World J Urol (2017) 35:1301–1320
DOI 10.1007/s00345-017-2008-6

TOPIC PAPER

Epidemiology of stone disease across the world

Igor Sorokin[1] · **Charalampos Mamoulakis**[2] · **Katsuhito Miyazawa**[3] · **Allen Rodgers**[4] ·
Jamsheer Talati[5] · **Yair Lotan**[1]

Received: 21 October 2016 / Accepted: 24 January 2017 / Published online: 17 February 2017

Abstract Nephrolithiasis is a highly prevalent disease worldwide with rates ranging from 7 to 13% in North America, 5–9% in Europe, and 1–5% in Asia. Due to high rates of new and recurrent stones, management of stones is expensive and the disease has a high level of acute and chronic morbidity. The goal of this study is to review the epidemiology of stone disease in order to improve patient care. A review of the literature was conducted through a search on Pubmed®, Medline®, and Google Scholar®. This review was presented and peer-reviewed at the 3rd International Consultation on Stone Disease during the 2014 Société Internationale d'Urologie Congress in Glasgow. It represents an update of the 2008 consensus document based on expert opinion of the most relevant studies. There has been a rising incidence in stone disease throughout the world with a narrowing of the gender gap. Increased stone prevalence has been attributed to population growth and increases in obesity and diabetes. General dietary recommendations of increased fluid, decreased salt, and moderate intake of protein have not changed. However, specific recommended values have either changed or are more frequently reported. Geography and environment influenced the likelihood of stone disease and more information is needed regarding stone disease in a large portion of the world including Asia and Africa. Randomized controlled studies are lacking but are necessary to improve recommendations regarding diet and fluid intake. Understanding the impact of associated conditions that are rapidly increasing will improve the prevention of stone disease.

Keywords Epidemiology · Kidney stones · Nephrolithiasis · Renal calculi · Urolithiasis

Introduction

Nephrolithiasis is a highly prevalent disease worldwide with rates ranging from 7 to 13% in North America, 5–9% in Europe, and 1–5% in Asia [1–3] There is growing evidence for an increasing incidence of stones in the United States (US) with recent data finding an overall prevalence of stone disease in 8.8% of the population (men 10.6%, women 7.1%) which is an increase from the 5.2% prevalence of kidney stone disease from 1988 to 1994 [4, 5]. This rise was also documented over a 40-year period in Japan where the estimated annual incidence of first-episode upper urinary tract stones in 2005 was 134.0 per 100,000 (192.0 in men and 79.3 in women) compared with 54.2 per 100,000 in 1965 [6]. The annual incidence has increased in all age groups except during the first three decades of life, and the peak age for both men and women has also increased [6]. Iceland also has documented an increase in prevalence from 7 to 24 per 100,000 for men over age 40 and from 7 to 21 per 100,000 for women over 50 years of age during a 24-year period [7, 8].

✉ Yair Lotan
Yair.Lotan@utsouthwestern.edu

[1] Department of Urology, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390, USA

[2] Department of Urology, University General Hospital of Heraklion, University of Crete, Medical School, Heraklion, Crete, Greece

[3] Department of Urology, Kanazawa Medical University, Uchinada, Ishikawa, Japan

[4] Department of Chemistry, University of Cape Town, Cape Town, South Africa

[5] Section of Urology, Aga Khan University, Karachi, Pakistan

There is significant variation in rates based on geography, climate, diet, fluid intake, genetics, gender, occupation, and age. Due to the high rates of new and recurrent stones, management of stones is expensive and the disease has a high level of morbidity both acute and chronic. Understanding the epidemiology of stone disease can allow improved patient care and possibly prevention. The goal of this review is to update the work of the prior international consultation on urologic diseases (ICUD) regarding stone disease [9, 10]. A literature search was conducted through Pubmed®, Medline®, and Google Scholar® for English articles using keywords kidney stones, epidemiology, nephrolithiasis, diet intake and urolithiasis, fluid intake and urolithiasis, associated conditions of kidney stones, metabolic syndrome, obesity and nephrolithiasis, diabetes and nephrolithiasis, gout and nephrolithiasis, climate and nephrolithiasis, seasonal factors and nephrolithiasis, and occupation and nephrolithiasis. Studies identified through the search which evaluated current or new risk factors for stone disease internationally since the prior consultation updates comprised the bulk of this 2014 update. The current review is based on the evidence synthesis of the authors of this study highlighting studies which have added value to our understanding of stone disease. This review is limited given that it lacks formalized PRISMA guidelines and is based on expert opinion which may cause selection bias. This review was presented and peer-reviewed at the 3rd International Consultation on Stone Disease during the 2014 Société Internationale d'Urologie Congress in Glasgow.

Risk factors

Age

The incidence of stones varies by age with low incidence in childhood and the elderly and peaks in the fourth to sixth decades of life [11, 12]. An important factor to consider related to age is that incidence and prevalence of kidney stones represent two different entities. While incidence represents new stones, prevalence represents any stone during a period of time. As such, lifetime prevalence increases over time with age, and a population that is older may appear to have a higher stone prevalence when in fact, the incidence of stones is identical to a population that is younger on average. For example, in Japan, the prevalence increased from 4.7 and 2.1% for men and women in 1965, to 15.1 and 6.8% in 2005, respectively [13]. Over this time period, the percent of the population that was elderly increased from 9.7 to 27.9% which may have played a role [13].

There are several studies that found that stone disease is more common in working aged adults and then decreases in older individuals [14–16]. The increase in incidence of stones in middle age may be related to diet, work, and lifestyle changes [17].

There are differences in stone composition in different age groups. Calcium oxalate dihydrate (COD) stones are more common in young stone former in both sexes. In Europe, stone composition differs by age in addition to being affected by gender [16, 18] with COD being 5 times more common in younger people compared to elderly [15], while the contribution of COD declined (between 3 and 5%) for each decade from 20 to 80 years [15]. The amount of calcium oxalate monohydrate (COM) as the main component reached a peak somewhere between 40 and 70 years. In their series in patients up to 10 years of age, calcium phosphate constituted ~40% of cases, but then declined. In 20–29 year old females, calcium phosphate predominates (35% of stone) (mainly carbonate apatite). Infection stones occur at the two extremes of life.

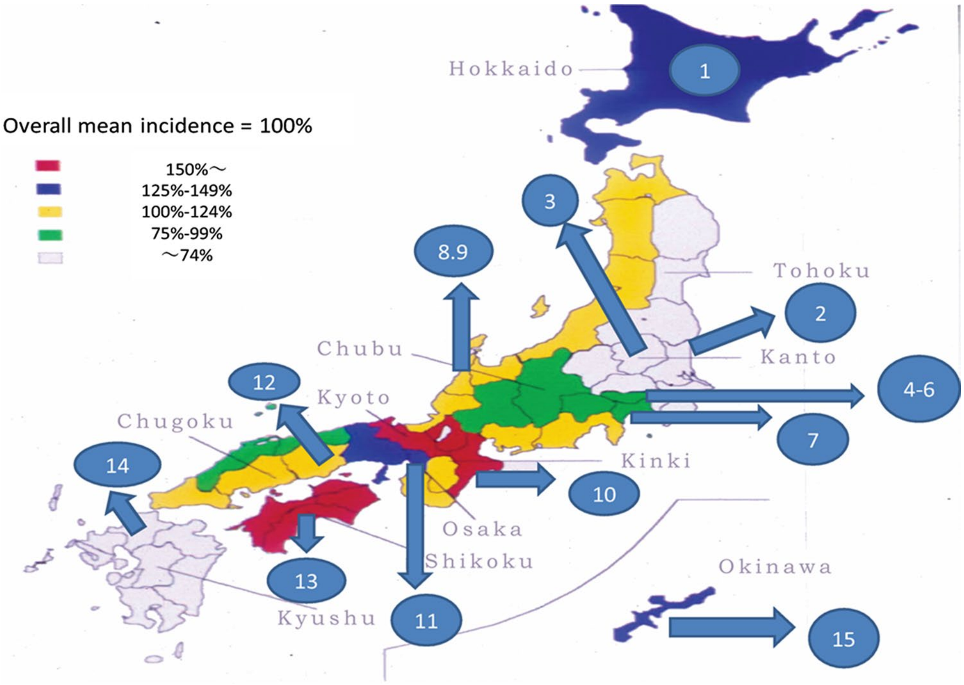
Gender ratios and nephrolithiasis

Overall, there has been a persistent male predominance in prevalence and incidence of kidney stones over a century. Literature suggests a gender ratio (GR) of ~1.5–2.5 across the world [19, 20]. However, there has been some evidence of a narrowing of the gender gap which may be related to changes in diet and increase in metabolic syndrome including rates of obesity and diabetes. In the US, the GR moved from 3:1 to 1.3:1 in 30 years (1970–2000) with rates for women increasing annually at a rate of 1.9% and men decreasing by 1.7% annually [21]. Scales et al. noted a change in GR from 1.7:1 to 1.3:1 (over the period 1997–2002); Strope et al. noted a 52% increase in the nephrolithiasis-related discharges of women as compared to a 22% increase in men; and the GR of stone patients discharged from hospitals, changed from 2 (1998) to 1.6 (2004) [4, 22]. On the other hand, these changes are not consistent worldwide. An analysis of 224,085 stones from 22 German centers noted an increasing GR reaching 2.7 in 2006 (from 1.86 in 1977) [16]. There is not much evidence that socioeconomic issues explain gender disparities [23]. Higher GR is seen in affluent Saudi Arabia (4:1) and Taiwan (GR 3.94) and a low GR (1.91), for upper urinary tract stones in Ouagadougou (Burkina Faso) [24–26].

Geographic variation of incidence and prevalence of nephrolithiasis

There is clear geographic variation in stone incidence worldwide. Even throughout a particular country, the incidence may have a drastic range (Fig. 1). The variation in incidence and prevalence is impacted by many factors with differing magnitude of impact on stone formation.

Fig. 1 Geographical distribution of various incidence rates of stone disease in Japan which have not changed in the last 20 years. Figure taken from Ref. [13], where it appears as "Figure 12.1, Geographical distribution of the annual incidence of urolithiasis in Japan…"



Factors such as seasonal temperature variation, genetics, water, environmental temperature, latitude, pollution, affluence, availability of technology, dietary habits, and age distribution among others interact with each other in complex ways.

There are also considerable issues with availability of data and sources of documentation for stones. Some countries have well-established sources of information such as the US but even the Urologic Diseases of America project used various databases for inpatient and outpatient care to try to capture the prevalence of disease since there is no centralized repository [27]. Differences in use of technology such as computed tomography (CT) can impact prevalence since CT scans have a higher sensitivity for stones than plain radiographs and ultrasound. On the other hand, routine use of ultrasound in examination may identify asymptomatic stones and can impact overall prevalence of disease. For example, ultrasound has been noted to identify asymptomatic stones in 2.1% of subjects in Denmark [28], 2% in Japan [29], and in 3% of subjects in Pakistan [30]. This may account for some of the increased prevalence noted in count in these countries.

One of the major problems in determining the prevalence of disease is that information from a third to a half of the population of the world is either non-existent or not accessible in the English speaking world. There is a lack of reliable large-scale studies from two major population blocks in South Central Asia and Sub-Saharan Africa (>1.8 billion people) and an inaccessibility of data for another 1 billion (+) population in China, because of language (Table 1).

