Dependence of Oxalate Absorption on the Daily Calcium Intake

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Abstract. Two to 20% of ingested oxalate is absorbed in the gastrointestinal tract of healthy humans with a daily 800 mg calcium intake. Calcium is the most potent modifier of the oxalate absorption. Although this has been found repeatedly, the exact correlation between calcium intake and oxalate absorption has not been assessed to date. Investigated was oxalate absorption in healthy volunteers applying 0.37 mmol of the soluble salt sodium [$^{13}C_2$]oxalate in the calcium intake range from 5 mmol (200 mg) calcium to 45 mmol (1800 mg) calcium. Within the range of 200 to 1200 mg calcium per day, oxalate absorption depended linearly on the calcium intake. With 200 mg calcium per day, the mean absorption (± SD) was 17% ± 8.3%; with 1200 mg calcium per day, the mean absorption was 2.6% ± 1.5%. Within this range, reduction of the calcium supply by 70 mg increased the oxalate absorption by 1% and *vice versa*. Calcium addition beyond 1200 mg/d reduced the oxalate absorption only one-tenth as effectively. With 1800 mg calcium per day, the mean absorption was 1.7% ± 0.9%. The findings may explain why a low-calcium diet increases the risk of calcium oxalate stone formation.

For decades, a mainstay in the treatment of patients with calcium (Ca) urinary stones has been a Ca-restricted diet (1). Reduction of the Ca content of the diet reliably reduced the amount of Ca excreted in urine. This reduction was believed—but never proven—to reduce the risk of Ca stone formation. By contrast, already in 1969, a Ca-restricted diet was shown to increase the gastrointestinal absorption of oxalate (2), leading to increased amounts of oxalate in the urine and an increased risk of the formation of Ca oxalate stones. Therefore, to reduce oxalate absorption and the resulting risk of formation of Ca oxalate stones, high-Ca supplements were routinely prescribed to obese patients after ileal bypass surgery (3). The reason why these contradictory and confusing recommendations persisted for so long is the lack of prospective studies and the fact that analysis of oxalate remained unreliable (4) until the mid-1980s. Consequently, the amount of dietary oxalate excreted in urine and its role for renal stone formation were underestimated (5). The fact that a low-Ca diet emerged as a risk factor for Ca oxalate calculi and that a high-Ca diet emerged as a protective factor in two large epidemiologic studies (6,7) is still frequently ignored. Hence, the advice to restrict Ca may still be given to patients with recurrent Ca oxalate urinary stones.

We wanted to clear up the confusion generated by the contradictory results as well as the contradictory recommendations, and to answer the following question: To what extent does Ca intake influence the gastrointestinal oxalate absorption? Therefore, we quantitatively measured the dependence of oxalate absorption on Ca intake. The physiologic daily dietary Ca intake lies between approximately 370 and 1200 mg. Intakes of less than 300 mg Ca never occur in adults except in those who reduce food intake to lose weight. Higher Ca intakes occur in people who drink milk as their main liquid or in subjects receiving Ca supplementation.

Materials and Methods

Volunteers

We tested eight healthy volunteers, three women and five men, aged between 20 and 59 yr, weight range 49 to 92 kg, body mass index range 17 to 26 kg/m^2. Individual data are given in Table 2. The volunteers had no history of gastrointestinal or renal disease, and urinalyses with dipsticks were normal before each test. The study was approved by the ethics committee of the Faculty of Medicine of the University of Bonn. Informed consent was obtained from all volunteers.

Standardized [$^{13}C_2$]Oxalate Absorption Test

The standardized [$^{13}C_2$]oxalate absorption test was developed (8) by using the original [^{14}C]oxalate absorption test (9) as a model. The reference range for oxalate absorption was determined for 120 volunteers (10). There was no gender dependence of oxalate absorption. In brief, an identical standard diet was provided on subjects on two consecutive days. At 08:00 on day 2, a capsule soluble in gastric juice containing 50 mg (0.37 mmol) sodium [$^{13}C_2$]oxalate corresponding to 33.8 mg [$^{13}C_2$]oxalic acid was ingested with water. Urine was completely collected in intervals of 12 h on day 1 and in intervals of 6, 6, and 12 h on day 2. Absorbed oxalate (labeled as well as unlabeled) was excreted rapidly and completely via the kidneys (biologic half-

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life 1.5 h). Its absorption was expressed as the percentage of the labeled dose recovered in the 24-h urine after dosing.

