 overweight men and women (110%-165% of desirable body weight) with high-normal BP.[30] The short-term weight loss and BP responses were similar to TOHP I. As in TOHP I, the BP-reduction effect declined over time, mirroring weight regain. At 36 months, the average BP effects were as follows: weight loss group (average 2-kg weight loss), −1.3/−0.9 mm Hg; sodium reduction group, −1.2/−0.7 mm Hg; and combined intervention group, −1.1/−0.6 mm Hg. As in the Hypertension Prevention Trial, TOHP II did not show an additive effect of BP reduction by combining lifestyle interventions.

The DASH Diet

In 1995, the NHLBI funded the landmark diet and BP study named simply the Dietary Approaches to Stop Hypertension, or DASH trial.[31] In 1997, the main results of the DASH diet trial were published,[32] illustrating for the first time that dietary intervention alone (holding body weight and sodium intake [3 g/d] constant) significantly lowers systolic and diastolic BP by an average of 11.4 and 5.5 mm Hg in hypertensives and by 3.5 and 2.1 mm Hg in nonhypertensive study participants, respectively. The magnitude of BP reduction is similar to that achieved by BP-lowering drug monotherapy for mild HTN.[33]

The DASH diet is rich in fruits, vegetables, and whole grains, with the additional inclusion of legumes, nuts, lean poultry, fish, and low-fat or fat-free dairy products (Table 2). The diet allows only a small amount of red meat, sweets, and added sugars. The DASH diet is exceptionally high in potassium, calcium, magnesium, and fiber, particularly when compared with the typical American diet. In the following section, we take a closer look at each individual mineral, keeping in mind that it is consumption of the whole DASH dietary pattern that is most effective for reducing BP.

Potassium, calcium, and magnesium. Studies have shown that a high dietary intake of the minerals potassium, calcium, and magnesium has a BP-lowering effect. (Note: the scientific evidence

Table 2.

Dietary Approaches to Stop Hypertension (DASH) Diet[a]

Food Group	Number of Servings			Sample Servings of Food
	1200-Calorie Diet	2000-Calorie Diet	2600-Calorie Diet	
Grains and grain products	6 per day	6-8 per day	10-11 per day	• 1 slice bread • ½ cup dry cereal • ½ cup cooked pasta
Vegetables	3-4 per day	4-5 per day	5-6 per day	• 1 cup raw green leafy vegetables • ½ cup cooked vegetables • ½ cup vegetable juice
Fruit	4 per day	4-5 per day	5-6 per day	• 1 medium fruit • ¼ cup dried fruit • ½ cup fruit juice
Fat-free or low-fat dairy	2-3 per day	2-3 per day	3 per day	• 1 cup fat-free milk • 1½ oz reduced-fat cheese
Meat, poultry, fish	3-6 per day	6 or less per day	6 per day	• 1 oz broiled or baked lean meats, skinless poultry, or fish • 1 egg
Nuts, seeds, beans	3 per week	4-5 per week	1 per day	• 1½ oz nuts • ½ cup cooked beans • 2 tbsp peanut butter
Added fats and oils	2 per day	2-3 per day	3 per day	• 1 tsp oil • 1 tbsp regular salad dressing • 2 tbsp light salad dressing
Sweets and added sugars	0	5 or less per week	2 or less per day	• 1 tbsp sugar • 1 tbsp jelly or jam • ½ cup sorbet

[a]The DASH diet can be accessed at http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf.

supporting the BP-regulating benefit of these minerals comes primarily from those obtaining the minerals from food rather than from supplements.)

According to the Third National Health and Nutrition Examination Survey (NHANES III), individuals with high dietary potassium intakes exhibit significantly lower BP.[34] As noted previously, the DASH trial data further support the beneficial effects of a potassium-rich diet on BP.[32] In the American Heart Association scientific statement on dietary approaches to prevent and treat HTN, the strength of the scientific evidence in support of the BP-lowering effect of dietary potassium is extensive.[3] Conversely, dietary potassium restriction increases both SBP and DBP in hypertensive and normotensive individuals.[35] It should also be noted that although the BP-lowering evidence for a high dietary intake of potassium is robust, data from individual trials examining the influence of supplemental potassium chloride on BP in hypertensive patients has been inconsistent.[36]

Just how much potassium does one need to consume to achieve BP reduction benefits? Original DASH diet participants consumed approximately 4400 mg/d of potassium, which lowered BP by an average of −2.8/−1.1 mm Hg in people with normal BP and by an average of −7.2/−2.8 mm Hg in people with HTN. However, the current recommendation is 4.7 g/d of potassium, a value based on data derived from more recent clinical trials.[3] There are caveats regarding potassium consumption: If an individual has a medical condition that impairs kidney function and/or is on certain medications that hinder potassium excretion, intake of potassium must be restricted

because of the potential for adverse cardiac effects from hyperkalemia.[3]

African Americans have a higher prevalence of HTN and poorer cardiovascular and renal outcomes than white Americans.[37] It has been hypothesized that blacks may be more salt sensitive and may consume less potassium than white Americans—as such, the DASH diet was shown to be particularly beneficial among this segment of the population.[37] Potassium has a greater BP-lowering effect in blacks compared with whites,[38] making the DASH diet an exceptionally attractive option for African Americans.

The earlier DASH diet also showed that consuming calcium-rich low-fat or fat-free dairy foods in addition to a diet high in fruits and vegetables lowers SBP and DBP further than a diet high in fruits and vegetables alone (the combination group increased dietary calcium intake to approximately 1265 mg/d). Meta-analyses of clinical trials on ingesting calcium in the form of supplements (400-2000 mg/d) also provide evidence of a modest BP-lowering effect: −0.9 to 1.4 mm Hg SBP and −0.2 to 0.8 mm Hg DBP, respectively.[39-41]

What is less well known is the joint role that calcium and vitamin D play in controlling BP. The Women's Health Study followed a large (28 886) prospective cohort of middle-aged and older women for 10 years.[42] Intake of low-fat dairy products, calcium, and vitamin D from food had an independent, inverse association with risk of HTN. As with calcium, the risk of HTN was reduced in the higher quintiles of dietary vitamin D (but did not change with supplemental vitamin D). The results of the Women's Health Study are observational; therefore, it may be the overall healthy diet that results in BP reduction rather than the vitamin D itself.

Results from the Women's Health Initiative, a randomized trial of more than 36 000 healthy postmenopausal women, support the advice to encourage consumption of calcium and vitamin D–rich foods as opposed to supplements. In the Women's Health Initiative, supplemental calcium (1000 mg) plus vitamin D3 (400 IU) were taken daily. Results

showed that the supplements did not reduce BP over 7 years of follow-up or attenuate the risk of developing pre-hypertension or HTN.[43] Therefore, it appears prudent to suggest the inclusion of nutritious calcium-rich foods that also include vitamin D, such as low-fat or fat-free dairy products or dairy substitutes, as the ideal complementary strategy to a diet rich in fruits, vegetables, and fiber, for BP regulation.

