# Diet and calcium stones

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**Objective:** To review the current literature on the dietary modification of urinary risk factors as a means of reducing the likelihood of recurrent stone formation and to develop practical dietary recommendations that might be useful to this end.
**Data sources:** MEDLINE was searched for English-language articles published from 1983 to 1990. Additional references were selected from the bibliographies of identified articles.
**Study selection:** Nonrandomized trials and retrospective reviews were included because of a paucity of randomized controlled trials.
**Data synthesis:** Information on the dietary intake of calcium, oxalate, protein, sodium and fibre and on alcohol and fluid intake was used to develop practical guidelines on dietary modification.
**Conclusion:** Dietary modification plays an important role in the reduction of urinary risk factors in patients with calcium stone disease of the urinary tract. As an initial form of prevention attention should be directed toward moderating the intake of calcium, oxalate, protein, sodium and alcohol and increasing the intake of fibre and water. Future research should include an assessment of the long-term reduction of dietary and urinary risk factors and the rates of recurrence of calcium stones.

**Objectif :** Examiner la littérature récente sur la modification alimentaire des facteurs de risque urinaires comme moyen de réduire la probabilité d'une formation de calculs à répétition et pour élaborer des recommandations alimentaires pratiques qui pourraient être utiles à cette fin.
**Sources de données :** MEDLINE a fait l'objet d'une recherche documentaire pour des articles en anglais publiés de 1983 à 1990. Des références supplémentaires ont été choisies dans les bibliographies des articles repérés.
**Sélection d'études :** On a inclu des essais non randomisés et des études rétrospectives en raison de la rareté des essais randomisés et contrôlés.
**Synthèse des données :** Des renseignements sur l'apport alimentaire de calcium, d'oxalate, de protéines, de sodium et de fibres et sur l'apport d'alcool et de liquides ont servi à élaborer des lignes directrices pratiques sur la modification de l'alimentation.
**Conclusion :** La modification alimentaire joue un rôle important dans la réduction des facteurs de risque urinaires chez les patients qui souffrent de calculs calciques des voies urinaires. Comme première mesure préventive, on devrait prendre soin de modérer l'apport de calcium, d'oxalate, de protéines, de sodium et d'alcool, et d'augmenter l'apport de fibres et d'eau. Les recherches futures devraient comporter une évaluation de la réduction à long terme des facteurs de risque alimentaires et urinaires et des taux de récurrence des calculs calciques.

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Evidence of renal stones has been recorded since the time of the ancient Greeks and Romans. However, it is estimated that renal stones are at least 10 times more common now than they were at the beginning of the century.[1] They vary in chemical composition, but calcium stones are the most prevalent in industrialized nations. About 80% of calcium stones comprise calcium oxalate and 5% calcium phosphate; the composition of the remainder is mixed.[2,3] Renal stones are three times more common in men than in women, and the peak age of occurrence is between 30 and 50 years. They tend to recur in most patients.

Nephrolithiasis is a multifactorial condition that occurs when the chemical environment favours crystallization. The urine must be supersaturated with the salts of the crystals (e.g., calcium oxalate and calcium phosphate), and urinary inhibitors must be absent, present in low concentrations or functionally ineffective. These inhibitors include small ions such as magnesium and citrate as well as polyanions of high molecular weight such as glycosaminoglycans.[1,3] Certain urinary factors can influence the chemical environment and increase the risk of calcium stone formation: a low volume of urine, increased urinary excretion of calcium, oxalate or uric acid, a persistently low or high urinary pH and a low concentration of inhibitors.[4] A low urine volume, hyperoxaluria, hyperuricosuria and a low inhibitor concentration are associated with a greater risk of stone formation than hypercalciuria or changes in pH.[5]

Many epidemiologic variables can modify the urinary risk factors; these include age, sex, heredity, occupation, social class and affluence, geographic location and climate, and diet.[6] Of these, diet — including fluid intake — is the only one that can be easily changed and that has a marked effect on all urinary risk factors.[7,8] Despite the recognized importance of diet in prevention much of the literature continues to focus on pharmacologic means of controlling the risk factors. Although pharmacologic treatment may be effective the long-term side effects are unknown, the drugs are expensive, and it is not clear how long a patient needs to take them.

The precise nature of the interaction between diet and calcium nephrolithiasis is unknown; however, the effect of various nutrients on the urinary risk factors continues to be a topic of much research. The nutrients that have been associated with calcium stone disease are dietary calcium, oxalate, protein, sodium and fibre as well as alcohol and fluids. The interrelation of these nutrients and nephrolithiasis will be the focus of this article. We reviewed the current literature on the dietary modification of urinary risk factors as a means of reducing the likelihood of recurrent stone formation and developed practical dietary recommendations to this end.

