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The use of anthropometric measures in the prediction of incident gout: results from a Swedish community-based cohort study

P Wändell[1], A Andreasson[2,3], H Hagström[3], MC Kapetanovic[4,5], AC Carlsson[1]

[1]Division of Family Medicine and Primary Care, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Huddinge, Sweden
[2]Stress Research Institute, Stockholm University, Stockholm, Sweden
[3]Department of Medicine, Solna, Karolinska Institutet, Stockholm, Sweden
[4]Department of Clinical Sciences, Lund, Section for Rheumatology, Lund University, Lund, Sweden
[5]Section for Rheumatology in Lund and Malmö, Skåne University Hospital, Lund, Sweden

Objectives: To study associations between different anthropometric measures and incident gout, and to find the best predictive measure.
Method: We used the baseline investigation from the Malmö Diet and Cancer study, excluding cases of prevalent gout (n = 28 081). Cox regression for each anthropometric measurement was calculated per standard deviation increment for men and women, with hazard ratios (HRs) and 95% confidence intervals (CIs), using a hospital diagnosis of incident gout (M10) during follow-up as the outcome. Incremental C-statistics for each anthropometric measure were used to determine the measure with the best predictive capacity, in models adjusted for age, socio-economic data, lifestyle factors, comorbidities, and antihypertensive medications.
Results: The study population included 11 049 men and 17 032 women, with 633 incident gout cases, 393 in men (3.6%) and 240 in women (1.4%). For both men and women, the five anthropometric measurements with highest C-statistics were weight, body mass index (BMI), waist circumference (WC), hip circumference, and waist-to-height ratio; in men, the measurement with the highest C-statistic was BMI (0.7361; fully adjusted HR 1.52, 95% CI 1.39–1.68), and in women WC (0.8085; fully adjusted HR 1.62, 95% CI 1.46–1.81). The increment in C-statistic with anthropometric measures was good, around 0.035. Waist-to-hip ratio, waist-to-hip-to-height ratio, body fat percentages, and especially A Body Shape Index had lower C-statistics.
Conclusions: Both BMI and WC showed good predictive ability for incident gout. The clinically used cut-offs for BMI and WC appeared to be relevant in the assessment of increased risk of gout.

Gout is a common disorder, with a prevalence in Sweden between 1.4% and 1.8% (1–3), depending on the definition. A systematic review and meta-regression analysis concluded that there is a large variation in the prevalence of gout depending on the sex distribution, with a higher prevalence in men, the continent being studied, and the definition of gout (4). According to an Australian review, the prevalence of gout in Australia has increased during a 30 year period from 0.5% to 1.7% (5).

Obesity is increasing worldwide and has been proposed as the main upstream driver of cardiometabolic risk factors and associated outcomes such as hypertension, diabetes, and myocardial infarction (6). Moreover, obesity is the leading risk factor for mortality in the world (7). Obesity is also predictive of incident gout, with greater body mass index (BMI) showing an increased risk of gout in both men and women (8–10). However, BMI is an imperfect measure of obesity since it risks misclassifying people with a large muscular mass as overweight or obese (11), and less is known about the association between other anthropometric measures and the development of gout.

Gout is associated with several concomitant diseases that may influence the health of the patient with gout (12). Gout is a metabolic disorder, and gout and hyperuricaemia are associated with insulin resistance (13), the metabolic syndrome, and type 2 diabetes mellitus (14). There is also a strong relationship between gout and hyperuricaemia with hypertension, and with diuretic treatment (12, 15), especially with thiazide diuretics (16, 17). Chronic

Per Wändell, Division of Family Medicine and Primary Care, NVS Department, Karolinska Institutet, Alfred Nobels Allé 23, SE-141 83 Huddinge, Sweden.
E-mail: per.wandell@ki.se

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heart failure (12) and chronic kidney disease (4) are also more common in patients with gout. Besides, overconsumption of alcohol is a strong risk factor for developing gout (18).