Magnesium is the third mineral that the most successful group in the original DASH trial consumed in high amounts, averaging approximately 480 mg/d. The science supporting the BP-lowering effect of dietary magnesium is inconsistent. Observational studies show an inverse relationship between dietary magnesium intake and BP,[44] whereas a meta-analysis of 20 randomized clinical trials found no effect of dietary magnesium on BP.[45] However, it should be noted that the studies in the later meta-analysis used magnesium supplementation (at a median dosage of 15.4 mmol/d) as opposed to boosting dietary intake of magnesium from food—which appears to be the preferred method. Nevertheless, considering the distinct BP-lowering effect of the DASH diet as a whole, it is of interest to identify the original DASH average daily mineral intakes and a list of foods rich in each (Table 3).

Despite the benefit of using a DASH-type diet as adjunctive lifestyle therapy in the treatment of HTN, the dietary profile of Americans diagnosed with HTN has deteriorated since the results of the DASH trial were first disseminated, and the original DASH message has faded among this population.[46] Given that healthy dietary choices are a highly effective means of attenuating the disease, efforts to promote the DASH-type eating pattern to patients must be reestablished among practicing clinicians and the nation as a whole.

Sodium Restriction

Salt (sodium chloride) saturates the American food supply, with the average man in the United States consuming 10.4 g of salt/d and the average woman consuming 7.3 g/d.[47] This amount vastly

exceeds the maximum daily amount of 3.7 g of salt (2300 mg of sodium) currently recommended by the US Departments of Agriculture and Health and Human Services for maintaining good health. It should be noted that most of the sodium in our diet does not come from the salt shaker; approximately 80% comes from daily intake of processed and restaurant foods.[48]

According to a report of the Council on Science and Public Health regarding sodium and cardiovascular disease,[48] the rise in BP with age and the prevalence of HTN are directly related to sodium intake. The Intersalt study,[49] a large population study, showed that populations with low intakes of sodium (less than 1265 mg/d) have low BP and exhibit a blunted age-related rise in BP with age. In fact, considerable scientific evidence exists linking excess salt ingestion to HTN, in addition to the data from observational studies linking high salt intake with increased cardiovascular events.[50]

Despite this evidence, dietary salt intake in the US is on the rise, posing a population problem of great magnitude. This disturbing situation prompted scientists to postulate projected health benefits of instituting a population-wide reduction of salt intake. In a study published recently in *The New England Journal of Medicine*, Bibbins-Domingo et al[47] used a computer model to forecast potentially achievable health benefits of reducing salt intake population-wide by up to 3 g/d (1200 mg of sodium/d). The results revealed that a relatively small reduction in salt intake could drastically improve public health. The projected decline in the annual number of new cases of coronary heart disease was 60 000 to 120 000; stroke, 32 000 to 66 000; and myocardial infarction, 54 000 to 99 000; also there was a reduction in the number of all-cause mortality by 44 000 to 92 000. These findings emphatically support a call to action for the federal government to regulate salt concentrations in processed, prepared, and restaurant foods and for the Food and Drug Administration to educate consumers by requiring warning labels to be placed on high-sodium foods.

351

Table 3.

Original DASH Diet Minerals Related to BP Regulation and Suggested Amounts

Mineral	Recommended Amount (mg/d)[a]	Sample Foods (mg)[b]
Calcium	1260	• Tofu, prepared with calcium sulfate (½ cup), 861 • Calcium-enriched orange juice (1 cup), 500 • Sardines (3-oz can with bones), 351 • Ricotta cheese, part skim (½ cup), 337 • Calcium-fortified soy milk (1 cup), 300 • 1 cup fat-free milk, 300 • 1 cup fat-free yogurt, 300 • Canned salmon with bones, 180 • Frozen yogurt (1 cup), 174 • Grated parmesan cheese (2 Tbsp), 138 • Artichoke, boiled (1 medium), 135
Magnesium	480	• Spinach (1 cup, steamed), 157 • Swiss chard (1 cup, chopped), 150 • Beet greens (1 cup, steamed), 98 • Ready-to-eat, high-fiber cereal (½ cup), 89 • Lentils (1 cup, cooked), 71 • Beans, black (½ cup), 60 • Wild rice (1 cup, cooked), 53 • Walnuts (1 oz, 14 halves), 45 • Sesame seeds (1 Tbsp.), 32 • Cocoa powder (1 Tbsp.), 26 • Coffee, brewed, decaffeinated, espresso (1 fl oz), 24 • Whole-grain bread (1 slice), 20
Potassium	4400	• Beet greens (1 cup, steamed), 1309 • Baked potato (1 medium with skin), 926 • Spinach (1 cup steamed), 840 • Avocado (half), 487 • Banana (1 medium), 422 • Orange juice (¾ cup), 357 • Tomatoes (1 cup cherry tomatoes), 353 • Apricots (1 oz dried), 325 • Beans, black, cooked without salt (½ cup), 305 • Kale (1 cup steamed), 296 • Cantaloupe (1 large wedge, 1/8 melon), 272 • Kiwi (1 medium), 237 • Figs (1 oz dried), 190 • Watermelon (1 cup), 173 • Grapefruit (½ fruit), 156 • Low-sodium V8 (1 cup), 820 • Raisins (½ cup), 543 • Brussels sprouts (1 cup steamed), 496

Abbreviations: DASH, Dietary Approaches to Stop Hypertension; BP, blood pressure.
[a]Recommended mineral amounts are the average amount consumed by participants in the original DASH combination diet[26] because no value for magnesium is available under current recommendations.
[b]Nutrition analysis was taken from the USDA National Nutrient Database, http://www.nal.usda.gov/fnic/foodcomp/search/.

Randomized controlled clinical trials have collectively demonstrated the benefits of sodium reduction for BP control in both normotensive and hypertensive individuals.[51-54] A meta-analysis by Feng and MacGregor[55] published in the *Journal of Human Hypertension* was based on 17 trials involving modest salt reduction in hypertensive patients and 11 similar trials in normotensive people. Salt intake was reduced by an average of 5 g/d, resulting in a mean drop in SBP/DBP of −4.96/−2.73 in hypertensive and −2.03/−0.97 mm Hg in normotensive people.

