Patterns of Sodium and Potassium Excretion and Blood Pressure in the African Diaspora

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Abstract

Habitual levels of dietary sodium and potassium are correlated with age-related increases in blood pressure (BP) and likely play a role in this phenomenon. Although extensive published evidence exists from randomized trials, relatively few large-scale community surveys with multiple 24-hour urine collections have been reported. We obtained three 24-hour samples on 2,704 individuals from Nigeria, Jamaica and the US to evaluate patterns of intake and within-person relationships to blood pressure. The average (±s.d.) age and weight of participants across all three sites were 39.9±8.6 years and 76.1±21.2 kg, respectively, and 55% of the total participants were females. Sodium excretion increased across the East-West gradient (e.g., 123.9±54.6, 134.1±48.8, 176.6±71.0 (±s.d.) mmol, Nigeria, Jamaica and US, respectively), while potassium was essentially unchanged (e.g., 46.3±22.9, 40.7±16.1, 44.7±16.4 (±s.d.) mmol, respectively). In multivariate analyses both sodium (positively) and potassium (negatively) were strongly correlated with blood pressure (p < 0.001); quantitatively the association was stronger, and more consistent in each site individually, for potassium. Within-population day-to-day variation was also greater for sodium than for potassium. Among each population group a significant correlation was observed between sodium and urine volume, supporting the prior finding of sodium as a determinant of fluid intake in free-living individuals. These data confirm the consistency with the possible role of dietary electrolytes as hypertension risk factors, reinforcing the relevance of potassium in these populations.

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Keywords

blood pressure; sodium excretion; potassium excretion; African Diaspora

Introduction

A broad base of evidence from epidemiologic studies, clinical trials and animal experimental data support the causal role of both sodium and potassium in the development of high blood pressure (BP).[1–3] Particular interest also exists in the relative impact of sodium and potassium in populations of African origin.[4, 5] High rates of hypertension are observed in Afro-origin groups in much of the western hemisphere and the UK,[6] and hypertension is now emerging as a major problem in sub-Saharan Africa as well.[7] As public health campaigns to reduce sodium consumption start to gain momentum; data on current levels of intake are necessary to help formulate targets and to monitor trends.[8] The inter-relations between sodium, potassium and other components of the diet also require further study. Previous survey data has suggested that daily intake of sodium is correlated with fluid volume, and this relationship has also been documented in a controlled clinical trial.[9, 10] In the setting where a substantial proportion of fluids are heavily sugar sweetened this could increase the risk of positive energy balance.[11, 12] The generalizability of the sodium-fluid volume relationship in free-living individuals deserves further study.

A marked gradient in hypertension prevalence exists across the course of the historical African Diaspora from West Africa to the Caribbean and North America. [13] This gradient is consistent with the pattern of all the lifestyle factors that are known to influence risk of hypertension, including an increase in sodium intake.[13, 14] Randomized trials have also shown a similar response in BP to sodium reduction in Nigerians and Jamaicans as seen in African Americans.[15] Because obesity, physical inactivity and other known risk factors are all at higher levels in the US, however, complicated interactions among these factors may be occurring and it is difficult to quantify the role of electrolytes in isolation.

To further clarify the distributions and impact of dietary sodium and potassium on BP in the African Diaspora we conducted a comparative study of community-based samples from southwest Nigeria, Kingston, Jamaica and metropolitan Chicago, Illinois. Individual electrolyte intake varies widely from day to day,[16] therefore multiple 24-hour samples were collected to characterize the associations at the individual level in each cultural setting. We hypothesized an East-West gradient in electrolytes excretions.

Methods

Participant recruitment

Participants were recruited from residential communities by local trained research staff. A screening visit was used to determine if the participants met study criteria. In order that BP could be evaluated as a continuous trait only persons not on treatment for hypertension were enrolled. Pregnant women were also excluded from participating in the study. To insure the inclusion of sufficient numbers of participants with obesity we recruited from the upper and

lower tertiles of the site-specific body mass index (BMI) in Nigeria and Jamaica. The protocol was reviewed and approved by the Institutional Review Board at Loyola University Medical Center, the University Hospital of the West Indies/University of the West Indies/ Faculty of Medical Sciences Ethics Committee, Mona, Kingston, Jamaica, and the Joint Ethical Committee of the University of Ibadan/University College Hospital, Ibadan, Nigeria. The consent process was presented in English or Yoruba (Nigeria) and written informed consent was obtained from all participants.

Measurement Procedures

Height and weight were measured using a beam balance and a stadiometer, as previously described.[13] BP was measured 4 times in the brachial fossa in the sitting position with an automated device (Omron HEM-412C) previously evaluated in our field settings,[17] and the mean of the last two measurements was used in the analysis. At the initial clinic visit participants' willingness to complete three 24-hour urine collections was ascertained and they were instructed on the procedures and provided collection materials. The first morning void was excluded and all urine, including the next first morning void, was collected. Collection days could be, but were not necessarily, consecutive. Eligible participants were then requested to complete 3 24-hour urine collections, recording the time of initiation and the ending. Data were adjusted to 24-hours. Sodium and potassium were measured using flame photometry. As a result of budgetary constraints urinary creatinine was measured only among participants from Jamaica.

