

# Potential renal acid load of foods and its influence on urine pH

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## ABSTRACT

The purpose of this study was to calculate the potential renal acid load (PRAL) of selected, frequently consumed foods. A physiologically based calculation model was recently validated to yield an appropriate estimate of renal net acid excretion (NAE); the model depends primarily on nutrient intake data. When nutrient data from actual food composition tables were used, the calculation model yielded PRAL values that ranged from an average maximum of 23.6 mEq/100 g for certain hard cheeses over 0 mEq/100 g for fats and oils to an average minimum of approximately -3 mEq/100 g for fruits and fruit juices and vegetables. By means of these PRAL data (summed according to the amounts of foods and beverages consumed daily and by an estimate of excretion of organic acids [based on body size]), the daily NAE can be calculated. This calculation methodology, primarily based on PRAL, allows an appropriate prediction of the effects of diet on the acidity of urine. For practical applicability in dietetic prevention of recurrent urolithiasis or in other fields of dietetics, the additionally determined correlation ( $r = .83$ ;  $P < .001$ ) between NAE and urine pH can be used to ascertain NAE target values for a desired urine pH modification. *J Am Diet Assoc.* 1995; 95:791-797.

Urolithiasis plays a quantitatively important role among the urologic diseases. Renal hydrogen ion excretion (ie, the urine pH) is generally accepted as a urinary risk factor in most types of urinary stone disease (1). Although scientists have known for several decades that the composition of the diet influences the urine pH (2), only recently has clear, experimental evidence been provided that it is possible to efficiently modify or adjust the urine pH by purely dietetic means (3-5).

Because previous methodologic efforts failed to provide reliable estimates of the net acid loads produced by diets (2), we tested a different, physiologically based calculation model developed for the prediction of renal net acid excretion (NAE) from nutrient intake data (4). This calculation model, which takes into account the mineral and protein composition of foods, the average intestinal absorption rates of the respective nutrients, sulfur metabolism, and urinary excretion of organic acids, proved to be appropriate for the prediction of NAE (4). In addition, we were able to predict and adjust the urine pH of healthy adults (5) when we used this model along with the correlation (shown in this article) between the analytically determined NAE and the urine pH. On the basis of these findings and earlier studies on protein hydrolyzates (6), synthetic amino acid mixtures (6), and milk formulas (7,8) — each confirming the applicability of the calculation model — it now appears justified and possible to estimate the potential renal acid load (PRAL) of foods.

The purpose of this study was to calculate and specify the PRAL of selected, frequently consumed foods (quoted per 100 g) and to demonstrate how to use these data for the estimation of NAE and for prediction of the corresponding urine pH in persons consuming definite diets. In addition to the dietetic prevention of recurrent urolithiasis, our findings could be of practical relevance in the areas of urinary tract infections, osteoporosis, and sports nutrition.

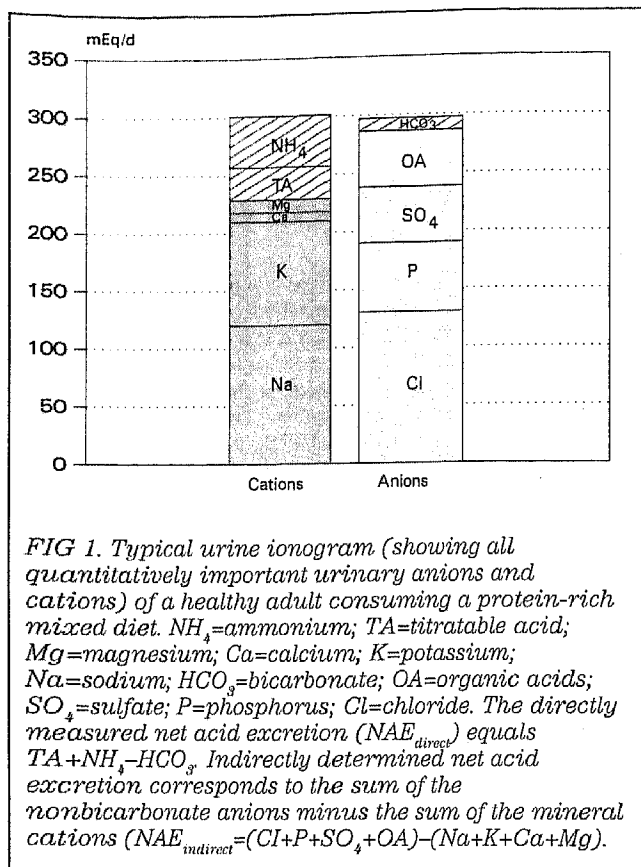
## MATERIAL AND METHODS

### Calculation Model

The method used for calculation of the PRAL of foods is based on the calculation model we developed for prediction of NAE from nutrient intake data (4). The *directly* determined NAE (based on urine analyses) is calculated in the conventional manner as the sum of titratable acid and ammonium minus bicarbonate (Figure 1). As is discernible from Figure 1, NAE can also be determined *indirectly* from the difference of the sum of the remaining important urinary anions — chloride, phosphorus, sulfate, and organic acids (nonbicarbonate anions) — minus the sum of the mineral cations — sodium, potassium, calcium, and magnesium. The amounts of these electrolytes in urine are determined primarily by nutritional intake. In the case of organic acids, the major determinant is body surface area or simply body weight (9). If growth, mineral losses through the skin, and transient, metabolic non-steady-state conditions are not considered (which appears to be a rational approach for healthy, nonpregnant adults under nor-

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**Table 1**  
Average intestinal net absorption rates for minerals and protein in adults consuming mixed diets and derived factors for conversion of nutrient intakes (milligrams per 100 g food) to milliequivalents (quoted per 100 g food) estimating the food-induced urinary excretion of the corresponding electrolytes

