Blood Pressure and Nutrient Intake in the United States

David A. McCarron, Cynthia D. Morris
Holly J. Henry, John L. Stanton

The relation between diet and cardiovascular disease has been the focus of substantial investigative effort for several decades (*1*). The observed reduction in cardiovascular death rates in the United States over the past 30 years has been attributed, in part, to changing dietary patterns (*1, 2*). Nevertheless, hypertensive cardiovascular disease remains the principal cause of morbidity and mortality in America (*1*). Life-style factors, particularly dietary patterns (*3*), have been implicated as major contributors to the continued prevalence of high blood pressure in the United States.

Summary. A data base of the National Center for Health Statistics, Health and Nutrition Examination Survey I (HANES I), was used to perform a computer-assisted, comprehensive analysis of the relation of 17 nutrients to the blood pressure profile of adult Americans. Subjects were 10,372 individuals, 18 to 74 years of age, who denied a history of hypertension and intentional modification of their diet. Significant decreases in the consumption of calcium, potassium, vitamin A, and vitamin C were identified as the nutritional factors that distinguished hypertensive from normotensive subjects. Lower calcium intake was the most consistent factor in hypertensive individuals. Across the population, higher intakes of calcium, potassium, and sodium were associated with lower mean systolic blood pressure and lower absolute risk of hypertension. Increments of dietary calcium were also negatively correlated with body mass. Even though these correlations cannot be accepted as proof of causation, they have implications for future studies of the association of nutritional factors and dietary patterns with hypertension in America.

The analysis of epidemiologic data presented in this article was prompted by three facts. First, a large data base of the National Center for Health Statistics, Health and Nutrition Examination Survey I (HANES I) (*4*), has not been analyzed for possible associations between the demographics of hypertension in the United States and exposure to all relevant nutrients in the diet. Second, the hypothesized relation of nutrients such as sodium (*5, 6*), potassium (*7, 8*), cholesterol (*9, 10*), and calcium (*11, 12*) to the development of hypertensive cardiovascular disease has yet to be confirmed in the United States. Third, specific nutrients, particularly the mineral ions, serve fundamental functions in the regulation of both cardiac output and peripheral resistance, the principal determinants of blood pressure in humans. Utilizing HANES I, we sought to address these issues, recognizing that the results do not prove causality, but do provide valuable insight for future studies of the pathogenesis and treatment of hypertensive cardiovascular disease.

Sample Composition

HANES I collected measures of health and nutrition obtained from interviews and examinations of 20,749 persons (*4*). The sample was scientifically designed to be representative of the U.S. civilian noninstitutionalized population 1 to 74 years of age.

Data tapes from HANES I were obtained from the National Center for Health Statistics. We used tape 4704 (24-hour food consumption), tape 4701 (version 2, dietary frequency and adequacy), tape 4233 (medical examination), and tape 4111 (anthropometry, goniometry, skeletal age, bone density, and cortical thickness) for the analysis. HANES I data on nutrient intake were analyzed with guidelines provided by Adams (*13*) and Pennington and Church (*14*).

Data on blood pressure and nutrient consumption were obtained in 20,749 persons for a 24-hour period. To form the cohort used for our analysis, all individuals under age 18 were excluded, leaving 13,671 persons. Subsequently, the following questions from the medical history questionnaire were used to exclude pregnant women and people with a history of hypertension:

1) "During the past 6 months, have you used any medicine, drugs, or pills for . . . high blood pressure?"

2) "Are you on a special diet? Low salt?"

3) "Are you pregnant now?"

Individuals answering affirmatively to any of these questions were excluded from the analysis, leaving 10,419 persons. In addition, 47 individuals for which data on one of the 17 nutrients were missing were eliminated, leaving 10,372 subjects for the analysis.

In the course of our analysis, we used three definitions to form hypertension groups: (i) systolic pressure of 140 mmHG or above, (ii) systolic pressure of 160 mmHg or above, and (iii) the upper 10 percent of systolic pressures in age-, sex-, and race-specific subgroups. Twenty-four-hour nutrient consumption was analyzed for calories, protein, fat, carbohydrates, calcium, phosphorus, iron, sodium, potassium, vitamin A, thiamin, riboflavin, preformed niacin, vitamin C, saturated fats, oleic acid, linoleic acid, and cholesterol. Average nutrient intake was calculated for each blood pressure group; for subgroups based on age, race, sex, and body mass index; and for alcohol consumption patterns by using Statistical Programs for the Social Sciences (SPSS). Additional SPSS programs for regression analysis, analysis of variance, and multivariate discriminant analysis were used (*15*).

The demographic composition of the 10,372 subjects is shown in Table 1. The age, race, and sex distribution was similar to the overall composition of the HANES I population (*16*). There was a slight overrepresentation of women because there were more women volunteers than men.

David A. McCarron is an associate professor of medicine and is director of the Oregon Hypertension Program, Cynthia D. Morris is a research epidemiologist, and Holly J. Henry is a research associate in the Division of Nephrology and Hypertension, Oregon Health Sciences University, Portland 97201. John L. Stanton is an associate professor of marketing at the Institute for Research in Food Consumption and Nutrition, Temple University, Philadelphia, Pennsylvania 19122. Address requests for reprints to D.A.M.

Nutrient and Caloric Intakes Among Groups: Results

Table 2 shows mean intakes of 11 nutrients and of total calories for several blood pressure groups. Percentage differences in nutrient intake between groups are also listed. Except as noted, intakes were less in hypertensives.

