

The New England Journal of Medicine

© Copyright, 2001, by the Massachusetts Medical Society

VOLUME 344

JANUARY 4, 2001

NUMBER 1



EFFECTS ON BLOOD PRESSURE OF REDUCED DIETARY SODIUM AND THE DIETARY APPROACHES TO STOP HYPERTENSION (DASH) DIET

FRANK M. SACKS, M.D., LAURA P. SVETKEY, M.D., WILLIAM M. VOLLMER, PH.D., LAWRENCE J. APPEL, M.D.,
GEORGE A. BRAY, M.D., DAVID HARSHA, PH.D., EVA OBARZANEK, PH.D., PAUL R. CONLIN, M.D.,
EDGAR R. MILLER III, M.D., PH.D., DENISE G. SIMONS-MORTON, M.D., PH.D., NJERI KARANJA, PH.D., AND PAO-HWA LIN, PH.D.,
FOR THE DASH-SODIUM COLLABORATIVE RESEARCH GROUP

ABSTRACT

Background The effect of dietary composition on blood pressure is a subject of public health importance. We studied the effect of different levels of dietary sodium, in conjunction with the Dietary Approaches to Stop Hypertension (DASH) diet, which is rich in vegetables, fruits, and low-fat dairy products, in persons with and in those without hypertension.

Methods A total of 412 participants were randomly assigned to eat either a control diet typical of intake in the United States or the DASH diet. Within the assigned diet, participants ate foods with high, intermediate, and low levels of sodium for 30 consecutive days each, in random order.

Results Reducing the sodium intake from the high to the intermediate level reduced the systolic blood pressure by 2.1 mm Hg ($P < 0.001$) during the control diet and by 1.3 mm Hg ($P = 0.03$) during the DASH diet. Reducing the sodium intake from the intermediate to the low level caused additional reductions of 4.6 mm Hg during the control diet ($P < 0.001$) and 1.7 mm Hg during the DASH diet ($P < 0.01$). The effects of sodium were observed in participants with and in those without hypertension, blacks and those of other races, and women and men. The DASH diet was associated with a significantly lower systolic blood pressure at each sodium level; and the difference was greater with high sodium levels than with low ones. As compared with the control diet with a high sodium level, the DASH diet with a low sodium level led to a mean systolic blood pressure that was 7.1 mm Hg lower in participants without hypertension, and 11.5 mm Hg lower in participants with hypertension.

Conclusions The reduction of sodium intake to levels below the current recommendation of 100 mmol per day and the DASH diet both lower blood pressure substantially, with greater effects in combination than singly. Long-term health benefits will depend on the ability of people to make long-lasting dietary changes and the increased availability of lower-sodium foods. (N Engl J Med 2001;344:3-10.)

Copyright © 2001 Massachusetts Medical Society.

HYPERTENSION affects almost 50 million people in the United States and places them at higher risk for cardiovascular diseases.^{1,2} Furthermore, this risk increases with progressive elevations in blood pressure, beginning at even normal levels of blood pressure.³ The Dietary Approaches to Stop Hypertension (DASH) trial demonstrated that a diet that emphasizes fruits, vegetables, and low-fat dairy products, that includes whole grains, poultry, fish, and nuts, that contains only small amounts of red meat, sweets, and sugar-containing beverages, and that contains decreased amounts of total and saturated fat and cholesterol lowers blood pressure substantially both in people with hypertension and those without hypertension, as compared with a typical diet in the United States.⁴ The DASH diet is now recommended in national guidelines.^{1,5} Clinical trials have shown that reducing the sodium chloride content of typical diets in the United States or northern Europe lowers blood pressure,⁶⁻⁸ and guidelines recommend reducing the daily dietary sodium

From the Endocrine-Hypertension Division and the Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston (F.M.S., P.R.C.); the Duke Hypertension Center and the Sarah W. Stedman Center for Nutritional Studies, Duke University School of Medicine, Durham, N.C. (L.P.S., P.-H.L.); the Kaiser Permanente Center for Health Research, Portland, Ore. (W.M.V., N.K.); the Welch Center for Prevention, Epidemiology, and Clinical Research, Johns Hopkins University, Baltimore (L.J.A., E.R.M.); the Pennington Biomedical Research Center, Baton Rouge, La. (G.A.B., D.H.); and the Division of Epidemiology and Clinical Applications, National Heart, Lung, and Blood Institute, Bethesda, Md. (E.O., D.G.S.-M.). Address reprint requests to Dr. Sacks at the Nutrition Department, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115, or at fsacks@hsph.harvard.edu.

