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**Review Article**

# Association between Dietary Practices and Calcium Oxalate Stone Formation in Urinary Tract among the Patients of Urolithiasis

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**Abstract:** Kidney stones are a major health problem around the globe. It occurs due to multiple reasons. In this review, the association between dietary intake and calcium oxalate stone formation in the urinary tract is studied. The relationship between calcium intake and its contribution in calcium crystals formation has been found to be inverse. Oxalate intake has a directly proportional relationship with the crystal formation in urinary tract. Moreover, the contribution of Vitamin D, sodium, protein and citrate in urolithiasis has also been studied.

**Keywords:** Urinary Tract Stones, Calcium Oxalate, Urinary Excretion.



## INTRODUCTION

Kidney stones are a multifactorial disorder and can be a result of one or combined influence of epidemiology, biochemical, genetic and dietary factors. There are four types of kidney stones.(Kasote, Jagtap, Thapa, Khyade, & Russell, 2017) Stones of calcium Oxalate are the most frequently formed kind of stones. Calcium oxalate stones are usually formed due to abnormally increased levels of calcium, oxalate, cysteine and very little water. They are formed through a mechanism of super saturation, which is the ratio of crystals and their solubility. When super saturation is below 1, which occurs when there is an imbalance between calcium, oxalate and water, it enhances the crystal formation and Promote their growth. The increased concentration of stone causes an increase in the probability of accumulation of crystals thus forming the stones.(Worcester & Coe, 2010) Calcium stones are formed due to increased absorption, reabsorption or renal leak. Recurrent calcium stone formers have unusually increased excretion of oxalate.(Robijn, Hoppe, Vervaet, D'haese, & Verhulst, 2011) Moreover, increased intake of ascorbic acid and protein also causes an increase in oxalate production. (Pearle *et al.*, 2014) The oxalate binds with free calcium forming the calcium oxalate crystals (Türk *et al.*, 2016).

According to studies, humans had always suffered from bladder and kidney stones. Back then bladder stones were the most prevalent kind of stones but in the past 100 years it has been observed that kidney stones are most prevalent ones.(López & Hoppe, 2010) In the past few decades, a trend was noticed that the occurrence of stone diseases was increasing in industrialized countries. In a village near Millan, the prevalence in the last three decades increased from 6.8 to 10.1% in men i.e. by 49% and in women it increased from 4.9 to 5.8% i.e. 18%. In US, the frequency of stones increased from 3.8% from 5.2% within 15 years. In Japan, the incidence of stones increased from 4.0 to 5.4% in 10 years. In Germany, an increase of 1.0 to 4.7% was observed (Knoll *et al.*, 2011). Pakistan might have the highest incidence rate of renal kidney stones. According to three studies published, the prevalence rate in Pakistan was 2.0% between 1991 to 2003.(Romero, Akpinar, & Assimos, 2010) According to Indus Hospital community cohort, the prevalence rate of kidney diseases in Pakistan is increasing due to various reasons which include sedentary lifestyle, unhealthy eating habits and misuse of medications. The overall prevalence rate for kidney diseases in Pakistan is 16.6% while 8% for moderate kidney diseases and 8.6% for mild kidney disorders (Alam, Amanullah, Baig-Ansari, Lotia-Farrukh, & Khan, 2014).

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The calcium absorption rate is quite low, if an adequate Intake (AI) of 1000 mg diet is taken then only 300 mg calcium is absorbed which means 70% of the calcium remains unabsorbed. Considering this absorption rate it was proposed that each serving of meal or every 100 kcal should provide 30 mg of calcium. Based on the absorption capacity of calcium, calcium sources are divided into good, potential and bad food sources. The good sources are those which have a higher tendency of getting the calcium absorbed and these include foods like dairy products including cheese, ice-creams etc., turnip greens and dark green leafy vegetables. Canned foods of Salmon and sardines containing the bones also have comparatively high amounts of calcium (Titchenal & Dobbs, 2007).

The second most important component of calcium oxalate stones is oxalate whose crystallization is mainly due to hypercalciuria, hyperoxaluria, hypocitraturia. The end product of breakdown of amino acids is oxalate. Plant sources such as spinach, berries, nuts, beets, tea, wheat bran etc. and chocolate are also potentially rich sources of oxalate. The oxalate binds with calcium forming the calcium oxalate crystals. Along with calcium and oxalate; proteins, citrate, sodium, refined sugars also contribute in the formation of calcium oxalate stones (Robertson, 2016).