When the definition of hypertension as a systolic blood pressure of 160 mmHg or above was applied (Table 2), 9.2 percent of the population was classified as hypertensive. Reported caloric intake was 15.8 percent lower in individuals who met the criterion for high blood pressure. Only intakes of calcium and linoleic acid were lower (19.6 and 21.2 percent, respectively) in the hypertensives when compared to their 15.8 percent reduction in total caloric intake. A similar pattern was observed when the more liberal definition of hypertension (systolic blood pressure of at least 140 mmHg) was applied. Multivariate discriminant analysis showed calcium to be the nutrient whose intake was most predictive of hypertension (systolic pressure of at least 160 mmHg) after controlling for age, race, and sex. In addition, controlling for alcohol consumption patterns (number of drinks per week) did not change the observed relation between lower calcium intake and higher blood pressures. Carbohydrates, vitamin C, and linoleic acid were the nutrients that entered the multivariate analysis after calcium.

The second comparison of nutrient intake in Table 2 is based on the definition of hypertension as the upper 10 percent of systolic blood pressures for the following age groups: 18 to 24, 25 to 34, 35 to 44, 45 to 54, 55 to 64, and 65 to 74 years. This definition adjusts for the

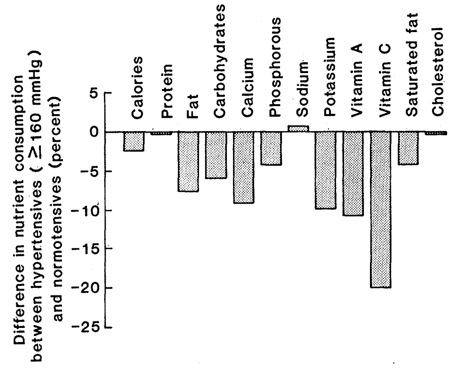


Fig. 1. Standardized mean differences in nutrient intakes between hypertensives (≥ 160 mmHg) and normotensives.

increase in blood pressure with age in our society (*17*) and for those individuals at greatest risk for cardiovascular complications. Intakes of calcium, potassium, vitamin A, and vitamin C were all reduced (7.6, 7.1, 11.8, and 11.9 percent, respectively) in subjects above the 90th percentile. Linoleic acid, fat, and carbohydrate were eliminated as nutrients that were reduced in the hypertensives.

Because not only age but also sex and race are correlates of blood pressure in the United States (*18*), we defined hypertension as the upper 10 percent of individuals, based on systolic blood pressure, for their respective age, race, and sex group. This analysis ensures that identified differences in nutrient intake are not simply related to demographic characteristics. After inclusion of age, race, and sex, it was found that potassium, calcium, vitamin A, and vitamin C were the nutrients whose intakes were significantly lower in hypertensive subjects, as determined by partial F tests ($P < 0.001$ for potassium and vitamins A and C and $P < 0.003$ for calcium).

With the epidemiologic technique of direct rate standardization, we standardized means for each nutrient for age, race, and sex, using the entire subgroup as the reference population. Figure 1 depicts the standardized differences on the basis of the 160-mmHg cutoff. By this calculation, calcium, potassium, vitamin A, and vitamin C are the four nutrients anticipated to be reduced the most in individuals with high blood pressure.

Body mass index (kilograms per square meter) is a strong correlate of blood pressure (*19*). Consequently, this variable was added to age, race, and sex. Intakes of calcium, potassium, vitamin A, and vitamin C were all significantly less ($P < 0.001$, partial F test) in the hypertensive subjects with this final adjustment. Figure 2A depicts total calories for the various blood pressure and body mass index groups. Caloric intake in both lean and obese hypertensive individuals was less than in the corresponding normotensive groups. Figure 2B depicts the values for calcium intake for the normotensive and hypertensive body mass index groups. Two points are evident. First, none of the hypertensive subgroups had a mean intake of calcium equal to the current recommendation of the National Academy of Sciences (800 mg/day) (*20*). Second, higher intake of calcium was negatively correlated with body mass index ($R = -0.588$). Calcium intake decreased with increasing body mass index.

Potassium intake (Fig. 2C) showed a pattern similar to that of calcium, but sodium intake (Fig. 2D) did not differ on the basis of blood pressure or body mass index. Neither cholesterol consumption (Fig. 2E) nor phosphorus intake (Fig. 2F) were consistently different among the

Table 1. Age, race, and sex distribution of subjects under various definitions of high blood pressure.

Subjects	Total		< 160 mmHg		≥ 160 mmHg		Lower 90th percentile		Upper 10th percentile	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
18 to 24	2,066	20	2,054	99.4	12	0.6	1,895	92	171	8
25 to 34	2,322	22	2,297	98.9	25	1.1	2,117	91	205	9
35 to 44	1,933	19	1,871	96.8	62	3.2	1,758	91	175	9
45 to 54	1,216	12	1,095	90	121	10	1,095	90	121	10
55 to 64	838	8	705	84	133	16	762	91	76	9
65 to 74	1,997	19	1,472	74	525	26	1,783	89	214	11
Total	10,372		9,494	92	878	8	9,410	91	962	9
					Race					
White	8,645	83	7,997	84	648	74	7,856	84	789	82
Nonwhite	1,727	17	1,497	16	230	26	1,554	16	173	18
					Sex					
Male	4,170	40	3,742	39	428	49	3,776	40	394	41
Female	6,202	60	5,752	61	450	51	5,634	60	568	59

Table 2. Average nutrient intakes in hypertensives and normotensives. Values for nutrients are means ± standard deviations.

