Dietary potential renal acid load and renal net acid excretion in healthy, free-living children and adolescents[1–3]

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ABSTRACT

Background: There is increasing evidence that acid-base status has a significant effect on high-intensity physical performance, urolithiasis, and calcium metabolism. Experimental studies in adults showed that renal net acid excretion (NAE) can be reliably estimated from the composition of diets.

Objective: We investigated whether a reasonable estimation of NAE is also possible from the dietary records of free-living children and adolescents.

Design: Healthy children (aged 8 y; $n = 165$) and adolescents (aged 16–18 y; $n = 73$) each collected a 24-h urine sample and completed a weighed diet record on the same day. Urinary NAE was analyzed (NAE_{an}) and estimated (NAE_{es}). Potential renal acid load (PRAL), the diet-based component of NAE_{es}, corrects for intestinal absorption of ingested minerals and sulfur-containing protein. A urinary excretion rate of organic acids (OAs) proportional to body surface area was assumed for the complete estimate ($NAE_{es} = PRAL + OA_{es}$).

Results: Significant ($P < 0.001$) correlations between NAE_{es} and NAE_{an} were seen in the children ($r = 0.43$) and the adolescents ($r = 0.51$). A simplified estimate based on only 4 components of dietary PRAL (protein, phosphorus, potassium, and magnesium) yielded almost identical associations. Mean simplified NAE_{es} (32.6 ± 13.9 and 58.4 ± 22.0 mEq/d in the children and the adolescents, respectively) agreed reasonably with NAE_{an} (32.4 ± 15.5 and 52.8 ± 24.3 mEq/d, respectively).

Conclusions: Predicting NAE from dietary intakes, food tables, and anthropometric data is also applicable during growth and yields appropriate estimates even when self-selected diets are consumed. The PRAL estimate based on only 4 nutrients may allow relatively simple assessment of the acidity of foods and diets. *Am J Clin Nutr* 2003;77:1255–60.

KEY WORDS Biomarkers, children, adolescents, dietary record, food table, mineral intake, nutrient bioavailability, potential renal acid load, protein intake, renal net acid excretion, 24-h urine collection

INTRODUCTION

Acid-base status is becoming increasingly important in nutritional medicine and related fields. For example, in sports medicine, alkalization has been shown to increase the capacity for high-intensity exercise (1, 2). In clinical nutrition, the use of infant and preterm formulas that are not appropriately composed and that contain excessive amounts of acid equivalents was shown to cause catabolic effects including growth retardation of the infants (3–6). Similar negative effects can occur if inadequately composed synthetic amino acid mixtures and protein hydrolyzates are fed (7). Several nephrologic diseases, such as urolithiasis and renal insufficiency, require both control and manipulation of acid-base status (8–12).

Furthermore, acid-base status appears to be important in osteology. In vitro studies have shown a measurable calcium efflux from bone under acidosis-like metabolic conditions (13). Accordingly, in epidemiologic surveys, strong positive associations have been observed between rates of hip fracture in women and indexes of dietary animal protein intake (a major source of endogenous acid production) (14, 15). In contrast, the ingestion of doses of alkalizing potassium bicarbonate sufficient to neutralize endogenous acid was shown to improve calcium balance, reduce bone resorption, and increase the rate of bone formation in postmenopausal women (16).

Today, there is a general consensus that diet can markedly affect acid-base status and that a person's acid load can be specifically manipulated by dietary means (9, 17–21). An established method of estimating acid loads of foods or diets is by calculating the potential renal acid load (PRAL) (9, 22, 23). PRAL provides an estimate of the production of endogenous acid that exceeds the level of alkali produced for given amounts of foods ingested daily. The concept of PRAL calculation is physiologically based and takes into account different intestinal absorption rates of individual minerals and of sulfur-containing protein, as well as the amount of sulfate produced from metabolized proteins. This method of calculation was experimentally validated in healthy adults, and it showed that, under controlled conditions, acid loads and renal net acid excretion (NAE) can be reliably estimated from diet composition (18). The purpose of the present study was to investigate whether a reasonable estimation of acid loads (quantified as NAE in 24-h urine samples) is also possible from dietary records of free-living children and adolescents.