Dietary practices influence the development of calcium oxalate renal stones. The relation between calcium oxalate stones and calcium intake has been found to be inverse i.e. the lower the calcium intake the greater the risk calcium crystallization. Low calcium intake increases the mechanism of calcium reabsorption from the bones thus increased urinary excretion of calcium. High intake of sodium also influences the excretion rate of calcium. Increased sodium in serum induces the reabsorption of calcium from the bones, depleting the calcium of bones meanwhile increasing the urinary excretion of calcium (SILVI SHAH & Calle, 2016).

Hypocitraturia is also a contributing factor, reduced intake of citrate promote the calcium oxalate crystallization. If adequate citrate is taken, the crystals of calcium, oxalate or CaOx will be dissolved instead of accumulating in the renal tract. Potassium citrate has been investigated to reduce the calcium excretion in urine and also normalize the pH of urine (Daudon, Bazin, & Letavernier, 2015).

The second important component of calcium oxalate stones; oxalate has a direct relation with dietary intake. Increased consumption of foods products containing oxalate and metabolizing oxalate as an end product have been found to increase the incident of calcium oxalate stones. Refined sugars and animal protein, also contribute in renal stone formation. Increased intake of animal protein has an important role in oxalate stone formation. The amino acids metabolize and produce oxalate as an end-product. Also high animal protein intake increases the acid load which creates metabolic acidosis (Abbagani, Gundimeda, Varre, Ponnala, & Mundluru, 2010).

Calcium and vitamin D supplementation do not cause or contribute in calcium stone formation. However, Vitamin C is also a determinant of calcium oxalate renal stones. Vitamin C or ascorbic acid supplementation has been found to increase the oxalate concentration and excretion as a result increasing the incidence of oxalate crystals formation. While ascorbic acid increases the risk of stones to two folds, no other multivitamin supplementation has been found to be associated with formation of kidney stones.(SILVI SHAH & Calle, 2016) The male: female ratio has been 2.7:1 implying that males are more prone to kidney stones as compared to women but the average age was 43.9 despite the gender (Daudon *et al.,* 2015).

Kidney diseases are recognized as a significant health problem throughout the world. The previous literature showed that there are multiple contributing factors in the increased prevalence of calcium oxalate stone formation in urinary tract, including; lifestyle, health status, dietary habits and factors influence overall health. This increase in renal stones incidence consequently increase the health care cost thus contributing to the economic burden. The main aim of this review is to determine the association between the dietary practices and calcium oxalate stone formation in the patients with calcium oxalate stones in urinary tract.

**Association between dietary intake and calcium oxalate stone formation**

Urolithiasis is calculi present in renal tract due to super saturation of urine and salts and minerals such as uric acid, calcium phosphate, and calcium oxalate etc. The size of the calculi or stone can be as small as a pebble also it can the size of a stag horn with branches. Renal stones are fairly common and often recurrent. Also, they are associated with diseases like hypertension, diabetes, obesity (Bangash, Shigri, Jamal, & Anwar, 2011)

**Oxalate**

Oxalate in urinary tract is considered dangerous as it can lead to the development of calcium oxalate stones and almost 75% stones in urinary system are due to calcium oxalate. Basically oxalate in urinary tract comes from the diet and from internal formulation of oxalate from ingested or metabolically generated precursors (Morgan & Pearle, 2016).

Oxalate has no functional role in human body, it is derived from food.(Morgan & Pearle, 2016) Diet contributes about 50% of excretion of urinary oxalate. There are some foods that contains high amount of oxalic acid such as spinach, wheat bran, cereals, sorrel, mangold, vegetables and rhubarb. Higher consumption

of oxalate leads to higher discharge of urinary oxalate even in individuals that are healthy without disturbing the metabolism of oxalate. Increased intestinal absorption of oxalate can leads to urinary oxalate.(Holmes, Knight, & Assimos, 2016) One way which can prevent recurrence of stones of calcium oxalate is to reduce the intake of products that contain high amount of oxalic acid or oxalate. Microbiological approach can also reduce intestinal absorption of oxalate. A bacterium that colonizes the human colon and also colons of other vertebrates and which uses oxalic acid for energy is *Oxalobacter formigenes*. Studies showed that decrease excretion of urinary oxalate is linked with enteric colonization by *O. formigenes due to oxalate degradation in gut thus absorption in intestines is reduced. Similarly when O.formigenes is absent than absorption of oxalate in intestines can be increased causing high levels of plasma oxalate and urinary oxalate thus the possibility of oxalate stones can be increased (Siener et al., 2013).*