The BP response to dietary sodium restriction varies: blacks, middle-aged and older people, and individuals with chronic diseases such as diabetes, HTN, and kidney disease tend to exhibit a greater response.[3] In the TOHP II lifestyle intervention trial of prehypertensive overweight men and women discussed previously, the sodium reduction group exhibited a significant SBP decline over 36 months, which led to a decrease in HTN through the 48-month follow-up when compared with the usual-care group. A cross-sectional dose–response relationship between sodium intake and BP change was demonstrated, with larger BP reductions corresponding to lower levels of dietary sodium. Furthermore, a long-term follow-up of the sodium reduction interventions in 2 completed lifestyle intervention trials, TOHP I and TOHP II (assessed 10-15 years after the original trial), showed a 25% to 30% lower risk of cardiovascular events.[56]

Combining sodium reduction with other lifestyle modifications has an additive BP-lowering effect. For example, the addition of sodium restriction to the original DASH diet illustrated the greater BP-lowering response.[57] The DASH–Sodium trial involved completion of a 12-week feeding intervention by 390 multiracial participants with high-normal BP. Participants were randomized to 3 consecutive 30-day intervention feeding periods where the original DASH diet or the control diet (typical American diet) was combined with 3 levels of sodium intake: 50 mmol/d (1.2 g), 100 mmol/d (2.3 g), and 150 mmol/d (3.5 g) for a

2100-kCal diet.[53] Results showed that the DASH diet lowered BP in all 3 sodium-restricted groups, reinforcing the notion that the DASH diet alone is effective lifestyle therapy for reducing BP. A second finding was that restricting sodium lowers SBP and DBP at all 3 sodium levels in both the control diet and the DASH diet groups. Sequential reductions in BP corresponding to sodium level among patients following either diet illustrated that the lowest sodium intake produced the greatest reduction. A third finding was that the combined effect of the DASH diet with the greatest sodium restriction produced greater BP-lowering results than either intervention alone. In fact, the largest decrease in BP was observed between the high-sodium control diet group and the low-sodium DASH diet group (−8.9/−4.5 mm Hg, respectively).

Therefore, the optimal dietary plan appears to be a DASH diet combined with a sodium restriction of 1.2 g/d, as even greater reductions in BP are observed when compared with the DASH diet alone or sodium restriction alone. And finally, the DASH–Sodium trial also demonstrated that the findings of a BP-lowering benefit of the DASH diet and sodium restriction are applicable to several subgroups: men and women, blacks and those of other races, and individuals with HTN or without.[57]

Reduced Sodium-to-Potassium Ratio

Early research demonstrating that both dietary sodium and potassium intake are related to BP appeared in 1988 in the American Heart Association journal *Circulation*.[58] In this study, the relationship between BP and dietary intake of sodium and potassium was estimated from a 24-hour diet recall in an older group of 584 men and 718 women living in southern California. The investigators found that in both men and women, every unit ratio increase in sodium/potassium ratio reflected a SBP/DBP increase of +2.4/+1.7 and +2.2/+1.0 mm Hg, respectively.

A meta-analysis of 67 randomized sodium or potassium trials by Danish researchers revealed that a reduced intake of sodium and an increased intake of potassium independently have a positive effect on BP. The average pooled data for unweighted SBP/DBP changes for the sodium and potassium trials was −4.1/−2.5 mm Hg and −3.3/−2.1 mm Hg, respectively.[59]

The large Intersalt study examined the relationship between BP and 24-hour urinary excretion of sodium and potassium in more than 10 000 men and women, aged 20 to 59, from 52 centers around the world.[49] (Sodium/potassium urinary excretion is a surrogate measure for the amounts typically consumed in the diet.) Results showed a clear, positive correlation between sodium intake and BP and an inverse relationship between dietary potassium and BP. The authors suggest that a high ratio of potassium to sodium intake could potentially blunt the rise in BP that often accompanies aging.

During phase II of the TOHP,[60] repeated measures of participants' 24-hour urinary sodium and potassium excretion were collected and analyzed over the 4-year follow-up period in 2275 middle-aged overweight men and women (110%-165% of desirable body weight) with high-normal BP. Results showed that over the 10 to 15 years of follow-up, the sodium-to-potassium excretion ratio exhibited a direct linear relationship with a risk of having a heart attack or stroke, with a 24% increased risk per unit of the ratio elevation. According to the authors, monitoring intake of both minerals together is the most effective strategy for fighting HTN and heart disease. (The extent of BP lowering from potassium depends on levels of salt intake, and the effect is subadditive when simultaneously reducing sodium intake and increasing potassium intake).[3]

Moderate Alcohol Consumption

A direct dose–response relationship between alcohol intake and risk of HTN exists, particularly when alcohol intake exceeds 2 drinks per day.[61] The alcohol mortality risk is a J-shaped curve, with heavy drinkers exhibiting the greatest risk, light-to-moderate drinkers exhibiting the lowest risk, and an intermediate risk for abstainers.[62] The relationship between heavy alcohol drinking and an increase in BP is also well documented.[63,64] According to the US Department of Agriculture 2005 dietary guidelines, 1 drink is defined as 12 oz of beer, 5 oz of wine, or 1.5 oz of spirits, with moderate alcohol consumption defined as 1 drink/d for women and up to 2 for men. Heavy drinking has been defined as exceeding this amount.[65] In a population-based study, Stranges et al[64] confirmed that heavy alcohol consumption (≥2 drinks/d) is associated with a higher risk for HTN.

The Atherosclerosis Risk in Communities Study was a longitudinal cohort study of 8334 free-living, healthy, middle-aged participants followed for 6 years.[66] Results showed an increased risk of incident HTN in those who consumed large amounts of ethanol, defined as ≥210 g/wk. The consumption of low-to-moderate amounts of alcohol was found to be a risk factor for HTN only in black men.

A combined analysis of data derived from 2 large prospective cohorts—28 848 women from the all-female Women's Health Study and 13 455 men from the all-male Physicians' Health Study—showed that alcohol affects men and women differently. Light-to-moderate alcohol intake decreased the risk of HTN in women (a J-shaped association) but increased the risk in men in a linear fashion with no BP benefits for light-to-moderate consumption. Consistent between genders was the tendency for heavier alcohol consumption—more than 2 drinks a day—to increase the risk of developing HTN in both men and women.[63]

The scientific consensus appears to be that moderating excessive alcohol intake can lower BP by up to 4 mm Hg on average.[65] A meta-analysis of 15 randomized controlled clinical trials showed that in heavy drinkers (defined as a mean baseline alcohol consumption of 3-6 drinks/d), reducing intake is associated with significant BP reduction.[61] An average of a 67% reduction in alcohol intake reduced SBP/DBP by a net change of −3.31/−2.04 mm Hg, respectively. The rise in BP as a result

of excess alcohol intake is largely reversible within 2 to 4 weeks of abstinence or extreme moderation.[67]

Regular Aerobic Exercise

Ample research demonstrates that physical activity lowers the risk of developing HTN and is an effective BP-lowering treatment for diagnosed HTN.[68,69] Epidemiological studies clearly show an inverse relationship between physical activity level and BP,[70] with higher physical activity levels reducing risk of incident HTN by approximately 15%.[71,72] Several meta-analyses of randomized clinical trials demonstrate that chronic dynamic aerobic endurance training lowers both resting BP and ambulatory BP in prehypertensive, hypertensive, and normotensive people.[70-72]

Whelton et al[71] conducted a meta-analysis of 54 randomized clinical trials (2419 participants) to determine the effects of aerobic exercise on BP. Results showed that a regular aerobic exercise program had an impressive BP lowering effect in previously sedentary individuals, with reductions in SBP and DBP of −3.84/−2.58 mm Hg, respectively. BP was lowered in hypertensive, normotensive, overweight, normal weight, black, white, or Asian people. It was also shown that BP was reduced by aerobic exercise in hypertensive people who had a normal body mass, hence the conclusion that exercise results in BP reductions independent of weight loss.