Even when available, comparisons are difficult: because of differences in period of study (years); per capita income changes which affect availability of technology; type of population studied (people accessing hospital, or sample of communities); method of study (self-reported, retrospective review of radiological evidence; population-based ultrasound); institutional characteristics (academic, community based, rural, or city); population demographics (ethnic mix, and age), access to care and changes in economy [13, 31]. Available data on incidence and prevalence rates in countries are summarized in Table 2, constructed by mostly adding pertinent information to and utilizing the template provided in the 2007 consultation. For completeness and comparison, some of previous data in that table are included as are data from Africa (though little is available on incidence).

There are also some unique populations who are predisposed to stones that can skew prevalence data independently of geography. For example there is a very high prevalence rate (46%) among Hmong (originally from the highlands of Lao) populations who have settled in US (in whom 24% of stones are staghorn) [32]. Similarly there is a high prevalence in the Hubei province in China (20%) in contrast to the rest of China [33] and in native Saudis who have 2.5 times the prevalence as compared to non-native Saudis in Saudi Arabia [34]. Some populations may be protected from upper urinary tract stone disease such as sub-Saharan Africans (see Fig. 2 map of Africa) who have

Table 1 An assessment of what proportion of the world's population [145] has extant figures on prevalence and incidence

Country	Population (millions)	Availability of information (expert opinion)
China	1361	Information on (large) population-based studies emerging
India	1240	Information on incidence elusive
US	318	Well documented
Indonesia	249	Limited information
Brazil	201	Limited information
Pakistan	186	Incidence figures unrealistic extrapolations from hospitals which are difficult to access by the populations included in the hinterland
Nigeria	174	Hospital-based data
Russia	143	Information well documented, but not generally available
Japan	127	Well documented over at least four decades
Australia	23	Limited information
Canada	35	Assumed as equivalent to US
Europe	490	Well documented to date and was homogenous; but with new European union members, population demographics have changed
Sub-Saharan Africa	800+	Paucity of information

persistently lower rate even after migration to North America, where they have a lower self-reported prevalence compared with Hispanic and non-Hispanic whites [4]. Other populations have demonstrated a rapid increase in stone disease such as in Kerala (South India) [35] from dietary changes on attaining affluence from job-related migration to United Arab Emirates and in Japan, where the incidence doubled over a 40-year time period, both in men and women [6, 13, 36].

The urinary metabolic evaluation and stone analysis reveal interesting variations from country to country. For example, hypercalciuria is common in US but rare in Thailand and South Asia. Hyperoxaluria is common in 61% of a cohort from NE Peninsular Malaysia, [37] in 25–29% in India, [38], and only 1.3% in Thailand [39]. This has a significant impact of types of stone disease which can also vary within countries. Figures 3 and 4 show different distributions of stones in China and Japan.

More efforts will be necessary to improve understanding of regional variation of stone disease and prevalence in countries with high populations but relatively sparse literature (China and India) in order to improve individualized recommendation based on risk factors of varying local, temperature, diet, and genetics.

Climate and seasonal factors

Regions with higher mean annual temperature have increased risk for stones due to the impact of temperature on fluid status and urine volume [40, 41]. In the US there is a twofold higher prevalence of stones in the Southeastern US compared to the Northern part of the country [40]. A study of individuals living in villages in Israel found that those living in hot climate formed more stones than those residing in temperate climate conditions [42]. Another study found higher stone incidence in British naval personnel stationed in tropical climates as compared with those posted in the UK [43]. The impact of change in temperature was also well documented in a study of military personal deployed to Kuwait from March through August 2003 where at one military hospital a total of 182 patients were diagnosed with 218 symptomatic stones [44]. The mean time to the formation of symptomatic urinary calculi was 93 days with a standard deviation of 42 days.

Within each geographic location there are variations in temperature related to seasonal changes which have been described as "stone season." Several studies have found that increases in temperature in summer months result in an increase in stone formation. A study evaluating total admissions to emergency departments were obtained from the Taiwan National Health Insurance Research Database (1999–2003) which provided monthly urinary calculi attack rates per 100,000 of the population [45]. The seasonal trends in the monthly urinary calculi attack rates revealed a peak in July–September, followed by a sharp decline in October. Another study from Saudi Arabia evaluated 307 renal stones analyzed during 1 year period from September 2000 to August 2001 from different hospitals in Riyadh and found that maximum number of stones were analyzed in peak summer months [34].

Trends in global warming will likely result in shifting and expansion of areas at increased risk for stone formation [46]. A study modeling the impact of climate change on stone disease found that the fraction of the U.S. population

Table 2 Prevalence and incidence of stones by continent incidence are reported as per 100,000 and prevalence as a percent, except where quoted

Country/nation/region/city	Author (references)	Prevalence	Incidence	Additional notes
Sub-Saharan Africa				
Nigeria	Mbonu [146]	‘Extremely low’	13	Covers a 5-year period; 14.8% stones in children; >80% of calculi secondary to obstruction, infection and immobilization
Nigeria	Ekwere [147]		19.1	21% lower-tract stones. Relatively high incidence (contrast other Nigerian centers) explained as due to high consumption of calcium/magnesium-rich sea foods and vegetables, as well as the soft water of the South-East. Endemic bladder stones were absent in the series
Nigeria	Monu [148]		6.3	Gender (M:F) ratio of 4:1
Nigeria (Lagos University Teaching hospital)	Esho [149]		7	45 stones were recorded in a period during which 636,735 Nigerian patients were seen in the clinics 80% resulted from obstruction, stasis, infection, or metabolic disorders. This contrasts sharply with stones in Caucasians in which ~66% are idiopathic Children and females were rarely affected by urinary stones. In an autopsy review, only two adult were found to have stones out of a total of 5022 bodies
America North				
North America	Scales [4]	Overall:8.8% Males (M): 10.6% Females (F): 7.1%		White vs Black non-Hispanic odds ratio 0.37 White vs Hispanic Odds ratio 0.6 An increase in stone disease compared to NHANES III cohort, across black and Hispanic and white populations Black individuals less likely to report stone US 2007–2010 NHANES

Table 2 (continued)

Country/nation/region/city	Author (references)	Prevalence	Incidence	Additional notes
	Staemetoulou [5]	1988–1994 M: 6.3% F: 4.1%		Source: United States National Health and Nutrition Examination Survey (II and III), population-based, cross-sectional studies 15,364 adult US residents (1976–1980); 16,115 in 1988–1994 1988–1994, non-Hispanic African Americans had reduced risk of disease compared to non-Hispanic Caucasians (1.7 vs. 5.9%, $P < .05$), and Mexican Americans (1.7 vs. 2.6%, $P < .05$) Age-adjusted prevalence 6.6% in the South 3.3% in the West
America: South				
Argentina (City : Buenos Aires)	Pinduli [2]	Lifetime prevalence(2006) Overall: 3.96% M: 4.35% F: 3.62%		Cross-sectional study questionnaire administered by trained volunteer $N = 1{,}086$ data of 1998. No stone in those <20 years In subjects over 19 years, 5.14% (5.98% M; 4.49% F); 20–39 age group 2.75% ; >60 7.79%
Brazil	Korkes [150, 151]		Data from Brazil concern populations of which 85% are Caucasian and hence give insufficient or obscured information on the native population	69,039 admissions (Brazilian Public Health System Database) for stone in 2010. Brazil population 198.7 M. Admissions for urinary disease (0.61% of all hospital admissions): 63.2% white; 35.8% African descent; 0.2% (Braz) Indians. (Note 49.7 and 49.5% of general population are White or African descent). Admissions for urinary lithiasis increased 69% from 1996 to 2010
Asia: Mainland China, Islands, Korea, and SE Asia				
China	Zeng [20]	4.0% 4.8% M/3.0F		1.169651 million people addressed; Ultrasound scan Nationally representative sample
China	Luo [152]	1–5% From as low as 0.201; to 4.87% in Shenzen to 20% (in Hubei)	1.5–2 (22.7 in the plains)	More stones in south than in north. Stone cases admissions <14% of uro-admissions north of Yangtze river; and 22–45%. In the South. Guizhou has highest proportion (59%) of admissions Prevalence 1.6% in medical personnel, farmers, fishermen, service personnel and organizers, traffic wardens, surgeons, soldiers pilots. Cement workers >the offices. (7.11%)

Table 2 (continued)

Country/nation/region/city	Author (references)	Prevalence	Incidence	Additional notes
		0.1–2%		188,697 individuals surveyed across all parts of China From the National Survey Consortium on Urinary System Diseases. The Han, Miao, Zhuang, and Tong nationalities have high incidence rates (57.1, 46.5, 41.8, 40.8)
		1–5% prevalence	1.4 in 1985 1.01 in 1983	In Dongguan Guangdong province
			0.653 in 1986 0.202 in 1977	In Guangxi Zhuang
China (Hong Kong)	Chan [153]	2.5%		Telephone interviews of 1010 citizens (>18 years) in 2007 Prevalence 6.9% if family incidence included
Taiwan	Lee [25]	9.6% 14.5% M 4.3% F		Postal survey to 27,758 people, 0.2% of the adults in Taiwan. 4588 valid respondents (2002)
Korea (Seoul)	Kim [154]	Standardized lifetime prevalence 3.5% 6.0% M; 1.8% F		$N=2643$, City of Seoul (2002)
Thailand 1997	Yanagawa [155]	Disease rate of 16.9%		367 persons in 3 villages in the rural areas of Khon Kaen province (NE Thailand); questionnaire and abdominal ultrasound. A high incidence of dRTA has been noted with low citrate excretion in Lao–Thai
Asia-Japan				
Japan	Ogawa [36]	4-(1965) 10.8% (2005) Lifetime prevalence 15.1% M; 6.8% F	1340 (2005) 437 (1965)	Stone Belt : Kyoto, Osaka, Shikoku Hokkaido districts Less common in Tohoku, northern Kanto and Kyushu Ca stones more important and infection stones less frequent in urban areas Uric acid in S Kyushu, Shikoku, southern Kinki (Kyoto + Osaka) district
South and Central Asia (India, Pakistan, Iran; for former Soviet Republics, see Eurasia East) and Middle East and North African Belt				
Iran	Safarinejad [156]	5.7% (4.2–5.4) 6.1% M 5.3% F 15–29 years: 0.9% 60–69 years: 8.2%	145.1 (2005)	$N=7649$ (Iran's population 72 million people) Ethnic mix Arab, Fars, Kurdish, Turkish

Table 2 (continued)

