Association between urinary potassium, urinary sodium, current diet, and bone density in prepubertal children[1–3]

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ABSTRACT

Background: Our understanding of the role of nutrients in bone development in children is limited.

Objective: We examined the associations between urinary potassium, urinary sodium, usual dietary intake, and bone mineral density (BMD) in prepubertal children.

Design: This was a cross-sectional study of 330 boys and girls aged 8 y. Urinary measures were assessed in a single, timed, overnight urine specimen. Usual diet was assessed with a food-frequency questionnaire completed by a parent or guardian. BMD at the femoral neck, lumbar spine, and total body was measured by dual-energy X-ray absorptiometry.

Results: Urinary potassium correlated significantly with BMD at all sites (femoral neck: $r = 0.20$, $P < 0.001$; lumbar spine: $r = 0.19$, $P = 0.001$; total body: $r = 0.24$, $P < 0.001$). After adjustment for confounders (primarily lean body mass), this association was lower in magnitude but remained significant at 2 sites with a consistent trend at the third (femoral neck: $P = 0.15$; lumbar spine: $P = 0.046$; total body: $P = 0.028$). Urinary sodium was not associated with BMD at any site. No nutrient or food intake estimate was associated with BMD, although urinary potassium correlated significantly with potassium intake ($r = 0.14$, $P = 0.016$) and fruit and vegetable intake ($r = 0.12$, $P = 0.033$).

Conclusions: Urinary potassium was associated with both dietary intake and BMD independent of lean body mass in these well-nourished, calcium-replete young children. These findings should be confirmed in further longitudinal studies. Nevertheless, this association is likely to represent dietary intake of potassium and suggests that measurement of urinary potassium is superior to food-frequency questionnaires for assessing potassium intake in this age group. *Am J Clin Nutr* 2001;73:839–44.

KEY WORDS Bone mineral density, urinary potassium, prepubertal children, dietary intake, urinary sodium, food-frequency questionnaire

INTRODUCTION

Fractures are a major public health problem in both men and women (1). Bone mineral density (BMD) is one of the major predictors of osteoporotic fractures in the elderly (2) and of fractures in children (3). BMD at any particular time is the result of the amount of bone gained in early life (ie, peak bone mass) and subsequent bone loss (4). Physical activity and, to a lesser extent, diet (particularly calcium intake) during adolescence and early adulthood have been implicated as determinants of peak bone mass (5, 6). Although most adult bone mass is attained before the age of 14 y (7), relatively little is known about the effect of genetic or lifestyle factors on bone acquisition during the time period from birth to puberty. Some reports suggest beneficial effects on bone density in prepubertal children of calcium supplementation (8), physical activity (9–11), and winter sunlight exposure (9). There is little information about the role of other nutrients in children, although carbonated beverages may increase the risk of fractures in adolescence (12, 13).

The results of recent studies in elderly men and women (14) and in premenopausal women at both axial (15) and peripheral (16) bone sites suggest that potassium and magnesium intake also may be important in maintaining bone mass, possibly through maintenance of a mild metabolic alkalosis. Furthermore, studies in adults implicated sodium intake as a risk factor for urinary calcium loss (17), bone loss (18), and high bone resorption (19). Few studies have examined the role of these nutrients in bone health in children. A report by Matkovic et al (20) indicated that urinary sodium is one of the most important determinants of urinary calcium excretion, implying that dietary sodium may be a determinant of bone mass in children aged 8–13 y (even though these authors found no association between urinary sodium and bone mass). However, neither an acute load of sodium chloride nor potassium citrate influenced urinary calcium excretion in prepubescent girls (21), whereas a similar loading regimen decreased urinary calcium in adults (22), suggesting possible differences between bone modeling in children and remodeling in adults. In this cross-sectional study, we therefore asked the following study questions: *1*) Is urinary potassium excretion, urinary sodium excretion, or both related to bone mass in prepubertal boys and girls? *2*) Is the current dietary intake of these

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same children [as assessed by food-frequency questionnaire (FFQ)] associated with bone mass?

