SAMUŁA, Sebastian, MAZUR, Michał, WAZ, Dorota, SZARŁOWICZ, Jakub, GOLISZEK, Zofia, SOBEK, Karolina Łucja, TABIN-BARCZAK, Wiktoria, SOKOŁOWSKA, Aldona, FIKAS, Klaudia and CHWALISZEWSKI, Kamil. Comparison of the Influence of Vegetarian and Mediterranean Diets on the Formation of Kidney Stones. Quality in Sport. 2025;37:57055. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.37.57055
https://apcz.umk.pl/QS/article/view/57055

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 16.12.2024. Revised: 03.01.2025. Accepted: 03.01.2025 Published: 07.01.2025.

Comparison of the Influence of Vegetarian and Mediterranean Diets on the Formation of Kidney Stones

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Abstract
Introduction and Purpose:
Kidney stone disease is a significant global health concern with increasing prevalence worldwide. Diet plays a critical role in both the prevention and development of this condition. The aim of this study was to compare the impact of vegetarian and Mediterranean diets on the risk of kidney stone formation, focusing on the metabolic mechanisms and nutritional composition of each diet.
Materials and Methods:
The review is based on a detailed analysis of peer-reviewed studies from major scientific databases such as PubMed. The selected studies focus on the mechanisms of kidney stone

formation, the influence of nutritional factors on this process, and the nutritional profiles of vegetarian and Mediterranean diets.
Results:
The vegetarian diet, rich in citrate, magnesium, and potassium, demonstrates urine-alkalizing properties that counteract kidney stone formation. Reducing meat intake in this diet lowers the risk of hyperuricosuria and uric acid stone formation. However, the vegan version of the vegetarian diet, which excludes dairy, may increase the risk of oxalate stones due to reduced calcium intake. Conversely, the Mediterranean diet offers a more balanced approach, combining the benefits of high fruit, vegetable, olive oil, and whole grain consumption with moderate intake of dairy, fish, and poultry. The presence of polyphenols in olive oil and the anti-inflammatory properties of this diet further support metabolic health and reduce the risk of kidney stones.
Conclusion:
The Mediterranean diet appears more effective in reducing kidney stone risk due to its flexibility and broader range of health benefits. It combines the advantages of the vegetarian diet with additional benefits provided by dairy, healthy fats, and moderate animal protein intake. Its comprehensive preventive effects and adaptability to patient needs make it a valuable dietary model for kidney stone prevention and management.

Keywords:
kidney stone disease; Mediterranean diet; vegetarian diet; metabolic factors; kidney stone prevention; dietary factors; recurrence prevention.

Introduction

Kidney stone disease (nephrolithiasis) is a well-known medical condition characterized by a high prevalence worldwide. The deposition of inorganic substances (such as salt crystals) along with organic components (e.g., macromolecules present in urine) in the renal parenchyma or the calyceal-pelvic system leads to the formation of kidney stones. Kidney Stone Disease (KSD) is common in nearly all regions of the world, and its prevalence continues to increase in many areas [1]. The global prevalence of nephrolithiasis ranges from 2% to 15%, with significant regional differences. The rate is approximately 13% in North America, 5–9% in Europe, and 1–5% in Asia [2]. In the United States, about 10.6% of men and 7.1% of women suffer from kidney stone disease, a prevalence comparable to that of diabetes (9.7%) [4].
Analyses of data from the NHANES study revealed a higher prevalence of KSD in men than in women in the adult population of the United States [2]. Nevertheless, in recent years, the prevalence in men has remained stable (11.6% in 2007–2008 and 11.9% in 2017–2018), while it has increased in women (from 6.5% in 2007–2008 to 9.4% in 2017–2018) [1]. Similar global trends indicate a decreasing male-to-female ratio in the context of kidney stone disease, reflecting a rise in cases among women, such as those reported in emergency departments [2].
Kidney stone disease also has a high recurrence rate, ranging from 35% to 50% after the first renal colic episode [2]. If patients fail to adhere to appropriate preventive measures (metaphylaxis), the recurrence rate can reach 10–23% annually, 50% within 5–10 years, and 75% within 20 years of the first episode. Recurrences of kidney stones can lead to numerous complications, including pyelonephritis, urinary tract infections, kidney failure, and even

urinary tract cancers [3]. Additionally, kidney stones can result in severe chronic health issues such as hypertension, chronic kidney disease, and, in extreme cases, end-stage renal disease [4].

The increase in its prevalence over recent decades can be attributed to environmental changes, including inadequate diets and limited physical activity. Diets high in animal protein and salt but low in fiber and plant-based proteins are known to increase the risk of lithogenesis. Similarly, low fluid intake and dietary changes, such as the popularity of high-protein diets or increased fructose consumption, also contribute to a heightened risk of kidney stone formation [2].

The economic burden associated with kidney stone disease is significant. As early as 2005, the annual costs of treating kidney stone disease in the United States exceeded $5 billion. With the growing number of individuals with obesity and diabetes, these costs are estimated to increase by an additional $1.24 billion per year through 2030 [4].

Mechanism of kidney stones formation

Calcium nephrolithiasis is the most common form of this disease, accounting for 75% of all cases. It is primarily associated with the presence of calcium oxalate, and less frequently with calcium phosphate. The formation of kidney stones involves several stages, including nucleation, crystal growth, and aggregation. A critical aspect of this process is the supersaturation of urine with calcium oxalate compounds, which is influenced by various factors, both promotive and inhibitory. Promotive factors include low urine volume, high urinary excretion of calcium, oxalate, and uric acid. On the other hand, inhibitors of kidney stone formation include citrate, magnesium, potassium, and other organic substances such as nephrocalcin [1].