Country/nation/region/city	Author (references)	Prevalence	Incidence	Additional notes
Iran	Basiri [157]		136	12 ecologic regions (inclusive of 30 provinces) were sampled Imaging-proven positive cases included; questionnaire filled out by interview
Iran	Ketabchi [158]	1.9% (in Kerman)		In Kerman alone
India (Rajasthan)	Singh [159]	Prevalence 4.6 times higher in regions with fluoride levels 3.5–4.9 ppm than those with levels <3.5		
India (Kerala)	Marickar [35]	Lifetime prevalence 2.643% 3.256% M 1.535% F		
Saudi Arabia		Lifetime: 20%		
Kuwait	ElRashaid [160]		23.9	Incidence 6.9 in the pediatric age group; 33.4 in adults; 73.6 in the elderly
Australia and New Zealand				
Australia	Lee [161]		131 (2002–7)	Quoting Australian Institute of Health and Welfare, Hospital Statistics 1996–2007
New Zealand	Davidson [162]		105	58% had stones in the ureter; 15% silent stones. Population: 90% of Euro origin; 3% Maori and 1.5% from Pacific Islands. They could not confirm the anecdotal impression that Maori and Polynesian islanders have a higher incidence
Europe-West				
Spain	Sanchez-Martin [163]	5.06%	730 (mentioned as 0.73%)	Summary of 16 papers on incidence (5 papers), prevalence (8 papers); and both (3 papers). Seven papers are nationwide and 9 are about local areas
Germany	Hesse [164]	4.7% M, 4.0% F (2001) 9.7% of men 50–64 year already had had a stone 5.9% of women by 50–64 year had already had a stone	2000: 1.47 0.84 M/0.63 F 1979: 0.54	*N* = 4000 Recurrence 42%
Europe	Osther [17]	5–10%		
Italy	Trinchieri [165]	1998:10.1% M, 5.8% F 1986: 6.8% M, 4.9% F	All: 0.4% Men: 0.6% Women: 0.2%	Questionnaires to two random samples of households in a village Prevalence of renal stones was significantly higher in 1998 than in 1986 only among males aged 31–40 and 51–60 years

Table 2 (continued)

Country/nation/region/city	Author (references)	Prevalence	Incidence	Additional notes
Iceland	Indridason [166]	Men: 4.3% (8.8% in 60–69 years) Women: 3.0% (5.0% in 60–69 years)	Men: 562 Women: 197	Period 1967–1991. 9039 M and 9619 F: response to question: Have u ever been diagnosed with stone, the prevalence was 8–19% in males depending upon geographical location in Iceland, and 3–5% in females. The incidence was 562/100k males 197 in females
Iceland	Edvardsson [8]		108 in the first 5-year interval of the study 138 in the last interval	24-year period, asymptomatic stones increased from 7 to 24% in M and 7–21 in F
Sweden	Ljunghall [167]	13.7%		In 2322 men. Only a quarter had been admitted to hospital
Turkey (Eurasia)	Muslumanoglu [168]	11.1%	1700 (author states 1.7%)	Representative sample, 2468 participants 18–70 years; 33 Turkish provinces, face-to-face interviews. Individuals with Turkish origin had a statistically significant decreased risk of urolithiasis ($p = 0.006$)
Eurasia–Russia				
Far Eastern Federal District			529.9- (2002) 714.7 (2008)	Highest ‘incidence’ of current Russian districts
Southern Federal District			360.6 (2002) 510.3 (2008)	Least ‘incidence’
See frequency index column for city/province		2–3% of working population has urolithiasis	In 1980: Moscow 17; Ukraine 53; Kyrgyzstan48; Turkmenistan 24 Uzbekistan 30 South Caucasus 35	
Altai Krai	Novikov [169]		1483 (2008)	
Ukraine			6580 (2006)	
Rep Belarus			1030 (2004)	
Tajikistan			2560 (2005)	

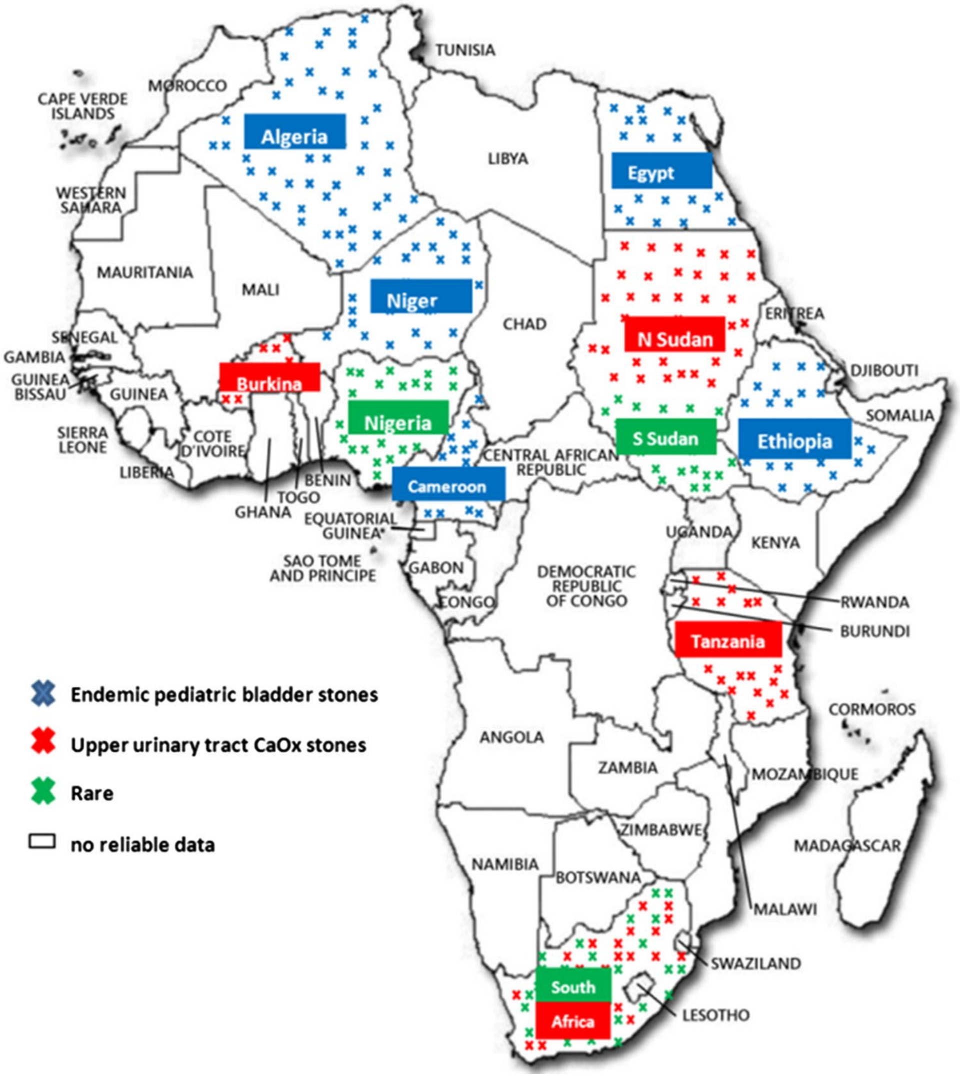


Fig. 2 Distribution of stone disease in Africa. Figure taken from Ref. [33], where it appears as "Figure 8.1 Distribution of Urinary tract stone disease in Africa)" p. 68

living in high-risk zones for nephrolithiasis will grow from 40% in 2000 to 56% by 2050, and to 70% by 2095. There is a predicted increase of 1.6–2.2 million lifetime cases of nephrolithiasis by 2050, which increases expenditures by 25%.

Diet

In the ICUD consensus document published in 2008, aspects of the relationship between urolithiasis and diet are compared to the updated ICUD 2008–2014 recommendations combined with the American Urological Association (AUA) guidelines [47], which are presented in Table 3. Since 2008, our search identified 30 articles [48–78]. Only six studies involving human subjects have been published since this time and of these, only two of them were RCTs [70, 74]. Moreover, only the former investigation had stone recurrence as its end point for establishing the efficacy or otherwise of the dietary protocols under investigation. Scrutiny of the rest of the articles reveals that the core dietary risk factors—calcium, oxalate, animal protein, carbohydrates, and sodium—remain unchanged. There were no "new" dietary risk factors which were proposed as being significant, although dietary fat was mentioned in two articles [60, 65]. The implied recommendations which emerged from these articles were to reduce the intake of saturated fats and to increase the intake of omega-3 essential fatty acids.

Several additional details (qualitative and quantitative) which were not described in the 2008 Consensus Document emerged during the present review. Regarding the protective effect of (modestly) increasing dietary calcium, Sellaturay et al. suggested that adding two glasses of milk per day to the diet is strongly associated with a decreased risk of kidney stones [49]; Worcester and Coe provided a more quantitative recommendation of 800–1000 mg/day of dietary calcium [55]. The most recent AUA guidelines increased the recommendation to 1000–1200 mg/

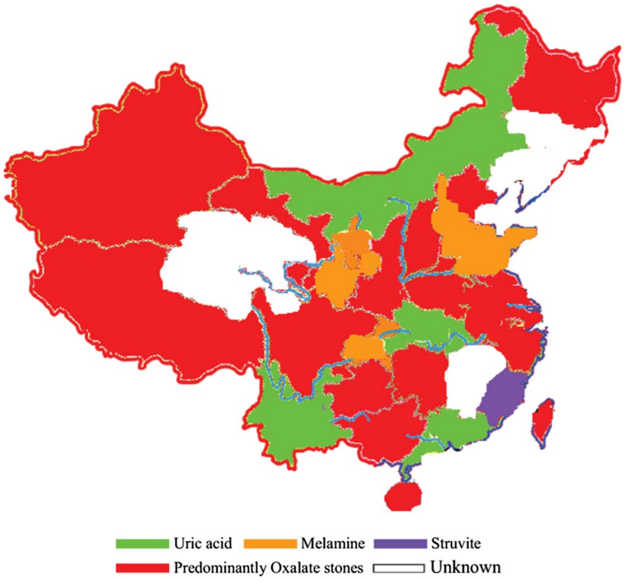


Fig. 3 Distribution of stone type in China. Figure taken from Ref. [33], where it appears as "Figure 6.4 Types of stone in different territories of China")

day of dietary calcium [47]. With respect to the intake of dietary oxalate, comprehensive lists of oxalate-rich foods were given in two articles [55, 65] and a guideline for the upper limit for oxalate intake was recommended [55]. The limits on protein intake which have been recommended since 2008 are fairly consistent [55, 60, 65]. Johri et al. reported that 40–50 g protein is approximately equivalent to 140–160 g animal flesh, irrespective of whether it is red meat, fish, or poultry [65]. Since sodium and salt (NaCl) are often used interchangeably, care has to be exercised in interpreting the suggested upper limits recommended in different articles: 2–3 g Na/day [79], 6 g NaCl/day (equivalent to 2.4 g Na/day) [60], and 100 mmol Na/day (equivalent to 2.3 g Na/day) [55]. Supplemental calcium is not regarded favorably [49, 55]. Restriction of vitamin C intake continues to be advised [56, 60, 61], with an upper limit of 1500 mg/day being recommended [60]. The role of vitamin D supplementation is controversial. Vitamin D supplementation is regarded as a risk factor [56] and over-repletion of vitamin D can be deleterious as noted in one study [60]. Others have noted that vitamin D therapy, if indicated, should not be withheld solely on the basis of stone disease [77]. Monitoring these patients may be difficult given that one study did not show a relationship between serum vitamin D level and 24-h urine calcium excretion in stone formers [78]. Therefore, repletion should be done carefully. The deleterious effects on urinary stone risk factors caused by high protein, low carbohydrate, ketogenic (Atkins) diets have been reinforced due to increase in urinary calcium and intracellular acidosis [51, 64, 80].