In a more recent meta-analysis of 44 randomized controlled intervention trials (2674 participants) studying the effects of aerobic exercise training on resting BP, average SBP and DBP declined by −2.6/−1.8 mm Hg in normotensive participants and −7.4/−5.8 mm Hg in hypertensives.[70] Another comprehensive meta-analysis (72 trials) reported the effects of aerobic exercise training on both resting BP and ambulatory BP in some 4000 normotensive or hypertensive participants.[72] Training induced significant net reductions in both resting and ambulatory SBP and DBP of −3.0/−2.4 mm Hg and −3.3/−3.5 mm Hg, respectively. The reduction in BP was more pronounced in the 30 hypertensive study groups (−6.9/−4.9 mm Hg) compared with the normotensive groups (−1.9/−1.6 mm Hg), illustrating the effectiveness of using exercise as means to both treat and prevent HTN. The authors concluded that chronic aerobic endurance exercise training induces an adaptive reduction in BP partially by decreasing systemic vascular resistance involving the sympathetic nervous system and the renin–angiotensin system.

What is the optimal exercise prescription for treating HTN? The American College of Sports Medicine (ACSM) Position Stand on Exercise and Hypertension recommends a program of primarily aerobic endurance exercise as an integral component of the lifestyle prescription for the prevention, treatment, and control of HTN.[68] The ACSM suggests the following guidelines for individuals with diagnosed HTN:

Frequency: on most, preferably all, days of the week
Intensity: moderate
Time: ≥30 minutes of continuous or accumulated physical activity per day
Type: primarily endurance physical activity supplemented by resistance exercise

The more recent 2008 Department of Health and Human Services (DHHS) *Physical Activity Guidelines Advisory Committee Report*'s conclusion on frequency, intensity, and mode of exercise that yields important reductions in both SBP and DBP is consistent with the ACSM guidelines.[69] Both aerobic exercise and resistance exercise reduce BP, but the evidence backing aerobic exercise is more substantial. According to the DHHS advisory report, the following exercise prescription yields reproducible effects on BP reduction in hypertensives:

Frequency: performed 3 to 5 times per week
Intensity: moderate- to high-intensity exercise
Time: 40 minutes of aerobic exercise training

Type: aerobic exercise involving more than 800 MET (metabolic equivalent) minutes per week (or 12 miles per week moderate and/or vigorous). (Note: For weight loss of 5% or more, the DHHS recommends a greater volume of physical activity, or 1560 MET minutes or more per week—equivalent to walking about 45 minutes per day at 4 miles per hour or about 70 minutes per day at 3 miles per hour, or jogging 22 minutes per day at 6 miles per hour.)

Regarding the ideal exercise prescription intensity, Cornelissen et al[73] demonstrated that 10 weeks of aerobic endurance training at either a high or low intensity reduced SBP at rest, during exercise, and during recovery in a similar manner. Exercise training programs involved walking/jogging/running for 23 minutes, cycling for 23 minutes, and stepping for 5 minutes for a total of 51 minutes per session 3 days per week at either 33% or 66% of heart rate reserve. The findings suggest that frequency, time, and mode are more important components of the exercise prescription than intensity when it comes to eliciting BP reduction.

Combination Lifestyle Therapy for Optimal BP Management

Evidence supporting the contention that a combination of multiple lifestyle strategies simultaneously is beneficial for maximizing BP reduction comes from numerous randomized clinical trials.[18,52,74-76] The NHLBI-sponsored PREMIER trial, for example, was a 6-month multicenter trial investigating the effects of a combination of well-established lifestyle interventions on BP of prehypertensive and stage-1 hypertensive, free-living individuals.[52,77] Participants (810 men and women) were randomly assigned to 1 of 3 groups: (1) a composite of established lifestyle recommendations (weight loss, sodium restriction, exercise, and moderation of alcohol consumption), (2) DASH diet plus established lifestyle recommendations, and

(3) an "advice-only" comparison group consisting of diet and lifestyle advice given in a single 30-minute session. After the treatment period, the "established plus DASH" group showed an absolute mean decrease in BP of −11.1/−6.4 mm Hg from baseline to 6 months; the "established" group showed an absolute mean decrease in BP of −10.5/−5.5 mm Hg; and the "advice-only" group showed an absolute decline of −6.6/−3.8 mm Hg. The PREMIER study illustrates that a multifaceted combination of BP-lowering lifestyle strategies is well tolerated among free-living individuals. Yet the study did not show a strong additive effect of combining lifestyle treatments, which may be partly because of the relatively low baseline BP of participants.[78]

Several additional randomized controlled studies have, however, demonstrated a powerful additive effect of combination therapy.[18,74,75] Cox et al[18] showed that in sedentary overweight men, combining calorie reduction with a program of regular vigorous exercise exhibited a synergistic effect on reducing BP. Another study, a large, randomized, multicenter controlled trial of nonpharmacological interventions in older people (TONE), used a combination of sodium reduction with weight loss to treat HTN.[79] Participants (975 men and women aged 60-80 years, overweight and nonoverweight) were randomized to 1 of 3 lifestyle interventions (sodium reduction, weight loss, or combined) or to a usual-care group (control). After 29 months, results revealed that the sodium-reduction group lowered the need for antihypertensive medications by 31%, the weight loss group by 36%, and the combination by 53% when compared with the usual-care group; hence, the authors' conclusion that a combination of modest lifestyle changes (such as losing weight and cutting salt intake) is a safe and feasible method for eliminating or lessening the need for hypertensive medication in elderly Americans.

The Diet, Exercise, and Weight Loss Intervention Trial (DEW-IT) was a comprehensive lifestyle modification study demonstrating that combination adjuvant BP-lowering lifestyle therapy (a low-calorie, sodium-restricted DASH diet combined with a moderate-intensity exercise program) can substantially reduce BP and improve BP control in overweight hypertensive individuals on antihypertensive medication.[74] In DEW-IT, 44 hypertensive overweight adults on a single BP medication were randomized to either a lifestyle or a control group. At the end of the 9-week intervention, mean weight loss in the lifestyle group was 5.5 kg, with a corresponding mean reduction in 24-hour ambulatory SBP and DBP of −10.5 mm Hg and −5.9 mm Hg, respectively.

The ENCORE study showed that adding exercise and weight loss lifestyle interventions boost the BP-lowering effect of the DASH diet.[76] In this clinical trial, 144 overweight and obese unmedicated outpatients with prehypertension or stage 1 HTN were randomized to the DASH diet alone, to DASH combined with a weight management program (a weekly cognitive-behavioral therapy session combined with aerobic exercise 3 times a week), or to the usual diet for 4 months. The main outcome was a significant reduction in clinic-measured SBP/DBP by −16.1/−9.9 mm Hg in the DASH plus weight management group, by −11.2/−7.5 mm Hg in the DASH alone group, and by −3.4/−3.8 mm Hg in the usual-care group. This recent landmark study confirms the beneficial BP-lowering effect of the DASH diet compared with the typical American diet in unmedicated hypertensive overweight men and women. The study also highlights the added value of combining exercise and weight loss with the DASH diet for maximizing BP reduction in this population.[76]

Novel BP-Lowering Lifestyle Interventions

Several less-well-established lifestyle modifications are gathering scientific backing in support of their potential ability to reduce BP. Although promising, the research on these novel therapies is preliminary at best and far from conclusive. Many more published trials will be needed before definitive conclusions can be drawn regarding the BP-lowering effectiveness of the treatments.

