Association between blood pressure and urinary electrolytes in a population of nonurban-dwelling Nigerians

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Abstract

Background: Little is known about the association between blood pressure and urinary electrolytes in young adult, nonurban-dwelling, sub-Saharan Africans. This study attempts to provide such data in a Nigerian population.

Patients and Methods: Four hundred Nigerians (50% female) aged 19-40 years were studied. Their blood pressures (BPs), anthropometric variables, and overnight urinary sodium, and potassium concentrations (UNaC and UKC respectively) were measured using standard procedures. Associations between measures of BP and the other parameters were examined using appropriate statistical tools.

Results: UNaC was correlated significantly with only diastolic BP (DBP) (r = +0.105, P = 0.037). Similarly, UNaC was significantly associated with DBP and mean BP (MBP) (β = +0.158, P = 0.018 and β = +0.155, P = 0.020, respectively). UKC was not associated with, nor correlated with, any measure of BP. There was no significant mean difference (P > 0.05) between the sexes for measures of BP and urinary electrolytes.

Conclusion: Urinary sodium (but not potassium) concentration was weakly correlated with only DBP, and weakly associated with only DBP and MBP in the studied population. The results support (modestly) the hypothesis that dietary sodium intake may be related to elevated blood pressure.

Key words: Association, blood pressure, electrolytes, urinary sodium and potassium

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Introduction

Cardiovascular diseases (CVDs) (especially hypertension) are multifactorial diseases that cannot be ascribed to a single gene or environmental factor. This makes understanding the patho-etiologic mechanisms underlying hypertension difficult[1] and identifying a nutrient that could be manipulated to reverse CVD morbidity and mortality akin to finding the Holy Grail of nutrition and cardiovascular science.[2] Sodium intake has often been associated with elevations in blood pressure. But the evidence is still controversial. Some observational and experimental studies support an independent, positive relationship between sodium intake and high blood pressure,[3-6] while some others support an inverse (or lack of) association between sodium intake and high blood pressure.[7-10] As a result of these differences, there is still no consensus on the role of dietary sodium in hypertension. The effect of potassium intake on blood pressure, like sodium intake, is controversial. It has also been reported variously to be inversely related, or not related at all, to increases in blood pressure and cardiovascular events.[11-15]

Morbidity and mortality from CVD are lower in women than in men throughout middle age.[16,17] Estrogens, acting

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through the regulation of sodium and water, are thought to be responsible for this sex difference in CVD morbidity and mortality,[18,19] and exogenous estrogen administration during and after menopause is known to maintain the female CVD risk advantage.[17]

Since hypertension, a strong predictor of CVD, is creeping in steadily on developing countries, and may arise from disturbances in sodium and potassium metabolism, this study investigated the relationship between urinary sodium and potassium excretion and blood pressure in a population of nonurban Nigerian adults, who have not reached the age limits stipulated as risks for coronary artery disease (CAD) by the US National Cholesterol Education Program (NCEP) (≥45 years for men, and ≥55 years for women),[20] and whose diets are still largely traditional and definitely far off the salted high-fat and low-fiber diets of the industrialized countries, or the city/urban centers in developing countries.

Patients and Methods

Male and female adults resident in Anyigba, Kogi State, Nigeria (age range: 19-40 years) were recruited for this population-based study. Participants were randomly recruited over a 4-month period. The individuals were properly informed about the aims and objectives of the study and those who gave an informed consent were allowed to participate in the study. Exclusion criteria included being a current smoker or having a history of smoking, current use of alcohol, pregnancy, morbid obesity (BMI >35), a known case of hypertension/current use of antihypertensive medication, and any other overt or suspected infirmity. The study protocol was prepared in accordance with the Helsinki Declaration and was approved by the Board of the Department of Biochemistry, Kogi State University, Anyigba.