Group	Calories (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Calcium (mg)	Phosphorus (mg)	Sodium (mg)	Potassium (mg)	Vitamin A (IU)	Cholesterol (mg)	Vitamin C (mg)	Linoleic acid (g)
Normotensive (< 160 mmHg)	1908 ± 940	76 ± 41	79 ± 47	210 ± 107	735 ± 557	1172 ± 648	2175 ± 1414	2230 ± 1200	4894 ± 8011	381 ± 299	84 ± 90	8.5 ± 7.6
Hypertensive (≥ 160 mmHg)	1606 ± 737	65 ± 34	66 ± 38	176 ± 87	591 ± 435	985 ± 506	1916 ± 1158	1902 ± 1012	4723 ± 7462	360 ± 280	75 ± 79	6.7 ± 5.7
Difference	15.8%	14.5%	16.5%	16.2%	19.6%	16.0%	11.9%	14.7%	3.5%	5.5%	10.7%	21.2%
Adjusted for age												
Normotensive (lower 90th percentile)	1884 ± 928	75 ± 41	78 ± 47	208 ± 106	728 ± 550	1160 ± 641	2156 ± 1393	2218 ± 1190	4938 ± 8089	379 ± 297	84 ± 90	8.4 ± 7.6
Hypertensive (upper 10th percentile)	1872 ± 923	76 ± 42	77 ± 47	202 ± 104	673 ± 538	1120 ± 620	2129 ± 1423	2060 ± 1174	4355 ± 6738	383 ± 303	74 ± 88	8.3 ± 6.7
Difference	0.6%	1.3%	1.3%	2.9%	7.6%	3.4%	1.3%	7.1%	11.8%	1.0%	11.9%	1.2%
Adjusted for age, race, and sex												
Normotensive (lower 90th percentile)	1893 ± 935	76 ± 41	78 ± 47	208 ± 107	729 ± 552	1164 ± 645	2162 ± 1408	2220 ± 1196	4948 ± 8131	380 ± 297	84 ± 88	8.4 ± 7.6
Hypertensive (upper 10th percentile)	1779 ± 846	72 ± 39	73 ± 42	194 ± 98	665 ± 510	1079 ± 578	2066 ± 1276	2030 ± 1107	4206 ± 6076	370 ± 295	75 ± 107	7.8 ± 6.3
Difference	6.0%	5.3%	6.4%	6.7%	8.8%	7.3%	4.4%	8.6%	15.0%	2.6%	10.7%	7.1%

various blood pressure and body mass index groups.

Figure 3 shows the relation of daily consumption of calcium, potassium, and sodium to systolic blood pressure for the entire cohort. For this analysis, the population was grouped by increments of 75 mg for calcium intake, 150 mg for potassium intake, and 150 mg for sodium intake. Mean systolic blood pressure was then calculated for each group. Standard deviations varied between 16 and 26 mmHg for calcium, 14 and 27 mmHg for potassium, and 11 to 20 mmHg for sodium intake, and increased slightly in states of both extremes of nutrient intake. The number of individuals per stratum was not less than 60 for calcium, 20 for potassium, and 31 for sodium. For each of the three nutrients, increased consumption was negatively correlated with systolic blood pressure ($r = -0.604$, -0.461, and -0.279 for calcium, potassium, and sodium, respectively).

The proportions of individuals with systolic blood pressures above 160 mmHg for increments of reported calcium (75 mg), potassium (150 mg), and sodium (150 mg) consumption are shown in Fig. 4. The risk of being hypertensive increased with decrements in the ingestion of each of these nutrients, including sodium. Calcium intakes of less than 300 mg/day were associated with a risk of 11 to 14 percent, while intakes greater than 1500 mg carried a risk of 3 to 4 percent. Potassium intakes under 900 mg carried a risk of 12 to 14 percent and intakes above 4200 mg/day a risk of 4 to 5 percent. Sodium intakes below 1600 mg had an associated risk of 9 to 12 percent; intakes above 4800 mg, 2 to 4 percent, the lowest observed.

Figure 5A depicts an individual's risk of having a systolic blood pressure in the upper 10 percent for his respective age, race, and sex cohort on the basis of calcium intake (75-mg increments). This analysis controls for variability in the prevalence of hypertension in different age, race, and sex groups as well as for variability in the calcium requirements of different subgroups (such as premenopausal and postmenopausal females). For the entire population, the risk of being above the 90th percentile was 3 to 4 percent if reported calcium intake was 1600 mg/day or greater. Below this consumption level, an individual's risk increased in a roughly linear fashion. The risk increased to 11 to 12 percent at intakes below 300 mg/day. Results for potassium are shown in Fig. 5B. At a potassium consumption level of 4600 mg or greater, the risk was approximately 5 percent that, within a demographic subgroup, an individual would be in the upper 10 percent of the blood pressure profile.

Table 3 shows the results of using multivariate discriminant analysis with U.S. government food groups to predict hypertension (systolic pressure of at least 160 mmHg) in subjects over 34 years of age. Ninety-six percent of all the identified hypertensive individuals were above this age. After controlling for age, race, and sex, differences in dairy product consumption proved to be the best predictor of hypertension. The variables listed correctly identified 79.6 percent of all the individuals with a systolic blood pressure of at least 160 mmHg.

Evaluation of HANES I Analysis

HANES I is a scientifically designed, representative sample of the U.S. population and its nutrition habits (*4*). Calcium, potassium, vitamin C, and vitamin A are the nutrients that distinguish Americans at greatest risk for hypertensive cardiovascular disease from those with lower pressures and thus less risk.

The HANES I data were intended to provide useful epidemiologic insights into relations among various nutrients, demographic correlates, and measures of wellness. The associations and correlations of various nutrient intake patterns with systolic blood pressure that emerged in this analysis do not prove causality (*15*), but do suggest the relation of a given nutrient to the blood pressure profile of the United States and whether the pattern of its consumption reflects or predicts the demographics of high blood pressure.

The sample size and the individual medical histories allowed us to identify a demographically representative population of adult Americans free of known hypertensive cardiovascular disease and who denied intentional modification of their dietary habits. Combining the medical data with the survey's comprehensive assessment of each individual's diet on the previous day provides a unique opportunity to address the relation between diet and blood pressure in healthy Americans. The 24-hour dietary recall design used in HANES I is imprecise for extrapolating to an individual's lifetime exposure to nutrients but is the best technique for cross-sectional analyses intended to identify differences among specific populations (*21*).

