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ORIGINAL ARTICLE

Blood pressure and urinary excretion of electrolytes in Spanish schoolchildren

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Despite the importance of hypertension in adults, its effects on child health are poorly understood. This cross-sectional epidemiological study was designed to look for a relationship between elevated blood pressure (BP) in children and 24-h urinary excretion of sodium (Na) and potassium (K), and between BP and dietary salt intake. The study population was all 59 856 schoolchildren aged 6 to 14 years in the province of Almería in southern Spain, among whom 613 participants were chosen randomly for study. We measured 24-h urinary Na and K concentrations, systolic and diastolic BP, body weight and height. There was a weak correlation between Na excretion and systolic BP ($r = 0.18$, 95% confidence interval 0.10–0.26), and between K excretion and systolic BP ($r = 0.49$, 95% CI = 0.04–0.20). Body weight was the variable that best correlated with systolic ($r = 0.49$, 95% CI = 0.43–0.55) and diastolic BP, and with Na excretion ($r = 0.48$, 95% CI = 0.42–0.55). Multiple regression analysis also showed that body weight was the variable that best correlated with systolic BP (b = 0.58), although the variables in the equation explained little of the total variability in BP (26%). These correlations were significant at $P < 0.05$. In conclusion urinary electrolytes correlated poorly with BP in a sample of Spanish schoolchildren. Body weight was the only variable that showed a weak relationship with BP and Na excretion.

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Introduction

Cardiovascular diseases are among the leading causes of death and illness,[1] and give rise to large health care costs in developed countries. Large clinical trials[2,3] have shown that elevated blood pressure (BP) in adults is a prime risk factor for cardiovascular diseases, cerebral vascular disorders, cardiac insufficiency, renal insufficiency, and to a lesser degree, coronary artery disease. Despite the influence of arterial hypertension in adults, its effects on child health are poorly known. Studies in children are hampered by technical factors, the lack of normal reference values for different geographical regions, and the widespread assumption that high BP is not a childhood problem. The first set of distribution curves of BP in children was published in 1977 by the Task Force of the National Heart Association of the US National Heart, Lung and Blood Institute.[4] The influence of diet on BP has been a controversial topic in recent decades. In 1944, Kemper[5] showed that a diet of vegetables, rice and water lowered BP. In his 1985 meta-analysis, MacGregor[6] concluded that patients who benefited the most from restricted sodium (Na) intake were the oldest subjects who had the highest BPs. Basic research has also shed light on the relation between Na and BP. The Intersalt study[7] showed a relationship between salt intake and BP within populations, but not between populations.

In children the relationship between BP and urinary excretion of electrolytes has been little studied. It is crucial to identify factors in children that may lead to hypertension during adulthood, and to determine whether these factors can be modified or avoided. The present study was designed to determine the relationship between BP and urinary Na excretion, and between BP and dietary salt intake, in children aged 6 to 14 years.

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Subjects and methods

Subjects and setting

From the 59 856 students (aged 6 to 14 years inclusive) registered in 107 public primary schools in the province of Almería, southern Spain, for the school years 1993–1994, we obtained a random sample of 613 children (308 boys and 305 girls). Maximum sampling error at the 95% confidence level (CI) was ±0.14.[8] Inclusion criteria were aged between 6 and 14 years, and current registration in a public school in the province of Almería. We excluded from the sample children with any of the following: (a) metabolic or chronic disease (liver disease, kidney disease, type I or II diabetes mellitus, hypo- or hyperpotassemia; (b) pharmacological treatment during the previous 3 months with corticosteroids, insulin, diuretics, oral contraceptives or other drugs that could affect BP values; (c) surgery or serious infectious disease during the previous 6 months; or (d) children who were judged to be too agitated or nervous during the physical examination. Of the 613 children initially recruited, a total of 60 were excluded from the analysis for the following reasons: type I diabetes mellitus ($n = 1$); previous treatment with oral or inhaled corticosteroids ($n = 5$, all with asthma); surgery or major infection during the previons 6 months ($n = 4$, appendicitis); agitation or nervousness during physical examination ($n = 13$).

