

Chapter 2

General Nutrition Guidelines for All Stone Formers

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There are multiple nutritional influences on kidney stone risk, regardless of stone type. These factors include energy balance as it relates to body composition, fluid intake, sodium intake, purine intake from animal flesh, and the acid load of the diet. A 24-h urine collection is used to identify metabolic and environmental risk factors. An assessment of the patient's habitual diet and dietary pattern is used to identify which of these risk factors has a nutritional contributor. When results from the urine analysis are used in combination with a thorough nutrition assessment by a registered dietitian (RD), a comprehensive and individualized nutrition care plan can be developed to address the dietary contributors to stone risk factors.

While it is recommended to tailor nutrition therapy to each patient based on his/her demonstrated risk factors, much as is done in pharmacologic therapy where the medication addresses a measured aberration, there are a few guidelines that all stone formers may follow, regardless of the type of stones they form and of their individual risk factors. This chapter provides the rationale for and strategies to implement these recommendations.

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Energy

Obesity is associated with higher risk for kidney stones through multiple mechanisms. Obese patients tend to have higher urinary excretion of oxalate and uric acid [1], and lower urinary citrate [2]. Obesity is also associated with increased sodium and phosphorus excretion, and urinary pH is inversely associated with body weight. Insulin resistance, frequently seen in obese patients, has also been associated with defects in renal ammonia production, while hyperinsulinemia is reported to increase urinary calcium excretion [1].

Because obesity plays an important role in metabolic stone disease, assessment of a patient's BMI and consideration of the effect of weight on stone risk factors is a part of the nutrition assessment. BMI is calculated either through the use of BMI charts or using the following equation: $BMI = (\text{weight in kg}) / (\text{height in m}^2)$. A patient with a BMI of 25.0 or greater is overweight, while a patient with a BMI of 30.0 or higher is classified as obese.

Overweight and obese patients should be counseled that weight loss is an important goal for reducing stone risk factors. It is helpful to provide patients with an estimate of their daily calorie needs to promote either energy balance or weight loss. There are multiple equations used to predict energy expenditure. While the Harris–Benedict equation is still used in clinical practice, recent data suggest that the most accurate equations for calculating resting metabolic rate (RMR) are the Mifflin St. Jeor and Livingston equations. These equations are seen below with W=weight (kg), H=height (cm), A=age (years) [3]. RMR is then multiplied by one of the activity factors below (Table 2.1) to determine total energy expenditure.

- *Mifflin St. Jeor*

$$\text{Women: RMR} = 10W + 6.25H - 5A - 161$$

$$\text{Men: RMR} = 10W + 6.25H - 5A + 5$$

- *Livingston*

$$\text{Women: RMR} = 248 \times W^{0.43356} - 5.09A$$

$$\text{Men: RMR} = 293 \times W^{0.4330} - 5.92A$$

TABLE 2.1. Activity factors for calculating total energy expenditure.

| Activity level | Activity factor |
|---------------------|-----------------|
| Very light activity | 1.3 |
| Light activity | 1.5 |
| Moderate activity | 1.6 |
| Heavy activity | 1.9 |

Fluids

Stone formers may have lower 24-h urine volumes than healthy controls, and increasing fluid intake in patients with a history of stones will decrease stone risk. Increasing fluid intake decreases the concentration of calcium, oxalate, phosphorus, and uric acid in the urine and decreases relative supersaturation of calcium oxalate, brushite, and uric acid [4].

Fluid needs vary between individuals. The daily fluid intake goal needs to be individualized based on a target urine output of at least 2.5 L daily. Extra-renal losses from perspiration, respiration, and stool vary considerably between individuals based on comorbidities, perspiration, occupation, climate, and activity level [5]. Provider considerations in developing individualized daily fluid intake goals include:

1. Patients with chronic loose stools or diarrhea will have increased extra-renal losses that need to be compensated for with increased fluid intake.
2. Patients with occupations or activities that require time outside in the heat with increased sweat losses will require increased fluid intake to compensate.
3. Urinary incontinence, frequent nocturia, occupations with lack of access to restroom for extended periods of time (such as truck drivers, airline pilots, and elementary school teachers) may need a personalized fluid schedule.

