Arthritis Research UK

Condition
Gout

Gout

This booklet provides information and answers to your questions about this condition.

What is gout?

Gout is often said to be the most painful form of arthritis. In this booklet we'll explain what causes it, how it can be treated and what you can do to reduce the risk of further attacks of gout. We'll also suggest where you can find out more about living with gout.

At the back of this booklet you'll find a brief glossary of medical words – we've underlined these when they're first used in the booklet.

What's inside?

Gout can be intensely painful. Fortunately, there are a number of treatments available that can ease the pain and others that can reduce the risk of further attacks or even get rid of the problem altogether. With suitable treatment, gout is unlikely to result in permanent joint damage.

At a glance
Gout

Gout is caused by a build-up of urate, which forms crystals in the joints.

What are the symptoms of gout?

Gout can have some painful symptoms, including:

- intense and rapidly developing pain in the affected joint (often the big toe)
- affected joints feeling hot and very tender to the touch
- affected joints looking swollen
- skin that looks shiny and often red.

What causes it?

Gout occurs when the body is unable to flush out excess uric acid or urate (produced by the body's own cells by the breakdown of food). When urate builds up above a certain level it can form crystals of sodium urate, particularly in the joint cartilage. Occasionally these crystals escape from the cartilage and trigger sudden painful inflammation of the joint lining.

What treatments are there?

Initial treatments for acute attacks include:

- ice packs
- non-steroidal anti-inflammatory drugs (NSAIDs)
- colchicine tablets
- steroids (injected into the joint or muscle, or as tablets).

Longer term treatments aim to lower urate levels and reduce the risk of further attacks. These include:

- allopurinol or febuxostat (which reduce urate production by the body)
- uricosuric drugs (which increase excretion of urate by the kidney).

How can I help myself?

Try the following to reduce your risk of attack:

- Lose weight if overweight.
- Eat less purine-rich foods (e.g. offal, oily fish, yeast extracts).
- Avoid dehydration by drinking plenty of water.
- Drink less alcohol (3–4 units per day for men, 2–3 units per day for women).
- Increase your intake of foods rich in vitamin C.

What is gout?

Gout is an intensely painful form of arthritis – it's said to be as painful as childbirth. Attacks of gout usually come on very quickly (doctors describe the sudden onset of symptoms as acute), often during the night. Of all the forms of arthritis, gout is the one we understand the best, and this has led to a range of therapies to treat acute attacks and control the condition.

Gout is the most common type of inflammatory arthritis, affecting 1.4% of adults in the UK. It affects more men than women and can affect men of any age. Women rarely develop gout before the menopause but may do so later in life. With more people living longer, gout is starting to become more common in women.

At one time it was thought that gout was caused simply by eating and drinking too much. While it's true that over-indulging in alcohol or food can make attacks of gout more likely, that's not the whole story.

People get gout because of certain chemical processes that take place within the body. A substance called urate builds up (sometimes because the kidneys aren't able to get rid of it quickly enough) and forms crystals in the joints, which can lead to painful inflammation. A tendency to attacks of gout may be inherited from a parent or a grandparent.

What are the symptoms of gout?

Urate crystals cause inflammation, meaning the joint becomes intensely painful, red, hot and swollen (see Figure 1). The skin over the joint often appears shiny and may peel. Attacks typically affect the big toe and usually start at night. The symptoms develop over just a few hours. Any contact with the affected joint is painful – even the weight of the bedclothes can cause pain.

Although gout most often affects the big toe, other joints in the legs and arms may also be affected, including the midfoot, ankles, knees, elbows, wrists and fingers. If several joints are inflamed at once this is called polyarticular gout. It's very rare to have gout in joints towards the centre of the body such as the spine, shoulders or hips.

Urate crystals can also collect outside of the joints and even be visible under the skin, forming small, firm white lumps called tophi (see Figure 2). These aren't usually painful but sometimes they break down and discharge pus-like fluid containing gritty white material, the urate crystals themselves.

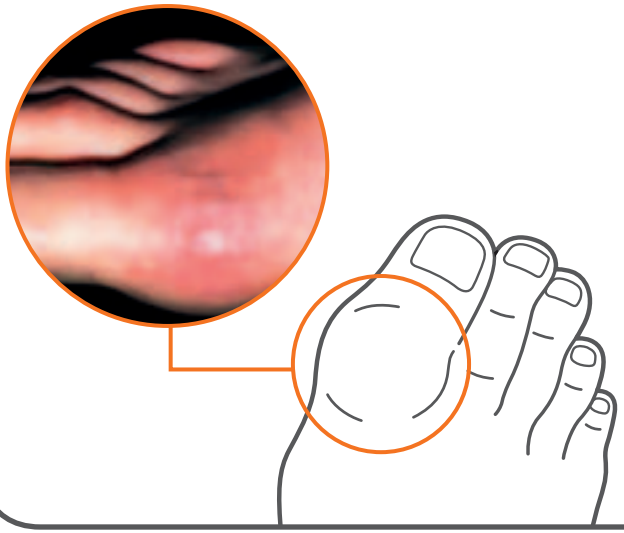

Figure 1
Gout affecting the big toe

Gout most commonly affects the big toe. The joint becomes red, hot, swollen and extremely painful.

