REVIEW OF LITERATURE
CHAPTER - 2

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1. Blacklock, (1981) reported that Calcium Oxalate stone formation is rare in primitive societies suggesting that this condition is preventable. People who have formed a Calcium Oxalate stone are at high risk of forming another stone.

2. Robertson, (1980) reported that drinking water increases the volume of urine. In the process substances which form kidney stones are dilated, reducing the risk that they will form into a stone. For this reason, people with a history of kidney stones should drink a plenty of water often two quarts /day. It's particularly important for people in hot climates to increase their fluid intake, to help prevent stones.

3. Shuster et al. (1992) views that increases in fluid is helpful. Some research has linked soft drinks (especially cola beverages) that contain phosphoric acid with a higher risk of stone formation.

4. Shah, (1980) reported that bran, a type of fibre, reduces the absorption of Calcium which in turn causes urinary Calcium to fall.

5. Brinkley et al. (1981-93) views that many food contains Oxalate, but only a few spinach, rhubarb, beetgreens, nuts, chocolates, tea, bran, almonds, peanuts and strawberries appear to increase urinary Oxalate significantly.

6. Robertson and Peacock, (1982) - views that vegetarians are
at lower risk of forming stones.

7. Coe, et al. (1992) - views that less than half of the patients who form Calcium stones have measurable hypercalciuria. In these there is increasing evidence that a low urinary citrate output may occur, particularly in women.

8. Johansson, (1982-88)-reported that both magnesium and Vitamin B₆ are used by the body to convert Oxalate into other substances. A lack of either nutrients can increase urinary oxalate and increase the risk of forming a stone.

9. Sharman, (1992) - reported that hypercalciuria is the excretion of >7.5mmol/day in women and 10mmol/day in men, or >0.1mmol/kg/day of Calcium in either sex when the patient is screened on a defined 50-mmol/day Calcium intake.

10. McCance et al. (1942) - reported that first demonstrated the importance of protein in facilitating calcium absorption. The probable explanation is that amino acids liberated in the course of protein digestion form very soluble calcium salts which are easily absorbed.

11. McCance & Widdowson (1942) - published the first human experiments on the dietary influence of phytic acid, by feeding volunteers on breads of varying composition & comparing the calcium content of the diet, with the amount excreted in the urine & faeces, they arrived at the following conclusions:
   (i) Calcium is much less freely absorbed from diet consisting largely of brown bread than from those consisting largely of white.
   (ii) The amount of calcium absorbed from diet containing
brown bread could be raised by adding calcium to the bread.

(iii) The absorption of calcium from white bread could be prevented by adding Sodium Phytate.

(iv) The absorption of calcium from brown bread diet could be improved by removing phytic acid from the bread.

12. Dans et. al (1970) reported that the urine normally contains 100 to 350 mg Ca/day. The amount varies greatly from person to person & according to (Nordin Personal Communication), in women, the fasting overnight level of calcium excretion increases after the menopause.

13. Consolazio et. al (1962) suggests that men working in extreme heat may lose over 100 mg / hour of Calcium in the sweat.

14. Whedon (1964) reported under extreme heat conditions the sweat may contribute 30% of the total calcium output. Normally this loss is about 15 mg/ day & insignificant.

15. Nicholls & Nimalasuriva (1939) Walker & Arvidsson (1954), Mysore, Murthy et.al (1955) & Peru Hegsted et.al (1952) - showed that growing ceylonese children often maintained a positive calcium balance on intakes of about 200 mg Ca/day. Their observations have since been amply supported by observations made on citizens.

16. Malm (1958) - has made a most thorough study of the adaptation of 26 Norwegian prisoners to diets low in calcium. The subjects were healthy men aged 20 to 69 years, some of whom were investigated continuously for a year.
or longer, 4 illustrates a metabolic study of a man in whom, after several months on a low calcium diet, the faecal loss was so reduced that he was virtually in balance. "Of the 26 subjects, 22 adapted satisfactorily, 10 rapidly & 12 slowly as in the example. One man made a slight adaptation, but in 3 there was no adaptation. These studies have been cited as examples of 'adaptation' to a low calcium intake. Adults who have grown accustomed over a long period to a calcium intake greatly in excess of their true needs, may no longer absorb enough calcium to keep themselves in equilibrium when their intake is suddenly reduced under the conditions of a short term experiment.

16. Dowdle et al. (1960) - Views that vitamin D certainly promotes calcium absorption from the gut. It is possible that the parathyroid hormone also assists the active transport of calcium across the gut wall.

17. Galante et al. (1968) - Showed another hormone which rapidly lowers an artificially raised plasma calcium is produced by the parafollicular or C cells present in the thyroid, parathyroid & thymus glands, & is known as Calcitonin.