Nutrients <sup>a</sup>	% Absorption	References	Urinary ions <sup>a</sup>	Conversion factors <sup>b</sup> (mg Intake → mEq excretion)
Na	95	26	Na	0.0413
K	80	26,27	K	0.0205
Ca	25 <sup>c</sup>	28	Ca	0.0125
Mg	32 <sup>c</sup>	29	Mg	0.0263
Cl	95	26	Cl	0.0268
P	63 <sup>c</sup>	28	PO <sub>4</sub>	0.0366
Protein	75	30	SO <sub>4</sub>	0.4888 × 10 <sup>-3</sup>

<sup>a</sup>Na = sodium; K = potassium; Ca = calcium; Mg = magnesium; Cl = Chloride; P = phosphorus; PO<sub>4</sub> = phosphate; SO<sub>4</sub> = sulfate.  
<sup>b</sup>To yield the conversion factors, % absorption was divided by the respective atomic weight (Na=23.0; K=39.1; Ca=40.1; Mg=24.3; Cl=35.5; P=31.0) and by 100, thereby allowing the estimation of the urinary excretion of the nutrients (expressed in mmol). For Ca and Mg, the ionic valence (×2), and for P the grade of dissociation of pH 7.4 (×1.8), was also considered (leading to the unit mEq). For total protein, an average content of 2.4% methionine and 2.0% cysteine was assumed (4). The atomic weights of methionine (149.2) and cysteine (121.2) were used to estimate the metabolized mEq SO<sub>4</sub> (=mmol SO<sub>4</sub> ×2) converted from absorbed amounts of sulfur-containing amino acids.  
<sup>c</sup>Values have been calculated from the regression equations for the daily mineral intake and the corresponding urinary electrolyte excretion rates (as described in the references). For calculation, an average daily intake of the respective nutrient according to the US Recommended Dietary Allowance (31) (reference adults, 25 to 50 years old) was assumed (Ca=800 mg/day; P=800 mg/day; Mg=315 mg/day—Mg allowances diverging for women and men were averaged).

mal living conditions), urinary excretion of electrolytes corresponds to amounts absorbed intestinally. Thus, renal excretion of NAE-relevant electrolytes (ie, NAE itself) can be estimated from nutrient intake and anthropometric data (4).

For our study, the average, absorbable amounts of all relevant nutrients (representing NAE<sub>indirect</sub> without organic acids, see Figure 1) were estimated for selected foods and beverages from data on the nutrient composition of foods (per 100-g edible portion, as provided by current food tables) and from average net absorption rates taken from the literature (Table 1). Organic acid excretion, the primarily diet-independent component of the NAE<sub>indirect</sub>, is not immediately considered for estimation of foodborne PRAL. However, when estimating the NAE of persons consuming known amounts of definite foods, daily organic acid excretion must be taken into account.

As can be deduced from the equations given in the legend of Figure 1, the indirect determination of NAE (and consequently estimation of the PRAL) involves adding anions and subtracting cations with partly different charges and dissociation properties. To manage this adequately, the units of measure of the relevant base- and acid-forming elements (milligrams per 100 g food) were converted to milliequivalents (mEq) as described in Table 1. With respect to the metabolic conversion of organically bound sulfur to sulfate, the average content of sulfur-containing amino acids in protein was also considered.

For a further characterization of foods according to particular nutrient categories that appear to be major determinants of the respective acid- or base-forming potential, urinary phosphorus excess and alkali excess were calculated as described in the notes to Table 2. The nutrient data used for PRAL calculation were taken nearly exclusively from reference 10. Additionally used sources are given in the footnotes to Table 2.

**Subjects**

Twenty-four-hour urine samples from 63 volunteers aged 16 to 49 years were analyzed for parameters of acid-base status to characterize the relationship between renal NAE and urine pH. The subjects were participants in studies of the Research Institute of Child Nutrition (Dortmund, Germany). All subjects described themselves as being healthy and none had a past medical history of renal, endocrine, metabolic, or cardiovascular disease. Because of the lack of an adequately large sample of adult female volunteers who collected timed 24-hour urine samples, only males were included in the investigation.

Males with a high protein intake (170±60 g/day) were drawn from a study group of young bodybuilders (16 to 29 years old) in whom the effects of dietary protein on metabolic and renal parameters were investigated (11,12). A corresponding control group (11,12) consisted of adults and adolescents (>16 years old) consuming normal mixed diets (protein intake=98±20 g/day). To yield urine pH data related to low NAE values, specimens of males who were consuming a lactovegetarian diet (comparable to that described previously [4]—protein content=50 g/day) were also analyzed.

Three subjects were excluded from the final set of data because of an incomplete 24-hour urine collection (n=1) or extreme ingestion of protein supplements (protein intake>300 g/day) associated with biochemical indications of chronic renal acid stimulation (n=2). Therefore, 60 subjects were considered in regression analysis.

**Sample Collections, Analyses, and Statistics**

Collections of timed 24-hour urine samples and measurements of the parameters of acid-base status (urinary pH, titratable acid, ammonium, and bicarbonate) were carried out as described previously (4). The correlation between urinary pH

**Table 2**  
Nutrient<sup>a</sup> content (10) and estimated potential renal acid load (PRAL)<sup>b</sup> of 114 frequently consumed foods and beverages (related to 100-g edible portion)