*Study was done by Holmes et al. in which individuals were given controlled diet in oxalate. Results declared that consuming foods that have oxalate ranging from 100mg/day to 750mg per day showed 2mg rise in urinary oxalate per 100mg. Another study was done in which diet containing oxalate less than 100mg/day was given. Results indicated that a large amount of oxalate from diet was absorbed because oxalate was present in the form of soluble oxalate anion. Another study was done in which diet containing oxalate in ultra-low amounts (10mg/day). Results revealed that the amount of oxalate absorbed was 2.5mg (25%). Study on males having calcium oxalate stones previously hypercalciuria was done. Individuals were asked not to consume foods that are high in oxalate. Normal calcium intake, animal intake and low sodium was also assessed in the individuals suffering from calcium oxalate stones. Intake of oxalate rich foods was low before and during the study, 143mg before the study and 121 during the study. The results showed that the excretion of urinary oxalate (7.3mg/day) was reduced. The reduction in discharge of urinary oxalate was just due to dietary modification (Morgan & Pearle, 2016).*

*Another study showed that* intake of a lactic acid bacterial probiotic reduces excretion of oxalate in people suffering from idiopathic hyperoxaluria. *A study was done on 50,000 males to find-out the relationship between kidney stones and vitamin C consumption. In the tissues, vitamin C can be broken down to oxalate.*[16] *Men taking vitamin C less than 105mg/day have less risk of kidney stones than men taking vitamin C in amounts greater than 218mg/day have higher risk of kidney stones. Vitamin C in the form of supplementation, greater than 1000mg/day also increases the risk of kidney stones.(Ivanovski & Drüeke, 2013) Elevating magnesium intake to bind oxalate in the gut also decreases absorption of oxalate but magnesium in supplementation form has no effect in the development of urolithiasis.(Thomas, Elinder, Tiselius, Wolk, & Åkesson, 2013)* From the members of solute-linked carrier 26 (SLC26) anion-exchanging family, oxalate in dietary form is absorbed. Kidney excrete serum oxalate which is delivered from external sources and internal sources (liver and gut) via tubular secretion and glomerular filtration. High urinary concentration of both oxalate and calcium leads to formation of calcium oxalate stones in kidney. Oxalic acid is destroyed by oxalobacter formigenes thus causing excretion or secretion of oxalate in colon. For oxalate handling, different members of the SLC26 anion-exchanging family are involved, depending upon renal tubular segment and intestinal segment. GRHPR, AGT, hydroxyl-pyruvate reductase or glyoxylate reductase, glyoxylate transferase (Trinchieri, 2013).

**Calcium**

Various studies have been there which shows the result of dietary calcium on urine oxalate excretion. Along with an increased dietary consumption of calcium which can lower intestinal assimilation of oxalate. By lowering the intake of calcium or its severe limitation can cause hyperoxaluria as well as the bone mineral loss. Daily excretion of oxalate in the urine is up to 40% due to dietary sources but its immersion in the intestine depends primarily on Calcium intake in our diet. Limitation of animal protein, salts and ample intake of calcium can decrease in over-abundance of calcium oxalate stones (Thomas *et al.*, 2013).

A research was conducted in order to see the relationship of Calcium and vitamin D supplements with kidney stones in postmenopausal women and also to check all metabolic changes that are caused due to intake of supplements to determine the risk of stone formation. The study was carried out on 53 women who had osteoporosis and not receiving adequate calcium before the study. Study was continued for a whole year in which the blood tests and kidneys ultrasonography were performed as well as urine calcium levels were also measured after a whole year these tests were again carried out with the treatment of 1000 mg/day of dietary calcium and 400 IU/day vitamin D showed a weak association with kidney stones formation. The result concluded that oral intake of calcium and vitamin D does not effect on urinary calcium excretion rate and kidney stone formation in postmenopausal women (Trinchieri, 2013)

Another study was lead on men for 5 years to check hypercalciuric stones through checking their urine output and concluded that the lesser the amount of calcium in diet the lesser it is worthwhile in the deterrence of its happening again than a diet with a normal amount of calcium (Haghighi, Samimagham, & Gahardehi, 2013).