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SUBJECTS AND METHODS

Subjects, anthropometric measures, and dietary recording

The study was performed in a cross-sectional sample of 165 healthy children aged 8 y and 73 adolescents aged 16–18 y. The subjects were all participants in the Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study, an ongoing observational study of the interrelations among nutrition, growth, and metabolic and endocrine changes during childhood and adolescence. The study was approved by the institutional review board of the Research Institute of Child Nutrition Dortmund, and informed parental consent and each child's oral assent were obtained before entry into the study. Besides age, a further inclusion criterion was the completion of a 3-d weighed diet record and the collection of one 24-h urine sample during the 3-d period. Only the nutrient intakes recorded on the day of urine collection were used for the estimation of NAE and PRAL.

To exclude major errors in the 24-h urine collection, those samples that were reported to contain incomplete micturitions or that showed a daily creatinine excretion rate < 0.1 mmol/kg body wt, or both (24), were not considered. Details of the urine collection procedure were reported recently (24). Subjects were asked to follow their usual diets on the day of urine collection. Parents of the children (or the adolescents themselves) weighed and recorded all foods and fluids consumed and all leftovers with the use of electronic food scales (Wedo Digi 2000; Werner Dorsch GmbH, Rödermark, Germany). Product information from wrappers, cartons, and other containers of new or special food items not included in the food tables (*see* below) were kept and evaluated with the dietary records by our dietitians (25).

To validate dietary recording, the ratio of reported energy intake to predicted basal metabolic rate was calculated according to the method of Goldberg et al (26) with prior calculation of basal metabolic rate according to the method of Schofield (27). Records with individual ratios of energy intake to basal metabolic rate of < 1.06 were excluded from this study. The nutrient and energy contents of foods were taken from European standard nutrient tables (28–30), preferentially from those of Souci et al (30). Around the time of completion of the dietary record and urine collection, all children were examined by a pediatrician, and anthropometric measurements were obtained. Body weight was measured to the nearest 0.1 kg and height was measured to the nearest 0.1 cm with the use of an electronic scale (Seca 753 E; Seca Weighing and Measuring Systems, Hamburg, Germany) and a digital, telescopic, wall-mounted stadiometer (Harpenden; Holtain Ltd, Crymych, United Kingdom), respectively.

Quantification of urinary analytes and NAE

Acid-base status was determined in the freshly thawed 24-h samples. Urine pH, titratable acid (TA), ammonium (NH_4), and bicarbonate (HCO_3) were measured according to the method of Lüthy et al (31). Quantification of organic acids (OAs) was carried out according to the method of van Slyke and Palmer (32). NAE was calculated from the analytic data in the conventional manner as the sum of TA plus NH_4 minus HCO_3. Aliquots of all 24-h urine samples were stored (< −20 °C) for subsequent analysis. The cations potassium, magnesium, and calcium were measured by flame atomic absorption spectrometry (Perkin Elmer 1100 Spectrometer; Perkin Elmer, Überlingen, Germany); the detection limit is 0.01 mmol/L, and the intraassay and interassay precision is < 5%. Sulfate and phosphate were measured with a Dionex 2000 i/SP Ion chromatograph with an ion Pac AS4A column (Dionex GmbH, Idstein, Germany). Urinary creatinine was measured with the use of the kinetic Jaffé test (33) on a creatinine analyzer (Beckman-2; Beckman Instruments Inc, Fullerton, CA) according to the manufacturer's instructions.

Calculation of PRAL and estimation of urinary NAE

Because the sum of cations excreted in urine equals the sum of anions, urinary NAE (Σcationic TA + NH_4 − anionic HCO_3) is also equal to the difference between the sum of the major urinary nonbicarbonate anions (chloride, phosphate, sulfate, and OAs) minus the sum of the non-TA and non-NH_4 cations (sodium, potassium, magnesium, and calcium). The amounts of these nonbicarbonate anions and mineral cations in urine, except OAs, are primarily influenced by nutritional intake. OAs are mainly determined by body surface area and can thus be estimated from anthropometric measurements [OA_{es} (mEq/d) = body surface $area_{individual} \times 41/1.73$] (18, 34). The diet-based estimate of the urinary difference of nonbicarbonate anions (without OAs) and mineral cations is PRAL. To calculate PRAL, average intestinal net absorption rates of relevant nutrients (including protein to estimate sulfate) must be considered together with the grade of dissociation of phosphate at pH 7.4 and the ionic valence of magnesium and calcium (9, 18). On the basis of these PRAL-determining factors (and after the respective atomic weights are taken into account), nutrient-specific conversion factors are obtained that allow the calculation of PRAL directly from dietary intakes (9):