The aim was to study the association between different anthropometric measures and the development of incident gout in men and women, and to find out whether any measure is superior in predicting incident gout.

Method

All women born between 1923 and 1950, and men born between 1923 and 1945, living in Malmö city, Sweden, were invited to the Malmö Diet and Cancer study (MDC study) during the period March 1991 to September 1996 (19, 20). After exclusion of subjects with prevalent gout at baseline (n = 17), 28 081 subjects remained (11 049 men and 17 032 women). Written informed consent was obtained from all participants, and the study was approved by the Ethical Committee at Lund University, Lund, Sweden (LU 51/90), and the Regional Ethical Board in Stockholm (2017/1374-31).

Outcome was assessed by a clinical diagnosis of gout (M10) in the Swedish National Patient Register during follow-up.

The examinations were carried out by trained nurses at the screening centre. Standing height was measured with a fixed stadiometer calibrated in centimetres. Weight was measured to the nearest 0.1 kg using a balance beam scale with subjects wearing light clothing and no shoes. BMI was calculated as weight (kg) divided by the square of the height (m^2). Waist circumference (WC) was measured as the circumference (cm) between the lowest rib margin and iliac crest, and hip circumference (cm) as the largest circumference between the waist and thighs. Waist-to-hip ratio (WHR) was defined as the ratio of circumference of waist to hip. Waist-to-height ratio (WHTR) was defined as the ratio of waist to height. Waist-to-hip-to-height ratio (WHHR) was defined as the ratio of WHR to height. A Body Shape Index (ABSI) was calculated according a special formula (21). Bioelectrical impedance analysers (BIAs) were used for estimating body composition, and body fat percentage and the lean-to-fat ratio were calculated using an algorithm, according to procedures provided by the manufacturer (BIA-103 single-frequency analyser; RJL Systems, Detroit, MI, USA). We used the inverted lean-to-fat ratio as a fat-to-lean ratio to express the excess risk. Basal metabolism (in kcal/day) was estimated from dietary habits.

Socio-demographic factors registered were age, immigrant status (foreign-born or Swedish-born), marital status (married, single, divorced, or widowed), highest educational level (basic up to 8 years, middle level 9–10 years, high-school level more than 10 years but not university level, and university level).

Lifestyle factors were registered, i.e. smoking habits (never smoked, former smokers, and current smokers), leisure-time physical activity score (assessed as number of minutes of 18 different activities multiplied by an activity coefficient (22), and used as a continuous variable), and alcohol intake (estimated total amount of pure alcohol, in g/day; and total amount of each of beer, wine, and spirits, in g/day).

Regarding prevalent comorbidities, these were registered as diabetes and cardiovascular diseases (i.e. coronary heart events, including therapeutic procedures, stroke, heart failure, and atrial fibrillation). Antihypertensive drugs at baseline were categorized into four groups, i.e. diuretic drugs, beta-blocking agents, calcium-receptor blocking agents, and renin–angiotensin II–aldosterone system (RAAS) blockers.

Statistical analysis

Each anthropometric measure was standardized with its standard deviation (sd) in men and in women, separately. Cox regression models were used to calculate risk [hazard ratios (HRs) with 95% confidence intervals (CIs)] of incident gout in men and women in three models, using baseline data for exposition and adjustment factors and standardized by sd units. Model 1 with age-adjusted HRs; Model 2 with additional adjustment for socio-economic data (marital status, immigrant status, and educational level) and lifestyle data (physical activity level, smoking habits, and alcohol intake); and Model 3, as Model 2 but with adjustment also for medical data (diabetes, cardiovascular diseases, and antihypertensive treatment). Harrell's C was calculated for the full models for each measure to assess prediction (23). In addition, we estimated p-values for the differences in likelihood ratio between the full models with and without each anthropometric measurement. We also tested the p-values for the five anthropometric measures with the highest C-statistic values against each other to test whether any of them was significantly better than the others. Stata version 14.2 (StataCorp, College Station, TX, USA) software was used for analyses.

