

Dietary recommendations and treatment of patients with recurrent idiopathic calcium stone disease

W. G. Robertson¹

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Abstract This review describes the various dietary regimens that have been used to advise patients on how to prevent the recurrence of their calcium-containing kidney stones. The conclusion is that although there is some general advice that may be useful to many patients, it is more efficacious to screen each patient individually to identify his/her main urinary, metabolic, nutritional, environmental, and lifestyle risk factors for stone-formation and then tailor specific advice for that particular patient based on the findings from these investigations. If the patient can be motivated to adhere strictly to this conservative approach to the prophylactic management of their stone problem over a long time period, then it is possible to prevent them from forming further stones. This approach to stone management is considerably less expensive than any of the procedures currently available for stone removal or disintegration. In the UK, for each new stone episode prevented by this conservative approach to prophylaxis it is calculated to save the Health Authority concerned around £2000 for every patient treated successfully. In the long term, this accumulates to a major saving within each hospital budget if most stone patients can be prevented from forming further stones and when the savings are totalled up country-wide saves the National Exchequer considerable sums in unclaimed Sick Pay and industry a significant number of manpower days which would otherwise be lost from work. It is also of immense relief and benefit to the patients not to have to suffer the discomfort and inconvenience of further stone episodes.

Keywords Dietary management · Metabolic screening · Urolithiasis · Fluid intake · Calcium intake · Oxalate intake · Animal protein intake · Salt intake · Sugar intake

Abbreviations

CaOx Calcium oxalate
UA Uric acid
 P_{sf} Biochemical probability of forming stones
PRAL Potential renal acid load

Introduction

Urolithiasis has become a major problem throughout the world not only for the patients and doctors concerned, but also for the budgets of the health authorities which are having to fund the cost of the increasingly expensive urological management of the patients who are referred to hospital to have their stones removed or disintegrated [1–4]. Since the urological management of patients does not cure the underlying cause of the stones in the vast majority of patients, most will return with a recurrence of their problems at a later date which adds to the problem created by the already increasing incidence of new stones in most countries world-wide [3, 4]. To reduce the burden on urology departments, therefore, emphasis is now being focused on how to prevent patients experiencing further stone episodes after removal of their first stone(s). Since the pharmacological management of stone-formers usually requires them to take some form of medication for the remainder of their lives, emphasis is now being placed on more conservative forms of management through changing the diet and lifestyle of patients.

The main object in the prevention of stone recurrence must be to reduce the biochemical risk of forming

✉ W. G. Robertson
robertsonwilliam67@gmail.com

¹ Nuffield Department of Surgical Sciences, John Radcliffe Hospital, Oxford OX3 9DU, UK

stones. Usually this is directed at reducing the supersaturation of urine with respect to the patient's stone type but may also require an increase in the excretion of urinary inhibitors of crystallisation or a decrease in the excretion of urinary promoters of crystallisation [5]. It is important to emphasise, however, that the treatment regime should be tailored to the specific metabolism of the patient concerned, as defined by the risk factors identified through a comprehensive metabolic, nutritional, environmental and lifestyle screening procedure [5]. It is clear that, if you take 100 stone-formers and feed them all exactly the same foods and drinks for a week and then measure their 24-h urine compositions at the end of the week, these will vary widely from patient to patient. This is because of inter-individual variability in the digestion, intestinal absorption, metabolism and renal handling of the various constituents of their diets. As the old adage says, "One man's food is another man's poison". In addition, some studies suggest that it is those individuals in the population who are most sensitive to a given dietary, environmental, metabolic or lifestyle stimulus that frequently end up with the most extreme excretions of the various urinary risk factors mentioned above [6]. They are the people who are most likely to form stones. Therefore, each patient should be treated as an individual and not as a member of some wide-ranging heterogeneous group classified under the broad heading of "idiopathic calcium stone-formers".

For many years, diet has been considered to be a major risk factor for so-called idiopathic calcium stone-formation [5, 7–11]. Many dietary factors have been implicated in the causation of these stones including an insufficient fluid intake [9–14], high or low intakes of calcium [10, 15] or fibre [5]; high intakes of oxalate [16–18], animal protein [8, 19–21], sodium [22, 23] or refined sugars [24, 25]; and low intakes of magnesium [26, 27] or potassium [28]. Reversing these dietary abnormalities has been tried with varying degrees of success on the urinary risk factors for stones and on the rate of stone recurrence. The object of this review is to examine (a) the evidence that diet does play a role in idiopathic calcium stone-formation, (b) the logic behind changing the composition of the diet in a given patient, and (c) how best can this form of prevention of stone recurrence be managed in the average Metabolic Stone Clinic. The data are taken from various reports in the literature and from personal experience over many years of manipulating the diets of stone-formers attending Private or National Health Service (NHS) Stone Clinics in the UK for comprehensive screening of their risk factors for stone-formation.

Screening procedure

To treat the patients as individuals, however, requires more than just a simple dietary screen. It also requires some knowledge of the patient's metabolic status and lifestyle characteristics, a point that has been emphasised in the recently published AUA Guidelines for the screening of patients with kidney stones which comprises a comprehensive assessment of the patient's metabolic and nutritional risk factors for stone-formation [29]. This recommends the following range of investigations (my personal comments are added in brackets)—the analysis of a blood sample and a snap (spot) urine sample taken at the same time (preferably after the patient has fasted overnight), at least one 24-h urine collection on his/her free home diet (never as an in-patient) and a nutritional screen on each patient. This goes beyond the scope of most existing major screening procedures in the USA, such as Litholink, which only infers the dietary intakes of a few nutrients from the composition of a 24-h urine collected by the patient, although it does include the other screening tests described in the AUA Guidelines. One existing screening procedure which covers all of the tests recommended in the AUA Guidelines is LITHOSCREEN which, in addition, includes a lifestyle questionnaire to check for potential extrinsic risk factors that can often account for apparent anomalies in the composition of urine from a given patient when compared with that predicted from his/her diet and metabolism alone [5, 30]. In my experience, only when you have the combined metabolic, nutritional and lifestyle data on a given patient is it possible to account completely for the composition of his/her urine which, in turn, determines the composition of his/her stones. The full LITHOSCREEN procedure for the screening of patients with stones is currently in preparation for publication although elements of it can be found in various publications [5, 13, 30].

As part of the LITHOSCREEN procedure the patient is required to record his/her intakes of all foods and drinks during a 7-day period while on his/her free, home diet (never as an in-patient). The dietary analysis programme employed yields data on total fluid intake (including the water contained in the various foods consumed), calorific intake, total calcium, calcium derived from dairy produce (which is the most absorbable source of calcium in the diet), magnesium, sodium, potassium, phosphate, oxalate, chloride, total protein, protein derived from meat + fish + poultry, protein derived from dairy produce, protein derived from fruit, vegetables and cereals, purine, fat, carbohydrates, starch, potential renal acid load (PRAL), sugars, and fibre.