Other authors were Mikel Aickin, Ph.D., Kaiser Permanente Center for Health Research, Portland, Ore.; Marlene M. Most-Windhauser, Ph.D., Pennington Biomedical Research Center, Baton Rouge, La.; Thomas J. Moore, M.D., Merck, West Point, Pa.; and Michael A. Proschan, Ph.D., and Jeffrey A. Cutler, M.D., National Heart, Lung, and Blood Institute, Bethesda, Md.

intake to 100 mmol (equivalent to 2.3 g of sodium or 5.8 g of sodium chloride) or less.¹

We undertook this trial to address several questions relevant to the prevention and treatment of hypertension. Does reducing the level of sodium from the average intake in the United States (approximately 150 mmol per day, which is equivalent to 3.5 g of sodium, or 8.7 g of sodium chloride) to below the currently recommended upper limit of 100 mmol per day lower blood pressure more than reducing the sodium level only to the recommended limit? We hypothesized that it would, on the basis of both the blood-pressure levels in populations with an average consumption of less than 60 mmol of sodium per day⁹ and data from incompletely controlled¹⁰⁻¹⁴ or small-scale¹⁵ clinical trials. Does the DASH diet lower the blood pressure beyond the level achievable by simply reducing sodium intake? What is the combined effect of the DASH diet and reduced sodium intake? The extent to which the reduction of the sodium level, in the context of a typical United States diet and in combination with the DASH diet, lowers blood pressure in people without hypertension is a much-debated⁶⁻⁸ issue critical to the prevention of hypertension.

METHODS

Study Design

The study was a multicenter, randomized trial comparing the effects on blood pressure of three levels of sodium intake in two diets among adults whose blood pressure exceeded 120/80 mm Hg, including those with stage 1 hypertension (a systolic blood pressure of 140 to 159 mm Hg or a diastolic blood pressure of 90 to 95 mm Hg). The design of the trial, which was conducted from September 1997 through November 1999, has been described in detail elsewhere.¹⁶ The three sodium levels were defined as high (a target of 150 mmol per day with an energy intake of 2100 kcal, reflecting typical consumption in the United States¹), intermediate (a target of 100 mmol per day, reflecting the upper limit of the current national recommendations¹), and low (a target of 50 mmol per day, reflecting a level that we hypothesized might produce an additional lowering of blood pressure). The daily sodium intake was proportionate to the total energy requirements of individual participants, so that larger or very active persons would receive more food and therefore more sodium than smaller or less active persons.

The two diets were a control diet⁴ typical of what many people in the United States eat, and the DASH diet, which emphasizes fruits, vegetables, and low-fat dairy foods; includes whole grains, poultry, fish, and nuts; and contains smaller amounts of red meat, sweets, and sugar-containing beverages than the typical diet in the United States.^{4,17} The DASH diet (originally termed the "combination diet"⁴) also contains smaller amounts of total and saturated fat and cholesterol and larger amounts of potassium, calcium, magnesium, dietary fiber, and protein than the typical diet.^{4,17} The nutrient composition of the diets was calculated and monitored with the use of chemical analysis. Specific dietary patterns were composed to achieve the high, intermediate, and low levels of sodium in both the control and the DASH diets. Participants were provided with all of their food, including snacks and cooked meals. Taste tests were performed to ensure that the diets were palatable.

During a two-week run-in period, eligible persons ate the high-sodium control diet. Participants were then randomly assigned to follow one of the two diets according to a parallel-group design. They ate their assigned diet at each of the three sodium levels for 30 consecutive days in random order in a crossover design. Each

participant's energy intake was adjusted to ensure that his or her weight remained constant throughout the study. Each of four clinical centers conducted the trial in four or five cohorts of participants. The primary outcome was systolic blood pressure at the end of each 30-day period of dietary intervention, and the secondary outcome was diastolic blood pressure. The study was approved by the human subjects committees of the centers, and written informed consent was given by each participant.

Criteria for Eligibility

To be eligible participants had to be at least 22 years old and to have an average systolic blood pressure on three screening visits of 120 to 159 mm Hg and an average diastolic blood pressure of 80 to 95 mm Hg. We targeted an enrollment that was 50 percent blacks and 50 percent women. The criteria for exclusion were heart disease, renal insufficiency, poorly controlled hyperlipidemia or diabetes mellitus, diabetes requiring insulin, special dietary requirements, intake of more than 14 alcoholic drinks per week, or the use of antihypertensive drugs or other medications that would affect blood pressure or nutrient metabolism.

Measurements

Blood pressure was measured with random-zero sphygmomanometers while participants were seated at three screening visits,

TABLE 1. BASE-LINE CHARACTERISTICS OF THE PARTICIPANTS.*

CHARACTERISTIC	DASH DIET (N=208)	CONTROL DIET (N=204)
Age (yr)	47±10	49±10
Female sex (%)	59	54
Race or ethnic group (%)		
Black	57	56
Non-Hispanic white	40	40
Asian or other	3	5
Hypertension (%)†	41	41
Blood pressure (mm Hg)‡		
Systolic	134±10	135±10
Diastolic	86±5	86±4
Body-mass index§	29±5	30±5
Waist circumference (cm)	96±12	100±14
Urinary sodium (mmol/day)¶	158±79	152±72
Educational level (%)		
High-school graduate	12	17
Attended college	41	32
College graduate	45	48
Annual income (%)		
<\$30,000	32	34
\$30,000–\$60,000	33	41
>\$60,000	35	25

*Plus-minus values are means ±SD. Because of rounding, not all percentages total 100.

†Hypertension was defined as an average systolic blood pressure of 140 to 159 mm Hg or an average diastolic blood pressure of 90 to 95 mm Hg during the three screening visits.

‡Base-line blood pressure was the average of three screening measurements and two measurements during the run-in period.

§The body-mass index is the weight in kilograms divided by the square of the height in meters.