Intraindividual variability (10) of oxalate absorption is noteworthy even under the standardized conditions of our test procedure. Thus, each volunteer was tested three times. The analytical procedure for the [$^{13}C_2$]oxalate measurement in urine has been previously published (8). Organic acids were extracted from a 0.1-ml aliquot of acidified urine and derivatized with N-methyl-*tert*-butyldimethylsilyltrifluoroacetamide. Samples were measured by gas chromatography–mass spectrometry. The ions *m/z* 261.3, 263.3, and 276.3 were used to quantify the unlabeled oxalic acid, the labeled oxalic acid, and the internal standard, [2-^{13}C]malonic acid, respectively.

Further Analytes Measured in Urine

Calcium and magnesium were measured by standard clinical chemistry procedures. Citrate was measured with the citrate lyase assay (Roche Diagnostics, Mannheim, Germany).

Diet

The standard diet was composed of regular foodstuffs according to the 1995 recommendations of the German Society of Nutrition. Information regarding compositions of the diet was partially obtained from the suppliers; the rest was calculated with a computer program for dietary counseling, PRODI 4.4 (11). The standard diet contained 2500 kcal, 83 g protein, 350 g carbohydrates, 96 g fat, 800 mg (20 mmol) Ca, 750 mg (31 mmol) Mg (data calculated), and 63 mg (0.7 mmol) oxalic acid per day (measured by an HPLC enzyme reactor method (12)). Because tabulated Ca content data were used, the actual Ca contents may have deviated by ±25 mg/d from the quoted ones. Composition of the standard diet and time schedule are given in Table 1.

Different Calcium Contents and the Required Deviations from the Standard Diet

1800 mg Ca. 1000 mg (25 mmol) Ca effervescent tablet (2500 mg $CaCO_3$ + 4374 mg citric acid) was provided as a supplement, with the capsule containing labeled oxalate. Meals and liquid intake were unchanged.

1200 mg Ca. 400 mg (10 mmol) Ca was provided as two film tablets (2 × 950 mg Ca citrate tetrahydrate) as supplement with the labeled oxalate. Meals and liquid intake were unchanged.

600 mg Ca. 200 mg (5 mmol) Ca less was provided. Meals were unchanged, and mineral water (Ca content 166 mg/L) was substituted for local tap water (Ca content 39 mg/L).

370 mg Ca. 430 mg (10.75 mmol) less Ca was provided. In meals, yogurt was replaced by half a slice of bread with 5 g margarine and 25 g strawberry preserves. As a substitute for cream cheese, 30 g salami was added. The mineral water was substituted by a brand very low in minerals (Ca content 1.7 mg/L, Mg content 0.67 mg/L; 304 mg Mg was added to the 1.4 L). This diet contained 2400 kcal.

Table 1. Composition and the time schedule of the standard meals, taken on the day prior to and on the test day

Solids		
Breakfast	9:00	2 bread rolls (100 g)
		10 g margarine
		25 g strawberry preserves
		30 g cold sliced meat
Snack	11:00	1 muesli bar (25 g)
		1 banana (150 g)
Lunch	14:00	60 g turkey ragout
		140 g creamy sauce
		120 g mixed vegetables
		120 g pasta
Snack	17:00	1 apple (150 g)
		150 g yogurt with fruit (3.5% fat)
Supper	20:00	2 slices of brown (rye-wheat) bread
		10 g margarine
		30 g salami
		17 g cream cheese
		1 tomato (50 g)
		1 apple (150 g)
Late meal	22:00	1 slice of brown bread
		5 g margarine
		17 g cream cheese
		1 tomato (50 g)
Liquids (2.2 L/day)		
300 ml coffee	9:00	
700 ml mineral water[a] until	14:00	
700 ml mineral water and 500 ml apple juice until	22:00	

[a] The mineral water used contained the following ions per litre: 268 mg Na^+, 35 mg K^+, 231 mg Mg^{2+}, and 166 mg Ca^{2+} and chloride 19.6 mg, sulphate 18 mg, fluoride 0.3 mg, and hydrogen carbonate 2350 mg.