18. Catt (1970) - reported that preparations of this hormone can lower the serum calcium in parathyroid in man. This is probably due to a direct effect on bone. Calcitonin has an important role in maintaining the normal plasma concentrations. A useful review of how hormone may control calcium homeostasis.

19. Lightwood (1952) - St. Mary Hospital London, reported that idiopathic hypercalcemia of infants was first described in many
parts of Britain.

20. **Forfar & Tompsett (1959) & Mitchell (1967)** - Hypercalcemia usually starts between the ages of 5 to 8 months. The infants affected suffer from loss of appetite, vomiting, wasting, constipation, flabby muscles & a characteristic facial appearance. The concentration of ionised calcium in the blood is raised, as may be the blood urea, plasma cholesterol and blood pressure. Abnormal calcification may be present in the heart and kidneys.

21. **West et al. (1971)** - Hypercalcemia may also occur in adults as a result of hyperparathyroidism or through excessive intake or hypersensitivity to vitamin D or from prolonged excessive intake of milk and absorbable alkali for the treatment of peptic ulcer by patients with coexisting renal disease, the milk alkali syndrome. It can be reduced by treatment with porcine calcitonin.

22. **Peacock et al. (1968)** - Reported that idiopathic hypercalciuria is a condition usually associated with increased alimentary absorption of calcium.

23. **Dent et al. (1964)** - Suggest that hypercalciuria may be reduced by limiting the calcium intake by restricting the intake of milk & cheese and by giving 14 g of sodium phytate daily or 15 g of cellulose phosphate daily. Both of these substances increase faecal calcium & may thereby reduce urinary calcium. Bendrofluazide in a dose of 5 mg/day reduces urinary calcium by about 30%. As calcium salts are more soluble in acid urine, the urine should be kept acid if necessary by drugs.
24. Zarembski and Hodgkinson (1962) - Reported that usually 80 to 90 % of the oxalate in the diet is not absorbed, but passed in the faeces as insoluble calcium salts. Typical British diets contain from 70 to 150 mg of oxalate; over half of this is provided by 5 cups of tea. Rhubarb & spinach contain large quantities 250 - 800 mg/100 g. Beetroot & parsley contain 100 -200 mg/100g & most beans about 25 mg /100 g & most beans about 25 mg /100 g & other vegetables, fruits, meat & fish contains less than 5 g /100 g. Bread & other cereal products have a content of 5 - 20 mg /100g.

25. Crawhall et.al (1959) - Showed that if patient has passed a stone which is mostly oxalate, & if he continues to pass gravel, or urine with large number of oxalate crystals, & if pain & dysuria persist, it is wise to eliminate rhubarb & spinach from the diet & to restrict greatly tea & coffee drinking. Restriction of other foods containing oxalates is not justified by a process of trial & error. Patients with oxaluria have been treated with folic acid 5 mg & pyridoxine 10 mg in the hope of diverting glycine metabolism towards Serine & so away from oxalate.” No dietary restrictions are necessary for people who had an oxalate stone removed & are then free of symptoms, & whose urine contains only a few oxalate crystals. Large quantities of fluids should be drunk.

26. McCarrison et al (1942) - Produced both renal & vesical calculi in rats by feeding diets low in vitamin A & rich in calcium. These diets had little resemblance to human diets, & so his results must be interpreted with caution.
27. **Sinclair (1953)** - Reported that there is no conclusive evidence that dietary factors play any part in the aetiology of renal calculi. The diet may well be in some way responsible for vesical calculi, but these do not arise as a result of a simple deficiency of any known nutrient.

28. **Parfitt et al. (1964)** - Predisposes to the formation of calculi. An excretion of above 300 mg/24 hours in men and 250 mg/24 hours in women has been termed 'hypercalciuria'. This is present in about 8% of the healthy population in whom balance studies shows low faecal outputs of calcium. Hypercalciuria also occurs in hyperparathyroidism, after prolonged use of corticosteroids in renal tubular acidosis, sarcoidosis, malignant disease of bone & in other disease. "In all of these conditions stones may be formed". However in the great majority of cases of renal calculi, no known cause can be found. Of the known cause hyperparathyroidism is the most common.

29. **Archer et al.(1957)** - reported that primary hyperoxaluria is due to an excessive formation of oxalate in the tissues, probably by conversion from glycine. There is no evidence either of excessive absorption of oxalate from the alimentary tract or a failure of absorption by the renal tubules. The increased oxalate precipitates out in the urine & forms stones.