## Literature review

MEDLINE was searched for English-language articles published from 1983 to 1990 that dealt with dietary modification of the urinary risk factors of calcium stone formation. Additional references were selected from the bibliographies of identified articles. Nonrandomized trials and retrospective reviews were included because of a paucity of randomized controlled studies. Articles were included if the studies involved human subjects and reliably measured the parameters of dietary and urinary risk factors. Animal studies and those of pharmacologic intervention were excluded.

## Dietary risk factors

### *Calcium*

Although a connection has been made between high dietary calcium intake and an increased risk of stone formation there are difficulties in comparing data on dietary intake and urinary excretion of calcium. Some studies have analysed patient-recorded diet journals, dietitian-elicited information on intakes, balance studies or radioisotope-labelled calcium absorption; others have failed to specify the details of the 24-hour urine collections and have differed in their techniques of calcium analysis. Furthermore, the variation in the incidence of hypercalciuria may have been a true one or a reflection of how the 24-hour urine collections were made and the diets standardized.[9] This section emphasizes the major themes of current research.

Dietary calcium is absorbed from the intestines with the help of vitamin D and parathyroid hormone; any excess is excreted in the urine. A direct relation has been found between calciuria and a calcium intake of up to 800 mg/d; above this level there was no further increase in calcium excretion.[8] This suggests that when dietary levels of calcium are high, absorption is adjusted. There is a reduced absorption of calcium at high intakes in healthy subjects, but people with idiopathic stone formation show increased calcium absorption because of an altered response to vitamin D.[10] Also, a linear relation exists between stone formation and a calcium intake of up to 1000 mg/d, and the risk of stone formation increases sharply with intakes greater than 1000 mg/d.[3,8] This indicates that reducing the calcium intake to less than 800 mg/d could decrease calcium excretion in the urine and reduce the risk of stone formation. This may be even more important for patients who lack the ability to decrease calcium absorption at high intakes.

Most studies have indicated that a decrease in calcium intake by stone-forming patients results in

decreased calcium excretion.[11-13] However, these studies have also indicated that decreasing the calcium intake without also decreasing the oxalate intake leads to increased oxalate excretion, which carries an even greater risk for subsequent stone formation. This increase is the result of a greater concentration of free oxalate in the intestine. Excess calcium and oxalate usually form complexes in the intestine that are then excreted in the feces. When the calcium intake is limited the reduced amount of unabsorbed calcium left in the intestine results in more free oxalate, which is then absorbed by passive diffusion in the colon.[13,14] The rise in urinary oxalate excretion disappears when a dietary oxalate restriction is also imposed.[13]

The dietary calcium restriction must be carefully controlled not only to reduce the risk of stone formation but also to avoid a negative calcium balance. A severe calcium restriction, to less than 400 mg/d, is not recommended, because over a long period it may result in osteoporosis from excessive resorption of calcium from bone.[15,16] A calcium restriction to 400 to 650 mg/d has been found to reduce the amount of calcium excreted in patients with stones, but the precise dietary prescription will also have to take into account the patient's age and sex.[10,17] For example, the risk of calcium nephrolithiasis from high-normal or even supplemental calcium intake is probably low in most postmenopausal women because of the impaired intestinal absorption of calcium and blunted calciuric response.[18] It may be wiser to emphasize adjustments in the other urinary risk factors in this group. A moderate oxalate restriction should also be applied.

## *Oxalate*

After urine volume, hyperoxaluria may be the most important risk factor in calcium nephrolithiasis. Small increases in oxalate excretion have a pronounced effect on the activity product of calcium oxalate in the urine.[8,14] The normal amount of oxalate excreted is less than 40 to 50 mg/d.[14] The dietary component of urinary oxalate is derived from the breakdown of dietary ascorbate and oxalate. Dietary oxalate accounts for only 10% to 15% of urinary oxalate, but any dietary excess is excreted in the urine since oxalate is a metabolic end product and is not further degraded in the body. In fact, 90% of an injected dose of radioisotope-labelled oxalate is excreted in the urine within 24 to 36 hours.[19]

The amount of oxalate excreted in urine has been found to be higher in people with stones than in healthy subjects at all levels of intake. This suggests that the former group absorbs more oxalate, consumes more oxalate-producing substances such as ascorbate or metabolizes more oxalate precursors. Intakes of up to 180 mg/d appear to have little effect on the urinary oxalate excretion, but intakes above this amount lead to a marked increase in the amount excreted.[8] This implies that although dietary oxalate is a relatively small contributor to urinary oxalate, foods high in oxalate can promote hyperoxaluria and increase the risk of stone formation. Although only 2% to 12% of oxalate is absorbed from foods an increase in oxalate excretion of up to 500% above normal is noted after the ingestion of high-oxalate foods.[14,20] Patients with hyperoxaluria should not eat such foods (Table 1).

