

# The Role of Calcium in the Prevention of Kidney Stones

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Nephrolithiasis is a common and important condition. Several lines of evidence suggest that increased urinary calcium increases the risk of kidney stones. Since dietary calcium raises urinary calcium, it has been common practice to reduce calcium intake in stone-formers who hyperabsorb calcium from the intestine, although no trial has yet been designed to directly demonstrate the effectiveness of calcium restriction.

In contrast, some have suggested that calcium restriction may be harmful due to resultant hyperoxaluria and risk of bone loss. In fact, two powerful prospective observational studies have suggested that increased dietary calcium reduces the risk of the first kidney stone. However, calcium was not the only variable, since those with the highest quintile of calcium intake also ingested more fluid, potassium, magnesium and phosphate. Moreover, the otherwise thorough analysis was not adjusted for alkali intake, which may prevent stones, or oxalate intake, which may increase stone risk.

Due to limitations in available data, future prospective studies should be designed to probe the effect of specific interventions with calcium, both dietary and supplemental, on urinary parameters and stone formation, particularly in hypercalciuric stone-formers, who may respond conversely. For now, dietary calcium should be gradually increased in stone-formers as guided by the urinary calcium, and hypocalciuric agents should be added as necessary.

## Key teaching points:

- Nephrolithiasis is a common and important condition.
- High urinary calcium is a risk factor for stone disease and low bone density.
- Urinary calcium is increased by sodium, calcium and acid load and decreased by phosphate and alkali load.
- Observational studies suggest that high calcium intake prevents an initial stone event, but these patients also ingested more fluid, potassium, magnesium and phosphate.
- Current recommended calcium intake for the general population is three to four servings of dairy daily.
- Calcium intake in hypercalciuric stone-formers should be one serving of dairy with gradual increments to three servings a day as guided by the urinary calcium. Thiazides are often additionally necessary.

## INTRODUCTION

Nephrolithiasis is a common, morbid and expensive disease. Prevalence and incidence are estimated at 5% to 10% and 100 to 300/100,000/year respectively [1,2]. Moreover, 50% will have another stone over the subsequent six years [3]. By a 2 or 3:1 ratio, nephrolithiasis is more common in men and Caucasians than in women or African-Americans respectively [4]. One of the most painful conditions known, kidney stones may require surgical removal. Less commonly, severe infection or even loss of the kidney occurs. The yearly cost of urolithiasis in the U.S. was estimated at \$1.83 billion in 1993 [5]. For these reasons, prevention of stone formation is of great importance.

Many dietary measures have been routinely prescribed to achieve this goal, but few studies have directly assessed the protection against stone recurrence. The purpose of this paper is to review the available data regarding the effect of dietary calcium in the prevention of kidney stones.

## THE ROLE OF URINE CALCIUM IN STONE DISEASE

Most kidney stones, generally composed primarily of a poorly soluble salt with a small amount of protein, contain calcium as a main constituent [6]. The direct cause of stones is

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unknown and likely multifactorial, but a urinary physiologic abnormality can be identified in >95% of patients [7]. Hypercalciuria, the most common of these, increases the risk of stones by raising the saturation of stone-forming salts and reducing the endogenous stone-inhibitors [8,9].

Hypercalciuria has been variously defined in subjects on random or restricted diet. On random diet, urine calcium exceeds 300 mg/day in men, 250 mg/day in women or 4 mg/kg/day in either gender in hypercalciurics [10]. On a diet restricted in salt and calcium (100 mmol and 10 mmol respectively), urine calcium >200 mg/day is considered hypercalciuria [7]. Clinical studies have added further supportive evidence to the pathogenic role of hypercalciuria. Placebo-controlled studies have examined the effect of thiazides, which reduce urinary calcium, on stone recurrence. Clearcut efficacy was demonstrated even with patients who had normal baseline urinary calcium [11,12]. Also, high urinary calcium is a risk factor for recurrent stone disease [3,13]. Thus, urinary calcium appears to play an important pathogenetic role in the formation of kidney stones.

**EFFECT OF DIET ON URINARY CALCIUM (Table 1)**

It is well known that dietary or supplemental calcium intake increases urinary calcium. Four variables are important to consider when evaluating the effect of dietary calcium on urinary calcium: calcium bioavailability and dose and intestinal adaptation and function. Foods have different calcium bioavailability. Spinach, for example, contains much more calcium than kale, but due to its poor absorbability (spinach 5.1%, kale 40.9%), a serving of spinach delivers less calcium than kale [14,15]. Also, the fractional calcium absorption of a given dose of calcium decreases with increasing doses, probably due to the saturation of active absorption processes, and there may be a plateau around 500 mg of calcium [16]. Therefore, a given calcium dose will be better absorbed in small divided doses than in one large dose [17].