An investigation was carried out on eight healthy cats who received three canned diets of different calcium content in the 12.2 (A), 18.5 (B) and 27.0 (C) g Ca/kg dry matter. All of the following diets were fed for 17 days after seven days the blood samples of cats were collected along with urine and feces which were collected over (2x4 days). In them, urinary oxalate, urinary pH, the concentration of Parathyroid hormones (PTH), serum Calcium and Phosphate and urinary and fecal minerals were analyzed. Calcium oxalate urolithiasis were detected using a microscope. The results showed that the Calcium obtained from the diet lowers the level of PTH and Ca and enhance the faecal concentration of potassium and calcium, but it did not affect the urinary fasting pH or urinary oxalate and calcium concentration. Calcium excretion in cats is free from dietary calcium levels when they have been fed with higher calcium content containing bones (Dogliotti *et al.,* 2013).

A study was conducted on 108 patients who has idiopathic recurrent calcium oxalate stones and they were analyzed. The stones were examined using a urine analysis, x-ray diffraction test and scanning electron microscopy before the dietary intervention. They were provided with dietary interventions for the urine abnormality of a specific 24hr period. After the whole month, the 24hr urinalysis were repeated and the values was again taken to compare the before and after of taking dietary interventions. Urine samples were collected and their pH, uric acid, calcium, oxalate, magnesium, phosphorus and the total volume were analyzed. An auto-analyzer was used to measure calcium content. If the calcium excreted in the urine was more than 250mg/day then calcium ingestion was limited to 400-600mg/day.(Passlack & Zentek, 2013).

**Sodium**

Dietary sodium chloride has an association with urinary excretion of calcium. The amount of intake of sodium should be 2300mg/day (Pearle *et al.,* 2014).

A study defined the result of intake of salt on urinary calcium. A high intake of salt causes hypervolemic state by the kidney by lowering proximal tubular sodium reabsorption. The slight increase in sodium in the food can increase calciuria in a healthy individual. For example, if the salt in the diet is increased by 3.5g it increases the risk of hypercalciuria by 1.63 folds. So, it was concluded that patients with idiopathic kidney calculi show hypercalciuria with the elevation in dietary salt. In the same way if the dietary salt is increased by 5% it increases urine calcium concentration by 40mg/day (Kıraç *et al.,* 2013).

Previous study which is being performed in this regard to analyze recurrent kidney stone formers to check the effect of low sodium and low calcium diet on urinary risk profile. The patients with recurrent calcium oxalate stones are checked for the outcome of low sodium diet on total urinary volume and the impact of low sodium and low calcium diet on urinary oxalate excretion. The experiments were done on 169 patients out of whom 49 were hypercalciuria with borderline. The diet caused a notable decrease in 24hr urinary sodium and calcium excretion from 201± 89 to 128± 88mmol/day of sodium, and for calcium 5.67± 3.01 to 4.06± 2.46mmol/day approximately. Hence, proved that diet which is low in sodium and calcium in recurrent oxalate stones patients results in a notable decrease in urinary calcium excretion (Afsar *et al.,* 2016).

An investigation was carried out to access oxidative stress that is caused due to high sodium which is involved in calcium oxalate crystal formation. The experiment was conducted on male rats that were provided with 3 types of sodium diet 1. Sodium depleted diet, 2. Normal sodium diet, 3. High sodium diet. The high sodium and normal sodium diets were attained by drinking water which has 3% and 0.3% of NaCl in it. Along with it the rats were given sodium depleted diet which has a 5% hydroxyl-L-proline for 7 - 42 days to generate calcium oxalate deposition. Results showed that high sodium increased calcium excretion as well as it generates a large amount of formation of crystals in the hyperoxaluric kidney it is mainly due to loss of anti-crystallization defense and oxidative injury (Seeger *et al.,* 2017)

A random study was conducted on 30 female mice for 3 weeks to mouse chow, they were given high fructose and high sodium diet. Their urine was occasionally collected for 30 days as well as their body weight was noticed twice daily. There was no difference in body weight when the result was obtained but high sodium and fructose diet altered the development of stone in children by decreasing stone inhibitors (Huang & Ma, 2015).

