ARTHRITIS & RHEUMATISM
Vol. 63, No. 10, October 2011, pp 3136–3141
DOI 10.1002/art.30520

Prevalence of Gout and Hyperuricemia in the US General Population

The National Health and Nutrition Examination Survey 2007–2008

Yanyan Zhu,[1] Bhavik J. Pandya,[2] and Hyon K. Choi[1]

***Objective*. To estimate the prevalence of gout and hyperuricemia based on the latest nationally representative sample of US men and women (National Health and Nutrition Examination Survey [NHANES] 2007–2008).**

***Methods*. Using data from 5,707 participants in NHANES 2007–2008, we estimated the prevalence of gout and hyperuricemia. During home interviews for NHANES 2007–2008, all participants were asked about a history of health professional– or physician-diagnosed gout. Our primary definition of hyperuricemia was a serum urate level of >7.0 mg/dl for men and >5.7 mg/dl for women. We explored potential secular trends in these estimates and their possible explanations by comparing them with estimates based on 18,825 participants in NHANES-III (1988–1994).**

***Results*. The prevalence of gout among US adults in 2007–2008 was 3.9% (8.3 million individuals). The prevalence among men was 5.9% (6.1 million), and the prevalence among women was 2.0% (2.2 million). The mean serum urate levels were 6.14 mg/dl among men and 4.87 mg/dl among women, corresponding to hyperuricemia prevalences of 21.2% and 21.6%, respectively. These estimates were higher than those in NHANES-III, with differences of 1.2% in the prevalence of gout (95% confidence interval [95% CI] 0.6, 1.9), 0.15 mg/dl in the serum urate level (95% CI 0.07, 0.24), and 3.2% in the prevalence of hyperuricemia (95% CI 1.2, 5.2). These differences were substantially attenuated after adjusting for body mass index and/or hypertension.**

***Conclusion*. These findings from nationally representative samples of US adults suggest that the prevalence of both gout and hyperuricemia remains substantial and may have increased over the past 2 decades, which is likely related to increasing frequencies of adiposity and hypertension.**

Gout is an inflammatory arthritis associated with hyperuricemia that is triggered by the crystallization of uric acid within the joints. Gout leads to substantial morbidity by causing severe pain. Furthermore, emerging evidence suggests that gout is strongly associated with the metabolic syndrome (1) and may lead to myocardial infarction (2–4), type 2 diabetes mellitus (5), and premature death (3,6).

The prevalence of gout in the US more than doubled between the 1960s and the 1990s (7), but it is unknown whether this trend continues in the new millennium. To address this issue, we determined the prevalence of gout based on the latest US National Health and Nutrition Examination Survey (NHANES) conducted in 2007 and 2008. We also calculated the prevalence of hyperuricemia and mean serum urate levels, using the same NHANES data. Finally, we compared these estimates with those from NHANES-III (1988–1994).

PATIENTS AND METHODS

Study population. The NHANES studies are designed to assess the health and nutritional status of adults and

Supported by Takeda Pharmaceuticals International.

[1]Yanyan Zhu, PhD, Hyon K. Choi, MD, DrPH: Boston University School of Medicine, Boston, Massachusetts; [2]Bhavik J. Pandya, PharmD: Takeda Pharmaceuticals International, Deerfield, Illinois.

Dr. Choi has received honoraria for service on advisory boards for Takeda Pharmaceuticals, URL Pharma, Savient Pharmaceuticals, and Novartis Pharma (less than $10,000 each) and has received research funding for other projects from Takeda Pharmaceuticals.

Address correspondence to Hyon K. Choi, MD, DrPH, Section of Rheumatology and the Clinical Epidemiology Unit, Boston University School of Medicine, 650 Albany Street, Suite 200, Boston, MA 02118. E-mail: hchoius@bu.edu.

