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Original article

National prevalence of gout derived from administrative health data in Aotearoa New Zealand

Doone Winnard[1], Craig Wright[2], William J. Taylor[3], Gary Jackson[4], Leanne Te Karu[5], Peter J. Gow[6], Bruce Arroll[7], Simon Thornley[8], Barry Gribben[9] and Nicola Dalbeth[6,10]

Abstract

Objective. Previous small studies in Aotearoa New Zealand have indicated a high prevalence of gout. This study sought to determine the prevalence of gout in the entire Aotearoa New Zealand population using national-level health data sets.

Methods. We used hospitalization and drug dispensing claims for allopurinol and colchicine for the entire Aotearoa New Zealand population from the Aotearoa New Zealand Health Tracker (ANZHT) to estimate the prevalence of gout in 2009, stratified by age, gender, ethnicity and socio-economic status ($n = 4\,295\,296$). Results were compared with those obtained from an independent large primary care data set (HealthStat, $n = 555\,313$).

Results. The all-ages crude prevalence of diagnosed gout in the ANZHT population was 2.69%. A similar prevalence of 2.89% was observed in the HealthStat population standardized to the ANZHT population for age, gender, ethnicity and deprivation. Analysis of the ANZHT population showed that gout was more common in Māori and Pacific people [relative risk (RR) 3.11 and 3.59, respectively], in males (RR 3.58), in those living in the most socio-economically deprived areas (RR 1.41) and in those aged >65 years (RR >40) (*P*-value for all <0.0001). The prevalence of gout in elderly Māori and Pacific men was particularly high at >25%.

Conclusion. Applying algorithms to national administrative data sets provides a readily available method for estimating the prevalence of a chronic condition such as gout, where diagnosis and drug treatment are relatively specific for this disease. We have demonstrated high gout prevalence in the entire Aotearoa New Zealand population, particularly among Māori and Pacific people.

Key words: gout, epidemiology, database, public health.

Introduction

Gout is an inflammatory arthritis that occurs due to monosodium urate crystal deposition in synovial joints. This disease can cause severe joint pain and musculoskeletal disability [1, 2], and is associated with metabolic syndrome, hypertension, diabetes and cardiovascular disease [3–8]. In recent decades the incidence and prevalence of gouty arthritis has increased worldwide [9–11]. The increasing rates of gout are likely to be due to increasing longevity, modification of dietary intake and the obesity epidemic [12]. Studies reporting gout epidemiology have used a variety of case definitions and data collection methods, including self-reported physician-diagnosed gout in population surveys and analysis of health-care

[1]Planning and Funding, Counties Manukau District Health Board, Auckland, [2]Health and Disability Intelligence Unit, Ministry of Health, [3]Rehabilitation Teaching & Research Unit, Department of Medicine, University of Otago, Wellington, [4]Health Partners Consulting Group, Auckland, [5]Māori Pharmacists Association, Taupo, [6]Department of Rheumatology, Counties Manukau District Health Board, Auckland, [7]Department of General Practice & Primary Health Care, University of Auckland, [8]Epidemiology and Biostatistics, School of Population Health, University of Auckland, [9]CBG Health Research Limited and [10]Department of Medicine, University of Auckland, Auckland, New Zealand.

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Correspondence to: Doone Winnard, Counties Manukau District Health Board, 19 Lambie Drive, Private Bag 94052, South Auckland Mail Centre, Manukau City, Auckland 2241, New Zealand.
E-mail: doone.winnard@middlemore.co.nz

CLINICAL SCIENCE

data sets [8–11, 13–23]. Previous sub-national studies in Aotearoa New Zealand have suggested a high prevalence of gout, particularly in Māori people [15–23]. However, the prevalence of gout using entire-nation databases has not been reported. The aim of this study was to determine the prevalence of gout in the entire New Zealand population using nationwide health data sets.