A study was conducted retrospectively to analyse recurrent kidney stone formers in order to check out the effect of low calcium diet along with low sodium diet on the urinary risk profile. It was carried out on 169 patients with CaOx stones, out of which 49 were with hypercalciuria at baseline. The diet caused reduction in 24-h urinary calcium and sodium excretion e.g., from5.67 ± 3.01 to 4.06 ± 2.46 mmol/d for calcium and for sodium 201 ± 89 at baseline to 128 ± 88mmol/d. Whereas, the urine volume remained unchanged. Hence a diet low in calcium and sodium cause notable reduction of urinary calcium excretion, but it does not affect urine volume. (Seeger *et al.,* 2017)

**Protein**

According to AUA guidelines, patients with calcium oxalate stones should limit their non-dairy protein intake.(Pearle *et al.,* 2014) To study the effect of protein intake, observational studies were conducted. A few studies showed that protein is a contributing factor while other did not. Several studies showed that

high protein can increase the risk of calcium oxalate incidence as an effect of increased purines, urine acidification and low pH. Increased protein intake results in an increased catabolism of endogenous sulfuric acid from methionine and cysteine, which cannot be metabolized. The dietary acidic load of protein results in increased calcium excretion which when viewed from pathophysiological aspect indicated that increased calcium excretion leads to increased calcium salt crystallization (Ross *et al.,* 2013).

A research was conducted to study the prospective association between dietary intake of different types of proteins and their possible contribution in calcium oxalate through three cohort studies with large sample size. In these three studies, HPFS established in 1986 had 51,529 participants who were men aged between 40 to 75 years. NHS I established in 1976 had an enrollment of 121,700 women aged between 30 to 55 women and NHS II was established in 1989 with 116,430 participants who were women 25 to 42 years old. In all these studies, participants were asked to fill a questionnaire which contained all the information about their diet, medical history. Subsequently updated questionnaire were provided throughout the research. The results showed that while vegetable protein and dairy protein did not contribute in the calcium crystallization, animal source nondairy protein was associated with an increased risk of calcium crystallization leading to eventual stone formation (Marckmann, Osther, Pedersen, & Jespersen, 2015).

A study was conducted to explore the association between renal acid load produced by animal protein and its contribution in increased calcium excretion and predisposing the risk of stone formation. In this study, 157 recurrent stone formers were recruited and 144 Italians were enrolled as the control group. Patients who were suffering from any other disease along with nephrolithiasis were excluded. Control groups were supposed to be completely healthy with normal serum excretions of creatinine and calcium. Nutritional analysis of both the groups were performed through three day food intake diary. The nutritional content of every food was determined through a software called Dietosystem to estimate the dietary intake of purines and sulphuric acid producing components. Also, potential renal acid load was calculated through the software using the previously validated methods.

**PRAL** = 0.49 x dietary proteins (g/day) + 0.0366 x dietary phosphate (mg/day) – 0.021 x dietary potassium (mg/day) – 0.026 x dietary magnesium (mg/day) – 0.013 x dietary calcium (mg/day)

After regular urine and blood concentration tests and statistical analysis of data it was concluded that PRAL and animal protein intake was not correlated with calcium excretion in a direct relation but it has a negative correlation with citrate excretion. Hypocitraturia was observed in stone formers due to high PRAL levels which meant decreased citrate excretion, influenced by high acid load produced by animal protein intake, increases the incidence of calcium crystallization (Ferraro, Mandel, Curhan, Gambaro, & Taylor, 2016).

Another research was carried out aimed to study the association of dietary intake of women and recurrent idiopathic calcium stones. 143 females were enrolled with recurrent calcium stones. They did not have any significant comorbidity or a drug therapy that would interfere with the risk of stone formation. A control group of 170 women was also enrolled who had volunteered. All the women in both the groups were asked to fill a non-consecutive three day food intake diary. The foods were then analyzed by a dietician and nutrient composition was analyzed through Dietosystem. The components which were particularly known to be involved in the incidence of nephrolithiasis were especially focused. A food frequency questionnaire for last 2 months was asked to fill to further analyze the dietary habits. The stone former group was divided into three groups based on their age. It was concluded that women who were developing stones before the age of 30 had a higher intake of meat and the dietary habits of ICN formers and control group differed (Vezzoli *et al.,* 2015).