SUBJECTS AND METHODS

In 1988 there were 6779 live births in Tasmania, an Australian island state whose population is predominantly white. Of these infants, 1380 were identified as being at higher risk of sudden infant death syndrome according to previously published criteria (9) and were selected to take part in a longitudinal study. In brief, children were selected on the basis of a composite risk score comprising their birth weight, season of birth, sex, maternal age, duration of second stage of labor, and maternal intention to breast-feed. In southern Tasmania, 735 infants met these criteria. The mothers of 696 infants (95%) agreed to an in-hospital interview. The children were then approached in 1996 to take part in an additional study. After 8 y, we were able, through the use of school lists, to locate 551 of these subjects (80%), which closely approximates the number likely to be available in Tasmania according to the Australian Bureau of Statistics data on annual outward migration rates.

This study was granted approval by the University of Tasmania Ethics Committee (Human Experimentation). Subjects whose parents or guardians provided informed consent took part in a study that included measurement of bone mass, anthropometry, and questionnaire assessment by a parent or guardian of the duration of breast-feeding, current maternal and paternal smoking and socioeconomic factors, current diet, competitive sports participation in the past 12 mo (yes or no), and winter sunlight exposure (4-point scale). Height and weight were measured with the subjects in light clothing by using transportable combined scales and a stadiometer (model 707, capacity 200 kg; Seca, Hamburg, Germany). Questions on socioeconomic status related to maternal education (4-point scale), paternal employment status (2 categories: employed or unemployed/pensioner), and marital status (6 categories).

Urinary sodium, potassium, and creatinine excretion were assessed in a single, timed, overnight nonfasting urine collection. Aliquots of each sample were diluted with neutral pH buffer and analyzed in duplicate for sodium and potassium with use of ion-specific electrodes in an Ektachem 750 XRC analyzer and for creatinine by the Ektachem method (Kodak, Rochester, NY). The CVs for these measurements were 0.6% for sodium, 1.1% for potassium, and 5.4% for creatinine. Urinary sodium and potassium concentrations were then converted to daily excretion rates by using the multipliers of 8-h urinary excretion identified by Dyer et al (23) of 3.8 for sodium and 5.0 for potassium.

The usual dietary intake of each child was measured with a semiquantitative FFQ that was completed by the child's mother or guardian. The reference period for the FFQ was the 12 mo before the examination. The FFQ consisted of 159 food categories and associated standard serving sizes appropriate for 8-y-old children. The questionnaire also included 24 questions on supplementary dietary practices such as the usual type of spreadable fat used and the type of milk used. The FFQ was modified from an instrument validated for an adult population (24), which in turn was derived from an instrument widely used in Australia (25). In a pilot test of the FFQ, the questionnaire was completed at home by 6 children aged 8–10 y and their parents. Information from these individuals resulted in some modifications to the food list and the standard serving sizes. The performance of the FFQ was not compared with another method of measuring usual dietary intake; however, work conducted in Australia indicates the utility of this method in Australian children (26). Frequencies of intake of the standard serving size less than once per month were not counted toward the nutrient estimate. Mean usual daily nutrient intake was estimated from the FFQ by using nutrient values assigned to each food category in the Australian Tables of Food Composition (NUTTAB95; National Food Authority, Canberra, Australia) and by incorporating information from the supplementary questions. Subjects who did not complete an entire page of the questionnaire or who reported consuming ≤15 FFQ categories were deemed to have inadequately completed the questionnaire and were not included in the data analysis.

Total-body, lumbar spine, and femoral neck bone mass were measured by dual-energy X-ray absorptiometry with a QDR2000 densitometer on the array setting (Hologic Inc, Waltham, MA). The software version was 4.76A. Bone mass was examined as bone mineral content (BMC), BMD, and bone mineral apparent density (BMAD). BMAD (g/cm^3) is an approximation of the volumetric density of bone and is calculated by dividing site-specific BMD by the square root of the area at that site (27). Precision estimates in vivo are not available for our subjects but are 1–2% in adults. The longitudinal CV for our instrument during 1996 for daily measurements of a spine phantom was 0.54%.

All statistical analyses were conducted with SPSS (version 8.0 for WINDOWS; SPSS Inc, Chicago). Pearson's product-moment correlation analysis was used to examine associations between the study factors and bone density at 3 sites. Unpaired *t* tests or Mann-Whitney *U* tests (where appropriate) were used to compare means. Linear regression analysis was then used to examine whether significant associations were independent of potential confounders or were mediated by body size or composition. Confounders included sex, height and weight, lean body mass, maternal smoking during pregnancy, breast-feeding in early life, dietary calcium, sports participation, category of sun exposure, and urinary sodium. Dietary variables were considered individually because of a high degree of collinearity when considered as continuous variables. Dietary variables were examined as crude nutrient or food estimates divided by total energy intake to provide an estimate of usual dietary composition (ie, nutrient/energy). P values < 0.05 (two-tailed) or 95% CIs not including the null point were regarded as statistically significant. Adjustment for multiple comparisons was not performed.