With long-term consistent calcium intake, intestinal adaptation occurs. So, those with high calcium intake will decrease intestinal calcium absorption, and those on low intake will upregulate absorption [18]. Finally, there is a wide range of individual intestinal absorption depending on the subject's clinical status (vitamin D deficiency, diarrhea, absorptive hypercalciuria, etc.). For these reasons, it is difficult to estimate an individual's increment in urinary calcium to a given quantity

**Table 1.** Effect of the Diet on Urinary Calcium

Increases Urine Ca	Decreases Urine Ca
Minerals: Ca, Na, Mg, Al	Minerals: K, PO <sub>4</sub>
Carbohydrate	Fiber
Acid Load (animal flesh)	Alkali Load (fruits and vegetables)

dietary calcium. In general, however, each 100 mg of dietary calcium increases urinary calcium by 8 mg/day in normal volunteers and 20 mg/day in hypercalciuric subjects [19,20].

Several other dietary factors are known to influence urinary calcium in normal volunteers and usually to a greater extent in hypercalciuric patients. Sodium is believed to increase urinary calcium by decreasing renal calcium reabsorption. Continued renal calcium loss is compensated by activation of the vitamin D axis and, in turn, increased intestinal absorption of calcium [21]. On average, each 100 mmol of sodium is believed to increase urinary calcium by 32 mg/day [19]. Magnesium increases urinary calcium excretion by inhibiting renal calcium reabsorption [22,23]. Potassium or phosphate deficiency increases urinary calcium whereas potassium or phosphate treatment reduces urinary calcium [24,25]. In fact, treatment with aluminum-containing phosphate binders is known to exacerbate hypercalciuria [26]. The key dietary sources of potassium are bananas, citrus fruits and many vegetables. Phosphorus, though present in many foods, is mainly provided by dairy and animal flesh. Phosphate may be present as a monobasic salt (H<sub>2</sub>PO<sub>4</sub><sup>-</sup>), which confers an acid load, or as a dibasic salt (HPO<sub>4</sub><sup>-2</sup>), which provides an alkali load. The monobasic salt is a less effective hypocalciuric agent due to the provision of acid load [27].

Acid load, primarily induced by animal flesh ingestion, increases urinary calcium, presumably by decreased renal calcium reabsorption and increased bone resorption [28,29]. The opposite occurs with alkali load [30], which is usually provided in the diet by fruits and vegetables. The measured acidity of food does not predict its metabolic effect on the body because some acid salts such as citrate are metabolized to bicarbonate and because sulfur-containing amino acids may be metabolized to sulfuric acid. Carbohydrate loads have been reported to increase urinary calcium to a greater degree in hypercalciurics and their family members than normal volunteers [31]. Finally, dietary fiber is believed to bind calcium in the intestine and, in turn, lower urinary calcium [32].

**RISK OF DIETARY CALCIUM RESTRICTION**

Given the important effect of diet on urinary calcium, hypercalciuric stone-formers traditionally have been counseled to increase fluid intake and to follow a diet restricted in calcium, sodium and animal flesh. However, the only dietary manipulation that has been prospectively proven to be effective in reducing stone recurrence is increasing fluid intake (55% decrease) [33]. In fact, calcium restriction has been questioned due to the potential risk of hyperoxaluria, a risk factor for stone-formation, and bone loss.

**Oxalate**

Several studies have demonstrated that severely restricted calcium intake may result in hyperoxaluria [34–37]. Unfortunately, most studies compared free diet to an instructed restricted calcium diet with or without specific oxalate recommendations. Therefore, actual dietary intake and response are difficult to interpret. In general, though, increased calcium intake reduces urinary oxalate by 0.5 to 1.1 mg/100 mg calcium [38,39].

Two studies utilized constant metabolic diet. Marshall *et al.* examined the importance of calcium and oxalate interaction in a 4-phase study [34]. Eight stone-formers and eight normal volunteers collected daily 24-hour urine while consuming each of four different constant metabolic diets for four days: low calcium (250 mg)/oxalate (50 mg), low calcium/normal oxalate, high calcium/low oxalate and high calcium (1000 mg)/normal oxalate (150 mg). Interestingly, on restricted oxalate diet, urine oxalate did not increase with calcium restriction (Table 2). When oxalate was increased by 100 mg, urine oxalate increased by 10 to 12 mg on the low calcium diet, but only five mg on high calcium (Table 2). Findings described by Breslau *et al.* further emphasize the importance of dietary oxalate [40]. They studied 15 normal volunteers randomized to three different constant metabolic diets. While keeping calcium constant at 440 mg/day, dietary oxalate content was increased from 188 mg/d to 392 mg/d. Urine oxalate increased from 26 to 39 mg/day. In combination, this data suggests that urine oxalate will not increase with calcium restriction when oxalate is simultaneously restricted; however, as oxalate intake increases, urine oxalate will rise more in those with calcium restriction. Studies in stone-formers with hyperoxaluria further suggest that for a given oxalate load, the hyperoxaluric response will be exaggerated [38].