The associations between diet and stone disease are not always conclusive or consistent with several meta-analyses finding inconclusive results [52, 59]. Interestingly, Goldfarb et al. pointed out in the 2008 Consensus Document that the association of a high protein diet with stone prevalence has not been uniformly reported in epidemiological studies nor has any RCT demonstrated that a low animal protein diet has benefits with respect to prevention of stone formation [81]. While, Dussol et al. found that a low animal protein diet administered over 4 years, did not protect against stone recurrence in 175 idiopathic calcium stone formers, [70] the association between a high animal protein diet and nephrolithiasis continues to be widely reported and, as a result, restriction of dietary animal protein is commonly advocated and practiced [49, 50, 53–56, 60, 64, 65]. At this time, there is insufficient compelling evidence to either discount or advocate the beneficial effects of a reduced dietary intake of animal protein with respect to reducing stone formation.

In summary, the present review has shown that since publication of the 2008 Consensus Document, there has not

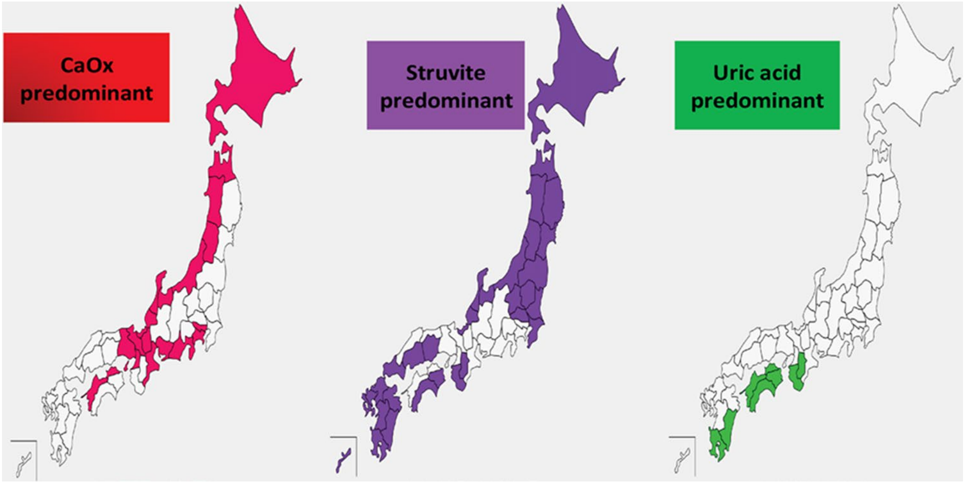


Fig. 4 Geographical distribution of stone type in Japan. Figure taken from Ref. [13], where it appears as "Figure 12.2, Geographical distribution of stone composition in Japan…"

Table 3 Summary of dietary and supplemental findings and recommendations from the ICUD Consensus Document 2008 compared with the updated literature and AUA guidelines

Dietary component	Recommendations and comments (ICUD 2008)	Updated recommendations (ICUD 2014 and AUA)
Ca	Avoid severe dietary Ca restriction; recommend modest restriction in hypercalciuric SFs No specific recommendation given regarding raising dietary Ca	Avoid severe dietary Ca restriction Maintain Ca intake at 1000–1200 mg/day
Ox	Restrict intake of nuts, chocolate, brewed tea, spinach, broccoli	Restrict Ox intake to <100 mg/day Restrict intake of nuts, chocolate, brewed tea, spinach, broccoli, rhubarb, beetroot, soya beans, tofu, peanut butter, okra, yams, sesame seeds, tahini, raspberries, figs, plums, wheat bran
Protein	Modestly restrict animal protein (red meat, fish, poultry) to 6–8 ounces/day	Modestly restrict animal protein (red meat, fish, poultry) to 0.8–1.0 g/kg/day
Carbohydrate	High intake identified as a dietary risk factor, but no specific recommendation given	Restrict refined carbohydrates to <20 g/day
Na	Limit sodium intake to 2–3 g/day	Limit sodium intake to ≤100 mEq/day (2300 mg/day)
Ca supplement	No specific recommendation given	Avoid
Vit C supplement	Limit intake of vit C to <1 g/day	Limit intake of vit C to <1.5 g/day
Vit D supplement	Explicit recommendation not given, but restriction is inferred	If indicated, vit D supplementation should not be withheld solely on the basis of stone disease. However, over-repletion may be detrimental so careful repletion is recommended
Atkins diet	Explicit recommendation not given, but avoidance is inferred	Avoid

Table 4 Summary of cited findings, authors' interpretations, and authors' recommendations

Type of fluid	Cited findings and authors' interpretations
Water	Significant decrease in stone reduction Urine output volume >2.5 l/day recommended
Coffee, tea, alcoholic beverages	Associated with reduced risk of stone formation
Soda	Conflicting results
Orange juice	No association with stone risk
Grapefruit juice	Increased risk of stone formation
Lemonade	Conflicting results

been any major shift in global expert opinion about dietary risk factors for kidney stone formation, nor has there been any new compelling evidence to unambiguously demonstrate an association between dietary interventions and stone recurrence. The recommendations given in Table 4 must be regarded as guidelines, rather than rigid rules. As is common practice, each patient must be evaluated individually. Dietary interventions can then be tailored according to the patient's specific metabolic profile.

Fluids

The impact of fluid intake on kidney stone pathogenesis and prophylaxis was briefly discussed in three chapters in the ICUD Consensus Document of 2008 (Table 4) [79–81]. Low fluid intake is a significant risk factor for kidney stone formation and that conversely, a regimen in which intake is increased will decrease the risk. This has been confirmed in more recent reviews. In those cases where authors provided recommendations, the desired urine output was recommended as being >2 l/day [48, 50, 60, 64, 65]. An output of >2.5 l day was advised in one article [68] and was strongly recommended in the most recent AUA guidelines [47]. In order to achieve output volumes of these magnitudes, fluid intake of 2.0–2.5 l/day was recommended [50, 64, 65]. A recent modeling study by Lotan et al. demonstrated that compliance with water intake in stone patients would result in significantly fewer stone recurrences with concomitant cost savings of millions of Euros/Dollars for the national budget [82]. Increasing water intake is therefore an established strategy in the treatment of kidney stone patients.

Four human studies have addressed changing composition of urine [83–86]. Some fruit juices (orange, apple, lime) have successfully raised urinary citrate excretion and urinary pH, both of which are regarded as favorable changes [87]. Inconsistent urinary responses have been reported for other juices such as grapefruit, cranberry and blackcurrant, and for sodas [87]. No updated literature on benefits of lemonade therapy have emerged since the 2nd ICUD statement. The previous literature has shown inconsistent results for lemonade therapy and benefits may be derived solely from maintaining adequate urine volume [88, 89]. Currently, there is insufficient evidence to recommend any forms of beverage (fruit juice, soda, energy drinks) for reduction of stone disease.

Occupational factors

Occupational history is considered an important component of stone formers' evaluation. Occupation risk include heat exposure resulting in dehydration or conditions that do not permit frequent hydration due to lack of access to water, or bathroom facilities, which may result in lower fluid intake, decreased urine output, and susceptibility to stone formation [90, 91]. Certain occupations such as steel workers, glass makers, and machinists have been associated with increased stones due to exposure to high heat and humidity [92].

Direct occupational exposure to toxic substances may also predispose to stone formation. Cadmium, a well-known nephrotoxic agent, as well as oxalic acid contained in the paint have been suggested as potential examples of substances responsible for a higher prevalence of urolithiasis in welders and spray painters [93–98]. In addition, a recent study has provided evidence that occupational exposure to trimethyltin, a by-product of plastic stabilizers known to inhibit H^+/K^+ ATPase activity in renal intercalated cells and alter urinary pH, is associated with an increased prevalence of kidney stones, with positive associations seen between exposure levels at the workplace or employment length and the development of stones [99].

Inheritance and genetics

Urinary tract stone disease including the most common form, idiopathic calcium oxalate urolithiasis, is considered to result from the interaction of heritable factors (single or multiple genes; polygenic or monogenic forms of urolithiasis) and their interplay with environmental influences. Although the genetic component is often considered part of the stone formers' differential diagnosis, people with known genetic causes appear to be few. The familial association of calcium oxalate urolithiasis and primary hypercalciuria, the most common metabolic risk factor, has been corroborated by several familial studies [100–102]. The collective estimation for the likelihood of kidney stone formers having affected first-degree or more distant family members is 15–65%; only 5–20% of non-stone formers have relatives with nephrolithiasis [103, 104]. Estimates of heritability in twin studies are higher (52–56%), with lower concordance in dizygotic than monozygotic twins (17 vs 32%, respectively) [105–107].

Recent genetic advances have revealed several candidate genes involved in renal calcium disorders/stone diseases such as NKCC2, ROMK, and ClCkb/Barttin, underlying renal salt excretion; claudin-14, -16, and -19, underlying renal calcium excretion; CaSR and KLOTHO that provide a sensing mechanism for renal salt, water, and calcium homeostasis regulation; F2, FN1, and HSPG2 encoding the stone inhibitor proteins prothrombin, fibronectin, and heparan sulfate proteoglycan, respectively; and MTNR1A, with unknown as yet mechanism [108–119]. The cellular mechanism for renal trans-epithelial calcium transport depends on these gene products' concerted action. Despite the fact that their individual pathogenic contribution in stone formation remains obscure, perturbation of their expression/function compromises different steps in the integrated pathway for calcium reabsorption, providing a physiological basis for diagnosing/managing renal stone diseases.

Monogenic forms are characterized by more severe phenotypes and progressive renal function impairment. Dent's disease, primary hyperoxaluria, adenine phosphoribosyltransferase (APRT) deficiency (2,8-dihydroxyadeninuria), hypoxanthine–guanine phosphoribosyl-transferase (HPRT) deficiency, and familial hypomagnesemia with hypercalciuria and nephrocalcinosis (FHHNC) in particular are associated with end-stage renal disease. Defective transporter, channel, and receptor proteins in the renal tubule and in other non-renal epithelia, as well as enzymes have been recognized to be responsible for these phenotypes [120]. Recent literature has also emerged distinguishing between the more recognized homozygous and heterozygous form of cystinuria. Heterozygous patients presented at a later age for their first stone episode had mixed stone compositions, had higher male predominance, and lower incidence of hyperuricemia [121].

Associated diseases

While there are certainly many diseases that may lead to increased risk of kidney stones, we highlight ones based on population issues that are increasing in prevalence.

Obesity

The prevalence and incident risk of kidney stone disease was found to increase with weight and body mass index (BMI) in both men and women from two large prospective cohort studies [122, 123]. The relative risk for stone formation in men weighing more than 100.0 kg vs less than 68.2 kg was 1.44 [95% confidence interval (CI), 1.11–1.86; $p = .002$ for trend]. A higher risk was also reported for men who gained more than 15.9 kg from the age of 21 years and women from the age of 18 years than in those whose weight did not change: 1.39 (95% CI 1.14–1.70; $p = .001$ for trend) for men, 1.70 (95% CI 1.40–2.05; $p < .001$ for trend) for older women, and 1.82 (95% CI, 1.50–2.21; $p < .001$ for trend) for younger women.

Other studies have shown that obese stone formers ($\text{BMI} \geq 30$ kg/m^2) are more likely to have a lower urine pH, hyperuricosuria, hypercalciuria, and hypocitraturia than non-obese stone formers [124], and body weight was shown to be inversely related to urine pH in a large population of kidney stone formers [125]. The pathophysiological mechanisms underlying the increase in acid excretion may be related to obesity/insulin resistance [126]. A higher body weight has been linked to insulin resistance, which may be responsible for the more acidic urine of heavier individuals [125, 127]. Previous studies have also suggested that renal lipotoxicity may be an integral part of this complex pathophysiology by impairing Na^+/H^+ exchange and NH_4^+ secretion in the proximal tubules of obese individuals [128, 129].