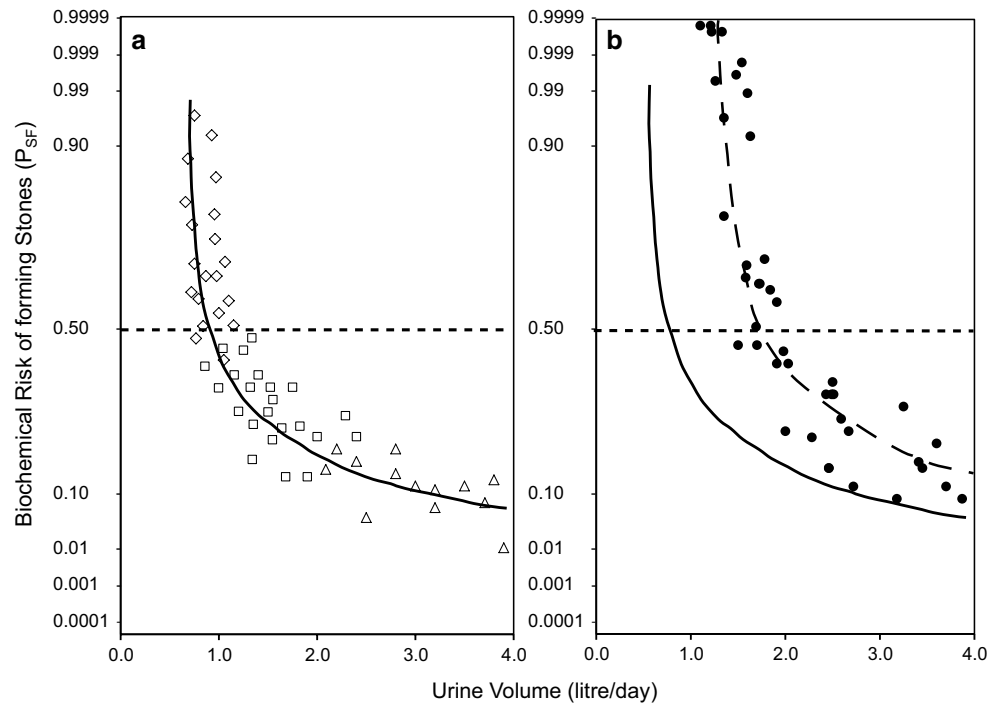


Fig. 1 The relationship between the biochemical risk of forming calcium-containing stones (P_{SF}) in **a** a group of normal subjects studied on a low (*open diamonds*), normal (*open squares*) and high (*open triangles*) fluid intake and **b** a group of untreated idiopathic calcium

stone-formers (*closed circles*) but including some who were on a self-prescribed high fluid intake. The trend lines for the normal subjects are shown as *continuous lines* and for the stone-formers as a *dashed line*

Dietary constituents potentially involved in calcium stone-formation

Fluid intake

Since the days of Hippocrates the most commonly used “dietary” strategy for reducing the recurrence of idiopathic calcium stones has been to advise the patient “to drink more fluid” [12]. But how much more and in what form should the additional fluid be taken? Figure 1a shows the biochemical risk of stones (P_{sf}) [13] in a group of normal subjects studied on a low, normal and high fluid intake in relation to their corresponding 24-h urine volumes. This shows that the risk of forming stones in the normal population rises sharply into the high risk region for stone-formation (P_{sf} values >0.5 on a probability scale ranging between 0 and 1) as urine volume drops below about 0.9 l/day. Figure 1b shows the trend line in normals based on the data in Fig. 1a together with the data obtained from a group of idiopathic calcium stone-formers studied in the Stone Clinic, some of whom were untreated and some were already on a self-prescribed high fluid regimen. This shows that the data from the stone-formers are shifted to the right compared with those of the normal subjects because the patients have a range of other urinary abnormalities such

as hypercalciuria, mild hyperoxaluria, hypocitraturia, etc., which increase their risk of forming stones at all levels of urinary volume. In the case of the stone-formers, the resulting trend line rises sharply into the danger zone when urine volume drops below about 1.6 l/day. Figure 1b shows that to get the P_{sf} value of his/her urine down to a safe level (<0.5), the answer to the question—“How much should I drink?”—is that the patient should be encouraged to consume sufficient fluid to enable him/her to excrete consistently at least 2.2–2.5 l/day *under all circumstances*. However, for patients with the most extreme urinary abnormalities, this might require them having to excrete closer to 3 or 4 l/day if they are only told to drink more fluids with no additional dietary advice.

How much the patient has to drink to achieve these urinary volume targets depends on many additional factors usually identifiable from the lifestyle questionnaire referred to above. This includes questions such as—does the patient sweat much under normal conditions, does he/she have night sweats, does he/she suffer from stress, does he/she exercise strenuously and sweat when doing so, does he/she work in a hot environment, does his/her work involve a moderate-to-high degree of physical activity, does he/she travel frequently by air (especially long-haul flights), does he/she travel regularly on business trips or on vacation to

countries with hot climates, and does he/she suffer from frequent bouts of diarrhoea or regularly take laxatives? In all of these situations, the patients will have to consume considerably more fluid to achieve a urine volume of 2.2–2.5 l/day.

Since the LITHOSCREEN procedure yields data on both total fluid intake and the corresponding 24-h urine volume collected from the patient as his/her dietary screen, this generally makes it possible to account for large discrepancies between the two (i.e., more than 200–250 ml/day) from the patient's lifestyle data. Of course, urine volume never matches exactly the total fluid intake of the patient since we all lose water from the body in many ways during the day from various bodily functions such as sweating, insensible fluid loss, the water vapour content of our breath, and the water content of our faeces. The comparison between fluid intake and urine volume then allows the physician to assess and advise the patient on how much actual additional fluid he/she should take to compensate for the fluid losses incurred by one or more of the above potential dehydrating factors.

There are some situations, however, where a high fluid intake may be difficult or inconvenient for the patient—for example, (a) if the patient has any bowel disorder, such as chronic diarrhoea or Crohn's disease, that might make it difficult for them to adhere to a high fluid diet, (b) if the patient has had any bowel surgery, such as an ileostomy or a colectomy, which might make it difficult for them to tolerate a high fluid intake, (c) if the patient has an enlarged prostate and finds it inconvenient to go more frequently to the toilet, or (d) if the patient has an occupation, such as taxi-driving or working in a restaurant kitchen or an operating theatre, that makes it difficult to go to the toilet for long periods during working hours. These groups of patients are difficult to treat by means of a high fluid intake alone. I usually encourage them to consume as much as they can without them being inconvenienced or embarrassed and try to find alternative approaches to their stone prevention.

In answer to the question about what form the additional fluid intake should take, I usually advise against (a) sugary drinks (since refined sugar increases the intestinal absorption of calcium), (b) black tea and black coffee, particularly instant coffee, (because of their high content of readily absorbable oxalate), and (c) fizzy drinks with a high acid phosphate content (since this adds to the potential renal acid load of the diet). Consequently, I usually advise the patient to take his/her extra fluid in the form of water. Of course, this leads to the question of what type of water should they consume—hard, soft, mineral, filtered or bottled?

There is no consistent evidence that populations living in hard water areas have a higher prevalence of stones than those living in areas where the water is soft [31–34].

Anecdotally, it has been found that if the patient lives in a hard water area and happens also to be a hyperabsorber of calcium (which is a feature of a relatively small proportion of calcium stone-formers), then drinking 4–5 l of unfiltered tap water can result in a urinary calcium excretion of over 12 mmol/day. By advising that patient to change his water consumption to bottled water (which in the UK generally has a very low content of calcium) this reduced urinary calcium excretion of one of my patients, who lived in a very hard water area, from over 12 mmol/day down to 6–7 mmol/day. However, hard water usually has an accompanying high content of magnesium, which some consider to be a mild inhibitor of calcium stone-formation, partially as a complexing agent for oxalate in the intestine (thereby decreasing the availability of oxalate for absorption in the large bowel) and partially as a complexing agent for oxalate in urine (thereby decreasing the relative supersaturation of urine with respect to calcium oxalate [34]). Furthermore, the effect of drinking more fluid (even if it is from a hard water area) will consistently dilute the urinary concentrations of all the other risk factors for stone-formation. Thus, drinking hard water may at the same time be both detrimental in promoting and beneficial in preventing stone-formation; so that the net effect is probably “stone-neutral”. However, from personal experience, one major problem in treating patients who live in hard water areas, such as London and the south-east of England, is that any injunction to “drink more water” is met with the response that “my local tap water is not pleasant to drink, doc. So, I find it difficult to drink a lot”. I usually advise these individuals to switch to filtered or bottled water instead.