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Edited by Marc C Hochberg, Alan J Silman, Josef S Smolen, Michael E Weinblatt and Michael H Weisman.

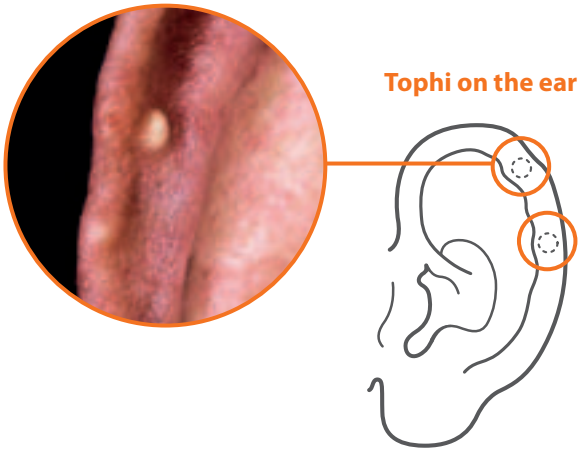

Figure 2
Tophi caused by urate collecting under the skin

Urate may collect under the skin forming small white pimples (tophi), but these aren't usually painful.

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Edited by Marc C Hochberg, Alan J Silman, Josef S Smolen, Michael E Weinblatt and Michael H Weisman.

What causes gout?

Gout occurs in people who have a higher-than-normal level of urate in the bloodstream. About two-thirds of the urate in our bodies comes from the breakdown of purines which are present in the cells of our bodies. The other third comes from the breakdown of purines in some of the foods we eat.

But the presence of urate in the blood does not itself lead to gout. It's normal and healthy to have some urate in the bloodstream, and the level is usually higher in men than in women. When urate levels start to build up, the body normally rids itself of any excess urate through the kidneys into the urine.

If the body is making too much urate or if the kidneys are unable to remove excess urate effectively, then urate levels start to rise. If the level goes above a certain critical concentration (the saturation point), it's possible for urate to form crystals of sodium urate. These crystals mainly form in and around joint tissues, especially joints at the ends of the legs and arms, such as the finger and toe joints.

Figure 3 is a joint showing urate crystals. The crystals gradually build up in the cartilage and after some years can spill out into the joint cavity (this is known as crystal shedding). The hard, needle-shaped crystals come into contact with the soft lining of the joint (the synovium) and make it very inflamed very quickly. The inflammation process dissolves the few crystals that have got loose inside the joint, and the attack gradually settles over a few days or weeks, depending on how many crystals were shed.

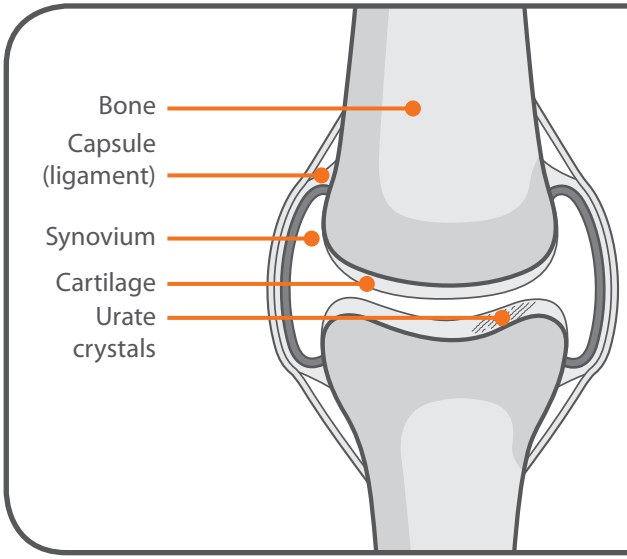


Figure 3 Cross-section of joint with urate crystals

Cross-section of a joint showing urate crystals deposited in the cartilage.

Apart from causing sudden attacks of inflammation, a build-up of crystals can eventually lead to the formation of stones called tophi in and around the joints. These hard tophi can grow and cause pressure damage to the joint cartilage and bone. This is just like the damage caused by osteoarthritis and can cause more regular, daily pain when the joints are used. At this stage the condition is often called chronic tophaceous gout. Some tophi may be seen and easily felt under the skin, but by the time they're visible from the outside the unseen part of the tophi may be quite extensive.

