

[Musings from the Chiefio](#)

[Oxalate, Gout, Arthritis and Autism](#)

Posted on [29 May 2011](#) by [E.M.Smith](#)

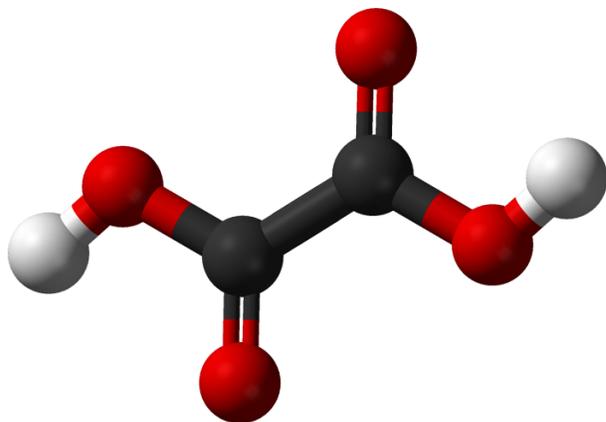
In doing the research on Oxalate and Kidney Stones I ran into several other “odd” connections of oxalate with illness. My overall conclusion is that “this is bad stuff” and generally ought to be avoided.

It has a connection to gout, forming the “seed crystals” on which the gouty deposits form. It has a connection to autism, though more tenuous. It has a connection to arthritis of some sorts; and there are even fungi and plants that use oxalic acid as a form of chemical warfare agent.

So some of the things put here will just be “gee whiz!” pointers to odd things, and some will be a more in depth look.

Oxalic Acid

The basis for all this hoopla is Oxalic Acid. It’s a bit strange, as acids go, in that it’s an ‘edge case’ where things are taken to extremes. The body has a lot of ‘organic acids’ in it. The fatty acids that make up most of our cell wall interiors, for example. There are three fatty acids stuck on a glycerine backbone (so they are called “tri – glycerides”). Fatty acids are a long chain of carbons with hydrogens around it, then at the end it gets a double bonded oxygen stuck to the last carbon, and an “OH” off the end. -COOH is how it is usually written. For Oxalic acid, you take two of these “end bits” and stick them back to back. HOCCOOH or:



Oxalic Acid. Black spheres are Carbon, red Oxygen, white Hydrogen

[Original Image](#)

Now if you are thinking that a small molecule with polar spots like those oxygens and with a couple of hydrogens just stuck out the end might be “reactive”, and that if it has ends that mimic something common in our metabolism it might get sucked into reactions and screw them up; well, you would be very right.

Oxalic Acid is often used as a strong cleaner:

http://en.wikipedia.org/wiki/Oxalic_acid

Oxalic acid’s main applications include cleaning or bleaching, especially for the removal of rust, e.g. Bar Keepers Friend is an example of a household cleaner containing oxalic acid. About 25% of produced oxalic acid is used as a mordant in dyeing processes. It is used in bleaches, especially for pulpwood.

[...]

Oxalic acid is used in the restoration of old wood.

Yup, that stuff you paint on the gray fence to make it look fresher again? Oxalic acid.

Its reducing properties are used in platinotype, the early photographic platinum/palladium printing process. Oxalic acid is also used for cleaning ‘grubbiness’ from dirty leather to get back to the flesh of the leather, before reintroducing preservatives.

Vaporized oxalic acid, or a 3.2% solution of oxalic acid in sugar syrup, is used by some beekeepers as a miticide against the parasitic varroa mite.

Oxalic acid is rubbed onto completed marble sculptures to seal the surface and introduce a shine.

Doesn’t sound exactly like something you would want to eat...

In humans, oxalic acid has an oral LDLo (lowest published lethal dose) of 600 mg/kg

So a 100 kg person (about my size) would take about 60 grams to kill them at the low end.

Oxalates in Foods

From:

<http://en.wikipedia.org/wiki/Oxalate>

Food	Serving size	mg/Oxalate
Beet greens, cooked	1/2 cup	916
Purslane, leaves, cooked	1/2 cup	910
Rhubarb, stewed, no sugar	1/2 cup	860
Spinach, cooked	1/2 cup	750
Beets, cooked	1/2 cup	675

Chard, Swiss, leaves cooked	1/2 cup	660	
Rhubarb, canned	1/2 cup	600	
Spinach, frozen	1/2 cup	600	
Beets, pickled	1/2 cup	500	
Poke Greens, cooked	1/2 cup	476	
Endive, raw	20 long leaves	273	
Cocoa, dry	1/3 cup	254	
Dandelion greens, cooked	1/2 cup	246	
Okra, cooked	8 - 9 pods	146	
Potatoes, sweet, cooked	1/2 cup	141	
Kale, cooked	1/2 cup	125	
Peanuts, raw	1/3 cup (1-3/4 oz)	113	
Turnip greens, cooked	1/2 cup	110	
Chocolate, unsweetened	1 oz.	91	
Parsnips, diced, cooked	1/2 cup	81	
Collard greens, cooked	1/2 cup	74	
Pecans, halves, raw	1/3 cup (1-1/4 oz)	74	
Tea, leaves (4 min. infusion)	1 level tsp in 7 oz water		72
Cereal germ, toasted	1/4 cup	67	
Gooseberries	1/2 cup	66	
Potato, Idaho white, baked	1 medium	64	
Carrots, cooked	1/2 cup	45	
Apple, raw with skin	1 medium	41	
Brussel sprouts, cooked	6 - 8 medium	37	
Strawberries, raw	1/2 cup	35	
Celery, raw	2 stalks	34	
Milk chocolate bar	1 bar (1.02 oz)	34	
Raspberries, black, raw	1/2 cup	33	
Orange, edible portion	1 medium	24	
Green beans, cooked	1/2 cup	23	
Chives, raw, chopped	1 tablespoon	19	
Leeks, raw	1/2 medium	15	
Blackberries, raw	1/2 cup	13	
Concord grapes	1/2 cup	13	
Blueberries, raw	1/2 cup	11	
Currants, red	1/2 cup	11	
Apricots, raw	2 medium	10	

Raspberries, red, raw	1/2 cup	10
Broccoli, cooked	1 large stalk	6
Cranberry juice	1/2 cup (4 oz)	6

So a 1 cup serving of beets, purselane, rhubarb, or even spinach has more than 1 gram of oxalates in it. In this case not all as the free acid, but bound to some other ions. (Often as MgOxalate or CaOxalate, though others also exist. The concept of “soluble oxalate” is important as the MgOxalate and CaOxalate tend to just ‘pass on through’. It is the other oxalates, the soluble ones, that cause the problems).

Also note that the oxalates will have different toxicities than the free acid. So while these are not directly comparable, they do give an “order of magnitude” understanding. At three meals a day, you would get 60 grams of oxalic acid as oxalate in under a month from those high roller foods. Clearly there are opportunities for “problems” as well as clearly there is something protective going on.

Fungi

Here the turf gets very muddy. Some plants use oxalic acid as a defence. Some molds and fungi use it for offense. Clearly it is involved in the battle on both sides.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2634576/>

Oxalic acid is thought to be a key factor of the early pathogenic stage in a wide range of necrotrophic fungi. We have recently published that oxalic acid induces Programmed Cell Death (PCD) in *Arabidopsis thaliana* cells. This cell death results from an early anionic efflux which is a prerequisite for the synthesis of ethylene and the PCD. Complementary experiments have been carried out by using seedlings of *A. thaliana*. The effects of millimolar concentrations of oxalic acid were analysed on *A. thaliana* seedlings. A treatment with a 3 mM oxalic acid solution does not alter the development of the plants but induces the transcription of defence related genes which are anion channel dependant. Moreover, our results suggest that a pre-treatment of the seedlings with oxalic acid is able to confer the resistance of *A. thaliana* against *Sclerotium rolfsii*. Regarding our results, we suggest that **oxalic acid plays two distinct roles, depending on the concentration: a high concentration of oxalic acid induces a large PCD and then contribute to the progression of the fungi. However, at low concentration it is able to induce the establishment of a resistance of the plant against the fungi.**

http://www.redorbit.com/news/science/134512/relationship_between_oxalate_oxalate_oxidase_activity_oxalate_sensitivity_and_white/index.html

Relationship Between Oxalate, Oxalate Oxidase Activity, Oxalate Sensitivity, and White Mold Susceptibility in *Phaseolus Coccineus*

Posted on: Thursday, 10 March 2005, 03:00 CST

ABSTRACT

Chipps, T. J., Gilmore, B., Myers, J. R., and Stotz, H. U. 2005. Relationship between oxalate, oxalate oxidase activity, oxalate sensitivity, and white mold susceptibility in *Phaseolus coccineus*. *Phytopathology* 95:292-299.

Sclerotinia sclerotiorum is a necrotrophic pathogen that devastates the yields of numerous crop species, including beans. The disease in common bean and pea is referred to as white mold. We examined the relationship between oxalate, an established virulence factor of *S. sclerotiorum*, and partial white mold resistance of scarlet runner bean (*Phaseolus coccineus*). *P. coccineus* genotypes PT 255956 ('Mayan White Runner') and PI 535278 (Tars-046A) were more resistant than susceptible 'Wolven Pole'. Sensitivity to oxalate ranked highest for Wolven Pole, lowest for PI 255956, and intermediate for PI 535278. Oxalate concentrations were similar in infected stem tissues of the partially resistant lines and lower than Wolven Pole. Moreover, oxalate oxidase and superoxide dismutase activities were absent in the more resistant lines but induced in Wolven Pole. Collectively, these results suggest that genetic differences in susceptibility to *S. sclerotiorum* among different *P. coccineus* lines are partially dependent on oxalic acid.

Additional keywords: activity gels, oxalate-deficient mutant fungus, *Phaseolus vulgaris*, straw test.

Sclerotinia sclerotiorum (Lib.) de Bary is a necrotrophic pathogen that infects 64 families, 225 genera, and 361 species of plants (32). It causes white mold of common bean (*Phaseolus vulgaris* L.), and in extreme cases, yield losses approach 100% (17). This disease is the most important limitation on common bean production in the United States (30). White mold reduces the size of seeds as well as the number of seeds and pods produced per plant (19).