Written authorisation was obtained from the parents for their child to take part in the study, and the parents completed a questionnaire that requested information about their child's antecedents (previous illnesses, pharmacological treatments, smoking and drug use), and about family antecedents of diabetes, hypertension, hypercholesterolaemia, hypertriglyceridaemia, myocardial infarction, cerebrovascular disease, obesity, smoking and drug use. Each child underwent a physical examination to measure BP and pulse (two determinations), body weight and height. A 24-h urine sample was obtained from each child for analyses of Na, potassium (K), and creatinine concentrations.

Measurements and laboratory analyses

Blood pressure was measured with Littman (St. Paul, MN, USA) adult and pediatric stethoscopes and Riester cuffs (Jungingen, Germany) of two sizes (8 H 13, range 13–21 and 13 H 24, range 24–32), and with Diplomat Presameter (Jungingen, Germany) table-top mercury sphygmomanometers. The first (systolic BP) and fifth Korotkoff sounds (diastolic BP) were taken as valid, and the recommendations of the British Hypertension Society[9] were followed. Blood pressure was measured twice at a 15 second interval; mean systolic BP and mean diastolic BP were considered the arithmetic mean of the two values. Height and weight were measured with a Seca 713 (Sussex, England) scale. Body mass index (BMI) (Quetelet index (QI)) was calculated as weight (kg)/height (m^2). Body surface area was calculated as weight$^{0.425}$ H height$^{0.725}$ H 71.84.[10] Urine specimens were collected in disposable wide-mouthed 2-L flasks.

To determine electrolyte concentrations in urine, the sample volume was recorded and part of the sample was centrifuged to remove impurities. Sodium and K were measured in the supernatant with a Biomedical Nova 5 selective electrode and the Nova 5 Fluids/Na/K/reagents pack. Chem-Sep level 6 solution (Boehringer Mannheim, Germany) was used as a quality control referent.

To check that the urine samples accurately represented the 24-h collection period, we measured creatinine in diluted samples (1:10) with the Jaffé method without protein removal (Boehringer Mannheim) in a Hitachi 704 autoanalyzer. As a quality control we used commercial Precinorm U (low level) and Precipath U (high level) solutions (Boehringer Mannheim). Reference values ranged from 8 to 22 mg/kg/24 h for children aged 6 to 11 years, and from 8 to 30 mg/kg/24 h for adolescents aged 12 to 14 years. Urine samples that yielded values outside these ranges were analysed with the Creatinine Enz-PAP kit (enzymatic creatinine-phenylaminopyridone, Boehringer Mannheim), an enzymatic method that detects possible interferences due to high creatinine concentrations. We found no significant differences in creatinine measurements obtained with these two methods. A total of 60 urine samples (9.3%) were excluded from study on the grounds of inadequate collection technique when creatinine concentration was below 7 mg/kg/24 h (insufficient volume of urine) or above 30 mg/kg/24 h (excess volume). Samples with a 24-h collection volume of 300 mL or lower were also excluded from study, and data from these subjects were not included in the statistical analysis.

Statistical analysis

Student-Fischer *t*-tests were used to compare independent means for quantitative variables, and chi-squared tests were used to compare proportions. Pearson's linear correlation was calculated for bivariate quantitative variables, with simple and multiple linear regression. All quantitative variables were analysed with the Kolmogorov-Smirnoff test for normal distribution. The results for all quantitative variables are expressed as the mean ± standard deviation (s.d.). Differences were considered statistically significant at $P < 0.05$.

Results

Table 1 shows the mean values ∀ s.d. for variables in boys and girls. Urinary excretion of Na, K, CI and creatinine was significantly higher in boys than in girls. Sodium excretion was higher in boys than in

Table 1 Anthropometric variables and 24-h urinary electrolyte concentrations in a sample of Spanish schoolchildren aged 6 to 14 years