We chose systolic blood pressure to define our populations because the risk of cardiovascular complications appears to be more closely associated with that measure of arterial pressure than with diastolic blood pressure (*22, 23*). The three different definitions of hypertension allowed us to test whether the definition of blood pressure utilized influenced the nutrients that would be identified and whether the differences that emerged were predictive of hypertension in those individuals at greatest risk for cardiovascular disease, the upper 10 percent of the U.S. population. By adjusting the blood pressure groups so that the hypertensive population was defined as the upper 10 percent of individuals controlled for age, race, sex, and body mass index, we were able to determine which nutritional variables were associated with hypertension independent of these demographic and anthropometric correlates of blood pressure. In the younger age ranges, the upper 10 percent definition results in some individuals being classified as hypertensive even though they do not meet an arbitrary definition (such as ≥ 140 mmHg). Nevertheless, these individuals are likely to be in the same high-risk population in the future as the blood pressure profile increases with age (*17*).

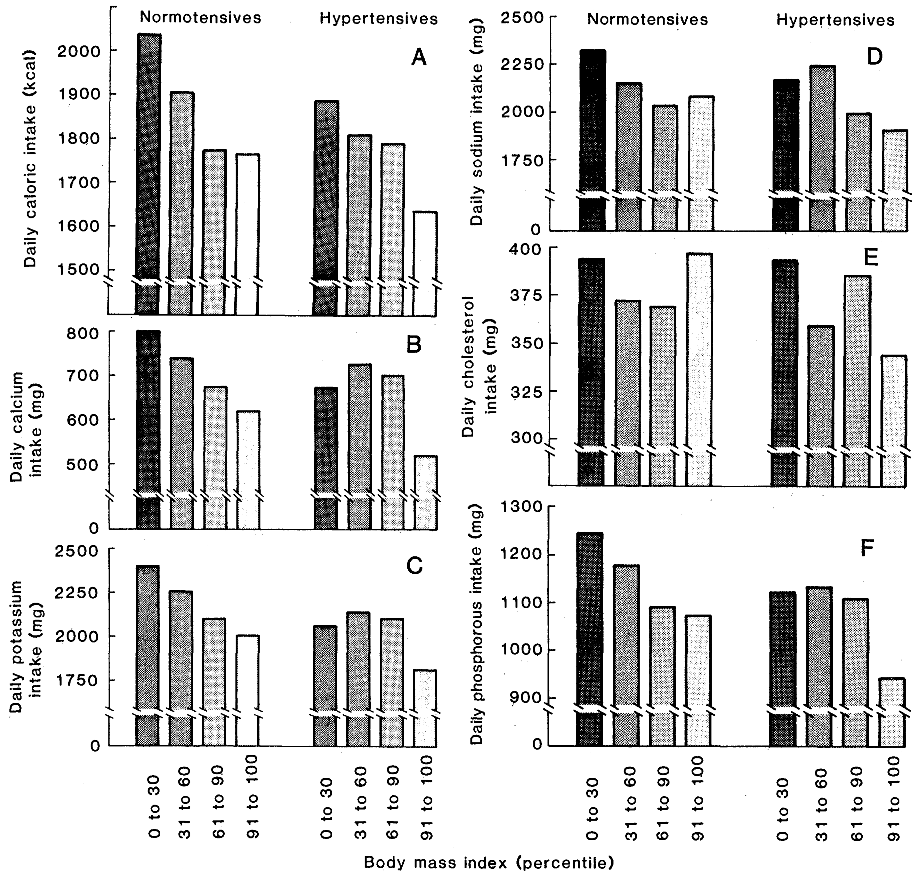
Regardless of the definition of hypertension applied to the population we studied, and, in part, independent of the effects of age, race, sex, body mass index, or alcohol consumption, lower consumption of four nutrients—calcium, potassium, vitamin A, and vitamin C—was statistically associated with hypertension. Ackley *et al.* (*12*), in a study of dairy products and blood pressure, also noted that the association was independent of these same variables.

Nutrition and Blood Pressure

Associations: Implications

Calcium. Calcium was the nutrient for which reduced intake was most consistent in hypertensive individuals, regardless of how that population was defined. Furthermore, across the entire cohort of 10,372 subjects, increasing systolic blood pressure was correlated most strongly with decrements in daily calcium ingestion. The observed range of the reduction in calcium intake in the hypertensives (7.6 to 19.6 percent) is similar to that in a recently reported diet survey (*11*). In that survey, low intake of calcium was the nutritional factor that distinguished subjects with essential hypertension from those with normal blood pressures. Our findings are also consistent with epidemiologic observations (*24–26*), spanning several decades, that have suggested an association between adequate exposure to calcium and protection against various cardiovascular disorders. In addition, recent clinical reports have linked disordered calcium metabolism to human hypertension (*27–30*).