The supersaturation of urine with solutes is a key factor in the formation of kidney stones, occurring when the concentration of dissolved substances exceeds their solubility limit. This supersaturation can be influenced by the balance between crystallization inhibitors and promoters, urine volume, and pH [5].

Urine pH is a critical determinant of the solubility of stone components. Calcium phosphate and struvite are less soluble in alkaline urine, whereas uric acid and cystine form stones in an acidic environment [5]. Moreover, most individuals with urinary supersaturation do not develop kidney stones, indicating the necessity of a nucleation site. Such sites may include damaged epithelial cells, Randall's plaques, or other structures that facilitate crystal growth and aggregation [6].

Genetic and anatomical factors also play significant roles in kidney stone formation. Hereditary patterns of calcium, citrate, oxalate, and uric acid excretion are well-documented. Additionally, inherited disorders such as cystinuria, primary hyperoxaluria, and Dent disease can lead to stone formation. Anatomical abnormalities, such as medullary sponge kidney, renal cystic diseases, or urothelial diverticula, increase the risk of lithogenesis through urinary stasis and localized changes in lithogenic factors [5].

The formation of calcium oxalate (CaOx) stones, the most common type of kidney stones, can occur through two primary mechanisms. The first, known as the intratubular mechanism, involves the supersaturation of crystallizable salts within the renal tubules, leading to crystal formation. These crystals are retained on the apical side of tubular epithelial cells, facilitated by the overexpression of crystal-binding proteins such as annexin A1, α-enolase, or heat shock protein 90. These retained crystals grow and aggregate until they obstruct the tubular lumen [1].

The second mechanism, called the interstitial mechanism, involves the formation of Randall's plaques within the renal interstitium. These plaques, primarily composed of calcium phosphate (CaP) hydroxyapatite crystals, may erode into the renal calyceal system, where they serve as nucleation sites for calcium oxalate (CaOx) crystals, ultimately leading to stone formation [1].

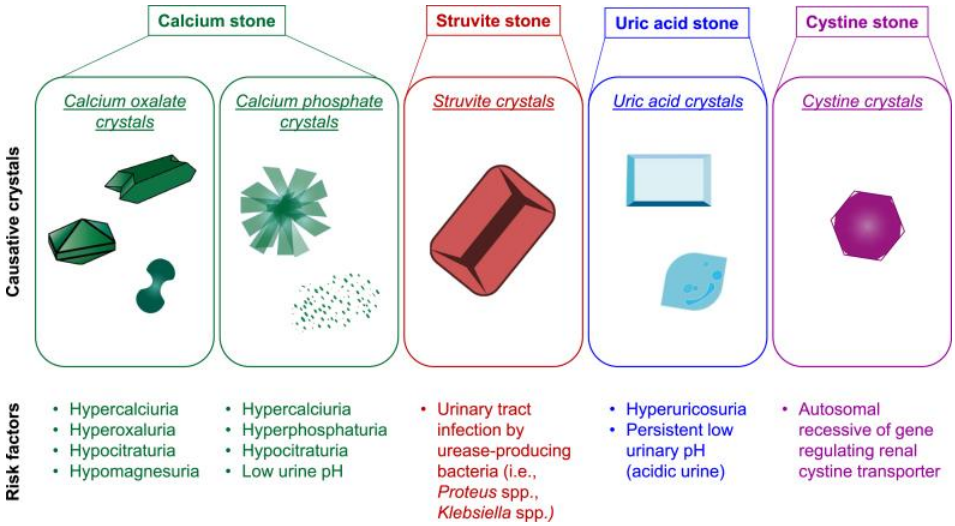

Figure 1. Type of kidney stones and their risk factors [1].

Calcium stones

Calcium stones, the most common type of kidney stones, are primarily composed of calcium oxalate (CaOx) or calcium phosphate (CaP), which can occur in pure or mixed forms [1]. The main risk factors for the formation of these stones (Figure 1) include hypercalciuria, hyperoxaluria, hyperuricosuria, hypocitraturia, and low urine volume [6].

Hypercalciuria, or excessive urinary calcium excretion, often results from metabolic disorders such as primary hyperparathyroidism or chronic metabolic acidosis. In such cases, treatment involves addressing the underlying conditions, such as parathyroidectomy or potassium citrate supplementation to reduce acidosis and improve urinary citrate levels [6]. The European Association of Urology (EAU) also recommends the use of thiazides in patients with calcium phosphate stones and concurrent hypercalciuria, as these drugs reduce urinary calcium excretion and lower the risk of new stone formation [7].

The gastrointestinal system plays a crucial role in the pathophysiology of idiopathic calcium oxalate nephrolithiasis, as intestinal absorption impacts calcium and oxalate metabolism. Terms such as "absorptive hypercalciuria" and "enteric hyperoxaluria" highlight the close interplay between the gut and kidneys in the development of these stones. Particular attention has been drawn to gut microbiota, especially *Oxalobacter formigenes*, which has the ability to degrade oxalates. However, studies on the role of microbiota in lithogenesis have produced conflicting results, leaving many questions unanswered [8].

Reduced urinary citrate levels (hypocitraturia) are among the most significant risk factors for the formation of calcium stones. Citrates act as crystallization inhibitors by binding calcium and reducing the supersaturation of lithogenic substances in the urine [6]. Citrate supplementation is particularly important for calcium phosphate stones, which often form in alkaline urine (pH > 6.0). However, excessive alkalization can increase the risk of calcium phosphate crystallization, so pH levels above 6.5 should be avoided [7].