	Boys (n = 274)	*Girls (n = 279)*	*Overall (n = 553)*
Age (years)	10.3 ± 2.6	10.4 ± 2.5	10.3 ± 2.5
Weight (kg)	41.8 ± 14.2	40.6 ± 12.6	41.2 ± 13.4
Height (cm)	143.8 ± 14.9	143.2 ± 13.3	143.6 ± 14.1
BMI (kg/m²)	19.6 ± 3.9	19.3 ± 3.7	19.5 ± 3.8
Na (mEq/24 h)	142.2 ± 70.4	125.6 ± 53.5*	136.3 ± 63.3
K (mEq/24 h)	41.2 ± 15.7	37.2 ± 15.2**	39.2 ± 15.5
Na/K	3.7 ± 1.3	3.6 ± 1.3	3.6 ± 1.3
Creatinine (mg/24 h)	751.4 ± 316.8	645.4 ± 236.9*	697.9 ± 281.4
SBP (mm Hg)	110.3 ± 14.4	111.4 ± 14.7	110.9 ± 14.6
DBP (mm Hg)	60.6 ± 9.5	61.6 ± 9.2	61.1 ± 9.4

SBP = systolic blood pressure; DBP = diastolic blood pressure. *$P < 0.001$; **$P < 0.01$.

girls at all ages except 6 years, and the tendency to increase with age was clearer in boys than in girls. Urinary Na excretion in girls was similar at 6 and 10 years of age. The difference between sexes was significant at 10, 12 and 13 years ($P < 0.05$) (Figure 1). The findings for K excretion were similar, and the difference between sexes was significant at 9 and 11 years of age ($P < 0.05$) (Figure 2). The Na/K ratio ranged from 2.98 to 4.19, meaning that Na excretion, and indirectly Na intake, was three- to four-fold higher than K excretion in all age subgroups.

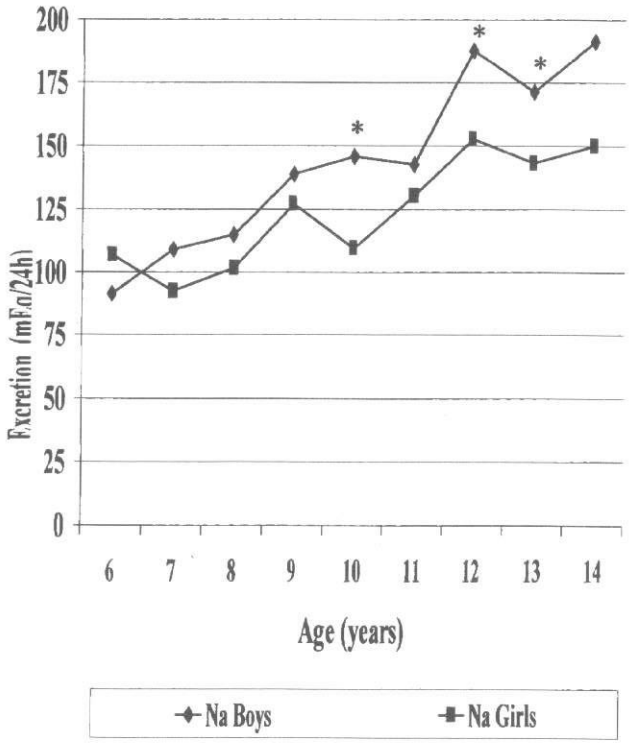

Figure 1 Mean urinary sodium excretion in a sample of Spanish schoolchildren. *$P < 0.05$.