200 mg Ca. From the 370-mg Ca diet, the following items were omitted without substitution: muesli bar, mixed vegetables and creamy sauce, tomatoes, apple juice, and one apple. This diet contained only 1950 kcal.

Statistical Analyses

SPSS (SPSS, Chicago, IL) for Windows, release 8.0.0, was used for statistical analyses. Results of descriptive statistics are given as mean ± SD. The significance of differences was calculated by the nonparametric Wilcoxon test for paired samples; $P \leq 0.05$ was considered significant.

Results

All reported values refer to urine collected in the 24 h after intake of the labeled sodium oxalate. Individual mean oxalate absorption data (± SD) for all volunteers at their different Ca levels are listed in Table 2. Figure 1 indicates the dependence of the oxalate absorption on the Ca intake. In the range of 370 to 1800 mg Ca/d, mean values for the eight volunteers, three tests each at each Ca content, were plotted. The absorption value of 16.92% at the unphysiological low Ca intake of 200 mg is the mean of only 18 tests from six of the eight individuals who volunteered for this additional test. The error bars are the SD for all individual tests at each Ca concentration (*i.e.*, intra- and inter-individual variability). Mean oxalate absorption values were significantly different from the mean absorption with 800 mg Ca/d, with $P < 0.01$ for the absorption with 600 mg Ca/d. Absorption values with 200, 370, 1200, and 1800 mg Ca/d differed, with $P < 0.001$. In the range of physiologic Ca intakes from 370 to 1200 mg/d, the mean oxalate absorption can be described by a straight line, $y = -0.0143x + 19.761$; coefficient of correlation, -0.9997. This linear function may be expressed as the following rule of thumb: In the range of normal dietary Ca contents, an increase of the Ca intake by 70 mg reduced the oxalate absorption by 1% and *vice versa*. This linear relation is only applicable to Ca intakes of up to 1200 mg/d. Additional Ca beyond 1200 mg/d reduced oxalate absorption only slightly and was only one-tenth as effective as in the range up to 1200 mg Ca/d.

When Figure 1 is examined, four results are striking: (*1*) The oxalate absorption decreased over the whole range with increasing intake of Ca. (*2*) The interindividual spread was minimal with the 1000 mg Ca supplement (1800 mg/d Ca intake; Table 2). Below 1200 mg Ca/d, the interindividual spread and the intraindividual variability were large. (*3*) Over the range of calcium intakes from 200 to 1200 mg Ca/d, mean oxalate absorption was a linear function. (*4*) However, over the whole range tested (*i.e.*, 200 to 1800 mg Ca/d), the oxalate absorption was clearly not a linear function of the Ca intake.

Table 3 lists total urinary oxalate excretion (endogenous + absorbed dietary oxalate + absorbed labeled oxalate) and urinary calcium excretion. The urinary oxalate excretion decreased only slightly with decreasing oxalate absorption in our tests because a low-oxalate diet was applied for the test. Nevertheless, the difference of the mean oxalate excretion between the 24 tests with 370 mg and the 24 tests with 800 mg Ca/d (0.439 and 0.348 mmol oxalate/d, respectively) was significant ($P < 0.001$). The urinary Ca excretion rose from 2.99 mmol/d with a dietary Ca of 370 mg/d to 3.97 mmol/d with 800 mg Ca/d ($P < 0.001$). Urinary citrate increased marginally with increasing Ca intake. The magnesium excretion (range of means 6.22 ± 2.08 to 7.44 ± 2.09 mmol/24 h) and the urine volume (range of means 2.25 ± 0.41 to 2.74 ± 0.43 L) were not significantly different.

