GliSODin – its Unique Place in the Clinician’s Toolbox

When we launched GliSODin in Australia in 2006, this unique and unusual product was initially regarded with a mixture of suspicion and intrigue. Practically no-one was talking about Superoxide dismutase, much less Nutrigenomics. Now, close to a decade later, it has become one of the most-respected nutraceuticals available, even though its clinical application is not always well-understood.

The journal Nutrition has just published a Review paper, “Therapeutic value of oral supplementation with melon superoxide dismutase and wheat gliadin combination” which discusses the many aspects of GliSODin’s clinical application.

To fully appreciate this article, it may be important to highlight key aspects of GliSODin’s nutrigenomic mechanism of action. The full paper which can be downloaded free of charge from the link in Reference #1 below addresses the most clinically-relevant aspects of this remarkable nutraceutical.

The Gliadin issue – critical nutrigenomic ‘activator’

One of the greatest objections we hear from clinicians is that GliSODin contains gliadin; in fact, we are often asked why we don’t remove it and use something else! The answer is very simple; the tiny amount of gliadin in each capsule (8.3 mg) is the nutrigenomic activator which acts as the weak stressor needed to ‘switch on’ the cell’s antioxidant and other defences; this is exactly the same mechanism Mother Nature has used for the millions of years animals have existed on this planet!

The gene which codes for the cell’s primary antioxidant enzyme Superoxide dismutase (SOD) is rapidly upregulated in this process. GliSODin acts as a trigger to initiate the process.

GliSODin is known to upregulate:
- SOD (Superoxide dismutase)
- GPx (Glutathione peroxidase)
- Cat (Catalase)
- Interleukin-10 (a key anti-inflammatory cytokine)

Melon Extract + Gliadin

Melon + Gliadin. The data shown in Table 2 above illustrate that when GliSODin (a combination of Extramel melon extract + gliadin) is administered, the plasma antioxidant enzymes increase significantly compared with placebo, close to doubling on average over a 28-day period.

Melon alone (Extramel). When a similar dosing study compared the effect of GliSODin with the melon extract alone (Extramel), there was no significant effect (Table 1). This is because Extramel as a simple melon extract has no ability of its own to upregulate gene expression.
Pharmacokinetic data over 28 days

The graph at right taken from the same paper by Vouldoukis et al. clearly shows that the Extramel does not produce any significant increase in SOD activity and its effect is much the same as for gliadin alone. Both are inert.

GliSODin however which combines both Extramel and gliadin significantly increases the SOD activity. These proof-of-concept findings are confirmed in the clinical trial by Cloarec where GliSODin was shown to reverse intima medial thickening in the carotid arteries of human subjects over a 2-year period.

But what about gluten intolerance?

The 8.3 mg of gliadin per capsule is equivalent to the amount of gluten in a tiny breadcrumb. In non-celiac gluten intolerance, this quantity does not appear to produce adverse effects in patients; in fact, many of those with gut dysbiosis or other inflammation-based intestinal disorders find that their symptoms clear when they take GliSODin at the recommended 2 capsules daily, 12 hours apart.

This anomaly is quite likely related to the increased synthesis of anti-inflammatory Interleukin-10 when taking GliSODin.

SOD at the core of mitochondrial function

Every single breath of oxygen we inhale generates the superoxide radical – now known to be an important signalling molecule to activate cellular defences. Kept under appropriate control, it is an essential part of normal cell function – untethered and it can wreak havoc, setting up chain reactions of damage that can ultimately lead to disease.

The mitochondria are the first organelles to be affected by such damage and the damaging effects can be amplified because the production of energy in the form of ATP can be significantly impaired by excessive superoxide radical.

Because chronic disease is known to be underpinned by oxidative stress and ATP is essential in so many cellular activities, it makes good sense to address superoxide at its origins in the mitochondria.

*Here lies GliSODin’s forte!*

Christine Houghton

*Nutritional Biochemist*

---