Diabetes mellitus

Epidemiological studies have recently implicated diabetes mellitus (DM) as a risk factor for the development of kidney stones [130, 131]. The relationship between DM and kidney stones has been linked to lower urine pH due to the effects of insulin resistance on ammoniagenesis [127]. Regarding the composition of kidney stones, the formation of uric acid stones was shown to be significantly higher in patients with DM. A study of 2464 calculi from 272 (11%) patients with type 2 diabetes and 2192 without type 2 diabetes found that the proportion of uric acid stones was 35.7% in patients with type 2 diabetes and 11.3% in patients without type 2 diabetes ($p < .0001$) [132].

Hypertension

Evidence for the association between hypertension and kidney stone disease has been inconsistent. Two longitudinal studies with follow-ups of 8 years showed that the incidence of kidney stones was significantly higher in hypertensive patients [133, 134]. In contrast to these smaller studies in which a large number of patients were lost to the follow-up, the incidence of kidney stone disease was independent of hypertension in a prospective analysis of two large cohorts [135, 136]. The findings of the recent Third National Health and Nutrition Examination Survey (NHANES) found that the likelihood of self-reported hypertension was 69% higher in female stone formers than in non-stone formers but these differences were not seen in men [137]. Consumption of a Dietary Approaches to Stop Hypertension (DASH)-style diet was prospectively examined in three cohort studies (the Health Professionals Follow-Up Study, Nurses' Health Study, and Nurses' Health Study II) and was associated with a marked decrease in the risk of kidney stone formation and incident kidney stones [138]. A DASH-style diet may reduce the risk of kidney stone formation by increasing urinary citrate levels and volume [139].

Gout

The findings of a nationally representative sample of American adults showed that even after adjusting for current age and BMI, as well as gender, race, and hypertension, patients with gout were still 49% more likely to have a history of kidney stones [140]. These results were confirmed in men in the prospectively conducted Health Professionals Follow-up Study. The risk of subsequent incident kidney stones in age-adjusted (RR 2.06) and multivariate-adjusted models (RR 2.12) was higher in patients with a confirmed diagnosis of gout than in those without gout. Men with gout were shown to be twofold more likely to develop incident kidney stones than men without gout [141].

Metabolic syndrome

Metabolic syndrome (MetS) also has been associated with kidney stone disease. MetS is a disorder of energy utilization and storage, diagnosed by a co-occurrence of three out of five of the following medical conditions: abdominal (central) obesity, elevated blood pressure, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol (HDL) levels. The prevalence of a self-reported history of kidney stones was shown to increase with the number of MetS traits from 3% with 0 traits to 7.5% with 3 traits and 9.8% with 5 traits. After adjusting for age and other covariates, the presence of 2 or more traits significantly increased the likelihood of self-reported kidney stone disease. The presence of 4 or more traits was associated with an approximately twofold increase in the likelihood of self-reported kidney stone disease in the NHANES [142]. MetS was also associated with a twofold higher level of echographic evidence of kidney stones in Caucasians in Southern Italy [143], and the presence of MetS had an OR of 1.25 for kidney stone prevalence evaluated using computed tomography or ultrasonography in 34,895 individuals who underwent general health screening tests in Korea [144].

Conclusions

There has been a rising incidence in stone disease throughout the world with a narrowing of the gender gap. Increased stone prevalence has been attributed to population growth and increases in obesity and diabetes. There is need for more studies regarding stone prevalence in Asia and Africa as well as identification of regional and ethnic variation in risk. General dietary recommendations of increased fluid, decreased salt, and moderate intake of protein have not changed. However, improvements have been made as specific recommended values have either changed or are more frequently reported. Still, randomized controlled studies are necessary to improve future recommendations regarding diet and fluid intake. Further research into impact of age and gender on stone disease will allow improved recommendations for different age/gender groups. Understanding the impact of associated conditions, especially those such as metabolic syndrome which are rapidly increasing, will improve policy decisions and prevention of stone disease overall.

Author contributions Igor Sorokin: Manuscript writing/editing, data analysis. Charalampos Mamoulakis: Manuscript writing/editing. Katsuhito Miyazawa: Manuscript writing/editing. Allen Rodgers: Manuscript writing/editing. Jamsheer Talati: Manuscript writing/editing. Yair Lotan: Protocol/project development, data analysis, manuscript writing/editing.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards The authors have abided by the statement of ethical standards for manuscripts submitted to World Journal of Urology.

References

1. Ramello A, Vitale C, Marangella M (2000) Epidemiology of nephrolithiasis. J Nephrol 13(Suppl 3):S45–S50
2. Pinduli I, Spivacow R, del Valle E, Vidal S, Negri AL, Previgliano H, Farias Edos R, Andrade JH, Negri GM, Boffi-Boggero HJ (2006) Prevalence of urolithiasis in the autonomous city of Buenos Aires, Argentina. Urol Res 34(1):8–11. doi:10.1007/s00240-005-0003-7
3. Medina-Escobedo M, Zaidi M, Real-de Leon E, Orozco-Rivadeneyra S (2002) Urolithiasis prevalence and risk factors in Yucatan, Mexico. Salud Publica de Mexico 44 (6):541–545
4. Scales CD, Jr., Smith AC, Hanley JM, Saigal CS, Urologic diseases in America P (2012) Prevalence of kidney stones in the United States. Eur Urol 62(1):160–165. doi:10.1016/j.eururo.2012.03.052
5. Stamatelou KK, Francis ME, Jones CA, Nyberg LM, Curhan GC (2003) Time trends in reported prevalence of kidney stones in the United States: 1976–1994. Kidney Int 63(5):1817–1823. doi:10.1046/j.1523-1755.2003.00917.x
6. Yasui T, Iguchi M, Suzuki S, Kohri K (2008) Prevalence and epidemiological characteristics of urolithiasis in Japan: national trends between 1965 and 2005. Urology 71(2):209–213. doi:10.1016/j.urology.2007.09.034
7. Indridason OS, Birgisson S, Edvardsson VO, Sigvaldason H, Sigfusson N, Palsson R (2006) Epidemiology of kidney stones in Iceland: a population-based study. Scand J Urol Nephrol 40(3):215–220. doi:10.1080/00365590600589898
8. Edvardsson VO, Indridason OS, Haraldsson G, Kjartansson O, Palsson R (2013) Temporal trends in the incidence of kidney stone disease. Kidney Int 83(1):146–152. doi:10.1038/ki.2012.320
9. Trinchieri A, Curhan G, Karlsen S, Jun Wu K (2003) Epidemiology. In: Segura J, Conort P KS (eds) Editions 21, Proceedings of 1st International Consultation On Stone Disease, Paris
10. Curhan GC, Goldfarb D, Trinchieri A (2008) Epidemiology of stone disease. In: Denstedt J, Khoury S (eds) Stone disease. Health Publications, Paris, pp 9–20
11. Saigal CS, Joyce G, Timilsina AR, Urologic Diseases in America P (2005) Direct and indirect costs of nephrolithiasis in an employed population: opportunity for disease management? Kidney Int 68(4):1808–1814. doi:10.1111/j.1523-1755.2005.00599.x
12. Hiatt RA, Dales LG, Friedman GD, Hunkeler EM (1982) Frequency of urolithiasis in a prepaid medical care program. Am J Epidemiol 115(2):255–265
13. Ogawa Y (2012) Epidemiology of stone disease over a 40-year period in Japan. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 89–96
14. Croppi E, Ferraro PM, Taddei L, Gambaro G, Group GEAFS (2012) Prevalence of renal stones in an Italian urban population: a general practice-based study. Urol Res 40(5):517–522. doi:10.1007/s00240-012-0477-z
15. Daudon M, Dore JC, Jungers P, Lacour B (2004) Changes in stone composition according to age and gender of patients: a multivariate epidemiological approach. Urol Res 32(3):241–247. doi:10.1007/s00240-004-0421-y
16. Knoll T, Schubert AB, Fahlenkamp D, Leusmann DB, Wendt-Nordahl G, Schubert G (2011) Urolithiasis through the ages: data on more than 200,000 urinary stone analyses. J Urol 185(4):1304–1311. doi:10.1016/j.juro.2010.11.073
17. Osther PJS (2012) Epidemiology of kidney stones in the European Union. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 3–12
18. Trinchieri A, Coppi F, Montanari E, Del Nero A, Zanetti G, Pisani E (2000) Increase in the prevalence of symptomatic upper urinary tract stones during the last ten years. Eur Urol 37(1):23–25. doi:10.1159/000020094
19. Walker V, Stansbridge EM, Griffin DG (2013) Demography and biochemistry of 2800 patients from a renal stones clinic. Ann Clin Biochem 50 (Pt 2):127–139. doi:10.1258/acb.2012.012122
20. Zeng Q, He Y (2013) Age-specific prevalence of kidney stones in Chinese urban inhabitants. Urolithiasis 41 (1):91–93. doi:10.1007/s00240-012-0520-0
21. Lieske JC, Pena de la Vega LS, Slezak JM, Bergstralh EJ, Leibson CL, Ho KL, Gettman MT (2006) Renal stone epidemiology in Rochester, Minnesota: an update. Kidney Int 69 (4):760–764. doi:10.1038/sj.ki.5000150
22. Strope SA, Wolf JS, Jr., Hollenbeck BK (2010) Changes in gender distribution of urinary stone disease. Urology 75 (3):543–546, 546 e541. doi:10.1016/j.urology.2009.08.007
23. Asper R (1984) Epidemiology and socioeconomic aspects of urolithiasis. Urol Res 12(1):1–5
24. Abomelha MS, Al-Khader AA, Arnold J (1990) Urolithiasis in Saudi Arabia. Urology 35(1):31–34
25. Lee YH, Huang WC, Tsai JY, Lu CM, Chen WC, Lee MH, Hsu HS, Huang JK, Chang LS (2002) Epidemiological studies on the prevalence of upper urinary calculi in Taiwan. Urol Int 68(3):172–177. doi:10.1159/000048445
26. Kabore FA, Kambou T, Zango B, Ouattara A, Simpore M, Lougue-Sorgho C, Lechevalier E, Karsenty G (2013) Epidemiology of a cohort of 450 urolithiasis at the Yalgado Ouedraogo University hospital of Ouagadougou (Burkina Faso). Progres Urol 23 (12):971–976
27. Pearle MS, Calhoun EA, Curhan GC, Urologic Diseases of America P (2005) Urologic diseases in America project: urolithiasis. J Urol 173(3):848–857. doi:10.1097/01.ju.0000152082.14384.d7