Food group and food	Energy	Protein	Na	K	Ca	Mg	P	Cl	SO <sub>4</sub> <sup>b</sup>	PEX <sup>b</sup>	ALEX <sup>b</sup>	PRAL <sup>b</sup>
	<i>kcal</i>	<i>g</i>	<i>mg</i>						<i>mEq</i>			
<b>Beverages</b>												
Beer, draft	32	0.3	12	38	11	9	13	32	0.1	0.1	0.4	-0.2
Beer, pale (Vollbier, hell) <sup>c</sup>	45	0.5	5	38	4	9	28	35	0.2	0.7	0.0	0.9
Beer, stout, bottled	37	0.3	23	45	8	8	17	48	0.1	0.3	0.6	-0.1
Coca-Cola	39	0.0	8	1	4	1	15	10	0.0	0.5	0.1	0.4
Cocoa, made with semi-skimmed milk	57	3.5	70	170	120	20	100	100	1.7	1.6	3.7	-0.4
Coffee, infusion, 5 minutes	2	0.2	0	66	2	6	2	0	0.1	-0.1	1.4	-1.4
Mineral water (Apollinaris) <sup>d</sup>	0	0.0	43	3	9	10	0	14	0.0	-0.4	1.5	-1.8
Mineral water (Volvic) <sup>d</sup>	0	0.0	1	1	1	1	0	1	0.0	0.0	0.0	-0.1
Red wine	68	0.2	10	130	7	11	14	18	0.1	0.1	2.6	-2.4
Tea, Indian, infusion	0	0.1	0	17	0	1	1	0	0.0	0.0	0.3	-0.3
White wine, dry	66	0.1	4	61	9	8	6	10	0.0	-0.1	1.1	-1.2
<b>Fats and oils</b>												
Butter	737	0.5	11	15	15	2	24	17	0.2	0.6	0.3	0.6
Margarine	739	0.2	800	5	4	1	12	1,200	0.1	0.4	1.0	-0.5
Olive oil	899	0.0	0	0	0	0	0	0	0.0	0.0	0.0	0.0
Sunflower seed oil	899	0.0	0	0	0	0	0	0	0.0	0.0	0.0	0.0
<b>Fish</b>												
Cod, fillets	76	17.4	77	320	16	23	170	110	8.5	5.4	6.8	7.1
Haddock	73	16.8	120	300	18	23	170	160	8.2	5.4	6.8	6.8
Herring	234	16.8	67	340	33	29	210	76	8.2	6.5	7.7	7.0
Trout, brown, steamed	135	23.5	88	370	36	31	270	70	11.5	8.6	9.3	10.8
<b>Fruits, nuts, and fruit juices</b>												
Apple juice, unsweetened	38	0.1	2	110	7	5	6	3	0.0	0.0	2.3	-2.2
Apples, 15 varieties, flesh and skin, average	47	0.4	3	120	4	5	11	0	0.2	0.2	2.6	-2.2
Apricots	31	0.9	2	270	15	11	20	3	0.4	0.3	5.5	-4.8
Bananas	95	1.2	1	400	6	34	28	79	0.6	0.1	6.1	-5.5
Black currants	28	0.9	3	370	60	17	43	15	0.4	0.4	7.3	-6.5
Cherries	48	0.9	1	210	13	10	21	0	0.4	0.3	4.3	-3.6
Grape juice, unsweetened	46	0.3	7	55	19	7	14	6	0.1	0.1	1.3	-1.0
Hazelnuts	650	14.1	6	730	140	160	300	18	6.9	5.0	14.7	-2.8
Kiwi fruit	49	1.1	4	290	25	15	32	39	0.5	0.5	5.1	-4.1
Lemon juice	7	0.3	1	130	7	7	8	3	0.1	0.0	2.6	-2.5
Orange juice, unsweetened	36	0.5	10	150	10	8	13	9	0.2	0.1	3.2	-2.9
Oranges	37	1.1	5	150	47	10	21	3	0.5	-0.1	3.2	-2.7
Peaches	33	1.0	1	160	7	9	22	0	0.5	0.5	3.3	-2.4
Peanuts, plain	564	25.6	2	670	60	210	430	7	12.5	9.5	13.6	8.3
Pears, 3 varieties, flesh and skin, average	40	0.3	3	150	11	7	13	1	0.1	0.2	3.2	-2.9
Pineapple	41	0.4	2	160	18	16	10	29	0.2	-0.3	2.6	-2.7
Raisins	272	2.1	60	1,020	46	35	76	9	1.0	1.3	23.1	-21.0
Strawberries	27	0.8	6	160	16	10	24	18	0.4	0.4	3.0	-2.2
Walnuts	688	14.7	7	450	94	160	380	24	7.2	8.5	8.9	6.8
Watermelon	31	0.5	2	100	7	8	9	0	0.2	0.0	2.1	-1.9
<b>Grain products</b>												
Bread, rye flour, mixed <sup>c,e</sup>	211	6.4	537	185	23	0	136	827	3.1	4.7	3.8	4.0
Bread, rye flour <sup>c,e</sup>	194	6.8	527	291	43	0	198	812	3.3	6.7	6.0	4.1
Bread, wheat flour, mixed <sup>c,e</sup>	233	6.2	553	177	17	0	127	852	3.0	4.4	3.6	3.8
Bread, wheat flour, whole meal <sup>c,e</sup>	198	7.0	380	270	63	92	196	585	3.4	4.0	5.6	1.8
Bread, white wheat	235	8.4	520	110	110	24	91	820	4.1	1.3	1.8	3.7
Cornflakes	360	7.9	1,110	100	15	14	50	1,820	3.9	1.3	-0.9	6.0
Crispbread, rye	321	9.4	220	500	45	100	310	370	4.6	8.2	9.4	3.3
Noodles, egg	391	12.1	180	260	28	43	200	277	5.9	5.8	5.3	6.4
Oat flakes, rolled oats (Haferflocken) <sup>c</sup>	355	12.5	5	335	54	139	391	61	6.1	10.0	5.4	10.7
Rice, brown	357	6.7	3	250	10	110	310	230	3.3	8.3	-0.9	12.5
Rice, white, easy cook	383	7.3	4	150	51	32	150	10	3.6	4.0	3.0	4.6
Rice, white, easy cook, boiled	138	2.6	1	54	18	11	54	4	1.3	1.5	1.0	1.7
Rye flour, whole	335	8.2	1	410	32	92	360	0	4.0	10.4	8.4	5.9
Spaghetti, white	342	12.0	3	250	25	56	190	25	5.9	5.2	4.6	6.5
Spaghetti, whole meal	324	13.4	130	390	31	120	330	210	6.5	8.5	7.7	7.3
Wheat flour, white, plain	341	9.4	3	150	15	20	110	81	4.6	3.3	1.0	6.9
White flour, whole meal	310	12.7	3	340	38	120	320	38	6.2	8.1	6.1	8.2
<b>Legumes</b>												
Beans, green/French beans	24	1.9	0	230	36	17	38	9	0.9	0.5	4.5	-3.1
Lentils, green and brown, whole, dried	297	24.3	12	940	71	110	350	87	11.9	9.0	17.4	3.5
Peas	83	6.9	1	330	21	34	130	39	3.4	3.6	5.8	1.2