There are several factors that can affect the level of urate in our bodies:

- An inherited (genetic) tendency may make some people's kidneys less efficient at clearing urate, even though the kidneys are otherwise completely normal and healthy. This is one of the most common causes, especially when there are several family members affected.
- The bigger the body the more urate is produced each day, so being overweight or obese may overwhelm the kidneys' ability to get rid of the urate effectively.
- High levels of cholesterol and lipids (fats) in the blood (hyperlipidaemia), high blood pressure and late-onset (Type 2) diabetes all reduce the kidneys' ability to excrete urate efficiently, and all these conditions tend to be associated with raised urate levels. This combination of problems is often called metabolic syndrome.

- Kidney disease may mean that your kidneys aren't able to process urate as well as they should.
- Tablets such as diuretics (water tablets) drain water from the body and reduce the kidneys' ability to get rid of urate effectively. Other drugs that do this include low-dose aspirin and ciclosporin.
- Rarely, in chronic blood disorders where the body produces too many blood cells, urate produced by the breakdown of those cells may be more than the kidneys can cope with.

Where a definite cause can be identified (such as kidney disease or regular use of diuretic drugs), the condition is described as secondary gout. However, most gout is primary and is usually due to a combination of factors, for example through having inherited kidneys that aren't very efficient at getting rid of urate and then getting a little overweight.

If you're prone to gout, several things can encourage urate crystals to shake loose from the cartilage and trigger an acute attack. These can include:

- a knock or injury to the joint
- an illness, such as pneumonia or flu, that makes you feverish
- an operation – this also puts your temperature up a little
- excessive eating and drinking of alcohol
- periods of dehydration.

Similar attacks can be caused by calcium crystals that are deposited in joint cartilage and then shed into the joint space. This type of crystal arthritis (termed acute calcium pyrophosphate crystal arthritis or pseudogout) affects the knee and other joints more than the big toe and is most common in people with osteoarthritis.

See Arthritis Research UK booklet *Calcium crystal diseases (pseudogout).*

What is the outlook?

The frequency of attacks is very variable. Some people have an attack only every few years, while others have attacks every few months. In time, though, the frequency of attacks tends to increase and new joints are often affected.

Although the acute attacks of gout are very dramatic, the inflammation subsides relatively quickly and the attacks themselves probably don't cause long-term joint damage. However, a continued build-up of urate crystals and formation of hard tophi can damage the cartilage and bone of joints, leading to long-term (chronic) arthritis.

With modern treatments and possibly some changes to your diet and lifestyle, this type of damage can usually be prevented by bringing urate levels in your tissues down below the saturation point at which crystals form.

With treatment, once the attack has passed then further pain and joint damage is unlikely.

Lowering your urate levels will prevent new crystals forming and slowly dissolve the crystals that are already there. It may take as long as 2 years of treatment to completely clear your body of urate crystals, but once they're gone then the risk of acute attacks of gout and of further joint damage from tophi is removed.

Because gout is associated with metabolic syndrome, you should pay special attention to these other aspects of your health. If gout is left untreated, urate can sometimes form stones in the kidneys, so tests may be needed to check how well the kidneys are working.

How is gout diagnosed?

What tests are there?

A diagnosis is often based on your symptoms and an examination of the joints, but your doctor may suggest the following tests:

A blood test can measure the amount of urate in the blood. A raised level of urate strongly supports a diagnosis of gout but can't confirm it. For one thing, not everyone with a raised level of urate will develop crystals in the joint tissues. Also, it's possible for urate levels in the blood to be normal at the time of an acute attack.

X-rays of joints will reveal joint damage in long-standing and poorly controlled gout. However, they're rarely helpful in confirming the diagnosis because they're usually normal in the early years of having gout.

Examination of joint fluid (synovial fluid) can be taken from a joint through a needle and examined under a microscope, where any urate crystals can be clearly seen. This test can confirm the diagnosis but isn't always practical. In particular, it's difficult, and sometimes uncomfortable for the patient, to obtain fluid from a small joint such as the big toe. However, it may be possible to identify a few urate crystals in a sample of fluid taken from the knee, even if you've not yet had an attack of gout at the knee. A fine needle inserted into a tophus under the skin can also be used to identify urate crystals.

What treatments are there for gout?

There are two main aspects to the treatment of gout (see Figure 4). These are:

- treating the acute attack of inflammation
- ongoing treatment to get rid of urate crystals and reduce the level of urate in the blood.