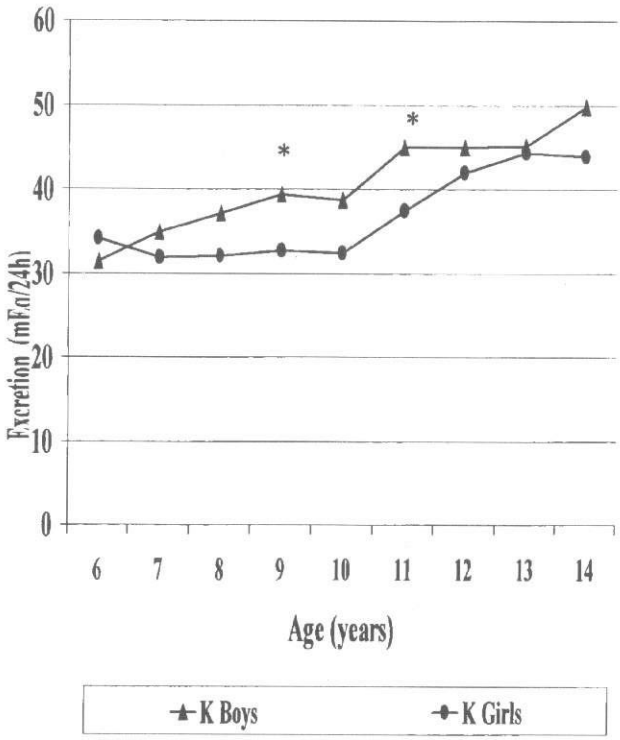

Figure 2 Mean urinary potassium excretion in a sample of Spanish schoolchildren. *$P < 0.05$.

Table 2 shows the correlation between urinary K excretion and the other variables. All correlations were significant ($P < 0.05$) except the relationships between urinary K excretion and diastolic BP. When boys and girls were analysed separately, these correlations were found in the former group, but in girls systolic BP did not correlate with Na excretion. In both sexes and at all ages, urinary Na excretion correlated significantly with K excretion ($r = 0.63$, $P < 0.001$).

Table 2 Correlations (r) of 24-h urinary K excretion with blood pressure (BP) and anthropometric variables

	Boys (n = 274) K	*Girls (n = 279) K*	*Overall (n = 553) K*
SBP (mm Hg)	0.16*	0.08	0.12*
DBP (mm Hg)	0.05	0.07	0.05
K (mEq/24 h)	–	–	–
Height (cm)	0.40*	0.35*	0.37*
Weight (kg)	0.44*	0.37*	0.40*
BMI (kg/m²)	0.34*	0.27*	0.30*
Age (years)	0.32*	0.31*	0.32*
BS	0.42*	0.39*	0.40*

SBP = systolic BP; DBP = diastolic BP; BMI = body mass index; BS = body surface area, calculated as weight$^{0.425}$ H height$^{0.725}$ H 71.84.^{25} *$P < 0.05$.

Table 3 Results of multivariate linear regression analysis with sodium excretion (mEq/24 h) as the dependent variable. Coefficient (B) and explained variability (R^2)

	Overall					
	Na Exc		*K Exc*		*Na/K Exc*	
	B	*P*	*B*	*P*	*B*	*P*
Weight	**2.32**	0.05	**0.41**	0.05	**0.02**	0.05
Height	**0.36**	NS	**0.24**	0.05	**−0.01**	NS
SBP	**−0.20**	NS	**−0.11**	0.05	**−0.00**	NS
DBP	**−0.40**	NS	**−0.08**	NS	**−0.00**	NS
Age	**−0.96**	NS	**−0.72**	NS	**−0.06**	NS
R^2	**0.25**		**0.18**		**0.04**	–
Constant	**45.58**	NS	**7.55**	NS	**3.93**	0.05

SBP = systolic blood pressure; DBP = diastolic blood pressure; NS = not significant; *P* = significance in comparison with the other independent variables. **Na Exc (Overall)** = 45.58 + 2.32 × **Weight** + 0.36 × **Height** − 0.20 × **SBP** − 0.40 × **DBP** − 0.96 × **Age; K Exc (Overall)** = 7.55 + 0.41 × **Weight** + 0.24 × **Height** − 0.11 × **SBP** − 0.08 × **DBP** − 0.72 × **Age; Na/K Exc (Overall)** = 3.93 − 0.02 × **Weight** − 0.01 × **Height** − 0.00 × **SBP** − 0.00 × **DBP** − 0.06 × **Age**.

Table 3 shows the coefficients in the multiple regression equation (b) obtained with Na excretion, K excretion and Na/K ratio as the dependent variables. Once the influence of height, systolic BP, diastolic BP and age was accounted for, body weight was the only variable significantly related with Na excretion: Na excretion increased 2.32 mEq per kilogram. The coefficients for systolic and diastolic BP were small, negative and nonsignificant, suggesting that these variables had little effect on Na excretion. The variables included in the equation explain only 25% of the variability (R^2) in Na excretion, 18% in K excretion, and only 4% in Na/K ratio in our sample of Spanish schoolchildren. Weight was the sole variable that showed a constant and significant correlation with Na excretion, K excretion and Na/K ratio, once the confusing effect of the other variables of the equation was ruled out.