$$\begin{aligned}\text{PRAL (mEq/d)} = {} & 0.49 \times \text{protein (g/d)} \\ & + 0.037 \times \text{phosphorus (mg/d)} \\ & - 0.021 \times \text{potassium (mg/d)} \\ & - 0.026 \times \text{magnesium (mg/d)} \\ & - 0.013 \times \text{calcium (mg/d)} \end{aligned} \quad (1)$$

For the estimation of total urinary NAE, OA was considered in addition to PRAL ($NAE_{es} = PRAL + OA_{es}$).

In contrast to earlier studies that evaluated a limited number of foods and beverages (3, 4, 9, 18, 19), sodium and chloride were omitted from the present calculation of PRAL because, for some of the foods recorded, either the chloride data are missing from the food tables used, or, in the case of processed (salted) foods, they deviate unrealistically by more than ± 10% from the respective sodium values (9). This omission implies a certain insensitivity of our calculation to large differences in the intakes of these elements. As calculated from differences in urinary chloride and sodium excretion after the controlled ingestion of different diets, the resulting estimation error for PRAL can be as much as 16 mmol/d (18). However, in 24-h urine samples from randomly selected children and adolescents from the DONALD Study, the mean urinary ratio of sodium to chloride was found to be 1.02 (35).

Statistical analysis

Data are presented as means ± SDs. Pearson's correlation coefficients, a paired *t* test, and two-way analysis of variance (factors: age group and sex) were performed. In clinical comparison of a new measurement technique with an established one, the Bland-Altman limits of agreement (36) are usually calculated, but that was not done in the present study because measurement (NAE_{an}) and estimation (NAE_{es}) techniques were compared. Instead, Pitman's test (37) was used to determine statistically whether NAE_{es} may allow a better assessment of acid-base status (criterion

TABLE 1
Baseline characteristics (anthropometric, nutritional, and urinary) of the study population according to age and sex[1]

Age group and sex	Body height	Body weight	BMI	Intake		Urinary creatinine
				Energy	Total protein	
	cm	*kg*	*kg/m²*	*kJ/d*	*g/d*	*mmol/d*
8 y old						
Males (n = 82)	132.8 ± 5.8[2]	28.8 ± 4.3	16.3 ± 1.6	7706 ± 1478[3]	56.7 ± 14.9[4]	4.9 ± 1.1[3]
Females (n = 83)	131.0 ± 4.8	28.1 ± 4.5	16.3 ± 2.0	6871 ± 1360	50.8 ± 13.0	4.3 ± 0.8
16–18 y old						
Males (n = 44)	179.8 ± 6.8[3]	67.1 ± 9.2[4]	20.7 ± 2.2	11993 ± 2937[3]	95.3 ± 29.0[3]	13.6 ± 3.0[3]
Females (n = 29)	168.2 ± 5.4	61.0 ± 8.5	21.5 ± 2.6	8709 ± 2162	65.2 ± 20.9	10.3 ± 1.5

[1] $\bar{x}$ ± SD.
[2–4] Significantly different from females (unpaired t test): [2] $P < 0.05$, [3] $P < 0.001$, [4] $P < 0.01$.

variable: analyzed 24-h NAE) than does a recently proposed alternative, diet estimate (20). Residuals of the regressions of the criterion variable with both estimation models were calculated (residuals A and B); the sum (A + B) and the difference (A − B) were calculated, and then A + B was correlated with A − B. If this correlation differed significantly from zero, the residual with the smaller SD was the model with better fit. Significance was set at $P < 0.05$, and all tests were two-tailed. Analyses were performed with the use of SAS for WINDOWS software (38).