Submitted for publication March 8, 2011; accepted in revised form June 21, 2011.

children in the US. These studies are based on a representative sample of the noninstitutionalized US civilian population that is selected using a multistage, stratified sampling design. The survey is unique in that it combines interviews, physical examinations, and various laboratory data (http://www.cdc.gov/nchs/nhanes/about_nhanes.htm). In the current analysis, we used the 2 most recent NHANES studies, NHANES-III (1988–1994) and NHANES 2007–2008, in which participants were asked about the presence of gout. After a home interview, participants were invited to attend examination sessions during which blood and urine specimens were obtained. NHANES 2007–2008 included 5,707 participants (2,797 men and 2,910 women), and NHANES-III (1988–1994) included 18,825 participants (8,816 men and 10,009 women), all of whom were at least 20 years of age and underwent a medical examination. In the current study, we analyzed individuals for whom complete information was available for gout status (n = 5,699 in NHANES 2007–2008 and n = 18,822 in NHANES-III) and serum urate levels (n = 5,313 in NHANES 2007–2008 and n = 15,838 in NHANES-III).

Assessment of gout. During the home interviews for NHANES 2007–2008, all participants were asked, "Has a doctor or other health professional ever told you that you had gout?" Similarly, during the home interview for NHANES-III (1988–1994), participants were asked, "Has a doctor ever told you that you had gout?"

Serum urate measurement and definitions of hyperuricemia. In both NHANES 2007–2008 and NHANES-III, serum urate levels were measured by oxidization with the specific enzyme uricase to form allantoin and H_2O_2. Details of the quality control procedures have been published elsewhere (8) (http://www.cdc.gov/nchs/data/nhanes/nhanes3/cdrom/nchs/manuals/labman.pdf); the serum urate levels measured in NHANES studies have been used in previous studies (9–11). The values for serum urate levels are reported in milligrams per deciliter and can be converted to micromoles per liter by multiplying by 59.48. Our primary definition of hyperuricemia was a serum urate level of >7.0 mg/dl in men and >5.7 mg/dl in women, based on the NHANES-III laboratory definition (8). We also examined the potential impact of alternative definitions of hyperuricemia regardless of sex (i.e., a serum urate level of >7.0 mg/dl, which is above the supersaturation point, and a serum urate level of >6.0 mg/dl, which is a widely accepted therapeutic target) (12–14).

Statistical analysis. All statistical analyses were performed using Stata survey commands (i.e., SVY) to adjust for clusters and strata of the complex sample design and to incorporate sample weights (Stata version 11.1). These procedures generated estimates for the total noninstitutionalized civilian population of the US. The prevalence of gout and hyperuricemia as well as mean serum urate levels were first calculated in the US adult population and then according to sex, race, and age. Temporal changes in these estimates were assessed based on 95% confidence interval (95% CI) estimates of the difference between time-period prevalence estimates. Additionally, we performed logistic regression to evaluate the association between the temporal order of the 2 NHANES studies and the prevalence of gout and calculated the odds ratios (ORs) before and after adjusting for potential contributors to changes in the risk of gout. We adjusted for these factors in a stepwise manner, including age, sex, body mass index (BMI), hypertension, use of diuretics, and alcohol intake. For all measures, we calculated the 95% CIs. All reported *P* values are 2-sided.

RESULTS

Prevalence of gout in the US in 2007–2008. In NHANES 2007–2008, the mean age of the participants was 47 years, 48.2% were men, and 69.4% were white. The overall prevalence of gout among US adults was 3.9%, which corresponded to an estimated 8.3 million adults with gout in the US in 2007 and 2008 (Table 1). The prevalence of gout was 5.9% among men (6.1 million) and 2.0% among women (2.2 million). The prevalence of gout increased with age, with the lowest prevalence (0.4% [0.2 million]) in individuals ages 20–29 years and the highest prevalence (12.6% [1.2 million]) among those ages 80 years or older (Table 1). The prevalence of gout among individuals ages 65 years or older (i.e., the group that receives Medicare) was 9.8%, which corresponded to an estimated 3.5 million US adults in this age group with gout.