¶Base-line urinary sodium was determined from a 24-hour urine collection during the screening period, when the participants were eating their customary, self-selected diets. Data were missing for four participants in the DASH-diet group.

twice during the run-in period, weekly during the first 3 weeks of each of the three 30-day intervention periods, and at five clinic visits during the last 9 days (at least two during the final 4 days) of each intervention period. During the screening period and during the last week of each intervention period, a 24-hour urine collection was obtained. The participants and the dietary staff were unaware of the outcome data; the personnel involved in the collection of the outcome data were unaware of participants' diet assignment. We assessed participants' adherence to the diet by reviewing their daily food diaries, having them eat their weekday lunches or dinners on site, and measuring 24-hour urinary excretion of sodium, potassium, phosphorus, and urea nitrogen. Side effects were monitored by means of questionnaires regarding symptoms and illnesses. According to the study protocol, a systolic blood pressure of more than 170 mm Hg or a diastolic blood pressure of more than 105 mm Hg at a single visit was considered to necessitate a second measurement; if the reading was sustained, the participant was referred to his or her physician for further evaluation and treatment.

Statistical Analysis

The analyses were structured according to a two-by-four design to compare the two diets (control and DASH) during the four periods (the run-in period and three intervention periods). The base-line blood pressure used for the analyses was the average of the measurements taken during the screening and run-in periods, and the blood pressure used for the end of each intervention period was the average of the last five measurements. A unified generalized-estimating-equation¹⁸ model with an exchangeable covariance matrix was used for all primary analyses. Blood pressure was the outcome. The base-line blood pressure, the clinical center, and the cohort were represented in the model as fixed effects, whereas the intervention periods were included as random effects to allow for within-person correlation among blood-pressure measurements. The model included indicators of the cohort, the clinical center, and the carryover effect from the previous intervention. Results were similar with and without carryover in the model. Indicators for the subgroups specified in the study protocol (hypertensive status, race, and sex) and for the relevant interactions with the effects of the diet assignments and sodium levels were included in the subgroup analyses.

The linearity of the effects of sodium within the control diet or the DASH diet was assessed by comparing the decrease in blood pressure from the high to the intermediate level of sodium with the decrease from the intermediate to the low level of sodium. Multiple comparisons were accounted for by means of the method of Holm¹⁹;

the resulting adjusted P values could be compared to 0.05 to determine significance.²⁰ The adjusted P values were used for the blood-pressure changes in the total cohort, but not in subgroups, as specified in the study protocol. All analyses were performed according to the intention-to-treat approach; in 22 instances, missing blood-pressure measurements during an intervention period, including those owing to a participant's withdrawal from the study, were replaced by base-line values. The planned sample size of 400 was calculated in order to provide the study with a power of 90 percent to detect a difference in systolic blood pressure of 2.1 mm Hg between sodium levels, and a difference of 3.0 mm Hg between the DASH and control diets at each sodium level.

RESULTS

The base-line characteristics of the participants are shown in Table 1. A total of 95 percent of the participants assigned to the DASH-diet group (198 of 208) and 94 percent of those assigned to the control-diet group (192 of 204) completed the study and provided blood-pressure measurements during each intervention period. Mean urinary sodium levels averaged 142 mmol per day during the high-sodium period, 107 mmol per day during the intermediate-sodium period, and 65 mmol per day during the low-sodium period (Table 2). The levels of urinary potassium, phosphorus, and urea nitrogen (reflective of the intake of fruit and vegetables, dairy products, and protein, respectively) were higher in the DASH-diet group than in the control-diet group, and were nearly identical for all three sodium levels. Weight remained stable, as intended.

The reduction of sodium intake significantly lowered systolic and diastolic blood pressure in a stepwise fashion, with both the control diet and the DASH diet (Fig. 1). The level of dietary sodium had approximately twice as great an effect on blood pressure with the control diet as it did with the DASH diet ($P < 0.001$ for the interaction). There was a greater response of blood pressure to progressively lower levels of sodi-

TABLE 2. URINARY EXCRETION AND BODY WEIGHT ACCORDING TO DIETARY SODIUM LEVEL AND ASSIGNED DIET.

VARIABLE	HIGH SODIUM LEVEL		INTERMEDIATE SODIUM LEVEL		LOW SODIUM LEVEL	
	DASH DIET	CONTROL DIET	DASH DIET	CONTROL DIET	DASH DIET	CONTROL DIET
	mean \pm SD					
Urinary excretion						
Sodium						
mmol/day	144 \pm 58	141 \pm 55	107 \pm 52	106 \pm 44	67 \pm 46	64 \pm 37
g/day	3.3 \pm 1.3	3.3 \pm 1.3	2.5 \pm 1.2	2.4 \pm 1.0	1.5 \pm 1.0	1.5 \pm 0.8
Potassium						
mmol/day	75 \pm 27	40 \pm 14	81 \pm 31	41 \pm 14	81 \pm 29	42 \pm 14
g/day	2.9 \pm 1.1	1.6 \pm 0.5	3.2 \pm 1.2	1.6 \pm 0.5	3.2 \pm 1.1	1.6 \pm 0.5
Phosphorus (mg/day)	778 \pm 285	666 \pm 248	825 \pm 350	646 \pm 264	783 \pm 286	672 \pm 243
Urea nitrogen (g/day)	11.5 \pm 4.0	9.6 \pm 3.2	12.4 \pm 4.5	9.7 \pm 3.4	11.8 \pm 4.1	10.0 \pm 3.3
Creatinine (g/day)	1.4 \pm 0.5	1.5 \pm 0.5	1.5 \pm 0.6	1.5 \pm 0.6	1.4 \pm 0.5	1.6 \pm 0.6
Weight (kg)	82.3 \pm 14.5	85.3 \pm 15.6	82.1 \pm 14.4	85.1 \pm 16.0	82.2 \pm 14.5	85.0 \pm 15.7

um intake. In the control diet, a reduction in the sodium intake of about 40 mmol per day from the intermediate sodium level lowered blood pressure more than a similar reduction in the sodium intake from the high level ($P=0.03$ for systolic blood pressure, $P=0.045$ for diastolic blood pressure).