RESULTS

Anthropometric baseline characteristics, dietary intakes of energy and total protein, and 24-h urinary creatinine output of the subjects are shown in **Table 1**. In both age groups, correlations between estimated and analyzed urinary electrolytes were highly significant for sulfate, phosphate, and potassium ($P < 0.001$) and OAs ($P < 0.01$) and significant ($P < 0.05$) for magnesium (**Table 2**). Correlations for calcium were significant ($P < 0.05$) only in the 8-y-old children. Comparable clear associations were seen for both forms of NAE_{es} (with or without calcium inclusion) with NAE_{an} (Table 2). Correlation coefficients remained in the same range after simplified NAE and NAE_{an} were corrected for energy intake. Correspondingly, the simplified PRAL, which is also based on intakes of only 4 nutrients (without calcium), correlated highly significantly ($P < 0.001$) with the sum of the corresponding analyzed urine variables (Table 2). Significant ($P < 0.05$) differences between estimated and analyzed mean values of all electrolytes were seen in the 8-y-old children. In the adolescent group, most of the differences were not significant. Differences in relative values (means of estimated values as a percentage of analyzed means) were largest for calcium. Absolute differences were in the range of 0.6 (for magnesium in 8-y-old boys) to 9.6 mE/d (for potassium in male adolescents).

TABLE 2
Comparison and correlation of potential renal acid load (PRAL), estimated net acid excretion (NAE_{es}), and estimates of single urinary determinants of NAE (sulfate, phosphate, potassium, magnesium, calcium, and organic acids) with the respective data analyzed in 24-h urine samples[1]

	8-y-old group					16–18-y-old group				
	Boys (n = 82)		Girls (n = 83)			Males (n = 44)		Females (n = 29)		
	Estimated	Analyzed	Estimated	Analyzed	r	Estimated	Analyzed	Estimated	Analyzed	r
Sulfate (mEq/d)	27.7 ± 7.3	26.0 ± 7.6[2]	24.8 ± 6.3	21.4 ± 6.0[2]	0.53[3]	46.6 ± 14.2	39.7 ± 12.1[2]	31.9 ± 10.2	30.4 ± 7.7	0.61[3]
Phosphate (mEq/d)	40.6 ± 11.3	34.1 ± 9.7[2]	35.5 ± 10.2	28.5 ± 8.0[2]	0.56[3]	62.5 ± 21.6	56.8 ± 19.4	44.7 ± 12.3	43.2 ± 11.2	0.54[3]
Potassium (mEq/d)	52.2 ± 14.3	45.9 ± 13.3[2]	46.3 ± 13.1	37.9 ± 11.9[2]	0.54[3]	76.7 ± 25.6	67.1 ± 22.6[2]	62.0 ± 22.2	65.3 ± 21.4	0.54[3]
Magnesium (mEq/d)	6.8 ± 1.9	6.2 ± 1.8[2]	6.5 ± 2.4	5.8 ± 1.5[2]	0.26[3]	10.2 ± 3.5	9.7 ± 3.6	8.0 ± 2.2	8.0 ± 2.6	0.27[4]
Calcium (mEq/d)	10.9 ± 3.9	2.6 ± 1.7[2]	9.1 ± 4.0	2.5 ± 1.6[2]	0.25[5]	16.0 ± 7.1	6.6 ± 4.1[2]	11.9 ± 3.9	6.4 ± 3.2[2]	0.15
Organic acids (mEq/d)	24.5 ± 2.2	29.0 ± 6.4[2]	24.0 ± 2.1	24.9 ± 5.0	0.53[3]	43.8 ± 3.5	50.5 ± 10.4[2]	40.1 ± 2.9	45.2 ± 9.4[2]	0.43[5]
NAE_{es} (mEq/d)[6]	22.8 ± 13.9	37.0 ± 17.7[2]	22.5 ± 12.3	27.8 ± 11.4[2]	0.43[3]	50.1 ± 20.8	59.8 ± 26.0	34.8 ± 14.9	42.3 ± 17.2[2]	0.51[3]
Simplified NAE_{es} (mEq/d)[7]	33.7 ± 14.7	37.0 ± 17.7	31.6 ± 13.1	27.8 ± 11.4[2]	0.43[3]	66.1 ± 22.5	59.8 ± 26.0	46.6 ± 15.4	42.3 ± 17.2	0.54[3]
Simplified NAE_{es} and NAE_{an}, energy corrected ($mEq \cdot kJ^{-1} \cdot d^{-1} \times 10^3$)	4.4 ± 1.8	4.9 ± 2.3	4.7 ± 2.0	4.2 ± 2.0[2]	0.41[3]	5.6 ± 1.9	5.1 ± 2.3	5.5 ± 2.0	5.1 ± 2.3	0.51[3]
Simplified PRAL (mEq/d)[8]	9.2 ± 14.4	8.1 ± 14.7	7.6 ± 12.6	6.1 ± 11.2	0.53[3]	22.3 ± 22.1	18.9 ± 22.9[2]	6.6 ± 15.5	−0.13 ± 22.6	0.47[3]
Simplified PRAL compared with NAE_{an}					0.38[3]					0.51[3]