RESULTS

There were a total of 330 participants (215 boys, 115 girls), representing a response rate of 60% of those available in 1996 or 47% of those selected in the original cohort in 1988. Characteristics of the participants and excluded subjects are shown in **Table 1**. Subjects with missing or inadequate FFQ information were not significantly different from the participants in terms of age, sex distribution, weight, height, sports participation, sun exposure, and BMD. However, they were less likely to have been breast-fed or to have mothers with tertiary education and more likely to have mothers who smoked and unemployed fathers. Dietary intake data are shown in **Table 2**. Calcium intake was high: 1336 mg/d. The distribution of other nutrients indicated a well-nourished population, although there was evidence of both under- and overreporting.

TABLE 1
Comparison of participants and excluded subjects[1]

Variable	Adequate dietary data (n = 262)	Inadequate dietary data (n = 68)	P[2]
Percentage male (%)	65	66	0.84[3]
Age (y)	8.20 ± 0.33[4]	8.22 ± 0.33	0.66
Height (cm)	127.7 ± 5.5	127.8 ± 6.6	0.88
Weight (kg)	27.9 ± 5.4	28.1 ± 6.2	0.73
Sports participation (%)	61	67	0.36[3]
Proportion breast-fed (%)	58	37	0.002[3]
Winter sun exposure (≈h/d)	2.73	2.65	0.57[3]
Mothers who smoked (%)	46	61	0.03[3]
Mothers with tertiary education (%)	19	8	0.032[3]
Fathers unemployed (%)	17	27	0.052[3]
Urinary potassium (mmol/d)	32 ± 21	38 ± 27	0.13
Urinary sodium (mmol/d)	73 ± 43	78 ± 53	0.36
Femoral neck BMD (g/cm^2)	0.63 ± 0.08	0.64 ± 0.08	0.34
Lumbar spine BMD (g/cm^2)	0.60 ± 0.07	0.61 ± 0.07	0.66
Total-body BMD (g/cm^2)	0.77 ± 0.04	0.78 ± 0.06	0.49

[1] BMD, bone mineral density.
[2] Unpaired t test unless noted otherwise.
[3] Mann-Whitney U test.
[4] $\bar{x}$ ± SD.

In unadjusted correlation analyses, urinary potassium excretion was positively associated with BMD at all sites (femoral neck: $r = 0.20$, $P < 0.001$; lumbar spine: $r = 0.19$, $P = 0.001$; total body: $r = 0.24$, $P < 0.001$) and with BMAD at the 2 anatomical sites (femoral neck: $r = 0.18$, $P = 0.002$; lumbar spine: $r = 0.13$, $P = 0.015$). Similar observations were made in quartile-based analyses (**Figure 1**). When we used the raw data, we found that children in the highest quartile of urinary potassium had higher BMD at all sites than did children in the lowest quartile.

In addition and importantly, urinary potassium correlated significantly with lean body mass ($r = 0.21$, $P < 0.001$), potassium intake ($r = 0.14$, $P = 0.02$), and fruit and vegetable intake ($r = 0.12$, $P = 0.03$). In multivariate analysis, urinary potassium was associated with both potassium intake ($P = 0.029$) and lean body mass ($P < 0.001$).

In multivariate analyses, the associations between urinary potassium and BMD were lower in magnitude but remained significant at the lumbar spine and total-body sites, with a consistent trend at the femoral neck (**Table 3**), indicating a dose-response relation. The lower magnitude was primarily due to adjustment for body composition. However, after adjustment, the differences between the highest and lowest quartile remained positive but became nonsignificant at all sites (femoral neck: 1.0%; lumbar spine: 2.1%; total body: 1.8%).

In correlation analyses, urinary sodium excretion was significantly positively associated with BMD at all sites (femoral neck: $r = 0.13$, $P = 0.014$; lumbar spine: $r = 0.14$, $P = 0.019$; total body: $r = 0.18$, $P = 0.001$). However, this association was no longer evident at any site after adjustment for body size and potential environmental confounders.