Dark Chocolate

Cocoa is a rich source of the main subclass of polyphenols, the flavanols. Data are starting to accumulate suggesting that consumption of flavanol-rich cocoa products, such as dark chocolate and cocoa beverages, has a measurable BP-lowering effect. A recent systematic review and meta-analysis of 10 randomized clinical trials comprising 297 participants[80] showed that SBP and DBP were lowered by −4.5 and −2.5 mm Hg, respectively, following daily cocoa ingestion (of varying amounts) for periods of 2 to 18 weeks. In 2007, a smaller meta-analysis (5 randomized clinical trials with 173 normotensive and hypotensive participants) examined the effect of flavonol-rich dark chocolate and cocoa on BP and also reported beneficial hypotensive effects.[81] Mean SBP and DBP were −4.7 and −2.8 mm Hg lower compared with cocoa-free controls. Flavanols have been shown to induce arterial vasodilation by increasing the endothelial production of nitric oxide, which the authors state is the likely mechanism for the reduction in BP from consuming flavanol-rich cocoa products.

A randomized clinical trial tested the effects of ingesting a daily dose of dark chocolate on BP in 44 men and women with prehypertension or stage 1 HTN (not taking medication) over 18 weeks.[82] Participants were divided into 2 groups. One group consumed a 30-calorie piece of dark chocolate (6.3 g)—high in flavonoid polyphenols—and the other a matching 5.6-g dose of polyphenol-free white chocolate daily. Patients who ate the dark chocolate exhibited a mean reduction in SBP/DBP of −2.9/−1.9 mm Hg without any accompanying drop in body weight. In contrast, the white chocolate eaters had no change in BP from baseline values.

New research in animals supports the potential for cocoa fiber to reduce BP in hypertensive rats. Sánchez and colleagues[83] randomly divided 20 male rats into 2 groups, 1 receiving tap water (control) and the other a solution of water

with added cocoa fiber extract for a period of 17 weeks. The cocoa-fed group exhibited a reduction in SBP by 10 to 15 mm Hg. Another rodent study examining the acute BP-lowering effects of cocoa compared with a common antihypertensive medication had similar findings. Hypertensive rats fed 300 mg/kg body weight of a high-polyphenol cocoa powder had SBP lowered to the same degree as a 50-mg/kg dose of captopril.[84] Thus, preliminary data derived from both animal and human studies suggest that ingesting flavanols may have promise as an effective lifestyle strategy in the treatment of HTN.

Soy

Epidemiological data suggest that long-term usual intake of soy foods has a small but beneficial effect on BP. The Shanghai Women's Health Study is a large population-based longitudinal study of healthy middle-aged and elderly Chinese women. Soy food intake was significantly and inversely related to both SBP and DBP measured 2 to 3 years after baseline assessment, particularly among the older women. The adjusted mean SBP for elderly women was 4.9 mm Hg lower, and the DBP was 2.2 mm Hg lower in women who consumed ≥25 g soy protein/d compared with those consuming <2.5 g/d.[85]

Randomized clinical trials also support the BP-lowering effect of eating soy foods, especially when substituting vegetable protein in place of animal protein in postmenopausal normotensive and hypertensive women, according to research published in the *Archives of Internal Medicine*.[86] A randomized, controlled crossover study investigated the effects of applying a National Cholesterol Education Program Therapeutic Lifestyle Changes diet with or without 25 g of soy protein daily (in the form of ½ cup of unsalted soy nuts) for two 8-week periods. Results showed that the hypertensive women had a mean decrease in SBP of 9.9% and a 6.8% decrease in DBP on the soy diet (−15.0/−6.0 mm Hg, respectively) compared with the control diet. Normotensive women exhibited a 5.2%

decrease in SBP and a 2.9% decrease in DBP on the soy diet −6.0/−2.0 mm Hg, respectively, compared with the control diet, with no change in BMI or exercise in either group. The BP-lowering effect of soy, according to the researchers, was comparable with those seen with antihypertensive medications.

Several additional randomized clinical trials have shown that short-term soy intake significantly reduces both SBP and DBP,[87-90] with substantially greater reductions noted in hypertensive compared with normotensive participants.[89] A randomized, controlled, double-blind multicenter trial conducted in the People's Republic of China further supports the potential BP-lowering effect of soy.[87] More than 300 men and women with prehypertension or stage 1 HTN were randomly assigned either 40 g of soybean protein per day or a placebo for 12 weeks. The net decrease in SBP/DBP for hypertensive and normotensive participants was −8.0/−5.0 mm Hg and −2.0/−1.0 mm Hg, respectively.

It should be noted that other small clinical trials have reported no statistically significant findings.[91] Therefore, despite the promising results, the data on the BP-lowering effect of soy is still preliminary, and much more research is needed to confirm the positive BP effects of replacing some animal protein with soy protein.

Relaxation Therapy

Chronic emotional and psychological stress has been implicated as a causal element in the development of sustained HTN.[92] Therefore, a variety of stress-reduction lifestyle therapies have been proposed as methods to ameliorate the harmful effects of stress. In fact, the Canadian government has suggested stress management as part of its lifestyle therapeutic approach for select individuals with HTN.[14] Relaxation lifestyle treatments fall under 4 general categories: (1) physical techniques such as yoga, breathing exercises, and progressive muscle relaxation; (2) biofeedback; (3) autogenic training relaxation techniques; and (4) cognitive-behavioral

therapy such as guided imagery, talk therapy, and meditation.

A recent Cochrane review of 25 randomized controlled trials (1198 participants) using different types of relaxation therapies for the management of primary HTN in adults[93] showed that a regular program of relaxation therapy results in a small yet statistically significant reduction in SBP and DBP compared with controls (mean SBP difference of −5.5 and −3.5 mm Hg, respectively). The review concluded that progressive muscle relaxation, biofeedback, and cognitive-behavioral therapies were the types of relaxation treatments most likely to be effective. The author did note that many of the studies were of poor quality with unexplained variation between trials, suggesting that the causal association between relaxation techniques and BP reduction is weak. These conclusions concurred with older analyses that also failed to find statistically significant BP effects with stress reduction programs.[94,95] Those analyses cautioned that many of the stress reduction BP trials were not well designed and tended to overestimate the treatment effect, and therefore, much more research is needed.