[1] $\bar{x}$ ± SD. NAE_{an}, analyzed NAE.
[2] Significantly different from estimated, $P < 0.05$ (paired t test).
[3] $P < 0.001$.
[4] $P < 0.05$.
[5] $P < 0.01$.
[6] With calcium.
[7] Without calcium. Estimated = sulfate + phosphate − potassium − magnesium + organic acids.
[8] PRAL = estimated sulfate + phosphate − potassium − magnesium. PRAL compared with the sum of corresponding urinary analytes (analyzed PRAL).

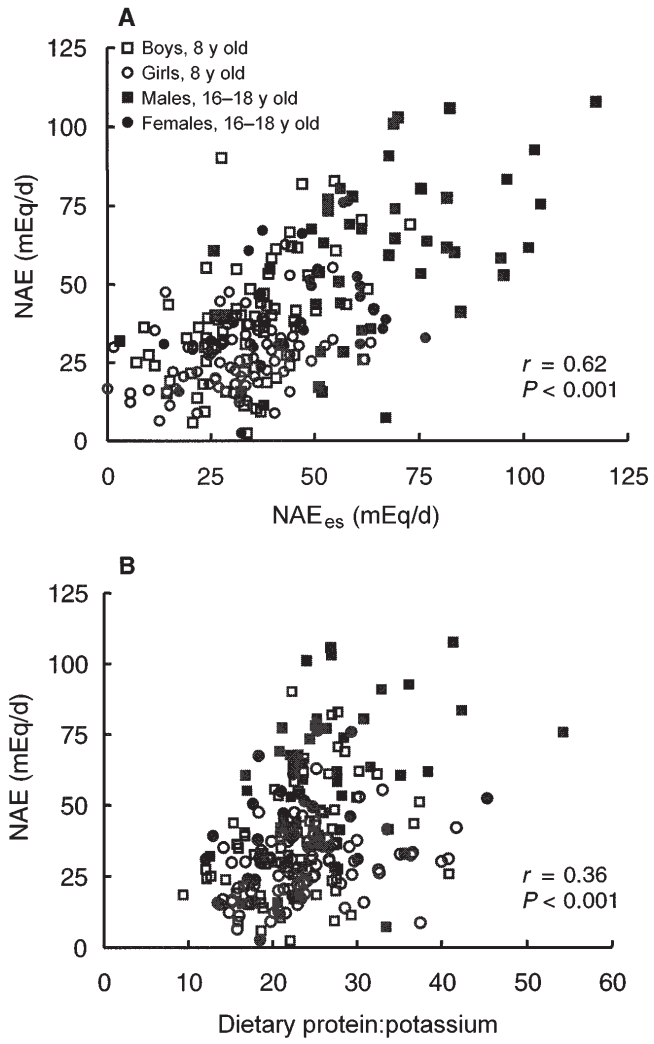

FIGURE 1. Twenty-four–hour urinary net acid excretion (NAE) in children and adolescents plotted against A) simplified estimated NAE (NAE_{es}) and B) the ratio of dietary protein to potassium (8-y-old group: $n = 82$ boys, 83 girls; 16–18-y-old group: $n = 44$ males, 29 females).