We also assessed HRs by Cox regression for categories of BMI (< 25, 25–29.9, and ≥ 30 kg/m^2) and WC (< 94, 94–102, and > 102 cm for men, and < 80, 80–88, and > 88 cm for women) using the three above-mentioned models.

Results

Baseline data for men and women are shown in Tables 1 and 2, with socio-demographic and lifestyle data in Table 1, and medical data, including different anthropometric measurements, in Table 2.

The results for the 10 anthropometric measures and their association with incident gout are shown in Table 3. Total patient-years of follow-up were 553 882, comprising

Table 1. Baseline characteristics of men and women in the Malmö Diet and Cancer study: socio-demographic and lifestyle data.

	Men	Women
	(n = 11 049)	(n = 17 032)
Age (years)	59.2 ± 7.1	57.4 ± 7.9
Marital status		
Married	8 005 (72.5)	10 300 (60.5)
Single	1 117 (10.1)	1 556 (9.1)
Divorced	1 564 (14.2)	3 452 (20.3)
Widowed	357 (3.2)	1 712 (10.1)
Immigrant status	1 335 (12.1)	2 019 (11.9)
Education		
Basic (≤ 8 years)	5 068 (46.0)	6 699 (39.4)
Middle (9–10 years)	2 169 (19.7)	5 160 (30.4)
High (≥ 11 years)	2 323 (21.1)	2 608 (15.4)
University	1 461 (13.3)	2 522 (14.8)
Smoking		
Never	3 112 (28.2)	7 526 (44.2)
Former	4 775 (43.2)	4 725 (27.8)
Current	3 156 (28.6)	4 775 (28.1)
Total physical activity	8 363 ± 7 084	7 926 ± 6 334
Alcohol (g/day)*		
Total amount of pure alcohol	15.5 ± 16.0	7.7 ± 8.7
Beer	227.5 ± 255.6	83.9 ± 115.5
Wine	49.1 ± 78.4	45.0 ± 62.1
Spirits	12.9 ± 23.7	3.2 ± 8.6

Data are shown as mean ± sd or n (%).
*Intake of alcohol is an estimated value for pure alcohol; the value for specific alcoholic beverages is the total amount of each beverage (not estimated as pure alcohol intake).

Table 2. Baseline characteristics of men and women in the Malmö Diet and Cancer study: medical data.

	Men	Women
	(n = 11 049)	(n = 17 032)
Weight (kg)	81.7 ± 12.1	68.0 ± 11.7
BMI (kg/m^2)	26.2 ± 3.5	25.4 ± 4.2
WC (cm)	93.7 ± 10.1	77.9 ± 10.6
Hip circumference (cm)	99.3 ± 7.1	97.9 ± 9.6
WHR	0.94 ± 0.06	0.79 ± 0.05
WHTR	0.53 ± 0.06	0.47 ± 0.07
WHHR	0.54 ± 0.04	0.49 ± 0.04
ABSI	0.080 ± 0.004	0.071 ± 0.007
Body fat (%)	20.7 ± 5.0	30.8 ± 5.0
Fat-to-lean ratio	0.33 ± 0.14	0.65 ± 0.26
Basal metabolism (kcal/day)	1 874 ± 207	1 457 ± 125
Diseases		
Diabetes	646 (5.9)	578 (3.4)
Cardiovascular diseases	872 (7.9)	365 (2.1)
Antihypertensive drugs		
Diuretics	563 (5.1)	1 228 (7.2)
Beta-blockers	1 318 (11.9)	1 555 (9.1)
Calcium blockers	767 (6.9)	641 (3.8)
RAAS blockers	522 (4.7)	322 (1.9)

Data are shown as mean ± sd.
BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHTR, waist-to-height ratio; WHHR, waist-to-hip-to-height ratio; ABSI, A Body Shape Index; RAAS, renin–angiotensin II–aldosterone system.