Methods

Gout prevalence was estimated using two independent data sources. The Aotearoa New Zealand Health Tracker (ANZHT) population was used to determine the overall prevalence of gout and also stratified by age, gender, ethnicity and socio-economic status. (Other papers presenting data from this population may refer to the denominator population simply as the NZHT population. Aotearoa is the name given to New Zealand by the indigenous Māori population.) To assess the accuracy of the prevalence of gout derived from this source, prevalence was also calculated using HealthStat, a separate primary care database that collects information in a different way from the ANZHT data and cannot be linked to the ANZHT data set.

ANZHT population

For the main prevalence study, we defined the denominator population based on Aotearoa New Zealand health service contact and attempted to align with Statistics New Zealand's census-derived usually resident definition (resident for >3 months). Health services contact includes:

(i) primary care consultation or current enrolment;
(ii) public hospital event (admission or discharge);
(iii) laboratory testing claims;
(iv) community pharmaceutical dispensing; and
(v) community aged and disability support events.

All New Zealand residents are assigned a unique alphanumeric code at the time of their first contact with the health-care system, the National Health Index (NHI), which is linked to most routinely collected national health databases. The NHI can be encrypted and linked anonymously to various databases. The denominator population for this study refers to people who were registered with a NHI, were alive on 30 June 2009 and had any form of health services contact in New Zealand from 1 July 2008 to 30 June 2009. Persons without resident status were excluded if they did not receive services for a period >3 months—to align with Statistics New Zealand's definition of usually resident. We identified a denominator health services contact population of 4 295 296 people as on 30 June 2009. This was 99.5% of the Statistics New Zealand estimated usually resident population ($n = 4\,315\,800$) for the same period.

Ethnicity data for this population were taken from the 2009 second quarter primary care enrolment database and the NHI extract for the 2009 second quarter. In keeping with other ethnicity reporting in Aotearoa New Zealand health data, ethnicity was prioritized from multiple ethnic codes in the following order: Māori, Pacific peoples, Asian, European and Other New Zealanders.

Socio-economic status was measured using the NZDep2006 Index of Deprivation. NZDep2006 is a small area-based ordinal scale of socio-economic deprivation based on nine variables from the New Zealand Census—income, employment, receiving a means-tested benefit, home ownership, access to car or telephone, household living space, educational qualifications and social support. The NZDep2006 quintiles range from 1 (least deprived) to 5 (most deprived) and are assigned to people living in a defined geographical area (meshblock) rather than to individuals. For this study, the NZDep2006 Index was taken either from the primary care enrolment register for 2009 second quarter or, in the absence of a meshblock code for the domicile on the primary care database, from the most recent health domicile code on the NHI identifier.

People were identified as having gout in the ANZHT population if they had either received a discharge diagnosis of gout (ICD-9 274, ICD-10 M10) from a public hospital admission from 1988 to 2009 or been dispensed allopurinol or colchicine from a community pharmacy between 2001 and 2009. Such individuals also had to be still alive and living in Aotearoa New Zealand, evidenced by some form of recorded health contact during July 2008–June 2009. For individuals who had been diagnosed with leukaemia or lymphoma (ICD-10-AM C80-C96) in the previous 24 months, dispensing of allopurinol was excluded as an indicator of gout. Allopurinol and probenecid are the only two registered urate-lowering drugs available in New Zealand. Allopurinol is not recommended for the treatment of asymptomatic hyperuricaemia in New Zealand. Probenecid was not included in the algorithm, as it is used infrequently in Aotearoa New Zealand for the treatment of gout, and is often used to increase antibiotic blood levels for the treatment of bacterial infections in primary care.