Patient strategies in complying with daily fluid intake goals include:

1. Divide the day into 2–3 sections and consume a target volume of fluids in each section. For example, instruct the patient to consume 1 L fluids in each of three 5-hour

- sections of the day—e.g., from 7 a.m. to 12 p.m., 12 p.m. to 5 p.m., 5 p.m. to 10 p.m.
2. Translate liters to ounces, quarts, or any other volume equivalent that makes sense to each patient.
 3. Emphasize low-calorie or no-calorie, low sugar beverages. Also encourage diversity. If patients don't like to drink water, recommend tea, sparkling flavored waters, or water with lemon or a small amount of 100 % fruit juice, or low sodium vegetable juice.
 4. Ask patients to carry a water bottle with either visible volume markings or a container of known volume, such as 1 L (approximately 32-oz.) container. The patient can then be instructed to fill and drink the contents of the bottle three or more times daily.
 5. Adjust fluid intake schedules as needed to accommodate patients' concerns. For nocturia, concentrating fluid intake earlier in the day is helpful. For urinary incontinence, higher fluid intake should be at times when the patient is at home or otherwise has access to facilities.

Sodium

The average sodium intake of Americans is estimated to be about 3,000 mg/day or about double the Dietary Reference Intake of 1,500 mg for ages 9–50. After age 50 and up to age 70, daily sodium requirement is 1,300 mg and lowers further after age 70–1,200 mg. High dietary sodium intake can increase urinary calcium excretion, thus increasing the potential for the formation of calcium-containing stones such as calcium oxalate stones. Sodium expands the extracellular volume and competes with calcium ions for reabsorption in the renal tubule. High sodium intake thus leads to increased sodium being reabsorbed in the renal tubule, leading to increased excretion of calcium [5]. High sodium intake has been shown to lead to increased risk of cystine stones and greater urinary saturation of brushite and monosodium urate. High sodium intake also has the ability to decrease urinary excretion of citrate, an inhibitor of calcium oxalate stone formation [6].

About 75 % of salt intake in the US comes from salt added during processing or manufacturing, not salt added at the table or during cooking. Foods with high sodium content include processed and packaged foods such as deli meats, frozen and canned meals, and certain condiments (e.g., soy sauce). Many people eat bread multiple times daily, and with 200–300 mg sodium per slice, bread is frequently a major source of daily sodium. In contrast, fresh meats, legumes, unprocessed whole grains, fruits, and vegetables are naturally low in sodium. The salt shaker is generally a much smaller contributor to daily sodium intake than processed foods [5]. When buying packaged foods, instruct patients to look for foods that are “sodium free” or “salt free” or “low sodium.” Purchasing “reduced sodium” options is helpful but does not guarantee that a food is actually low in sodium. Reduced sodium merely means the food has 25 % less sodium than regular version (Table 2.2).

Citrate

Urinary citrate is an inhibitor of calcium oxalate and phosphate stones through the binding of citrate to calcium to form a soluble complex, leaving calcium unavailable to bind to oxalate or phosphate. Urinary citrate is affected by intake of citrate in the diet, renal acid load, potassium intake, and the presence of frequent diarrhea.

Dietary Citrate

Urinary citrate levels can be altered by dietary intake of citrate. The richest sources of dietary citrate are citrus fruits with lemon and lime being the most concentrated sources. Other citrus fruits have considerably less citrate including oranges, and grapefruit [7,8]. Strategies for increasing dietary citrate include:

1. Drink 4 oz. lemon or lime juice diluted in water daily. (Note: patients should always be instructed to dilute the juice in order to prevent damage to tooth enamel)

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2. Reduce dietary acid load by reducing portions of meat, eggs, and fish, while increasing fruit and vegetable intake
3. Reduce dietary sodium

Renal Acid Load

High dietary acid load may impact urinary citrate levels through enhanced renal reabsorption of citrate. The Potential Renal Acid Load (PRAL) for a particular food takes into account the quantity of chloride, phosphate, sulfate (the acidifying components) and sodium, potassium, calcium, and magnesium (the alkalizing components). Foods that have a positive PRAL include meat, fish, eggs, cheese, grains, and legumes, with animal products generally having a higher dietary acid load per serving than grains or legumes. Dairy products like yogurt and milk are neutral, while fruits and vegetables are generally alkalizing to varying degrees [9].