207 613 for men and 346 269 for women. In total, there were 633 incident gout diagnoses (2.3%), 393 in men (3.6%) and 240 in women (1.4%). The C-statistic without any anthropometric measure for the full model was 0.7013 in men and 0.7719 in women. For both men and women, the five anthropometric measurements with highest C-statistics were weight, BMI, WC, hip circumference, and WHTR (0.7317–0.7361 in men, 0.8036–0.8085 in women), resulting in an incremental difference in C-statistic around 0.035. BMI had the highest C-statistic in men (0.7361) and WC had the highest C-statistic in women (0.8085). We also estimated the p-value for difference in likelihood ratio for the full models with and without respective anthropometric measurements, finding statistical significance for all measurements except for ABSI in women (p = 0.092). When testing the models with the different anthropometrics against each other, for men weight against WC had a p-value of 0.052, with otherwise significant differences between the five anthropometric measurements with the highest C-statistics. For women, no statistically significantly differences in likelihood ratio for the full models were found between the five anthropometric measurements with the highest C-statistics, while differences were found for the five most important anthropometric measurements against the rest of the measurements.

We also tested the HRs per unit; for men, BMI had a fully adjusted HR of 1.13 (95% CI 1.10–1.16) and WC 1.04 (95% CI 1.03–1.05), and for women, BMI had a fully adjusted HR of 1.12 (95% CI 1.09–1.15) and WC 1.05 (95% CI 1.04–1.06).

We tested to exclude alcohol and exposure to diuretics for men and for women in the models with the highest C-statistics. For men, excluding alcohol gave a C-statistic of 0.7272, i.e. a decrease in C-statistic of 0.0089, and excluding use of diuretics a C-statistic of 0.7360, i.e. a decrease in C-statistic of 0.0001. For women, excluding alcohol gave a value of 0.8058, i.e. a decrease in C-statistic of 0.0027, and excluding diuretics a value of 0.8037, i.e. a decrease in C-statistic of 0.0048.

The HRs for categories of BMI and WC for men and for women are shown in Table 4. The percentages of gout cases in the three BMI categories (< 25, 25–29.9, and ≥ 30 kg/m^2) were, respectively, 2.0%, 4.1%, and 6.2% for men, and 0.8%, 1.5%, and 3.6% for women. The percentages of gout cases in the three WC categories (men < 94, 94–102, and > 102 cm, and women < 80, 80–88, and > 88 cm) were, respectively, 2.5%, 3.8%, and 6.3% for men, and 0.8%, 1.6%, and 3.9% for women.

Discussion

The main finding of this study was that anthropometric measures that included weight (weight, BMI) or circumference of the waist (WC, WHTR) or hip had the highest C-statistics, although the highest values differed between men and women. The increased predictability for incident gout was more than 3%, which is regarded as a high value.

Table 3. Cox regression models for incident gout in men and women in the Malmö Diet and Cancer study, per standard deviation increment for each anthropometric variable.