Patients experiencing problems with gastrointestinal absorption may be at greater risk than others for hyperoxaluria. This is particulary true for those with steatorrhea: calcium in the intestine combines with free fatty acids to form soaps; this leaves more free oxalate to be absorbed. It is also thought that the fatty acids and bile salts may increase the colon's permeability to oxalate and thereby result in greater absorption and subsequent urinary excretion.[14] Such patients should be placed on a low-oxalate, low-fat diet that includes medium-chain triglycerides and a calcium supplement to decrease oxalate absorption.[4,19]

## *Protein*

A high intake of animal protein is linked with an increased risk of stone formation.[8] Purine is found primarily in animal proteins. Uric acid is a metabolite of purine, but its level in urine can also be affected by diet.[21] A consistent linear relation between purine intake and uric acid excretion has been described in hyperuricosuric people with calcium oxalate stone formation.[22] A dramatic increase in uric acid excretion has also been obser-

Table 1: Foods high in oxalate that should be avoided in the prevention of calcium stones

| Food | Serving size | Amount of oxalate per serving, mg |
|---|---|---|
| Baked beans | 250 mL | 50 |
| Blackberries | 250 mL | 66 |
| Chocolate | 28 g | 35 |
| Cocoa | 15 mL | 35 |
| Gooseberries | 250 mL | 132 |
| Leeks | 250 mL | 89 |
| Peanuts | 250 mL | 288 |
| Rhubarb | 125 mL | 1092 |
| Rutabaga | 250 mL | 32 |
| Spinach | 250 mL | 1350 |
| Squash | 250 mL | 40 |
| Sweet potato | 1 (medium) | 63 |
| Swiss chard and beet greens | 250 mL | 1000 |
| Tea | 250 mL | 25 |

ved after the diet of healthy subjects was supplemented with ribonucleic acid (purine).[23] Others have shown that in patients with a diet high in animal protein the excretion of urate was almost doubled.[24]

Hyperuricosuria has been associated with an increased risk of calcium stone formation and is present in nearly one-third of all people with calcium oxalate stones. There are three theories of how uric acid promotes calcium stone formation. The first suggests that similarities in crystal structure promote epitaxial growth of calcium oxalate on uric acid in supersaturated urine.[25] The second proposes that uric acid adsorbs onto the surface of the polyanionic inhibitors of calcium oxalate and therefore interferes with their action and provides the opportunity for stone formation.[4] The third suggests that glutamic acid adheres to the surface of the uric acid crystals and thus encourages the calcium and oxalate ions to aggregate and crystallize.[25]

A high-protein intake leads to an increased net acid excretion; this may help explain why a high-protein diet increases urinary calcium excretion.[26] Such a diet also lowers the urinary pH and can cause an increase in uric acid excretion as a result of the dissociation of sodium urate. A low pH may reduce the effectiveness of the polyanionic inhibitors and decrease the urinary excretion of citrate.[24]

Sulfur-containing amino acids such as methionine (found in dietary protein) are believed to have a hypercalciuric effect. Because calcium sulfate complexes that develop within the renal tubule are not easily reabsorbed they are excreted and result in an increased urinary calcium level.[23] Methionine is found primarily in animal proteins. It has been shown that healthy subjects who consume a regular diet excrete more calcium than those on a vegetarian diet.[27] Other studies have observed increased calcium excretion in subjects who progressed from a vegetarian diet or a diet low in animal protein to high intakes of animal protein.[28,29]

In light of the evidence implicating a high intake of animal protein with hyperuricosuria, hypercalciuria and a low urinary pH it had been proposed that people with renal stone formation follow a vegetarian diet. This was not without problems, since an increase in the intake of vegetable protein can lead to an increased excretion of oxalate.[27,28] People with calcium stones who have hyperuricosuria are therefore advised simply to avoid excessive intakes of animal protein. Modest protein restrictions, to less than 240 g (8 oz) of meat, fish or poultry daily, have been found to reduce the incidence of hyperuricosuria. Patients should also be cautioned against the overconsumption of foods particularly high in purine.[30]

### *Sodium*

There is a close association between the urinary excretion of sodium and calcium because of the many common sites of absorption along the renal tubule. Minor changes in dietary sodium intake have been found to result in significant changes in the 24-hour excretion of urinary calcium in subjects with stones. Hypercalciuric patients with stones whose calcium intake was restricted to 700 mg/d and whose sodium intake was increased from 80 to 200 mmol/d were found to excrete an extra 97 mg of calcium daily for each 100-mmol/d increase in urinary sodium.[31] There has been a suggestion that people with calcium stones are more sensitive than the rest of the population to the calciuric effects of sodium.[32] Mild sodium restriction — avoidance of the use of salt and the intake of high-sodium foods — is therefore recommended for hypercalciuric patients.