Table 4 shows the results of multiple regression analysis with BP as the dependent variable. Again, body weight was the variable that showed the clearest relationship with BP, and a significant effect was found in both sexes and all age subgroups. For example, systolic BP in boys increased 0.58 mm Hg per kilogram of body weight. In contrast, the coefficients for urinary Na and K excretion were small and showed no significant correlation with BP. The variables included in the equation accounted for 33% (R^2) of the variability in systolic BP and 19.8% of the variability in diastolic BP in boys, and for 20% of the variability in systolic BP but only 8% of the variability in diastolic BP in girls.

Discussion

Sodium intake and blood pressure

We could not establish a relationship between urinary excretion of Na (or, indirectly, dietary intake of Na) and BP: our findings thus suggest that the BP values found in our sample of Almería schoolchildren were independent of Na intake. In contrast, body weight correlated directly with both BP and Na intake.

To test whether food intake in children affected BP, Jenner *et al*[11] did a cross-sectional study of 884 Australian 9-year-olds, and found that nutrient intake and BP were related only after adjustment for energy intake. These authors found in inverse relationship between diastolic BP and fibre consumption in boys. In girls, systolic BP correlated inversely with protein and cholesterol intake after adjustment for calorie intake. This study found no relationship between BP an the intake of carbohydrates, Na, K, calcium or magnesium. More recent studies have identified weight during childhood (measured as BMI or body surface area) as an important variable in the subsequent development of hypertension in the adult. Some authors have found that low birth weight is associated with a higher BP as an adult.[12–14]

Diets of different Na content have been tested to

Table 4 Results of multivariate linear regression analysis with systolic and diastolic blood pressure (BP) (mm Hg) as dependent variables. Coefficient (B) and explained variability (R^2)

	Boys				*Girls*				*Overall*			
	SBP		*DBP*		*SBP*		*DBP*		*SBP*		*DBP*	
	B	*P*	*B*	*P*	*B*	*P*	*B*	*P*	*B*	*P*	*B*	*P*
Weight	0.58	0.05	0.51	0.05	0.58	0.05	0.32	0.05	**0.58**	0.05	**0.41**	0.05
Height	0.27	NS	−0.15	NS	0.20	NS	−0.02	NS	**0.23**	0.05	**−0.08**	NS
Na Exc	−0.01	NS	−0.01	NS	−0.01	NS	−0.01	NS	**−0.01**	NS	**−0.01**	NS
K Exc	−0.07	NS	−0.04	NS	−0.06	NS	−0.01	NS	**−0.065**	NS	**−0.03**	NS
Age	−1.65	0.05	−0.55	NS	−1.46	NS	−0.64	NS	**−1.51**	0.05	**−0.61**	NS
R^2	0.33	–	0.198	–	0.20	–	0.08	–	**0.26**	–	**0.14**	–
Constant	65.05	0.05	70.15	0.05	78.07	NS	59.81	0.05	**72.52**	0.05	**64.47**	0.05

NS = not significant; *P* = significance in comparison with the other independent variables.
SBP (Overall) = 72.52 + 0.58 × **Weight** + 0.23 × **Height** − 0.01 × **Na Exc** − 0.065 × **K Exc** − 1.51 × **Age**
DBP (Overall) = 64.47 + 0.41 × **Weight** − 0.08 × **Height** − 0.01 × **Na Exc** − 0.03 × **K Exc** − 0.61 × **Age**

