General points

- Data is not presented where the number of people was below 10, to preserve confidentiality.
- People were assigned to their district health board (DHB) of domicile; where more than one domicile was recorded, we selected the most recent value. This was the same for their primary health organisation (PHO).
- We analysed ethnicity data by prioritised ethnic group (Māori, Pacific peoples, non-Māori, non-Pacific). For people reporting multiple ethnic groups, we selected the most recent value.
- Since the rates for Asian populations were similar to those of the European/Other population, and in some DHBs Asian populations were small, we combined these into a non-Māori, non-Pacific group. Data where ethnicity was not specified was excluded from the ethnicity analysis, meaning the sum of ethnicity data does not equal the total.

Exclusions

- The year of dispensing is the same as or after the year of death.
  OR
- The patient category is either J or Y (juvenile or youth).

Acknowledgements

The Health Quality & Safety Commission (the Commission) acknowledges the contribution of the gout expert advisory group in developing this Atlas of Healthcare Variation domain:

- Professor Nicola Dalbeth, (chair) rheumatologist, Department of Rheumatology, Auckland District Health Board (DHB) and Department of Medicine, University of Auckland
- Dr Doone Winnard, public health physician, Counties Manukau Health
- Leanne Te Karu, pharmacist prescriber – general practice, University of Auckland
- Dr Peter Gow, rheumatologist, Middlemore Hospital
- Prof Tony Dowell, professor of primary health care and general practice, University of Otago, Wellington.

Disclaimer

The Commission is responsible for the content of the gout Atlas domain and methodology; any errors of fact or interpretation are ours alone.

**Standard deviation**

Data is presented as standard deviation from the mean.

Standard deviation is a statistical measure of variation from a mean. Assuming that recorded instances are normally distributed (ie, they are in the usual ‘bell-shaped curve’), 68 percent of all recorded instances would be expected to be within one standard deviation either side of the mean and 95 percent within two standard deviations. The two ‘middle’ shades will be within one standard deviation of the mean.

**Confidence intervals**

Upper and lower confidence intervals were calculated to 95 percent level of confidence. All changes in indicator values listed in the landing page are statistically significant changes.
**Indicator #1**  
The prevalence of identified gout in those enrolled with primary health organisations (PHOs), by DHB (percent)

**Numerator**  
Those aged 20 years and over identified with gout using the HealthTracker method¹ (discharge diagnosis of gout (ICD 9 274, ICD 10 M10) from a public hospital admission from 1 January 1988 to 31 December 2019 or who have been dispensed gout-specific urate-lowering therapy (allopurinol, febuxostat, benzbromarone) or colchicine from a community pharmacy between 2001 and 2019) and excluding patients with a diagnosis of leukaemia or lymphoma (ICD 10 C81–C96). Only those enrolled with a PHO in each year were included.

**Denominator**  
Statistics New Zealand (Stats NZ) population aged 20 and over, using 2019 projections.

**Data source**  
Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry (numerator).  
Stats NZ: population projections (denominator).

**Analysis**  
By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

**Comments**  
The method used to calculate gout prevalence is similar to that described by Winnard et al (2012). This method is believed to underestimate prevalence by around 20 percent (Jackson et al 2012).

**Commentary**  
*Why is this important?*  
Rates of gout are particularly high in male Māori and Pacific peoples, meaning DHBs with a high Māori and/or Pacific population have higher prevalence.

**Indicator #2**  
People with identified gout who received urate-lowering therapy at least once in a year, by DHB (percent)

**Numerator**  
Number of people aged 20 years and over with a identified gout who were dispensed a gout-specific urate-lowering therapy (allopurinol, febuxostat, benzbromarone) at least once in a year from 1 January 2012 to 31 December 2019.

**Denominator**  
Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.

**Data source**  
Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.

**Analysis**  
By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

**Medicines**  
Urate-lowering therapy: 1026 allopurinol, 4026 febuxostat, 3754 and 4003 benzbromarone.

¹ See page 5 for details on HealthTracker definitions.
Commentary

Why is this important?