Table 4 shows the ion activity product index AP_{CaOx} (23) of the 24-h urine after ingestion of the labeled oxalate. The AP_{CaOx} index for a 24-h urine sample is calculated as follows:

$$AP_{CaOx} = \frac{1.9 \cdot Calcium^{0.84} \cdot Oxalate}{Citrate^{0.22} \cdot Magnesium^{0.12} \cdot Volume^{1.03}}$$

The AP_{CaOx} index did not increase despite increasing urinary excretion of Ca with increased Ca intake. With a mean value of approximately 0.5 and the maximal value of 1.205, the AP_{CaOx} was far below the critical value of two.

Table 2. Mean oxalate absorption values [%] ± intra-individual (SD) from the three [$^{13}C_2$]oxalate absorption tests for each volunteer at her/his different Ca levels and means [%] of all volunteers ± inter-individual SD

Volunteer initials	Gender	Age (yr)	Weight (kg)	BMI (kg/m^2)	Ca Intake (mg/d Ca)					
					Depleted			Standard	Supplemented	
					200	370	600	800	1200	1800
VS	F	27	63	21	19.1 (5.4)	13.8 (0.6)	12.4 (2.3)	6.9 (2.1)	1.2 (0.3)	1.4 (0.1)
KS	F	27	49	17	ND[a]	16.0 (1.4)	12.1 (1.4)	8.3 (2.7)	1.4 (0.8)	1.7 (0.8)
GM	F	26	54	20	16.9 (1.6)	13.5 (1.4)	11.2 (1.2)	12.3 (4.1)	2.9 (1.1)	2.1 (2.4)
UG	M	59	84	24	13.7 (3.8)	11.6 (1.1)	7.2 (1.3)	3.2 (0.4)	2.6 (1.3)	1.4 (0.4)
SM	M	39	79	26	ND	11.6 (2.8)	10.6 (2.9)	7.6 (2.2)	4.0 (2.5)	2.0 (0.4)
HP	M	26	92	26	9.7 (1.2)	16.8 (2.3)	14.6 (4.3)	12.2 (4.5)	3.3 (1.2)	1.4 (0.2)
GC	M	24	67	21	24.4 (1.9)	19.9 (1.7)	14.7 (1.2)	9.6 (3.9)	3.1 (1.9)	1.5 (0.2)
JN	M	20	60	19	17.7 (11.6)	11.8 (2.2)	8.3 (3.3)	6.4 (3.2)	2.4 (0.6)	2.0 (1.2)
Mean ± (SD)					16.92 (4.98)	14.37 (2.98)	11.39 (2.69)	8.32 (3.05)	2.61 (0.94)	1.69 (0.304)

[a] ND, not determined.