***S. sclerotiorum* uses a variety of mechanisms to kill plant cells, penetrate plant tissues, and colonize host plants. secretion of oxalate and cell wall degrading enzymes appears to play an important role in pathogenesis (25). Mutants deficient in oxalic acid production provided conclusive proof for a role of this dicarboxylic acid in virulence (16). These types of mutants can partially regain virulence when grown on media containing sodium succinate (16), which is an inducer of oxalic acid production (27). However, the production of oxalate by wild-type *S. sclerotiorum* is still five times higher than in oxalate-deficient mutants grown in medium containing succinate (16).**

So Oxalic acid and Oxalates are important to cellular degradation and damage.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC183509/>

Our results show that all of the brown rot fungi tested produce oxalic acid in liquid as well as in semisolid cultures. *Gloeophyllum trabeum*, which accumulates the lowest amount of oxalic acid during decay of pine holocellulose, showed the highest polysaccharide-depolymerizing activity. Semisolid cultures inoculated with this fungus rapidly converted ¹⁴C-labeled oxalic acid to CO₂ during cellulose depolymerization. The other brown rot fungi also

oxidized ¹⁴C-labeled oxalic acid, although less rapidly. In contrast, semisolid cultures inoculated with the white rot fungus *Coriolus versicolor* did not significantly catabolize the acid and did not depolymerize the holocellulose during decay. **Semisolid cultures of *G. trabeum* amended with desferrioxamine, a specific iron-chelating agent, were unable to lower the degree of polymerization of cellulose or to oxidize ¹⁴C-labeled oxalic acid to the extent or at the rate that control cultures did. These results suggest that both iron and oxalic acid are involved in cellulose depolymerization by brown rot fungi.**

OK, why am I looking at that article? (Other than the neat idea that iron and oxalate are used in the depolymerization of cellulose... that might come in handy as an idea for turning trees into sugar...) It's that iron and oxalate are both seen in Gout. We'll come back to that.

The main “takeaway” here is that this stuff is around, and widely used in nature by plants and fungi. Often for purposes of breaking things down.

<http://www.science.oregonstate.edu/bpp/faculty/stotz/stotz.htm>

Most fungi that attack fruits and vegetables are necrotrophic pathogens that kill plant cells prior to colonization. *Botrytis cinerea*, the causal agent of gray mold, attacks more than 200 plant species, including the genetic model organisms, such as *Arabidopsis* and tomato (*Lycopersicon esculentum*). Three different approaches will be used to enhance resistance of tomato to *B. cinerea*. (1) Utilization of wild tomato relatives to isolate quantitative trait loci that confer resistance or susceptibility to *B. cinerea*. (2) Genomic studies of infection-related changes in fungal and plant gene expression to unravel molecular mechanisms underlying pathogen virulence and host defense. (3) Isolation and characterization of antimicrobial peptides that inhibit fungal growth in vitro.

In addition, the interaction between a closely related fungal pathogen, *Sclerotinia sclerotiorum*, and bean (*Phaseolus vulgaris*) is studied in collaboration with Jim Myers in the Department of Horticulture to find new approaches for enhancing resistance to white mold. **Potential contributions of oxalate and reactive oxygen species to genetic differences in resistance to *S. sclerotiorum* among bean species are emphasized.**

Got that? Oxalate and oxygen species together can make for “issues” in attack by white mold.

<http://ejournal.sinica.edu.tw/bbas/content/1997/2/bot382-01.html>

More recently, tolerance to white mold was discovered in the white bean ExRico 23 in Ontario (Tu and Beversdorf, 1982). **The mechanism of resistance appears to be associated with its tolerance to oxalic acid secreted by the white mold fungus** (Tu, 1985). ExRico 23 was registered for commercial planting in Ontario and has since gained worldwide acceptance as a cultivar and as a main source for genetical resistance in white bean breeding.

Basically, various molds and fungi can use oxalic acid and oxalates (with the right oxidative environment) to attack and break down a bunch of different tissue types and protective membranes.

My hypothesis is that this can happen in animals, too, and that it can happen at low concentrations of oxalic acid or oxalates.

The body will try to defend against that; but sometimes the defense can be discomforting in its own right.

Plant Poisons

<http://emedicine.medscape.com/article/817016-overview>

Plant exposures are some of the most frequent poisonings reported to poison control centers. Exposures to plants containing oxalate crystals, such as Philodendron and Dieffenbachia, are among the most common toxic plant exposures reported in the US.

For the past 200 years, the irritant properties of the Dieffenbachia plant have had various uses, including punishing slaves and treating gout, impotence, and frigidity. Today, plants containing oxalate are admired for their ornamental beauty and found in public places and homes.

The following plants contain oxalates:

- Anthurium (Anthurium species)
- Arum, Araceae (Arisaema species)
- Caladium (Caladium bicolor)
- Calla lily (Zantedeschia species)
- Chinese evergreen (Aglaonema species)
- Dieffenbachia (Dieffenbachia species) (see the image below)
- Jack-in-the pulpit (Arisaema triphyllum) (see the image below)
- Monstera, Ceriman (Monstera deliciosa)
- Nephtytis (Syngonium podophyllum)
- Philodendron (Philodendron species)
- Pothos or Hunter's robe (Epipremnum aureum)
- Skunk cabbage (Symplocarpus foetidus) (see the images below)

Well, perhaps this explains some of the “pain” from having calcium oxalate “kidney stone” fragments left laying about in the kidney after they broke up the stone... If plants are using these crystals to cause pain and discomfort, they are most likely not high on the list of things you want forming inside your body.

http://www.wrongdiagnosis.com/p/plant_poisoning_calcium_oxalate_crystals/symptoms.htm

Calcium oxalate crystals is a chemical found naturally in plants such as dumb cane and rhubarb leaves. The amount of calcium oxalate crystals varies amongst species of plant. **The crystals are quite sharp and abrasive and ingestion of plants containing them can cause abrasive and irritation injuries. Eating large amounts can cause kidney and liver damage and even death**

in serious cases. More detailed information about the symptoms, causes, and treatments of Plant poisoning — Calcium oxalate crystals is available below.

[...]

Corneal abrasion – eye exposure

Tearing eyes – eye exposure

Eye pain – eye exposure

Light sensitivity – eye exposure

Calcium oxalate crystal deposit on cornea – eye exposure

Dysphonia – ingestion exposure

Burning lips – ingestion exposure

Lip irritation – ingestion exposure

Mouth irritation – ingestion exposure

Mouth pain – ingestion exposure

Mouth swelling – ingestion exposure

Vomiting – ingestion exposure

Tongue swelling – ingestion exposure

Diarrhea – ingestion exposure

Digestive upset – high ingestion exposure

So that's what happens if you eat some in a plant...

Arthritis Connections

<http://ndt.oxfordjournals.org/content/23/10/3362.full>

Abstract

We report a case of a woman with secondary oxalosis after jejunoileal bypass surgery for obesity, who presented with oxalate stone disease and renal insufficiency requiring dialysis. Thirty years after surgery, longstanding osteoarticular symptoms were recognized as oxalate arthritis. Eventually, she also developed oxalate vasculitis, which improved with corticoid treatment and intensification of dialysis. Work-up for kidney transplantation **revealed AA amyloidosis on gastric and colonic biopsies**. Since no other cause of chronic inflammation could be identified, **it was concluded that the amyloidosis was secondary to oxalate arthritis and vasculitis**. To our knowledge, this is the first report on this association.

Hmmm... “amyloid” is seen in Alzheimer’s. Perhaps, that, too, could be worth a look. Just a bit further down...

http://www.ehow.com/about_5716642_calcium-oxalate-crystals-joint-pain.html

Calcium Oxalate Crystals & Joint Pain

Calcium oxalate crystals are hard and strong, like bone, arranged in a repeating pattern. The crystals’ sharp edges are abrasive grinding surfaces, which may injure body tissue and cause joint damage and pain. A large calcium crystal deposit can produce a bulging tendon in the arm,

for example, which causes discomfort and pain in the upper arm and shoulder. The inflamed tendon between the bones in the shoulder makes it difficult to raise the arm laterally or outward.

Calcium Crystals in Arthritis

Calcium crystals may be calcium phosphate or calcium oxalate. Calcium crystals embedded in joints usually do not interfere with the working of the joint. The calcium crystals usually become dislodged and disappear.

The rough grinding surfaces of the calcium phosphate or oxalate crystals may inflame tendons in a condition known as calcific tendinitis, or the crystals may cause hot painful swollen “pseudogout.” In pseudogout, calcium crystals form in joints. With gout uric acid crystals form in joints.

Calcific tendinitis usually occurs in the shoulder. Chondrocalcinosis, or calcium crystals in cartilage, tears at the hyaline cartilage at the end of a bone. Calcium phosphate crystals usually cause chondrocalcinosis. Calcium chondrocalcinosis from calcium oxalate is rare and occurs only in dialysis patients.

Chronic calcium crystal arthritis may be more troublesome, painful and severe than most osteoarthritis, but usually there is no difference between osteoarthritis with crystals and osteoarthritis without crystals.

OK, at this point a minor “anecdote”. As the family member had a kidney stone, we were swapping her over to a low oxalate diet. Along the way, this meant that I had a load of canned chile, spinach, beets, etc. on the shelves to “get rid of”. So I started eating more of “all that stuff” while serving the non-oxalate foods to her.

About 2 weeks later, I had a bout of “right shoulder pain”. I’d had little bits before (though, in retrospect, they got worse when my family decided to “go vegetarian” and I was suddenly eating a lot more spinach, beans, whole wheat, chard, ...).

In the last week, I’ve deliberately “cut back” a lot on those foods, even for me, and I’ve added the Magnesium laced water and Ca foods to my dinners (to bind oxalic acid and soluble oxalates into insoluble and take it “on through”...).