### *Fibre*

Patients with stones have been found to have a lower intake of dietary fibre than healthy subjects.[8] Recent research has focused on the potential protective effect of dietary fibre in preventing the recurrence of stone formation. A sharp decline in the recurrence rate was found in patients who consumed 30 g of a preparation of wheat and soya bran daily.[33] Other studies have noted a reduced frequency of recurrence among hypercalciuric patients given 20 g of rice bran daily. The effect was even more pronounced in those who had had recurrences the most frequently.[34]

The exact nature of the interaction between dietary fibre and calcium is still unknown, but several theories have been proposed. The first suggests that phytic acid, found in bran, binds to calcium in the intestine, and the complexes are then passed in the stool. The second proposes that the intestinal transit time is decreased by fibre and may allow less time for calcium absorption. Finally, bran may simply bind mechanically to dietary calcium. One or more of these interactions may occur at any given time.[35,36]

If the phytate mechanism were predominant an increase in oxalate excretion would be expected, since there would be less calcium available for binding in the intestine. Only a few researchers have observed an increase in oxalate excretion with fibre supplementation. Patients whose diets were supplemented with 20 g of a rice bran mixture daily showed a slight increase in such excretion.[34] An increase was also observed at first in patients who received a supplement of 24 g of wheat bran daily, but this effect decreased with time.[37]

The oxalate content of the bran itself may be

responsible for the increased excretion of oxalate. A group of 17 hypercalciuric patients on a low-calcium, low-oxalate diet was studied with and without the addition of 30 g of fibre as unprocessed bran.[38] The bran contained 164 mg of oxalate. No increase in oxalate excretion was observed among the patients who received the fibre, but the reduction in the rate of excretion was not as great as in the subjects not given the supplement. This was attributed to the oxalate content of the bran. The 30 g of fibre used in this study is equivalent to 78.2 g of unprocessed bran, over twice the amount used in another study.[38]

The effect of two forms of rice bran, one defatted and the other with the phytate removed, has also been examined. The phytate-free rice bran was found to be less effective than the defatted bran in reducing the rate of calcium excretion, but the difference was less than one would expect if phytate were the only factor involved.[39] This finding favours the suggestion of mechanical binding with calcium, which would not necessarily lead to an increased rate of oxalate excretion unless the oxalate content of the fibre supplement were high. The oxalate content of various sources of fibre has been analysed with the following results: soya 17 mg/100 g, wheat 240 mg/100 g, rice 123 mg/100 g and corn 7 mg/100 g.[40] Therefore, adding 20 to 30 g of wheat or rice fibre could increase a person's oxalate intake by 48 to 72 mg and 25 to 37 mg respectively. However, only 2% to 12% of the oxalate found in foods is absorbed, and recent studies have shown no evidence of increased oxalate excretion with fibre supplementation. One study found no variation in oxalate excretion after 90 days of supplementation with 28 g of wheat bran.[36] Another observed a decrease in such excretion with 30 g of a wheat and soya bran mixture daily.[41] Finally, one study placed hypercalciuric patients with stones on a diet low in calcium, oxalate and animal protein for 3 months. At the end of this time two fibre biscuits (10 g of fibre as wheat and corn) were added to the diet. The modified diet alone resulted in reduced levels of urinary calcium and oxalate, but the added fibre led to further decreases in both.[42] This suggests that dietary fibre supplements are most effective when used in conjunction with a modified diet.

The effect of fibre remains an area of active research with many unanswered questions. All studies to date have looked at increasing the amount of fibre through supplementation; the effect of increasing the dietary fibre content remains to be examined.

### *Alcohol*

The effects of alcohol on the risk of stone formation have not been extensively investigated. Recent evidence suggests an almost linear correlation between alcohol consumption and levels of urinary calcium and uric acid.[7] Beer (particularly draught beer) is known to contain oxalate and guanosine, which is metabolized to uric acid in the body.[6,43] On the other hand, there might be some benefit from beer because of its diuretic effect, but this has not been studied. Alcohol should therefore be consumed in moderation by people with calcium stones.