No nutrient or food intake density estimates (including total energy) from the FFQ were associated with BMD at any site, except for potassium and vegetable intakes, which were negatively associated with total-body BMD (**Table 4**). After adjustment for confounders, both of these associations became nonsignificant. The lack of association between BMD at any site and dietary variables persisted in multivariate analysis (data not shown) and when other methods of analysis such as the residual method or energy-adjusted method were used or when the analysis was restricted to subjects with energy intakes <12 000 kJ/d.

DISCUSSION

In this study of well-nourished, calcium-replete children, we found consistent, positive associations between urinary potassium excretion estimated from a single, timed, overnight urine specimen and bone mass at all sites. In part, these associations were attributable to body size and composition, but the associations persisted after adjustment for these variables, indicating an effect independent of lean body mass, most likely through diet.

Potassium intake is reported to be beneficial in the maintenance of bone mass in premenopausal women (15, 16) and in elderly men and women (14). The mechanism of this association may be a reduction in endogenous acid production with a consequent decrease in urinary calcium excretion. In adults, dietary alkaline potassium, such as potassium bicarbonate (28–30) or potassium citrate (22), reduces 24-h urinary calcium excretion and promotes a more positive calcium balance (29–31). In short-term studies, administration of 50 mmol potassium bicarbonate decreases acute urinary calcium excretion in adults (22). However, in a similar study of children given potassium citrate providing 32 mmol K (1.2 mmol K/kg body wt), there was no change in urinary calcium excretion, although net acid excretion decreased (21). Notably, rats also do not respond to a load of alkaline potassium with a decrease in urinary calcium excretion (32, 33). The differences in bone metabolism between children and adults (ie, bone modeling compared with remodeling) may be involved in the difference in calciuric response to alkaline potassium between adults and children. Alternatively, potassium intake may be a marker of a high-quality diet or may indicate a diet rich in plant components favorable to bone formation such as magnesium, vitamin C, and phytoestrogens.

The observation that urinary potassium but not estimated potassium intake was associated with bone mass is important. Whereas intakes of potassium, fruit, and vegetables correlated

TABLE 2
Dietary intake of subjects included in the analysis[1]

Intake	Value
Nutrient	
Calcium (mg/day)	1336 ± 541 (283–3754)[2]
Magnesium (mg/d)	306 ± 88 (96–644)
Phosphorus (mg/d)	1569 ± 503 (573–3931)
Potassium (mg/d)	3484 ± 1042 (1153–7892)
Protein (g/d)	83 ± 25 (27–195)
Fat (g/d)	90 ± 31 (33–246)
Carbohydrate (g/d)	315 ± 89 (77–636)
Total energy (kJ/d)	9814 ± 2727 (3150–21945)
Food group	
Milk (mL/d)	590 (0–2040)[3]
Vegetable (servings/wk)	29 (1–68)
Fruit (servings/wk)	16 (0–48)
Fish (servings/wk)	1.3 (0–15)
Meat (servings/wk)	16 (0–44)

[1] n = 262.
[2] $\bar{x}$ ± SD; range in parentheses.
[3] $\bar{x}$; range in parentheses.

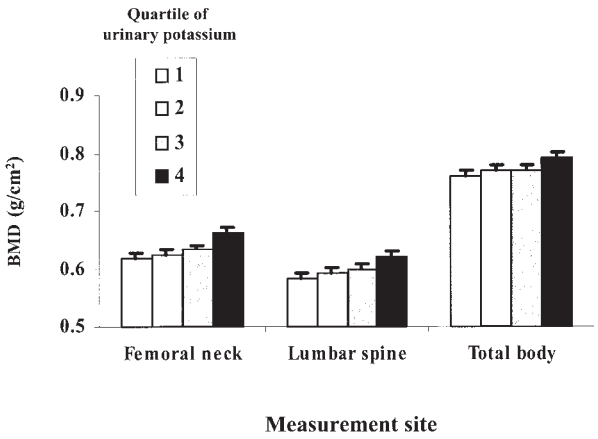

FIGURE 1. Relation between quartile of urinary potassium and mean (±SE) bone mineral density (BMD) at various sites. Median values for each quartile of urinary potassium in ascending order were 12, 22, 35, and 59 mmol/d. There was a consistent dose-response relation between urinary potassium and BMD (femoral neck: $r = 0.18$, $P = 0.001$; lumbar spine: $r = 0.19$, $P = 0.001$; total body, $r = 0.21$, $P < 0.001$). Children in the highest quartile of urinary potassium had higher BMD at all sites than did children in the lowest quartile (femoral neck: 6.8%, $P = 0.001$; lumbar spine: 6.3%, $P = 0.001$; total body: 4.3%, $P < 0.001$).