Statistical analysis

Each participant's sodium and potassium excretion values were calculated as the product of concentrations and total urine volume corrected to 24 hours. Average total excretion was computed as the mean of each participant's values for the three collections. As part of the data cleaning process, any collection for which sodium and potassium excretion values did not meet the following criteria was discarded on the assumption of incomplete urine collection:

$$\mathrm{Na} \geq \lambda$$
$$K \geq (\lambda/\mathrm{mean\,Na}) * \mathrm{mean}\,K$$

Na and K are respectively the excretion of sodium and potassium of each collection; mean Na and K represent mean excretion of an individual's three collections for sodium and potassium, respectively; λ was chosen to correspond to the bottom 1st percentile of sodium excretion distribution within each site and was roughly equal to 40 for Maywood, 30 for Jamaica and 20 for Nigeria. After applying the above criteria, mean excretion for sodium and potassium were then recalculated for every participant while participants having less than two collection values for sodium or potassium (25 out of 946 for Maywood, 24 out of 1003 for Jamaica and 25 out of 829 for Nigeria) were excluded from the analytical dataset. The final analytical dataset used for subsequent analyses consisted of 2704 adults including 921 from Maywood site, 979 from Jamaican site and 804 from Nigerian site. As a comparison with the above criteria for identifying incomplete 24-hour urine, we applied two creatinine-based strategies for the Jamaican participants for whom creatinine excretion data

were available. The first strategy was that developed by the World Health Organization (WHO)[18] and it classified subjects with a ratio of urinary creatinine (mg/d) to body weight (kg) of <10.8 or >25.2 as having incomplete urine. The second strategy was that by Malekshah *et al*,[19] it classified subjects with a ratio of urinary creatinine (mg/d) to body weight (kg) of <11.0 or >20.0 as having incomplete urine. We observed that the number of samples identified as having complete urine by these 2 strategies differed by just 5 from the number identified through the criteria that we used. The 5 samples were classified by our criteria as having incomplete urine. The correlation of excreted sodium through the WHO and Malekshah *et al* strategies versus our criteria was 0.9982.

All statistical analyses were performed using SAS 9.1 (SAS Institute, Cary, NC, USA). Using the pooled data, associations between BP and the urinary electrolytes were examined via linear regression of BP on urinary sodium, potassium and sodium-potassium ratio with adjustment for age, sex, age*sex interaction and body size. We included both ions in the regression models with BP as the outcome variable. No non-linear term was included in the fitted model because model with only linear terms provided better fit to the data. The same analyses were also performed using each site-specific data separately. We used the standard normal transformed urinary electrolytes in all regression analyses because the raw data indicated evidence of skewness.

Results

Descriptive statistics

Characteristics of the study participants are presented in Table 1. In both Table 1 and following text, mean values are presented with standard deviations (±s.d.). Mean age and BMI in the pooled data were 39.9±8.6 years and 27.0±7.4 kg/m^2, respectively. The age distribution was similar among sites; however mean BMI ranged from 31 in Jamaica to 23 in Nigeria. Female participants constituted 55% of the pooled study sample, whereas site-by-site proportions of female participants ranged from 38% for Maywood, 52% for Nigeria to 75% for Jamaica. Participants in Maywood were on average slightly older than their counterparts in Jamaica and Nigeria with the mean ages being 41.9±7.5, 39.6±8.1 and 38.1±9.7 years, respectively. Participants in the Jamaican site had higher BMI (30.9±7.0 kg/m^2) compared to those in Maywood (26.8±7.7 kg/m^2) and Nigeria (22.5±4.4 kg/m^2) sites, primarily as a consequence of the gender distribution. All sites were significantly different (p-values < 0.05) from each other in terms of mean age and BMI.

Average BP across sites was 120.9±17.2 and 73.5±11.9 mmHg respectively for systolic and diastolic, with higher values being observed in Maywood. Mean urinary sodium and potassium were 145.5±63.1 and 43.7±18.6 mmol/24 hours, respectively. Maywood participants had significantly higher average 24 hours urinary sodium excretion compared to participants from the other sites (Maywood: 176.6±71.0 mmol, Jamaica: 134.1±48.8 mmol and Nigeria: 123.9±54.6 mmol); Nigerian participants had slightly higher urinary potassium excretions. Mean sodium-to-potassium ratio was 3.6±1.5 overall and ranged from 4.1±1.4 in Maywood to 2.9±1.2 in Nigeria and 3.6±1.5 in Jamaica. Compared with other sites, urinary potassium was significantly lower in Jamaican site, whereas both urinary sodium and sodium to potassium ratio were significantly different (p < 0.05) among the three sites. The

intraclass correlation coefficient (ICC) for urinary sodium, which reflects reproducibility, was 0.38, 0.41 and 0.44 for US, Jamaican and Nigerian sites, respectively. Urinary potassium ICC was 0.35 for Maywood, 0.56 for Jamaica and 0.37 for Nigeria. At each site and in the pooled data, males had significantly higher (p-values < 0.05) electrolyte excretion than females.