The HealthStat population

The HealthStat database contains primary care records from a sample of 103 New Zealand general practices. Practices that contribute information to this database originally represented 10% of the population in 2005, and at December 2009 contained data for 555 313 patients (12.9% of the New Zealand population). The HealthStat data we accessed covered the interval from 1 October 2005 to 31 December 2009, but only patients registered with HealthStat practices on 31 December 2009 were included, to align with the denominator used for the ANZHT cohort. HealthStat data are collected by automatic electronic upload each week and include consultation dates and types, diagnoses, prescriptions and complete registers every 3 months for defining denominators. All data are anonymized but linked by a unique practice-level identifier. This identifier is different from the NHI, so the HealthStat data cannot be directly linked to the ANZHT data. People were identified as having gout in this cohort if they had either a primary care practitioner record of a diagnosis of gout or a prescription of

allopurinol or colchicine for at least 1 month ordered by a primary care practitioner and recorded in the database during the period 1 October 2005 to 31 December 2009.

HealthStat practices are not randomly selected from primary care practices across New Zealand, and the ethnicity of their patients differs from the ANZHT population. Of the HealthStat population, 25.9% identified as Māori and 7.5% as Pacific, compared with 12.3% and 5.9% of the ANZHT population, respectively. Similar proportions were aged ⩾65 years (10.8% for HealthStat and 12.8% for the ANZHT population).

Statistical methods

To estimate the relative risks (RRs), 95% CIs and *P*-values for comparisons with each reference category within age groups, genders, ethnic groups and the New Zealand deprivation quintiles, we fitted a modified Poisson regression model with main effects [24]. The SAS 9.1 version of PROC GENMOD was used for estimation. These estimates were adjusted for first-order effects but not interactions. Prevalence estimates were also calculated and age-standardized to the WHO reference population to facilitate international comparisons.

Ethical considerations

All unit record data were non-identifiable, analysis was undertaken by the institution holding the data and no contact was made with the study population. Therefore, ethical review was not required, in accordance with New Zealand Ministry of Health Guidelines.

Results

All-ages gout prevalence in the ANZHT and the HealthStat primary care populations

The all-ages prevalence of gout in the ANZHT population was 2.69% (115 399/4 295 296) and the all-ages crude prevalence of recorded primary care diagnosed gout in the HealthStat population was 3.05% (16 956/555 313) (Table 1). A prevalence of 2.89% was observed in the HealthStat population standardized to the ANZHT population for age, gender, ethnicity and deprivation (Table 1). For the ANZHT population, gout case ascertainment was made in 3.0% using hospital admission criteria, 83.4% using medication dispensing criteria and 13.5% using both criteria. For the HealthStat population, gout case ascertainment was made in 39.3% using general practitioner gout diagnosis criteria, 19.4% using medication prescribing criteria and 41.3% using both criteria.

Gout prevalence and risk factors for gout in those aged ⩾20 years in the ANZHT population

The prevalence of gout for those ⩾20 years of age in the ANZHT population was 3.75% (114 318/3 047 172) (Table 1). In the ANZHT population, the risk of being identified with gout in Māori and Pacific people was over threefold higher than those of European ancestry (Table 2). Overall prevalence was 3.24% in European adults, 6.06% in Māori adults and 7.63% in Pacific adults (Table 2). The prevalence for men was 3.6 times that of women, and there was a steep increase in prevalence with increasing age (Table 2). The risk of being identified as having gout was 41% higher in those living in the most socio-economically deprived areas compared with people in the most advantaged areas, after controlling for age, gender and ethnicity (Table 2). The crude gout prevalence was 9.62% in adult Māori men and 12.32% in adult Pacific men (Table 3). Prevalence of gout in older Māori and Pacific men was particularly high; affecting at least one-quarter of Pacific men and one-third of Māori men ⩾65 years (Fig. 1A). Similarly, prevalence in older Māori and Pacific women was high, affecting between 12 and 25% of those ⩾65 years (Fig. 1B).

The prevalence of gout, standardized by age and gender, showed steeper socio-economic deprivation gradients, particularly for Māori and Pacific groups (Fig. 2). Analysis of age-specific gout prevalence rates by gender and the highest and lowest socio-economic deprivation quintiles showed higher prevalence at earlier ages for more deprived populations (Fig. 3).