A study conducted to evaluate the American dietary trends in the past 40 years using National Health and Nutrition Examination Survey reported rates to compare the United States Department of Agriculture's food distribution data with the rates for stone disease during the same period. After analyzing the data it was established that increased protein consumption has led to increased incidence of calcium nephrolithiasis. Several controlled studies showed that limiting protein significantly decreased the occurrence of kidney stones.(Meschi *et al.,* 2012) A population based case control study was carried out in Italy. Dietary habits of 123 stone formers were compared with 123 individuals of control group. Only patients who had calcium stones were included and any other stone composition mixed with calcium oxalate were excluded. 24-Hr dietary recall was used along with an interview conducted to ask the frequency of particular food groups to analyze the dietary intake and calculate PRAL. The data demonstrated that in recurrent stone formers the PRAL was comparatively higher which meant meat, eggs and cheese contribute in increasing the acid load and eventually increasing the risk of calcium oxalate stone formation (De, Liu, & Monga, 2014)

An animal study was carried out to observe the relevance of high dietary protein intake and risk of calcium oxalate urolithiasis. After approval from Animal Welfare Committee, eight adult cats were

included in the study. They were given four same experimental diets. First three diets had high protein quality and low but different crude protein content while the fourth diet had a comparable crude protein but low quality protein. After analyzing the ingredients and quality of experimental diets, renal and faecal excretion of cats it was concluded that, renal calcium and renal oxalate excretion increased due to high protein intake. However, when the influence of quality was studied, it was observed that oxalate excretion was lowest in high quality protein intake. (Trinchieri, Maletta, Lizzano, & Marchesotti, 2013).

Another study was done to check weather animal protein intake cause acid load by influencing calcium and citrate excretion. In this study, 144 controls and157 consecutive calcium stone former participants were involved. Software was used in order to check the nutrient intake from a three day food intake diary it also estimated potential renal acid load (PRAL, mEq/day). The results showed that 35% of stones were hypercalciuric (n=55). Their phosphate and sodium excretion was higher in normocalciuric stone formers (Vezzoli *et al.,* 2015).

Several studies indicate that high intake of animal protein can increase the risk of calcium crystallization and subsequent stone formation, meanwhile, studies have also referred that increased intake of vegetable or plant sourced proteins can reduce the incidence of stone formation in renal tract. The vegetarian diet provides urinary alkalization thus reducing the acidification produced by sulphuric acid during the metabolism of proteins nitrogenous content. This alkalization reduces the calcium excretion and eventually reducing the risk of calcium crystallization (Paßlack, Burmeier, Brenten, Neumann, & Zentek, 2014).

**Citrate**

A study was conducted on 477 participants who have higher prevalence of calcium oxalate and calcium phosphate stones. They were given salt of citrate orally for six months which significantly reduced the stone size in them. Hence, conclusion was made that the citrate salt prevents the stone formation along with further growth of stones in participants (Phillips *et al.*, 2015).

Another study was carried out whose aim was to identify potential COM crystal dissolution compounds. Deionized water was used to treat COM crystals. The activities of these crystals were evaluated by semi- quantitative analysis of crystal size, phase contrast, number and total mass, and spectrophotometric oxalate-dissolution assay. The results declared that citrate had remarkable effect on COM crystals as demonstrated by crystals mass, size and number. In addition to this citrate can separate 85% of COM crystals from renal tubular cell surface (Chutipongtanate, Chaiyarit, & Thongboonkerd, 2012).

## CONCLUSION

Different studies conducted to investigate the association between dietary intake and calcium oxalate urinary tract stones formation in urolithiasis patients. Numerous studies results showed that reducing dietary intake of oxalate can significantly reduce urinary excretion of oxalate. While calcium has been found to have an inverse relationship with dietary intake and urinary excretion. Reduced amount of dietary calcium intake can increase the chances of calcium binding with oxalate and forming crystals in urinary tract. Studies also showed that low sodium intake helped reducing urinary excretion of sodium. Animal sources of proteins have also been found to significantly contribute in calcium oxalate crystallization. Moreover, increased intake of citrine foods reduced the super saturation of calcium oxalate crystallization. Hence, making a few changes in dietary intake can reduce the chances of recurring calcium oxalate stones in the patients of urolithiasis.