Correlation coefficients between urinary electrolytes, urine volume, BMI and body weight are displayed in Table 2. In this analysis it was our intent to examine the pattern of relationships linking electrolytes, fluid volume, and body size. In all sites and in the pooled sample urinary sodium was better correlated with volume of urine than potassium (differences between the correlations are significant with p < .01). Both urinary sodium and potassium were also more strongly associated with body weight than BMI (p < .01), as would be anticipated since weight is a better proxy for energy intake than BMI.[20, 21] The inter-relation of potassium with body size was notably weaker in Nigeria.

The relationship between sodium / potassium and BP by BMI categories are shown in Figure 1. Participants in higher BMI categories had higher urinary sodium excretion, although the absolute differences were small and apparent only for the open-ended extreme quantiles (Figure 1). A similar modest relationship was observed between BMI and BP (Figure 1). The pattern of distribution of urinary potassium and BP appeared similar for all BMI categories. There was evidence of moderate positive skewness (<2.0) for both sodium and potassium when expressed per kg of body weight (Figure 2).

Blood pressure and urinary sodium / potassium in pooled data

Coefficients from regression analyses relating urinary sodium and potassium to blood pressure as the outcome variable are presented in Table 3. For both systolic and diastolic BP three separate models were fitted – without adjustment for BMI or weight; with adjustment for BMI; and with adjustment for body weight. All models included age, sex, age*sex interaction, and two dummy variables for site. The regression coefficients indicated positive relationship between BP and urinary sodium with or without adjustment for BMI or body weight in the presence of urinary potassium. For 1 standard deviation (SD) change in urinary sodium (i.e., 63 mmol), there was an associated change of 1.13 mmHg (p=0.0048) or 1.01 mmHg (p=0.0125) in SBP in models with adjustment for BMI or body weight, respectively. Similarly, for every 1 SD change in urinary sodium in models with BMI or body weight, there was an associated 0.41 or 0.30 mmHg (p>0.05) change in DBP.

The regression coefficients for urinary potassium on BP were inverse in the presence of urinary sodium irrespective of whether BMI or body weight was adjusted for. For every 1 SD change in urinary potassium (i.e., 19 mmol), there was a change of 1.55 mmHg in SBP (p<0.0001) and 0.96 mmHg in DBP (p=0.0002) (Table 3).

Sodium-to-potassium ratio was positively and significantly related to both SBP and DBP with or without adjustment for BMI or body weight (Table 3). For each unit change in sodium-to-potassium ratio, there are at least 0.99 mmHg and 0.54 mmHg changes in SBP and DBP, respectively. As observed in the models for the electrolytes, the model with

weight explained slightly more of the variation than the model with BMI (e.g., for SBP, 14.9% vs. 14.7%, and DBP, 15.8% vs. 15.4%) (Table 3).

Electrolyte-blood pressure relationships by site

The electrolyte-BP associations were less consistent for each site separately. The relationship for sodium was only significant (p<0.05) in Maywood for SBP (Table 4). The strength of the relation between SBP and sodium excretion based on the coefficients expressed as mmHg per mmol increased from Nigeria to Maywood with Jamaica being intermediate. The pattern was however opposite for DBP, with Maywood and Nigeria having the lowest and highest coefficients, respectively (Table 5). The relationship between potassium and BP was significant in Maywood (p<0.005) (Tables 4 & 5). Sodium-to-potassium ratio was positively related to both SBP and DBP in all sites and again significant in Maywood (p<0.05) for both SBP and DBP; in Nigeria (p<0.05) for DBP (Tables 4 & 5).

Tables 4 and 5 also show sodium-blood pressure relationships within each site when urinary potassium was not included in the model. Estimates of potassium-blood pressure relationships when urinary sodium was not included in the model are also reported. As can be seen in Table 4, there was a significant relationship between sodium intake and blood pressure in the absence of potassium and without adjustment for BMI or body weight in all sites with the exception of Nigeria. Test of differences indicated a moderate significant (p < 0.043) difference between the regression coefficients for the Nigerian and Jamaican sites. While this seems to indicate that the Jamaican population could be more sensitive to dietary salt intake than the Nigerian population, we however note that we have limited power to investigate true interactions between the sites. Across sites, the regression coefficients indicated a positive relationship between SBP and urinary sodium in the absence of urinary potassium with or without adjustment for BMI or body weight (Table 3). Similarly, the coefficients indicated an inverse relationship between BP and urinary potassium across sites in the absence of urinary sodium (Table 3). The strength of these relationships was, however, weaker than that observed when the electrolytes were jointly modeled (Table 3). Covariates-adjusted site-specific regression lines for the relationship between BP and the electrolytes are displayed in Figure 3.