The NAE_{es} that included calcium underestimated NAE_{an} in all groups, significantly so in the 8-y-old children and in the adolescent girls. In comparing simplified NAE and PRAL (both without calcium) with the corresponding analyzed values, overestimations were seen (except for simplified NAE_{es} in 8-y-old boys), but most were not significant (Table 2). Correspondingly, when the sexes were combined, means of simplified NAE_{es} coincided with NAE_{an} in 8-y-old children (32.6 ± 13.9 versus 32.4 ± 15.5 mEq/d) and adolescents (58.4 ± 22.0 versus 52.8 ± 24.3 mEq/d). The sex differences that were present for simplified NAE_{es} and NAE_{an} were no longer significant after these variables had been corrected for individual energy intake. For respective mean values, *see* Table 2; *F* values from two-way analysis of variance for the factor sex were 11.5 ($P < 0.001$) for simplified NAE_{es}, 25.3 ($P < 0.001$) for NAE_{an}, 0.4 ($P = 0.5$) for energy-corrected simplified NAE_{es}, and 3.1 ($P = 0.08$) for energy-corrected NAE_{an}. Both simplified NAE_{es} and the ratio of protein to potassium are shown in **Figure 1** to be significantly ($P < 0.001$) correlated with NAE_{an}. However, the correlation coefficient was lower for the relation with protein:potassium, which is an alternative estimate of the alkalizing or acidifying potential of diets (20). Pitman's test yielded significantly smaller SDs of the residuals for the regression of NAE_{es} on NAE_{an} when compared with protein:potassium.

DISCUSSION

The results of this cross-sectional study indicate that an appropriate prediction of average urinary NAE is possible for healthy children and adolescents with the use of a simplified model for calculating PRAL. The PRAL estimate, which is based on food consumption according to a 24-h weighed diet record and the corresponding intakes of only 4 nutrients (as specified in standard food composition tables), was highly significantly correlated with NAE_{an} in 24-h urine samples.

Similar associations with NAE_{an} were seen for NAE_{es}, ie, for the PRAL plus an anthropometrically based estimate of the daily OA production rate, which usually varies moderately among different diets (18). The inclusion of estimated OA allowed a fairly good prediction of the total amount of actual NAE in children and adolescents. This absolute NAE prediction did not improve when dietary calcium was considered along with the intakes of protein, phosphate, potassium, and magnesium. As is widely recognized and also observed in the present study, the association between dietary estimated and renally excreted calcium is very weak. Data on urinary anion and cation balance regularly indicate that renal calcium output contributes less to overall urinary NAE than does any other mineral cation or nonbicarbonate anion (18, 21, 34). In a previous diet experiment, during which calcium intake was more strictly controlled, an overestimation of urinary calcium output by ≤3-fold was observed (18). This was similar to the present misestimations. Finally, renal calcium excretion is influenced by acid-base status itself as well as by sodium chloride ingestion. For all these reasons, it appears appropriate to use the simplified NAE_{es} (and PRAL calculation formula) that is based on only 4 dietary components and that no longer takes dietary calcium into account.

Overall, NAE_{es} that included calcium underestimated NAE_{an} in all groups (mostly significant), whereas simplified NAE_{es} and PRAL (both without calcium) moderately (mostly nonsignificantly) overestimated NAE_{an} and analyzed PRAL, respectively. There was one exception to this uniform direction of estimation effects: in 8-y-old boys, simplified NAE underestimated NAE_{an}. The reason for this is not quite clear. Possibly, the higher urinary output of OA in prepubertal boys than in prepubertal girls might have contributed to an elevated NAE_{an}. Usually, children with similar body surface area have comparable urinary excretion of OA (34). Despite the fact that OA is mainly determined by anthropometric measures, there is also a dietary influence. Fruit can contain considerable amounts of aromatic OAs in the form of phenolic and benzoic acids. Unlike dietary OAs such as citrate, malate, and succinate, aromatic OAs are not metabolically oxidized to bicarbonate and water but are metabolically inactivated (detoxified) and excreted (mainly via the kidney) as acids, partly in the form of hippuric acid. Recently, 15 phenolic and benzoic acids were identified in cranberries, and phenolic acid was the most abundant (39). The additional acidifying effect of such plant-derived OAs is not considered in our NAE estimation model and is probably one reason for the differences seen between estimated and analyzed OAs. Another source of inaccuracy stems from the fact that the sulfur content of different proteins varies, which certainly has an impact on the endogenously generated acidity

resulting in sulfate. However, our formulas do not take this into account. A more differentiated PRAL model might have reduced the discrepancies seen between estimated and analyzed sulfate values in our children and adolescents. A correspondingly revised PRAL model should differentiate between certain foods or food groups, depending on the amount of sulfur per gram of protein.