	Model 1	Model 2	Model 3		p†
	HR (95% CI)	HR (95% CI)	HR (95% CI)	C-statistic*	Likelihood ratio test
Men					
Weight	1.58 (1.44–1.73)	1.56 (1.42–1.71)	1.49 (1.36–1.64)	0.7341	< 0.001
BMI	**1.60 (1.47–1.75)**	**1.60 (1.46–1.75)**	**1.52 (1.39–1.68)**	**0.7361**	< 0.001
WC	1.62 (1.48–1.77)	1.59 (1.45–1.75)	1.52 (1.38–1.67)	0.7352	< 0.001
Hip circumference	1.49 (1.38–1.61)	1.49 (1.38–1.619)	1.44 (1.33–1.57)	0.7317	< 0.001
WHR	1.38 (1.26–1.51)	1.33 (1.21–1.47)	1.26 (1.14–1.39)	0.7153	< 0.001
WHTR	1.58 (1.45–1.73)	1.58 (1.44–1.73)	1.49 (1.35–1.64)	0.7328	< 0.001
WHHR	1.27 (1.15–1.40)	1.25 (1.12–1.38)	1.18 (1.06–1.30)	0.7088	0.002
ABSI	1.19 (1.08–1.31)	1.15 (1.05–1.28)	1.12 (1.02–1.23)	0.7050	0.027
Body fat (%)	1.33 (1.21–1.46)	1.29 (1.17–1.42)	1.24 (1.12–1.37)	0.7111	< 0.001
Fat-to-lean ratio	1.26 (1.16–1.37)	1.23 (1.12–1.34)	1.19 (1.08–1.30)	0.7092	< 0.001
Basal metabolism	1.47 (1.34–1.62)	1.47 (1.33–1.62)	1.41 (1.28–1.56)	0.7268	< 0.001
Women					
Weight	1.75 (1.58–1.93)	1.75 (1.59–1.94)	1.58 (1.43–1.76)	0.8036	< 0.001
BMI	1.72 (1.57–1.90)	1.73 (1.56–1.91)	1.55 (1.39–1.73)	0.8042	< 0.001
WC	**1.82 (1.65–2.01)**	**1.82 (1.64–2.01)**	**1.62 (1.46–1.81)**	**0.8085**	< 0.001
Hip circumference	1.68 (1.54–1.83)	1.68 (1.53–1.84)	1.55 (1.40–1.71)	0.8045	< 0.001
WHR	1.42 (1.31–1.54)	1.41 (1.29–1.53)	1.29 (1.17–1.44)	0.7842	< 0.001
WHTR	1.80 (1.63–1.99)	1.79 (1.61–1.98)	1.59 (1.42–1.78)	0.8067	< 0.001
WHHR	1.40 (1.27–1.55)	1.37 (1.23–1.52)	1.23 (1.09–1.38)	0.7802	0.001
ABSI	1.05 (1.02–1.08)	1.05 (1.02–1.08)	1.05 (1.01–1.08)	0.7729	0.092
Body fat (%)	1.90 (1.65–2.19)	1.89 (1.64–2.19)	1.63 (1.40–1.89)	0.7803	< 0.001
Fat-to-lean ratio	1.61 (1.42–1.84)	1.60 (1.40–1.83)	1.43 (1.25–1.64)	0.7846	< 0.001
Basal metabolism	1.68 (1.51–1.88)	1.70 (1.52–1.90)	1.56 (1.39–1.74)	0.7978	< 0.001

Model 1 with age-adjusted hazard ratios (HRs) and 95% confidence intervals (CIs); Model 2 with adjustment for socio-demographic data (age, marital status, immigrant status, and educational level) and lifestyle data (physical activity level, smoking habits, and alcohol intake); and Model 3 as Model 2 but with adjustment also for medical data (diabetes, cardiovascular diseases, and antihypertensive treatment).
*C-statistics were assessed for the full model. The highest C values (in bold) were found for BMI in men and WC in women.
†p-Value for likelihood ratio test comparing the full models with and without the respective anthropometric measure.
BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHTR, waist-to-height ratio; WHHR, waist-to-hip-to-height ratio; ABSI, A Body Shape Index; RAAS, renin–angiotensin II–aldosterone system.

Compared with other studies, BMI had a high predictive value (8). However, what was unexpected was that WHR, WHHR, and especially ABSI showed lower C-statistics. We also tested basal metabolism to see whether this factor showed higher C-statistics than BMI and WC, which it did not. In general, the C-statistics showed higher values for women than for men, which is expected as being a man is a risk factor in itself for gout (4). The rate of gout in the highest versus the lowest BMI category was three times higher in men and 4.5 times higher in women, and in the highest versus the lowest WC category 2.5 higher times in men and 4.9 higher times in women, thus showing a higher relative risk with increasing anthropometric measurements in women.