Long-term urate-lowering therapy is used to prevent gout flares and prevent tophus formation, bony erosions and permanent disability in people with gout. Many studies have demonstrated underutilisation of urate-lowering therapy continuously and long term. The results of this indicator are combined with indicator #3 to prompt questions and actions about ‘the persistence gap’ (see commentary for indicator #3).

What questions does this prompt?

- Why is there variation in the rate of urate-lowering therapy dispensing between DHBs?
- What factors might be contributing to low urate-lowering therapy use?

Indicator #3  People with identified gout who received urate-lowering therapy regularly, by DHB (percent)

<table>
<thead>
<tr>
<th>Numerator</th>
<th>Number of people aged 20 years and over with identified gout who were dispensed gout-specific urate-lowering therapy (allopurinol, febuxostat, benzbromarone) for three or four quarters in a year from 1 January 2012 to 31 December 2019.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denominator</td>
<td>Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.</td>
</tr>
<tr>
<td>Data source</td>
<td>Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.</td>
</tr>
<tr>
<td>Analysis</td>
<td>By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).</td>
</tr>
<tr>
<td>Medicines</td>
<td>Urate-lowering therapy: 1026 allopurinol, 4026 febuxostat, 3754 and 4003 benzbromarone.</td>
</tr>
</tbody>
</table>
| Commentary | Why is this important?
Long-term urate-lowering therapy is used to prevent gout flares and prevent tophus formation, bony erosions and permanent disability in people with gout. Many studies have demonstrated underutilisation of urate-lowering therapy. The map for this indicator suggests there is wide variation in the regular dispensing of urate-lowering therapy to those with a diagnosis of gout.

What questions does this prompt?

- Why is there variation in the rate of urate-lowering therapy dispensing between DHBs?
- Why are rates of urate-lowering therapy dispensing in Māori and Pacific peoples with gout higher than rates for non-Māori, non-Pacific populations but lower for regular dispensing?
- What other factors might be contributing to a persistent gap in urate-lowering therapy use?
<table>
<thead>
<tr>
<th>Indicator #4</th>
<th>People with identified gout dispensed funded non-steroidal anti-inflammatory drugs (NSAIDs), by DHB (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numerator</td>
<td>People with identified gout aged 20 years and over dispensed publicly funded NSAIDs.</td>
</tr>
<tr>
<td>Denominator</td>
<td>Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.</td>
</tr>
<tr>
<td>Data source</td>
<td>Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.</td>
</tr>
<tr>
<td>Analysis</td>
<td>By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).</td>
</tr>
<tr>
<td>Medicines</td>
<td>NSAIDs: diclofenac sodium, ibuprofen, ketoprofen, mefenamic acid, meloxicam, naproxen, naproxen sodium, tenoxicam, celecoxib and sulindac.</td>
</tr>
<tr>
<td>Comment</td>
<td>This indicator only includes NSAIDs dispensed at community pharmacies, ie, not medicines given in hospital or purchased over the counter. Note that the NSAID dispensing may not necessarily be related to gout symptoms.</td>
</tr>
<tr>
<td>Commentary</td>
<td><strong>Why is this important?</strong> NSAIDs are often the first line of treatment for acute gout flares and some can be purchased from a pharmacy without a prescription. This indicator does not capture over-the-counter NSAID use and does not assess the indication for NSAID use. This indicator may reflect people experiencing acute gout flares who are not on long-term preventive therapy for gout. Gout management guidelines state that people experiencing frequent (two or more) acute gout flares per year should be offered long-term urate-lowering therapy. Although NSAIDs are effective at treating acute gout flares, these medicines have important side effects including kidney injury and peptic ulcer disease.</td>
</tr>
<tr>
<td></td>
<td><strong>What questions does this prompt?</strong></td>
</tr>
<tr>
<td></td>
<td>• Should some of those receiving NSAIDs for gout flares be treated with long-term urate-lowering therapy?</td>
</tr>
<tr>
<td></td>
<td>• Would providing long-term urate-lowering therapy offer a better risk:benefit ratio?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indicator #5</th>
<th>People with identified gout who were dispensed funded NSAIDs but not dispensed urate-lowering therapy, by DHB (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numerator</td>
<td>People with identified gout aged 20 years and over, dispensed publicly funded NSAIDs in a year but not dispensed gout-specific urate-lowering therapy (allopurinol, febuxostat, benz bromarone) in the same year.</td>
</tr>
<tr>
<td>Denominator</td>
<td>Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.</td>
</tr>
</tbody>
</table>
Data source: Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.