Participants had their blood pressure measured on a single visit, between 8 am and 10 am daily, using sphygmomanometry and appropriate cuff sizes. Each subject rested for an initial 10 minutes before three separate readings were taken (after a 2 minutes interval each) in a serene environment. The average of the second and third readings per subject was recorded. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken at the first and fifth Korotkoff sounds respectively. Pulse pressure (PP) was calculated as the difference between SBP and DBP. Mean blood pressure (MBP) was calculated as [SBP + (DBP × 2)]/3. The same trained personnel took all blood pressure measurements.

Measurement of weight (to the nearest 1 kg) and height (to the nearest 1 cm) were taken, with subjects dressed in light clothing, and the BMI calculated as weight (kg)/ height (m)^2. Self-reported age (at last birthday) for each participant was recorded.

Overnight urine was collected during the first urination after awakening in the morning and during awakenings at night (where applicable), from each subject, who had been properly educated on how to collect the samples, prior to sample collection. The samples were taken immediately to our laboratory and frozen, until analyzed. Urinary sodium and potassium ions' concentrations were determined by potentiometry – the ion-selective electrodes method.[21]

Descriptive statistics was done to get the characteristics of the subjects (stratified by urinary sodium and potassium tertiles), represented as means ± standard deviations. Differences between means for the sexes and tertiles were checked for significance using the one-way ANOVA test. The Pearson's product moment correlation was used to assess correlations between variables. Multivariate linear regression models were used to examine the association between the variables and measures of blood pressure (SBP, DBP, PP, and MBP). A significant threshold of $P < 0.05$ was employed for the ANOVA, correlation, and regression analyses. Analyses were performed using SPSS for windows version 17.0 (SPSS Inc., Chicago, IL, USA).

Results

Four hundred subjects (50% females) participated in the study. The mean age of the participants was 25.2 ± 5.2 years (26.5 ± 5.8 for females and 23.7 ± 3.6 years for males). Males were significantly ($P = 0.024$) younger than the females. Females weighed significantly ($P = 0.021$) less than the males (63.3 ± 14.9 kg vs. 66.6 ± 9.7 kg) and were significantly ($P = 0.017$) shorter than the males (1.6 ± 0.07 m vs. 1.7 ± 0.07 m). Conversely, the females had significantly ($P = 0.012$) higher BMI compared to the males (24.8 ± 8.3 kg/m^2 vs. 22.2 ± 3.1 kg/m^2). Urinary sodium excretion was statistically similar ($P = 0.251$) between the sexes (190.1 ± 85.3 mEq/l in females and 198.5 ± 96.5 mEq/l in males), and urinary potassium excretion was also statistically similar ($P = 0.095$) between the sexes (74.2 ± 31.8 mEq/L in females and 71.8 ± 29.1 mEq/L in males). In a similar fashion, the urinary sodium/potassium ratio was statistically similar ($P = 0.113$) between the sexes (3.4 ± 1.7 for females and 3.1 ± 1.3 for males).

Mean SBP was not significantly different ($P = 0.375$) between the sexes (125.9 ± 17.3 mmHg for males and 121.8 ± 12.1 mmHg for females). Similarly, mean DBP was 78.8 ± 12.2 mmHg for females and 79.0 ± 12.7 for males, and the difference was not statistically significant ($P = 0.423$). Following the same pattern, mean PP values were 45.1 ± 11.1 mmHg for females and 46.9 ± 15.3 mmHg for males, while mean MBP values were 91.8 ± 12.9 mmHg for females and 94.6 ±° 12.3 mmHg for males. In both

Table 1: Distribution of age, anthropometric variables, urinary electrolytes, and measures of blood pressure stratified by tertiles of urinary sodium concentration