Discussion

Hypertension is more frequent in persons of African descent in some regions of the Western Hemisphere and Europe, however, the underlying explanation for this important health differential remains elusive.[22, 23] The role of dietary electrolytes has been a major focus for investigations on the cause of the higher blood pressures in U.S. blacks compared to whites, along with psychosocial factors.[22, 23] Relatively little research has attempted to examine the relationship between sodium and potassium intake in West Africa or other parts of the African Diaspora. Our previous international study demonstrated a consistent increase in blood pressure and hypertension prevalence from West Africa (Nigeria, Cameroon), to the Caribbean (Jamaica, Barbados, St. Lucia) and the U.S.[13] Intake of sodium and potassium varies markedly day-to-day, however, and in the original prevalence survey the goal was to estimate population mean values, with only a single 24-hour urine on a sub-set of the total

sample.[13] For the analyses presented here we recruited a new sample of untreated middle-aged individuals and required 3 complete collections for inclusion in the study. As observed in previous cross-sectional surveys, the blood pressure-sodium association was weak, and became non-significant in all 3 sites after inclusion of BMI in the regression model. For potassium the association remained significant after adjustment for body size in Maywood (DBP) and Nigeria (SBP). In analyses that considered the combined effect of sodium and potassium, however, the associations were significant for at least one ion and blood pressure in all three sites, whether the electrolytes were entered separately in the regression model or combined as a ratio. The associations were more consistent for potassium ($p < 0.01$). While the statistical power to examine differences in effect size by site was limited, it would also appear that similar electrolyte-blood pressure relationships exist across these populations with very different background prevalences of hypertension and other cardiovascular risk factors, although the findings in Jamaica were less clear-cut.

Early in the course of research on the relationship between sodium and blood pressure it was recognized that intra-individual variability was very substantial, relative to inter-individual variation, thereby biasing associations toward the null.[16] In a study of adolescents with 7 consecutive 24-hour collections to reduce this bias correlations were observed between sodium and blood pressure in the range of 0.10 – 0.12.[24] This finding was generally replicated in INTERSALT, which also confirmed the relationship by the population level.[25] Whether this relationship reflects the "true" habitual level or simply short-term fluctuations in blood pressure with changing dietary intake is not known. The voluminous data from randomized trials are the primary basis for the consensus that dietary electrolytes play a crucial role in the causation of hypertension.[1–4, 26–29] Our previous comparative trial in Nigeria and Jamaica demonstrated quantitatively similar responses to sodium reduction in normotensive individuals.[30]

The most notable outcome from the present analyses is an increase in the association with blood pressure when both sodium and potassium were considered jointly. A substantial body of literature has demonstrated differences in the effect of oral potassium on blood pressure among blacks in the U.S. compared to whites.[31–35] In addition, reduced urinary excretion of potassium has been noted in population surveys in the U.S. and South Africa, even under conditions of potassium supplementation.[33–35] These observations have been linked to renal mechanisms via the renin-angiotensin system, and have also been proposed as an explanation of the differential responsiveness of whites and blacks to diuretics.[31] Although previous small studies suggested that blacks had greater non-renal excretion of potassium, a recent tightly controlled metabolic study found no difference in potassium loss by sweat or feces, while confirming lower urinary excretion.[34] Clearly a long-term net positive potassium balance is not possible so these findings cannot be explained. The 6 week duration of the recent supplementation study further rules out the possibility of depleted potassium stores at baseline as the cause. Whatever the physiologic explanation, a meta-analysis of feeding trials confirmed that blacks experience larger BP reduction with potassium supplementation compared to whites.[29]

An unresolved problem in the analysis of survey data on dietary electrolytes and BP is the appropriateness of adjustment for body size. As observed in our data, larger body size is

associated with greater sodium intake, which of course reflects greater calorie intake. This co-linearity cannot be eliminated and it is entirely possible that the quantity of sodium/ potassium presented to the kidney is the important causal factor and the association of body size with BP is in fact confounded by the electrolytes, at least in part. If that were the case, adding body size to the analysis would represent "over adjustment", resulting in an underestimate of the true effect of sodium in particular. Since body size is measured with orders of magnitude greater accuracy then the epidemiologic analyses can only approximate the underlying quantitative relationships.[12] In a test for the relationship between body size and blood pressure, as in the reports by Kotchen et al[36] we also observed a plateau effect for the relationship between body size and blood pressure (data not shown) and therefore have chosen not to include it in the regression models.