Hyperoxaluria, or excessive urinary oxalate excretion, may result from a diet high in oxalates, intestinal absorption disorders, or genetic defects. Treatment involves reducing oxalate intake, calcium supplementation to bind oxalates in the gut, and the use of vitamin B6 to support glyoxylate metabolism [6].
The treatment and prevention of calcium stones require an individualized approach, combining dietary management, the use of crystallization inhibitors such as citrate, and pharmacological therapy, including thiazides in appropriate cases [6][7]. The role of gut microbiota in lithogenesis remains an area requiring further research, but the influence of the gastrointestinal system on calcium and oxalate metabolism underscores the need for a comprehensive approach to kidney stone prevention [8].

Uric acid stones

Uric acid stones consist of uric acid crystals, which typically crystallize in acidic urine with a pH below 5.5 [1][5]. Uric acid, with a pKa of 5.35, exists predominantly in its poorly soluble protonated form in acidic environments, promoting supersaturation and precipitation as stones. As urinary pH increases, uric acid transforms into the more soluble urate anion, which is a key mechanism for both treatment and prevention of these stones [7].
Uric acid stones are the most common type of radiolucent stones and are frequently associated with other crystals, forming mixed stones [1][5]. Their formation is also influenced by low urinary volume and hyperuricosuria, defined as uric acid excretion exceeding 800 mg/day in men and 750 mg/day in women [5]. This type of stone is particularly prevalent in patients with type 2 diabetes, obesity, and metabolic syndrome, highlighting a link between renal tubular insulin resistance and low urinary pH [7].
The treatment of uric acid stones focuses on alkalizing urine and reducing its supersaturation. Potassium citrate and sodium bicarbonate are recommended as primary alkalizing agents, with the goal of achieving a urinary pH between 6.0 and 6.5. This range increases the solubility of uric acid while avoiding excessive alkalization, which could promote apatite precipitation [6][7]. Potassium citrate is preferred; however, for patients with kidney disease or a risk of hyperkalemia, sodium bicarbonate serves as an alternative. The dose of alkalizing agents (30–60 mEq/day) should be adjusted while monitoring urinary pH using indicator strips [7].
The use of pioglitazone to address insulin resistance can also yield beneficial effects, such as raising urinary pH. Additionally, recommendations including regular physical activity, weight reduction, and glycemic control effectively support therapeutic efforts [7].
Maintaining adequate hydration is equally crucial, as increased urine volume reduces the supersaturation of lithogenic components and limits the risk of stone recurrence [6][7]. By combining dietary, pharmacological, and lifestyle interventions, it is possible to effectively prevent the formation and recurrence of uric acid stones.

Struvite stones

Struvite stones, also known as infection stones, are composed of magnesium ammonium phosphate ($MgNH_4PO_4 \cdot 6H_2O$) and often occur in combination with other components, such as calcium oxalate (CaOx) or calcium phosphate (CaP), particularly in the form of carbapatite [1][6]. The formation of these stones is closely associated with urinary tract infections caused by urease-producing bacteria, including *Proteus* spp., *Klebsiella* spp., *Pseudomonas* spp., and enterococci [6][7]. These bacteria hydrolyze urea, releasing hydroxide ions, which lead to urinary alkalization and promote the formation of struvite crystals [6].

Struvite stones are characterized by rapid growth and can develop into "staghorn" stones, making their treatment particularly challenging. Both the European Association of Urology (EAU) and the American Urological Association (AUA) recommend antibiotic therapy before and after surgical procedures, such as percutaneous nephrolithotomy (PCNL) or extracorporeal shockwave lithotripsy (ESWL), until stone sterilization is achieved. Antibiotic therapy should be guided by culture results and typically lasts 1–2 weeks before the procedure. Interestingly, some studies suggest that antibiotics may aid in reducing the size of struvite stones or facilitating their spontaneous expulsion; however, long-term antibiotic use is not recommended due to the risk of resistance [7].
In supportive treatment, urease inhibitors such as acetohydroxamic acid (AHA, Lithostat) are used to reduce ammonia production and lower urinary pH, thereby preventing the formation of new stones [6][7]. AHA works through irreversible and non-competitive urease inhibition, which helps delay stone growth and reduces the risk of recurrence compared to placebo. However, the use of AHA is limited due to side effects such as tremors, hemolytic anemia, headaches, and nausea [7].
In summary, the treatment of struvite stones requires a multidimensional approach that includes stone removal, infection control, and recurrence monitoring. The application of antibiotics, urease inhibitors, and appropriate surgical interventions enables more effective management of this challenging clinical condition [6][7].

Cystine stones
Cystine stones are a rare type of kidney stone, accounting for approximately 1–2% of cases in adults and 6–8% in children, making cystinuria the most common Mendelian genetic disorder causing nephrolithiasis [1][5]. This condition arises from inherited defects in amino acid transport in the kidneys and intestines, leading to increased urinary excretion of cystine [6]. Mutations in the SLC3A1 or SLC7A9 genes result in abnormalities in amino acid transporters, causing reduced reabsorption of cystine and its excessive presence in the urine [1][5].
Cystine, a homodimer of the amino acid cysteine, has limited solubility in urine, particularly at a pH below 6.5, which promotes its precipitation, crystallization, and the formation of cystine stones [1][6]. These stones are characterized by rapid growth and frequent recurrence, which can lead to significant kidney function impairment from an early age. Typical pathological changes include interstitial fibrosis and obstruction of collecting ducts [6].
The diagnosis of cystinuria involves analyzing family history, examining the stones, and measuring urinary cystine excretion. In healthy individuals, urinary cystine excretion is approximately 30 mg per day, while in patients with cystinuria, it can exceed 400 mg per day [6]. The solubility of cystine increases in alkaline urine, and achieving a pH above 7 reduces its supersaturation. The goal of urine alkalization is to maintain a pH in the range of 7.0–7.5, where cystine becomes soluble, without reaching a pH level high enough to promote the formation of calcium phosphate stones. The recommended alkalizing treatment is potassium citrate at a dose of 30–60 mEq/day. Urine pH should be monitored daily to ensure it remains within the target range [7].
Preventive therapy also includes maintaining high fluid intake throughout the day and night to keep urinary cystine concentrations below 243 mg/L [6]. Additionally, the use of potassium alkali effectively regulates urine pH and reduces the risk of stone recurrence [6][7].