Discussion

This study has allowed estimation of the prevalence of gout for the entire Aotearoa New Zealand population using national-level administrative databases of health care and medication use. We demonstrate a high overall national prevalence of gout of 2.69% and a prevalence of 3.75% in people aged ⩾20 years. We also confirm that

Table 1 Gout prevalence in ANZHT and HealthStat populations

Source (method)	Gout	Population	Prevalence (95% CI), %
ANZHT all ages	115 639	4 295 296	2.69 (2.67–2.71)
HealthStat all ages (crude)	16 956	555 313	3.05 (3.00–3.10)
HealthStat all ages (standardized)[a]	16 956	555 313	2.89 (2.87–2.91)
ANZHT aged ⩾20years	114 318	3 047 172	3.75 (3.73–3.77)
HealthStat aged ⩾20years (crude)	16 898	373 607	4.52 (4.45–4.59)
HealthStat aged ⩾20years (standardized)[a]	16 898	373 607	4.06 (4.00–4.12)

[a]Standardized for ANZHT age–gender–ethnicity-NZDep.

Table 2 Prevalence of gout in the ANZHT population, aged ⩾20 years

Demographic factor	Gout	Population	Prevalence, %	RR (95% CI)	*P*-value
Ethnic group					
European/other	73 272	2 258 073	3.24	1.00	
Asian	4598	234 312	1.96	0.98 (0.92–1.05)	0.5583
Māori	22 689	374 531	6.06	3.11 (2.94–3.28)	<0.0001
Pacific	13 759	180 256	7.63	3.59 (3.22–4.01)	<0.0001
NZDep quintile 2006[a]					
1	15 431	571 156	2.70	1.00	
2	16 829	561 393	3.00	1.05 (0.98–1.14)	0.1804
3	21 935	616 549	3.56	1.17 (1.09–1.26)	<0.0001
4	26 519	663 286	4.00	1.24 (1.16–1.34)	<0.0001
5	33 074	621 888	5.32	1.41 (1.31–1.52)	<0.0001
Gender					
Female	28 255	1 607 547	1.76	1.00	
Male	86 063	1 439 625	5.98	3.58 (3.34–3.85)	<0.0001
Age group, years					
20–24	734	288 775	0.25	1.00	
25–29	1425	266 296	0.54	2.23 (1.76–2.82)	<0.0001
30–34	2282	266 009	0.86	3.69 (2.87–4.73)	<0.0001
35–39	3820	310 634	1.23	5.57 (4.47–6.93)	<0.0001
40–44	5567	311 703	1.79	8.20 (6.64–10.12)	<0.0001
45–49	8012	321 040	2.50	11.85 (9.76–14.38)	<0.0001
50–54	9939	280 481	3.54	17.35 (14.48–20.77)	<0.0001
55–59	12 482	245 380	5.09	25.58 (21.50–30.44)	<0.0001
60–64	14 052	217 502	6.46	34.15 (28.68–40.67)	<0.0001
65–69	13 736	168 252	8.16	43.58 (36.52 – 52.00)	<0.0001
70–74	13 111	126 896	10.33	56.89 (47.43–68.24)	<0.0001
75–79	12 120	102 430	11.83	69.56 (57.89–83.59)	<0.0001
80–84	9378	77 131	12.16	78.36 (64.32–95.47)	<0.0001
⩾85	7660	64 643	11.85	91.07 (72.70–114.08)	<0.0001

Each factor in the table is controlled for all of the other factors. *P*-value refers to comparison with the reference population. [a]Data not available in 12 900 people, as the address data were not sufficient, or no NZDep score has been calculated for the area as the population is too small.

Table 3 Gender- and ethnic-specific prevalence of gout in the ANZHT population, aged ⩾20 years; crude and age-standardized to the WHO standard population

Ethnicity	Men (crude ANZHT), %	Men (age-standardized), %	Women (crude ANZHT), %	Women (age-standardized), %
Māori	9.62	11.73	2.99	4.00
Pacific	12.32	13.53	3.45	4.07
European	5.12	3.69	1.52	0.93
Asian	3.53	3.65	0.80	0.98
All	5.98	4.93	1.76	1.31

gout is more common among Māori and Pacific people, males, people with advancing age and people living in socio-economically deprived areas. The analysis raises a number of issues about methods of assessment of gout prevalence in large population-based studies and gout epidemiology, and has implications for clinical and public health practice.