In summary, within the precision of the measurement process, we have shown an association between sodium and potassium in all regions of the West African Diaspora. As previously reported, we find a somewhat more consistent effect of potassium compared to sodium, and an inter-dependency between the two. No effect modification by BMI or geographic region could be appreciated. Substantial reduction in cardiovascular disease burden could be achieved with alteration in electrolyte composition of the contemporary diet.[37, 38] The data reported here suggest that public health campaigns to prevent hypertension can use a consistent message in all of the countries represented in this study. Improved survey methods are also needed to insure that reliable population estimates can be obtained to monitor trends as campaigns are mounted to reduce intake.

Acknowledgments

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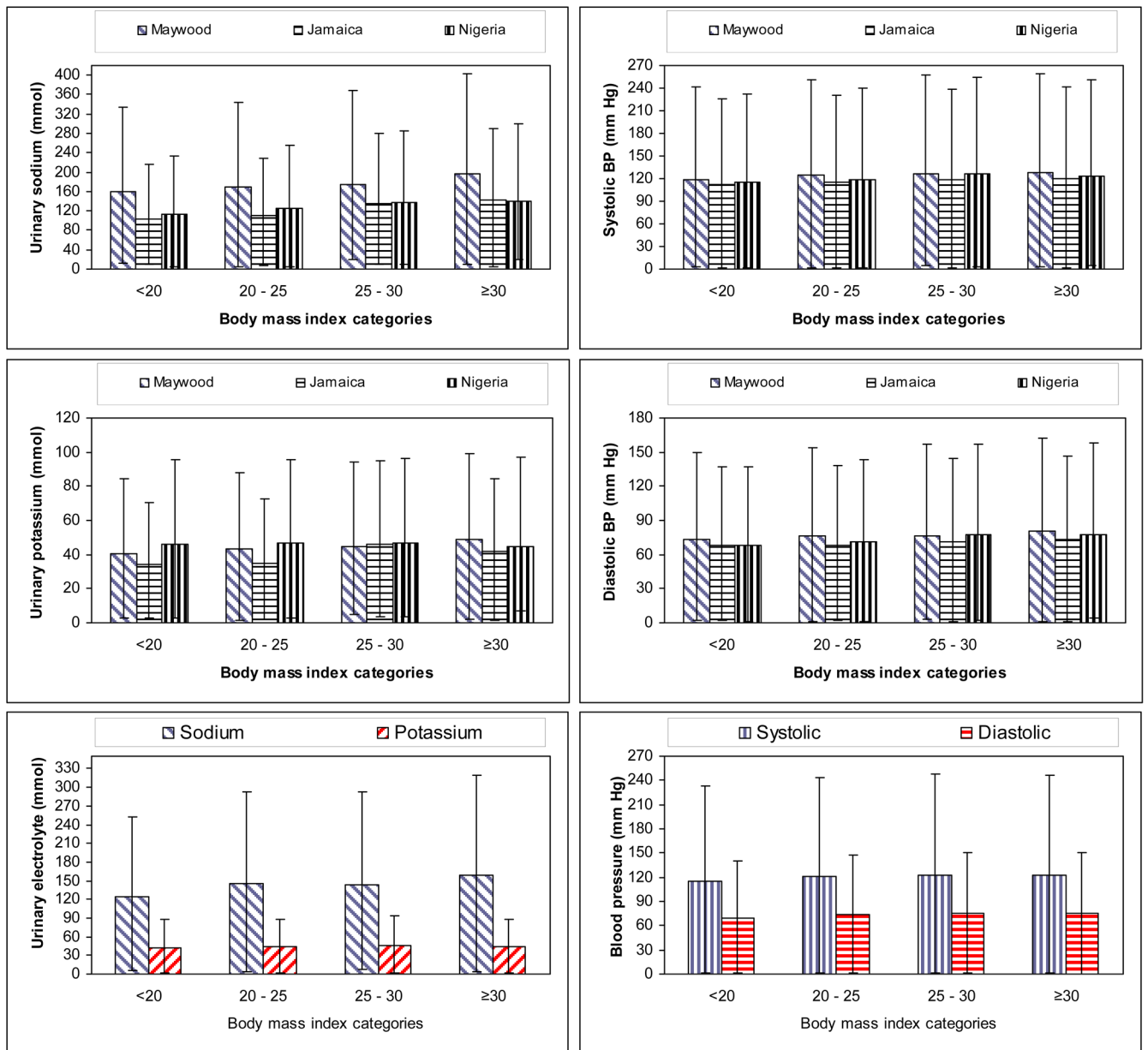
Figure 1.
Distribution of urinary electrolytes (left column) and blood pressure (right column) by body mass categories within sites (top and middle rows) and across sites (bottom row)
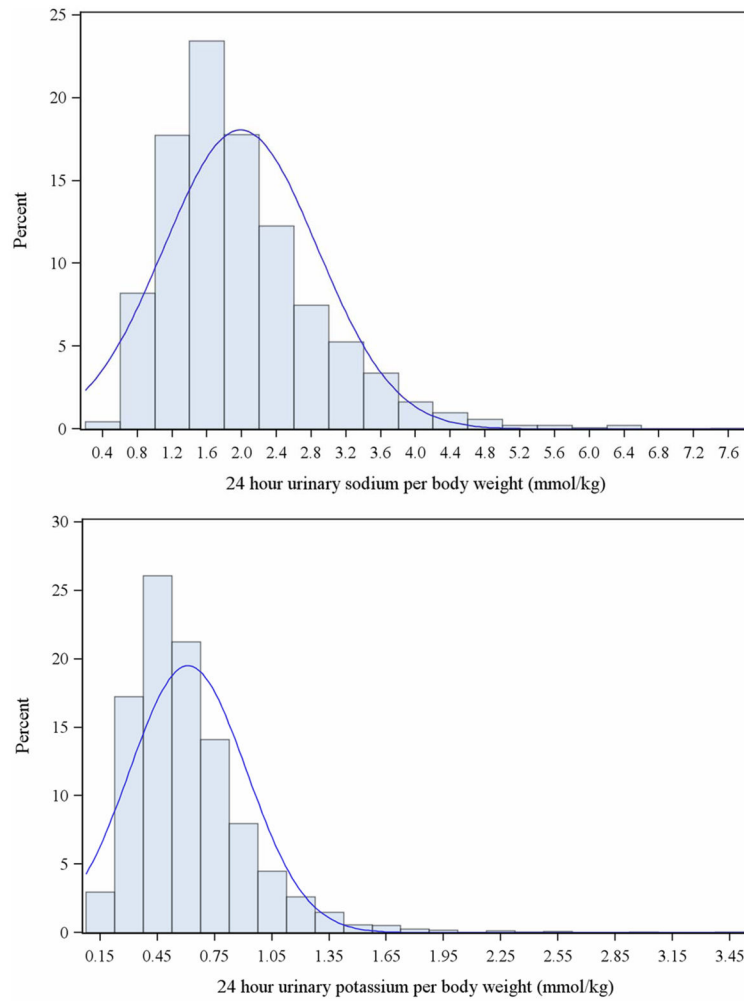
Figure 2.
Variability of per body weight urinary sodium (Top) and potassium (Bottom)
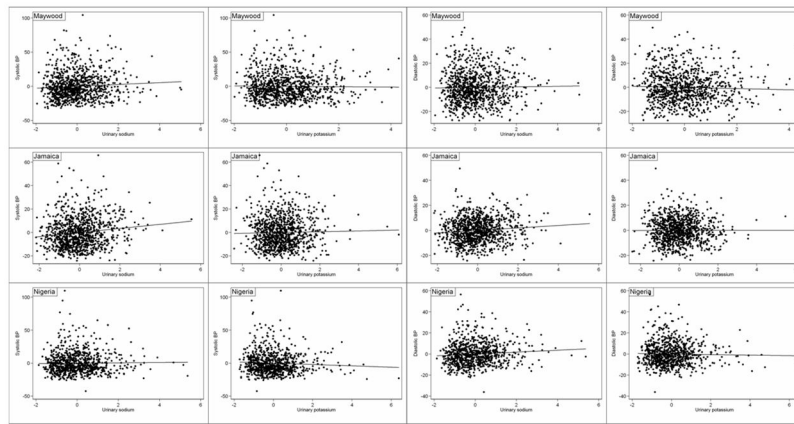
Figure 3.
Site-specific regression lines (adjusted for age, sex and age-by-sex interaction) for systolic (columns 1 and 2) and diastolic (columns 3 and 4) blood pressure against standardized urinary sodium (columns 1 and 3) and potassium (columns 2 and 4)