30. **Halstead & Valyasevi (1967)** - Contain important epidemiological studies of bladder stones in different regions in Thailand & interesting accounts to Thai life and culture, pertinent to dietary habits, nutritional intakes, feeding practices & urinary constituents. Marked differences were found
between the diets consumed by 16 farm families resident in a small village in which bladder stone was epidemic compared to that consumed by 15 families, predominantly shopkeepers & govt. workers in Ubol,( a town of 27,000 inhabitants in which the incidence of stone was 14 - fold lower ). The town families consumed ordinary rice predominantly, while the villagers ate glutinous rice. Village diet was monotonous and consisted of rice, vegetables, uncooked 'fermented fish' with infrequent supplings of fruits and animal protein, while the town families ate rice with cooked fermented fish, a great variety of fruits and other animal protein prepared in a variety of ways.

The per capita energy intake of the two groups was similar. The calcium and phosphate intakes were lower in the village diets than in the town diets. The excretion of calcium magnesium, oxalate and uric acid was consistently higher and the excretion of phosphate and sulphate strikingly lower in the villages, where bladder stones was epidemic, than in the town of Ubol.

These reports strongly support the view that "the high incidence of bladder stones in parts of Thailand is mainly due to dietetic causes". It is said to report that the prevention of bladder stones in Thai children still awaits solutions and we hope that this failure will stimulate and not discourage further investigations.

31. Robertson et al.(1969) - reported that crystals of calcium oxalate are present in all urines and are probably the result of precipitation from a supersaturated solutions of cations and anions. The crystals are small and normally washed out when
the urine is voided. Various factors in the urine may inhibit precipitation and prevent the rapid growth of crystals. Citric acid is present in molar concentrations comparable to that of Calcium. As it binds Calcium ions to form a soluble complex, citrate might allow more free Calcium ions to pass into solution.

32. **Hodgkinson (1962)** - has measured citrate excretion in healthy persons and in those with renal calculus. Low rates of excretion were found in 19% of those with calculus, but were always associated with poor renal function. Measures to increase citrate excretion are unlikely to be of use in preventing the recurrence of renal calculus.

33. **Dent and Sutor (1971)** - reported that crystals of Calcium Oxalate grow abnormally fast in the urines of patients with recurrent renal stone formation. This was found to be due to the urine containing reduced amounts of an inhibitor present in all urines.

34. **DeLuca (1969), & Froser & Kodicek (1970)** - reported that Vitamin D is necessary for the formation of normal bone. Its main site of action is in the small intestine where it promotes the absorption of Calcium and Phosphorus from the gut, but it also has a direct action of bone, kidneys and perhaps other tissues. These actions depend on the conversion of cholecalciferol in the body into two more active hormones.

35. **Lawson et al (1971)** - Suggested that first cholecalciferol is carried in the blood bound to an alpha-globulin to the liver. There it is either stored or converted into 25-hydroxycholecalciferol
(25-1,25 dihydroxycholecalciferol (1,25-DCC). This is secreted by the kidney into the blood stream and is probably the hormone which acts on the target tissues.

36. DeLuca (1969) - reported that, in the small intestine 1,25 DCC enters the epithelial cells where it unmasks in the nuclei, a specific DNA, so initiating the synthesis in the cytoplasm of a specific Calcium binding protein; this serves for the active transport of calcium from the brush border facing the lumen of the gut across the cell and hence into the circulation. The resulting raised concentration of plasma Calcium promotes bone deposition. This is regulated by Calcitonin and Parathyroid hormone, but the vitamin probably has a direct local action, perhaps by initiating a cellular transport system for Calcium.

37. Hartles (1970) - reported that Vitamin D has also a direct action on the kidneys where it promotes tubular absorption of phosphate. In men an early feature of rickets is increased urinary excretion of phosphate with a fall in plasma phosphate to a point which may interfere with mineralisation of bone.

38. Halstead (1981) - reported that epidemiological evidence suggests that a single-cereal diet (wheat, rice or millet) may be a significant causative factor of epidemic bladder calculi. This theory may explain the 'So called bladder stone belt' extending from the middle east across India and the far east from Thailand to Indonesia. Maize, the staple food in much of Africa, does not recommend reducing dietary calcium intake in patients with recurrent calcium-rich kidney stones. However, a low intake of dietary calcium can lead to a negative calcium
balancthat would likely increase the risk of osteoporosis in the long run. (seem HCC). This is carried to the kidney where it is further oxygenated to asociated with bladder stones.