## REFERENCES

1. Abbagani, S., Gundimeda, S. D., Varre, S., Ponnala, D., & Mundluru, H. P. (2010). Kidney stone disease: etiology and evaluation. *International journal of applied biology and pharmaceutical technology*, *1*(1), 175-182.
2. Afsar, B., Kiremit, M. C., Sag, A. A., Tarim, K., Acar, O., Esen, T., . . . Kanbay, M. (2016). The role of sodium intake in nephrolithiasis: epidemiology, pathogenesis, and future directions. *European journal of internal medicine*, *35*, 16-19.
3. Alam, A., Amanullah, F., Baig-Ansari, N., Lotia-Farrukh, I., & Khan, F. S. (2014). Prevalence and risk factors of kidney disease in urban Karachi: baseline findings from a community cohort study. *BMC research notes*, *7*(1), 179.
4. Bangash, K., Shigri, F., Jamal, A., & Anwar, K. (2011). Spectrum of renal stones composition; chemical analysis of renal stones. *International Journal of Pathology*, *9*(2), 63-66.
5. Chutipongtanate, S., Chaiyarit, S., & Thongboonkerd, V. (2012). Citrate, not phosphate, can dissolve calcium oxalate monohydrate crystals and detach these crystals from renal tubular cells. *European journal of pharmacology*, *689*(1-3), 219-225.
6. Daudon, M., Bazin, D., & Letavernier, E. (2015). Randall's plaque as the origin of calcium oxalate kidney stones. *Urolithiasis*, *43*(1), 5-11.
7. De, S. K., Liu, X., & Monga, M. (2014). Changing trends in the American diet and the rising prevalence of kidney stones. *Urology*, *84*(5), 1030-1033.
8. Dogliotti, E., Vezzoli, G., Nouvenne, A., Meschi, T., Terranegra, A., Mingione, A., . . . Soldati, L.