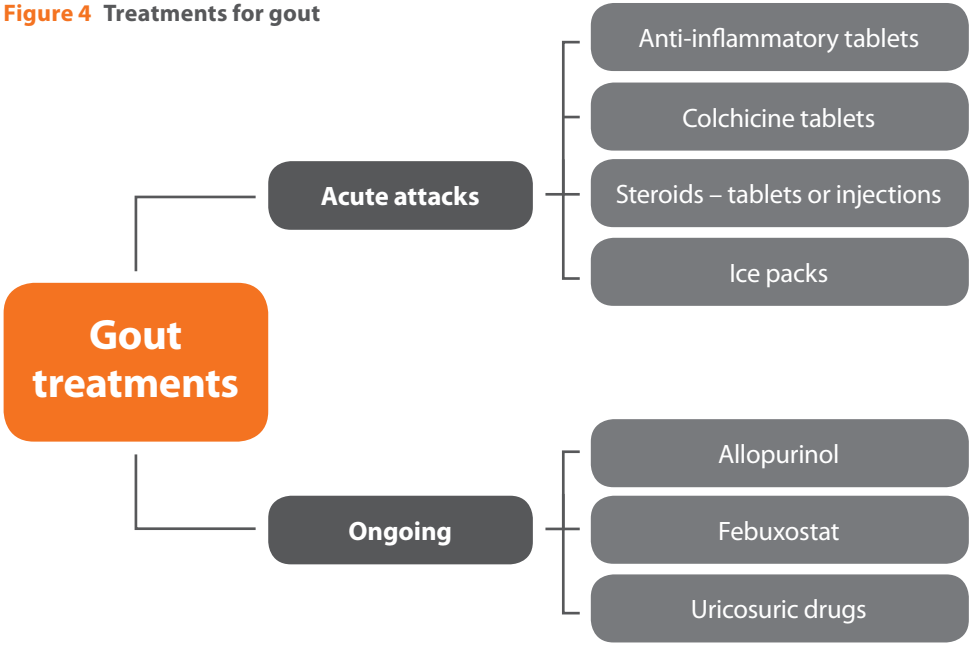
If you only have occasional attacks of gout then you'll probably only need treatment to deal with those. But if your attacks become more frequent or blood tests show that urate levels are building up, you may need ongoing treatment to reduce the urate in your blood and the risk of further attacks.

Treatments for acute attacks of gout

The two most commonly used drug treatments for acute attacks of gout are non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine.

If you've had attacks of gout before, be on the lookout for early signs of another attack and take the prescribed treatment as soon as possible. The earlier you can start treating an acute attack the better.

Figure 4 **Treatments for gout**



If you've had an attack before, be on the lookout for early signs of another attack and take your prescribed treatment as soon as possible. The earlier you start treating an acute attack the better.

If you only have occasional attacks of gout then you'll probably only need treatment to deal with those attacks.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Acute attacks of gout are often treated with oral non-steroidal anti-inflammatory drugs (NSAIDs). They can relieve pain and possibly reduce some of the inflammation. Examples include ibuprofen, naproxen, diclofenac and etoricoxib.

Like all drugs, NSAIDs can sometimes have side-effects, but your doctor will take precautions to reduce the risk of these – for example, by prescribing the lowest effective dose for the shortest possible period of time.

NSAIDs can cause digestive problems (stomach upsets, indigestion, or damage to the lining of the stomach) so in most cases NSAIDs will be prescribed along with a drug called a proton pump inhibitor (PPI), which will help to protect the stomach.

NSAIDs also carry an increased risk of heart attack or stroke. Although the increased risk is small, your doctor will be cautious about prescribing NSAIDs if there are other factors that may increase your overall risk – for example, smoking, circulation problems, high blood pressure, high cholesterol or diabetes.

See Arthritis Research UK drug leaflet *Non-steroidal anti-inflammatory drugs.*

Colchicine

Colchicine is derived from the crocus plant. It's not a painkiller but it's often very effective at damping down the inflammation caused by the crystals interacting with the joint lining. As with NSAIDs, colchicine tablets should be taken at the very beginning of an attack. Your doctor may let you keep a supply so you can start taking them at the first signs.

The recommended dose is 0.5 mg two to four times per day, depending on your size, age and whether you have other health problems. Some people are unable to take colchicine because they have side-effects such as nausea, vomiting or diarrhoea. For this reason it's best to start at a low dose and only increase it if there's no upset.

Colchicine can also be taken longer term at a dose of 0.5 mg once or twice a day to suppress the tendency to have gout attacks. However, like NSAIDs, colchicine won't reduce the urate level in the blood, so it won't help to get rid of the urate crystals or prevent long-term joint damage.