determine the effect of salt intake on BP. In a study of infants, Hofman *et al*[15] found that systolic BP was 2.1 mm Hg lower in babies who were fed a low-Na diet than in the group fed a normal salt diet. These findings were confirmed by Lucas *et al*.[16] It is not known whether excessive salt intake during infancy, childhood and adolescence sensitises the vascular system and makes subjects more likely to have hypertension as adults. At the age of 8 years, children who had been given a highly salted milk formula as infants showed no greater preference for saltier food than did children given an unsalted formula. The results of this study led many to question the practice, widespread in the USA during the 1960s, of sharply reducing the Na content in baby formulae.[17] Grobbee and Hofman[18] reviewed the data from 13 studies (age range 10–70 years) and concluded that dietary salt restriction decreased BP, and noted that the decreases were greater in subjects who had a higher initial BP and who were older. Cooper *et al*[19] found that in 16-year-old adolescents, reducing Na intake from 110 to 45 mEq per day during 24 days did not significantly lower BP. Other population-based studies of children have failed to demonstrate a relationship between Na intake and BP. Zwiauer[20] and Knuiman[21] studied European children aged 8 and 9 years, and Geleijnse *et al*[22] published a longitudinal, prospective study of 7 years duration of 233 children aged 5 to 17 years; neither group found a significant association between Na excretion and changes in BP. In Spain, Luque *et al*,[23] in a study similar in design to ours, were the only group to find a significant relationship (r approaching unity) between Na excretion and BP in a sample of children aged 6–7 and 10–14 years in Torrejón; however, when they adjusted for body weight in each group, the correlation disappeared, despite the fact that these children consumed 160–220 mmol Na/day.

The findings of studies that compared populations show a clear relation between Na intake and hypertension in adults; however, this association has been difficult to document from data of single-population studies.[7] Several reasons may help to explain why research in western adult and child populations does not usually bring to light a direct relationship between Na intake and BP: we suspect that the values of urinary Na excretion (and hence Na intake) in our sample of children were predominantly in the range where further increases are not associated with increasing BP. The normal range of urinary Na excretion varies widely in adults (70–200 mEq/day). This degree of variability (23.3–383.8 mEq/day) in the child and adolescent population we tested makes it difficult to prove a clear relationship with BP. The relation between increasing age and rising BP in subjects who consume large amounts of salt may also camouflage the relation between salt intake and BP. Increases in BP as a result of excess dietary Na intake may not become evident until a certain age. An Na intake of 60–70 mmol/day may not have any noticeable effect on BP. In most western societies, daily salt intake easily exceeds this amount. Because of genetic variability, not all individuals in a natural population respond equally to changes in Na intake. Forty percent of all normotensive subjects are salt-sensitive.[7] Because of the cross-sectional design of most studies (except for cohort studies), salt intake and BP are measured at a single point in time. Hypertension appears only after a period of development, and it is thus more important to determine life-long Na intake rather than salt intake at a given moment in a subject's lifetime. Other ions (eg, calcium, magnesium and potassium), and obesity, can obscure the relationship between Na and BP. Further studies may show that there is, in fact, no relationship between Na intake and BP.

Potassium intake and blood pressure

We found evidence against an association between K intake and BP in adolescents aged 12 to 14 years. No relation could be shown in any of the younger age subgroups, even when boys and girls were analysed separately. Nonetheless, the weak relationship is of interest, as it is consistent with earlier findings in adult blacks.[24–29] McGarvey *et al*[30] found that maternal K intake during pregnancy correlated inversely with diastolic BP measured when their babies had reached the age of 6 and 12 months. A number of studies have reported an inverse relationship between BP and K intake during infancy and childhood.[20,22,31] Khaw and Barrett-Connor[32] found that for every 10 mmol of K in the diet, systolic BP decreased in normotensive subjects by 1.7 mm Hg. They noted that the relative risk of stroke was 2.6 times greater in men and 4.8 times greater in women at the low range of K intake in comparison with subjects who consumed more of this ion, and found this association to be independent of the influence of dietary K supplementation on BP.

Conclusions

Excess weight and obesity in children and adolescents have also been shown to be important in the appearance of hypertension in the adult.[31,33] These findings are compatible with the results of the present study. Nonetheless, our findings are insufficient in themselves to justify the conclusion that there is no potential benefit in restricting dietary salt intake in children. Dietary habits at this age may lead to patterns of dietary behaviour in adults that place them at increased risk of developing hypertension.

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