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PERSPECTIVES IN PRACTICE

**Table 2 (cont'd)**  
Nutrient<sup>a</sup> content (10) and estimated potential renal acid load (PRAL)<sup>b</sup> of 114 frequently consumed foods and beverages (related to 100-g edible portion)

Food group and food	Energy	Protein	Na	K	Ca	Mg	P	Cl	SO <sub>4</sub> <sup>b</sup>	PEX <sup>b</sup>	ALEX <sup>b</sup>	PRAL <sup>b</sup>
	<i>kcal</i>	<i>g</i>	← <i>mEq</i> →			← <i>mg</i> →			← <i>mEq</i> →			
<b>Meat and meat products</b>												
Beef, lean only	123	20.3	61	350	7	20	180	59	9.9	6.0	8.1	7.8
Chicken, meat only	121	20.5	81	320	10	25	200	78	10.0	6.5	7.8	8.7
Corned beef, canned	217	26.9	950	140	14	15	120	1,430	13.1	3.8	3.8	13.2
Frankfurters <sup>c</sup>	274	9.5	980	98	34	9	130	1,509	4.6	4.1	2.0	6.7
Liver sausage <sup>c</sup>	310	12.9	860	170	26	12	230	1,324	6.3	7.8	3.5	10.6
Luncheon meat, canned <sup>e</sup>	313	12.6	1,050	140	15	8	200	1,617	6.2	6.9	2.9	10.2
Pork, lean only	147	20.7	76	370	8	22	200	71	10.1	6.6	8.8	7.9
Rump steak, lean and fat	197	18.9	51	330	6	20	210	49	9.2	7.1	7.6	8.8
Salami <sup>c</sup>	491	19.3	1,850	160	10	10	160	2,849	9.4	5.5	3.3	11.6
Turkey, meat only	107	21.9	54	300	8	23	190	48	10.7	6.2	7.1	9.9
Veal, fillet	109	21.1	110	360	8	25	260	68	10.3	8.8	10.1	9.0
<b>Milk, dairy products, and eggs</b>												
Buttermilk <sup>c</sup>	39	3.5	57	147	109	16	90	100	1.7	1.5	2.7	0.5
Camembert <sup>c</sup>	297	20.9	650	100	350	21	310	1,001	10.2	6.4	2.1	14.6
Cheddar-type, reduced fat	261	31.5	670	110	840	39	620	1,110	15.4	11.2	0.2	26.4
Cheese, Gouda	375	24.0	910	91	740	38	490	1,440	11.7	7.7	0.9	18.6
Cottage cheese, plain	98	13.8	380	89	73	9	160	550	6.7	4.7	2.8	8.7
Creams, fresh, sour	205	2.9	41	110	93	10	81	81	1.4	1.5	1.8	1.2
Eggs, chicken, whole	147	12.5	140	130	57	12	200	160	6.1	6.3	4.2	8.2
Eggs, white <sup>f</sup>	36	9.0	190	150	5	11	33	170	6.6	0.9	6.4	1.1
Eggs, yolk	339	16.1	50	120	130	15	500	140	7.9	16.3	0.8	23.4
Fresh cheese (Quark) <sup>c</sup>	112	12.5	35	87	85	11	165	130	6.1	4.7	-0.3	11.1
Full-fat soft cheese <sup>c</sup>	313	8.6	330	150	110	9	130	508	4.2	3.1	3.1	4.3
Hard cheese, average of 4 types	405	24.7	620	82	670	24	470	980	12.1	8.2	1.0	19.2
Ice cream, dairy, vanilla	194	3.6	69	160	130	13	110	110	1.8	2.1	3.2	0.6
Milk, whole, evaporated	151	8.4	180	360	290	29	260	250	4.1	5.1	8.1	1.1
Milk, whole, pasteurized and sterilized	66	3.2	55	140	115	11	92	100	1.6	1.6	2.5	0.7
Parmesan	452	39.4	1,090	110	1,200	45	810	1,820	19.3	13.5	-1.5	34.2
Processed cheese, plain <sup>c</sup>	330	20.8	1,320	130	600	22	800	2,033	10.2	21.2	2.7	28.7
Yogurt, whole milk, fruit	105	5.1	82	210	160	16	130	150	2.5	2.3	3.7	1.2
Yogurt, whole milk, plain	79	5.7	80	280	200	19	170	170	2.8	3.2	4.5	1.5
<b>Sugar, preserves, and sweets</b>												
Chocolates, milk	529	8.4	120	420	220	55	240	270	4.1	4.6	6.3	2.4
Honey	288	0.4	11	51	5	2	17	18	0.2	0.5	1.0	-0.3
Madeira cake <sup>c</sup>	393	5.4	380	120	42	12	120	585	2.6	3.6	2.5	3.7
Marmalade	261	0.1	18	44	35	4	13	7	0.0	-0.1	1.5	-1.5
Sugar, white	409	0.0	0	2	2	0	0	0	0.0	0.0	0.0	-0.1
<b>Vegetables</b>												
Asparagus	25	2.9	1	260	27	13	72	60	1.4	2.0	3.8	-0.4
Broccoli, green	33	4.4	8	370	56	22	87	100	2.2	1.9	5.2	-1.2
Carrots, young	30	0.7	40	240	34	9	25	39	0.3	0.3	5.5	-4.9
Cauliflower	34	3.6	9	380	21	17	64	28	1.8	1.6	7.4	-4.0
Celery	7	0.5	60	320	41	5	21	130	0.2	0.1	5.6	-5.2
Chicory	11	0.5	1	170	21	6	27	25	0.2	0.6	2.9	-2.0
Cucumber	10	0.7	3	140	18	8	49	17	0.3	1.4	2.5	-0.8
Eggplant	15	0.9	2	210	10	11	16	14	0.4	0.2	4.0	-3.4
Leeks	22	1.6	2	260	24	3	44	59	0.8	1.2	3.8	-1.8
Lettuce, average of 4 varieties	14	0.8	3	220	28	6	28	47	0.4	0.5	3.4	-2.5
Lettuce, iceberg	13	0.7	2	160	19	5	18	42	0.3	0.3	2.2	-1.6
Mushrooms, common	13	1.8	5	320	6	9	80	69	0.9	2.6	4.9	-1.4
Onions	36	1.2	3	160	25	4	30	25	0.6	0.7	2.7	-1.5
Peppers, <i>Capiscum</i> , green	15	0.8	4	120	8	10	19	19	0.4	0.3	2.1	-1.4
Potatoes, old	76	2.1	7	360	5	17	37	66	1.0	0.8	5.9	-4.0
Radish, red	12	0.7	11	240	19	5	20	37	0.3	0.4	4.4	-3.7
Spinach	25	2.8	140	500	170	54	45	98	1.4	-1.9	13.4	-14.0
Tomato juice	14	0.8	230	230	10	10	19	400	0.4	0.3	3.5	-2.8
Tomatoes	17	0.7	9	250	7	7	24	55	0.3	0.6	4.0	-3.1
Zucchini	18	1.8	1	360	25	22	45	45	0.9	0.8	6.2	-4.6