Two powerful observational studies by Curhan *et al.* have suggested that high calcium intake prevents stone formation in men and women [41,42]. The study design was similar for both trials. Intake of various dietary constituents was measured at baseline by a single validated semi-quantitative food frequency questionnaire [43] (and every four years thereafter for the later study). The subjects, health care workers with no previous history of kidney stones, were divided by quintile of dietary calcium intake (adjusted for energy intake) and followed prospectively for symptomatic kidney stone formation. So, if a

subject consumed a diet high in calcium and total calories, his energy-adjusted calcium intake would be lower than his actual calcium intake.

As seen in Table 3, those with the highest quintiles of calcium intake also enjoyed higher intakes of fluid, potassium, magnesium, phosphate and animal protein-constituents of dairy. After controlling for confounding variables, the relative risk of kidney stones for the subjects in the highest quintile of calcium intake relative to the lowest quintile was 0.66 in men and 0.65 in women (Table 4). The authors hypothesized that this protection was likely due to intestinal binding of oxalate by calcium.

A study by Lemann *et al.* has provided data to substantiate this possibility [39]. Using the same semiquantitative food frequency questionnaire, the authors measured three consecutive 24-hour urine samples on 94 normal men and women. They found that dietary calcium did seem to reduce urinary oxalate, but this effect already reached a plateau at 664 mg of calcium intake, the mean intake of the 2<sup>nd</sup> lowest quintile in Curhan’s study in men. Of note, the key protection in both men and women occurred between the 1<sup>st</sup> and 2<sup>nd</sup> quintiles (Table 4).

This combination of three studies is very compelling. Despite the important strengths of these studies, unavoidable weaknesses inherent to epidemiological studies limit them (Table 5). A potential strength is that, even after adjusting for total energy intake, calcium intake was still a significant predictor of an initial stone. On the other hand, it has been noted that larger individuals, who likely have higher general intakes to maintain their larger mass, excrete more calcium and oxalate, key risk factors for stone formation [39]. Therefore, the underrepresentation of their true calcium intake by energy-adjustment may potentially involve bias toward increased stone risk. Likewise, the overrepresentation of calcium intake in those with a small dietary intake may involve bias toward stone prevention. Also, the authors could not directly account for alkali or oxalate intake with their questionnaire. Since alkali intake may prevent stones [44], and oxalate intake may increase stone risk [45], these factors have conceivable impact.

Curhan *et al.* also examined the effect of calcium supplementation on the risk of first kidney stone [41,42]. In men, the risk was not significant, but in women, they found a 20% increased risk (confidence interval 1.02–1.41) of any dose of calcium supplement compared to no supplement use. They

**Table 2.** Mean 24-hour Urine Calcium and Oxalate on Four Different Constant Metabolic Diets

Dietary		Urinary			
Ca, mg/d	Oxalate, mg/d	Stone-formers		Normals	
		Calcium, mg/d	Oxalate, mg/d	Calcium, mg/d	Oxalate, mg/d
1000	150	353	34	188	35
1000	50	365	29	198	30
250	150	200	40	126	45
250	50	190	30	124	33

Adapted from reference 34.

**Table 3.** Diet Characteristics by Energy-Adjusted Dietary Calcium Intake in Men and Women

Dietary Intake	Quintile of Calcium Intake									
	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Calcium, mg/d	516	664	783	937	1326	430	564	672	811	1119
Potassium, mg/d	3165	3308	3425	3582	3964	2703	2917	3050	3191	3458
Magnesium, mg/d	350	362	372	385	418	265	286	301	314	334
Phosphorus, mg/d	1159	1258	1337	1441	1701	921	1015	1097	1193	1404
Fluid, mL/d	1789	1859	1915	1985	2167	1802	1903	1974	2074	2321
Animal Protein, g/d	59	61	63	65	71	50	52	53	56	62

Adapted from references 41 and 42.