weakly but significantly and independently with urinary potassium, these measures did not correlate with BMD. It appears likely that this discrepancy was due to measurement error and that biological measures of exposure may better estimate dietary intake in young children than do questionnaire methods. It is surprising that a single overnight measure of potassium excretion explained ≈5% of the variation in bone mass, with large differences between the highest and lowest quartiles. However, a proportion of this was the result of the association between urinary potassium and body size (particularly lean body mass). After adjustment (primarily for lean body mass), the difference in BMD between the highest and lowest quartiles of urinary potassium was 1–2%. This was not significant but there was still evidence supporting a dose-response relation at 2 sites and a consistent trend at the femoral neck.

There are several reasons we did not find an association between salt intake and bone mass. There may, in fact, be no relation because bone may adapt to changes in urinary calcium excretion by increasing calcium absorption (assuming dietary calcium intake is adequate). It is more likely that we were unable to link salt intake with bone mass because a single 24-h urinary sodium measure is only an approximation of habitual sodium (or potassium) intake (34). Day-to-day variation in the sodium excretion of individuals is so great that regression dilution bias may reduce the slope of the regression line of sodium on a dependent variable such as blood pressure to one-quarter of its true value (35); this may well apply to bone mass also. We previously reported a significant association between urinary sodium and urinary calcium in prepubescent girls (21). On the converse, any effect on bone mass might be detectable only with repeated measurements of both urinary sodium and bone mass. Although we controlled for several important potential confounders, the present study was cross-sectional in design and a longitudinal approach may be required to reduce regression dilution bias and to accurately assess habitual sodium and potassium intakes. Discrepancies among other studies in the literature may also be explained by study design. The 2 studies that found an association (albeit weak) were longitudinal studies in adults with repeated measurement (18, 36); the 3 studies that reported negative findings were either cross-sectional (19, 20) or measured sodium intake with a suboptimal technique (questionnaire) administered on only one occasion 16 y before the measurement of bone mass (37). In light of these points, the lack of association between sodium excretion and bone mass in our sample does not provide strong evidence that an association does not exist.

We found no association between the children's current calcium intake and BMD. Other investigators reported similar findings (38); however, dietary supplementation with calcium appears to have a beneficial effect on BMD in prepubertal children (8). The resolution of this apparent paradox may be that

TABLE 3
Univariate and multivariate associations between urinary potassium and bone mineral density (BMD) in 8-y-old children

	Femoral neck BMD (g/cm²)	Lumbar spine BMD (g/cm²)	Total-body BMD (g/cm²)
Univariate analysis			
β[1]	0.20	0.19	0.24
P	<0.001	0.001	<0.001
Partial R^2 (%)[2]	4.1	3.6	5.6
Adjusted step 1[3]			
β[1]	0.07	0.09	0.12
P	0.13	0.047	0.015
Partial R^2 (%)[2]	0.8	1.3	1.9
Model R^2 (%)[4]	41	36	35
Adjusted step 2[5]			
β[1]	0.07	0.09	0.11
P	0.15	0.046	0.028
Partial R^2 (%)[2]	0.7	1.4	1.7
Model R^2 (%)[4]	43	42	36

[1] Standardized to allow comparison of effect sizes at all 3 sites.
[2] Refers to urinary potassium only.
[3] Adjusted for sex and lean body mass.
[4] Refers to the model including all listed variables.
[5] Adjusted as for step 1 and for current height, current weight, winter sun exposure, sports participation, dietary calcium, breast-feeding in early life, and maternal smoking during pregnancy.