Nevertheless, research on individual stress reduction modalities continues to surface, suggesting the potential effectiveness of these techniques on BP. Slow breathing exercises (6 cycles/min) have been shown to result in an acute decrease in BP in hypertensive patients—with an accompanying increase in baroreflex sensitivity.[96] The authors suggest that this technique appears to be potentially beneficial in the management of HTN. A randomized controlled observer-blind pilot trial aimed at determining the effect of contemplative meditation combined with breathing exercises supports meditation as an antihypertensive strategy.[92] Participants included 52 people with pharmacologically untreated HTN randomly assigned to 8 weeks of meditation/breathing exercise treatment or no intervention under both resting conditions and mental stress. The results showed that the average BP during mental stress at follow-up was a substantial 11 mm Hg lower in the treatment

group compared with controls. Hence, preliminary research indicates that meditation and breathing exercises can be effective antihypertensive therapy in individuals with HTN exposed to mental stress.

A recent meta-analysis of well-designed randomized controlled clinical trials on stress reduction and BP calculated the effects of various stress reduction approaches: biofeedback, relaxation-assisted biofeedback, progressive muscle relaxation, stress management training, and transcendental meditation. Results showed that only the transcendental mediation program showed statistically significant effects, with reductions in SBP and DBP of −5.0 and −2.8 mm Hg, respectively.[97]

Summary and Application

The epidemic of HTN in the United States and the associated adverse health consequences necessitates a population strategy emphasizing the practice of healthy lifestyle measures among all Americans for the primary prevention and clinical management of HTN. The best approach to employing therapeutic lifestyle changes is to prescribe them in combination. Alone or ideally in concert, these lifestyle strategies have the potential to lower BP and/or enhance the effectiveness of hypertensive medications.

A summary of the 6 established lifestyle interventions for the prevention and treatment of HTN are listed here followed by the 3 novel interventions with less proven efficacy:

1. Maintain and/or achieve a normal body weight for adults aiming for a BMI <25.
2. Reduce sodium intake to <2.5 g/d and ideally 1.5 g/d.
3. Follow a DASH-type dietary pattern, consuming at least 4700 mg/d of potassium (from food).
4. Follow a DASH-type dietary pattern, including daily intake of vitamin D–fortified low-fat or fat-free dairy products (providing at least 1200 mg/d of calcium) and foods rich in magnesium (providing at least 480 mg/d).

5. For those who drink, moderate alcohol consumption should be limited to ≤2 alcoholic drinks/d (men) and ≤1 alcoholic drink/d (women).
6. Engage in regular aerobic exercise such as brisk walking (≥40 minutes on most and preferably all days of the week).
7. Eat a small amount of dark chocolate daily.
8. Include 25 g of soy protein/d in place of animal protein.
9. Practice healthful stress management techniques daily such as meditation or slow breathing exercises.

It should be noted that the last 3 recommendations are novel BP-lowering lifestyle strategies with preliminary and far from conclusive supporting scientific evidence.

A silent killer, HTN remains the most common primary diagnosis in the United States and is a significant public health concern as it is a major risk factor for coronary heart disease, our nation's leading cause of death. Irrefutable scientific evidence supports adoption of healthful lifestyle intervention strategies as a highly effective tactic in preventing new-onset HTN and as valuable adjunctive therapy in treating diagnosed HTN. In light of these findings, it would appear prudent to answer the World Health Organization's[4] call to action to reduce the sodium intake of all Americans to a reasonable level by having our government institute regulations for food manufacturers and restaurateurs, requiring them to reduce the salt content of our food. In addition, as recommended by JNC 7,[1] a combination of the BP-lowering lifestyle intervention strategies described in this article should be recommended as the first line of therapy for the prevention of HTN and in combination with medication for the optimal management of high BP and its sequelae to reduce the burden of the most preventable cause of premature morbidity and mortality in developed and developing countries.[15] Health professionals should actively promote BP-lowering lifestyle strategies that will help control the mass

public health problem of rampant HTN. Instituting population-wide modest lifestyle changes is a cost-effective, practical approach to addressing the BP problem and will contribute greatly to improving the health and longevity of our nation's citizens. AJLM

References

1. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289:2560-2571.

2. Lloyd-Jones D, Adams RJ, Brown TM, et al; for the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics: 2010 update: a report from the American Heart Association. *Circulation*. 2010;121:e46-e215.

3. 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.

4. World Health Organization. *World Health Report 2002: reducing risks, promoting healthy life*. Geneva, Switzerland: World Health Organization; 2002.

5. 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.

6. Franco OH, Peeters A, Bonneux L, de Laet C. Blood pressure in adulthood and life expectancy with cardiovascular disease in men and women. *Hypertension*. 2005;46:280-286.

7. Wright JM, Musini VM. First-line drugs for hypertension. *Cochrane Database Syst Rev*. 2009;(3):CD001841.

8. Wald DS, Law M, Morris JK, Bestwick JP, Wald NJ. Combination therapy versus monotherapy in reducing blood pressure: meta-analysis on 11,000 participants from 42 trials. *Am J Med*. 2009;122:290-300.

9. Neal B, MacMahon S, Chapman N. Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs. *Lancet*. 2000;355:1955-1964.

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

11. Wexler R, Aukerman G. Nonpharmacologic strategies for managing hypertension. *Am Fam Physician*. 2006;73:1953-1956.

12. Dolor RJ, Yancy WS, Owen WF, et al. Hypertension Improvement Project (HIP): study protocol and implementation challenges. *Trials*. 2009;10:13.

13. Whelton PK, He J, Appel LJ, et al; for the National High Blood Pressure Education Program Coordinating Committee. Primary prevention of hypertension: clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA*. 2002;288:1882-1888.

14. Khan N, Hemmelgarn B, Padwal R, et al. The 2009 Canadian Hypertension Education Program recommendations for the management of hypertension: part 2—therapy. *Can J Cardiol*. 2009;25:287-298.

15. Williams B, Poulter NR, Brown MJ, et al. Guidelines for management of hypertension: report of the fourth working party of the British Hypertension Society, 2004—BHS IV. *J Hum Hypertens*. 2004;18:139-185.

16. Ebrahim S, Smith GD. Lowering blood pressure: a systematic review of sustained effects of non-pharmacological interventions. *J Public Health Med*. 1998;20:441-448.

17. Forman JP, Stampfer MJ, Curhan GC. Diet and lifestyle risk factors associated with incident hypertension in women. *JAMA*. 2009;302:401-411.

18. Cox KL, Puddey IB, Morton AR, et al. Exercise and weight control in sedentary overweight men; effects on clinic and ambulatory blood pressure. *J Hypertens* 1996;14:779-790.

19. Davis BR, Blaufox MD, Oberman A, et al. Reduction in long-term antihypertensive medication requirements: effects of weight reduction by dietary intervention in overweight persons with mild hypertension. *Arch Intern Med*. 1993;153:1773-1782.

20. Schotte DE, Stunkard AJ. The effects of weight reduction on blood pressure in 301 obese patients. *Arch Intern Med*. 1990;150:1701-1704.

21. Dornfeld LP, Maxwell MH, Waks AU, Schroth P, Tuck ML. Obesity and hypertension: long-term effects of weight reduction on blood pressure. *Int J Obes*. 1985;9:381-389.