<sup>a</sup>Key: Na = sodium; K = potassium; Ca = calcium; Mg = magnesium; P = phosphorus; Cl = chloride.

<sup>b</sup>The characteristic postabsorption determinants of PRAL are also presented; these are the primarily protein-dependent urinary sulfate excretion: SO<sub>4</sub>; the phosphate excess: PEX (PEX [mEq] = PO<sub>4</sub> - Ca - Mg); and the alkali excess: ALEX (ALEX [mEq] = Na + K - Cl). Each is estimated from the corresponding nutrient data by the conversion factors described in Table 1; PRAL (mEq of Cl + PO<sub>4</sub> + SO<sub>4</sub> - Na - K - Ca - Mg) also corresponds to SO<sub>4</sub> + PEX - ALEX.

<sup>c</sup>Data were derived from reference 32.

<sup>d</sup>Data were derived from the manufacturer's literature (Apollinaris, Bad Neuenahr-Ahrweiler, Germany; Volvic, Puy-de-Dome, France).

<sup>e</sup>For those processed (ie, salted) foods for which the tabulated Cl contents deviated by more than ± 10% from the values determined under the assumption of an equimolar Na and Cl content, Cl was calculated from the listed Na data on an equimolar basis, ie, Cl (mg) = Na (mg) × 1.54.

<sup>f</sup>For egg white protein, known to have a particularly high methionine and cysteine content, a 1.5-fold higher conversion factor (ie, 0.7332 × 10<sup>-3</sup>) was used to estimate renal sulfate excretion. Methionine and cysteine content related to 100 g protein is approximately 1.5-fold higher for egg white than, eg, for beef (32).

and renal NAE was assessed using Pearson correlation coefficients, and the corresponding regression equation was obtained from simple linear regression analysis. The average pH values obtained for definite NAE ranges are given as arithmetic means (Figure 2). The statistical procedures and the calculations of phosphorus excess, alkali excess, and PRAL (each per 100 g food) from the nutrient values of the food tables were conducted with the Statistical Package for the Social Sciences (SPSS/PC+, version 4.0, SPSS, Chicago, Ill).

## RESULTS

The calculated acid-forming potential (or base-forming potential) of more than 100 frequently consumed foods and beverages is listed along with postabsorptive urinary determinants, sulfate, phosphorus excess, and alkali excess, according to nine main food groups in Table 2. The calculation model yielded PRAL values ranging from a maximum of 34.2 mEq/100 g (parmesan cheese) over 0 mEq/100 g for fat and oils to a minimum of -21 mEq/100 g (raisins). Among the raw (ie, nondried) fruits, the base-forming potential was similar to that of vegetables. This can also be seen from Table 3, which gives the average PRAL values for certain groups and subgroups of foods. Along with fruits and fruit juices and vegetables, alkali-rich, low-phosphorus beverages have the lowest (ie, negative) PRAL values. According to Table 3, these foods are followed, in the order of gradually increasing average PRAL values, by alkali-poor (low-phosphorus) beverages, fats and oils, milk and noncheese dairy products, bread, noodles, and flour. Fish, meat and meat products, and cheeses are the food groups with the highest PRAL.

Table 4, an example of an extremely simplified diet, indicates that the exchange of only a few foods (protein-rich or alkali-poor vs alkali-rich) can markedly alter the daily intake of acid equivalents. Table 4 also demonstrates that the renal NAE is not affected solely by food-dependent acid loads but also by an important individual factor: the daily excretion rates of organic acids. Use of the average PRAL values (listed in Table 3) instead of the data calculated for single foods yielded moderately different estimates for the diet-induced daily acid loads.

The correlation found for urine pH and renal NAE is depicted in Figure 2. The corresponding regression equation and regression line, as well as the single pH values (each representing the mean of a certain NAE interval of 40 mEq), indicate that consumption of diets with an estimated renal NAE of about 100 mEq (diet A in Table 4) and about 30 mEq (diet B in Table 4) should result in average 24-hour urine pH of about 5.9 and 6.6, respectively. In other words, corresponding urine pH values are attainable with diets yielding daily PRAL values of either approximately 60 or -10 mEq/day.

## DISCUSSION

According to the calculation model described, negative PRAL values (indicating an excess of the base-forming potential of foods) were nearly exclusively found in the vegetable and fruit groups. In contrast, the highest acid loads originated in cheese, followed by meat, fish, and grain products. Similar trends, although with marked deviations for individual foods, were observed by other investigators whose calculations were based on acid-alkaline ash analyses (13,14) or on current food tables (2). However, none of these researchers considered the (average) net absorption rates of the relevant minerals.