Prevalence of hyperuricemia and mean serum urate levels in the US in 2007–2008. The overall prevalence of hyperuricemia among US adults was 21.4%, which corresponded to an estimated 43.3 million individuals with hyperuricemia (Table 2). The prevalence of hyperuricemia was 21.2% among men (20.7 million) and 21.6% among women (22.6 million). The prevalence of

Table 1. Prevalence of gout and number of affected adults in the US, NHANES 2007–2008*

	Prevalence, % (95% CI)	No. of affected US adults, million
Overall	3.9 (3.3, 4.4)	8.3
Sex		
Male	5.9 (4.7, 7.1)	6.1
Female	2.0 (1.5, 2.5)	2.2
Race/ethnicity		
White	4.0 (3.3, 4.8)	6.0
African American	5.0 (3.3, 6.6)	1.2
Mexican American	1.5 (1.0, 2.0)	0.3
Other	3.4 (1.2, 5.6)	0.8
Age category, years		
20–29	0.4 (0.0, 0.9)	0.2
30–39	1.3 (0.5, 2.0)	0.5
40–49	3.3 (1.8, 4.9)	1.5
50–59	3.7 (3.0, 4.4)	1.5
60–69	8.0 (5.8, 10.3)	2.0
70–79	9.3 (6.5, 12.0)	1.5
80+	12.6 (10.1, 15.1)	1.2

* The data were adjusted for clusters and strata of the complex sample design of the National Health and Nutrition Examination Survey (NHANES) 2007–2008, with incorporation of sample weights. 95% CI = 95% confidence interval.

Table 2. Prevalence of hyperuricemia, number of US adults with hyperuricemia, and mean serum urate levels, NHANES 2007–2008*

	Prevalence of hyperuricemia, % (95% CI)†	No. of US adults with hyperuricemia, million†	Prevalence of hyperuricemia >7 mg/dl, % (95% CI)	No. of US adults with hyperuricemia >7 mg/dl, million	Serum urate level, mean (95% CI) mg/dl
Overall	21.4 (19.6, 23.3)	43.3	13.2 (11.7, 14.6)	26.6	5.48 (5.41, 5.56)
Sex					
Male	21.2 (18.9, 23.5)	20.7	21.2 (18.9, 23.5)	20.7	6.14 (6.06, 6.23)
Female	21.6 (18.9, 24.4)	22.6	5.7 (4.4, 6.9)	5.9	4.87 (4.79, 4.94)
Race/ethnicity					
White	22.1 (19.7, 24.4)	31.3	13.4 (11.4, 15.4)	19.0	5.51 (5.41, 5.60)
African American	25.7 (22.8, 28.6)	5.3	15.3 (13.1, 17.6)	3.2	5.53 (5.40, 5.67)
Mexican American	17.1 (14.2, 19.9)	2.9	11.4 (8.5, 14.4)	2.0	5.36 (5.21, 5.50)
Other	16.7 (13.5, 19.8)	3.8	11.0 (7.7, 14.4)	2.5	5.39 (5.25, 5.54)
Age category, years					
20–29	18.6 (14.0, 23.1)	6.9	13.0 (9.7, 16.4)	4.8	5.36 (5.25, 5.46)
30–39	15.3 (12.9, 17.7)	5.7	9.7 (7.1, 12.3)	3.6	5.33 (5.22, 5.44)
40–49	17.9 (14.9, 20.9)	7.5	10.6 (8.0, 13.1)	4.4	5.40 (5.26, 5.53)
50–59	22.6 (18.6, 26.6)	8.6	14.5 (11.2, 17.9)	5.5	5.56 (5.46, 5.66)
60–69	27.7 (22.9, 32.5)	6.7	15.5 (11.4, 19.7)	3.8	5.66 (5.52, 5.80)
70–79	31.5 (27.8, 35.1)	4.8	17.5 (14.6, 20.3)	2.6	5.78 (5.66, 5.91)
80+	36.8 (29.0, 44.7)	3.2	20.7 (16.4, 25.0)	1.8	5.79 (5.58, 6.00)