(2013). Nutrition in calcium nephrolithiasis. Journal of translational medicine, 11(1), 109.
9. Ferraro, P. M., Mandel, E. I., Curhan, G. C., Gambaro, G., & Taylor, E. N. (2016). Dietary Protein and Potassium, Diet–Dependent Net Acid Load, and Risk of Incident Kidney Stones. *Clinical Journal of the American Society of Nephrology*, *11*(10), 1834-1844.
10. Haghighi, A., Samimagham, H., & Gahardehi, G. (2013). Calcium and vitamin D supplementation and risk of kidney stone formation in postmenopausal women. *Iranian journal of kidney diseases*, *7*(3), 210-213.
11. Holmes, R. P., Knight, J., & Assimos, D. G. (2016). Lowering urinary oxalate excretion to decrease calcium oxalate stone disease. *Urolithiasis*, *44*(1), 27-32.
12. Huang, H.-S., & Ma, M.-C. (2015). High sodium-induced oxidative stress and poor anticrystallization defense aggravate calcium oxalate crystal formation in rat hyperoxaluric kidneys. *PloS one*, *10*(8), e0134764.
13. Ivanovski, O., & Drüeke, T. B. (2013). A new era in the treatment of calcium oxalate stones? *Kidney international*, *83*(6), 998-1000.
14. Kasote, D. M., Jagtap, S. D., Thapa, D., Khyade, M. S., & Russell, W. R. (2017). Herbal remedies for urinary stones used in India and China: a review. *Journal of Ethnopharmacology*, *203*, 55-68.
15. Kıraç, M., Küpeli, B., İrkilata, L., Gülbahar, Ö., Aksakal, N., Karaoğlan, Ü., & Bozkırlı, İ. (2013). Effects of dietary interventions on 24-hour urine parameters in patients with idiopathic recurrent calcium oxalate stones. *The Kaohsiung journal of medical sciences*, *29*(2), 88-92.
16. Knoll, T., Schubert, A. B., Fahlenkamp, D., Leusmann, D. B., Wendt-Nordahl, G., & Schubert, G. (2011). Urolithiasis through the ages: data on more than 200,000 urinary stone analyses. *The Journal of urology*, *185*(4), 1304-1311.
17. López, M., & Hoppe, B. (2010). History, epidemiology and regional diversities of urolithiasis. *Pediatric nephrology*, *25*(1), 49.
18. Marckmann, P., Osther, P., Pedersen, A. N., & Jespersen, B. (2015). High-protein diets and renal health. *Journal of Renal Nutrition*, *25*(1), 1-5.
19. Meschi, T., Nouvenne, A., Ticinesi, A., Prati, B., Guerra, A., Allegri, F., . . . Gambaro, G. (2012). Dietary habits in women with recurrent idiopathic calcium nephrolithiasis. *Journal of translational medicine*, *10*(1), 63.
20. Morgan, M. S., & Pearle, M. S. (2016). Medical management of renal stones. Bmj, 352, i52.
21. Paßlack, N., Burmeier, H., Brenten, T., Neumann, K., & Zentek, J. (2014). Relevance of dietary protein concentration and quality as risk factors for the formation of calcium oxalate stones in cats. *Journal of nutritional science*, *3*.
22. Passlack, N., & Zentek, J. (2013). Urinary calcium and oxalate excretion in healthy adult cats are not affected by increasing dietary levels of bone meal in a canned diet. *PloS one*, *8*(8), e70530.
23. Pearle, M. S., Goldfarb, D. S., Assimos, D. G., Curhan, G., Denu-Ciocca, C. J., Matlaga, B. R., . . . Turk, T. M. (2014). Medical management of kidney stones: AUA guideline. *The Journal of urology*, *192*(2), 316-324.
24. Phillips, R., Hanchanale, V. S., Myatt, A., Somani, B., Nabi, G., & Biyani, C. S. (2015). Citrate salts for preventing and treating calcium containing kidney stones in adults. *Cochrane Database of Systematic Reviews*(10).
25. Robertson, W. (2016). Dietary recommendations and treatment of patients with recurrent idiopathic calcium stone disease. *Urolithiasis*, *44*(1), 9-26.
26. Robijn, S., Hoppe, B., Vervaet, B. A., D'haese, P. C., & Verhulst, A. (2011). Hyperoxaluria: a gut–kidney axis? *Kidney international*, *80*(11), 1146-1158.
27. Romero, V., Akpinar, H., & Assimos, D. G. (2010). Kidney stones: a global picture of prevalence, incidence, and associated risk factors. *Reviews in urology*, *12*(2-3), e86.
28. Ross, S. S., Masko, E. M., Abern, M. R., Allott, E. H., Routh, J. C., Wiener, J. S., . . . Lipkin, M. E. (2013). The effect of dietary sodium and fructose intake on urine and serum parameters of stone formation in a pediatric mouse model: a pilot study. *The Journal of urology*, *190*(4S), 1484-1489.
29. Seeger, H., Kaelin, A., Ferraro, P. M., Weber, D., Jaeger, P., Ambuehl, P., . . . Mohebbi, N. (2017). Changes in urinary risk profile after short-term low sodium and low calcium diet in recurrent Swiss kidney stone formers. *BMC nephrology*, *18*(1), 349.
30. Siener, R., Bangen, U., Sidhu, H., Hönow, R., Von Unruh, G., & Hesse, A. (2013). The role of Oxalobacter formigenes colonization in calcium oxalate stone disease. *Kidney international*, *83*(6), 1144-1149.
31. SILVI SHAH, M., & Calle, J. C. (2016). Dietary and medical management of recurrent nephrolithiasis. *Cleveland Clinic Journal of Medicine*, *83*(6), 463.
32. Thomas, L. D., Elinder, C.-G., Tiselius, H.-G., Wolk, A., & Åkesson, A. (2013). Dietary cadmium exposure and kidney stone incidence: a population-based prospective cohort study of men & women. *Environment international*, *59*, 148-151.
33. Titchenal, C. A., & Dobbs, J. (2007). A system to assess the quality of food sources of calcium. *Journal of Food Composition and Analysis*, *20*(8), 717-724.
34. Trinchieri, A. (2013). Diet and renal stone formation. *Minerva medica*, *104*(1), 41.
35. Trinchieri, A., Maletta, A., Lizzano, R., & Marchesotti, F. (2013). Potential renal acid load and the risk of renal stone formation in a case–

control study. *European journal of clinical nutrition*, *67*(10), 1077.
36. Türk, C., Petřík, A., Sarica, K., Seitz, C., Skolarikos, A., Straub, M., & Knoll, T. (2016). EAU guidelines on interventional treatment for urolithiasis. *European urology*, *69*(3), 475-482.
37. Vezzoli, G., Dogliotti, E., Terranegra, A., Arcidiacono, T., Macrina, L., Tavecchia, M., . . . Nouvenne, A. (2015). Dietary style and acid load in an Italian population of calcium kidney stone formers. Nutrition, *Metabolism and Cardiovascular Diseases*, *25*(6), 588-593.
38. Worcester, E. M., & Coe, F. L. (2010). Calcium kidney stones. *New England Journal of Medicine*, *363*(10), 954-963.