This study used routinely collected diagnostic coding and medication data from national administrative databases to explore the prevalence of gout in the entire Aotearoa New Zealand population. Previous studies reporting gout prevalence have used a variety of case definitions and methods for collecting case data. In part, the various case definitions reflect the challenge of diagnosing gout. Although identification of monosodium urate crystals in synovial fluid is the most specific and accurate method of diagnosing gout, this method is not feasible for large population-based studies. Clinical classification criteria exist for acute gout [25]; however, these criteria require individual patient assessments, and may not be

FIG. 1 Prevalence of gout in the ANZHT population. (**A**) In men, by age and ethnicity. (**B**) In women, by age and ethnicity.

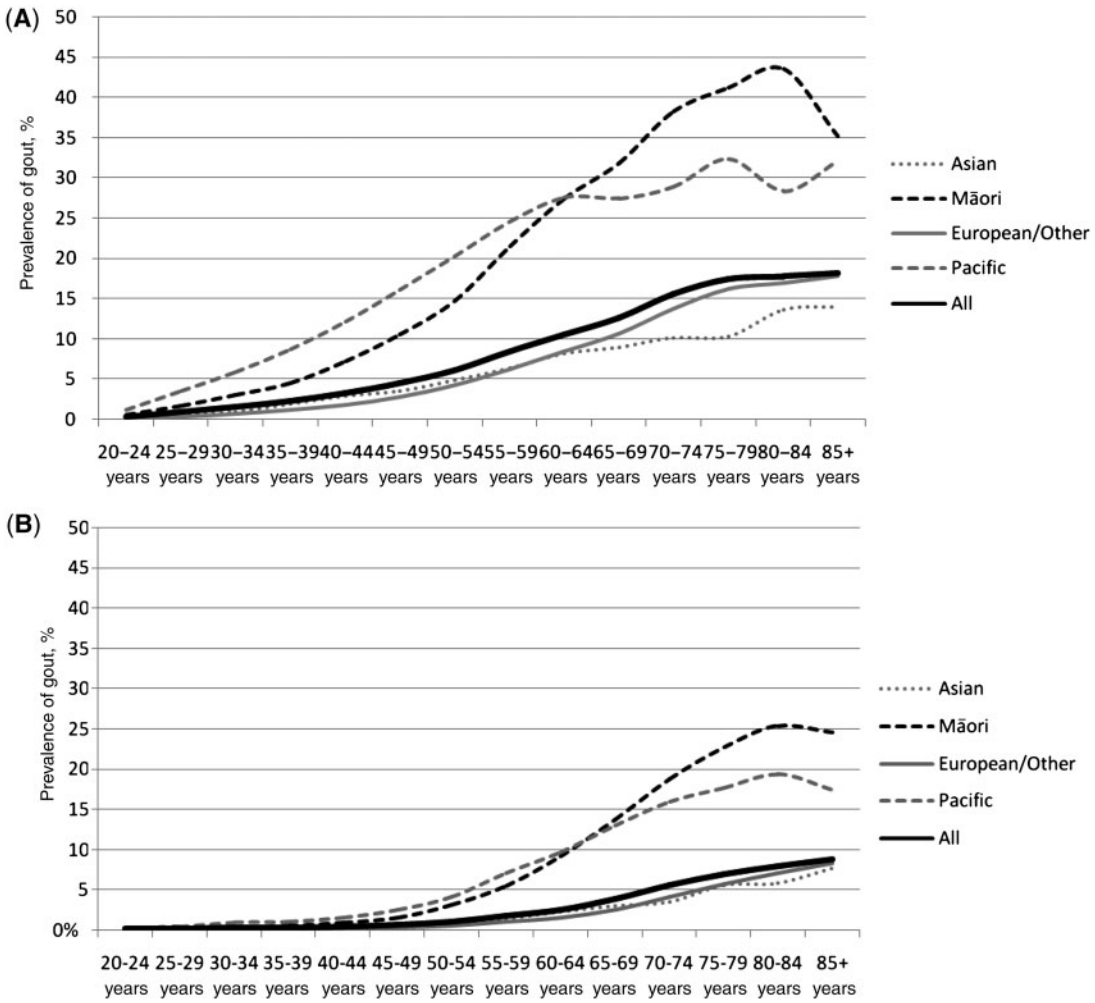


FIG. 2 Age- and gender-standardized prevalence of gout by ethnic group and NZDep2006 Index. The NZDep2006 quintiles range from 1 (least deprived) to 5 (most deprived).

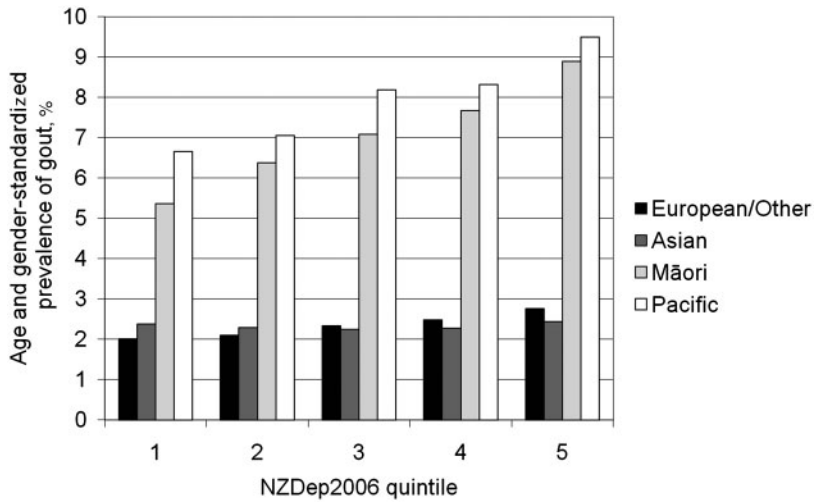
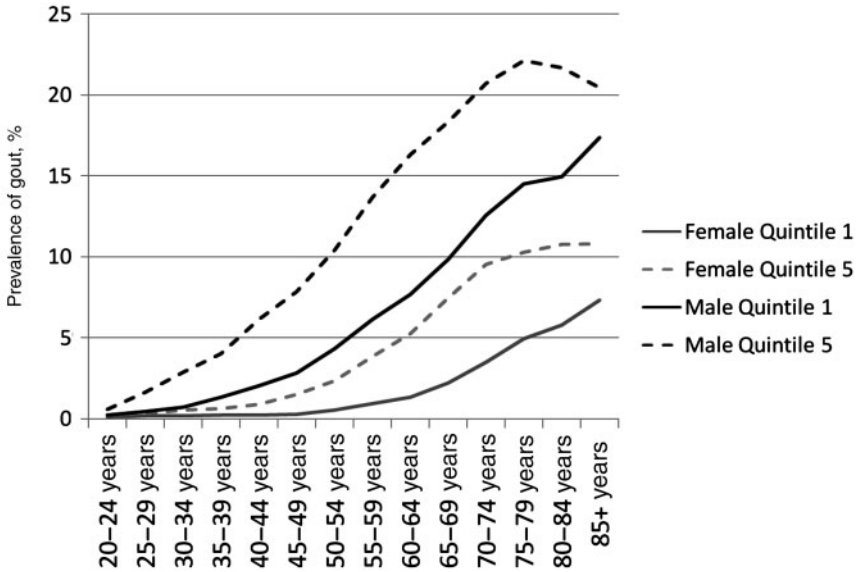


FIG. 3 Age-specific prevalence of gout by gender, and the highest and lowest socio-economic deprivation quintiles. The NZDep2006 quintiles range from 1 (least deprived) to 5 (most deprived).



suitable for diagnosis of gout between acute attacks or in large population-based studies.