28. Emamian SA, Nielsen MB, Pedersen JF, Ytte L (1993) Sonographic evaluation of renal appearance in 665 adult volunteers. Correlation with age and obesity. Acta Radiol 34(5):482–485
29. Oshibuchi M, Nishi F, Sato M, Ohtake H, Okuda K (1991) Frequency of abnormalities detected by abdominal ultrasound among Japanese adults. J Gastroenterol Hepatol 6(2):165–168
30. Buchholz NP, Abbas F, Afzal M, Khan R, Rizvi I, Talati J (2003) The prevalence of silent kidney stones—an ultrasonographic screening study. JPMA 53(1):24–25
31. Bartoletti R, Cai T, Mondaini N, Melone F, Travaglini F, Carini M, Rizzo M (2007) Epidemiology and risk factors in urolithiasis. Urol Int 79(Suppl 1):3–7. doi:10.1159/000104434
32. Portis AJ, Hermans K, Culhane-Pera KA, Curhan GC (2004) Stone disease in the Hmong of Minnesota: initial description of a high-risk population. J Endourol Endourol Soc 18(9):853–857. doi:10.1089/end.2004.18.853
33. Rodgers A (2012) Epidemiology: South Africa and Sub-Saharan Africa. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 67–71
34. Khan AS, Rai ME, Gandapur, Pervaiz A, Shah AH, Hussain AA, Siddiq M (2004) Epidemiological risk factors and composition of urinary stones in Riyadh Saudi Arabia. J Ayub Med Coll Abbottabad JAMC 16(3):56–58
35. Marickar, YM (2012) Epidemiology of stone disease in Kerala, South India. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 47–51
36. Yoshida O, Terai A, Ohkawa T, Okada Y (1999) National trend of the incidence of urolithiasis in Japan from 1965 to 1995. Kidney Int 56(5):1899–904 doi:10.1046/j.1523-1755.1999.00754.x
37. Hussein NS, Sadiq SM, Kamaliah MD, Norakmal AW, Gohar MN (2013) Twenty-four-hour urine constituents in stone formers: a study from the northeast part of Peninsular Malaysia. Saudi J Kidney Dis Transp 24(3):630–637
38. Ganesamoni K (2012) Epidemiology of stone disease in Northern India. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 47–52
39. Stitchantrakul W, Kochakarn W, Ruangraksa C, Domrongkitchaiporn S (2007) Urinary risk factors for recurrent calcium stone formation in Thai stone formers. J Med Assoc Thail 90(4):688–698
40. Soucie JM, Coates RJ, McClellan W, Austin H, Thun M (1996) Relation between geographic variability in kidney stones prevalence and risk factors for stones. Am J Epidemiol 143(5):487–495
41. Mandel NS, Mandel GS (1989) Urinary tract stone disease in the United States veteran population. I. Geographical frequency of occurrence. J Urol 142(6):1513–1515
42. Frank M, Atsmon A, Sugar P, De Vries A (1963) Epidemiological investigation of urolithiasis in the hot arid Southern region of Israel. Urol Int 15:65–76
43. Blacklock NJ (1965) The pattern of urolithiasis in the Royal Navy. J R Nav Med Serv 51(2):99–111
44. Evans K, Costabile RA (2005) Time to development of symptomatic urinary calculi in a high risk environment. J Urol 173(3):858–861. doi:10.1097/01.ju.0000152578.07262.1c
45. Chen YK, Lin HC, Chen CS, Yeh SD (2008) Seasonal variations in urinary calculi attacks and their association with climate: a population based study. J Urol 179(2):564–569. doi:10.1016/j.juro.2007.09.067
46. Brikowski TH, Lotan Y, Pearle MS (2008) Climate-related increase in the prevalence of urolithiasis in the United States. Proc Natl Acad Sci USA 105(28):9841–9846. doi:10.1073/pnas.0709652105
47. Pearle MS, Goldfarb DS, Assimos DG, Curhan G, Denu-Ciocca CJ, Matlaga BR, Monga M, Penniston KL, Preminger GM, Turk TM, White JR, American Urological A (2014) Medical management of kidney stones: AUA guideline. J Urol 192(2):316–324. doi:10.1016/j.juro.2014.05.006
48. Worcester EM, Coe FL (2008) Nephrolithiasis. Primary Care 35(2):369–391, vii. doi:10.1016/j.pop.2008.01.005
49. SSaF C (2008) The metabolic basis for urolithiasis. Surgery 26(4):136–140
50. Goldfarb DS (2009) In the clinic. Nephrolithiasis. Ann Intern Med 151 (3):ITC2
51. Hall PM (2009) Nephrolithiasis: treatment, causes, and prevention. Clevel Clin J Med 76 (10):583–591. doi:10.3949/ccjm.76a.09043
52. Fink HA, Akornor JW, Garimella PS, MacDonald R, Cutting A, Rutks IR, Monga M, Wilt TJ (2009) Diet, fluid, or supplements for secondary prevention of nephrolithiasis: a systematic review and meta-analysis of randomized trials. Eur Urol 56(1):72–80. doi:10.1016/j.eururo.2009.03.031
53. Lopez M, Hoppe B (2010) History, epidemiology and regional diversities of urolithiasis. Pediatr Nephrol 25(1):49–59. doi:10.1007/s00467-008-0960-5
54. Romero V, Akpinar H, Assimos DG (2010) Kidney stones: a global picture of prevalence, incidence, and associated risk factors. Rev Urol 12(2–3):e86–e96
55. Worcester EM, Coe FL (2010) Clinical practice. Calcium kidney stones. N Engl J Med 363(10):954–963. doi:10.1056/NEJMcp1001011
56. Sakhaee K, Maalouf NM, Sinnott B (2012) Clinical review. Kidney stones 2012: pathogenesis, diagnosis, and management. J Clin Endocrinol Metab 97(6):1847–1860. doi:10.1210/jc.2011-3492
57. Taylor EN, Curhan GC (2008) Fructose consumption and the risk of kidney stones. Kidney Int 73(2):207–212. doi:10.1038/sj.ki.5002588
58. Saint-Elie DT, Patel PV, Healy KA, Solomon T, Pattaras JG, Qian J, Master VA, Ogan K (2010) The impact of income and education on dietary habits in stone formers. Urology 76(2):307–313. doi:10.1016/j.urology.2009.11.028
59. Sorensen MD, Kahn AJ, Reiner AP, Tseng TY, Shikany JM, Wallace RB, Chi T, Wactawski-Wende J, Jackson RD, O'Sullivan MJ, Sadetsky N, Stoller ML, Group WHIW (2012) Impact of nutritional factors on incident kidney stone formation: a report from the WHI OS. J Urol 187(5):1645–1649. doi:10.1016/j.juro.2011.12.077
60. Nouvenne A, Meschi T, Guerra A, Allegri F, Prati B, Borghi L (2008) Dietary treatment of nephrolithiasis. Clin Cases Miner Bone Metab 5(2):135–141
61. Pak CY (2008) Medical stone management: 35 years of advances. J Urol 180(3):813–819. doi:10.1016/j.juro.2008.05.048
62. Tracy CR, Pearle MS (2009) Update on the medical management of stone disease. Curr Opin Urol 19(2):200–204. doi:10.1097/MOU.0b013e328323a81d
63. Goldfarb DS (2009) Prospects for dietary therapy of recurrent nephrolithiasis. Adv Chronic Kidney Dis 16(1):21–29. doi:10.1053/j.ackd.2008.10.010
64. Sayer J MS, Thomas D (2010) The medical management of urolithiasis. Br J Med Surg Urol 3(3):87–95
65. Johri N, Cooper B, Robertson W, Choong S, Rickards D, Unwin R (2010) An update and practical guide to renal stone management. Nephron Clin Pract 116 (3):c159–171. doi:10.1159/000317196
66. Ortiz-Alvarado O, Miyaoka R, Kriedberg C, Moeding A, Stessman M, Anderson JK, Monga M (2011) Impact of dietary counseling on urinary stone risk parameters in recurrent stone