### *Fluids*

Fluid intake is the most important dietary modification for patients with stones. It is also the only dietary recommendation that is applicable to all forms of nephrolithiasis, regardless of cause.[4] It has been recognized as an important factor in the prevention of kidney stone recurrence since the time of Hippocrates, who told his patients to drink more water.

A decreased fluid intake leads to a low urine volume and increased concentrations of all stone-forming salts. This risk of stone formation is increased with urine volumes of less than 1 L/d and decreased with volumes of more than 2 to 2.5 L/d.[6,11] Some investigators have argued that increasing the urine volume decreases the concentration of urinary inhibitors and thus magnifies the risk of stones. No evidence of this has been found.[44]

At least 250 mL of fluid should be taken with each meal, between meals, before bedtime and when the patient gets up at night to void. This will ensure that the fluid intake is spread out over the day and that the urine is not concentrated. At least half the fluid should be taken as water. The composition of the remainder is up to the patient, unless his or her diet is restricted in calcium or oxalate, in which case overdependence on milk, tea, hot chocolate, draught beer and citrus juices must be avoided. It is also important for patients with stones to increase their fluid intake in hotter weather and after vigorous exercise.[6,11]

## Compliance

Many who argue against dietary intervention suggest that the diet is unpalatable and unlikely to be followed for any extended period; they therefore advocate pharmacologic therapy in its place. Although such therapy can be effective its long-term side effects are unknown, and it is expensive. No single medication can influence all the urinary risk factors, and it is often difficult to use separate drugs to treat each factor. Furthermore, there is no evidence to suggest that patients would be more compliant with their medications than with their dietary

modification. When 62 patients with idiopathic stone formation were followed for 6 to 43 months it was found that 70% were compliant with all the recommended changes in their diet and another 24% were partially compliant.[45]

Patients should receive individualized instruction from a qualified dietitian so that dietary compliance is encouraged. The instruction should be as simple and the diet as unrestricted as possible. The objective is to limit excesses rather than overall intake and to tailor the dietary modification to the urinary risk factors. This was clearly shown in a study involving people with stones who were advised only to increase their fluid intake and avoid excesses of calcium and protein: 58% of the patients showed no new stone growth after 62 months.[46]

Patients must be made aware of the urinary risk factors for stone formation. This will help them understand the importance of diet in preventing stone recurrence and should encourage compliance. Dietary compliance also depends on the appropriateness of the diet prescription. Patients may need help in adapting the diet to their particular lifestyle. Regular follow-up visits and reviews of changes in urinary risk factors are essential. They serve to reinforce the need for the diet, address problems and concerns and provide the extra motivation and encouragement required for continued compliance.

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## *Conferences*
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**Mar. 16-19, 1992:** Cardiovascular Conference at Lake Louise
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Montreal Grand Hotel
Donald Gilbert, Quebec Epilepsy Association, 4839-3175 Cote Ste. Catherine Rd., Montreal, PQ H3T 1C5; (514) 342-6877, fax (514) 342-1909

**Mar. 22-26, 1992:** International Symposium — Pollinosis in the Mediterranean Area
Herzlia, Israel
Conference coordinator, ORTRA Ltd., PO Box 50432, Tel Aviv 61500, Israel

**Mar. 22-27, 1992:** Congress of Dietetics
Jerusalem
Conference coordinator, Kenes USA, 903-271 Madison Ave., New York, NY 10016; (212) 986-8300

**Mar. 23-24, 1992:** University of Toronto Institute of Health Management Seminars on Continuous Quality Improvement in Healthcare, Series 1, part 3: Methods and Tools for Quality Improvement (part 4, Apr. 14, 1992)
Toronto
Fern Greenbaum, Conference Communications Plus, PO Box 573, Stn. Z, 413 Eglinton Ave. W, Toronto, ON M5N 2Z6; (416) 489-5932, fax (416) 489-0119

**Mar. 23-29, 1992:** International Conference on Reduction of Drug-Related Harm
Melbourne
Mr. W. Stonach, Alcohol and Drug Foundation, PO Box 529, South Melbourne, VIC 3205, Australia

**Mar. 26-27, 1992:** Theoretical and Clinical Perspectives (sponsored by the Rotman Research Institute, Baycrest Centre for Geriatric Care)
Toronto
Education Department, Baycrest Centre for Geriatric Care, 3560 Bathurst St., Toronto, ON M6A 2E1; (416) 789-5131, ext. 2365

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