As portrayed in this analysis, the average calcium intake for the U.S. popula-



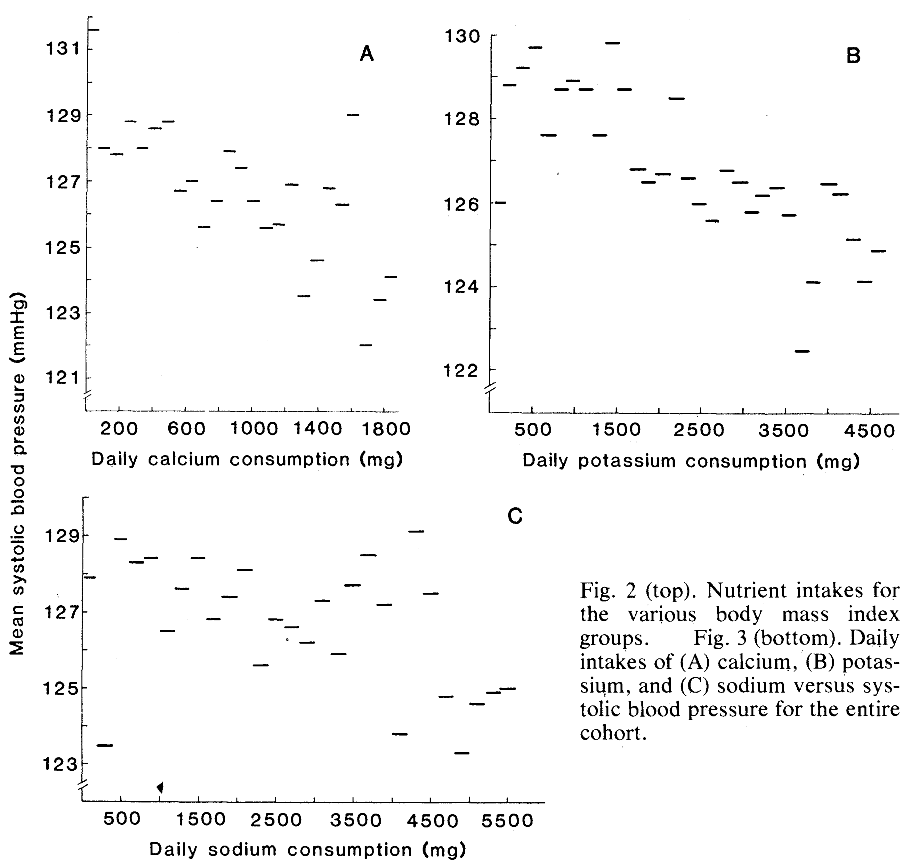


Fig. 2 (top). Nutrient intakes for the various body mass index groups. Fig. 3 (bottom). Daily intakes of (A) calcium, (B) potassium, and (C) sodium versus systolic blood pressure for the entire cohort.

tion at greatest risk for hypertensive cardiovascular disease is significantly less than the 800 mg/day recommended by the National Academy of Sciences. In addition, the calcium-depleting effects of the high content of protein, phosphorous, sodium, and alcohol in the U.S. diet may necessitate a calcium intake above 800 mg/day in some individuals if the current minimum daily requirement is to be met (*31*).

Potassium. The relation of potassium consumption to blood pressure was similar to that of calcium. However, the observed differences in potassium intake between hypertensives and normotensives were consistently less than those noted for calcium. Both epidemiologic observations and animal studies have suggested that an increased dietary potassium intake is protective against the development of hypertensive cardiovascular disease (*32*).

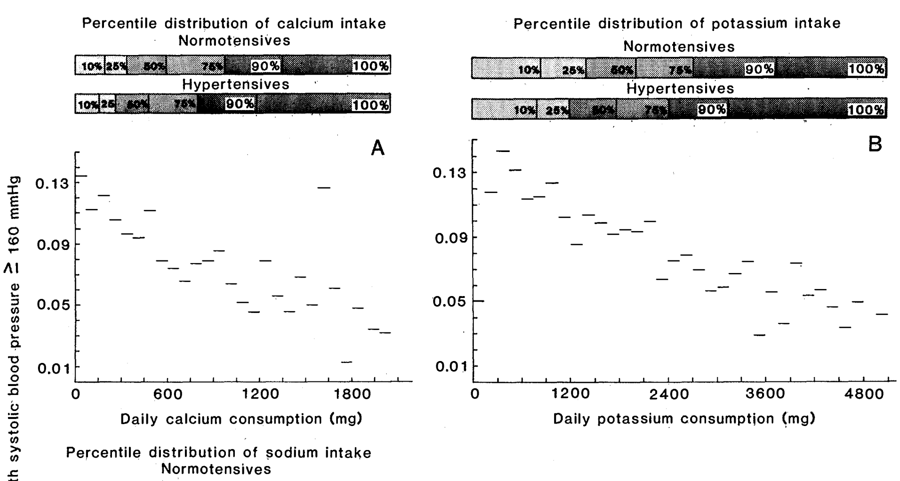
Potassium may influence cell membrane and intracellular mechanisms that contribute to vascular smooth muscle cell regulation as well as humoral and volume factors (*33*) that are functionally important in cardiovascular regulation. For calcium the putative mechanisms are less certain. While the cation is essential for smooth muscle contraction (*33*), a membrane stabilization and vascular smooth muscle relaxation effect (*34, 35*) appears to be an equally important action. The effect of calcium on cell membranes may be related, in part, to inhibitory actions on membrane-associated calcium channels (*36, 37*). Whatever specific pathway is involved, it is apparent that calcium contributes to both vascular smooth muscle cell relaxation and contraction (*33*).

The observed relation between calcium and potassium intake and blood pressure provides substantive evidence of nutrient interactions. Dairy products, which account for an individual's principal exposure to both calcium (60 to 70 percent) and potassium (35 to 45 percent) (*38*), were the food group whose consumption best predicted whether an individual over the age of 34 was hypertensive. The greater an individual's consumption of dairy products, the less likely it was that he or she was hypertensive. Increased consumption of dairy products would be associated with a correction in potassium and calcium deficits.

Vitamins A and C. Intake of vitamins A and C differed between the blood pressure groups when the populations were defined as the upper 10 percent and lower 90 percent of systolic blood pressures, as adjusted for demographic variables in the standardized analysis. With the simpler definitions of hypertension (≥ 140 or ≥ 160 mmHg), a consistent difference was less evident. A role of a deficiency of either or both of these vitamins in the pathogenesis of hypertension has not, to our knowledge, been previously postulated. The lower intake of these two vitamins in hypertensives may reflect, in part, their close association in the diet with calcium (vitamin A) and potassium (vitamin C).