Diet ingredients

Fluids

Adequate hydration is one of the most important preventive measures in reducing the formation and recurrence of kidney stones, regardless of their composition or individual risk factors. Increasing fluid intake results in urine dilution, which decreases the concentration of lithogenic substances and reduces their contact time with renal tubular epithelium. Additionally, a higher urine volume promotes the excretion of crystals, lowering the risk of their aggregation [9].
Studies confirm the effectiveness of increased fluid intake. In a randomized study involving 199 patients with idiopathic calcium oxalate stones, individuals who increased their water intake to achieve a urine volume of at least 2 L/day demonstrated 2.5 times greater urine volume, a lower stone recurrence rate, and a longer time to recurrence compared to the control group without specific recommendations. Systematic reviews and meta-analyses have shown that a urine volume exceeding 2.0–2.5 L/day significantly reduces the risk of recurrence [9].
Fluid intake recommendations may vary depending on the type of kidney stones. For most types of kidney stones, it is advised to maintain a urine output of at least 2.0–2.5 L per day. More intensive hydration is required for cystine stones, where the recommended urine volume is significantly higher than for other types of stones, at a minimum of 3.0 L per day. Maintaining this urine output reduces the concentration of cystine in the urine below the solubility threshold (1.3 mmol/L at a pH of 6.0). A study among adult patients with cystinuria demonstrated that a urine volume exceeding 3.0 L per day significantly reduces the risk of stone recurrence [9].
Adequate hydration is also an effective measure in the prevention of primary kidney stone formation. Cohort studies have shown that higher fluid intake protects against stone formation, although evidence from randomized trials in this area is limited [9]. In clinical practice, regular monitoring of urine output and tailoring fluid intake to the patient's needs are particularly important.

Mineral water
Mineral water, which contains magnesium and calcium as well as bicarbonates, can support alkalization therapy and reduce the risk of kidney stone formation by increasing urine pH and promoting the excretion of citrate, a natural crystallization inhibitor. Bicarbonates are potent alkalizing agents that lower the supersaturation of lithogenic components in urine, making bicarbonate-rich water an effective element in the prevention of calcium oxalate and uric acid stones [9].
Studies show that consuming 1.5–2.0 L of mineral water per day containing more than 1800 mg/L of bicarbonates increases urine pH, citrate, and magnesium excretion while decreasing the supersaturation of calcium oxalate and uric acid in urine. The observed increase in urinary calcium excretion in such cases does not negatively affect the risk of stone formation [9].
Alkalization therapy using mineral water is particularly beneficial for calcium oxalate, uric acid, and cystine stones, but it is not recommended for calcium phosphate or struvite stones. Due to the significant variability in calcium content among bottled waters (ranging from 13 to 581.6 mg/L), patients should pay attention to the composition to avoid excessive calcium intake [9].

Fruit juices and fructose
Fruit juices, especially those rich in citrates, can support kidney stone prevention by increasing urine pH and citrate excretion—natural inhibitors of crystallization. Citrus juices, such as orange, lemon, and lime, are particularly valued in this context. Orange juice raises urine pH and citrate excretion, though its effect on the risk of calcium oxalate stones remains

inconclusive. Due to its high sugar content, moderate consumption or dilution with water is recommended [9].
Lemon and lime juices contain higher concentrations of citrates compared to orange juice. However, the degree of dilution and the presence of non-alkalizing forms of citric acid are key factors influencing the preventive effectiveness of these beverages. Apple and blackcurrant juices also increase citrate excretion and urine pH but may simultaneously contribute to elevated oxalate concentrations. Cranberry juice produces mixed effects, ranging from reducing the risk of stones in healthy individuals to increasing the risk in patients with nephrolithiasis [9].
Fructose is a dietary macronutrient that has garnered particular interest in the context of kidney stone formation. A diet high in fructose, commonly found in processed foods and soft drinks, is strongly associated with an increased risk of nephrolithiasis [9][10]. High fructose intake enhances urinary excretion of calcium and oxalates, as confirmed in both animal models with magnesium deficiency and healthy individuals [10].
Fructose also contributes to insulin resistance, which is linked to low urine pH, thereby increasing the risk of uric acid stone formation. Although the mechanisms by which fructose affects urine composition are not yet fully understood, evidence suggests its influence through epigenetic mechanisms, such as the regulation of ncRNA and DNA methylation, which may impact the expression of genes responsible for the transport of lithogenic and anti-lithogenic substances [10].

Tea and coffee
Tea and coffee, two of the most popular beverages worldwide, may have a protective effect against the risk of kidney stone formation. Numerous genetic and population studies have shown an inverse relationship between coffee and caffeine consumption and the risk of nephrolithiasis [9][11]. For example, a study involving over 7,000 cases of kidney stones and 530,000 individuals without a history of nephrolithiasis found that higher coffee consumption was associated with a reduced risk of stone development. Similarly, daily consumption of at least one serving of coffee reduced the risk by approximately 26% compared to less frequent consumption [11].
The protective mechanisms of coffee and caffeine include their diuretic effect, which increases urine flow and reduces the risk of crystallization. Caffeine also decreases the adhesion of calcium oxalate crystals to renal tubular epithelial cells, while compounds such as citrates and trigonelline found in coffee further enhance its protective effect [11][12].
Tea consumption also demonstrates a beneficial effect in reducing the risk of kidney stone formation. Studies confirm that tea, due to its high flavonoid content—potent antioxidants—not only reduces the risk of stone formation but also helps prevent chronic diseases such as cancer and cardiovascular disorders. These effects align with earlier research indicating that tea consumption can significantly lower the risk of urinary stone formation [12].
Despite these benefits, the type of tea matters. Black and green teas contain higher levels of oxalates, which may increase the risk of calcium oxalate stones in susceptible individuals. Herbal and fruit teas have lower oxalate content and may be a safer choice for patients with kidney stones [9].

