

Prize cherry for gout treatment

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What is gout?

Men are six times more susceptible to gout than women, and women are very unlikely to develop gout before menopause. The disease is unlikely to strike a man under the age of 30, but it is common in middle-aged and older adults. Genetics probably play a significant role in the disease, but those who consume a purine-rich diet including a lot of red meat, poultry and fish will be more prone, especially if this is accompanied by heavy alcohol consumption and insufficient water intake. Obesity is also a risk factor.

Gout is actually a form of arthritis, hence a sometimes used term, 'gouty arthritis'. The pain in an acute episode or attack is caused by swelling of joints, which become red, swollen and stiff. The most commonly affected joint is the big toe, but ankles, heels, knees, wrists, fingers and elbows can also be affected.

The disease is caused by an excessive concentration of uric acid in the blood (hyperuricemia), uric acid being the final breakdown product following the metabolism of dietary purines. In the vast majority of gout sufferers, the high uric acid concentration is the result of under-excretion of uric acid, rather than over-production¹. Uric acid is normally filtered by the glomeruli of the kidney, resorbed in the proximal tubule and then secreted distally, to be excreted via the urine. In around 98% of patients with primary hyperuricemia and gout, renal management of uric acid has somehow become defective². The high uric acid concentration within the body leads to the formation of angular monosodium urate crystals in the joints, which in turn causes inflammation, so precipitating the gout attack. An episode may typically last from a few days to two weeks, but unless lifestyle changes or treatment regimens are implemented, attacks tend to occur with increasing frequency.

It should be noted that asymptomatic hyperuricemia is common and does not necessarily progress to clinical gout.³

Diagnosis

The principal diagnosis is made by detection of urate crystals in joints, tissues or blood. Hyperuricemia is defined as a serum uric acid concentration above 7 mg per dL (420 µmol per L). This concentration is also the limit of solubility for monosodium urate in plasma. With uric acid concentrations of 8 mg per dL (480 µmol per L) or greater, urate crystals are more likely to form in tissues.

Risk factors

- Alcohol: Heavy alcohol consumption, especially of beer, is related to increased risk, particularly if this is accompanied by drinking insufficient (less than 1.5 litres) water daily.
- Gender: Men are six times more prone to suffer from hyperuricemia, the precursor condition that leads to gout. Women become more susceptible following menopause given that hormonal changes predispose them to hyperuricemia.
- Genetics: Since children of parents with gout have a 20% chance of developing the condition, a genetic association is an associated risk.
- Obesity: Obese people are more likely to develop gout.
- Purine-rich diet: Consumption of a purine-rich diet contributes to elevated uric acid concentrations within the body, especially when accompanied by compromised excretion. Foods such as red meat, organ meat and oily fish (such as salmon, sardines, and herring) are particularly rich in purines.
- Some medications: Certain diuretics, high dose niacin supplements, aspirin (taken in low doses), cyclosporine (e.g. Neoral® or Sandimmune®), and some anti-cancer drugs, have also been associated with gout.⁴

Prevention

As with any disease, prevention is better than cure. Prevention is based on avoiding hyperuricemia. Therefore, the following should be implemented:

- Alkalisating diet: Diet rich in fruits and vegetables, low or free of meat and fish, and low or free of dairy.
- Alcohol consumption: Do not exceed two units per day (16 g pure alcohol).
- Magnesium supplements: Magnesium helps to alkalisate the body. Depending on food intake, 300-600 mg of elemental magnesium daily in a supplement is a useful additional source.
- Avoidance of a purine-rich diet: Avoid high intakes of red meat, poultry, oily fish, dried peas, etc. all known to be rich in purines.
- Weight management: Use nutritional and lifestyle approaches to help reduce weight in case of patients who are obese or even over-weight.

A range of natural treatment options

There are a wide range of botanicals that have been used traditionally to treat gout. None have been subject to intensive research via randomised controlled trials. However, some have strong support among practitioners because of a long folklore of use as well as continued anecdotal evidence of efficacy from a clinical environment. In addition a small number have supporting scientific evidence from clinical trials.

Following is an incomplete list of different natural products with a history of traditional use for treatment of gout:

- Aconite
- Agrimony
- Bilberry
- Bromelain
- Chondroitin sulphate
- Chamomile
- Nux vomica
- Pycnogenol
- Sour cherry
- Water, containing fresh lemon or lime

Cherries rule

It is rare that you encounter any medicine or natural product that yields an intended treatment effect on the vast majority of persons. For example, of 2500 orthodox treatments evaluated by the peer review journal *BMJ Clinical Evidence*, only 13% are rated as beneficial.⁵

One study in particular in the peer review literature points to the importance of cherries as a treatment regimen. However, this study was conducted on sweet, rather than tart or sour cherries.

The paucity of gout treatment specific literature is anomalous given the long history of traditional use, as well as a 1950 scientific review, which promoted daily cherry consumption as means of alleviating gout attacks.⁶

The study at US Department of Agriculture, University of California at Davis, published in 2003, looked at plasma urate levels, antioxidants and inflammatory markers of 10 women consuming two servings (280 g) of sweet cherries following an overnight fast. The results were striking. The USDA team demonstrated clearly that urate excretion increased significantly (from 202 ± 13 to 350 ± 33 $\mu\text{mol}/\text{mmol}$ creatinine after 3 h, $P < 0.01$) when the baseline urate levels were compared against the post-dose levels, while plasma urate levels decreased (from 214 ± 13 to 183 ± 15 $\mu\text{mol}/\text{L}$ 5 h post-dose, $P < 0.05$). The authors of the study concluded: "The decrease in plasma urate after cherry consumption supports the reputed anti-gout efficacy of cherries."⁷

A supporting animal study from China has recently revealed the importance of cherries in reducing inflammation associated with induced arthritis in rats. The study showed not only an increase in antioxidant effects, but also a

reduction in the inflammatory markers TNF-alpha and prostaglandin E2.⁸

However, many practitioners and consumers are only too familiar with the fact that sweet cherries (*Prunus avium* L.) are considerably **less effective** than **tart or sour cherries** (*Prunus cerasus* L.), the latter being considerably **richer in proanthocyanidins** which are likely to be the key components within cherry juice that yield the benefits for gout sufferers.⁹

The **very high antioxidant** capacity of **sour cherries**, such as the **Montmorency variety**, has been confirmed by numerous studies. It has also been shown that the antioxidant capacity of fruit extracts, as measured by ORAC and TEAC assays, does not vary significantly between varieties or cultivars. ORAC values in the order of 2500 µmol TE/100 g fresh weight are typical of high quality extracts.¹⁰

Apart from the specific role of cherries in reducing plasma urate levels, hence lowering the risk of formation of urate crystals responsible for acute gout attacks, cherries have been shown to yield beneficial effects with regard to a range of other inflammatory conditions.^{11,12}

About the Author

Dr Robert Verkerk BSc MSc DIC PhD is an internationally acclaimed expert in sustainability, with 25 years of experience particularly in the areas of agriculture and healthcare. After gaining his MSc and PhD from Imperial College London, he continued his research for a further 7 years as a postdoctoral Research Fellow. In 2002, Dr Verkerk founded the Alliance for Natural Health (ANH), which has become one of the world's leading campaigners promoting the right to natural health. He also acts as a consultant to a wide range of natural health interests around the world through ANH's sister organization, ANH Consultancy Ltd.

CherryActive is made from 100% Montmorency Cherries and is available from:
www.cherryactive.com.my - Tel: 601 6201 3408 - Email: info@cherryactive.com.my

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