The DASH diet, as compared with the control diet, resulted in a significantly lower systolic blood pressure at every sodium level and in a significantly lower diastolic blood pressure at the high and intermediate sodium levels (Fig. 1). It had a larger effect on both systolic and diastolic blood pressure at high sodium levels than it did at low ones ($P<0.001$ for the interaction).

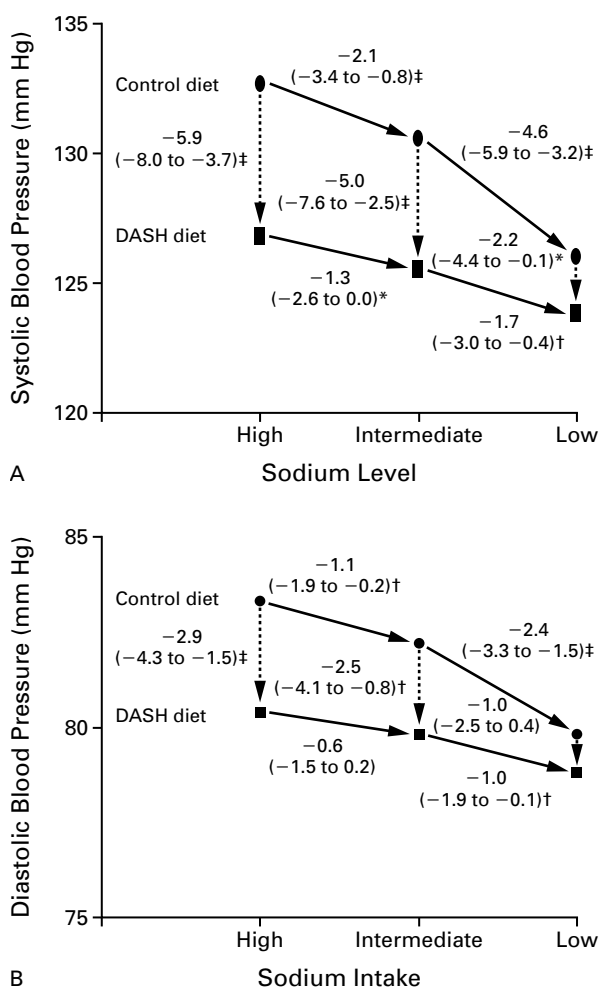
As compared with the high-sodium control diet, the low-sodium DASH diet produced greater reductions in systolic and diastolic blood pressure than either the DASH diet alone or a reduction in sodium alone (Fig. 1). The reductions in blood pressure caused by the combination of dietary interventions were smaller than they would have been if the effects of each dietary intervention were strictly additive ($P<0.001$ for the interaction).

Reducing the sodium intake from the high to the low level, with either the control diet or the DASH diet, reduced systolic blood pressure in participants with and in those without hypertension (among blacks as well as among participants of other races or ethnic

groups), and in men and women (Fig. 2). The effects of sodium were greater in participants with hypertension than in those without hypertension (interaction, $P=0.01$ on the control diet; $P=0.003$ on the DASH diet), in blacks on the control diet than in participants of other races or ethnic groups on that diet ($P=0.007$), and in women on the DASH diet than in men on that diet ($P=0.04$). As compared with the combination of the control diet and a high level of sodium, the combination of the DASH diet and a low level of sodium lowered systolic blood pressure by 11.5 mm Hg in participants with hypertension (12.6 mm Hg for blacks; 9.5 mm Hg for others), by 7.1 mm Hg in participants without hypertension (7.2 mm Hg for blacks; 6.9 mm Hg for others), and by 6.8 mm Hg in men and 10.5 mm Hg in women ($P<0.001$ in all subgroups). The combination of the two dietary interventions lowered systolic blood pressure more in participants with hypertension than in those without hypertension ($P=0.004$), and more in women than in men ($P=0.02$).

Figure 1. The Effect on Systolic Blood Pressure (Panel A) and Diastolic Blood Pressure (Panel B) of Reduced Sodium Intake and the DASH Diet.