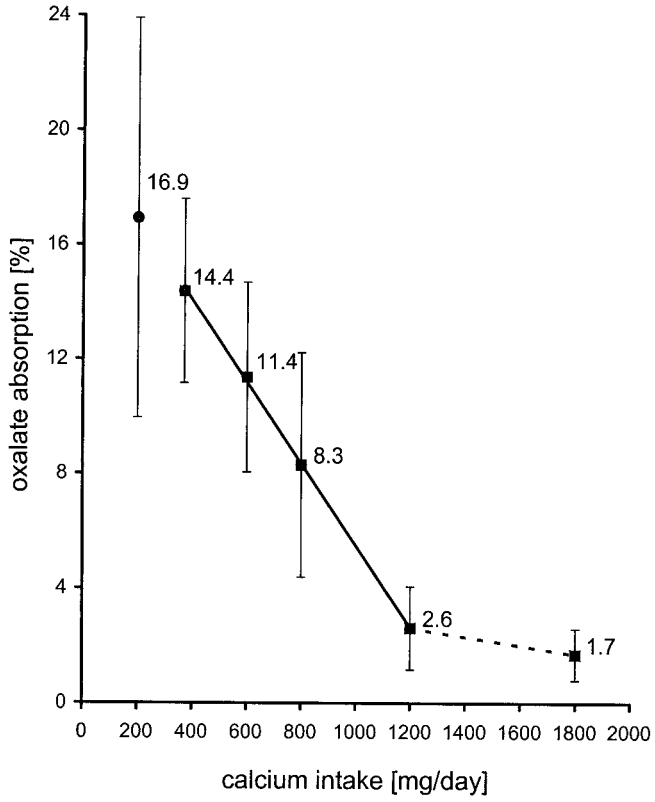

Figure 1. Mean oxalate absorption values from six volunteers at 200 mg Ca/d (circle) and from all eight volunteers (squares) for the other Ca intakes. Error bars are SD for all individual tests at each Ca intake. For the range of physiologic Ca intakes, 370 to 1200 mg Ca/d, the graph was a straight line with an r of -0.9997. The dotted connecting line from Ca intakes of 1200 to 1800 mg/d indicates the reduced slope—*i.e.*, the reduced efficacy of additional Ca. The end points of the dotted line were confirmed by 8 and 18 additional tests, respectively (data not shown).

Discussion

In the study presented here, we showed that exogenous Ca leads to a linear reduction of oxalate absorption within the range of a daily intake of 200 to 1200 mg Ca. Strong interactions between dietary Ca and dietary oxalate affecting the oxalate absorption have been described (2, 13–19) as well as denied (20). Ca supplements have been shown to lower the oxalate absorption in several reliable studies (for a review, see (21)). On the other hand, Ca supplements emerged as a potential risk factor for urolithiasis in a large epidemiologic study (7). However, there are no systematic studies on the magnitude of the influence of the Ca intake on the gastrointestinal oxalate absorption. Such studies could not be performed because the necessary number of [^{14}C]oxalate applications exceeded the permitted dose of radioactivity. For the same reason, intraindividual variability of oxalate absorption was not studied at all with [^{14}C]oxalate. Oxalate absorption varies considerably in the same person. Intraindividual variability depends on such known highly variable physiologic parameters as gastric emptying, intestinal transit time, amount of chyme, and amount and composition of the intestinal flora in the gastrointestinal tract. The effect of these variable physiologic parameters is wiped out only by a 1000-mg Ca supplement given simultaneously with the oxalate test dose. Under the standardized conditions of our oxalate absorption test (10), the mean intraindividual coefficient of variation was 38% (*i.e.*, the mean intraindividual SD was 3.39% $\pm$ 1.68%). This range of SD (1.7% to 5.1%) corresponds to the effect of a difference of about 100 to 360 mg Ca in the diet within the range of 400 to 1200 mg Ca/d (Figure 1). Beyond 1200 mg Ca/d, only very small differences of oxalate absorption occur at all. These changes are identical to or smaller than the SD of 1% found for 1800 mg Ca. Such small differences make analysis of the relation between Ca intake and oxalate absorption extremely difficult. Erroneous results and contradictory statements about the effect of Ca supplements can thus be explained.

From normal to unphysiologically low Ca intakes, the linear extrapolation seems justified. The mean oxalate absorption from the six volunteers with 200 mg Ca/d neatly fits the straight line despite the reduced caloric intake (1950 kcal instead of 2500 kcal). However, linear extrapolation of the absorption line to the right, from low to high Ca intakes, would suggest that a dose of about 1400 mg Ca/d completely suppresses the oxalate absorption. Such a suppression does not happen. Even with 1800 mg Ca/d, there was always a small oxalate absorption just as after a dose of calcium oxalate (22).

Our study assessed the absorption of a soluble oxalate salt in the gastrointestinal tract or—in terms of pharmacokinetics—the absolute bioavailability of oxalate. The majority of oxalate-containing foodstuffs also contain oxalate or contain it preferentially as calcium salt. Fortunately, the relative bioavailability of this oxalate will be somewhat lower. How much lower depends on the physiologic factors of the person (*e.g.*, extent of chewing, pH of the gastric juice) and also on the properties and processing of the plant material. However, relative bioavailability of oxalate in different foodstuffs was not the topic of our investigation.