	Age (years)	Weight (kg)	Height (m)	BMI (kg/m²)	Sodium (mEq/l)	Potassium (mEq/l)	Sodium/potassium	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	MBP (mmHg)
I[st]											
F	29.3 ± 6.4	64.8 ± 12.8	1.6 ± 0.06	24.8 ± 6.3	102.6 ± 24.9	46.2 ± 22.9	2.6 ± 1.1	121.8 ± 11.7	76.4 ± 9.4	45.4 ± 9.4	91.6 ± 9.4
M	24.0 ± 4.0	65.9 ± 9.1	1.7 ± 0.06	21.8 ± 2.5	88.4 ± 28.7	44.5 ± 23.0	2.5 ± 1.3	122.8 ± 17.8	75.7 ± 9.4	47.1 ± 14.9	91.4 ± 10.7
P	<0.001	0.56	<0.001	<0.001	<0.01	0.78	0.83	0.73	0.73	0.45	0.94
T 2[nd]	27.0 ± 6.1	65.3 ± 11.3	1.7 ± 0.08	23.5 ± 5.2	96.3 ± 27.5	45.4 ± 22.9	2.5 ± 1.2	122.3 ± 14.7	76.1 ± 9.5	46.1 ± 12.1	91.5 ± 10.0
F	26.1 ± 5.9 (<0.01)	63.0 ± 14.6 (0.35)	1.6 ± 0.07 (0.19)	24.2 ± 5.4 (0.40)	202.8 ± 31.9 (<0.01)	79.9 ± 37.0 (<0.01)	3.8 ± 6.2 (0.02)	122.9 ± 19.1 (0.70)	78.1 ± 12.3 (0.43)	44.8 ± 12.2 (0.81)	93.0 ± 13.8 (0.49)
M	23.7 ± 3.1 (0.67)	66.7 ± 10.2 (0.72)	1.7 ± 0.07 (0.28)	22.4 ± 2.7 (0.54)	193.2 ± 35.1 (<0.01)	76.4 ± 33.7 (<0.01)	3.0 ± 1.2 (0.36)	127.9 ± 17.0 (0.09)	80.7 ± 14.9 (0.03)	47.3 ± 16.4 (0.95)	96.4 ± 13.6 (0.03)
P	<0.01	0.06	<0.01	0.03	0.04	0.56	0.11	0.08	0.22	0.28	0.11
T 3[rd]	24.9 ± 4.9	64.8 ± 12.8	1.7 ± 0.09	23.3 ± 4.4	198.2 ± 33.7	78.2 ± 35.4	3.4 ± 4.6	125.3 ± 18.2	79.3 ± 13.6	46.0 ± 14.4	94.7 ± 13.7
F	24.2 ± 4.4 (<0.01)	62.7 ± 11.9 (0.32)	1.6 ± 0.07 (0.10)	25.4 ± 7.3 (0.55)	293.8 ± 19.3 (<0.01)	96.4 ± 41.6 (<0.01)	3.7 ± 1.7 (0.04)	120.8 ± 20.9 (0.75)	75.8 ± 16.4 (0.78)	45.0 ± 12.1 (0.89)	90.8 ± 17.1 (0.75)
M	23.3 ± 2.8 (0.35)	66.0 ± 8.7 (0.99)	1.7 ± 0.08 (0.04)	22.5 ± 3.3 (0.45)	302.1 ± 27.4 (<0.01)	94.8 ± 49.4 (<0.01)	3.9 ± 1.8 (0.01)	126.9 ± 16.2 (0.18)	80.6 ± 13.2 (0.03)	46.3 ± 15.5 (0.72)	96.0 ± 12.2 (0.04)
P	0.26	0.12	<0.01	<0.01	0.12	0.808	0.68	0.06	0.04	0.62	0.03
T	23.6 ± 3.5	64.6 ± 10.2	1.7 ± 0.09	23.7 ± 5.5	298.8 ± 24.7	95.5 ± 46.3	3.8 ± 1.7	124.4 ± 18.4	78.6 ± 14.7	45.8 ± 14.1	93.9 ± 14.6

1st, 2nd, and 3rd represent first, second and third tertiles, respectively. F, M, *P*, and T represent females, males, probability value, and total respectively. SBP, DBP, PP, and MBP represent systolic blood pressure, diastolic blood pressure, pulse pressure and mean blood pressure, respectively. *P* values in brackets represent values for comparisons with the corresponding value (sex-wise) in the first tertile.