Today my shoulder is pain free and some other odd minor joint pains have just gone away. Partly from the Epsom Salts baths, partly from the Dasani Water (and some pseudo-Dasani I’ve made from 1/2 tsp MgSO₄, 1/4 tsp KCl in 10 liters of water.)

Proof? Nope. But a strong suspicion is now raised for me... Is there a case where microscopic CaOxalate crystals might form in joints but not be seen by an arthritis doctor as he’s just not looking on that scale? Given all the OTHER things that use oxalic acid and oxalates as a tissue attack system, is it really a good idea to just ignore oxalate intake even if you “only” have mild arthritis or of a sort the doctor assures you can not be oxalate related?

Given that the whole “joint wearing out” story just seems made up from nothing (I’d have thought the ‘wear’ would have been worse when I was a 20 something and running / skiing / hiking / digging much more...). Given that, I’ve always suspected that perhaps there was a “wearing agent” added with age. A reasonable hypothesis to explore would be that perhaps a microscopic dusting of oxalate is all it would take to cause “degeneration”. It seems to work for molds...

The “downside”? You would need to have creamed spinach instead of plain, have your chard with a glass of Dasani water or chew a tums with dinner. Somehow I’m not seeing a lot of downsides... Oh, and put some whipped cream on the Rhubarb Pie...

Crystal Formation

A specific concentration of calcium oxalate in solution is one condition required for calcium oxalate crystal formation. Inhibitor proteins usually prevent the crystals from developing in blood, urine or soft tissue. Promoters facilitate calcium crystal formation.

Crystals form when the serum, urine or soft tissue balance between inhibitors and promoters is conducive to crystal formation. **Osteoarthritis and aging alter the inhibitor-promoter balance.** An acute attack of inflammation and pain starts in the arm and shoulder within 12 to 36 hours after crystals embed in tissue surrounding the shoulder joint. Pain in the upper arm and shoulder worsen, and you have difficulty raising your arm outward.

Inhibition of Crystal Formation

Kidney stones are one of the most common urinary tract problems. Kidney stones are hard masses of calcium oxalate, calcium phosphate or uric acid crystals. Normal urine is frequently supersaturated with calcium oxalate, but most humans do not form stones.

Uropontin, a protein similar to pontin proteins found in bone, plasma and breast milk, occurs in urine. Uropontin has an inhibitory effect on calcium oxalate crystal growth, which indicates that the pontins have a regulatory role in calcium crystal formation.

Kidney Failure and Arthritis

During chronic renal failure, calcium oxalate crystals are deposited in synovial fluid and cartilage. Under the microscope, the calcium oxalate crystals look like calcium phosphate crystals associated with knee osteoarthritis. Joint disease in chronic renal failure may be the result of calcium oxalate deposits.

OK, again we see the association of oxalate issues with uric acid issues. We also have urine as “frequently supersaturated” with oxalate. If the urine is that concentrated, what are the odds that some is left floating around in the blood as the kidneys are already dumping all they can...

Also note that the place the stuff gets deposited is in the joints. “Synovial fluid and cartilage”. That implies to me that folks in a marginal state (of low but not failed kidney function, or of low

hydration – and most folks are under hydrated; or have excess intake of oxalate) and can easily have some crystal deposition in joints. As this stuff is highly insoluble, once deposited there it will be harder to re-dissolve.

All in all, this is looking to me like it is well worth it to take a lower oxalate meal plan and some added citrate and water in the diet, along with a bump up in the Mg levels (via that Mg rich mineral water with Total Dissolved Solids over 1/2 gram) and adding some dairy or other calcium source to any meal with an oxalate bearing plant in it. I also note that there is specific reference to the lowered level of protective proteins with aging. To me it looks like it all “fits”.

That article goes on to warn that taking a calcium supplement might increase calcium oxalate crystal formation. (Part of why I like the Mg water solution better than eating Tums, but as long as taken WITH the food, the complex of CaOxalate ought not to make it to the blood...).

<http://images.rheumatology.org/viewphoto.php?albumId=75676&imageId=2861681>

Has an interesting connection to “inflammatory bowel disease”:

Extensive soft-tissue calcifications are seen in the hands of this 35-year-old male who presented with progressive stiffness of the hands and localized swelling at the elbows. Aspiration from the deposits at the elbow revealed calcium oxalate crystals. **Calcium oxalate arthritis occurs most commonly in chronic renal failure patients on dialysis but can also complicate inflammatory bowel disease or primary oxalosis.** The arthritis presents as an acute monoarthritis or polyarthritis, and chronic arthritis may develop. The crystals may be either dipyrarnidal or polymorphic chunks or rods.

Gee... bowel disease can make things worse... Haven't we seen a connection between bowel issues (“leaky gut” or “leaky bowel” and folks with Autism? Might there be some connection there too?

Autism

<http://www.sott.net/articles/show/215309-The-Role-of-Oxalates-in-Autism-and-Chronic-Disorders>

The Role of Oxalates in Autism and Chronic Disorders

William Shaw, PhD

The Weston A. Price Foundation for Wise Traditions in Food, Farming, and the Healing Arts
Sun, 19 Sep 2010 13:21 CDT

At this point I do have to point out that the Weston A Price Foundation has had a fair number of rocks tossed at it for being a bit less than stellar in some science done. I can't vouch for the veracity nor for the potential slander of the attackers.

A mummy that had been preserved for a couple of thousand years in the high desert of Chile was discovered upon X-ray examination to have a very large oxalate stone in the kidney, about the

size of a golf ball. The discovery of this ancient sufferer is testimony to the fact that kidney stones and oxalate toxicity have afflicted humans for a very long time.

Oxalates (the salt form of oxalic acid) are extremely painful when deposited in the body. About eighty percent of kidney stones are caused by oxalates and they are by far the most common factor in kidney stone formation. There is also a large degree of genetic variability in the ability to detoxify the chemicals that produce oxalates. Perhaps twenty percent of the population has a genetic variance that increases their likelihood of producing oxalates, even when not consuming a high-oxalate diet.

Stoned

Oxalates can form all throughout the kidney and the urinary tract, and can also form in the ureter as well as in the bladder. These star-shaped crystalline stones cause pain as the pressure in the urinary filtrate builds up, and perhaps also by tearing into the walls of the urinary tract itself.

Some kidney stones acquire a stag horn shape, while some oxalate crystals resemble pieces of coral. The crystals do have a lot of calcium in them just as coral does. Oxalate crystals appear in different colors. Some are black and almost look the color of Indian arrowheads made of obsidian. On page 41 is shown a picture of a kidney with one of the oxalate crystals imbedded in it. You can see that the crystal is very pointed. Some of these have extremely sharp ends that cause severe pain.

[...]

Not Just In Kidneys

Even though **oxalate crystals are most common in the kidney, they also can form in virtually any other tissue in the body**, including the brain and the blood-brain barrier. Oxalate crystals resembling pieces of glass can form in the heart muscle. As the heart muscle contracts, these pieces of oxalate crystals actually tear into the tissue. If these crystals are deposited in skeletal muscle, normal movement and exercise can be very painful. I'm convinced this is also one of the factors responsible for **fibromyalgia**. **Oxalates may also cause thyroid disease as they react in thyroid tissue.**

Oxalate crystals can form in the bone. The oxalate crystals can become so dense they actually push the bone marrow cells out of the bones, leading to severe anemia. Deficiencies of red blood cells as well as white blood cells may result due to the oxalate depositions in the bones. Oxalates can likewise cause osteoporosis. The oxalates form in the bone marrow and alter the structure of the bone matrix so the bone is much weaker and prone to breakage.

Other diseases in which oxalates may play a role include **arthritis, joint pain and interstitial cystitis.**

[...]

A Fungal Origin

An unexpected finding is the fact that oxalate crystals are produced in very high amounts by molds and fungus. **Aspergillus – a common organism that causes infection in humans and also is found in the black fungi that you see in your bathroom – produces oxalates.**

I remember I was in San Juan, Puerto Rico, at the old fort that overlooks the sea. There was a lot of black mold on the walls and I could see stalactites coming down. The stalactites in this case were formed from calcium oxalate. Aspergillus produces these oxalates, and these stones will form any place that has infection by the fungus.

In the case of sinus infection, mold and fungus, not bacteria, are the most common causes of infection. A colleague of mine, who is an eye, nose and throat specialist, X-rayed a patient's sinuses and found large oxalate crystals in her sinuses, which disappeared after anti-fungal treatment.

Large oxalate crystals have also been isolated from the lungs of people who had Aspergillus infection of the lungs. The deposits can also form in the skin where they create black areas and necrotic lesions in people with very high oxalate levels.

Well, all that is sounding pretty darned miserable. It also leads me to think that mold is not my friend ;-)

Oxalates and Autism

I first became interested in this topic because of improvements that were noted in autistic children by the researcher Susan Owens. It was Owens who collected the data showing that many autistic children had frequent urination of small volume and found that the phenomenon was associated with oxalates. She also found that these children often manifested gastrointestinal symptoms such as diarrhea and stomach pain. **They may also have pain in the urinary tract. That pain is relieved when a low oxalate diet is instituted. Owens also found that children had improved cognitive, academic and motor skills once the amount of oxalates in their diets was sharply reduced. The same dietary measures helped reduce pain in their muscles and feet, and also brought about a reduction in abnormal behavior and self-abuse as well.**

Eighty percent of people with genetic diseases that cause them to produce kidney stones die before the age of twenty. These genetic diseases, which belong to a class of disorders called hyperoxaluria, are frequently fatal unless the victim receives both a liver and a kidney transplant. Sometimes even after the transplants people die because the oxalates are deposited in tissues all throughout the body. The oxalates will come out of the bones or the muscles and then form in the transplanted kidneys and still kill the person.

More than a third of children with autism have oxalate values as high as people who have these rare genetic disorders, even though these autistic children do not have the disorders.

The question naturally arose: If they don't have this genetic disorder, why are their oxalates so high?