Several studies have shown that a high fluid intake is effective in decreasing the recurrence rate of stones in patients with idiopathic calcium stones [35–40]. Theoretically, the only situation where drinking more fluid is likely to be of doubtful benefit is (a) in the case of those patients whose *sole* risk factor for stone-formation is a deficiency in the excretion of one or more of the inhibitors of crystallisation, such as citrate and magnesium and (b) in those patients whose main risk factor is the passage of an excessively acid or alkaline urine. By further diluting the already low concentrations of the protective inhibitors in urine, this may aggravate the risk of forming new stones even though the resulting higher urine volume may also dilute the urinary concentrations of calcium, oxalate, and uric acid. In patients whose dominant risk factor is passing excessively acidic or alkaline urine, dilution per se has virtually no effect on urinary pH and so theoretically drinking more fluid may not significantly reduce the overall biochemical risk of forming further stones in this sub-group of stone-formers.

A summary of the advantages and disadvantages of a high fluid intake and of the various dietary treatments

Table 1 A summary of the advantages and disadvantages of various dietary measures to decrease the biochemical risk of forming recurrent stones in idiopathic calcium stone-formers

Dietary measure (recommended)	Potential advantages	Potential disadvantages	Net effect on stone risk
High total fluid intake (>2.5 l/day)	↓ Urinary concentrations of Ca, Ox, UA	↓ Urinary concentrations of Mg, citrate	↓ RS CaOx ↓ RS CaP
Low oxalate intake (<1.5 mmol/day)	↓ Excretion of Ox	Difficult to maintain	↓ RS CaOx
Low calcium intake (not recommended)	↓ Excretion of Ca	↑ Excretion of Ox	δ↑ RS CaOx ↓ RS CaP
High calcium intake (20–25 mmol/day)	↓ Excretion of Ox	↑ Excretion of Ca by calcium hyperabsorbers	↓ RS CaOx δ↑ RS CaP
Low animal protein diet (30–40 g/day)	↓ Excretions of Ca, Ox, UA ↑ Excretion of citrate	Difficult to maintain	↓ RS CaOx ↓ RS CaP
Phosphate supplements (not recommended)	↓ Excretion of Ca ↑ Excretion of pyrophosphate	Stimulation of parathyroids?	↓ RS CaOx δ↑ RS CaP
High magnesium intake (>16 mmol/day)	↑ Excretion of Mg ↓ Excretion of Ox	Gastrointestinal side-effects	↓ RS CaOx
Magnesium hydroxide supplements (10 mmol/day)	↑ Excretion of Mg ↓ Excretion of Ox	Gastrointestinal side-effects	↓ RS CaOx
High potassium diet (>70 mmol/day)	↑ Excretion of citrate	δ↑ Urinary pH?	↓ RS CaOx
Potassium citrate supplements (20 mmol/day = 60 mmol/day of potassium)	↓ Excretion of Ca ↑ Excretion of citrate	↑ Urinary pH	↓ RS CaOx ↑ RS CaP
High fibre diet (18–22 g/day)	↓ Excretion of Ca ↑ Urinary concentrations of Mg, Citrate	↓ Urine volume ↑ Urinary concentrations of Ox, UA	δ↑ RS CaOx δ↓ RS CaP
Low sodium intake (<140 mmol/day)	↓ Excretion of Ca	Difficult to maintain	δ↓ RS CaOx δ↓ RS CaP
Low sugar intake (<100 g/day)	↓ Excretion of Ca	Difficult to maintain	δ↓ RS CaOx δ↓ RS CaP

RS CaOx, relative supersaturation of urine with respect to calcium oxalate; RS CaP, relative supersaturation of urine with respect to calcium phosphate; ↑, increase; ↓, decrease; δ, slight; UA, uric acid

described below for the management of idiopathic calcium stone-formers is contained in Table 1.

Oxalate intake

Risk factor analysis has shown that next to a low urine volume, the second most important individual risk factor for calcium-containing stones is mild hyperoxaluria [13, 16]. The average so-called “upper limit of normal” for urinary oxalate is considered to be around 0.45 mmol/day in most series in the West although that figure is much higher in the oil-rich Gulf States in the Middle East [17, 41, 42]. Risk factor analysis shows that at a urinary oxalate of 0.45 mmol/day, the risk of forming stones in the average individual is already increased by a factor of about 1.7. In this context, it is important to recognise that risk is a continuous function such that the probability of forming stones is already increasing by the time urine reaches the so-called upper (or lower) limit of normal for each of the seven risk factors found to be of importance for the formation of calcium- and/or uric acid-containing stones. It is not

a discrete function that immediately becomes significant at some arbitrary upper (or lower) limit for the variable concerned. Furthermore, risk is generally not a linear function in relation to the urinary excretion of the various risk factors involved in stone-formation; it has been shown to increase either geometrically or logarithmically with a linear increase (or decrease) in the values of each of the various risk factors [13].

It has been assumed for many years that a diet high in oxalate-containing foods is likely to increase the risk of forming stones [43–45]. On the other hand, there is not yet any solid evidence that decreasing oxalate intake has a beneficial effect of the recurrence rate of stones although it does reduce the urinary excretion of oxalate [46] and with it the relative supersaturation of urine with respect to calcium oxalate [46, 47]. The reasons for the lack of data on the recurrence rate of stones on a low oxalate diet are multiple—(a) the need for a universally accepted and fully reliable method for measuring oxalate in biological media and in foods (the widely used enzymatic method for measuring urinary oxalate may underestimate oxalate particularly in

urines with a high calcium content [43]), (b) the sparsity of data on the bioavailability of oxalate in various foods and (c) an incomplete knowledge of the ability of the intestine to absorb oxalate from stomach to large bowel.

Some foods are particularly high in oxalate content. These include rhubarb, spinach, beetroot, parsley, okra (ladies' fingers), plantains, soya beans and some soya products such as tofu, yams, baked potatoes including the skin, wheat bran, rice bran, nuts and nut-containing foods, peanut butter, sesame seed-containing foods, black pepper, chocolate and chocolate-containing foods, instant coffee, and tea. Most fruits, vegetables and unrefined cereals contain a low to moderate amount of oxalate but if these foods are extracted and concentrated, such as in the case of cranberry juice tablets, the resulting oxalate content can be very high [48]. Thus, one 450 mg tablet of cranberry concentrate contains around 2 mmol of oxalate and taking the recommended dosage of two tablets per day would add approximately 4 mmol of oxalate to the diet—a twofold to threefold increase in the normal daily intake of that dietary constituent.

Very little is known about the food content of the immediate metabolic precursors of oxalate, such as hydroxypyruvate, L-glycerate, glycollate, and glyoxylate [49]. Hydroxyproline, which is another potential precursor of oxalate and is present in relatively high quantities in the collagen of meat, fish and poultry protein, is known to increase the urinary excretion of oxalate in animals [50, 51] and humans [52]. As a result of these metabolic processes, some studies have shown that there is a relationship between urinary oxalate and animal protein consumption [19, 53] although others have not found this [54]. The reasons for this discrepancy are unclear, although one study has suggested that, for some reason, only about 30 % of patients are sensitive to meat protein in terms of its effect on urinary oxalate excretion [53].

Ascorbic acid is another potential precursor of oxalate but there is some debate as to whether or not increased ingestion of vitamin C actually increases urinary oxalate. It would appear that if precautions are taken with the procedure for the collection of urine and with the method of urinalysis, then ingestion of ascorbic acid in doses of 1–2 g/day does increase urinary oxalate by approximately 20–30 % [55–58], although others disagree [59]. As a precaution, I usually advise patients who are taking vitamin C supplements not to exceed a daily dose of 1 g.