In the present study, lower absolute values were found for both the acid-forming and the base-forming potential, especially for food groups with the highest and the lowest (ie, the negative) acid excess per 100 g food. Thus, compared with the earlier calculations, the total range of the potential acid or base

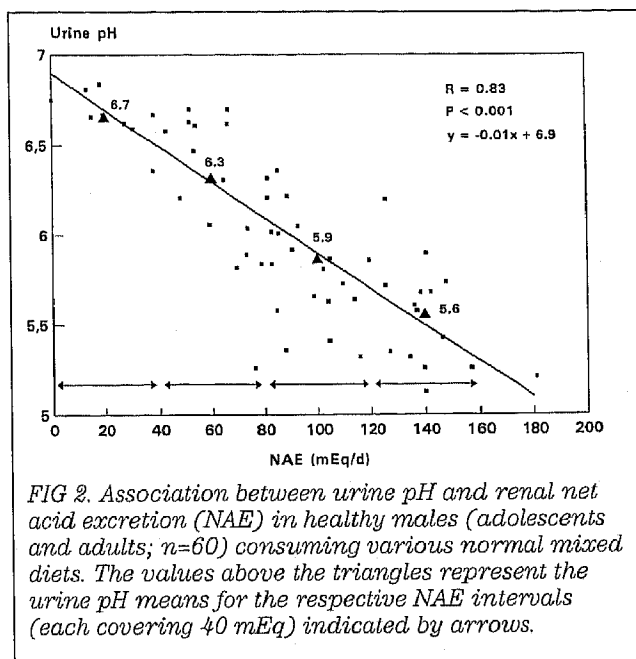


FIG 2. Association between urine pH and renal net acid excretion (NAE) in healthy males (adolescents and adults; n=60) consuming various normal mixed diets. The values above the triangles represent the urine pH means for the respective NAE intervals (each covering 40 mEq) indicated by arrows.

Table 3

Average potential renal acid loads (PRAL)<sup>a</sup> of certain food groups and combined foods (related to 100-g edible portion)

Food group	PRAL (mEq)
<b>Beverages</b>	
Alkali-rich and low phosphorus <sup>b</sup>	-1.7
Alkali-poor and low phosphorus <sup>c</sup>	0
<b>Fats and oils</b>	0
<b>Fish</b>	7.9
<b>Fruits and fruit juices<sup>d</sup></b>	-3.1
<b>Grain products<sup>e</sup></b>	
Bread	3.5
Flour	7.0
Noodles, spaghetti	6.7
<b>Meat and meat products</b>	9.5
<b>Milk and dairy products</b>	
Milk and noncheese products <sup>g</sup>	1.0
Cheeses with lower protein content <sup>h</sup>	8.0
Cheeses with higher protein content <sup>i</sup>	23.6
<b>Vegetables<sup>j</sup></b>	-2.8

<sup>a</sup>Data represent the arithmetic mean of the PRAL values of the respective foods listed in Table 2.

<sup>b</sup>Beverages (phosphorus <30 mg/100 g) with several times higher sodium + potassium content compared to chloride, for example, red wine, white wine, certain mineral (soda) waters, and coffee.

<sup>c</sup>Beverages (phosphorus <30 mg/100 g) with similar sodium + potassium vs chloride content. Cocoa (alkali- and phosphorus-rich) also falls in this PRAL category. Because of a medium phosphorus content (eg, 28 mg/100 g) some European pale beers have a relatively high PRAL value (about 1 mEq/100 g).

<sup>d</sup>Without dried fruits.

<sup>e</sup>Irrespective of the type of flour (whole meal or white, plain).

<sup>f</sup>Primarily whey based.

<sup>g</sup>Less than 15 g protein per 100 g.

<sup>h</sup>More than 15 g protein per 100 g.

<sup>i</sup>Without asparagus (very low alkali excess) and spinach (very high alkali excess).

**Table 4**

Estimation of daily renal net acid excretion (NAE) for a woman<sup>a</sup> consuming a fictitious diet with a relatively high (diet A) or low (diet B) potential renal acid load (PRAL)

Food	Diet A						Food	Diet B					
	Intake (g/d)	Energy (kcal/d)	Protein (g/d)	PRAL <sup>b</sup> (mEq/100 g)	PRAL <sup>b</sup> (mEq/d)	PRAL <sup>c</sup> (mEq/d)		Intake (g/d)	Energy (kcal/d)	Protein (g/d)	PRAL <sup>b</sup> (mEq/100 g)	PRAL <sup>b</sup> (mEq/d)	PRAL <sup>c</sup> (mEq/d)
Bread, wheat flour	200	466	12.4	3.8	7.6	7.0	Bread, wheat flour	200	466	12.4	3.8	7.6	7.0
Cottage cheese	350	343	48.3	8.7	30.5	28.0	↔ Tomatoes	300	51	2.1	-3.1	-9.3	-8.4
Turkey	200	214	43.8	9.9	19.8	19.0	↔ Turkey	200	214	43.8	9.9	19.8	19.0
Cucumber	200	20	1.4	-0.8	-1.6	-5.6	↔ Carrots	300	90	2.1	-4.9	-14.7	-8.4
Spaghetti	120	410	14.4	6.5	7.8	8.0	↔ Potatoes	400	300	8.4	-4.0	-16.0	-11.2
Butter, margarine	102	753	0.4	0.0	0.0	0.0	Butter, margarine	147	1,085	0.5	0.0	0.0	0.0
		<b>2,206</b>	<b>120.7</b>		<b>64.1</b>	<b>56.4</b>			<b>2,206</b>	<b>69.3</b>		<b>-12.6</b>	<b>-2.0</b>
Daily urinary excretion of organic acids <sup>a</sup>					<b>39.8<sup>d</sup></b>	<b>41.6<sup>e</sup></b>						<b>39.8<sup>d</sup></b>	<b>41.6<sup>e</sup></b>
Daily NAE (estimated)					<b>103.9</b>	<b>98.0</b>						<b>27.2</b>	<b>39.6</b>

<sup>a</sup>An adult female 63 kg in weight and 163 cm in height.

<sup>b</sup>PRAL values taken from Table 2.

<sup>c</sup>Calculated from the average PRAL values listed in Table 3.