Steroids

If an acute attack of gout doesn't improve with NSAIDs or colchicine or if you're at risk of side-effects from these drugs, your doctor may prescribe a steroid injection into the joint or muscle, or a short course of steroid tablets (usually no more than a few days).

See Arthritis Research UK drug leaflets *Local steroid injections; Steroid tablets.*

Other treatments for acute attacks

Putting an ice pack on the affected area can reduce some of the swelling, heat and pain, and they're very safe provided that you protect the skin from direct contact with the ice (commercial re-usable cooling pads are available). These should always be used in addition to any of the drug treatments for acute gout.

Resting the painful inflamed joint also takes some of the edge off the severe pain. A cage over the affected foot or knee to take the weight of the bedclothes can help.

Ongoing treatments to reduce urate

The drugs given to relieve an acute attack don't get rid of the urate crystals in your joints or reduce the level of urate in your blood. In some circumstances your doctor may recommend that you start taking a urate-lowering drug once a day with the aim of getting rid of the urate crystals. This is most likely if:

- attacks are occurring frequently
- you have tophi, kidney stones or any signs of joint damage
- tests show that you have a very high level of urate in your blood.

You may still have acute attacks when you first begin the treatment, so you may wish to take daily NSAIDs or colchicine to suppress inflammation while your urate level is brought down. You'll be at risk of acute attacks for at least 6 months and probably longer. It can take as long as 2 years to clear your body completely of urate crystals.

Urate-lowering drugs are usually very well tolerated, but you might have to stop using them if you have side-effects such as a rash or indigestion (dyspepsia). Aside from this, you should continue to take them indefinitely. Interruptions in dosage, especially in the early stages, can cause fluctuations in the urate level, which seem to trigger acute attacks.

It's also important to consider other ways of reducing your urate levels – for example, by losing weight if you're overweight and by changing your diet. If you have other features of metabolic syndrome (high blood pressure, high lipids, diabetes), good control of these will also help to reduce your urate levels.

Allopurinol

Allopurinol is the most commonly used urate-lowering drug. It works by reducing the body's production of urate and is usually taken once a day.

Having measured your blood urate level, your doctor will probably start you on a dose of 100 mg a day and then retest your blood urate level after about a month. If the urate level hasn't come down enough then your dose will be increased by 100 mg. You may need several dose increases, each of 100 mg, approximately every month, until you're at the right dose that keeps the blood urate level well below the saturation point. The maximum

dose of allopurinol is 900 mg but most people reach the target urate level by taking 200–500 mg. Allopurinol is available as 100 mg and 300 mg tablets so you don't need to take a lot of tablets if you need a higher dose.

Once the urate level is well below the saturation point, you should continue on that dose of allopurinol with repeat blood checks every 12 months or so to ensure that your urate level is still low. The main reason not to start with a large dose is that rapid reduction of urate levels can actually trigger an acute attack. This is probably because the crystals in your cartilage become smaller as they start to dissolve, which allows them to shake loose more easily and shed into the joint cavity. However, if urate levels are brought down slowly by gradually increasing the dose of allopurinol, it's much less likely that it will trigger an acute attack.

Increasing the dose gradually is also less likely to result in side-effects such as a rash, headache or nausea. If you do develop any side-effects soon after starting allopurinol, you should stop taking the tablets and see your doctor, who will advise whether you should restart the tablets and what precautions you should take.

It's also important to consider other ways of reducing your urate levels – for example, by losing weight if you're overweight and changing your diet.

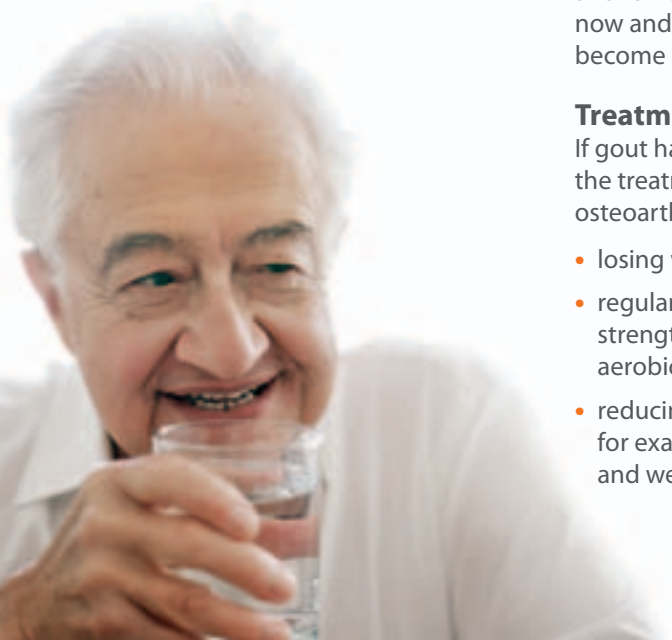
It's important to keep taking allopurinol. The most common reason for failure of allopurinol is the patient not taking the drug regularly or at the correct dose.