Table 2: Distribution of age, anthropometric variables, urinary electrolytes, and measures of blood pressure stratified by tertiles of urinary potassium concentration

	Age (years)	Weight (kg)	Height (m)	BMI (kg/m²)	Sodium (mEq/l)	Potassium (mEq/l)	Sodium/potassium	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	AvBP (mmHg)
I^st											
F	27.3 ± 6.3	61.9 ± 11.0	1.6 ± 0.07	23.8 ± 6.1	127.7 ± 57.5	36.2 ± 10.4	4.3 ± 6.3	119.4 ± 11.9	75.7 ± 10.3	43.6 ± 9.5	90.3 ± 9.9
M	23.5 ± 3.8	65.8 ± 9.2	1.7 ± 0.07	21.6 ± 2.5	131.4 ± 75.6	33.5 ± 10.6	3.9 ± 1.8	126.0 ± 18.7	76.0 ± 13.2	50.0 ± 16.1	92.7 ± 13.3
P	<0.01	0.05	<0.01	0.01	0.77	0.44	0.439	0.23	0.90	0.01	0.28
T 2^nd	25.7 ± 5.7	63.6 ± 10.4	1.7 ± 0.09	22.8 ± 4.9	129.1 ± 66.0	34.9 ± 10.5	4.1 ± 4.8	122.3 ± 15.7	75.8 ± 11.6	46.5 ± 13.3	91.3 ± 11.5
F	26.2 ± 5.9 (0.19)	63.6 ± 13.4 (0.42)	1.6 ± 0.07 (0.38)	24.5 ± 4.8 (0.44)	208.8 ± 74.1 (<0.01)	62.0 ± 7.9 (<0.01)	3.4 ± 1.3 (0.08)	120.3 ± 12.9 (0.75)	75.7 ± 10.6 (0.99)	44.6 ± 10.3 (0.69)	90.6 ± 10.3 (0.88)
M	23.4 ± 2.7 (0.83)	67.2 ± 8.9 (0.47)	1.7 ± 0.07 (0.02)	22.9 ± 2.7 (0.13)	219.9 ± 91.3 (<0.01)	64.8 ± 8.6 (<0.01)	3.5 ± 1.4 (0.35)	126.7 ± 16.6 (0.83)	81.5 ± 12.5 (0.01)	45.2 ± 15.2 (0.04)	96.5 ± 12.0 (0.08)
P	<0.01	0.07	<0.01	0.05	0.38	0.45	0.94	0.03	0.01	0.80	0.01
T 3^rd	24.3 ± 4.6	65.6 ± 11.2	1.7 ± 0.08	23.6 ± 3.9	214.9 ± 78.0	63.5 ± 8.4	3.4 ± 1.4	123.8 ± 15.3	78.9 ± 12.0	44.9 ± 13.2	93.9 ± 11.6
F	26.8 ± 6.0 (0.60)	65.7 ± 15.4 (0.06)	1.6 ± 0.07 (0.40)	26.1 ± 7.4 (0.01)	233.8 ± 61.3 (<0.01)	119.8 ± 26.4 (<0.01)	2.0 ± 0.6 (<0.01)	126.8 ± 23.8 (0.01)	79.5 ± 16.0 (0.08)	47.3 ± 13.3 (0.12)	95.2 ± 17.9 (0.02)
M	24.0 ± 3.4 (0.59)	65.4 ± 10.0 (0.84)	1.7 ± 0.08 (0.30)	22.1 ± 3.3 (0.53)	244.3 ± 79.4 (<0.01)	121.1 ± 39.2 (<0.01)	2.1 ± 0.7 (<0.01)	125.3 ± 16.0 (0.80)	79.6 ± 12.8 (0.12)	45.7 ± 15.2 (0.07)	94.8 ± 11.9 (0.35)
P	<0.01	0.89	<0.01	<0.01	0.41	0.72	0.86	0.62	0.96	0.51	0.84
T	25.4 ± 5.1	65.5 ± 12.9	1.7 ± 0.09	24.1 ± 6.0	239.1 ± 70.9	120.4 ± 33.3	2.0 ± 0.6	126.0 ± 20.2	79.5 ± 14.4	46.5 ± 14.3	95.0 ± 15.1

1^st, 2^nd, and 3^rd represent first, second and third tertiles, respectively. F, M, *P*, and T represent females, males, probability value, and total respectively. SBP, DBP, PP, and MBP represent systolic blood pressure, diastolic blood pressure, pulse pressure, and mean blood pressure, respectively. *P* values in brackets represent values for comparisons with the corresponding value (sex-wise) in the first tertile.