<sup>d</sup>Estimation of daily excretion of organic acids (OA):

OA (mEq/d) =  $\frac{\text{body surface area (m}^2\text{)} \times 41 \text{ (mEq/d/1.73 m}^2\text{)}}{1.73 \text{ (m}^2\text{)}}$

<sup>e</sup>Simplified estimation of daily excretion of OA using individual body weight (BW) (9): OA (mEq/d) = BW × 0.66.

excess of foods is reduced by our physiologically based calculation model. Differences are also discernible for foods from food groups with lower (absolute) PRAL values, for example, milk, -5.0 mEq (14), -2.7 mEq (13), -3.3 mEq (2), +0.7 mEq (actual value); peas, -1.3 mEq (14), -1.2 mEq (13), +1.2 mEq (actual value); and whole-wheat bread, +7.3 (14), +6.7 mEq (13), +1.8 mEq (actual value). These discrepancies probably occurred because none of the former evaluation models took into account the bioavailability of nutrients.

In addition, the earlier acid-ash diet calculation methodologies did not offer the possibility of predicting the probable urine pH levels produced by certain diets. As we have shown in healthy men (5), urine pH can be predicted reliably and consequently can be adjusted to a target pH when the PRAL values presented here (for the prognosis of diet-induced NAE) are used with the regression equation for renal NAE and urine pH depicted in Figure 2. Although this regression equation was obtained from measurements in men, the corresponding prediction of urine pH for women will be valid because a characteristic effect of gender on renal NAE does not exist (4,9).

**IMPLICATIONS AND APPLICATIONS**

A formula for the prediction of urine pH using nutrient intake data has been developed for cats (15), a species with a high incidence of struvite stones. To our knowledge no such prognostic method has been available hitherto for human beings consuming normal mixed diets. Based on PRAL values presented herein, which consider mean intestinal absorption rates for individual nutrients and postabsorptive metabolism of sulfur-containing amino acids, it is possible to estimate the diet-dependent component of daily renal NAE, that is, the daily PRAL. This can be achieved by simply adding all single PRAL values of foods and beverages according to their daily ingested amounts (see Table 4). Another NAE component, the excretion of organic acid which depends primarily on body weight (or body surface area) and is relatively constant for each individual, must also be considered (Table 4) to yield the total NAE that finally determines each person's urine pH level.

No optimal estimation of actual NAE will be obtained (even based on exact nutrient intake data for the day of urine collection) if dietary composition differs markedly between the day of the 24-hour urine collection and the preceding day. Our calculation model requires a certain steady state of nutrient intake, that is, a relatively constant food supply at least for 2 days. Marked deviations between predicted and measured urine pH values can also occur for certain persons, for example, as a result of an inherent reduced renal ability to produce ammonium — renal tubular acidosis type IV (16), or depending on the classification, type II (17). Such a lowered NAE capacity will result, for each acid load (ie, for each given NAE range), in a markedly lower urine pH level than that depicted in Figure 2. This phenomenon which, as a pathological finding, is rare in the normal population, may occur to a much weaker degree in healthy subjects.

In addition to the aforementioned sources of inaccuracies and variations involved in predicting urine pH, there may be differences between the tabulated nutrient data (used for PRAL calculation) and the actual values due to inherent nutrient variations of natural foods and differences in their processing and preparation (18). Furthermore, the results of predicting renal NAE (and consecutively the urine pH of subjects consuming certain diets) depend on accurate food consumption data. In regard to estimation errors related to the normal nutrient variations of foods, it seems appropriate (at least for a rough and rapid survey of the effects of diet on urine pH) to use the mean PRAL values, which have been averaged for definite foods and food groups in Table 3.

Several diseases encountered in dietetics could benefit from application of dietary means to modify urine pH in people. One of these is urolithiasis, especially in a case of confirmed diagnosis of calcium phosphate or struvite stones or a case of uric acid lithiasis or cystine stones. In these two cases, a generally accepted basic principle of therapy or recurrence prevention consists of urine acidification or alkalization, respectively.

For magnesium ammonium phosphate (struvite) or calcium phosphate stones, both of which are poorly soluble at higher

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urine pH values, solubility can be increased and precipitation inhibited by adequate decreases of urine pH. However, no dietary methods to prevent recurrence by improving the solubility of these phosphate stones (infective calculi) is possible when patients have renal tubular acidosis (19). In patients with uric acid lithiasis or cystine stones, adjusting urine pH to near 6.8 is recommended. This urine pH level is attainable in healthy subjects by purely dietetic measures (4,5).

In a case of noninfectious calcium oxalate stones, a pH increase can be of benefit (3,8). Therefore, the recommended high fluid intake (20) should be achieved by drinking alkalizing, ie, potassium/alkali-rich (low-phosphorus) beverages but not coffee, because coffee contains relatively high amounts of oxalate and excess intake seems to increase renal calcium losses (21).

About 0.5% of all boys and 3% to 5% of all girls (data from Germany) become ill from urinary tract infection before reaching puberty (22). Increasing resistance to various antibiotics makes dietary treatment of this disorder an option that should be considered. Zimmermann (23) proposed an alteration between several days consuming an acidifying diet and several days consuming an alkali-rich diet; whether this approach is particularly effective in suppressing bacterial growth, and thus in overcoming infection, deserves detailed reinvestigation.