Analysis: By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

Medicines: 
- NSAIDs: diclofenac sodium, ibuprofen, ketoprofen, mefenamic acid, meloxicam, naproxen, naproxen sodium, tenoxicam, celecoxib and sulindac.
- Urate-lowering therapy: 1026 allopurinol, 4026 febuxostat, 3754 and 4003 benzbromarone.

Comment: This indicator only includes NSAIDs dispensed at community pharmacies, ie, not medicines given in hospital or purchased over the counter. Note that the NSAID dispensing may not necessarily be related to gout symptoms.

Commentary: Why is this important?
While it is not known whether it was gout that led to an NSAID being dispensed, high rates of NSAID dispensing without urate-lowering therapy use may reflect people using NSAIDs to manage their acute gout flares. International best practice indicates that people experiencing frequent gout flares (two or more per year) should be offered long-term urate-lowering therapy. Large variation may reflect patient preference and/or variable clinical practice.

This indicator does not capture those who use over-the-counter medicine.

What questions does this prompt?
- Are people with gout using more or fewer NSAIDs without urate-lowering therapy than might be expected?
- Should some of those receiving NSAIDs for acute gout flares be treated with urate-lowering therapy?

Indicator #6: People with identified gout dispensed any of colchicine, prednisone or funded NSAIDs in a year, by DHB (percent)

Numerator: People with identified gout, aged 20 years and over dispensed any of colchicine, prednisone or funded NSAIDs in a year.

Denominator: Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.

Data source: Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.

Analysis: By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

Medicines: Colchicine, prednisone, diclofenac sodium, ibuprofen, ketoprofen, mefenamic acid, meloxicam, naproxen, naproxen sodium, tenoxicam, celecoxib and sulindac.
Comment

This indicator only includes colchicine, prednisone or NSAIDs dispensed at community pharmacies, ie, medicines given in hospital or purchased over the counter are not included. Note that the NSAID dispensing may not necessarily be related to gout symptoms.

Commentary

Why is this important?

Acute gout flares are treated with NSAIDs, colchicine or oral corticosteroids such as prednisone. Gout management guidelines state that people experiencing frequent (two or more) acute gout flares per year should be offered long-term urate-lowering therapy. Although NSAIDs are effective at treating acute gout flares, these medicines have important side effects including kidney injury and peptic ulcer disease.

This indicator does not capture over-the-counter NSAID use and does not assess the indication for NSAID use.

What questions does this prompt?

- Should some of those receiving colchicine, prednisone or NSAIDs for gout flares be treated with long-term urate-lowering therapy?
- Would providing long-term urate-lowering therapy offer a better risk:benefit ratio?

Indicator #7

People with identified gout dispensed any of colchicine, prednisone or funded NSAIDs but not urate-lowering therapy in a year, by DHB (percent)

Numerator

People with identified gout, aged 20 years and over dispensed any of colchicine, prednisone or funded NSAIDs in a year but not dispensed gout-specific urate-lowering therapy (allopurinol, febuxostat, benzbromarone) in the same year.

Denominator

Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.

Data source

Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Mortality Collection, New Zealand Cancer Registry.