39. Curhan GC. et al.,(2004) - fortunately, now suggests that dietary calcium does not promote kidney stones, but those consuming more foods rich in calcium and potassium,and drinking lots of fluids,have already been shown to have a lower risk of forming kidney stones. The most recent study, published in the April 2004 issue of the Archives of Internal Medicine, looked at the effect of diet on kidney stone formation in younger women. More than 96,000 women in the Nurses' Health Study aged 27 to 44 years participated in this 8-year study. Those who ate the most calcium-rich foods were found to be 27% less to form kidney stones compared to those who ate the least. While taking supplemental calcium did not appear to increase risk, it didn't lower it. Other dietary factors that lowered kidney stone formation risk were eating foods high in phytates-a chemical in high fibre foods such as whole grains that binds minerals (37% risk reduction), drinking lots of fluids (32% risk reduction), and eating animal protein (16% risk reduction). Eating foods rich in sugar (sucrose) raised risk of kidney stone formation by 31%.

40. Broadus AE, et al.,(1984) - reported that hypercalciuria (increased calcium in the urine) is an important risk factor for kidney formation. Many patients with kidney stones are known to have intestinal hyperabsorption of calcium.

41. Ramello A et al (2000) - About 13% of Americans will
experience at least one occurrence of symptomatic kidney stones in their lifetime.

42. Massey LK et al (1993) - found that oxalic acid occurs naturally in many plant foods, Spinach, rhubarb, cocoa powder, chocolate, beets, beet greens, peppers, strawberries, tea (both black and green), peanuts, wheat germ and bran contain sufficient oxalic acid to increase urinary oxalate excretion. Increased oxalic acid in the urine raises the Tiselius Risk Index (TSI) and presumably increases the risk of calcium oxalate-rich kidney stones. One study showed that most of the above mentioned oxalic acid-containing foods do significantly increase urinary oxalate excretion.

43. Robertson WG, Peacock M et. al. (1980) - Reported that 6 to 8 glasses of water / day usually are enough to keep urine dilute and prevent crystallization. Kidney stone sufferers in hot climates should be specially certain to drink enough water.

44. Sutton RA, et. al. (1994) - reported that vitamin C can be oxidized to oxalic acid in the human body. Large doses of Vitamin C have been shown to increase oxalic acid excretion in the urine.

45. Willet WC et al (1997) - recommended that those at high risk of kidney stones start taking calcium supplements but this may not be wise. In a 12 year long prospective study of more than 90,000 nurses, this same group of researchers found that women who consumed more dietary calcium from food also experienced a reduced risk of developing kidney stones.

46. Pak C, (1997) - reported that patients who are prescribed calcium supplements may do better if the supplements are
taken in a divided dose with meals. This should reduce oxalic acid absorption. By contrast, taking a large single dose of calcium before bed may very well increase the risk of calcium-rich kidney stones forming. A supplement of potassium and magnesium citrate may help prevent stone formation when calcium supplements are taken.

47. Allen LH, et. al, (1981) - suggests that a small amount of dietary protein can be converted to oxalic acid in the body and excreted as such in the urine. In addition, dietary protein is known to enhance calcium excretion in the urine.

48. Robertson WG, et. al,(1979) - reported that both an increase in oxalate and calcium in the urine can result from increasing dietary protein intake will usually increase the TSI. As a result increased dietary protein will likely contribute to kidney stone formation. The risk of kidney stone formation seems greater from animal than vegetable proteins.

49. Hiatt RA, et.al, (1996) - reported that a randomized controlled trial found that a diet low in animal protein and higher in vegetable protein significantly reduced the recurrence of kidney stones in high-risk subjects.

50. Peacock M, et. al, (1979) - showed that a vegetarian diet was associated with lower excretion of calcium, oxalate and uric acid in the urine.

51. Silver J, et. al, (1983) - reported that dietary salt intake has long been known to contribute increased calcium loss in the urine in both normal subjects and in those with hypercalciuria.

52. Pak CY, (1998) - reported that a diet with more plant foods
would contain more citrate, magnesium and potassium. Many patients with calcium oxalate stones have low levels of citrate, potassium and magnesium in their urine. This is important because citric acid is known to reduce the tendency for calcium oxalate crystals to form. Potassium citrate is particularly effective in reducing stone formation in patients who do not have hypercalciuria but do have a low level of urinary citrate excretion. Urinary citrate drops with increasing urinary acid content so a diet high in whole grains, fruits and vegetables may be more effective than potassium citrate supplementation alone because such a diet will reduce urine acidity and increase potassium, magnesium and citric acid intake.

53. Ettenger B, et. al, (1997) - showed that potassium magnesium citrate may be more effective than potassium citrate alone.

54. Kenney JJ ( ) - reported that excessive salt intake increases the risk of kidney stones, osteoporosis, hypertension, stroke, kidney failure and heart disease and also may promote stomach and kidney cancer.

55. Borghi L, et.al.(2002) - showed in a recent 5 years randomized controlled study compared the study of two diets in 120 men with recurrent calcium oxalate stones.

