

## GOUT

There are 2 reasons why we might want to consider the purine content of the diet. High purines are implicated in the elevated uric acid associated with gout. Also, purine content is a significant factor in dietary choices of Ketogenic and Glucogenic patients.

First consider gout. Contrary to common mythology, gout is not caused by eating too much meat. It is not even caused by eating too much beans. Once a person has gout associated with elevated uric acid, eating a lot of meat or even a moderate amount of beans will exacerbate the gouty arthritic symptoms, but the high purine in the content of the diet was not the cause of gout to start with. One of the most common causes of elevated uric acid is mycotoxins --- mold toxins in the home and work environment. However --- your patients with a Ketogenic Imbalance do not metabolize purines very efficiently, so, they are more likely to develop gout in response to mycotoxins in the environment than are the average patient, and certainly more likely than a patient who is Glucogenic and handles purines quite easily.

Now consider the dietary choices for Glucogenic and Ketogenic patients. Glucogenic patients thrive on a high protein diet, particularly adenine, one of the four purine bases. Ketogenic patients are pushed deeper into a Ketogenic Imbalance by high purine foods. The high adenine for Glucogenic patients and low adenine for Ketogenic patients comes from the original Watson paradigm from which the NUTRI-SPEC Glucogenic/Ketogenic Balance system is derived.

We have had a fair amount of experience with gout, which is a condition that I have always found perplexing. Given the nature of the disease, we might expect that it would have a typical pain pattern associated with it --- either Acid or Alkaline or Anaerobic or Dysaerobic pain pattern --- yet that has never seemed to be the case. We have usually been able to significantly improve the pain associated with the gout but always had to test carefully to find the right NUTRI-SPEC approach. The pain pattern in gout seems to be Anaerobic or Dysaerobic with about equal frequency. One problem is that these patients almost always have an acid urine which leads to a Dysaerobic interpretation of tests, but in many of these cases the saliva pH has been a better indicator than the urine pH (acid saliva = Anaerobic, alkaline saliva = Dysaerobic).

Another interesting note about gout is that the disease involves the abnormal metabolism of xanthines. I always wondered why no one ever recommended avoiding the xanthines in coffee, tea, and chocolate for gout patients. I decided to do that years ago and I cannot offer any objective evidence that it made any difference, but I have always

suspected that having the patients avoid caffeine, theophylline and theobromine may have helped somewhat.

Other essential facts regarding gout:

- Most gout patients are obese
- 52% have Type IV hyperlipidemia
- 36% have hypertension
- (Hyperuricemics without gout do not necessarily show hyperlipidemia and hypertension.)
- Elevated [triglycerides] in gout is associated with fatty infiltration of the liver, but especially in obese subjects.
- Thiazide diuretics precipitate gout attacks by decreasing uric acid excretion.
- High SpGr = nitrogen eliminated as uric acid = causes low UpH  
Low SpGr = nitrogen eliminated as ammonia = causes high UpH  
Normal SpGr = nitrogen eliminated as urea  
Water Restriction = increased purine catabolism = increases urine uric acid = low UpH

All gout patients must strictly follow 3 rules:

- a) If they are on a diuretic they must stop taking it, as diuretics can cause gout, and always exacerbate it.
- b) No fruit or juice.
- c) No fried foods since they convert to pro-inflammatory prostaglandins.

We recommend you follow the Pain Control protocol --- looking for increase/decrease in pain associated with increase/decrease in Urine pH and/or Saliva pH.