Allopurinol can affect some other tablets, especially warfarin and azathioprine. If you have to take either of these drugs for any reason, you **must** tell the doctor who prescribes them that you're also taking allopurinol. The dose of the other drug may need to be adjusted.

See Arthritis Research UK drug leaflets *Allopurinol; Azathioprine.*

Febuxostat

Febuxostat is another drug that has recently become available. Like allopurinol, it reduces the body's production of uric acid, but unlike allopurinol it's broken down by the liver and is therefore particularly useful in people with kidney problems who can't take a high enough dose of allopurinol.



Febuxostat comes in just two doses. The starting dose is 80 mg, which is quite strong and may trigger acute attacks. It's therefore recommended that you take a daily NSAID or colchicine for at least 6 months to help protect against this. If urate levels aren't reduced after a month, the dose of febuxostat can be increased to 120mg daily.

Other urate-lowering drugs

Uricosuric drugs, which include sulfinpyrazone, benzbromarone and probenecid, work by flushing out more urate than normal through the kidneys. These drugs may not be suitable if you've had kidney stones or similar disorders. They're not widely used in the UK, but they may be a useful alternative if allopurinol isn't suitable for you.

Other drugs for treating acute attacks and for lowering urate are in development now and it's likely that new drugs will become available in due course.

Treatment of joint damage

If gout has already caused joint damage, the treatment will be the same as for osteoarthritis, including:

- losing weight if overweight
- regular daily exercises (both muscle-strengthening exercise and general aerobic exercise)
- reducing strain on the affected joint – for example, by pacing your activities and wearing appropriate footwear

- painkillers – e.g. paracetamol, codeine
- anti-inflammatory creams and gels
- anti-inflammatory tablets
- topical capsaicin cream
- steroid injections into the painful joint
- surgery, including joint replacement.

See Arthritis Research UK booklets *Osteoarthritis; Osteoarthritis of the knee.*

Self-help and daily living

Diet and nutrition

There are a number of changes you can make to your diet to ease attacks of gout. The most useful things you can do are:

- losing weight
- drinking less alcohol
- drinking plenty of water.

Weight loss

Losing weight, if you need to, is the most effective dietary treatment for gout because it can significantly reduce the urate levels in your body.

Weight loss should be gradual and combined with daily exercise. Extreme weight loss or starvation diets increase cell breakdown in the body, which can raise urate levels.

We don't recommend Atkins-type weight-loss diets for people who are prone to gout. These diets include a lot of meat and are therefore high in animal proteins. These are high in purines, which break down to produce urate.

Fluids

Drinking plenty of water may reduce the risk of triggering an attack and may also reduce the risk of urate crystallising in joint tissues. If you have kidney stones you may need as much as 3.5 litres (6 pints) a day. Even if you don't have kidney stones you should aim for at least 1 litre (2 pints) of fluid a day.

You can include some other fluids besides water in this total but not beer or other alcoholic drinks. However, many soft drinks contain large amounts of sugar, in the form of fructose. Keep these to a minimum as fructose sugar is likely to increase the level of urate in the blood. Diet soft drinks don't appear to increase the risk of gout.

There's some research which suggests that drinking coffee regularly may help by increasing excretion of urate through the kidneys. This doesn't appear to be due to caffeine but to some other factor which hasn't yet been identified.

Drinking a glass of skimmed milk every day may help to prevent attacks of gout.

Alcohol

Excessive alcohol consumption, especially beer and spirits, is associated with gout. A moderate intake of wine doesn't appear to increase the risk. It's important for many reasons to keep your alcohol intake well below the levels recommended by the government: 3–4 units a day for men and 2–3 units a day for women.

Remember that units are calculated from the strength of the drink as well as the quantity. See Figure 5 for approximate units of alcohol in some popular drinks.

It's a good idea to have at least two alcohol-free days a week – but without compensating over the remaining five days.