Current PRAL data could be valuable in applying the metabolic benefits of increased alkali ingestion to athletic performance in certain sport disciplines (24) and to mineral balance and skeletal metabolism in postmenopausal women (25). ■

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## References

1. Peacock M, Robertson WG. Pathogenesis of urolithiasis. In: Schneider H-J, ed. *Urolithiasis: Etiology, Diagnosis*. Berlin, Germany: Springer; 1985:245-334.
2. Dwyer J, Foulkes E, Evans M, Ausman L. Acid/alkaline ash diets: time for assessment and change. *J Am Diet Assoc*. 1985; 85:841-845.
3. Siener R, Hesse A. Einfluß verschiedener Kostformen auf die Harnzusammensetzung und das Kalziumoxalat-Steinbildungsrisiko. *Z Ernahrungswiss*. 1993; 32:46-55.
4. Remer T, Manz F. Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. *Am J Clin Nutr*. 1994; 59:1356-1361.
5. Remer T, Manz F. Dietary protein as a modulator of the renal net acid capacity: evidence that an increased protein intake improved the capability of the kidney to excrete ammonium. *J Nutr Biochem*. In Press.
6. Manz F, Schmidt H, Schärer K, Bickel H. Acid-base status in dietary treatment of phenylketonuria. *Pediatr Res*. 1977; 11:1084-1087.
7. Kalthoff H, Manz F, Diekmann L, Stock GJ. Suboptimal mineral composition of cow's milk formulas: a risk factor for the development of late metabolic acidosis. *Acta Paediatr Scand*. 1990; 79:743-749.
8. Manz F, Schmidt H. Retrospective approach to explain growth retardation and urolithiasis in a child with long-term nutritional acid loading. *Z Ernahrungswiss*. 1992; 31:121-129.
9. Manz F, Vecsei P, Wesch H. Renale Säureausscheidung und renale Molenlast bei gesunden Kindern und Erwachsenen. *Monatsschr Kinderheilkd*. 1984; 132:163-167.
10. Holland B, Welch AA, Unwin ID, Buss DH, Paul AA, Southgate DAT. *McCance and Widdowson's the Composition of Foods*. 5th ed. Cambridge, England: The Royal Society of Chemistry; 1991.
11. Höhler M, Decher-Splithoff E, Kersting M, Ternes ML, Manz F. Funktionsbelastung des Stoffwechsels und der Niere bei Kraftsportlern mit eiweißreicher Kost. *Dtsch Z Sportmed*. 1994; 45:92-103.
12. Manz F, Remer T, Decher-Splithoff E, Höhler M, Kersting M, Kunz C, Lausen B. Effects of a high protein intake on renal acid excretion in bodybuilders. *Z Ernahrungswiss*. 1995; 34:10-15.
13. Gonick HC, Goldberg G, Mulcare D. Reexamination of the acid-ash content of several diets. *Am J Clin Nutr*. 1968; 21:898-903.
14. Langendorf H. Säure-Basen-Gleichgewicht und chronische acidogene und alkalogene Ernährung. *Z Ernahrungswiss*. 1963; 2 (suppl):1-33.
15. Kienzle E, Schuhknecht A. Untersuchungen zur Struvitsteindiätetik: 1. Einfluß verschiedener Futterrationen auf den Harn-pH-Wert der Katze. *DTW*. 1993; 100:198-203.
16. Schärer K, Manz F. Hereditäre Tubulopathien. In: Losse H, Renner E, eds. *Klinische Nephrologie*. vol 2. Stuttgart, Germany: Thieme; 1982:312-349.
17. Carlisle EJJ, Donnelly SM, Halperin ML. Renal tubular acidosis (RTA): Recognize the Ammonium defect and pHorget the urine pH. *Pediatr Nephrol*. 1991; 5:242-248.
18. Pennington JAT, Wilson DB. Daily intakes of nine nutritional elements: analyzed vs calculated values. *J Am Diet Assoc*. 1990; 90:375-381.
19. Lutzeyer W, Hering F. Drug therapy of urinary calculi and prevention of recurrence. In: Schneider H-J, ed. *Urolithiasis: Therapy, Prevention*. Berlin, Germany: Springer; 1985:1-73.
20. Lemann J. Composition of the diet and calcium kidney stones. *N Engl J Med*. 1993; 328:880-881.
21. Hasling C, Sondergaard K, Charles P, Mosekilde L. Calcium metabolism in postmenopausal women is determined by dietary calcium and coffee intake. *J Nutr*. 1992; 122:1119-1122.
22. Schofer O, Beetz R, Mannhardt W, Schulte-Wissermann H. Welche Art von Abwehrschwäche besteht bei Kindern mit rezidivierenden, nicht-obstruktiven Harnwegsinfektionen? *Kinderarzt*. 1993; 24:715-724.
23. Zimmermann W. Natürliche Urologika: Harnsteinprophylaxe, Harnwegsinfekte, Diätformen. Teil 1. *Fortschr Med*. 1988; 106:18-20.
24. Gao JP, Costill DL, Horswill CA, Park SH. Sodium bicarbonate ingestion improves performance in interval swimming. *Eur J Appl Physiol*. 1988; 58:171-174.
25. Sebastian A, Harris ST, Ottaway JH, Todd KM, Morris RC. Improved mineral balance and skeletal metabolism in postmenopausal women treated with potassium bicarbonate. *N Engl J Med*. 1994; 330:1776-1781.
26. Documenta Geigy. *Wissenschaftliche Tabellen*. 7th ed. Stuttgart, Germany: Thieme, 1975:658-661.
27. Kesteloot H, Joossens JV. The relationship between dietary intake and urinary excretion of sodium, potassium, calcium and magnesium: Belgian interuniversity research on nutrition and health. *J Hum Hypertens*. 1990; 4:527-533.
28. Stanbury SW. Intestinal absorption of calcium and phosphorus in adult man in health and disease. In: Bickel H, Stern J, eds. *Inborn Errors of Calcium and Bone Metabolism*. London, England: MTP Press; 1976:21-45.
29. Maschio G, D'Angelo A, Mioni G, Ossi E. *Il metabolismo idro-elettrolitico nelle malattie del rene*. Padova, Italy: Piccin Editore; 1972:60-68.
30. Schulman JD. Sulfur metabolism. In: Schulman JD, ed. *Cystinosis*. Washington, DC: Government Printing Office; 1973. Dept of Health, Education, and Welfare publication NIH 72-249.
31. Food and Nutrition Board. *Recommended Dietary Allowances*. 10th ed. Washington, DC: National Academy Press; 1989:32-33.
32. Souci SW, Fachmann W, Kraut H. *Food Composition and Nutrition Tables 1989/90*. 4th ed. Stuttgart, Germany: Wissenschaftliche Verlagsgesellschaft; 1989.