Table 3: Pearson's correlation coefficients for comparisons between age, anthropometric variables, urinary electrolytes, and measures of blood pressure

	Age (years) r (*P*)	Weight (kg) r (*P*)	Height (m) r (*P*)	BMI (kg/m^2) r (*P*)	Sodium (mEq/L) r (*P*)	Potassium (mEq/l) r (*P*)	Sodium/ potassium r (*P*)
SBP (mmHg)	+0.136 (0.007)	+0.220 (<0.001)	+0.107 (0.032)	+0.118 (0.019)	+0.064 (0.200)	+0.043 (0.398)	−0.034 (0.493)
DBP (mmHg)	+0.123 (0.014)	+0.229 (<0.001)	+0.050 (0.319)	+0.124 (0.013)	+0.105 (0.037)	+0.087 (0.084)	−0.091 (0.069)
PP (mmHg)	+0.056 (0.261)	+0.063 (0.208)	+0.089 (0.076)	+0.032 (0.528)	−0.017 (0.734)	−0.027 (0.596)	+0.042 (0.399)
MBP (mmHg)	+0.141 (0.005)	+0.249 (<0.001)	−0.081 (0.108)	+0.134 (0.007)	+0.098 (0.051)	+0.077 (0.127)	−0.076 (0.132)

SBP = Systolic blood pressure; DBP = Diastolic blood pressure; PP = Pulse pressure; MBP = Mean blood pressure, respectively

Table 4: Data from the regression analysis showing association between measures of blood pressure and the other studied variables

	Sex r (*P*)	Age (years) r (*P*)	Weight (kg) r (*P*)	Height (m) r (*P*)	BMI (kg/m^2) r (*P*)	Sodium (mEq/L) r (*P*)	Potassium (mEq/L) r (*P*)	Sodium/ potassium r (*P*)
SBP (mmHg)	+0.142 (0.026)	+0.157 (0.007)	+0.176 (0.018)	−0.016 (0.825)	−0.020 (0.786)	+0.114 (0.089)	−0.043 (0.527)	−0.029 (0.611)
DBP (mmHg)	+0.124 (0.051)	+0.126 (0.029)	+0.252 (0.001)	−0.098 (0.161)	−0.072 (0.316)	+0.158 (0.018)	−0.044 (0.508)	−0.096 (0.089)
PP (mmHg)	+0.064 (0.332)	+0.080 (0.183)	−0.014 (0.853)	+0.073 (0.318)	+0.043 (0.565)	−0.004 (0.957)	−0.012 (0.859)	+0.054 (0.358)
MBP (mmHg)	+0.145 (0.022)	+0.153 (0.008)	+0.244 (0.001)	−0.072 (0.303)	−0.056 (0.431)	+0.155 (0.020)	−0.048 (0.469)	−0.076 (0.175)

SBP = Systolic blood pressure; DBP = Diastolic blood pressure; PP = Pulse pressure; MBP = Mean blood pressure, respectively

cases, the difference between the sexes was not significant ($P > 0.05$).

Table 1 shows the distribution of the data in tertiles of urinary sodium concentration, while Table 2 shows the distribution of the same data in tertiles of urinary potassium concentration. Table 3 shows data from the correlation analysis while Table 4 shows data from the regression analysis. Aside anthropometric variables, only urinary sodium was associated with any measure of BP.