Sodium. Regardless of the definition of hypertension and the demographic variables controlled for, hypertensives tended to consume less sodium than normotensives. This conclusion is consistent with a recent report from the National Center for Health Statistics (*39*) that identified an association of increased salty snack food consumption and frequent salt shaker use with lower blood pressures in the HANES I data. Of all the nutrients measured in the HANES I survey, sodium intake is subject to the greatest underestimation. The dietary recall data do not account for discretionary sodium added during food preparation and at the table. Previous studies have suggested that discretionary sodium accounts for 20 to 40 percent of total sodium consumption in the United States (*38*). Other diet surveys indicate that an individual's discretionary sodium use parallels the sodium content of the food consumed (*40, 41*); still others have demonstrated that dietary recall information on sodium consumption parallels 24-hour urinary sodium excretion (*42, 43*). Consequently, it seems likely that the lower dietary sodium content of the hypertensives in our study reflects reduced intake of the nutrient among hypertensives in general. Because the individuals that formed our sample denied a history of hypertension and intentional manipulation of their diet, the lower sodium intake reported by individuals at greatest risk for hypertension-related cardiovascular disease cannot be ascribed to changes in dietary habits related to concerns about hypertension and cardiovascular disease. Earlier reports, based on the HANES I data, that addressed the relation of sodium intake to measures of wellness suggested a similar inverse relation between dietary sodium and high blood pressure in the United States (*39–41*).

Previous attempts to establish the putative link between sodium consumption and blood pressure in the United States have failed (*6, 44*). The HANES I population, however, was specifically identified so that observations based on its analysis could be extrapolated to the entire U.S. population (*4*). Unlike any previous assessment of sodium's relation to blood pressure in more limited U.S.



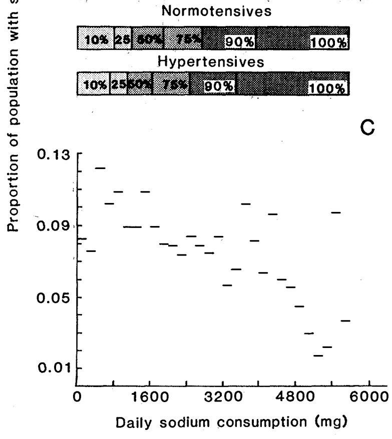


Fig. 4. Proportion of individuals with systolic blood pressures ≥ 160 mmHg versus increments of daily (A) calcium, (B) potassium, and (C) sodium consumption.

Table 3. Multivariate discriminant analysis of food groups in subjects 35 to 74 years old and having systolic blood pressures of < 160 mmHg or ≥ 160 mmHg, controlled for age, race, and sex.

Food group	Order of variable entry	Standardized canonical discriminant function coefficient
Dairy products	1	−0.1344
Fruit and vegetable juices	2	−0.1017
Nonsugar beverages	3	−0.0738
Fats and oils	4	−0.0644
Organ meats	5	−0.0660
Mixed protein dishes	6	−0.0564
Shellfish	7	−0.0555
Desserts	8	−0.0549

surveys, HANES I provides the opportunity to evaluate the association of blood pressure with extremes of sodium intake. With HANES I, sufficiently large numbers of individuals can be identified who spontaneously reported diets either relatively low or relatively high in sodium (Fig. 4C). While "salt-sensitive" individuals are not specifically identified, it is evident that subjects reporting low-sodium diets are at two to three times greater risk of being hypertensive than those who report a high sodium intake. Such cross-sectional dietary recall data are not necessarily indicative of individual patterns but do indicate the characteristics of the population as a whole.

The relation between greater sodium consumption and lower blood pressure is consistent with both nutritional and physiological interactions of nutrients. First, as mentioned above, dairy products are a significant source of sodium as well as calcium and potassium. Second, the actions of sodium are closely linked to those of calcium and potassium at both the cellular and organ levels (*45–47*).

Cholesterol. Cholesterol consumption and serum cholesterol concentration have been considered concurrent risk factors with high blood pressure for the development of cardiovascular disease in Western societies (*3, 48*). In our study cholesterol intake did not differ consistently between hypertensive and normotensive individuals.

Obesity. The known link between obesity and blood pressure has not been explained by past investigations (*18, 19, 49*). Excessive sodium intake in the obese subject was long thought to be a contributing factor (*50*). However, controlled clinical investigations in the past few years have largely discounted that explanation (*51, 52*). Our analysis suggests that obesity-related hypertension is not associated with excessive sodium consumption. Furthermore, the total caloric intake of obese subjects was less than that of lean individuals across the HANES I population, a finding noted previously (*53*). Hypertensives also reported a lower caloric intake than normotensives. This was evident principally in the leanest and most obese subgroups of hypertensives. It is unlikely that the heavier subjects and hypertensive subjects intentionally underreported their caloric intake for several reasons. First, the inverse relation between body mass index and calories was strong and continuous, even within the leaner portion of the population. Second, none of the subjects perceived him or herself as hypertensive. Third, the association between a significant reduction in calories and elevated blood pressure was apparent even in the leanest 30th percentile of the hypertensive population (Fig. 2A).

It appears that nutritional deficiencies and not excesses are what distinguish overweight or hypertensive individuals from normal subjects in the United States. Caloric restriction increases the risk of further reducing an individual's exposure to nutrients that may be essential for maintaining normal mean arterial pressures.

Conclusion

Our analysis of the HANES I survey suggests the following:

1) There are predictable nutritional differences between individuals with high blood pressure and those with normal blood pressure.

2) Deficiencies rather than excesses are the principle nutritional patterns that characterize the hypertensive person in America.

3) Reduced consumption of calcium and potassium is the primary nutritional marker of hypertension, with reductions in vitamins A and C also being noted.

4) Dairy products are the food group for which reduced consumption is most closely related to high blood pressure in the United States.

5) These observations are largely independent of age, race, sex, body mass index, and alcohol consumption.

6) Diets low in sodium are associated with higher blood pressures, while high-sodium diets are associated with the lowest blood pressures.