* The data were adjusted for clusters and strata of the complex sample design of the National Health and Nutrition Examination Survey (NHANES) 2007–2008, with incorporation of sample weights. 95% CI = 95% confidence interval.
† Hyperuricemia was defined as serum urate level of >7.0 mg/dl in men and >5.7 mg/dl in women.

serum urate levels >7 mg/dl regardless of sex was 13.2% overall (26.6 million), with a prevalence of 21.2% among men and 5.7% among women. The prevalence of serum urate levels >6 mg/dl regardless of sex was 32.8% overall (66.3 million), with a prevalence of 50.4% among men and 16.3% among women. The overall mean serum urate level was 5.48 mg/dl (95% CI 5.41, 5.56), corresponding to a mean level of 6.14 mg/dl (95% CI 6.06, 6.23) among men and 4.87 mg/dl (95% CI 4.79, 4.94) among women. The prevalence of hyperuricemia increased with age, with the highest prevalence (36.8% [3.2 million]) among individuals ages 80 years or older (Table 2). The prevalence of hyperuricemia among individuals ages 65 years or older was 31.4%, which corresponded to an estimated 10.7 million US adults in this age group with hyperuricemia.

Comparison with NHANES-III estimates. The prevalence of gout in NHANES 2007–2008 (3.9% [95% CI 3.3, 4.4]) was significantly higher than the prevalence estimate in NHANES-III (2.7% [95% CI 2.3, 3.0]), with a difference of 1.2% (95% CI 0.6, 1.9) (Table 3). The age-adjusted difference was 1.0% (95% CI 0.4, 1.7). Similarly, the prevalence of hyperuricemia in NHANES 2007–2008 was significantly higher than that in NHANES-III (1988–1994), with an unadjusted difference of 3.2% (95% CI 1.2, 5.2) and an age-adjusted difference of 2.4% (95% CI 0.7, 4.2). Correspondingly, the mean serum urate level significantly increased over

Table 3. Unadjusted and age-adjusted comparison of the prevalence of gout and hyperuricemia among US adults between NHANES-III (1988–1994) and NHANES 2007–2008*

	NHANES-III	NHANES 2007–2008	Difference
Prevalence of gout			
Unadjusted	2.7 (2.3, 3.0)	3.9 (3.3, 4.4)	1.2 (0.6, 1.9)
Age-adjusted	2.9 (2.5, 3.3)	3.9 (3.4, 4.5)	1.0 (0.4, 1.7)
Prevalence of hyperuricemia			
Unadjusted	18.2 (17.2, 19.3)	21.4 (19.7, 23.2)	3.2 (1.2, 5.2)
Age-adjusted	19.1 (18.1, 20.0)	21.5 (20.1, 23.0)	2.4 (0.7, 4.2)
Mean serum urate level, mg/dl			
Unadjusted	5.33 (5.29, 5.37)	5.48 (5.41, 5.55)	0.15 (0.07, 0.24)
Age-adjusted	5.36 (5.32, 5.40)	5.49 (5.44, 5.53)	0.13 (0.07, 0.18)

* Values are the percent (95% confidence interval). The data were adjusted for clusters and strata of the complex sample design of the National Health and Nutrition Examination Survey (NHANES) 2007–2008, with incorporation of sample weights.

Table 4. Odds ratios for gout between NHANES-III and NHANES 2007–2008*

	Patients with gout	
	NHANES-III (n = 466)	NHANES 2007−2008 (n = 259)
Unadjusted	1.0 (referent)	1.75 (1.50, 2.04)
Adjusted		
Age and sex	1.0 (referent)	1.74 (1.48, 2.04)
Age, sex, and race	1.0 (referent)	1.74 (1.48, 2.05)
Age, sex, race, and BMI	1.0 (referent)	1.44 (1.22, 1.70)
Age, sex, race, BMI, and hypertension	1.0 (referent)	1.21 (1.02, 1.43)
Age, sex, race, BMI, hypertension, and diuretics	1.0 (referent)	1.19 (1.01, 1.41)
Age, sex, race, BMI, hypertension, diuretics, and alcohol	1.0 (referent)	1.17 (0.99, 1.39)

* Values are the odds ratio (95% confidence interval), based on logistic regression for the association between the odds of gout in the National Health and Nutrition Examination Survey III (NHANES-III) and NHANES 2007–2008, with stepwise adjustments of potential contributors. BMI = body mass index.

the same period of time (difference of 0.15 mg/dl [95% CI 0.07, 0.24]).