22. Fagerberg B, Andersson OK, Isaksson B, Bjorntorp P. Blood pressure control during weight reduction in obese hypertensive men: separate effects of sodium and energy restriction. *BMJ*. 1984;288:11-14.

23. Gelber RP, Gaziano M, Manson JE, Buring JE, Sesso HD. A prospective study of body mass index and the risk of developing hypertension in men. *Am J Hypertens*. 2007;20:370-377.

24. Vasan RS, Larson MG, Leip EP, Kannel WB, Levy D. Assessment of frequency of progression to hypertension in non-hypertensive participants in the Framingham Heart Study: a cohort study. *Lancet*. 2001;358:1682-1686.

25. Aucott L, Rothnie H, McIntyre L, Thapa M, Waweru C, Gray D. Long-term weight loss from lifestyle intervention benefits blood pressure? A systematic review. *Hypertension*. 2009;54:756-762.

26. Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2003;42:878-884.

27. Mark AL. Weight reduction for treatment of obesity-associated hypertension: nuances and challenges. *Curr Hypertens Rep*. 2007;9:368-372.

28. Hypertension Prevention Trial Research Group. The Hypertension Prevention Trial: three-year effects of dietary changes on blood pressure. *Arch Intern Med*. 1990;150:153-162.

29. The Trials of Hypertension Prevention Collaborative Research Group. The effects of non-pharmacological interventions on blood pressure of persons with high normal levels: results of the Trials of Hypertension Prevention (phase I). *JAMA*. 1992;267:1213-1220.

30. The Trials of Hypertension Prevention Collaborative Research Group. Effects of weight loss and sodium reduction on blood pressure and hypertension incidence in overweight people with high-normal blood pressure: The Trials of Hypertension Prevention (phase II). *Arch Intern Med*. 1997;157:657-667.

31. Sacks FM, Obarzanek E, Windhauser MM, et al; and the DASH Investigators. Rationale and design of the Dietary Approaches to Stop Hypertension Trial (DASH): a multicenter controlled feeding study of dietary patterns to lower blood pressure. *Ann Epidemiol*. 1995;5:108-118.

32. Appel L, Moore TJ, Obarzanek E, et al; for the DASH Collaborative Research Group. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997;336:1117-1124.

33. The Treatment of Mild Hypertension Research Group. The treatment of mild hypertension study: a randomized, placebo-controlled trial of a nutritional-hygienic regimen along with various drug monotherapies. *Arch Intern Med*. 1991;151:1413-1423.

34. Hajjar IM, Grim CE, George V, Kotchen TA. Impact of diet on blood pressure and age-related changes in blood pressure in the US population: analysis of NHANES III. *Arch Intern Med*. 2001;161:589-593.

35. Krishna GG, Kapoor SC. Potassium depletion exacerbates essential hypertension. *Ann Intern Med*. 1991;115:77-83.

36. Grimm RH, Neaton JD, Elmer PJ, et al. The influence of oral potassium chloride on blood pressure in hypertensive men on a low-sodium diet. *N Engl J Med*. 1990;322:623-624.

37. Douglas JG, Bakris GL, Epstein M, et al. Management of high blood pressure in African Americans: Consensus Statement of the Hypertension in African Americans Working Group of the International Society on Hypertension in Blacks. *Arch Intern Med*. 2003;163:525-541.

38. 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.

39. Allender PS, Cutler JA, Follmann DA, Cappuccio FP, Pryer J, Elliott P. Dietary calcium and blood pressure: a meta-analysis of randomized clinical trials. *Arch Intern Med*. 1996;124:825-831.

40. 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.

41. Cappuccio FP, Elliott P, Allender PS, Pryer J, Follmann DA, Cutler JA. Epidemiologic association between dietary calcium intake and blood pressure: a meta-analysis of published data. *Am J Epidemiol*. 1995;142:935-945.

42. Wang L, Manson JE, Buring JE, Lee I-M, Sesso HD. Dietary intake of dairy products, calcium, and vitamin D and the risk of hypertension in middle-aged and older women. *Hypertension*. 2008;51:1073-1079.

43. Margolis, KL, Ray RM, Van Horn L, et al; for the Women's Health Initiative Investigators. Effect of calcium and vitamin D supplementation on blood pressure: The Women's Health Initiative Randomized Trial. *Hypertension*. 2008;52:847-855.

44. Mizushima S, Cappuccio FP, Nichols R, Elliott P. Dietary magnesium intake and blood pressure: a qualitative overview of the observational studies. *J Hum Hypertens*.1998;12:447-453.

45. Jee SH, Miller ER, Guallar F, Singh VK, Appel LJ, Klag MJ. The effect of magnesium supplementation on blood pressure: a meta-analysis of randomized clinical trials. *Am J Hypertens*. 2002;15:691-696.

46. Mellen PB, Gao SK, Vitolins MZ, Goff DC. Deteriorating dietary habits among adults with hypertension: DASH dietary accordance, NHANES 1988-1994 and 1999-2004. *Arch Intern Med*. 2008;168:308-314.

47. Bibbins-Domingo K, Chertow GM, Coxson PG, et al. Projected effect of dietary salt reductions on future cardiovascular disease. *N Engl J Med*. 2010;362:590-599.

48. Dickinson BD, Havas S; for the Council on Science and Public Health, American Medical Association. Reducing the population burden of cardiovascular disease by reducing sodium intake: a report of the Council on Science and Public Health. *Arch Intern Med*. 2007;167:1460-1468.

49. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *BMJ*. 1988;297:319-328.

50. Perry IJ. Salt, science and politics. *J Hum Hypertens*. 2003;17:1-3.

51. Macgregor GA, Sagnella GA, Markandu ND, Singer DRJ, Cappuccio FP. Double-blind study of three sodium intakes and long-term effects of sodium restriction in essential hypertension. *Lancet*. 1989;334:1244-1247.

52. Svetkey LP, Erlinger TP, Vollmer WM, et al. Effect of lifestyle modifications on blood pressure by race, sex, hypertension status, and age. *J Hum Hypertens*. 2005;19:21-31.

53. Vollmer WM, Sacks FM, Ard J, et al; for the DASH–Sodium Collaborative Research Group. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH–Sodium Trial for the DASH–Sodium Trial. *Ann Intern Med*. 2001;135:1019-1028.

54. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? III—Analysis of data from trials of salt reduction. *BMJ*. 1991;302:819-824.

55. Feng JH, 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.

56. Cook NR, Cutler JA, Obarzanek E, et al. Long-term effects of dietary sodium on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP). *BMJ*. 2007;334:885-888.

57. Sacks FM, Svetkey LP, Vollmer WM, et al; for the DASH–Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med*. 2001;344:3-10.

58. Khaw KT, Barrett-Connor E. The association between blood pressure, age, and dietary sodium and potassium: a population study. *Circulation*. 1988;77:53-61.

59. Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomized trials. *J Hum Hypertens*. 2003;17:471-480.

60. Cook NR, Obarzanek E, Cutler JA, et al; for the Trials of Hypertension Prevention Collaborative Research Group. Joint effects of sodium and potassium intake on subsequent cardiovascular disease: the trials of hypertension prevention follow-up study. *Arch Intern Med*. 2009;169:32-40.