In population surveys and cohort studies, self-report of physician-diagnosed gout is a common means of case-definition [10, 13, 14, 26, 27]. Data from such surveys have been derived either from general health surveys containing a question about gout diagnosis [e.g. the National Health and Nutrition Examination Survey (NHANES) in the USA][10] or surveys more specifically focused on musculoskeletal conditions (e.g. a house-to-house survey focused on symptoms of rheumatic disease in an aboriginal settlement in Australia) [14].

As in our study, algorithms based on diagnostic coding and pharmaceutical prescribing have also been used to identify cases of gout in population-based studies [8, 11]. However, prevalence studies based on health-care databases have described more limited populations than our study, which instead uses national-level databases and includes all residents living in Aotearoa New Zealand. The 1999 UK gout epidemiology study used the national General Practice Research Database; at the time of that study the general practices participating in the database provided primary health care to ~3% of the UK population. The age, gender and geography of the study population were similar to the total UK census population [8]. The UK General Practice Research Database is comparable with our HealthStat comparison study, although in the UK study, gout case definition was based only on physician coding for a diagnosis of gout only rather than also including prescribing data. In a study more comparable with our methodology, an administrative claims database for an insured managed care population in the USA was used to calculate the prevalence of gout [11] using encounter claim diagnosis or pharmacy claim for a gout-related medication (allopurinol, probenecid, colchicine or sulphinpyrazone) in the absence of a cancer requiring allopurinol.

In our study, the case definition of gout has been made by an algorithm based on national administrative data. The use of administrative data has the advantage of not requiring complex and expensive surveys and may have more predictable biases than unknown biases related to survey response rates. Several observations point to the validity of this algorithm. Similar prevalences were observed between the ANZHT and the HealthStat analyses even though the information is collected in a different manner; the ANZHT data, is based on hospital diagnosis and dispensing data, whereas HealthStat uses primary care diagnosis and prescribing data. Despite different criteria weighting using these different models, similar prevalence was obtained using the two data sets. The slightly higher prevalence in the HealthStat population may be due to the oversampling of Māori in this population. Further, when we compare our results with other studies, recognized risk factors for gout (increasing age, male sex, Māori ethnicity) were identified with a similar magnitude as previous studies (see supplementary Tables S1 and S2, available as Supplementary data at *Rheumatology* online) [8, 10, 11, 13–23]. This algorithm provides a template to allow comparison in gout prevalence between different countries and to study changes in prevalence over time.

A study such as ours requires a population who are all assigned a unique identifier; in our case, the NHI identifier. Using this identifier for enumerating both the numerator and denominator means we avoided numerator/denominator bias. This is particularly important in relation to assigned ethnicity because of historical issues related to undercounting of our indigenous Māori population [28]. A further advantage of involving the total health contact population for the country is that examining trends over

time will be less complicated by shifts in the denominator population compared with the shifts inherent in the use of managed care population data.

Our study is limited by the accuracy of our method for detecting a diagnosis of gout. Sensitivity for the tracker population is limited by the fact that only those who present to the health system and are diagnosed or fill their prescription for colchicine or allopurinol are counted. The slightly higher prevalence in the HealthStat population, which counts prescribing rather than dispensing of medication, may also indicate that the true prevalence could be higher than that reflected in the ANZHT population. Qualitative research and clinical experience indicates that people with gout may self-manage their gout flares by purchasing over-the counter NSAIDs directly from a pharmacy, borrowing medication or using alternative therapies that are not captured in health systems data [29]. In Aotearoa New Zealand, the use of traditional Māori remedies (rongoā) is important among the Māori community. Thus our data may be an underestimate of the true prevalence of gout in the community. Capture–recapture modelling is one method that may estimate the extent of this otherwise unmeasured burden of gout, and this analysis will be reported in a separate paper.

