GLAUCOMA

1. The abnormal fluid dynamics of glaucoma appears to be diphasic in nature. In other words, it tends to be associated with either an Anaerobic or a Dysaerobic Imbalance. I have had several glaucoma patients who tested Anaerobic and experienced a "miraculous" drop in eye pressure on taking Oxy Tonic. I tentatively concluded that glaucoma was an Anaerobic condition. (Note that one of the signs of an Anaerobic Imbalance is exophthalmos.) However, subsequent to that I had other Nutri-Spec Doctors find Dysaerobic Imbalances on glaucoma patients and experienced equally fine clinical response using Oxy D-plus. (I have never found a Dysaerobic case myself.)

   I am certain the benefits to glaucoma patients from Oxy D-plus supplementation was from the glycerol component of Oxy D-plus. We have written extensively on the major roll glycerol plays in fluid dynamics. Glycerol relates particularly to movement of water and electrolytes between the three body fluid compartments on a systemic level, and facilitates movement of water and electrolytes through cellular membranes. Decreasing ocular pressure (as in glaucoma) is one of the benefits from glycerol supplementation. So, if you find a glaucoma patient does not test Anaerobic, we suggest you supplement with glycerol, which is your Electro Tonic. Electro Tonic is almost entirely glycerol, and therefore should be much more beneficial then Oxy D-plus.

   SUMMARY: Glaucoma is likely to respond to either Oxy Tonic or Electro Tonic.

2. Revici's input on glaucoma: I remember either in discussion with Revici or in articles he wrote his explicit association between glaucoma and an Anaerobic/Anabolic Imbalance. Regrettably, in his book he does not elaborate. There are 3 places in his book --- pages 110, 226, and 370-371 in which he talks about inducing either exophthalmia or enophthalmia in test animals by subjecting them to Anabolic or Dysaerobic influences, respectively.

   Specifically, after injection of cholesterol or other sterols, or certain noxious substances, or scalding an animal in hot water, or by strong mechanical trauma --- all Anaerobic stressors --- the central nervous system responded with many symptoms --- including exophthalmia. In contrast, injecting animals with free fatty acids or other Dysaerobic/Catabolic substances that push into a Catabolic state of shock induced enophthalmia. ----- Revici saw these same dualistic changes in intraocular pressure in patients who were either chronically Anaerobic or Catabolic.

3. Metformin for glaucoma?

   First --- I state emphatically, that the nasty drugs prescribed for glaucoma should be avoided if at all possible.

   Metformin has very definitely been shown to decrease the risk of glaucoma. There are many mechanisms proposed by which it achieves this risk reduction, and probably more than one of those mechanisms are valid.

   However --- the greatest risk reduction is in Type II diabetics --- a decreased risk of between 20 and 25 percent. I doubt that this risk reduction applies to those who are not Type II diabetic. --- Why do I say that?

   Two of the mechanisms by which Metformin is proposed to decrease development of glaucoma are its inhibiting of glycation --- the advanced glycation end-products typical of diabetics, but not the rest of us. One other mechanism by which glaucoma risk is reduced is by Metformin's activation of AMPK enzyme --- which yields the same benefits as calorie restriction and exercise, and inhibits fat storage and reduces triglyceride synthesis. --- In other words, it inhibits all the nasty metabolic consequences of Type II diabetes (Metabolic Syndrome).

   ----- So, since two of the major and most likely effects of Metformin in decreasing glaucoma risk are mechanisms unique to Type II diabetes, I suspect that the risk reduction for those who are not diabetic is significantly less than the 20% found in those with Insulin Resistance (Metabolic Syndrome).

Other mechanisms proposed for Metformin's benefits on glaucoma include:

- Stimulating autophagy (--- there is an article on your NUTRI-SPEC website on autophagy) --- a good cellular housecleaning.

- Inhibition of excess glial cell activity --- a part of many neurodegenerative diseases.

- Inhibition of NF-kappa-B --- one of the most important pro-inflammatories, and a direct stimulator of pro-inflammatory cytokines, particularly TNF-α.

4. Two of the major provokers of NF-kappa-B are yeast/mold/fungal exposure and UVB irradiation ….

- Fungal cell walls are composed of a glucan that stimulates fibroblast NF-kappa-B nuclear binding activity, along with pro-inflammatory Interleukin-6.

- UVB irradiation activates NF-kappa-B along with inducible nitric oxide synthase and TNF-α, specifically in the cornea, resulting in inflammatory responses and oxidative damage.

5. NF-kappa-B is kept under control reasonably well by ADAPTO-MAX, OXY-MAX, and TAURINE. These adaptogens, and particularily the Taurine also control Inducible Nitric Oxide Synthase. Those supplements, along with Oxygenic A also specifically control TNF-α.

   Also specific for NF-kappa-B inhibition is the probiotic L reuteri found in two of our Immuno-Synbiotic products.

6. Exophthalmia is also typical of Sympathetic Imbalance and hyperthyroid.