The mean systolic and diastolic blood pressures are shown for the high-sodium control diet. The mean changes in blood pressure are shown for various sodium levels (solid lines), and the mean differences between the two diets at each level of sodium intake are shown. Unidirectional arrows are used for simplicity, although the order in which participants were given the sodium levels was random with a crossover design. The numbers next to the dotted lines connecting the data points are the mean changes in blood pressure. The 95 percent confidence intervals are given in parentheses. There was a significant difference in systolic blood pressure between the high-sodium and low-sodium phases of the control diet (mean, -6.7 mm Hg; 95 percent confidence interval, -5.4 to -8.0 ; $P<0.001$) and the DASH diet (mean, -3.0 mm Hg; 95 percent confidence interval, -1.7 to -4.3 ; $P<0.001$) and between the high-sodium phase of the control diet and the low-sodium phase of the DASH diet (mean, -8.9 mm Hg; 95 percent confidence interval, -6.7 to -11.1 ; $P<0.001$). There was also a significant difference in diastolic blood pressure between the high-sodium and low-sodium phases of the control diet (mean, -3.5 mm Hg; 95 percent confidence interval, -2.6 to -4.3 ; $P<0.001$) and of the DASH diet (mean, -1.6 mm Hg; 95 percent confidence interval, -0.8 to -2.5 ; $P<0.001$) and between the high-sodium phase of the control diet and the low-sodium phase of the DASH diet (mean, -4.5 mm Hg; 95 percent confidence interval, -3.1 to -5.9 ; $P<0.001$). Asterisks ($P<0.05$), daggers ($P<0.01$), and double daggers ($P<0.001$) indicate significant differences in blood pressure between groups or between dietary sodium categories.



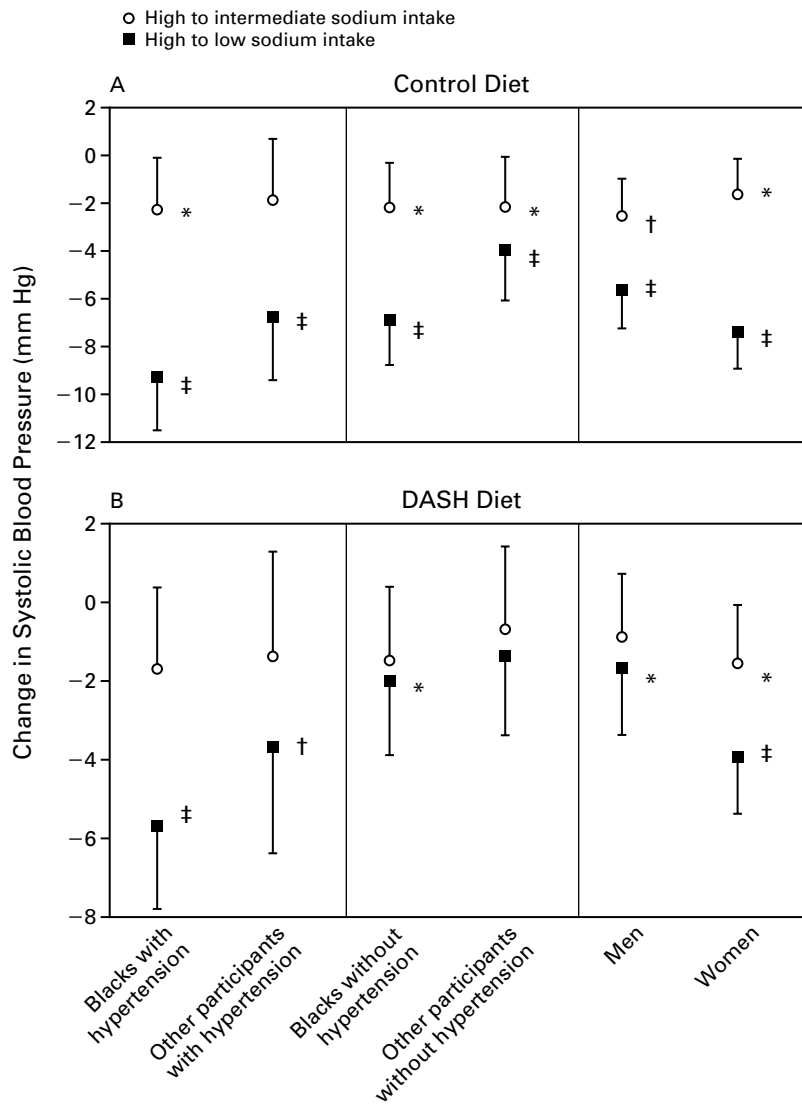


Figure 2. The Effect on Systolic Blood Pressure of Dietary Sodium Intake during the Control Diet (Panel A) and the DASH Diet (Panel B), According to Subgroup.

The error bars represent the 95 percent confidence limits of the changes in blood pressure for each subgroup. Hypertension was defined as an average systolic blood pressure of 140 to 159 mm Hg or an average diastolic blood pressure of 90 to 95 mm Hg during the three screening visits. The “other” category of race and ethnic group is composed primarily of non-Hispanic whites (see Table 1). Asterisks ($P \leq 0.05$), daggers ($P < 0.01$), and double daggers ($P < 0.001$) indicate significant differences between levels of sodium intake.

A systolic blood pressure of more than 170 mm Hg or a diastolic blood pressure of more than 105 mm Hg occurred in 36 participants in the control-diet group and in 7 in the DASH-diet group; in 18 participants during the period of high sodium intake, 22 during intermediate sodium intake, and 3 during low sodium intake; and in no participant during the low-sodium phase of the DASH diet. None of these participants reached the predefined threshold for sustained elevat-

ed blood pressure¹⁶ that necessitated referral for anti-hypertensive pharmacologic therapy. The participants tended to report fewer symptoms during periods of reduced sodium intake. Headache was reported at least once by 47 percent of the participants during the high-sodium phase of the control diet, by 39 percent during the low-sodium phase of the control diet, and by 36 percent during the low-sodium phase of the DASH diet ($P < 0.05$ for both comparisons with the high-

sodium phase of the control diet). The number of participants who did not complete an intervention period was similar during all three sodium levels (seven during the high-sodium phase, seven during the intermediate-sodium phase, and eight during the low-sodium phase).