56. Curhan SG, et. al, (1994) - suggests that it is important for physicians to rule out secondary causes of kidney stone formation. When infection, hyperparathyroidism and renal tubular acidosis are promoting kidney stone formation it is important that this primary disease process be the focus of medical treatment. (increased calcium
in the urine) is an important risk factor for kidney formation. Many patients with kidney stones are known to have intestinal hyperabsorption of calcium. Animal protein was reduced (to a target of 52 gm/day) and dietary salt intakes was cut by about 50% (the target was actually only 1150 mg sodium/day). The risk of new kidney stone formation over 5 years was reduced by 51% in the low-salt and meat group compared to the second group in which calcium intake was reduced to only 10 mmol/day.

57. Ruben Flocks, (1939) - urologist, first recognized hypercalciuria as a clinically significant entity associated with renal stone disease.

58. Hassapidou MN, et. al.,(1999) - Increased levels of urinary calcium also increases the risk of stone formation. Consumption of animal protein from meat, dairy, poultry, or fish increases urinary calcium. Perhaps for this reason, consumption of animal protein has been linked to an increased risk of forming stones.

59. Ettinger B, Pak CY, Citron JT et. al.,(1997) - The level of potassium in food is much higher than the small amounts found in supplements. Some citrate research conducted with people who have a history of kidney stones involves supplementation with a combination of potassium citrate and magnesium citrate. In one double-blind trial, the recurrence rate of kidney stones dropped from 64% to 13% for those receiving high amounts of both supplements.

60. Pak CY, (1999) - In that trial, people were instructed to take six pills per day, enough potassium citrate to provide 1,600 mg of
potassium and enough magnesium citrate to provide 500 mg of magnesium. Both placebo and citrate groups were also advised to restrict salt, sugar, animal protein, and foods rich in oxalate. Other trials have also shown that potassium and magnesium citrate supplementation reduces kidney stone recurrences.

61. Levine BS, Rodman JS, et al., (1994) - Citric acid (citrate) is found in many foods and may also protect against kidney stone formation.

62. Seltzer MA, Low RK, et al., (1996) - The best food source commonly available is citrus fruits, particularly lemons. One preliminary trial found that drinking 2 litres (approximately 2 quarts) of lemonade / day improved the quality of the urine in ways that are associated with kidney stone prevention.

63. Curhan GC, Willet WC, et al., (1998) - Lemonade was far more effective in modifying these urinary parameters than orange juice. The lemonade was made by mixing 4 oz lemon juice with enough water to make 2 litres. The smallest amount of sweetener possible should be added to make the taste acceptable. Further study is necessary to determine if lemonade can prevent recurrence of kidney stones. Drinking grapefruit juice has been linked to an increased risk of kidney stones in two large studies.

64. Shah PJR, (1980) - Grapefruit juice actually causes kidney stone recurrence or is merely associated with something else that increases risks remains unclear; some doctors suggest that people with a history of stones should restrict grapefruit juice
intake until more is known. Bran, a rich source of insoluble fibre, reduces the absorption of calcium, which in turn causes urinary calcium to fall.

65. Ebisuno S, Morimoto S, Yoshida T., et.al.,(1986) - In one trial, risk of forming kidney stones was significantly reduced simply by adding one-half ounce of rice bran per day to the diet.

66. Rao PN, Gordon C, et.al.,(1982) - Oat and wheat bran are also good sources of insoluble fibre and are available in natural food stores and supermarkets. Before supplementing with bran, however, people should check with a doctor, because some people—even a few with kidney stones—don’t absorb enough calcium. For those people, supplementing with bran might deprive them of much-needed calcium. People who form kidney stones have been reported to process sugar abnormally.

67. Lemann J Jr, Piening WF, Lennon EJ,(1969) - Sugar has also been reported to increase urinary oxalate, and in some reports, urinary calcium as well.

68. Gaby AR, (1996) - As a result, some doctors recommend that people who form stones avoid sugar.

69. Robertson WG, Peacock M, et.al.,(1980) - To what extent, if any, such a dietary change decreased the risk of stone recurrence has not been studied and remains unclear. Drinking water increases the volume of urine. In the process, substances that form kidney stones are diluted, reducing the risk of kidney stone recurrence. For this reason, people with a history of kidney stones should drink at least two quarts per day. It is particularly
important that people in hot climates increase their fluid intake to reduce their risk.

70. **Hollinberry PW, Massey LK, (1986)** - Drinking coffee or other caffeine-containing beverages increases urinary calcium.

71. **Keil DP, Felson DT, Hannan MT, et.al., (1990)** - Long-term caffeine consumers are reported to have an increased risk of osteoporosis.