We correlated the amount of oxalate in autistic children with other biochemical parameters and found there was a high correlation with the sugar arabinose, which is a Candida marker. **It appears that the main reason for the high oxalates in children with autism is because of the Candida problem, which is prevalent in autism. Arabinose is very low in normal children and very high in those with autism. We found in my earliest research that treatment with the anti-fungal drug Nystatin markedly decreased this compound. In addition, autistic symptoms such as hyperactivity, lack of eye contact, and aggressive behavior markedly decreased as well. Because of the dramatic reduction in symptoms, anti-fungal treatment has become one of the most common therapies in autism in the world today.**

Hmmm.... Here we come full circle back to those fungi attacking the host by making oxalic acid. I can't help but wonder if a Calistoga Water with its high TDS would help bind that oxalic acid and make the digestive tract less hospitable to the little buggers... perhaps a "Yogurt and Dasani" lunch is not so "fru fru" after all...

That link goes on to greater depth on fibromyalgia, Zellweger Syndrom, and others.

In Zellweger the high levels of oxalate are found to correspond to mental retardation.

They recommend 300 to 350 mg of Calcium Citrate, per meal, with meals as the best way to bind oxalate and get it out of the body. They also recommend Vit-B6 along with other treatments.

The optimum dosage is approximately 300-350 mg calcium as calcium citrate for a total of 1000 mg (one gram) of calcium a day. If you're taking this you don't need additional sources of calcium. An even better approach would be to use magnesium citrate. The adult dosage is about 300-400 mg a day. Some practitioners recommend up to 1000 mg but many people report problems with diarrhea if they exceed 400 mg. Again, a divided dose would be best, taking the magnesium citrate with each meal.

Some other supplements that can be very useful include probiotics and anti-fungal medication to help to control Candida. The probiotic bacteria have enzymes that break down oxalates.

The amino acid arginine helps to prevent the depositing of oxalates in the tissues. The omega-3 fatty acids and cod liver oil are also very effective in preventing oxalate deposition. The omega-6 fatty acids, mostly from commercial vegetable oils, behave in the reverse, and accelerate the deposition of oxalate.

The supplement that is most helpful is vitamin B6. This costs only pennies a day and is extremely safe. I take 100 mg every single day. I recommend just the pyridoxine form. I know the type called P5P is also used but personally I don't think you get the additional benefit by the P5P.

There are a number of medical tests for oxalate status that we use at Great Plains Laboratory. We have a urine panel to measure oxalates and we can also test for yeast markers. We typically find that where the yeast marker is very high, the oxalate marker is also very high. We also test for vitamin B6.

With these measures, kidney stones are largely preventable. This is good news because oxalate buildup can do a lot of damage.

OK, that's quite a load...

I think that these folks are associated with the Weston Price folks:

<http://www.greatplainslaboratory.com/home/eng/oxalates.asp>

OXALATES CONTROL IS A MAJOR NEW FACTOR IN AUTISM THERAPY

Oxalates: Test Implications
for Yeast & Heavy Metals

By: Dr. William Shaw

[...]

Many parents who told me of adverse vaccine reactions of their children reported that their child was on antibiotics at the time of vaccination. Yeast overgrowth, commonly associated with antibiotic usage, might lead to increased oxalate production and increased combination with mercury, slowing mercury elimination if oxalates were so high that they deposited in the bones with attached mercury. It would be interesting to see if increased elimination of heavy metals occurs after oxalate elimination by antifungal therapy and low oxalate diet. In addition, oxalates from the diet or from yeast/fungus in the gastrointestinal tract bind calcium, magnesium, and zinc, perhaps leading to deficiencies even when dietary sources should be adequate.

I've seen this speculated in other pages as well. Autistics are known to excrete very little mercury. Mercury Oxalate is even less soluble than Calcium Oxalate. So perhaps the basic problem is an oxalate problem and the "mercury" issue is just acting as a "marker" for the presence of excess oxalate? I could easily see a case where a child has a fungal / oxalate problem; a vaccination is given, and all sorts of things precipitate out in "odd places" and doing who knows what.

Add to that the historic connection of "leaky gut" to autism and you have a direct pathway for excess oxalate to enter the body. And, as seen here, oxalic acid is a pretty bad actor...

Oxalates in the urine are much higher in individuals with autism than in normal children (Figure 1). As a matter of fact, 36% of the children on the autistic spectrum had values higher than 90 mmol/mol creatinine, the value consistent with a diagnosis of genetic hyperoxalurias while none of the normal children had values this high. 84% of the children on the autistic

spectrum had oxalate values outside the normal range (mean \pm 2 sd). None of the \pm 2 sd). None of the children on the autistic spectrum had elevations of the other organic acids associated with genetic diseases of oxalate metabolism, indicating that oxalates are high due to external sources.

They have a laundry list of “treatments” that look to me like it needs some evidence of efficacy, but it does look exhaustive:

Use antifungal drugs to reduce yeast and fungi that may be causing high oxalate. Children with autism frequently require years of antifungal treatment. I have noticed that arabinose, a marker used for years for yeast/fungal overgrowth on the organic acid test at The Great Plains Laboratory, is correlated with high amounts of oxalates (Table 2 and Figure 2) and arabinose has been found to be an important fuel for fungal oxalate production (5). Candida organisms have been found surrounding oxalate stones in the kidney (9).

Give supplements of calcium citrate to reduce oxalate absorption from the intestine. Citrate is the preferred calcium form to reduce oxalate because citrate also inhibits oxalate absorption from the intestinal tract. The best way to administer calcium citrate would be to give it with each meal. Children over the age of 2 need about 1000 mg of calcium per day. Of course, calcium supplementation may need to be increased if the child is on a milk-free diet. The most serious error in adopting the gluten-free, casein-free diet is the failure to adequately supplement with calcium.

Try N-Acetyl glucosamine to stimulate the production of the intercellular cement hyaluronic acid to reduce pain caused by oxalates (17).

Give chondroitin sulfate to prevent the formation of calcium oxalate crystals (18).

Vitamin B6 is a cofactor for one of the enzymes that degrade oxalate in the body and has been shown to reduce oxalate production (19).

Increase water intake to help to eliminate oxalates.

Excessive fats in the diet may cause elevated oxalate if the fatty acids are poorly absorbed because of bile salt deficiency. Nonabsorbed free fatty acids bind calcium to form insoluble soaps, reducing calcium ability to bind oxalate and reduce oxalate absorption (20). If taurine is low in the plasma amino acid profile, supplementation with taurine may help stimulate bile salt production (taurocholic acid), leading to better fatty acid absorption and diminished oxalate absorption.

Probiotics may be very helpful in degrading oxalates in the intestine. Individuals with low amounts of oxalate-degrading bacteria are much more susceptible to kidney stones (21). Both *Lactobacillus acidophilus* and *Bifidobacterium lactis* have enzymes that degrade oxalates (22).

Increase intake of essential omega-3 fatty acids, commonly found in fish oil and cod liver oil, which reduces oxalate problems (23). High amounts of the omega-6 fatty acid, arachidonic acid, are associated with increased oxalate problems (24). Meat from grain fed animals is high in arachidonic acid.

Take supplements of vitamin E, selenium, and arginine which have been shown to reduce oxalate damage (25, 26).

Undertake a low oxalate diet. This may be especially important if the individual has had Candida for long periods of time and there is high tissue oxalate buildup. There may be an initial bad reaction lasting several days to a week after starting the diet since oxalates deposited in the bones may begin to be eliminated as oxalates in the diet are reduced.

Evaluate vitamin C intake. Vitamin C can break down to form oxalates. However, in adults, the amount of oxalate formed did not increase until the amount exceeded 4 g of vitamin C per day (27). A large study of more than 85,000 women found no relation between vitamin C intake and kidney stones (28). In addition, an evaluation of 100 children on the autistic spectrum at The Great Plains Laboratory revealed that there was nearly zero correlation between vitamin C and oxalates in the urine (Table 2). Megadoses (more than 100 mg/Kg body weight per day) of vitamin C were shown to markedly reduce autistic symptoms in a double blind placebo controlled study (29) so any restriction of vitamin C needs to be carefully weighed against its significant benefits.

It then goes on for a long ways talking about metabolism and diet and various and sundry other things.

The key takeaway for me was that we again have a correlation of a known “bad actor” with a variety of bad results. That, and they have listed some novel ideas about what might be helpful.

This guy thinks that it is Vitamin K that causes the oxalate problem:

<http://gutresearch.com/v1.html>

In this paper I am proposing that a deficiency in Vitamin K causes unregulated calcium movement and deposition in the body of the autistic child, and that unregulated calcium is a cause of many of the symptoms associated with autism. I am also proposing that a Vitamin K deficiency is the cause of the calcium oxalate crystals found in many autistic children.

Calcium, in tandem with the neurotransmitter glutamate, is essential to the functioning of the excitatory cells of the nervous system: once glutamate opens the neuronal cell's calcium channel, calcium pours into the channel and triggers the neuron to fire. The concentration of glutamate within the nervous system is therefore carefully regulated by the nervous system (specifically the astrocytes, which can be negatively affected by mercury and by neurotoxins produced by Lyme spirochetes) because excess glutamate will keep the calcium channels open, allowing calcium to continue to enter, and excite, the neurons. Dr. Russell Blaylock, among others, has written extensively about the neurotoxicity associated with an excess of glutamate.¹⁶ However, I believe that unregulated calcium may play an unappreciated role in triggering the incessant neuronal firing and resultant cell death that are a hallmark of excess glutamate in the nervous system. If a child is unable to regulate calcium due to a Vitamin K deficiency, that child may display signs of glutamate toxicity and uncontrolled neuronal firing that manifest as the cluster of behavioral disorders called autism.

In the context of “leaky gut” syndrome, one must wonder what happens to oxalate levels and to Vit-K levels under those conditions.

At any rate, there is some indicia of both calcium and oxalic acid levels being “off” in autism and that path likely needs more exploration than I can give it in this posting. That those defects could be exacerbated by any of antibiotic regimen, vaccine and / or mercury injection, and the presence of various fungi just makes for one unholy mess to sort out. My spouse reports that she remembers a “yeast free diet” that was helpful for some of the autistic kids she has taught; so that adds to the evidence for a “fungi and oxalate” link.