Discussion

This was a cross-sectional study of adults who (from the point of age) are at low risk for CAD. This study sought to investigate the relationship(s) between urinary electrolytes (sodium and potassium) and measures of BP. We are not aware of any precedent study of this nature, in a nonurban sub-Saharan African community. The studied community is gradually undergoing transition (nutritionally and epidemiologically) but is still far-off the habits of urban/city centers (especially in terms of the consumption of processed salty foods, and decreased physical exertion). The results of the present study confirm the positive relationship between age, anthropometric variables and BP, and show that only DBP and MBP were associated, albeit weakly, with urinary sodium concentration, while neither urinary potassium concentration nor the sodium/potassium ratio was significantly associated with any measure of BP. Furthermore, the results show that the (premenopausal) females studied had comparable BP values to those of their significantly younger male counterparts.

The relationship between BP and salt is still controversial, and there is evidence that even within the same community, different conclusions can be drawn from studies of the association between salt intake and BP.[22,23] Our results are however consistent with the relationship between DBP and sodium excretion in some earlier studies,[24,25] and between mean BP and sodium excretion also reported earlier.[26] SBP was however not associated with urinary sodium in this study, a phenomenon earlier reported by Cheung *et al*.[25] The absence of association or correlation between urinary sodium and both SBP and PP, contrasts with the presence of significant but weak association between urinary sodium and both DBP and MBP. Contrary to these findings, the WHO Cardiovascular Diseases and Alimentary Comparison (WHO-CARDIAC) study showed a significant association between both SBP and DBP and urinary sodium (though only in men and postmenopausal women).[27] This difference may be explained by the fact that we studied only premenopausal women and young men, as against the wider age spectrum in the WHO-CARDIAC study.

Appreciating the relationship between sodium and BP is difficult. Guyton had postulated that the maintenance of renal sodium excretion and sodium balance requires increased BP,[28] such that the relationship between the two is thought to be an important factor in the long-term control of BP. Human studies have however shown that the relationship between BP and urinary sodium excretion varies with age, gender, and BP state.[26,29,30] It is possible that increases in salt intake increase DBP and MBP which in turn increase urinary sodium excretion in a Guytonian fashion.

The inverse relationship between urinary potassium and BP reported by other workers[11-14] could not be confirmed by our study. Our findings are also at variance with the positive association reported between Na^+/K^+ ratio and BP,[11] but is however consistent with the reports of a lack of association between potassium intake and coronary events[15] and lack of association between urinary potassium and DBP.[25]

Gender-related differences in the process of hypertension are yet to be completely resolved. Though some reports have found urinary sodium excretion to be significantly higher[29] or significantly lower[31] in females than in males, our study found no significant difference in the urinary sodium or potassium concentration between the sexes. This could be because the females we studied were premenopausal and the males were even younger. Meneton *et al.* had noted that a reduced ability of the kidneys to excrete salt (either due to aging, disease or as a result of hormones) was intrinsic in the relationship between salt and BP.[1] Interestingly, our regression analysis shows that (even after correcting for age and anthropometric variables) sex was significantly associated with all the measures of BP, but not PP. Despite this, there was no significant difference between the mean values for the measures of BP between the sexes. This observation could be explained by the fact that while the females were significantly older than the males, the males were significantly heavier than the females. The distribution of these two factors that were associated with SBP, DBP, and MBP between the sexes, coupled with the "risk-advantage" that premenopausal women have over age-matched men,[17] may have brought the BP values of the different sexes to a point where they were statistically similar.