We performed logistic regression analyses by pooling the 2 NHANES studies together to evaluate the potential impact of key risk factors for gout on the increasing trend identified above. Compared with the NHANES-III cohort, the age- and sex-adjusted OR for gout in NHANES 2007–2008 was 1.74 (95% CI 1.48, 2.04) (Table 4). Although the OR remained the same after adjusting for race, further stepwise adjustment for BMI and hypertension reduced the OR to 1.21 (95% CI 1.02, 1.43). When we additionally adjusted for treatment with diuretics and alcohol consumption, the association was further attenuated and became nonsignificant (multivariate OR 1.17 [95% CI 0.99, 1.39]).

Similarly, compared with the NHANES-III cohort, the age-, sex-, and race-adjusted OR for hyperuricemia in NHANES 2007–2008 was 1.18 (95% CI 1.09, 1.28). After adjusting for BMI or hypertension, this OR was attenuated substantially and became nonsignificant (OR 0.96 [95% CI 0.89, 1.05] and OR 1.05 [95% CI 0.97, 1.14], respectively). Correspondingly, the unweighted mean serum urate level in NHANES 2007–2008 was 0.16 mg/dl (95% CI 0.12, 0.21) higher than the level observed in NHANES-III. After adjusting for age, sex, race, and BMI or hypertension, these differences were attenuated substantially (difference of −0.02 mg/dl [95% CI −0.07, 0.02] and difference of 0.05 mg/dl [95% CI 0.00, 0.09], respectively).

DISCUSSION

In this nationally representative sample of US men and women, we observed that the prevalence of both gout and hyperuricemia was substantial. Specifically, health professional– or physician-diagnosed gout in 2007–2008 was reported in 3.9% of US adults, which translates into 8.3 million US adults. Approximately 43.3 million US adults (21.4%) met sex-specific criteria for hyperuricemia (i.e., serum urate levels of >7.0 mg/dl for men and >5.7 mg/dl for women), whereas the prevalence of hyperuricemia defined as a serum urate level of >7 mg/dl was 13.2% (26.6 million US adults). These latest prevalence estimates help determine the burden of these conditions on the US healthcare system, especially in an increasingly aging population.

According to the data gathered before the new millennium, the disease burden of gout was already substantial and increasing worldwide. For example, a multicenter study of general practices in the UK showed that the prevalence of gout in 1991 had increased 3-fold compared with estimates from the 1970s (15). Similarly, the prevalence of gout has been noted to be increasing in New Zealand (16) and urban African communities (17). The Rochester Epidemiology project showed that the incidence of primary gout (i.e., without diuretic exposure) doubled between the 1970s and 1990s, whereas the proportion of gout associated with diuretic use decreased significantly during that period (18). Furthermore, a study based on a US managed-care population showed that the overall prevalence of gout or hyperuricemia requiring serum urate–lowering or other gout medication in 1999 increased by 80% compared with that in 1990 (19). The prevalence estimates of gout in the US based on NHANES-III (1988–1994) (20) were >2% in men older than age 30 years and women older than age 50 years (20). The prevalence increased with age, to 9% in men and 6% in women older than age 80 years (20). Western diets, sedentary lifestyle, an increased frequency of obesity and hypertension, and increased use of diuretics and aspirin have been suspected to contribute to this increase before the new millennium (21).