In relation to specificity, relying on pharmaceutical claims may lead to the inclusion of people who have been prescribed allopurinol for other indications. However, the exclusion of patients with haematological malignancies is expected to largely mitigate this problem. Allopurinol prescription for asymptomatic hyperuricaemia is very uncommon in Aotearoa New Zealand. Colchicine is used for other rare conditions such as auto-inflammatory diseases or auto-immune serositis, but the infrequency of these disorders suggest that false-positive cases would be few. Both data sets used in this study rely on clinical diagnosis of gout and subsequent treatment, particularly in primary health care. Diagnosis in this setting is likely to be presumptive rather than confirmed with joint aspiration, but this caveat will also hold for other epidemiological studies based on algorithms using prescribing and diagnosis data or self-report of physician diagnosis.

The prevalence of gout estimated for the Aotearoa New Zealand health services contact population, both for all ages (2.69%) and those aged $\geqslant$20 years (3.75%), is higher than previous estimates from other countries (see supplementary Table S1, available as supplementary data at *Rheumatology* Online) [8, 10, 11, 13, 14]. The reasons for these differences are not clear, but may represent differences in ethnicity in the Aotearoa New Zealand population, increased awareness of gout within primary care or different prescribing behaviours. Previous research suggests Māori and Pacific peoples do not excrete uric acid as effectively as those of European ancestry [22], and genetic studies link this under-excretion to variation in renal urate transporter genes in Polynesian peoples [30, 31]. The differences between results for ethnic groups in our study highlights the importance of disaggregating data to avoid masking important differences in the burden of disease for population subgroups. The result for Māori of 6.06% is similar to those reported in the Australian Aboriginal community study [14] and in excess of total population figures internationally (see supplementary Table S1, available as supplementary data at *Rheumatology* Online) [8, 10, 11, 13, 14]. The Māori prevalence in our study is difficult to compare with previous New Zealand studies because of the different methods used. However, the prevalences do appear to be comparable with these studies (see supplementary Table S2, available as supplementary data at *Rheumatology* Online) [15–23], and the method of this study is more nationally representative than previous studies. The high gout prevalence for Pacific peoples living in Aotearoa New Zealand of 7.63% is consistent with a recent estimate using a primary care database [19]. Our analysis shows that gout is more common and has earlier onset in those living in more socio-economically deprived areas, with this finding remaining after adjustment for ethnicity. These ethnicity and socio-economic results highlight gout as an important burden of disease for populations who already have significant health disparities. Further research to determine the factors contributing to higher prevalence and earlier onset of gout in areas of greater socio-economic deprivation will be important. Furthermore, the increase in the prevalence of gout with age, at levels much higher than previously reported, has implications for clinical service planning for both primary health care and specialist services, given the demographic trends of increasing life expectancy and ageing [32]. While the exact nature of the role of serum urate in diabetes and cardiovascular disease remains contested [33], the onset of gouty arthritis can also identify a population likely to have coincident metabolic risk [34, 35], and therefore represents an important opportunity for intervention to modify other disease trajectories [36].

In summary, by applying algorithms based on diagnostic coding and drug dispensing claims to nationwide health data sets we have demonstrated a high overall gout prevalence of 2.69% for the entire population of Aotearoa New Zealand and a prevalence of 3.75% in those aged $\geqslant$20 years. We have confirmed higher risk in Māori and Pacific people, males, in people living in more socio-economically deprived areas and much greater risk with advancing age. This study provides further support for public health and primary care measures that address risk factors and improve management of gout.

Rheumatology key messages

- Algorithms based on diagnostic coding and drug dispensing claims can be used to estimate national gout prevalence.
- Ethnicity, social deprivation, male gender and increasing age are associated with higher gout prevalence.
- Further public health and primary care measures are needed to address gout risk factors.

Disclosure statement: The authors have declared no conflicts of interest.

Supplementary data

Supplementary data are available at *Rheumatology* Online.

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