This study may be limited by a number of factors. First, salt intake was assessed from one overnight urine collection. This provides only a crude estimate of short-term salt intake.[32] However, there is evidence that both 24-hour and overnight sodium excretion estimate the daily intake reasonably well.[15,33] Second, we did not determine the phases of the menstrual cycle of the females that took part in the study. It is known that estrogen and progesterone levels vary between the phases of the menstrual cycle, and can influence sodium handling in the body, and thus affect BP.[18,19] However, due to cultural practices that regard menstruating women as "unclean," women in the locality we studied are often timid to talk about such matters. The females therefore could not volunteer the information we needed. The effect this would have had on our data is minimized, however, by the fact that in young normotensive females, the BP response to sodium is not affected by hormonal status.[28]

The strengths of this study include the description of data from an under-(or maybe un-) studied racial subgroup. Our sample population was also young, thereby allowing us rule out age-related confounding factors which may affect the relationship between BP and urinary electrolytes. This, however, may be responsible for the weak associations and correlation recorded in this study. Our sample size is also large, especially when viewed from the prism of cultural practices and belief systems in many African communities that make people hesitant to take part in this type of study, especially when no honoraria is given to participants. The weak correlations and associations however warrant a careful interpretation of the data presented in this report. It is however interesting that salt intake (as is inducible from urinary sodium concentration) is associated modestly with increased BP even in a population where salt consumption comes almost entirely from salt added to food while cooking or at the table. An earlier study in hypertensive Nigerians had shown that the higher the salt intake, the higher the BP in men.[34] These clearly imply that a well-planned campaign aimed at reducing the quantity of salt intake in this (and similar) population(s) may help in reducing morbidity and mortality from CVDs.

Conclusion

We studied the relationship between BP and urinary electrolytes in a young nonurban sub-Saharan African population, and found that urinary sodium (but not potassium) concentration was associated significantly and positively (albeit weakly) with DBP and MBP. Though a cautious interpretation of the data is warranted, they appear to support the hypothesis that dietary sodium intake is related to blood pressure elevations. Public health campaigns aimed at encouraging a reduction in the quantity of salt consumed in food is advocated.

References

1. Menton P, Jeunemaitre X, DeWardener HE, MacGregor GA. Links between dietary salt, renal salt handling, blood pressure, and cardiovascular disease. Physiol Rev 2005;85:679-715.
2. McCaron DA. Dietary sodium and cardiovascular and renal disease risk factors: Dark horse or phantom entry? Nephrol Dial Transplant 2008;23:2133-7.
3. McMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, *et al.* Blood pressure, stroke, and coronary heart diease. Part 1. Prolonged differences in blood pressure: Prospective observational studies corrected for the regression dilution bias. Lancet 1990;335:765-74.
4. Graudal NA, Galloe AM, Garred P. Effects of sodium restriction on blood pressure, rennin, aldosterone, catecholamines, cholesterols, and triglyceride: A meta-analysis. JAMA 1998;279:1383-91.
5. Mancilha-Carvallo JJ, deOlivveira R, Esposito RJ. Blood pressure and electrolyte excretion in Yanomamo Indians, an isolated population. J Hum Hypertens 1989;3:309-14.
6. He J, Tell GS, Tang YC, Mo PC, He GQ. Relation of electrolytes to blood pressure in men. The Yi people study. Hypertension 1991;17:327-32.
7. Alderman MH, Cohen H, Madhavan S. Dietary sodium intake and mortality: The National Health and Nutrition Examination Survey (NHANES 1). Lancet 1998;351:781-5.
8. Tunstall-Pedoe H, Woodward M, Tavendale R, A'Brook R, McCluskey MK. Comparison of the prediction by 27 different factors of coronary heart disease