Despite the fact that magnesium (similar to calcium) contributes only modestly to the base-forming potential of total mineral cations (18, 21, 34), and despite merely moderate correlations between dietary and urinary magnesium, it proves advantageous to leave this mineral in the NAE calculation formula for an appropriate prediction of NAE_{an}. For the other dietary determinants of endogenous base and acid release, stronger correlations were observed between intakes and 24-h renal excretion rates. Correlation coefficients for potassium, phosphate, and sulfate ranged from 0.54 to 0.61, which agreed fairly well with the associations seen in diet studies, in which potassium, phosphate, and nitrogen (analyzed in 24-h urine samples) were used as biomarkers to validate dietary intakes (40–42). Although the differences between the measured and the estimated urinary excretion values for the above minerals were mostly significant, the absolute mean differences were not so large and were within the range of our previously reported controlled-diet study in adults (18). This overall reasonable agreement and reproducibility emphasize the high degree of reliability of weighed dietary records of food intake in the children and adolescents in the present study. However, the weighed dietary record is not in common use in the United States or in many other countries. Moreover, food tables differ. Therefore, reproducibility of our findings on mineral and NAE prediction may vary according to the dietary assessment tools used.

Diet-dependent changes in acid-base balance, which frequently are closely related to overall protein intake, have long been considered less relevant in preventive medicine. However, during the last years, increased evidence shows that dietary protein intake may have an important influence on skeletal health. Both negative and positive effects on bone values have been observed in animal, epidemiologic, and experimental diet studies (eg, 13–15, 43–45). There has not yet been appropriate evaluation of whether, in those studies indicating an anabolic effect of high protein intakes on bone (46, 47), a concomitantly greater consumption of alkalizing fruit and vegetables may have counterbalanced some of the potential detrimental acid effects of protein. One reason for this omission could be the lack of convenient methods for appropriately quantifying acid and alkali loads (22). A simplified approach based on 2 key dietary (and urinary) components has been suggested by Frassetto et al (20). They found that the ratio of dietary protein to potassium clearly predicts NAE. In addition, the PRAL of > 100 individual food items, as calculated according to our multiple-constituent model, correlated highly with the respective ratio of protein to potassium (20). Accordingly, the question arises as to whether it still is justified to use a more complex NAE prediction model.

One argument in favor of our prediction model is that, for specific population groups, the total NAE can be reasonably estimated from whole-food diets. This is not possible with the use of protein:potassium. In addition, in the young people we studied, NAE_{es} provided a significantly better fit with NAE_{an} than did protein:potassium. Frassetto et al (20) did not find a higher predictive ability of the multiple-dietary component estimate (sulfate + phosphate − potassium − magnesium) than of protein:potassium. One reason for this could be that they did not include estimates for average intestinal net absorption rates of individual nutrients in their calculation. However, both the present study and that of Frassetto et al (20) show that the readily available protein:potassium is useful for a rapid assessment of the alkalizing or acidifying potential of foods and diets.

Bearing in mind that our NAE (PRAL) calculation model has yielded reasonable estimates for the average diet-dependent acid-base status of groups, the model is certainly not precise enough to predict clinical findings in individual subjects. Furthermore, intestinal nutrient absorption (an important determinant of acid-base status) can vary considerably between persons and cannot be predicted from diet. In epidemiologic studies, however, it should be possible to detect such dietary PRAL levels, for groups and perhaps for certain persons, that have a greater or lesser risk of developing clinically relevant symptoms. For example, daily PRAL levels ≈0 mEq/d (corresponding to an NAE range of 40–50 mEq/d in adults) usually lead to 24-h urine pH values that clearly exceed a level of 6.0, and they may thus prevent the precipitation of uric acid and cystine stones in predisposed patients (8). On the other hand, PRAL values > 70–80 mEq/d (NAE > 120 mEq/d) can result in so-called maximum renal acid stimulation (5, 21), a physiologic condition during which a greater renal calcium loss occurs (21).

In conclusion, our method for predicting NAE from dietary intakes, food tables, and anthropometric data is also applicable during growth and, with the use of weighed dietary records, yields appropriate estimates. The PRAL estimate based on only 4 nutrients appears to provide a reasonable tool for the relatively simple assessment of food and dietary acidity. With such a tool, it may be possible to determine whether long-term high acid loading through diet may have adverse effects on bone or other biological variables. However, our prediction model should be retested with alternative dietary assessment methods and food tables that are in common use in the United States or Canada.

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