DISCUSSION

This trial produced several key findings that are important for the prevention and treatment of hypertension. First, the DASH diet lowered blood pressure at high, intermediate, and low levels of sodium intake, confirming and extending the findings of the previous DASH study.⁴ Thus, the benefits of following the DASH diet have now been shown to apply throughout the range of sodium intakes, including those recommended for the prevention and treatment of hypertension. Second, blood pressure can be lowered in the consumers of either a diet that is typical in the United States or the DASH diet by reducing the sodium intake from approximately 140 mmol per day (an average level in the United States) to an intermediate level of approximately 100 mmol per day (the currently recommended upper limit¹), or from this level to a still lower level of 65 mmol per day. Moreover, reducing the sodium intake by approximately 40 mmol per day caused a greater decrease in blood pressure when the starting sodium intake was already at the recommended level than when it was at a higher level similar to the average in the United States. These results provide a scientific basis for a lower goal for dietary sodium than the level currently recommended.

Third, the combined effects on blood pressure of a low sodium intake and the DASH diet were greater than the effects of either intervention alone and were substantial. In participants with hypertension, the effects were equal to or greater than those of single-drug therapy.^{21,22} The combined effects were not as great as would be estimated on the basis of strict additivity, perhaps because low levels of sodium attenuated the hypotensive effects of potassium in the DASH diet^{23,24} or because the high potassium or calcium content of the DASH diet attenuated the effects of low levels of sodium.²⁵⁻²⁷ Nevertheless, the combination of the two interventions achieved the greatest effect on blood pressure, and therefore, both — not just one or the other — merit recommendation. The DASH diet and the low sodium level were well tolerated, with no increase in symptoms or dropouts. However, long-term health benefits remain to be demonstrated and will depend on the ability of people to make long-lasting dietary changes, including the consistent choice of lower-sodium foods.

We found that the reduction of dietary sodium significantly lowered the blood pressure of persons without hypertension who were eating a diet that is typical in the United States. These results should settle the controversy over whether the reduction of sodium

has a worthwhile effect on blood pressure in persons without hypertension. This controversy stemmed in part from the apparently divergent results and interpretations of individual trials and meta-analyses.⁶⁻⁸ Because of differences in the designs, quality, and study populations of the trials and the subjectivity involved in judgments about which studies to include in meta-analyses, a single, large, well-controlled trial with a diverse population provides the most reliable estimates of the effects of treatments.

In our study the dietary intake was controlled and the influences of behavioral factors, programs of dietary education, and varying degrees of adherence to the diets were minimized, so that we measured only true biologic effects. This method offers the optimal approach for determining the effects of diet on blood pressure. The variation in the results in persons without hypertension among previous trials and meta-analyses were probably caused in large part by variable adherence to the prescribed reduction in sodium, inadequate trial design, small samples, or limitations in analysis and presentation, rather than by the lack of a biologic effect of sodium on blood pressure.

We found that the level of dietary sodium and assignment to the control or the DASH diet each had a substantial effect on the blood pressure of blacks, confirming previous findings.²⁸⁻³³ Blacks have a higher rate of hypertension and the resulting cardiovascular disease than other racial and ethnic groups in the United States. We speculate that a greater sensitivity to the deleterious effects of diet could contribute to the high prevalence of hypertension in blacks. These findings justify the intensification of public health and therapeutic efforts to induce dietary change among blacks.

The attainment of a lower sodium level in the population as a whole presents challenges, since sodium is widely prevalent in the food supply, and since most of the daily sodium intake comes from salt in processed foods rather than from table salt.³⁴ The first report on U.S. dietary goals by the Senate Select Committee for Nutrition and Human Needs recommended a goal of 3 g of sodium chloride per day (52 mmol of sodium),³⁵ but concern about the feasibility of achieving this goal led to an increase of the goal to 5 g of sodium chloride.³⁶ Hence, efforts to reduce sodium intake must ultimately rely both on consumers' selection of low-sodium foods and, perhaps more important, on the increased availability of low-sodium products.

Our results should be applicable to most people in the United States. Approximately 50 percent of the adult population of the United States and 80 percent of those 50 years of age or older have a blood pressure of at least 120/80 mm Hg,³⁷ which is the upper limit of optimal blood pressure¹ and which was the lower limit of the eligibility requirements for blood pressure for our trial. Furthermore, epidemiologic studies suggest that diets low in sodium and high in potassium

blunt the rise in blood pressure that normally occurs with age.⁹ The intervention periods in our trial were, of necessity, brief — just 30 days. Still, the effect of the reduction in dietary sodium on blood pressure tends to persist over time to the extent that adherence to the lower-sodium diet is maintained.^{7,15,38} In conclusion, our results provide support for a more aggressive target for reduced sodium intake, in combination with use of the DASH diet, for the prevention and treatment of elevated blood-pressure levels.