Our findings suggest that the substantial prevalence of both gout and hyperuricemia has been sustained during the last 2 decades and indeed may still be increasing. Specifically, in comparison with the estimates from NHANES-III (1988–1994), we observed significant increases in the prevalence of gout and in serum urate levels. An obvious potential explanation for the former observation is that NHANES 2007–2008 inquired about

health professional– or physician-diagnosed gout, whereas NHANES-III asked about physician-diagnosed gout. However, our pooled analysis of the NHANES-III and NHANES 2007–2008 studies suggests that the difference in the prevalence of gout could also be related to an increased frequency of hypertension, obesity, diuretic use, and alcohol intake, because these factors substantially reduced the secular trend. Furthermore, our analysis of the outcome of hyperuricemia and serum urate levels indicated that the substantial difference in these outcomes could be explained by increasing obesity or hypertension in recent years, as compared with the NHANES-III study period (22,23). It should be noted that these latter measures in the NHANES studies are based on laboratory tests and thus do not rely on self-reports by participants (as was the case for the assessment of gout).

To help halt or reduce the increasing burden of gout and hyperuricemia, prospective data on modifiable risk factors for these conditions should be considered, including lifestyle and dietary factors (obesity, alcohol, fructose, purine-rich fatty foods), certain drugs (thiazide and loop diuretics), and disease conditions (hypertension, renal insufficiency, and heart failure) (24). Furthermore, these approaches should take into account both associated benefits and associated risks in a holistic manner, because gout is often associated with many important comorbidities including the metabolic syndrome and an increased future risk of cardiovascular disease and mortality (24). For example, reducing body weight with daily exercise (25–27) and limiting intake of red meat (28,29) and sugary beverages (10,30–32) would help reduce uric acid levels, the risk of gout, insulin resistance, and comorbidities. Heavy drinking should be avoided (33,34), while moderate drinking, consumption of sweet fruits, and seafood intake (particularly oily fish) should be tailored to the individual, considering their anticipated health benefits against cardiovascular disease (24). Dairy products, vegetables, nuts, legumes, fruits (those that are less sugary), and whole grains are healthy dietary choices for the comorbidities of gout and would likely help prevent gout by reducing insulin resistance and thus inducing urinary urate excretion (24,28). Coffee drinking (regular or decaffeinated) (35–37) and vitamin C supplementation (38) may be considered as long-term preventive measures, because they can lower serum urate levels as well as the risk of gout and some associated comorbidities (24).

The strengths and limitations of our study deserve comment. This study was performed in a nationally representative sample of US men and women; thus, the findings are likely to be generalizable to the US population. Unlike estimates of serum urate levels that are based on objective measures, gout prevalence estimates in the NHANES studies are based on self-reports and are thus likely inflated, similar to other condition estimates based on the NHANES studies. However, we cannot rule out the possibility that the survey might have missed gout cases that were not diagnosed by health care professionals. Data from the Sudbury study showed that 44% of self-reported cases of gout could be validated according to the Rome or New York criteria (39). However, the validation rate from a physician cohort (Johns Hopkins Precursor Study [25]) was much higher: 80% according to the American College of Rheumatology (ACR) survey criteria for gout (40) applied by mail and 100% by mail combined with medical record review (41). Similarly, the validation rate of self-reported gout in a recent large prospective cohort of male health professionals (Health Professionals Follow-Up Study [26]) was ~70% according to the ACR survey criteria as assessed by a mailed survey (28). However, even if the true prevalences of gout were 50% lower, they would still be substantial. Furthermore, our gout trend data were consistent with the results based on increasing serum urate levels, the precursor of gout.

In conclusion, these findings from nationally representative samples of US adults indicate that the prevalences of gout and hyperuricemia continue to be substantial in the new millennium. These data also suggest that the scope of these conditions may have been increasing in the US over the past 2 decades, and this trend may be explained by the increasing frequency of risk factors for hyperuricemia, such as obesity and hypertension (22,23). Better management of these factors could help prevent further increases in the burden of gout, hyperuricemia, and some associated complications in the US.

AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. Choi had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Zhu, Pandya, Choi.

Acquisition of data. Zhu, Choi.

Analysis and interpretation of data. Zhu, Pandya, Choi.