Gout is an inflammatory disorder, where a higher BMI and higher proportion of fatty tissue, with its pro-inflammatory effect, further increase the inflammatory burden in the body, and therefore also the risk of cardiovascular and other metabolic diseases. Thus, our findings could be expected, but it is of importance to show that other anthropometric measures do not have superior predictive values to BMI, WC, or weight. ABSI is shown to be more strongly associated with cardiovascular mortality (24), but showed a lower predictive value for gout in the present study. However, ABSI was created to predict mortality, and the predictive values for different outcomes do differ among different anthropometric measurements.

We performed adjustment for different confounders. Alcohol-induced hyperuricaemia, associated with incident gout, could be due to lower excretion and increased production of urate (25), which is why it is important to adjust for alcohol intake. There are also differences between different alcoholic beverages and the gout risk, with beer in particular, and also spirits, increasing the risk, whereas wine does not seem to have this effect (26). However, adjusting for socio-demographic and lifestyle factors of interest (physical activity, smoking, and alcohol intake) did not affect HRs other than marginally, while adjusting for medical factors, such as comorbidity (diabetes and cardiovascular diseases) (1) and cardiovascular medications did (12, 15–17), especially for women. When excluding alcohol in the full models, the C-statistics showed a higher decrease for

Table 4. Cox regression models for incident gout in men and women in the Malmö Diet and Cancer study, by categories of body mass index (BMI) and waist circumference (WC).

	All	Gout cases	Model 1	Model 2	Model 3
	n	n (%)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Men					
BMI (kg/m^2)					
< 25	4 145	83 (2.0)	1 (ref)	1 (ref)	1 (ref)
25–29.9	5 462	221 (4.1)	1.97 (1.53–2.53)	1.94 (1.50–2.50)	1.83 (1.42–2.37)
≥ 30	1 426	88 (6.2)	3.46 (2.56–4.67)	3.31 (2.43–4.50)	2.84 (2.07–3.88)
WC (cm)					
< 94	5 776	144 (2.5)	1 (ref)	1 (ref)	1 (ref)
94–102	3 324	127 (3.8)	1.56 (1.23–1.98)	1.50 (1.18–1.90)	1.43 (1.12–1.82)
> 102	1 926	121 (6.3)	2.95 (2.31–3.75)	2.79 (2.18–3.58)	2.46 (1.91–3.16)
Women					
BMI (kg/m^2)					
< 25	9 018	69 (0.8)	1 (ref)	1 (ref)	1 (ref)
25–29.9	5 674	87 (1.5)	1.79 (1.30–2.46)	1.78 (1.29–2.45)	1.58 (1.14–2.18)
≥ 30	2 314	83 (3.6)	4.26 (3.09–5.87)	4.21 (3.03–5.86)	3.09 (2.20–4.36)
WC (cm)					
< 80	10 728	83 (0.8)	1 (ref)	1 (ref)	1 (ref)
80–88	3 759	59 (1.6)	1.84 (1.32–2.58)	1.79 (1.27–2.50)	1.58 (1.12–2.22)
> 88	2 511	97 (3.9)	4.79 (3.56–6.44)	4.61 (3.41–6.23)	3.45 (2.52–4.73)

Missing data: BMI for men n = 16 and women n = 26; WC for men n = 23 and women n = 34.
Model 1 with age-adjusted hazard ratios (HRs) and 95% confidence intervals (CIs); Model 2 with adjustment for socio-demographic data (age, marital status, immigrant status, and educational level) and lifestyle data (physical activity level, smoking habits, and alcohol intake); and Model 3 as Model 2 but with adjustment also for medical data (diabetes, cardiovascular diseases, and antihypertensive treatment).

men than for women, while the opposite was the case when excluding diuretics. This is not surprising, as men, on average, have a higher alcohol consumption than women, and women have a higher average rate of diuretic treatment than men. Still, the increment in C-statistics using anthropometric values compared to excluding them had a higher value, around 0.035, compared to the increment by alcohol intake (0.0089 for men) or diuretic use for women (0.0048).