**Table 4.** Risk of Symptomatic Stones by Quintile of Energy-Adjusted Dietary Calcium Intake in Men and Women

Variable	Quintile of Calcium Intake					
	Women			Men		
	1	2	5	1	2	5
Mean dietary calcium intake, mg/d	430	564	1119	516	664	1326
Multivariate RR of stones	1.0	0.83	0.65	1.0	0.74	0.66
95% CI	0.57–0.97	0.57–0.97	0.57–0.97	0.57–0.97	0.57–0.97	0.49–0.90
n for entire trial	45,619			91,731		
Follow-up interval, years	4			12		
Age at baseline, years	40 to 75			34 to 59		

Adapted from references 41 and 42.

**Table 5.** Strengths and Weakness of Available Epidemiologic Studies

Strengths	Weaknesses
Huge sample size	Based on rare food frequency questionnaires
Excellent follow-up	Calcium was not the only difference in diets
Careful regression analysis	Did not account for alkali or oxalate intake
Evaluated women and men	Adjustment for energy intake could be a confounding variable

explained the contradicting findings by noting that most of the queried subjects tended to take calcium apart from meals; this would raise urine calcium without the benefit of intestinal oxalate binding. The data seems physiologically untenable because the full effect was present even at intakes <100 mg/d (Table 6). Even if the full observed increased risk is corroborated in future studies,

**Table 6.** Risk of Adjusted Relative Risk for Kidney Stone with Calcium Supplementation

Variable	Intake of Supplemental Calcium, mg/d			
	None	1–100	101 to 500	>500
Multivariate RR	1.0	1.26	1.18	1.21
95% CI	—	0.79–2.00	0.98–1.43	0.96–1.52
Person-years	305,164	14,228	151,202	96,731

Adapted from reference 42.

the incidence rate of stones in treated women would increase minimally from 1 to 1.2/1000 patients/year [46].

Two studies, one in normal and the other in stone-forming women, have examined the effect of calcium supplementation on calcium oxalate [47,48]. The study in normal women was performed on constant metabolic diet, so it is more interpretable [47]. After one month of calcium citrate (1000 mg in divided doses), urine calcium increased by about 50% and oxalate decreased by about 15%. However, after three months of treatment, calcium oxalate saturation was not significantly higher than baseline, possibly due to intestinal adaptation and calcium complexation by citrate. Similarly, the study in stone-forming women revealed no significant change in calcium oxalate saturation after six months of treatment with calcium citrate [48]. Though suspicion has been raised, the available data is not yet highly suggestive that calcium supplementation increases the stone formation risk in normal women.

**Bone Density**

In general, hypercalciuric stone-formers have low bone density (–5% to –15% compared to normocalciuric stone-formers) [49–54]. Moreover, some patients appear to develop negative calcium balance with severe calcium restriction despite their tendency to hyperabsorb calcium from the intestine [13]. Therefore, concern has been raised about severe calcium restriction.

In a retrospective study, Fuss *et al.* compared the radial

bone densities in 60 patients who had followed a low-calcium diet to 63 subjects who were on an unrestricted diet [55]. They found that the bone mineral content at the distal radius was significantly reduced in those on low calcium diet compared to the free diet group. Despite circumstantial evidence, it is still unclear whether the well-documented low bone mass in hypercalciuric stone-formers is due to overzealous dietary advice or their underlying disease. Evidence to the latter is that 30% of hypercalciuric children examined within one year of diagnosis already have low bone mass [56].

## CONCLUSIONS

In summary, hypercalciuria is a risk factor for kidney stone formation and low bone mass. Though dietary calcium increases urinary calcium, recent epidemiologic data suggests that high calcium intake may, in contrast, reduce risk of an initial stone. The observational data should be interpreted with some caution because calcium was not the only dietary difference, and an unmeasured but associated confounding variable may have been present.

For the normal population, full dietary calcium recommendations should be advocated. There is ample evidence that adequate calcium intake should protect against bone loss if not also against stone formation. In the hypercalciuric stone-forming population, a more prudent course should be advised. Along with increased fluid intake and restriction of salt, oxalate and animal flesh, its members should be advised to start consistent intake of one serving of dairy daily. Dietary calcium should be gradually increased as guided by the urinary calcium, and hypocalciuric agents should be added as necessary.

Future studies should be designed to examine the specific intervention of increased calcium intake, both by diet and by supplement, on urinary parameters and stone formation, particularly in hypercalciuric stone-formers who may respond conversely. Moreover, we must still determine whether the potential protection against stones is provided by calcium or by other factors present in dairy. Also, in hypercalciuric stone-formers, more work must be done to elucidate the direct cause of bone loss.

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