ROLE OF THE STUDY SPONSOR

Dr. Pandya of Takeda Pharmaceuticals International participated in the study design and review and interpretation of results. Takeda Pharmaceuticals funded the project and provided editorial and proofreading assistance. Drs. Zhu and Choi were responsible for the

final content of the manuscript and for the decision to submit it for publication. Publication of this manuscript was not contingent on approval from Takeda.

REFERENCES

1. Choi HK, Ford ES, Li C, Curhan G. Prevalence of the metabolic syndrome in patients with gout: the Third National Health and Nutrition Examination Survey. Arthritis Rheum 2007;57:109–15.
2. Abbott RD, Brand FN, Kannel WB, Castelli WP. Gout and coronary heart disease: the Framingham Study. J Clin Epidemiol 1988;41:237–42.
3. Choi HK, Curhan G. Independent impact of gout on mortality and risk for coronary heart disease. Circulation 2007;116:894–900.
4. Krishnan E, Baker JF, Furst DE, Schumacher HR. Gout and the risk of acute myocardial infarction. Arthritis Rheum 2006;54: 2688–96.
5. Choi HK, De Vera MA, Krishnan E. Gout and the risk of type 2 diabetes among men with a high cardiovascular risk profile. Rheumatology (Oxford) 2008;47:1567–70.
6. Krishnan E, Svendsen K, Neaton JD, Grandits G, Kuller LH. Long-term cardiovascular mortality among middle-aged men with gout. Arch Intern Med 2008;168:1104–10.
7. Lawrence RC, Felson DT, Helmick CG, Arnold LM, Choi H, Deyo RA, et al, for the National Arthritis Data Workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States: Part II. Arthritis Rheum 2008;58: 26–35.
8. Centers for Disease Control and Prevention. The Third National Health and Nutrition Examination Survey (NHANES III 1988–94) reference manuals and reports [CD-ROM]. Hyattsville (MD): National Center for Health Statistics; 1996.
9. Choi HK, Ford ES. Prevalence of the metabolic syndrome in individuals with hyperuricemia. Am J Med 2007;120:442–7.
10. Nguyen S, Choi HK, Lustig RH, Hsu CY. Sugar-sweetened beverages, serum uric acid, and blood pressure in adolescents. J Pediatr 2009;154:807–13.
11. Afzali A, Weiss NS, Boyko EJ, Ioannou GN. Association between serum uric acid level and chronic liver disease in the United States. Hepatology 2010;52:578–89.
12. Becker MA, Schumacher HR Jr, Wortmann RL, MacDonald PA, Eustace D, Palo WA, et al. Febuxostat compared with allopurinol in patients with hyperuricemia and gout. N Engl J Med 2005;353: 2450–61.
13. Zhang W, Doherty M, Bardin T, Pascual E, Barskova V, Conaghan P, et al. EULAR evidence based recommendations for gout. Part II: management. Report of a task force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT). Ann Rheum Dis 2006;65:1312–24.
14. Perez-Ruiz F, Liote F. Lowering serum uric acid levels: what is the optimal target for improving clinical outcomes in gout? Arthritis Rheum 2007;57:1324–8.
15. Harris CM, Lloyd DC, Lewis J. The prevalence and prophylaxis of gout in England. J Clin Epidemiol 1995;48:1153–8.
16. Klemp P, Stansfield SA, Castle B, Robertson MC. Gout is on the increase in New Zealand. Ann Rheum Dis 1997;56:22–6.
17. Cassim B, Mody GM, Deenadayalu VK, Hammond MG. Gout in black South Africans: a clinical and genetic study. Ann Rheum Dis 1994;53:759–62.
18. Arromdee E, Michet CJ, Crowson CS, O'Fallon WM, Gabriel SE. Epidemiology of gout: is the incidence rising? J Rheumatol 2002;29:2403–6.
19. Wallace KL, Riedel AA, Joseph-Ridge N, Wortmann R. Increasing prevalence of gout and hyperuricemia over 10 years among older adults in a managed care population. J Rheumatol 2004;31: 1582–7.