I would also note that there is a link to the potential for a loss of a beneficial bacterium from antibiotic use:

Calcium dysregulation appears to play an important role in the development of calcium oxalate deposits in humans, a topic whose relationship to autism is currently being explored by autism researchers. Oxalic acid is an organic dicarboxylic acid produced by plants, sometimes in abundance, in order to manage and store calcium. Oxalic acid can be produced endogenously by humans in situations of deficiency of certain vitamins and it can be produced by various species of fungi including *Aspergillus niger*. Oxalic acid is highly corrosive, with a pH of approximately 1.4-1.6. Much but not all of the oxalic acid in plants is bound to calcium, thereby making it insoluble. **When oxalate containing plants are eaten by humans, the soluble and insoluble oxalates are normally degraded in the GI tract by the anaerobic bacterium *Oxalobacter formigenes*, which is easily destroyed by antibiotics.** Insoluble oxalates that are not degraded in the GI tract tend to pass out in the stool. The soluble oxalate can also bind to calcium consumed in food, thereby becoming insoluble. However, if the soluble oxalate is not either degraded by bacteria or bound to calcium consumed in food, then it can be absorbed through the intestinal membrane. Soluble oxalate, whether absorbed by the digestive tract, produced by the human liver, or produced by infectious fungi, will either bind to calcium and other minerals and become insoluble or will be carried into cells on the same transporters that carry sulfate, bicarbonate, and chloride. (Oxalic acid binds to cations, including calcium, zinc, sodium, potassium, and magnesium.

[...]

c It is worth remembering that most vaccines contain glutamate in various forms. Infants have not developed the enzyme system necessary to handle exogenous loads of glutamate, so the regular injection of glutamate from the many infant vaccines could either initiate or accelerate the process of neuronal hyperexcitation.

d The vaccine preservative thimerosal, if excreted through the baby’s biliary system into the stool, would also have acted as an antibiotic on gastrointestinal flora.

Once again we see issues circling around the impact on the gut lining and to some extent on the population of bacteria and fungi found there.

For now, though, we must move on.

Alzheimer's

OK, I have **absolutely no idea if there is any truth to this at all**; but I'm going to quote it and live by it as a bit of "self confirmation bias":

<http://chemistry.about.com/b/2008/02/10/drinking-beer-to-prevent-alzheimers.htm>

Silicon in silicic acid can be metabolized (as opposed to eating silicon-containing quartz or glass, which won't do you any favors). The silicon affects the bioavailability of aluminum, which is neurotoxic and associated with Alzheimer's disease. A natural source of silicic acid is beer. According to at least one study consumption of "moderately high levels" of beer reduced the absorption of aluminum by the digestive tract and accumulation of aluminum in the brain. It's worth pointing out you might expect better results from bottled beer than beer in a can (though modern cans are coated). I have to wonder how much beer is considered therapeutic versus the amount that kills too many brain cells to confer a benefit. It was an interesting acid fact, anyway.

The referenced article says:

http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=ShowDetailView&TermToSearch=17697731&ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

Abstract

Aluminium (Al), a neurotoxin, has lately been implicated as one of the possible causal factors contributing to Alzheimer's disease. Because silicon (Si) intake can affect the bioavailability of aluminium, the object of the present study was to assess whether moderate beer consumption might, as a source of dietary Si, affect the toxicokinetics of Al and thereby limit that element's neurotoxicity. The results obtained confirmed that at **moderately high levels of beer intake the Si present in the beer was able to reduce Al uptake in the digestive tract and thus was able to slow the accumulation of this metal in the body, brain tissue included**. In consequence, moderate beer consumption, due to its content in bioavailability silicon, possibly affording a protective factor for preventing Alzheimer's disease, could perhaps be taken into account as a component of the dietary habits of the population.

"Moderately high levels of beer intake"... I can work with that ;-)

So now we know that having beer with that Polish with Kraut and Mustard is, like "totally health food" dude!

I feel a need for a therapeutic trip to the baseball park coming on ;-)

What? You think I'm joking?

This link:

<http://www.elements4health.com/the-health-benefits-of-turmeric.html>

Describes how turmeric (the stuff that makes mustard yellow) has various health benefits. It also notes that it can raise the level of oxalic acid in the urine.

Turmeric was used in ancient times on the Indian subcontinent to treat various illnesses such as rheumatism, body ache, skin diseases, intestinal worms, diarrhoea, intermittent fevers, hepatic disorders, biliousness, urinary discharges, dyspepsia, inflammations, constipation, leukoderma, amenorrhoea, and colic.

The health benefits of turmeric include possible cancer prevention, promising Alzheimer's treatment, and powerful anti-inflammatory properties.

[...]

Alzheimer's

Preliminary studies suggest that turmeric has a potential role in the treatment of Alzheimer's, and further studies are underway in this regard.

Alzheimer's involves amyloid beta (Abeta) accumulation, oxidative damage, and inflammation, and risk is arguably reduced with increased antioxidant and anti-inflammatory consumption. Turmeric's phenolic pigment curcumin has potent anti-inflammatory and antioxidant activities and can suppress oxidative damage, inflammation, cognitive deficits, and amyloid accumulation. When fed to aged mice with advanced amyloid accumulation, curcumin labeled plaques and reduced amyloid levels and plaque burden. This data suggests that low dose curcumin effectively disaggregates Abeta as well as prevents fibril and oligomer formation, supporting the rationale for curcumin use in clinical trials preventing or treating Alzheimer's.

Using blood samples from Alzheimer's patients, researchers have found that bisdemethoxycurcumin, the active ingredient of curcuminoids found in turmeric root boosted immune cells called macrophages to clear amyloid beta.

In-vitro studies have found curcuminoids (a mixture of curcumin, bisdemethoxycurcumin and demethoxycurcumin) found in turmeric, to possess acetylcholinesterase (Alzheimer's has been linked to a deficiency in the brain neurotransmitter acetylcholine) inhibitory and memory enhancing activities, demonstrating that curcuminoids mixture might be better than curcumin as a treatment for Alzheimer's.

[...]

The consumption of supplemental doses of turmeric can significantly increase urinary oxalate levels, thereby increasing risk of kidney stone formation in susceptible individuals.

What is unclear is the degree to which the higher oxalate levels are due to more flushing out of existing oxalates vs creation of new ones.

That article goes on to tout the health benefits of turmeric in diseases as wide spread as cancer and being a general anti-inflammatory (so useful for arthritis as well)

In this other link:

<http://www.cancer.org/Treatment/TreatmentsandSideEffects/ComplementaryandAlternativeMedicine/HerbsVitaminsandMinerals/turmeric?sitearea=ETO>

we find that Turmeric not only has a load of anti-cancer properties, but also may have a tendency to reduce the plaques that form in Alzheimer's.

Early research has suggested that curcumin may help lower “bad” cholesterol, reduce inflammation, and help with arthritis symptoms, although more reliable human studies are still needed. Tests of curcumin in HIV disease have been mixed and have generally not shown it to be helpful. In studies of mice, **curcumin appeared to help with blocking the plaques and proteins that cause problems in the brain during Alzheimer's disease.**

[...]

A recent safety study in humans suggested that **curcumin changes metabolism of oxalate**, a substance that can form kidney stones. The researchers urged caution in use of this supplement by people with other conditions that make them susceptible to kidney stones.

Yet it raises urine oxalate levels. So is this because it is making more oxalate or helping the body to dump the load it already has? We don't know... (I did find a guy trying to patent the use of oxalic acid and oxalates as a cure for Alzheimer's and Cancer based on the thesis that it is toxic and kills cancer cells first... but it looked rather hokey to me – involving stories of they guy's dog and candy bars IN the patent application...) At any rate, it looks like there is an involvement of oxalate, but on which side is a big vague...

Gout

There is an interesting connection between oxalates and gout. Not only do calcium oxalate and uric acid both form kidney stones, but both can be found in joints causing joint pain. I have to think there is a similarity of metabolism going on here.

<http://en.wikipedia.org/wiki/Gout>

Gout (also known as podagra when it involves the big toe)[1] is a medical condition usually characterized by recurrent attacks of acute inflammatory arthritis—a red, tender, hot, swollen joint. The metatarsal-phalangeal joint at the base of the big toe is the most commonly affected (~50% of cases). However, it may also present itself as tophi, kidney stones, or urate nephropathy. It is caused by elevated levels of uric acid in the blood which crystallize and are deposited in joints, tendons, and surrounding tissues.

Again we note the arrival of arthritis and kidney stones in the same topic...

For some folks, the cause looks to be a metabolic elevation of the uric acid levels:

Long-standing elevated uric acid levels (hyperuricemia) may result in other symptomatology, including hard, painless deposits of uric acid crystals known as tophi. Extensive tophi may lead

to chronic arthritis due to bone erosion. Elevated levels of uric acid may also lead to crystals precipitating in the kidneys, resulting in stone formation and subsequent urate nephropathy [...]

Cause

Hyperuricemia is the underlying cause of gout. This can occur for a number of reasons, including diet, genetic predisposition, or underexcretion of urate, the salts of uric acid. Renal underexcretion of uric acid is the primary cause of hyperuricemia in about 90% of cases, while overproduction is the cause in less than 10%. About 10% of people with hyperuricemia develop gout at some point in their lifetimes. The risk, however, varies depending on the degree of hyperuricemia. When levels are between 415 and 530 $\mu\text{mol/L}$ (7 and 8.9 mg/dL), the risk is 0.5% per year, while in those with a level greater than 535 $\mu\text{mol/L}$ (9 mg/dL), the risk is 4.5% per year.

We have a connection to diet, but also note that “underexcretion of urate”. So the kidneys are involved in the process as well...

The wiki goes on to cover the connection with eating a lot of meat and seafood, along with some genetic correlation and even the use of diuretics (that would include coffee and tea), aspirin, and even niacin as potential triggers.

But there is an interesting “oddity”. The body normally does not precipitate uric acid. Normally it stays in solution.