Implicit in the application of the nutrient and blood pressure interactions we have characterized from the HANES I

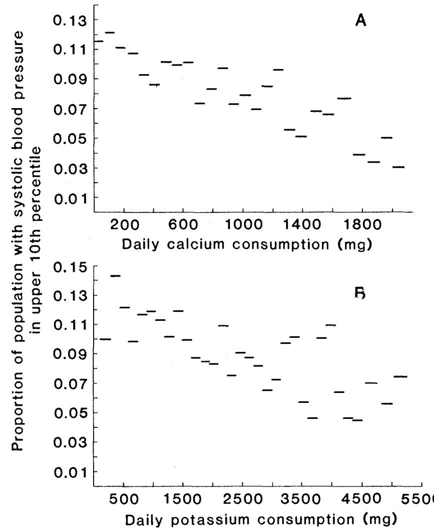


Fig. 5. Proportion of the population with systolic blood pressures in the upper 10th percentile versus increments of daily (A) calcium and (B) potassium intake.

data is a note of caution. Clinical use of sodium- or cholesterol-restricted diets for patients with high blood pressure or cardiovascular disease must be monitored closely to avoid inadvertent, simultaneous reduction in calcium and potassium intakes below current recommended daily allowances (*38*). In addition, these epidemiologic data raise the important question of whether sodium restriction is routinely advisable in many hypertensives (*6, 54*). The identified "salt-sensitive" patient and patients with compromised cardiac or renal function would be the obvious exceptions.

It must be emphasized that these findings do not prove causality. They simply indicate potentially important relations among nutrients and blood pressure regulation in humans. It is possible that low consumption of dairy products (the major source of calcium) serves as a marker of hypertension; however, if the dietary patterns of smokers and individuals with sedentary or stressful life-styles involve low intake of dairy products, one cannot be sure from these findings which is the cause and which the effect. Only future clinical and basic laboratory investigations can ascertain the importance of these correlations in the application of health measures intended to reduce the prevalence of hypertensive cardiovascular disease in adult Americans. If validated, our observations do not indicate that it is routinely necessary to ingest any nutrient, including calcium or potassium, above the current recommended levels. Rather, they suggest the consumption of a diet balanced in all the essential nutrients and appropriate for the individual's level of physical activity.

References and Notes

1. R. I. Levy and J. Moskwitz, *Science* **217**, 121 (1982).
2. W. J. Walker, *N. Engl. J. Med.* **308**, 649 (1983).
3. J. Stamler, *Circulation* **58**, 3 (1978).
4. Department of Health, Education, and Welfare, *DHEW Publ. (HRA) 77-1310* (1977).
5. L. Tobian, *Ann. Intern. Med.* **98**, 729 (1983).
6. J. H. Laragh and M. S. Pecker, *ibid.*, p. 735.
7. H. G. Langford, *ibid.*, p. 770.
8. G. A. Perera, *J. Clin. Invest.* **32**, 633 (1953).
9. R. B. Shekelle *et al.*, *N. Engl. J. Med.* **304**, 65 (1981).
10. A. B. Nichols, C. Ravenscroft, D. E. Lamphiear, L. D. Ostrander, Jr., *Am. J. Clin. Nutr.* **29**, 1384 (1976).
11. D. A. McCarron, C. D. Morris, C. Cole, *Science* **217**, 267 (1982).
12. S. Ackley, E. Barrett-Connor, L. Suarez, *Am. J. Clin. Nutr.* **38**, 457 (1983).
13. C. F. Adams, *U.S. Dep. Agric. Agric. Handb. No. 456* (1975), pp. 1–291.
14. J. A. T. Pennington and H. N. Church, *Food Values of Portions Commonly Used* (Harper & Row, New York, 1980).
15. Multivariate analysis provides a theoretical framework that allows simultaneous consideration of several dependent variables. Specifically, multiple regression analysis is an extension of straight-line regression analysis involving only one independent variable to a situation involving more than one independent variable. Although the usual parametric tests of hypothesis in multiple linear regression analysis are robust, these data did not violate the assumptions, as shown by residual analysis. Discriminant analysis, which can be considered an extension of multiple regression analysis, determines whether and to what extent the independent variables can discriminate a dichotomous dependent variable, and may be particularly appropriate when a threshold exists, as in hypertension.
    These methods may produce goodness of fit to the data (in the sense that a parabola can fit an arc of a sine curve adequately), yet the coefficient may bear little relation in magnitude and may even be of opposite sign to coefficients obtained with the full, correct model. This occurs when an important variable is omitted or if nonlinearity is overlooked, in which case a bias can be introduced into all coefficients (the reason for the "correlation does not equal causation" warning). Tests of significance of coefficients against an alternative of zero cannot, therefore, be interpreted meaningfully unless one can place a bound on the possible bias in the coefficient that is attributable to the effect of excluded variables. Equally, the lack of statistical significance cannot be taken as evidence of lack of effect of the variable.
16. Department of Health, Education, and Welfare, *DHEW Publ. (PHS) 79-1658* (1979).
17. J. M. Kotchen, H. E. McKean, T. A. Kotchen, *Hypertension* **4** (Suppl. 3), 128 (1982).
18. J. L. Stanton, L. E. Braitman, A. M. Riley, C. S. Khoo, J. L. Smith, *ibid.*, p. 135.
19. R. J. Havlik, H. Hubert, R. R. Fabsitz, M. Feinlieb, *Ann. Intern. Med.* **98**, 855 (1983).
20. National Research Council, *Recommended Dietary Allowances* (National Academy of Sciences, Washington, D.C., ed. 9, 1980).
21. G. Block, *Am. J. Epidemiol.* **115**, 492 (1982).
22. W. B. Kannel, T. Gordon, M. J. Schwartz, *Am. J. Cardiol.* **27**, 335 (1971).
23. S. W. Rabkin, F. A. L. Mathewson, R. B. Tate, *Ann. Intern. Med.* **88**, 342 (1978).
24. H. A. Schroeder, *J. Am. Med. Assoc.* **172**, 1902 (1960).
25. F. W. Stitt, M. D. Crawford, D. G. Clayton, J. N. Morres, *Lancet* **1973-I**, 122 (1973).
26. D. A. McCarron, J. Stanton, H. J. Henry, C. D. Morris, *Ann. Intern. Med.* **98**, 715 (1983).
27. D. A. McCarron, *N. Engl. J. Med.* **307**, 226 (1982).
28. ——, *Hypertension* **2**, 162 (1980).
29. F. W. Lafferty, *Arch. Intern. Med.* **141**, 1761 (1981).
30. D. A. McCarron, *Hypertension* **4** (Suppl. 3), 27 (1982).
31. L. H. Allen, *Am. J. Clin. Nutr.* **35**, 783 (1982).
32. R. L. Tannen, *Ann. Intern. Med.* **98**, 773 (1983).
33. H. Kuriyama, I. Yushi, H. Suzuki, K. Kitamura, T. Itoh, *Am. J. Physiol.* **243**, H641 (1982).
34. D. F. Bohr, *Science* **139**, 597 (1963).
35. R. C. Webb and D. F Bohr, *Am. J. Physiol.* **235**, C227 (1978).
36. R. Eckert and D. Ewald, *Science* **216**, 730 (1982).
37. L. Hurwitz, L. J. McGuffee, P. M. Smith, S. A. Little, *J. Pharmacol. Exp. Ther.* **220**, 382 (1982).
38. A. M. Engstrom and R. C. Tobelman, *Ann. Intern. Med.* **98**, 870 (1983).
39. Department of Health and Human Services, *DHHS Publ. (PH5) 83-1676* (1983).
40. M. J. Fregley, *Ann. Intern. Med.* **98**, 792 (1983).
41. H. S. Schwerin, J. L. Stanton, A. M. Riley, Jr., B. E. Brett, *Am. J. Clin. Nutr.* **35**, 1319 (1982).
42. P. Pietinen, *Ann. Nutr. Metab.* **26**, 90 (1982).
43. J. Schlacter, P. H. Harper, M. E. Radin, A. W. Caggiula, R. H. McDonald, W. F. Diven, *Hypertension* **2**, 695 (1980).
44. R. Cooper, K. Liu, M. Trevisan, W. Miller, J. Stamler, *ibid.* **5**, 135 (1983).
45. D. A. McCarron, H. J. Henry, C. D. Morris, *ibid.* **4** (Suppl. 3), 2 (1982).
46. A. W. Jones, in *The Cardiovascular System*, vol. 2, *Content and Fluxes of Electrolytes*, D. F. Bohr *et al.*, Eds. (American Physiology Society, Bethesda, Md., 1980), pp. 253–299.
47. S. M. Friedman, *Ann. Intern. Med.* **98**, 753 (1983).
48. C. J. Gluck, *Am. J. Clin. Nutr.* **32**, 2703 (1979).
49. H. P. Dustan, *Ann. Intern. Med.* **98**, 860 (1983).
50. L. Dahl, L. Silver, R. W. Christie, *N. Engl. J. Med.* **258**, 1186 (1958).
51. E. Reisin *et al.*, *N. Engl. J. Med.* **298**, 1 (1978).
52. M. L. Tuck, J. Sowers, L. Dornfeld, G. Kledzik, M. Maxwell, *ibid.* **304**, 930 (1981).
53. T. Gordon, M. Fisher, B. M. Rifkind, *Am. J. Clin. Nutr.* **39**, 152 (1984).
54. F. O. Simpson, *Clin. Sci.* **57**, 463s (1979).