Alcohol
The relationship between alcohol consumption and the risk of kidney stone formation remains unclear. A cross-sectional study based on five cycles of NHANES data found no significant correlation between alcohol consumption (both lifetime and in the past 12 months) and a

history of kidney stones. There was no association between the amount or frequency of alcohol intake and the prevalence of kidney stones, even among heavy drinkers [13].
Although alcohol is often consumed socially, excessive consumption is linked to numerous health issues, such as diabetes and obesity, both of which are known risk factors for kidney stones. Furthermore, excessive alcohol intake may lead to kidney damage, inflammation, and fibrosis, which could also increase the risk of stone formation [13].
Despite the lack of a direct connection between alcohol consumption and kidney stones, coexisting conditions that indirectly influence stone risk should be considered. Further research in this area is needed, with larger sample sizes and more detailed data on kidney stone history [13].

Uric acid
Elevated serum uric acid (SUA) levels are an independent risk factor for kidney stone formation, even in individuals without symptoms of gout. A cross-sectional study conducted in a multiethnic cohort from southwestern China demonstrated a significant association between hyperuricemia and the prevalence of kidney stones. This risk increased with SUA levels in both men and women, even after adjusting for potential confounding factors [14].
The definition of hyperuricemia, as updated in the 2021 expert consensus, specifies SUA levels > 420 μmol/L for men and > 360 μmol/L for women. However, studies have shown that the risk of kidney stone formation increases at levels below these thresholds. For instance, in men, SUA levels above 356 μmol/L and in women above 265 μmol/L were associated with a marked increase in stone formation risk [14].
Gender differences in the prevalence of kidney stones may be attributed to anatomical differences in the urinary tract as well as lifestyle factors, such as higher rates of smoking and alcohol consumption among men [14].

Vitamins
The role of vitamin D in kidney stone formation is particularly significant. Adequate dietary calcium intake reduces the risk of calcium stones by limiting intestinal absorption of oxalates; however, calcium supplementation between meals increases urinary calcium excretion, potentially raising the risk of stone formation [15].
The active form of vitamin D (1,25-dihydroxycholecalciferol) and levels of 25-hydroxycholecalciferol are typically elevated in individuals with kidney stones, especially those with hypercalciuria. In such patients, additional vitamin D supplementation may further increase the risk of stone formation. To minimize this risk, it is recommended to monitor urinary calcium excretion [15].
Vitamin C may also increase the risk of calcium oxalate stone formation as it is metabolized into oxalate and excreted in urine. Studies have shown that supplementation of vitamin C at doses exceeding 1000 mg/day increases the risk of a first kidney stone by 41% [15].
Sodium chloride
Sodium chloride consumption can increase the risk of kidney stone formation, primarily by promoting urinary calcium excretion. High salt intake facilitates this process by inhibiting tubular calcium reabsorption due to the expansion of extracellular fluid volume caused by sodium [9][10].
Intervention studies have shown that increasing sodium intake by 100 mmol (2300 mg) per day raises urinary calcium excretion by approximately 1 mmol. Although one cohort study identified a correlation between sodium intake and kidney stone formation, other studies have

not confirmed these findings, potentially due to difficulties in accurately estimating salt intake using dietary questionnaires [9].
A randomized controlled trial involving 210 patients with idiopathic calcium oxalate stones demonstrated that a low-sodium diet effectively reduced urinary calcium excretion compared to a control diet [10]. The recommended daily sodium intake is less than 100 mmol (2300 mg) or 6 g of table salt [9][10].

Calcium
Calcium plays a crucial role in the pathogenesis of kidney stones. High urinary calcium excretion is one of the main risk factors for stone formation, with calcium oxalate (CaOx) and calcium phosphate (CaP) being the most common stone components. Although it was once believed that a low-calcium diet reduces the risk of kidney stones, current evidence suggests otherwise. Calcium intake of 1000–1200 mg/day decreases the risk of stone formation by limiting intestinal oxalate absorption [9][10].
Evidence also indicates that recommendations to restrict milk and dairy product consumption may lead to increased intake of animal protein, which can adversely affect kidney stone risk. While the role of dietary calcium in inducing epigenetic changes predisposing to nephrolithiasis is not yet fully understood, studies suggest that a low-calcium diet may contribute to such changes. For instance, a study on mice demonstrated that a low-calcium diet during pregnancy was associated with insulin resistance in offspring through an epigenetic inheritance mechanism. In humans, insulin resistance is implicated in the pathogenesis of uric acid stone formation [10].

Oxalates
Urinary oxalate is a significant risk factor for the formation of calcium oxalate (CaOx) stones. Increased oxalate excretion significantly elevates urinary supersaturation with calcium oxalate, promoting stone formation. In a study involving 134 patients with recurrent calcium oxalate stones, elevated oxalate excretion was identified as a primary factor contributing to disease recurrence [9].
The main sources of oxalate are dietary intake and endogenous synthesis. Excessive dietary oxalate consumption, such as 600 mg/day, can increase urinary oxalate excretion by over 50%. To reduce oxalate content, cooking methods, such as boiling spinach, are recommended. Endogenous oxalate production involves precursors like ascorbic acid and hydroxyproline [9]. Beverages, including fruit juices and teas, contain significant amounts of oxalate, which must be considered in the diet of patients with nephrolithiasis. Additionally, excessive vitamin C intake can increase urinary oxalate excretion, as it is metabolized into oxalate [9].