The triggers for precipitation of uric acid are not well understood. While it may crystallize at normal levels, it is more likely to do so as levels increase. Other factors believed to be important in triggering an acute episode of arthritis include cool temperatures, rapid changes in uric acid levels, acidosis, articular hydration, and extracellular matrix proteins, such as proteoglycans, collagens, and chondroitin sulfate. The increased precipitation at low temperatures partly explains why the joints in the feet are most commonly affected. Rapid changes in uric acid may occur due to a number of factors, including trauma, surgery, chemotherapy, diuretics, and stopping or starting allopurinol.

[...]

Hyperuricemia is a classic feature of gout; gout occurs, however, nearly half of the time without hyperuricemia, and most people with raised uric acid levels never develop gout. Thus, the diagnostic utility of measuring uric acid level is limited. Hyperuricemia is defined as a plasma urate level greater than 420 $\mu\text{mol/L}$ (7.0 mg/dL) in males and 360 $\mu\text{mol/L}$ (6.0 mg/dL) in females. Other blood tests commonly performed are white blood cell count, electrolytes, renal function, and erythrocyte sedimentation rate (ESR). However, both the white blood cells and ESR may be elevated due to gout in the absence of infection. A white blood cell count as high as $4.0 \times 10^9/\text{L}$ (40,000/mm³) has been documented.

OK... So normally sodium urate stays in the blood and does not crystallize out. Normally you can have a variety of urate levels and not get gout... Perhaps there is a “co factor” that leads to the precipitation?

The wiki on Oxalate says:

In the body, oxalic acid combines with divalent metallic cations such as calcium (Ca^{2+}) and iron(II) (Fe^{2+}) to form crystals of the corresponding oxalates which are then excreted in urine as minute crystals. These oxalates can form larger kidney stones than can obstruct the kidney tubules. An estimated 80% of kidney stones are formed from calcium oxalate. Those with kidney disorders, **gout**, rheumatoid arthritis, or certain forms of chronic vulvar pain (vulvodynia) are **typically advised to avoid foods high in oxalic acid**. Methods to reduce the oxalate content in food are of current interest.

Magnesium (Mg^{2+}) oxalate is 567 times more soluble than calcium oxalate, so the latter is more likely to precipitate out when magnesium levels are low and calcium and oxalate levels are high.

Magnesium oxalate is a million times more soluble than mercury oxalate. Oxalate solubility for other metals decreases in the order $\text{Ca} > \text{Cd} > \text{Zn} > \{\text{Mn, Ni, Fe, Cu}\} > \{\text{As, Sb, Pb}\} > \text{Hg}$. **The highly insoluble iron(II) oxalate appears to play a major role in gout, in the nucleation and growth of the otherwise extremely soluble sodium urate.** This explains why gout usually appears after age 40, when ferritin levels in blood exceed 100 ng/dl. Beer is rich in oxalate and iron, and ethanol increases iron absorption and magnesium elimination, so beer intake greatly increases the risk of a gout attack.

So I must wonder to what extent raising the Magnesium level would help keep that oxalate in solution and prevent a nucleation site from forming... It also looks, at first glance, like a low oxalate diet along with chelating oxalate in the gut via added magnesium and calcium with meals might be helpful in reducing the oxalate load to make those nucleation sites.

None of this would detract from the typical advice to avoid meats, fish, and other urate raising foods, but it would be an adjunct in perhaps removing the trigger that lets that “normally soluble” sodium urate for crystals.

Some less common treatments were listed here:

http://www.lef.org/protocols/immune_connective_joint/gout_01.htm

Cherries and cherry extract

Cherries are rich in antioxidants such as anthocyanins, catechins, chlorogenic acid, flavonol glycosides, and melatonin. Studies have shown that cherry extract can reduce uric acid concentration among women, though the cause of this reduction is unknown (Jacob RA et al 2003). Cherries and their extracts traditionally have been used to treat gout (Fam AG 2005), and one small case series documented decreased duration and severity of gout attacks in three people on cherry-supplemented diets (Blau LW 1950).

Chinese herbs

Certain Chinese medicinal plants were tested for xanthine oxidase inhibitory activity (preventing the conversion of xanthine, a purine metabolite, to uric acid). The most active was the methanol

extract of Chinese cinnamon (*Cinnamomum cassia*), followed by *Chrysanthemum indicum* (Asteraceae) and *Lycopus europaeus* (Labiatae). Among water extracts, the strongest inhibition was observed with *Polygonum cuspidatum* (Polygonaceae), (Kong LD et al 2000). These herbs have been used in China to suppress gout (Kong LD et al 2000).

Vitamin C

In a recent study, the effect of 500 mg of vitamin C daily on serum uric acid levels was compared to placebo in 184 healthy adults. The vitamin C increased the estimated glomerular filtration rate, a measure of kidney function. After two months, the test subjects had reduced serum uric acid compared to controls, suggesting that vitamin C might be beneficial in preventing and managing gout and other urate-related diseases (Huang HY et al 2005).

Grape seed procyanidins

Grape seed procyanidins were found to have uric acid-lowering effects in rats with hyperuricemia. The procyanidin-treated animals exhibited normal growth compared to animals treated with allopurinol, which exhibited some retarded growth (Wang Y et al 2004b).

Because of gout's close association with inflammation, gout patients should also consider Life Extension's anti-inflammatory recommendations. For more information on nutrients and supplements that help combat inflammation, please see the Inflammation protocol.

So it looks like adding some cherries and grapes helps (especially if you swallow some of the grape seeds ;-)) and keep your vitamin C levels high.

http://en.wikipedia.org/wiki/Uric_acid

Has an interesting observation about urate solubility:

Generally, the solubilities of uric acid and its alkali metal and alkaline earth metal salts in water are rather low and all exhibit greater solubility in hot water than cold, allowing for easy recrystallization. **This low solubility is significant in the etiology of gout.** The solubility of the acid and its salts in ethanol is very low or negligible. In ethanol water mixtures, the solubilities are somewhere between the end values for pure ethanol and pure water.

It then has a chart of solubilities. I note that KUrates solubility is roughly double that of NaUrates. This implies that using "no salt" salt with KCl in it instead of NaCl has a chance of improving the overall solubility level of urate in the blood.

Further, this statement:

"In humans, about 70% of daily uric acid disposal occurs via the kidneys, and in 5-25% of humans, impaired renal (kidney) excretion leads to hyperuricemia."

Implies that an added run rate of water through the kidneys ought to "wash out" more of the uric acid / urate from the blood.

Basically, it looks like drinking a load of water (couple of liters) and having some potassium in it would help flush out the urate a bit better.

One could also move to Ashland Oregon, which has “Lithia Park” with Lithia water running in the fountains. Why? Lithium salts of uric acid are fairly soluble:

One treatment for gout, in the 19th century, had been administration of lithium salts; lithium urate is more soluble. Today, inflammation during attacks is more commonly treated with NSAIDs, and urate levels are managed with allopurinol.

There is also the interesting observation that some things can cause abnormally low uric acid levels. Why these are not followed as treatments is an interesting question. And might the inversion of some of these states be involved in high uric acid levels?

Low uric acid

Causes of low uric acid

Low uric acid (hypouricemia) can have numerous causes.

Low dietary zinc intakes cause lower uric acid levels. This effect can be even more pronounced in women taking oral contraceptive medication.

Xanthine oxidase is an Fe-Mo enzyme, so people with Fe deficiency (the most common cause of anemia in young women) or Mo deficiency can experience hypouricemia.

Xanthine oxidase loses its function and gains ascorbase function when some of the Fe atoms in XO are replaced with Cu atoms. Accordingly, people with high Cu/Fe can experience hypouricemia and vitamin C deficiency, resulting in oxidative damage. Since estrogen increases the half life of Cu, women with very high estrogen levels and intense blood loss during menstruation are likely to have a high Cu/Fe and present with hypouricemia.

So it looks to me like having a higher Cu level and a lower Fe or Zn level can cause your uric acid levels to drop. OK. So might not one wish to try dropping the added Iron in the diet and adding some Cu? And maybe avoid zinc supplements?

Zn inhibits Cu absorption, helping to reduce the high Cu/Fe in some people with hypouricemia. Fe supplements can ensure adequate Fe reserves (ferritin above 25 ng/dl), also correcting the high Cu/Fe

OK, so these three interact, too. So drop the Zn levels, the copper ought to rise (and maybe even add some as a supplement).

The Gouty foods list?

In humans, purines are excreted as uric acid. Purines are found in high amounts in animal food products, such as liver and sardines. A moderate amount of purine is also contained in beef, pork,

poultry, fish and seafood, asparagus, cauliflower, spinach, mushrooms, green peas, lentils, dried peas, beans, oatmeal, wheat bran and wheat germ.[

Examples of high purine and Fe sources include: sweetbreads, anchovies, sardines, liver, beef kidneys, brains, meat extracts (e.g., Oxo, Bovril), herring, mackerel, scallops, game meats, and gravy.

Well, that's quite a list to avoid!

Then there is this interesting bit per lead:

<http://www.ncbi.nlm.nih.gov/pubmed/11422760>

Lead chelation therapy and urate excretion in patients with chronic renal diseases and gout.

Lin JL, Yu CC, Lin-Tan DT, Ho HH.

Source

Division of Nephrology and Rheumatology, Department of Internal Medicine, Chang Gung Memorial Hospital, Lin-Kou Medical Center, Chang Gung Medical College and University, Taipei, Taiwan, Republic of China. jllin99@hotmail.com

Abstract

BACKGROUND:

It is known that chronic renal insufficiency (CRI) patients with gout may have subtle lead poisoning. In addition, gout episodes frequently aggravate progressive renal insufficiency because of the use of nephrotoxic drugs and urate deposition. Our study was arranged to evaluate the causal effect of environmental lead exposure on urate excretion in CRI patients.