The bioavailability of oxalate in different foods and the composition of the accompanying ingested food may be additional factors in determining how much oxalate is actually absorbed from a given diet [43, 60]. It has been shown that oxalate ions present in a soluble form are more readily absorbed than oxalate present as crystals of calcium oxalate [61] or oxalic acid. In support of this observation, it

has been found that the simultaneous consumption of dairy produce along with high oxalate-containing foods may reduce their availability of oxalate for intestinal absorption. Thus, oxalate is better absorbed from black tea or coffee, especially instant coffee (all of which have a high content of oxalate [62]), when they are consumed in the absence of added milk compared with, for example, tea containing a little milk [63], presumably because the calcium in the milk precipitates the oxalate released from the tea and makes it less available for intestinal absorption. Similarly, oxalate is better absorbed from spinach consumed without the addition of any dairy produce than when served with it, such as in creamed spinach [64]. It has been calculated that, even under normal gastric conditions of pH (~2), any CaOx crystals that are formed might not all be redissolved within the period of time that they remain in the stomach [43]. If these crystals pass out of the stomach into the small intestine, they are unlikely ever to be re-dissolved under the more alkaline conditions existing in the remainder of the intestinal tract and release oxalate that might potentially be absorbed in the large bowel [43].

For the same reason, calcium from other dietary sources may exert a major effect on oxalate absorption. At high concentrations of calcium in the intestinal tract, most free oxalate will precipitate and is unlikely to be re-dissolved in the more alkaline environment of the small intestine. Based on this principle, a high calcium intake has been used to reduce the intestinal absorption and urinary excretion of oxalate in patients with enteric hyperoxaluria [65–67]. This produced a concomitant reduction in the relative supersaturation of urine with respect to calcium oxalate [46, 68, 69].

The fibre content of foods may also affect the bioavailability of oxalate for intestinal absorption [70] by a different mechanism. For example, the fibre in spinach and sugar beet may reduce the intestinal absorption of oxalate from these foods by physically “locking up” the crystals of CaOx in these products and making them less likely to be dissolved and release oxalate which may become available for absorption [71].

Oxalobacter formigenes, an anaerobe which is located in the human colon and has an obligate requirement for oxalate as a source of energy [73], may reduce the amount of soluble oxalate that is available for passive absorption by metabolising oxalate in the large intestine [72]. The hypothesis is that any reduction in the colonisation of the large bowel with *O. formigenes* will lead to an increase in the amount of soluble oxalate available for passive absorption at that site and so will inevitably increase the urinary excretion of oxalate. There is some evidence to support this in studies in stone-formers which show first, that absence of *O. formigenes* is associated with an increase in urinary oxalate [74] and, second, that stone-formers generally have lower levels of colonisation with *O. formigenes* than

non-stone-formers [75–78]. Once the level of colonisation with *O. formigenes* has dropped for whatever reason (for example, after a course of antibiotics), it is said to be difficult to recolonise the gut with the anaerobe.

It has also been claimed that urinary oxalate excretion can be reduced in humans by oral administration of a mixture of five strains of freeze-dried lactic acid bacteria, each of which has some ability to metabolise oxalate [79], but the effectiveness of this form of treatment has been challenged by others [47]. Nevertheless, the concept of destroying oxalate in the gut may have some real therapeutic value in terms of preventing the recurrence of CaOx stones if some way can eventually be found to introduce an enzyme that can metabolise oxalate under the conditions of pH and ionic environment existing in the large bowel [80].

In terms of trying to reduce the risk of stone recurrence in idiopathic calcium stone-formers, I usually advise the patients against the consumption of the high oxalate-containing foods mentioned above. The main aim of this line of approach is to bring dietary oxalate below about 1.5 mmol/day (135 mg/day) (Table 1). I also advise those patients who have been told by their family practitioner to reduce their intake of dairy produce or who have picked up this misguided advice from the Internet, to return to a normal intake of calcium. The aim is to get the oxalate/calcium molar ratio of their diets down below 0.1. If the patients are invited out for dinner and faced with a dish containing one of the high oxalate-containing foods mentioned above and do not wish to upset the person who has cooked the meal by refusing to eat it, I advise them to drink an additional large glass of water (>300 ml) during the course of the meal to dilute out any potential increase in their urinary oxalate excretion.

Controlling the urinary excretion of oxalate would undoubtedly constitute a major break-through in the prevention of recurrence of CaOx stones. To achieve this, however, a complete understanding of the various endogenous and exogenous factors that affect the urinary excretion of oxalate is necessary. This should include (a) an accurate knowledge of the dietary intake of oxalate and its bioavailability in the various parts of the intestine, (b) a full understanding of the factors that control the intestinal absorption of oxalate at various sites in the intestinal tract including the stomach [43], (c) elucidation of the dietary intake and metabolism of the immediate precursors that lead to the formation of oxalate in the body.

Calcium intake

Low calcium intake

It has been common practice, especially among family practitioners, to advise calcium stone-formers to restrict

their intake of high calcium-containing foods, such as milk, cheese, yoghurt, and other dairy products, on the principle that hypercalciuria is considered (perhaps wrongly [16, 43]) to be *the* major risk factor for calcium stone-formation. Based on this assumption, the logical conclusion is that reducing the dietary intake of calcium will reduce the urinary excretion of that mineral and with it the risk of stone recurrence. Unfortunately, although urinary calcium excretion may decrease on this regimen, there is no evidence that stone recurrence decreases in parallel [81]. The reason for this is that reducing dietary calcium appears to decrease the precipitation of oxalate in the intestine and make oxalate more freely available for passive absorption in the large bowel resulting in an increase in the urinary excretion of oxalate [6, 68, 82]. The net effect of these changes is to cause an increase in urinary supersaturation with respect to calcium oxalate [68, 82]. Epidemiological evidence in support of this possibility comes from the large study carried out by Curhan et al. [10] which showed that, as dietary calcium falls below about 720 mg/day (18 mmol/day), the prevalence of stones progressively increases. At the other end of the scale, there is little or no evidence that increasing the intake of calcium beyond 1000 mg/day (25 mmol/day) increases the risk of forming calcium-containing stones in the majority of the population. However, if the patient is a hyperabsorber of calcium *and* is consuming a high calcium intake then some limitation of dietary calcium may be considered with the reservation that if, in addition, the patient also has a renal leak of calcium, care must be taken not to put the patient into negative calcium balance by over-reducing his/her intake of that mineral. For the majority of calcium stone-formers, a low calcium diet is not recommended (Table 1).

High calcium intake

In the oil-rich Gulf States of the Middle East, the effect of a diet naturally low in calcium (on average about 50 % of that in the West) but very high in oxalate (3–4 times the average normal intake in the West) leads to a situation where the ratio of oxalate/calcium in the diet is about 5–6 times that in the West [17, 42]. The result is that hypercalciuria (>6 mmol/day) is seen only very rarely whereas mild hyperoxaluria (>0.45 mmol/day) is common in about 70–80 % of the population [17, 41, 42]. The fact that the life-time prevalence of stone-formation in men in these countries is over 20 % compared with 8–15 % in the West appears to emphasise the relative importance of mild hyperoxaluria over hypercalciuria as the main risk factor for calcium oxalate (CaOx) stone-formation [13]. Since the apparent lack of calcium in the diet seems to be one of the factors leading to the high prevalence of mild hyperoxaluria [6, 68, 82], adding supplements of 1 g (25 mmol) of

calcium per day (given as calcium citrate) reduced urinary oxalate excretions into the normal range with only a minimal increase in urinary calcium excretion [42, 68, 83]. The reason for the latter appears to be due to the low vitamin D status among Arab populations [84] who cover their skin and so are exposed to less UV light than might be anticipated from populations living in countries with wall-to-wall sunshine for much of the year. In addition, since their skin is pigmented, they tend to absorb less UV radiation than white populations. The low vitamin D status leads to a reduced ability of the intestine to absorb calcium and may explain why adding 1 g of additional calcium to the diet of the Middle Easterners had only a minor effect on the urinary excretion of calcium [42, 83]. Unfortunately, it was not possible to continue the high calcium regimen for long enough to determine whether or not this form of treatment actually reduced the recurrence rate of stone-formation in this particular population.