Figure 5 Approximate units of alcohol in some popular drinks

Drink	Units
Beer, lager, stout	
Ordinary strength (4% abv)	
Bottle (330ml)	1.3 units
Can (440ml)	1.8 units
Pint (568 ml)	2.3 units
Premium strength (5% abv)	
Bottle (330 ml)	1.7 units
Can (440 ml)	2.2 units
Pint (568 ml)	2.8 units
Lager	
Super strength (9% abv)	
Bottle (330 ml)	3 units
Can (440 ml)	4 units
Pint (568 ml)	5.1 units
Cider	
Ordinary strength (6% abv)	
Bottle (330 ml)	2 units
Can (440 ml)	2.6 units
Pint (568 ml)	3.4 units

Drink	Units
Cider (cont...)	
Strong (9% abv)	
Bottle (330 ml)	3 units
Can (440 ml)	4 units
Pint (568 ml)	5.1 units
Wine, red or white (13% abv)	
Standard glass (175 ml)	2.3 units
Large glass (250 ml)	3.2 units
Gin, rum, vodka, whisky (40% abv)	
Small measure (25 ml)	1 unit
Large measure (35 ml)	1.4 units
Sherry, port (20% abv)	
Standard measure (50 ml)	1 unit
Alcopops (5% abv)	
Bottle (275 ml)	1.4 units

NOTE: These figures are based on typical strengths for the drinks shown; actual strengths vary from one brand to another. Bear in mind also that 'measures' poured at home are often much larger than the standard pub measures shown.

Other diet tips

Limiting your intake of foods that are particularly high in purines may be helpful, whether or not you need to lose weight. These include:

- **red meat and offal** – e.g. beef, kidneys, liver, sweetbreads
- **oily fish** – e.g. anchovies, fish roes, herring, mackerel, sardines
- **foods rich in yeast extracts** – e.g. Marmite, Bovril, Vegemite.

Aim to reduce the amount of protein you get from meat by eating one less portion of meat or fish per day, for example. This can be replaced by other sources of protein, such as beans, eggs, pulses or low-fat dairy products.

Vitamin C encourages the kidneys to excrete more urate, so a diet rich in vitamin C may be beneficial. This is another reason to make sure your diet includes plenty of fruit and vegetables. There's some evidence that cherries may be beneficial – either the fruit or the juice, fresh or preserved – though it's unclear whether this is just due to the vitamin C content.

Complementary medicine

There's little evidence for many of the other natural or herbal remedies and supplements available for gout. These include celery seeds, garlic, artichokes and saponins (natural compounds found in peas, beans and some other vegetables).

Generally speaking, though, complementary and alternative therapies are relatively well tolerated, but you should always discuss their use with your doctor before starting treatment. There are some risks associated with specific therapies.

In many cases the risks associated with complementary and alternative therapies are more to do with the therapist than the therapy. This is why it's important to go to a legally registered therapist, or one who has a set ethical code and is fully insured.

If you decide to try therapies or supplements you should be critical of what they're doing for you, and base your decision to continue on whether you notice any improvement.

Ongoing research is greatly increasing our understanding of what triggers gout and how new therapies may be developed to treat it.

Research and new developments

Ongoing research is greatly increasing our understanding of what triggers gout and how new therapies may be developed to treat it. Meanwhile, a preliminary study funded by Arthritis Research UK aims to find out if more people with gout could benefit from treatment including urate-lowering drugs, together with dietary and weight loss advice, rather than simply treating the symptoms of acute attacks. This could lead to a full-scale clinical trial to establish the most effective package of treatment for people with gout.

Glossary

Azathioprine – a drug commonly used to help prevent rejection of transplanted organs. It works by suppressing the body's immune system and is also used in rheumatoid arthritis to prevent the immune system attacking the joints.

Cartilage – a layer of tough, slippery tissue that covers the ends of the bones in a joint. It acts as a shock-absorber and allows smooth movement between bones.

Dehydration – a condition where the normal water content of your body is reduced. The human body is about two-thirds water. The amount of water in the body only has to be reduced by a few percent before the chemical balance in the body is affected. Dehydration can be caused by illness, exhaustion or not drinking enough fluids.

Diabetes – a medical condition that affects the body's ability to use glucose (sugar) for energy. The body needs insulin, normally produced in the pancreas, in order to use glucose. In diabetes the body may produce no insulin or not enough insulin, or it may become resistant to insulin. When the body is unable to use glucose obtained from foods, the level of sugar in the blood increases. If untreated, raised blood sugar can cause a wide variety of symptoms.

Inflammation – a normal reaction to injury or infection of living tissues. The flow of blood increases, resulting in heat and redness in the affected tissues, and fluid and cells leak into the tissue, causing swelling.

Non-steroidal anti-inflammatory drugs (NSAIDs) – a large family of drugs prescribed for different kinds of arthritis that reduce inflammation and control pain, swelling and stiffness. Common examples include ibuprofen, naproxen and diclofenac.

Osteoarthritis – the most common form of arthritis (mainly affecting the joints in the fingers, knees, hips), causing cartilage thinning and bony overgrowths (osteophytes) and resulting in pain, swelling and stiffness.