72. **Curhan GC, Willet WC, Speizer FE, et.al., (1998)** - suggesting that the increase in urinary calcium caused by caffeine consumption may be significant. However, coffee consists mostly of water, and increasing water consumption is known to reduce the risk of forming a kidney stone. Many doctors are concerned about the possible negative effects of caffeine consumption in people with a history of kidney stones, preliminary studies in both men and women.

73. **Curhan GC, Willet WC, et.al. (1996)** have found that coffee and tea consumption is actually associated with a reduced risk of forming a kidney stone. These reports suggest that the helpful effect of consuming more water by drinking coffee or tea may compensate for the theoretically harmful effect that caffeine has in elevating urinary calcium. Therefore, the bulk of current research suggests that it is not important for kidney stone formers to avoid coffee and tea. The findings of some but not all studies suggest that consumption of soft drinks may increase the risk of forming a kidney stone.

74. **Conte A, Piza P, et.al. (1999)** - The phosphoric acid found in these beverages is thought to affect calcium metabolism in
ways that might increase kidney stone recurrence risk. Nutritional supplements that may be helpful I P-6 (inositol hexaphosphate, also called phytic acid) reduces urinary calcium levels and may reduce the risk of forming a kidney stone.

75. Grases F, et al. (1999) - In one trial, 120 mg per day of IP-6 for 15 days significantly reduced the formation of calcium oxalate crystals in the urine of people with a history of kidney stone formation.

76. Marshall RW, Hodkinson A, et al. (1972) - In the past, doctors have sometimes recommended that people with a history of kidney stones restrict calcium intake because a higher calcium intake increases the amount of calcium in urine. However, calcium (from supplements or food) binds to oxalate in the gut before either can be absorbed, thus interfering with the absorption of oxalate. When oxalate is not absorbed, it cannot be excreted in urine. The resulting decrease in urinary oxalate actually reduces the risk of stone formation.

77. Lemann J Jr, (1993) - The reduction in urinary oxalate appears to outweigh the increase in urinary calcium.

78. Sowers MFR, Wood C, et al. (1998) - In clinical studies, people who consumed more calcium in the diet were reported to have a lower risk of forming kidney stones than people who consume less calcium.

79. Curhan GC, et al. (1997) - However, while dietary calcium has been linked to reduction in the risk of forming stones, calcium supplements have been associated with an increased risk in a large study of American nurses.
80. Curhan GC, et.al.(1996) - The researchers who conducted this trial speculate that the difference in effects between dietary and supplemental calcium resulted from differences in timing of calcium consumption. Dietary calcium is eaten with food, and so it can then block absorption of oxalates that may be present at the same meal. In the study of American nurses, however, most supplemental calcium was consumed apart from food.

81. Pak CY, et.al.(1987) Calcium taken without food will increase urinary calcium, thus increasing the risk of forming stones; but calcium taken without food cannot reduce the absorption of oxalate from food consumed at a different time. For this reason, these researchers speculate that calcium supplements were linked to increased risk because they were taken between meals. Thus, calcium supplements may be beneficial for many stone formers, as dietary calcium appears to be, but only if taken with meals. When doctors recommend calcium supplements to stone formers, they often suggest 800 mg per day in the form of calcium citrate or calcium citrate malate, taken with meals. Citrate helps reduce the risk of forming a stone.

82. Levine BS, et.al.(1994) Calcium citrate has been shown to increase urinary citrate in stone formers, which may act as protection against an increase in urinary calcium resulting from absorption of calcium from the supplement.

83. Pak CY, et.al. (1987) Despite the fact that calcium supplementation taken with meals may be helpful for some, people with a history of kidney stone formation should not take
calcium supplements without the supervision of a healthcare professional. Although the increase in urinary calcium caused by calcium supplements can be mild or even temporary.

84. Bataille IP, et.al. (1983) - Some stone formers show a potentially dangerous increase in urinary calcium following calcium supplementation; this may, in turn, increase the risk of stone formation.

85. Bataille IP, et.al. (1983) - People who are "hyperabsorbers" of calcium should not take supplemental calcium until more is known. Using a protocol established years ago in the Journal of Urology, 24-hour urinary calcium studies conducted both with and without calcium supplementation determine which stone formers are calcium "hyperabsorbers."

86. Broadus AE et.al (1984) - Any healthcare practitioner can order this simple test. Increased blood levels of vitamin D are found in some kidney stone formers.

87. Rao PN, Blacklock NJ, (1983) - Until more is known, kidney stone formers should take vitamin D supplements only after consulting a doctor.

88. Nath R et.al. (1990) - Both magnesium and vitamin B6 are used by the body to convert oxalate into other substances. Vitamin B6 deficiency leads to an increase in kidney stones as a result of elevated urinary oxalate.

89. Mitwali A et.al. (1988) - Vitamin B6 is also known to reduce elevated urinary oxalate in some stone formers who are not necessarily B6 deficient.