Analysis

By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

Medicines

Colchicine, prednisone, diclofenac sodium, ibuprofen, ketoprofen, mefenamic acid, meloxicam, naproxen, naproxen sodium, tenoxicam, celecoxib and sulindac.

Urate-lowering therapy: allopurinol, febuxostat, benzbromarone.

Comment

This indicator only includes colchicine, prednisone or NSAIDs dispensed at community pharmacies, ie, not medicines given in hospital or purchased over the counter. Note that the NSAID dispensing may not necessarily be related to gout symptoms.

Commentary

Why is this important?

Acute gout flares are treated with NSAIDs, colchicine or oral corticosteroids such as prednisone. This indicator may reflect people...
experiencing acute gout flares who are not on long-term preventive therapy for gout. Gout management guidelines state that people experiencing frequent (two or more) acute gout flares per year should be offered long-term urate-lowering therapy. Although NSAIDs are effective at treating acute gout flares, these medicines have important side effects including kidney injury and peptic ulcer disease.

This indicator does not capture over-the-counter NSAID use and does not assess the indication for NSAID or prednisone use.

What questions does this prompt?

- Are people with gout using more or less colchicine, prednisone or NSAIDs without urate-lowering therapy than might be expected?
- Should some of those receiving colchicine, prednisone or NSAIDs for acute gout flares be treated with urate-lowering therapy?

### Indicator #8 Serum urate testing in the six months following urate-lowering therapy dispensing, by DHB (percent)

**Numerator**
Number of people with identified gout aged 20 years and over who had a recorded serum urate laboratory test in the six months following gout-specific urate-lowering therapy (allopurinol, febuxostat, benzbromarone) dispensing.

**Denominator**
Those aged 20 years and over with an indication of gout using the HealthTracker method (numerator indicator #1) who were dispensed urate-lowering therapy in a year.
Only those enrolled with a PHO in each year were included.

**Data source**
Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Laboratory Claims Mortality Collection, New Zealand Cancer Registry.

**Analysis**
By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).
Laboratory testing was calculated in the years after the year of diagnosis, e.g., people diagnosed during the year 2014 were included in the cohort for 2015–19, but not for 2014.

**Medicines**
Urate-lowering therapy: 1026 allopurinol, 4026 febuxostat, 3754 and 4003 benzbromarone.

**Comment**
This indicator differs from that reported for gout in the Equity Explorer (www.hqsc.govt.nz/atlas/equity-explorer).
Commentary

Why is this important?
Best practice guidelines recommend that people with gout who are prescribed urate-lowering therapy should have their serum urate level monitored at least every six months. Long-term urate-lowering therapy is not recommended for people with asymptomatic hyperuricaemia. The data presented in this indicator cannot tell us what the result was, only whether or not a test was performed.

What questions does this prompt?
- Why is serum urate testing not occurring at recommended intervals in most patients with gout?
- Why is there variation in rates of serum urate testing between DHBs?

Indicator #9
The number of admissions with a primary diagnosis of gout in those identified with gout, by DHB (rate per 100,000)

Numerator
The number of admissions in people aged 20 years and over with a publicly funded hospital discharge with primary diagnosis of gout (ICD 9 274, ICD 10 M10), by DHB.

Denominator
Those aged 20 years and over with identified gout using the HealthTracker method (numerator indicator #1). Only those enrolled with a PHO in each year were included.

Data source
Ministry of Health: National Minimum Dataset, Pharmaceutical Collection, PHO enrolments, Laboratory Claims Mortality Collection, New Zealand Cancer Registry.

Analysis
By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).

Commentary
Why is this important?
Variation in admission rates to hospital due to gout may indicate different admission criteria between DHBs, poor gout control or limited access to primary care.

What questions does this prompt?
- How much of the variation is due to differing prevalence of disease and how much is modifiable?
- Could some of the high admission rates be prevented by more intensive primary care management?
- Do some of these admissions reflect higher disease severity?