Phosphates
Phosphate plays a critical role in cellular metabolism and bone health, with most of it stored in bones and the remainder circulating in serum. Its concentration is influenced by diet, intestinal absorption, and urinary excretion, regulated by hormones such as parathyroid hormone (PTH), fibroblast growth factor 23 (FGF23), and vitamin D [10].
Renal tubular phosphate loss can lead to supersaturation of urine with calcium phosphate, increased intestinal absorption of calcium and phosphate, and elevated urinary calcium excretion, all of which promote stone formation. Additionally, it impacts other lithogenic parameters, such as uric acid, oxalate, and citrate, and may result in bone demineralization [10].

Magnesium

Magnesium, primarily found in bones and soft tissues, plays a significant role in preventing kidney stone formation due to its interaction with calcium and phosphates, which are key components in stone development. It acts as a crystallization inhibitor by binding oxalate and forming the more soluble magnesium oxalate, thereby reducing the risk of stone formation [10].

Studies have shown that magnesium supplementation increases the excretion of magnesium and citrate while decreasing oxalate excretion, limiting the formation of new stones. Magnesium deficiency has been associated with inflammatory conditions such as type 2 diabetes and metabolic syndrome, both of which elevate the risk of kidney stones. In animal models, magnesium deficiency has been linked to epigenetic changes in genes associated with inflammation and apoptosis [10].

Fruits and vegetables

The consumption of fruits and vegetables plays a critical role in the diet of individuals with kidney stones, providing an adequate supply of alkalizing compounds and citrates. The benefits of consuming these foods are linked to their alkalizing effects, which reduce the risk of stone formation. Metabolic acidosis increases the reabsorption and metabolism of citrates in renal tubules, whereas alkalosis or citrate supplementation enhances their urinary excretion, offering protective effects against stones. A high dietary acid load is associated with increased calciuria, reduced citraturia, and a higher risk of stone formation [16].

Citrates play a pivotal role in alkalizing urine and counteracting stone formation. They bind with calcium, inhibiting the crystallization and aggregation of calcium oxalate, thus preventing the formation of larger stones [16].

For individuals with calcium kidney stones, a diet rich in fruits and vegetables is currently recommended, while the intake of animal proteins and salt should be significantly reduced. These dietary modifications improve urinary pH and increase the excretion of potassium, citrates, and magnesium, significantly reducing the risk of stone formation. Conversely, the removal of fruits and vegetables from the diet reverses these effects, increasing the excretion of calcium and ammonium, leading to urinary oversaturation with calcium oxalate and calcium phosphate [16].

Animal and plant proteins

Proteins play a crucial role in the diet; however, their type and quantity significantly impact the risk of kidney stone formation. High consumption of animal proteins from non-dairy sources (poultry, meat, fish, eggs) combined with a low intake of alkalizing foods increases the risk of stone formation. This leads to a negative calcium balance, low urinary pH, and reduced excretion of citrates, potassium, and magnesium. Animal proteins also enhance purine metabolism, contributing to hyperuricosuria in uric acid and calcium stones, and may influence oxalate excretion in patients with idiopathic calcium oxalate stones [16].

On the other hand, plant-based proteins have not been associated with an increased risk of stone formation, and a diet rich in fruits and vegetables can reduce the risk of kidney stones by up to 45%. Additionally, the consumption of dairy proteins has a protective effect in the context of kidney stone formation [16].

Reducing protein intake in the diet can improve metabolic risk factors for kidney stone formation. A study on patients with hypercalciuria demonstrated that lowering protein consumption to 0.8 g/kg/day, combined with a daily calcium intake of 955 mg, improved

urinary parameters by reducing calcium, oxalate, and uric acid excretion, while increasing citrate excretion [16].
The source of protein is crucial—plant-based and dairy proteins appear to be neutral or protective, whereas animal proteins from non-dairy sources increase the risk of kidney stones [16].

Vegetarian diet
Vegetarianism encompasses various types of diets, differing in their level of restrictiveness. It is most commonly understood as the exclusion of meat from the diet, although less restrictive dietary patterns also fall within this category. Examples include flexitarians, who consume meat occasionally, such as once a week, and pescatarians, who avoid meat except for fish and seafood. Lacto-ovo vegetarians abstain from meat but consume animal-derived products such as eggs and dairy. A strict vegetarian diet excludes all animal-derived products. Veganism, a broader concept, not only encompasses a strict vegetarian diet but also involves avoiding products and items that exploit animals, such as cosmetics or clothing. For practical purposes, the term "vegan diet" is often used to describe a strict vegetarian diet [17].
The prevalence of vegetarianism varies by region. Asia has the highest rate of adherence to this diet, with 19% of the population following vegetarian practices, significantly boosted by India, where nearly 40% of people avoid meat. In Africa and the Middle East, approximately 16% of the population are vegetarians, compared to 8% in Central and South America. The lowest rates are observed in North America (6%) and Europe, where only 5% of the population are vegetarians [17].
The benefits of a vegetarian diet, as confirmed by the American Dietetic Association, include improved health outcomes as well as the prevention and treatment of various diseases. Research indicates a lower risk of developing cardiovascular diseases, and improvements in biochemical parameters have been noted in some types of cancer. Vegetarians are also characterized by a more active lifestyle and reduced consumption of harmful products [18].