and death in men and women of the Scottish heart health study: Cohort study. BMJ 1997;315:722-9.
9. Alderman MH, Madhavan S, Cohen H, Sealey JE and Laragh JH. Low sodium is associated with greater risk of myocardial infarction among treated hypertensive men. Hypertension 1995;25:1144-52.
10. Cohen HW, Halpern SM, Fang J, Alderman MH. Sodium intake and mortality in the NHANES II follow-up study. Am J Med 2006;119:275.e7-e14.
11. Khaw KT, Barrett-Connor E. The association between blood pressure, age and dietary sodium and potassium: A population study. Circulation 1988;77:53-61.
12. Whelton PK, He J, Cutler JA, Brancati FL, Appel IJ, Follmann D, *et al.* Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials. JAMA 1997;277:1624-32.
13. Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: A meta-analysis of randomized trials. J Hum Hypertens 2003; 17: 471-80
14. Khaw KT, Barrett-Connor E. Dietary potassium and stroke-associated mortality: 1. 12 year prospective population study. N Engl J Med 1987;316:235-40.
15. Geleijnse JM, Witteman JCM, Stijnen T, Kloos MW, Hofman A, Grobbee DE. Sodium and potassium intake and risk of cardiovascular events and all-cause mortality: The Rotherdam study. Eur J Epidemiol 2007;22:763-70.
16. Eaker ED, Chesebro JA, Cacks FM, Wenger NK, Whisnat JP, Winston M. American Hearts Association medical/scientific statement: Cardiovascular disease in women. Special report. Circulation 1993;88:1999-2000.
17. Kafoneck SD. Post menopausal hormone replacement therapy and cardiovascular risk reduction. Drugs 1994;47 (Suppl 2):16-24.
18. Groffon JT, Dustan H, Share L, Brooks DP. Vasopressin secretion in normotensive black and white men and women on normal and low sodium diets. J Endocrinol 1986;108:191-9.
19. Lee Ma, Bohn M, Paul M, Ganten D. Tissue rennin-andiotensin systems. Their role in cardiovascular disease. Circulation 1993;87:V7-13.
20. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholestrol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) JAMA. 2001;285: 2486-97.
21. Langhoff E, Steinees L Potentiometric analysis for sodium and potassium in biological fluids. Clin Chem 1982;28:170-2.
22. Beard TC, Blizzard L, O'Brien DJ, Dyer T. Association between blood pressure and dietary factors in the dietary and nutritional survey of British adults. Arch Intern Med 1997;152:243-8.
23. Smith WC, Crombie IK, Tavendale RT, Gulland SK, Tunstall-Pedoe H. Urinary electrolyte excretion, alcohol consumption and blood pressure in the Scottish heart health study. BMJ 1988;297:329-30.
24. Intersalt: An international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. Intersalt Coopreative Research Group. BMJ 1988;297:319-28.
25. Cheung BM, Ito SP, Cheung AH, Lau CP. Diastolic blood pressure is related to urinary sodium excretion in hypertensive Chineese patients. QJM 2000;93:163- 8.
26. Centozona L, Castoldi G, Chianca R, Busca G, Golin R, Zanchetti A, *et al.* Short-term analysis of the relationship between blood pressure and sodium excretion in normotensive subjects. Clin Sci 2000;98:495-500.
27. Yamori Y, Nara Y, Mizushima S, Sawamura M, Horie R. Nutritional factors for stroke and major cardiovascular diseases: International epidemiological comparison of dietary prevention. Health Rep 1994;6:22-7.
28. Guyton AC. Renal function curves: A key to understanding the pathogenesis of hypertension. Hypertension 1987;10:1-6.
29. Jankunas R, Volbekas V, Stakisaitis D, Driziene Z. Urinary sodium: Gender-related differences and relation to blood pressure in adolescents. Acta Med Lithuanica 2002;9:86-92.
30. Harshfield GA, Alpert BS, Pulliam DA, Willey ES, Somes GW, Stepleton FB. Sodium excretion and racial differences in ambulatory blood pressure patterns. Hypertension 1991;18:813-8.
31. Dyer AR, Stamler R, Grimm R, Stamler J, Berman R, Gosch FC, *et al.* Do hypertensive patients have a different diurnal pattern of electrolyte excretion? Hypertension 1987;10:417-24.
32. Bruce NG, Cook DG, Sharper AG, Ratcliffe JG. Casual urine concentrations of sodium, potassium and creatinine in population studies of blood pressure. J Hum Hypertens 1990;4:597-602.
33. Luft FC, Fineberg NS, Sloan RS. Estimating dietary sodium intake in individuals receiving a randomly fluctuating intake. Hypertension 1982;4:805-8.
34. Olubodun JOB, Akingbade OA, Abiola OO. Salt intake and blood pressure in Nigerian hypertensive patients. Int J Cardiol 1997;59:185-8.

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