Table 1

Characteristics of study participants

	Pooled	Maywood	Jamaica	Nigeria
Number (% females)	2704 (55)	921 (38)	979 (75)	804 (52)
Age (years)[†]	39.9 ± 8.6	41.9 ± 7.5	39.6 ± 8.1	38.1 ± 9.7
Weight (kg)[†]	76.1 ± 21.2	78.6 ± 22.1	85.4 ± 19.6	61.8 ± 12.1
Height (cm)	168.0 ± 8.8	171.6 ± 9.0[‡]	166.4 ± 8.1	165.8 ± 8.1
BMI (kg/m^2)[†]	27.0 ± 7.4	26.8 ± 7.7	30.9 ± 7.0	22.5 ± 4.4
Systolic BP (mmHg)	120.9 ± 17.2	125.0 ± 19.9[‡]	118.5 ± 13.2	119.2 ± 17.3
Diastolic BP (mmHg)	73.5 ± 11.9	77.2 ± 13.3[‡]	71.5 ± 9.7	71.5 ± 11.7
Urine volume (L/24 hours)[†]	1.4 ± 0.6	1.6 ± 0.5	1.1 ± 0.5	1.5 ± 0.6
Urinary sodium (mmol/24 hours)[†]	145.5 ± 63.1	176.6 ± 71.0	134.1 ± 48.8	123.9 ± 54.6
Urinary potassium (mmol/24 hours)	43.7 ± 18.6	44.7 ± 16.4	40.7 ± 16.1[‡]	46.3 ± 22.9
Urinary sodium : potassium ratio[†]	3.6 ± 1.5	4.1 ± 1.4	3.6 ± 1.5	2.9 ± 1.2