Mediterranean diet
The Mediterranean diet is a dietary pattern rooted in the traditional eating habits of the Mediterranean Basin region. It reflects the diverse culinary practices and local products that have formed the basis of the region’s diet for centuries [19, 20]. The Mediterranean diet emphasizes the consumption of whole grains such as oats and barley, legumes (e.g., beans, lentils, and chickpeas), fresh vegetables, fruits, and nuts, such as walnuts and pine nuts [19, 20]. These foods provide the body with substantial amounts of fiber, vitamins, minerals, and bioactive compounds like polyphenols, which contribute to the health benefits associated with the this diet.
A key characteristic of the Mediterranean diet is the significant use of olive oil, particularly extra virgin olive oil, as the primary fat source. Olive oil is rich in monounsaturated fatty acids, which support cardiovascular health, and antioxidants such as vitamin E and polyphenols, which exhibit anti-inflammatory and anti-cancer properties [19, 21]. The diet minimizes the intake of saturated fats, distinguishing it from many other traditional diets worldwide.
This diet limits the intake of dairy products and red meat, focusing instead on fish and poultry as the primary sources of protein. Fish, especially fatty fish, provide valuable omega-3 fatty acids, which have anti-inflammatory properties and support heart and brain health [19, 20]. The Mediterranean diet encourages moderate consumption of dairy products, primarily in the form of fermented items like yogurt and cheese, which supply probiotics and calcium.

Moderate wine consumption, particularly red wine, is also an integral part of this diet, with its polyphenols, such as resveratrol, potentially benefiting cardiovascular health [21].
In terms of macronutrient proportions, the Mediterranean diet offers a balanced energy distribution: approximately 40–50% from carbohydrates, 15–20% from proteins, and 30–40% from fats. Importantly, the fats in this diet primarily come from olive oil, nuts, and fish, making it more healthful than diets rich in saturated fats [19].
Thanks to its unique composition, the Mediterranean diet is regarded as one of the healthiest dietary patterns worldwide. It not only promotes cardiovascular health but also helps prevent metabolic diseases, such as type 2 diabetes, and certain cancers. Numerous studies confirm that adherence to the Mediterranean diet is associated with increased life expectancy and improved quality of life.

The Impact of a Vegetarian Diet on Kidney Stones formation
Following a vegetarian diet involves high consumption of fruits, vegetables, legumes, nuts, seeds, and whole grains while reducing or completely eliminating meat. Numerous studies confirm that the citrates found in plant-based foods provide alkalizing properties, which can increase urine pH and reduce the risk of stone formation. Furthermore, these citrates bind calcium, limiting its crystallization and reducing the aggregation of existing crystals. A vegetarian diet also provides components such as magnesium and potassium, which bind oxalates in the gastrointestinal tract, thereby reducing their bioavailability and excretion in the urine. At the same time, this diet lowers purine intake by partially or completely excluding meat. This results in a significant reduction in the risk of hyperuricosuria and the formation of uric acid stones.
It is important to note that the vegan variation of a vegetarian diet, which completely excludes animal products, may be less beneficial in preventing kidney stones. The absence of dairy products in such a diet leads to lower calcium intake, which can increase oxalate absorption in the intestines. This, in turn, may elevate oxalate concentrations in the urine.

The Impact of Mediterranean Diet on Kidney Stones formation
The Mediterranean diet, characterized by high consumption of fruits, vegetables, legumes, olive oil, nuts, and whole grains, along with reduced intake of red meat and processed foods and moderate consumption of fish and poultry, demonstrates protective effects against kidney stone formation. Research indicates that these benefits arise from the diet's alkalizing properties and the presence of natural crystal inhibitors. Citrates in fruits and vegetables bind calcium, preventing its crystallization and reducing the aggregation of existing crystals. Olive oil, rich in polyphenols with anti-inflammatory properties, improves metabolic health and indirectly protects against kidney stone development. Whole grains, such as whole wheat bread and barley, are sources of phytates, which, along with magnesium found in leafy greens, nuts, and seeds, exhibit protective effects by binding oxalates in the gastrointestinal tract, reducing their absorption, and thereby preventing their excretion in urine. The diet is also rich in potassium, which reduces the risk of hypercalciuria by lowering urinary calcium levels. Similarly, limited sodium intake, achieved by avoiding processed foods, contributes to this protective effect.
Moderate dairy consumption provides an adequate amount of calcium, which binds oxalates in the gastrointestinal tract, reducing their bioavailability. Additionally, the low intake of red meat reduces purine levels, significantly lowering the risk of hyperuricosuria and the subsequent formation of uric acid stones.

Conclusions

The Mediterranean diet, with its more balanced approach, appears to have greater potential for reducing the risk of kidney stone formation. It effectively combines the benefits of a vegetarian diet, such as high consumption of fruits, vegetables, legumes, and whole grains, with the advantages of moderate dairy intake and healthy fats derived from olive oil. An additional strength of the Mediterranean diet is its inclusion of animal proteins, primarily fish and poultry, while limiting red meat intake, thereby reducing the risk of hyperuricosuria. The presence of polyphenols with anti-inflammatory properties further supports overall metabolic health, which is particularly significant for individuals with metabolic syndrome or excess weight.

The described benefits of the Mediterranean diet, along with its greater flexibility, which allows for easier adaptation to individual needs and preferences, make it more acceptable and effective for long-term adherence. These characteristics make the Mediterranean diet a particularly valuable lifestyle modification. For this reason, it should be widely promoted as part of kidney stone prevention strategies and as supportive care in managing the condition.

Disclosures

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All authors have read and agreed with published version of the manuscript.

Funding Statement – No applicable.
Institutional Review Board Statement – Not applicable.
Informed Consent Statement – Not applicable.
Data Availability Statement – The authors confirm that the data supporting this study are available in the article's references.
Conflict of Interest – Authors declare no conflict of interest.