20. Kramer HM, Curhan G. The association between gout and nephrolithiasis: the National Health and Nutrition Examination Survey III, 1988–1994. Am J Kidney Dis 2002;40:37–42.
21. Bieber JD, Terkeltaub RA. Gout: on the brink of novel therapeutic options for an ancient disease [review]. Arthritis Rheum 2004;50:2400–14.
22. Ogden CL, Carroll, MD. Prevalence of overweight, obesity, and extreme obesity among adults: United States, Trends 1960–1962 through 2007–2008. URL: http://www.cdc.gov/nchs/data/hestat/obesity_adult_07_08/obesity_adult_07_08.pdf.
23. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. JAMA 2003;290:199–206.
24. Choi HK. A prescription for lifestyle change in patients with hyperuricemia and gout. Curr Opin Rheumatol 2010;22:165–72.
25. Roubenoff R, Klag MJ, Mead LA, Liang KY, Seidler AJ, Hochberg MC. Incidence and risk factors for gout in white men. JAMA 1991;266:3004–7.
26. Choi HK, Atkinson K, Karlson EW, Curhan G. Obesity, weight change, hypertension, diuretic use, and risk of gout in men: the health professionals follow-up study. Arch Intern Med 2005;165: 742–8.
27. Williams PT. Effects of diet, physical activity and performance, and body weight on incident gout in ostensibly healthy, vigorously active men. Am J Clin Nutr 2008;87:1480–7.
28. Choi HK, Atkinson K, Karlson EW, Willett WC, Curhan G. Purine-rich foods, dairy and protein intake, and the risk of gout in men. N Engl J Med 2004;350:1093–103.
29. Choi HK, Liu S, Curhan G. Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid: the Third National Health and Nutrition Examination Survey. Arthritis Rheum 2005;52:283–9.
30. Gao X, Qi L, Qiao N, Choi HK, Curhan G, Tucker KL, et al. Intake of added sugar and sugar-sweetened drink and serum uric acid concentration in US men and women. Hypertension 2007;50: 306–12.
31. Choi JW, Ford ES, Gao X, Choi HK. Sugar-sweetened soft drinks, diet soft drinks, and serum uric acid level: the Third National Health and Nutrition Examination Survey. Arthritis Rheum 2008; 59:109–16.
32. Choi HK, Curhan G. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. BMJ 2008;336: 309–12.
33. Choi HK, Atkinson K, Karlson EW, Willett WC, Curhan G. Alcohol intake and risk of incident gout in men: a prospective study. Lancet 2004;363:1277–81.
34. Choi HK, Curhan G. Beer, liquor, and wine consumption and serum uric acid level: the Third National Health and Nutrition Examination Survey. Arthritis Rheum 2004;51:1023–9.
35. Choi HK, Curhan G. Coffee, tea, and caffeine consumption and serum uric acid level: the Third National Health and Nutrition Examination Survey. Arthritis Rheum 2007;57:816–21.
36. Choi HK, Willett W, Curhan G. Coffee consumption and risk of incident gout in men: a prospective study. Arthritis Rheum 2007;56:2049–55.
37. Choi HK, Curhan G. Coffee consumption and risk of incident gout in women: the Nurses' Health Study. Am J Clin Nutr 2010;92: 922–7.
38. Choi HK, Gao X, Curhan G. Vitamin C intake and the risk of gout in men: a prospective study. Arch Intern Med 2009;169:502–7.
39. O'Sullivan JB. Gout in a New England town: a prevalence study in Sudbury, Massachusetts. Ann Rheum Dis 1972;31:166–9.
40. Wallace SL, Robinson H, Masi AT, Decker JL, McCarty JD, Yu TF. Preliminary criteria for the classification of the acute arthritis of primary gout. Arthritis Rheum 1977;20:895–900.
41. Roubenoff R. Gout and hyperuricemia. Rheum Dis Clin North Am 1990;16:539–50.