Supported by cooperative agreements and grants from the National Heart, Lung, and Blood Institute (U01-HL57173, to Brigham and Women's Hospital; U01-HL57114, to Duke University; U01-HL57190, to Pennington Biomedical Research Institute; U01-HL57139 and K08 HL03857-01, to Johns Hopkins University; and U01-HL57156, to Kaiser Permanente Center for Health Research) and by the General Clinical Research Center Program of the National Center for Research Resources (M01-RR02635, to Brigham and Women's Hospital, and M01-RR00722, to Johns Hopkins University).

We are indebted to the study participants for their sustained commitment to the DASH–Sodium Trial; to the Almond Board of California, Beatrice Foods, Bestfoods, Cabot Creamery, C.B. Foods, Dannon, Diamond Crystal Specialty Foods, Elwood International, Hershey Foods, Hormel Foods, Kellogg, Lipton, McCormick, Nabisco U.S. Foods Group, Procter & Gamble, Quaker Oats, and Sun-Maid Growers for donating food; to Frost Cold Storage for food storage; to the members of the external Protocol Review Committee — Janice A. Derr, Ph.D., Richard D. Mattes, Ph.D., Lemuel A. Moye, M.D., Ph.D., Jeremiah Stamler, M.D. (chair), and Jackson T. Wright, M.D., Ph.D.; and to the members of the Data and Safety Monitoring Board — Avital Cnaan, Ph.D., Janice A. Derr, Ph.D., Richard Grimm, M.D. (chair), Richard D. Mattes, Ph.D., Jeremiah Stamler, M.D., and Jackson T. Wright, M.D., Ph.D.

APPENDIX

In addition to the authors, other members of the DASH–Sodium Collaborative Research Group include the following: *Brigham and Women's Hospital and Harvard Medical School, Boston (clinical center)* — N. Alexander, J. Belmonte, F. Bodden, L. Cashman, B. Cox, J. Dyer, A. Ghosh, J. Hackett, E. Hamilton, T. Holiday, J. Karimbakis, C. Larson, M. McCullough, D. McDonald, P. McVinnay, D. Moeller, P. Morris, M. Myrie, K. Osborn, E. Penachio, S. Redican, J. Sales, J. Swain, Z. Than, K. Weed; *Duke University Medical Center, Durham, N.C. (clinical center)* — J. Abbott, K. Aicher, J. Ard, J. Baughman, M. Baughman, B. Brown, A. Bohannon, B. Graves, K. Hoben, J. Huang, L. Johnson-Pruden, T. Phelps, C. Plaisted, L. Reams, P. Reams, T. Ross, F. Rukenbrod, E. Ward; *Johns Hopkins University, Baltimore (clinical center)* — J. Abshere, D. Bengough, L. Bohlman, J. Charleston, L. Clement, C. Dahne, F. Dennis, S. Dobry, K. Eldridge, T. Erlinger, A. Fouts, C. Harris, B. Horseman, M. Jehn, S. Kritt, J. Lambert, E. Levitas, P. McCarron, N. Muhammad, M. Nagy, B. Peterson, D. Rhodes, V. Shank, T. Shields, T. Stanger, A. Thomas, E. Thomas, L. Thomas, R. Weiss, E. Wilke, W. Wong; *Kaiser Permanente Center for Health Research, Portland, Oreg. (coordinating center)* — M. Allison, S. Baxter, N. Becker, S. Craddick, B. Doster, C. Eddy, D. Ernst, A. Garrison, S. Gillespie, R. Gould, T. Haswell, L. Haworth, F. Heinitz, M. Hornbrook, K. Kirk, P.A. LaChance, R. Laws, M. Leitch, W.R. Li, L. Massinger, M. McMurray, G. Meltesen, G. Miranda, S. Mitchell, N. Redmond, J. Reinhardt, J. Rice, P. Runk, R. Schuler, C. Souvanlasy, M. Succ, T. Vogt; *National Heart, Lung, and Blood Institute, Bethesda, Md. (sponsor)* — C. Brown, M. Evans; *Pennington Biomedical Research Center, Baton Rouge, La. (clinical center)* — C. Champagne, S. Crawford, F. Greenway, J. Ihrig, B. Kennedy, J. Perault, D. Sanford, A. Sawyer, S. Smith, R. Tulley, J. Vaidyanathan; *Virginia Polytechnic Institute, Blacksburg, Va. (food-analysis coordinating center)* — K. Phillips; *Washington University School of Medicine, St. Louis (core laboratory)* — T. Cole.

REFERENCES

1. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1997;157:2413-46. [Erratum, *Arch Intern Med* 1998;158:573.]