[†] Estimates significantly different among all sites;

[‡] Site estimate significantly different from those of other two sites;

Values are mean ± SD or count (percentage). BP, blood pressure

Table 2

Correlations between urinary electrolytes, BMI and body weight

		Sodium	Potassium	Urine volume	Weight
Pooled:	Potassium	0.503			
	Urine volume	0.389	0.259		
	Weight	0.303	0.101	0.032[†]	
	BMI	0.209	0.047	−0.025[†]	0.922
Maywood:	Potassium	0.608			
	Urine volume	0.347	0.273		
	Weight	0.266	0.258	0.138	
	BMI	0.219	0.207	0.094	0.923
Jamaica:	Potassium	0.417			
	Urine volume	0.544	0.413		
	Weight	0.376	0.179	0.200	
	BMI	0.281	0.128	0.142	0.908
Nigeria:	Potassium	0.608			
	Urine volume	0.265	0.076		
	Weight	0.253	0.098	0.168	
	BMI	0.174	0.013[†]	0.167	0.855

[†]Correlation not significant (p>0.05), all others are significant

Table 3

Association of blood pressure and urinary electrolytes in pooled data

Intercept	Coefficients for the following variables in the model[//]					Adjusted R² (%)	p - value		
	Na[†]	K[†]	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
Systolic blood pressure									
113.43	2.03	–1.48				11.4	<.0001	0.0001	
101.88	1.13	–1.50		0.50		14.6	0.0048	<.0001	
100.32	1.01	–1.55			0.18	14.9	0.0125	<.0001	
114.30	1.19					11.0	0.0004		
102.81	0.28			0.50		14.1	0.4126		
101.33	0.13				0.18	14.4	0.6968		
114.68		–0.45				10.6		0.1597	
101.84		–0.96		0.53		14.4		0.0024	
100.15		–1.07			0.19	14.7		0.0007	
109.07			1.24			11.5			<.0001
98.62			0.99	0.49		14.7			<.0001
97.24			0.99		0.17	14.9			<.0001
Diastolic blood pressure									
65.69	1.13	–0.91				11.0	<.0001	0.0005	
56.29	0.41	–0.93		0.41		15.5	0.1430	0.0003	
54.96	0.30	–0.96			0.15	15.9	0.2829	0.0002	
66.23	0.62					10.7	0.0080		
56.86	–0.12			0.41		15.1	0.6140		
55.59	–0.24				0.15	15.4	0.3049		
66.40		–0.33				10.5		0.1358	
56.28		–0.73		0.42		15.4		0.0008	
54.91		–0.82			0.15	15.8		0.0002	
63.06			0.74			11.1			<.0001

Intercept	Coefficients for the following variables in the model[¶]				Adjusted R² (%)	p - value		
	Na[†]	K[†]	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
54.70			0.54	0.39		15.4			0.0006
53.56			0.54		0.14	15.8			0.0006

[¶] Age, sex, site and age-by-sex interaction were included in all models;

[†] Coefficients expressed as mmHg of blood pressure per 1 standard deviation change in urinary sodium (63.1 mmol) and potassium (18.6 mmol); Na, urinary sodium; K, urinary potassium; Na:K, ratio of urinary sodium topotassium; R^2, coefficient of determination

Table 4

Association of systolic blood pressure and urinary electrolytes by site

Intercept	Coefficients for the following variables in the model∥					Adjusted R² (%)	p - value		
	Na†	K†	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
Maywood									
111.04	2.41	−1.73				8.3	0.0026	0.0294	
97.52	1.71	−2.17		0.50		11.5	0.0307	0.0058	
95.41	1.69	−2.32			0.18	11.9	0.0325	0.0032	
110.69	1.35					8.0	0.0334		
97.71	0.44			0.48		10.8	0.4964		
95.86	0.34				0.17	11.1	0.5961		
110.53		−0.28				7.5		0.6616	
96.40		−1.18		0.53		11.1		0.0651	
94.25		−1.35			0.19	11.5		0.0357	
104.63			1.55			8.7			0.0006
92.33			1.40	0.48		11.8			0.0016
90.44			1.42		0.17	12.1			0.0013
Jamaica									
107.93	1.94	−0.48				8.2	<.0001	0.3108	
98.94	0.93	−0.44		0.42		12.1	0.0477	0.3392	
97.36	0.80	−0.49			0.15	12.4	0.0911	0.2905	
108.82	1.75					8.2	<.0001		
99.75	0.75			0.42		12.1	0.0807		
98.27	0.61				0.15	12.4	0.1637		
108.72		0.34				6.5		0.4339	
98.41		−0.09		0.46		11.8		0.8404	
96.74		−0.19			0.16	12.2		0.6542	
103.58			0.82			7.3			0.0034