Minimizing intake of meat and fish, and encouraging intake of fruits and vegetables to five or more servings of fruits and vegetables per day is an important way to help reduce acid load. Tips for increasing fruit and vegetable intake include:

1. Add vegetables like mushrooms, kale, or tomatoes to scrambled eggs or a frittata
2. Add a piece of fruit like berries, apple, or banana to breakfast
3. Snack on clementines or an apple with peanut butter or berries with yogurt, or raw vegetables with hummus
4. Have a big salad for lunch with high-protein vegetables like black or garbanzo beans
5. Keep carrots, celery, or snap peas on hand to add as an easy side dish
6. Reheat frozen vegetables to accompany any meal
7. Sauté mushrooms with garlic and a green vegetable like broccoli or kale
8. Blend up berries or other fruit into a smoothie for a healthy dessert

TABLE 2.2. Foods with high sodium content to limit if intake is excessive.

| | | |
|--|--|--|
| Cheese | Deli meats and cured meats and fish (such as smoked salmon or lox) | Breakfast meats like sausage and bacon |
| Frozen meals | Canned soups | Canned vegetables |
| Chips, pretzels, and other salty snacks | Fast food meals | Canned and jarred tomato sauces and salsas |
| Some breakfast cereals | Vegetable juices like V8 | Casseroles made with canned soups |
| Breads, bagels, rolls, and baked goods | Hot dogs, bratwurst, sausages, braunschweiger | Pizza and lasagna |
| Pickles and olives | Some spice blends with added salt | Some salad dressing |
| Ramen noodles and other dried noodle packets | Boxed meals like macaroni and cheese or hamburger helper, rice-a-roni type meals/sides | |

9. Eat spaghetti squash or zucchini noodles as a substitute for pasta
10. Eat butternut squash, sweet potatoes, or carrots as a starch with dinner

Potassium Intake

Urinary potassium excretion is also highly correlated with urinary citrate, and hypokalemia has been established as a cause of hypocitraturia. In hypokalemia, intracellular pH decreases, causing the secretion of hydrogen ions in the kidney resulting in hypocitraturia [10]. Potassium is widely distributed in the food supply with meat, potatoes, fruits, and vegetables all providing varying amounts. Especially good food sources of potassium are potatoes, bananas, tomatoes, oranges, and spinach. High potassium foods also tend to be foods with a lower PRAL, so these foods have the added benefit of reducing overall acid load.

Chronic Diarrhea

Chronic diarrhea can have multiple etiologies including Crohn's disease, ulcerative colitis, short bowel, celiac disease, or other etiologies. Frequent diarrhea results in increased losses of bicarbonate in the stool leading to increased renal absorption of citrate. Managing the underlying cause of the diarrhea is the best way to manage this cause of hypocitraturia. Using dietary or supplemental soluble fiber can help to bulk the stool thus preventing or slowing diarrhea. Good food sources of soluble fiber include apples, pears, bananas, oatmeal, and chia seeds. Probiotic foods and supplements may also be helpful in managing diarrhea [5].

Summary

Body weight, fluid and sodium intake, and urinary citrate levels can all contribute to stone risk in the majority of stone formers. Dietary changes can be effective ways to reduce stone risk factors such as obesity, low urine volume, hyperoxaluria, hypercalciuria, and hypocitraturia. While a thorough assessment of a patient's current diet is essential in order to provide targeted nutrition therapies, the general recommendations provided in this chapter may, without inducing any harm, benefit most all calcium and uric acid stone formers.

References

1. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA*. 2005;294(4):455–62.
2. Asplin JR. Obesity and urolithiasis. *Adv Chronic Kidney Dis*. 2009;16(1):11–20.
3. Frankenfield DC. Bias and accuracy of resting metabolic rate equations in non-obese and obese adults. *Clin Nutr*. 2013;32:976–82.
4. Borghi L, Meschi T, Amato F, Briganti A, et al. Urinary volume, water and recurrences in idiopathic calcium nephrolithiasis: a 5-year randomized prospective study. *J Urol*. 1996;155:839–43.

5. Penniston KL, Nakada SY. Diet and alternative therapies in the management of stone disease. *Urol Clin North Am*. 2013;40:31–6.
6. Sakhaee K, Harvey J, Padalino P, et al. The potential role of salt abuse on the risk for kidney stone formation. *J Urol*. 1993; 150:310–2.
7. Penniston KL, Steele TH, Nakada SY. Lemonade therapy increases urinary citrate and urine volumes in patients with recurrent calcium oxalate stone formation. *J Urol*. 2007;70(5): 856–60.
8. Seltzer MA, Low RK, McDonald M, et al. Dietary manipulation with lemonade to treat hypocitraturic calcium nephrolithiasis. *J Urol*. 1996;156:907–9.
9. Remer T. Potential renal acid load of foods and its influence on pH. *J Am Diet Assoc*. 1995;95(7):791–7.
10. Domrongkitchaiporn S, Stitchantrakul W, Kochakarn W. Causes of hypocitraturia in recurrent calcium stone formers: focusing on urinary potassium excretion. *Am J Kidney Dis*. 2006;48(4):546–54.

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