Based on the above study on Arab populations and taking into account the evidence from Curhan's study [10], it would be predicted that a high calcium diet might be beneficial in the treatment of calcium stone-formation in Western countries [85, 86]. However, few researchers have yet had the courage of their convictions to try this. The potential beneficial effect of a high intake of calcium on the risk of forming stones was confirmed in a study from Switzerland [87] although providing additional calcium in the form of high Ca-containing mineral water did not reduce urinary oxalate excretion in an equivalent study carried out on German stone-formers [88]. The only sub-group of calcium stone-formers in whom calcium supplements might not work are those who are hyperabsorbers of calcium, although it might be argued that any reduction in urinary oxalate that should result from such a high intake of calcium would more than offset the effect of any increase in urinary calcium on the biochemical risk of forming further stones, particularly if the patients were already hypercalciuric before treatment. The reason for this is that, paradoxically, it is the ion present in lower concentration (in this case, oxalate) that controls the risk of forming CaOx crystals and the resultant volume of CaOx crystalluria [89] and this explains the relative importance of small increases in urinary oxalate compared with large increases in urinary calcium on the risk of forming calcium oxalate stones [16]. The general aim in Western countries is to get the dietary intake of calcium of patients within the range 20–25 mmol/day (800–1000 mg/day) for normal absorbers of calcium and 18–20 mmol/day (720–800 mg/day) for hyperabsorbers of calcium.

Animal protein

There is considerable evidence that populations who consume high quantities of animal protein, particularly that

derived from meat, fish and poultry, have a much higher prevalence of stones when compared with populations whose diet is more vegetarian-based [8, 19–21, 90]. Studies on stone patients and normal control subjects show that when they are put on a high animal protein diet both groups excrete more calcium, oxalate and uric acid but less citrate and their urines become more acidic than on a normal animal protein diet [19, 21]. Basically, a high animal protein diet is highly acidogenic because of its higher acid-ash content arising from its higher sulphur content than that of protein derived from fruit and vegetables. The high acidity of animal protein causes urine to become more acidic which, in turn, leads to an increase in urinary calcium (probably caused by the acid interfering with the tubular reabsorption of calcium) and a decrease in urinary citrate (caused by the acid increasing the tubular reabsorption of citrate). Urinary oxalate increases from the possible metabolism of amino acids such as hydroxyproline, phenylalanine, tyrosine, and tryptophan. It has been suggested that some individuals, possibly as a result of polymorphism in alanine:glyoxylate transferase Pro11Leu, may have an increased ability to metabolise one or more of these amino acids to oxalate [91], thereby possibly accounting for the mixed reports on the effect of a high animal protein diet on urinary oxalate [19, 92]. Urinary uric acid increases as a result of the high purine content of meat, fish and poultry. Thus, five out of the seven urinary risk factors found to be important in the causation of calcium-containing stones [13] are adversely affected by a high animal protein diet and the biochemical risk of forming stones (P_{sf}) increases markedly [13] with respect to both calcium oxalate and uric acid [19]. Another feature of the populations in the oil-rich Gulf States of the Middle East is that they tend to consume meat or fish or poultry at three main meals per day. This gives them an even higher average intake of animal protein than in the USA and contributes to their very high life-time prevalence of stones by increasing their urinary excretions of oxalate and uric acid and decreasing their urinary pH and excretion of citrate [42]. The net effect of these changes is to increase the risk of forming both calcium oxalate and uric acid stones [42, 93].

Vegetarians, on the other hand, have lower urinary excretions of calcium, uric acid and oxalate (unless they consume high oxalate-containing foods) and pass slightly more alkaline urine than omnivores [94]. The net effect of these changes is to reduce markedly the biochemical risk of forming stones [94, 95]. As a result, they have a cross-sectional prevalence of stones which is about one third of that in the general population in the UK [96, 97].

Studies on reducing animal protein intake have shown that the abnormal urine biochemistry associated with a high animal protein diet is corrected by reducing the intake of meat, fish and poultry [98, 99] and the recurrence of stones

is decreased when compared with a control group who received no dietary advice [100]. In my experience, however, it is difficult to maintain patients on a low animal protein intake when they have been used to consuming a diet high in meat, fish and poultry. People tend to revert to what they like to eat and, since patients with urolithiasis generally feel well most of the time, it is often difficult to motivate them to adhere to a restrictive diet over a long period. My advice to the so-called “purine gluttons” is to consume meat or fish or poultry only once per day and then a helping of no more than 120–150 g (about 4–5 oz). This is equivalent to about 30–40 g of protein and added to the protein from other food sources gives a total daily protein intake of 70–80 g (Table 1). For stone disease, I draw no distinction between red meat and white meat or between meat and fish and poultry. A high consumption of any of these foods appears to increase the risk of forming stones. However, there are some, such as oily fish, anchovies, caviar and offal, that contain higher than average quantities of purine and I usually advise patients to avoid these altogether.

Phosphate supplements

There is no epidemiological evidence that a diet high in phosphate increases the risk of forming calcium-containing stones. On the contrary, phosphate supplements containing 2 g of phosphorus per day were recommended for many years as a treatment to prevent stone recurrence [101, 102] (Table 1). This was based on the principle that they reduced the urinary excretion of calcium and increased that of pyrophosphate, considered at that time to be an important protective factor against the formation of calcium-containing stones [103, 104]. However, the phosphate supplements had to be given in a neutral or mildly alkaline form since, if given in the acid form, they increase the urinary excretion of calcium through the inhibitory effect of the increased throughput of acid on the tubular reabsorption of calcium [105].

This form of preventative treatment was claimed to reduce the recurrence rate of calcium stone-formation [101, 102] but has been largely discontinued because of concerns that it might eventually lead to a small decrease in plasma ionised calcium concentration and, consequently, to stimulation of the parathyroids. It is also an unsuitable form of treatment for stone patients with any degree of renal impairment. Consequently, this form of therapy has gone out of favour in recent years.

Magnesium intake

In the risk factor model of stone-formation, a low urinary excretion of magnesium emerged as the weakest (although

still significant) of the seven urinary risk factors for calcium stone-formation [13]. It has been shown, however, that there is an inverse relationship between the incidence of stones and magnesium intake [34]. As far as the mechanism is concerned, magnesium is known to be a weak inhibitor of the crystallisation of calcium salts in urine and is known to bind oxalate to form a complex that is considerably more soluble in urine than the corresponding complex of oxalate with calcium. For these reasons, some researchers have suggested that magnesium supplements given in the form of magnesium hydroxide or magnesium citrate might be useful in preventing the recurrence of calcium-containing stones [106, 107].

The main problem with magnesium is that there is a limit as to how much can safely be prescribed in supplement form because of possible side-effects, such as gastrointestinal discomfort and diarrhoea. Some success was reported on the effect of magnesium hydroxide supplements on stone recurrence [106] but this form of treatment is rarely used nowadays. There is some evidence that magnesium in the form of potassium magnesium citrate may be more tolerable and effective in reducing the risk of forming calcium-containing stones [108].

From my own observations in the UK, urinary magnesium excretion has decreased in the general population by about 20–25 % over the past 45 years or so. This appears to be partly due to a decrease in the consumption of brown and wholemeal bread and some magnesium-containing fruits and vegetables. Magnesium deficiency is rare, however, and usually results from excessive losses through diarrhoea or chronic alcoholism or from the use of thiazides rather than from a low intake of the mineral. Humans absorb about 25–30 % of their daily intake of the magnesium so any marked reduction in intake will have a noticeable effect on its excretion in urine. It might be preferable, therefore, to increase magnesium intake naturally through the consumption of foods which have a high magnesium content. Indeed, increasing magnesium intake has been claimed to decrease the urinary excretion of oxalate and with it the risk of stone recurrence [109]. I normally advise patients to consume at least 16 mmol (approximately 380 mg) of magnesium per day. Food-stuffs with a high magnesium content include brown and wholemeal bread, bran-containing cereals, brown rice, wholemeal pasta, beans, lentils, and certain fruits and vegetables such as seeds, bananas and dried fruit and particularly those green vegetables containing chlorophyll of which magnesium is a key constituent. Unfortunately, several of the high magnesium-containing foods also contain some oxalate and a few, such as nuts and spinach, have a very high content of oxalate and, therefore, should be avoided.