Intercept	Coefficients for the following variables in the model¶					Adjusted R² (%)	p - value		
	Na†	K†	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
96.65			0.43	0.44		12.0			0.1182
95.28			0.43		0.16	12.4			0.1164
Nigeria									
115.60	1.39	−1.86				11.2	0.0603	0.0125	
99.91	0.35	−1.40		0.83		15.0	0.6382	0.0552	
98.36	0.10	−1.40			0.31	15.3	0.8927	0.0559	
116.17	0.24					10.6	0.6762		
99.81	−0.54			0.86		14.7	0.3533		
98.25	−0.79				0.32	15.0	0.1800		
116.16		−0.99				10.9		0.0889	
99.76		−1.19		0.84		15.0		0.0377	
98.29		−1.34			0.31	15.4		0.0197	
111.09			1.44			11.4			0.0058
97.29			1.00	0.79		15.0			0.0511
96.20			0.99		0.28	15.2			0.0540

¶ Age, sex and age-by-sex interaction were included in all models;

† Coefficients expressed as mmHg of blood pressure per 1 standard deviation change in urinary sodium (Maywood: 71.0, Jamaica: 48.8, Nigeria: 54.6 mmol) and potassium (Maywood: 16.4, Jamaica: 16.1, Nigeria: 22.9 mmol); Na, urinary sodium; K, urinary potassium; Na:K, ratio of urinary sodium to potassium; R^2, coefficient of determination

Table 5

Association of diastolic blood pressure and urinary electrolytes by site

Intercept	Coefficients for the following variables in the model‖					Adjusted R² (%)	p - value		
	Na†	K†	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
Maywood									
66.92	0.85	−1.00				4.6	0.1179	0.0640	
54.84	0.23	−1.39		0.45		10.3	0.6634	0.0084	
53.04	0.22	−1.52			0.16	10.9	0.6851	0.0039	
66.72	0.24					4.3	0.5802		
54.96	−0.59			0.43		9.7	0.1767		
53.33	−0.67				0.15	10.2	0.1227		
66.74		−0.49				4.4		0.2554	
54.69		−1.26		0.45		10.3		0.0034	
52.89		−1.40			0.16	11.0		0.0011	
64.01			0.71			4.8			0.0215
53.55			0.58	0.41		9.9			0.0516
52.02			0.60		0.14	10.3			0.0446
Jamaica									
62.91	1.19	−0.50				3.5	0.0005	0.1609	
57.37	0.57	−0.48		0.26		6.2	0.1101	0.1747	
56.26	0.47	−0.50			0.10	6.6	0.1867	0.1496	
63.84	0.99					3.4	0.0015		
58.25	0.38			0.26		6.1	0.2453		
57.21	0.28				0.10	6.5	0.4035		
63.40	0.00					2.4		0.9898	
57.05	−0.26			0.28		6.1		0.4245	
55.90	−0.33				0.10	6.5		0.3101	
60.28			0.57			3.2			0.0062

Intercept	Coefficients for the following variables in the model[¶]					Adjusted R² (%)	p - value		
	Na[†]	K[†]	Na:K	BMI (kg/m²)	Weight (kg)		Na	K	Na:K
56.11			0.34	0.26		6.3			0.1030
55.21			0.34		0.10	6.7			0.1056
Nigeria									
59.73	1.74	−1.35				13.8	0.0004	0.0065	
45.18	0.77	−0.93		0.77		20.9	0.1105	0.0519	
43.74	0.54	−0.92			0.28	21.5	0.2654	0.0525	
60.15	0.90					13.1	0.0199		
45.11	0.19			0.79		20.7	0.6247		
43.66	−0.04				0.29	21.3	0.9078		
60.43		−0.27				12.6		0.4928	
44.84		−0.45		0.80		20.8		0.2248	
43.39		−0.59			0.29	21.5		0.1114	
56.41			1.13			13.7			0.0012
42.97			0.71	0.77		21.1			0.0355
41.76			0.69		0.28	21.7			0.0395

[¶] Age, sex and age-by-sex interaction were included in all models;

[†] Coefficients expressed as mmHg of blood pressure per 1 standard deviation change in urinary sodium (Maywood: 71.0, Jamaica: 48.8, Nigeria: 54.6 mmol) and potassium (Maywood: 16.4, Jamaica: 16.1, Nigeria: 22.9 mmol); Na, urinary sodium; K, urinary potassium; Na:K, ratio of urinary sodium to potassium; R^2, coefficient of determination