TABLE 4
Unadjusted Pearson correlation coefficients between children's current dietary intake and bone mineral density (BMD) at the age of 8 y

	Femoral neck BMD (g/cm^2)	Lumbar spine BMD (g/cm^2)	Total-body BMD (g/cm^2)
Nutrient density			
Calcium (mg/kJ)	−0.01	−0.06	−0.03
Phosphorus (mg/kJ)	−0.01	−0.04	−0.02
Magnesium (mg/kJ)	−0.05	−0.03	−0.05
Potassium (mg/kJ)	−0.05	−0.05	−0.14[1]
Protein (g/kJ)	−0.05	−0.00	−0.09
Fat (g/kJ)	−0.08	−0.01	−0.00
Carbohydrate (g/kJ)	0.07	−0.02	0.03
Food group			
Meat (portions/kJ)	−0.06	0.05	−0.09
Fish (portions/kJ)[2]	−0.02	0.05	0.03
Fruit (portions/kJ)	0.02	0.03	−0.04
Vegetables (portions/kJ)	0.01	0.01	−0.15[1]
Milk (portions/kJ)	0.03	0.03	−0.02

[1] $P < 0.05$.
[2] Log transformed.

calcium intake above a certain threshold does not contribute further to bone mineralization. Many of the supplement studies showing positive results were conducted in populations consuming a relatively low background calcium intake. Our sample had a high mean daily calcium intake of 1336 mg. Our FFQ overestimated calcium intake in comparison with shorter questionnaires (39), but both FFQs correlated modestly but significantly in estimating calcium intake 8 y apart in premenopausal women (40).

An alternative explanation of the apparent lack of association between current diet and BMD in this well-nourished population is that the estimate of diet intake may have entailed large measurement error, particularly given that no other nutrients were associated with BMD in this sample. In general, children aged >7 y can provide the necessary information for accurate dietary assessment provided abstract concepts are not required of children aged <11 y. Parents often provide surrogate information but can accurately estimate only the food the child has eaten in their company. Parents and children together provide the best dietary information (41).

FFQs are a quick means of assessing dietary intake with low respondent burden and a high response rate. Work in the Bogalusa Study of children showed that children in grades 8–10 are accurate in reporting frequency (42). Further work by this group showed an FFQ to be a valid and reliable tool for describing the protein eating pattern of adolescents (43). In a study in which children aged 11 and 12 y completed their own semiquantitative FFQs (26), a questionnaire containing 175 food items was administered at the beginning and at the end of the study and was also sent to parents by mail. Over the next 3 mo the children filled out fourteen 24-h records. Mean nutrient intakes obtained from the FFQ derived from the child or parent were higher than intakes obtained from the dietary records. Because dietary records filled out by children without parent contribution were shown to have lower energy estimates than those obtained from doubly labeled water estimates of energy expenditure (44), the FFQ data we present may better represent total intake even though, in most cases, data were not derived from both parents and children simultaneously.

This study has several potential limitations. The children who took part are not representative of Tasmanian children. They were originally selected on the basis of having a high risk of sudden infant death syndrome. As a result, the sample had a high proportion of males, premature infants, teenage mothers, and mothers who smoked during pregnancy. These findings suggest that this group was of a lower socioeconomic status than is the Tasmanian population as a whole. In addition, subjects who were excluded because of providing inadequate dietary information appeared also to be of lower socioeconomic status than the participants, with higher rates of smoking and unemployment and lower rates of breast-feeding and lower tertiary education levels. According to Miettinen (45), to be generalizable to other populations, an analytic cohort study does not have to be representative of the community from which the sample was selected provided key criteria are met regarding the definition of eligible participants, the sample size, and a proper distribution of determinants, modifiers, and confounders. The present study fulfils all 3 criteria. The study population was explicitly defined and the sample size was adequate; furthermore, it had considerable heterogeneity of exposure to factors of causal interest. In addition, there were no significant differences in other important explanatory factors, and bone mass and adjustment for potential environmental confounders did not alter the urinary potassium–bone mass associations. Overall, these observations suggest that the associations we report may be generalizable to other prepubertal populations, even though there are no normative data on sodium and potassium excretion in Australian children.

In conclusion, urinary potassium was associated with BMD independent of lean body mass in these well-nourished, calcium-replete children. These findings should be confirmed in longitudinal studies that include repeated measurement of urinary potassium to better estimate usual intake and minimize regression dilution bias. Nevertheless, this association is likely to represent dietary intake of potassium and suggests that urinary potassium is superior to FFQs for assessing potassium intake in this age group.

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