A clear dose response was noted when the association between BMI and WC categories was studied, indicating that these cut-offs are clinically relevant as cut-offs to assess the relative risk of gout. In men, BMI was superior in predicting gout compared to all other anthropometric measurements, and in women weight, BMI, WC, hip circumference, and WHTR were superior to the rest of the tested anthropometric measurements. Thus, there seem to be differences between different anthropometric factors in predictive value for different disorders, with the waist-to-stature ratio or WHTR showing higher predictive value in predicting disorders such as cardiovascular diseases and diabetes (27, 28).

There are some limitations to this study. The diagnoses were clinically based, with the risk of both underestimation and overestimation. Diagnoses were taken from the Swedish National Patient Register, where diagnoses from primary care are not included. According to another Swedish study, two-thirds of patients were cared for in primary care, but there seemed to be no large differences according to symptom pattern (29). However, in that study, the positive predictive values (PPVs) in relation to different definitions of gout were estimated for patients from primary care and rheumatology care. In relation to Rome, New York, and American Rheumatism Association (ARA) definitions for at least one gout diagnosis, for primary care PPVs were 7%, 3%, and 21%, respectively, while for rheumatology care PPVs were 64%, 61%, and 68%, respectively (29). Thus, using only hospital discharge diagnoses could be justified. Any risk of underestimation and overestimation of gout should affect men and women similarly. Thus, the diagnosis of gout was a clinical one, as was the diagnosis of chronic kidney disease, with no possibility of checking these diagnoses according to diagnostic criteria. However, the quality of Swedish registers is found to be high (30), including that of the National Patient Register (31). Data on lifestyle habits were self-reported. We have used only baseline data, and the situation might have changed during follow-up. Urate levels were not available in the present study, but if they had been available we would have included them in the analyses.

The strengths of the study are the large, well-characterized, population-based cohort (19, 20), with low loss to follow-up, good registration of outcomes, and inclusion of many different confounders. Measurements were not self-reported, but were performed by an experienced research nurse.

Conclusion

We found that for men BMI, and for women WC, showed the highest predictive ability for gout, with increased predictability of more than 3% compared to using other available risk factors only. The clinically used cut-offs for BMI and WC appeared to be relevant in the assessment of increased risk of gout.

Disclosure statement

The authors have no conflicts of interest to declare.