61. Xin X, He J, Frontini MG, Ogden LG, Motsami OI, Whelton PK. Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trial. *Hypertension*. 2001;38:1112-1117.

62. Klatsky AL. Alcohol and cardiovascular health. *Physiol Behav*. 2010;100:76-81.

63. Sesso HD, Cook NR, Buring JE, Manson JE, Gaziano JM. Alcohol consumption and the risk of hypertension in women and men. *Hypertension*. 2008;51:1080-1087.

64. Stranges S, Wu T, Dorn JM, et al. Relationship of alcohol drinking pattern to risk of hypertension: a population-based study. *Hypertension*. 2004;44:813-819.

65. Kodavali L, Townsend RR. Alcohol and its relationship to blood pressure. *Curr Hypertens Rep*. 2006;8:338-344.

66. Fuchs FD, Chambless LE, Whelton PK, Nieto FJ, Heiss G. Alcohol consumption and the incidence of hypertension: the atherosclerosis risk in communities study. *Hypertension*. 2001;37:1242-1250.

67. Puddey IB, Beilin LJ. Alcohol is bad for blood pressure. *Clin Exp Pharmacol Physiol*. 2006;33:847-852.

68. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. Exercise and hypertension: American College of Sports Medicine Position Stand. *Med Sci Sports Exerc*. 2004;36:533-552.

69. Physical Activity Guidelines Advisory Committee. *Physical Activity Guidelines Advisory Committee Report, 2008*. Washington, DC: US Department of Health and Human Services; 2008.

70. Fagard RH. Effects of exercise, diet and their combination on blood pressure. *J Hum Hypertens*. 2005;19:S20-S24.

71. Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med*. 2002;136:493-503.

72. Cornelissen VA, Fagard RH. Effects of endurance training on blood pressure, blood pressure: regulating mechanisms, and cardiovascular risk factors. *Hypertension*. 2005;46:667-675.

73. Cornelissen VA, Verheyden B, Aubert AE, Fagard RH. Effects of aerobic training intensity on resting, exercise and post-exercise blood pressure, heart rate and heart-rate variability. *J Hum Hypertens*. 2010;24:175-182.

74. Miller ER, Erlinger TP, Young DR, et al. Results of the Diet, Exercise, and Weight Loss Intervention Trial (DEW-IT). *Hypertension*. 2002;40:612-618.

75. Puddey IB, Parker M, Beilin LJ, Vandongen R, Masarei JR. Effects of alcohol and caloric restrictions on blood pressure and serum lipids in overweight men. *Hypertension*.1992;20:533-541.

76. Blumenthal JA, Babyak MA, Hinderliter A, et al. Effects of the DASH diet alone and in combination with exercise and weight loss on blood pressure and cardiovascular biomarkers in men and women with high blood pressure: the ENCORE study. *Arch Intern Med*. 2010;170:126-135.

77. Appel LJ, Champagne CM, Harsha DW, et al; for the Writing Group of the PCRG. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. *JAMA*. 2003;289:2083-2093.

78. Beilin LJ, Burke V, Puddey IB. Letter to the editor. *JAMA*. 2003;290:887.

79. Whelton PK, Appel LJ, Espeland MA, et al; for the TONE Collaborative Research Group. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). *JAMA*. 1998;279:839-846.

80. Desch S, Schmidt J, Kobler D, et al. Effect of cocoa products on blood pressure: systematic review and meta-analysis. *Am J Hypertens*. 2010;23:97-103.

81. Taubert D, Roesen R, Schömig E. Effect of cocoa and tea intake on blood pressure. *Arch Intern Med*. 2007;167:626-634.

82. Taubert D, Roesen R, Lehmann C, et al. Effects of low habitual cocoa intake on blood pressure and bioactive nitric oxide: a randomized controlled trial. *JAMA*. 2007;298:49-60.

83. Sánchez D, Quiñones M, Moulay L, Muguerza B, Miguel M, Aleixandre A. Changes in arterial blood pressure of a soluble cocoa fiber product in spontaneously hypertensive rats. *J Agric Food Chem*. 2010;58:1493-1501.

84. Cienfuegos-Jovellanos E, Quiñones M, Muguerza B, Moulay L, Miguel M, Aleixandre A. Antihypertensive effect of a polyphenol-rich cocoa powder industrially processed to preserve the original flavonoids of the cocoa bean. *J Agric Food Chem*. 2009;57:6156-6162.

85. Yang G, Shu X-O, Jin F, et al. Longitudinal study of soy food intake and blood pressure among middle-aged and elderly Chinese women. *Am J Clin Nutr*. 2005;81:1012-1017.

86. Welty FK, Lee KS, Lew NS, Zhou J-R. Effect of soy nuts on blood pressure and lipid levels in hypertensive, prehypertensive, and normotensive postmenopausal women. *Arch Intern Med*. 2007;167:1060-1067.

87. 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.

88. Burke V, Hodgson JM, Beilin LJ, Giangiulioi N, Rodgers P, Puddey IB. Dietary protein and soluble fiber reduce ambulatory BP in treated hypertensives. *Hypertension.* 2001;38:821-826.

89. Rivas M, Garay RP, Escanero JF, Cia P Jr, Cia P, Alda JO. Soy milk lowers BP in men and women with mild to moderate essential hypertension. *J Nutr.* 2002;132:1900-1902.

90. Appel LJ. The effects of protein intake on BP and cardiovascular disease. *Curr Opin Lipidol.* 2003;14:55-59.

91. Washburn S, Burke GL, Morgan T, Anthony M. Effect of soy protein supplementation on serum lipoproteins, blood pressure, and menopausal symptoms in perimenopausal women. *Menopause.* 1999;6:7-13.

92. Manikonda JP, Störk S, Tögel S, et al. Contemplative meditation reduces ambulatory blood pressure and stress-induced hypertension: a randomized pilot trial. *J Hum Hypertens.* 2008;22:138-140.

93. Dickinson HO, Campbell F, Beyer FR, et al. Relaxation therapies for the management of primary hypertension in adults: a Cochrane review. *J Hum Hypertens.* 2008;22:809-820.

94. Eisenberg DM, Delbanco TL, Berkey CS, et al. Cognitive behavioral techniques for hypertension: are they effective? *Ann Intern Med.* 1993;118:964-972.

95. Jacob RG, Chesney MA, Williams DM, et al. Relaxation therapy for hypertension: design effects and treatment effects. *Ann Behav Med.* 1991;13:5-17.

96. Joseph CN, Porta C, Casucci G, et al. Slow breathing improves arterial baroflex sensitivity and decreases blood pressure in essential hypertension. *Hypertension.* 2005;46:714-718.

97. Rainforth MV, Schneider RH, Nidich SI, Gaylord-King C, Salerno JW, Anderson JW. Stress reduction programs in patients with elevated blood pressure: a systematic review and meta-analysis. *Curr Hypertens Rep.* 2007;9:520-528.