2. National High Blood Pressure Education Program Working Group report on primary prevention of hypertension. *Arch Intern Med* 1993;153:186-208.
3. Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks: US population data. *Arch Intern Med* 1993;153:598-615.
4. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med* 1997;336:1117-24.
5. Facts about the DASH diet. Bethesda, Md.: National Heart, Lung, and Blood Institute, 1998. (NIH publication no. 98-4082.)
6. Cutler JA, Follmann D, Allender PS. Randomized trials of sodium reduction: an overview. *Am J Clin Nutr* 1997;65:Suppl:643S-651S.
7. 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;302:819-24. [Erratum, *BMJ* 1991;302:939.]
8. Graudal NA, Galloe AM, Garred P. Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride: a meta-analysis. *JAMA* 1998;279:1383-91.
9. 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;297:319-28.
10. Hatch FT, Wertheim AR, Eurman GH, Watkin DM, Froeb HF, Epstein HA. Effects of diet in essential hypertension. III. Alterations in sodium chloride, protein and fat intake. *Am J Med* 1954;17:499-513.
11. Chapman CB, Gibbons T, Henschel A. The effect of the rice-fruits diet on the composition of the body. *N Engl J Med* 1950;243:899-905.
12. Parfrey PS, Markandu ND, Roulston JE, Jones BE, Jones JC, MacGregor GA. Relation between arterial pressure, dietary sodium intake, and renin system in essential hypertension. *BMJ* 1981;283:94-7.
13. Gill JR, Gullner HG, Lake CR, Lakatua DJ, Lan G. Plasma and urinary catecholamines in salt-sensitive idiopathic hypertension. *Hypertension* 1988;11:312-9.
14. Kawasaki T, Delea CS, Bartter FC, Smith H. The effect of high-sodium and low-sodium intakes on blood pressure and other related variables in human subjects with idiopathic hypertension. *Am J Med* 1978;64:193-8.
15. MacGregor GA, Markandu ND, Sagnella GA, Singer DR, Cappuccio FP. Double-blind study of three sodium intakes and long-term effects of sodium restriction in essential hypertension. *Lancet* 1989;2:1244-7.
16. Svetkey LP, Sacks FM, Obarzanek E, et al. The DASH diet, sodium intake and blood pressure trial (DASH-sodium): rationale and design. *J Am Diet Assoc* 1999;99:Suppl:S96-S104.
17. Karanja NM, Obarzanek E, Lin PH, et al. Descriptive characteristics of the dietary patterns used in the Dietary Approaches to Stop Hypertension Trial. *J Am Diet Assoc* 1999;99:Suppl:S19-S27.
18. Liang K-Y, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika* 1986;73:13-22.
19. Holm S. A simple sequentially rejective multiple test procedure. *Scand J Stat* 1979;6:65-70.
20. Aicikin M, Gensler H. Adjusting for multiple testing when reporting research results: Bonferroni vs Holm methods. *Am J Public Health* 1996;86:726-8.
21. Materson BJ, Reda DJ, Cushman WC, et al. Single-drug therapy for hypertension in men: a comparison of six antihypertensive agents with placebo. *N Engl J Med* 1993;328:914-21. [Erratum, *N Engl J Med* 1994;330:1689.]
22. 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;151:1413-23.
23. Grimm RH Jr, 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;322:569-74.
24. Whelton PK, He J, Cutler JA, et al. The effects of oral potassium on blood pressure: meta-analysis of randomized controlled clinical trials. *JAMA* 1997;277:1624-32.
25. Krishna GG, Miller E, Kapoor S. Increased blood pressure during potassium depletion in normotensive men. *N Engl J Med* 1989;320:1177-82.
26. Saito K, Sano H, Furuta Y, Fukuzaki H. Effect of oral calcium on blood pressure response in salt-loaded borderline hypertensive patients. *Hypertension* 1989;13:219-26.
27. Rich GM, McCullough M, Olmedo A, Malarick C, Moore TJ. Blood pressure and renal blood flow responses to dietary calcium and sodium intake in humans. *Am J Hypertens* 1991;4:642S-645S.
28. Luft FC, Grim CE, Fineberg N, Weinberger MC. Effects of volume expansion and contraction in normotensive whites, blacks, and subjects of different ages. *Circulation* 1979;59:643-50.
29. Miller JZ, Weinberger MH, Daugherty SA, Fineberg NS, Christian JC, Grim CE. Heterogeneity of blood pressure response to dietary sodium restriction in normotensive adults. *J Chronic Dis* 1987;40:245-50.
30. Voors AW, Dalferes ER Jr, Frank GC, Aristimuno GG, Berenson GS.

Relation between ingested potassium and sodium balance in young blacks and whites. *Am J Clin Nutr* 1983;37:583-94.

31. Zemel MB, Gualdoni SM, Sowers JR. Sodium excretion and plasma renin activity in normotensive and hypertensive black adults as affected by dietary calcium and sodium. *J Hypertens* 1986;4:Suppl 6:S343-S345.
32. Flack JM, Ensrud KE, Mascioli S, et al. Racial and ethnic modifiers of the salt-blood pressure response. *Hypertension* 1991;17:Suppl:1-115-1-121.
33. 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;159:285-93.
34. Mattes RD, Donnelly D. Relative contributions of dietary sodium sources. *J Am Coll Nutr* 1991;10:383-93.
35. Select Committee on Nutrition and Human Needs, U.S. Senate. Dietary goals for the United States. Washington, D.C.: Government Printing Office, February 1977.
36. *Idem*. Dietary goals for the United States. 2nd ed. Washington, D.C.: Government Printing Office, December 1977.
37. 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;25:305-13.
38. Kumanyika SK, Hebert PR, Cutler JA, et al. Feasibility and efficacy of sodium reduction in the Trials of Hypertension Prevention, phase I. *Hypertension* 1993;22:502-12.

Copyright © 2001 Massachusetts Medical Society.