90. Berkow R et.al. (1977) - Years ago, the Merck Manual
recommended 100-200 mg of vitamin B6 and 200 mg of magnesium per day for some kidney stone formers with elevated urinary oxalate.


92. Will EJ, et.al. (1979) - Optimal supplemental levels of vitamin B6 and magnesium for people with kidney stones remain unknown. Some doctors advise 200-400 mg per day of magnesium. While the effective intake of vitamin B6 appears to be as low as 10-50 mg per day, certain people with elevated urinary oxalate may require much higher amounts, and therefore require medical supervision. In some cases, as much as 1,000 mg of vitamin B6 per day (a potentially toxic level) has been used successfully.

93. Lindberg J, Pak CYC, et.al. (1979) - Doctors who do advocate use of magnesium for people with a history of stone formation generally suggest the use of magnesium citrate because citrate itself reduces kidney stone recurrences. As with calcium supplementation, it appears important to take magnesium with meals in order for it to reduce kidney stone risks by lowering urinary oxalate.

94. Piesse JW (1985) - It has been suggested that people who form kidney stones should avoid vitamin C supplements, because vitamin C can convert into oxalate and increase urinary oxalate.

95. Wandzilak TR et.al. (1984) - Initially, these concerns were questioned because the vitamin C was converted to oxalate.
after urine had left the body.

96. Levine M (1999) - However, newer trials have shown that as little as 1 gram of vitamin C per day can increase urinary oxalate levels in some people, even those without a history of kidney stones.

97. Auer BL et al. (1998) - In one case report, a young man who ingested 8 grams per day of vitamin C had a dramatic increase in urinary oxalate excretion, resulting in calcium-oxalate crystal formation and blood in the urine.

98. Curhan GC, Willet WC et al. (1996) - On the other hand, in preliminary studies performed on large populations, high intake of vitamin C was associated with no change in the risk of forming a kidney stone in women, 86 and with a reduced risk in men.

99. Baggio B et al. (1991) - This research suggests that routine restriction of vitamin C to prevent stone formation is unwarranted. However, until more is known, people with a history of kidney stones should consult a doctor before taking large amounts (1 gram or more per day) of supplemental vitamin C. Chondroitin sulphate may play a role in reducing the risk of kidney stone formation. One trial found 60 mg per day of glycosaminoglycans significantly lowered urinary oxalate levels in stone formers.

100. Amudam P et al. (1999) - Chondroitin sulphate is a type of glycosaminoglycan. A decrease in urinary oxalate levels should reduce the risk of stone formation. In a double-blind trial, supplementation with 200 IU of synthetic vitamin E per day was found to reduce several risk factors for kidney stone formation.
in people with elevated levels of urinary oxalate.

101. Suphiphat V et.al. (1993) - Refer to the individual supplement for information about any side effects or interactions. Herbs that may be helpful. Two trials from Thailand reported that eating pumpkin seeds reduces urinary risk factors for forming kidney stones.

102. Suphakam VS et. al. (1987) - One of those trials, which studied the effects of pumpkin seeds on indicators of the risk of stone formation in children, used 60 mg per 2.2 pounds of body weight—the equivalent of only a fraction of an ounce per day for an adult.

103. Barcelo P et.al. (1993) - Citrate, or citric acid, is an ordinary component of our diet, present in high amounts in citrus fruits. Citrate binds with calcium in the urine, thereby reducing the amount of calcium available to form calcium oxalate stones. It also prevents tiny calcium oxalate crystals from growing and massing together into larger stones. Finally, it makes the urine less acidic, which inhibits the development of both calcium oxalate and uric acid stones. One form of citrate supplement, potassium citrate, was approved by the FDA in 1985 for the prevention of two kinds of kidney stones: calcium stones (including calcium oxalate stones) and uric acid stones. In a 3-year double-blind study of 57 people with a history of calcium stones and low urinary citrate levels, those given potassium citrate developed fewer kidney stones than they had previously. In comparison, the group given placebo had no change in their rate of stone formation.
104. Ettinger B, Pak CY, et al. (1997) - Lemon juice may be preferable, as it has almost five times the citrate of orange juice. A small study found that drinking 2 liters of lemonade a day doubled urinary citrate in people with decreased urinary citrate.

105. Seltzer MA et al. (1996) - Avoid regular consumption of grapefruit juice, though: in one large-scale study, women drinking 8 ounces of grapefruit juice daily increased their risk of stones by 44%. It was first thought that citrate supplements were only helpful against kidney stones in individuals who didn't excrete the normal amount of citrate in their urine.

106. Gerster H (1997) - Early studies suggested that high vitamin C intake could increase oxalate in the urine and thereby potentially raise the risk of kidney stones. However, more recent research indicates that this conclusion was due to imperfect measurement techniques.