55. We are indebted to D. Krakower for statistical analysis, J. Utterback for preparing the manuscript, and P. P. McCarron for editorial assistance. Special appreciation is extended to W. M. Bennett for his continued professional support of this research. Supported by grants-in-aid from the M. J. Murdock Charitable Trust and the R. Blaine Bramble Medical Research Foundation. C.D.M. is a recipient of grant support from the Medical Research Foundation of Oregon and is an N. L. Tartar research fellow.

Inherently Safe Reactors and a Second Nuclear Era

Alvin M. Weinberg and Irving Spiewak

David Lilienthal, in his book *Atomic Energy, A New Start* (*1*), was among the first to call upon nuclear technologists to design a reactor that was inherently safe. He saw such a device as being necessary for a new start in atomic energy. Without such a forgiving reactor, Lilienthal doubted that nuclear energy could regain the public's confidence, which had been so badly shattered by the Three Mile Island incident. The book appeared in 1980. At that time it was received with skepticism by the reactor community. The review of the book by one of us, for example, suggested that it was easy enough for a nontechnologist like Lilienthal to call for an inherently safe reactor, but that the fundamental characteristics of the fission process, in particular the afterheat, made such a goal all but unattainable (*2*).

Nevertheless, in May of 1980, the Institute for Energy Analysis, under the sponsorship of the Department of Energy, convened a 2-day workshop at which the possibility of designing a practical, inherently safe reactor was discussed. In attendance were many of those responsible for setting nuclear energy on its present course: M. Benedict of Massachusetts Institute of Technology, K. Cohen and E. Schmidt of General Electric, P. Cohen of Westinghouse, J. Dietrich of Combustion Engineering, M. Edlund of Babcock and Wilcox, P. Fortescue of General Atomic, K. Davis, then of Bechtel and later deputy secretary of energy, H. Kendrick of Department of Energy, U. Gat of Oak Ridge National Laboratory, and H. G. MacPherson, J. A. Lane, E. P. Epler, M. W. Firebaugh, and the authors, associated with the Institute itself.

We concluded that a serious study of more forgiving, or perhaps even inherently safe, reactors was a good idea, but the study would have to begin by assessing the safety of existing light-water reactors and of incremental improvements to light-water reactors (*3*). Most of the participants in the workshop believed that such a reexamination would confirm the widely held view that light-water reactors were as safe as any reactors that might compete economically with them.

A year later the Andrew W. Mellon Foundation granted the Institute for En-

Alvin M. Weinberg is director of the Institute for Energy Analysis, Oak Ridge Associated Universities. Irving Spiewak, formerly section head in the Engineering Technology Division, Oak Ridge National Laboratory, is a Mellon Fellow at the Institute for Energy Analysis.