High Ca in the diet as well as Ca supplements were both described as means to reduce oxalate absorption in patients (2,13–18,21). Interpretation of the published data as well as of our first results was hampered by the lack of information on the variation of the oxalate absorption, as well on interindividual variation as on the physiologic, intraindividual variation. These variations are extensive. Under our standard conditions with 800 mg Ca/d, we found a range of oxalate absorptions from 2% to 20% for 120 healthy volunteers (10). The range of intraindividual coefficients of variation from 26 volunteers (including the eight described here) tested three times was 10.5% to 80.8% under these standardized conditions (10). Therefore, by using a small number of volunteers tested only once, a correlation in the range of 800 to 1800 mg Ca intake could be incorrectly described by one straight line, indicating a maximum oxalate absorption for a zero Ca intake of about 10%, the value frequently cited. Such an incorrect extrapolation is the explanation of the widespread belief that gastrointestinal oxalate absorption is only marginally affected by Ca intake.

However, we showed a distinct and drastic change of the slope of the oxalate absorption curve. As can be derived from the curve in Figure 1, an additional 700 mg Ca added to a

Table 3. Effects of dietary calcium on urinary oxalate/calcium excretion values (mmol/24 h) (means of three tests each)

Volunteer	Ca intake (mg/d Ca)					
	Depleted			Standard	Supplemented	
	200	370	600	800	1200	1800
VS	0.404/3.81	0.402/3.63	0.424/6.10	0.404/3.98	0.300/5.17	0.347/5.88
KS	ND	0.291/2.38	0.238/3.61	0.209/4.62	0.155/5.88	0.203/6.39
GM	0.368/2.46	0.304/3.20	0.266/3.98	0.267/4.62	0.267/5.35	0.196/6.01
UG	0.310/2.55	0.405/2.80	0.347/2.83	0.354/3.61	0.354/3.92	0.259/5.00
SM	ND	0.539/3.38	0.455/3.60	0.371/3.15	0.371/4.74	0.308/4.99
HP	0.468/4.08	0.541/4.07	0.507/4.62	0.460/6.54	0.460/6.11	0.481/7.26
GC	0.468/2.83	0.502/3.26	0.332/3.87	0.430/4.16	0.423/5.18	0.342/4.66
JN	0.432/1.85	0.528/1.16	0.338/1.31	0.286/1.07	0.323/1.82	0.313/1.92
Mean	0.413/2.87	0.439/2.99	0.363/3.74	0.348/3.97	0.338/4.77	0.306/5.26
± SD	0.096/0.89	0.106/0.99	0.099/1.42	0.097/1.59	0.112/1.42	0.099/1.60

Table 4. Mean AP_{CaOx} index [L^{-1}] from the three [$^{13}C_2$]oxalate absorption tests for each volunteer at her/his different Ca levels

Volunteer	Ca intake (mg/d Ca)					
	Depleted			Standard	Supplemented	
	200	370	600	800	1200	1800
VS	0.584	0.611	0.600	0.418	0.584	0.596
KS	ND	0.298	0.383	0.351	0.284	0.346
GM	0.365	0.343	0.351	0.416	0.474	0.329
UG	0.282	0.506	0.292	0.466	0.382	0.359
SM	ND	0.640	0.534	0.377	0.569	0.489
HP	0.763	1.099	0.929	0.973	1.015	1.206
GC	0.589	0.532	0.357	0.553	0.605	0.496
JN	0.417	0.323	0.198	0.177	0.224	0.254
Mean	0.495	0.544	0.455	0.466	0.517	0.509
± SD	0.197	0.263	0.243	0.250	0.263	0.317

low-Ca diet with 370 mg Ca/d will reduce oxalate absorption by 10%. However, an additional 700 mg Ca added to a 1200 mg/d Ca diet will reduce oxalate absorption by only 1%. This fact explains why Ca supplements added to a low-Ca diet lower the risk of Ca oxalate crystal formation. The decrease of oxalate absorption and the resulting decrease in oxalate excretion overcompensates the increase in Ca excretion. Ca supplements given in addition to a high-Ca diet can decrease oxalate absorption only marginal without overcompensating the increased Ca excretion. Therefore, information about the daily Ca intake of a patient is required before advising the patient on an increase of the consumption of Ca-rich foodstuffs or prescription of Ca supplements.