107. Simon JA et al. (1999) - In large-scale observational studies, individuals who consume large amounts of vitamin C have shown either no change or a decreased risk of kidney stone formation.

108. Curhan GC, Willet WC et al. (1997) - Several studies have shown that a higher dietary calcium intake has been associated with fewer calcium stone events in both men and women.

109. Borghi L et al. (2002) - Further, a study in 120 Italian hypercalciuric calcium oxalate stone-formers demonstrated that a diet with normal calcium, low sodium, and low animal protein resulted in reduced frequency of calcium stones compared
with a low-calcium diet.

110. Asplin JR (2002) - In this study, both diets were associated with a reduction in urinary calcium; however, urinary oxalate excretion rose in the low-calcium-diet group and fell in the high-calcium-diet group. The reduction in urinary oxalate excretion in individuals on a normal calcium diet was attributed to the intestinal binding of dietary oxalate by dietary calcium, thus lessening the amount of free oxalate available for absorption. Although both groups had reduced calcium oxalate saturation of their urine, the normal-calcium-diet group had a more significant reduction. Compared with the patients on a low calcium diet, the patients on the normal calcium, low sodium, low protein diet had a 50% reduction in stone risk at 5 years. In some calcium stone formers, the mechanism for the increased rate of calcium oxalate stones is the presence of hyperoxaluria.

111. Leumann E et al (1986-1993) - The hyperoxaluria is usually secondary to high dietary oxalate intake due to ingestion of foods or liquids containing large quantities of oxalate. Some of these foods and liquids include baked beans, collards, green beans, rhubarb, tea, cocoa, peanut butter, and vegetable soup. In other cases, the hyperoxaluria occurs in the setting of gastrointestinal malabsorption, seen in patients with inflammatory bowel disease. When patients malabsorb fat, dietary calcium binds to the fat rather than to dietary oxalate (which is the norm). This results in a larger amount of unbound intestinal oxalate that passes into the colon, from which it is absorbed. In patients with ileostomies (colon excluded) this
enhanced oxalate absorption does not occur. A rare cause of hyperoxaluria is the inherited condition known as primary hyperoxaluria.

112. Curhan GC, Willet WC et.al. (1998) - Avoid regular consumption of grapefruit juice, though: in one large-scale study, women drinking 8 ounces of grapefruit juice daily increased their risk of stones.

113. Pak CY (1994) - It was first thought that citrate supplements were only helpful against kidney stones in individuals who didn't excrete the normal amount of citrate in their urine.

114. Roman-Smith H et.al. (1993) - Since calcium oxalate is responsible for most kidney stones, avoiding foods containing oxalate may be helpful.

115. Brinkley L, Pak CYC, et.al. (1981) - Foods to be avoided include spinach, beet greens, nuts, chocolate, peanuts, bran, and strawberries.

116. Hollingbery PW et.al. (1986) - Caffeine should be avoided by anyone with kidney stones, as it appears to increase urinary calcium.

117. Curhan GC, Willet WC, et.al. (1993) - Animal proteins are linked to increased kidney stones.

118. Robertson WG, Peacock M, et.al. (1982) - A vegetarian diet will reduce the risk of stone formation.

119. Silver J et.al. (1983), Curhan GC et.al. (1993), Shah PJR (1980) - Salt increases urinary calcium excretion, and should be limited. Both Potassium and Bran Fiber reduce urinary calcium levels.
120. Marshall RW et al. (1972) - They are best taken in natural form, rather than as supplements. The following supplements may be effective in preventing and treating kidney stones: Calcium supplements cause oxalate to bind while in the stomach, before it can be absorbed into the urinary tract. This causes a decrease in urinary oxalate, which reduces the risk of kidney stones.

121. Curhan GC et al. (1993) - reported that the increased calcium in the urine may cause some kidney stones, overall higher calcium intake appears to lower kidney stone formation risk.

122. Rao PN et al (1983) - suggests that calcium should not be taken with Vitamin D, as this can increase calcium absorption and increase the risk of stone formation.

123. Baggio B et al. (1991) - Both chondroitin sulfate and glucosamine sulfate can help reduce urinary oxalate levels.

124. Lindberg J et al. (1990) - Magnesium helps the body to convert oxalate into other substances. Supplementation with Magnesium can reduce urine levels of both calcium and oxalate.

125. Ettiniberg B et al. (1988) - Vitamin B6, like Magnesium, is used to convert oxalate, and may be helpful in reducing the risk of kidney stone formation.

126. Piesse JW et al. (1985) - Supplements to Avoid - Vitamin C should be avoided by those who suffer from kidney stones, as it can convert to oxalate.