formers. J Endourol Endourol Soc 25(3):535–540. doi:10.1089/end.2010.0241

67. Kurtz MP, Eisner BH (2011) Dietary therapy for patients with hypocitraturic nephrolithiasis. Nat Rev Urol 8(3):146–152. doi:10.1038/nrurol.2011.9
68. Xu H, Zisman AL, Coe FL, Worcester EM (2013) Kidney stones: an update on current pharmacological management and future directions. Expert Opin Pharmacother 14(4):435–447. doi:10.1517/14656566.2013.775250
69. Penniston KL, Nakada SY (2009) Effect of dietary changes on urinary oxalate excretion and calcium oxalate supersaturation in patients with hyperoxaluric stone formation. Urology 73(3):484–489. doi:10.1016/j.urology.2008.10.035
70. Dussol B, Iovanna C, Rotily M, Morange S, Leonetti F, Dupuy P, Vazi A, Saveanu A, Loundou A, Berland Y (2008) A randomized trial of low-animal-protein or high-fiber diets for secondary prevention of calcium nephrolithiasis. Nephron Clin Pract 110 (3):c185–194. doi:10.1159/000167271
71. Thomas E, von Unruh GE, Hesse A (2008) Influence of a low- and a high-oxalate vegetarian diet on intestinal oxalate absorption and urinary excretion. Eur J Clin Nutr 62(9):1090–1097. doi:10.1038/sj.ejcn.1602832
72. Stoller ML, Chi T, Eisner BH, Shami G, Gentle DL (2009) Changes in urinary stone risk factors in hypocitraturic calcium oxalate stone formers treated with dietary sodium supplementation. J Urol 181(3):1140–1144. doi:10.1016/j.juro.2008.11.020
73. Nouvenne A, Meschi T, Guerra A, Allegri F, Prati B, Fiaccadori E, Maggiore U, Borghi L (2009) Diet to reduce mild hyperoxaluria in patients with idiopathic calcium oxalate stone formation: a pilot study. Urology 73 (4):725–730, 730 e721. doi:10.1016/j.urology.2008.11.006
74. Nouvenne A, Meschi T, Prati B, Guerra A, Allegri F, Vezzoli G, Soldati L, Gambaro G, Maggiore U, Borghi L (2010) Effects of a low-salt diet on idiopathic hypercalciuria in calcium-oxalate stone formers: a 3-mo randomized controlled trial. Am J Clin Nutr 91(3):565–570. doi:10.3945/ajcn.2009.28614
75. Knight J, Assimos DG, Easter L, Holmes RP (2010) Metabolism of fructose to oxalate and glycolate. Horm Metab Res 42 (12):868–873. doi:10.1055/s-0030-1265145
76. Lotan Y, Pearle MS (2011) Cost-effectiveness of primary prevention strategies for nephrolithiasis. J Urol 186(2):550–555. doi:10.1016/j.juro.2011.03.133
77. Leaf DE, Korets R, Taylor EN, Tang J, Asplin JR, Goldfarb DS, Gupta M, Curhan GC (2012) Effect of vitamin D repletion on urinary calcium excretion among kidney stone formers. Clin J Am Soc Nephrol CJASN 7(5):829–834. doi:10.2215/CJN.11331111
78. Eisner BH, Thavaseelan S, Sheth S, Haleblian G, Pareek G (2012) Relationship between serum vitamin D and 24-hour urine calcium in patients with nephrolithiasis. Urology 80(5):1007–1010. doi:10.1016/j.urology.2012.04.041
79. Pearle MS, Asplin JR, Coe FL, Rodgers AL, Worcester EM (2008) Medical management of urolithiasis. In: Denstedt J, Khoury S (eds) Stone disease. Health Publications, Paris, pp 59–82
80. Assimos DG (2008) Evaluation of the stone former. In: Denstedt J, Khoury S (eds) Stone disease. Health Publications, Paris, pp 33–36
81. Goldfarb DTA, Curhan G (2008) Epidemiology of stone disease. In: Denstedt J, Khoury S (eds) Stone disease. Health Publications, Paris, pp 11–19
82. Lotan Y, Buendia Jimenez I, Lenoir-Wijnkoop I, Daudon M, Molinier L, Tack I, Nuijten MJ (2013) Increased water intake as a prevention strategy for recurrent urolithiasis: major impact of compliance on cost-effectiveness. J Urol 189(3):935–939. doi:10.1016/j.juro.2012.08.254
83. Passman CM, Holmes RP, Knight J, Easter L, Pais V, Assimos DG (2009) Effect of soda consumption on urinary stone risk parameters. J Endourol Endourol Soc 23(3):347–350. doi:10.1089/end.2008.0225
84. Baia Lda C, Baxmann AC, Moreira SR, Holmes RP, Heilberg IP (2012) Noncitrus alkaline fruit: a dietary alternative for the treatment of hypocitraturic stone formers. J Endourol Endourol Soc 26(9):1221–1226. doi:10.1089/end.2012.0092
85. Tosukhowong P, Yachantha C, Sasivongsbhakdi T, Ratchanon S, Chaisawasdi S, Boonla C, Tungsanga K (2008) Citraturic, alkalinizing and antioxidative effects of limeade-based regimen in nephrolithiasis patients. Urol Res 36(3–4):149–155. doi:10.1007/s00240-008-0141-9
86. Aras B, Kalfazade N, Tugcu V, Kemahli E, Ozbay B, Polat H, Tasci AI (2008) Can lemon juice be an alternative to potassium citrate in the treatment of urinary calcium stones in patients with hypocitraturia? A prospective randomized study. Urol Res 36(6):313–317. doi:10.1007/s00240-008-0152-6
87. Rodgers AL, Webber D, de Charmoy R, Jackson GE, Ravenscroft N (2014) Malic acid supplementation increases urinary citrate excretion and urinary pH: implications for the potential treatment of calcium oxalate stone disease. J Endourol Endourol Soc 28(2):229–236. doi:10.1089/end.2013.0477
88. Koff SG, Paquette EL, Cullen J, Gancarczyk KK, Tucciarone PR, Schenkman NS (2007) Comparison between lemonade and potassium citrate and impact on urine pH and 24-hour urine parameters in patients with kidney stone formation. Urology 69(6):1013–1016. doi:10.1016/j.urology.2007.02.008
89. Kang DE, Sur RL, Haleblian GE, Fitzsimons NJ, Borawski KM, Preminger GM (2007) Long-term lemonade based dietary manipulation in patients with hypocitraturic nephrolithiasis. J Urol 177 (4):1358–1362. doi:10.1016/j.juro.2006.11.058 **(discussion 1362; quiz 1591)**
90. Curhan GC (2007) Epidemiology of stone disease. Urol Clin North Am 34(3):287–293. doi:10.1016/j.ucl.2007.04.003
91. Goldfarb DS, Arowojolu O (2013) Metabolic evaluation of first-time and recurrent stone formers. Urol Clin North Am 40(1):13–20. doi:10.1016/j.ucl.2012.09.007
92. Atan L, Andreoni C, Ortiz V, Silva EK, Pitta R, Atan F, Srougi M (2005) High kidney stone risk in men working in steel industry at hot temperatures. Urology 65(5):858–861
93. Ferraro PM, Bonello M, Frigo AC, D'Addessi A, Sturniolo A, Gambaro G (2011) Cadmium exposure and kidney stone formation in the general population—an analysis of the National Health and Nutrition Examination Survey III data. J Endourol Endourol Soc 25(5):875–880. doi:10.1089/end.2010.0572
94. Elinder CG, Edling C, Lindberg E, Kågedal B, Vesterberg O (1985) Assessment of renal function in workers previously exposed to cadmium. Br J Ind Med 42(11):754–760
95. Trevisan A, Gardin C (2005) Nephrolithiasis in a worker with cadmium exposure in the past. Int Arch Occup Environ Health 78(8):670–672
96. Ding X, Zhang Q, Wei H, Zhang Z (2011) Cadmium-induced renal tubular dysfunction in a group of welders. Occup Med (Lond) 61 (4):277–279
97. Vitayavirasuk B, Junhom S, Tantisaeranee P (2005) Exposure to lead, cadmium and chromium among spray painters in automobile body repair shops. J Occup Health 47(6):518–522
98. Laerum E, Aarseth S (1985) Urolithiasis in railroad shopmen in relation to oxalic acid exposure at work. Scand J Work Environ Health 11(2):97–100
99. Tang X, Li N, Kang L, Dubois AM, Gong Z, Wu B, Lai G, Yang A, Ruan X, Gao H, Zhu G, Ge Y, Zhang J, Lin Z, Olson JR, Ren X (2013) Chronic low level trimethyltin exposure and the risk of developing nephrolithiasis. Occup Environ Med 70(8):561–567. doi:10.1136/oemed-2012-101261

100. Resnick M, Pridgen DB, Goodman HO (1968) Genetic predisposition to formation of calcium oxalate renal calculi. N Engl J Med 278(24):1313–1318. doi:10.1056/NEJM196806132782403
101. Coe FL, Parks JH, Moore ES (1979) Familial idiopathic hypercalciuria. N Engl J Med 300(7):337–340. doi:10.1056/NEJM197902153000703
102. Curhan GC, Willett WC, Rimm EB, Stampfer MJ (1997) Family history and risk of kidney stones. J Am Soc Nephrol JASN 8(10):1568–1573
103. McGeown MG (1960) Heredity in renal stone disease. Clin Sci 19:465–471
104. Stechman MJ, Loh NY, Thakker RV (2007) Genetics of hypercalciuric nephrolithiasis: renal stone disease. Ann N Y Acad Sci 1116:461–484. doi:10.1196/annals.1402.030
105. Goldfarb DS, Fischer ME, Keich Y, Goldberg J (2005) A twin study of genetic and dietary influences on nephrolithiasis: a report from the Vietnam Era Twin (VET) Registry. Kidney Int 67(3):1053–1061. doi:10.1111/j.1523-1755.2005.00170.x
106. Hunter DJ, Lange M, Snieder H, MacGregor AJ, Swaminathan R, Thakker RV, Spector TD (2002) Genetic contribution to renal function and electrolyte balance: a twin study. Clin Sci (Lond) 103(3):259–265. doi:10.1042/cs1030259
107. Monga M, Macias B, Groppo E, Hargens A (2006) Genetic heritability of urinary stone risk in identical twins. J Urol 175(6):2125–2128. doi:10.1016/S0022-5347(06)00272-2
108. Esposito T, Rendina D, Aloia A, Formicola D, Magliocca S, De Filippo G, Muscariello R, Mossetti G, Gianfrancesco F, Strazzullo P (2012) The melatonin receptor 1 A (MTNR1A) gene is associated with recurrent and idiopathic calcium nephrolithiasis. Nephrol Dial Transp 27 (1):210–218. doi:10.1093/ndt/gfr216
109. Hou J (2013) The role of claudin in hypercalciuric nephrolithiasis. Curr Urol Rep 14(1):5–12. doi:10.1007/s11934-012-0289-2
110. Vezzoli G, Terranegra A, Aloia A, Arcidiacono T, Milanesi L, Mosca E, Mingione A, Spotti D, Cusi D, Hou J, Hendy GN, Soldati L, Network G, Paloschi V, Dogliotti E, Brasacchio C, Dell'Antonio G, Montorsi F, Bertini R, Bellinzoni P, Guazzoni G, Borghi L, Guerra A, Allegri F, Ticinesi A, Meschi T, Nouvenne A, Lupo A, Fabris A, Gambaro G, Strazzullo P, Rendina D, De Filippo G, Brandi ML, Croppi E, Cianferotti L, Trinchieri A, Caudarella R, Cupisti A, Anglani F, Del Prete D (2013) Decreased transcriptional activity of calcium-sensing receptor gene promoter 1 is associated with calcium nephrolithiasis. J Clin Endocrinol Metab 98(9):3839–3847. doi:10.1210/jc.2013-1834
111. Vezzoli G, Terranegra A, Soldati L (2012) Calcium-sensing receptor gene polymorphisms in patients with calcium nephrolithiasis. Curr Opin Nephrol Hypertens 21(4):355–361. doi:10.1097/MNH.0b013e3283542290
112. Rungroj N, Sudtachat N, Nettuwakul C, Sawasdee N, Praditsap O, Jungtrakoon P, Sritippayawan S, Chuawattana D, Borvornpadungkitti S, Predanon C, Susaengrat W, Yenchitsomanus PT (2012) Association between human prothrombin variant (T165M) and kidney stone disease. PLoS One 7(9):e45533. doi:10.1371/journal.pone.0045533
113. Vezzoli G, Terranegra A, Rainone F, Arcidiacono T, Cozzolino M, Aloia A, Dogliotti E, Cusi D, Soldati L (2011) Calcium-sensing receptor and calcium kidney stones. J Transl Med 9:201. doi:10.1186/1479-5876-9-201
114. Vezzoli G, Scillitani A, Corbetta S, Terranegra A, Dogliotti E, Guarnieri V, Arcidiacono T, Paloschi V, Rainone F, Eller-Vainicher C, Borghi L, Nouvenne A, Guerra A, Meschi T, Allegri F, Cusi D, Spada A, Cole DE, Hendy GN, Spotti D, Soldati L (2011) Polymorphisms at the regulatory regions of the CASR gene influence stone risk in primary hyperparathyroidism. Eur J Endocrinol 164(3):421–427. doi:10.1530/EJE-10-0915
115. Telci D, Dogan AU, Ozbek E, Polat EC, Simsek A, Cakir SS, Yeloglu HO, Sahin F (2011) KLOTHO gene polymorphism of G395A is associated with kidney stones. Am J Nephrol 33(4):337–343. doi:10.1159/000325505
116. Rungroj N, Sritippayawan S, Thongnoppakhun W, Paemanee A, Sawasdee N, Nettuwakul C, Sudtachat N, Ungsupravate D, Praihirunkit P, Chuawattana D, Akkarapatumwong V, Borvornpadungkitti S, Susaengrat W, Vasuvattakul S, Malasit P, Yenchitsomanus PT (2011) Prothrombin haplotype associated with kidney stone disease in Northeastern Thai patients. Urology 77 (1):249e217–223. doi:10.1016/j.urology.2010.07.494
117. Chou YH, Woon PY, Chen WC, Hsu YW, Chang JM, Hwang DY, Chiu YC, Kuo HC, Chang WP, Hou MF, Liu ME, Chang JG, Chang WC (2011) A genetic polymorphism (rs17251221) in the calcium-sensing receptor gene (CASR) is associated with stone multiplicity in calcium nephrolithiasis. PLoS One 6(9):e25227. doi:10.1371/journal.pone.0025227
118. Onaran M, Yilmaz A, Sen I, Ergun MA, Camtosun A, Kupeli B, Menevse S, Bozkirli I (2009) Heparan sulfate gene polymorphism in calcium oxalate nephrolithiasis. Urol Res 37(1):47–50. doi:10.1007/s00240-008-0167-z
119. Onaran M, Yilmaz A, Sen I, Ergun MA, Camtosun A, Kupeli B, Menevse S, Bozkirli I (2009) A HindIII polymorphism of fibronectin gene is associated with nephrolithiasis. Urology 74(5):1004–1007. doi:10.1016/j.urology.2009.05.010
120. Monico CG, Milliner DS (2012) Genetic determinants of urolithiasis. Nat Rev Nephrol 8(3):151–162. doi:10.1038/nrneph.2011.211
121. Elkoushy MA, Andonian S (2012) Characterization of patients with heterozygous cystinuria. Urology 80(4):795–799. doi:10.1016/j.urology.2012.04.062
122. Taylor EN, Stampfer MJ, Curhan GC (2005) Obesity, weight gain, and the risk of kidney stones. JAMA 293(4):455–462. doi:10.1001/jama.293.4.455
123. Curhan GC, Willett WC, Rimm EB, Speizer FE, Stampfer MJ (1998) Body size and risk of kidney stones. J Am Soc Nephrol JASN 9(9):1645–1652
124. Ekeruo WO, Tan YH, Young MD, Dahm P, Maloney ME, Mathias BJ, Albala DM, Preminger GM (2004) Metabolic risk factors and the impact of medical therapy on the management of nephrolithiasis in obese patients. J Urol 172(1):159–163. doi:10.1097/01.ju.0000128574.50588.97
125. Maalouf NM, Sakhaee K, Parks JH, Coe FL, Adams-Huet B, Pak CY (2004) Association of urinary pH with body weight in nephrolithiasis. Kidney Int 65(4):1422–1425. doi:10.1111/j.1523-1755.2004.00522.x
126. Maalouf NM (2011) Metabolic syndrome and the genesis of uric acid stones. J Renal Nutr 21(1):128–131. doi:10.1053/j.jrn.2010.10.015
127. Abate N, Chandalia M, Cabo-Chan AV Jr, Moe OW, Sakhaee K (2004) The metabolic syndrome and uric acid nephrolithiasis: novel features of renal manifestation of insulin resistance. Kidney Int 65(2):386–392. doi:10.1111/j.1523-1755.2004.00386.x
128. Bobulescu IA, Dubree M, Zhang J, McLeroy P, Moe OW (2008) Effect of renal lipid accumulation on proximal tubule Na+/H + exchange and ammonium secretion. Am J Physiol Renal Physiol 294 (6):F1315–1322. doi:10.1152/ajprenal.00550.2007
129. Bobulescu IA, Dubree M, Zhang J, McLeroy P, Moe OW (2009) Reduction of renal triglyceride accumulation: effects on proximal tubule Na+/H + exchange and urinary acidification. Am J Physiol Renal Physiol 297 (5):F1419-1426. doi:10.1152/ajprenal.00177.2009