Potassium intake

Potassium is known to inhibit the tubular reabsorption of citrate and thereby stimulate the urinary excretion of citrate [110]. Since citrate is an inhibitor of the crystallisation of calcium salts in urine and since it also forms a soluble complex with calcium, it is generally considered that a high urinary citrate is a protective factor against calcium stone-formation [111, 112]. It follows that hypocitraturia, one of the seven urinary risk factors identified in risk factor model [13], is a mild-to-moderate causal factor for the disorder. This is the basis of the wide use of potassium citrate supplements as a preventative treatment for calcium stone-formation [111, 112]. However, the dosage has to be high—in the order of 60 mmol/day additional potassium, equivalent to 20 mmol/day of potassium citrate (potassium citrate has three potassium ions for every citrate ion) (Table 1). This is claimed to correct the low excretion of citrate in patients with hypocitraturia, not exclusively through the 20 mmol of extra citrate ingested (which is largely metabolised) but through the effect of the markedly increased throughput of potassium on the renal excretion of citrate, as mentioned above. However, not all researchers agree with these findings [113]. Some patients respond to potassium citrate supplements by showing little or no increase in their urinary excretion of citrate. In my experience, as a result of the alkalinising effect of potassium citrate on urinary pH, some have progressed from being “pure” calcium oxalate stone-formers to form “mixed stones” containing a large proportion of calcium phosphate [114], a potential risk that has also been pointed out by Coe et al. [115]. In spite of these personal reservations about the efficacy of potassium citrate supplements, there are several reports that they do increase the urinary excretion of citrate in many patients thereby reducing the recurrence rate of stone-formation in idiopathic calcium stone-formers [111, 112]. The only problem is that many patients find potassium citrate unpleasant to take, partly because of the number of tablets to be consumed each day and partly because of its occasional side-effects. Perhaps, it would be better to increase the intake of potassium naturally through dietary means since it has been shown that there is a positive relationship between urinary citrate excretion and potassium intake [116]. High potassium-containing foods include white beans, baked potatoes (with skin), dried apricots, plain yoghurt, avocados, mushrooms, bananas, and tomatoes.

In the UK, potassium excretion has not changed much over the past 40 years or so, the average excretion being about 50–60 mmol/day. For although there has been a reduction in the consumption of potassium derived from fruit and vegetables in the population, this has largely been compensated for by the concomitant increase in the consumption of meat, fish and poultry, which also contain

appreciable quantities of potassium. Although the net effect of these changes has little or no effect on potassium excretion, the acidifying effect on urine of the transition towards a more acid-ash diet has caused a reduction in the urinary excretion of citrate through an increased tubular reabsorption of citrate. This effect of increased urinary acidity on citrate excretion is also evident in patients with so-called Metabolic Syndrome [117]. My advice to patients is to consume at least 70 mmol/day of potassium (Table 1).

My advice to both idiopathic calcium and uric acid stone-formers is to correct the imbalance between the acidifying and alkalinising factors in their diets if these are high in animal protein and low in fruit and vegetables to induce them to pass a 24-h urine with an average pH of around 6.0 and a normal urinary excretion of citrate of at least 2 mmol/day. Correction of the acidic urine in patients with Metabolic Syndrome may be more difficult to achieve by dietary means alone since the low urinary pH observed in these patients is due to a fundamental defect in the renal production of ammonia to buffer their hydrogen ion excretion, a defect that appears to be due to the type 2 diabetes component of the disorder [118, 119]. It has yet to be established whether or not supplements of potassium citrate will overcome the problem of the acidic urine found in this particular group of patients.

Fibre intake

Dietary fibre appears to have a double role as a risk factor for stone-formation. First, most dietary fibre, particularly that containing phytate, has the ability to bind calcium and, thereby, make it less available for absorption in the intestine [120]. A low fibre diet (<15 g/day), therefore, binds less calcium than a normal fibre diet (around 20 g/day), thereby making more calcium available for absorption. This may aggravate hypercalciuria in some patients, particularly those who are hyperabsorbers of calcium. A normal fibre diet may play some limited role in reducing the intestinal absorption and urinary excretion of calcium in these patients. However, there is an upper limit to this beneficial effect of dietary fibre. A diet which is very high in fibre (>30 g/day) may actually increase the risk of stones by binding water in the intestine and making it less available for absorption, this being the principle by which high-fibre laxatives work. In turn, this leads to the patient passing a lower urine volume than expected from their fluid intake, thereby increasing the risk of forming stones (personal observation). I generally advise patients to consume a diet that contains between 18 and 22 g/day of fibre (Table 1).

There is no published evidence that normalising the intake of fibre in patients with a low basal intake of that dietary constituent alone decreases the recurrence rate of stones. Theoretically, this measure might have a minor

beneficial effect through the ability of phytate to bind calcium in the intestine, thereby lowering urinary calcium excretion but it can never be a sole treatment regimen for idiopathic calcium stone-formers. However, it may be a useful adjunct to other dietary changes as long as the patient does not increase his/her intake of fibre excessively.

Salt intake

A diet that is high in salt often leads to hypercalciuria by causing a renal leak of calcium [22]. This is because sodium and calcium share a common transport mechanism in the distal tubule and, therefore, a high throughput of sodium ions through that part of the nephron interferes with the tubular reabsorption of calcium. The degree of hypercalciuria increases proportionately with the amount of sodium excreted in the urine. The body usually compensates for this loss of calcium by absorbing more calcium from the intestine or by stimulating the parathyroid glands.

However, a high throughput of sodium is not all bad in terms of stone risk since a high intake of salt is likely to stimulate the thirst response and make the patient drink more. The net effect on the overall risk of stones is difficult to predict when the diluting effect on urine of the additional fluid intake is taken into account. Nevertheless, a high salt diet is one of the scourges of modern society for many other reasons, the problem being that children may develop a taste for salt at an early age and find it difficult to reduce their intake as they mature into adulthood.

I try to persuade patients living in a temperate climate to limit their total intake of salt to no more than about 7–8 g/day (120–140 mmol/day) (Table 1). This includes all sources of salt in the diet such as salt inherent in food, salt added to pre-prepared foods or convenience foods, salty snacks, salt added during cooking and, particularly, salt added at the table. Since about 90 % of dietary sodium is absorbed through the intestine, this limitation will produce a urinary sodium of around 110–125 mmol/day. However, patients who live or work in a hot environment or who participate in strenuous physical work or exercise may require a higher intake to compensate for salt losses through sweating.

There is some evidence that reducing salt intake may have a beneficial effect on the biochemical risk of forming calcium-containing stones [121] but it is unlikely to be the sole form of stone management. However, it may be a useful adjunct to other dietary changes.

Refined sugar intake

It is well documented that a diet high in refined sugars stimulates the intestinal absorption of calcium and leads to hypercalciuria [122]. As with a high salt diet, a high

refined sugar diet is another scourge of modern society since sugar is an ingredient of many products such as confectionery, cakes, biscuits, fruit juices and fruit squashes, fizzy drinks, ice-cream, desserts, etc. Again, the problem is that children develop a “sweet tooth” at an early age and find it difficult to reduce their intake of sugary foods when they reach adulthood. The combination of a diet high in salt and high in refined sugars (part of the so-called “Bad Western Diet”—see below) often leads to gross hypercalciuria (>10 mmol/day) since the renal leak of calcium induced by the former is fed by the increased intestinal absorption of that mineral induced by the latter. I generally advise patients to limit their total intake of sugars to no more than 100 g/day (Table 1).