METHODS:

A cross-section study and a randomized, controlled trial were performed. Initially, 101 patients with CRI and without a history of previous lead exposure received ethylenediaminetetraacetic acid mobilization tests to assess body lead stores (BLS). Then, a clinical trial was performed; 30 CRI patients with gout and high-normal BLS and the changes of urate excretion in these patients were compared before and after lead chelating therapy. The treated group received four-week chelating therapy, and the control group received a placebo therapy.

RESULTS:

The BLS of patients with CRI and gout was higher than that of patients with CRI only, and none had subtle lead poisoning. The BLS, not the blood lead level (BLL), significantly correlated to indices of urate excretion in all CRI patients after related factors were adjusted. **In addition, after lead chelating therapy, urate clearance markedly improved after a reduction of the BLS of patients with CRI and gout** (study group 67.9 +/- 80.0% vs. control group 1.2 +/- 34.0%, P = 0.0056).

CONCLUSION:

Our findings suggest that the chronic low-level environmental lead exposure may interfere with urate excretion of CRI patients. Importantly, the inhibition of urate excretion can be

markedly improved by lead chelating therapies. These data shed light on additional treatment of CRI patients with gout; however, more studies are needed to confirm our findings.

So, if you have kidney insufficiency, perhaps there is some lead exposure in your past...

And maybe “getting the lead out” can help with any gout issues.

I’d also note that heavy metal oxalates are most likely not a good thing in the body and reducing them might just in general be a good thing.

And, finally, this paper has some very good detail on how urate gets generated and moves about. It would be very useful for any person with gout to find out if their gout was due to over production of urate, under excretion of urate, or some of the more exotic causes (such as some tumors listed in this article).

<http://ard.bmj.com/content/65/8/981.full.pdf>

In humans, only 5–10% of the filtered urate is finally excreted, the largest part being reabsorbed at the tubules,⁵ largely at the proximal convoluted tubule. Earlier studies had suggested that the urate is almost fully reabsorbed and that the urate excreted by the kidney is the result of tubular secretion, but more recent data suggest that secretion plays little part, and that excreted urate largely represents the filtered urate which escapes reabsorption

OK, so it gets reabsorbed. But if you have a very excessive flow of water, that reabsorption ought to become relatively hard to do. Frankly, had I gout, I’d try just flushing a couple of liters a day of water laced with KCl and MgSO₄ through and see what happened. I’ve made a “Dasani Water Analog” using 1/2 tsp of MgSO₄ and 1/4 tsp of KCl per 10 L of water. It’s taste isn’t too bad and it does flush a fair amount of water, Mg and K out the kidneys.

Hyperuricaemia and gout also occur as a result of the ingestion of different drugs such as ciclosporin²⁰ or low dose aspirin

So that “baby aspirin” for heart health is likely “right out the window”... as well as avoiding various diuretics

Hyperuricaemia is a widely publicised consequence of diuretic treatment, such that in the current guidelines on the management of hypertension, having gout is considered as a contraindication for the administration of diuretics. Diuretics induce hyperuricaemia by increasing urate reabsorption, though the exact mechanism has not been elucidated. It has been noted that hyperuricaemia occurs when diuretics produce sufficient salt and water loss as to result in volume contraction; this stimulates solute reabsorption at the proximal tubule,^{24 25} and this effect is corrected by administration of the lost fluid.²⁶

So if you take enough diuretics to reduce liquid volumes, the kidneys try to suck the water back in and drag the urate with it. OK, don’t do that. Drink a bucket of water and let it flush instead...

The article then goes on to look at the way some diuretics have different actions than others.

OK, what I'm seeing here is a multifaceted problem. It may be a metabolic issue, or a tumor or even just a "too much rich foods" issue. At the end of the day there is more urate being produced than the kidneys are dumping. Some of it then starts to form crystals and deposits, some times on top of IronOxalate.

I'd get the oxalates down to cut the nucleation sites, raise the water flow to "flush" more and reduce reabsorption, add some KCl (and maybe some lithium if I could find an easy source) and reduce nitrogenous foods (meats / fish). Then get my lead level checked (and perhaps chelated out) and play a bit with the Cu / Zn / Fe ratios.

Looks like a general renal sufficiency measure would be "good to know" along with a bit of testing for any "odd things" like those tumors that can cause the problem. Once you have a handle on where the urate problem comes from (too much rich food and beer; or metabolic problem; or just bum kidneys, perhaps from heavy metal exposure) then you can apply a "fix" for that thing that is better targeted. Given how much oxalates can screw up the kidneys, I'd generally try to get them down in any case.

I have the feeling like there is more to find out about gout. This is a "good start", but the picture is not complete. OTOH, it's midnight, I'm getting "fuzzy", and I ought to go to bed and revisit this tomorrow. So that's what I'm going to do. We'll see if this is in need of more "completion" then. So watch for an "update"...

In Conclusion

There is ample evidence that Oxalate is just not good for people and other living things. This paper even ties Crohn's Disease to higher oxalate levels (with resultant issues):

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1237322/?page=2>

It is only 3 pages long and on page 2 talks about a patient of 6 1/2 years old with arthritis. On the third page (# 317) it says:

Bile salt malabsorption because of ileal disease or ileal resection, or both, is common in Crohn's Disease. This results in increased absorption of Calcium Oxalate

So one could also explore things that modulate the bile salts. In any case, it does look like the gut activity, calcium, oxalate, and arthritis are tied together. The general impact of oxilate is negative for animals. It looks like in addition to being a cause of various pains, it also is the bringer of several diseases, and generally following a lower oxilate diet would be "a good thing". There is one guy talking about a modest potential for a beneficial effect of oxalic acid (as a killer of cancer cells and perhaps helpful for Alzheimer's plaque removal) but those are highly speculative.

In the end, it looks like getting a proper mix of the less thought about metals (Cu, Zn, Mg) would be a good thing, and drinking a lot more water is a very good thing.

Oh, and take your Vitamin B-6

http://www.rejuvenation-science.com/n_magnesium_kidney-stones.html

Magnesium and Vitamin B6 for Kidney Stone Prevention

149 patients with longstanding recurrent idiopathic calcium oxalate and mixed calcium oxalate/calcium phosphate renal stones received 100 mg of magnesium oxide 3 times a day and 10 mg of pyridoxine (vitamin B6) once a day for 4.5 to 6 years. The mean rate of stone formation fell by 92.3%, from 1.3 stones per patient per year prior to the study to 0.10 stones per patient per year during the study. No significant side effects occurred.

So it has a 92% efficacy. If it can do that for other oxalate deposits, too, perhaps it would even help with things like arthritis and gout...

Worth a bit of "research" if nothing else is giving relief...

About E.M.Smith

A technical managerial sort interested in things from Stonehenge to computer science. My present "hot buttons" are the mythology of Climate Change and ancient metrology; but things change...

[View all posts by E.M.Smith](#) →

This entry was posted in [Science Bits](#) and tagged [Gout](#), [Oxalate](#), [Oxalic Acid](#). Bookmark the [permalink](#).

1.  [Verity Jones](#) says:

[29 May 2011 at 11:59 am](#)

Wow E.M. that's a lot to take in. I got about halfway down and lunch beckons so I'll have to come back to it.

One thing though – the aluminium link in Alzheimers has been debunked as a probable artefact of sample preparation (staining). e.g.

<http://www.nature.com/nature/journal/v360/n6399/abs/360065a0.html>

2. [E.M.Smith](#) says:

[29 May 2011 at 6:28 pm](#)

@Verity:

Yup, it's a lot. The more I looked the more I kept finding. But "quantity has a quality all its own" and the simple fact that oxalate keeps popping up in so many different places as a toxic material and / or related to disease processes is "of import"; so the length has that impact.

Per aluminum in Alzheimer's: I'm completely neutral on that one. I'm pretty sure it's junk (as aluminum is just about everywhere and in everything and as folks drink aluminum hydroxide by the gallon as an antacid and we don't have them keeling over with Alzheimer's) **but** I just could not resist that "beer" angle ;-)

I'm just hoping that they have the effect right even if they have the mechanism wrong ;-0

I'm also a bit careful about calling something "debunked" based on one article with a negative finding. Proving a negative? "Damaged" to the thesis, for sure... but what if the aluminum does the "tangle causing" but just doesn't end up deposited IN the plaques? (I.e. what if the plaque is a secondary effect). So I'm "holding neutral" until it is clear what IS the cause.

At any rate, I'd wanted to look at Alzheimer's just to see if there was any known connection to oxalates (positive or negative) and basically got that one guy saying "eat more oxalic acid as a cure". That needed a "mention", but I couldn't bring myself to do it without a bit of "tongue in cheek" about it. (It just looked too nutty, even for me ;-). When the "beer" article popped up, that was it... Nice little "humor angle" to hang the other comment upon... and a justification for "Beer and Sausages" ;-)

The plaques are almost entirely an odd protein deposit (so not a metal issue) anyway. I suspect we'll find it is more like a "prion" as the cause. (Once the badly folded protein form is made, it 'refolds' other normal forms and causes them to deposit on it... repeat until plaque is large). But as it is damn near impossible to get anyone other than a prion specialist to think in terms of "broken protein shape causing disease" I doubt much will come of that muse).

Well, I spoke too soon!

A quick "Bing! Check" of "Alzheimer's prion" turned up several articles... Looks like other folks ARE looking at it.

<http://www.npr.org/templates/story/story.php?storyId=101145687>

In Alzheimer's, prion proteins appear to play a different role, says Stephen Strittmatter, one of the new study's authors and the Vincent Coates Professor of Neurology at Yale University School of Medicine.

Strittmatter says there's no evidence the prion proteins fold into an abnormal shape or actually cause Alzheimer's. Instead, they seem to interact with early stage plaques in the brain in a way that allows those plaques to damage brain cells.

Strittmatter's team made the discovery after looking at hundreds of thousands of molecules that occur naturally in the brain. The prion protein turned out to be the best at interacting with a protein called amyloid-beta, which is what forms the plaques in Alzheimer's

“At first they said, ‘No that can't be,’” Strittmatter says. “It's too bizarre that these two diseases would share this common protein.”

But he says it seemed less strange when they considered that both diseases affect brain cells and cause dementia.