References

1. Wandell P, Carlsson AC, Ljunggren G. Gout and its comorbidities in the total population of Stockholm. Prev Med 2015;81:387–91.
2. Dehlin M, Drivelegka P, Sigurdardottir V, Svard A, Jacobsson LT. Incidence and prevalence of gout in Western Sweden. Arthritis Res Ther 2016;18:164.
3. Kapetanovic MC, Hameed M, Turkiewicz A, Neogi T, Saxne T, Jacobsson L, et al. Prevalence and incidence of gout in southern Sweden from the socioeconomic perspective. RMD Open 2016;2:e000326.
4. Wijnands JM, Viechtbauer W, Thevissen K, Arts IC, Dagnelie PC, Stehouwer CD, et al. Determinants of the prevalence of gout in the general population: a systematic review and meta-regression. Eur J Epidemiol 2015;30:19–33.
5. Robinson PC, Taylor WJ, Merriman TR. Systematic review of the prevalence of gout and hyperuricaemia in Australia. Intern Med J 2012;42:997–1007.
6. Haslam DW, James WP. Obesity. Lancet 2005;366:1197–209.
7. Narayan KM, Ali MK, Koplan JP. Global noncommunicable diseases–where worlds meet. N Engl J Med 2010;363:1196–8.
8. Aune D, Norat T, Vatten LJ. Body mass index and the risk of gout: a systematic review and dose-response meta-analysis of prospective studies. Eur J Nutr 2014;53:1591–601.
9. Maynard JW, McAdams DeMarco MA, Baer AN, Kottgen A, Folsom AR, Coresh J, et al. Incident gout in women and association with obesity in the atherosclerosis risk in communities (ARIC) study. Am J Med 2012;125:717 e9–e17.
10. Juraschek SP, Miller ER 3rd, Gelber AC. Body mass index, obesity, and prevalent gout in the United States in 1988-1994 and 2007-2010. Arthritis Care Res (Hoboken) 2013;65:127–32.
11. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond) 2008;32(Suppl 3):S56–9.
12. Zhu Y, Pandya BJ, Choi HK. Comorbidities of gout and hyperuricemia in the US general population: NHANES 2007-2008. Am J Med 2012;125:679–87 e1.
13. Nakamura K, Sakurai M, Miura K, Morikawa Y, Nagasawa SY, Ishizaki M, et al. HOMA-IR and the risk of hyperuricemia: a prospective study in non-diabetic Japanese men. Diabetes Res Clin Pract 2014;106:154–60.
14. Li C, Hsieh MC, Chang SJ. Metabolic syndrome, diabetes, and hyperuricemia. Curr Opin Rheumatol 2013;25:210–16.
15. Gibson TJ. Hypertension, its treatment, hyperuricaemia and gout. Curr Opin Rheumatol 2013;25:217–22.
16. Reungjui S, Pratipanawatr T, Johnson RJ, Nakagawa T. Do thiazides worsen metabolic syndrome and renal disease? The pivotal roles for hyperuricemia and hypokalemia. Curr Opin Nephrol Hypertens 2008;17:470–6.
17. Sica DA, Carter B, Cushman W, Hamm L. Thiazide and loop diuretics. J Clin Hypertens (Greenwich) 2011;13:639–43.
18. Vamvakas S, Teschner M, Bahner U, Heidland A. Alcohol abuse: potential role in electrolyte disturbances and kidney diseases. Clin Nephrol 1998;49:205–13.
19. Berglund G, Elmstahl S, Janzon L, Larsson SA. The Malmo Diet and Cancer study. Design and feasibility. J Intern Med 1993;233:45–51.
20. Manjer J, Carlsson S, Elmstahl S, Gullberg B, Janzon L, Lindstrom M, et al. The Malmo Diet and Cancer study: representativity, cancer incidence and mortality in participants and non-participants. Eur J Cancer Prev 2001;10:489–99.
21. Krakauer NY, Krakauer JC. A new body shape index predicts mortality hazard independently of body mass index. PLoS One 2012;7:e39504.
22. Andreasson A, Carlsson AC, Onnerhag K, Hagstrom H. Waist/hip ratio better predicts development of severe liver disease within 20 years than body mass index: a population-based cohort study. Clin Gastroenterol Hepatol 2017;15:1294–301 e2.
23. Harrell FE Jr., Califf RM, Pryor DB, Lee KL, Rosati RA. Evaluating the yield of medical tests. JAMA 1982;247:2543–6.
24. Dhana K, Kavousi M, Ikram MA, Tiemeier HW, Hofman A, Franco OH. Body shape index in comparison with other anthropometric measures in prediction of total and cause-specific mortality. J Epidemiol Community Health 2016;70:90–6.
25. Choi HK, Atkinson K, Karlson EW, Willett W, Curhan G. Alcohol intake and risk of incident gout in men: a prospective study. Lancet 2004;363:1277–81.
26. 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.
27. Ho SY, Lam TH, Janus ED. Waist to stature ratio is more strongly associated with cardiovascular risk factors than other simple anthropometric indices. Ann Epidemiol 2003;13:683–91.
28. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global boundary value. Nutr Res Rev 2010;23:247–69.
29. Dehlin M, Stasinopoulou K, Jacobsson L. Validity of gout diagnosis in Swedish primary and secondary care - a validation study. BMC Musculoskelet Disord 2015;16:149.
30. Ludvigsson JF, Almqvist C, Bonamy AK, Ljung R, Michaelsson K, Neovius M, et al. Registers of the Swedish total population and their use in medical research. Eur J Epidemiol 2016;31:125–36.
31. Ludvigsson JF, Andersson E, Ekbom A, Feychting M, Kim JL, Reuterwall C, et al. External review and validation of the Swedish national inpatient register. BMC Public Health 2011;11:450.