Note: The denominator of this indicator has been changed to match the denominator for indicators #2–#7 so it is now displaying hospital admissions for people with gout, rather than for the general population.

This indicator shows differences in admission rates within the gout population. It isn't affected by the prevalence of gout.
<table>
<thead>
<tr>
<th>Indicator #10</th>
<th><strong>The number of admissions with a primary diagnosis of gout in the general population, by DHB (rate per 100,000)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Numerator</td>
<td>The number of admissions in people aged 20 years and over with a publicly funded hospital discharge with primary diagnosis of gout (ICD 9 274, ICD 10 M10), by DHB.</td>
</tr>
<tr>
<td>Denominator</td>
<td>Stats NZ population projection aged 20 years and over, using 2019 projections.</td>
</tr>
<tr>
<td>Analysis</td>
<td>By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).</td>
</tr>
</tbody>
</table>
| Commentary | **Why is this important?** <br>Variation in admission rates to hospital due to gout may indicate different admission criteria between DHBs, poor gout control or limited access to primary care.  
**What questions does this prompt?**  
- How much of the variation is due to differing prevalence of disease and how much is modifiable?  
- Could some of the high admission rates be prevented by more intensive primary care management?  
- Do some of these admissions reflect higher disease severity?  
This indicator shows the impact of a higher gout prevalence on admission rates. |

<table>
<thead>
<tr>
<th>Indicator #11</th>
<th><strong>The dispensing of funded NSAIDs in the rest of the New Zealand resident population, by DHB (percent)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Numerator</td>
<td>PHO-enrolled population aged 20 years and over who were dispensed NSAIDs, excluding those with identified gout.</td>
</tr>
<tr>
<td>Denominator</td>
<td>Stats NZ population projection aged 20 years and over, using 2019 projections.</td>
</tr>
<tr>
<td>Analysis</td>
<td>By year (2012–19), by age (20–44, 45–64, 65 years and over) and by ethnicity (Māori, Pacific peoples, non-Māori, non-Pacific).</td>
</tr>
<tr>
<td>Medicines</td>
<td>NSAIDs: diclofenac sodium, ibuprofen, ketoprofen, mefenamic acid, meloxicam, naproxen, naproxen sodium, tenoxicam, celecoxib and sulindac.</td>
</tr>
</tbody>
</table>
Comment

This indicator only includes NSAIDs dispensed at community pharmacies, ie, not medicines given in hospital or purchased over the counter.

HealthTracker definition

This definition aligns with the definition used by Winnard et al (2012) with two exceptions:
1. The cancer exclusion codes used in this indicator were C81–C96 (not C80–C96).
2. Benzbromarone and febuxostat were added to allopurinol in the definition to become urate-lowering therapy. Febuxostat, but not benzbromarone, was excluded alongside allopurinol as an indicator of gout when the relevant cancer(s) were diagnosed in the 24 months before the period end date.

Data sources used

- National Minimum Dataset
- Laboratories Collection
- Mortality Collection
- Pharmaceutical Collection
- PHO enrolment
- Cancer registrations.

Values of the main indicator variable

1 = Yes, the person showed an indication of having gout, as recorded in national health information systems.

A person is counted as having showed an indication of gout if they have one of the codes specified below (from any data collection) within the time period searched (eg, 12-month or lifetime).

National Minimum Dataset codes:
- ICD10 diagnosis codes: M10
- ICD 9 CM diagnosis codes: 274.

Pharmaceutical Collection:
- 1341 – colchicine
- 1026 – allopurinol*
- 4026 – febuxostat*
- 3754 – benzbromarone
- 4003 – benzbromarone.

* Dispensings of allopurinol or febuxostat are excluded as indications of gout if there is a diagnosis of malignant neoplasms of lymphoid, haematopoietic and related tissues (ICD-10-AM C81-C96) recorded in either the New Zealand Cancer Registry or in the publicly funded hospitalisations in the National Minimum Dataset in the 24 months before the period end date.

References