Proton pump inhibitor (PPI) – a drug that acts on an enzyme in the cells of the stomach to reduce the secretion of gastric acid. They're often prescribed along with non-steroidal anti-inflammatory drugs (NSAIDs) to reduce their side-effects.

Purines – nitrogen-containing compounds, found mostly in nucleic acids – DNA and RNA. The body breaks purines down to uric acid, which passes from the body via the urine.

Synovial fluid – the fluid produced within the joint capsule that helps to nourish the cartilage and lubricate the joint.

Urate – a salt of uric acid, which forms as old cells are broken down and foods are digested within the body. It's normally expelled in the urine but can sometimes build up and form crystals that are deposited in the joints or under the skin.

Warfarin – a drug used to prevent blood clots from forming or growing larger. It works by 'thinning' the blood, making it less 'sticky' and reducing the blood's ability to clot.

Where can I find out more?

If you've found this information useful you might be interested in these other titles from our range:

Conditions

- *Calcium crystal diseases (pseudogout)*
- *Osteoarthritis*
- *Osteoarthritis of the knee*

Self-help and daily living

- *Diet and arthritis*
- *Pain and arthritis*

Therapies

- *Complementary and alternative medicine for arthritis*

Drug leaflets

- *Allopurinol*
- *Azathioprine*
- *Non-steroidal anti-inflammatory drugs*
- *Local steroid injections*
- *Steroid tablets*

You can download all of our booklets and leaflets from our website or order them by contacting:

Arthritis Research UK
PO Box 177
Chesterfield
Derbyshire S41 7TQ
Phone: 0300 790 0400
www.arthritisresearchuk.org

Related organisations

The following organisations may be able to provide additional advice and information:

Arthritis Care
18 Stephenson Way
London NW1 2HD
Phone: 020 7380 6500
Helpline: 0808 800 4050
www.arthritiscare.org.uk

British Dietetic Association
5th Floor Charles House
148/9 Great Charles Street Queensway
Birmingham B3 3HT
Phone: 0121 200 8080

NHS alcohol information website
www.nhs.uk/Livewell/alcohol/Pages/Alcoholhome.aspx

UK Gout Society
PO Box 527
London
WC1V 7YP
www.ukgoutsociety.org

Notes

We're here to help

Arthritis Research UK is the charity leading the fight against arthritis.

We're the UK's fourth largest medical research charity and fund scientific and medical research into all types of arthritis and musculoskeletal conditions.

We're working to take the pain away for sufferers with all forms of arthritis and helping people to remain active. We'll do this by funding high-quality research, providing information and campaigning.

Everything we do is underpinned by research.

We publish over 60 information booklets which help people affected by arthritis to understand more about the condition, its treatment, therapies and how to help themselves.

We also produce a range of separate leaflets on many of the drugs used for arthritis and related conditions. We recommend that you read the relevant leaflet for more detailed information about your medication.

Please also let us know if you'd like to receive our quarterly magazine, Arthritis Today, which keeps you up to date with current research and education news, highlighting key projects that we're funding and giving insight into the latest treatment and self-help available.

We often feature case studies and have regular columns for questions and answers, as well as readers' hints and tips for managing arthritis.

Tell us what you think of our booklet

Please send your views to:
feedback@arthritisresearchuk.org
or write to us at:
Arthritis Research UK, PO Box 177, Chesterfield, Derbyshire S41 7TQ.

A team of people contributed to this booklet. The original text was written by Dr Mike Snaith with expertise in the subject. It was assessed at draft stage by consultant rheumatologist Prof. Bhaskar Dasgupta, Prof. Mike Doherty, lead rheumatology educator Kate Gadsby, senior lecturer in rheumatology Dr Ian Giles and honorary lecturer in rheumatology Dr Dorothy Pattison. An **Arthritis Research UK** editor revised the text to make it easy to read, and a non-medical panel, including interested societies, checked it for understanding. An **Arthritis Research UK** medical advisor, Prof. Anisur Rahman, is responsible for the content overall.

Arthritis Research UK

Get involved

You can help to take the pain away from millions of people in the UK by:

- Volunteering
- Supporting our campaigns
- Taking part in a fundraising event
- Making a donation
- Asking your company to support us
- Buying gifts from our catalogue

To get more **actively involved,** please call us **0300 790 0400** or e-mail us at enquiries@arthritisresearchuk.org

Or go to:
www.arthritisresearchuk.org

Providing answers today and tomorrow

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Derbyshire S41 7TD

Tel **0300 790 0400**
calls charged at standard rate

www.arthritisresearchuk.org

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