130. Taylor EN, Stampfer MJ, Curhan GC (2005) Diabetes mellitus and the risk of nephrolithiasis. Kidney Int 68(3):1230–1235. doi:10.1111/j.1523-1755.2005.00516.x
131. Kohjimoto Y, Sasaki Y, Iguchi M, Matsumura N, Inagaki T, Hara I (2013) Association of metabolic syndrome traits and severity of kidney stones: results from a nationwide survey on urolithiasis in Japan. Am J Kidney Dis 61(6):923–929. doi:10.1053/j.ajkd.2012.12.028
132. Daudon M, Traxer O, Conort P, Lacour B, Jungers P (2006) Type 2 diabetes increases the risk for uric acid stones. J Am Soc Nephrol JASN 17(7):2026–2033. doi:10.1681/ASN.2006030262
133. Cappuccio FP, Siani A, Barba G, Mellone MC, Russo L, Farinaro E, Trevisan M, Mancini M, Strazzullo P (1999) A prospective study of hypertension and the incidence of kidney stones in men. J Hypertens 17(7):1017–1022
134. Borghi L, Meschi T, Guerra A, Briganti A, Schianchi T, Allegri F, Novarini A (1999) Essential arterial hypertension and stone disease. Kidney Int 55(6):2397–2406. doi:10.1046/j.1523-1755.1999.00483.x
135. Madore F, Stampfer MJ, Rimm EB, Curhan GC (1998) Nephrolithiasis and risk of hypertension. Am J Hypertens 11(1 Pt 1):46–53
136. Madore F, Stampfer MJ, Willett WC, Speizer FE, Curhan GC (1998) Nephrolithiasis and risk of hypertension in women. Am J Kidney Dis 32(5):802–807
137. Gillen DL, Coe FL, Worcester EM (2005) Nephrolithiasis and increased blood pressure among females with high body mass index. Am J Kidney Dis 46(2):263–269. doi:10.1053/j.ajkd.2005.04.030
138. Taylor EN, Fung TT, Curhan GC (2009) DASH-style diet associates with reduced risk for kidney stones. J Am Soc Nephrol JASN 20(10):2253–2259. doi:10.1681/ASN.2009030276
139. Taylor EN, Stampfer MJ, Mount DB, Curhan GC (2010) DASH-style diet and 24-hour urine composition. Clin J Am Soc Nephrol CJASN 5(12):2315–2322. doi:10.2215/CJN.04420510
140. Kramer HM, Curhan G (2002) The association between gout and nephrolithiasis: the National Health and Nutrition Examination Survey III, 1988–1994. Am J Kidney Dis 40(1):37–42. doi:10.1053/ajkd.2002.33911
141. Kramer HJ, Choi HK, Atkinson K, Stampfer M, Curhan GC (2003) The association between gout and nephrolithiasis in men: The Health Professionals' Follow-Up Study. Kidney Int 64(3):1022–1026. doi:10.1046/j.1523-1755.2003.t01-2-00171.x
142. West B, Luke A, Durazo-Arvizu RA, Cao G, Shoham D, Kramer H (2008) Metabolic syndrome and self-reported history of kidney stones: the National Health and Nutrition Examination Survey (NHANES III) 1988–1994. Am J Kidney Dis 51(5):741–747. doi:10.1053/j.ajkd.2007.12.030
143. Rendina D, Mossetti G, De Filippo G, Benvenuto D, Vivona CL, Imbroinise A, Zampa G, Ricchio S, Strazzullo P (2009) Association between metabolic syndrome and nephrolithiasis in an inpatient population in southern Italy: role of gender, hypertension and abdominal obesity. Nephrol Dial Transp 24 (3):900–906. doi:10.1093/ndt/gfn548
144. Jeong IG, Kang T, Bang JK, Park J, Kim W, Hwang SS, Kim HK, Park HK (2011) Association between metabolic syndrome and the presence of kidney stones in a screened population. Am J Kidney Dis 58(3):383–388. doi:10.1053/j.ajkd.2011.03.021
145. The World Bank (2014) Population, total. http://data.worldbank.org/indicator/SP.POP.TOTL?end=2014&start=1960. Accessed 1 Oct 2016
146. Mbonu O, Attah C, Ikeakor I (1984) Urolithiasis in an African population. Int Urol Nephrol 16(4):291–296
147. Ekwere PD (1995) Urinary calculous disease in south-eastern Nigeria. Afr J Med Med Sci 24(3):289–295
148. Monu JU (1989) Pattern of urolithiasis in Benin City, Nigeria. J Natl Med Assoc 81(6):695–698
149. Esho JO (1976) Experience with urinary calculus disease in Nigerians as seen at the Lagos University Teaching Hospital. Nigerian Med J 6 (1):18–22
150. Korkes F, Schor N, Heilberg IP (2012) Epidemiology of stone disease in South America. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 61–66
151. Korkes F, Silva II, Heilberg IP (2011) Costs for in hospital treatment of urinary lithiasis in the Brazilian public health system. Einstein (São Paulo) 9 (4):518–522
152. Luo D, Li H, Wang K (2012) Epidemiology of stone disease in China. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 53–59
153. Chan SW, Ng CF, Man CW, Chung R, Li SK (2008) A report on a randomly sampled questionnaire survey about renal stone disease in Hong Kong. Hong Kong Med J 14(6):427–431
154. Kim H, Jo MK, Kwak C, Park SK, Yoo KY, Kang D, Lee C (2002) Prevalence and epidemiologic characteristics of urolithiasis in Seoul, Korea. Urology 59(4):517–521
155. Yanagawa M, Kawamura J, Onishi T, Soga N, Kameda K, Sriboonlue P, Prasongwattana V, Borwornpadungkitti S (1997) Incidence of urolithiasis in northeast Thailand. Int J Urol 4(6):537–540
156. Safarinejad MR (2007) Adult urolithiasis in a population-based study in Iran: prevalence, incidence, and associated risk factors. Urol Res 35(2):73–82. doi:10.1007/s00240-007-0084-6
157. Basiri A, Shakhssalim N, Khoshdel AR, Ghahestani SM, Basiri H (2010) The demographic profile of urolithiasis in Iran: a nationwide epidemiologic study. Int Urol Nephrol 42(1):119–126. doi:10.1007/s11255-009-9588-z
158. Ketabchi AA, Aziziolahi GA (2008) Prevalence of symptomatic urinary calculi in Kerman, Iran. Urol J 5(3):156–160
159. Singh PP, Barjatiya MK, Dhing S, Bhatnagar R, Kothari S, Dhar V (2001) Evidence suggesting that high intake of fluoride provokes nephrolithiasis in tribal populations. Urol Res 29(4):238–244
160. el-Reshaid K, Mughal H, Kapoor M (1997) Epidemiological profile, mineral metabolic pattern and crystallographic analysis of urolithiasis in Kuwait. Eur J Epidemiol 13(2):229–234
161. Lee MC, Bariol SV (2012) Epidemiology of stone disease in Australia. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 73–76
162. Davidson PJ, Sheerin IG, Frampton C (2009) Renal stone disease in Christchurch, New Zealand. Part 1: presentation and epidemiology. N Z Med J 122(1297):49–56
163. Sanchez-Martin FM, Millan Rodriguez F, Esquena Fernandez S, Segarra Tomas J, Rousaud Baron F, Martinez-Rodriguez R, Villavicencio Mavrich H (2007) Incidence and prevalence of published studies about urolithiasis in Spain. A review. Actas Urol Esp 31(5):511–520
164. Hesse A, Brandle E, Wilbert D, Kohrmann KU, Alken P (2003) Study on the prevalence and incidence of urolithiasis in Germany comparing the years 1979 vs. 2000. Eur Urol 44(6):709–713
165 Trinchieri A, Coppi F, Montanari E, Del Nero A, Zanetti G, Pisani E (2000) Increase in the prevalence of symptomatic upper urinary tract stones during the last ten years. Eur Urol 37(1):23–25
166 Indridason OS, Birgisson S, Edvardsson VO, Sigvaldason H, Sigfusson N, Palsson R (2006) Epidemiology of kidney stones in Iceland A population-based study 1. Scand J Urol Nephrol 40(3):215–220

167 Ljunghall S (1987) Incidence of upper urinary tract stones. Miner Electrolyte Metab 13(4):220–227

168 Muslumanoglu AY, Binbay M, Yuruk E, Akman T, Tepeler A, Esen T, Tefekli AH (2011) Updated epidemiologic study of urolithiasis in Turkey. I: Changing characteristics of urolithiasis. Urol Res 39(4):309–314. doi:10.1007/s00240-010-0346-6

169 Novikov A, Nazarov T, Startsev VY (2012) Epidemiology of stone disease in the Russian Federation and Post Soviet Era. In: Talati J, Tiselius HG, Albala DM, Ye Z (eds) Urolithiasis, basic science and clinical practice. Springer, London, pp 97–105