There is no good evidence that reducing sugar consumption per se actually reduces the recurrence rate of stones in the population although it has been recommended [123]. Again, it may be a useful adjunct to other dietary changes.

Potential renal acid load (PRAL)

In recent years attention has turned to the importance of the potential renal acid load of diets in relation to the risk of forming both CaOx- and UA-containing stones [124–126]. PRAL can be used to estimate the net acid excreted (NAE) by the patient. From this, an estimated 24-h urinary pH can be determined from previously published relationships between urinary pH and NAE [124]. The *expected* urinary pH estimated in this way can then be compared with the *actual* pH measured in a plain 24-h urine sample collected at the same time as the completion of the Diet Diary [30]. This can help with the diagnosis of acid–base disorders, such as dRTA, and renal acid buffering disorders, such as Metabolic Syndrome [125]. It can also identify those patients who had a urinary tract infection at the time of their investigations.

The “Bad Western Diet” for stone-formation

In recent years, the Western media have regularly run features about the deleterious effect of the so-called “Bad Western Diet”, which usually implies a diet high in fat, salt and refined sugars, on the general health of the population. In terms of urolithiasis, I would add to this combination of dietary excesses a high intake of meat + fish + poultry. The effect of the “Bad Western Diet” on the risk of forming both calcium oxalate and uric acid stones is summarised in Fig. 2.

- A baseline of a high animal protein diet in the population leads to hyperuricosuria, an acidic urine, mild hyperoxaluria, hypocitraturia, and hypercalciuria [19], which combine to increase the risk of forming both CaOx and UA stones.

Fig. 2 A diagram showing the essential features and effects of the so-called "Bad Western Diet" on the metabolic and urinary risk factors for the formation of calcium oxalate and uric acid stones

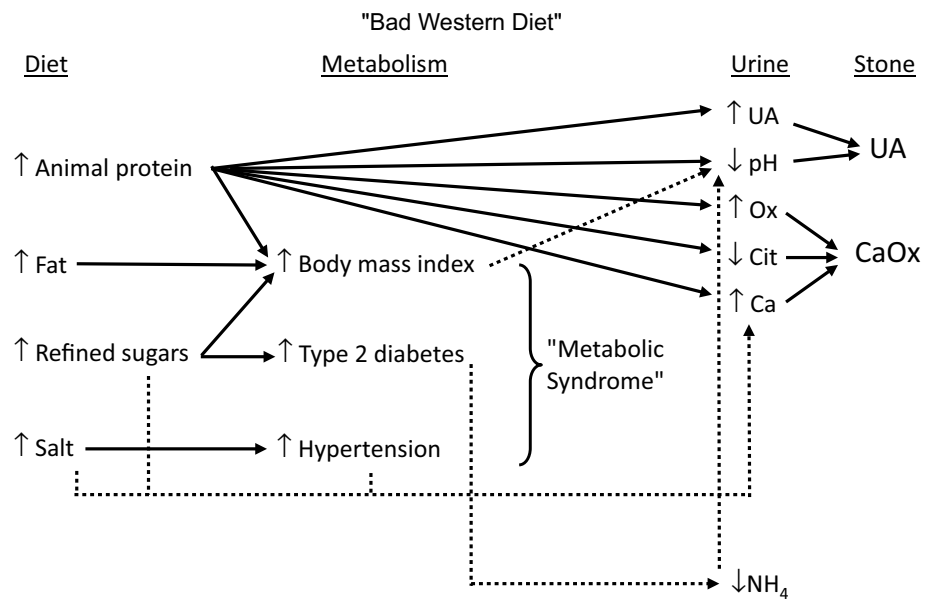
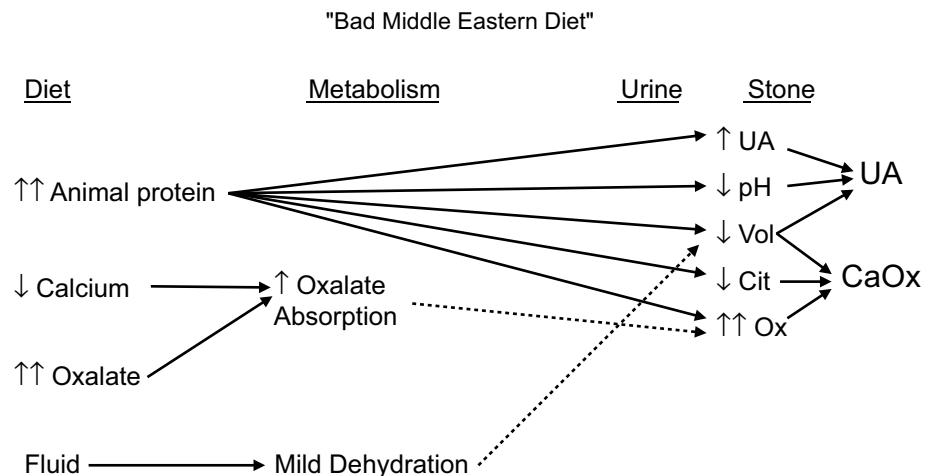


Fig. 3 A diagram showing the essential features and effects of the so-called "Bad Middle Eastern Diet" on the metabolic and urinary risk factors for the formation of calcium oxalate and uric acid stones



- The high fat diet, which usually accompanies this, tends to increase body mass index (BMI). In turn, obesity accentuates the acidity of urine and further increases the risk of forming UA-containing stones in the population.
- A high intake of refined sugars further increases BMI and with it the risk of type 2 diabetes. In turn, this causes reduced ammoniogenesis and a low urinary ammonium ion excretion and consequently leads to a more acidic urine than would be expected from the composition of the diet alone [118, 119]. The high intake of refined sugars also increases the intestinal absorption and urinary excretion of calcium. Taken together, these changes further increase the risk of forming both calcium oxalate and uric acid stones in the population.
- Finally, a high intake of salt increases the risk of hypertension. It also causes the kidneys to leak calcium and accentuate the risk of gross hypercalciuria, thereby

increasing the risk of forming CaOx-containing stones in the population.

The effect of this diet which increases BMI and the incidence of hypertension and type 2 diabetes in the population increases the incidence of "Metabolic Syndrome" in the population.

A similar set of dietary factors accounts for the very high incidence of UA- and CaOx-containing stones in the oil-rich Gulf States in the Middle East.

The "Bad Middle Eastern Diet" for stone-formation

A summary of the so-called "Bad Middle Eastern Diet" for stone-formation is shown in Fig. 3. This shares some

common factors with the “Bad Western Diet” and also leads to the formation of both CaOx- and UA-containing stones.

- The very high animal protein diet found in the Middle Eastern population leads to marked hyperuricosuria, a highly acidic urine, mild hyperoxaluria and marked hypocitraturia, which combine to increase the risk of forming both CaOx and UA stones [42]. As mentioned earlier, the incidence of hypercalciuria in the oil-rich states of the Middle East is very low because of the relatively low intake of calcium and the low prevailing level of vitamin D in the population. Even the high acid content of the local diet, which tends to cause a renal leak of calcium, is not enough to counter the effect of the low intestinal absorption of calcium observed in that population resulting from their low vitamin D status. Consequently, hypercalciuria is rarely a major risk factor for stone-formation in these countries.
- The combination of a low calcium and very high oxalate diet leads to a very high incidence of marked hyperoxaluria (0.5–0.9 mmol/day) [42] and a further substantial increase in the risk of forming CaOx-containing stones.
- Finally, the tendency to mild dehydration in the population as a consequence of the very hot, dry climate of the region reduces urine volume and further increases the risk of forming both calcium oxalate and uric acid stones.

My overall general advice to patients both in the West and the Middle East is to minimise the various excesses and deficiencies in their diets and drink more fluids, particularly in the form of water.