Under a low-oxalate diet, even in cases of oxalate hyperabsorption, dietary oxalate contributed only a small amount to the oxalate excreted. The majority of urinary oxalate is then endogenously derived. Even in the case of drastic absorption changes, only small changes in oxalate excretion could thus be expected. The situation is of course completely different under a high-oxalate diet. With supplemented 500 mg of oxalate from spinach, more than half of the excreted oxalate was of dietary origin (D. Zimmermann *et al.*, unpublished data). Under such conditions, the extent of absorption is highly critical for the oxalate excretion.

There has always been concern about the increased urinary excretion of Ca, a potential risk factor with Ca-rich diets. This concern was not supported by our data. The AP_{CaOx} index, a formula for the risk of Ca oxalate stone formation (23), did not increase with increasing Ca intakes (Table 4). The AP_{CaOx} index remained always well below the critical value of two. These increases of urinary Ca in volunteers were by no means critical with respect to Ca oxalate supersaturation. In patients who form Ca stones, however, Ca excretion should be monitored (1,24).

The dependence of the oxalate absorption from Ca intake explains the epidemiologic results obtained from large populations: a low-Ca diet increased the risk of urinary stone occurrence (6,7). Our data fit very well into the published

epidemiologic studies because we measured oxalate absorption in the same range of Ca intake as described for the cohorts in these studies. The quintile limits were within the steep part of our oxalate absorption line: *i.e.*, if all men and women questioned would eat the same oxalate-containing meal, the persons in the lowest Ca quintiles would absorb approximately three times the amount of oxalate than the ones in their highest quintiles.

By use of the same method, the [$^{13}C_2$]oxalate absorption test, we demonstrated that patients with idiopathic recurrent Ca oxalate stones have a significantly higher mean oxalate absorption than healthy volunteers (25). Patients with enteric hyperoxaluria (26) or with ileal resections >30 cm or ileal bypass surgery performed to treat severe obesity (3) have absorptions considerably more than 20%; absorptions of up to 50% have been described (3,9,14,26). Presently, the highest absorption we measured was 73%. The patient in question had a functioning colon but only 30 cm of small intestine left after surgery as a result of mesenterial artery infarction. This patient forms several stones per week (B. Hoppe, unpublished data). Further studies are required to study this matter. Some of these patients may still benefit from a Ca intake greater than 1200 mg/d. The tolerable upper intake level of 2.5 g Ca/d for adults represents a reasonable upper limit (27). Supplements greater than 3.6 g Ca/d supported stone formation even in patients who had undergone ileal bypass (3) and should never be prescribed.

In summary, we showed that reducing intake from 1200 to 400 mg Ca/d increases the absorption of dietary oxalate by fivefold. Gastrointestinal oxalate absorption depends linearly and strongly on the Ca intake up to 1200 mg Ca/d. The resulting increased urinary Ca excretion did not heighten the risk of crystal formation. Additional Ca beyond 1200 mg Ca/d reduced oxalate absorption further, but only marginally. For patients with Ca oxalate urinary stones who are high oxalate absorbers, the advice to avoid dairy products and to follow a Ca-restricted diet is a recipe for the generation of recurrent Ca oxalate stones. This conclusion, derived from the dependence of the gastrointestinal oxalate absorption from the Ca content of the diet, was recently independently confirmed by a prospective study of 120 patients who formed idiopathic Ca oxalate stones (24).

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