<http://www.nature.com/news/2009/090225/full/news.2009.121.html>

‘Harmless’ prion protein linked to Alzheimer's disease

Non-infectious form of prion protein could cause brain degeneration.

Non-infectious prion proteins found in the brain may contribute to Alzheimer's disease, researchers have found.

The surprising new results, reported this week in Nature¹, show that normal prion proteins produced naturally in the brain interact with the amyloid- β peptides that are hallmarks of Alzheimer's disease. Blocking this interaction in preparations made from mouse brains halted some neurological defects caused by the accumulation of amyloid- β peptide. It was previously thought that only infectious prion proteins, rather than their normal, non-infectious counterparts, played a role in brain degeneration.

The results have yet to be confirmed in humans, but suggest that targeting the non-infectious prion protein (PrP^c) could provide an alternative route to treating Alzheimer's disease. “The need is huge,” says Paul Aisen, an Alzheimer's researcher based at the neurosciences department of the University of California, San Diego. “And it's great news for the field when a new idea is brought forth with strong evidence that can lead to new therapeutic strategies.

So it's not QUITE a “prion disease” but it involves the same prion protein. Just in its normal folding...

Curiouser and curiouser... So what causes it to interact with the other protein in a broken way?... Maybe they need to look for a slicious acid connection and / or have a beer ;-)

3. [E.M.Smith](#) says:

[29 May 2011 at 7:07 pm](#)

Ah! Found an answer on “why is there oxalate in beer” (and what can I do to have my beer and avoid oxalates too...)

From:

<http://answers.yahoo.com/question/index?qid=20070723145527AAvMwah>

Why does a draught beer have an extremely high oxalate level, while a bottled beer has a very low level?

Been diagnosed with calcium-oxalate kidney stones and was researching foods with a high level of oxalate...this one I can't figure out! What is it about a draught that promotes oxalate?

and the “best answer” was:

Hey a great question!

Oh shi_ now I must think.

There really should not be more oxalates in draught than in bottled beer. Here is how I see it.

I actually had to get out my brewing textbook for this one.

Oxalates can form through fermentation when brewers yeast imparts them to the beer. They give problems to the brewer in something called haze (cloudiness). The oxalates are a fraction of haze causing compounds and are the form of micro crystals. **To get clear of the problem brewers have a trick, we “throw a haze” in the beer. This is done so we can filter it out.** So we chill the beer to near freezing temperatures and (calcium oxalates) oxlates form as well as other haze causing compounds. The beer is then filtered and the problem is solved. The filtering also removes solids such as the brewers yeast (more about yeast later).

So, when we buy a beer and chill it (hell we Americans give it a cold shock) the beer will not haze and it looks good to the drinker. Buy removing solids and compounds such as Calcium oxalate stability is increased.

You have a kidney conditions caused by (DRINK MORE WATER-GIRLfriend) oxalates and I am guessing perhaps uric acids also. So you would be concerned about the yeast in beer which is a source of oxalates.

Traditionally, the draught or kegged beers were not filtered or only lightly so . So yeast was passed into the beer in the keg(barrel). This is no longer the

practice so your beers are much lower in oxalates today than in the past. {beers today are highly filtered}

****Exception there are some craft beers which do only light filtrations or produce styles that traditionally have yeast in them i. e. Hefe Weizen.**

If your doctor has not stopped your from drinking beer then any of the large brewers beers should be safe for you. (get a # of how many u r allowed from her)

Good Luck. Good Health. Take Care.

email me if I can be of further help.

-Raidersman :)

Source(s):

Malting and Brewing Science Volume 2

J. S. Houghs, D. E. Briggs, R. Stevens and T. W. Young ISBN 0-412-16590-2

So there you have it. Drink all the cold filtered American P-water beer you want, it doesn't have anything in it anyway; but watch out for those "brewpubs" and Hefeweizen and anything else that looks cloudy or was not filtered. My expectation would be that German and Czech Lager and Pilsner styles ought to be just dandy too.

Or, in short form "have a cold clear one"...



4. [Verity Jones](#) says:

[29 May 2011 at 9:40 pm](#)

Well, keep drinking the beer, providing you don't suffer from gout.

Personally I am so fond of cherries I would happily eat large quantities daily as a preventative even though I probably have no/low risk of gout. Your article reminded me I had one of those wonderful Belgian Cherry Beers in the fridge. I'd almost forgotten about it, but I'm slowly sipping it now – wonderful.

As for aluminium / Alzheimer's – the original finding of Al in brain plaques caused a real health scare that has stayed in the public memory. That Nature paper I linked to was one of several showing that if brain sections were prepared without staining, Al was not present in plaques. Overall no causal link has been proven and this is generally accepted.
http://alzheimers.org.uk/site/scripts/documents_info.php?documentID=99

5. *Judy F.* says:

[29 May 2011 at 10:30 pm](#)

There is Weston A. Price again :)

I have a friend with gout, so I have been carefully reading through your post here. Now, my brain is tired. However, part way through, I started wondering if this in any way related to my fibromyalgia. And there it was. Now I will have to go through the whole thing again and do some thinking. Fibromyalgia is mostly diagnosed by a process of elimination, as opposed to diagnosis based on a test. In my case, the pain and stiffness is extreme a short time after using my muscles. If I mow the lawn, I get very stiff within 15 minutes of finishing mowing. After a few hours, I am less stiff and sore. I have always wondered if it was more of a lactic acid issue, since it comes on suddenly and decreases in a relatively short amount of time. Then I saw your fungal and sugar correlations and now I will have to think some more. The Naturopathic Doctor I see, recommends Calcium Citrate and Magnesium Lactate, as well as Vit D as supplements. I need to study more and see if those are the best formulations.

When I was in college, oh those many years ago, I had a microbiology professor talk about the Scientific Method. He said that all good scientists try to discover why something happens. I suspect that he was talking about correlation and causation. Normally, if “A” happens, and “B” is there when it happens, there is a connection. But then he told a story, which has stayed in my mind ever since. He told us to always keep in mind the Alien. Great, we thought, a professor of microbiology talking about Aliens. The story went like this: Suppose an Alien landed in New York, and walked down the street and got thirsty. He stopped into a bar and was watching and listening to all the people around him. He watched a man order a Whiskey and water, who then proceeded to get noisy, loud and disorderly as he kept drinking whiskey and water. Being an inquisitive alien,(the best kind) the alien went back the next night. He watched as another man ordered a Scotch and water and proceeded to get noisy, loud and disorderly as the night progressed. By this time the alien had an idea in mind, so returned the third night and watched as a patron ordered a Vodka and water. As expected, the third man got noisy, loud and disorderly. So the Alien came to his conclusion, as any good scientist would, that the common ingredient in the drinks each night was the water, therefore, water caused humans to get noisy, loud and disorderly.

There could be many things in the world where we can't see the connection because we are so busy looking for the “water” and don't see what is really causing the problem.

Jduy

6. [E.M.Smith](#) says:

[30 May 2011 at 6:42 am](#)

@Judy F:

It would be fairly easy to “do a test” via the low oxalate diet and see what happens.

As precipitation of things is pH dependent, a lactic acid surge could easily cause other things to shift their solubilities.

FWIW, the “subject” with kidney stones is using commercial bottled Dasani water (that has added Mg in it) along with chewing one “Tums” (that is a load of Ca Carbonate) with each meal that has oxalate foods. (i.e. not bothering at breakfast with scrambled eggs or Rice Crispies in milk...)

Other than that, it’s mostly been to avoid: Spinach, beans (though lentils are OK), beets / chard, and whole wheat while adding more milk, cheese and cream sauces...

So far, so good.

Best of luck and do keep us informed if anything interesting comes of it.

(I’ve a friend with fibromyalgia, so it matters...)

7. [E.M.Smith](#) says:

[30 May 2011 at 11:32 pm](#)

I’ve found a rather remarkably well written discussion of oxalate and the various bad things it can do / cause here:

<http://www.lowoxalate.info/>

One snippet in particular:

Now that we have six years of experience in reducing oxalate in more than 3500 people in the support group associated with this website and in more people indirectly through their physicians, **it seems clear that those who have had gut issues and reduce dietary oxalate are often seeing their gut function improve or normalize as they lose other chronic problems.**

Many who reduce dietary oxalate, but had NO obvious kidney issues, are **seeing major improvements in other chronic conditions**. Scientists must start looking

with fresh eyes at these other conditions and their potential association to oxalate. **So far, our list of conditions that improve includes fibromyalgia, interstitial cystitis, vulvodynia, depression, arthritis, and gut problems of all sorts, as well as autism and many other developmental disorders.** Some of these disorders have clear genetic links, but the gene defects may produce “leaky guts”, or oxalate problems all by themselves without the requirement of inflammation.

Scientists know that issues local to the kidneys raise risks for kidney stones. They also found out that urine oxalate levels in those with kidney stones do not tend to be very different from urine oxalate levels in the rest of us. Even so, reducing dietary oxalate can help reduce stones, especially if other treatments did not help.

Kidneys provide only one site of oxalate secretion, but oxalate is also secreted to skin, to saliva, to mucus in the lungs, and to the gut and stool. The relative importance of these other modes of secretion have not been adequately studied. Risk factors relevant to these other systems may account for why some people with oxalate issues do not have kidney stones or other signs of kidney disease, but may have serious effects in other places from an excess body burden of oxalate.

The body has antioxidant and other protections against oxalate which work when the body burden of oxalate is low and the antioxidant resources high. These systems may be overwhelmed by higher levels of oxalate in the body’s tissues, or when infection (frequent immunization?) overwhelms the body’s antioxidant systems. For this reason, as long as someone continues to consume a high oxalate diet when their gut is “leaky” or when they are sick, then, eventually, in time, the damage may begin to appear as some organs begin to be affected by a buildup of oxalate, or by the disruption of the mineral chemistry that oxalate may inflict.

So it looks like others have been down this path for a while and are seeing positive results in several areas.

As oxalate does nothing good, going on a low oxalate diet has little downside and much upside, IMHO.

That site is well worth a read.