General advice on the dietary management of patients with urolithiasis

In summary, there are some general pieces of advice that will work for some patients with idiopathic calcium and/or uric acid stones but, in my opinion, it is better to follow advice tailored specifically to their individual metabolism and lifestyle:

- Drink sufficient fluid to produce *consistently* a urine volume of at least 2.2–2.5 l/day.
- Eliminate as far as possible the consumption of foods that have a high content of oxalate.
- Regulate your intake of meat or fish or poultry protein to only once per day and then a helping of no more than 4–5 oz (120–150 g/day).
- Increase your intake of fresh fruit and vegetables with the exception of those which have a high content of oxalate.

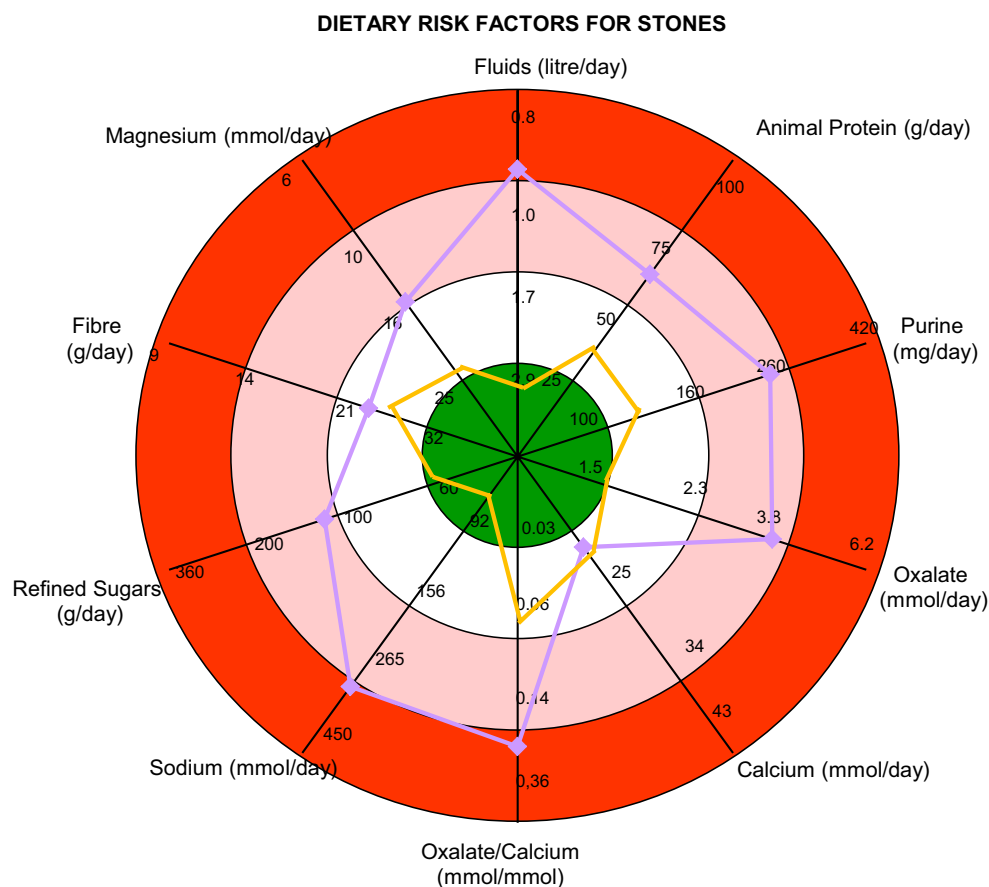
- Do *not* eliminate or markedly reduce your intake of calcium but maintain it at a reasonable level (around 20 mmol/day).
- Change your consumption of white bread to brown bread and your consumption of cereals to bran-containing products in order to correct a low fibre diet and increase your intake of magnesium BUT do not consume excessive quantities of high fibre foods unless you compensate for this by simultaneously increasing your fluid intake.
- Limit your intake of salt and salty foods.
- Limit your intake of sugar and sugar-containing foods.

It is my opinion, however, that it is more important for stone patients to be given more specific advice tailored to their particular metabolism [126], as defined by the risk factors identified by means of a detailed metabolic, nutritional and lifestyle screening procedure, rather than relying solely on a general broad instruction on how to change their diets, such as proposed above or in the DASH diet scheme [127–129] which may work for some patients but not for all. Although the detailed screening procedures, such as proposed in the AUA Guidelines [29] and in LITHOSCREEN [30] may initially appear to be costly, in the long run they will save the various health authorities money by reducing the necessity for urologists to remove or disintegrate the stones that will inevitably form in the future if the patients are not put on a prophylactic course of treatment tailored to their individual risk factors for stone-formation. Calculations have shown that for every stone episode prevented in this way, the National Health Service in the UK saves about £2000 [130] compared with leaving the patient with no personalised form of preventative treatment. In addition to the financial savings, it is clearly of considerable benefit to the patient not to have to suffer the discomfort, pain and inconvenience of experiencing further stone episodes. It also saves the National Exchequer considerable sums through unclaimed Sick Pay and industry a significant number of manpower days otherwise lost from work. Similar calculations in the USA have shown that significant cost savings can be made through the introduction of such programmes for stone prevention [131].

Motivation and compliance of stone patients with treatment regimen

One of the major problems in the prophylactic management of patients with urolithiasis, however, is the motivation to adhere to and compliance with whatever treatment they are prescribed [5, 132]. Generally, stone-formers feel well for most of the time, except when they are experiencing an attack of renal colic. It is often difficult, therefore,

Fig. 4 The target diagram showing the pattern of dietary intakes of various nutrients in a recurrent idiopathic calcium oxalate stone-former before (purple lines) and during (yellow lines) dietary management of his risk factors for stone-formation



to maintain their cooperation and motivation to adhere to their preventative treatment over a long period following their stone episode. If they do not have a recurrence of their problem within a few months of their episode, most stone-formers will regress to their original abnormal pattern of urine biochemistry within 3–6 months and will eventually produce another stone [131]. Once they have had several episodes of renal colic, however, it is often easier to motivate them on a more continuous basis. It is important, therefore, to review the patient regularly as an outpatient and to repeat at least a 24-h urine analysis, preferably annually but at least biennially, to ensure that they are adhering to the dietary regimen prescribed and to check that their biochemical risk of stones remains low. I have also found that it is helpful for patients to give them some visual objectives to aim for rather than just some numerical figure in a patient report. In the case of diet, I have devised a radar plot in the form of a target diagram as shown in Fig. 4. The plot is in the form of a series of coloured concentric rings ranging from green at the centre (the “bull’s eye”), through white, then pink, to red on the outside. The daily consumption of various nutrients are plotted along the radial axes of the diagram in such a way as to ensure that the minimum risk of stones occurs when the nutrient data points are each located within the

green “bull’s eye”. There is a slight risk of stones when the data are in the white ring, a moderate risk in the pink ring and the highest risk occurs when the data lie in the outer red ring of the target or even outside the target altogether. Similar target diagrams have been constructed for the various 24-h urinary risk factors for stone-formation and for the overall biochemical risk of forming various types of calcium- and uric acid-containing stones [133]. The target diagrams are used to show the patient his/her dietary status before treatment and how his/her observance of the prescribed treatment is progressing. The objective is to get each patient’s dietary risk factors as close as possible to the “bull’s eye”. The diagrams can be given to the patients to motivate them to adhere better to the particular form of prophylaxis prescribed and thereby increase the chances that they will experience fewer stone recurrences. Figure 4 shows an example of the target diagram for the dietary profile of a recurrent idiopathic calcium oxalate stone-former before and during dietary management to reduce his risk of forming further stones. This shows that he had several abnormal dietary risk factors on his basal free home diet with many of the data points lying in the pink and red zones of the target. After receiving nutritional advice, his dietary profile improved markedly with most of the data points lying within or close to the “bull’s eye” of

the target. He has not formed any more stones after several years on this treatment.

Compliance with ethical standards

Conflict of interest There is no conflict of interest involved in the writing of this review.

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