References:

1. Peerapen P, Thongboonkerd V. Kidney Stone Prevention. *Adv Nutr*. 2023;14(3):555-569. doi:10.1016/j.advnut.2023.03.002
2. Tamborino F, Cicchetti R, Mascitti M, et al. Pathophysiology and Main Molecular Mechanisms of Urinary Stone Formation and Recurrence. *Int J Mol Sci*. 2024;25(5):3075. Published 2024 Mar 6. doi:10.3390/ijms25053075
3. Wigner P, Grębowski R, Bijak M, Szemraj J, Saluk-Bijak J. The Molecular Aspect of Nephrolithiasis Development. *Cells*. 2021;10(8):1926. Published 2021 Jul 29. doi:10.3390/cells10081926

4. Ma Y, Cheng C, Jian Z, et al. Risk factors for nephrolithiasis formation: an umbrella review. *Int J Surg*. 2024;110(9):5733-5744. Published 2024 Sep 1. doi:10.1097/JS9.0000000000001719
5. Shastri S, Patel J, Sambandam KK, Lederer ED. Kidney Stone Pathophysiology, Evaluation and Management: Core Curriculum 2023. *Am J Kidney Dis*. 2023;82(5):617-634. doi:10.1053/j.ajkd.2023.03.017
6. Ene MA, Geavlete PA, Simeanu CE, Bulai CA, Ene CV, Geavlete BF. The effectiveness of citrates and pyridoxine in the treatment of kidney stones. *J Med Life*. 2023;16(6):856-861. doi:10.25122/jml-2023-0234
7. Segall M, Mousavi A, Eisner BH, Scotland K. Pharmacologic treatment of kidney stones: Current medication and pH monitoring. *Actas Urol Esp (Engl Ed)*. 2024;48(1):11-18. doi:10.1016/j.acuroe.2023.11.008
8. Ticinesi A, Nouvenne A, Chiussi G, Castaldo G, Guerra A, Meschi T. Calcium Oxalate Nephrolithiasis and Gut Microbiota: Not just a Gut-Kidney Axis. A Nutritional Perspective. *Nutrients*. 2020;12(2):548. Published 2020 Feb 20. doi:10.3390/nu12020548
9. Siener R. Nutrition and Kidney Stone Disease. *Nutrients*. 2021;13(6):1917. Published 2021 Jun 3. doi:10.3390/nu13061917
10. D'Ambrosio V, Ferraro PM, Lombardi G, Friso S, Gambaro G. Unravelling the Complex Relationship between Diet and Nephrolithiasis: The Role of Nutrigenomics and Nutrigenetics. *Nutrients*. 2022;14(23):4961. Published 2022 Nov 23. doi:10.3390/nu14234961
11. Yuan S, Larsson SC. Coffee and Caffeine Consumption and Risk of Kidney Stones: A Mendelian Randomization Study. *Am J Kidney Dis*. 2022;79(1):9-14.e1. doi:10.1053/j.ajkd.2021.04.018
12. Yang L, Wang L, Liu Y, et al. Causal associations between 45 dietary intake habits and urolithiasis: insights from genetic studies. *Transl Androl Urol*. 2024;13(7):1074-1084. doi:10.21037/tau-24-79
13. Zhou Z, Huang Z, Ai G, Guo X, Zeng G, Zhu W. Association between alcohol consumption and kidney stones in American adults: 2007-2016 NHANES. *Front Public Health*. 2023;11:1156097. Published 2023 Apr 14. doi:10.3389/fpubh.2023.1156097
14. Deng H, Zhang X, Cheng N, et al. Asymptomatic hyperuricemia associated with increased risk of nephrolithiasis: a cross-sectional study. *BMC Public Health*. 2023;23(1):1525. Published 2023 Aug 10. doi:10.1186/s12889-023-16469-y
15. Cupisti A, Giannese D, D'Alessandro C, et al. Kidney Stone Prevention: Is There a Role for Complementary and Alternative Medicine?. *Nutrients*. 2023;15(4):877. Published 2023 Feb 9. doi:10.3390/nu15040877
16. Ferraro PM, Bargagli M, Trinchieri A, Gambaro G. Risk of Kidney Stones: Influence of Dietary Factors, Dietary Patterns, and Vegetarian-Vegan Diets. *Nutrients*. 2020;12(3):779. Published 2020 Mar 15. doi:10.3390/nu12030779
17. Hargreaves SM, Raposo A, Saraiva A, Zandonadi RP. Vegetarian Diet: An Overview through the Perspective of Quality of Life Domains. *Int J Environ Res Public Health*. 2021;18(8):4067. Published 2021 Apr 12. doi:10.3390/ijerph18084067
18. Wang T, Masedunskas A, Willett WC, Fontana L. Vegetarian and vegan diets: benefits and drawbacks. *Eur Heart J*. 2023;44(36):3423-3439. doi:10.1093/eurheartj/ehad436

19. Sultan MI, Ibrahim SA, Youssef RF. Impact of a Mediterranean diet on prevention and management of urologic diseases. *BMC Urol*. 2024;24(1):48. Published 2024 Feb 26. doi:10.1186/s12894-024-01432-9
20. Rodriguez A, Curhan GC, Gambaro G, Taylor EN, Ferraro PM. Mediterranean diet adherence and risk of incident kidney stones. *Am J Clin Nutr*. 2020;111(5):1100-1106. doi:10.1093/ajcn/nqaa066
21. Kiani AK, Medori MC, Bonetti G, et al. Modern vision of the Mediterranean diet. *J Prev Med Hyg*. 2022;63(2 Suppl 3):E36-E43. Published 2022 Oct 17. doi:10.15167/2421-4248/jpmh2022.63.2S3.2745